

Dietary calcium intake and lead body burden in U.S. adolescents

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SUMMARY

Lead exposure is a concern for adolescents, partly because rapid growth can change how the body handles lead. Although rodent studies and small-scale human trials suggest that calcium can influence lead absorption and elimination, evidence from large human populations is limited. We hypothesized that adolescents with higher calcium intake would excrete more lead, reflected by higher urinary lead at a given blood lead concentration. To test this, we analyzed data from 1,861 U.S. adolescents aged 12–19 years in the National Health and Nutrition Examination Survey (NHANES), a survey designed to represent the civilian U.S. population, from 2009–2018. We modeled habitual calcium intake with the National Cancer Institute method, and used survey-weighted regression to account for demographic and renal covariates. Calcium intake varied across the study population and was generally lower in some demographics, including lower-income groups. After accounting for blood lead and other covariates, adolescents who met the age-specific Estimated Average Requirement for calcium had about 14% higher urinary lead than those who did not meet this requirement. Taken together, our results indicate that meeting calcium recommendations may be associated with more efficient lead elimination during adolescence, which has implications for how clinicians and researchers interpret lead biomarkers in adolescent groups.

INTRODUCTION

Lead poisoning is a public health concern, especially for children and adolescents whose brains and bodies are still developing (1). Although lead exposure in the U.S.—most often measured using blood lead concentrations—has declined over time, exposure has not been eliminated (2). Legacy sources such as deteriorating lead-based paint, contaminated soil, and aging plumbing are still common. Even at relatively low exposure levels, lead has been linked to negative neurodevelopmental and long-term health outcomes (3, 4). Because sources of lead exposure are unevenly distributed, these effects also disproportionately impact members of disadvantaged groups as well as racial and ethnic minority populations (5–7).

Adolescence is a particularly sensitive period for lead toxicity (2). Rapid growth at this stage increases bone turnover and mineral accumulation, raising the body's demand for calcium, a mineral that competes with lead for absorption and storage in the body (8, 9). Bone is also the primary long-term storage site for lead (2). During adolescence, changes in bone turnover may affect both the amount of lead retained in the body and the amount released into the bloodstream (2, 10).

Lead and calcium are closely linked through shared biological pathways that govern lead absorption, storage, and elimination (**Figure 1**). The two metals overlap in gastrointestinal absorption pathways such that calcium intake can affect how much ingested lead enters the bloodstream (2). Gut absorption of lead decreases when calcium and other mineral intakes are sufficient, likely because these minerals compete for shared intestinal transport pathways, as documented in adults and pregnant women (11, 12). Upon absorption, lead and calcium interact in the circulation process, during renal elimination, and in bone, where lead replaces calcium and increases lead storage when calcium intake is low (8, 9). Notably, many adolescents do not meet recommended calcium intake levels, and lower intake is more common in some demographic groups (13, 14). This combination suggests that diet may influence lead burden during adolescence, with calcium playing a role in lead absorption and retention.

The biology connecting calcium intake with lead retention has been supported by experimental work in rodents and in humans. In human trials, calcium supplementation lowered blood lead in pregnant women and in chronically exposed children (15, 16). However, whether these mechanisms translate into measurable differences in lead burden among adolescents has received limited attention (17). Blood lead is primarily an indicator of exposure, whereas urinary lead reflects renal elimination (2). Yet, few population-based studies have examined both biomarkers together across demographic groups (18, 19).

We hypothesized that adolescents with higher dietary calcium intake eliminate more lead, as reflected by higher urinary lead levels for a given blood lead concentration. To test this hypothesis, we analyzed data from U.S. adolescents aged 12–19 years in the National Health and Nutrition Examination Survey (NHANES). NHANES is a program sponsored by the Centers for Disease Control and Prevention (CDC) to track the health and nutritional status of the U.S. population (20). We evaluated dietary calcium intake alongside paired blood and urinary lead biomarkers to assess whether calcium intake is related to lead handling during adolescence. Consistent with our hypothesis, adolescents who met the calcium recommendation showed higher urinary lead at a given blood lead level after adjustment for other factors. These results may help refine strategies to protect adolescents who are disproportionately exposed to lead.

RESULTS

We first examined trends in lead exposure and dietary calcium intake across NHANES survey cycles from 2009–2018 (**Figure 2**). Over this period, we observed that blood lead concentrations appeared to decline, whereas dietary

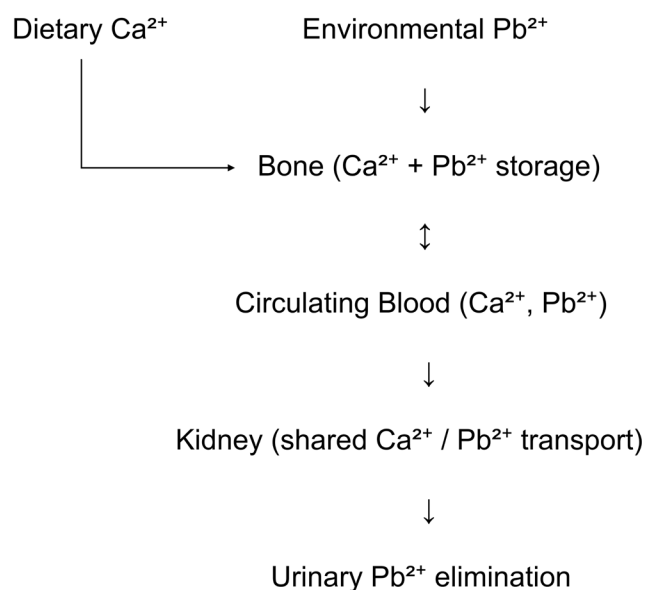


Figure 1: Conceptual framework for calcium–lead interactions in humans. Calcium (Ca²⁺) and lead (Pb²⁺) share pathways involving bone storage and resorption, blood circulation, and renal transport. Because the two ions compete for these shared mechanisms, calcium intake may influence lead retention and urinary elimination, forming the basis of the study hypothesis. The figure was created using Microsoft Word (version 2021).

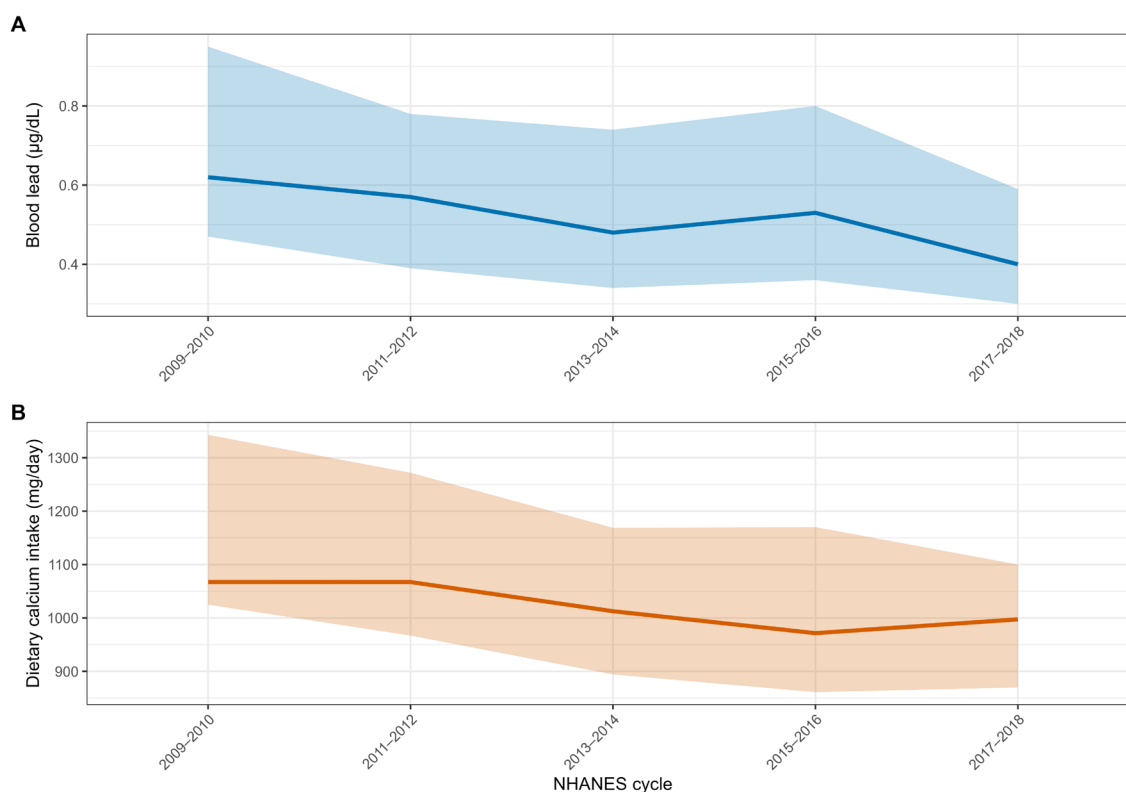


Figure 2: Trends in blood lead and dietary calcium intake across NHANES cycles (2009–2018). Survey-weighted medians and interquartile ranges of blood lead (A) and dietary calcium intake (B) among U.S. adolescents aged 12–19 years ($n = 1,861$). Shaded bands denote interquartile ranges (IQRs). All estimates reflect the complex survey design; values were log-transformed during estimation and converted back to original units for display. Figures were generated in R (version 4.3.1) using ggplot2.

calcium intake changed little. These trends provide context for the analyses that follow, which focus on differences between individuals rather than changes over time.

The Estimated Average Requirement (EAR) is the daily intake level established by the Institute of Medicine that is meant to estimate the nutrient needs of half the healthy individuals in a given population by age and sex group (21). We summarized pooled demographic, dietary, and biomarker characteristics stratified by calcium intake relative to the EAR. These included age, sex, race/ethnicity, household income, BMI, smoking exposure, and kidney function (**Table 1**). Following Dietary Reference Intakes, the EAR threshold was 1,100 mg/day for participants aged 12–18, and 800 mg/day for participants aged 19 years (21). Adolescents who met the EAR consumed about 260 mg/day more calcium than those below the EAR (median 1,190 vs. 933 mg/day, respectively; $p < 0.05$). We observed a modest but statistically significant difference in blood lead concentrations between calcium intake groups, with slightly higher levels among adolescents meeting the EAR (median 0.58 vs. 0.47 $\mu\text{g/dL}$; $p < 0.05$). Urinary lead concentrations were numerically similar and were not significantly different between groups (median 0.26 vs. 0.25 $\mu\text{g/L}$; $p = 0.24$), with substantially overlapping interquartile ranges for both blood and urinary lead (**Table 1**).

Calcium intake also differed across demographic groups. Adolescents who met the EAR were usually male, non-Hispanic White, and from households with higher income, as measured by the poverty-income ratio (PIR). Those below the EAR were more often female or non-Hispanic Black. Being overweight or obese was slightly more common among adolescents below the EAR. In contrast, renal function, assessed by estimated glomerular filtration rate (eGFR), did not differ meaningfully between the two calcium intake groups.

We then examined how dietary calcium intake, blood lead, and urinary lead were related using pooled data from all survey cycles (**Figure 3**). As expected, blood and urinary lead were moderately positively correlated (Spearman's rank correlation coefficient $\rho = 0.47$, $p < 0.01$). This finding is consistent with the fact that urinary lead reflects lead circulating in the blood (2). In contrast, dietary calcium showed weaker correlations with either lead biomarker. Because lead levels and calcium intake varied widely across individuals, we analyzed these factors together using the multivariable regression model to evaluate whether calcium intake was associated with urinary lead after accounting for blood lead.

We examined creatinine (Cr)-adjusted urinary lead ($\mu\text{g/g Cr}$) on the log scale in the multivariable regression model

Characteristics	Total (n = 1,861)	Below EAR (n = 1,125)	At/Above EAR (n = 736)
Medians (IQR)			
Nutritional indicators			
Dietary calcium (total), foods + supplements (mg/day) *	1044 (911–1183)	933 (852–1013)	1190 (1127–1275)
Total daily energy intake (kcal/day) *	1915 (1491–2460)	1779 (1376–2251)	2101 (1672–2710)
Lead indicators			
Urinary lead ($\mu\text{g/L}$)	0.26 (0.14–0.46)	0.25 (0.14–0.45)	0.26 (0.15–0.48)
Blood lead ($\mu\text{g/dL}$) *	0.51 (0.36–0.77)	0.47 (0.33–0.70)	0.58 (0.40–0.83)
Renal measures			
Renal function (eGFR, mL/min/1.73 m ²)	105.0 (91.5–124.0)	105.0 (92.0–122.0)	105.0 (91.1–126.0)
Urinary creatinine (mg/dL)	128.0 (76.5–188.0)	127.0 (76.7–186.0)	131.0 (76.5–192.0)
Percentages in categories (standard error)			
Demographics			
Sex *			
Male	51.8 (1.3)	23.5 (2.4)	85.7 (1.9)
Female	48.2 (1.3)	76.5 (2.3)	14.3 (1.9)
Age (yr) *			
16–19	46.6 (1.4)	43.7 (2.0)	50.2 (2.2)
12–15	53.4 (1.4)	56.3 (2.0)	49.8 (2.2)
Race/ethnicity *			
Non-Hispanic White	50.0 (3.1)	38.2 (3.5)	64.1 (2.9)
Non-Hispanic Black	15.1 (1.7)	23.9 (2.5)	4.5 (1.0)
Mexican Americans	15.9 (1.9)	16.1 (2.7)	15.6 (1.9)
Other races	19.0 (1.6)	21.8 (1.8)	15.8 (1.9)
Body mass index (kg/m ²) *			
Obese/overweight (> 85 th percentile)	35.8 (1.6)	38.5 (2.5)	32.5 (2.5)
Normal (5 th –85 th percentile)	60.8 (1.6)	59.5 (2.4)	62.4 (2.8)
Underweight (< 5 th percentile)	3.4 (0.7)	2.0 (0.7)	5.1 (1.1)
Smoke exposure *			
Yes	12.8 (1.3)	9.8 (1.5)	16.6 (1.7)
No	87.2 (1.3)	90.2 (1.5)	83.4 (1.7)
Poverty-income ratio *			
≥ 1.85	55.1 (1.9)	50.2 (2.4)	61.5 (2.4)
< 1.85	44.9 (1.9)	49.8 (2.4)	38.5 (2.4)

Table 1: Characteristics of U.S. adolescents aged 12–19 years by calcium intake relative to the Estimated Average Requirement (EAR). Continuous values are reported as medians and interquartile ranges. Categorical values are expressed as weighted percentages with standard errors in parentheses. Differences between groups were assessed with Wald F tests (log-transformed continuous measures) and Rao–Scott χ^2 tests (categorical measures). EARs for calcium were set at 1,100 mg/day for ages 12–18 years and 800 mg/day for age 19. An asterisk (*) denotes a statistically significant difference ($p < 0.05$) between the below-EAR and at/above-EAR groups, based on Wald F tests for continuous measures and Rao–Scott χ^2 tests for categorical measures. Abbreviations: eGFR, estimated glomerular filtration rate; IQR, interquartile range; yr, year.

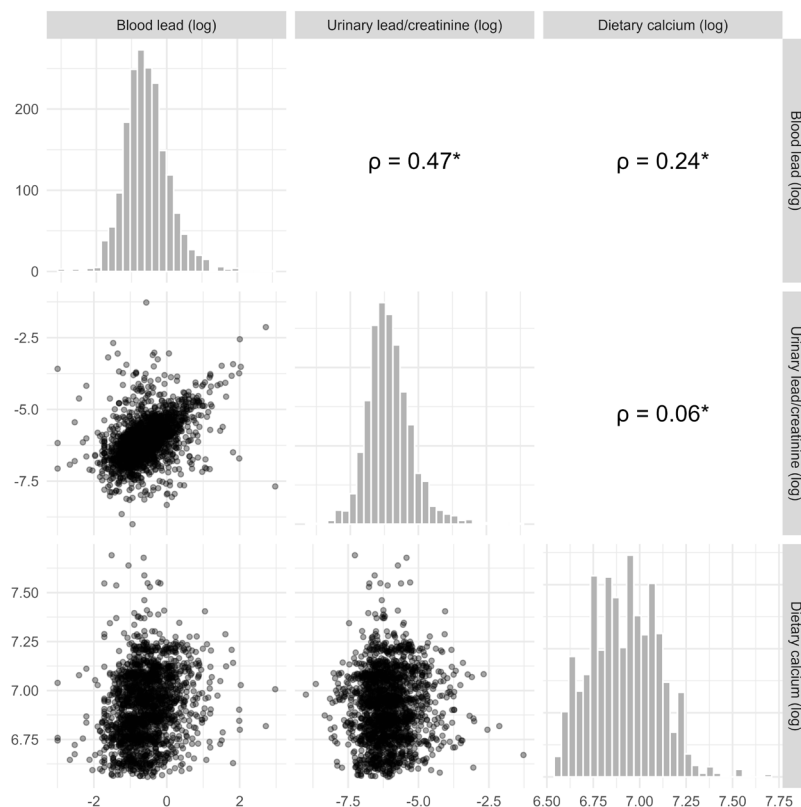


Figure 3: Correlation matrix of lead biomarkers and dietary calcium intake. Pairwise relationships among log-transformed blood lead, creatinine-adjusted urinary lead, and dietary calcium intake in U.S. adolescents (NHANES 2009–2018). Diagonal panels show histograms, lower panels show scatterplots, and upper panels show Spearman's rank correlation coefficients (ρ) with significance indicated as $*p < 0.05$. Figures were generated in R (version 4.3.1) using gplot2.

Outcome variable: Urinary lead ($\mu\text{g/g Cr}$, logged)	β (SE)	p
Dietary calcium intake (Ref: below EAR) *	0.134 (0.063)	0.04
Blood lead ($\mu\text{g/dL}$, logged) *	0.437 (0.120)	< 0.001
Age (yr) *	-0.059 (0.012)	< 0.001
Sex (Ref: Male) *	0.246 (0.068)	< 0.001
Body mass index		0.05
Obese/overweight	-0.058 (0.054)	
Normal	Ref	
Underweight	0.341 (0.166)	
Race/ethnicity *		0.03
Non-Hispanic White	Ref	
Non-Hispanic Black	-0.070 (0.067)	
Mexican Americans	0.174 (0.070)	
Other races	-0.084 (0.062)	
Smoke exposure (Ref: No) *	0.148 (0.066)	0.02
Poverty-income ratio (Ref: ≥ 1.85) *	0.151 (0.069)	0.03
eGFR (Ref: ≥ 90 mL/min/1.73 m ²) *	-0.193 (0.049)	< 0.001

Table 2: Multivariable regression model predicting creatinine-adjusted urinary lead ($\mu\text{g/g Cr}$). Creatinine-adjusted urinary lead and blood lead were log-transformed. The blood lead coefficient represents the percent change in urinary lead for a 1% increase in blood lead. Calcium intake included food and supplement sources with energy intake adjustment. Estimated Average Requirements (EARs) for calcium were 1,100 mg/day for ages 12–18 years and 800 mg/day for age 19 years. Survey-weighted Wald F tests were used to derive p-values. An asterisk (*) denotes statistical significance at $p < 0.05$. Abbreviations: Cr, creatinine; EAR, Estimated Average Requirement; eGFR, estimated glomerular filtration rate (categorized as ≥ 90 vs. < 90 mL/min/1.73 m²); Ref, reference group; SE, standard error; β , regression coefficient.

(Table 2). For categorical predictors, beta coefficients (β) represent fold differences in urinary lead compared with a reference group (the categories marked 'Ref'; the calcium reference is the below-EAR group). For predictors that were also log-transformed, such as blood lead, coefficients indicate the percentage shift in urinary lead corresponding to a 1% increment in the predictor. As expected, blood lead was a strong positive predictor of urinary lead ($\beta = 0.437$, standard error [SE] = 0.120, $p < 0.001$). Calcium intake, modeled as meeting the EAR, showed a modest but consistent association with urinary lead after adjusting for demographic and kidney-related factors. For example, a coefficient of 0.134 (SE = 0.063) corresponds to about 14% higher urinary lead among adolescents meeting the EAR compared with those below it ($e^{0.134} \approx 1.14$) (Table 2). The results were similar in sensitivity analyses using raw urinary lead ($\mu\text{g/L}$) with urinary creatinine included as a separate covariate (data not shown).

DISCUSSION

In this population-based study, we examined whether dietary calcium intake is associated with lead handling during adolescence. Using NHANES data from 2009–2018, we found a moderate association between blood lead and urinary lead. At a given blood lead concentration, adolescents who met the age-specific EAR for calcium had approximately 14% higher creatinine-adjusted urinary lead than those with lower than the recommended calcium intake.

These results are consistent with existing evidence on

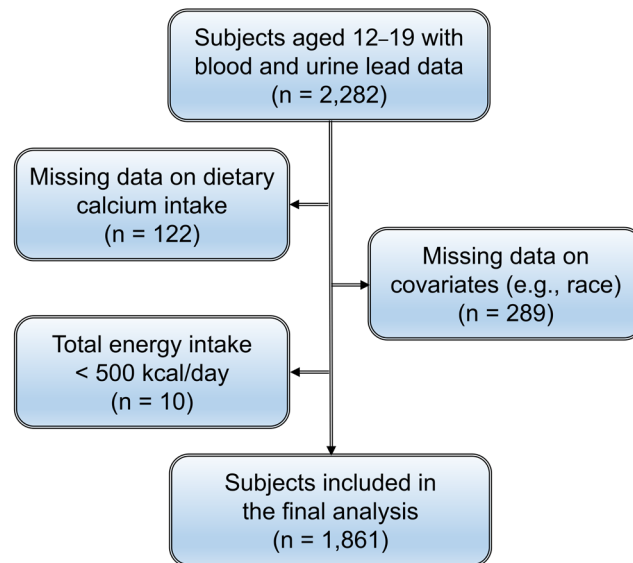


Figure 4: Flow diagram of participant selection for adolescents aged 12–19 years from NHANES 2009–2018. Participants were dropped for missing calcium intake data, incomplete covariate information, or energy intake below the plausibility threshold, yielding a final sample of 1,861. The figure was created using Microsoft PowerPoint (version 2021).

the biological relationship between calcium and lead (22, 23). Because the two elements share biological pathways involved in intestinal absorption and bone storage, calcium intake may influence how much lead is absorbed and retained, particularly during adolescence when bone remodeling is active (8, 9). Our findings, though, are more about how lead leaves the body than how it is taken in or stored. Because lead and calcium share transport in the kidney, enough calcium may help move more lead into the urine, which is a route studied less than gut absorption (**Figure 1**) (8, 9). The association we observed was modest, but it remains biologically plausible given the relatively low lead exposures in this population. Although exposure has declined over the years of the NHANES study, no level of lead in the body is safe, and its burden still falls most heavily on low-income and racial and ethnic minority communities (**Figure 2**) (1, 4).

We also found that urinary lead varied with age, kidney function, and race/ethnicity, consistent with established kinetic analyses of how lead is distributed and cleared in the body (10, 24). Of these, kidney function is the most direct. Adolescents with lower eGFR excreted less lead, which fits the idea that urinary lead tracks how much the kidney filters. Calcium intake also differed by income and race/ethnicity, with lower intake seen among adolescents from lower-income households and some racial and ethnic minority groups (13, 14). These findings suggest that the pattern we observed is shaped by more than calcium intake alone. The unadjusted comparisons illustrate this finding (**Table 1**). Adolescents meeting the EAR had slightly higher blood lead than those below it. This finding likely reflects the demographic differences described above rather than an effect of calcium, and adjusting for these differences isolates the association with calcium intake.

Several limitations of our study should be noted. The cross-sectional design precludes causal inference. Both blood and urinary lead reflect dynamic processes influenced by recent exposure, bone turnover, and kidney function (10,

24). We estimated calcium intake from dietary recalls, which may be misclassified despite the use of established methods (25). We used dietary calcium rather than serum calcium because intake more directly reflects the absorption and excretion processes examined, and serum calcium reflects short-term status compared to dietary intake. Although the directed acyclic graph (DAG) analysis identified a minimal set of covariates for adjustment, residual confounding from unmeasured dietary or environmental factors is still possible. Finally, because calcium intake differed by sex in this sample, we included sex in all adjusted models to reduce potential confounding, although some residual sex-related differences may remain.

In summary, lead exposure and calcium insufficiency are both common among U.S. adolescents (13, 14). Our findings suggest that adolescents with lower calcium intake may excrete less lead in urine relative to blood lead levels. These results extend the rodent and small-trial evidence to a nationally representative sample. If confirmed in longitudinal or interventional studies, calcium status could be relevant to the interpretation of lead biomarkers and to dietary guidance for lead-exposed youth.

MATERIALS AND METHODS

Study population and data source

Data used for the study were sourced from NHANES 2009–2018, conducted by the National Center for Health Statistics of the CDC (20). The NHANES sampling framework employs a stratified, multistage probability design intended to capture the health characteristics of the U.S. civilian, noninstitutionalized population. The primary focus of the study was on adolescents aged 12–19 years who had available measurements of blood lead, urinary lead, and dietary calcium intake. Participants were excluded if they were missing calcium intake data or key demographic information or if they had total energy intake below 500 kcal/day, which is a threshold used to flag implausible dietary reports (26). After

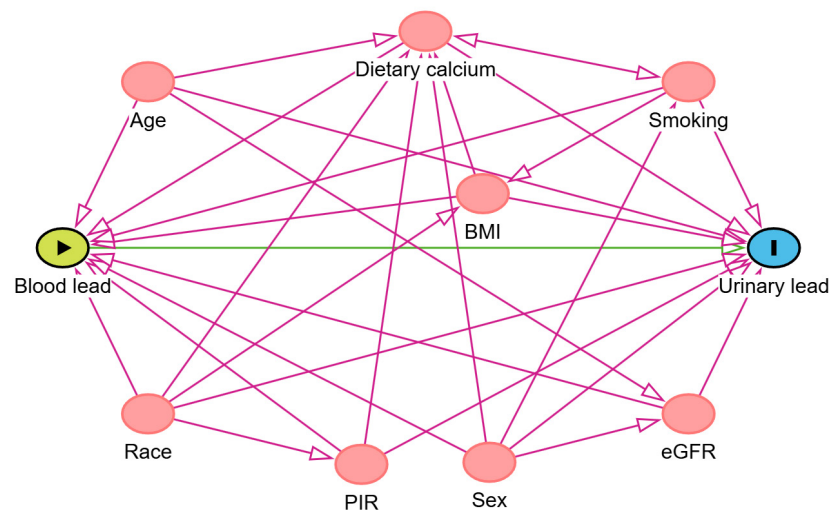


Figure 5: Directed acyclic graph (DAG) showing the framework used to guide covariate selection. Green arrows indicate the primary pathway linking blood lead and urinary lead and pink arrows show other links among covariates that may confound or influence this relationship. The graph shows how dietary calcium may influence both lead biomarkers and how it connects to age, body mass index (BMI), sex, race/ethnicity, socioeconomic status (PIR), smoking status, and kidney function (eGFR). The graph was generated using DAGitty (<http://www.dagitty.net/dags.html>).

these exclusions, the final analytic sample consisted of 1,861 adolescents (**Figure 4**). All NHANES data collection and laboratory procedures followed standardized CDC protocols, with appropriate ethical approval and informed consent.

Lead biomarker measurements

Lead in blood and urine was quantified via inductively coupled plasma–mass spectrometry (ICP-MS) across the five NHANES cycles included in this study. Blood lead was measured in all participants, with a detection limit of approximately 0.25 $\mu\text{g}/\text{dL}$. Urinary lead was measured using the same analytical method, with detection limits improving across survey cycles (from 0.10 $\mu\text{g}/\text{L}$ in 2009–2010 to 0.03 $\mu\text{g}/\text{L}$ in 2017–2018). To account for variation in urine dilution between individuals, urinary lead concentrations were adjusted for creatinine and expressed as $\mu\text{g}/\text{g}$ creatinine. Nearly all blood lead and most urinary lead measurements were above the detection limit. Values falling below the detection limit were assigned a substituted concentration of the limit of detection (LOD) divided by the square root of two, consistent with established NHANES analytic practice.

Assessment of calcium intake

Dietary calcium intake was estimated from two 24-hour dietary recalls—one administered in person during the NHANES examination and a second conducted by telephone. Supplement-derived calcium was incorporated using responses from the 30-day dietary supplement questionnaire. To estimate habitual intake, the National Cancer Institute method was applied, which accounts for random within-person variability across recall days (25, 27). This approach yields a more stable estimate of longer-term dietary patterns than a single recall alone (25, 27).

Covariates

Demographic and health-related covariates were selected based on prior literature linking them to lead exposure or lead clearance. These covariates included age, sex, and race/ethnicity. The poverty-income ratio (PIR) was used as a proxy for household socioeconomic position. Body mass index (BMI) percentile was included to reflect differences in body composition and developmental stage. Serum cotinine was used to capture tobacco smoke exposure, and eGFR was included as an indicator of renal function. A DAG was constructed, further informing covariate selection by identifying the smallest set of variables needed to control for confounding pathways (**Figure 5**) (28–30).

Statistical analysis

Dietary calcium intake was evaluated relative to age-specific EARs to place intake in a nutritional context. Based on Institute of Medicine guidelines, the EAR threshold for calcium was defined as 1,100 mg/day for ages 12–18 and 800 mg/day for age 19 (21). Modeled calcium intake from both foods and supplements was compared with these thresholds, and participants were classified as either below or at/above the EAR. Demographic, dietary, and laboratory characteristics were summarized by calcium intake category. Continuous variables were assessed for skewness and log-transformed when appropriate. Survey-weighted Rao–Scott χ^2 tests were used to compare categorical variables, and Wald F tests were used for continuous variables (31, 32). Every analysis incorporated the NHANES sample weights, stratification variables, and primary sampling units to produce nationally representative estimates.

The primary outcome was creatinine-adjusted urinary lead, expressed as $\mu\text{g}/\text{g}$ creatinine ($\mu\text{g}/\text{g}$ Cr), and analyzed on the log scale. Blood lead was also log-transformed. Survey-weighted linear regression was used to model

urinary lead as a function of dietary calcium intake. Models controlled for blood lead and for covariates identified through the DAG. Total energy intake was also included to account for overall dietary quantity. Calcium intake was examined as a categorical variable based on EAR status. A *p*-value threshold of 0.05 was used to evaluate statistical significance. Analyses were performed in R (version 4.3.1; survey package) and independently verified using SUDAAN 10.0.1 (Research Triangle Institute) (33, 34).

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