MEDDOCS Open Access Publisher

ISSN: 2639-9210

Annals of Dentistry and Oral Health

Open Access | Research Article

The Association Between High-Sensitivity C-Reactive Protein (hs-CRP) and Root Caries: NHANES Analysis of Systemic Inflammation and Dental Disease

Edmond Kubi Appiah, BSc, BDS, MPH*; Joseph Nimako-Boateng, BDS, MSc, DDPH (RCS, Eng)

Drexel University, Dornsife School of Public Health, Philadelphia, United States of America, University of Ghana Dental School, Ghana.

***Corresponding Author(s): Edmond Kubi Appiah**

Drexel University, Dornsife School of Public Health, Philadelphia, United States of America. University of Ghana Dental School, *Ghana*.

Email: eka44@drexel.edu

Received: Oct 13, 2025

Accepted: Oct 28, 2025

Published Online: Nov 04, 2025

Journal: Annals of Dentistry and Oral Health

Publisher: MedDocs Publishers LLC

Online edition: http://meddocsonline.org/

Keywords: C-reactive protein; Root caries; NHANES; Oral health disparities; Iinflammation.

Abstract

Background: Systemic inflammation, as indicated by elevated high-sensitivity C-reactive protein (hs-CRP), may also be involved in the development of oral conditions such as root caries. This study examines the relationship between CRP level and the prevalence of root caries while adjusting for sociodemographic and clinical factors.

Methods: We analyzed data from 3,342 adults in the 2017–2018 National Health and Nutrition Examination Survey (NHANES). Root caries status (present/absent) and CRP categories (low, intermediate, high risk) were assessed.

Results: The prevalence of root caries was 13.1% (weighted). Participants with root caries had significantly higher mean hs-CRP levels (4.40 mg/L vs. 3.26 mg/L, *p*=0.002) and higher rates of diabetes, smoking, lower education, and lower income. In the fully adjusted model, individuals in the high CRP risk category had lower odds of being caries-free compared to those in the low-risk category (aOR=0.59, 95% CI: 0.35–1.01). Additional predictors of root caries included Hispanic ethnicity (aOR=1.57), Black race (aOR=0.65), lower educational attainment (e.g., <High School: aOR=0.18), and current smoking (aOR=0.26).

Conclusion: Higher CRP levels have also been associated with an increased risk of root caries, suggesting systemic inflammation may be implicated in oral disease. These findings highlight the significance of holistic medical-dental care in preventing inflammation and chronic disease as part of oral health promotion among socially disadvantaged populations.

Introduction

Root Caries (RC) is a growing public health concern, particularly among aging populations [1], with approximately 1 in 6 older Americans affected [2]. If left untreated, root caries can lead to serious complications, including inflammation of the periodontium, infections, and abscess formation [18]. Although RC is preventable, it remains a major contributor to diminished oral health-related quality of life, potentially leading to tooth loss and negatively impacting overall well-being in adults [19]. Root caries is most commonly defined as a soft, irregularly shaped, dark-colored, progressive lesion that is either confined

Cite this article: Appiah EK, Boateng JN. The Association Between High-Sensitivity C-Reactive Protein (hs-CRP) and Root Caries: NHANES Analysis of Systemic Inflammation and Dental Disease. Ann Dent Oral Health. 2025; 8(2): 1060.

to the root surface or extends to undermine the enamel at the cementoenamel junction, though clinical evidence indicates it originates on the root surface [3].

Several risk factors, including high sugar intake, decreased salivary flow, and poor oral hygiene, are well-documented to contribute to the development of both coronal and root caries [4]. Lifestyle factors such as tobacco use are also significantly associated with the occurrence of root caries lesions [17]. Emerging evidence suggests that systemic inflammation may also contribute to the progression of root caries [5].

C-Reactive Protein (CRP) is an acute-phase protein synthesized by the liver in response to inflammation in the body [6,7]. Its production is regulated by pro-inflammatory cytokines such as Interleukin-6 (IL-6), Interleukin-1β (IL-1β), and Tumor Necrosis Factor-α (TNF-α) [14,15]. High-sensitivity C-reactive protein (hs-CRP) is a more sensitive form of standard CRP tests, capable of detecting subtle changes in CRP levels within the normal range that would typically go unnoticed by conventional tests [6,8]. Hs-CRP is considered a biomarker of systemic inflammation [16] and has been linked to cardiovascular diseases [9,10,11] diabetes [12] and periodontal disease [13]. Elevated hs-CRP levels reflect an upregulated inflammatory state, which may influence oral health by altering immune responses, impairing tissue repair, or exacerbating periodontal breakdown—key factors in root surface exposure and subsequent caries development.

Recent studies have explored the associations between systemic inflammation and oral diseases, but very few have specifically examined the relationship between hs-CRP and root caries. Given that systemic inflammation can contribute to salivary dysfunction, altered oral microbiota [20,21], and impaired remineralization capacity, hs-CRP may serve as a biomarker for identifying individuals at elevated risk for RC.

This study investigated the relationship between High-Sensitivity C-Reactive Protein (hs-CRP) and root caries among U.S. adults using data from the 2017-2018 NHANES. The aim of the present study was to determine whether adults with elevated systemic inflammation (hs-CRP ≥3.0 mg/L) had greater odds of root caries compared to those with lower inflammation levels (hs-CRP <3.0 mg/L). Root caries was assessed through standardized clinical examinations conducted by trained dentists, with presence defined as any detectable cavitation or softened area on exposed root surfaces.

Materials and methods

Study design

This study employed a cross-sectional design to investigate the association between High-Sensitivity C-Reactive Protein (hs-CRP), a biomarker of systemic inflammation, and root caries in U.S. adults. A cross-sectional approach was suitable for assessing the relationship between inflammation and dental disease at a single time point, allowing for an evaluation of how systemic inflammation may influence oral health across different demographic groups, including age, gender, race/ethnicity, and socioeconomic status.

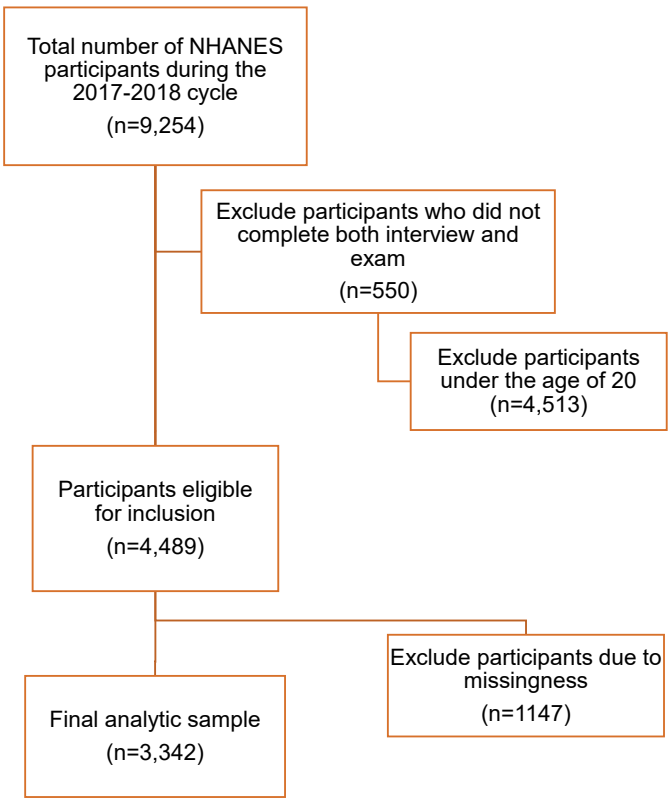
Data source

Data were obtained from the National Health and Nutrition Examination Survey (NHANES) 2017–2018. NHANES is a nationally representative survey conducted by the National Center for Health Statistics (NCHS) at the Centers for Disease Control and Prevention (CDC) [22]. The survey collects comprehensive health data through interviews, physical examinations, and laboratory tests, including blood samples for hs-CRP and dental examinations for caries assessment. NHANES uses a multistage, stratified probability sampling design, oversampling certain populations (e.g., older adults, racial/ethnic minorities) to ensure robust statistical estimates [23,24]. All participants provided written informed consent at the time of recruitment [25].

Study population

The current analysis utilized data from the 2017-2018 cycle. Individuals were eligible for inclusion if they completed both the interview and examination. Participants under the age of 30 were excluded from this analysis (n=4513). A total of 4,489 participants were eligible for the study.

The final analytic sample comprised participants with information on the outcome (RC), exposure (hs-CRP), and all covariates (age, gender, race, income, education, diabetes, and smoking status). Of the 4,489 participants who were eligible for the study, 1,147 were further excluded due to missingness. The final analytic sample consisted of 3,342 (STROBE DIAGRAM) (Figure 1) participants aged 30 and above who completed the interview and examination and provided information on all the analytic variables.



Strobe diagram

Measures

Outcome variable: root caries:

The outcome variable for this study was the presence of root caries, assessed at the subject level using data from the NHANES 2017–2018 Oral Health Examination. Root caries was evaluated only in participants aged 18 years and older and only on teeth exhibiting gingival recession, which was defined as apical displacement of the gingival margin exposing the root surface. Trained dental examiners conducted a standardized full-mouth visual-tactile assessment using a surface-reflecting mirror and a No. 23 explorer. The examination followed a spe-

cific sequence beginning in the maxillary right quadrant and proceeding clockwise through all four quadrants. Teeth were examined for exposed root surfaces, and any areas suggestive of root caries were evaluated for texture, discoloration, and tactile softness. Only visible and accessible root surfaces were included, with subgingival inspection avoided to reduce the risk of bleeding and because root caries rarely occur entirely subgingivally. NHANES classified root caries at the participant level using "whole mouth" scoring codes: Code 1: Root caries detected, Code 2: No root caries detected, and Code 9: Cannot be assessed.

For this study, root caries was treated as a binary variable. Participants who received a code of 1 were classified as having root caries (i.e., outcome = 1), while those with a code of 2 were considered free of root caries (i.e., outcome = 0). Participants coded as 9 (cannot be assessed) were excluded from the analysis. The NHANES root caries examination protocol is based on diagnostic criteria established in prior NHANES cycles (1999–2004), ensuring methodological consistency over time. Lesions were considered root caries only if they were located on exposed root surfaces and confirmed by tactile softness using an explorer. The complete examination methodology, including detailed diagnostic criteria and scoring procedures, is documented in the NHANES Oral Health Examiners Manual (2018), available through the National Center for Health Statistics (www.cdc.gov/nchs/nhanes).

Exposure variable: High-sensitivity c-reactive protein (hs-CRP):

The exposure variable in this study was high-sensitivity C-reactive protein (hs-CRP). CRP concentrations were measured in serum samples using a two-reagent immunoturbidimetric assay performed at the University of Minnesota's Advanced Research Diagnostics Laboratory. The procedure involved incubating samples with Tris buffer, followed by the addition of latex particles coated with mouse anti-human CRP antibodies. For the NHANES 2017–2018 cycle, a new instrument (Roche Cobas 6000 chemistry analyzer) was used. LBXHSCRP was the NHANES code used to identify CRP levels. For analysis, hs-CRP was treated as a categorical variable based on clinical cutoffs: Low risk: <1.0 mg/L, Intermediate risk: 1.0–3.0 mg/L, and High risk: >3.0 mg/L. Participants with missing hs-CRP data were excluded from the multivariable analysis.

Covariates and adjustment variables

Several covariates were included in the analysis to account for potential confounding factors. These variables were grouped into sociodemographic factors, general behavior, and general health condition.

Sociodemographic factors: Participants aged 30 and older were grouped into the following age categories: 30–49 years, 50–64, and 65 years and older. Gender was classified as either male or female. Race and ethnicity were categorized as Non-Hispanic White, Non-Hispanic Black, Hispanic, and Non-Hispanic Other. Educational attainment was grouped into three levels: less than high school, high school graduate or GED, and some college or higher. Income was categorized into '< $20k' and ' $20k+'.

General behavior: Smoking Status: Defined as "never smoked," "current smoker," or "former smoker."

General health conditions: Diabetes Mellitus: Diabetes status was determined using glycated hemoglobin (HbA1c) values (LBXGH) from laboratory testing. Participants were classified into three categories based on American Diabetes Association guidelines: Normal: HbA1c <5.7% (coded as 1), Prediabetes: HbA1c 5.7% to 6.4% (coded as 2) and Diabetes: HbA1c ≥6.5% (coded as 3).

Statistical analysis

Descriptive statistics were used to describe the study sample. Categorical variables like gender, race/ethnicity, smoking status, and education were reported in terms of unweighted frequencies and weighted percentages, while continuous variables like age and hs-CRP and glycohemoglobin values were summarized as weighted means with standard errors. Table 1 displays the overall distribution of key sociodemographic and clinical covariates.

Bivariate analyses were conducted to compare characteristics between participants with and without root caries. Survey-weighted chi-square tests (Rao–Scott adjusted) were used for categorical variables, and survey-weighted t-tests were used for continuous variables. These comparisons are shown in Table 2, along with corresponding p-values. A significance level of $p \leq 0.05$ was used to determine statistical significance.

To evaluate the association between systemic inflammation and root caries, multivariable logistic regression models were fitted. The outcome variable was the root caries, and the primary exposure was the high-sensitivity CRP (hs-CRP) risk category (low, intermediate, or high). Model 1 included unadjusted estimates. Model 2 adjusted for potential confounders, including age group, gender, race/ethnicity, and educational attainment. Model 3 was further adjusted for smoking status and diabetes. Odds Ratios (ORs), Adjusted Odds Ratios (aORs), and 95% Cconfidence Intervals (CIs) are presented in Table 3. All analyses accounted for the complex, multistage probability sampling design of NHANES by applying the appropriate strata, cluster, and sampling weights using the SURVEY procedures in SAS version 9.4 (SAS Institute, Cary, NC). This ensured nationally representative estimates of the U.S. adult population. Statistical tests were two-sided.

Results

Table 1 presents the unweighted and weighted distributions of sociodemographic, health, and inflammatory characteristics among U.S. adults aged 30 years and older from NHANES 2017–2018 when accounting for the complex survey design.

The prevalence of root caries was 16.5% in the unweighted sample and 13.1% in the weighted population. Systemic inflammation, as measured by high-sensitivity C-reactive protein (hs-CRP), showed a mean level of 4.31 mg/L (unweighted) and 3.48 mg/L (weighted), with 36.8% of participants classified as high risk (≥3.0 mg/L) and 26.9% as low risk (<1.0 mg/L).

The study population had a weighted mean age of 38.4 years, with 43.2% aged 30–49 years and 23.0% aged 65 years or older. Racial/ethnic distribution revealed that Non-Hispanic White adults comprised the majority (66.1%), followed by Hispanic (14.1%) and Non-Hispanic Black (9.5%) adults. Most participants (89.6%) reported annual household incomes of $20,000 or more. Regarding health status, diabetes was present in 16.0% of the unweighted sample (11.7% weighted), while prediabetes affected 33.3% (28.6% weighted). Current smokers represented 15.1% of the weighted population.

Notable differences emerged between unweighted and weighted estimates for education and income. While 18.4% of the unweighted sample had less than a high school education, this decreased to 9.9% after weighting. Similarly, the proportion of low-income adults (<$20,000 annually) was 17.4% unweighted but 10.4% weighted, reflecting NHANES' oversampling of certain demographic groups. All weighted estimates include standard errors to account for the survey's stratified sampling design.

Table 1: Intra-oral photograph illustrating the degree of displacement of the left incisor (#21), gingival bleeding and the degree of coronal fracture of the right incisor (#11).

Characteristic	N (Unweighted %) [Weighted % (SE)]
Glycohemoglobin (%)	5.93(1.11) [5.64(0.02)]
HS C-Reactive Protein (mg/L)	4.31(8.71) [3.48(0.13)]
Age(years)	54.70(14.65) [38.43(0.53)]
Age Groups	
30-49	1,294(38.7%) [43.2%(1.5)]
50-64	1,143(34.2%) [33.8%(1.5)]
65+	905(27.1%) [23.0%(1.5)]
Race/Ethnicity	
Hispanic	744(22.3%) [14.1%(1.7)]
NH White	1,218(36.5%) [66.1%(2.8)]
NH Black	718(21.5%) [9.5%(1.4)]
NH Other	662(19.8%) [10.3%(1.4)]
Annual Household Income	
<$20k	580(17.4%) [10.4%(0.9)]
$20k+	2,762(82.7%) [89.6%(0.9)]
Gender	
Male	1,607(48.1%) [47.9%(1.0)]
Female	1,735(51.9%) [52.1%(1.0)]
Educational Attainment	
Less than HS grad	614(18.4%) [9.9%(0.8)]
HS grad	736(22.0%) [24.2%(1.2)]
Some college	1,089(32.6%) [31.5%(1.6)]
College grad	903(27.0%) [34.4%(2.4)]
Smoking Status	
Never Smoked	1,945(58.2%) [58.1%(1.7)]
Former Smoker	853(25.5%) [26.9%(1.1)]
Current Smoker	544(16.3%) [15.1%(1.1)]
Diabetes Status	
Normal	1,694(50.7%) [59.7%(1.1)]
Prediabetes	1,114(33.3%) [28.6%(1.1)]
Diabetes	534(16.0%) [11.7%(0.7)]
CRP Category	
Low risk	899(26.9%) [28.2%(1.3)]
Intermediate risk	1,213(36.3%) [36.7%(1.5)]
High risk	1,230(36.8%) [35.2%(1.3)]
Root Caries	
No	2,790(83.5%) [86.9%(1.4)]
Yes	552(16.5%) [13.1%(1.4)]

Total sample: Unweighted N = 3,342

Notes: Unweighted data shown as n(%) for categorical variables, mean(SD) for continuous variables

Weighted data shown as %(SE) for categorical variables, mean(SE) for continuous variables

All percentages are column percentages

Table 2 presents the distribution of sociodemographic, health, and inflammatory characteristics stratified by root caries status among U.S. adults aged 30 years or older from NHANES 2017-2018 (N=3,342).

Adults with root caries were significantly older (61.2 vs 52.8 years, p<0.001) and showed poorer glycemic control(mean HbA1c, 6.42% vs 5.79%, p<0.001). Systemic inflammation levels were markedly higher in the root caries group, with mean hs-CRP of 4.40 mg/L compared to 3.26 mg/L (p=0.002), and 60.1% of participants fell into the high-risk CRP category, compared to 32.2% (p<0.001). The age distribution revealed striking disparities, with 59.1% of root caries cases occurring in adults ≥65 years compared to just 20.7% of those without root caries.

Race and ethnicity were strongly associated with root caries ($p < 0.001$). Non-Hispanic Black (32.2% unweighted; 15.3% weighted) and Non-Hispanic Other/multiracial individuals (26.6% unweighted; 26.3% weighted) showed disproportionately higher rates of root caries compared to Non-Hispanic Whites (18.7% unweighted; 42.1% weighted). Conversely, Non-Hispanic Black adults accounted for 32.2% of root caries cases despite comprising only 7.6% of unaffected individuals. Socioeconomic gradients were apparent, with the root caries group having higher proportions of individuals with less than high school education (35.7% vs 15.0%, p<0.001) and annual incomes <$20,000(24.1% vs 16.0%, p=0.008).

Health behavior differences included higher current smoking rates among those with root caries (35.0% vs 12.6%, p<0.001). Diabetes prevalence was substantially higher in the root caries group (42.6% vs 10.7%, p<0.001), with only 28.8% having normal glycemic status compared to 55.0% of those without root caries.

Table 3 presents the results of multivariable logistic regression models examining the association between high-sensitivity C-Reactive Protein (CRP) risk categories. In the unadjusted model (Model 1), individuals in the high-risk CRP category had significantly lower odds of being root caries–free compared to those in the low-risk group (OR = 0.70; 95% CI: 0.48–0.77; p<0.01). This association remained statistically significant after adjusting for age, gender, race/ethnicity, and education in Model 2 (aOR=0.58; 95% CI: 0.35–0.94; p<0.05). However, in the fully adjusted model (Model 3), which additionally accounted for smoking and diabetes status, the association attenuated and was no longer statistically significant (aOR=0.59; 95% CI: 0.35–1.01).

Several covariates showed significant associations with root caries. Compared to college graduates, participants with lower educational attainment had progressively reduced odds of being root caries–free. In Model 3, those with less than a high school education had an aOR of 0.18 (95% CI: 0.10–0.32; p<0.001), and those with some college education had an aOR of 0.32(95% CI: 0.21–0.48; p<0.001). Current smoking was a strong predictor of root caries, with current smokers having significantly lower odds of being caries-free than never-smokers (aOR=0.26; 95% CI: 0.16–0.45; p<0.001). Former smoking and diabetes status were not significantly associated with root caries in the final model. Race/ethnicity also showed associations in Model 3. Non-Hispanic Black participants had significantly lower odds of being root caries–free compared to non-Hispanic Whites (aOR=0.65; 95% CI: 0.45–0.95; p<0.05). The association for Hispanic participants attenuated and was no longer statistically significant in the fully adjusted model. Age group and gender were not significantly associated with root caries in the adjusted models.

Table 2: Unweighted and Weighted Distribution of Select Covariates by Root Caries Status, NHANES (2017–2018).
Total Sample: N = 3,342

Covariate	No Root Caries (Unweighted %, Weighted %)	Root Caries (Unweighted %, Weighted %)	Total (Unweighted %, Weighted %)	P-value
Age (years)	52.8±0.25	61.2±0.54	54.7±0.26	<0.001
Glycohemoglobin (%)	5.79±0.02	6.42±0.06	5.93±0.02	<0.001
HS-CRP (mg/L)	3.26±0.12	4.40±0.32	3.48±0.13	0.002
Age Group				<0.001
1. 30–49	1243(44.6%) (46.6%)	51(9.2%) (11.6%)	1294(38.7%) (43.2%)	
2. 50–64	968(34.8%) (35.5%)	175(31.7%) (29.7%)	1143(34.2%) (33.8%)	
3. 65+	579(20.7%) (17.9%)	326(59.1%) (58.7%)	905(27.1%) (23.0%)	
Gender				0.732
1. Male	1345(48.2%) (47.7%)	262(47.5%) (48.9%)	1607(48.1%) (47.9%)	
2. Female	1445(51.8%) (52.3%)	290(52.5%) (51.1%)	1735(51.9%) (52.1%)	
Race and Ethnicity				<0.001
1. Hispanic	620(22.2%) (13.2%)	124(22.5%) (16.3%)	744(22.3%) (14.1%)	
2. NH White	1115(39.9%) (70.1%)	103(18.7%) (42.1%)	1218(36.5%) (66.1%)	
3. NH Black	540(19.3%) (7.6%)	178(32.2%) (15.3%)	718(21.5%) (9.5%)	
4. NH Other	515(18.4%) (9.1%)	147(26.6%) (26.3%)	662(19.8%) (10.3%)	
Annual Household Income				0.008
1. <$20k	447(16.0%) (8.7%)	133(24.1%) (19.1%)	580(17.4%) (10.4%)	
2. ≥$20k	2343(84.0%) (91.3%)	419(75.9%) (80.9%)	2762(82.6%) (89.6%)	
Educational Attainment				<0.001
1. Less than HS	417(15.0%) (6.3%)	197(35.7%) (21.6%)	614(18.4%) (9.9%)	
2. HS Grad	616(22.1%) (22.6%)	120(21.7%) (27.7%)	736(22.0%) (24.2%)	
3. Some College	949(34.1%) (32.9%)	140(25.4%) (27.2%)	1089(32.6%) (31.5%)	
4. College Grad	808(29.0%) (38.2%)	108(19.6%) (23.5%)	903(27.0%) (34.4%)	
Smoking Status				<0.001
1. Never	1734(62.2%) (64.3%)	211(38.2%) (37.6%)	1945(58.2%) (58.1%)	
2. Former	705(25.3%) (25.1%)	148(26.8%) (30.5%)	853(25.5%) (26.9%)	
3. Current	351(12.6%) (10.6%)	193(35.0%) (31.9%)	544(16.3%) (15.1%)	
Diabetes Status				<0.001
1. Normal	1535(55.0%) (63.2%)	159(28.8%) (36.8%)	1694(50.7%) (59.7%)	
2. Prediabetes	956(34.2%) (28.5%)	158(28.6%) (28.9%)	1114(33.3%) (28.6%)	
3. Diabetes	299(10.7%) (8.2%)	235(42.6%) (34.3%)	534(16.0%) (11.7%)	
CRP Risk Category				<0.001
1. Low Risk	804(28.8%) (30.4%)	95(17.2%) (18.2%)	899(26.9%) (28.2%)	
2. Intermediate Risk	1088(39.0%) (37.4%)	125(22.6%) (34.4%)	1213(36.3%) (36.7%)	
3. High Risk	898(32.2%) (32.2%)	332(60.1%) (47.5%)	1230(36.8%) (35.2%)	

Table 3: Multivariable logistic regression models describing the relationship between CRP risk category and root caries prevalence, NHANES 2017–2018 (n=3342).
(Low CRP risk is reference; outcome: Absence of root caries)

Variable	Model 1 OR [95% CI]	Model 2 aOR [95% CI]	Model 3 aOR [95% CI]
CRP Risk Category			
High Risk	0.70 [0.48, 0.77]**	0.58 [0.35, 0.94]*	0.59 [0.35, 1.01]
Intermediate Risk	0.98 [0.67, 1.04]	0.77 [0.51, 1.16]	0.77 [0.51, 1.17]
Low Risk(ref)	1.00 [1.00, 1.00]	1.00 [1.00, 1.00]	1.00 [1.00, 1.00]

Age Category			
30–49	–	1.11 [0.76, 1.62]	1.35 [0.96, 1.90]
50–64	–	1.00 [0.73, 1.37]	1.10 [0.85, 1.43]
65+(ref)	–	1.00 [1.00, 1.00]	1.00 [1.00, 1.00]
Gender			
Female	–	1.15 [0.87, 1.53]	1.02 [0.75, 1.37]
Male(ref)	–	1.00 [1.00, 1.00]	1.00 [1.00, 1.00]
Race/Ethnicity			
Hispanic	–	1.82 [1.07, 3.09]*	1.57 [0.94, 2.61]
NH Black	–	0.65 [0.45, 0.93]*	0.65 [0.45, 0.95]*
NH Other	–	0.76 [0.47, 1.24]	0.73 [0.46, 1.17]
NH White(ref)	–	1.00 [1.00, 1.00]	1.00 [1.00, 1.00]
Education			
HS Grad	–	0.24 [0.16, 0.37]***	0.31 [0.21, 0.46]***
<HS Grad	–	0.14 [0.08, 0.23]***	0.18 [0.10, 0.32]***
Some College	–	0.27 [0.18, 0.40]***	0.32 [0.21, 0.48]***
College Grad(ref)	–	1.00 [1.00, 1.00]	1.00 [1.00, 1.00]
Smoking Status			
Current Smoker	–	–	0.26 [0.16, 0.45]***
Former Smoker	–	–	0.76 [0.55, 1.05]
Never Smoked(ref)	–	–	1.00 [1.00, 1.00]
Diabetes Status			
Diabetes	–	–	0.67 [0.43, 1.03]
Prediabetes	–	–	1.08 [0.81, 1.45]
Normal(ref)	–	–	1.00 [1.00, 1.00]

Notes: Outcome: *No root caries(rootcaries_cat2 = No)*
OR: Unadjusted Odds Ratio; aOR: Adjusted Odds Ratio
***p < 0.001, **p < 0.01, *p < 0.05
Model 1: Unadjusted
Model 2: Adjusted for sociodemographic variables including age group, gender, race/ethnicity, and educational attainment.
Model 3: Fully adjusted model including all variables in Model 2 plus smoking status and diabetes status.

Discussion

This study explored the association between systemic inflammation, as measured by High-Sensitivity C-Reactive Protein(hs-CRP), and root caries prevalence in U.S. adults.

The findings revealed that participants in the high CRP risk category had significantly lower odds of being caries-free, reflecting a positive association between systemic inflammation and root caries prevalence.

Our study found the prevalence of root caries to be unweighted (16.5%) and in the (13.1%) weighted population. This finding is consistent with studies conducted in the United States between 2017 and 2020, which reported a root caries prevalence of approximately 14% among U.S. adults [26]. The results highlight root caries as a significant dental problem, particularly given its association with older adults [27]. The elevated levels of hs-CRP (weighted mean: 3.48 mg/L) suggest a high inflammatory burden among the study population. These values exceed the American Heart Association's threshold for cardiovascular risk assessment (≥2.0 mg/L) [28], highlighting potential systemic health implications.

In this study, adults with root caries demonstrated significantly poorer glycemic control (p<0.0001). This was consistent with a study done in 2024, which showed a significant association between higher HbA1c levels and an increased risk of untreated root caries (Odds Ratio [OR]: 1.11, 95% CI: 1.01-1.22, $p < .05$) [29]. Elevated blood glucose levels can lead to various pathological changes, including salivary gland dysfunction and decreased salivary flow [30], which are significant risk factor for root caries.

In this study, significant racial and ethnic differences were observed in the root caries prevalence. Non-Hispanic Black adults represented 32.2% of individuals with root caries, indicating a disproportionately high burden. These findings are consistent with prior analyses of NHANES datasets. For example, Badr and Sabbah found that non-Hispanic Black adults had significantly higher odds of untreated root caries (OR 1.88, 95% CI 1.17, 3.01) [31]. Similarly, Griffin et al., noted in their report that root caries were significantly higher among non-Hispanic Blacks (40%) compared with non-Hispanic Whites (less than 20%) [32]. There could be a potential explanatory factor to these consistent findings, which may be related to historical inequities to dental care access. For example, a study that used the behavioral risk factor surveillance system (BRFSS) data for 2014 found that both Hispanics and non-Hispanic Blacks were less likely to visit a dentist in the past year [33]. Additionally, there have been some biological considerations regarding this, as studies have shown that genetic variations in enamel formation genes influence caries risk [34]. The higher root caries prevalence among individuals with lower education (35.7% vs. 15.0%, $p<0.001$) highlight education as a key determinant of oral health. Similar findings have been observed among American adults, where individuals with higher educational attainment were more likely to visit the den-

tist regularly, highlighting education-based disparities in dental care utilization [35].

In the unadjusted logistic regression model, individuals in the high-risk CRP category (≥3.0 mg/L) had lower odds of being root caries–free. These findings align with previous research by Al Shammari et al. which showed a positive association between elevated CRP levels and a higher mean number of dental caries, with an Adjusted Mean Ratio (AMR) of 1.7. Logistic regression analysis further indicated that individuals with higher CRP levels had a 50% greater likelihood of developing dental caries (AOR=1.5; 95% CI: 1.2–1.9; p<0.01) [36]. The narrow confidence interval (0.48-0.77) demonstrates greater precision than similar studies, attributable to NHANES' large sample size and standardized measurements.

The sustained statistical significance of the association between elevated CRP levels and root caries after adjusting for demographic variables (aOR=0.58, 95% CI: 0.35–0.94, p<0.05) in Model 2 highlights a likely independent link between systemic inflammation and root caries risk. The reduction in effect from the unadjusted OR of 0.70 to the adjusted OR of 0.58 indicates that demographic factors account for 17% of the CRP–caries association, suggesting a considerable portion remains independently driven by inflammation.

The loss of statistical significance after adjusting for smoking and diabetes status suggests these factors explain much of the apparent CRP-caries association. This finding mirrors results from study done by Sim et al using data from the 2016–2018 Korea National Health and Nutrition Examination Survey (KNHANES), which showed that current smokers had a higher risk of periodontal disease compared to non-smokers [37]—aligning with the attenuation pattern observed in our study. The biological plausibility of this mediation is well-established. *Streptococcus mutans* is recognized as the primary pathogen in dental caries due to its strong acid resistance, acidogenicity, and ability to form biofilms [38]. Notably, nicotine has been shown to enhance both the biofilm formation and metabolic activity of *S. mutans*, further contributing to its cariogenic potential [39]. Hyperglycemia similarly contributes to elevated production of inflammatory factors like CRP, IL-6, and TNF- *α [40]* and which increases caries susceptibility. Our findings suggest these shared pathways may largely account for the initial unadjusted association.

A major strength of this study is the use of a large, nationally representative dataset (NHANES 2017–2018), which enhances the generalizability of findings to the U.S. adult population. Additionally, the study benefits from standardized clinical assessments of root caries and high-quality laboratory measurement of hs-CRP levels, both of which strengthen the validity of the exposure and outcome measures.

Importantly, this study addresses a markedly underexplored area in the literature. To date, very few studies have investigated the association between systemic inflammation, as measured by high-sensitivity C-reactive protein, and dental root caries in adults. While inflammation has been widely studied in relation to periodontal disease and systemic conditions, its specific link to root caries remains largely neglected. By focusing on this intersection of systemic and oral health, this study contributes unique insights and helps fill a critical gap in oral health epidemiology.

Nevertheless, the study has limitations. The cross-sectional design precludes causal inference. Residual confounding from unmeasured variables—such as oral hygiene practices, dietary factors, or access to dental care—may also influence the observed associations.

Conclusion and future direction

This study provides compelling evidence of an inverse association between high-sensitivity C-reactive protein(hs-CRP) levels and the likelihood of being root caries–free among U.S. adults. Elevated systemic inflammation, as reflected by hs-CRP, was associated with increased odds of root caries in both unadjusted and partially adjusted models, though the association attenuated after adjusting for behavioral and clinical risk factors such as smoking and diabetes. Socioeconomic disparities—particularly in education—and current smoking status emerged as robust predictors of root caries, highlighting the multifactorial nature of oral health inequities.

Very few studies have explored the intersection of systemic inflammation and root caries risk using nationally representative data, making this analysis a valuable contribution to both dental and public health literature

Future studies should follow individuals over time to better understand how systemic inflammation contributes to the development and progression of root caries. There is also a need to test whether improving modifiable risk factors like smoking, diabetes control, or oral hygiene can help lower both CRP levels and the risk of root surface decay.

Author declarations

Funding

This research received no external funding.

Consent to participate

All NHANES participants provided written informed consent at the time of recruitment. The present analysis used de-identified, publicly available NHANES data.

Human ethics and consent to participate declarations

The NHANES protocol was approved by the National Center for Health Statistics(NCHS) Research Ethics Review Board. Written informed consent was obtained from all participants at the time of data collection. The present study was a secondary analysis of de-identified public-use data and was exempt from additional institutional review board approval.

References

1. Zhang W, McGrath C, Lo ECM. A comparison of root caries diagnosis based on visual-tactile criteria and DIAGNOdent in vivo. J Dent. 2009; 37: 509–513.

2. Badr F, Sabbah W. Inequalities in untreated root caries and affordability of dental services among older American adults. Int J Environ Res Public Health. 2020; 17: 8523.

3. Katz RV. The clinical identification of root caries. Gerodontology. 1986; 5: 21–24.

4. Turner MD, Ship JA. Dry mouth and its effects on the oral health of elderly people. J Am Dent Assoc. 2007; S15–S20.

5. Gavriilidou N, Belibasakis G. Root caries: The intersection between periodontal disease and dental caries in the course of ageing. Br Dent J. 2019; 227: 1063–1067.

6. Back JL. Cardiac injury, atherosclerosis, and thrombotic disease. In: McPherson A, editor. Henry's clinical diagnosis and management by laboratory methods. 14th ed. Elsevier; 2022. p. 267–275.

7. Luan Y, Yao Y. The clinical significance and potential role of C-reactive protein in chronic inflammatory and neurodegenerative diseases. Front Immunol. 2018; 9: 1302.

8. Helal I, Zerelli L, Krid M, et al. Comparison of C-reactive protein and high-sensitivity C-reactive protein levels in patients on hemodialysis. Saudi J Kidney Dis Transpl. 2012; 23: 477–483.

9. Buckley DI, Fu R, Freeman M, Rogers K, Helfand M. C-reactive protein as a risk factor for coronary heart disease: A systematic review and meta-analyses for the U.S. Preventive Services Task Force. Ann Intern Med. 2009; 151: 483–495.

10. Danesh J, Collins R, Appleby P, Peto R. Association of fibrinogen, C-reactive protein, albumin, or leukocyte count with coronary heart disease: Meta-analyses of prospective studies. JAMA. 1998; 279: 1477–1482.

11. Oluleye OW, Folsom AR, Nambi V, Lutsey PL, Ballantyne CM. Troponin T, B-type natriuretic peptide, C-reactive protein, and cause-specific mortality. Ann Epidemiol. 2013; 23: 66–73.

12. Dregan A, Charlton J, Chowienczyk P, Gulliford MC. Chronic inflammatory disorders and risk of type 2 diabetes mellitus, coronary heart disease, and stroke: A population-based cohort study. Circulation. 2014; 130: 837–844.

13. Gomes-Filho IS, Coelho JMF, Cruz SS, Passos JS, Freitas COT, Farias NSA, et al. Chronic periodontitis and C-reactive protein levels. J Periodontol. 2011; 82: 969–978.

14. Ebersole JL, Cappelli D. Acute-phase reactants in infections and inflammatory diseases. Periodontol 2000. 2000; 23: 19–49.

15. Bennett JC, Plum F. The acute phase response. In: Cecil textbook of medicine. 20th ed. Vol. 2. Saunders; 1996. p. 1535–1537.

16. Panichi V, Migliori M, Di Pietro S, Taccola D, Andreini B, Metelli MR, et al. The link of biocompatibility to cytokine production. Kidney Int. 2000; S96–S103.

17. Christensen LB, Bardow A, Ekstrand K, Fiehn NE, Heitmann BL, Qvist V, et al. Root caries, root surface restorations and lifestyle factors in adult Danes. Acta Odontol Scand. 2015; 73: 467–473.

18. Laudenbach JM, Simon Z. Common dental and periodontal diseases: Evaluation and management. Med Clin North Am. 2014; 98: 1239–1260.

19. León S, Rivera M, Payero S, Correa-Beltrán G, Hugo FN, Giacaman RA. Assessment of oral health-related quality of life as a function of non-invasive treatment with high-fluoride toothpastes for root caries lesions in community-dwelling elderly. Int Dent J. 2019; 69: 58–66.

20. Peng X, Cheng L, You Y, Tang C, Ren B, Li Y, Xu X, Zhou X. Oral microbiota in human systemic diseases. Int J Oral Sci. 2022; 14: 14.

21. Bhattarai KR, et al. The imprint of salivary secretion in autoimmune disorders and related pathological conditions. Autoimmun Rev. 2018; 17: 376–390.

22. Xie R, Liu X, Wu H, Liu M, Zhang Y. Associations between systemic immune-inflammation index and abdominal aortic calcification: Results of a nationwide survey. Nutr Metab Cardiovasc Dis. 2023; 33: 1437–1443.

23. Kalyani BS, Qian J, Hansen RA. Utilization patterns of antihypertensive drugs among the chronic kidney disease population in the United States. Clin Ther. 2015; 37: 188–196.

24. Jones CA. Hypertension and renal dysfunction: NHANES III. J Am Soc Nephrol. 2003; 14: S71–S75.

25. Tang M, Liu M, Zhang Y, Xie R. Association of family income to poverty ratio and vibration-controlled transient elastography quantified degree of hepatic steatosis in U.S. adolescents. Front Endocrinol. 2023; 14: 1160625.

26. Mobin T, Khan TZ, Mobin A, Tahir MR, Imran Q. The association between seeking urgent dental care and the prevalence of root caries among patients in the United States: National Health and Nutrition Examination Survey 2017–2020. Cureus. 2023; 15: e41797.

27. Gati D, Vieira AR. Elderly at greater risk for root caries: A look at the multifactorial risks with emphasis on genetic susceptibility. Int J Dent. 2011; 2011: 647168.

28. Arnett DK, Blumenthal RS, Albert MA, Buroker AB, Goldberger ZD, Hahn EJ, et al. 2019 ACC/AHA guideline on the primary prevention of cardiovascular disease: A report of the American College of Cardiology/American Heart Association Task Force on Clinical Practice Guidelines. Circulation. 2019; 140: e596–e646.

29. He L, Shi L, Zhu JH. Association between diabetes status, glycaemic control and untreated root caries among United States adults. Int Dent J. 2025; 75: 537–544.

30. Díaz Rosas CY, Cárdenas Vargas E, Castañeda-Delgado JE, Aguilera-Galaviz LA, Aceves Medina MC. Dental, periodontal and salivary conditions in diabetic children associated with metabolic control variables and nutritional plan adherence. Eur J Paediatr Dent. 2018; 19: 119–126.

31. Griffin SO, Jones JA, Brunson D, Griffin PM, Bailey WD. Burden of oral disease among older adults and implications for public health priorities. Am J Public Health. 2012; 102: 411–418.

32. Singhal A, Jackson JW. Perceived racial discrimination partially mediates racial-ethnic disparities in dental utilization and oral health. J Public Health Dent. 2022; 82: 63–72.

33. Opal S, Garg S, Jain J, Walia I. Genetic factors affecting dental caries risk. Aust Dent J. 2015; 60: 2–11.

34. Lee W, Kim SJ, Albert JM, Nelson S. Community factors predicting dental care utilization among older adults. J Am Dent Assoc. 2014; 145: 150–158.

35. AlShammari A, AlSaleh S, AlKandari A, et al. The association between dental caries and serum CRP in the U.S. adult population: Evidence from NHANES 2015–2018. BMC Public Health. 2024; 24: 2210.

36. Sim KY, Jang YS, Nerobkova N, Park EC. Association between smoking and periodontal disease in South Korean adults. Int J Environ Res Public Health. 2023; 20: 4423.

37. Takahashi N, Nyvad B. The role of bacteria in the caries process: Ecological perspectives. J Dent Res. 2011; 90: 294–303.

38. Huang R, Li M, Gregory RL. Effect of nicotine on growth and metabolism of Streptococcus mutans. Eur J Oral Sci. 2012; 120: 319–325.

39. Gasecka A, Siwik D, Gajewska M, et al. Early biomarkers of neurodegenerative and neurovascular disorders in diabetes. J Clin Med. 2020; 9: 2807.