HHS Public Access

Author manuscript

Clin Nutr. Author manuscript; available in PMC 2019 August 01.

Published in final edited form as:

Clin Nutr. 2018 August ; 37(4): 1306–1312. doi:10.1016/j.clnu.2017.06.001.

Diet-borne systemic inflammation is associated with prevalent tooth loss

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SUMMARY

Background & aims—The deleterious effect of cariogenic dietary patterns on tooth loss is well characterized, but the contribution of diet-borne systemic inflammation to loss of teeth remains uncharted. Recent efforts have unveiled a protective role of single nutrients to periodontal health. However, the assessment of overall diet as a modifiable risk factor for oral health remains elusive. Thus, the aim of this study was to assess the association between diet-borne systemic inflammation and tooth loss in a representative sample of the US adult non-institutionalized population.

Methods—A cross-sectional analysis of a sample of participants of the 2009–2010 and 2011–2012 continuous NHANES receiving an oral exam and providing dietary recall data was performed. Dietary inflammatory potential was assessed by the Dietary Inflammatory Index (DII),

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Availability of data and material

The datasets supporting the conclusions of this article are available in the publicly available NHANES repository: http://wwwn.cdc.gov/Nchs/Nhanes/Search/Nhanes_continuous.aspx.

Conflict of interest statement

Dr. Hébert owns controlling interest in Connecting Health Innovations LLC (CHI), a company planning to license the right to his invention of the dietary inflammatory index (DII) from the University of South Carolina in order to develop computer and smart phone applications for patient counseling and dietary intervention in clinical settings. Dr. Michael Wirth and Dr. Nitin Shivappa are employees of CHI.

Drs. Kotsakis, Chrepa, Koyanagi and Tyrovolas declare that they have no competing interests.

Statement of Authorship

GK, VC and ST designed the study, performed the analysis and interpretation of the data and preparation of the manuscript, NS, JH, MW and AK contributed to analysis of data and critical review of the manuscript for important intellectual content.

a composite measure computed based on the association between nutrients and systemic pro-inflammatory cytokine levels. The outcome measure was prevalent tooth loss. Numbers of missing teeth were regressed across quartiles of the DII using multivariable linear regression models.

Results—6887 eligible NHANES participants were included in the analysis; participants in the highest quartile of the DII index (pro-inflammatory diet) had an average [95% CI] of 0.84 [0.24, 1.45] additional more teeth lost as compared to those in the lowest quartile of DII (anti-inflammatory diet) ($p = 0.015$), after adjusting for known confounders. This significant association remained in subgroup analyses, including the lowest tertiles of energy-adjusted carbohydrate intake, and in persons aged ≥50 years.

Conclusions—Adherence to an anti-inflammatory diet is associated with fewer missing teeth. These results suggest protective dietary patterns as a modifiable protective factor for tooth loss in the US adult population and support the incorporation of tooth loss prevention in the agenda of dietary public health interventions to prevent chronic inflammatory diseases.

Keywords

NHANES; Periodontal diseases; Tooth loss; Inflammation; Diet; Nutrition

1. Introduction

Tooth loss is a prevalent morbid condition that substantially contributes to the global burden of disease, similarly to conditions as widely variable as rheumatoid arthritis and ovarian cancer [1]. Recent discoveries revealing the independent association of missing teeth with incident cardiovascular events and mortality have sparked interest in the investigation of correlates of tooth loss, i.e. edentulism [2,3]. Tooth loss is a well-defined, definitive condition that is the outcome of either one of two conditions; dental caries or periodontitis. Dental caries are largely attributed to cariogenic dietary habits, with carbohydrate-rich diet being a key modifiable risk factor for disease. The effect that dietary habits have on tooth loss is such that a diet restricting fermentable carbohydrates (e.g. simple sugars and complex carbohydrates) can prevent caries [4]. However, periodontitis, the major cause of tooth loss in adults over 40 years of age [5], and diet are considered unrelated.

Periodontitis is a prevalent chronic inflammatory disease that ultimately leads to tooth loss [6]. The inflammatory component of periodontitis is not constrained to oral tissues, but seems to be contributing to chronic systemic low-grade inflammation [7]. Epidemiologic data indicate that periodontitis patients have elevated serum levels of inflammatory biomarkers, such as C-reactive protein (CRP) levels [8], compared to individuals free of periodontitis. Results also point to a reduction in systemic inflammatory marker concentrations following periodontal therapy [9]. Despite periodontitis being among the most prevalent chronic diseases in adults with its severe form affecting nearly one out ten US adults ≥30 years [10], modifiable risk factors for this disease are not well-established, other than oral hygiene and smoking [11]. Periodontitis and tooth loss in adults have been linked to obesity, diabetes and cardiovascular disease via chronic low-grade inflammation as their common denominator [3,7]. Therefore, when viewed through the prism of systemic

inflammation, periodontitis may be associated with environmental factors that contribute to a heightened systemic pro-inflammatory state.

Recently, single nutrients such as n–3 polyunsaturated fatty acids have been shown to hold promise for regulating inflammation in the periodontium via increasing pro-resolving lipid mediators to regulate the immune-mediated destructive inflammatory response [12,13]. Less is known regarding the overall effect of diet on tooth loss. The limited available evidence reports a protective effect of dietary caloric restriction on surrogate indices of tooth loss in non-human primates [14]. Nevertheless, studies on the overall effect of diet on chronic inflammatory diseases have unveiled a wealth of associations between pro-inflammatory diet and risk for cardiovascular diseases, cancer, and mortality [15–17]. The potential of diet to resolve or dysregulate the inflammatory response associated with tooth loss related to periodontal inflammation remains unclear and warrants further investigation. Thus, the aim of this study was to assess the association between diet-borne systemic inflammation and prevalent tooth loss in a representative sample of the US adult non-institutionalized population that participated in the 2009–2010 and 2011–2012 continuous National Health and Nutrition Examination Surveys (NHANES). Specifically, it was hypothesized that persons with more pro-inflammatory diet, as measured by the Dietary Inflammatory Index (DII)™, would have greater tooth loss compared to those with more anti-inflammatory diet (i.e., lower DII scores) [18].

2. Materials and methods

The NCHS Research Ethics Review Board approved the study protocol of 2009–2010 (protocol 2005–06), and 2011–2012 (protocol 2011–17) NHANES, and all participants provided written informed consents.

2.1. Study population

The continuous NHANES is a cross-sectional survey that assesses the health and nutritional status of US persons via interviews and physical examinations [19]. Continuous NHANES utilize a complex survey design to obtain a nationally representative, stratified, multistage probability sample of the civilian non-institutionalized US population. The current analysis included data from participants aged ≥30 years old who had completed an oral examination (teeth count) recorded at a mobile exam center. They also had nutritional assessment by trained interviewers as part of the 2009–10 and 2011–12 continuous NHANES waves [19]. Individuals were excluded if they: had incomplete teeth count data or incomplete dietary recall assessment data; had fractured teeth; had not undergone periodontal examination; or if they were missing important covariate data. The NCHS Research Ethics Review Board approved the study protocol of 2009–2010 (protocol 2005–06), and 2011–2012 (protocol 2011–17) NHANES, and all participants provided written informed consents.

2.2. Outcome assessment

In the 2009–2010 and 2011–12 NHANES, 7145 and 8073 persons underwent complete oral health examinations at the mobile examination centers, respectively. For oral assessments, we utilized missing tooth counts recorded by trained and calibrated examiners. All

NHANES examiners were calibrated against a reference examiner and underwent an annual retraining session. For the primary outcome assessment, missing teeth were recorded as the number of edentulous sites or sites with dental implant replacements [20].

2.3. Dietary assessment

The DII was used as the exposure variable. The DII is an index developed based on the association of various food components to the levels of specific inflammatory biomarkers; i.e., interleukin (IL)-10, IL-4, IL-6, IL-1β, tumor necrosis factor (TNF)-α, and CRP (Suppl. Table 1) [18]. This index has been has been designed to capture diet's pro-inflammatory potential; its development and validation of the DII has been described in detail elsewhere [18,21]. Briefly, nearly 2000 research articles published between 1950 and 2010 examining the relationship between 45 different food parameters (mostly micro and macro nutrients, flavonoids, and some individual food items) and inflammation were reviewed (Suppl. Table 1). Studies showing a positive association between the food parameters and pro-inflammatory cytokines (i.e., IL-1β, IL-6, tumor necrosis factor [TNF]-α, and CRP), or a negative association with anti-inflammatory cytokines (IL-4 and IL-10) received a value of "+1". If the food parameters were associated with reduced pro-inflammatory or increased anti-inflammatory cytokines, the article received a value of "−1". Null values were set to "0", and these scores were weighted based on study design. For example, randomized control trials received the greatest weight, while preclinical studies the lowest weight. These scores and the weights were used to create pro- and anti-inflammatory fractions for each food parameter [18]. The anti-inflammatory fraction was subtracted from the pro-inflammatory fraction to create the "article effect score" for each of the 45 food parameters [18]. DII calculation is linked to a regionally representative world database. The world database contains standard means and deviations for the 45 food parameters from 11 populations around the world (i.e., United States, United Kingdom, Bahrain, Mexico, Australia, South Korea, Taiwan, India, New Zealand, Japan, and Denmark) [18].

For DII calculation in the NHANES participants, dietary information from the available 24-hour dietary recall were utilized. The DII food parameters available included carbohydrates; protein; fat; alcohol; fiber; cholesterol; saturated, monounsaturated, and polyunsaturated fatty acids; omega-3 and omega6- polyunsaturated fatty acids; niacin; vitamins A, B1, B2, B6, B12, C, D, E; iron; magnesium; zinc; selenium; folic acid; beta carotene; and caffeine. A z-score was created for each food parameter by subtracting the world standard means from the participants estimated intake, then dividing this by its standard deviation. This was then converted to a percentile and centered by doubling the value and subtracting "1". The product of the literature-derived inflammatory effect score and the centered percentile for each food parameter was summed across all food parameters to create the overall DII score. Higher (i.e., more positive) scores indicate more pro-inflammatory diets and negative values are more anti-inflammatory [18]. To control for the effect of total energy intake, the DII was calculated per 1000 calories of food consumed. As per DII calculation, the lower the DII score the more anti-inflammatory the dietary consumption.

In addition to DII calculation, total carbohydrate intake information was obtained from the data and energy-adjusted against total daily energy intake. Increased fermentable

carbohydrate consumption defines persons at high risk for tooth loss due to decay; the second most frequent reason for tooth loss in adults [5]. Thus, we aimed to control for this important potential confounder in the relationship between inflammatory diet and tooth loss due to periodontal inflammation.

2.4. Assessment of variables used for adjustment

All analyses included extensive adjustment for covariates related to both tooth loss and low-grade inflammation, as previously described [20]. These included demographic and socioeconomic variables, behavioral risk factors and chronic inflammatory conditions and diseases. The demographic and socioeconomic variables adjusted for were age, gender, race/ethnicity, educational level, and poverty-income-ratio (PIR). PIR was assessed as the ratio of family income to poverty based on the poverty guidelines of the Department of Health and Human Services. A PIR < 1.0 corresponds to a family income level below the official poverty threshold. Behavioral risk factor assessments included cigarette smoking (current, past, never smoker) and physical activity level (none, low, moderate or high) assessed with the Global Physical Activity Questionnaire using conventional cutoffs (http://www.who.int/chp/steps/GPAQ/en/) [20]. BMI was calculated from weight and height measurements (<25 kg/m^2, 25–29.9 kg/m^2, or ≥30 kg/m^2) [20]. We further adjusted for systemic conditions/diseases that are associated with chronic systemic inflammation: a) obesity; b) diabetes mellitus; c) hypercholesterolemia; d) hypertension. To assess the cumulative effect of these comorbidities on tooth loss we coded a 5-level dummy variable with scores ranging from “0” (representing no comorbidities) to “4” (representing positive status for all four assessed diseases/conditions). In detail: a) obesity was defined as BMI ≥ 30 kg/m^2 based on weight and height measured at the mobile examination centers (MECs); diabetes mellitus (type 2) was defined as either one of: glycated hemoglobin A1C ≥ 6.5; fasting blood glucose levels ≥ 120 mg/dl; or self-reported prior diagnosis of diabetes, based on the American Diabetes Association diagnostic criteria [20]; c) hypercholesterolemia was defined by the presence of at least one of: total serum cholesterol levels ≥200 mg/dL; or self-reported prior diagnosis of hypercholesterolemia [22]; d) hypertension referred to either one of: systolic blood pressure ≥140 mmHg; diastolic blood pressure ≥90 mmHg; or self-reported prior diagnosis of hypertension.

2.5. Statistical analysis

Descriptive statistics were estimated for quartiles of the DII and reported as percentages or means (SD). To assess the association of the DII and tooth loss we estimated the numbers of missing teeth and regressed this value across quartiles of the DII scores. For the main analysis, multivariable linear regression models were constructed with number of missing teeth as the outcome, DII as the exposure and extensive confounder adjustment. Confounder selection was based on previous reports from this sample population and included age, sex, race/ethnicity, poverty-income-ratio, education level, smoking status, physical activity level, BMI and a comorbidities variable, as defined above [20]. A sensitivity analysis excluding persons at the highest tertile of total carbohydrate intake (a correlate of dental caries) was performed to minimize confounding by tooth loss due to caries. We further performed a subgroup analysis in persons >50 years of age because the risk of tooth loss due to periodontal inflammation increases with age [10]. All analyses used appropriate sampling

weights for the two NHANES cycles (i.e., two-year mobile examination sampling weights × 0.5) to account for the complex survey design and to yield nationally-representative estimates. Missing values were excluded from the analyses. Standard errors were calculated based on Taylor series linearization. The level of statistical significance was set at $p < 0.05$ and analyses were performed in Stata Statistical Software: Release 14 (College Station, TX: StataCorp LP).

3. Results

Following exclusion of participants with incomplete oral health data or dietary recall assessments, data from 6887 participants of the 2009–10 and 2011–12 continuous NHANES aged ≥30 years old were analyzed using appropriate sampling weights (Fig. 1).

The prevalence of edentulism excluding third molars was 65.1% in the overall sample and 62.7% in dentates, while the mean [SD] tooth loss was 5.2 [7.9]. Prevalence of edentulism among the upper quartile of DII was 65.9%, while the prevalence for the lower quartile of DII was 62.9%, respectively (Table 1, Fig. 2).

In crude analyses, tooth counts varied significantly across DII quartiles ($p = 0.003$), with a mean [95% CI] difference of 1.13 [0.56, 1.70] additional missing teeth for participants in the 4th quartile (pro-inflammatory diet) as compared to the 1st quartile (anti-inflammatory diet).

After adjusting for demographics and environmental variables including smoking, participants in the upper DII quartile were associated with 0.84 [0.24, 1.45] additional missing teeth ($p = 0.015$) as compared to the lowest quartile (Table 2).

Results were robust in the analysis excluding persons in the highest tertile of energy-adjusted carbohydrate consumption; participants in the upper quartile of DII had 0.80 [0.17, 1.42] additional missing teeth ($p = 0.027$) than participants in the lower DII quartile (Table 3). The subgroup analysis in adults aged ≥50 also found the results to be consistent, with persons in the upper DII quartile having 1.19 [0.31, 2.06] additional missing teeth than those in the lower quartile ($p = 0.020$) (Table 3).

4. Discussion

Adherence to an anti-inflammatory diet is associated with lower odds for tooth loss in this population. In detail, participants consuming an anti-inflammatory diet (lower DII quartile) had an average of one more tooth present as compared to those consuming a pro-inflammatory diet ($p = 0.015$) in adjusted models. These results were consistent and stronger in individuals with low carbohydrate intake or >50 years of age, i.e. in subpopulations that suffer tooth loss primarily due to chronic inflammatory periodontal disease. These results suggest protective dietary patterns as a modifiable protective factor for tooth loss based on a representative sample of the US adult population.

This is the first report of an association between overall dietary patterns and tooth loss. A plenitude of previous research efforts have documented the key role of fermentable carbohydrate consumption in the pathogenesis of dental caries, but the data on the effect of

diet on inflammatory periodontal diseases is scarce. The majority of existing studies have focused on specific nutrients for the treatment or prevention of periodontitis [23]. Thus far, their results have been inconclusive, with limited promising results in cases of nutritional supplementation for underlying micronutrient deficiencies below dietary reference intake values, such as hypovitaminosis D [24]. Specific nutrients have been associated with reduction in inflammatory biomarkers that are present in periodontal tissue destruction [23]. Nonetheless, it is important to assess nutrients as part of the overall dietary effect, as they represent a set of modifiable risk factors for inflammatory diseases. Notably, diet is a shared risk factor among cardiovascular diseases and other inflammatory diseases [15,25]. The opportunity to directly address the prevention of these important non-communicable diseases with dietary modification aimed to reduce the systemic inflammatory burden results in a cost-effective addition to the public health intervention quiver.

The biological plausibility for the association of diet-borne inflammation to tooth loss is strong. Initiation of periodontitis is primarily attributed to teeth-adherent bacterial communities that have a pathogenic potential; i.e. are “dysbiotic” [26]. Nonetheless, the pathogenesis of periodontitis is immune-mediated and a susceptible host is a prerequisite for periodontitis to occur [27]. When viewed through the prism of systemic inflammation, periodontitis may be associated with environmental factors that contribute to a systemic pro-inflammatory state. The consistent finding of increased prevalence of periodontitis among diabetic patients as compared to normoglycemic controls is exemplary of the contribution of systemic inflammatory on periodontitis onset and progression [28,29]. As previously stated, periodontitis is initiated by oral bacteria therefore the effect of diet-borne inflammation seems to be contributory rather than etiologic.

The present study has several strengths; most notably it is the first study to document an association between overall dietary patterns and tooth loss. This paves the way for the recognition of diet as a novel modifiable risk factor for tooth loss due to its contribution to a systemic pro-inflammatory state. The breadth of information available in the NHANES dataset allowed us to utilize highly adjusted models that included adjustment for sociodemographic variables, known risk factors for periodontitis as well as other systemic inflammatory conditions in an effort to minimize confounding. Furthermore, we performed a subgroup analysis excluding the highest tertile of energy-adjusted fermentable carbohydrate consumption to exclude persons in high risk for tooth loss due to caries. An additional subgroup analysis in persons aged ≥50 years was performed since tooth loss due to periodontal inflammatory disease increases with age. Results of all subgroup analyses found results of the main model to be robust. Lastly, based on the design of the NHANES the results are generalizable to the US non-institutionalized adult population.

The limitations of this investigation also must be considered. Tooth loss is an end-state condition and the temporal mismatch between the dietary data and the time of tooth loss limits the conclusions of this analysis [30]. Confirmatory longitudinal studies or clinical trials are warranted to evaluate causality and temporal trends. Furthermore, the strong confounding effect of smoking may not be adequately adjusted for by controlling for currency of smoking in the adjusted models and further validation of the present findings in non-smoking populations is necessary. Additionally, the continuous NHANES does not

record data on measures of gingival inflammation (e.g. bleeding on probing of the gingivae), which would allow the direct simultaneous assessment of dietary inflammatory potential and gingival inflammation. Data on oral hygiene efficacy is also not available. Oral hygiene awareness may be an important confounder to consider in prospective studies as it is apparent that associations exist between overall health awareness, lifestyle and diet, which could moderate the estimates observed in this study. Moreover, our findings share the limitations of results from epidemiological studies that rely on self-reported measures of dietary intake. Further research is needed to identify the components of the DII that drive this association with tooth loss. Specifically, given the importance of this condition, intervention studies to assess reduction of dietary inflammatory potential as a preventive measure in order to translate the present findings to applicable preventive measures.

5. Conclusions

Adherence to an anti-inflammatory diet is associated with fewer missing teeth. The present results suggest protective dietary patterns as a modifiable protective factor for tooth loss in the US adult population. Results further corroborate that tooth loss shares modifiable risk factors with other chronic inflammatory diseases, thus incorporation of tooth loss prevention in the agenda of dietary public health interventions to prevent chronic inflammatory diseases is a direct and cost-effective approach for improving population oral health.

Supplementary Material

Refer to Web version on PubMed Central for supplementary material.

Acknowledgments

The authors are thankful to the NHANES investigators and personnel. The authors would also like to thank Xiwen Liao, Seattle, WA for her assistance with generating the figures for this manuscript.

Funding sources

Georgios Kotsakis received an academic career & teaching scholarship by the American Academy of Periodontology Foundation. Stefanos Tyrovolas's work was supported by the Foundation for Education and European Culture (IPEP), the Sara Borrell postdoctoral programme (reference no. CD15/00019) from the Instituto de Salud Carlos III (ISCIII – Spain) and the Fondos Europeo de Desarrollo Regional (FEDER). Ai Koyanagi's work was supported by the Miguel Servet contract financed by the CP13/00150 project, integrated into the National R + D + I and funded by the ISCIII – General Branch Evaluation and Promotion of Health Research – and the FEDER. Drs. Wirth, Shivappa, and Hébert were supported by grant number R44DK103377 from United States National Institute of Diabetes and Digestive and Kidney Diseases.

Appendix A. Supplementary data

Supplementary data related to this article can be found at http://dx.doi.org/10.1016/j.clnu.2017.06.001.

Abbreviations

BMI	Body-mass Index
CRP	C-reactive protein

DII	Dietary Inflammatory Index
IL	Interleukin
NHANES	National Health and Nutrition Examination Surveys
PIR	Poverty-income-ratio

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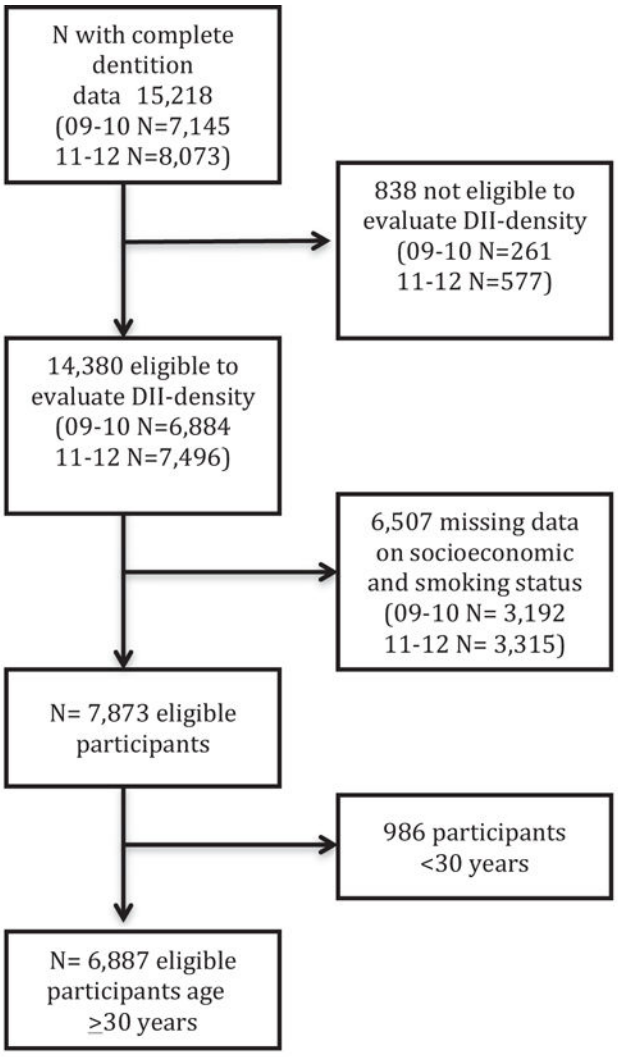

Footnote: The present report was conducted in compliance with the STrengthening the Reporting of OBservational studies in Epidemiology (STROBE) guidelines

Fig. 1.
Sample selection flowchart.

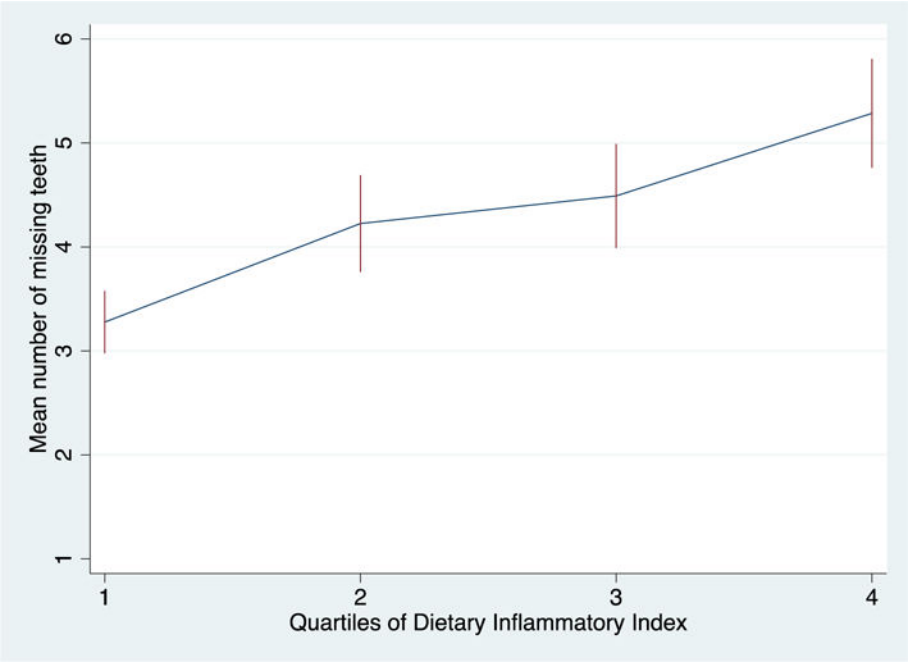

Fig. 2.
Age-adjusted mean tooth loss across quartiles of DII (Dietary Inflammatory Index).

Table 1

Sample characteristics by Quartiles of the Dietary Inflammatory Index (DII) in the 2009–2010 & 2011–2012 continuous NHANES.

	Quartile 1 DII	Quartile 2 DII	Quartile 3 DII	Quartile 4 DII	p-value[b]
N (N weighed)	1722 ($3.6*10^7$)	1722 ($3.2*10^7$)	1722 ($3.4*10^7$)	1721 ($3.5*10^7$)	
DII (SD)	2.46 (1.00)	0.33 (0.40)	0.96 (0.41)	2.47 (0.60)	<0.001
Male (%)	40.9	45.9	53.4	54.5	<0.001
Age	54.8	52.8	51.7	49.5	<0.001
Education (%)					<0.001
<9th grade	5.9	8.0	5.5	5.4	
9–11th grade	7.0	9.6	12.7	15.5	
High school	15.7	21.0	21.9	26.1	
Some college	26.8	28.3	30.2	29.9	
≥College graduate	44.5	33.1	29.5	23.0	
Physical activity (%)					<0.001
High	31.9	32.0	30.0	30.8	
Low	41.4	44.3	49.8	50.3	
Moderate	26.8	23.6	20.3	18.9	
Smoke (%)					<0.001
Current	8.7	14.1	22.3	28.3	
Never	59.0	58.1	51.1	48.3	
Past	32.3	27.8	26.6	23.3	
BMI (%)					<0.001
Underweight	1.5	1.1	0.9	1.0	
Healthy weight	31.7	28.1	22.6	21.2	
Overweight	36.6	37.7	36.4	32.2	
Obese	30.2	33.1	40.1	45.7	
Race (%)					<0.001
Black	6.6	9.0	11.7	14.5	
Hispanic	10.2	16.1	12.4	9.9	
White	74.8	65.6	70.3	71.8	
Other	8.4	9.2	5.7	3.8	

	Quartile 1 DII	Quartile 2 DII	Quartile 3 DII	Quartile 4 DII	p-value[b]
Edentulism (%)[a]	62.8	65.5	66.1	66.1	0.41
Tooth count[a] (SD)	23.4 (7.0)	22.7 (8.4)	22.7 (8.0)	22.3 (8.2)	<0.001
Tooth count in dentates[a] (SD)	24.6 (4.9)	24.4 (5.5)	24.3 (5.5)	23.9 (5.7)	0.05
Energy-intake (SD)					
Sugar	0.21 (0.07)	0.21 (0.08)	0.20 (0.08)	0.23 (0.10)	<0.001
Carbohydrate	0.51 (0.09)	0.49 (0.09)	0.47 (0.09)	0.48 (0.09)	<0.001

Abbreviation: DII Dietary Inflammatory Index; BMI Body Mass Index.

[a]Excluding 3rd molars.

[b]p-values arise from ANOVA for continuous variables or chi-square tests for categorical variables. All analyses include survey-sampling weights; standard errors were calculated based on Taylor series linearization.

Table 2

Adjusted model for tooth loss by quartiles of Dietary Inflammatory Index (DII) in the 2009–2010 & 2011–2012 continuous NHANES (N = 6825).

Variables	Levels	Mean	95% CI	p-value[b]
DII	1st quartile	Ref		
	2nd quartile	0.52	[0.09, 0.96]	0.03[a]
	3rd quartile	0.69	[0.14, 1.23]	0.03[a]
	4th quartile	0.84	[0.24, 1.45]	0.02[a]
Age (years)	Per year	0.23	[0.09, 0.96]	<0.001[a]
Sex	Female	Ref		
	Male	−0.36	[−0.73, 0.01]	0.08
Race/ethnicity	Non-hispanic white	Ref		
	Non-hispanic black	1.48	[0.91, 2.01]	<0.001[a]
	Hispanic	−1.01	[−1.62, −0.39]	0.01[a]
	Other	1.22	[0.65, 1.78]	0.01[a]
Poverty-income-ratio	Not poor	Ref		
	Poor	1.38	[0.76, 1.99]	<0.001[a]
Education	<9th grade	Ref		
	9–11th grade	−1.36	[−2.46, −0.26]	0.03[a]
	High school	−3.05	[−3.99, −2.11]	<0.001[a]
	Some college	−4.66	[−5.57, −3.75]	<0.001[a]
	≥College graduate	−6.09	[−7.10, −5.07]	<0.001[a]
Current smoking	Current	Ref		
	Former	−2.29	[−3.27, −1.32]	<0.001[a]
	Never	−3.62	[0.09, 0.96]	<0.001[a]
Physical activity	High	Ref		
	Moderate	−0.08	[−0.51, 0.36]	0.74
	Low	0.67	[0.21, 1.13]	0.01[a]
Body-mass index (BMI)	Normal weight	Ref		
	Overweight	−0.21	[−0.82, 0.40]	0.52
	Obese	0.02	[−0.74, 0.78]	0.92
Comorbidities[a]	Per number of comorbidities	0.32	[0.03, 0.61]	0.05[a]

Abbreviation: DII Dietary Inflammatory Index.

[a]Obesity; diabetes mellitus; hypercholesterolemia; hypertension.

[b]The level of statistical significance was set at $p < 0.05$.

Table 3

Adjusted model[b] for tooth loss by quartiles of Dietary Inflammatory Index (DII) in subgroups of NHANES participants in the 2009–2010 & 2011–2012 NHANES.

		Age > 50 years (N = 3935)			Lowest two tertiles of carbohydrate intake (N = 3649)		
		Mean	**95% CI**	**p-value[a]**	**Mean**	**95% CI**	**p-value[a]**
DII	1st quartile	Ref			Ref		
	2nd quartile	0.66	[−0.18, 1.50]	0.15	0.67	[−0.07, 1.40]	0.10
	3rd quartile	0.76	[−0.01, 1.52]	0.07	0.54	[−0.22, 1.29]	0.19
	4th quartile	1.19	[0.31, 2.06]	0.02[a]	0.80	[0.17, 1.42]	0.03[a]

Abbreviation: DII Dietary Inflammatory Index; CI Confidence interval.

[a]The level of statistical significance was set at $p < 0.05$.

[b]The model is adjusted for age, sex, race, wealth, education, smoking, BMI, physical activity and comorbidities.