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How does the social “get under the gums”? The role of socio-economic position in the oral-systemic health link

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ABSTRACT

OBJECTIVES: To evaluate the extent of association between systemic inflammation and periodontal disease in American adults, and to assess whether socio-economic position mediated this relationship.

METHODS: We used data from the National Health and Nutrition Examination Survey (NHANES IV) (2001–2010). Systemic inflammation was defined by individual and aggregate (cumulative inflammatory load) biomarkers (C-reactive protein, white blood cell counts, neutrophil counts, and neutrophil: lymphocyte ratio). Loss of attachment and bleeding on probing were used to define periodontal disease. Poverty:income ratio and education were indicators of socio-economic position. Covariates included age, sex, ethnicity, smoking, alcohol, and attendance for dental treatment. Univariate and multivariable logistic regressions were constructed to assess the relationships of interest.

RESULTS: In a total of 2296 respondents, biomarkers of systemic inflammation and cumulative inflammatory load were significantly associated with periodontal disease after adjusting for age, sex, and behavioural factors. Socio-economic position attenuated the association between markers of systemic inflammation and periodontal disease in the fully adjusted model.

CONCLUSION: Socio-economic position partly explains how systemic inflammation and periodontal disease are coupled, and may thus have a significant role in the mechanisms linking oral and non-oral health conditions. It is of critical importance that the social and living conditions are taken into account when considering prevention and treatment strategies for inflammatory diseases, given what appears to be their impactful effect on disease processes.

KEY WORDS: Socio-economic position; social determinants of oral health; oral–systemic health; systemic inflammation; periodontal disease

La traduction du résumé se trouve à la fin de l'article.

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The oral–systemic health link continues to emerge as an area of new interest and study from clinical and public health policy standpoints.[1] This stems largely from this field's significant implications in advancing knowledge on the biological processes linking these groups of diseases, and in innovating strategies for their diagnosis, prevention and treatment. Yet, many of the underlying pathways between oral and systemic conditions remain unknown.

Mounting evidence has shown bidirectional relationships between periodontal disease and several other health conditions, with evidence suggesting that inflammation plays a pivotal role in these relationships.[2–4] However, the risk factors involved in these inflammatory reactions are yet to be elucidated. Indeed, devising novel disease prevention strategies requires identifying these risk factors and understanding how they affect the disease process. For example, oral health policy researchers have suggested that effective disease prevention should target risk factors that are common to both oral and systemic diseases, including health-related behaviours and health-system factors.[5] Nonetheless, there has been ongoing critique of targetting these risk factors given their limited ability in explaining oral and non-oral diseases and related health inequalities.[5,6] Meanwhile, the "fundamental causes" of disease are continually argued to lie within the socio-political factors as the key determinants of health.[5–7] Indeed, this conforms with the general susceptibility view of disease causation, which suggests that there are common, rather than specific risk factors that affect people's vulnerability and susceptibility to a wide range of chronic conditions.[8]

Previous research has proposed that disease is not merely the product of cellular and molecular cascades taking place within the body, but rather the result of an interplay between social factors, such as socio-economic position, and biological ones, such as the immune system.[9,10] These were suggested to interact through various reciprocating pathways, eventually leading to an increased risk of oral and/or systemic conditions.[9]

Given the growing interest in the role of social and living conditions in bringing about disease – most notably, the impact of socio-economic conditions on the mechanisms of

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inflammation – the need to understand the interplay between social and biological factors has continued to gain importance and is now being brought to the forefront of clinical and public health research.

In this study, we aimed to investigate the role of socio-economic position as a structural factor in linking oral and systemic inflammation. Using a nationally representative sample of American adults, we assessed the extent of association between systemic inflammation and periodontal disease, and whether socio-economic factors mediated this relationship.

METHODS

Study design

Data for this research were publicly available and obtained from the Continuous National Health and Nutrition Examination Survey (NHANES IV). NHANES uses a stratified multistage probability sampling design with a sample representative of the non-institutionalized American population. The survey collects a variety of cross-sectional data via questionnaires, physical examinations and laboratory assessments. For this study, we used NHANES IV cycles that spanned the years from 2001 to 2004 and 2009 to 2010, for adults aged 20–85 years who had received a clinical periodontal examination and laboratory assessments. Cycles 2005–2006 and 2007–2008 did not include periodontal examinations and were therefore excluded. Individual participants were excluded if they had reported having diabetes or a cardiovascular condition, or if they had recently experienced an episode of acute infection (e.g., sore throat, common cold), which could contribute to increased inflammation.

Variables

Biomarkers of Systemic Inflammation

Systemic markers of inflammation included C-reactive protein, white blood cell counts, segmented neutrophil counts, and neutrophil:lymphocyte ratio, obtained from laboratory data of C-reactive protein assays and complete blood counts. The choice of these biomarkers was based on their significant role in both periodontal and systemic inflammation. C-reactive protein is an acute phase protein (a direct and quantitative measure of the acute phase reaction) that reflects local and systemic events accompanying inflammatory responses (e.g., vasodilatation, platelet aggregation, neutrophil chemotaxis, and release of lysosomal enzymes). It is associated with several inflammatory conditions, including periodontal disease. Total white blood cell counts are also a measure of systemic inflammation and were suggested to mediate the relationship between systemic and periodontal inflammations.[11] Derived from the differential white blood cell count, neutrophils are cells of the innate immune system that play a crucial role in systemic inflammation and periodontal disease, and which have recently been employed in understanding the oral–systemic health links.[12,13] Neutrophil:lymphocyte ratio has also been used in several studies as an index of systemic inflammation and as a prognostic factor for a number of diseases.[14,15] In addition to these four biomarkers, we created a clustered dichotomous variable for cumulative inflammatory load based on the American Association for Clinical Chemistry cut-off values for neutrophils (7.5×10^9 cells/L) and white blood cells (11.0×10^9 cells/L).[16]

Periodontal Disease Assessment

The NHANES periodontal examination is performed according to a random half-mouth method (excluding third molars). Pocket depths (*P*), clinical attachment loss (AL) and bleeding on probing (BP) measurements were assessed at three sites per tooth (mid-facial, mesial and distal). For the 2009–2010 cycle, a full-mouth examination was performed using six sites per tooth. To standardize measurements across NHANES cycles, we first created variables to indicate the extent of periodontal disease. These were the extent of $P \geq 4$ mm, the extent of AL ≥ 3 mm, and the extent of BP, each calculated as the ratio of sites showing the periodontal characteristic to the total number of sites probed. We then created a binary variable for periodontal disease as having at least 1% of sites with BP and $P \geq 4$ mm, or 1% of sites with BP and AL ≥ 3 mm. These periodontal disease indicators were similar to ones used in previous NHANES studies.[17,18]

Risk Factor Assessment

Poverty:income ratio and years of education were used as indicators of socio-economic position. Poverty:income ratio was calculated in NHANES by dividing family income by the poverty thresholds, specific to family size, as well as the appropriate year and state according to the American Department of Health and Human Services guidelines. If family income was reported as a range value, the midpoint of the range was used to compute the variable. Poverty:income ratio values in NHANES range from 0 to 5, with the cut-off of 1 indicating an individual or family as below the poverty threshold. Education was defined as the highest grade or level of education completed. We dichotomized this variable into <12 years (did not complete high school) and ≥12 years of education (completed high school). Other variables included age, sex, ethnicity, and behavioural factors (smoking status, amount of alcohol consumption, and dental attendance).

Statistical analysis

NHANES uses a complex, multistage probability sampling design to select participants who are representative of the civilian, non-institutionalized US population.

While oversampling of certain population subgroups is generally done in national surveys to increase the reliability and precision of health status indicator estimates for these groups, sample weights are used to produce an unbiased national estimate.[19] Here, we used NHANES-constructed sample weights to take into account survey non-response, oversampling, post-stratification, and sampling error, and accordingly used survey commands in STATA IC 14.1 throughout the analysis. Descriptive statistics were calculated. Testing for the confounding effect of variables was carried out using a backward stepwise process with a cut-off value of 10% change in the coefficient of periodontal disease.

Using univariate logistic regression, we first examined the crude association between the individual and aggregate markers of systemic inflammation and periodontal disease. We then constructed multivariable logistic regression models to assess the extent of association between systemic inflammation and

periodontal disease while accounting for the effects of demographic, behavioural and socio-economic factors. For this, we first adjusted the model for age, sex, and behavioural factors (smoking, alcohol and dental attendance). Socio-economic position indicators were then added to the model to test for their effect.

RESULTS

General characteristics

A total of 2296 respondents were included in this study, after excluding those ineligible for participation. The mean age of respondents was 46.02 ± 16.6 (mean ± SD), with 62% being male (Table 1). Whites, Hispanics and blacks represented 56%, 25% and 16% of the sample respectively. Almost 72% had completed a high school education and were above the poverty threshold with a mean poverty:income ratio of 2.8 ± 1.6 (mean ± SD). Just over 20% of the participants had a total annual household income of <$20,000, whereas 23% had an annual household income of >$75,000. Periodontal disease was observed in 25% of the sample participants. White blood cells, neutrophils and C-reactive protein were shown to have a mean ± SD of 7.29 ± 2.16 ($\times 10^9$ cells/L), 4.31 ± 1.72 ($\times 10^9$ cells/L) and 0.43 ± 0.93 mg/dL respectively. Only 7% of the sample were shown to have above-threshold cumulative inflammatory load.

An increased risk of periodontal disease was observed in males, and in association with age, as well as with behavioural factors such as smoking (OR = 1.66, 95% CI: 1.11–2.49) and alcohol consumption (OR = 1.44, 95% CI: 1.10–1.88). Meanwhile, dental attendance was associated with lower periodontal disease risk (OR = 0.5, 95% CI: 0.3–0.6). No significant differences were observed in periodontal disease risk when comparing ethnic groups.

Table 1. General characteristics of study participants

Characteristics	*n* (%)	Mean ± SD
n = 2296		
Age (years)		46 ± 16.6
Sex (males)	1510 (61.9)	
Annual household income		
<$10,000	146 (6.5)	
$10,000–$19,999	314 (14)	
$20,000–$34,999	464 (20.7)	
$35,000–$54,999	512 (22.8)	
$55,000–$74,999	290 (12.9)	
≥$75,000	512 (22.8)	
Poverty:income ratio		2.8 ± 1.6
Education		
<12 years (no high school)	672 (27.5)	
≥12 years (completed high school)	1766 (72.4)	
Periodontal disease (binary)	602 (24.6)	
Extent of pocket depth (mm)		3.3 ± 9.8
Extent of bleeding from probing		11.8 ± 18.1
Extent of loss of attachment (mm)		10.1 ± 20.6
Neutrophil count ($\times 10^9$ cells/L)		4.3 ± 1.7
White blood cell counts ($\times 10^9$ cells/L)		7.2 ± 2.1
Neutrophil:lymphocyte ratio		2.2 ± 1.2
C-reactive protein (mg/dL)		0.4 ± 0.9
Cumulative inflammatory load	208 (8.5)	
Smoking status (smokers)	1266 (51.9)	
Alcohol consumption*	1114 (45.6)	
Dental attendance†	42.8	

* At least one episode of having more than three drinks per day in the past year.
† Visited the dentist at least once in the past six months.

Socio-economic position associates with systemic inflammation and periodontal disease

Socio-economic position had a protective association with periodontal disease. One unit increase in poverty:income ratio and obtaining >12 years of education reduced periodontal disease risk by 13% and 59% respectively. Similarly, the risk of systemic inflammation was shown to be significantly reduced with higher socio-economic position. Individuals with higher education had less risk of systemic inflammation, as demonstrated by all individual and aggregate biomarkers, with the exception of neutrophil:lymphocyte ratio. Higher poverty:income ratio was associated with reduced C-reactive protein levels (Table 2).

Effect of socio-economic position on the association between systemic inflammation and periodontal disease

Crude analysis showed individual and aggregate markers of systemic inflammation to be significantly associated with periodontal disease. For example, a unit increase in white blood cells, neutrophil counts and cumulative inflammatory load was shown to increase periodontal disease risk by 3.3, 2.1 and 2 times respectively (Table 3, Model 1). A greater extent of loss of periodontal attachment, pocket depth, and bleeding from probing were also shown to be associated with increased white blood cells and neutrophils in the unadjusted models. Meanwhile, C-reactive protein was only associated with the loss of periodontal attachment. Adjusting for age, sex, ethnicity, and behavioural

Table 2. Association between indicators of socio-economic position and biomarkers of systemic inflammation and periodontal disease

	Education OR (95% CI)	Poverty:income ratio OR (95% CI)
Periodontal disease	0.4 (0.2–0.6)*	0.8 (0.7–0.9)*
Systemic inflammatory markers		
WBC	0.3 (0.1–0.7)**	0.3 (0.1–0.8)*
PMN	0.5 (0.2–0.9)*	0.6 (0.3–1.3)†
NLR	0.8 (0.7–1.0)†	1.0 (0.7–1.3)†
CRP	0.4 (0.2–0.7)*	0.38 (0.1–0.7)*
CIL	0.6 (0.4–0.9)*	0.7 (0.4–1.3)†

Note: WBC = white blood cells; PMN = neutrophils; NLR = neutrophil:lymphocyte ratio; CRP = C-reactive protein; CIL = cumulative inflammatory load.
*** $p < 0.001$; ** $p < 0.01$; * $p < 0.05$; † not significant.

Table 3. The effect of socio-economic position on the association between systemic inflammation and periodontal disease

Variables	Periodontal disease		
	Model 1 OR (95% CI)	Model 2 OR (95% CI)	Model 3 OR (95% CI)
WBC	3.3 (1.7–6.3)***	3.2 (1.9–5.3)***	3.0 (1.7–5.2)**
PMN	2.1 (1.2–3.6)**	2.1 (1.3–3.3)**	2.1 (1.3–3.3)**
NLR	1.2 (0.7–2.1)†	1.2 (0.7–1.9)†	1.2 (0.8–1.9)†
CRP	1.1 (1.0–1.3)*	1.1 (1.0–1.3)*	1.0 (0.8–1.1)†
CIL	2.0 (1.0–3.9)*	1.9 (1.0–3.4)*	1.8 (0.9–3.5)†

Note: Model 1: unadjusted; Model 2: adjusted for age, sex, ethnicity and behaviour; Model 3: adjusted for poverty:income ratio and education.
*** $p < 0.001$; ** $p < 0.01$; * $p < 0.05$; † not significant.

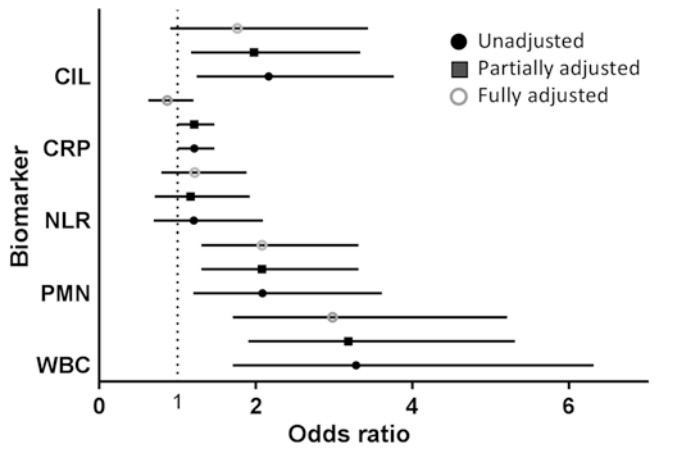

Figure 1. Socio-economic position attenuates the association between systemic inflammation and periodontal disease. Odds ratios (95% CI) of periodontal disease in association with systemic inflammatory markers are shown for the crude model, partially adjusted model (for age, sex, ethnicity and behaviour) and fully adjusted model (for socio-economic position).

factors in multivariable logistic regressions did not attenuate these relationships (Table 3, Model 2). In the fully adjusted model, the effects of poverty:income ratio and education were demonstrated by the attenuation of cumulative inflammatory load and C-reactive protein. While blood cell counts remained statistically significant, they were also partly attenuated by adjusting for socio-economic position (Table 3, Model 3) (Figure 1).

DISCUSSION

In this study, we found periodontal disease risk to be significantly associated with higher levels of systemic inflammation, supporting previous studies that demonstrated a relationship between various inflammatory conditions and periodontal disease.[20–22] This association remained statistically significant after controlling for factors known to trigger both systemic and periodontal inflammation, such as smoking and alcohol consumption and independent inflammatory conditions such as cardiovascular disease and diabetes.

In addition, the systemic inflammation–periodontal disease association was partly attenuated by socio-economic position, indicating the latter's role in the inflammatory processes linking oral and systemic diseases; a role that may outweigh that of health behaviours. Previous research has highlighted the ability of social conditions to "*get under the skin*" to affect one's biological systems and hence the body's ability to fend off disease.[9,10,23] The effect of socio-economic position observed in this analysis supports this concept, where social and economic conditions that determine an individual's position on the social hierarchy can potentially influence immune functions – indicated here by markers of systemic inflammation – and eventually affect one's risk of periodontal inflammation.

Furthermore, our findings are consistent with previous studies emphasizing the role of socio-economic position in linking oral and systemic health outcomes. For example, in a study conducted on middle-aged American army men during the Vietnam War, income and education were shown to be stronger explanatory factors of the relationship between oral health and all-cause mortality compared to health behaviours.[24] Indeed, behavioural explanations of oral and systemic diseases have long been debated by social epidemiologists as factors that lie along the pathway to disease rather than being "causals".[7,25] Many calls to enhance oral disease prevention policies, such as the London Charter on Oral Health Inequalities and the Global Oral Health Inequalities Research Agenda (GOHIRA), have thus focused on the importance of understanding and tackling "upstream" social and economic factors.[26,27]

In this analysis, indicators of socio-economic position were similarly associated with oral and systemic inflammation, where higher income and education reduced the risk of both inflammatory conditions. This similarity supports the notion that oral and systemic diseases share common causals that lie within the social environment, and once again, speaks to the importance of understanding the underlying mechanisms by which the social environment brings about inflammation.

Our study has the advantage of providing evidence on the role of structural and social factors in oral and systemic inflammation by analyzing a large, nationally representative dataset of American adults and by quantifying the impact of socio-economic and behavioural factors in the oral–systemic health connection. It further provides insight into the notion of "biological embodiment", whereby social conditions penetrate the body and interact with its functions to bring about disease. However, a few limitations were encountered in this analysis. Variables on oral health behaviours such as tooth brushing were not available in NHANES IV, and information on the availability of dental insurance was also limited to a few cycles. Thus, it was not possible to include these variables in the analysis. Dental attendance was however used as a proxy to tooth cleaning and dental insurance. This was based on previous findings that showed individuals who had no dental insurance having reported the least number of dental visits, according to both American and Canadian data.[28,29] Also, this study cannot support causal inferencing due to the cross-sectional nature of NHANES IV. Longitudinal studies that can better investigate the causal relationship between social and biological factors in disease outcomes are needed in the future.

While this study aimed to develop a better understanding of the social pathways linking oral and systemic health, more studies will be needed that further investigate other pathways, such as the role of psychosocial stress and healthsystem-related factors in the biological processes leading to oral disease.

Unraveling the mechanisms by which oral and systemic inflammatory diseases are linked has become a critically important yet under-investigated area of biomedical research in general, and dental research in specific. This study has examined how these conditions may be related from a novel angle by adding the social dimension to the equation. Clearly, it is imperative to further investigate how such social factors regulate physiological and pathophysiological processes. This should not only develop a better understanding of how disease occurs but should also aid in developing novel loci for intervention, leading to more effective prevention policies and therapeutic strategies that essentially target the social determinants of health.

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RÉSUMÉ

OBJECTIFS : Évaluer le degré d'association entre l'inflammation systémique et la maladie parodontale chez les Américains adultes et déterminer si le statut socioéconomique intervient dans une telle association.

MÉTHODE : Nous avons utilisé les données de la National Health and Nutrition Examination Survey (NHANES IV) (2001–2010). L'inflammation systémique a été définie par des biomarqueurs (protéine C-réactive, numération des leucocytes, numération des neutrophiles et ratio neutrophiles/lymphocytes) individuels et globaux (charge inflammatoire cumulative). Selon notre définition, la maladie parodontale était caractérisée par la perte d'attache et le saignement au sondage. Le ratio pauvreté/revenu et le niveau d'instruction ont été nos indicateurs du statut socioéconomique. Les covariables étaient l'âge, le sexe, l'ethnicité, le tabagisme, la consommation d'alcool et la réception de soins dentaires. Nous avons construit des modèles de régression logistique univariés et multivariés pour déterminer les relations d'intérêt.

RÉSULTATS : Sur notre échantillon de 2 296 répondants, les biomarqueurs d'inflammation systémique et la charge inflammatoire cumulative présentaient une corrélation significative avec la maladie parodontale après élimination des effets de l'âge, du sexe et des facteurs comportementaux. Le statut socioéconomique atténuait l'association entre les indicateurs d'inflammation systémique et la maladie parodontale dans le modèle entièrement rajusté.

CONCLUSION : Le statut socioéconomique explique en partie l'association entre l'inflammation systémique et la maladie parodontale; il pourrait donc jouer un rôle important dans le mécanisme qui associe les affections buccodentaires et non buccodentaires. Lorsqu'on envisage des stratégies de prévention et de traitement des maladies inflammatoires, il faut impérativement tenir compte des conditions sociales et des conditions de vie, car elles semblent avoir un impact sur les processus morbides.

MOTS CLÉS : Statut socioéconomique; déterminants sociaux de la santé buccodentaire; santé buccodentaire–santé systémique; inflammation systémique; maladie parodontale