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Original Contribution

Diet-Induced Overweight and Obesity and Periodontitis Risk: An Application of the Parametric G-Formula in the 1982 Pelotas Birth Cohort

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We aimed to estimate hypothetical effects of habits (smoking, alcohol consumption, and fat and carbohydrates consumption) combined with diet-induced overweight/obesity on the risk of periodontitis. The risk of any periodontitis, moderate/severe periodontitis, and the combination of bleeding on probing (BOP) and clinical attachment loss (CAL) was estimated using the parametric g-formula in adults aged 31 years from the 1982 Pelotas Birth Cohort in Brazil. Individuals in this cohort have been followed since birth. Hypothetical conditions were set independently for each risk factor and in combination for the entire population. A total of 539 participants had oral examinations in 2013. The cumulative 31-year risk under no intervention was 33.3% for any periodontitis, 14.3%, for moderate/severe periodontitis, and 14.7%, for BOP and CAL. According to our statistical approach, diet-induced overweight/obesity increased the risk of all outcomes: 11% (overweight) and 22% (obesity) higher risk of periodontitis; 12% (overweight) and 27% (obesity) higher risk of moderate/severe periodontitis; 21% (overweight) and 57% (obesity) higher risk of CAL and BOP. When overweight/obesity was combined with other unhealthy habits, the risk was even greater. Our findings suggest that the combination of diet-induced obesity with other risk factors may increase the risk of periodontitis. Further research in the field is required to corroborate our study.

cohort studies; diet; g-formula; interventions; nutritional status; obesity; overweight; periodontal disease

Abbreviations: BMI, body mass index; BOP, bleeding on probing; CAL, clinical attachment loss; OHS, Oral Health Study; RR, risk ratio; SD, standard deviation.

Periodontitis is an infectious inflammatory condition that affects the supporting tissue of teeth (1). In addition to its infectious component, its establishment and progression also depend on the quality of the host immune-system response, which is influenced by health habits and systemic conditions (1–6). Severe periodontitis has been ranked as the sixth most prevalent chronic disease worldwide (7, 8). Furthermore, given the combination of high population growth with greater life expectancy and a significant reduction in tooth loss, an increase in the prevalence of periodontitis is expected (7). This is a major concern for health professionals and policy-makers, because periodontitis has been identified as a potential risk factor for other chronic inflammation-related conditions (e.g., cardiovascular diseases) (9–11).

Overweight and obesity can be defined as diseases in which excess body fat has been accumulated, adversely affecting the health of an individual (12). It is an emerging chronic disease associated with relevant morbidity and mortality (12, 13). In the 1982 Pelotas Birth Cohort, a population-based cohort in Brazil, the prevalence of overweight and obesity has increased over time, as it has elsewhere in the world. In 1986, when participants were aged 4 years, only 7% were considered to have excess weight, whereas in 2012, at age 30 years, the rate had risen to approximately 60% (14). The increase of overweight and obesity in the 1982 Pelotas Birth Cohort participants might be attributed to a variety of factors, predominantly dietary changes (e.g., consumption of ultraprocessed food), as revealed in previous studies (15, 16).

Obesity in general increases the risk of many systemic diseases, such as type 2 diabetes mellitus, cardiovascular disease, and cancer (17, 18).

The association between obesity and periodontal disease has been supported by systematic reviews (19, 20). However, few studies adopted longitudinal designs, and there is a lack of information from middle- and low-income countries (21–23). Some mechanisms have been proposed to explain this complex relationship (19, 20). Excess adipose tissue, by downgrading adiponectin levels, creates a reservoir of circulating proinflammatory mediators inducing chronic inflammation (24). This systemic low-grade inflammation increases the susceptibility of obese subjects to infectious challenges by down-regulating the local immune response (17, 25).

It is plausible that obesity and periodontitis share common risk factors, such as smoking and unhealthy diet (2–5). The influence of the simultaneous occurrence of several risk factors on health indicators is greater than the isolated effect of a single factor, suggesting a clustering effect of the chronic diseases (26). The literature has provided useful information regarding the identification of such clustered behaviors, but little is known about the effects of modifying them. Another difficulty lies in the fact that some exposures, such as obesity, cannot be allocated in a randomized intervention. Hence, longitudinal observational studies provide the best approach for investigating this question.

In the medical and dental fields, studies investigating the association between obesity and systemic chronic conditions in later life have rarely considered the cumulative nature of obesity during the life course. Additionally, they have not employed analytical approaches that account for time-varying confounders or distinguish between confounders and mediators (27). This may have led to biased or controversial findings (28).

Statistical methods have been proposed for incorporating time-varying confounders and for handling mediation appropriately using observational studies (29). These methods fall under the heading of g-formula (30, 31) and can be especially useful when the explanatory variables are measured at several different points in time for each subject. This approach also allows the estimation of the population risk of a disease under hypothetical scenarios, providing useful information for health professionals and policy-makers (32). The parametric g-formula uses a counterfactual framework. When studying obesity as exposure, studies have shown that counterfactuals cannot be defined solely using the cutoff points for categories of body mass index (BMI) (33, 34). However, a definition of obesity or a definition of BMI that is specific to an intervention may be used. In this study we used the definition of diet-induced overweight/obesity. Here the counterfactual can be defined as 0 for good/recommended diet for healthy living and 1 for unhealthy diet. In this way, we can have a clear definition of the counterfactual. Hereafter, we use the terms overweight and obesity to refer to diet-induced overweight and obesity.

In this study we aimed to investigate the effects of obesity and overweight on the risk of periodontal disease in adulthood. We also sought to estimate the effect of unhealthy habits combined with diet-induced overweight and obesity on periodontitis by simulating hypothetical scenarios using the parametric g-formula. According to previous investigations, life-course obesity and overweight can lead to a greater risk of periodontal disease development (35). Thus, the null hypothesis of this study is that neither overweight nor obesity will influence the risk of periodontitis.

METHODS

The 1982 Pelotas Birth Cohort

In 1982, the 3 maternity hospitals in Pelotas, Brazil, were visited daily, and those children whose parents lived in the urban areas of the city ($n = 5{,}914$) were examined and their mothers were interviewed (14). Since then, those individuals have been examined on several occasions. In 2012, all individuals aged 30 years were contacted for interview and health examination, including anthropometric measurements and blood tests ($n = 3{,}701$). These interviews covered issues related to socioeconomic condition, dietary habits, smoking, alcohol consumption, and health conditions. More details can be found elsewhere (14).

In 1997, when participants were aged 15 years, 900 of the 5,914 individuals were randomly selected for the Oral Health Study (OHS) nested in the larger study. Of the 900 participants, 888 were clinically examined. In 2006, at age 24 years, these 888 participants were contacted for another oral health examination; 720 were examined. In 2013, the same 888 individuals were contacted for a third oral health assessment. All teeth were examined for the presence of periodontal disease (bleeding on probing (BOP) and assessment of probing depth and gingival margin level to identify clinical attachment loss (CAL)), dental caries, and other dental and oral conditions. Six dentists were trained, and they performed the oral examinations at the participants' homes. Dentists underwent theoretical and practical training on 25 individuals. The lowest intraclass correlation coefficient for pocket depth and gingival margin level was 0.85. Methodological aspects of the OHS in the 1982 Pelotas Birth Cohort Study are discussed in depth elsewhere (36). This study was approved by the ethics committee of the Federal University of Pelotas. Written informed consent was obtained from all enrolled individuals.

Outcomes

Periodontal examinations comprised a full-mouth probing at 6 sites per tooth using a periodontal probe (PCP-2; Hu-Friedy Manufacturing Company, Rotterdam, the Netherlands). Two sets of criteria were adopted for this study. The presence and severity of periodontitis were categorized according to the American Academy of Periodontology and the Center for Diseases Control and Prevention (37) as a participant being healthy or having mild, moderate, or severe periodontitis. Following that, 2 different dichotomous variables were created. The first variable distinguished all those individuals with any periodontitis compared with those considered healthy. The second variable was dichotomized to compare the individuals with moderate/severe periodontitis with those who were healthy or presented mild periodontitis, which was the reference category. The presence of periodontitis was also assessed according to Baelum

and Lopez (38) as a participant having at least 1 site with simultaneous occurrence of CAL ≥4 mm and BOP.

Nutritional status assessment

During each visit, subjects were weighed and their heights measured. To estimate the cumulative effects of obesity on periodontitis, we used different measures of obesity for the measurements taken at different ages (z scores for measurements taken at ages 4 years and 15 years; BMI for measurements taken at ages 23 years and 30 years). Obesity at age 4 years was defined according to the following cutoffs: eutrophic (z score for age and sex ≤2 standard deviations (SD)), overweight (z score for age and sex >2–≤3 SD), and obese (z score for age and sex >3 SD). The adoption of these cutpoints was based on recommendations from the World Health Organization Growth Reference Group and the European Childhood Obesity Group (39, 40). Also, we performed a preliminary analysis using definitions for age 4 years of a z score for age and sex >1 SD and ≤2 SD (overweight) and a z score for age and sex >2 SD (obese), and no relevant changes in our results were noted (41).

For age 15 years, the categories were defined as eutrophic (z score for age and sex ≤1 SD), overweight (z score for age and sex >1–≤2 SD), and obese (z score for age and sex >2 SD) (42). At ages 23 and 30, BMI was calculated as weight divided by the square of height (kg/m^2). Subjects were classified as eutrophic (<25), overweight (≥25 and <30), or obese (≥30) (12). Portable scales (Uniscale; Seca GmbH & Co, Hamburg, Germany) and locally built anthropometers were used to assess weight and height, respectively. These measures were obtained using the techniques proposed by Lohman et al. (43). Further methodological details are found elsewhere (44).

Covariates

Baseline covariates. The following variables collected at baseline, in 1982, were set as baseline confounders: sex, maternal schooling at birth (≤8 years; ≥9 years), and household income at birth (categorized into tertiles and converted to a dichotomous variable in which the second and third tertiles were grouped) (45).

Time-varying variables. Potential pathways to periodontal disease were identified through a directed acyclic graph (46). Based on the literature, we decided to include a set of time-varying covariates in the analyses: smoking status (current or former smoker; never smoker), type 2 diabetes mellitus (blood glucose level of ≥126 mg/dL), and hypertension (systolic blood pressure of ≥140 mm Hg and/or diastolic blood pressure of ≥90 mm Hg). Alcohol consumption was assessed in grams per day. Percentage of dietary energy from fat and carbohydrate consumption was assessed through a semiquantitative, interviewer-administered food frequency questionnaire (47). The variables "alcohol consumption" and "fat and carbohydrate consumption" were categorized into tertiles and converted to a dichotomous variable in which the first and second tertiles were grouped. Figure 1 displays a simplified directed acyclic graph demonstrating possible causal relationships between the variables. A more detailed figure is available in Web Figure 1 (available at http://aje.oxfordjournals.org/).

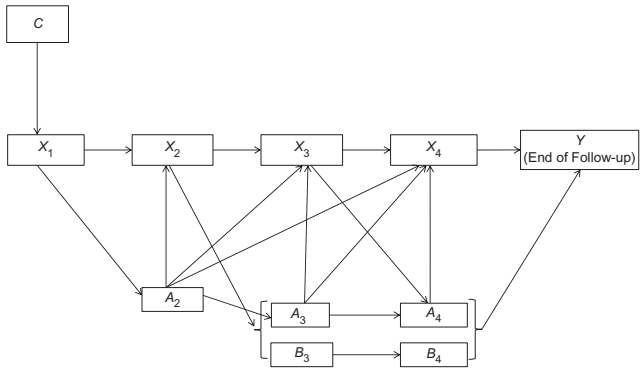

Figure 1. Simplified directed acyclic graph depicting the relationship between time-varying exposure, outcomes, and time-varying covariates, 1982 Pelotas Birth Cohort, Brazil, 1982–2013. *X* represents time-varying exposure (obesity), *Y* represents outcomes (periodontal disease), and *A* (smoking status) and *B* (type 2 diabetes mellitus, hypertension, alcohol consumption, dietary habits) represent time-varying covariates. *C* represents baseline confounders.

Hypothetical changes based on risk factors

Based on the literature from randomized controlled trials and observational studies, we defined 15 hypothetical scenarios using the time-varying variables to analyze the potential effects of overweight, obesity, smoking, high consumption of alcohol, and high consumption of fat and carbohydrates on periodontitis for the entire population. We decided not to directly modify diabetes and hypertension in the hypothetical interventions, because those conditions can be indirectly modified by the other specified risk factors (e.g., diet-induced reduction in weight or reduction in alcohol consumption). Table 1 shows the covariates used to model the incidence of periodontitis in this study.

Data analyses

The parametric g-formula was used to estimate the standardized risk of periodontal disease under hypothetical interventions in the population. This analytical approach has been used previously with different outcomes, such as cardiovascular disease (48) and diabetes (49).

The Monte Carlo approach was used to simulate hypothetical interventions. For a simulated hypothetical cohort, we used a sample of size 10,000, where these samples were drawn from a known probability distribution (e.g., normal). Using these samples we estimated the relative risk of periodontitis and the effects of intervention. The bootstrap method was used to estimate the standard errors as well as the confidence intervals of the relative risks estimated in the hypothetical interventions. For this later estimation, we used 1,000 resamples of size 10,000.

Table 1. Covariates Used to Model the Incidence of Periodontitis in the 1982 Pelotas Birth Cohort, Brazil, 1982–2013

Variables	Years Assessed	Type of Model When Used as a Dependent Variable	Functional Form When Used as a Predictor
Nonmodifiable			
Time period		Not predicted	Period indicators
Sex	1982	Not predicted	Indicator
Household income	1982	Not predicted	Indicator
Maternal schooling	1982	Not predicted	Indicator
Modifiable			
Nutritional Status	1986, 1997, 2005, 2012	Nested logistic	Categories[a]
Smoking	1997, 2005, 2012	Logistic	Indicator
Alcohol	2005, 2012	Logistic	Indicator
Diet	2005, 2012	Logistic	Indicator
Indirectly Modifiable			
Hypertension	2005, 2012	Logistic to failure	Indicator
Diabetes	2005, 2012	Logistic to failure	Indicator

[a] Categories were eutrophic, overweight, and obese and defined according to body weight *z* score or body mass index.

The values of time-varying covariates for each time interval were drawn from the distribution estimated via regression models after setting obesity and other risk factors to the values specified by each specific scenario. Because some time-varying covariates were not measured in all visits, only the most recent measurement was used, and a term for interaction between the most recent measurement and the time since that measurement was included in the model.

For each model, we estimated the outcome risk associated with a respective hypothetical intervention. Additionally, we set the risk under no intervention as the reference category, and we estimated the population risk ratio for each selected scenario. All analyses were conducted using SAS, version 9.4 (SAS Institute, Inc., Cary, North Carolina), and the GFORMULA macro implemented by Taubman et al. (32), available at http://www.hsph.harvard.edu/causal/software. Code used for our analysis is provided in the Web Appendix 1.

RESULTS

Of the 888 individuals interviewed for the OHS in 1997, 539 individuals were examined in 2013. Thus, the present analysis was conducted using only 539 individuals (61% of the 1997 OHS sample). A flow chart of the sample selection is displayed in Figure 2. The observed risks of having mild, moderate, or severe periodontitis at age 31 were 20%, 13%, and 1%, respectively. The observed risk of any type of periodontitis was 34%, and the observed risk of moderate or severe periodontitis was 14%. The observed risk of having at least 1 site with the combination of CAL and BOP was 15%. Table 2 shows the distribution of periodontal disease according to sociodemographic characteristics and nutritional status during the life course. Information regarding other health-related characteristics is shown in Web Tables 1 and 2.

The simulated risks under no intervention on any type of periodontitis, moderate or severe periodontitis, and the combination of CAL and BOP were 33%, 14%, 15% respectively. Table 3 shows the estimated effects of different hypothetical interventions on periodontitis. Overall, both overweight and obesity increased the risk of any periodontitis (risk ratio

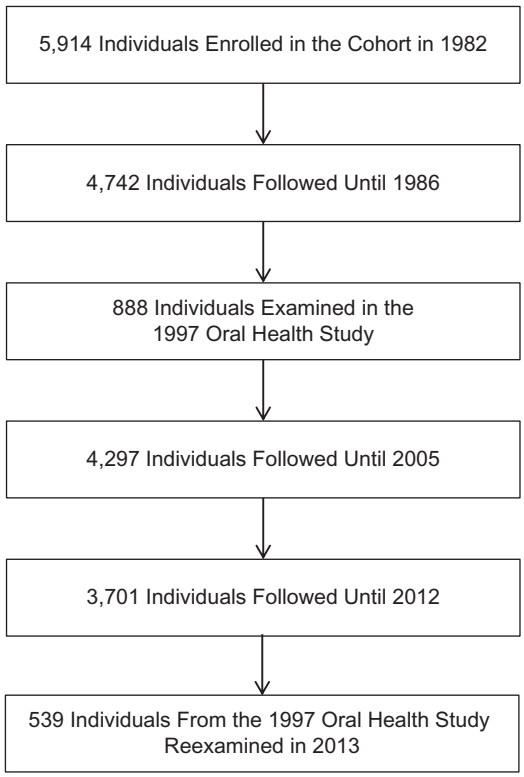

Figure 2. Flow chart depicting selection of the sample, 1982 Pelotas Birth Cohort, Brazil, 1982–2013.

Table 2. Sociodemographic and Anthropometric Characteristics of the Sample During the Life Course According to Periodontal Outcomes ($n = 539$), 1982 Pelotas Birth Cohort, Brazil, 1982–2013

Characteristic	Participants With Periodontitis on Examination in 2013		
	Any Periodontitis, %	Moderate/Severe Periodontitis, %	CAL and BOP, %
Baseline Covariates			
Sex			
Male	42.9	18.3	18.0
Female	31.6	10.1	12.4
Household income			
First tertile	39.2	15.7	15.9
Second and third tertiles	33.1	11.0	13.9
Maternal schooling			
≤8 years	32.3	14.4	14.8
≥9 years	31.9	13.9	16.7
Time-Varying Exposure			
Weight category by *z* score (1986)[a]			
Eutrophic	36.5	14.4	15.5
Overweight	46.7	13.3	5.3
Obese	25.0	0	12.5
Weight category by *z* score (1997)			
Eutrophic	34.4	13.3	14.7
Overweight	50.0	21.6	20.5
Obese	37.0	11.0	12.2
BMI category (2005)[b]			
Eutrophic	34.2	13.1	12.9
Overweight	43.1	17.2	22.1
Obese	45.2	14.4	19.4
BMI category (2012)			
Eutrophic	29.3	9.9	8.2
Overweight	45.8	19.9	24.4
Obese	46.6	20.4	26.8

Abbreviations: BMI, body mass index; BOP, bleeding on probing; CAL, clinical attachment loss; SD, standard deviation.

[a] Weight categories at age 4 years by *z* score were eutrophic (*z* score for age and sex ≤2 SD), overweight (*z* score for age and sex >2–≤3 SD), and obese (*z* score for age and sex >3 SD). At age 15 years, the definitions were eutrophic (*z* score for age and sex ≤1 SD), overweight (*z* score for age and sex >1–≤2 SD), and obese (*z* score for age and sex >2 SD).

[b] Body mass index was calculated as weight (kg)/height $(m)^2$, and the categories were eutrophic (<25), overweight (≥25 and <30), or obese (≥30).

(RR) = 1.11 for overweight and RR = 1.22 for obesity). Except for alcohol consumption, the other risk factors also increased the risk of periodontitis. When hypothetical risk factors were combined with overweight and obesity, the risk of periodontitis increased. Individuals who were overweight smokers with high consumption of fat and carbohydrates and of alcohol had a 44% higher risk of periodontitis; those with obesity in combination with smoking and high consumption of fats, carbohydrates, and alcohol had an almost 62% higher risk of periodontitis compared with those under no intervention.

Table 4 shows the risk of moderate and severe periodontitis. Individuals who were overweight and those who were obese had a greater risk of moderate/severe periodontitis (RR = 1.12 for overweight and RR = 1.27 for obesity). Similarly, smoking increased the risk (RR = 1.18), whereas diet had no influence and alcohol consumption slightly reduced the risk. The combination of overweight with smoking increased the risk of moderate/severe periodontitis to 32%; when obesity was associated with smoking, the risk was even greater (RR = 1.48). Compared with no intervention, the risk ratio for being overweight, a smoker, and consuming a high amount of fat and carbohydrates was 1.35, and for those who were obese in combination with those other factors it was 1.52.

Table 3. Periodontitis Risk Under Hypothetical Interventions, 1982 Pelotas Birth Cohort, Brazil, 1982–2013

Intervention	31-Year Risk of Periodontitis, %[a]	95% CI	Population RR	95% CI	Population RD, %	95% CI	No. Needed to Intervene
No intervention	33.29	26.99, 40.25	1.00	Referent	0		
Overweight	36.93	30.30, 44.04	1.11	0.94, 1.35	3.64	−2.44, 10.77	27
Obesity	40.72	27.98, 46.63	1.22	1.01, 1.67	7.44	−4.66, 22.21	13
Smoking	35.84	25.31, 48.82	1.08	0.80, 1.39	2.56	−6.31, 12.42	39
Unhealthy diet	36.58	28.49, 46.20	1.10	0.83, 1.49	3.29	−6.50, 15.15	30
High alcohol consumption	31.80	22.33, 42.46	0.96	0.72, 1.27	−1.48	−9.89, 8.37	NA
Overweight and smoking	39.60	27.76, 52.58	1.19	0.88, 1.67	6.31	−4.25, 19.94	16
Overweight and unhealthy diet	42.42	33.76, 52.48	1.27	0.93, 1.72	9.14	−2.48, 20.58	11
Overweight and high alcohol consumption	35.39	26.44, 44.45	1.06	0.77, 1.46	2.10	−0.88, 2.76	48
Overweight, smoking, and unhealthy diet	45.09	32.07, 59.33	1.35	0.92, 1.99	11.80	−3.03, 26.95	8
Overweight, smoking, unhealthy diet, and high alcohol consumption	47.95	33.59, 62.10	1.44	0.98, 2.13	14.66	−0.58, 31.07	7
Obesity and smoking	43.44	28.46, 61.90	1.30	0.84, 2.00	10.15	−5.80, 30.22	10
Obesity and unhealthy diet	48.47	33.47, 62.53	1.46	0.92, 2.07	15.18	−2.84, 31.17	7
Obesity and high alcohol consumption	39.13	28.63, 50.83	1.18	0.78, 1.74	5.85	−8.37, 20.70	17
Obesity, smoking, and unhealthy diet	51.06	33.49, 68.16	1.53	0.97, 1.40	17.77	−0.89, 38.78	6
Obesity, smoking, unhealthy diet, and high alcohol consumption	53.85	36.51, 70.87	1.62	1.04, 2.45	20.56	1.36, 41.21	5

Abbreviations: CI, confidence interval; RD, risk difference; RR, risk ratio.
[a] The observed risk was 33.39%.

Alcohol consumption reduced the risk in all hypothetical interventions.

Overweight and obesity also increased the risk of CAL and BOP (Table 5). Individuals who were overweight had a 21% higher risk than those under no intervention, while those who were obese had a 50% higher risk. Smokers had an 11% greater risk of CAL and BOP, but diet did not materially change the risk. High consumption of alcohol reduced the risk of CAL and BOP (RR = 0.88). The risk ratio for overweight combined with smoking was 1.50 and for obesity combined with smoking was 2.01. When unhealthy diet was included, overweight individuals and smokers had a 57% greater chance of CAL and BOP, and those who were obese had more than twice the risk (RR = 2.09). In the combined interventions, alcohol consumption did not materially change the risk.

We replicated our analyses using a random order for the covariates in the model. The risks under no intervention and the other parameters were not substantially altered, thus indicating the robustness of our models.

DISCUSSION

To the best of our knowledge, this is the first study to have investigated the influence of obesity/overweight on periodontal health using a longitudinal design and adjustment for time-varying covariates. The present study's findings suggest a dose-response relationship between overweight or obesity during the life course and the occurrence of periodontitis in adulthood. Moreover, a combination of detrimental health behaviors with overweight/obesity appears to increase the risk of periodontitis in this population. These findings reject the null hypothesis that neither overweight nor obesity influence the risk of periodontitis. A positive association between obesity/overweight and periodontitis has been demonstrated (20, 21). However, the source of evidence presented limitations regarding the analytical approach employed. To our knowledge, no study has addressed either childhood obesity or the cumulative effects of obesity during the life course on periodontitis in later life. Obesity developed in early life could be a result of unhealthy behaviors that are set in this younger age and might have repercussions later in life.

Previous reports could not address the effects of overweight and obesity on periodontitis because a bidirectional association between those conditions could exist. Our results clearly showed the cumulative effects of both overweight and obesity on periodontitis in later life. It has been hypothesized that low-grade inflammation significantly alters the immune-system response threshold, making obese/overweight individuals more susceptible to infectious diseases than eutrophic ones (17, 50).

Our findings also suggest that the combination of multiple detrimental health behaviors influences the risk of periodontitis. The behaviors involved are well-known risk factors for cardiovascular disease, cancer, and premature mortality (51). Individuals who accumulated unhealthy behaviors had

Table 4. Moderate and Severe Periodontitis Risk Under Hypothetical Interventions, 1982 Pelotas Birth Cohort, Brazil, 1982–2013

Intervention	31-Year Risk of Periodontitis, %[a]	95% CI	Population RR	95% CI	Population RD, %	95% CI	No. Needed to Intervene
No intervention	14.30	9.59, 18.56	1.00	Referent	0		
Overweight	16.16	11.17, 20.75	1.12	0.84, 1.50	1.86	−2.44, 6.19	56
Obesity	18.28	10.99, 28.99	1.27	0.76, 2.14	3.98	−4.44, 14.08	26
Smoking	16.96	8.76, 26.70	1.18	0.67, 2.08	2.66	−5.69, 11.50	39
Unhealthy diet	14.80	7.67, 25.09	1.03	0.59, 1.65	0.50	−7.42, 9.66	235
High alcohol consumption	13.55	7.75, 20.27	0.94	0.63, 1.38	−0.75	−5.80, 4.84	NA
Overweight and smoking	18.94	10.58, 29.32	1.32	0.69, 2.50	4.64	−4.84, 16.22	22
Overweight and unhealthy diet	16.62	9.00, 26.22	1.16	0.59, 1.98	2.32	−7.16, 11.54	45
Overweight and high alcohol consumption	15.27	9.36, 22.15	1.06	0.67, 1.75	0.97	−5.64, 7.70	111
Overweight, smoking, and unhealthy diet	19.46	9.16, 32.24	1.35	0.55, 2.80	5.16	−7.66, 19.61	20
Overweight, smoking, unhealthy diet, and high alcohol consumption	18.44	8.25, 33.56	1.28	0.47, 3.00	4.14	−8.88, 21.04	25
Obesity and smoking	21.24	11.84, 33.13	1.48	0.66, 3.27	6.94	−5.51, 21.64	15
Obesity and unhealthy diet	18.77	9.59, 30.80	1.31	0.59, 2.40	4.47	−7.55, 17.03	23
Obesity and high alcohol consumption	17.32	9.99, 27.67	1.21	0.65, 2.26	3.02	−6.39, 13.03	34
Obesity, smoking, and unhealthy diet	21.79	10.31, 37.85	1.52	0.57, 3.36	7.49	−7.28, 24.98	13
Obesity, smoking, unhealthy diet, and high alcohol consumption	20.71	8.89, 38.14	1.44	0.52, 3.28	6.41	−8.89, 25.57	16

Abbreviations: CI, confidence interval; RD, risk difference; RR, risk ratio.
[a] The observed risk was 14.31%.

a higher risk of periodontitis, indicating that this oral condition is similarly influenced by systemic factors. Because obese individuals are more likely to experience neglected health care, the coexistence of poor health behaviors is expected, increasing the risk of periodontitis. Other investigators have demonstrated that the combined effect of multiple risk factors on mortality is greater than individual risk factors, suggesting a cluster effect (51, 52). The importance of such findings to the public health perspective is beyond dispute, given that poor behaviors frequently coexist.

Smoking, diet, and alcohol consumption are closely related to obesity and periodontitis. Smoking has been identified as a risk factor for chronic periodontitis in adults (53) with a cumulative effect on periodontal attachment loss; thus, it is expected that the older the individual is, the greater the effect. Given the age of the individuals enrolled in this cohort, this fact could explain a reduced influence of smoking on periodontitis in our study. Excessive carbohydrate and fat consumption may lead to increased inflammatory activity and oxidative stress inducing a hyperinflammatory state, which has been associated with periodontitis and obesity (54, 55). Although information related to the quality of carbohydrate (simple or complex) and types of fat (saturated, unsaturated, trans) was not considered in the present analyses, a high consumption of ultraprocessed food with a predominance of simple carbohydrates and saturated fat has been observed in this cohort (16). Furthermore, the consumption of food rich in polyunsaturated fat, such as nuts and fishes, is relatively low in Brazil; the typical Brazilian diet is composed of rice, black beans, white bread, and butter (56). The association between alcohol consumption and periodontal disease, however, is not conclusive (57, 58). Studies revealed that alcohol could have both protective and detrimental effects on periodontal tissues depending on the dose at which it is consumed (59).

Our study had several strengths that should be highlighted. First, the prospective longitudinal data originated from a population-based birth cohort that has provided robust information since early life. The use of repeated measures for exposure and risk factors also supported the robustness of our findings. Second, the use of the parametric g-formula allowed us to appropriately adjust our analyses for time-varying confounders as well as estimating the effects of joint hypothetical simulations, which is relevant for policy-makers and clinicians (30, 60). Our methodological choice was based on the rising prevalence of overweight/obesity attributed to dietary changes in this cohort (15, 16). According to previous studies, increase in the consumption of ultraprocessed food in this cohort was responsible for the incidence of overweight and obesity among participants. Third, the similarity between the simulated risks of the different outcomes with the corresponding observed risks suggests an absence of model misspecification in the calculation of estimates using the parametric g-formula. Finally, we should emphasize the reliability of our measures: Data were obtained from clinical examinations in addition to the use of established classifications for periodontitis.

Table 5. Risk of Clinical Attachment Loss and Bleeding on Probing Under Hypothetical Interventions, 1982 Pelotas Birth Cohort, Brazil, 1982–2013

Intervention	31-Year Risk of Periodontitis, %[a]	95% CI	Population RR	95% CI	Population RD, %	95% CI	No. Needed to Intervene
No intervention	14.70	9.36, 19.19	1.00	Referent	0		
Overweight	17.78	10.82, 24.72	1.21	0.83, 1.63	3.08	−2.42, 8.63	32
Obesity	21.97	11.07, 34.84	1.50	0.73, 2.58	7.27	−3.95, 21.53	14
Smoking	16.24	3.43, 31.37	1.11	0.23, 2.64	1.54	−12.60, 18.87	64
Unhealthy diet	14.39	7.56, 22.37	0.99	0.52, 1.61	−0.31	−7.01, 7.15	NA
High alcohol consumption	12.80	6.28, 22.84	0.88	0.49, 1.52	−1.90	−8.53, 6.96	NA
Overweight and smoking	21.96	7.54, 38.58	1.50	0.54, 3.01	7.26	−7.33, 25.92	14
Overweight and unhealthy diet	17.45	9.20, 26.95	1.19	0.65, 2.15	2.75	−5.80, 11.95	36
Overweight and high alcohol consumption	16.86	9.71, 25.65	1.15	0.72, 1.82	2.16	−4.57, 10.10	46
Overweight, smoking, and unhealthy diet	22.99	7.25, 44.69	1.57	0.53, 3.31	8.29	−6.54, 24.93	12
Overweight, smoking, unhealthy diet, and high alcohol consumption	21.82	8.05, 43.23	1.49	0.57, 3.22	7.12	−5.71, 26.80	14
Obesity and smoking	29.44	7.49, 57.23	2.01	0.54, 4.65	14.74	−6.98, 43.19	7
Obesity and unhealthy diet	21.59	10.46, 37.61	1.47	0.67, 3.28	6.89	−5.45, 24.17	14
Obesity and high alcohol consumption	22.29	8.60, 38.32	1.52	0.72, 1.82	7.59	−5.65, 25.23	13
Obesity, smoking, and unhealthy diet	30.63	9.33, 68.01	2.09	0.66, 5.01	15.93	−5.05, 33.80	6
Obesity, smoking, unhealthy diet, and high alcohol consumption	30.84	10.74, 64.90	2.10	0.79, 4.79	16.14	−2.92, 49.20	6

Abbreviations: CI, confidence interval; RD, risk difference; RR, risk ratio.
[a] The observed risk was 15.15%.

Our study was not free of limitations. First, we obtained measures of alcohol consumption and diet only from early adulthood. Because some detrimental habits start in adolescence, this could underestimate the effects of such habits in the hypothetical scenarios. Second, we used BMI to classify the nutritional status of the individuals as recommended by the World Health Organization. Previous studies have demonstrated that waist circumference is a more reliable measurement, because abdominal obesity, independent of general obesity, is associated with a higher risk of chronic diseases (61, 62). Hence, abdominal obesity should be included in future studies. Third, our estimates should not be extrapolated to other populations with different distributions of risk factors, because the g-formula standardizes the risk to the distribution of risk factors in the particular population under study.

Regardless of the limitations, our study demonstrated the influence of increasing overweight/obesity on periodontitis in adults from a Brazilian birth cohort. Severe periodontitis is ranked among the 10 most prevalent chronic conditions in the global burden of diseases (7, 8), and the increase of obesity will further add to the burden of periodontitis. Our findings suggest that the combination of diet-induced obesity with other risk factors may increase the risk of periodontitis. However, further research in the field is required in order to corroborate our study. Even though our results should be carefully considered from the clinical perspective, they suggest the adoption of a common risk approach as a rationale to prevent periodontal disease.

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