J Clin Periodontol 2004; 31: 484–488 doi: 10.1111/j.1600-051X.2004.00503.x
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Journal of Clinical Periodontology

Alcohol consumption and periodontal disease

The Third National Health and Nutrition Examination Survey

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Tezal M, Grossi SG, Ho AW, Genco RJ. Alcohol consumption and periodontal disease the third National Health and Nutrition Examination Survey. J Clin Periodontol 2004; 31: 484–488. doi: 10.1111/j.1600-051X.2004.00503.x. © Blackwell Munksgaard, 2004.

Abstract

Objective: This study was carried out to evaluate the effect of alcohol consumption on the severity of periodontal disease.

Material and Methods: This cross-sectional study employed 13,198 subjects of the Third National Health and Nutrition Examination Survey (NHANES III) aged 20 and older who have at least six natural teeth. Alcohol intake was represented both as a continuous variable and dichotomized using 5, 10, 15, and 20 drinks/week as cut-points. Periodontal disease was represented by clinical attachment loss (CAL) and was assessed both as a continuous variable and dichotomized as <1.5 mm and $\geqslant 1.5$ mm. Independent effect of alcohol on CAL was assessed by weighted multiple linear and logistic regression analyses adjusting simultaneously for the effects of age, gender, race, education, income, smoking, diet, diabetes, gingival bleeding, number of remaining teeth.

Results: There was a significant linear relationship between number of drinks per week and log CAL ($p = 0.0001$). Odds ratios for the risk of attachment loss using 5, 10, 15, and 20 drinks/week as cut-points were 1.22 [1.02–1.47], 1.39 [1.13–1.71], 1.54 [1.22–1.93], and 1.67 [1.25–2.23], respectively.

Conclusion: Alcohol consumption may be associated with increased severity of CAL in a dose-dependent fashion. Prospective studies and studies of mechanism are needed to confirm the role of alcohol as a risk factor for periodontal disease.

Key words: alcohol; epidemiology; periodontal disease

Accepted for publication 4 August 2003

Alcohol consumption is part of the majority (90%) of people's life, although the amount, frequency and pattern vary. Men (40–50%) have temporary alcohol-induced problems, 10% of men and 3–5% of women develop alcoholism (Novacek et al. 1995).

The effect of alcohol on periodontal disease has been assumed to be the result of self-neglect due to chronic alcohol consumption and most studies have examined only the effect of alcoholism on oral tissues (Larato 1972, Movin 1981, Novacek et al. 1995, Sakki et al. 1995). The effect of different levels of alcohol intake that does not necessarily reach ''alcoholism'' levels on periodontal tissues has not been assessed adequately.

Supported by USPHS grant DE04898.

We previously studied the effect of alcohol on periodontium in the Erie County study population (Tezal et al. 2001). Results from that study indicated that self-reported alcohol consumption was significantly related to gingival inflammation and attachment loss after controlling for major confounders, and that this relationship was dose dependent. In the present study, we examined the same relationship in a different population performed by different methodology to assess whether the results found in Erie County population could be confirmed.

The purpose of this study was to assess the relationship between alcohol intake and severity of periodontal disease in a large probability sample of U.S. population.

Materials and Methods

A secondary analysis of the cross-sectional data from the Third National Health and Nutrition Examination Survey (NHANES III) was performed. The NHANES III employed a multistage, stratified, clustered sampling, representing the total civilian, non-institutionalized population, two months of age and over, in the 50 states and the District of

Colombia of the United States. More detailed information on NHANES III can be found in the Plan and Operation of the Third National Health and Nutrition Examination Survey (National Center for Health Statistics 1994). Inclusion criteria for the present study were the age 20 and older, and the presence of at least six natural teeth. A total of 13,198 subjects who met these criteria were included.

Number of drinks in the past 12 months, assessed by means of interviewed questionnaires, was calculated multiplying the number of drinks per day on a drinking day by the number of days the subject consumed alcohol. Number of drinks per week was obtained dividing this number by 52. Alcohol intake was analyzed separately both as a continuous variable and dichotomized using cut-points of 5, 10, 15, and 20 drinks per week.

Severity of periodontal disease was represented by clinical attachment loss (CAL), and was used both as a continuous variable and dichotomized using 1.5 mm as the cut-point, which is the upper quartile of its distribution in the NHANES III population. The measurements were performed using National Institute for Dental Research (NIDR) periodontal probe, on buccal and mesiobuccal sites of each tooth on randomly assigned one upper and one lower quadrant selected at the beginning of the examination. CAL was derived from two measurements: (1) the distance from the free gingival margin (FGM) to the cemento-enamel junction (CEJ), and (2) the distance from the FMG to the bottom of the sulcus (probing depth (PD)). CAL was calculated by subtracting the distance FGM–CEJ from PD. These measurements were performed in mobile examination centers (MECs). Examiners received formal training and calibration before and during the study. Intra-examiner reliability assessments were based on replicate examinations conducted on random recall samples of roughly 20 study participants at each of the 89 NHANES III survey locations. Inter-examiner reliability was evaluated by comparing each examiner with the ''gold standard'' examiner (National Center for Health Statistics 1994).

Information on covariates was obtained by household interviews and by examinations at MEC and at houses, and included age (years), smoking (packyears), gender, race/ethnicity (non-Hispanic black, non-Hispanic white, Mexican-American and other), education ($\leqslant$7, 8–11, 12 and $\geqslant$13 years), income ($<$US$ 30,000 and $\geqslant$$ 30,000), presence of diabetes mellitus (fasting plasma glucose $<$126 mg/dl, $\geqslant$126 mg/dl), and number of remaining teeth. Dietary intake of total calory in grams was available from 24 h dietary recall data. Information on plaque was not available. Therefore, we used percent sites with gingival bleeding as a covariate since there is a tight relationship between the presence of plaque and gingival bleeding.

Statistical analyses

Univariate analyses included means, standard deviations, frequencies, and percentages of variables of interest. Bivariate analyses were used to assess associations among variables (collinearity, confounding and effect modification) and to construct the models for multiple regression analyses, and consisted of correlation matrix, χ^2- and *t*-tests. Finally, multiple linear and logistic regression analyses were used to assess the independent effect of alcohol on CAL after adjusting the effect of confounders. Possible interaction of alcohol consumption with smoking was tested in the multiple regression analyses. The distribution of CAL was skewed. Therefore, log transformation of CAL was used in multiple linear regression analyses. Statistical analyses were weighted to account for sampling design and non-response to produce national estimates using WesVar statistical software.

Results

Table 1 describes the characteristics of the study population by periodontal disease status. Diseased group had higher levels of age, smoking, diabetes and percent sites of gingival bleeding but lower levels of education, income, total calory intake and number of remaining teeth. Frequency of non-Hispanic whites and non-Hispanic blacks in diseased group were similar but both were significantly higher compared with the frequency of Hispanics.

Table 2 describes clinical variables (CAL, PD, gingival bleeding and number of remaining teeth) by drinking groups. Trend analyses (unadjusted linear regressions) showed significant linear relationships between number of drinks per week and all clinical variables.

Table 1. Characteristics of the study population by clinical attachment loss

	CAL $<$ 1.5 mm	CAL $\geqslant$ 1.5 mm	*p*-value[‡]
Age*	38 ± 15	57 ± 16	$<$0.0001
Gender[†]			
Female	5309 (79.1)	1407 (20.9)	$<$0.0001
Male	3936 (65.5)	2071 (34.5)	
Race/ethnicity[†]			
Non-Hispanic white	3293 (70.5)	1380 (29.5)	$<$0.0001
Non-Hispanic black	2607 (71.3)	1048 (28.7)	
Hispanic	2962 (76.7)	902 (23.3)	
Education[†]			
$\leqslant$7 years	1099 (55.2)	893 (44.8)	$<$0.0001
8–11 years	1726 (66.3)	878 (33.7)	
12 years	3085 (76.1)	967 (23.9)	
$>$12 years	3278 (82.2)	708 (17.8)	
Income[†]			
$<$$30,000	5061 (70.0)	2172 (30.0)	$<$0.0001
$\geqslant$$30,000	3413 (77.8)	971 (22.2)	
Smoking (packyears)*	4.4 ± 11.3	15.8 ± 23.9	$<$0.0001
Diabetes[†]			
No	8563(74.5)	2927 (25.5)	$<$0.0001
Yes	341 (44.5)	425 (55.5)	
Total calories (kcal)*	2205.9 ± 1068.3	1975.6 ± 989.8	$<$0.0001
Remaining teeth*	25 ± 4	19 ± 6	$<$0.0001
GB (%)*	10.2 ± 14.6	16.3 ± 20.8	$<$0.0001

CAL, clinical attachment loss; GB, percent of sites with gingival bleeding; Diabetes, fasting plasma glucose – no: $<$126 mg/dl and yes: $\geqslant$126 mg/dl.

*Mean ± SD.

[†]Count (%).

[‡]*p*-values were derived from *t*-tests for continuous variables and from χ^2-tests for categorical variables.

Fig. 1 compares mean CAL in high and low level drinkers using 5, 10, 15 and 20 drinks/week as cut-points. The difference in mean CAL between high and low drinkers increases as the cut-point for alcohol consumption gets higher suggesting a dose–response relationship.

Fig. 2 presents adjusted odds ratios (ORs) and their 95% confidence intervals (CIs) from multiple logistic regression analyses controlling for age, gender, race, education, income, smoking, diet, diabetes, number of remaining teeth, gingival bleeding. Adjusted ORs and their 95% CIs were 1.22 [1.02–1.47], 1.39 [1.13–1.71], 1.54 [1.22–1.93], and 1.67 [1.25–2.23] using 5, 10, 15 and 20 drinks/week as cut-points. We can see that even after controlling the effect of confounding variables, the risk of attachment loss increases in a consistent manner by increasing levels of alcohol consumption implying that the relationship is dose dependent. There was also a significant linear relationship between number of drinks per week and log CAL (0.0001) in multiple regression analysis adjusting for the same covariates as in the multiple logistic regression analyses.

There was no significant interaction between smoking and alcohol consumption in any of the regression models implying that the relationship between alcohol consumption and periodontal disease does not vary with the levels of smoking.

Table 2. Description of clinical variables by drinking groups

	Drinks/week			
	⩾5	⩾10	⩾15	⩾20
CAL (mm)	1.22 ± 1.12*	1.31 ± 1.19	1.36 ± 1.23	1.40 ± 1.25
PD (mm)	1.62 ± 0.54	1.69 ± 0.56	1.74 ± 0.59	1.74 ± 0.54
GB (% sites)	10.97 ± 15.7	12.25 ± 16.6	13.40 ± 17.3	13.89 ± 17.9
Remaining teeth	23.91 ± 5.2	23.86 ± 5.3	23.90 ± 5.2	23.83 ± 5.2

CAL, clinical attachment loss; PD, probing depth; GB: gingival bleeding.
*Mean ± SD.

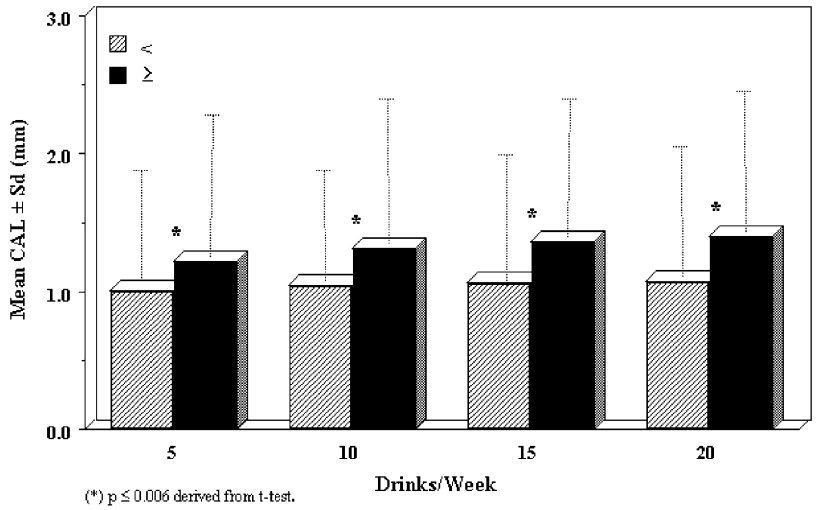
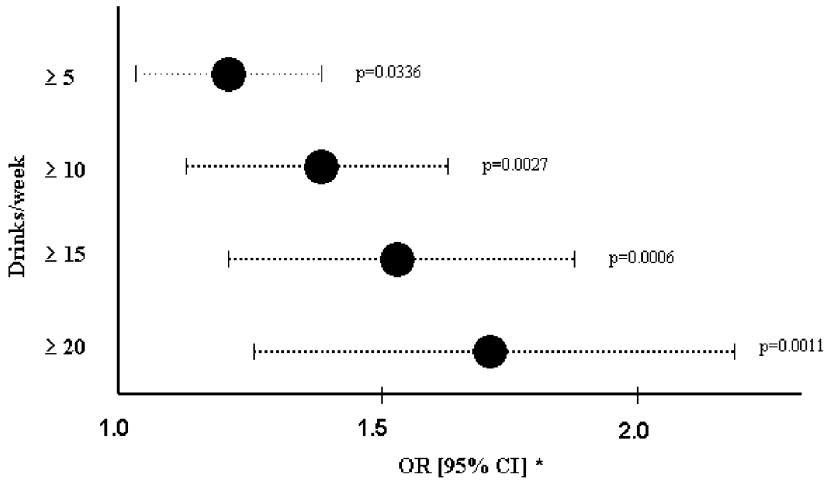


Fig. 1. Mean clinical attachment loss by alcohol consumption.



* Odds ratios and their 95% confidence intervals were derived from multiple logistic regression analyses adjusting for age, gender, race, education, income, smoking diet, diabetes, number of remaining teeth and gingival bleeding.

Fig. 2. Association between alcohol consumption and clinical attachment loss.

Discussion

The present study found a moderate but consistent dose-dependent relationship between alcohol consumption and periodontal disease. Adjusted OR ranged between 1.22 and 1.67 and increased gradually for increasing levels of alcohol consumption. Chronic diseases are usually multifactorial. The risk factors act together to cause disease, and it is not unusual for a chronic-disease-risk factor to have a low effect size. For example, plaque is the cause of periodontal disease but it has had consistently low or non-significant effect sizes for either attachment loss or alveolar bone loss in multiple regression models. Still, we always include it in risk models because it causes periodontal disease in the presence of other risk factors such as smoking. Smoking has a much higher effect size compared with plaque but it does not mean that smoking is more important than plaque in the etiology of periodontal disease. ORs for smoking usually range between 1.5 and 5. In this study ORs for alcohol ranged between 1.22 and 1.67 which is comparable with the risk of moderate smoking.

The effect of alcohol on periodontal disease has been commonly explained by poor oral hygiene of chronic alcoholic consumers. However, biologic plausibility of this relationship exists and could be explained by alcohols

adverse effect on host defense (defective neutrophil function and complement deficiency), clotting mechanism (defective prothrombine and vitamin K activity), bone metabolism (increased resorption, decreased formation), healing (vitamin B-complex and protein deficiency) and direct toxic effect on periodontal tissues (Christen 1983, Farley et al. 1985, Gottsegen 1993, Holbrook and Barret-Conner 1993, Moniz 1994).

The results of this study are consistent with Erie County study results (Tezal et al. 2001) which showed that self-reported alcohol consumption was related to CAL in a dose-dependent fashion, with ORs of 1.36 [1.02–1.80] and 1.44 [1.04–2.00] for $\geqslant 5$ and $\geqslant 10$ drinks/week, respectively, after controlling for major confounders. Slightly increased ORs in the Erie County study may be due to higher level of periodontal disease in this population and higher validity of the measurements of periodontal disease. In Erie County study, probing measurements were performed on all present teeth except the third molars with a constant force electronic probe on six sites per tooth. In the NHANES III, these measurements were performed using a hand probe (NIDR) on only one upper and one lower quadrant at buccal and mesiobuccal sites, which may lead to underestimation of the disease severity. In addition, alcohol intake in the NHANES III was assessed only for the last 12 months.

There are only two other studies in previous literature that relates alcohol consumption to CAL indirectly: in a case–control study (Movin 1981) with 30 cirrhotic and 43 controls matched for age, sex and education, cirrhotic and control groups exhibited no significant difference in attachment loss but patients suffering from cirrhosis for more than 3 years showed significantly greater loss of attachment, as well as more plaque and calculus compared with a disease duration of less than 3 years. Authors suggested that aggravation of the periodontal conditions was related to increasing neglect of teeth as cirrhotic condition aggravates. In this study bivariate analysis was used, which does not allow adjusting covariates simultaneously. Therefore we cannot draw any conclusion from this study. In another study (Novacek et al. 1995), in order to assess the role of alcoholism and cirrhosis on CAL, four groups of subjects were compared: 64 alcoholics with cirrhosis, 33 non-alcoholics with cirrhosis, 68 alcoholics without cirrhosis and 71 healthy control subjects. Presence of cirrhosis was significantly associated with increased loss of attachment in a stepwise multiple linear regression analysis adjusting for oral hygiene, age, time since last dental examination and smoking. In this study, alcohol consumption was not assessed quantitatively.

In a cross-sectional study (Sakki et al. 1995) with 780 subjects of 55-year-old Finnish subjects, alcohol consumption of 3.5 drinks/week was significantly related to the frequency of PD >3 mm (OR: 2.52, 95% CI: 1.40–4.54) in stepwise logistic regression controlling for smoking, and toothbrushing frequency. Although PD does not reflect previous loss of periodontium, this study suggests an association between alcohol intake and periodontal disease.

There was no significant interaction between smoking and alcohol in any of the regression models implying that the relationship between alcohol consumption and periodontal disease does not depend on the level of smoking. In the Erie County study, interaction between alcohol and smoking was not significant either (Tezal et al. 2001). In other previous studies interactions were not assessed.

We also evaluated the effect of each type of alcoholic beverage separately. The only information available on individual types of drinks was ''times per month'' of consumption. To evaluate the effect of a specific type of drink, we used ''times/month'' of beer, wine and hard liquor in separate regression models. Adjusting for the effect of confounders, beer ($p = 0.01$) and hard liquor ($p = 0.03$) consumption had significant linear relationships with CAL. However, wine was not significantly related to CAL. These results on types of drinks are not reliable and should not be used to draw conclusions since the variable ''times/month'' does not include information on the amount of alcohol consumed and there was no information about whether those individuals consume only that particular type of drink or in combination with other types of drinks. On the other hand, differences among different types of drink may reflect lifestyle of the consumers. For example, we can speculate that wine and beer consumers represent different lifestyles. Beer is usually consumed in much larger amount at once than wine and diet of beer and wine consumers may be dramatically different.

In NHANES III, alcohol consumption was assessed only for the last year. Lifetime history of alcohol consumption would be more valuable since periodontal disease is a chronic disease. The net result of alcohol's effect on periodontal disease may depend on dose, frequency, timing, and pattern (regular, random, periodic, etc.) of its consumption. Accuracy of information on alcohol consumption is usually inadequate. Different questions elicit varying degrees of reliability and validity (Alanko 1984, Giovannucci et al. 1991, Webb et al. 1991). Most researchers agree that the direction of bias is negative, a result of under-reporting (Embree & Whitehead 1993). To have clearer answers, more precise quantitation of alcohol consumption needs to be obtained.

We can conclude that alcohol consumption may be related to CAL and the relationship appears to be dose dependent. Further studies to confirm this relationship and to test possible underlying mechanisms are needed.

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