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Amount and type of alcohol and periodontitis in the Copenhagen City Heart Study

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Abstract

Aim: The aim of this study was to study the association between alcohol consumption and periodontitis assessed as clinical attachment loss (CAL) and bleeding on probing (BOP) in a cross-sectional design.

Material and Methods: The study included 1,521 adults aged 20–95 years, who underwent an oral examination including full-mouth registration of CAL and BOP. Alcohol was ascertained using a food-frequency questionnaire. The association between total and type-specific alcohol and periodontitis was assessed by means of multivariate logistic regression.

Results: A lower odds ratio (OR) for CAL (defined as mean $\geqslant$3 mm) was observed in men consuming 21–34 [OR = 0.51, 95% confidence interval (CI), 0.27–0.95] and 35+ drinks/week (OR = 0.34, 95% CI, 0.15–0.79) compared with men drinking 1–13 drinks/week. Also, men with a weekly wine consumption of more than 14 drinks compared with men who reported no wine intake had lower OR for CAL (OR = 0.24; 95% CI, 0.09–0.62). A higher OR for BOP (defined as $\geqslant$25%) among male abstainers was observed (OR = 1.79, 95% CI, 1.03–3.12) compared with men in the light-drinking group (1–13 drinks/week). No significant association was observed for either CAL or BOP in women.

Conclusions: The results indicate that higher alcohol consumption, particularly intake of wine, is inversely associated with CAL in men. Such an association is not found in women.

Key words: alcohol consumption; epidemiology; periodontal disease; risk factor

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Studies of the past decade have suggested a positive association between high alcohol consumption and periodontitis (Sakki et al. 1995, Tezal et al. 2001, 2004, Pitiphat et al. 2003, Shimazaki et al. 2005, Okamoto et al. 2006, Jansson 2008). Also, studies have related alcohol abuse (Harris et al. 1997) and alcohol-induced hepatic cirrhosis (Movin 1981, Novacek et al. 1995) to periodontal status. However, the studies differed according to the definition of periodontitis and results were diverse.

Several issues limit the interpretation of the results of past studies. The majority of these studies have been cross-sectional (Sakki et al. 1995, Tezal et al. 2001, 2004, Shimazaki et al. 2005), while fewer were longitudinal (Pitiphat et al. 2003, Okamoto et al. 2006, Jansson 2008). Only two of the above-mentioned population studies were performed in Caucasians. One study included 527 dentate 55-year-old citizens from Finland and found lifestyle, including dietary and smoking habits, alcohol consumption and physical activity, independently related to periodontal pocketing (Sakki et al. 1995). Another study describing the association of alcohol consumption with dental health in 477 Swedish adults found no relation to periodontal disease (Jansson 2008). Also, although women and men appear to have different alcohol pharmacokinetics (Mumenthaler & Taylor 1999), none of the previous alcohol-periodontitis studies include separate analyses for women and men. Moreover, while intake of different types of alcohol may have different effects on mortality (Gronbaek et al. 2000), few have focused on possible

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differences among beer, wine and spirits in relation to periodontitis. An American study evaluated the significance of alcoholic beverage type, but revealed no apparent differences in the association with periodontal variables (Tezal et al. 2001). The effect of frequency of alcohol consumption has also been described. The authors reported a significant linear relationship between frequency of beer and hard liquor consumption (times/month), and clinical attachment loss (CAL). However, the study lacked information on type-specific amount of alcohol consumed (Tezal et al. 2004). In the Health Professionals Follow-up Study, no clear indication of an association between specific types of beverages and periodontitis was observed (Pitiphat et al. 2003).

In accordance with the previous findings, it was our hypothesis that high alcohol consumption is related to poor periodontal status. The objective of the present study was to examine the association between amount and type of alcohol consumed and periodontitis in female and male adults participating in the Copenhagen City Heart Study (CCHS).

Material and Methods

Study population

The CCHS is a prospective cohort study of risk factors and general health. The first examination took place in 1976–1978 where 14,223 persons aged 20 years or more participated. A detailed description of the study procedure has been published previously (Appleyard 1989).

Of the 6,237 participants from the 2001–2003 examination of CCHS, we randomly selected 2,951 for the present cross-sectional study. In 2004–2007, the sample was invited to a periodontal examination at the School of Dentistry, University of Copenhagen. A total of 1,597 (54%) persons participated in the clinical examination. Age of participants ranged from 20 to 95 years, and more than 97% of the population was of Caucasian origin. Thirty-four individuals were edentulous and another 41 individuals were excluded due to missing CAL measurements. One participant did not answer the items concerning drinking habits. Therefore, the final study population consisted of 1,521 participants.

The study was approved by the Biomedical Research Ethics Committees for Copenhagen and Frederiksberg Municipalities (KF 11-078/03), and the participants signed informed written consent before enrolling.

Questionnaire variables

A questionnaire was mailed to each participant and completed shortly before the examination. The items concerned health-related behaviours including alcohol intake, smoking habits, socioeconomic status and medical history. To avoid misunderstandings, the answers were reviewed by the clinical examiners who were not aware of the specific hypothesis of the study.

Assessment of alcohol consumption

For the present study, participants' alcohol intake was assessed as weekly intake of beer, wine and spirits stated in average drinks. A standard Danish drink contains approximately 12 g of alcohol. The categorization of alcohol consumption was based on the Danish sensible drinking limits, i.e. a maximum of 14 drinks/week for women and 21 drinks/week for men. Participants were classified in five groups according to their usual weekly intake of alcohol: Abstainers (< 1 drink/week), light drinkers (1–6 drinks/week for women and 1–13 drinks/week for men), moderate drinkers (7–13 drinks/week for women and 14–20 drinks/week for men), heavy drinkers (14–20 drinks/week for women and 21–34 drinks/week for men) and excessive drinkers ($\geqslant$21 and $\geqslant$35 for women and men, respectively). Type-specific alcohol groups were categorized as < 1, 1–5, 6–10 and > 10 drinks/week for women and < 1, 1–6, 7–14 and > 14 drinks/week for men for beer, wine and spirits, respectively.

Periodontal examination

All the permanent teeth present, except third molars, were examined at six sites of each tooth. The clinical registrations were performed by a dentist (J. K.) and three dental hygienists, who were calibrated before and throughout the study to control inter- and intra-examiner variability. Plaque was registered as 1 for visible plaque, if necessary after using the probe across the tooth surface, and 0 for no plaque, modified from Silness & Löe (1964). Probing depth (PD) was measured with a manual periodontal probe 0.5 mm in diameter (product: 8 BG, Løco; Denmark). Bleeding on probing (BOP) was registered 15 s after probing at each site. CAL is the distance from the cemento-enamel junction to the bottom of the pocket, i.e. to the tip of the periodontal probe. The distance from the free gingival margin to the cemento-enamel junction was measured and CAL was calculated by subtracting this value from PD. Gingival recession was recorded as a negative value and thereby added to PD. If the cemento-enamel junction was missing because of a restoration and could not be estimated from the neighbouring tooth, the CAL measurement was registered as missing. For each individual, plaque and bleeding scores were determined as percentage of sites with plaque and bleeding, respectively, relative to the total number of sites examined. Mean CAL was categorized as a binary outcome variable based on the 75 percentile (< 3 and $\geqslant$3 mm), and likewise, the BOP score was categorized in groups of < 25% and $\geqslant$25%.

Other covariates

Age at baseline was included in the adjusted models as a continuous variable. Participants reported their smoking status as ''never smoker'', ''ex-smoker'' or ''current smoker''. Current and ex-smokers reported the amount and type (cigarettes, cheroots, cigars, pipe or mixed) of tobacco smoked, and the total daily tobacco consumption was calculated by equating a cigarette to 1 g, a cheroot to 3 g and a cigar to 5 g of tobacco. Number of pack years was calculated as daily consumption of tobacco divided by 20 multiplied by years of smoking, and categorized as 0, 0.1–19.9 and $\geqslant$20.0 pack years. The educational level of participants was divided into three categories: less than 11 years of schooling, 11–12 years of schooling and more than 12 years of schooling. Annual household income was categorized as < 200,000 DKK (corresponding to approximately 40,000 US$), 200–600,000 DKK and > 600,000 DKK (> 120,000 US$). Leisure time physical activity was divided into two groups of light physical activity of less than 4 h/week or more. The body mass index (BMI) (defined as weight/height^2) was categorized as normal weight (< 25 kg/m^2), overweight (25–29.9 kg/m^2) and obese ($\geqslant$30 kg/m^2). Participants were

classified as having diabetes based on self-reported information. Number of teeth was categorized in three groups of <24, 24–27 and 28. The plaque score was categorized in quartiles based on the distribution in the cohort.

Statistical analyses

Contingency tables described the distribution of covariates across levels of alcohol consumption. Multivariate logistic regression analyses were applied to examine the cross-sectional relationship between alcohol consumption and CAL, and BOP, respectively, adjusting for the effects of the above-mentioned covariates. To assess the dose–response relationships of alcohol consumption to periodontal variables, trend analyses were computed by treating the median values of each category of alcohol consumption as a continuous variable. Tests for interaction by gender did not reveal any statistically significant interactions. The *p*-value for the test for interaction between total alcohol intake and gender was $p = 0.67$. For beer, wine and spirits, $p = 0.68$, 0.17 and 0.76, respectively. However, data for women and men were analysed separately, not because of statistical interaction, but because alcohol pharmacokinetics is believed to be gender dependent, and because drinking habits may vary with gender. Possible effect modification by smoking, age, BMI, physical activity and diabetes on the relation between alcohol and CAL, and between alcohol and BOP was examined by including a product term for alcohol and each of the variables mentioned one at a time, categorized as described previously (Hyman 2006). The statistical analyses were performed using the statistical software package SAS, version 9.1 (SAS Institute, Cary, NC, USA).

Results

Study population

General characteristics of the study population by weekly alcohol consumption for women and men are shown in Table 1. In the study population, 54% were women with a mean age of 52.8 years. Men were slightly older with a mean age of 54.7 years. Almost 25% of both women and men had a weekly alcohol intake above the recommended level, while 12% of women and 11% of men reported alcohol abstinence. Twenty-four percent of women and 29% of men were current smokers, and the proportions of current smokers as well as the mean number of pack years were highest in the two heavy-drinking groups in both women and men. The proportion of men reporting low income was smallest between medium and heavy drinkers. Low physical activity was reported from half of the participants. The proportion of male participants with low physical activity followed a U-shaped curve, as the medium drinkers had the lowest proportion, and the abstainers and excessive drinkers had the highest proportion. Four percent of women and 6% of men reported a diagnosis of diabetes. There was no difference in mean number of teeth between the five alcohol groups among women, whereas a statistically significant difference among male participants was observed, the lowest mean number found in abstainers.

Relation between total alcohol consumption and CAL

In the multivariate logistic regression model, a lower odds ratio (OR) for CAL was observed in men with a total weekly alcohol consumption of 21–34 drinks [OR, 0.51; 95% confidence interval (CI), 0.27–0.95], and in men with a total weekly alcohol consumption of 35+ drinks (OR, 0.34; 95% CI, 0.15–0.79) compared with men in the light-drinking group (1–13 drinks/week). Trend analyses showed significant decreases in the ORs of CAL ($p = 0.002$) for men with increasing alcohol consumption. No significant interactions were observed between alcohol and smoking ($p = 0.66$), age ($p = 0.81$), physical activity ($p = 0.18$), BMI ($p = 0.27$) or diabetes ($p = 0.66$).

For women, no significant association between total amount of alcohol and CAL was observed (Table 2). No significant interactions were observed between alcohol and smoking ($p = 0.14$), age ($p = 0.96$), physical activity ($p = 0.08$), BMI ($p = 0.08$) or diabetes ($p = 0.87$).

Relation between type-specific alcohol consumption and CAL

The type-specific alcohol analyses generally showed lower ORs for CAL among men with higher intake of alcohol. The results were statistically significant among men with a weekly wine consumption of more than 14 drinks compared with men who reported no wine intake (OR, 0.24; 95% CI, 0.09–0.62). Again, trend analyses showed significant decreases in the ORs of CAL ($p = 0.009$) for men with increasing intake of wine. In women, no significant relation between type-specific alcohol and CAL was observed (Table 3).

Relation between alcohol consumption and BOP

In the multivariate logistic regression model including BOP as the outcome variable, a significant association among male abstainers was found (OR, 1.79; 95% CI, 1.03–3.12) compared with men in the light-drinking group (1–13 drinks/week). No significant interactions were observed between alcohol and smoking ($p = 0.83$), age ($p = 0.20$), physical activity ($p = 0.18$), BMI ($p = 0.12$) or diabetes ($p = 0.36$).

For women, there was no significant association between total amount of alcohol and BOP (Table 4). No significant interactions were observed between alcohol and smoking ($p = 0.10$), age ($p = 0.63$), physical activity ($p = 0.32$), BMI ($p = 0.23$) or diabetes ($p = 0.89$).

Relation between type-specific alcohol consumption and BOP

When classifying participants according to type-specific alcohol intake (Table 5), the multivariate logistic regression model showed that men drinking wine and spirit had an inverse relation to BOP. The results were statistically significant for men consuming 7–14 drinks of wine per week (OR, 0.46; CI, 0.24–0.91) compared with men without wine intake. The ORs for higher BOP among men seemed to be decreasing with higher intake of any type of alcohol; however, the trend analyses revealed these results as statistically insignificant. In women, a tendency towards a higher OR for BOP with higher consumption of spirits was found ($p = 0.05$).

Discussion

To our knowledge, this is the first study reporting gender-specific analyses of the association between alcohol consumption and periodontitis severity. We found an inverse relationship between high total alcohol intake and CAL among men. Trend tests showed tendencies towards lower ORs for CAL among male participants with increasing intake

Table 1. Characteristics of the study population according to weekly alcohol consumption

		Average alcohol intake (drinks/week)				
		<1	1–6	7–13	14–20	⩾21
Women						
Number (%)	817 (54)	101 (12)	328 (40)	218 (27)	102 (13)	68 (8)
Mean age, years (SD)	52.8 (15.2)	51.3 (17.6)	50.2 (16.1)	53.9 (14.3)	58.1 (12.0)	55.9 (10.6)
Persons with low education* (%)	309 (38)	43 (43)	117 (36)	83 (38)	39 (38)	27 (40)
Persons with middle education* (%)	290 (35)	32 (32)	110 (34)	81 (37)	40 (39)	27 (40)
Persons with high education* (%)	216 (26)	26 (26)	100 (31)	53 (24)	23 (23)	14 (21)
Persons with low income† (%)	445 (54)	63 (66)	189 (58)	110 (52)	46 (46)	37 (55)
Persons with middle income† (%)	157 (19)	21 (22)	58 (18)	43 (20)	24 (24)	11 (16)
Persons with high income† (%)	198 (24)	12 (13)	78 (24)	59 (28)	30 (30)	19 (28)
Never smokers (%)	355 (43)	47 (47)	146 (45)	99 (45)	39 (38)	24 (35)
Ex-smokers (%)	265 (32)	27 (27)	114 (35)	71 (33)	34 (33)	19 (28)
Current smokers (%)	197 (24)	27 (27)	68 (21)	48 (22)	29 (28)	25 (37)
Pack years‡, mean (SD)	10 (15)	24 (18)	17 (14)	24 (21)	26 (22)	32 (20)
Mean BMI, kg/m^2 (SD)	24.3 (4.3)	25.0 (4.6)	24.3 (4.6)	24.1 (4.1)	24.7 (4.2)	23.8 (3.4)
Persons with low physical activity§ (%)	431 (53)	62 (61)	184 (56)	101 (46)	51 (50)	33 (49)
Persons with high physical activity§ (%)	386 (47)	39 (39)	144 (44)	117 (54)	51 (50)	35 (51)
Persons with diabetes (%)	31 (4)	3 (3)	13 (4)	9 (4)	3 (3)	3 (4)
Plaque score, mean (SD)	31.1 (21.7)	34.0 (22.8)	31.2 (23.1)	30.5 (20.5)	28.8 (19.0)	31.8 (20.6)
Number of teeth, mean (SD)	25.3 (4.6)	25.4 (4.3)	25.1 (5.2)	25.4 (4.4)	25.6 (3.6)	25.5 (3.9)
		<1	1–13	14–20	21–34	⩾35
Men						
Number (%)	704 (46)	74 (11)	337 (48)	116 (16)	126 (18)	51 (7)
Mean age, years (SD)	54.7 (14.4)	57.5 (13.6)	52.3 (15.7)	54.2 (13.6)	57.7 (12.5)	60.0 (9.2)
Persons with low education* (%)	303 (43)	36 (49)	124 (37)	52 (45)	66 (52)	25 (49)
Persons with middle education* (%)	208 (30)	18 (24)	106 (32)	35 (30)	37 (30)	12 (24)
Persons with high education* (%)	191 (27)	20 (27)	105 (31)	29 (25)	23 (18)	14 (27)
Persons with low income† (%)	350 (50)	48 (65)	174 (53)	48 (41)	52 (41)	28 (56)
Persons with middle income† (%)	165 (23)	17 (23)	74 (22)	28 (24)	34 (27)	12 (24)
Persons with high income† (%)	182 (26)	9 (12)	83 (25)	40 (34)	40 (32)	10 (20)
Never smokers (%)	242 (34)	21 (28)	140 (42)	36 (31)	38 (30)	7 (14)
Ex-smokers (%)	260 (32)	31 (42)	107 (32)	57 (49)	46 (37)	19 (37)
Current smokers (%)	202 (29)	22 (30)	90 (27)	23 (20)	42 (33)	25 (49)
Pack years‡, mean (SD)	18 (27)	24 (18)	32 (30)	25 (24)	31 (21)	42 (32)
Mean BMI, kg/m^2 (SD)	25.9 (3.8)	26.4 (4.8)	25.8 (4.0)	25.9 (3.2)	26.2 (3.2)	26.1 (3.7)
Persons with low physical activity§ (%)	360 (51)	45 (61)	172 (51)	45 (39)	69 (55)	29 (57)
Persons with high physical activity§ (%)	344 (49)	29 (39)	165 (49)	71 (61)	57 (45)	22 (43)
Persons with diabetes (%)	44 (6)	8 (11)	18 (5)	6 (5)	7 (6)	5 (10)
Plaque score, mean (SD)	42.8 (24.8)	49.5 (25.4)	41.4 (25.2)	40.5 (23.7)	43.3 (24.0)	46.6 (24.9)
Number of teeth, mean (SD)	24.4 (5.7)	23.1 (6.6)	24.4 (5.9)	25.6 (4.5)	24.8 (4.5)	23.4 (7.0)

*Defined as <11, 11–12 and >12 years of schooling.
†Corresponding to <200,000, 200–600,000 and >600,000 DKK/year.
‡Calculated among current smokers.
§Defined as <4 and >4 h/week.[]BMI, body mass index.

of total alcohol. The ORs for women showed the same tendency, but the results and the test for trend were statistically insignificant. There was significantly decreased ORs for BOP among men reporting high alcohol intake compared with men reporting abstinence. For women, no pattern between alcohol consumption and BOP was observed. Type-specific analyses among men showed tendencies towards an inverse association between higher alcohol intake and CAL. In addition, the association between type-specific alcohol and BOP showed lower ORs for BOP among men consuming more drinks of wine or spirits.

As most other studies on alcohol and periodontitis (Tezal et al. 2001, 2004, Nishida et al. 2005, Shimazaki et al. 2005), the design of the present study was cross-sectional. An obvious problem with cross-sectional studies is that exposure and outcome are measured at the same time, their inter-related sequence being unknown. However, it is unlikely that people change their drinking habits because of their awareness of a periodontitis diagnosis. Therefore, despite the design of the study, it seems plausible to assume that alcohol intake is the risk factor for periodontitis and not vice versa.

Another limitation of our study is that only 54% of the invited people participated and people choosing to participate may have a different risk profile than those who decline. This is why caution should be taken to generalize from our findings. However, representation of participants with different stages of both alcohol drinking habits and perio-

Table 2. Association between weekly alcohol consumption and clinical attachment loss (CAL)

Alcohol (drinks/week)	OR (95% CI)*	OR (95% CI)†	OR (95% CI)‡
Women			
<1	1.45 (0.85–2.48)	1.18 (0.58–2.37)	1.19 (0.56–2.51)
1–6	1.00 (Reference)	1.00 (Reference)	1.00 (Reference)
7–13	1.30 (0.86–1.98)	1.07 (0.64–1.80)	1.05 (0.60–1.84)
14–20	1.30 (0.76–2.22)	0.71 (0.37–1.36)	0.85 (0.43–1.70)
21+	1.37 (0.74–2.54)	0.70 (0.33–1.48)	0.82 (0.37–1.82)
p-value for trend	0.44	0.15	0.42
Men			
<1	1.38 (0.80–2.38)	0.98 (0.50–1.94)	1.03 (0.50–2.13)
1–13	1.00 (Reference)	1.00 (Reference)	1.00 (Reference)
14–20	0.67 (0.40–1.10)	0.50 (0.26–0.93)	0.53 (0.27–1.05)
21–34	0.92 (0.58–1.45)	0.52 (0.29–0.92)	0.51 (0.27–0.95)
35+	0.94 (0.48–1.83)	0.31 (0.14–0.67)	0.34 (0.15–0.79)
p-value for trend	0.32	<0.001	0.002

*Crude odds ratios (OR) and 95% confidence intervals (CI) derived from logistic regression analysis.
†Adjusted for age and smoking.
‡Adjusted for age, smoking, educational level, income, body mass index (BMI), physical activity, diabetes, number of teeth and plaque score.

Table 3. Association between weekly intake of beer, wine, and spirits and clinical attachment loss (CAL)

	OR (95% CI)*	OR (95% CI)†	OR (95% CI)‡
Women			
Beer (drinks/week)			
<1	1.00 (Reference)	1.00 (Reference)	1.00 (Reference)
1–5	0.79 (0.54–1.15)	1.06 (0.66–1.70)	1.13 (0.68–1.87)
6–10	1.02 (0.45–2.30)	0.86 (0.30–2.46)	0.82 (0.26–2.57)
>10	0.54 (0.11–2.58)	0.33 (0.06–1.78)	0.26 (0.04–1.79)
p-value for trend	0.54	0.20	0.18
Wine (drinks/week)			
<1	1.00 (Reference)	1.00 (Reference)	1.00 (Reference)
1–5	0.71 (0.43–1.16)	0.91 (0.47–1.75)	0.84 (0.42–1.68)
6–10	0.85 (0.48–1.51)	0.72 (0.35–1.49)	0.69 (0.32–1.53)
>10	0.86 (0.48–1.54)	0.63 (0.30–1.32)	0.71 (0.32–1.58)
p-value for trend	0.70	0.18	0.46
Spirit (drinks/week)			
<1	1.00 (Reference)	1.00 (Reference)	1.00 (Reference)
1–5	1.24 (0.84–1.83)	1.05 (0.64–1.71)	0.97 (0.58–1.65)
6–10	1.78 (0.69–4.62)	0.99 (0.31–3.10)	1.20 (0.36–3.94)
>10	0.96 (0.10–8.89)	0.51 (0.05–5.48)	1.07 (0.10–11.28)
p-value for trend	0.70	0.63	0.91
Men			
Beer (drinks/week)			
<1	1.00 (Reference)	1.00 (Reference)	1.00 (Reference)
1–6	0.78 (0.47–1.30)	0.97 (0.51–1.82)	1.12 (0.56–2.24)
7–14	0.75 (0.37–1.49)	1.01 (0.43–2.36)	0.99 (0.39–2.51)
>14	0.65 (0.33–1.31)	0.51 (0.22–1.16)	0.55 (0.22–1.33)
p-value for trend	0.32	0.11	0.06
Wine (drinks/week)			
<1	1.00 (Reference)	1.00 (Reference)	1.00 (Reference)
1–6	0.58 (0.34–0.96)	0.66 (0.34–1.27)	0.63 (0.31–1.30)
7–14	0.51 (0.27–0.94)	0.45 (0.21–0.99)	0.45 (0.19–1.05)
>14	0.38 (0.19–0.76)	0.26 (0.11–0.59)	0.24 (0.09–0.62)
p-value for trend	0.01	<0.001	0.009
Spirits (drinks/week)			
<1	1.00 (Reference)	1.00 (Reference)	1.00 (Reference)
1–6	1.36 (0.92–2.01)	1.14 (0.70–1.88)	1.21 (0.71–2.05)
7–14	2.32 (1.03–5.21)	0.69 (0.25–1.92)	0.78 (0.26–2.33)
>14	1.61 (0.45–5.74)	0.80 (0.17–3.63)	0.67 (0.15–3.00)
p-value for trend	0.12	0.52	0.42

*Crude odds ratios (OR) and 95% confidence intervals (CI) derived from logistic regression analysis.
†Adjusted for age and smoking.
‡Adjusted for age, smoking, educational level, income, body mass index (BMI), physical activity, diabetes, number of teeth and plaque score.

dontitis allows us to believe that the relative risks found in the present study are also applicable to people other than those examined.

In the present study, data on alcohol intake were self-reported and the validity of such data may be questioned, in particular with respect to underreporting. However, a previous study evaluating self-reported alcohol consumption compared with a more detailed dietary interview revealed very little or no systematic variation for the three types of alcohol between the two methods (Gronbaek & Heitmann 1996).

The assessment of periodontal disease in epidemiologic studies varies and there is no generally approved case definition for use in population-based surveillance of periodontitis (Page & Eke 2007). Thus, a wide range of methods from self-reported data over full-mouth registrations of CAL and PD to different index systems has been used, and therefore comparison of results is impaired.

Clearly, periodontitis manifestations in the form of attachment loss are expressed as a continuum, the endpoint manifestation being tooth loss and any definition of periodontitis is arbitrary. In the present study, either mean CAL $\geqslant$ 3 mm or BOP $\geqslant$ 25% was chosen as outcome variable. While CAL reflects accumulated historical disease activity, BOP is a measure of current periodontal inflammation. Such definitions do not reflect the number of lost teeth due to periodontitis and thus underestimate manifestations of actual disease status. The consequence of underestimated outcome variables could be an even stronger association between alcohol consumption and periodontitis.

Whereas periodontitis is initiated by bacterial accumulations, the pathogenesis of tissue breakdown involves the inflammatory host response (Van Dyke & Serhan 2003). Several biological effects of alcohol consumption on host defence mechanisms, including decreased inflammatory response and altered cytokine production may explain an association between alcohol and periodontitis [for review, see Szabo (1999)]. The link between alcohol and health is influenced by several factors such as drinking patterns, amount and type of alcohol consumed, and by age and gender (Gronbaek 2004, Romeo et al. 2007). Moreover, wine seems to enhance the immune response by being

Table 4. Association between weekly alcohol consumption and bleeding on probing (BOP)

Alcohol (drinks/week)	OR (95% CI)*	OR (95% CI)†	OR (95% CI)‡
Women			
<1	1.21 (0.73–1.99)	1.13 (0.68–1.88)	1.02 (0.60–1.73)
1–6	1.00 (Reference)	1.00 (Reference)	1.00 (Reference)
7–13	1.12 (0.76–1.65)	1.05 (0.71–1.56)	1.10 (0.73–1.67)
14–20	0.83 (0.49–1.40)	0.72 (0.42–1.23)	0.86 (0.49–1.52)
21+	1.33 (0.75–2.35)	1.08 (0.60–1.93)	1.24 (0.67–2.30)
p-value for trend	0.85	0.60	0.73
Men			
<1	2.04 (1.20–3.46)	1.90 (1.11–3.24)	1.79 (1.03–3.12)
1–13	1.00 (Reference)	1.00 (Reference)	1.00 (Reference)
14–20	0.91 (0.57–1.47)	0.90 (0.56–1.47)	0.98 (0.59–1.62)
21–34	0.77 (0.48–1.24)	0.70 (0.43–1.13)	0.78 (0.47–1.29)
35+	0.87 (0.44–1.73)	0.74 (0.37–1.49)	0.81 (0.39–1.67)
p-value for trend	0.04	0.01	0.06

*Crude odds ratios (OR) and 95% confidence intervals (CI) derived from logistic regression analysis.
†Adjusted for age and smoking.
‡Adjusted for age, smoking, educational level, income, body mass index (BMI), physical activity, diabetes, number of teeth and plaque score.

Table 5. Association between weekly intake of beer, wine and spirits and bleeding on probing (BOP)

	OR (95% CI)*	OR (95% CI)†	OR (95% CI)‡
Women			
Beer (drinks/week)			
<1	1.00 (Reference)	1.00 (Reference)	1.00 (Reference)
1–5	0.79 (0.55–1.12)	0.85 (0.59–1.21)	0.91 (0.62–1.32)
6–10	1.03 (0.48–2.22)	1.07 (0.49–2.33)	1.26 (0.56–2.86)
>10	0.98 (0.28–3.47)	0.80 (0.22–2.86)	0.71 (0.18–2.77)
p-value for trend	0.94	0.88	0.93
Wine (drinks/week)			
<1	1.00 (Reference)	1.00 (Reference)	1.00 (Reference)
1–5	0.94 (0.59–1.50)	1.00 (0.63–1.61)	1.04 (0.64–1.70)
6–10	0.79 (0.45–1.36)	0.76 (0.43–1.33)	0.87 (0.48–1.57)
>10	0.74 (0.42–1.29)	0.66 (0.37–1.17)	0.77 (0.42–1.43)
p-value for trend	0.17	0.045	0.22
Spirit (drinks/week)			
<1	1.00 (Reference)	1.00 (Reference)	1.00 (Reference)
1–5	1.16 (0.81–1.67)	1.13 (0.78–1.63)	1.07 (0.72–1.57)
6–10	2.50 (1.02–6.13)	2.31 (0.93–5.72)	2.56 (0.99–6.58)
>10	1.78 (0.29–11.06)	1.60 (0.25–10.04)	2.46 (0.37–16.46)
p-value for trend	0.07	0.12	0.05
Men			
Beer (drinks/week)			
<1	1.00 (Reference)	1.00 (Reference)	1.00 (Reference)
1–6	0.93 (0.56–1.54)	0.99 (0.59–1.65)	1.02 (0.60–1.75)
7–14	0.77 (0.38–1.56)	0.84 (0.41–1.72)	0.84 (0.40–1.77)
>14	1.02 (0.52–2.01)	1.09 (0.54–2.17)	1.15 (0.56–2.38)
p-value for trend	0.97	0.96	0.89
Wine (drinks/week)			
<1	1.00 (Reference)	1.00 (Reference)	1.00 (Reference)
1–6	0.64 (0.39–1.06)	0.67 (0.40–1.12)	0.73 (0.42–1.25)
7–14	0.41 (0.22–0.77)	0.39 (0.21–0.74)	0.46 (0.24–0.91)
>14	0.48 (0.24–0.94)	0.45 (0.23–0.90)	0.59 (0.28–1.24)
p-value for trend	0.01	0.006	0.08
Spirits (drinks/week)			
<1	1.00 (Reference)	1.00 (Reference)	1.00 (Reference)
1–6	1.05 (0.71–1.53)	1.01 (0.69–1.48)	0.93 (0.63–1.40)
7–14	1.13 (0.48–2.66)	0.90 (0.38–2.14)	0.77 (0.31–1.92)
>14	0.44 (0.09–2.12)	0.37 (0.08–1.78)	0.29 (0.06–1.47)
p-value for trend	0.46	0.23	0.12

*Crude odds ratios (OR) and 95% confidence intervals (CI) derived from logistic regression analysis.
†Adjusted for age and smoking.
‡Adjusted for age, smoking, educational level, income, body mass index (BMI), physical activity, diabetes, number of teeth and plaque score.

protective against infection (Romeo et al. 2007). Gender-dependent differences in alcohol pharmacokinetics and immune-inflammatory reactions may explain some of the different effects of alcohol consumption encountered (Mumenthaler & Taylor 1999). Beside the influence of alcohol on the host reactions, another effect might be attributed to the antimicrobial effect, similar to the effect obtained by some mouthrinses containing alcohol. However, in the present study, the analyses included plaque as a covariate and therefore a possible plaque-inhibiting effect of alcohol is already accounted for.

Among men, the results of the present study indicate that moderate alcohol consumption is inversely associated with CAL, which is in contrast to the results of the previous studies demonstrating increased ORs for periodontitis with increasing alcohol consumption (Sakki et al. 1995, Tezal et al. 2001, 2004, Pitiphat et al. 2003, Nishida et al. 2004, Shimazaki et al. 2005, Okamoto et al. 2006). An obvious difference is that the present study included more than 97% participants of Caucasian origin compared with the referred studies mainly including individuals of Japanese origin or different American background. This may partly explain the diverse results obtained.

The previously published studies have adjusted for confounding variables, including gender, but no other studies of the association between alcohol consumption and periodontitis have so far reported gender-specific analyses (Sakki et al. 1995, Tezal et al. 2001, 2004, Pitiphat et al. 2003, Nishida et al. 2004, Shimazaki et al. 2005, Okamoto et al. 2006, Jansson 2008). While two studies included only male participants (Pitiphat et al. 2003, Okamoto et al. 2006), for unknown reasons, the six other studies mentioned did not perform gender-specific analyses.

In addition to the effect of ethanol alone, wine has previously been suggested to possess a beneficial effect on mortality as well as on cardiovascular diseases (Gronbaek 2004). However, previous studies have failed to reveal differences between specific types of alcoholic beverages and periodontitis (Tezal et al. 2001). Moreover, others have reported a tendency towards a positive relation of intake of red wine and self-reported periodontitis, the results, however, being statistically insignificant (Pitiphat et al. 2003).

The relation between alcohol and some systemic diseases has been reported as a J-shaped curve, i.e. a light or moderate alcohol intake is associated with a lower risk of coronary heart disease and total mortality compared with abstinence and heavy drinking (Corrao et al. 2000, Di Castelnuovo et al. 2006). Based on these findings, the possible periodontitis-protective effect of alcohol for participants with a low to moderate alcohol consumption compared with abstinence or alcohol abuse has been discussed (Tezal et al. 2001, Nishida et al. 2004, Shimazaki et al. 2005). However, the results only seemed to follow a J-shaped curve before adjustment for relevant covariates (Tezal et al. 2001). In univariate analysis, we also found a statistically significant J-shaped relation between total weekly alcohol consumption and mean CAL in women indicating that 1–6 drinks/week has a protective effect on CAL compared with both abstinence and higher intake of alcohol; however, only before adjustment (data not shown).

To further assess the association between alcohol and periodontitis, an obvious future task is to establish prospective cohort studies with participants free of periodontitis at baseline. In addition, experimental studies to determine the biological influence of alcohol consumption on periodontal disease are needed. The present study is intended to serve as a baseline examination allowing for future comparative follow-up studies on participants with no current attachment loss.

In conclusion, the results of the present study did not support the hypothesis that total or type-specific alcohol consumption is an independent risk factor for periodontitis. Based on the present results, higher alcohol consumption appears to show an inverse association with attachment loss in men.

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Clinical Relevance

Scientific rationale for the study: Previous studies have related alcohol consumption to periodontitis with conflicting results. Alcohol consumption has different pharmacokinetics in women and men, and intake of various types of alcohol may have different effects on periodontal health. The present study examined the association between total and type-specific alcohol intake, and CAL and BOP in 1,521 female and male adults.

Principal findings: In multivariate analyses, no association between alcohol consumption and either CAL or BOP was observed in women. An inverse relationship between total alcohol intake and CAL was found among men. In type-specific analyses, men with a weekly wine intake of more than 14 drinks compared with men who reported no wine intake had lower ORs for CAL. In male abstainers, a higher OR for BOP was observed, compared with men with light alcohol consumption.

Practical implications: This study showed that higher alcohol consumption appears to have an inverse association with attachment loss in men, but not in women.