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Effect of alcohol consumption on the prevalence and severity of periodontal disease: A meta-analysis

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Abstract
Background: Alcohol intake is the third largest risk factor for disease and disability in the world. Long-term excessive alcohol consumption affects bone metabolism and may play a role in bone loss along with the decreased salivary flow, halitosis, and increased tendency to periodontal disease.
Objective: To assess the influence of alcohol consumption on the prevalence and severity of periodontal disease.
Search and Selection Methods: A search for studies on alcohol and periodontal diseases was conducted in the following databases: PubMed (MEDLINE, Cochrane Library), Web of Science (WoS), and Scopus.
Data Analysis: For dichotomous outcomes, the estimates of effects of an intervention were expressed as odds ratios (OR) using Mantel-Haenszel (M-H) method and, for continuous outcomes, the estimates of effects of an intervention were expressed as mean differences (MD), both with 95% confidence intervals.
Results: 21 studies were included in this meta-analysis. Drinkers were 1.51 times more likely to develop periodontitis than non-drinkers ($p<0.001$). Likewise, heavy drinkers increased BY 2.51 times the periodontitis risk ($p=0.03$). Alcohol consumption worsened periodontal parameters, causing a significant increase in plaque index levels (MD: 4.06; $p=0.04$), probing depth (MD: 0.33; $p<0.001$), and clinical attachment loss (MD:0.33; $p<0.01$), but not bleeding on probing ($p>0.05$).
Conclusions: Alcohol intake increases the periodontitis risk. This risk is even higher the more alcohol is consumed.

Keywords: alcohol drinking; ethanol; periodontal diseases; periodontics

Introduction
The World Health Organization considers alcohol consumption as the third largest risk factor for disease and disability in the world. Alcohol intake influences the immune system with a wide range of adverse effects on different human body organ systems, including the oral cavity [1]. The high consumption of alcoholic beverages is related to various cardiovascular diseases, obesity, strokes, oral diseases, or several cancers. Alcohol intake can affect the oral cavity and the upper digestive tract, causing morphological, metabolic, and functional changes [2]. Periodontitis is a chronic inflammatory disease associated with a dysbiotic bacterial biofilm in a susceptible host that leads to progressive loss of the teeth-supporting structures [3]. Long-term heavy alcohol use affects bone metabolism contributing to bone loss along with the decreased salivary flow, halitosis, and increased tendency for periodontal disease. However, the real effect of alcohol ingestion on periodontal disease is still under investigation, and the findings are not conclusive [4]. This study aimed to assess the influence of alcohol consumption on the prevalence and severity of the periodontal disease.

Materials and Methods
The two authors (ARA and LBB) performed all research steps (search, study selection, data extraction, and evaluation). Later, they jointly selected the articles to consider in this study.

Search strategy
A search for studies on alcohol consumption and periodontal diseases was conducted in the databases PubMed (MEDLINE, Cochrane Library), Web of Science (WoS), and Scopus. A combination of Medical Subjects Headings (MeSH) and free-text terms were used as a search strategy for each database. The searched terms were: ("ethanol"[MeSH Terms] OR "alcohol drinking"[MeSH Terms] OR "alcohol") AND "periodontitis"[MeSH Terms]; "alcohol" AND "periodon*"; TITLE-ABS-KEY (("alcohol" AND ("periodon*")). There were no restrictions about the date or publication language. The exclusion criteria were: a) articles with a relevant risk of bias (score <6 points on the Newcastle-Ottawa methodological quality assessment scale) [5], b) articles with no full-text availability, c) articles without clinical data, and d) studies with non-usable data.

Assessment of methodological quality

The methodological quality of the articles was screened using the Newcastle-Ottawa (NOS) methodological quality assessment scale composed of eight items that evaluate three dimensions (selection, comparability, exposure) [5]. Considering the score obtained, the studies are classified as high quality (≥7 points), moderate quality (4-6 points), and low quality (1-3 points).

Statistic analysis

Data were processed with the RevMan 5.4 meta-analysis software (The Cochrane Collaboration, Oxford, UK). For dichotomous outcomes, the odds ratio (OR) with the Mantel-Haenszel (MH) Chi-square formula was used, and For continuous outcomes, the inverse variance (IV) for the mean difference (MD) was applied, both with 95% confidence intervals (95% CI). Heterogeneity was determined according to the Higgins statistic (I^2). The random-effects model was employed in case of high heterogeneity (I^2>50%). P<0.05 was set as the minimum level of significance.

Results

Study selection

In the initial electronic search, 1346 records were found (300 in PubMed, 685 in WoS, and 361 in Scopus) between the years 1971 and 2021. 695 articles were removed based on the exclusion criteria: a) articles with a relevant risk of bias (<6 points) according to the NOS methodological quality assessment scale [5] (n=198), b) articles with no full-text availability (n=99), c) studies without clinical data (n=185), and d) studies with non-usable data (n=212). After applying these criteria, 21 studies were included in this meta-analysis (Figure 1). The main descriptive characteristics and the methodological quality according to the NOS quality scale of the twenty-one studies [6-26] included in this meta-analysis are shown in Table 1. A total of 66,132 participants, 38,386 males (58%) and 27,746 females (42%) were considered in these articles. The screened studies covered eleven countries: Brazil (5 studies), South Korea (3 studies), Japan (3 studies), the USA (2 studies), Denmark (2 studies), Finland (1 study), Sri Lanka (1 study), Thailand (1 study), Australia (1 study), Colombia (1 study), and Nigeria (1 study). Considering the NOS quality scale [5], two articles (9.5%) had 6 points, sixteen articles (76.2%) got 7 points, and three articles (14.3%) reached 8 points.

Figures

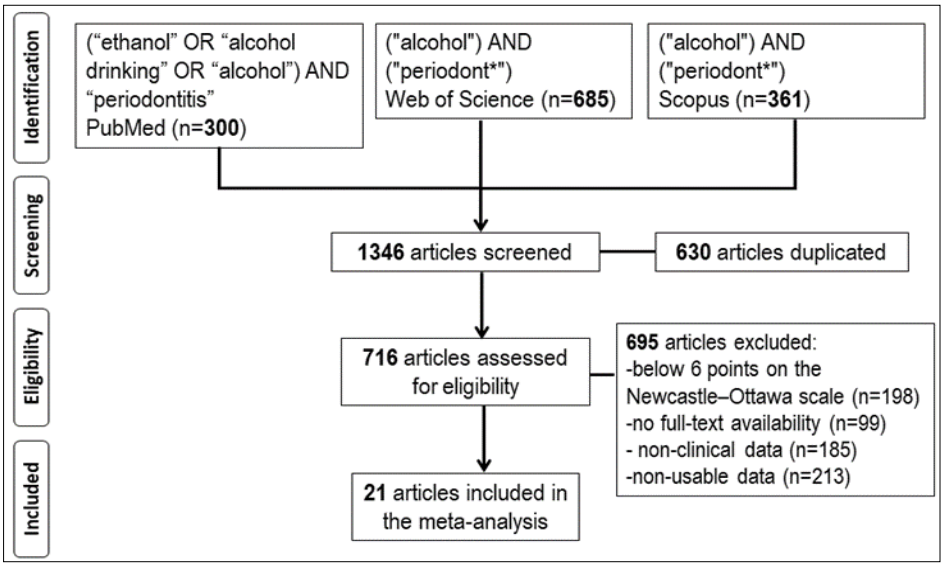

Fig 1: Flow diagram of study selection.

Tables

Table 1: Description and methodological quality evaluation of the twenty-one articles included in this meta-analysis.

Study, year	Country	Study population	Alcohol consumption evaluation	Other parameters analyzed	NOS
Tezal, 2001 [6]	USA	1371 (710M, 661F; 48.1y)	drinks/week (0, 5, 10)	education level, income level, smoking, PI, GB, CAL.	7
Torrungruang, 2005 [7]	Thailand	2005 (1492M, 513F; na)	non user former user current user	Age, education level, income level, smoking, PI, diabetes, BMI, periodontitis.	7
Okamoto, 2006	Japan	1323 (1323M, 0;	g. alcohol/day	Smoking, missing teeth,	7

[8]		43.5y)	0 g./d 1-20 g./d >20 g./d	periodontitis.	
Amaral Cda, 2008 [9]	Brazil	98 (98M, 0F; 43.8y)	Alcoholics No Yes	education level, income level, smoking, residential place, PD, CAL, BOP	6
Kongstad, 2008 [10]	Denmark	1521 (704M, 817F; 53.7y)	drinks/week (<1, 1-6, 7-13, 14-20, ≥ 21)	education level, income level, smoking, diabetes, BMI, physical activity, number of teeth, PI, periodontal disease.	7
Jamieson, 2010 [11]	Australia	425 (205M, 220F; na)	non user user	age, smoking, other drugs, income level, residential place, oral hygiene habits, periodontal disease.	7
Lages, 2012 [12]	Brazil	542 (250M, 292F; na)	Alcohol intake no/occasional Moderate intensive dependance	Age, education level, income level, smoking, BMI, diabetes, PI, CAL, BOP, missing teeth.	7
Costa, 2013 [13]	Brazil	705 (341M, 364F; na)	Alcohol intake no/occasional AUDIT ≥ 8	Age, education level, income level, diabetes, BMI, periodontal disease.	8
Park, 2014 [14]	South Korea	20229 (8645M, 11584F; 46.5y)	AUDIT (0-7, 8-14, 15-19, ≥20)	Education level, income level, residential place, smoking, BMI, diabetes, hypertension, oral hygiene habits, periodontal disease.	7
Hach, 2015 [15]	Denmark	168 (77M, 91F; na)	light user moderate user heavy user	age, education level, income level, smoking, physical exercise, BMI, diabetes, PI, BOP, periodontal disease.	8
Tanner, 2015 [16]	Finland	13819 (13564M, 255F; 19.1y)	non user moderate user intensive user	education level, smoking, oral hygiene habits, DMFT, CPI, BOP.	7
Akpata, 2016 [17]	Nigeria	500 (260M, 240F; na)	non/low user chronic user type of alcohol consumed	age, education level, income level, smoking, residential place, diabetes, periodontal disease.	7
Borba, 2016 [18]	Brazil	77 (36M, 41F; na)	non user user	age, education level, income level, smoking, diabetes, periodontal disease.	6
Maya, 2017 [19]	Colombia	467 (112M, 355F; 53.3y)	non user user	education level, income level, smoking, diabetes, number or teeth, PD, CAL, BOP, periodontitis.	7
Gay, 2018 [20]	USA	7062 (3509M, 3553F; 50.0y)	drinks/week (0, 1-7, ≥ 8)	education level, income level, smoking, BMI, oral hygiene habits, PD, CAL, periodontitis.	7
Suwama, 2018 [21]	Japan	439 (236M, 203F; 70.0y)	Alcohol intake non (0 g.) light-moderate (1-39 g.) heavy (≥40 g.)	foods, BMI, oral hygiene habits, PD, CAL, periodontal disease.	8
Islam, 2019 [22]	Japan	738 (646M, 92F; 40.7y)	Alcohol intake no daily	smoking, BMI, hypertension, oral hygiene habits, stress level, periodontitis.	7
Lee, 2019 [23]	South Korea	9798 (3717M, 6081F; 46.8y)	Alcohol intake no <2 times/week ≥2 times/week	education level, income level, smoking, BMI, foods, oral hygiene habits, periodontitis.	7
Wellappuli, 2019 [24]	Sri Lanka	720 (720M, 0F; na)	Former user Current user	age, education level, smoking, betel chewing, periodontitis.	7
Costa, 2020 [25]	Brazil	138 (67M, 71F; na)	Alcohol intake non/occasional moderate intense	age, education level, income level, smoking, diabetes, PI, PD, CAL, BOP, missing teeth	7
Han, 2020 [26]	South Korea	3987 (1674M, 2313F; na)	non user user	age, education level, income level, smoking, diabetes, oral	7

				hygiene habits, cholesterol, lipoproteins.	

USA: United States of America; M: male; F: female; y: mean age in years; na: not available; PI: plaque index; GB: gingival bleeding index; CAL: clinical attachment loss; PD: probing depth; BOP: bleeding on probing; DMFT: decayed, missing, and filled teeth; CPI: community periodontal index; BMI: body mass index; g. alcohol/day: grams of alcohol per day; AUDIT: alcohol use disorders identification test; NOS: Newcastle-Ottawa methodological quality scale.

Periodontitis risk

Sixteen studies [7, 8, 11-15, 17-20, 22-26] analyzed the periodontitis risk in alcohol drinkers and non-drinkers (Figure 2). Alcohol drinkers increased 1.51-fold the periodontitis risk with a highly statistically significant relationship (OR=1.51; 95%CI: 1.20 to 1.91; p<0.001).

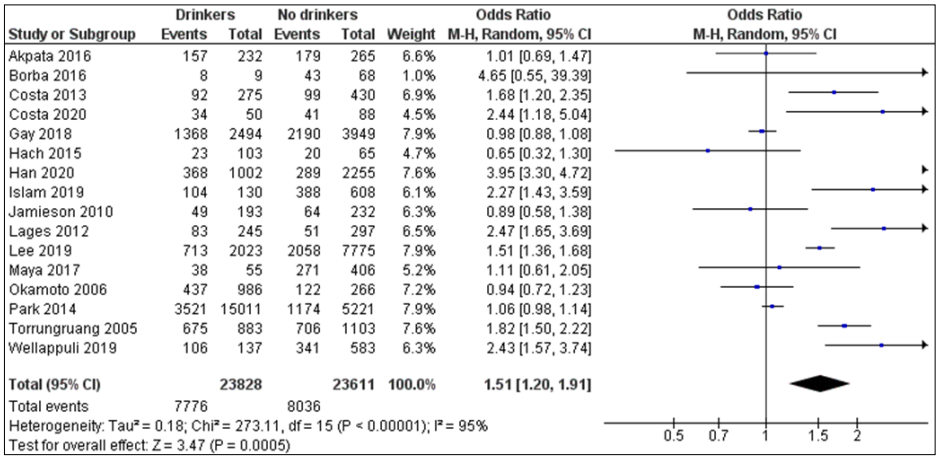

Fig 2: Study data and forest plot graph for the presence of periodontitis in subjects with and without alcohol consumption.

Other Parameters

Table 2 presents the analysis of other parameters such as periodontitis severity in drinkers, alcohol consumption degree in periodontitis patients, plaque index (PI), probing depth (PD), clinical attachment level (CAL), and bleeding on probing (BOP). Three studies [7, 17, 18] evaluated the severity of periodontitis in drinkers. Alcohol consumption did not affect the severity of periodontitis with no statistically significant relationship (OR=1.06; 95% CI: 0.66 to 1.71; p=0.08). On the other hand, six studies [8, 12-14, 20, 23] analyzed the level of alcohol consumption in periodontitis patients. Periodontitis patients were 2.51 times more likely to be heavy drinkers with a highly statistically significant association (OR=2.51; 95% CI: 1.08 to 5.85; p<0.001). Four studies [6, 10, 12, 25] considered the plaque index (PI) in both groups (drinkers and non-drinkers). Regular drinkers had a PI 4.06 units higher than non-drinkers, with statistically significant differences (MD=4.06; CI95%: 0.17 to 7.94; p=0.04). Five studies [9, 12, 20, 21, 25] assessed the probing depth (PD), finding in drinkers a PD of 0.33 mm higher compared to non-drinkers, with a highly significant statistical relationship (MD=0.33; 95% CI: 0.14 to 0.52; p<0.001). Six studies [6, 9, 12, 20, 21, 25] screened the clinical attachment loss (CAL) according to alcohol consumption. Drinkers showed a CAL 0.33 mm higher, with a statistically significant association (MD=0.33; 95% CI: 0.12 to 0.54; p<0.01). Other four studies [9, 12, 16, 25] examined the bleeding on probing (BOP). Alcohol intake did not have a relevant influence on this periodontal parameter. In the statistical analysis, no significant differences were found (MD=0.17; 95% CI: -0.31 to 0.65; p=0.49).

Table 2: Evaluation of different parameters regarding alcohol consumption (drinkers/non-drinkers).

Parameter	Ref.	Outcome	OR/MD	(95% CI)	I^2	P-value
Periodontitis severity in drinkers	[7, 17, 18]	severe periodontitis	OR: 1.06	(0.66 to 1.71)	60%	0.80
Alcohol consumption degree in periodontitis patients	[8, 12-14, 20, 23]	heavy drinkers	OR: 2.51	(1.08 to 5.85)	99%	0.03*
Plaque index (PI)	[6, 10, 12, 25]	drinkers	MD: 4.06	(0.17 to 7.94)	60%	0.04*
Probing depth (PD)	[9, 12, 20, 21, 25]	drinkers	MD: 0.33	(0.14 to 0.52)	98%	<0.001*
Clinical attachment level (CAL)	[6, 9, 12, 20, 21, 25]	drinkers	MD: 0.33	(0.12 to 0.54)	97%	<0.01*
Bleeding on probing (BOP)	[9,12,16,25]	drinkers	MD: 0.17	(-0.31 to 0.65)	0%	0.49

Ref.: references; OR: Odds Ratio; MD: mean difference; (95% CI): 95% confidence interval; I^2: Higgins statistic for heterogeneity (percentage); *statistically significant.

Discussion

In the present meta-analysis on the influence of alcohol consumption on periodontal disease, data from 21 studies have been included. In this study, alcohol consumption increased 1.51 times the probability of developing periodontitis with a highly statistically significant relationship ($p<0.001$). Of the 21 studies that analyzed this parameter, eleven of them [7, 12-14, 17-19, 23-26] corroborated the higher prevalence of periodontitis among drinkers; while the remaining five [8, 11, 15, 20, 22] did not confirm this higher frequency in drinkers. The findings that support the potential link between alcohol consumption and periodontitis are contradictory; some studies establish it, while others do not find it. The mechanisms that justify this association are not yet fully elucidated. Several possible explanations have been proposed. 1) Alcohol consumption would affect polymorphonuclear neutrophils (PMNn), the main phagocytic cells, inhibiting the phagocytosis. Alternatively, there would be an increase in bacterial penetration, and proliferation on the gingival tissues, favoring periodontal inflammation. 2) Alcohol intake could have toxic effects on the periodontium and alter the production of inflammatory cytokines by monocytes, allowing further microbial proliferation. 3) Some of these inflammatory cytokines as tumor necrosis factor-alpha (TNF-α) or interleukins 1 (IL-1) and 6 (IL-6), are closely related to the development of periodontitis [25]. Chronic alcohol intake may lead to impaired immune function, reducing host immune defense mechanisms against periodontal pathogens. Two cytokines (TNF-α and IL-6) account for the initiation and persistence of systemic inflammation that favors the progression and severity of periodontitis. Higher serum levels of these cytokines have also been found in periodontitis patients compared to periodontally healthy individuals [20]. Alcohol consumption induces the suppression of bone turnover and stimulates bone resorption that together with poor oral hygiene habits, determine an increased risk of periodontal disease [17]. In the present study, the severity of periodontitis was not prominently conditioned by alcohol consumption ($p=0.80$). The three studies [7, 17, 18] that considered the severity of periodontitis did not find statistically significant results. Being a drinker did not increase the severity of periodontitis. Nevertheless, most studies only consider the frequency of alcohol consumption but not the amount consumed, making it difficult to determine the real influence of this habit on periodontitis. Similarly, there are also different criteria in the classification of the severity of periodontitis that, together with other confounding factors, do not allow the association between the harmful habit and periodontal disease to be established [7]. In this study, higher alcohol consumption increased 2.51-fold the periodontitis risk with a statistically significant association ($p=0.03$). All the studies [8, 12-14, 20, 23] that evaluated the amount of alcohol consumed agreed in pointing out a proportional relationship between the increase in consumption and the increase in periodontitis prevalence. As previously mentioned, the role of alcohol intake on periodontal disease is controversial, with an aggravating or protective effect according to the studies consulted [23]. The literature supports the beneficial effects of light-moderate alcohol consumption in cardiovascular disorders or type 2 diabetes mellitus. The same could occur in the case of periodontitis. This beneficial effect is attributed to resveratrol that exerts important actions as an antioxidant, anti-inflammatory, antiangiogenic, antimutagenic, and proapoptotic agent. Resveratrol modulates the immune response acting on the transcription of growth factors, cytokines, or interleukins, just as controlling the synthesis of prostaglandins and cell cycle regulatory proteins [14]. Periodontal disease is a multifactorial disorder characterized by chronic local and systemic inflammation. Low alcohol consumption could inhibit the production of proinflammatory cytokines and vasoactive substances, decreasing inflammation. However, the large variability in both evaluation methods of alcohol consumption and periodontitis criteria diagnosis would determine these contradictory findings. According to the available epidemiological evidence, whether or not alcohol intake has a real beneficial effect on the periodontium remains not answered nowadays [20]. In the present meta-analysis, drinkers had higher plaque indices (PI) than non-drinkers with statistically significant differences ($p=0.04$). Of the four studies that determined the PI, three [6, 12, 25] found a higher PI index in drinkers, while only one [10] did not observe it. Dental biofilms are a necessary factor for the development of periodontal diseases, and chronic dental plaque is considered a primary etiologic agent of periodontitis. Alcohol would act as an added factor that promotes the accumulation of dental plaque and, therefore, favors periodontal disease [12]. However, the potential antimicrobial effect of alcohol should also be considered, similar to the effect obtained by some alcohol mouthwashes, reducing dental plaque accumulation [10]. In this study, drinkers had a greater probing depth (PD) with a highly statistically significant relationship ($p<0.001$). All the studies [9, 12, 20, 21, 25] that determined this periodontal parameter confirmed this higher PD in alcoholic beverages consumers. It seems that alcohol developes an adverse effect on various periodontal parameters such as PD, clinical attachment loss (CAL), or bleeding on probing (BOP), although its influence on plaque index (PI) tends to be lower [9]. Moreover, drinkers had a greater clinical attachment loss (CAL) compared to the non-drinkers, with a statistically significant association ($p<0.01$). In contrast, in the case of bleeding on probing (BOP), alcohol consumption did not significantly affect it ($p=0.49$). When assessing CAL, all the studies [6, 9, 12, 21, 25] except one [20] with non-significant results, confirmed this higher CAL in drinkers. These higher CAL values in drinkers may be related to the increase in bacterial lipopolysaccharides in these subjects. Alcohol induces the activation of TLR receptors in many cell types, such as monocytes or macrophages, which leads to an increase both in the production of proinflammatory cytokines and periodontal damage [21]. Drinkers show a higher prevalence of periodontitis along with worse periodontal parameters such as PD, CAL, and BOP [12]. This study has some limitations. In many studies, different evaluation criteria were established for periodontitis and alcohol consumption, creating high heterogeneity and forcing a cautious interpretation of the results. Also, the heterogeneity did not allow adequate analysis of the possible dose-response effect between alcohol consumption and periodontitis. The type of

alcoholic beverage (beer, wine, spirits, etc.) was not adequately assessed. Residual confounding factors could not be eliminated, preventing the true influence of alcohol on periodontitis from being established.

Conclusions

In this study, drinkers were 1.51 times more likely to develop periodontitis than non-drinkers ($p<0.001$). Likewise, heavy drinkers increased by 2.51-fold the periodontitis risk ($p=0.03$). Alcohol consumption worsened periodontal parameters, causing a significant increase in plaque index levels (MD: 4.06; $p=0.04$), probing depth (MD: 0.33 mm; $p<0.001$) and clinical attachment loss (MD: 0.33 mm; $p<0.01$), but not bleeding on probing ($p>0.05$).

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