

Correlations between supra- and subgingival clinical parameters in smokers and individuals who have never smoked

Correlações entre parâmetros clínicos supra e subgingivais em fumantes e em indivíduos que nunca fumaram

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Resumo

Introdução: O hábito de fumar é um fator de risco importante na prevalência, progressão e gravidade das doenças periodontais e parece suprimir a resposta inflamatória marginal no periodonto. **Objetivo:** Correlacionar Índice de Placa visível (IPV) e Índice de Sangramento Gengival (ISG) em fumantes e indivíduos que nunca fumaram, bem como correlacionar ISG e o sangramento à sondagem (SS) nesses dois grupos. **Material e método:** Foram utilizados dados de baseline de um estudo quasi-experimental, no qual 11 pacientes fumantes e 14 indivíduos que nunca fumaram foram submetidos a exames clínicos periodontais no período de setembro de 2010 e outubro de 2011. **Resultado:** A correlação entre IPV e ISG foi positiva para ambos os grupos, sendo forte e estatisticamente significativa nos indivíduos que nunca fumaram e moderada nos fumantes. Com relação ao ISG e SS houve moderada correlação para os fumantes e fraca para os indivíduos que nunca fumaram. **Conclusão:** Fumantes apresentam uma correlação entre IPV e ISG de menor força em relação aos indivíduos que nunca fumaram resultando em um sangramento gengival marginal menos pronunciado.

Descritores: Tabaco; índice periodontal; inflamação; periodontite; gengivite.

Abstract

Introduction: Smoking is a risk factor for prevalence, severity and progression of periodontal disease and appears to suppress marginal periodontium inflammatory response. **Purpose:** To correlate Visible Plaque Index (VPI) and Gingival Bleeding Index (GBI) in smokers and never-smokers, as well as GBI and bleeding on probing (BOP) in these groups. **Material and method:** We used baseline data of one quasi-experimental study in which 11 smokers and 14 subjects who never smoked were submitted to clinical periodontal examinations between September 2010 and October 2011. **Result:** The correlation between VPI and GBI was positive for both groups, it was strong and statistically significant in subjects who had never smoked and moderate in smokers. Regarding GBI and BOP correlations were moderate for smokers and weaker for individuals who had never smoked. **Conclusion:** Smokers have lower strength correlation between VPI and GBI compared to individuals who had never smoked resulting in a less pronounced marginal gingival bleeding.

Descriptors: Tobacco; periodontal index; inflammation; periodontitis; gingivitis.

INTRODUCTION

Gingivitis, which refers to inflammation of the protective tissues of the teeth caused by the presence of supragingival biofilm, develops prior to the establishment of subgingival biofilm, which can initiate periodontitis^{1,2}. There is high prevalence of gingivitis and periodontitis in both developed and developing countries, with differences in periodontitis prevalence being observed among different age groups³.

The correct clinical diagnosis of periodontal diseases is directly related to the presence of inflammatory signs in response to the accumulation of microbial biofilms⁴. Factors, such as the smoking habit, seem suppress the marginal inflammatory response in the

periodontium⁵. However, it remains unclear as to whether bleeding in the base of the periodontal pockets during probing is suppressed in smokers.

The smoking habit is the most important behavioral risk factor to impact the prevalence, progression, and severity of periodontitis. Smoking is associated with other chronic diseases, such as lung and oral cancers and cardiovascular diseases⁶. This habit compromises periodontal health⁷ due to the negative impact of tobacco on the patient's responses to biofilm, periodontal treatment, and healing, leading to higher risk in those patients who have more severe periodontal disease⁸.

Inflammation of the periodontal tissues can be evaluated by the presence of bleeding as an objective and easy-to-evaluate sign. Together with other clinical data, interpretation of bleeding, either by the gingival bleeding index (GBI) or bleeding on probing (BOP), can permit an accurate diagnosis of periodontal diseases and aid in treatment planning. The GBI reflects inflammatory changes in the gingival margin caused by supragingival biofilm, whereas BOP reflects inflammation in the base of the periodontal pocket caused by subgingival biofilm⁹. Studies have shown that GBI is the main clinical parameter of marginal gingival inflammation. However, inflammatory indicators are suppressed, bleeding of the gingival margin is less pronounced¹⁰⁻¹², and inflammatory signs, such as erythema and swelling, are less evident in smokers compared to individuals who have never smoked¹³. These conditions can make it difficult to diagnose periodontal disease in smokers. Moreover, some studies have not found reduced gingival blood flow in smokers^{14,15}. Thus, the literature is not consistent with regard to whether smokers and never-smokers differ in terms of their gingival bleeding parameters. The aim of this study was to correlate the visible plaque index (VPI) and BOP with GBI in smokers and in never-smokers.

MATERIAL AND METHOD

Study Population

This study refers to the baseline examination of patients participating in a quasi-experimental study¹⁶. The sample consisted of individuals diagnosed with chronic periodontitis¹⁷ who requested treatment at the Dentistry Course of the Federal University of Santa Maria (UFSM) in southern Brazil between September 2010 and October 2011. Eligible individuals had at least 12 teeth in their mouth and a probing pocket depth (PPD) of 5 mm or more in at least four teeth, with no indication of extraction or periodontal-endodontic lesions, and had no contraindications to dental treatment. Individuals reported either a minimal consumption of 10 cigarettes per day for the past 6 months (smoker/test group) or a history of never smoking (never-smoker/control group). Individuals with any of the following conditions were excluded: 1) a history of diabetes or hormone replacement therapy, 2) pregnancy, 3) regular use of antibiotics or anti-inflammatory drugs in the past 6 and 3 months, respectively, 4) use of medications that cause increased gingival volume, and 5) any periodontal treatment in the last 12 months.

Ethical Considerations

Eligible subjects were informed about the purpose of the study and were invited to participate. All included patients agreed to participate and signed an informed consent form. This study was conducted in accordance with the Declaration of Helsinki and was approved by the Research Ethics Committee of the UFSM.

Evaluation Methods and Reproducibility

Participants were interviewed to obtain demographic, socioeconomic, behavioral, and medical data. Two calibrated and trained examiners collected clinical parameters, including the VPI,

GBI, PPD, plaque retentive factors (PRFs), and clinical attachment level (CAL), at six sites per tooth (distobuccal, buccal, mesiobuccal, mesiolingual, lingual, and distolingual). Clinical parameters were not measured on the third molars, teeth indicated for extraction, or teeth with periodontal-endodontic lesions. BOP results were categorized as BOP1 (small point bleeding), BOP2 (blood flowing through the groove area), and total BOP (BOP1 + BOP2)⁹. All tests were performed with a manual periodontal probe (CP 15 UNC, Neumar, Brazil) at a dental unit in the Periodontics Clinic of the UFSM.

Examiners were trained to perform all clinical examinations and calibrated for PPD and CAL. Weighted Kappa coefficients (± 1 mm) for examiners 1 and 2 were 0.97 and 0.96, respectively, for PPD and 0.88 and 0.70, respectively, for CAL. Interexaminer values were 0.94 for PPD and 0.84 for CAL.

Measured Outcomes

The primary outcome was the correlation between the GBI and VPI values. The correlation between the GBI and BOP was considered the secondary outcome.

Statistical Analysis

Data were analyzed with the Statistical Package for Social Sciences (SPSS for Windows, version 21.0, SPSS Inc., Chicago, IL, USA). Means were obtained for all data. Standard deviations were obtained for continuous variables and frequency distributions for categorical variables. Pearson's correlation coefficient (r) was used to determine the correlations of GBI with BOP1, BOP2, and VPI in the two groups. The significance level was 5%.

RESULT

The sample comprised 25 subjects, including 11 smokers and 14 never-smokers (Figure 1). The groups were similar with respect to income, age, and gender ($P > 0.05$). The mean duration of the smoking habit among smokers was 25 years, with an average consumption of 20 cigarettes per day. Compared to never-smokers, smokers had four times less bleeding in the gingival margin, despite presenting twice as many PRFs and one-third more visible plaque. Smokers showed almost twice as much suppuration on probing and increased clinical attachment loss. There were no statistically significant differences in PPD, total BOP, BOP1, or BOP2 between the two groups (Table 1).

All observed correlations were positive (Table 2). The correlation between VPI and GBI was moderate in smokers ($r = 0.35$) but strong in never-smokers ($P < 0.05$). Correlations of GBI with BOP1 and BOP2 were moderate in smokers (both $r = 0.35$) and low in never-smokers ($r = 0.18$ and $r = 0.03$, respectively).

DISCUSSION

Smokers presented a moderate correlation between VPI and GBI, whereas a strong correlation was observed among never-smokers. Thus, despite their higher rates of visible plaque, smokers had fewer

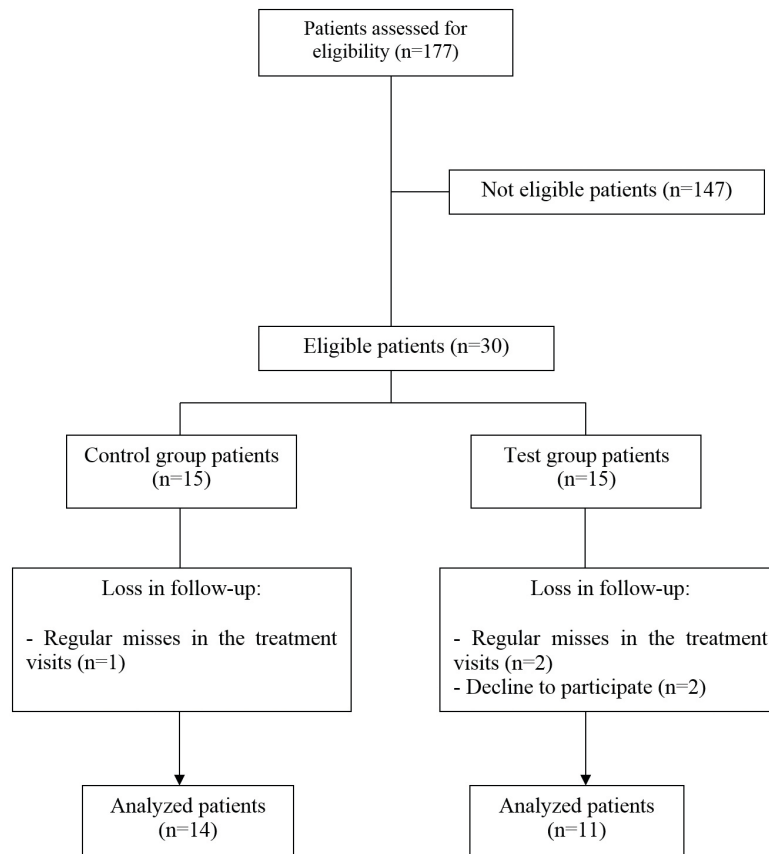


Figure 1. Flowchart.

Table 1. Demographic, socioeconomic, behavioral and clinical variables of sample (% , mean \pm standard deviation)

Variable	Never smokers	Smokers	P
N	14	11	
Age (years)	47.64 \pm 10.81	47.00 \pm 7.81	0.87
Gender male (%)	35.70	36.40	0.97#
Income \leq 3 standard salaries (%)	64.30	54.50	0.62#
Income $>$ 3 standard salaries (%)	35.70	45.50	
Cigarettes number for day	0 \pm 0	19.27 \pm 3.28	
Years of smoking	0 \pm 0	25.68 \pm 9.52	
Teeth number	25.35 \pm 2.97	21.09 \pm 5.14	0.02*
VPI	53.20 \pm 24.55	71.36 \pm 17.04	0.03*
GBI	32.64 \pm 15.27	8.50 \pm 5.95	0.00*
RPF	18.03 \pm 15.70	36.48 \pm 12.77	0.00*
PPD (mm)	3.26 \pm 0.44	3.46 \pm 0.47	0.25
PPD 1-3 mm	69.02 \pm 9.02	55.73 \pm 13.18	0.00*
PPD 4-6 mm	24.29 \pm 7.17	39.16 \pm 13.10	0.00*
PPD \geq 7 mm	6.67 \pm 5.46	5.10 \pm 5.47	0.53*
CAL (mm)	2.95 \pm 1.01	4.94 \pm 1.53	0.00*
SP	9.18 \pm 4.88	17.11 \pm 11.87	0.13
BOP1	29.79 \pm 15.06	18.20 \pm 8.07	0.09
BOP2	41.10 \pm 16.08	48.27 \pm 22.60	0.50
Total BOP	70.89 \pm 13.00	66.48 \pm 19.02	0.49

Chi-Square, * Statistically significant difference between groups ($P < 0.05$); VPI, Visible Plaque Index; GBI, Gingival Bleeding Index; RPF, Retentive Plaque Factors; PPD, Probing Pocket Depth; CAL, Clinical Attachment Level; SP, Suppuration; BOP, Bleeding on Probing.

Table 2. Correlations and r^2 values

	Smokers	Never Smokers
VPI e GBI	($r=0.35$)	($r=0.54$)**
GBI e BOP1	($r=0.35$)	($r=0.18$)
GBI e BOP2	($r=0.35$)	($r=0.03$)

* Pearson Correlation Coefficient; ** Statistically significant difference ($P < 0.05$).

signs of marginal inflammation compared to individuals who had never smoked. The higher correlations of GBI with BOP1 and BOP2 in smokers compared to never-smokers may indicate that the marginal bleeding in smokers is more reflective of subgingival inflammation than it is in individuals who have never smoked.

Limitations of this study include the fact that the number of cigarettes smoked and the duration of the smoking habit were self-reported. Biochemical markers, such as cotinine, may be a reliable alternative to determining tobacco use¹⁸. Furthermore, it was not possible for the investigators to be blinded, due to the presence of smoking-related features (e.g., halitosis and dental staining) in patients.

Several studies have investigated the effect of smoking on the suppression of inflammatory indicators, because the biological mechanisms that lead to increased periodontitis in these patients have not been well elucidated. Bergström et al.¹⁹ induced gingivitis in dental students with similar plaque index values. The vascular response was less pronounced in smokers when compared to those who had never smoked. Photographic evaluation of the vascular changes demonstrated that smoking led to a reduction in the number of blood vessels. This finding can explain the masking of inflammatory signs and, therefore, the reduction in the average GBI in smokers. Dietrich et al.⁵ reported that smoking caused a strong, chronic, and dose-dependent effect in the suppression of gingival bleeding. In the present study, the moderate correlation between VPI and GBI in smokers and strong correlation in never-smokers corroborate the results of the aforementioned studies.

Smoking leads to an increase in the thickness of the gingival epithelium by affecting the proliferation of cells in the basal layer

and the stratum corneum (keratin layer). This increased thickness occurs regardless of periodontal status and can reduce the signs of gingival inflammation during infection^{20,21}. However, one study demonstrated that although smoking has an effect on cell proliferation, the increased epithelial thickness is mostly associated with the inflammatory condition of the periodontium²².

In this study, the correlation between GBI and BOP (1 and 2) was moderate in smokers and weak in patients who had never smoked. Thus, unlike in the gingival margin, smoking does not attenuate bleeding in the base of the periodontal pockets. This result is in agreement with other studies²³⁻²⁵, in which smokers had higher BOP values than never-smokers. Furthermore, the presence of marginal gingival bleeding (GBI) in smokers may suggest that the inflammatory infiltrate is associated with deeper regions of the periodontal pockets. Therefore, the likelihood of inflammation in the base of the periodontal pockets is increased relative to individuals who have never smoked.

Accordingly, more attention should be given to marginal bleeding in smokers. This condition may suggest the presence of subgingival infection, because the suppressed inflammatory response only seems to occur at the gingival margin and not in the base of the periodontal pockets. Overall, the GBI does not appear to be an accurate clinical marker for detecting gingivitis and monitoring oral hygiene habits in smokers. In these individuals, an assessment of the gingival margin condition must include the VPI, obtained at different times, to determine the patient's true oral hygiene condition.

CONCLUSION

Compared to individuals who have never smoked, smokers present a lower correlation between VPI and GBI, resulting in less pronounced gingival margin bleeding. Therefore, gingivitis must be carefully diagnosed in these patients. When present in smokers, marginal gingival bleeding may represent a subgingival infection, as indicated by the greater correlation between GBI and BOP (1 and 2) compared to never-smokers.

REFERENCES

1. American Academy of Periodontology – AAP. Glossary of periodontal terms. 4th ed. Chicago: AAP; 2001.
2. Schätzle M, Loe H, Bueglin W, Anerud A, Boysen H, Lang NP. Clinical course of chronic periodontitis. I. Role of gingivitis. *J Clin Periodontol*. 2003 Oct;30(10):887-901. <http://dx.doi.org/10.1034/j.1600-051X.2003.00414.x>. PMID:14710769
3. Albandar JM, Rams TE. Global epidemiology of periodontal diseases: an overview. *Periodontol 2000*. 2002; 29(1):7-10. <http://dx.doi.org/10.1034/j.1600-0757.2002.290101.x>. PMID:12102700
4. Armitage GC. Periodontal diagnoses and classification of periodontal diseases. *Periodontol 2000*. 2004; 34(1):9-21. <http://dx.doi.org/10.1046/j.0906-6713.2002.003421.x>. PMID:14717852
5. Dietrich T, Bernimoulin JP, Glynn RJ. The effect of cigarette smoking on gingival bleeding. *J Periodontol*. 2004 Jan;75(1):16-22. <http://dx.doi.org/10.1902/jop.2004.75.1.16>. PMID:15025212
6. Doll R. Uncovering the effects of smoking: historical perspective. *Stat Methods Med Res*. 1998 June;7(2):87-117. <http://dx.doi.org/10.1191/096228098668199908>. PMID:9654637
7. Bergström J. Periodontitis and smoking: an evidence-based appraisal. *J Evid Based Dent Pract*. 2006 Mar;6(1):33-41. <http://dx.doi.org/10.1016/j.jebdp.2005.12.018>. PMID:17138394

8. Bergström J. Tobacco smoking and risk for periodontal disease. *J Clin Periodontol*. 2003 Feb;30(2):107-13. <http://dx.doi.org/10.1034/j.1600-051X.2003.00272.x>. PMID:12622851
9. Lie MA, Timmerman MF, Van der Velden U, van der Weijden GA. Evaluation of 2 methods to assess gingival bleeding in smokers and non-smokers in natural and experimental gingivitis. *J Clin Periodontol*. 1998 Sept;25(9):695-700. <http://dx.doi.org/10.1111/j.1600-051X.1998.tb02509.x>. PMID:9763323
10. Bergström J, Preber H. The influence of cigarette smoking on the development of experimental gingivitis. *J Periodontol Res*. 1986 Nov;21(6):668-76. <http://dx.doi.org/10.1111/j.1600-0765.1986.tb01504.x>. PMID:2948000
11. Danielsen B, Manji F, Nagelkerke N, Fejerskov O, Baelum V. Effect of cigarette smoking on the transition dynamics in experimental gingivitis. *J Clin Periodontol*. 1990 Mar;17(3):159-64. <http://dx.doi.org/10.1111/j.1600-051X.1990.tb01080.x>. PMID:2319002
12. Bergström J, Floderus-Myrhed B. Co-twin control study of the relationship between smoking and some periodontal disease factors. *Community Dent Oral Epidemiol*. 1983 Apr;11(2):113-6. <http://dx.doi.org/10.1111/j.1600-0528.1983.tb01367.x>. PMID:6573237
13. Bergström J. Tobacco smoking and chronic destructive periodontal disease. *Odontology*. 2004 Sept;92(1):1-8. <http://dx.doi.org/10.1007/s10266-004-0043-4>. PMID:15490298
14. Mavropoulos A, Brodin P, Rösing CK, Aass AM, Aars H. Gingival blood flow in periodontitis patients before and after periodontal surgery assessed in smokers and non-smokers. *J Periodontol*. 2007 Sept;78(9):1774-82. <http://dx.doi.org/10.1902/jop.2007.060472>. PMID:17760548
15. Baab DA, Oberg PA. The effect of cigarette smoking on gingival blood flow in humans. *J Clin Periodontol*. 1987 Aug;14(7):418-24. <http://dx.doi.org/10.1111/j.1600-051X.1987.tb01547.x>. PMID:2957396
16. Ardaís R, Mário TG, Boligon J, Kantorski KZ, Moreira CH. The effect of smoking on bleeding on probing after nonsurgical periodontal therapy: a quasi-experimental study. *Braz Oral Res*. 2014 Jan-Feb;28(1):1-7.; published online October 21, 2014. <http://dx.doi.org/10.1590/1807-3107BOR-2014.vol28.0058>. PMID:25337935
17. Tonetti MS, Claffey N, and the European Workshop in Periodontology group C. Advances in the progression of periodontitis and proposal of definitions of a periodontitis case and disease progression for use in risk factor research. Group C consensus report of the 5th European Workshop in Periodontology. *J Clin Periodontol*. 2005; 32(6 Suppl):210-3. <http://dx.doi.org/10.1111/j.1600-051X.2005.00822.x>. PMID:16128839
18. González YM, De Nardin A, Grossi SG, Machtei EE, Genco RJ, De Nardin E. Serum cotinine levels, smoking, and periodontal attachment loss. *J Dent Res*. 1996 Feb;75(2):796-802. <http://dx.doi.org/10.1177/00220345960750021001>. PMID:8655777
19. Bergström J, Persson L, Preber H. Influence of cigarette smoking on vascular reaction during experimental gingivitis. *Scand J Dent Res*. 1988 Feb;96(1):34-9. PMID:3422504.
20. Villar CC, Lima AF. Smoking influences on the thickness of marginal gingival epithelium. *Pesqui Odontol Bras*. 2003 Jan-Mar;17(1):41-5. <http://dx.doi.org/10.1590/S1517-74912003000100008>. PMID:12908058
21. van Oijen MG, Gilsing MM, Rijkse G, Hordijk GJ, Sloopweg PJ. Increased number of proliferating cells in oral epithelium from smokers and ex-smokers. *Oral Oncol*. 1998 July;34(4):297-303. [http://dx.doi.org/10.1016/S1368-8375\(98\)80011-0](http://dx.doi.org/10.1016/S1368-8375(98)80011-0). PMID:9813726
22. Gültekin SE, Sengüven B, Karaduman B. The effect of smoking on epithelial proliferation in healthy and periodontally diseased marginal gingival epithelium. *J Periodontol*. 2008 Aug;79(8):1444-50. <http://dx.doi.org/10.1902/jop.2008.070645>. PMID:18672994
23. Müller HP, Stadermann S, Heinecke A. Longitudinal association between plaque and gingival bleeding in smokers and non-smokers. *J Clin Periodontol*. 2002 Apr;29(4):287-94. <http://dx.doi.org/10.1034/j.1600-051X.2002.290403.x>. PMID:11966925
24. Müller HP, Stadermann S. Multivariate multilevel models for repeated measures in the study of smoking effects on the association between plaque and gingival bleeding. *Clin Oral Investig*. 2006 Dec;10(4):311-6. <http://dx.doi.org/10.1007/s00784-006-0067-y>. PMID:16896834
25. Farina R, Tomasi C, Trombelli L. The bleeding site: a multi-level analysis of associated factors. *J Clin Periodontol*. 2013 Aug;40(8):735-42. <http://dx.doi.org/10.1111/jcpe.12118>. PMID:23713685

CONFLICTS OF INTERESTS

The authors declare no conflicts of interest.

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