SEVERITY OF PERIODONTAL DISEASE IN SMOKERS

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Palavras-chave: Hábitos de Fumar. Introdução: O objetivo do presente estudo foi avaliar a gravidade da doença periodontal em uma população de adultos com doença gengival ou periodontal que Doenca Periodontal. Perda do Anexo atende no servico de periodontia da Faculdade de Odontologia da Universidade Clínico Periodontal. Nacional de Rosario. Métodos: O estudo avaliou as condições periodontais em fumantes e não fumantes. A amostra foi composta por 400 indivíduos: 268 não fumantes (SN) e 132 fumantes ativos (E). A classificação das doenças gengivais e periodontais foi baseada nos critérios da American Association of Periodontology (2017), recrutando pacientes com diagnóstico de gengivite, estágio II, III e IV de periodontite. Cada uma dessas patologias foi relacionada às diferentes faixas etárias para avaliar os riscos de perda de inserção clínica. As variáveis periodontais estudadas foram: profundidade de sondagem (DP), nível de inserção (AL), número de dentes presentes (TP), índice de higiene bucal (IOH), mobilidade dentária (M) e sangramento à sondagem (BOP). Uma análise multivariada também foi realizada para determinar o grau de responsabilidade dos diferentes fatores de risco, como idade, gênero, ser fumante e os anos de duração desse hábito, em relação à extensão e gravidade da doença periodontal. **Resultados**: Vinte e nove por cento das mulheres e quarenta por cento dos homens eram fumantes. As variáveis periodontais nos grupos de S e NS comportaram-se da seguinte forma: PD para NS 4.19 (\pm 0.67) e para S 5.37 (\pm 0.64); AL para NS 3.43 (\pm 1.28) e para S 4.30 (±1,43); BOP para NS 41 (±23,76) e para S 43,28 (±23,56); OHI para NS 1,75 (±0,61) e para S 1,82 (± 0,53); TP para NS 21,38 (± 6,13) e para S 21,20 (± 6,60); e M para NS 1,65 (± 0,74) e para S 2,10 (± 0,65). Conclusão: Os estágios II e III da periodontite se comportaram de maneira semelhante nos dois grupos, mas guando analisamos o estágio IV da periodontite, esta foi mais prevalente em fumantes, independentemente de sua idade. Quando a população foi estratificada de acordo com a idade dos indivíduos, o estágio IV da periodontite foi mais prevalente em pessoas idosas que não fumavam e em indivíduos jovens com menos de 40 anos que tinham o hábito. As variáveis periodontais PD, CAL e M foram encontradas com valores mais severos no grupo S.

ABSTRACT

Introduction: The objective of the present study was to evaluate the severity of periodontal disease in a population of adults with gingival or periodontal disease who assist at the periodontics service of the Faculty of Dentistry of the National University of Rosario. Methods: The study evaluated the periodontal conditions in smokers and non smokers. The sample consisted of 400 individuals: 268 non-smokers (NS) and 132 active smokers (S). The classification of gingival and periodontal diseases was based on the criteria of the American Association of Periodontology (2017), recruiting patients with diagnoses of gingivitis, stage II, III and IV of periodontitis. Each one of these pathologies was related to the different age groups to assess the risks of clinical attachment loss. The periodontal variables studied were: probing depth (PD), attachment level (AL), number of teeth present (TP), oral hygiene index (OHI), tooth mobility (M) and bleeding on probing (BOP). A multivariate analysis was also carried out to determine the degree of responsibility of the different risk factors, such as age, gender, being a smoker and the years of that habit duration, in relation to the extent and severity of the periodontal disease. Results: Twenty-nine per cent of women and fourty percent of men were smokers. The periodontal variables in the groups of S and NS behaved in the following way: PD for NS 4.19 (\pm 0.67), and for S 5.37 (\pm 0.64); AL for NS 3.43 (±1.28), and for S 4.30 (±1.43); BOP for NS 41 (±23.76), and for S 43.28 (±23.56); OHI for NS 1.75 (±0.61), and for S 1.82 (±0.53); TP for NS 21.38 (±6.13), and for S 21.20 (±6.60); and M for NS 1.65 (±0.74), and for S 2.10 (±0.65). **Conclusion:** Stage II and III of periodontitis behaved similarly in both groups, but when we analysed stage IV of periodontitis, it was more prevalent in smokers, regardless of their age. When the population was stratified according to the age of the individuals, stage IV of periodontitis was more prevalent in older people who did not smoke and in young individuals under 40 who had the habit. PD, CAL and M periodontal variables were found with more severe values in the S group.

Keywords: Smoking Habbits. Periodontal Disease. Periodontal Clinical Attachment Loss.

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INTRODUCTION

The habit of smoking is considered as a risk factor for periodontal disease. ^{1,2} Tobacco smoke has some 4000 to 5000 toxic substances, some of which are known to be carcinogenic elements for humans. A higher prevalence of periodontal disease has been reported in individuals who smoke.^{3,4}

The clinical evidence shows that a greater bone loss occurs in smokers due to the mineral content of the bone being affected and its quality diminished.^{5,6} The habit of smoking is strongly associated with the severity of the disease, the number of teeth lost, and refractory and recurrent periodontitis.^{7,8,9}

In general, smokers have a lower response to different types of gingival and periodontal therapies, both from the point of view of healing times and in the parameters of final wound healing.^{10,11,12,13} Smokers immune system is depleted in terms of response effectiveness, and a plausible explanation is based on the fact that smokers present vascular constriction in their gingiva. This would lead to a minimal presence of cellular defence elements at the critical site of the infection, together with a decrease in the titer and avidity of the antibodies.^{14,15,16,17}

The objective of the present study was to determine the severity of the periodontal pathology in a group of patients attending the periodontics service of the Dental School of Rosario and to assess the relationship with other variables that may increase or decrease the severity of periodontal lesions.

MATERIALS AND METHODS

This epidemiological cross-sectional study (number of ethics commitee approval: 17552/269) of a group of patients who attended the periodontics service of the Faculty of Dentistry of Rosario covered a three-month period for collecting information obtained from oral clinical examination. Four hundred patients with periodontal disease not treated were observed. The following variables were taken as an inclusion criterion: the absence of systemic diseases that may be of risk for periodontal diseases, the absence of any type of periodontal gingival therapy in the last year and the patient who had consumed antibiotics of any kind in the last six months. The following were used as exclusion criteria: the presence of removable partial dentures, the use of orthodontic therapies in the last two years, and the regular intake of any type of medication that can modify the immunological and morphological parameters of the gingiva. Passive smoking patients who had regular contact with smoke were excluded from the study.

attachment level (AL), number of teeth present (TP), oral hygiene index (OHI), tooth mobility (M) and bleeding on probing (BOP).

All the variables were performed on mesial and distal sites of the teeth with the exception of the third molars, and the supernumerary teeth were excluded from the examination.

To examine the PD, AL and BOP, the measuring instrument used was the Marquis probe, graduated at 3, 6, 9 and 12 mm, with a tip of 0.5 mm in diameter. The records were taken with two measurements in the mesial and distal faces close to the union with the vestibular and lingual or palatal faces of all the teeth studied. The gingival and periodontal pathologies were classified according to the criteria of the American Association of Periodontology in regard to the severity of the nosological entity in stage II, III and IV of periodontitis and by the extent of the destruction with localised parameters (when it affects up to 30% of sites or dental faces) and generalised (when the level of involvement is more than 30% of the sites).^{18,19} For the BOP, we follow the criteria of Van der Velden (1979),²⁰ with which we determine the faces of positive ones such as those that bled when the probe was removed or within 30 seconds after being removed.

To examine the M, we follow the criteria of Miller (mobility index) a cotton clamp was used and categorised into four ranks: grade 0 without mobility, grade 1 vestibular – lingual or palatal mobility, grade 2 adds mobility towards mesial and distal, and grade 3 adds intrusion.

The oral hygiene examination was determined by visual inspection through a modified Greene and Vermillion index (1964).²¹

Half of the vestibular and lingual faces were not observed so as to avoid incorporating measurement biases into the study due to the presence of gingival recessions that could have been due to traumatic causes and not to infectious causes compatible with the nature of initiation and progression of periodontal diseases.

Smokers were defined as those who smoked cigarettes with pulmonary aspiration of smoke and consumption of blonde cigarettes.

Two different groups – non-smokers and smokers – were formed. Subsequently, the number of cigarettes consumed daily and the accumulated years of the habit were assessed.

The measurements were made by a single calibrated examiner and with the methodology blind regarding knowledge about the presence of smoking.

All individuals were informed about their participation in the epidemiological study and asked to sign a consent from. The study was adapted to the norms of the bioethics committee of the National University of Rosario.

The variables studied were: probing depth (PD),

Statistical analysis

The individuals were taken as the unit of analysis. To assess the proportion of smokers within the population, a continuous Goodman confidence interval was used.

For the relationship between the periodontal variables and the independent variable measured (smoking), a Kruskal–Wallis test was used.

A logistic regression analysis was implemented to categorise the different risk predictors and their influence on insertion loss as a gold parameter for measuring the final loss of periodontal support and to categorise the resulting disease.

The predictors studied were: age as an acquired risk factor and unfolded in categories (up to 30 years, from 30 to 40 years, from 40 to 50 years and more than 50 years of age), gender as an innate risk factor, the presence of smoking in three categories (not present, up to 10 cigarettes per day and more than 10 cigarettes per day) and the accumulated time since the habit began (observed as up to 10 years and more than 10 years).

The response variable was also categorised to facilitate the epidemiological analysis in four groups: clinical insertion loss d" 4mm (ICP d" 4mm), clinical insertion loss e" 5mm (ICP e" 5mm), clinical insertion loss in up to 30% of the sites (ICP < 30%), and clinical insertion loss in more than 30% of the sites (ICP > 30%)

All the variables were collected in all the sites (dental faces measured).

In the multivariate model, each predictor was expressed with its influence on the response variable accompanied by its OR and a confidence interval of 95%.

Epidat and Epi Info epidemiological programs (OMS) were used for data management, and SPSS[®] was used for the calculation of the tests and modelling.

The probability of type I error was set equal to 0.05 to obtain statistical significance.

RESULTS

The sample population consisted of 400 individuals categorised according to gender, with 250 women with an average age of 44.22 (95% CI 41.27–47.17) and 150 men with an average age of 40.36 (95% CI 34.86–42.60).

In reference to those smoking cigarettes, 268 did not smoke (NS) and 132 did (S). Regarding the age of the participants, there were no significant differences between the groups, with an average age of 42.99 (95% CI 38.27–43.69) and an age range between 16 and 73 years for the NS group, and an average age of 40.85 (95% CI 35.46–42.70) and an age range between 15 and 69 years for the S group (Table 1).

Twenty-nine per cent of the women and 40% of the men were smokers. The distribution of the absolute numbers is shown in Table 1.

When we determined the number of cigarettes consumed daily, the average was 13.64 cigarettes (95% CI 10.67–16.60) with a range of consumption of 5 to 50 cigarettes per day. When we observed the years since the smoking habit began, the average was 18.41 years (95% CI 9.65-22.17) with a range of 3 to 50 years. Periodontal variables in the groups of S and NS behaved in the following way: PD for NS 4.19 (± 0.67), and for S 5.37 (0.64), with a p value of 0.005; AL for NS 3.43 (1.28), and for S 4.30 (1.43), with a p value of 0.039; BOP for NS 41 (23.76), and for S 43.28 (23.56), with a p value of 0.545; OHI for NS 1.75 (0.61), and for S 1.82 (0.53), with a p value of 0.463; TP for NS 21.38 (6.13), and for S 21.20 (6.60), with a p value of 0.80; and M for NS 1.65 (0.74), and for S 2.10 (0.65), with a p value of 0.021. The values of PD, AL and M were statistically significant, with an aggravation in the S group (Table 2).

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		Non smokers	Smokers	
Women age:44.22	Ν	178	72	
(95Cl 41.27-47.17)	%	71%	29%	
Men age: 40.36	Ν	90	60	
(95Cl 34.86-42.60)	%	60%	40%	
Total		268	132	

 Table 1: Distribution of patients (non smokers and smokers) according to gender.

Note: Age of Non Smokers 42.99(95Cl 38,27-43.69) range from 16 to 73 years, smokers - 40,85 (95Cl 35,46-42,70) range from 15 to 69 years.

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	Non-smokers	Smokers	<i>p</i> value
Ν	268	132	
Probing depth (n ± SD)	4.19 (±0.67)	5.37 (±0.64)	0.005*
Attachment level ($n \pm SD$)	3.43 (±1.28)	4.30 (±1.43)	0.039*
Bleeding on probing ($n \pm SD$)	41 (±23.76)	43.28 (±23.56)	0.545
Oral hygiene index (n ± SD)	1.75 (±0.61)	1.82 (±0.53)	0.463
Teeth present (n ± SD)	21.38 (±6.13)	21.20 (±6.60)	0.80
Mobility (n ± SD)	1.65 (±0.74)	2.10 (±0.65)	0.021*

Table 2: Evaluation of the behaviour of the variables in the different groups.

Note: * Mann-Whitney test. Significant difference at the level of significance = p < 0.05. * Significant data.

Within the cross-sectional design, the prevalence of the different nosological entities was obtained in relation to the presence or absence of the habit, which were expressed in absolute frequencies. Individuals in the NS group had 13% gingivitis associated with plaque, 59.4% stage III of periodontitis, and 27.5% severe periodontitis. Individuals in group S were distributed as 8.3% with plaque-associated gingivitis, 51.7% with stage II and III of periodontitis, and 40% with stage IV of periodontitis (Table 3).

 Table 3: Relationship between periodontal diagnosis and smoking.

		Non-smokers N (%)	Smokers N (%)
Deviedentel	Gingivitis	35 (13.0%)	11 (8.3%)
Periodontal Diagnosis	Stage II and III Periodontitis	160 (59.4%)	68 (51.7%)
	Stage IV Periodontitis	73 (27.5%)	53 (40.0%)

When we separated the populations of NS and S according to age (younger or older than 40 years of age), we observed the following: in the NS group, there was a higher prevalence of plaque-associated gingivitis in younger individuals, a slightly higher prevalence of stage II and III of periodontitis in those under 40 and a greater prevalence of stage IV of periodontitis in older individuals (Table 4).

Table 4: Relationship between age and diagnosis in the group of non-smokers.

	Under 40 y	vears of age	Over 40 ye	ears of age	To	tal
	Smokers	Non smokers	Smokers	Non smokers	Smokers	Non smokers
Gingivitis	11 (17.2%)	33 (26.2%)	0 (0%)	2 (1.4%)	11 (8.3%)	35 (13.0%)
Stage II and						
III periodontitis	37 (58.6%)	85 (67.7%)	31 (45.2%)	75 (52.1%)	68 (51.7%)	160 (59.4%)
Stage IV						
periodontitis	16 (24.1%)	8 (6.2%)	37 (54.8%)	65 (46.6%)	53 (40.0%)	73 (27.5%)
Total	64 (48.0%)	126 (47.0%)	68 (52.0%)	142 (53.0%)	132 (100.0%)	268 (100.0%)

For the S group, percentages remained similar except for individuals younger than 40 years, who showed a significant increase in the prevalence of severe periodontitis (Table 4). The logistic regression analysis was carried out through a study of risk predictors, including age (up to 30 years, from 30 to 40 years, from 40 to 50 years and more than 50 years), gender, the presence of smoking (not present, up to 10 cigarettes per day and more than 10 cigarettes per day) and the duration of the habit (up to 10 years and more than 10 years), and crossing them with response variables, "CAL d" 4mm, "CAL e" 5mm, localised CAL < 30% and generalized CAL > 30%). The results of the variables were accompanied by their odds ratio and its corresponding 95% confidence interval (Table 5). As the ages increases the smoking patients is 2 or 3 more likely to develop periodontal disease in both gender. Patients who smoke more than 10 cigarettes per day have 4 or 5 more chances of developing periodontal disease, this possibility became worse as time passes.

Table 5: Predictors and their relation	n with age, gender a	nd smoking habits.
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PREDICTOR	"CALd"4mm OR CI95%	"CAL e"5mm OR CI95%	CAL < 30% OR CI 95%	CAL>30% OR CI 95%
Age				
Up to 30 years	0.99 (0.85–1.20)	0.82 (0.75-1.01)	1.58 (1.06–2.35)	1.25 (0.99-1.89)
From 30 to 40 years	0.97 (0.81–1.19)	0.89 (0.65-1.09)	1.75 (1.02–2.45)	1.45 (1.03–2.03)
40 to 50 years	1.18 (0.95–1.45)	1.21 (1.01–2.34)	2.64 (1.94–3.39)	3.01 (2.45-3.89)
More than 50 years	1.99 (1.20-2.99)	2.28 (1.79-3.21)	3.89 (2.45-4.38)	3.45 (2.68–3.99)
Gender				
Female	0.93 (0.81–1.03)	0.87 (0.78-1.03)	0.89 (0.79–1.04)	0.93 (0.80-1.19)
Male	0.95 (0.78–1.04)	0.85 (0.74-1.04)	1.07 (0.88–1.45)	1.05 (0.85-1.42)
Smoking Habits				
Not present	0.98 (0.75–1.25)	0.88 (0.75-1.02)	0.99 (0.88–1.41)	1.09 (0.87-1.47)
Up to 10 cigarettes per day	2.48 (2.40–4.41)	3.71 (2.48-5.30)	2.99 (2.01-3.89)	3.47 (2.98–4.74)
More than 10 cigarettes per d	lay 3.89 (3.01–4.47)	3.97 (2.99–5.01)	3.88 (2.89–4.57)	4.59 (3.84-6.10)
Habit time				
Up to 10 years	3.57 (2.90-4.84)	3.43 (2.78–4.89)	3.48 (2.78–4.35)	4.49 (2.99–5.84)
More than 10 years	4.20 (2.29–5.89)	4.79 (3.41-5.02)	5.45 (3.53-6.81)	6.08 (5.01-7.89)

DISCUSSION

When we take an epidemiological and descriptive approach to the the population (pacients who assit at the Periodontics Service of the Faculty of Dentistry) in terms of the prevalence of periodontal disease, we observe that there was a higher prevalence of gingivitis in the NS group and in younger individuals. Stage II and III of periodontitis had a similar prevalence in both groups, but when we analysed stage IV of periodontitis, it was more prevalent in smokers, regardless of their age.

When we studied the age of individuals (under and over 40 years old) and crossed it with the variable "smoking", we observed that gingivitis was more prevalent in younger individuals, regardless of habit.

Stage II and III of periodontitis was similarly distributed in the S and NS groups. Severe periodontitis was more prevalent in older individuals who did not smoke, but the most valuable finding was the prevalence of severe periodontitis in young individuals under 40 years old who had the habit, which is a warning sign in terms of the destructive ability of tobacco use on oral tissues and at early ages.

In reference to the relationship between periodontal variables studied and presence of the habit, (PD), (AL) and (M) were found with more severe values in the S group. Other

studies also determined a higher rate of CAL and periodontal tissues in smokers.^{22,23,24}

The OHI and TP variables remained similar in both groups and even when compared with populations previously studied by our research team,^{25,26} and in comparison with other published works.^{27,28}

As periodontal disease has a multi-causal aetiological factor, it is not possible to analyse it only from the descriptive epidemiological point of view, and we delve into it more precisely by means of modelling carried out with a multivariate analysis through the logistic regression of the different risk predictors that may influence the severity and extent of CAL.

This analysis was useful for a more specific discrimination of risk indicators that may have greater or lesser weight on the variables studied and to establish precedents in the performance of subsequent analytical epidemiological studies.

The multivariate analysis was accompanied by its odds ratio and its 95% confidence interval.

Analysing the results obtained, we can deduce that the positive influence of age on slight CAL (equal to or less than 4mm) was similar to that on the severe CAL (equal to or greater than 5 mm). It also had similar influence on the extent of localised CAL (in less than 30% of the sites) and on the generalised CAL (more than 30% of the sites). This indicates that older individuals have a higher probabilities of losing periodontal tissue regardless of whether they are smokers.⁶

Gender was not a determiner of risk for the CAL in this study, which is unlike other studies that suggested the male gender as having a higher risk of CAL.^{29,30,31}

The presence of smoking definitely enhanced the extent and severity of periodontal disease, showing that individuals who smoked more than 10 cigarettes per day had the highest probabilitys of the risk estimators, the chances increased almost four times for localizated periodontits, and almost five times for generalized periodontitis; thus determining the dose-dependent nature of smoking with the periodontal pathology previously exposed in the literature.³²

Finally, the number of years for which the habit persists remained a strong predictor of risk of periodontal tissue loss, demonstrated by high figures of estimators for those who smoked for more than 10 years consecutively.

If we perform a thorough analysis of the variables discussed here, we can observe that regardless of age and gender, smoking more than 10 cigarettes per day and for a time period greater than 10 years considerably aggravates both the extent and the severity of the loss of periodontal insertion.

The descriptive epidemiological analysis of the sample also determines a higher prevalence of loss of periodontal insertion in young individuals under 40 who smoke.

Analytical epidemiological studies are useful to observe the degree of responsibility of these and other risk factors and their relationship with periodontal disease.³³

CONCLUSIONS

Stage II and III of periodontitis behaved similarly in both groups, but when we analysed stage IV of periodontitis, it was more prevalent in smokers, regardless of their age. When the population was stratified according to the age of the individuals, stage IV of periodontitis was more prevalent in older people who did not smoke and in young individuals under 40 who had the habit. PD, CAL and M periodontal variables were found with more severe values in the S group.

The extent and the severity of the loss of periodontal insertion have a direct association with the quantity of cigarettes (more than 10 cigarettes per day) and time of smoking (more than 10 year) in spite of age and gender.

REFERENCES

1. Page R, Offenbacher S, Schroeder HE, Seymour GJ, Kornman KS. Advances in the pathogenesis of periodontitis. Summary of developments, clinical implications and future directions. Periodontol 2000 1997; 14: 216–246. 2. Have J, Wattles J, Crowley M, Mandell R, Joshipura K, Kent RL. Evidence for cigarette smoking as a major risk factor for periodontitis. J Periodontol 1993 January; 64 (1): 16–23.

3. Rees T. Drugs and oral disorders. Periodontology 2000, 1998; 18: 21–36

4. The tobacco epidemic: A crisis of startling dimensions. www.who.org

 Arno A, Schein O, Lovdal A, Waerhaug J. Alveolar bone loss as a function of tobacco. Acta Odontol Scand 1959; 17: 3–9.
 Bergström J, Sören E, Preber H. Cigarette Smoking and Periodontal Bone Loss. J Periodontol 1991; 62: 242–246.

7. J. To I Figueras. Tobacco and cancer: From the Epidemiological Association to molecular evidence. Med Clin (Barc) 1999; 112: 589–594.

8. Ah MKB, Johnson GK, Kaldahl WB, Patil KD, Kalkwarf KL. The effect of smoking on the response to periodontal therapy. J Clin Periodontol 1994; 21: 91–97.

9. Grossi SA, Zambon JJ, Ho AW. Assessment of risk for periodontal disease. 1. Risk indicators for attachment loss. J Periodontol 1994; 65: 260–267.

10. Mandel I. Smoke signals: An alert for oral disease. J Am Dent Assoc 1994; 125: 872–877.

11. Bastiaan R, Reade PC. The effect of tobacco smoking on oral and dental tissues. Aus Dent J 1976; 21: 308.

12. Kristoffersen T. Periodontal condition in Norwegian soldiers. An epidemiological and experimental study. Scand J Dent Res 1970; 8: 34.

13. Sheiham A. Periodontal disease and oral cleanliness in tobacco smokers. J Periodontol 1971; 42: 259–263.

14. Bergström J, Preber H. Tobacco use as a risk factor. J Periodontol 1994; 65: 545–550.

15. MacFarlane G, Herzberg M, Wolff L, Hardie N. Refractory periodontitis associated with abnormal polymorphonuclear leukocyte phagocytosis and cigarette smoking. J Periodontol 1992; 63: 908–913.

16. James J, Sayers N, Drucker D, Hull P. Effects of tobacco products on the attachment and growth of periodontal ligament fibroblast. J Periodontol 1999; 70: 518–525

17. Ratka-Kruger P, Neukranz E, Raetzke P. Guided tissue regeneration procedure with bioresorbable membranes versus conventional flap surgery in the treatment of intrabony periodontal defect. J Clin Periodontol 2000; 27: 120–127.

18. The American Academy of Periodontology. Annals Vol. 4, International Workshop for a Classification of Periodontal Disease and Conditions. Vol. 4 No.1, 1999.

19. The American Academy of Periodontology. Proceedings of the Word Workshop in Clinical Periodontics Chicago: A.A.P. 1989, 1/23-24.

20. Van der Velden U. Probing force and the relationship of the probe tip to the periodontal tissues. J Clin Periodontol 1979 Apr; 6 (2): 106–14.

21. Greene JC, Vermillion JR. The simplified oral hygiene index. J Am Dent Assoc 1964 Jan; 68: 7–13.

22. Bolin A, Lavstedt S, Frithiof L, Henrikson CO. Proximal alveolar bone loss in a longitudinal radiographic investigation. IV. Smoking and some other factors influencing the progress in individuals with at least 20 remaining teeth. Acta Odontol Scand 1986; 44: 263–269.

23. Beck JD, Cusmano L, Green-Helms W, Koch GG, Offenbacher S. A 5-year study of attachment loss in community-dwelling older adults: incidence density. J Periodont Res 1997; 32: 506–515.

24. Machtei EE, Dunford R, Hausmann E, et al. Longitudinal study of prognostic factors in established periodontitis patients. J Clin Periodontol 1997; 24: 102–109.

25. Eficacia de la higiene oral en adultos jóvenes fumadores y no fumadores. Feser G, Funosas E, Marí G, Escovich L. Av. Odontoestomatol 2003; 19-3: 141–148.

26. Dental death rate prevalence in smoker and non smoker adults with periodontal disease. Feser G, Funosas E, Marí G, Escovich L. Med Oral Patol Oral Cir Bucal. 2005 Jul 1;10 Suppl 2:E109-16. 27. Bergström J, Eliasson S, Preber H. Cigarette smoking and periodontal bone loss. J Periodontol 1991; 62: 242–246.

28. Kerdvongbundit V, Wikesjö UME. Effect of smoking on periodontal health in molar teeth. J Periodontol 2000; 71: 433–437.

29 U.S. Public Health Service, National Institute of Dental Research. Oral Health of United States Adults; National Findings. Bethesda, MD: National Institute of Dental Research; 1987. NIH publication number 87-2868.

30. U.S. Public Health Service, National Center for Health Statistics. Periodontal disease in adults, United States 1960-1962. Washington, DC: Government Printing Office; 1965. PHS publication number 1000, Series 11 No. 12.

31. Van der Velden U, Abbas F, Van Steenbergen TJ, et al. Prevalence of periodontal breakdown and presence of Actinobacillus actinomycetemcomitans in subjects with attachment loss. J Periodontol 1989; 60: 604–610.

32. Do LG, Slade GD, Roberts-Thomson KF, Sanders AE. Smoking-attributable periodontal disease in the Australian adult population. J Clin Periodontol 2008; 35: 398–404.

33. Genko RJ, Borgnakke WS. Risk factors for periodontal disease. Periodontol 2000.2013. Jun; 62 (1): 59.94.