JOURNAL OF APPLIED ORAL SCIENCE
www.fob.usp.br - www.scielo.br
J Appl Oral Sci *2004; 12(4): 273-9*

INFLUENCE OF AGE, SEX, PLAQUE AND SMOKING ON PERIODONTAL CONDITIONS IN A POPULATION FROM BAURU, BRAZIL

INFLUÊNCIA DA IDADE, SEXO, PLACA BACTERIANA E FUMO NAS CONDIÇÕES PERIODONTAIS EM UMA POPULAÇÃO DE BAURU, BRASIL

Mariana Schützer RAGGHIANTI[1], Sebastião Luiz Aguiar GREGHI[2], José Roberto Pereira LAURIS[3], Adriana Campos Passanezi SANT'ANA[2], Euloir PASSANEZI[4]

1- MSc, Graduated Student, Department of Periodontology, Bauru Dental School, University of São Paulo.
2- PhD, Assistant Professor, Department of Periodontology, Bauru Dental School, University of São Paulo.
3- PhD, Assistant Professor, Department of Public Health, Bauru Dental School, University of São Paulo.
4- PhD, Associate Professor, Department of Periodontology, Bauru Dental School, University of São Paulo.

Corresponding address: Mariana Schützer Ragghianti - Rua Araújo Leite n.23-32, apto.102 - Altos da Cidade - CEP: 17043-070 Bauru-SP. Email: mragghianti@yahoo.com - telefones: (14) 32239054 / 97949098

Received: November 11, 2003 - Returned for modification: May 5, 2004-11-08 - Accepted: August 17, 2004

ABSTRACT

Epidemiology is the study of health and disease in populations, and of how these conditions are influenced by heredity, biology, physical environment, social environment, and personal behavior. There are many epidemiological studies in Brazilian population but few about the influence of some risk factors in periodontal conditions. This cross-sectional study was performed to assess the influence of age, sex, plaque and smoking on periodontal disease in a population from Bauru (Brazil). Data concerning periodontal status were collected from 380 patients in the University of São Paulo (USP). Measurements of periodontal pocket depths (PPD), clinical attachment levels (CAL), plaque index (PI) of four sites in all teeth were registered. The influence of age, sex and smoking habits on the periodontal parameters were statistically evaluated using descriptive statistical and ANOVA. The correlation between plaque and periodontal parameters was analyzed by Pearson's correlation coefficient. The results showed an increase in the mean of periodontal destruction (PPD and CAL) and a higher number of sites with severe losses with increasing age. Correlation among percentage of sites with plaque and periodontal parameters (PPD and CAL) were positive but weakly related. The male group showed significantly higher means of CAL than the female. Smokers had significantly higher PPD and CAL means than non-smokers. Aging, smoking habit, male sex, and percentage of sites with plaque were associated with a great increase of periodontal destruction, being important factors in the diagnosis of the periodontal disease in this Brazilian population.

Uniterms: Epidemiology; Periodontal disease; Aging; Sex; Smoking.

RESUMO

A Epidemiologia é o estudo da saúde e da doença nas populações e de como esses estados são influenciados pela hereditariedade, biologia, ambiente físico e social e comportamento pessoal. Existem vários estudos epidemiológicos na população brasileira porém poucos sobre a influência de fatores de risco nas condições periodontais. Este estudo transversal objetivou avaliar a influência da idade, sexo, placa bacteriana e fumo na doença periodontal em uma amostra da cidade de Bauru. Os dados sobre as condições periodontais foram obtidos de 380 pacientes da Universidade de São Paulo, sendo registradas medidas de profundidade de sondagem, nível de inserção clínica e índice de placa de 4 sítios de todos os dentes. A influência da idade, sexo e fumo foram avaliadas utilizando-se a Estatística descritiva e a Análise de Variância (ANOVA) e o papel da placa bacteriana, por meio do coeficiente de correlação de Pearson. Os resultados mostraram um aumento nas médias de profundidade de sondagem e nível de inserção e um maior número de sítios com perdas severas com o avanço da idade. A correlação entre a porcentagem de sítios com placa e os parâmetros periodontais foi positiva porém fraca. O sexo masculino apresentou médias significantemente maiores de nível de inserção do que o sexo feminino. Os fumantes obtiveram médias significantemente maiores de profundidade de sondagem e de nível de inserção quando comparados aos não-fumantes. A idade, hábito de fumar, sexo masculino e porcentagem de sítios com placa estão associados a maior destruição periodontal, sendo fatores importantes no diagnóstico da doença periodontal nessa população brasileira.

Unitermos: Epidemiologia; Doença periodontal; Idade; Sexo; Fumo.

INTRODUCTION

Risk assessment has become increasingly important in the prevention of chronic diseases and has recently been applied to oral diseases. This observation has triggered interest in identifying susceptible individuals as well as the factors that put them at higher risk to develop these diseases[15]. This identification is one of the challenges facing Periodontology today.

Changes in our knowledge on the etiology of periodontal disease, and the recognition of the potential importance of susceptibility factors as they affect initiation and progression of periodontal disease, have led to intense study of specific risk factors for periodontal disease. Epidemiological investigations have played an essential role in helping to elucidate these risk factors for disease and determine the treatment needs of populations.

It was previously believed that the population was universally susceptible to periodontal disease[29]. This view has changed since authors reported that 5% to 20% of the population suffer from severe forms of destructive periodontitis[3]. This observation led to the proposal that there are susceptibility factors or risk factors that modulate susceptibility or resistance to destructive periodontitis[13].

Cross-sectional studies allow the identification of risk factors by determining associations between attributes and disease outcome with no inference on causality. A risk factor is an environmental exposure, aspect of behavior, or an inherent characteristic which is associated with disease[3,13]. The term "determinant" is often used synonymously with risk factor in literature, but for clarity is best reserved for risk factors that cannot be modified, for example age and sex[3]. The term "risk indicator" can be used to describe a risk factor associated with the disease, which is identified from case-control or cross-sectional studies[13].

In the past 2 decades increasing attention has been focused on identifying these risk factors for the initiation and progression of adult periodontitis. In Brazil, there are many epidemiological evaluations of periodontal disease in the population but rare studies about the influence of some risk factors in periodontal conditions. Some risk factors found to be associated with higher prevalence of periodontal disease include greater age[1,15,16,23,25], male sex[7,17], bacterial plaque[1,25] and smoking[6,19,24,25] in others populations. Therefore, the aim of the present study were to evaluate the association of age, sex, bacterial plaque and smoking with periodontal disease in a sample from Bauru, a medium size city in São Paulo (Brazil).

MATERIAL AND METHODS

This cross-sectional study was performed in a randomly sample of 380 subjects with age above 20 years old that were searching for dental treatment at the University of São Paulo. A questionnaire about general health and dental care habits was used in combination with the examination. Data recorded during questionnaire included age, sex and self-reported smoking habits (current smoker or non-smoker). A total of 380 dentate individuals were divided into 4 age groups from 20 to above 50 years.

The examinations were carried out in the dental clinics of the University of São Paulo in Bauru (Brazil) using mouth mirrors and a UNC 15 periodontal probe (*Hu-Friedy*) with 1mm graduations and a diameter of 0.4mm. Only one experienced examiner measured the same clinical parameters throughout the study using the same instruments. The measurements recorded and the diagnostic criteria used were the following:

Visible Plaque Index was recorded according to Ainamo, Bay[2] (1975). The occurrence of clearly visible plaque was considered to be a positive indication scored with number 1 and no plaque visible was scored with 0;

Periodontal pocket depth (PPD) was calculated as the distance in mm from the gingival margin to the base of the crevice/periodontal pocket;

Clinical attachment level (CAL) was measured as the distance in mm from a fixed, reproducible point (cement-enamel junction) to the bottom of the crevice/periodontal pocket.

The sequence of scoring was the following: plaque index, pocket depths and attachment levels. All clinical measurements were made on 4 sites per tooth (mesial, buccal, distal and lingual surfaces) on all existing teeth, including third molar. The subjects must had at least 20 teeth. The probe was inserted into the pocket as near as possible to the long axis of the tooth and the more severe of theses measurements was recorded.

The data obtained were evaluated using descriptive statistical with means and percentages. Means values were calculated for PPD and CAL and percentages for plaque index. An analysis of variance test (ANOVA) was selected to determine whether some type of relationship existed among the groups under study and subsequently, to identify statistically significant differences between them. When statistical differences were detected in at least one group, Tukey's test (significance level = 5%) was used to identify these groups. Pearson's correlation coefficient ($p<0,001$) was used to evaluate the correlation between periodontal parameters and plaque index.

This study was approved by Ethics Committee in Research from University of São Paulo in February (2001).

RESULTS

This paper presents the periodontal variables studied by age, plaque, sex and smoking habits.

Age: The distribution of the patients was 100 subjects in each age cohorts (20-29 years, 30-39 years and 40-49 years). The last group (above 50 years) had fewer participants (80 subjects) because of the difficulty to find patients with at least 20 teeth.

When mean PPD and CAL were evaluated by age cohort we found that mean PPD increased from 20-29 years to 40-49 years and maintained thereafter (Table 1). Statistically

significant differences were evident between some age cohorts. Mean CAL increased with age and there was a statistically difference between age cohorts (Table 1). It can be noted a higher tendency of increasing in CAL means compared to PPD means, which denotes a compromising by gingival recession with increasing age.

Another evaluation of the relation between age and periodontal disease included the number and percentage of sites with CAL ≤ 3mm, 4mm, 5mm, 6mm, 7mm and ≥ 8mm (Table 2). There was a higher percentage of sites with minor measurements and lower percentage of sites with major measurements in all age cohorts. About 75% of the sites evaluated showed CAL ≤ 3mm, 20% obtained measurements of 4-6mm and only 4.22% of the sites measured more than 6mm. There was an increase in the percentage of sites with higher measurements (CAL ≥ 4mm) and a decrease in the percentage of sites with lower measurements (CAL ≤ 3mm) with increasing age (Table 2).

Plaque : The prevalence of bacterial plaque in this sample was 100%, this implies that all subjects had at least one site with plaque. The percentages of sites with plaque for each age cohort were 84.1, 84.7, 86, 86.2. The % of plaque index (PI) did not vary significantly between age cohorts, there was only a small increase in the % of sites with plaque with increasing age.

The correlations between PPD and PI, CAL and PI were given by Pearson's correlation coefficient. The correlations among these periodontal parameters were positive. Higher means of PPD and CAL were weakly associated to higher percentages of sites with plaque (Table 3).

Sex: Our sample included 61.5% female and 38.4% male participants distributed among four age groups. In the three first age cohorts women predominated and the group above 50 years had a higher percentage of men.

The differences in the measurements of PPD and CAL according to gender are shown in Table 4. PPD and CAL means were higher in male participants in all age cohorts. However, there were statistical differences only for CAL measurements among male and female groups.

When considering the PI, higher percentages of sites with plaque was observed among men than among women in all age groups. The difference among them was statistically significant and are represented in Table 4.

Smoking habits: The sample was divided according to smoking habits in smokers (S) and non-smokers (NS). 15-21% of the individuals in all age groups were smokers.

The differences of PPD and CAL means between smokers and non-smokers are shown in Table 5. It was observed that PPD and CAL means were higher for the smokers group. These differences among smokers and non-smokers were statistically significant.

TABLE 1- Mean (SD) PPD and CAL by age cohort. Statistically significant differences between age cohorts are shown with different letters

Age range (years)	PPD (mm)	SD	CAL (mm)	SD
20-29	2.61[a]	0.57	2.69[a]	0.59
30-39	2.75[ab]	0.60	3.02[b]	0.73
40-49	2.89[b]	0.70	3.41[c]	0.98
50+	2.89[b]	0.74	3.69[c]	1.04
total	2.78	0.66	3.18	0.92

SD = standard deviation

ANOVA and Tukey´s test (p<0,05)

DISCUSSION

A risk factor for periodontal disease is a factor which predisposes individuals to the development of severe periodontal destruction. In this paper, the influence of age, sex, plaque and smoking habit in periodontal conditions were evaluated in a cross-sectional sample of 380 individuals from Bauru (Brazil).

Significant association between age and periodontal conditions was found, since the means of pocket depths

TABLE 2- Number and % of sites at different CAL by age cohort

	Age range (years)			
CAL	20-29	30-39	40-49	+50
≤ 3mm	9282 (86.6%)	7895 (78.3%)	6637 (69.2%)	4466 (63.2%)
4mm	836 (7.8%)	1043 (10.3%)	1233 (12.8%)	992 (14%)
5mm	312 (2.9%)	516 (5.1%)	732 (7.6%)	684 (9.7%)
6mm	150 (1.4%)	310 (3.1%)	413 (4.3%)	380 (5.4%)
7mm	58 (0.5%)	124 (1.2%)	260 (2.7%)	226 (3.2%)
≥ 8mm	85 (0.8%)	194 (1.9%)	318 (3.3%)	317 (4.5%)
total	10723	10082	9593	7065

ANOVA

and attachment levels increased with increasing age. This relation was confirmed with the analysis of the percentages of sites with established CAL measurements. The percentage of sites with greater attachment loss levels increased with increasing age. These findings are not unexpected due to the cumulative effect of the disease. Results of periodontal disease prevalence, or extent and severity from epidemiologic studies show more periodontal disease in older age groups as compared to younger groups[1,15,16,23,25].

Periodontitis is an inflammation of the gingival tissues together with loss of both the attachment of the periodontal ligament and bony support. The standard clinical measures for periodontitis are clinical attachment level and probing pocket depth. In this sample, periodontal disease status was determined by measurement of both pocket probing depths and clinical attachment levels. Mean pocket probing depths increased from the youngest age cohort up to 40-49 years, but the mean was maintained in older age cohort. Dowsett, et al.[11] (2001) reported decrease of this mean in elderly age cohorts. Clinical attachment levels were measured in order to provide a more accurate assessment of past disease activity[11]. As expected, mean CAL increased with age and, in accord with previous findings[8,11,26], recession contributed increasingly to attachment loss in the older age cohorts. PPD is related to age, though less directly than CAL[3]. This is in support of the findings of Carlos, et al.[8]

TABLE 3- Correlation between PPD and PI and between CAL and PI by sextant

Sextants		PPD X PI		CAL X PI	
		r	p	r	p
M	01	0.24	<0.001	0.22	<0.001
A	02	0.35	<0.001	0.33	<0.001
X	03	0.32	<0.001	0.31	<0.001
M	04	0.28	<0.001	0.32	<0.001
A	05	0.40	<0.001	0.39	<0.001
N	06	0.23	<0.001	0.26	<0.001
D					

r= Pearson´s correlation coefficient

p= error probability (p<0,001)

TABLE 4- Means (SD) PPD, CAL and % (SD) of sites with plaque by age cohort according to gender (male-M and female-F). Statistically significant differences between sexes are shown with different letters

Age range (years)	PPD (mm)				CAL (mm)				Plaque index (%)			
	M	SD	F	SD	M	SD	F	SD	M	SD	F	SD
20-29	2.72[a]	0.62	2.56[a]	0.54	2.82[a]	0.66	2.62[b]	0.55	90.4%[a]	15.8	80.9%[b]	17.0
30-39	2.76[a]	0.46	2.74[a]	0.66	3.05[a]	0.59	3.00[b]	0.79	87.6%[a]	11.9	83.2%[b]	15.4
40-49	2.91[a]	0.65	2.88[a]	0.73	3.65[a]	1.12	3.28[b]	0.88	90.1%[a]	15.1	83.8%[b]	14.2
50+	2.91[a]	0.71	2.87[a]	0.79	3.85[a]	1.02	3.50[b]	1.04	87.0%[a]	14.5	85.3%[b]	14.5

SD = standard deviation

ANOVA and Tukey´s test (p<0,05)

TABLE 5- Mean (SD) PPD and CAL in smokers (S) and non-smokers (NS) by age cohort. Statistically significant differences between smokers and non-smokers are shown with different letters

Age range (years)	PPD (mm)				CAL (mm)			
	NS	SD	S	SD	NS	SD	S	SD
20-29	2.56[a]	0,54	2.80[b]	0,63	2.63[a]	0,57	2.91[b]	0.65
30-39	2.64[a]	0,51	3.09[b]	0,74	2.87[a]	0,58	3.52[b]	0.95
40-49	2.85[a]	0,72	3.02[b]	0,62	3.29[a]	0,81	3.81[b]	1.33
50+	2.81[a]	0,69	3.37[b]	0,86	3.59[a]	1,02	4.25[b]	0.96

SD = Standard deviation

ANOVA and Tukey´s test (p<0,05)

(1987) who concluded that pocket depth measures alone may underestimate the amount of past destructive periodontal disease.

Most studies suggest that periodontal disease is more severe in elderly people because of cumulative tissue destruction over a lifetime rather than an age-related, intrinsic deficiency or abnormality which affects periodontal susceptibility[13,30]. It is still unclear though whether aging per se is a risk factor for severe periodontal disease, or if its effect is due to the prolonged exposure of older subjects to true aetiologic factors.

Although the prevalence of periodontitis increases with age, Abdellatif, et al.[1] (1987) showed that the increase in prevalence was much more pronounced in the poor-oral-hygiene stratum then in the good one when the data were stratified by oral hygiene status. So the effect of age on the progression of periodontitis could be considered negligible when good oral hygiene is maintained. The authors concluded that periodontitis is mainly related to the oral hygiene status of subjects and that age could be considered as a correlate rather than a risk factor[1].

It may be that in extreme old age a general deterioration in immune function and tissue integrity may increase susceptibility to periodontal destruction but there is as yet nothing to suggest that this constitutes a problem of public health importance. Both increasing in proportion of older people and in retention of teeth increase the number of units at risk of periodontal breakdown and this certainly has public health implications[10].

We must consider that periodontal disease is not a static process; periods of progression are interspersed with periods of stability or repair. Various models have been put forward to describe disease progression; however, the regression component of the disease process has been largely overlooked. The study from Faddy, et al.[12] (2000) has shown that age have significant effects on the rate of disease regression. So, it is still unresolved whether the physiological changes of the aging process itself promote disease progression or whether the relationship between age and periodontal disease is merely the manifestation of past disease. It can be concluded that this relationship reflects both cumulative effect of the non-reversible component of tissue destruction and the effect of a reduced rate of repair[12].

Another uncertainty is the influence of sex in periodontal conditions. Clinical attachment loss of all levels of severity is generally more prevalent in males than in females[3]. In this sample, it was observed difference statistically significant between males and females participants related to clinical attachment level. This is in accord to studies[7,17] which found that periodontal disease affects males more severely than females at comparable ages without any clear justification for this difference.

The reasons for these sex differences have not been explored in detail, but are thought to be related to poorer oral hygiene and dental-visit behavior among males than to any genetic factor. It has been shown that males usually exhibit evidence of poorer oral hygiene than females[1]. However, when correcting for oral hygiene, socioeconomic status, and age, male sex is associated with more severe periodontal disease when either attachment loss or bone height is used as the dependent variable[15,16]. Some future studies are necessary to help understanding the small but definite increase in periodontal disease seen in males. These studies may reveal important destructive or protective mechanisms related to male or female sex[13].

Another aspect evaluated in this sample was the influence of plaque in periodontal conditions. Although the relationship of oral hygiene to periodontitis is not as straightforward as that seen with gingivitis, plaque is considered the primary aetiologic agent in the initiation of periodontal disease. Other local or systemic factors may affect the host response and increase plaque accumulation or modify the plaque, causing it to be more pathogenic.

In the present study, the vast majority of subjects claimed to brush their teeth but oral hygiene was almost universally poor, with widespread plaque evident. The conclusion from most cross-sectional studies in populations with poor oral hygiene is that plaque correlates poorly with severe periodontitis[4,5,26]. In this sample, the % of sites with plaque were weakly associated with periodontal destruction. Results from well-controlled studies also showed that the quantity of plaque accumulation was only weakly correlated with periodontitis[21]. On the other hand, in a study from Norderyd, et al. [25] (1998), they found that higher mean levels of plaque was significantly correlated with severe periodontal disease. Abdellatif, et al.[1] (1987) showed a strong association between disease and oral hygiene (odds ratio=20.52) and a weak association between disease and age.

Griffiths, et al.[14] (1988) asserted that plaque index may be of value for a number of other reasons, such as assessment of patient cooperation and motivation, but it has limited value as predictor of susceptibility and have no value at all as marker of disease activity.

Epidemiological data show that while there is generally more CAL in third-world populations, the deficient oral hygiene and consequent gingivitis in such populations does not always progress to periodontitis[4,5]. In the majority of the population, the absence of oral hygiene and oral health care will lead to gingivitis. Depending on the interaction between the bacterial flora and the host, some gingival lesions progress to advanced lesions while others do not. While the evidence is that oral deposits are likely to be a major contributing cause of periodontitis, it is likely that their effect is mediated by host response to a greater or lesser extent[1]. These findings emphasize the role of the host response in the development of clinical periodontitis, so it is not surprising that the identification of an infection by itself does not predict periodontitis very well. Then, not all gingivitis proceeds to periodontitis but gingivitis always precedes periodontitis. The best predictor of future attachment loss still appears to be past disease added to age[18]. Prediction of future disease, either in the person or at a site, is still an inexact procedure. So the importance in controlling dental plaque leaves in this affirmation. Until we have better predictors of progression from gingivitis to

periodontitis, it would be irresponsible to modify significantly the basic approach (plaque control) to management of periodontal disease.

A substantial number of studies[6,19,24,25] has established the association of smoking with impaired periodontal conditions. Furthermore, in the current study, a correlation between periodontal disease experience and smoking habit was reported. Smokers had more periodontal destruction (higher means of PPD and CAL) than non-smokers in all age groups.

We must also bear in mind that within the non-smokers group, there were former smokers who, at the time of examination, did not smoke. As has been seen in another study[17], the former smokers presents greater severity than non-smokers, but less than current smokers, which could have raised the mean of PPD and CAL severity for non-smokers.

Some decades ago, authors had the perception that greater levels of plaque and calculus in smokers than non-smokers fully accounted for the association between smoking and periodontal disease. In 1983, Ismail, et al.[20] found that after adjusting for potential confounding variables such as age, oral hygiene, gender and socioeconomic status, smoking remained a major risk indicator for periodontal diseases. Grossi and coworkers[15,16] found a direct and linear dose response between the level of smoking and destructive periodontitis, lending further support to smoking as a risk factor for periodontal disease.

Tobacco has a direct periodontal effect, and not because of poor hygiene. What we do not know yet is the specific mechanisms by which it acts. Both bacterial flora and host take part in the pathogenesis of periodontal disease and since no differences have been found in the periodontopathogenic bacterial population between smokers and non-smokers[27], it would appear that tobacco acts on the host through 2 main mechanisms: on the one hand systemically causing alterations of the immune response[22]. On the other hand, it acts locally through cytotoxic metabolytes and vasoactives liberated by the combustion of the latter affecting fibroblasts[28], and the vascular response[9]. Nicotine can impair the attachment of fibroblasts to root surfaces and may affect collagen synthesis and protein secretion[28], thus interfering with the host's natural repair mechanisms.

It is likely that smoking is a major risk factor for destructive periodontal disease and that modification of this risk factor is important in the treatment and prevention of periodontal disease.

In conclusion, the present paper reported that smoking, greater age, male gender and higher levels of plaque are possible risk factors for periodontal disease in this specific population. These factors must be considered in the diagnostic of the periodontal disease. A new generation of studies is needed not only to identify other potential risk factors for periodontal diseases, but also to determine the effective interventions directed to modulating important risk factors and to assess their effects on the initiation and progression of periodontal disease, and their effects on periodontal therapy.

Influence of age, sex, plaque and smoking on periodontal conditions in a population from Bauru, Brazil.

Influência da idade, sexo, placa bacteriana e fumo nas condições periodontais em uma população de Bauru, Brasil.

REFERENCES

1- Abdellatif HM, Burt BA. An epidemiological investigation into the relative importance of age and oral hygiene status as determinants of periodontitis. J Dent Res 1987 Jan; 66(1):13-8.

2- Ainamo J, Bay I. Problems and proposals for recording gingivitis and plaque. Int Dent J 1975 Dec; 25(4):229-35.

3- American Academy of Periodontology. Committee on Research, Science and Therapy. Epidemiology of periodontal diseases. J Periodontol 1996 Sept; 67(9):935-45.

4- Baelum V, Fejerskov O, Karring T. Oral hygiene, gingivitis and periodontal breakdown in adult Tanzanians. J Periodont Res 1986 May; 21(3):221-32.

5- Baelum V, Fejerskov O, Manji F. Periodontal diseases in adult Kenyans. J Clin Periodontol 1988 Aug; 15(7):445-52.

6- Bergström, J. Cigarette smoking as risk factor in chronic periodontal disease. Community Dent oral Epidem 1989 Oct; 17(5):245-7.

7- Brown LF, Beck JD, Rozier RG. Incidence of attachment loss in community-dwelling older adults. J Periodontol 1994 Apr; 65(4):316-23.

8- Carlos JP, Brunelle JA, Wolfe MD. Attachment loss vs. Pocket depth as indicators of periodontal disease: A methodologic note. J Periodont Res 1987 Nov; 22(6):524-5.

9- Clarke NG, Shepherd BC, Hirsch RS. The effects of intraarterial epinephrine and nicotine on gingival circulation. Oral Surg 1981; 52:577-81.

10- Douglass C, Gillings D, Sollecito W, Gammon M. The potential increase in the periodontal diseases of the aged population. J Periodontol 1983 Dec; 72(12):721-30.

11- Dowsett AS, Archila L, Segreto VA, Eckert GJ, Kowolik MJ. Periodontal disease status of the indigenous population of Guatemala, Central America. J Clin Periodontol 2001 Jul; 28(7):663-71.

12- Faddy MJ, Cullinan, MP, Palmer JE, Westerman B, Seymour GJ. Ante-dependence modeling in a longitudinal study of periodontal disease: the effect of age, gender, and smoking status. J Periodontol 2000 Mar; 71(3):454-9.

13- Genco RJ. Current view of risk factors for periodontal diseases. J Periodontol 1996 Sept; 67(9):1041-9.

14- Griffiths GS, Wilton JMA, Curtis MA, et al. Detection of high-risk groups and individuals for periodontal diseases: Clinical assessment of the periodontium. J Clin Periodontol 1988 Aug; 15(7):403-10.

15- Grossi SG, Genco EE, Machtei AW, et al. Assessment of risk for periodontal disease. I. Risk indicators for attachment loss. J Periodontol 1994 Mar;65(3):260-7.

16- Grossi SG, Genco RJ, Machtei EE et al. Assessment of risk for periodontal disease. II. Risk indicators for alveolar bone loss. J Periodontol 1995 Jan; 66(1):23-9.

17- Haber J, Wattles J, Crowley M, Mandell R, Joshipura K, Kent RL. Evidence for cigarette smoking as a major risk for periodontitis. J Periodontol 1993 Jan;64(1):16-23.

18- Haffajee AD, Socransky SS, Lindhe J, Kent RL, Okamoto H, Yoneyama T. Clinical risk indicators for periodontal attachment loss. J Clin Periodontol 1991 Feb; 18(2):117-25.

19- Haffajee AD, Socransky SS. Relationship of cigarette smoking to attachment level profiles. J Clin Periodontol 2001 Apr; 28(4):283-95.

20- Ismail AI, Burt BA, Eklund SA. Epidemiologic patterns of smoking and periodontal disease in the United States. J Amer dent Ass 1983 May; 106(5):617-21.

21- Lindhe J, Okamoto H, Yoneyama T, et al. Longitudinal changes in periodontal disease in untreated subjects. J Clin Periodontol 1989 Aug; 16(8):662-70.

22- MacFarlane GD, Herzberg MC, Wolff LF, Hardie NA. Refractory periodontitis associated with abnormal polimorphonuclear leucocyte phagocitytosis and cigarette smoking. J Periodontol 1992 Dec; 63(12):908-13.

23- Marshall-day CD, Stephens RG, Quigley LFJ. Periodontal disease: prevalence and incidence. J Periodontol 1955 July; 26(3):185-203.

24- Martinez-Canut P, Lorca A, Magán R. Smoking and periodontal disease severity. J Clin Periodontol 1995 Oct; 22(10):743-9.

25- Norderyd O, Hugoson A. Risk of severe periodontal disease in a Swedish adult population. A cross-sectional study. J Clin Periodontol 1998 Dec; 25(12):1022-8.

26- Okamoto H, Yoneyama T, Lindhe J, Haffajee A, Socransky S. Methods of evaluating periodontal disease data in epidemiological research. J Clin Periodontol 1988 Aug; 15(7):430-9.

27- Preber H, Bergström J, Linder LE. Occurence of periopathogens in smoker and non-smoker patients. J Clin Periodontol 1992 Sept; 19(8):667-71.

28- Raulin LA, McPherson JC, McQuade MJ, Hanson BS. The effect of nicotine in the attachment of human fibroblasts to glass and human root surfaces in vitro. J Periodontol 1988 May; 59 (5):318-24.

29- Scherp HW. Current concepts in periodontal disease research: Epidemiological contributions. J Amer dent Ass 1964; 68:667-75.

30- Van der Velden U. Effect of age on the periodontium, J Clin Periodontol 1984 May; 11(5):281-94.