

## Cigarette Smoking As a Risk Factor For Periodontal Disease Severity

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**Abstracts:** Objective: To evaluate the influence of cigarette smoking on severity of periodontal disease and to quantify the strength of this influence in relation to frequency and duration of smoking. Methods: Total of 223 male subjects aged between 20-55 years and suffering from mild to severe chronic periodontitis was assessed for their periodontal status. Clinical parameters recorded were PI, GI, BOP, GR, PPD, and CAL on four sites on each tooth present excluding 3<sup>rd</sup> molars. Results: On the role played by cigarette smoking, the results confirmed that its consumption increases the severity of periodontal disease in terms of clinical attachment loss. When compared between smokers and non-smokers, the smokers had mean CAL 2.67 times more than non-smokers. As the frequency and duration of smoking increased, the severity of periodontal disease in terms of GR, PPD, and CAL increased steadily. Conclusion: As per the results of this study, frequency and duration of cigarette smoking are directly proportional to periodontal disease severity indicating it as a risk factor.

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**Key words:** Cigarette smoking, Clinical attachment loss, Chronic periodontitis, Periodontal disease severity, Tobacco smoke.

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**Introduction:** Forty years have passed since the landmark U.S. Surgeon General's report in 1964 warned that smoking played a causative role in lung cancer and was associated with cardiovascular diseases. It has been estimated that there are 1.1 billion smokers worldwide and 182 million (16.6%) of them live in India<sup>1</sup>. Tobacco is used in smoking and smokeless forms in India. Among tobacco users, bidi (34%), cigarette smokers (31%) and those using smokeless forms (35%)<sup>2</sup>. The prevalence of smoking among 13-15 year old school going students in India ranges from 19.7-34.5%, even the lowest was considerably higher than the global median of current cigarette smoking (13.9%)<sup>1</sup>.

In the past 20 years, there has been an increasing awareness of the role of tobacco use on the prevalence and severity of periodontal diseases. Recently, there has been a great deal of attention accorded to smoking as a risk factor in periodontitis. There have been a number of studies that strongly suggest that smoking is related to periodontal destruction. When reviewed carefully performed epidemiological and case control studies, it has been indicated that smoking is an important risk factor for the loss of attachment and supporting alveolar bone in adult periodontitis<sup>3</sup>. This study reports the influence of cigarette smoking on severity of periodontal disease

**Material and Methods :** A sample of 223 patients with mild to severe periodontitis (who had at least 4 sites with pocket depth and/or attachment loss of  $\geq 4$ mm) was selected for the study. The patient's age ranged from 20-55 yrs which included both smokers (who smoked for at least 2 years and currently smoking) and non-smokers (who never smoked). All the patients were selected during the dental camps organized by the Department of Community Dentistry and Department of Periodontics, and those subjects visiting the out patient Department of Periodontics, College of Dental Sciences, Davangere, Karnataka. Subjects were screened and upon signing an approved informed consent, entered into the study.

A cross sectional observation was done with all the patients who are selected. The demographic data referring to age, daily cigarette consumption, duration of the habit and socio-economic status were recorded in the proforma. All the subjects received a clinical periodontal examination by using PCP UNC-15 periodontal probe. The single examiner who was blinded recorded the following parameters at 4 sites per tooth (mesial, distal, mid-buccal, and mid-palatal or mid-lingual) on all the teeth present excluding 3<sup>rd</sup> molars. The plaque (PI), gingival inflammation (GI), bleeding on probing (BOP) and gingival recession (GR) scores were

recorded dichotomously. Probing pocket depth (PPD) and clinical attachment loss (CAL) were recorded as linear measurements.

#### Statistical analysis

One way ANOVA was used for multiple group comparisons followed by student's t-test (unpaired) for group wise comparisons. Where data may not have been normally distributed, non-parametric Mann-Whitney test was used as an alternative to unpaired t-test.

**Result:** Table 1 shows the distribution of the population studied in which 107 (48%) were non-smokers with mean age 34.7 yrs and 116 (52%) were smokers with mean age 39.0 yrs. The patients from different socio-economic status were fairly distributed between smokers and non-smokers group with statistically non-significant difference ( $P>0.05$ ). Those who smoked, consumed an average  $8.0 \pm 3.7$  (2-19 cig/day) with a median value of 7 cig/day and had smoked for an average of  $10.5 \pm 4.4$  years (3-29 years) with a median value of 10 years.

**TABLE 1: PATIENT DEMOGRAPHICS (MEAN  $\pm$  SD)**

	Non-Smokers	Smokers	't' Value	'P' value
No. of Pt.	107 (48%)	116 (52%)	-	-
Age	$34.7 \pm 9.6$	$39.0 \pm 8.5$	3.57	$<0.001$ (HS)
SES				
Low	6	8	7.42*	$>0.05$ (NS)
Lower middle	34	19		
Upper Middle	27	38		
High	40	51		
Frequency (Cig/Day)	-	$8.0 \pm 3.7$ (2-19 Cig/day) Median – 7.0	-	-
Duration (Years)	-	$10.5 \pm 4.4$ (3 – 23 years) Median – 10.0	-	-

The smokers had more plaque score ( $87.9 \pm 10.3$ ) than non-smokers ( $70.6 \pm 14.0$ ) with statistically highly significant difference ( $P<0.001$ ) (Graph 1).

The mean value of percentage of sites with gingival inflammation in smokers was  $63.7 \pm 12.4$ , whereas in non-smokers it was  $67.3 \pm 16.7$  and the difference was not statistically significant ( $P>0.05$ ). The mean percentage of sites which bleed on probing was higher in non-smokers  $62.2 \pm 18.1$  than in smokers  $52.4 \pm 12.8$  and the mean difference was statistically highly significant with  $P<0.001$ . The mean of gingival recession score was  $18.7 \pm 13.3$  in smoker's whereas in non-smokers it was  $6.7 \pm 6.27$  and the difference was shown to be statistically highly significant ( $P<0.001$ ).

The mean percentage of sites with probing pocket depth  $\geq 4$  mm was  $21.5 \pm 11.0$  in smokers and it was  $10.7 \pm 6.5$  in non-smokers with statistically highly significant difference ( $P<0.001$ ). The mean percentage of sites with clinical attachment level  $\geq 4$ mm was  $26.5 \pm 13.1$  in smokers, whereas in non-smokers it was  $9.9 \pm 5.4$ . The difference was shown to be statistically highly significant ( $P<0.001$ ).

With regard to age, the smokers had shown higher values than non-smokers indicating the significant effect of smoking on periodontal variables. Also the socio-economic status (SES) in which the mean values were shown to reduce as the SES improved.

The amount of cigarette smoked also significantly increased the values of periodontal variables showing statistically significant differences for GR, and CAL ( $P<0.001$ ) (Table 2). Although there was increase in PPD, the difference was statistically non-significant ( $P<0.032$ ). Whereas GI and BOP scores were shown to reduce with amount of smoking. When compared between non-smokers and those who smoked 5cig/day, the percentage increase in GR, PPD, and CAL were 158, 101, and 168 respectively. As the frequency of smoking increased to 10-20 cig/day, the GR, PPD, and CAL mean scores were increased by 367%, 131% and 263% respectively (Table 2).

On analyzing the periodontal losses by duration of smoking, statistically significant differences are observed for every 5 years of smoking (Table 3). When compared between non-smokers and those who smoked for up to 5yrs, the percentage increase in GR, PPD, and CAL were 107, 21, and 77 respectively. As the duration of smoking increased

**TABLE 2: COMPARISON OF CLINICAL DATA ACCORDING TO FREQUENCY OF CIGARETTE SMOKING.**

Smoking Frequency Cig/Day	No. (%)	Plaque Score		Gingival Inflammation Score		Bleeding on Probing Score		Gingival Recession Score		Probing Pocket Depth		Clinical Attachment Level		
		Mean	SD	Mean	SD	Mean	SD	Mean	SD	Mean	SD	Mean	SD	
A	Upto 5	44	86.8	12.1	64.2	11.8	50.3	12.6	17.3	8.9	21.5	11.5	22.3	9.6
B	5 – 10	53	88.5	8.7	65.6	10.8	55.8	12.8	15.3	8.4	20.2	10.1	26.6	11.6
C	10 – 20	19	88.6	10.6	61.2	14.2	47.9	11.2	31.3	23.2	24.7	12.2	35.9	18.5
ANOVA			f = 0.41 P = 0.67 (NS)		f = 1.53 P = 0.22 (NS)		f = 3.86 P = 0.02 (S)		f = 12.6 P < 0.001 (HS)*		f = 1.15 P = 0.32 (NS)		f = 8.3 P < 0.001 (HS)	
d	Non-Smokers	107	70.6	14.0	67.3	16.7	62.2	18.1	6.7	7.27	10.7	6.5	9.9	5.4

**TABLE – 3: COMPARISON OF CLINICAL DATA ACCORDING TO DURATION OF CIGARETTE SMOKING.**

Duration of Smoking (Avg. No. of Years)	No. (%)	Plaque Score		Gingival Inflammation Score		Bleeding on Probing Score		Gingival Recession Score		Probing Pocket Depth		Clinical Attachment Level		
		Mean	SD	Mean	SD	Mean	SD	Mean	SD	Mean	SD	Mean	SD	
a	Upto 5	11	74.9	11.4	70.4	4.9	56.2	6.9	13.9	12.0	12.9	4.9	17.5	11.1
b	6 – 10	60	86.6	8.1	66.8	12.3	52.4	12.4	15.3	11.8	18.2	7.3	23.3	10.7
c	11 – 15	32	92.0	10.4	61.4	11.9	52.5	13.7	24.9	15.9	27.5	12.4	30.3	12.0
d	> 15	13	94.8	7.2	58.9	16.8	47.9	14.1	23.1	6.5	29.2	14.2	39.5	16.0
ANOVA			f = 12.8 P < 0.001 (HS)		f = 3.35 P = 0.02 (S)		f = 0.83 P = 0.48 (NS)		f = 5.05 P < 0.01 (S)*		f = 12.0 P < 0.001 (HS)		f = 100 P < 0.001 (HS)	
e	Non Smokers	107	70.6	14.0	67.3	16.7	62.2	18.1	6.7	6.27	10.7	6.5	9.9	5.4

to >15yrs, the GR, PPD, and CAL mean scores were increased by 245%, 173% and 299% respectively.

**Discussion:** The data of the present investigation indicates that the smokers had significantly more number of sites with gingival recession, probing pockets and clinical attachment loss  $\geq 4$  mm. This is in agreement with previous studies by Martinez-Canut 1995<sup>4</sup>; Haffajee AD et al 2001<sup>5</sup>; and Calsina G 2002<sup>6</sup>. When compared between the smokers and non-smokers, the smokers had the mean values 1.24 times more than non-smokers in relation to PI scores. This increased levels of plaque which have been observed in smokers have been tentatively attributed to personality traits leading to decreased oral hygiene habits in smokers.. Whereas the

gingival inflammation and bleeding on probing mean values were reduced by 1.05 and 1.18 times respectively. This reduced bleeding response could be due to the potential vasoconstrictive effect of nicotine as previously reported by Clarke 1981<sup>7</sup>. The mean values of GR, PPD and CAL were increased by 2.79, 2.0 and 2.67 times respectively, indicating the direct effect of cigarette smoking on periodontal tissues. In this study, periodontal status of cigarette smokers has been assessed with due consideration to age, socio-economic status as risk determinants. Age is considered as one of the risk determinants of periodontal disease i.e., the prevalence of periodontal disease varies with the advance of age. As the age increased, the severity of periodontal disease increased. The percentage of sites with

plaque, gingival inflammation, bleeding on probing and gingival recession increased with age. Again in each age group, the smokers had high percentage of sites with plaque, GR, PPD, CAL but low percentage of sites with GI and BOP. These findings were similar to the findings of early observational report made by Martinez-Canut et al 1995<sup>4</sup> & Haffajee et al 2001<sup>5</sup>.

The subjects selected were grouped into low, lower middle, upper middle and high socio-economic status according to their annual income as implemented by National Council of Applied Economic Research, New Delhi 1993-94<sup>8</sup>. Most of the subjects in our study belonged to middle and high socio-economic status. The low socio-economic group showed high mean values in all the clinical parameters and as the socio-economic status increased from low to high, the mean value of all the parameters were reduced significantly. This reflects that the population from low socio-economic group experienced more severe periodontal diseases than those of high socio-economic status. The occupation and the level of education were responsible for the decreased dental awareness and decreased frequency of dental visits when compared with more educated individuals of higher SES and also inability to have a consistent home care measures due to cost of the plaque control aids.

The increased frequency of cigarette smoking was shown to increase the mean values of plaque scores. But the difference was statistically non-significant. The gingival inflammation scores and bleeding on probing scores were shown to be decreased with increase in frequency of smoking because of the local vasoconstrictor effect of nicotine<sup>7</sup>. Periodontal disease variables like gingival recession (GR), probing pocket depth (PPD) and clinical attachment level (CAL) were increased with increase in frequency of smoking and this effect was clinically being evident even at less than 5 cigarettes smoked per day. This was in contrast to previous studies done by Martinez-Canut P. et al 1995<sup>4</sup>. In which clinically evident changes in periodontal disease were noticed only from 10cig/day onwards.

The duration of smoking has also shown to have a significant effect on the periodontal disease

severity. The statistical analysis showed that, there was a significant increase in GR, PPD, CAL for very 5 years of smoking. For each year of smoking, the GR, PPD, CAL score mean values were increased by 12.2%, 8.6% and 15% respectively. These findings were similar to previous studies done by Martinez-Canut P. et al 1995<sup>4</sup>.

**Conclusion:** Within the limits of our study, it can be concluded that cigarette smoking increases the severity of periodontal disease. This effect clinically being evident even less than 5cigarettes smoked per day for less than 5years. Therefore these results confirm that cigarette smoking is an important risk factor for periodontal disease severity. Although smoking is considered as one of the risk factors for periodontal disease, the exact mechanism of smoking leading to such increased severity remains to be unclear. Therefore future studies which consider the plasma, saliva and/or GCF nicotine or cotinine levels along with clinical parameters and host immunity levels should be carried out to rule out the possible mechanism of causation.

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