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The Clinical Course of Chronic Periodontitis

VI. The influence of lifelong smoking (20 years) on loss of attachment and teeth

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Abstract

Aim: to examine the long-term influence of smoking on periodontal health.

Material & Methods: The data derived from a 20-year longitudinal study of a group of Norwegian middle class males. The subjects were subset according to their smoking history. 119 non-smokers and 17 smokers were examined 20 years apart.

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Conclusions: Lifelong light smoking could be confirmed as a risk factor of periodontal disease progression. However, in this population, smoking did not significantly increase the risk for tooth loss.

Introduction

The significance of tobacco smoking and the occurrence of periodontal disease have been the subject of a long-standing debate. One of the first reports on the subject revealed that in Danish navy personnel there was a significant correlation between the occurrence of ulcerative gingivitis and tobacco consumption (Pindborg 1946, 1947). Subsequently, data from Sweden (Herluf 1950) showed that the frequency of periodontal pathology was greater in smoking than non-smoking dental students. Moreover, Norwegian investigations (Schei et al. 1959) supported the view of a positive correlation between loss of alveolar bone and tobacco consumption.

In a number of more recent studies the relationship between smoking and periodontal health or disease was, again, explored and seemed to confirm that smoking may be a putative risk factor for loss of periodontal attachment and alveolar bone (Ismail et al. 1983, Bergström et al. 1991, Horning et al. 1992, Haber et al. 1993, Bolin et al. 1993, Grossi et al. 1994). Furthermore, the severity of periodontal lesions was directly related to the quantity of cigarette consumption (Goultschin et al. 1990, Haber et al. 1993, Grossi et al. 1994, 1995, Norderyd & Hugoson 1998). Also, the prevalence of smoking seemed to be very high in patients with recurrent (refractory) periodontitis (MacFarlane et al. 1992), and it is maintained that the response to periodontal therapy was found to be less favorable in cigarette smokers than in non-smokers (Preber & Bergström 1986, 1990, Ah et al. 1994, Grossi et al. 1997, Kaldahl et al. 1996). Regenerative therapy in smokers resulted in treatment outcomes that were clearly less favorable than in non-smokers (Tonetti et al. 1995).

It has been found that smoking had an adverse effect on the immune and inflammatory systems (*for review see* Barbour et al. 1997). Decreased polymorphonuclear leucocyte (PMN) migration into the oral cavity of smokers was suggested already in 1969 (Eichel & Shahrik 1969). Subsequently, PMNs harvested from the gingival sulcus of smokers were shown to have reduced phagocytic capacity compared to PMNs from non-smokers (Kenny et al. 1977). Smokers yielded increased numbers of leukocytes in circulation, while fewer PMNs migrated into the gingival crevice/pocket. *In vitro* studies tended to show a direct inhibition of neutrophil and monocyte-macrophage defensive functions at

high concentrations of nicotine (Pabst et al. 1995). Abnormal PMN phagocytosis was associated with a high level of cigarette smoking (MacFarlane et al. 1992). Using a Markov chain statistical model for the analysis of longitudinal data, it has also been suggested that smoking inhibited the healing process rather than promoting the disease progression of periodontitis (Faddy et al. 2000).

Most studies examining the effects of smoking on the periodontium have used a cross-sectional design. While cross-sectional studies may be valuable in identifying possible risk indicators associated with disease, prospective studies with longitudinal assessments are necessary to provide evidence that a condition or feature may represent a true risk factor (Genco 1996). To date, few such studies are available to explore the long-term influence of smoking on periodontal health or disease progression.

In a 10 year follow-up study (Bergström et al. 2000), the influence of smoking on periodontal health was evaluated prospectively. Based on evidence of an increase in periodontal probing depths concomitant with periodontal bone loss in chronic smokers, it was suggested that smoking compromised periodontal health. In non-smokers, on the other hand, the periodontal conditions remained relatively unchanged over the 10-year follow-up period. Similar results were found in another longitudinal study mentioned above (Faddy et al. 2000).

In contrast, no significant relationship between smoking and loss of attachment was found in a 20-year longitudinal study of a Sri Lankan tea labourer population who never had practiced oral hygiene (Neely et al. 2001). Given the high level of periodontal disease progression in the absence of oral hygiene practices and no periodontal treatment rendered, the effect of local irritants may have overshadowed the effect of smoking on disease progression in that study.

The purpose of the present study was to explore the pattern of periodontal attachment loss in smokers or non-smokers who, during their adult life, had regular dental care and practiced good to excellent oral hygiene.

Material & Methods

The present report represents an analysis of data obtained from a longitudinal study of the initiation and progression of chronic periodontitis and tooth loss over major portions of adult life in middle class Norwegian man.

Source of Data

The study population has been described earlier (Løe et al. 1978a-c, Løe et al. 1986, Ånerud et al. 1991, Schätzle et al. 2004). The Norwegian group was established in Oslo in 1969 and consisted of 565 men between 17 and 30+ years of age and who had a minimum of 13 years of education. The group was randomly selected by the Norwegian Bureau of Statistics. They were all born and raised in the City of Oslo and had received regular systematic dental care since childhood. This population practiced oral home care on a daily basis and reported seeing their dentist on an annual basis.

With respect to smoking, the group was examined in 1969 and twenty years later in 1988, although the subjects had been examined longitudinally every 2 – 7 years. Thus only subjects present at baseline and in 1988 were considered in order to avoid overlapping of the various cohorts. At 1969, the participants were divided into 3 age groups (<20, 20-24 and 25-30 years of age) which resulted in groups after 20 years (35-39, 40-44 and 45-50 years of age) (Table 1). As a result of this division, 119 non-smokers and 17 lifelong smokers were compared after an observation period of 20 years.

Clinical Parameters

The examinations were performed in well-equipped clinical facilities at the Faculty of Odontology, University of Oslo and included assessments of the periodontal tissues and adjacent portions of the oral cavity and the dentition. At each appointment the participants answered questions regarding their personal dental care and smoking habits. Throughout the study, the same indices were scored by the same two investigators who were both experienced periodontists and well-trained and calibrated examiners (H.B., Å.Å.).

The following parameters were assessed (Løe et al. 1978a):

- Gingival Index (GI) (Löe & Silness 1963)
- Loss of attachment (LA) (Glavind & Löe 1967)
- Plaque Index (PII) (Silness & Löe 1964)
- Calculus Index (CII) (Löe 1967)

In the survey in 1981 and in all subsequent examinations, the distal and lingual surfaces were also included in the examinations, while before that survey, the examinations were restricted to buccal and mesial sites.

Tooth loss was identified when a tooth was present in 1969 and missing at any of the subsequent examinations. Information on the exact date or the reason for tooth loss was not collected. Third molars were not included in the evaluation at any time.

Subjects were stratified according to their smoking history into self-reported *Smokers* and *Non-smokers*.

The non-smoking cohort was made up of individuals who, at each examination, reported that they had never smoked. The smoking group consisted of all subjects, who, at every survey in which they participated, reported smoking of 2 or more cigarettes per day (Table 1).

Irregular smokers, who reported smoking at some examinations, but not at others, were eliminated from the analyses.

Data Analyses

As in most longitudinal studies of this size and length, a number of the patients dropped out and could not be followed up. Other subjects missed one or more examinations, but showed up at a later survey. Of the 565 subjects who started in the investigation in 1969, 136 showed up for the last examination, 20 years later and were the basis for evaluation in the present analysis (Table 1).

The Statistical Analysis System Package (SAS Institute Inc., Cary, N.C., USA) was used to calculate frequencies, mean values and the regression models. For the comparison of

the significant differences in the mean values between the non-smoking and smoking cohorts as stratified groups (mean values) and at different age levels, the Wilcoxon rank-sum test was used.

For the calculations of the Odds ratio, the logistic regression models (PROC LOGISTIC, SAS) were used to model the binary tooth loss variable as a function of smoking- habits and age. The level of significance was set at $\alpha = 0.05$.

Results

The baseline population consisted 565 men among those 94 corresponding to 16.6% were self declared smokers. Twenty years later, 203 subjects were re-examined in 1988. From those, 17 were self declared smokers representing a percentage of 8.4%. One hundred and nineteen could be identified as never smokers, representing 58.6%. The remainder 67 (33.0%) belonged to a group of intermittent smokers, that was excluded from the analysis. Hence, the same never smokers and smokers at survey 6 were used to compare their baseline data 20 years prior.

Clinical parameters

Plaque Index (PII):

The mean Plaque Index and the frequency distributions of the Plaque Index scores for both non-smokers and smokers are illustrated in Table 2, Figure 1. Before the age of 30 years, the mean PII scores for non-smokers and smokers were comparable (PII= 1.15 - 1.18 and 1.17-1.37, respectively). After 35 years of age and as the cohort approached 50 years, the smokers showed statistically significantly higher mean PII values than did non-smokers (PII= 1.46 *versus* 1.75).

Also after 35 years of age, there were fewer PII = 2 or 3 scores at sites of non-smokers (47-55%), as compared with smokers (56% - 77%) at different age levels.

Gingival Index (GI):

Before 30 years of age, the mean Gingival Index Scores (Table 3, Figure 2) were comparable or slightly higher in non-smokers. In cohorts older than 35 years of age, however, the smokers had significantly greater mean GI scores (GI = 0.90-1.23) than did the non-smokers (GI= 0.81-0.85).

The frequency distribution of various Gingival Index scores (Table 3) also indicated that there were fewer GI = 2 or 3 scores in the smokers (4-7%) than in non-smokers (8-14%) before 30 years of age. Approaching 50 years of age, non-smokers yielded significantly lower proportions of GI=2 or 3 scores (16%) when compared to the smokers (35%).

Calculus Index (CI):

Table 4 and Figure 3 show the frequency distribution of the different Calculus Index (CI) scores for both non-smokers and smokers over the various age ranges. Before the age of 30 years, non-smokers and smokers showed similar mean calculus scores. More than 80% of all the surfaces were calculus free. After the age of 35 and as the subjects approached 50 years of age, the prevalence of subgingival calculus (CI =2,3) increased substantially to 13 % to 33 % in smokers, whereas in the non-smokers, subgingival calculus showed very little increase over the age span (4 % - 8 %).

Loss of attachment:

The cumulative loss of attachment during the 20 years of observation is shown in Table 5, Figure 4. At baseline, the mean loss of attachment for non-smokers and smokers was 0.14 mm. In non-smokers, 87-97% of the sites did not yield a loss of attachment of more than 1 mm, while in smokers, the respective proportion was 87-96% (before the age of 30 years). Approaching 50 years of age, the cumulative mean loss of attachment was 1.57 mm in non-smokers and 2.31 mm in smokers, respectively. Moreover, there were more lesions with LOA \geq 4mm in smokers (7.5% versus 17.7%)(Table 5).

Figure 5 provides a scatter plot of the mean attachment loss and age in smokers and non-smokers. Age was significantly related to attachment level in both groups ($p < 0.001$). The slope of the regression line for the smokers was approximately 1.5- times steeper than that of the non-smokers, indicating that smoking had an increasing effect with increasing age. The slopes were significantly different from each other ($p < 0.05$).

Tooth Loss

The smokers did not lose significantly more teeth over the 20- year observation period than did the non-smokers (Table 6). In smokers, the initially 462 teeth present in 1969, 461 were retained after 20 years; i.e. 2 teeth or 0.43% were lost during the observation period. Non-smokers had lost 15 (0.46 %) of 3246 teeth present at baseline. The odds ratio for smokers to lose a tooth was 1.07 (C.L.: 0.24 - 4.67) compared to non-smokers, and was not statistically significant.

Discussion

The purpose of the present study was to evaluate the clinical parameters of health and disease in adult male lifelong smokers and non-smokers of a middle class population with good to excellent oral hygiene practices. On the basis of two clinical examinations 20 years apart, the data describe the accumulation of plaque and calculus and the attachment loss as well as tooth mortality before 50 years of age based on the participants' self-reported smoking history. While the baseline cohort presented with 16.6% smokers the re-evaluation 20 years later revealed only a proportion of 8.4% self declared lifelong smokers. This, in turn, means that the numbers of drop outs was 2-fold increased in smokers than non-smokers. The reason for this remains unknown. However, it may be speculated that life-long smokers may lack the interest to be re-examined. In the present study lifelong smokers have been defined as subjects that reported to have smoked 2 cigarettes or more a day for 20 years. This represents a definition that generally would address light smokers. While heavy smokers smoking 1 pack of cigarettes or more have often been identified as at risk for periodontal disease progression (e.g., Preber & Bergström 1986, Bergström 1989, Bergström et al. 1991, Haber et al. 1993, Grossi et al. 1994, 1997), the influence of light smoking on the progression of periodontitis has generally not been addressed. The middle class Norwegian males represent a well educated group, motivated to perform oral hygiene and maintaining a healthy dentition. In that respect the study population may not represent an average population and generalizeability of the results has to be questioned. Nevertheless, the effect of light, but lifelong cigarette smoking on the progression of loss of periodontal attachment throughout life is indisputable and documents that even lower exposure to cigarette smoking when performed lifelong may be detrimental to periodontal health.

Oral Hygiene standards:

In the smokers and the non-smokers younger than 30 years of age, there was little or no difference in the standard of oral hygiene as revealed by plaque accumulation. For the period of 35-50 years, however, the smokers consistently exhibited significantly greater

Plaque Index scores than did the non-smokers. These findings tended to confirm the long-held view that smokers display worse standards of oral hygiene than do non-smokers. They also stand in contrast to suggestions that tobacco use could act as an anti-plaque agent (Danielsen et al. 1990). In accordance with numerous studies over the years, increasing Plaque Index scores were accompanied by increasing Gingival Index values for all age levels of smokers as well as non-smokers.

Before the age of 30 years, there was very little subgingival calculus in either cohort, while in the later decades of life, the smokers exhibited consistently more subgingival calculus than did the non-smokers.

While the mean Plaque Index in smokers was only about 1.2 times greater than that of the non-smokers, the mean Calculus Index for smokers was almost 3 times higher than that of the non-smokers as the cohorts approached 50 years. This combined impact of plaque and calculus accumulation corresponded especially to the increasing mean Gingival Index scores and significantly more sites with bleeding gingiva in smokers of 35 years and older. Thus, it may be suggested that in smokers, this combined increase in plaque and calculus formation may have had significant detrimental effects both on the gingival health and the subsequent progression of the periodontal lesions.

Periodontal Changes:

Before 30 years of age, loss of attachment occurred at equally low levels in smokers and non-smokers. But with increasing age and especially at 35- 40 years of age periodontal lesions progressed at a higher rate in smokers than in non-smokers (Fig. 5). This finding is in accordance with that of Gunsolley et al. (1998) and Haffajee & Socransky (2001) who found that smokers with minimal periodontal destruction, exhibited greater loss of attachment than subjects with minimal disease who did not smoke.

It is generally known that with increasing age, there is an increase in loss of attachment. The amount of attachment loss from the age of 16 to 50 years was 1.43 mm for the non-smokers (0.14– 1.57 mm) and 2.17 mm for smokers, indicating that both smoking and ageing were independently associated with increased attachment loss. The relationship between smoking and attachment loss was significant at $p < 0.05$ (SAS GLM) and the

slope of the scatter plot that related age to mean attachment level was 1.5 times greater for smokers than for non-smokers. In contrast to the data of attachment loss, smokers did not have a significantly increased risk for tooth loss. This, in turn, may be explained on the basis of the fact that, in this dentally-minded middle class population, tooth loss was a relatively rare event (Schätzle et al. 2004).

By and large, these results coincide with those of several cross-sectional (Haber et al. 1993) long-term follow-up (Burt et al. 1990, Ismail et al. 1990) and longitudinal (Neely et al. 2001, Van der Velden et al. 2006, Hugoson et al. 2008) studies that smokers might be at higher risk for periodontal disease than non-smokers. On the other hand, knowledge of the mechanisms of this disease progression is skewed: How much is due to local factors; and how much is due to the direct or systemic effect of smoking itself and the chemicals involved?

This and other clinical studies have been unable to adequately answer these questions. In this context, two features which according to the data of the present study suggest that (1) the difference between the periodontal changes in those who consistently smoked for 20 years compared to those who never smoked, was relatively small (gingivitis level, loss of attachment) or absent (tooth loss) and that (2) the biggest difference in the two cohorts occurred in the accumulation of supra- and subgingival calculus, and that the combined impact of plaque and calculus in smokers might explain the modest increase in periodontal pathology.

Thus, it is concluded that this longitudinal study of middle class Scandinavian men with regular dental check-ups and daily oral hygiene practices, has shown that smokers have lost slightly more periodontal attachment than non-smokers; and that the major factor in this attachment loss is due to the increased accumulation of plaque as well as supra- and subgingival calculus as compared with that in non-smokers.

Furthermore, this study failed to document that smoking had an influence on tooth loss before 50 years of age.

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Clinical relevance

Scientific rationale: Longitudinal studies on the history of periodontitis progression in smokers and non- smokers are scarce. This cohort of Norwegian middle class males was followed for 20 years and their smoking habits were well-defined. Consequently, the effects of smoking in an otherwise well-maintained oral cavity could be evaluated.

Principle findings: Smoking resulted in higher plaque and calculus formation, especially after the age of 35 years. This led to greater loss of attachment in higher age groups. However, tooth loss was not affected.

Practical implications: Lifelong light smoking is a risk factor for the progression of periodontitis.

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Tables Legend:

- Table 1:** Survey and number of patients examined at each survey from 1969 to 1988
- Table 2:** Mean values and frequency distribution of Plaque Index Scores for Non-Smoker and Smoker by Age Group
- Table 3:** Mean values and frequency distribution of Gingival Index Scores for Non-Smoker and Smoker by Age Group
- Table 4:** Mean values and frequency distribution of Calculus Index Scores for Non-Smoker and Smoker by Age Group
- Table 5:** Mean values and frequency distribution of Pocket Depth Scores for Non-Smoker and Smoker by Age Group
- Table 6:** Mean values and frequency distribution of Recession Scores for Non-Smoker and Smoker by Age Group
- Table 7:** Mean values and frequency distribution of Loss of Attachment Scores for Non-Smoker and Smoker by Age Group
- Table 8:** Frequency Distribution of teeth lost for Non-Smokers and Smokers

Table1:

Survey and number of patients examined at each survey during 20 years

Age Group	Baseline		Follow up	
	Non-Smokers	Smokers	Non-Smokers	Smokers
< 20	25	3		
20-24	59	8		
25-30	35	6		
35-39			42	6
40-44			51	6
45-50			26	5
Patients per Survey	119	17	119	17

Table 2:**Mean values and frequency Distribution of Plaque Index (PII) Scores**

Age Group	Non-Smokers				Smokers			
	PII = 0	PII = 1	PII = 2,3	Mean PII	PII = 0	PII = 1	PII = 2,3	Mean PII
< 20	16.33%	52.34%	31.33%	1.15	16.67%	49.4%	33.93%	1.17
20-24	16.41%	51.58%	32.01%	1.16	11.59%	49.32%	39.09%	1.28
25-30	17.31%	47.19%	35.50%	1.18	10.44%	41.77%	47.78%	1.37
35-39	11.27%	38.73%	50.00%	1.39	9.43%	34.13%	56.44%	1.47
40-44	12.07%	40.80%	47.13%	1.35	7.62%	32.16%	60.21%	1.53
45-50	8.42%	37.03%	54.55%	1.46	1.74%	21.90%	76.36%	1.75

* significant differences ($p < 0.05$)

Table 3:**Mean values and frequency Distribution of Gingival Index (GI) Scores**

Age Group	Non-Smokers				Smokers			
	GI = 0	GI = 1	GI = 2,3	Mean GI	GI = 0	GI = 1	GI = 2,3	Mean GI
< 20	37.41%	54.47%	8.13%	0.71	45.24%	50.60%	4.17%	0.59
20-24	16.19%	70.06%	13.75%	0.98	22.05%	74.32%	3.64%	0.82
25-30	16.22%	72.82%	10.97%	0.95	6.01%	86.71%	7.28%	1.02
35-39	31.95%	51.39%	16.65%	0.85	24.85%	60.03%	15.12%	0.90
40-44	35.91%	47.13%	16.96%	0.81	22.56%	54.27%	23.17%	1.02
45-50	31.58%	52.11%	16.32%	0.85	12.60%	52.71%	34.69%	1.23

* significant differences ($p < 0.05$)

Table 4:**Mean values and frequency Distribution of Calculus Index (Cal) Scores**

Age Group	Non-Smokers				Smokers			
	Cal = 0	Cal = 1	Cal = 2,3	Mean Cal	Cal = 0	Cal = 1	Cal = 2,3	Mean Cal
< 20	93.12%	6.44%	0.44%	0.07	89.29%	10.12%	0.60%	0.11
20-24	85.67%	11.05%	3.28%	0.18	84.09%	14.09%	1.82%	0.18
25-30	84.15%	11.23%	4.62%	0.20	80.38%	11.39%	8.23%	0.28
35-39	73.69%	18.04%	8.27%	0.34	66.92%	20.81%	12.28%	0.45
40-44	79.99%	15.23%	4.78%	0.25	57.47%	19.36%	23.17%	0.67
45-50	74.90%	20.60%	4.50%	0.29	47.87%	19.77%	32.36%	0.86

* significant differences ($p < 0.05$)

Table 5:

Mean values and frequency Distribution of Loss of Attachment (LoA) Scores

Age Group	Non-Smokers					Smokers				
	LoA = 0, 1	LoA = 2, 3	LoA= 4, 5	LoA ≥ 6	Mean LoA	LoA = 0, 1	LoA = 2, 3	LoA= 4, 5	LoA ≥ 6	Mean LoA
< 20	97.57%	2.36%	0.07%	0.00%	0.14	96.43%	3.57%	0.00%	0.00%	0.14
20-24	90.85%	8.69%	0.40%	0.06%	0.53	91.03%	8.51%	0.46%	0.00%	0.40
25-30	86.71%	11.30%	1.72%	0.26%	0.63	87.25%	11.44%	1.31%	0.00%	0.75
35-39	64.03%	32.57%	3.34%	0.06%	1.29	44.48%	40.64%	12.16%	2.72%	1.98
40-44	56.60%	37.53%	5.46%	0.41%	1.49	42.56%	43.37%	13.11%	0.98%	1.97
45-50	52.56%	39.95%	7.33%	0.16%	1.57	34.07%	48.23%	15.49%	2.21%	2.31

* significant differences ($p < 0.05$)

Table 6:

Frequency Distribution of teeth lost for Non-Smokers and Smokers

	Non-Smokers				Smokers				Total			
	Retained		Lost		Retained		Lost		Retained		Lost	
Incisors	947	99.73%	1	0.11%	136	100.00%	0	0.00%	1083	99.91%	1	0.09%
Canines	476	100.00%	0	0.00%	68	100.00%	0	0.00%	544	100.00%	0	0.00%
Premolar	893	99.33%	6	0.67%	126	99.21%	1	0.79%	1019	99.32%	7	0.68%
Molars	930	99.15%	8	0.85%	131	99.24%	1	0.76%	1061	99.16%	7	0.84%
Total	3246	99.54%	15	0.46%	461	99.57%	2	0.43%	3707	99.54	17	0.46%

At baseline: 84 missing teeth

* significant differences ($p < 0.05$)

Figures Legend:

- Figure 1:** Mean Plaque Index Scores for Non-Smoker and Smoker by Age Group
- Figure2:** Mean Gingival Index Scores for Non-Smoker and Smoker by Age
- Figure 3:** Mean Calculus Index Scores for Non-Smoker and Smoker by Age Group
- Figure 4:** Mean Loss of Attachment for Non-Smoker and Smoker by Age Group
- Figure 5:** Scatter plot of mean attachment level (y-axis) and age (x-axis) in smokers and non-smokers.

Figure 1: Mean Plaque Index Scores for Non-Smoker and Smoker by Age Group

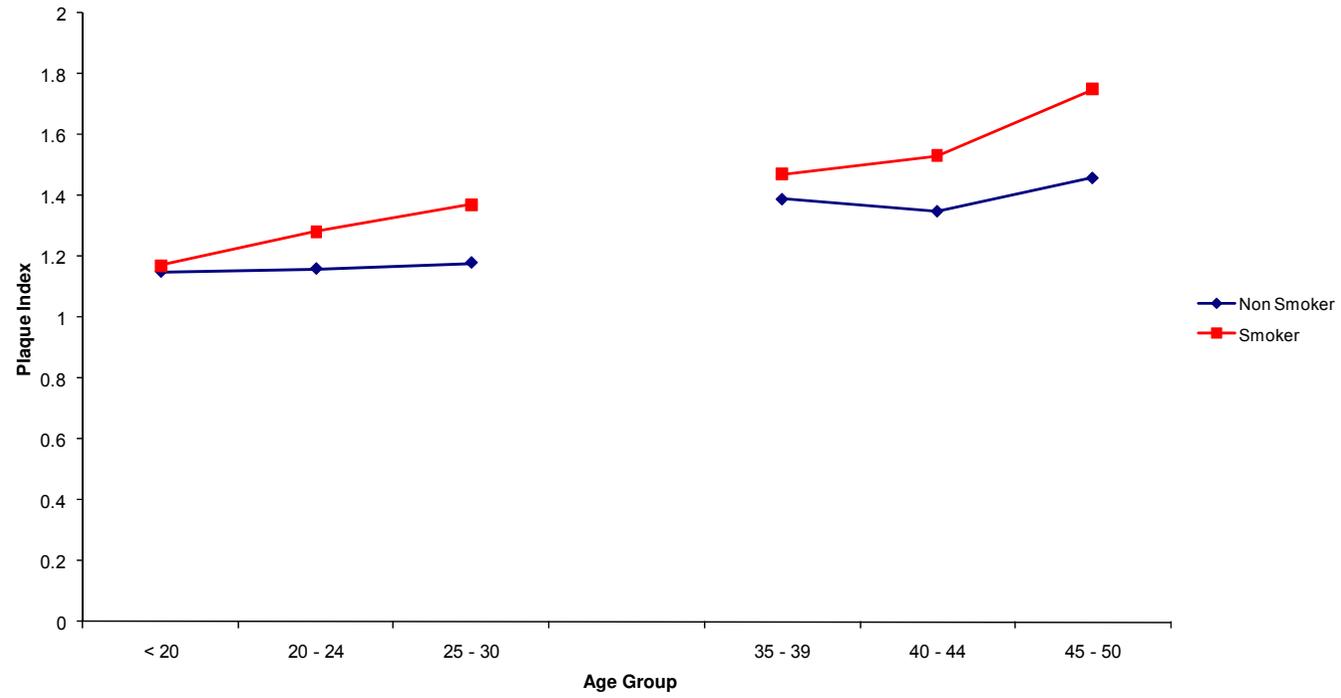


Figure2: Mean Gingival Index Scores for Non-Smoker and Smoker by Age

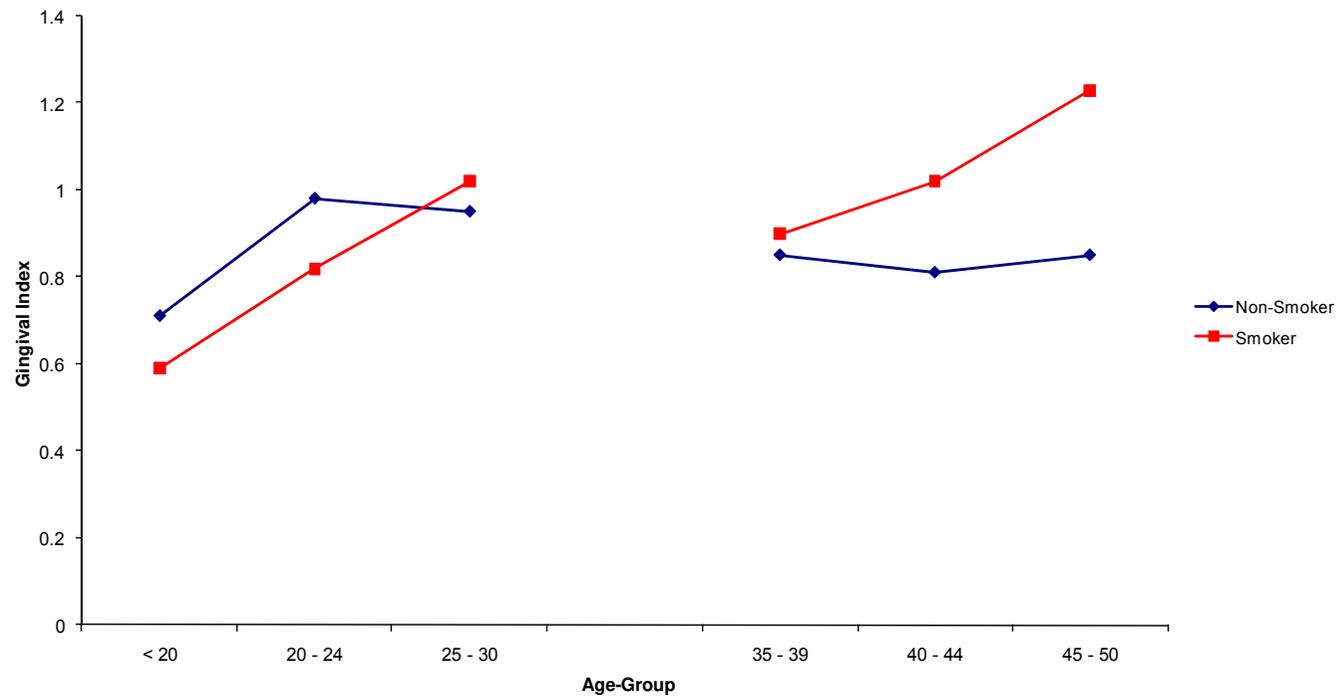


Figure 3: Mean Calculus Index Scores for Non-Smoker and Smoker by Age Group

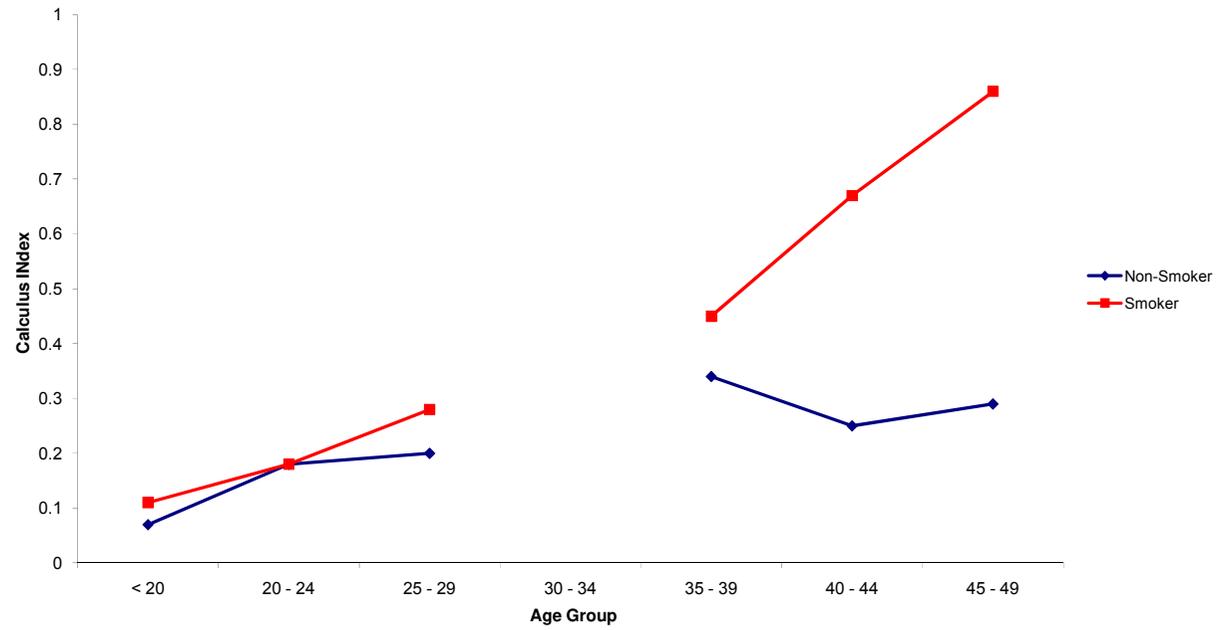


Figure 4: Mean Loss of Attachment for Non-Smoker and Smoker by Age Group

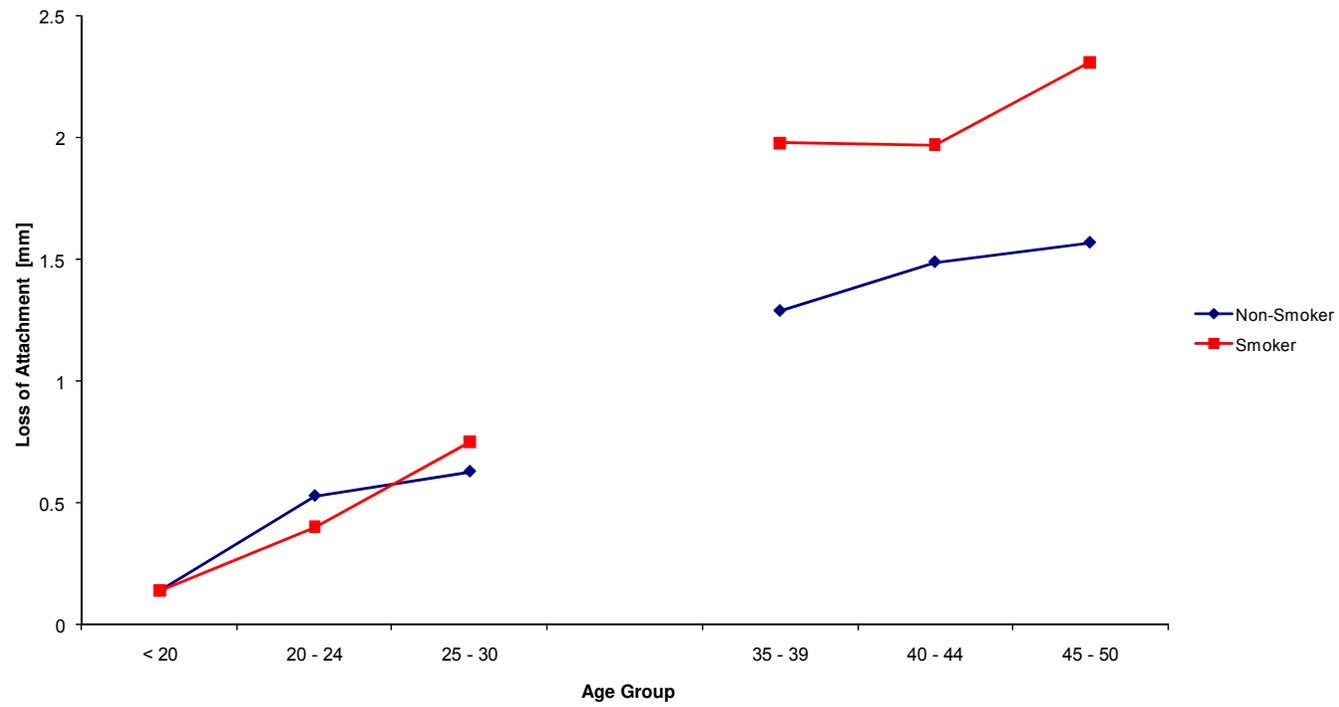


Figure 5: Scatter plot of mean attachment level (y-axis) and age (x-axis) in smokers and non-smokers.

