Natural History of Periodontal Disease in Adults: Findings from the Tecumseh Periodontal Disease Study, 1959-87

A.I. ISMAIL, E.C. MORRISON[1], B.A. BURT[2], R.G. CAFFESSE[1], and M.T. KAVANAGH

Faculty of Dentistry, Dalhousie University, Halifax, NS, Canada B3H 3J5; [1]Department of Periodontics, Dental Branch, University of Texas Health Science Center, Houston, Texas 77225; and [2]Program in Dental Public Health, School of Public Health, Ann Arbor, Michigan 48109

The purpose of this epidemiological study was to estimate the degree of change in periodontal attachment level in a sample of adults examined in 1959 and 1987 in Tecumseh, Michigan. Out of 526 individuals between the ages of five and 60 years in 1959, a sample of 325 resided within an 80-km-radius area in 1987. Of those, 167 were re-examined. Loss of periodontal attachment (LPA) was determined with a Michigan #0 probe on four tooth sites (disto-buccal, mid-buccal, mesio-buccal, mid-lingual) for all teeth present. Of the individuals contacted, 28 had lost all their teeth during the 28 years. Of the 167 adults examined, two refused periodontal probing. Out of the 165 adults with LPA measurements in 1987, only 22 (13.3%) had an average increased loss of 2 mm or more per person between 1959 and 1987; five adults (3.0%) had an average LPA increase of 3 mm or more, and only two adults (1.2%) had an average LPA increase of 4 mm or more. The attachment level in 59.3% of all the tooth sites examined in 1959 in the 165 individuals either did not change or changed within ± 1.0 mm. On the basis of bivariate analyses, the individuals with high LPA increase (≥2 mm) had the following characteristics significantly different from those with low LPA increase: They were older, smoked, had tooth mobility at baseline, higher gingivitis, plaque, calculus, and tooth mobility scores at follow-up, lower education level, and irregular dental attendance. However, in logistic regression analyses, only the following risk markers remained significantly associated with high LPA increase: age, smoking, and presence of tooth mobility.

J Dent Res 69(2):430–435, February, 1990

Introduction.

In oral epidemiology, most published work has concentrated on describing the distribution of oral conditions in populations and identifying the risk markers associated with these conditions [a risk marker is a factor associated with an increased probability of having a disease, but is not necessarily a causal factor (Last, 1983)]. Even for the most widely studied oral condition, dental caries, few studies of the natural history of the disease can be identified (Hughes *et al.*, 1982). The progression of periodontal disease in American adults has received even less investigation (Hughes *et al.*, 1982; Feldman *et al.*, 1987). Data from the North Carolina Studies (Hughes *et al.*, 1982) are not easily interpreted because they are expressed as Periodontal Index (Russell, 1956) averages.

The most comprehensive studies of the natural history of periodontal disease to date were carried out by Löe *et al.* (1978, 1986) in Sri Lanka and Norway. In each country, a group of adults was followed longitudinally. The first detailed analysis showed that the Norwegians had a lower severity of loss of periodontal attachment (LPA), compared with the Sri Lankan sample (Löe *et al.*, 1978). In the Norwegians, 40 years of age, the mean annual rates of attachment loss were 0.08 and 0.10 mm for interproximal and buccal surfaces, respectively. In the Sri Lankans, 40 years of age, the mean annual rates of attachment loss were 0.30 and 0.20 mm for the same surfaces.

The most recent analysis of the data (Löe *et al.*, 1986) collected from the Sri Lankan sample identified three distinct patterns of loss of attachment: none, moderate, or rapid. The percentages of individuals classified into these groups were 11, 81, and 8, respectively. Interestingly, there were no differences in oral hygiene status and gingivitis among the three groups throughout the follow-up period, although there was a tendency in the rapidly progressing group to have higher scores than the other two groups. Previous analysis (Löe *et al.*, 1978) of the data collected from the Norwegian sample showed no individuals with rapidly progressing loss of attachment.

Several recent epidemiological studies have found that LPA is widespread among adults (National Institute of Dental Research, 1987). However, the prevalence of advanced LPA found in several studies ranged between 5 and 30% (Hugoson and Jordan, 1982; Ismail *et al.*, 1987; National Institute of Dental Research, 1987; Johnson *et al.*, 1988). The risk markers associated with periodontal destruction are plaque, calculus, and gingivitis (Marshall-Day *et al.*, 1955; Carlos *et al.*, 1988). However, these clinical factors have not been shown to be reliable predictors of future periodontal breakdown (Badersten *et al.*, 1985; Lang *et al.*, 1986; Griffiths *et al.*, 1988).

This paper presents the findings of progression of loss of periodontal attachment level in a sample of adult residents of Tecumseh, Michigan, examined in 1959 and 1987. The purpose of the baseline study (Jamison, 1960, 1963) was to compare the findings of periodontal disease distribution measured by the Periodontal Index (PI) (Russell, 1956) and the Periodontal Disease Index (PDI) (Ramfjord, 1959). The purpose of the follow-up survey, carried out in 1987, was to investigate the changes in LPA over 28 years and to study the association of gingivitis, plaque, calculus, tooth mobility, smoking, dental visit pattern, and other socio-demographic factors with high loss of periodontal attachment (upper 10% of mean LPA distribution of the examinees).

Materials and methods.

The study was carried out in Tecumseh, Michigan, a city in southeast Michigan with a population of about 7000. Tecumseh was selected in 1956 by the Department of Epidemiology, School of Public Health, University of Michigan, as the site for community laboratory studies of chronic diseases (Epstein *et al.*, 1970). It was chosen by the Department of Epidemiology because it provided "stable and well-defined populations" with a variety of occupations and living conditions (Napier *et al.*, 1970) within driving distance from the university in Ann Arbor, Michigan.

Almost all persons living within the service area of 145 km^2 around Tecumseh were included in the sample (Napier *et al.*, 1970). In 1957, a house-to-house census in the designated area was carried out so that a household and kindred listing could

Received for publication May 29, 1989
Accepted for publication September 19, 1989
This investigation was supported in part by USPHS Research Grant DE-08051 from the National Institute of Dental Research, National Institutes of Health, Bethesda, MD 20892.
Data were collected when the first author was at the Faculty of Dentistry, McGill University, Montreal.

be completed. In addition, the population answered a self-administered questionnaire measuring experiences with selected chronic conditions and physical impairments (Napier *et al.*, 1970). There were about 8000 individuals in Tecumseh and its surrounding area at baseline (Napier *et al.*, 1970). The listing of households and their residents was used for division of the population into ten samples, each containing 10% of the population. Each sample included a proportional representation of all socio-economic strata.

The Jamison (1960) study included all non-institutionalized individuals, five years of age or older, who resided in the dwellings included in Sample V. Of the 801 sampled residents, 690 (86.1%) received a dental examination; 550 of these had permanent teeth. For the 1987 follow-up, 24 individuals born before 1900 were excluded, and, for practical reasons, the search was further restricted to include only those residing within an 80-km radius of Tecumseh.

Examination criteria.—The oral conditions measured during the follow-up examination in 1987 were: (a) dental plaque, (b) gingivitis, (c) calculus, (d) mobility, and (e) pocket depth and periodontal attachment level. All teeth were examined. Dental plaque was measured in the baseline study after application of a disclosing agent (Bismark Brown) and with use of Ramfjord's Plaque Index (Jamison, 1960). In the follow-up examination, no disclosing agent was used, and the Plaque Index of Silness and Löe was used instead (Löe, 1967). Gingivitis, calculus, mobility, pocket depth, and loss of periodontal attachment were measured in both the baseline and follow-up surveys, according to the methods developed by Ramfjord (1959). Loss of periodontal attachment was measured for four sites around each tooth, as described by Jamison (1960) (mesial-buccal, mid-buccal, distal-buccal, and mid-lingual). The same type of periodontal probe that was used during the baseline examination (Michigan #0) was used during the follow-up examination.

In addition to the dental examination, the participants answered a short questionnaire, administered by the coordinator of the project, which asked about income and education status, dental visit pattern, dental insurance, and smoking habits.

One examiner carried out all examinations in 1959; there were three in 1987. The three examiners underwent a two-day training session shortly before data collection. For assessment of diagnostic standardization, multiple examinations were carried out on nine participants during the study, with the most experienced periodontal examiner used as the "gold standard". The inter-examiner agreement with the gold standard was good for LPA within 2 mm (Kappa = 0.50-0.60). Agreement was lower for gingivitis because of the more subjective nature of the index used. No consistent examiner effect was demonstrated in the bivariate or regression analyses.

Statistical analysis.—Data were recorded on a portable microcomputer with a program written specifically for this study. The unit of analysis was the individual examinee. Site-specific analyses were carried out, but no hypotheses were tested on site data because of clustering effects of attachment level measurements within individuals (Donner and Banting, 1988). Recently, statistical methods have been described that will adjust for these clustering effects where all sites are categorized by the same co-variable (Donner, 1985; Donner and Banting, 1988). However, these methods do not address site-specific analyses where different sites within the same mouth are categorized by several co-variables, as was the case in this study.

While the mean pocket depth or attachment level is used as an indicator of a history of past periodontitis in epidemiological studies, in this study, the mean LPA scores were not normally distributed but rather were significantly skewed. Another problem encountered was the heterogeneity of the variances of the mean LPA and other dental variables at baseline and follow-up examinations. As a result, we decided to categorize the variables for analysis. Average gingivitis, calculus, and plaque scores *per* patient were categorized on the basis of the 33.3 and 66.7 percentiles (see Table 6 for tertile values). Values below the first tertile were designated as "low", those between the 33.3 and 66.7 percentiles were classified as "middle", and those larger than the 66.7 percentile were classified as "high". Mean mobility and number of missing teeth were also categorized by tertiles, except in cases where a large number of the participants had a score of "0". In such cases—namely, average mobility and number of missing teeth at baseline—the variables were dichotomized as follows: Scores equal to zero were classified as "low", and those larger than zero were classified as "high".

Because of the interest in identification of a high-risk group, and to detect differences in LPA that were independent of examiner variability (± 2 mm), we used the following definition of low and high LPA differences between the follow-up and baseline examinations: Those with a mean LPA difference of less than 2 mm over the 28 years were classified as "low LPA", while those with a mean LPA difference of 2 mm or more were classified as the "high-LPA" group. The high-LPA group was made up of 22 persons (13.3% of the individuals examined).

Testing for differences among the mean changes in LPA was carried out by means of ANOVA with Scheffé's tests. Chi-squared coefficients were computed when categorical data were analyzed. Odds ratios—defined as the increase in risk of belonging to the high-LPA group in exposed *vs.* unexposed individuals—were computed with 95% confidence intervals (Fleiss, 1981). Such odds ratios are estimates of the strength of association between degree of change in LPA over the 28 years and various risk markers. Only odds ratios with a lower confidence limit greater than one were considered clinically significant.

The effect of the risk markers on changing LPA over the 28 years was investigated with use of several multiple logistic regression models (Kleinbaum *et al.*, 1982). Because of multicollinearity among the risk markers, these included different sets of variables.

Results.

Table 1 shows the results of the contacts with the original 526 participants in the 1959 survey. No statistically or clinically significant differences were found between those who resided within the 80-km radius of Tecumseh and those who did not, with respect to baseline determinations of mean loss of periodontal attachment, mean number of missing teeth, or plaque, calculus, gingivitis, and mobility scores. Furthermore, no differences were found in mean baseline periodontal disease status between those examined in 1987 and those not examined.

Table 2 presents the number of teeth lost during the 28 years, the percentage of sites with $\geq$ 4 mm increased loss of attachment, and the percentage of these sites (with $\geq$ 4 mm LPA) that are interproximal for the examinees with high LPA. The percentages of tooth sites affected with $\geq$ 4.0 mm LPA in the 22 individuals ranged between 7 and 79% of all tooth sites examined. Only one individual in the high-LPA group had only a single buccal site affected, with a loss of $\geq$ 4.0 mm in the two teeth present in his/her mouth (17 teeth were lost between

TABLE 1
NUMBER OF INDIVIDUALS WITH PERMANENT TEETH IN 1959 AND WHO RESIDED WITHIN AN 80-KM RADIUS OF TECUMSEH IN 1987

Status in 1987	N
Within 80-km radius of Tecumseh:	
Examined in 1987	167*
Edentulous	28
Refused examination	52
Scheduled but did not keep appointment	37
Retired in Florida	16
Not contacted, no phone listed	12
Moved beyond 80-km radius	9
Deceased	4
Sub-total	325
Outside of 80-km radius area:	201
Total	526

*Loss of periodontal attachment and pocket depths were measured for only 165 individuals.

TABLE 2
NUMBER OF TEETH LOST AND INCREASE OF LPA BETWEEN 1959 AND 1987 IN THE 22 HIGH-LPA INDIVIDUALS

			% of Tooth Sites with LPA ≥ 4.0 mm	
Person	MT*	Total Sites†	A‡	B§
1	10	40	25.0	80.0
2	0	111	18.0	90.0
3	14	27	25.9	42.9
4	0	112	16.1	66.7
5	0	112	7.1	37.5
6	0	79	16.5	69.2
7	0	39	53.8	42.9
8	9	68	20.6	42.9
9	18	24	37.5	44.5
10	3	36	25.0	55.6
11	1	28	28.6	12.5
12	1	108	24.1	38.5
13	19	28	78.6	40.9
14	0	112	9.8	72.7
15	18	20	55.0	45.5
16	4	44	18.2	50.0
17	0	112	8.9	60.0
18	14	39	12.8	40.0
19	7	72	76.4	47.3
20	17	8	12.5	0.0
21	7	67	41.8	39.3
22	3	76	10.5	50.0

*MT = Number of teeth lost between 1959 and 1987.
†Total sites = Total number of tooth sites examined in 1987.
‡Percentage of total sites with ≥4.0 mm LPA increase.
§Of those sites with LPA ≥4.0 mm, the percentage that were interproximal.

1959 and 1987). In the remaining 21 individuals, an average of 52% of tooth sites with ≥ 4.0 mm LPA were interproximal.

The mean change in attachment loss by age cohort is presented in Table 3. The older cohorts had a greater mean increase in mean attachment loss from 1959 to 1987 than did the youngest cohort; however, the differences between cohorts in mean LPA increase were not statistically significant. The mean increase in LPA over 28 years in those born between 1900 and 1924 was 1.34 mm, while in those born between 1945 and 1954 the mean increase was 0.88 mm. The annual average LPA increase was 0.04 mm.

The percentage distribution of the 165 individuals categorized by magnitude of increase in average LPA, low and high, is presented in Fig. 1. About 5% of the youngest age cohort had a high increase in attachment loss, compared with 23% of the oldest age cohort. Such differences in percentage frequencies between cohorts were significant ($p < 0.05$). Fig. 2 presents the distribution of tooth sites by the degree of change in attachment level between 1959 and 1987. Only 13.4% of the tooth sites with attachment level measurements in 1959 and 1987 experienced a loss of 3 mm or more.

TABLE 3
MEAN DIFFERENCE IN LOSS OF PERIODONTAL ATTACHMENT (LPA) BETWEEN 1959 AND 1987 BY AGE COHORTS

		Mean LPA (± SD*)		
Year of Birth	n	1959	1987	Difference
1945-54	58	0.00 (0.00)	0.88 (0.66)	0.88 (0.66)
1935-44	36	0.09 (0.16)	1.30 (1.18)	1.21 (1.15)
1925-34	49	0.55 (0.66)	1.78 (1.07)	1.23 (0.78)
1900-24	22	0.94 (1.40)	2.29 (1.80)	1.34 (0.78)
Total	165	0.31 (0.71)	1.43 (1.19)	1.12 (0.85)

*SD = Standard deviation.

TABLE 4
PREVALENCE OF INCREASE IN LOSS OF PERIODONTAL ATTACHMENT (LPA) BETWEEN 1959 AND 1987 BY DEGREE OF INCREASE

Increase in LPA 1959-87	Percentage of Sites	Percentage of Individuals*	Percentage of Individuals with Mean LPA Difference†
≥ 2 mm	33.1	97.0	13.3
≥ 3 mm	15.0	87.9	3.0
≥ 4 mm	5.1	57.0	1.2

*With at least one site affected (n = 165).
†Mean LPA difference over all sites (n = 165).

TABLE 5
FREQUENCIES OF TOOTH SITES BY MILLIMETERS OF LOSS OF PERIODONTAL ATTACHMENT (LPA) IN 1959 AND 1987

LPA	LPA in 1987 by Tooth Site					Missing Teeth 1959-87	
in 1959	0	1-3	4-6	7+	MS*	MT†	%‡
0	4306	5551	469	34	889	140	6.3
1-3	258	1220	400	30	496	183	17.4
4-6	0	16	38	10	77	34	53.1
7+	0	0	0	5	42	19	86.4
Total	4564	6787	907	79	1504	376	10.9

*MS = Number of missing tooth surfaces.
†MT = Number of missing teeth.
‡% = Percentage of teeth lost during the 28 years by maximum LPA score of the tooth.

Table 4 presents, by degree of increase, the prevalence of LPA increase over 28 years. Only 5% of the 12,337 tooth sites present in 1959 and 1987 had an LPA increase of 4 mm or more over 28 years, while 33% of the tooth sites followed had a loss of 2 mm or more, with 97% of those individuals followed having at least one such site. Table 5 presents the distribution of tooth sites by their LPA at baseline and follow-up. In 1959, 81% of the tooth sites had no attachment loss, while in 1987, 37% percent of sites had no attachment loss. Of tooth sites present at both examinations, attachment loss in 66% either did not change or changed within ± 1.0 mm. The percentage of teeth lost during the 28 years increased as the mean baseline LPA *per* tooth increased. Only 6% of all teeth

TABLE 6
OBSERVED ODDS RATIOS OF THE ASSOCIATION BETWEEN DEGREE OF DIFFERENCE IN LOSS OF PERIODONTAL ATTACHMENT (LPA) BETWEEN 1959-87 AND RISK MARKERS, TECUMSEH

Variable	Odds Ratios (95% Confidence Intervals)*
Gender	2.16 (0.85, 5.47)
Education	3.05 (1.10, 8.43)
Dental Visits	3.09 (1.16, 8.24)
Dental Insurance	2.06 (0.78, 5.40)
Smoking	6.26 (2.42, 16.20)
Income	1.01 (0.41, 2.49)
Age	
1900-24/1945-54	5.39 (1.17, 24.93)
1925-34/1945-54	3.58 (0.89, 14.32)
1935-44/1945-54	3.67 (0.86, 15.72)
Baseline Dental Status (High *vs.* Low Status)*:	
Gingivitis	0.98 (0.36, 2.68)
Calculus	1.74 (0.58, 5.19)
Plaque	0.73 (0.25, 2.12)
Mobility	5.27 (1.88, 14.73)
Missing teeth	1.55 (0.62, 3.85)
Follow-up Dental Status (High *vs.* Low Status)*:	
Gingivitis	4.15 (1.27, 13.55)
Calculus	6.34 (1.72, 23.40)
Plaque	10.87 (2.36, 50.06)
Mobility	†
Missing Teeth	1.99 (0.72, 5.47)

*Definitions of LPA and dental status categories:
Low LPA difference: <2 mm; high LPA difference: ≥2 mm.
Low baseline gingivitis: <1.12; high baseline gingivitis: >1.60.
Low baseline calculus: <0.04; high baseline calculus: >0.37.
Low baseline plaque: <1.33; high baseline plaque: >1.79.
Low baseline mobility: 0; high baseline mobility: >0.
Low baseline missing teeth: 0; high baseline missing teeth: >0.
Low follow-up gingivitis: <0.91; high follow-up gingivitis: >1.25.
Low follow-up calculus: <0.84; high follow-up calculus: >1.57.
Low follow-up plaque: <0.75; high follow-up plaque: >1.20.
Low follow-up mobility: <0.08; high follow-up mobility: >0.36.
Low follow-up missing teeth: <1.00; high follow-up missing teeth: >3.72.
†One cell in the contingency Table had a frequency of 1.00.

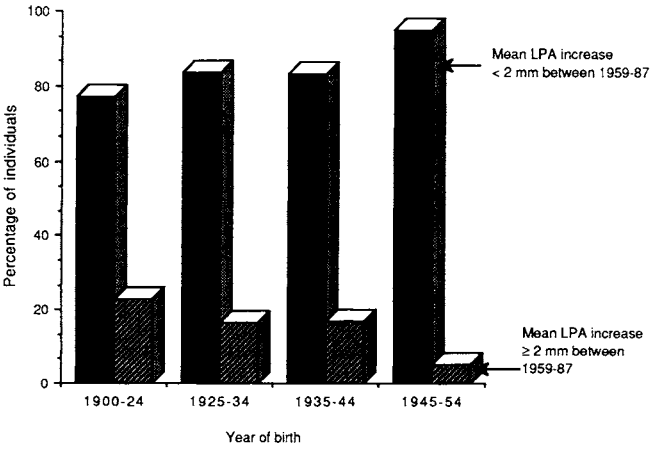

Fig. 1 — Percentage distribution of loss of periodontal attachment (LPA) *per* individual between 1959 and 1987 by year of birth. (Tecumseh, MI, 1959-87)

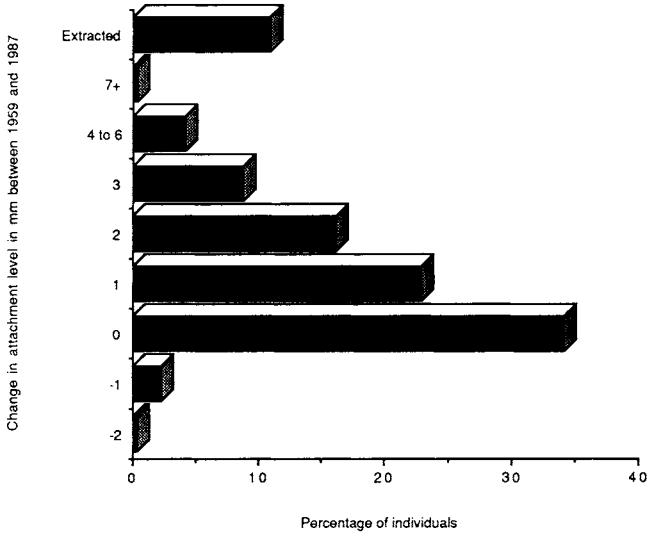

Fig. 2 — Percentage of tooth sites by degree of change in attachment level in mm between 1959 and 1987, Tecumseh, MI.

with no baseline attachment loss were extracted, compared with 53% of the teeth with baseline attachment loss between 4 and 6 mm.

Table 6 presents the odds ratios and their confidence intervals for the change in LPA for risk markers measured in the study. When changes in LPA were categorized as greater than or equal to 2 mm *vs.* less than 2 mm, the factors associated with elevated risk of high LPA increase included level of education, regularity of dental care, smoking, age, mobility at baseline, and gingivitis, calculus, and plaque at follow-up. Those who reported that they were smokers (approximately 20% of those examined) had six times higher odds of a high LPA increase, compared with non-smokers. Irregular dental visitors (approximately 20% of those examined) had a threefold higher risk of high attachment loss than regular attenders.

Table 7 presents the findings from the logistic regression analyses. A consistent and significant association was found between high LPA increases and smoking, age, and presence of tooth mobility. The odds of smokers having high LPA increases were 12-14 times the odds of non-smokers. The oldest age group had significantly higher odds than the youngest age groups. Calculus and gingivitis measured at either baseline or follow-up examinations were not found to increase the odds of high LPA increase. Plaque at follow-up significantly increased the odds of a high LPA increase, when considered independently of tooth mobility. However, as shown in Table 7, in models that included mobility at follow-up, the odds ratio for plaque was not significantly different from 1.00.

Discussion.

Bias is an inherent problem in longitudinal studies: There is a likelihood that persons remaining in a long-term study such as this one differ from those who did not. While no differences were found in the baseline levels of plaque, calculus, gingivitis, or loss of periodontal attachment between those examined and those who were not, the possibility remains that those participants seen in 1987 were in better oral health than those not seen. It is perhaps remarkable that during the 28 years only 10.9% of the teeth present at baseline were lost. Table 5 shows that a total of 376 teeth was lost over the 28 years in the 165 individuals examined for loss of periodontal attachment. Out of those teeth, 313 had an average LPA of 2 mm or less at baseline. However, when the frequency of lost teeth was dis-

TABLE 7
RESULTS OF LOGISTIC REGRESSION ANALYSES

Variable	B	S.E.	P	Odds Ratios
Baseline Model:				
Intercept	−0.93	0.33	<0.01	
Year of Birth				
1935-44/1945-54	1.70	0.83	<0.05	5.47
1925-34/1945-54	1.57	0.86	N.S.	4.81
1900-24/1945-54	2.34	0.98	<0.05	10.38
Smoking	2.65	0.63	<0.01	14.15
Mobility				
(high/low)	1.59	0.67	<0.05	4.90
Follow-up Model:				
Intercept	−2.06	0.43	<0.01	
Year of Birth				
1935-44/1945-54	1.48	0.91	N.S.	4.39
1925-34/1945-54	2.16	0.91	<0.05	8.67
1900-24/1945-54	2.47	1.01	<0.05	11.82
Smoking	2.44	0.69	<0.01	11.47
Mobility at follow-up				
(mid/low)	1.04	1.16	N.S.	2.83
(high/low)	2.25	1.12	<0.05	9.49
Plaque at follow-up				
(mid/low)	0.79	0.95	N.S.	2.20
(high/low)	1.62	0.88	N.S.	5.05

played by the maximum LPA score of the tooth (around the four examined tooth sites), a linear increase in frequency of tooth loss was observed with the increase in the maximum baseline LPA tooth score. There is, therefore, the possibility that many of the 376 teeth lost over the 28 years were affected by periodontal destruction, but whether the loss of periodontal attachment during the 28 years was a reason for the loss of teeth cannot be elucidated from the data collected.

As previously stated, recent epidemiological studies have shown that there are groups of individuals who have higher prevalence of advanced periodontal destruction than the population at large. Estimates of the prevalence of this group range between 5 and 30% of the population (Johnson *et al.,* 1988). In the Jamison (1960) study, the prevalence of individuals with an LPA of 6 mm or more in 1959-60 was about 4%, a prevalence that is similar to that reported in recent studies (Beck *et al.,* 1984; Ismail *et al.,* 1987; National Institute of Dental Research, 1987). No reliable information is available about longitudinal trends of LPA and pocket depth. The factors associated with high LPA increase, in the regression analysis, were smoking, age, presence of one or more mobile teeth, and high plaque scores at follow-up. Smoking has been correlated with periodontal disease (Ismail *et al.,* 1983); however, the mechanism by which smoking increases periodontal breakdown is not yet clear (Baab and Öberg, 1987). Because of the small number of mobile teeth at baseline (100 out of the 3487 teeth present in 1959) and the subsequent loss of 45% of these teeth during the 28 years, presence of tooth mobility in this study was used only as a risk marker of individuals with high LPA increase. Further analysis with the tooth used as the unit will be carried out for investigation of the association between tooth mobility, tooth loss, and loss of periodontal attachment.

A high plaque score at follow-up was a significant risk marker in the logistic model. However, when both plaque and mobility at follow-up were included, the odds ratio for plaque was not significantly different from 1.00. Gingivitis and calculus were associated with high LPA increase in the bivariate model; however, when they were correlated with high LPA increase within the multivariate context, their odds ratios did not remain significantly different from 1.00. This finding does not indicate that these factors are not clinically significant, but only that as discriminators between adults with low and high LPA increases, they were not as strong risk markers as age, smoking, plaque, and tooth mobility.

Further studies to define the risk markers at both the site-specific and individual levels will be needed for full determination of the risk equations for periodontal diseases. Risk assessment should be carried out on representative samples of the population (*i.e.*, diseased and healthy individuals) rather than on periodontal patients. There are few published microbiological and immunological risk-assessment studies of periodontal diseases carried out with a sample from the population (Carlos *et al.,* 1988). By reliance on only periodontal patients as a source of information about risk markers of destructive periodontal disease, risk-assessment studies will be over-represented by patients who have a relatively higher level of periodontal disease than the population at large, thereby introducing sampling bias (Ellenberg and Nelson, 1980).

In conclusion, the loss of periodontal attachment progressed very slowly in adults examined in this study, except for approximately 13% who showed a susceptibility to higher loss of attachment. The study identified the following risk markers associated with high LPA increase: age, smoking, and tooth mobility. Individuals with high LPA increases also had higher levels of gingivitis, calculus, and plaque at follow-up.

Acknowledgments.

We thank Dr. Homer C. Jamison for allowing us to use the data from the 1959 Tecumseh periodontal disease study; Dr. Sigurd P. Ramfjord for his valuable comments during the project; David Schottenfeld, MD, and Stevo Julius, MD, for their help; and the late Alice Exelby and Mrs. Virginia Cline, who patiently worked with us during the days and evenings of October, 1987.

REFERENCES

BAAB, D.A. and ÖBERG, P.A. (1987): The Effect of Cigarette Smoking on Gingival Blood Flow in Humans, *J Clin Periodontol* 14:418–424.

BADERSTEN, A.; NILVÉUS, R.; and EGELBERG, J. (1985): Effect of Non-surgical Periodontal Therapy. VII Bleeding, Suppuration and Probing Depth in Sites with Probing Attachment Loss, *J Clin Periodontol* 12:432–440.

BECK, J.D.; LAINSON, P.A.; FIELD, H.M.; and HAWKINS, B.F. (1984): Risk Factors for Various Levels of Periodontal Disease and Treatment Needs in Iowa, *Community Dent Oral Epidemiol* 12:17–22.

CARLOS, J.P.; WOLFE, M.D.; ZAMBON, J.J.; and KINGMAN, A. (1988): Periodontal Disease in Adolescents: Some Clinical and Microbiological Correlates of Attachment Loss, *J Dent Res* 67:1510–1514.

DONNER, A. (1985): A Regression Approach to the Analysis of Data Arising from Cluster Randomization, *Int J Epidemiol* 14:322–326.

DONNER, A. and BANTING, D. (1988): Analysis of Site-specific Data in Dental Studies, *J Dent Res* 67:1392–1395.

ELLENBERG, J.H. and NELSON, K.B. (1980): Sample Selection and the Natural History of Disease: Studies of Febrile Seizures, *J Am Med Assoc* 243:1337–1340.

EPSTEIN, F.H.; NAPIER, J.A.; BLOCK, W.D.; HAYNER, N.S.; HIGGINS, M.P.; JOHNSON, B.C.; KELLER, J.B.; METZNER, H.L.; MONTOYE, H.J.; OSTRANDER, L.D.; and ULLMAN, B.M. (1970): The Tecumseh Study. Design, Progress, and Perspectives, *Arch Environ Health* 21:402–407.

FELDMAN, R.S.; ALMAN, J.E.; and CHAUNCEY, H.H. (1987):

Periodontal Disease Indexes and Tobacco Smoking in Healthy Aging Men, *Gerodontics* 1:43–46.

FLEISS, J.L. (1981): **Statistical Methods for Rates and Proportions**, New York: John Wiley & Sons, pp. 61–64.

GRIFFITHS, G.S.; WILTON, J.M.A.; CURTIS, M.A.; MAIDEN, M.F.J.; GILLETT, I.R.; WILSON, D.T.; STERNE, J.A.C.; and JOHNSON, N.W. (1988): Detection of High-risk Groups and Individuals for Periodontal Diseases. Clinical Assessment of the Periodontium, *J Clin Periodontol* 15:403–410.

HUGHES, J.T.; ROZIER, R.G.; and RAMSEY, D.L. (1982): **Natural History of Dental Diseases in North Carolina. 1976-1977**, Durham, NC: Carolina Academic Press, pp. 103–114.

HUGOSON, A. and JORDAN, T. (1982): Frequency Distribution of Individuals Aged 20-70 Years According to Severity of Periodontal Disease, *Community Dent Oral Epidemiol* 10:187–192.

ISMAIL, A.I.; BURT, B.A.; and EKLUND, S.A. (1983): Epidemiologic Patterns of Smoking and Periodontal Disease in the United States, *J Am Dent Assoc* 106:617–621.

ISMAIL, A.I.; EKLUND, S.A.; STRIFFLER, D.F.; and SZPUNAR, S.M. (1987): The Prevalence of Advanced Loss of Periodontal Attachment in Two New Mexico Populations, *J Periodont Res* 22:119–124.

JAMISON, H.C. (1960): *Prevalence and Severity of Periodontal Disease in a Sample of a Population,* Ann Arbor, MI: Doctoral Dissertation, University of Michigan, pp. 1–153.

JAMISON, H.C. (1963): Some Comparisons of Two Methods of Assessing Periodontal Disease, *Am J Public Health* 53:1102–1106.

JOHNSON, N.W.; GRIFFITHS, G.S.; WILTON, J.M.A.; MAIDEN, M.F.J.; CURTIS, M.A.; GILLETT, I.R.; WILSON, D.T.; and STERNE, J.A.C. (1988): Detection of High-risk Groups and Individuals for Periodontal Diseases. Evidence for the Existence of High-risk Groups and Individuals and Approaches to their Detection, *J Clin Periodontol* 15:276–282.

KLEINBAUM, D.G.; KUPPER, L.L.; and MORGENSTERN, H. (1982): **Epidemiologic Research**, Belmont, CA: Lifetime Learning Publications, pp. 461–475.

LANG, N.P.; JOSS, A.; ORSANIC, T.; GUSBERTI, F.A.; and SIEGRIST, B.E. (1986): Bleeding on Probing. A Predictor of the Progression of Periodontal Disease?, *J Clin Periodontol* 13:590–596.

LAST, J.M. (1983): **A Dictionary of Epidemiology,** New York: Oxford University Press, p. 93.

LÖE, H. (1967): The Gingival Index, the Plaque Index, and the Retention Index Systems, *J Periodontol* 38:610–616.

LÖE, H.; ANERUD, A.; BOYSEN, H.; and MORRISON, E.C. (1986): Natural History of Periodontal Disease in Man. Rapid, Moderate, and No Loss of Attachment in Sri Lankan Laborers 14 to 46 Years of Age, *J Clin Periodontol* 13:431–440.

LÖE, H.; ANERUD, A.; BOYSEN, H.; and SMITH, M. (1978): The Natural History of Periodontal Disease in Man. The Rate of Periodontal Destruction Before 40 Years of Age, *J Periodontol* 49:607–620.

MARSHALL-DAY, C.D.; STEPHENS, R.G.; and QUIGLEY, L.F., Jr. (1955): Periodontal Disease: Prevalence and Incidence, *J Periodontol* 26:185–203.

NAPIER, J.A.; JOHNSON, B.C.; and EPSTEIN, F.H. (1970): The Tecumseh, Michigan, Community Health Study. In: **The Community as an Epidemiologic Laboratory**, I.I. Kesler and M.L. Levin, Eds., Baltimore: Johns Hopkins Press, pp. 25–46.

NATIONAL INSTITUTE OF DENTAL RESEARCH (1987): *Oral Health of United States Adults. National Findings*, Bethesda, MD: NIH Pub. No. 87-2868, pp. 69–79.

RAMFJORD, S.P. (1959): Indices for Prevalence and Incidence of Periodontal Disease, *J Periodontol* 30:51–59.

RUSSELL, A.L. (1956): A System of Classification and Scoring for Prevalence Surveys of Periodontal Disease, *J Dent Res* 35:350–359.