Dear students,
In order to complete the curriculum of the second term and finish the academic year (2019-2020) you need to submit 1 report and 1 presentation (Power Point). Both tasks will be assessed. Their marks + your attendance (Homework Tasks) will determine your final grade. The topics are:

1. Report (3 pages – Times New Roman, 12): "Maintaining healthy dental status – hygiene and nutrition (food and drinks)". Include Bibliography Section.
2. Presentation (Power Point; 8-10 slides). Discuss a dental issue by choice.

Submit both Report and Presentation to the e-mail: svetlanataneva@abv.bg, by 21.05. 2020.

Task (12 week): Discuss the methods of
Dental Plaque Study - 200 words

Dental plaque and oral health during the first 32 years of life

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Accumulation of plaque can cause the rapid onset of gingivitis, and long-term exposure to plaque can lead to the loss of periodontal attachment and bone support. Long-term exposure to plaque also can lead to the demineralization and destruction of the teeth through caries. Removal of plaque is an effective way to help prevent periodontal diseases and caries,[1,2] but if deposits remain on the teeth across time, destructive periodontal disease, caries or both may occur.

When considering the role of plaque in the etiology of caries and periodontal disease, time is perhaps the most important factor. Longitudinal research is an important part of determining the role plaque plays in oral disease; however, epidemiologic studies have focused primarily on the quantity and quality of plaque in a person's mouth at a given point in time. No large-scale epidemiologic research study has been undertaken regarding continuity and change in plaque levels across the long term (such as through childhood and into adulthood) and the association of the continuity of and change in plaque levels with oral health. A longitudinal examination of the quantity of plaque could show how it affects dental disease (particularly cumula-

ABSTRACT

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Background. Studies investigating the role of dental plaque in oral disease have focused primarily on the quantity and quality of plaque at a given point in time. No large-scale epidemiologic research has been conducted regarding the continuity and change in plaque levels across the long term and the association of plaque levels with oral health.

Methods. The authors used data from the Dunedin Multidisciplinary Health and Development Study. Collection of dental plaque data occurred at ages 5, 9, 15, 18, 26 and 32 years by means of the Simplified Oral Hygiene Index. The authors assessed oral health outcomes when participants were aged 32 years.

Results. The authors identified three plaque trajectory groups (high, n = 357; medium, n = 450; and low; n = 104) and found substantial, statistically significant differences in both caries and periodontal disease experience among those groups. For example, after the authors controlled for sex, socioeconomic status and dental visiting pattern, they found that participants in the high-plaque-trajectory group lost nearly five times more teeth owing to caries than did those in the low-plaque-trajectory group.

Conclusions. Across the long term, participants in the high-plaque-trajectory group were more likely to experience caries, periodontal disease and subsequent tooth loss than were those in the low- or medium-plaque-trajectory groups, and they experienced all those conditions with greater severity.

Clinical Implications. Improving oral health requires emphasizing long-term self-care, as well as providing broad public health and health promotion measures that promote and support oral self-care. This study's findings suggest that poor oral hygiene and smoking have a synergistic effect on periodontal disease experience.

Key Words. Longitudinal study; adult; socioeconomic status; oral hygiene; dental neglect; smoking.

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tive dental decay and decayed, missing and filled surfaces [DMFS]) throughout life.

The human body's response to plaque may differ according to a person's sex and race,[3] as well as according to his or her medical status and genotype. For example, children with type 1 diabetes mellitus have worse oral hygiene than do children who do not have diabetes, despite having similar oral hygiene practices.[4,5] Manual dexterity is a key factor in oral hygiene, and some people who have poor manual dexterity are unable to achieve a satisfactory level of oral cleanliness through self-care.[6]

People who are socially advantaged,[7] have high self-esteem[8] or are female[7] are more likely to brush frequently than are people who are socially disadvantaged, have low self-esteem or are male, respectively. People often have different motivations for toothbrushing, and people who are socially advantaged may be more likely to brush their teeth to avoid dental problems, whereas people who are socially disadvantaged may be more likely to brush their teeth for the sake of good appearance.[7] Flossing behaviors follow a pattern similar to that of toothbrushing behaviors,[9] as do more general hygiene behaviors. The findings of recent research have shown a link between general hygiene behaviors—such as hand washing—and oral hygiene practices.[10]

Sustained management of plaque is an important aspect of oral disease control. Frequent professional dental prophylaxis reduces the severity and progression of gingivitis and periodontal attachment loss (AL),[11] and scaling and other periodontal therapies are effective in maintaining periodontal attachment and reducing periodontal pocket depths (PDs).[12] On the other hand, people who experience the most periodontal AL tend to have greater amounts of plaque on their teeth.[13] For example, investigators in the Tecumseh Study found that people who became edentulous during the 28 years of follow-up had significantly higher plaque index scores at baseline than those who retained their teeth; however, the investigators found no statistically significant differences in plaque scores at baseline and the number of teeth lost among those who had not lost all of their teeth during the follow-up period.[14]

We conducted a study to describe plaque levels through childhood and early adulthood, and to determine the association of those plaque levels with oral health in adulthood. We also investigated the extent to which social inequalities in adult oral health may be mediated by poor oral hygiene habits throughout childhood and into adulthood.

PARTICIPANTS AND METHODS

Sample. The Dunedin Multidisciplinary Health and Development Study (DMHDS) is a longitudinal study of a birth cohort of children who were born at Queen Mary Hospital in Dunedin, New Zealand, between April 1, 1972, and March 31, 1973.[15] Investigators obtained perinatal data at the time of birth and defined the sample for the longitudinal study when the children were age 3 years. This sample initially comprised 1,037 children assessed within a month of their third birthdays and when they were aged 5, 7, 9, 11, 13, 15, 18, 21, 26 and 32 years. Barriers to people's participating were minimized by the DMHDS' paying for the costs of participation (for example, travel, lost wages and child care). More than 90 percent of the participants in the cohort self-identified themselves as being of New Zealand European origin. The University of Otago Human Ethics Committee (Dunedin, New Zealand) granted ethics approval for each assessment phase of the DMHDS.

Measures. In the DMHDS, collection of plaque data took place when participants were aged 5, 9, 15, 18, 26 and 32 years. At each assessment, examiners whose techniques were calibrated observed and scored the amount of plaque on teeth by means of a four-point scale (0 = no debris or stain detectable; 1 = soft debris covering no more than the cervical one-third of the tooth surface or extrinsic stains without other debris regardless of surface area covered; 2 = soft debris covering more than one-third but no more than two-thirds of the exposed tooth surface; and 3 = soft debris covering more than two-thirds of the exposed tooth surface) in accordance with Greene and Vermillion's[16] Simplified Oral Hygiene Index (OHI-S).

The examiner observed and scored six teeth (four posterior and two anterior) for each participant. For the posterior teeth, the first fully erupted tooth distal to the second premolar was examined in each quadrant. For maxillary molars, the buccal surfaces were scored, and for mandibular molars, the lingual surfaces were scored. For the anterior teeth, the labial surfaces of the maxillary right and the mandibular left central incisors were scored. If these incisors

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ABBREVIATION KEY. AL: Attachment loss. **BIC:** Bayesian information criterion. **DMFS:** Decayed, missing or filled surfaces of permanent teeth. **DMHDS:** Dunedin Multidisciplinary Health and Development Study. **GBTM:** Group-based trajectory modeling. **GR:** Gingival recession. **OHI-S:** Simplified Oral Hygiene Index. **PD:** Pocket depth. **SES:** Socioeconomic status.

were not present, the central incisor on the opposite side of the midline was scored. The examiner did not score teeth with full-coverage crowns and instead scored alternate teeth in the same group. If a participant did not have a tooth in one of the six groups, that tooth was excluded from the OHI-S score. The OHI-S score was computed as the mean of the plaque scores for the teeth that were observed and scored (index teeth).[16]

A total of 1,024 participants underwent at least one of the dental examinations at ages 5, 9, 15, 18, 26 and 32 years, and OHI-S data from at least three of these time points, including at age 32 years, were available for 911 participants. We investigated patterns of lifetime plaque exposure in the participants by using group-based trajectory modeling (GBTM), an approach that can be used to characterize developmental trajectories. We used a GBTM macro program that modeled the response variable (the OHI-S score) by using the censored normal distribution and fitted three plaque trajectory groups to the data. We assigned each participant automatically to one of these groups through GBTM.[17]

GBTM is based on a class of statistical models called finite mixture models. This type of modeling uses trajectory groups as a statistical device to approximate the unknown distribution of trajectories, which is convenient for summarizing trajectories in distinctive regions of a distribution that otherwise would be of unknown shape. In GBTM, a group is considered to be a collection of people who follow approximately the same developmental trajectory—an approximation of a more complex reality.[18] The number of groups in the model is determined by weighting the formal criteria obtained through maximum likelihood estimation together with explanatory power and usability in analyses. In group-based trajectory analysis, missing data are assumed to be missing at random. For the purposes of our research, we restricted analyses to those participants who had OHI-S data available for at least three time points.

The dental examiners were not aware of participants' socioeconomic statuses (SES) or responses to questionnaires they completed before the examinations. OHI-S plaque assessment was conducted before examining the teeth for caries and periodontal disease.

Adult outcomes. ***Caries and tooth loss.*** When participants were aged 32 years, two examiners (J.M.B. and W.M.T.) whose techniques were calibrated conducted dental examinations for caries and missing teeth; each examiner assessed approximately 50 percent of the participants. The intraclass correlation coefficient for intraexaminer reliability was 0.99 for both examiners, and for interexaminer reliability was 0.99.[19] Before conducting the examinations, the examiners adjusted the paper forms on which they recorded dental examination data to account for teeth that had been missing at the age 26 years assessment.

The examiners assessed the teeth for caries and restorations. They looked at four surfaces for anterior teeth (buccal, lingual, distal and mesial), and five surfaces (buccal, lingual, distal, mesial and occlusal) for posterior teeth (premolars and molars). The examiners diagnosed caries visually with the aid of a dental mouth mirror. They did not dry the teeth before the examination, they used dental explorers to remove plaque only (after the OHI-S assessment), and they did not obtain radiographs. If an examiner could not see a surface because it was covered by calculus or gingival tissue, the examiner excluded the surface from the examination and subsequent analyses.

The examiners estimated the accumulated tooth loss due to caries by observing the presence or absence of each tooth at the time of the examination. They asked the participant the reason for a tooth's absence. In this study, we included only those teeth that had been lost because of caries in the analyses of tooth-loss data.

Periodontal disease. Two examiners (J.M.B. and W.M.T.) conducted periodontal examinations when the participants were aged 32 years. One examiner assessed 495 participants, and the other examiner assessed 437 participants. They measured three sites per tooth (mesiobuccal, buccal and distolingual), and recorded gingival recession (GR) (the distance in millimeters from the cementoenamel junction to the gingival margin) and PD (the distance from the tip of the periodontal probe to the gingival margin) by using a color-coded periodontal probe, which has a rounded 1-mm tip and six alternating black and silver 2-mm bands. The examiners did not repeat periodontal examinations on the participants, but they did conduct replicate examinations on a separate groups of participants to allow for computation of reliability statistics, which we reported previously.[20] Intraclass correlation coefficients for the periodontal measurements pooled for the two examiners were 0.93 for mean GR, 0.68 for mean PD and 0.69 for mean AL. Of the calibration measurements, 99.6 percent of the AL measurements for the intraexaminer and interexaminer comparisons were within 2 mm, meaning that only 0.4 percent of replicated pairs differed by 3 mm or more.[20]

The examiners assessed gingivitis at the

same time they measured periodontal AL levels. They classified teeth that had one or more periodontal sites that bled subsequent to periodontal probing as "bleeding on probing."

SES. During the period that the participants were children (through age 15 years), we based the SES of participants' families on the parents' occupational status. We assigned occupations to one of six categories (for example, 6 = unskilled laborer, 1 = professional) on the basis of the education level and income associated with that occupation in New Zealand census data.[21] In the data analyses described below, the variable "childhood SES" was the average of the highest SES level of either parent, assessed repeatedly from birth through age 15 years. We used this method because measuring SES at a single point early in life does not describe cumulative exposure to low SES.[22] We classified participants as having low (groups 6 and 5), medium (groups 4 and 3) or high (groups 2 and 1) childhood SES. When the participants were adults, we calculated their SES at ages 18, 21, 26 and 32 years by using the same occupation-based method.

Smoking status. Trained interviewers obtained data regarding participants' smoking status when they were aged 32 years by asking the question "Have you smoked for one month or more of the previous 12 months?" We classified those who answered negatively as "nonsmokers" (n = 635, 69.7 percent) and those who answered positively as "smokers" (n = 276, 30.3 percent). The results of previous analyses from the DMHDS confirmed the validity of the self-report smoking measures used through measurement of cotinine levels in saliva.[23]

Education level. Trained interviewers assessed participants' levels of formal education when they were aged 32 years. We determined whether they had no formal educational achievements (n = 157, 17.2 percent), a New Zealand School Certificate (passed 11th grade) (n = 147, 16.1 percent), high school diploma or equivalent (n = 377, 41.4 percent) or a bachelor's degree or higher (n = 228, 25.0 percent). Education level data were not available for two participants.

Oral self-care. When the participants were aged 32 years, they answered self-reported questionnaires that included the question, "When do you usually brush your teeth?" The response options were "more than once a day," "once a day," "not every day" and "less than once a week or never." We dichotomized the respondents to this questions into two groups: those who indicated that they brushed at least once a day (n = 823, 90.3 percent) and those who brushed less than once per day (n = 85, 9.3 percent). Toothbrushing data were not available for three participants.

We determined the participants' flossing frequency when they were aged 32 years by asking, "When do you use dental floss?" The response options were never, rarely, sometimes or every day. We trichotomized respondents into those who never floss (n = 197, 21.6 percent), those who floss but not every day (n = 308, 33.8 percent) and those who floss daily (n = 403, 44.2 percent). Flossing data were not available for three participants.

Dental visits. Before the dental examination, we asked participants to estimate the number of months since their last dental visit. We recorded if a participant could not remember when his or her last dental visit was or indicated that she or he had never been to a dentist. We also asked participants, "What is your usual reason for visiting the dentist? Would it be for a problem or a checkup?" We classified participants who visited the dentist only because of problems as "episodic dental visitors" (n = 485, 53.2 percent), and those who normally attended for checkups as "routine" (n = 424, 46.5 percent). Two participants did not respond to this item.

Finally, we asked participants whether they had any known medical condition that would contraindicate a periodontal examination.

Data analysis. We used surface-level dental examination data to compute a modified DMFS index,[24] at age 32 years. For a tooth in which four or five surfaces were identified as being carious at the assessment before the extraction of the tooth, we assigned the tooth that number of carious surfaces instead. We adjusted DMFS scores for teeth that were missing because of caries when participants were aged 26 years according to their surface-level data at age 18 years, and we adjusted teeth missing because of caries only when participants were aged 32 years according to their surface status at age 26 years.[24] We included third molars in the analyses, as well as teeth with crowns.

We used the zero-inflated negative binomial model[25] to fit models for DMFS data. We used the zero-inflated Poisson to model tooth-loss severity (that is, number of teeth lost) and the extent of periodontal disease (that is, the percentage of affected sites), and we used logistic regression to fit models for tooth loss and periodontal disease prevalence (prevalence of one or more sites with ≥ 4 mm of periodontal AL). We used linear regression for modeling the percentage of teeth that had bleeding on probing. We used GBTM to model plaque trajectories by using plaque data collected from examinations at ages 5, 9, 15, 18, 26 and 32 years.

TABLE 1

Mean plaque scores at each time point.

ASSESSMENT	NO. OF PARTICIPANTS ASSESSED	MEAN PLAQUE SCORE (SD*)	PLAQUE TRAJECTORY GROUP (MEAN PLAQUE SCORE [SD])			NO. OF INCLUDED PARTICIPANTS
			Low	Medium	High	
Age 5 Years	922	0.95 (0.45)	0.84 (0.40)	0.97 (0.46)	1.13 (0.48)	876
Age 9 Years	661	0.71 (0.32)	0.60 (0.29)	0.74 (0.30)	0.92 (0.33)	639
Age 15 Years	756	1.11 (0.48)	0.85 (0.35)	1.19 (0.42)	1.72 (0.46)	753
Age 18 Years	864	0.75 (0.46)	0.47 (0.31)	0.82 (0.34)	1.39 (0.48)	859
Age 26 Years	926	0.87 (0.54)	0.47 (0.30)	0.99 (0.39)	1.71 (0.50)	908
Age 32 Years	929	0.76 (0.53)	0.41 (0.29)	0.85 (0.41)	1.62 (0.53)	908
At One or More Time Points	1,024†	0.87 (0.32)	NA‡	NA	NA	NA
At Three or More Time Points	953§	0.87 (0.31)	NA	NA	NA	NA
At Three or More Time Points, Including at Age 32 Years	911¶	0.86 (0.30)	NA	NA	NA	NA

* SD: Standard deviation.
† Total number of participants whose plaque levels were assessed at one or more time points.
‡ NA: Not applicable.
§ Total number of participants whose plaque levels were assessed at three or more time points.
¶ Total number of participants whose plaque levels were assessed at three or more time points, including age 32 years (for outcome variables).

We did not use the Bonferroni adjustment in our analyses, despite the fact that we made multiple comparisons. The Bonferroni adjustment increases the likelihood of type II error, which means that important differences may be deemed to be nonsignificant. Instead, we describe the tests of significance undertaken for each comparison we made and report effect sizes for each analysis, so that readers can compare effect sizes and make their own judgments as to the validity of the findings. We believe this was an appropriate way of addressing questions arising from making multiple comparisons.[26-28]

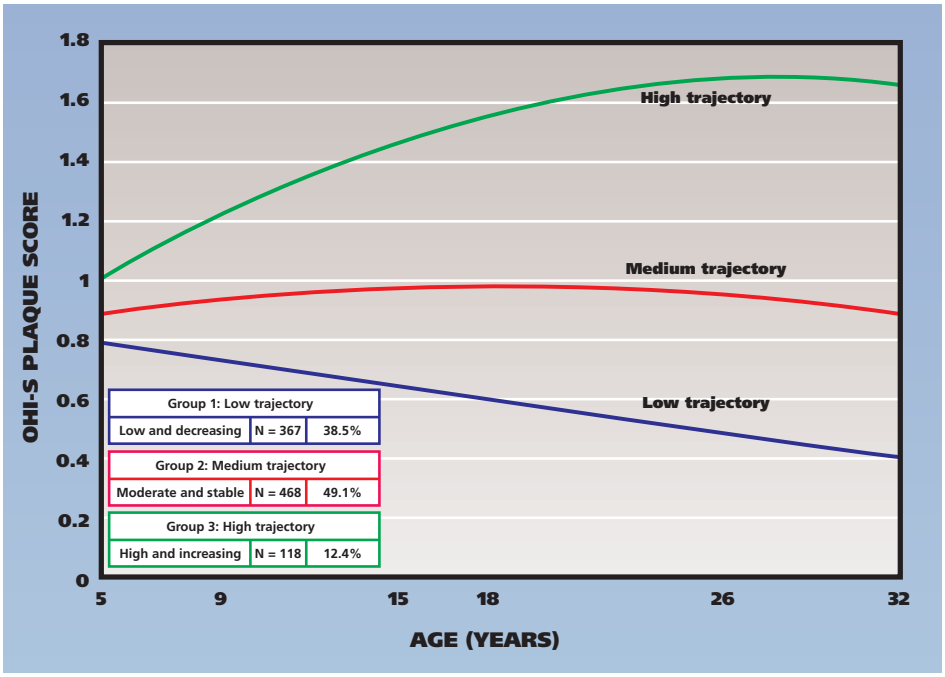

Figure 1. Plaque trajectory group plots. Simplified Oral Hygiene Index (OHI-S) plaque scores, according to age.

RESULTS

By age 32 years, 1,015 of the original participants were alive, and we assessed 972 (95.8 percent). Dental examination data for participants at age 32 years were available for 932 participants, including two completely edentulous participants and three participants who had natural teeth in only one or two groups. Plaque data were available for 1,024 participants at one

TABLE 2

Participants' oral hygiene practices at age 32 years, according to sex, childhood socioeconomic status, education level and smoking status.*†

FACTOR	TOOTHBRUSHING FREQUENCY AT AGE 32 YEARS (NO. [%])		DENTAL FLOSSING FREQUENCY AT AGE 32 YEARS (NO. [%])			DENTAL VISITING PATTERN AT AGE 32 YEARS (NO. [%])	
	Less Than Once Per Day	At Least Once Per Day	Never	Not Every Day	Daily	Routine	Episodic
Sex							
Female	16 (3.6)	431 (96.4)	68 (15.2)	140 (31.3)	239 (53.5)	238 (53.0)	211 (47.0)
Male	69 (15.0)	392 (85.0)	129 (28.0)	168 (36.4)	164 (35.6)	186 (40.4)	274 (59.6)
Childhood Socioeconomic Status							
High	7 (4.7)	142 (95.3)	22 (14.8)	45 (30.2)	82 (55.0)	97 (65.1)	52 (34.9)
Medium	48 (8.3)	531 (91.7)	124 (21.4)	210 (36.3)	245 (42.3)	262 (45.2)	318 (54.8)
Low	30 (17.1)	145 (82.9)	50 (28.6)	51 (29.1)	74 (42.3)‡	64 (36.6)	111 (63.4)
Education Level							
No formal educational achievements	37 (23.6)	120 (76.4)	60 (38.2)	46 (29.3)	51 (32.5)	33 (21.2)	123 (78.8)
New Zealand School Certificate	15 (10.3)	130 (89.7)	32 (22.1)	54 (37.2)	59 (40.7)	49 (33.3)	98 (66.7)
High school diploma or equivalent	29 (7.7)	347 (92.3)	80 (21.3)	132 (35.1)	164 (43.6)	190 (50.4)	187 (49.6)
Bachelor's degree or higher	4 (1.8)	224 (98.2)	25 (11.0)	74 (32.5)	129 (56.6)	152 (67.0)	75 (33.0)
Smoking Status							
Nonsmoker	38 (6.0)	596 (94.0)	117 (18.5)	209 (33.0)	308 (48.6)	354 (55.8)	281 (44.3)
Smoker	47 (17.2)	227 (82.9)	80 (29.2)	99 (36.1)	95 (34.7)	70 (25.6)	204 (74.5)

* All associations are statistically significant to $P < .001$ unless otherwise indicated.
† Data missing for a small number of participants were excluded from the table.
‡ $P = .006$.

or more time points and for 953 participants at three or more time points. Some of the 911 participants whose plaque levels were assessed at three or more time points also were assessed for caries at age 32 years, and 897 were assessed for periodontal disease (Table 1).

The mean plaque scores averaged about 0.87 across all the assessments (Table 1). Mean plaque scores were the highest when the participants were 15 years old. We included in the analysis 953 participants for whom data were available from at least three time points. Group-based trajectory analysis of OHI-S scores (the mean of the six individual scores) across time identified three distinct plaque trajectory groups across the participants' lifetimes (Figure 1). We selected a three-group model, as it provided a better fit than a two-group model, and it was more parsimonious than a four-group model. We used the Bayesian information criterion (BIC) as the criterion for model selection. The BIC value improved by only 0.1 percent for a four-group model over a three-group model, and the fourth group in the four-group model was similar to the largest group in the three-group model. Thus, we selected the three-group model.

The mean OHI-S plaque scores for each of these plaque trajectory groups were substantially different at each time point (Table 1, Figure 1). Group 1 had low levels of plaque (n = 367, 38.5 percent), group 2 had medium levels of plaque (n = 468, 49.1 percent) and group 3 had high levels of plaque (n = 118, 12.4 percent). We excluded from further analysis participants for whom data at age 32 years were unavailable. This left 357 participants (39.2 percent) in the low-trajectory group, 450 participants (49.4 percent) in the medium-trajectory group and 104 participants (11.4 percent) in the high-trajectory group. The mean scores by group at each time point are given in Table 1, together with their standard deviations.

We also investigated the differences in oral hygiene practices (toothbrushing and flossing) and dental visiting patterns, according to sex, childhood SES, education level and smoking status. We found that proportionally more male participants, smokers, those with low childhood SES and those with low education levels brushed their teeth less than once per day, flossed occasionally or never, and visited the dentist on an episodic basis than did female participants, nonsmokers, those with high childhood SES and those with high education levels. These differences are shown in Table 2 and were statistically significant.

There were almost three times as many male participants as female participants in the high-plaque-trajectory group (Table 3). Those who had low childhood SES or low education levels also were more likely to have had high levels of lifetime plaque experience than those who had high childhood SES. The same was true of smokers and episodic dental visitors. Poor oral hygiene practices (infrequent toothbrushing and flossing) also were associated strongly with being in the high-plaque-trajectory group.

Participants who had low childhood SES brushed their teeth less often at age 32 years than did those who had high childhood SES. At age 32 years, 30 of the 175 participants who had low childhood SES (17.1 percent) did not brush their teeth every day, and 48 of the 579 participants who had medium childhood SES (8.3 percent) and seven of the 149 participants who had high childhood SES (4.7 percent) reported that they did not brush their teeth every day ($\chi^2 = 17.0$, $P < .001$).

TABLE 3

Participants' lifetime plaque trajectories at age 32 years.

FACTOR	PLAQUE TRAJECTORY GROUP (NO. [%])			Total (No. [%])
	Low	Medium	High*	
TOTAL†‡	**357 (39.2)**	**450 (49.4)**	**104 (11.4)**	**911 (89.8)**
Sex				
Female	224 (49.9)	200 (44.5)	25 (5.6)	449 (49.3)
Male	133 (28.8)	250 (54.1)	79 (17.1)	462 (50.7)
Childhood Socioeconomic Status				
High	77 (51.7)	66 (44.3)	6 (4.0)	149 (16.4)
Medium	226 (39.0)	292 (50.3)	62 (10.7)	580 (63.7)
Low	52 (29.4)	89 (50.3)	36 (20.3)	177 (19.4)
Education Level				
No formal educational achievements	30 (19.1)	80 (51.0)	47 (29.9)	157 (17.2)
New Zealand School Certificate	38 (25.9)	90 (61.2)	19 (12.9)	147 (16.1)
High school diploma or equivalent	161 (42.7)	186 (49.3)	30 (8.0)	377 (41.4)
Bachelor's degree or higher	128 (56.1)	92 (40.4)	8 (3.5)	228 (25.0)
Smoking Status				
Smoker	73 (26.5)	139 (50.4)	64 (23.2)	276 (30.3)
Nonsmoker	284 (44.7)	311 (49.0)	40 (6.3)	635 (69.7)
Toothbrushing Frequency				
Less than once per day	12 (14.1)	35 (41.2)	38 (44.7)	85 (9.3)
At least once per day	345 (41.9)	412 (50.1)	66 (8.0)	823 (90.3)
Flossing Frequency				
Never	50 (25.4)	95 (48.2)	52 (26.4)	197 (21.6)
Not every day	116 (37.7)	167 (54.2)	25 (8.1)	308 (33.8)
Daily	191 (47.4)	185 (45.9)	27 (6.7)	403 (44.2)
Dental Visiting Pattern				
Routine	208 (49.1)	191 (45.0)	25 (5.9)	424 (46.5)
Episodic	149 (30.7)	258 (53.2)	78 (16.1)	485 (53.2)

* All associations significant to $P < .001$.
† Row percentage.
‡ Data missing for a small number of participants were excluded from the table.

The outcomes of multivariate modeling are presented in Table 4 and Figures 2 through 5 (page 423). Strong associations for the extent of caries experience (that is, DMFS) for the prevalence of unrestored caries and for the prevalence and severity of caries-associated tooth loss at age 32 years existed according to plaque trajectory group before and after controlling for putative confounding variables (for example, mean childhood SES, sex and dental visiting pattern).

TABLE 4

Models for dental health–related outcomes at age 32 years, according to plaque trajectory group, after controlling for putative confounding variables.*

OUTCOME	PLAQUE TRAJECTORY GROUP						NO. OF PARTICIPANTS
	Low (ref.)		Medium		High		
Caries-Related Measures							
Decayed, missing and filled teeth							911
Mean no. (SD†)	14.1 (11.9)		17.8 (15.1)		22.3 (16.5)		
IR‡ (95% CI§)¶	1.0		1.2 (1.1-1.4)		1.4 (1.2-1.7)		
Participants with one or more decayed tooth surfaces							911
No. (%)	144 (40.3)		249 (55.3)		83 (79.8)		
OR# (95% CI)**	1.0		1.4 (1.1-1.9)		3.4 (2.0-6.0)		
Tooth loss due to caries							911
Mean no. teeth lost due to caries (SD)	0.2 (0.7)		0.7 (1.5)		2.2 (4.8)		
IR (95% CI)††	1.0		1.8 (1.3-2.6)		4.0 (2.9-5.8)		
Participants with one or more teeth lost owing to caries							911
No. (%)	38 (10.6)		126 (28.0)		47 (45.2)		
OR (95% CI)**	1.0		2.8 (1.8-4.2)		4.8 (2.8-8.4)		
Periodontal Disease–Related Measures	**Nonsmokers**	**Smokers**	**Nonsmokers**	**Smokers**	**Nonsmokers**	**Smokers**	
Teeth with bleeding on probing							897
Mean percentage (SD)	5.7 (5.4)	7.1 (6.6)	7.9 (6.1)	10.4 (7.8)	15.3 (8.0)	16.5 (8.7)	
IR (95% CI)§§	1.0	1.1 (1.0-1.2)	1.2 (1.2-1.4)	1.5 (1.4-1.6)	2.1 (2.0-2.4)	2.1 (1.9-2.3)	
Sites with at least 4 millimeters of periodontal AL¶¶							897
Mean percentage (SD)	0.7 (2.7)	1.8 (4.9)	0.9 (3.2)	3.8 (6.0)	2.7 (8.1)	9.6 (15.3)	
IR (95% CI)§§	1.0	1.9 (1.5-2.3)	1.0 (0.9-1.2)	3.7 (3.2-4.4)	3.0 (2.4-3.7)	8.9 (7.5-10.4)	
Participants with one or more sites with at least 4 mm of periodontal AL							897
No. (%)	44 (15.6)	20 (27.8)	63 (20.7)	78 (57.4)	17 (42.5)	41 (65.1)	
OR (95% CI)**	1.0	1.6 (0.9-2.7)	1.1 (0.7-1.9)	3.4 (2.2-5.5)	2.7 (1.1-6.2)	5.8 (3.1-10.8)	

* Caries-related analyses controlled for childhood socioeconomic status, sex and dental visiting pattern. Periodontal disease–related variables also controlled for smoking status and interaction between smoking status and plaque trajectories.
† SD: Standard deviation.
‡ IR: Incidence risk ratio.
§ CI: Confidence interval.
¶ Zero-inflated negative binomial model was used.
# OR: Odds ratio.
** Logistic regression was used.
†† Zero-inflated Poisson regression model was used.
§§ Poisson regression was used.
¶¶ AL: Attachment loss.

For example, after we controlled for potential confounding variables, participants in the high-plaque-trajectory group had a 40 percent greater experience of caries (Figure 2), were 3.4 times more likely to have one or more decayed surfaces (Figure 3), had lost 4.0 times more teeth owing to caries (Figure 4) and were 4.8 times more likely to have lost a tooth owing to caries (Figure 5) than were those in the low-plaque-trajectory group. Participants in the medium-plaque trajectory group also were significantly worse off than were those in the low-plaque-trajectory group.

We found the same patterns for associations

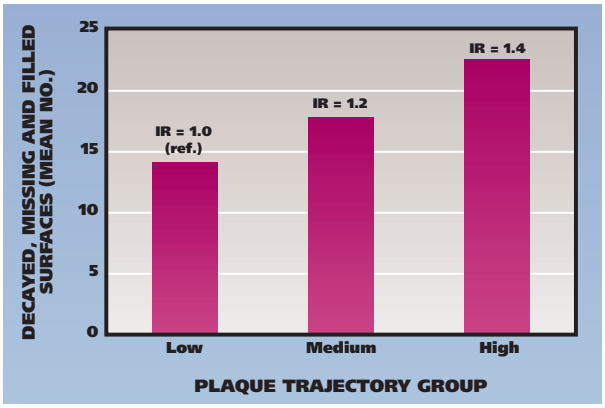

Figure 2. Mean number of decayed, missing and filled surfaces at age 32 years, according to plaque trajectory group. IR: Incidence risk ratio.

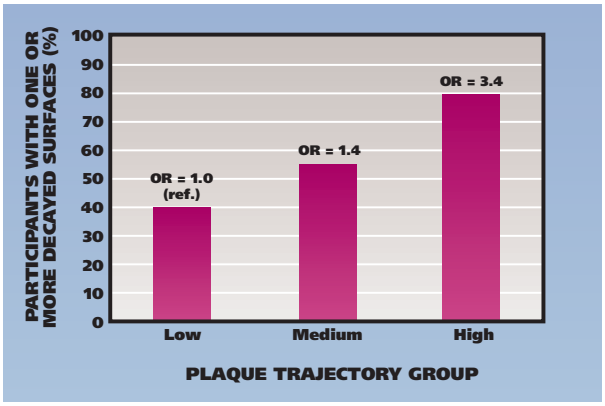

Figure 3. Percentage of participants with one or more decayed surfaces at age 32 years, according to plaque trajectory group. OR: Odds ratio.

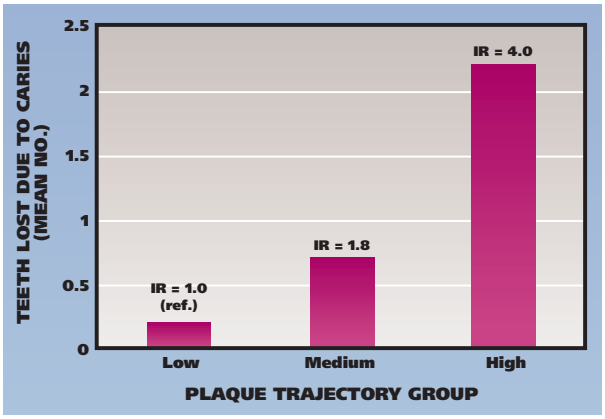

Figure 4. Mean number of teeth lost owing to caries at age 32 years, according to plaque trajectory group. IR: Incidence risk ratio.

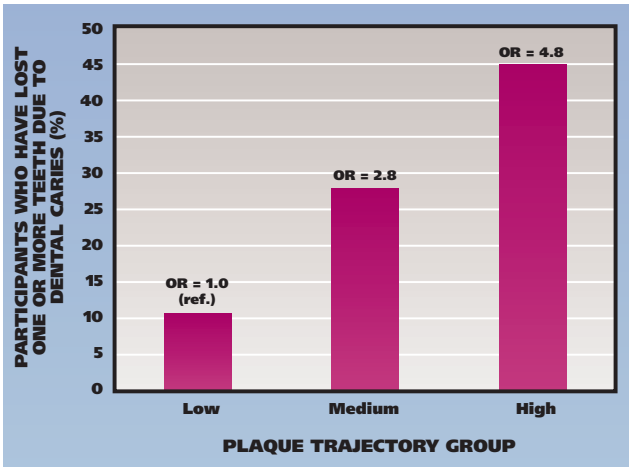

Figure 5. Percentage of participants who have lost one or more teeth owing to caries at age 32 years, according to plaque trajectory group. OR: Odds ratio.

of periodontal disease measures according to plaque trajectory group. Participants in the high-plaque-trajectory group were more likely to have bleeding on probing, to have one or more sites with at least 4 mm of periodontal AL and to have a greater proportion of sites with at least 4 mm of periodontal AL. Furthermore, the results of multivariate modeling revealed effect modification between smoking and plaque trajectory group for the risk of having one or more sites with at least 4 mm of periodontal AL and for the extent of having at least 4 mm of periodontal AL, but not for bleeding on probing. Smokers did not have significantly different rates of bleeding on probing from nonsmokers in any of the plaque trajectory groups; however, smokers had a substantially greater extent of having at least 4 mm of periodontal AL in the low-, medium- and high-plaque-trajectory groups than did nonsmokers, and smokers in the medium-plaque-trajectory group had a greater risk of having one or more sites with at least 4 mm of periodontal AL than did nonsmokers. The difference in risk between smokers and nonsmokers for having one or more sites with at least 4 mm of periodontal AL did not differ significantly in the low- or high-plaque-trajectory groups (Table 4, Figures 6 through 8).

DISCUSSION

To our knowledge, this is the first study to report on longitudinal dental plaque data from childhood and into adulthood. Lifetime exposure to plaque may be the key risk factor in cumulative dental diseases, such as caries experience and tooth loss due to caries, as well as a major risk factor for the eventual development of other conditions such as gingivitis and periodontal disease. The findings from our study support these propositions.

The limitations of our study included the fact that the data on caries experience may be underestimated, because the participants' teeth

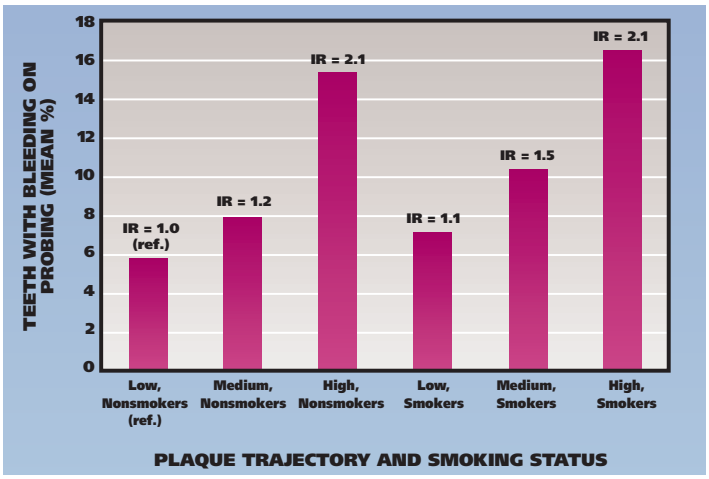

Figure 6. Mean percentage of teeth with bleeding on probing at age 32 years, according to plaque trajectory group and smoking status. IR: Incidence risk ratio.

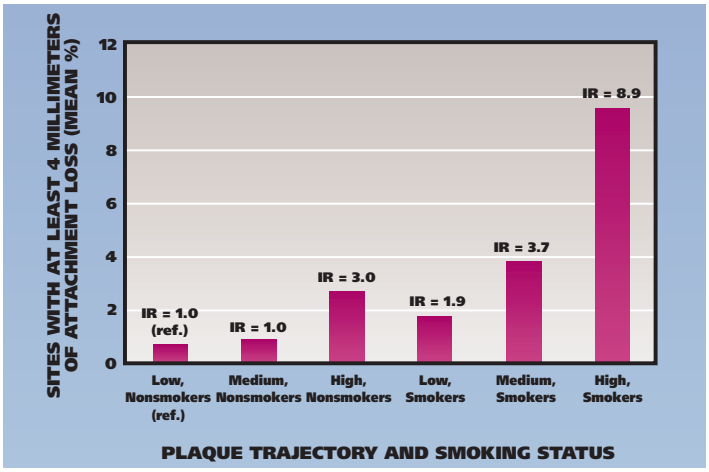

Figure 7. Mean percentage of sites with at least 4 millimeters of attachment loss at age 32 years, according to plaque trajectory group and smoking status. IR: Incidence risk ratio.

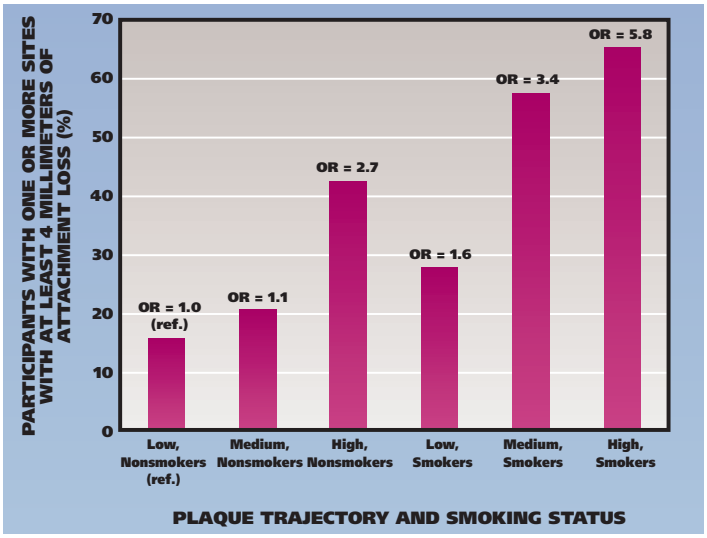

Figure 8. Percentage of participants with one or more sites with at least 4 millimeters of attachment loss at age 32 years, according to plaque trajectory group and smoking status. OR: Odds ratio.

were not dried before the caries examination, and radiographs were not obtained. A diverse collection of bacteria exists in plaque; however, the investigation of specific bacteria that may cause caries was outside the scope of our analysis. We restricted our analyses to the OHI-S data collected in the DMHDS (that is, the extent to which the surfaces of index teeth were covered by plaque). Another limitation may have been that different examiners were involved at different assessment points in the study. Although this situation is not ideal, it was unavoidable in a study of this length and magnitude. We observed some variation in mean plaque scores between ages. The variation across time could be due either to periods of poor self-care (for example, a greater tendency to neglect oral hygiene during the early childhood and teenage years) or to interexaminer variation in measuring plaque between ages. However, the effect of any such variation on the outcomes of the analyses should be minimized by the use of the group-based trajectory analysis data. We did not use plaque-disclosing solution in this study, so measurements of plaque in our study likely were underestimated.

In addition, there may be limitations to the applicability of findings from a New Zealand population to other populations throughout the world. However, as with any other single-cohort longitudinal study, the DMHDS produced data that are bounded by time and place. All populations experience plaque-related dental diseases, and the development of these chronic disorders across time most likely would be similar among various populations.

We found that smokers had particularly poor oral hygiene and were likely to have poorer self-care in general than were nonsmokers, and they were more likely to have poor plaque control. Similar findings have been reported in the dental literature, albeit with an unrepresentative convenience sample.[29] Male participants were as likely as smokers to have poor lifetime oral hygiene. We were unable to explain the sex difference, except that male participants may be less likely to be aware of their oral hygiene status[7] or may place less importance on it than female participants, and hence be less likely to maintain good oral hygiene.

Gingivitis (as measured by bleeding on probing) did not appear to be affected by

interaction between smoking status and oral hygiene. We observed similar levels of bleeding on probing in smokers and nonsmokers within each plaque trajectory group (Table 4). The finding of other studies have shown that smokers tend to have less bleeding on probing,[30,31] but we found no direct evidence of that in our study.

One finding in our study was an interaction between smoking status and oral hygiene in the multivariate models for periodontal disease. Nonsmokers in the high-plaque-trajectory group were more likely to have one or more sites with at least 4 mm of periodontal AL than the reference group (nonsmokers in the low-plaque-trajectory group). Smokers in the low-plaque-trajectory group were 1.6 times more likely to have one or more sites with at least 4 mm of periodontal AL than the reference group, but, when combined with smokers in the high-plaque-trajectory group, they had 5.8 times greater odds of having one or more sites with at least 4 mm of periodontal AL.

We observed similar findings regarding the extent of periodontal disease (that is, percentage of sites with at least 4 mm of periodontal AL). Smokers in the low-plaque-trajectory group had 1.9 times more sites with AL than did nonsmokers in the low-plaque-trajectory group but smokers in the high-plaque-trajectory group had 8.9 times more sites with AL. Nonsmokers in the high-plaque-trajectory group had a greater incidence of AL, but this was only 3.0 times greater than that of the reference group. These findings suggest that poor oral hygiene and smoking have a synergistic effect on a person's risk of developing periodontal disease and affects the severity of that disease.

There is no reason that plaque control should be a long-term problem for any able-bodied person. Plaque may be removed by mechanical debridement as a part of routine self-care or by a dental care professional. In addition, substantial reduction in plaque development may be obtained through the use of an appropriate mouthrinse, such as those containing chlorhexidine, or toothpaste, such as those containing triclosan. It is unlikely that the fact that people who have low SES tend to have greater levels of plaque across the long term is due to any systemic cause. Instead, it is likely to be due to differences in self-care behavior patterns among these plaque trajectory groups and differences in the affordability of oral hygiene aids. People who have high SES also may be less likely to neglect their oral hygiene. They also may be more likely to be routine dental visitors than are people with low SES[32] and thus are likely to be more exposed to better oral hygiene reinforcement than are episodic dental visitors. This is an example of a socioeconomic inequality in health, in that those who have the poorest oral hygiene and the poorest prognosis for good oral health earn the lowest income and thus are least able to afford dental care in a private-sector dental health care environment.

CONCLUSIONS

People follow identifiable trajectories of oral hygiene through their lives. These trajectories are, in part, determined by social and behavioral factors. Dental plaque trajectories are associated strongly with future oral health.

Improving oral health requires emphasizing long-term self-care, as well as providing broad public health and health promotion measures that advance and support oral self-care. ■

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