# Do Root Lesions Tend to Develop in the Same People Who Develop Coronal Lesions?

# James D. Beck, PhD; Claude W. Drake, DDS, MPH

# Abstract

Objectives: The three purposes of this study are to: (1) describe the relationship between the prevalence of coronal caries and root caries; (2) describe the relationship between the three-year incidence of coronal caries and root caries; and (3) if the two conditions are associated, develop a multiple regression model that identifies characteristics distinguishing people who had increments of both root caries and coronal caries from people who had increments of either coronal caries or root caries, or who had no new caries. Methods: Dental examinations and interviews were conducted in the homes of a randomly selected, stratified sample of people over the age of 65 years in five North Carolina counties. The relationships between coronal and root D and DF were analyzed through contingency table analyses, and ordinal logistic regression was used to identify characteristics that differentiated people who had both coronal and root D over the three years from people who had either coronal or root D and people who had no new disease. Results: Evidence of root and coronal caries in whites was much more likely to be in the form of fillings, while for blacks, it was more likely to be in the form of untreated decay. Prevalence rates of coronal and root D and DF were significantly associated for both blacks and whites. Incidence rates based on DF indicated that root and coronal caries were not associated in whites, but were associated in blacks. People more likely to experience both types of caries had more gingival recession at baseline, greater average attachment loss over the three years, and lactobacilli at baseline. In addition, the presence of Porphymonas gingivalis at three years was important for whites. Conclusions: It appears that coronal and root caries do tend to appear together in the same individuals, but fillings attenuate that relationship. The impact of dental treatment on the epidemiology of dental caries appears to be considerable and calls into question whether the F component of the caries index is related to disease as defined by epidemiologic criteria. [J Public Health Dent 1997:57(2):82-8]

Key Words: coronal caries, root caries, incidence, old age, periodontitis, lactobacilli, epidemiology.

The questions of whether root and coronal caries are the same disease, whether they have a similar etiology, or whether people with high levels of coronal caries are at higher risk of root caries have led to investigations of the relationship between the two conditions. Sumney et al. (1) found that most teeth with root caries had no coronal decay or fillings, indicating no relationship. Most studies, however, have found positive relationships between root and coronal caries experience. Schamschula et al. (2) showed a correlation of 0.34 between the two conditions in a primitive population, Banting et al. (3) found that people with root DF had more DF coronal surfaces, while Burt et al. (4) found a correlation between coronal DMFT and root caries. Beck et al. (5) found that the relative risk of having DF root caries was 2.6 if DF coronal caries was present. Vehkalahti et al. (6) found stronger associations than were found in other studies between the D components of coronal caries and root caries, indicating that including treated caries weakened the relationship. Fure and Zickert (7) also found the associations between unfilled coronal caries and unfilled root caries were stronger than when fillings were included. Locker et al. (8) found that the number of decayed coronal surfaces was related to the presence of one or more decayed root surfaces in a multivariable model and there was a weaker relationship when fillings were included.

All of the associations described above are based on data from crosssectional studies and cannot address the time sequence of the two conditions. Thus, it is possible that while coronal caries and root caries are associated, they are different entities and do not tend to occur in the same person at the same time. The only populationbased study to investigate the relationship between the incidence of root caries and coronal caries found that coronal decay at baseline was a significant predictor of root decay at 18 months in a multivariable model, and that people who had an increment of coronal caries had 1.78 times the odds of having a root caries increment (9). However, by 36 months there was not a statistically significant association between the people who had coronal caries increments and those who had new root caries (10).

The three purposes of this study are to describe the relationship between the prevalence of coronal caries and root caries; describe the relationship between the three-year incidence of coronal caries and root caries; and develop a multiple regression model that identifies characteristics distinguishing people who had increments of both root caries and coronal caries from people who had increments of either coronal caries or root caries, or who had no new caries.

Send correspondence and reprint requests to Dr. Beck, Kenan Professor and Chair, Department of Dental Ecology, CB# 7450, University of North Carolina, Chapel Hill, NC 27599. Internet: james\_beck@unc.edu. Dr. Drake is with the Department of Diagnostic Sciences, University of North Carolina at Chapel Hill. This project was supported by NIDR grant #R01-DE-08660. Participants in this study were selected from the Duke Established Populations for Epidemiologic Studies of the Elderly, which was performed pursuant to NIA contract #N01-AG-1-2102. Manuscript received: 7/20/95; returned to authors for revision: 12/22/95; accepted for publication: 2/19/96.

#### Methods

Sample Selection. Data for this paper were obtained from the Piedmont 65+ Dental Study, which is an ongoing longitudinal oral health survey of noninstitutionalized older adults initiated by investigators at the University of North Carolina School of Dentistry. The sample was part of a parent study conducted by the Duke University Center for Aging on the health status and functioning of older adults, with emphasis on those who had been underrepresented in previous studies (11). Thus, the sample was stratified disproportionately by race. This parent study consisted of a cohort of approximately 4,100 North Carolina residents over the age of 65 years residing in five contiguous counties. The Piedmont 65+ Dental Study involved a randomly selected subsample from this parent population following the same stratification process.

Dental survey examinations were completed at baseline, 18 months, and three years. This paper used data from the baseline examination of 448 blacks and 362 whites who had at least one natural tooth and from the three-year examination of 430 dentate subjects consisting of 221 blacks and 209 whites. Details regarding the study design for the Piedmont 65+ Dental Study have been reported previously in Graves et al. (12).

Data Collection. Clinical data, including coronal and root surface caries, were collected by five examiner teams, each composed of a dentist-examiner and a recorder who collected data at baseline and also examined and interviewed the same subjects at the three-year examinations. The examiners were standardized during a week-long training session before oral examinations. Replicate examinations were conducted on 50 subjects participating in the study. Each examiner was paired with the four other examiners and each pair examined a common subset of five subjects.

The interviews and examinations were conducted in the subjects' own homes. Examiners wore a headlamp, used a standard plane surface mirror, a #23 explorer, and periodontal probes in the clinical examinations. The recorder entered the clinical findings in code directly into a laptop computer.

Oral Examination Measures. Coronal caries examinations were conducted in accordance with the criteria described by Radike (13), e.g., only obvious caries was recorded. Root surface caries was diagnosed using the visual-tactile criteria as described by Katz (14) and modified to fit the criteria developed for the National Survey of Oral Health in US Employed Adults and Seniors, 1985–86 (15).

The clinical diagnosis of root caries was based on the visual observation of a discrete, well-defined, and discolored cavitation on the root surface and the tactile finding of softness upon probing with light pressure. Hardened discolored root surfaces due to arrested caries were not scored. The diagnosis was determined without use of radiographs, the teeth were not dried, and calculus was not removed prior to examination. Root caries was measured supragingivally on cementum and scored separately from cervical abrasion. When both coronal and root surfaces were affected by a single carious lesion, root caries, rather than coronal caries, was recorded if at least one-half of the lesion or restoration extended apically to the cementoenamel junction. A multiple-surface lesion or restoration was recorded if the lesion or restoration extended more than one-third of the way across the adjacent surface.

The criteria for the recording of caries distinguished between abrasion lesions and root lesions, with cervical abrasion being defined as a wedgeshaped defect, softly angled in the early stages and sharply angled in the later stages, with highly polished and exposed dentin (16). Root restorations due to caries and root restorations due to abrasion or erosion were distinguished according to the criteria established by Hand et al. (17). If the examiner could not distinguish between filled abrasions and filled root caries, the restorations were recorded as filled root caries.

Gingival recession was recorded on each of the four tooth surfaces. These surfaces were considered at risk for root caries if there was at least 1 mm of visible root surface between the gingival crest and the cemento-enamel junction. All teeth including third molars were evaluated, since it has been suggested that in the elderly the third molar may be of functional importance since it often replaces another molar as a result of tooth movements after extractions.

Caries adjacent to restorations was

individually recorded; however, when a surface was both decayed and filled, it was recorded as decayed and filled and analyzed as decayed. Broken restorations and lost restorations were recorded as filled surfaces. Remaining roots without clinical crowns or teeth with less than one-fourth of the clinical crown were scored as root fragments and included as coronal caries. Teeth that were lost between the baseline and three-year examinations were not included in the calculations since it is difficult to ascertain the cause of tooth loss. The number of missing teeth and the presence of fixed and removable dentures were recorded.

DFS increment for root and coronal caries was determined on the basis of a surface-by-surface comparison between baseline and the three-year follow-up examination. Net caries increments were calculated for each person by adding the number of surfaces with new caries or fillings on previously sound surfaces to the number of surfaces with new recurrent caries on previously filled surfaces, and then subtracting from this sum the number of surfaces with reversals due to examiner error. Reversals were defined as surfaces that were recorded as decayed or filled at the baseline examination, but were recorded as sound at the three-year follow-up. This finding was considered to be examiner error as recommended by WHO (18), because the diagnostic criteria for this study resulted in remineralization being an unlikely event. Caries attack rates for coronal and root caries were calculated by dividing the net caries increment by the number of surfaces at risk for caries, expressed as the proportion of new decayed and filled root surfaces per 100 surfaces at risk (for root caries, those surfaces with recession). A similar approach, the Root Caries Index (RCI), introduced by Katz (14) has been used widely in epidemiologic studies of root caries.

Periodontal indicators included measures of gingival recession, probing pocket depth and attachment loss; however, calculus and bleeding assessments were not used because almost everyone had both conditions. Measurements were taken at two sites on each remaining tooth (midbuccal and mesiobuccal sites) using an NIDR probe (15) and were rounded down to the next lowest whole millimeter. Attachment level measures were computed from the gingival recession and probing depth measures. Attachment loss was computed by subtracting the attachment level at three years from the baseline attachment level on a siteby-site basis and summing for the individual.

Samples of paraffin-stimulated whole saliva were collected for three minutes immediately before the baseline clinical examination, and the salivary flow rate was recorded as milliliters of saliva collected per minute. The salivary buffer capacity (final pH) was determined using the Dentobuff® system designed by Orion Diagnostica of Sweden (19). A value for the secretion rate that fell below 1 ml per min was considered a risk value. The buffer capacity of stimulated saliva was considered low at a final pH of  $\leq$ 4.5 using the Dentobuff indicator scale.

To detect the cariogenic microorganisms mutans streptococci and lactobacilli, salivary counts for these microbes were estimated by culturing the stimulated whole saliva on selective media (20,21). Commercially available testing kits were used to quantify the salivary concentration of mutans streptococci (Cariescreen) and lactobacilli (Bactotest). The microorganisms' levels were dichotomized and registered as high at  $\geq 10^5$  CFU/ml stimulated saliva and not high at lower levels, using the standard charts supplied by the manufacturers. The BANA assay (22) was used for detecting the presence of three periodontal pathogens: Treponema denticola, Porphyromonas gingivalis, and Bacteroides forsythus. Separate immunofluorescent assays for Actinobacillus actinomycetemcomitans, Prevotella intermedius, and Porphyromonas gingivalis were carried out from subgingival plaque samples as previously described (23,24). For both assays, the results were dichotomized as present or absent.

Analysis. Estimates of population proportions and rates and their standard errors were computed for the fivecounty area in North Carolina from which the study participants were selected using PC SUDAAN (25). This software used sampling weights to control for oversampling as well as accounting for the clustering in the sampling design. Chi-square tests with one degree of freedom were calculated using PC SUDAAN (25) for the associations between coronal caries and root caries that are summarized in Table 2.

The analytic methods used to develop logistic models for the incidence of disease have been described previously by Koch and Beck (26). A variety of modeling procedures were used to identify factors and predictors associated with disease progression. Two dichotomous outcome variables were used in the study, whether or not people developed coronal caries and whether or not people developed root caries over the three-year period. The outcome variable used in the logistic regression was ordinal and was created as follows: people who developed both coronal and root caries over the three-year period were given a score of "0," people who developed either coronal caries or root caries were scored "1," and people who had no caries increment over the three years were given a score of "2." Initially, items theoretically associated or that had been previously identified in the literature as being associated with caries incidence were identified using the Spearman rank correlation test, e.g., sex, age, salivary flow rate, buffering capacity, mutans streptococci counts, lactobacilli counts, income, and education. If the Spearman test showed some minimal association (*P*-value <.20), the variable became a candidate for the model. Each of the variables identified became candidates for the ordinal logistic regression model using PROC LOGISTIC in PC-SAS (27).

A forward selection procedure with entry set at P=.20 for the score statistic was used. After the initial selection was completed, variables not associated with disease progression with respect to their Spearman test were given a second chance to enter the model in a stepwise fashion with entry criteria set at P=.15. Then a final backward selection procedure was used to eliminate any of the variables that did not continue to be associated with disease. The backwards selection criterion was set at *P*=.10, since the goal of this analysis was exploratory. Once the final set of main variables was identified, squared main effect terms and pairwise interaction terms were evaluated for stepwise entry into the model with entry set at P=.05. Final models were then adjusted for the complex sampling frame using PC-SUDAAN (25).

### Results

Study Subjects and Participation. During the period between baseline and three-year follow-up, 89 subjects had died, 32 were too ill to continue participation, 63 had moved away from the area or could not be contacted for follow-up, and 135 refused to participate. Thirty-nine subjects became edentulous during this period and were no longer eligible for this study. Thus, of the 810 subjects examined at baseline, 452 (56%) were examined again at three years. Despite subject attrition due to death, medical disability, or refusal to participate, the balance of the representative groups was not affected. At the three-year followup, the baseline coronal and root caries scores for those subjects who were lost from the study were not significantly different from the scores of those who remained in the study. However, subjects who remained in the study had approximately one-half surface more that was filled than subjects who were lost

Caries Prevalence and Incidence. The baseline prevalence (percent of people with DF or D surfaces at baseline) and three-year incidence (percent of people with one or more new DF or D lesions) rates are presented in Table 1. Both the prevalence and incidence of coronal and root caries were higher for whites than for blacks. Coronal caries is more likely to be treated than root caries and the prevalence and incidence of caries in whites is much more likely to be composed of fillings and crowns in comparison to blacks. The incidence of root caries was lower than coronal caries; however, both conditions had substantial three-year incidence rates. In addition, root caries prevalence and incidence for blacks were much more likely to be in the form of unfilled decay than for whites. Thus, the two groups studied could be contrasted as: one group that has most of their caries prevalence and incidence in the form of treatment (whites) and one group that was much less likely to have caries prevalence and incidence in the form of treatment (blacks). Since missing teeth often confound caries patterns, it should be noted that at baseline whites had significantly more teeth present (mean=20.5, SE=0.6) than blacks (mean=16.7, SE=0.6). Blacks were significantly more likely to lose teeth over the three-year period (mean number

lost=2.2, SE=0.03) than whites (mean number lost=0.9, SE=0.2).

Table 2 presents the number and percent of people who had both coronal and root caries at baseline (prevalence) and who developed both root and coronal caries over the three-year period (incidence) for blacks and whites in terms of total caries experience (DF) and untreated caries only (D). This table summarizes results from eight 2 x 2 tables comparing the presence and absence of root caries in people who do and do not have coronal caries by presenting the cell for people who have both conditions. The P-value was a chi-square test calculated from the distributions in the four cells of each 2 x 2 table (coronal caries and root caries, coronal caries and no root caries, no coronal caries and root caries; no coronal caries and no root caries). As shown, both measures of caries prevalence (DFS and DS) show a significant association between the two conditions for both blacks and whites. In whites, the relationship is less significant for DFS than for DS, indicating that the F component is attenuating the relationship.

For blacks, the incidences of coronal caries and root caries are significantly associated irrespective of whether fillings are included. However, for whites, root and coronal caries incidence were not significantly associated for decayed and filled surfaces. The two conditions were significantly related only when unfilled decay was considered. Although not shown in this table, the nonsignificant relationship using DFS primarily occurred because 38.3 percent of the people with no coronal caries increment had a root caries increment compared to 16.7 percent of people with no coronal caries increment but having a root caries increment when only DS lesions were considered.

Root and Coronal Caries Models. Since root and coronal caries incidences were significantly associated for unfilled decay, we were interested in constructing logistic models to determine the characteristics associated with developing both unfilled coronal and root decay compared to developing just one of the conditions or developing neither of the conditions. Figure 1 presents the percentage of people who had both coronal and root caries, coronal caries only, root caries only, or no caries increment by whether or not

the increments include fillings. These incidence rates are provided to gain a better understanding of the conditions being modeled and the impact of not including fillings. Including fillings in the incidence rates increased the incidence for whites more than for blacks, especially for the incidence of both conditions and for coronal caries. When fillings are included, the incidence of coronal caries is greater than for root caries in both blacks and whites. Since coronal caries and root caries incidence were associated only for unfilled decay, we were limited to constructing a prediction model that used unfilled decay as the outcome variable.

Table 3 presents an ordinal logistic model for characteristics associated with the incidence of both root and coronal caries (D) over the three-year

period, either of the two conditions, or neither of the conditions. For this model, we are predicting people who had increments of both coronal caries and root caries, so the parameters are interpreted in the following manner. In instances where the parameter estimate is positive (+), the characteristic having a value of "1" is associated with a greater likelihood of having both conditions. Where the parameter estimate is negative (-), a score of "1" is associated with a smaller likelihood of having both conditions, meaning that the characteristic with a "0" score is associated with a greater likelihood of having both conditions. The absolute values of the parameter estimates indicate the strength of the association, with larger values indicating a stronger association. Main effects that were related to the incidence of both

 TABLE 1

 Coronal Caries and Root Caries Prevalence, Incidence, and Percent Decayed

Condition	Prevalence	% Decayed	Incidence	% Decayed
Coronal caries				
Black	78.4	30.9	44.4	34.3
White	94.5	7.2	47.0	17.4
Root caries				
Black	38.1	78.9	30.5	81.0
White	55.7	20.9	40.1	41.0

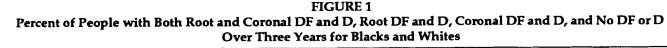
Prevalence=% of people with 1+DF.

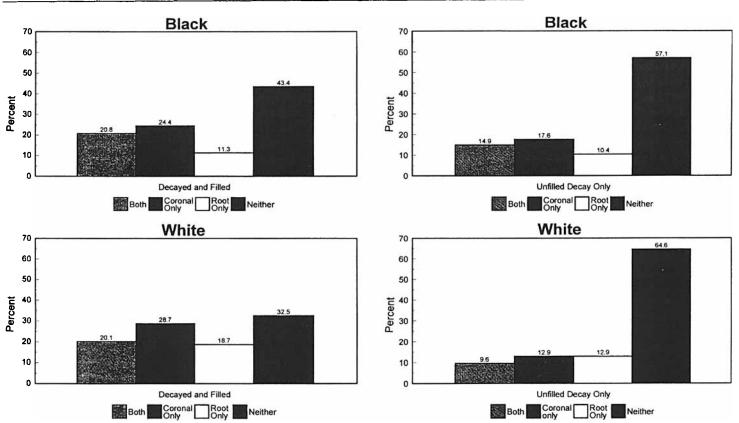
Incidence=% of people with 1+ new DF over 3 years.

 TABLE 2

 Associations Between Coronal Caries and Root Caries Prevalence and Incidence

Coronal Caries	Total N	N with Root Caries	% with Root Caries	P-value
Prevalence of 2 condi	tions:			
Blacks				
DFS	350	152	43.5	<.001 <.001
DS	254	111	43.8	
Whites		1 <b>9</b> 6	57 <i>.</i> 6	<.023
DFS	340			
DS	81	39	48.9	<.001
Incidence of the 2 cor	ditions:			
Blacks				
DFS	100	44	44.4	<.003
DS	72	31	43.2	.002
Whites		43	42.1	.623
DFS	102			
DS	47	19	40.7	<.020





conditions at the  $P \le .10$  level of significance were higher levels of gingival recession at baseline, having higher levels of attachment loss over the three years, and having a high lactobacilli score at baseline. Race and *P. gingivalis* at 36 months were not significant as main effects, but did form a significant interaction effect. The interaction indicated that those whites with *P. gingivalis* present at 36 months were more likely to develop both conditions, whereas *P. gingivalis* was not important for blacks.

## Discussion

In this study, there were two contrasting groups of people, one group that had most of its caries incidence in the form of treatment (whites) and one group that was less likely to have caries in the form of treatment (blacks). For both groups, a substantial proportion of people developed one or more new lesions over the three-year period, indicating that caries continues to occur throughout life in dentate populations.

The prevalence data from this study strongly indicated that coronal caries and root caries were associated with

TABLE 3 Ordinal Logistic Regression Model for Characteristics Associated with the Incidence of Unfilled Coronal and Root Caries\*

Characteristic	Parameter Estimate	SE	Significance Level	Odds Ratio	95% CI
Race (black=0, white=1)	0.267	0.297	0.368	1.31	0.73, 2.34
Worst gingival recession at baseline in mm	0.091	0.053	0.086	1.10	0.99, 1.22
P. gingivalis at 36 months (yes=0, no=1)	0.265	0.350	0.448	1.30	0.66, 2.59
Average attachment loss over 3 years	0.726	0.333	0.029	2.07	1.08, 3.97
Lactobacilli at base- line (0=not high, 1=high)	0.346	0.109	0.002	1.41	1.14, 1.75
Race x P. gingivalis at 36 months	-1.246	0.488	0.011		_

\*Ordinal variable (having both coronal and root caries=0, having either coronal or root caries=1, having neither coronal nor root caries=2).

each other in both high-treatment and low-treatment groups. A possible explanation for the association in hightreatment groups for the prevalence data and not for the incidence data is that the people who stayed in the study for three years are a biased subset of the people present at baseline, i.e., there is a selective survival problem. We reanalyzed the prevalence data using only the people who were in the study for three years and the results were the same. Thus, the different findings for the incidence and prevalence associations in the hightreatment group were not due to any type of selective survival bias in the study. In addition, this finding of a relationship between the two conditions agrees with similar findings on this subject from other studies that were reviewed in the introduction (2-8).

Investigating the association between coronal caries and root caries using prevalence data also can be problematic due to the inherent characteristics of prevalence data. For coronal caries, many of the restorations could have been placed decades earlier and before the person had many teeth at risk for root caries, which tends to occur later in life. This could result in significant associations between the two conditions that did not occur over the same time period, e.g., the coronal caries could have occurred earlier in life in the absence of root caries activity while the opposite pattern could have occurred for root caries. In addition, many of the teeth missing at baseline might have had caries prior to their extraction. Thus, associations based on historic evidence of the conditions can be misleading and, at best, are quite difficult to interpret.

The incidence rates for coronal caries and root caries were associated when most of the incidence was in the form of unfilled decay; however, the presence of a large proportion of restorations attenuated the relationship so that it was no longer significant. The additional cells that generated the findings presented in Table 2 that the incidence of DFS for the two conditions were not associated in whites indicate that there were quite a few people who received a coronal filling during the three years who had no new root lesions. When "decayed only" was the criterion for new caries lesions, those people with only coronal fillings were designated as having no new coronal caries and this resulted in a greater number of people falling into the category "no new coronal and root caries." This observation is substantiated by an additional analysis that followed 82 people who had both unfilled coronal and unfilled root lesions at baseline. During the three years, 18 people received coronal restorations; only five of those 18 (28%) also received restorations on their root lesions.

Why do the patterns of caries differ when measured by the D component as compared to the DF component? It might be that the receipt of dental treatment actually modifies the caries patterns in individuals so that those who receive care have different caries patterns than those who do not receive care. This explanation cannot be dismissed and might lead researchers to study that phenomenon more carefully and perhaps to stratify future samples based on receipt of care. Another explanation involves differences between the standardized diagnostic criteria for caries employed by the field examiners for whom the reliability is known and the diagnostic criteria used by dental practitioners in placing restorations. The diagnostic criteria used in this study emphasized cavitation and obvious caries, while practitioners might be filling teeth using other criteria. Thus, studies of groups that have a high level of care in which the diagnostic criteria for the caries study are not similar to the criteria for placing restorations will result in a study that has mixed, unreliable diagnostic criteria for caries. Consequently, we conclude that since the diagnostic criteria used in this study are known and because the reliability of the examiners was high, analyzing the data using only unfilled decay is the most appropriate alternative and that the two conditions are related.

Due to the problem just discussed and the fact that the two conditions are related when unfilled caries is the outcome, the outcome used in the ordinal logistic model was based on unfilled caries (D). This model (Table 3) indicated that people who are most likely to develop both types of D compared to developing just one type of D or no D at all are those who have more gingival recession at baseline, who have lactobacilli present at baseline, who experience more attachment loss during the time period, and who are white and have P. gingivalis present. The main effects in this model are consistent with what is known about coronal and root caries. The presence of lactobacilli in the model indicates that the conditions are related to the presence of an infection. Gingival recession at baseline is consistent with the fact that root D was already present in many people at the start of the study and the

presence of attachment loss over three years in the model indicates that periodontal disease was more likely to be progressing in those with more root caries. This finding is consistent with the supposition that new root lesions are more likely to occur in newly exposed dentin near the gingival margin. The interaction term, race and P. gingivalis, indicates that a putative periodontal pathogen is important for whites, but not for blacks. This finding does not imply that pathogens are not important for blacks, as other pathogens not measured in this study could be more important in this situation.

The results of this study lend a potential explanation to the findings reported by Hand et al. (10), who found that the three-year incidence rates of coronal and root caries were not significantly associated. That study also used the Radike criteria in diagnosing caries, assessed the relationship between the two conditions using the DF rate, and found that the population had a high rate of treatment, which also could have attenuated the relationship between the two conditions.

The implications of these types of results for future epidemiologic studies of caries are troubling. It appears that when standardized diagnostic criteria for coronal decay and root decay are used in a reliable manner, coronal decay and root decay do tend to appear together in the same individuals. The same association is seen in individuals or groups who have the vast majority of their caries in the form of unfilled decay. Thus, in this study, coronal decay and root decay (D) are associated in whites, but coronal caries and root caries (DF) are not. However, coronal caries and root caries (DF) are associated in blacks who have little F.

If this study had included only people with high levels of treatment, we could have reached a very different conclusion. The impact of dental treatment on the epidemiology of dental caries appears to be considerable and calls into question whether the F component of the caries index is related to disease as defined by epidemiologic criteria, a point already made by others (28,29), or whether treatment itself affects caries incidence patterns. This study shows the possible consequences of this problem on our understanding of the epidemiology of the diseases. It seems apparent that future studies cannot continue in the same vein, ignoring the impact of restorative treatment on disease epidemiology.

While we have no answers to this problem, potential strategies to consider may involve (1) disaggregating the typical descriptive and analytic epidemiology objectives for caries into two separate objectives: the epidemiology of unfilled carious lesions and the epidemiology of treated caries; (2) modifying epidemiologic diagnostic criteria to be more consistent with diagnostic criteria used by practitioners in placing fillings; or (3) including variables on practitioner clinical decision making in future attempts to create models of dental caries incidence. None of these strategies will entirely solve the problem. We tend to prefer the first strategy listed as an immediate response because it deals separately with both components of caries incidence. However, it is obvious that only reporting and modeling the D component means that both types of caries are being underreported (in this study, little underreporting in blacks and major underreporting in whites). However, using DF in groups that have a high proportion of F means that we are combining reliable data collected using standardized diagnostic criteria (the D component) with an F component that was generated by means of unknown diagnostic criteria that others have shown to have considerable variability (28,29). This methodology is not a sound one.

#### References

- 1. Sumney DL, Jordan HV, Englander HR. The prevalence of root surface caries in selected populations. J Periodontol 1973;44:500-4.
- Schamschula RG, Barmes DE, Keyes PH, Gulbinat W. Prevalence and interrelationship of root surface caries in Lufa, Papua New Guinea. Community Dent Oral Epidemiol 1974;2:295-304.
- Banting DW, Ellen RP, Fillery ED. Prevalence of root surface caries among institutionalized older persons. Community

Dent Oral Epidemiol 1980;8:84-8.

- Burt BA, Ismail AI, Eklund SA. Root caries in an optimally fluoridated and a high-fluoride community. J Dent Res 1986;65:1154-8.
- Beck JD, Hand JS Hunt RJ, Field HM. Prevalence of root and coronal caries in a noninstitutionalized older population. J Am Dent Assoc 1985;111:964-7.
- Vehkalahti M, Fajala M, Tuominen R, Paunio I. Prevalence of root caries in the adult Finnish population. Community Dent Oral Epidemiol 1983;11:188-90.
- Fure S, Zickert I. Prevalence of root surface caries in 55-, 65-, and 75-year-old Swedish individuals. Community Dent Oral Epidemiol 1990;18:100-5.
- Locker D, Slade GD, Leake JL. Prevalence of and factors associated with root decay in older adults in Canada. J Dent Res 1989;68:768-72.
- 9. Hand JS, Hunt RJ, Beck JD. Incidence of coronal and root caries in an older adult population. J Public Health Dent 1988; 48:14-19.
- Hand JS, Hunt RJ, Beck JD. Coronal and root caries in older Iowans: 36-month incidence. Gerodontics 1988;4:136-9.
- 11. Cornoni-Huntley J, Blazer DG, Lafferty ME, Everett DF, Brock DB, Farmer ME. Established populations for epidemiologic studies of the elderly. Vol 2. Resource data book. Bethesda, MD: National Institute on Aging, US Department of Health and Human Services, Public Health Service, National Institutes of Health, 1990; NIH pub no 90-495.
- Graves RC, Beck JD, Disney JA, Drake CW. Root caries prevalence in black and white North Carolina adults over age 65. J Public Health Dent 1992;52:94-101.
- Radike AW. Criteria for diagnosis of dental caries. In: Proceedings of the conference on the clinical testing of cariostatic agents, Oct 14-16, 1968. Chicago, IL: American Dental Association, 1972:87-8.
- Katz RV. Assessing root caries in populations: the evolution of the root caries index. J Public Health Dent 1980;40:7-16.
- 15. Miller AJ, Brunelle JA, Carlos JP, Brown LJ, Löe H. Oral health of United States adults. The national survey of oral health in US employed adults and seniors. National findings: 1985-1986. Bethesda, MD: US Department of Health and Human Services, Public Health Service, National Institutes of Health, 1987; NIH pub no 87-2868.
- 16. DePaola PF, Soparkar PM, Kent RL. Methodological issues relative to the quantification of root surface caries. Gerodontology 1989;8:3-8.
- 17. Hand JS, Hunt RJ, Reinhardt JW. The

prevalence and treatment implications of cervical abrasion in the elderly. Gerodontics 1986;2:167-70.

- Horowitz HS, Baume LJ, Backer-Dirks O, Davies GN, Slack GL. Principal requirements for controlled clinical trials of caries preventive agents and procedures. FDI Commission on Classification and Statistics for Oral Conditions. Int Dent J 1973;23:506-16.
- Frostell G. A colorimetric screening test for evaluating the buffering capacity of saliva. Swed Dent J 1980;4:81-6.
- Jordan HV. Cultural methods for the identification and quantification of Streptococcum mutans and lactobacilli in oral samples. Oral Microbiol Immunol 1986;1: 23-30.
- Jordan HV, Laraway R, Snirch R, Marmel M. A simplified diagnostic system for cultural detection and enumeration of *Streptococcus mutans*. J Dent Res 1987;66: 57-61.
- Loesche WJ, Syed SA, Stoll J. Trypsin-like activity in subgingival plaque: a diagnostic marker for spirochetes and periodontal disease? J Periodontol 1987;58:266-73.
- Bonta CY, Zambon JJ, Genco RJ, Neiders ME. Rapid identification of periodontal pathogens in subgingival plaque: comparisons of indirect immunofluorescence microscopy with bacterial culture for detection of Actinobacillus actinomycetemcomitans. J Dent Res 1985;64:793-8.
- 24. Zambon JJ, Reynolds HS, Chen P, Genco RJ. Rapid identification of periodontal pathogens in subgingival dental plaque. Comparison of indirect immunofluorescence microscopy with bacterial culture for detection of *Bacteroides gingivalis*. J Periodontol 1985;56(Spec Iss):32-40.
- 25. Shah BV, Barnwell BG, Hunt PN, Lavange LM. SUDAAN™ user's manual. Professional software for survey data analysis for multi-stage sample designs, release 5.50. Research Triangle Park, NC: Research Triangle Institute, 1991.
- Koch GG, Beck JD. Statistical methodologies useful for the analysis of data from risk-assessment studies. J Public Health Dent 1992;52:146-67.
- 27. SAS Institute Inc. SAS/STAT user's guide, version 6, 4th ed, vol 2. Cary, NC: SAS Institute Inc., 1989.
- Bader J, Shugars D, Rozier R. Epidemiologic caries assessments and dentists' treatment recommendations. Community Dent Oral Epidemiol 1993;21:96-101.
- Pitts NB, Fyffe HE. The effect of varying diagnostic thresholds upon clinical caries data for a low prevalence group. J Dent Res 1988;67:592-6.