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Multidimensional Causal Model of Dental Caries Development in Low-Income Preschool Children

SYNOPSIS

DESPITE THE DECLINE in the incidence of dental caries in the United States over the past several years, the condition remains a significant problem for the nation's poor children. Efforts to identify the factors responsible for caries development in samples of children of low socioeconomic status have primarily focused on a limited number of variables, and those have been predominantly biological (*mutans streptococci*, for example). Resulting models of caries development have usually shown good sensitivity but poor specificity. They have had limited implications for treatment.

In an effort to produce a comprehensive model of caries development, 184 low-income preschool children were clinically assessed for *mutans streptococci* and for decayed, missing, or filled surfaces of deciduous teeth twice, first at age 4 years (baseline) and again a year later (year 1 assessment). As the clinical assessments were being done, caretakers were being interviewed to obtain data from five domains: demographics, social status, dental health behaviors, cognitive factors such as self-efficacy (self-confidence) and controllability, and perceived life stress. Data were analyzed using a structural equations modeling approach in which variables from all domains, plus baseline decayed missing and filled surfaces and baseline mutans, were used together to create a model of caries development in the year 1 assessment.

Results confirmed earlier work that suggested that caries development at a 1-year followup was strongly dependent on earlier caries development. Early caries development in this sample was determined in part by mutans levels and by dental health behaviors. These behaviors themselves were accounted for partly by a cognitive factor. The results support the advantages of employing multidimensional models and provide some direction for intervention to reduce caries incidence.

> ognitive-behavioral models of disease are highly relevant to oral health behaviors because most oral health problems can be prevented or controlled through preventive behaviors or professional preventive care. But multidimensional models, particularly those consisting of both psychosocial and biological variables, have not

been widely used in the study of oral health diseases (1,2). Recent work in caries risk assessment and periodontal disease illustrates the potential contribution of psychosocial factors in understanding the disease process as well as improving prediction of disease risk (3). Our goal in this study was to develop a multidimensional model of caries development in preschool children of lowincome families that may shed light on the contribution of behavioral and psychological, as well as biological, variables.

Background: Models of Caries Development

The few models used to predict risk of disease in the oral health sciences are hampered by limited theoretical or conceptual frameworks regarding the expected relationships among the variables. Further, these models are simplistic in that they assume that all variables have direct effects on caries risk, when it is more likely that some variables have indirect effects on risk. Cognitive factors, for example, may influence behaviors, which in turn may directly influence caries risk. Four major groups of variables—biological, cognitive, behavioral, and demographic factors—have been studied previously in oral health risks.

Biological factors. Attention to developing statistical models to predict caries risk accurately has increased in recent years because the distribution of dental caries in children has changed. Contemporary studies show that the greatest prevalence of disease is concentrated in a minority of children. Bohannan and coworkers found that 60 percent of all caries occurred in 20 percent of the children (4). In the 1986 study of school age children by the Department of Health and Human Services, similar results were reported (5).

Biological predictors of caries, such as levels of *mutans* streptococci (S. mutans), lactobacillus, and salivary fluoride are characterized by high sensitivity, which is important in identifying those at risk of disease when the disease is highly prevalent (6-10). These models have poor specificity, however, because of a high false positive rate. That is, they are unable to distinguish who among those at risk of the disease will actually develop clinical caries. Specificity is increasingly important in evaluating prediction models as the incidence and prevalence of dental caries becomes concentrated in fewer children.

One consistent predictor of caries has been previous caries experience. When high-risk populations have been considered, prior caries has shown sensitivities of 57 percent and specificities of 85 percent (11) and 49 and 76 percent (12) for predicting caries of deciduous teeth in children. In multi-variate models, when biological and demographic factors have been considered, prior caries experience has been found to be one of the best predictors of caries increment (13, 14).

It is hoped that the addition of cognitive and behavioral variables may improve the overall predictive ability of models of caries development in low-income preschoolers. What is more important, however, is that the use of cognitive and behavioral variables may allow us to target interventions toward attitudes and health practices that may influence later dental health in this high-risk group.

Cognitive factors. Recent work in attribution theory has examined two complementary concepts, locus of control (15) and self-efficacy (16), in predicting or explaining health

behaviors. A limited number of studies have used these constructs in analyzing oral health behaviors or outcomes. Rotter's concept of locus of control refers to the beliefs of people regarding the source of control over events in their lives. Those whose locus of control is internal tend to believe that control over events resides within themselves, whereas those with an external locus of control tend to believe that events are controlled by forces external to them, such as powerful others or chance.

Studies using locus of control as a predictor of dental health behaviors have been inconsistent in their results. Some studies (17,18) have found no consistent relationship between locus of control and oral health behaviors and outcomes, while others have shown that having an external locus of control orientation is associated with poorer oral hygiene (19-21).

Perceptions of self-efficacy refer to the confidence of people in their ability to behave in certain ways (16). Selfefficacy is a powerful predictor of many health behaviors, including smoking cessation, weight loss, and adherence to preventive health programs (22). Few studies of oral health behaviors have examined self-efficacy, but some of these are provocative. McCaul and colleagues studied the ability of a social learning model to predict oral health behaviors (brushing, flossing) in a sample of college students (23). Those who had more self-efficacy about their ability to brush and floss, given the constraints in their lives, actually did brush and floss more frequently and had lower plaque scores. Tedesco and colleagues have shown that confidence in the ability to prevent periodontal disease significantly predicts adherence to oral hygiene regimens and that selfefficacy for a preventive regimen can be enhanced with intervention (24,25).

Stress. There is a large amount of literature on the relationship between stress and physical and mental health (26). The effects of stress on oral health problems has been studied, as well, but attention to this relationship has focused on selected oral health problems. For example, the effects of stress on acute necrotizing ulcerative gingivitis has been well documented (27-30). Two studies have assessed work stress and caries and periodontal disease. In a small case study, Freeman and Goss found that workers who reported more stress at work had more severe periodontitis (31). Marcenes and Shieham evaluated 164 male workers, ages 35-44, and found that periodontal disease was associated with higher work-related mental demands (32). Less attention has been paid in the literature to the effects of stress on oral health behaviors, but we would expect that those experiencing greater stress would be less likely to adhere carefully to oral hygiene recommendations for themselves or their children. Preventive behaviors may drop off as families are preoccupied with more pressing concerns.

The number of studies of stress and dental caries among children is small. Sutton suggests that the positive association between increased stress and higher rates of caries may be due to changes in oral hygiene behaviors and nutrition, saliva content, or blood supply (33). Tuutti and Lahti (34)and Lahti and coworkers (35) conceptualized and measured stress in terms of anxiety. Their work showed that parental anxiety regarding dental care, specifically the father's dental anxiety, was positively associated with a higher caries rate in their children. Parents' dental anxiety had a higher correlation with caries than did children's dental anxiety. In contrast, Brown and colleagues found a negative association between dental anxiety and caries rate for primary teeth (36).

Dental health behaviors. Three behaviors—tooth brushing, sugar consumption, and baby bottle use—have been studied extensively with regard to their effects on dental caries among children. Oral hygiene levels measured by plaque scores, self-reported tooth brushing, and use of a fluoride dentifrice have shown no consistent relationship with risk of caries in children (37). Several factors may account for the poor relationship between tooth brushing and caries risk. Even though children or their parents may brush their teeth frequently, the quality of brushing may be variable, brushing frequency may be overstated, and the effects of brushing may be more highly related to fluoride content of the toothpaste than to frequency of brushing.

In a recent review, Winter described the importance of fermentable carbohydrates, particularly refined sugar, in caries risk (38). He cautioned that caries development is a complex process involving factors such as host immune response, the characteristics of the oral microflora, and the amount and frequency of sucrose ingestion. Several investigators have shown a significant positive association between increased ingestion of sticky, high-sugar foods and increased risk of caries (37). Others have shown caries not to be associated with sugar consumption when other factors are considered (3,7,8,39). Hunt has suggested that measurement error may also account for inconsistent findings in the literature on the relationship between caries risk and sugar intake (37).

Putting a child to bed with a baby bottle clearly increases the risk of dental caries and contributes to a condition known as nursing caries, because the maxillary anterior teeth experience prolonged exposure to milk sugars (40). Others also have investigated duration of baby bottle use and have demonstrated that there is a higher risk of caries for those children who are bottle fed at an older age (7,8).

Sociodemographic factors. Sociodemographic factors have received considerable attention in the literature on caries risk assessment (1-3,37). Variables such as age, sex, race, and socioeconomic status have been included as control variables to assess the contribution of biological, behavioral, and cognitive factors in multivariate models of caries risk. Hunt reported that black children in the United States historically had lower caries rates than white children (37). Other recent surveys have shown that black and Hispanic children now have higher rates of decay than whites (5,10,41,42). Most investigators believe that differences in caries susceptibility between whites and nonwhites stem from socioeconomic and cultural factors.

Socioeconomic status has received considerable attention in evaluating caries risk and has been measured in a variety of ways, including parents' education, occupation, poverty status, and income. Regardless of measurement, studies in aggregate show children of higher social class generally have lower caries rates (3,37). Social class most likely has indirect effects on caries risk, in that social class is expected to affect behavioral norms about baby bottle use, use of preventive dental services, tooth brushing frequency and effectiveness, and sugar consumption. These behaviors will directly influence both mutans levels and caries experience.

Causal models. Our previous work analyzing caries risk among children ages 3 and 4 recruited from Head Start Programs in Connecticut showed that the inclusion of social and psychological variables in addition to biological variables did improve caries prediction (1). Although the best explanatory variable was the biological variable mutans level, the model with the highest predictive ability was one that included all variables. The significant variables were mutans, dental knowledge, dental locus of control, race-ethnicity, occupational status, stress, income, and baby bottle use. Children with higher levels of mutans whose parents had more external beliefs and reported more frequent baby bottle use were more likely to have caries than children with low mutans scores and parents who had less external beliefs and reported less frequent baby bottle use.

Stress and knowledge consistently had unexpected relationships with caries: those parents who reported less stress and had higher knowledge scores had children with higher caries prevalence. However, the strong effects of class were still evident, even within this relatively homogenous disadvantaged group. Those with lower incomes, those who were unemployed, and those who were nonwhite had greater risk of having children in the caries-positive group.

Discriminant analysis of followup data on 184 children who remained in the study after 1 year showed that clinical variables, the number of decayed, missing, and filled tooth surfaces (dmfs) and mutans level were the most important predictors of caries risk in the next year (43). Children who had higher dmfs and higher mutans levels were more likely to have decay in the followup period. These two variables alone explained 15 percent of the variation in decay. In addition, one behavioral variable, tooth brushing, was significant in the discriminant function but not in the predicted direction: more brushing was associated with more decay.

Causal modeling using structural equations with longitudinal data was indicated as the next step in our exploration of caries development for two reasons. Structural equation modeling is similar to linear regression-based path analysis, but with it, all projected paths can be examined simultaneously, and it yields statistics indicating how well a given model fits the available data. The ability to analyze complex models having both direct and indirect effects and employing variables with very different distributional properties was needed for several reasons.

First, the cross-sectional discriminant analysis suggested that behavioral and cognitive factors might have both direct and indirect effects on caries risk. As an example of an indirect effect, Tedesco and colleagues (25) showed that a cognitive factor like self-confidence did not have direct effects on gingivitis, but that it did influence oral hygiene behaviors, which in turn affected oral health outcomes. Thus, causal modeling could offer insights into the relationships among biological, cognitive, behavioral, and social variables that were not evident from discriminant analysis or logistic regression.

Second, the distribution of the dependent variablenumber of dmfs-was highly skewed and violated the assumptions of normality required for regression analysis. Special treatment of the variables, such as the use of polychoric correlations, was needed to analyze the data properly. Finally, unlike other techniques, structural equations modeling could take into account the influence of multiple concurrent variables as well as preceding ones and could test directional relationships. (Thus, a significant path coefficient indicates a directional, or causal, relationship.) The technique has a drawback in that sample sizes need to be close to 200 to insure reliable results.

Our study reports on the development of a multidimensional causal model of caries development in preschool children from low-income families. We believed that prior caries experience and the biological variable s. mutans would be the strongest predictors of caries risk, because they are most proximal to the outcome variable. We further believed that behavioral variables, measured by sugar intake, brushing frequency, and a baby bottle at bedtime would influence the mutans level directly and caries risk indirectly through the effects of mutans. Cognitive factors measured by dental knowledge, dental locus of control, dental care self-efficacy, and perceived stress would influence behaviors affecting caries risk. Sociodemographic factors including child's age, parents' age, race-ethnicity, education, income, and number of children would indirectly influence caries risk because of their direct effects on behaviors and attitudes toward oral health. All of these hypotheses would be tested in the stepwise construction of the final model.

Methods

Sample. The sample consisted of children ages 3 and 4 years and their caretakers recruited from a population enrolled in the Head Start Program in the City of Hartford and in New London County, Connecticut. Caretakers generally were the children's mothers, but in some cases, fathers, other relatives, and foster mothers were the primary caretakers. In the first year of the study, 460 children were examined clinically for caries and sampled for salivary mutans. Of the 460, complete data were obtained for 355 of their caretakers, who were interviewed regarding social, behavioral, and attitudinal characteristics of the families and the children in the study. At the beginning of the second year of the study, 184 of the original 355 children with complete data returned and were examined clinically, allowing collection of caries and mutans data, while their caretakers were re-interviewed.

Study protocol. Parents or other caretakers of eligible children were contacted by teachers or family advocates from Head Start in the fall of 1990 and were told about the study. Research assistants and dentists visited the Head Start schools in Hartford to examine children and conduct the interviews. Because New London County encompassed a larger geographic area, children and parents were examined and interviewed in two central locations. Transportation was provided when needed. The research assistant obtained the informed consent at the time of the examination and interview. Parents of children in the study received a small gift (value of less than \$10) as a token of appreciation.

Each child was given a clinical dental examination, and a saliva sample was obtained to measure mutans levels. In each year, one caretaker of each child was interviewed for 15-20 minutes to obtain data on sociodemographic characteristics, locus of control, dental self-efficacy, dental knowledge, sugar intake, and a measure of perceived life stress. All scales were pretested on pilot subjects before they were used on this sample.

Measurement of the variables. Dental caries. Each child was examined for dental caries with a mirror, explorer, and focusable flashlight by a trained dentist. Caries were identified using the criteria developed by Radike (44). An interexaminer reliability study of caries diagnosis, performed on a separate sample of Head Start children, yielded more than 90 percent agreement among the two raters using these methods. The same two raters participated both at baseline and at year 1 followup. Each tooth surface received a score of decayed, missing, filled, sealed, or sound. Stainless steel crowns were counted as five filled surfaces. Missing teeth were counted as 5 missing surfaces. (Teeth lost for reasons other than decay, such as displacement by a permanent tooth, were not counted as missing.) This resulted in a total score of decayed surfaces (ds) and decayed, missing, and filled surfaces (dmfs) for primary teeth for each child. The major dependent variable of this study was, therefore, the number of dmfs 1 year after the baseline measures had been taken.

Mutans. Saliva samples were obtained from each child by moistening a sterile wooden tongue depressor on the child's tongue. The tongue depressor was impressed onto plates containing media selective for *mutans streptococci* (45). Plates were incubated for 72 hours at 37° C in a CO_2 enriched environment, after which the number of colonyforming units (CFU) in the area of the tongue depressor was counted. Because of the skewed distribution of the count, the mutans variable in the study was scored on a 3point scale as follows: 0 = no detectable CFU; 1 = 1 to 50 CFU; and 2 = more than 50 CFU. This scoring scheme followed the trichotomous distribution of the data.

Sugar intake. Baseline data on sugar intake were obtained from the interview of the child's caretaker. Respondents were asked a series of questions about how often their child had eaten a list of seven foods high in sugar content during the last 2 weeks. Responses were: 1 (never) to 5 (more than once a day). Responses were summed to obtain a total index score that could range from 7 to 35.

Baby bottle use. Parents were asked whether their children ever took a bottle with milk or juice to bed at night. Those who responded yes were asked how frequently this occurred on a 6-point scale ranging from 0 (never) to 5 (every night).

Tooth brushing. Parents were asked how frequently brushing was done on a 6-point scale: never, less than once a day, once a day, twice a day, more than twice a day, or more than three times a day.

Dental knowledge. Dental knowledge was assessed by means of a questionnaire consisting of 10 items requiring a true or false response. Questions pertained to causes of caries, dental treatment, baby bottle use, and tooth brushing. The tests were scored by counting the total number of correct responses.

Dental health locus of control (externality). Dental health locus of control was a measure developed for this study. After a pretest, the scale consisted of seven statements about how much control over a dentist's behavior and how much information the parent desired. Respondents rated agreement with the statement on a six-point scale from strongly agree to strongly disagree. The scale could range from 7 to 42, with a higher score indicating higher externality or greater preference for others to have control. The scale had a Chronbach's alpha of .65.

Dental self-efficacy. A scale measuring dental self-efficacy, also developed specifically for this study, consisted of eight items describing specific preventive dental and treatment behaviors. Respondents were asked to rate confidence

Table I. Clinical oral health status of 184 low-income preschool children, baseline and year I, Connecticut, 1990–91

Oral health variable	Baseline	Year I
dmfs: ¹		
Mean	2.8	4.6
Standard deviation	6.9	9.2
No dmfs (caries free) (percent)	56	4 2
Mutans (CFU):		
Mean	48.9	43.8
Standard deviation	63	59
Percent of children with mutans colonies:		
0	21	23
I–50	45	50
51–150	33	27

'dmfs = decayed, missing or filled surfaces of deciduous teeth.

in their ability to perform the behaviors on a five-point scale ranging from 0 (not at all sure) to 4 (extremely sure). Responses were summed to give a possible range of 0 to 32. The internal reliability was Chronbach's alpha = .70.

Perceived stress. A revised version of the Holmes and Rahe Life Events Questionnaire was developed for this study (46). Parents were asked to indicate whether each of the 41 events listed in the questionnaire occurred to them and then, based on the work of Lazarus and Folkman (26), to rate how stressful they thought the event was on a sixpoint scale from 0 (no stress at all) to 5 (extremely stressful). Total baseline stress scores could range from 0 to 205.

Demographic variables. Several variables were included to determine the demographic characteristics of the family. These variables included race-ethnicity, age of the child and parent, family size, education of the parent, and income.

Results

Description of the sample. At baseline, complete data were obtained on 355 parents and their children out of a total of 460 approached. One year later, at the beginning of the second year of the study, interview data and data on caries and mutans were again obtained for 184 children and their parents out of the original 355. This represented an attrition rate of 48 percent. Data presented elsewhere indicate that there were no systematic differences between those who did and did not return for the second year of the study (1). Tables 1 and 2 present a summary of the clinical and social characteristics of the children.

The distribution of caries in this sample was positively skewed, with most children having few active or treated carious lesions. Most of the decay was experienced by a minority of the children. These trends continued largely unchanged into the next year, although more children experienced disease. The distribution of mutans also was positively skewed, with most children having either no detectable colonies or fewer than 51. The skewness was even more pronounced after 1 year; 73 percent of the children had fewer than 51 mutans colonies.

Table 2 shows that, at baseline, most children were about age 4 years in families of about four people. Parents generally were in their mid to late 20s and had a high school education. Most of the families had incomes of less than \$15,000 a year, and more than half had incomes of less than \$10,000. Blacks and Hispanics made up the majority of respondents.

Baby bottle use at bedtime also was a common practice, with 32 percent reporting that they put the child to bed with a bottle every night. Only 28 percent said that their children never used a baby bottle at night. Parents reported that children brushed their teeth at least once a day, and most reported twice a day. Moderate levels of sugar intake also were found. Parents appeared to be knowledgeable about factors that influence dental caries, reported moderate levels of perceived stress, and were confident of their ability to care for the oral health needs of themselves and their children. Table 2. Baseline demographic, behavioral, and attitudi-nal characteristics of 184 low-income preschool chil-dren, Connecticut, 1990, by percentages

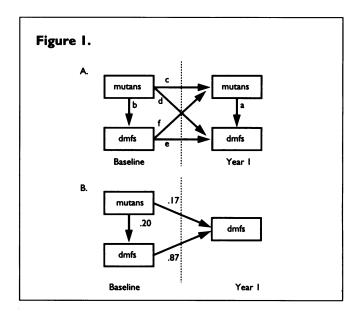
Category and variable	Percent	Mean	SD
Demographics			
Age (months)		46.3	5.7
Sex:			
Male	58	•••	
Female	42	•••	
Race-ethnicity:			
White	20		
Black	50	•••	
Hispanic	30		
Parent age (years)		28.5	7.2
Social status indicators			
Annual family income:			
Less than \$10.000	53		
\$10,000-15,000	29		
More than \$15,000	18	•••	
Parent education			
(years of school)		11.7	2.3
Number of persons in family		4.2	1.5
Dental health behaviors			
Baby bottle use at night:			
Never	28		
Some times	25		
Pretty often, very often	15		
Every night	32		
Brushing frequency:			
Once a day	24		
Twice a day	50		
More than twice a day	27		
Sugar intake index			
(range 7–28)		14.3	3.8
Cognitive factors			
Self-efficacy (range 8–32)		21.5	4.7
Externality (dental locus of			
control, range 7–39)		15.7	6.5
Dental knowledge			
(range 0–10)		7.8	1.4
Stress		•••	
Life stress (range 5–205)		17.2	15.0

NOTE: SD = standard deviation.

Causal modeling analyses. Overview. Causal model analyses with structural equations were done using LISREL 7 (47). Because of the relatively small sample size of 184, a generalized least squares procedure was used. The exploration of models proceeded as follows: first, an effort was made to create latent variables from those observed variables that fell in the same domain (for example, a dental health behavior variable, or a social status variable). Then, to limit the number of possible models that might have to be tested, constraints (to be described subsequently) were placed on the model in such a way that only certain predictions in certain directions would be allowed. Finally, models were created working backwards from the dependent variable, one domain at a time, within the constraints already established. Those paths that proved significant, and the variables that appeared as significant predictors, were retained in future models. At no time would all the variables in the data set be tested in the same model, since the usable size of the sample was not sufficient for a saturated model.

Measurement models: evaluating latent variables. The first measurement model analyzed was a latent social status variable, made up of family income, parent education, and number of people at home. Examination of the intercorrelations of the social status variables showed that only family size and income were correlated (r = .36, P < .001). A model of social status using these three variables failed to fit the data; that is, a coherent social status latent variable could not be formed. Similar results were found when latent variable models were assessed for dental health behavior (made up of tooth brushing frequency, dietary sugar, and baby bottle use), and for cognitive factors such as attitudes, beliefs, and knowledge (a confidence-knowledge latent variable made up of dental care self-efficacy, externality, and dental knowledge). The failure to find latent variables resulted in a final caries model using only directly observed variables.

Model constraints. Given the relatively large number of variables examined, certain constraints were instituted to insure that the number of models finally evaluated would be manageable. The first constraint was that the number of decayed, missing, or filled surfaces after 1 year (dmfs-year 1) would be the ultimate dependent variable and would not be employed in a model to predict other variables. The second constraint, based on our earlier results, was that our biological variable, mutans level, would be treated as a predictor of dmfs but nothing else. For example, the mutans level would not be placed in a model in which it predicted sugar intake.



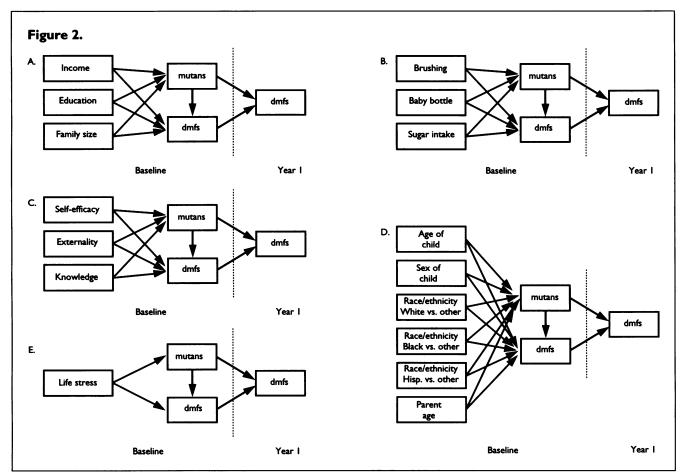
In Panel A, path a depicts the hypothesis that concurrent Year I mutans should predict Year I dmfs. Path b is the hypothesis that baseline mutans would be causal of baseline dmfs. Path c is the test of baseline mutans causing Year I mutans. Path d concerns baseline mutans causing Year I dmfs. Path e hypothesized that baseline dmfs would be predictive of Year I dmfs. Path f is the connection between baseline dmfs and Year I mutans. Panel B shows those paths that proved to be significant. Path coefficients (numbers between the variables) indicate the strength of association, in a predicted direction, between one variable and another. Higher values indicate higher strength of association. Path coefficients are standardized. Furthermore, because of its obvious predictive ability, mutans level would play a role in all models tested. Finally, the variables used to identify race-ethnicity would be used as predictors, but they could not be used as dependent variables. As an example, life stress could not be used as a cause of a participant's race-ethnicity.

Model building. The first relationships tested were between the baseline and year 1 disease and biological variables. The complete model tested is presented in figure 1, panel A. As indicated in the figure, paths were tested from mutans-year 1 to dmfs-year 1 and from baseline mutans and dmfs to year 1 mutans and dmfs. The only paths not tested were those from dmfs to concurrent or past mutans. (That is, we did not explore the possibility that dmfs could cause concurrent or past mutans.) Panel B shows the significant paths that resulted from the analysis. In this model, the only significant predictors of dmfs-year 1 were baseline mutans (t (182) = 4.4, P < .05) and baseline dmfs (t (182) = 28.1, P < .001).

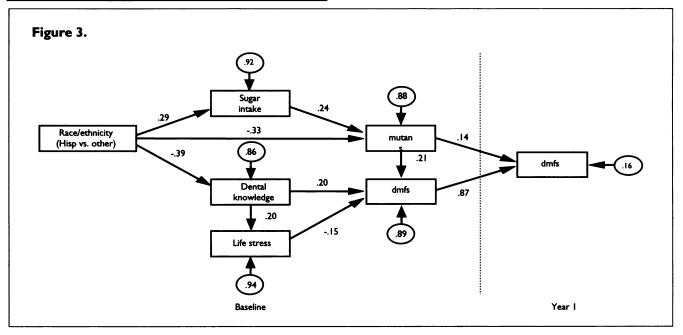
The path coefficients (the numbers between the variables) show the relative strength of the relationships between the predictor variables and the dependent variables. Higher values of the path coefficients indicate a stronger directional relationship. The path coefficients shown in the figures that follow are standardized, so that they may all be compared with each other.

Given the high correlation of baseline dmfs and dmfsyear 1 (r = .93), it seemed unlikely that any other variable would account for significant variance in the year 1 dependent variable. Nevertheless, a series of models was tested in which the domains of variables listed previously (that is, demographics, social status indicators, health behaviors, cognitive factors, and life stress) were tested in groups to predict dmfs-year 1. None of these models was predictive (none accounted for more than 3 percent of the variance in dmfs-year 1), nor were any of the individual paths significant. On the basis of these results we concluded that baseline dmfs, along with baseline mutans, would be the primary direct predictors, and that other variables would be modeled in such a way that their influence on dmfs-year 1 would be mediated through their effects on baseline dmfs.

Figure 2 depicts the various models that were tested next. Panel A of the figure shows the social status variables, panel B shows the health behavior variables, and so on. The measurement model analyses indicated that little intercorre-



Initial causal models of observed variables predicting to mutans and to dmfs. Panel A depicts the hypothesis that baseline social variables (income, education, and family size) will be predictive of baseline mutans and dmfs. Panel B shows dental health care behaviors being similarly tested. Panel C shows testing of cognitive factors. Panel D shows testing of demographics. Panel E is the model where life stress is used as the only predictor of mutans and dmfs.



Causal model made up of predictors found to be significant (at P < .05 level) in the analyses depicted in figure 2. Path coefficients (number between variables) are standardized. Higher values indicate higher strength of association. Psi values (in circles) represent residual variance of associated variables.

lation existed within classes of variables, so paths linking variables within classes, such as paths from self-efficacy to externality or from tooth brushing to baby bottle use frequency, were not tested at this point. None of the models shown in figure 2 fit the data very well (the average coefficient of determination was .03), but several of the individual paths were significant at the P < .05 level. As expected, the path from baseline mutans to baseline dmfs was significant (t (182) = 3.09, P < .01). So were the paths from baseline mutans to dmfs-year 1 (t (182) = 4.04, P < .001 and from baseline dmfs) t (182) = 27.7, P < .001).

Additionally, the path from dental knowledge (panel C) to baseline dmfs (t (182) = 2.52) and from life stress (Panel E) to baseline dmfs (t (182) = -2.02) were significant. The paths from sugar intake (Panel B) to baseline mutans level (t (182) = 2.96) and from Hispanic race-ethnicity (the dummy variable identifying Hispanics in panel B) to baseline dmfs (T (182) = -2.99) were also significant. These four variables were then placed in a new model.

The new model started out as saturated (that is, all possible paths in all directions were tested, except for a path from dmfs to mutans level). Nonsignificant paths were then removed, and the resulting model was tested (figure 3). This model fit the data relatively well

 χ^2 (10) = 10.85, P = .37, goodness-of-fit = .95,

coefficient of determination = .28.

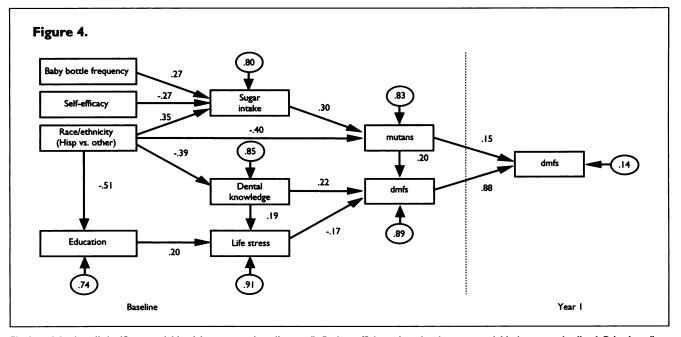
All paths shown were significant at the P < .05 level. As expected, all the paths from the left side of the figure to the right side are significant. The paths from race-ethnicity to sugar intake and from race-ethnicity to knowledge, however, were also significant, as was the path from knowledge to life stress. As in figure 1, panel B, path coefficients are shown indicating the strength of association between variables. In addition, psi values (in circles) are added. These values represent the residual variance of that associated variable. The amount of variance accounted for in a variable is therefore given by 1-psi (that is, for year 1 dmfs, the amount of variance accounted for in that variable is 1 - .16 = .84). The most potent predictor of dmfs-year 1 is baseline dmfs with a path coefficent of .87, but baseline mutans is predictive as well.

Finally, although the other 12 variables examined in this study did not predict either mutans level or dmfs, it was thought that they might predict some of the contributing variables from the new model. Therefore, another model was tested in which the remaining variables would be tested in relationships with the four predictors used in figure 3, namely race-ethnicity, sugar intake, dental knowledge, and life stress. All possible paths between these four variables and the remaining 12 were tested, as were the paths shown in figure 3. As before, nonsignificant paths were removed and the model was tested again. The final model is shown in figure 4. The total coefficient of determination of this model is .52. The model fits the data extremely well

 χ^2 (28) = 30.87, P = .47, goodness-of-fit = .99.

Discussion

The model in figure 3, with 84 percent of the variance accounted for, and in figure 4, with 86 percent, are highly predictive of dmfs-year 1 (as indicated by the psi values in the figures). This is a very high degree of predictive ability, but it becomes less surprising when one understands that the principal predictor of dmfs-year 1 is baseline dmfs. Even past



Final model using all significant variables (given constraints discussed). Path coefficients (number between variables) are standardized. Psi values (in circles) represent residual variance of associated variables.

mutans levels contributed only marginally, and concurrent mutans levels (mutans-year 1) contributed not at all. These findings, combined with the fact that no other baseline variable is predictive of dmfs-year 1, reinforces earlier conclusions by us (43) and others (11-14) that early development of caries strongly predicts caries progression. Conversely, those with few or no caries at 4 years are likely to have few caries a year later. Given these circumstances, it becomes even more important to determine the factors contributing to early caries, in this case represented by baseline dmfs.

In the final model (figure 4) 11 percent of the variance in baseline dmfs is accounted for. Although this is nothing like the 86 percent accounted for in dmfs-year 1, it is still of interest. Not surprisingly, concurrent mutans level is a strong contributor. Much more surprising is the strong contribution of dental knowledge and life stress, both of which were as predictive as the mutans variable. Oddly, the analyses indicate that the more knowledge a parent has, the more caries the child is likely to have. More knowledge is also associated with more report of life stress. And more stress is related to fewer caries.

One possible explanation is that the direction of some of the arrows is wrong. Perhaps parents acquire knowledge as a result of becoming informed when their children get caries. Because of the constraints of the study, this hypothesis was not tested directly. If the hypothesis were true, then dental knowledge should have either a negative or null impact on caries or caries change in the year 1 assessment. In fact, none of the baseline variables, including dental knowledge, has a direct association with dmfs-year 1.

The life stress finding is also unexpected. Those who report greater stress have children with fewer baseline caries. It may be that these high-stress people are simply hypervigilant, and report more stressful experiences. At the same time, they may behave in a more protective fashion (in a way not measured in this study) as a result of this vigilance. The positive contributions of education and dental knowledge supports the view that the parents who report higher stress may be those who have more knowledge generally. The one conclusion we can probably draw from these associations is that dental knowledge should not be considered protective against caries, and life stress should not be seen as cariogenic.

Another interesting association concerns the variable of race-ethnicity. The most important discrimination of ethnic groups came from the difference between Hispanics and other groups (whites and blacks for the most part). Examination of the distribution of both baseline and year 1 dmfs indicates that not only did Hispanics have lower mean dmfs at both assessment periods than did the other groups, but that they also had a small variance in the number of lesions. Judging by the final model in figure 4, low levels of dmfs in Hispanics in this sample is mediated by their lower levels of mutans. It is not clear, however, why Hispanics have lower levels of mutans.

A more understandable set of associations occurs in the prediction of mutans. Baseline mutans is the second-best predicted dependent variable of the model and is itself a strong predictor of disease. Of interest from an intervention point of view is that mutans level is strongly predicted by sugar intake levels. The results imply that reducing sugar intake would have a significant effect on mutans levels and thereby on dental caries. The model also suggests that, aside from race-ethnicity, the most important predictors of sugar intake are self-efficacy (that is, confidence in ability to care for one's dental health) and baby bottle use. Those mothers who are more likely to use a bottle at night are also more likely to have children with higher sugar intake. Those who are more confident in their ability to care for their teeth have children with lower sugar intake.

These results provide partial support for the theory of planned behavior as it would apply to parents' dental health care for their children (48). The theory of planned behavior suggests that attitudes form intentions and that intentions will drive health behaviors. One of the more important attitudes is a person's attitude toward oneself and one's abilities, that is, self-efficacy. Our results resemble those of McCaul and colleagues (49) as far as the importance of self-efficacy as a predictor of dental health behavior is concerned. McCaul and coworkers, however, also found that perception of controllability was a mediating variable. In our study, perceptions of controllability (as measured by dental locus of control scale) did not play a significant role, although this was possibly the fault of our instrument.

One variable that was not included in these models was access to care. We do not know, therefore, what measurement of barriers to dental care can contribute to prediction of caries risk. Future studies will need to address these kinds of variables.

Our model nevertheless provides a broad view of the process of caries development in low-income children. By using causal modeling we were able to investigate both direct and indirect predictors of caries rates and of mutans. The model indicates that caries development is more complex than usually conceptualized in the literature. A health behaviors model, in which attitudes and intermediate behaviors are considered, may be the most complete way to view the development of caries. From a clinical perspective, the model is also prescriptive.

On a concrete level, the results indicate that public health efforts must be directed at early primary prevention of caries; secondary prevention efforts are unlikely to be very successful. Children must be reached before they can develop caries. Our results also indicate that efforts should be made to intervene with caretakers of children at risk to prevent the use of night time baby bottles and to limit the use of refined sugar. Given the importance of self-efficacy in the consumption of refined sugar, such an intervention would take the form of an educational program that would not only provide information but also raise the confidence of caretakers in their ability to care for the dental health of the children in their charge.

A program that provides positive feedback (in the form of demonstrating reductions in plaque deposition, for example), plus reinforcement for good performance, should increase self-efficacy, which is raised through success experiences. We are currently exploring the development of such interventions with caretakers that could provide a way to achieve reductions in harmful behaviors and ultimately decrease caries development in this vulnerable population. The research was supported by National Institutes of Health grant No. DE-09217 awarded to Dr. Tinanoff.

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