Community Dent Oral Epidemiol 2007; 35: 449–458

COMMUNITY DENTISTRY AND ORAL EPIDEMIOLOGY

Dental caries and childhood obesity: roles of diet and socioeconomic status

Teresa A. Marshall[1], Julie M. Eichenberger-Gilmore[1], Barbara A. Broffitt[1], John J. Warren[1] and Steven M. Levy[1,2]

[1]Department of Preventive and Community Dentistry, College of Dentistry, University of Iowa, [2]Department of Epidemiology, College of Public Health, University of Iowa, Iowa City, IA, USA

Marshall TA, Eichenberger-Gilmore JM, Broffitt BA, Warren JJ, Levy SM. Dental caries and childhood obesity: roles of diet and socioeconomic status. Community Dent Oral Epidemiol 2007; 35: 449–458. © 2007 The Authors. Journal compilation © 2007 Blackwell Munksgaard

Abstract – *Objective:* Our objective was to determine (a) if caries and obesity were associated in a pediatric population and (b) if so, then to explore diet and socioeconomic status as additional risk factors. *Methods:* Subjects were recruited at birth and are members of the Iowa Fluoride Study. Data such as parental age, parental education levels and family incomes were obtained by questionnaire at recruitment. Children's primary dentition was examined and their weight and height measured at 4.5–6.9 years of age. Parental weight and height were measured when children were 7.6–10.9 years of age. Beverage and nutrient intake patterns were obtained from 3-day food and beverage diaries completed at 1, 2, 3, 4 and 5 years of age. *Results:* Children with caries had lower family incomes, less educated parents, heavier mothers and higher soda-pop intakes at 2, 3 and for 1–5 years than children without caries ($P < 0.05$). 'Overweight' children had less educated fathers and heavier parents than 'normal' weight children ($P < 0.05$). Children 'at risk' of overweight had higher caries rates than 'normal' or 'overweight' children ($P < 0.05$). In stepwise logistic regression models to predict caries experience, soda-pop intakes were displaced by mother's education, leaving 'at risk' of overweight and mother's education in the final model. *Conclusion:* Caries and obesity coexist in children of low socioeconomic status. Public health measures to improve dietary education and access to appropriate foodstuffs could decrease the risk of both diseases.

Key words: caries; diet; obesity; soda-pop; socioeconomic status

Teresa A. Marshall, Department of Preventive and Community Dentistry, College of Dentistry, N335 DSB, Iowa City, IA 52242-1010, USA
Tel: +1 319-335-7190
Fax: +1 319-335-7187
e-mail: teresa-marshall@uiowa.edu

Submitted 6 March 2006;
accepted 12 July 2006

Dental caries during childhood continues to be a significant public health concern, while childhood obesity is increasingly being cited as a major public health problem. The 2000 Surgeon General's Report stated that caries is the most prevalent chronic disease of childhood, affecting 58.6% of children aged 5–17 years (1). More recently, Glick reported that 41% of children aged 2–11 years participating in National Health and Nutrition Examination Survey (NHANES) 1999–2002 had primary caries, and that the prevalence and severity of primary caries is unchanged from NHANES 1988–1994 (2). Childhood obesity is less prevalent than dental caries, with 10.3% and 15.8% of children aged 2–5 and 6–11 years, respectively, participating in NHANES 1999–2002 being overweight (3). Unlike caries, childhood obesity rates have increased since NHANES 1988–1994 when 7.2% and 11.3% of children aged 2–5 and 6–11 years, respectively, were overweight (4). Historically, both dental caries and childhood obesity have occurred at disproportionately higher rates in individuals of minority background and/or low socioeconomic status.

Recent studies have identified an association between dental caries and obesity in childhood, and have suggested that obese children are at an increased risk for dental caries (5–7). These studies were cross-sectional in nature and unable to investigate causality. The concept of biological

doi: 10.1111/j.1600-0528.2006.00353.x

plausibility would suggest that neither the hypothesis 'obesity increases risk of caries' nor 'caries increases risk of obesity' is particularly logical. Rather, it is more realistic that a common risk factor increases the likelihood of both diseases, which are then observed in association.

Dental caries is a disease process in which repeated exposure to acids produced during bacterial fermentation of carbohydrates erodes the enamel. The erosion is progressive and can lead to cavitation of the enamel and, thereafter, of the dentin. Although necessary, the dietary component of the disease process is difficult to quantify. Sugars, modified starches and starches are all subject to fermentation; sugared soda-pop, confections and starches baked with sugars are considered highly cariogenic (8–12). Dietary habits, that is, when and how the beverage or food is consumed, can modify this risk with frequent consumption of sugars thought to increase risk (13). Children at highest risk for dental caries are disproportionately from minority households and/or live in poverty (1). Both the prevalence of decay and the lack of dental treatment increase with minority status and decreasing resources (14). Food choices, dietary habits and socioeconomic status (SES) are interrelated (15, 16); these interactions have not been investigated with respect to caries etiology.

Obesity is a disease process in which energy intakes exceed energy requirements resulting in the deposition of body fat. Obesity is defined as an excess of body fat and has both genetic and environmental origins. Although environmental obesity conjures images of indulgence, particularly on high-fat, sweetened treats, and limited physical activity, biological adaptation and food culture may be more responsible (17, 18). The transition from a hunter–gatherer society characterized by feast–famine cycles to our current industrial society characterized by plentiful food has been relatively rapid in evolutionary terms and may not have allowed sufficient time for biological adaptation (19, 20). Energy-dense, highly refined food choices (i.e. soda-pop, fast food), dietary habits (i.e. grazing, night-eating, binging) and food insecurity have been identified as potential contributors to the obesity epidemic (17, 18, 21–23). In the United States, childhood obesity is associated with low SES (24, 25). Drewnoski and Darmon (26) speculated that the relationship between obesity and SES is mediated by the low cost of energy-dense foods, particularly those high in fat and sugar. Relationships between obesity and dental caries, with potential mediation by dietary factors and SES, have not been thoroughly investigated. Knowledge of these relationships could lead to preventive health measures designed to decrease the prevalence and incidence of both obesity and dental caries.

We hypothesize that if obesity and caries occur more frequently in the same children, then a common risk factor drives both disease processes. The objectives of the current study were (a) to determine if caries and obesity were associated in a pediatric population and (b) if an association was observed, then to explore dietary factors and SES as risk factors.

Methods

Subjects

Children and their parents were participants in the Iowa Fluoride Study (IFS) (12, 13, 27–33) and the Iowa Bone Development Study (IBDS) (12, 13, 27–33), which are longitudinal investigations of the relationships between fluoride exposures, oral health measures and bone health. Mothers of newborn infants were recruited from March 1992 to February 1995 from eight Iowa hospitals for their children's participation. Children who participated in dental examinations and whose parents completed 3-day food and beverage diaries ($n = 427$) for them were the focus of this report. Fourteen subjects were excluded from analyses because their body mass index (BMI) was below the normal range. The Institutional Review Board at the University of Iowa approved all components of the IFS and IBDS, and written informed consent was obtained from mothers at recruitment and again at the time of examination.

Data collection

Demographic information was obtained from mothers at recruitment (Fig. 1). Parents were mailed IFS questionnaires and 3-day food and beverage diaries at 6 weeks of age; 3, 6, 9 and 12 months of age; every 4 months through 3 years of age; and every 6 months thereafter. IFS questionnaires were designed to obtain information regarding the children's beverage intakes, general health and oral health behaviors.

Children participated in clinical examinations of the primary dentition and dual-energy X-ray

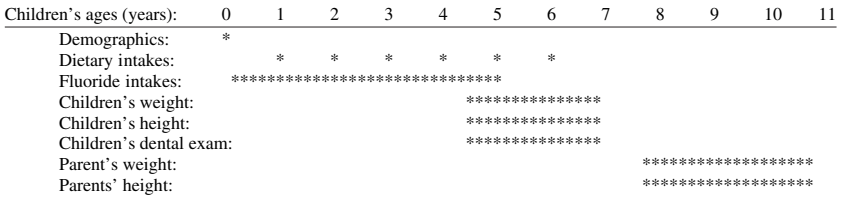

Fig. 1. Description of Iowa Fluoride Study.

absorptiometry scans for bone-density measures at 4.5–6.9 years of age. Parents also had bone-density scans and measurements of weight and height when their children were 7.6–10.9 years of age.

Children's dental caries experience

Examinations of the primary teeth were conducted at the General Clinical Research Center at The University of Iowa or one of several community locations by trained and calibrated dentist examiners using standardized, portable dental equipment (27, 28). The examination was visual, but a dental explorer was used to confirm questionable caries findings (27). Transillumination with the DenLite® system (Welch-Allyn Medical Products, Inc, Skaneateles Falls, NY, USA) augmented the visual/tactile examination.

For this study, caries experience was defined as the presence of at least one cavitated (d_{2-3}) or filled surface. The criteria for dental caries did not differentiate between cavitated enamel (d_2) and dentin (d_3) lesions, and have previously been reported (27). Inter-examiner agreement for caries experience at the person level was 92.3%, with a kappa statistic of 0.82.

Anthropometric measures

Weight and height were measured at the time of clinic visits in light clothing and without shoes. Weight was measured using a standard physician's scale. Height was measured using a stadiometer. Body mass index (kg/m^2) was calculated from these measurements.

Children were categorized as 'normal'; 'at risk' of overweight or 'overweight' using age- and gender-specific Centers for Disease Control and Prevention (CDC) definitions for BMI (34).

Parents were initially categorized as normal weight (BMI < 24.9), overweight (BMI 25.0–29.9) or obese (BMI > 30.0) using CDC definitions (34); CDC categorizations were used in analyses of maternal or paternal BMI categories. Parents were subsequently categorized using criteria defined by Whitaker et al. (35) as normal (mother's BMI < 27.3; father's BMI < 27.8) or obese (mother's BMI ≥ 27.3; father's BMI ≥ 27.8) to create a parental obesity category: 0, 1 or 2 obese parents.

Dietary data

Three-day food and beverage diaries targeted for completion at 1, 2, 3, 4, and 5 years of age or substitutes completed within 6 months were used in these analyses (12, 30). One- to 5-year intakes were estimated using area-under-the-curve analyses and required a minimum of four diaries, including the 1- and 5-year diaries (12, 30).

Parents and caregivers were asked to record all foods and beverages consumed by their child for 1 weekend day and 2 weekdays on each diary (12, 30). The 3-day food and beverage diaries were coded and verified by registered dietitians. Weighted averages based on weekend or weekday consumption were calculated to reflect average consumption during a week (12, 30). Energy, soda-pop and 100% juice intakes were included in analyses.

A relational database (Microsoft Access, version SR-1, Redmond, WA, USA) was used to analyze food and beverage records. Nutrient data were obtained from the USDA (Nutrient Database for Standard Reference 12, Agriculture Research Service, US Department of Agriculture, Washington, DC, USA), the Minnesota Nutrient Database (Nutrition Coding Center NDS-R, versin 4.01; Minneapolis, MN, USA) and manufacturers' values (12, 30).

Fluoride intake

Iowa Fluoride Study questionnaires completed from 6 weeks to 5 years of age were used to estimate fluoride intakes from water consumed as a beverage and added during preparation of select foods, other beverages, dietary fluoride supplements and fluoride dentifrices (12, 31, 33). Cumulative fluoride intakes (mg/day) were estimated from fluoride intakes from 6 weeks through 5 years of age using area-under-the-curve analy-

ses. Fluoride intakes were used as a control variable in models developed to predict caries risk.

Statistical analyses

Cross-sectional, secondary analyses were conducted using SAS (SAS, version 8.0; Cary, NC, USA). Subject characteristics were categorized and presented as percentages or median values (25th, 75th percentiles). Fisher's exact tests, chi-squared analyses and Kruskal–Wallis tests were used to compare baseline demographic variables between caries experience categories and among BMI categories. Spearman's correlations were used to test associations between BMI categories and dietary intakes. The Mantel–Haenszel correlation statistic was used to test linear associations and the general association statistic was used to test categorical differences between caries prevalence and BMI categories after stratifying by the child's age at the dental examination. Prevalence of dental caries by BMI category was adjusted for differences in age using direct standardization. A P-value < 0.05 was considered statistically significant.

Logistic regression models were developed to explain caries experience. All models included age at dental exam and cumulative fluoride intake. Sequential models were developed by entering the variables that were significant in bivariate analyses in the following order: child's BMI category, child's soda-pop intake, parental education, parental BMI and family income.

Results

Demographic characteristics by children's caries experience and children's BMI

Demographic characteristics of children and their parents at enrollment into the IFS are presented according to the children's caries experience and BMI category (Table 1). Gender, birth order and parental ages did not differ across caries experience nor BMI categories. Children with caries experience had lower family incomes than children without caries experience; family income did not differ among BMI categories. Parents of children with caries experience were less educated than parents of children without caries experience; fathers of

Table 1. Demographic characteristics of families at enrollment according to children's caries experience and body mass index (BMI) categories

	Caries experience		Children's BMI category		
Variable	Caries free (n = 296)	Caries present (n = 117)	'Normal' (n = 308)	'At risk' of overweight (n = 81)	'Overweight' (n = 24)
	Percentage[a]		Percentage[b]		
Gender					
Female	68	32	74	21	5
Male	75	25	76	18	7
First child					
Yes	74	26	73	20	7
No	70	30	76	20	5
	Median (25th, 75th percentile)[c]		Median (25th,75th percentile)[c]		
Family income category (1–7)[d]	5 (3, 6)	4 (3, 6)**	4 (3, 6)	4 (3, 6)	4 (2, 5)
Mother's age (years)	30 (26, 34)	29 (25, 33)	29 (26, 34)	30 (25, 34)	27 (25, 33)
Father's age (years)	31 (28, 35)	31 (28, 35)	31 (28, 35)	31 (26, 35)	30 (27, 32)
Mother's education level (1–7)[e]	6 (4, 6)	4 (3, 6)***	5 (4, 6)	5 (4, 6)	4 (4, 5)
Father's education level (1–7)[e]	5 (3, 6)	4 (3, 6)*	5 (4, 6)	5 (3, 6)	4 (3, 5)*

[a]Fisher's exact test was used to compare gender and birth order between caries experience categories.
[b]Chi-squared test was used to compare gender and birth order among BMI categories.
[c]Kruskal–Wallis test was used to compare family income, parental ages and parental education categories between caries experience categories and among BMI categories.
[d]Family income categories: 1 < $9999; 2 = $10 000–19 999; 3 = $20 000–29 999; 4 = $30 000–39 999; 5 = $40 000–49 999; 6 = $50 000–59 999; 7 = ≥$60 000.
[e]Educational levels: 1 = 8th grade or less; 2 = some high school; 3 = high school diploma/GED; 4 = some college; 5 = associate degree; 6 = bachelor's degree; 7 > bachelor's degree.
*P < 0.05; **P < 0.01; ***P < 0.001.

'overweight' children were less educated than fathers of children categorized as 'normal' or 'at risk'.

Children's anthropometric measures by children's caries and children's BMI

Children's median age-adjusted weight, height and BMI did not differ according to caries experience (data not shown). Children's median age-adjusted weight and height increased with BMI category (normal: 21.1 kg, 116 cm; at risk: 24.6 kg, 118 cm; overweight: 30.1 kg, 122 cm; both $P < 0.001$).

Parent's anthropometric measures by children's caries and children's BMI

Parents' median (25th, 75th percentile) weight, height and BMI according to children's caries experience and BMI category are presented in Table 2. Mothers of children with caries had higher weights and BMI values than mothers of children without caries. Both mothers and fathers of 'overweight' children had higher weights and BMIs than parents of 'normal' or 'at risk' children.

Children's diet by children's caries and children's BMI

Associations among children's median daily intakes of energy, soda-pop and 100% juice at ages 1, 2, 3, 4, 5 and for 1–5 years, caries experience and BMI categories were tested (data not shown). Neither energy nor 100% juice intakes at any age differed between children with and without caries. Children with caries had higher soda-pop intakes at ages 2, 3 and for 1–5 years than children without caries (median intakes at 2 years: 16 g vs. 0 g; 3 years: 32 g vs. 0 g; 1–5 years: 44 g vs. 28 g; all $P < 0.01$). At age 1 year, energy intakes differed among BMI categories (median intakes: 'normal' – 921 kcal; 'at risk' – 994 kcal; 'overweight' – 1077 kcal; $P < 0.01$). Neither soda-pop nor 100% juice intakes were associated with BMI categories at any age.

Children's diet by parents' BMI

Subsequently, Spearman's correlations between the children's dietary intakes and parents' BMI were investigated (data not shown). Mothers' BMI categories were positively associated with children's energy intakes at 1 ($r = 0.228$; $P < 0.001$) and for 1–5 ($r = 0.120$, $P = 0.046$) years and with soda-pop intakes at 1 ($r = 0.102$; $P = 0.033$), 2 ($r = 0.138$; $P = 0.009$), 3 ($r = 0.195$; $P < 0.001$), 5 ($r = 0.133$; $P = 0.025$) and for 1–5 ($r = 0.188$; $P = 0.002$) years. One hundred percent juice intakes were inversely associated with mothers' BMI categories at 2 ($r = -0.125$; $P = 0.018$) years. No significant associations were observed between children's dietary intakes and fathers' BMI categories. Children's energy intakes were positively associated with the number (i.e. 0, 1 or 2) of obese parents at 1 ($r = 0.181$; $P < 0.001$) and for 1–5 ($r = 0.126$; $P = 0.039$) years and soda-pop intakes at 1 ($r = 0.095$; $P = 0.050$), 2 ($r = 0.107$; $P = 0.044$), 3 ($r = 0.208$; $P < 0.001$), 5 ($r = 0.192$; $P = 0.001$) and for 1–5 ($r = 0.179$; $P = 0.003$) years. One hundred percent juice intakes were inversely associated with the number (i.e. 0, 1 or 2) of obese parents at 2 ($r = -0.118$; $P = 0.027$) years.

Table 2. Median (25th, 75th percentiles) mothers' and fathers' anthropometric measures according to children's caries experience and body mass index (BMI) categories

	Children's caries experience[a,b]		Children's BMI category[c,d]		
Variable	Caries free	Caries present	'Normal'	'At risk' of overweight	'Overweight'
Mother					
Weight (kg)	70 (61, 83)	75 (66, 91)**	69 (61, 82)	72 (66, 92)	91 (74, 103)***
Height (cm)	165 (161, 170)	165 (161, 171)	165 (160, 170)	166 (162, 170)	166 (162, 171)
BMI	25.5 (22.3, 30.0)	27.8 (23.6, 32.3)**	25.4 (22.3, 29.8)	27.3 (23.7, 32.4)	30.6 (28.7, 37.7)***
Father					
Weight (kg)	90 (80, 102)	90 (80, 104)	88 (79, 101)	92 (84, 101)	99 (88, 108)*
Height (cm)	179 (175, 184)	179 (175, 182)	179 (175, 183)	180 (174, 184)	176 (172, 181)
BMI	27.7 (25.4, 31.3)	28.1 (25.3, 31.4)	27.4 (24.9, 30.8)	28.5 (26.1, 31.2)	31.1 (29.4, 34.5)***

[a]Wilcoxon test was used to compare parental anthropometric measures between caries experience categories.
[b]$n = 296$ and 291 for mothers and fathers of children without caries and $n = 117$ and 115 for mothers and fathers of children with caries, respectively.
[c]Kruskal–Wallis test was used to compare parental anthropometric measures among children's BMI categories.
[d]$n = 308$ and 304 for mothers and fathers of children with 'normal' BMIs; $n = 81$ and 80 for mothers and fathers of children with 'at risk' of overweight BMIs; and $n = 24$ and 22 for children with 'overweight' BMIs, respectively.
*$P < 0.05$; **$P < 0.01$; ***$P < 0.001$.

Children's diet by demographic characteristics

Correlations between the children's dietary intakes and demographic characteristics were explored to identify relationships among predictor variables (data not shown). Children's energy intakes were inversely associated with mothers' ages at 2 ($r = -0.150$; $P = 0.005$) years and fathers' ages at 1 ($r = -0.113$; $P = 0.026$) and 2 ($r = -0.215$; $P < 0.001$) years. Children's soda-pop intakes were inversely associated with mothers' education at 2, 3, 4, 5 and for 1–5 years (r range: −0.180 to −0.308; all $P \geq 0.002$); fathers' education at 2, 3, 5 and for 1–5 years (r range: −0.124 to −0.216; all $P \leq 0.025$); mothers' age at 3, 4, 5, and for 1–5 years (r range: −0.131 to −0.174; all $P \leq 0.033$); fathers' age at 3, 4, 5, and for 1–5 years (r range: −0.146 to −0.171; all $P \leq 0.019$); and family income at ages 3, 4, 5, and for 1–5 years (r range: −0.136 to −0.190; all $P \leq 0.028$). Children's 100% juice intakes were inversely associated with mothers' ages at 1 ($r = -0.143$; $P = 0.004$), 4 ($r = -0.122$; $P = 0.047$) and for 1–5 ($r = -0.122$; $P = 0.049$) years; fathers' ages at 1 ($r = -0.128$; $P = 0.012$) and 3 ($r = -0.131$; $P = 0.031$) years and family income at 1 ($r = -0.115$; $P = 0.021$) year.

Children's caries by children's BMI and parent's BMI

Age-adjusted caries prevalence rates differed among children in different BMI categories; the 'at risk' of overweight category had the highest caries rate (Table 3). Caries rates were positively associated with mothers' BMI categories, but were not associated with fathers' BMI categories (Table 4).

Parent's BMI by demographic characteristics

Prior to developing multivariable models, associations between parental BMI categories and demographic characteristics were explored to identify relationships among predictor variables. Mothers' BMI categories were inversely associated with family income ($r = -0.204$; $P < 0.001$), mothers' education ($r = -0.147$; $P = 0.002$) and fathers' education ($r = -0.196$; $P < 0.001$). Fathers' BMI categories were inversely associated with fathers' education ($r = -0.127$; $P = 0.011$). The number (i.e. 0, 1 or 2) of obese parents was inversely associated with family income ($r = -0.139$; $P = 0.005$), mothers' education ($r = -0.130$; $P = 0.007$) and father's education ($r = -0.171$; $P < 0.001$.

Table 3. Children's age-adjusted caries prevalence[a] according to children's body mass index (BMI) category

Children's BMI Category[b]	
''Normal'' ($n = 308$)	27.4
''At Risk'' of Overweight ($n = 81$)	37.0
''Overweight'' ($n = 24$)	10.5*

[a]Percentage of children.
[b]The Cochran–Mantel–Haenszel general association statistic was used to test for categorical associations between caries prevalence and children's BMI category after stratifying by age at dental examination.
*$P < 0.05$.

Table 4. Children's age-adjusted caries prevalence[a] according to mothers' and fathers' body mass index (BMI) category and the number of obese parents

Mother's BMI Category[b]	
Normal ($n = 188$)	23.2
Overweight ($n = 127$)	30.0
Obese ($n = 116$)	33.8*
Father's BMI Category[b]	
Normal ($n = 100$)	30.3
Overweight ($n = 187$)	28.3
Obese ($n = 137$)	28.3
Number of Obese Parents[b]	
None ($n = 139$)	23.4
One ($n = 182$)	29.2
Two ($n = 103$)	33.9

[a]Percentage of children.
[b] The Mantel–Haenszel chi-squared statistic was used to test for linear associations between caries prevalence and BMI category after stratifying by age at dental exam.
*$P < 0.05$.

Models to predict children's caries experience

Sequential logistic regression models were developed to predict caries experience from variables that were significant in bivariate analyses; all models included age at dental examination and cumulative fluoride intake (Table 5). All variables except age and fluoride intakes were required to be statistically significant in order to be added to the models. Variables were dropped at later stages if they no longer showed a significant effect (i.e. their effect was decreased because of the addition of other variables). The children's 'at risk' of overweight category entered the second model and remained in the final model. Children's cumulative soda-pop intake entered the third model, but dropped significance when mother's education was added. Neither parental BMIs nor family income were significant additions to the models, leaving a final model with the children's 'at risk' of overweight status and mother's education as predictors of children's caries experience at 4.5–6.9 years of age. This final model suggests that,

Table 5. Logistic regression models[a] predicting children's caries experience ($n = 246$) at 4.5–6.9 years of age

Exposure variables	Odds ratio (95% CI)[b]	C[c]
Model 1		
Age at dental examination (years)	5.41 (2.24, 13.09)***	0.637
Fluoride intake (mg)	0.64 (0.24, 1.67)	
Model 2		
Age at dental examination (years)	6.18 (2.46, 15.51)***	0.683
Fluoride intake (mg)	0.67 (0.25, 1.74)	
Children 'at risk' of overweight	3.07 (1.51, 6.25)**	
Model 3		
Age at dental examination (years)	6.40 (2.54, 16.10)***	0.715
Fluoride intake (mg)	0.66 (0.24, 1.76)	
Children 'at risk' of overweight	3.20 (1.56, 6.58)**	
Soda-pop, 1–5 years (g)	1.01 (1.00, 1.01)*	
Final model		
Age at dental examination (years)	5.44 (2.11, 14.03)***	0.724
Fluoride intake (mg)	0.75 (0.29, 1.99)	
Children 'at risk' of overweight	3.02 (1.46, 6.25)**	
Mother's education level[d]	0.73 (0.58, 0.91)**	

[a]All models included age at dental exam and cumulative fluoride intake. Sequential models were developed by entering the variables that were significant in univariate analyses in the following order: children's body mass index (BMI) category, children's dietary variables, parental education, parental BMI and family income.
[b]Confidence interval.
[c]The area under the receiver–operating characteristic curve (C) is a measure of overall concordance between observed outcome (caries experience) and predicted values based on the logistic model.
[d]Educational levels: 3 = high school/GED; 4 = some college; 5 = associate degree; 6 = bachelor's degree.
*$P < 0.05$; **$P < 0.01$; ***$P < 0.001$.

for children at the median age of examination with median fluoride intakes and median maternal education levels, being 'at risk' of overweight doubled their caries risk (to 40% prevalence) compared with children who were normal or overweight (18% prevalence) (data not shown).

Discussion

Our observation that children 'at risk' of overweight have higher rates of caries experience than their 'normal'-weight peers is similar to reports by other investigators (5–7). The data are also consistent with the hypotheses that a common risk factor drives both disease processes and suggest a very complex picture.

Our data do not, however, explain the distribution of caries experience observed across our BMI categories. It is well established that obesity has both genetic and environmental components (35–37); and distinguishing between the contributions of each in an observational study is not possible. Children who present as overweight early in life could have a stronger genetic predisposition to obesity (38, 39), but one cannot rule out environmental factors. However, having been identified as 'overweight' early in life could have caught the attention of healthcare providers and parents who instituted restrictive dietary measures – measures which could have decreased caries risk of our 'overweight' subjects without impacting genetically predisposed obesity. In contrast, children identified as 'at risk' of overweight at age 5 years could be exhibiting the cumulative environmental effects of dietary factors; the same dietary factors could be responsible for the higher rates of tooth decay observed in these subjects.

Measures of SES (i.e. parental education and family income) were associated with both caries experience and obesity in our subjects, suggesting that our subjects are similar to those studied by others (14, 40–44). As we have reported previously, soda-pop intakes were predictive of caries in our subjects; neither energy nor 100% juice intakes were associated with caries (12). In a national study of US adults, Heller et al. (45) also reported increased caries risk with soda-pop, but not 100% juice. We did not find consistent associations between dietary measures examined (i.e. energy, soda-pop and 100% juice) and obesity in our subjects. These data are consistent with the observations of Skinner et al. (46) who did not find significant associations between 100% juice and measures of obesity in children aged 24–32 months, and Newby et al. (47) who did not find

significant associations between milk, soda-pop, 100% juice or fruit drink intakes and obesity measures in children 2–5 years. In contrast, Troiano et al. (48) reported higher proportions of energy from soft drinks by overweight children using data from multiple NHANES. Small, chronic increases in energy intakes leading to obesity are difficult to detect, and studies supporting associations between childhood obesity and energy intake are conflicting (49–51).

Low SES as measured by limited education and incomes has been associated with food insecurity; Mexican–American children residing in low SES households are less likely to meet the Food Guide Pyramid guidelines than other children (21). Vereecken et al. (52) reported that less-educated mothers were more likely to consume soft drinks and to permit their children to consume soft drinks than highly educated mothers. Skinner et al. (46) reported an inverse association between soda-pop intakes and parental ages. These reports are consistent with our data; dietary intakes of soda-pop by our subjects were associated with SES.

Our outcome variables were cross-sectional, which limits the ability to identify causative factors. However, the sum of the data, which is consistent with that of other investigators, suggests a complex scenario with a powerful public health message. First, caries and 'at risk' status coexisted in our subjects. Secondly, both 'at risk' status and soda-pop intakes were predictive of caries in a multivariate model adjusted for age at examination and fluoride intake. Finally, a measure of SES, mother's education, was a stronger predictor of caries than soda-pop intakes in the final model. The mechanism by which SES exerts this effect could be through inadequate knowledge of appropriate food choices and limited ability to procure such foods.

Limitations of this study include its cross-sectional nature and self-reporting of dietary data. Measures of SES were assessed once at the beginning of the study and could have changed during the ensuing years. Obesity is a complex issue with multiple etiological factors; our analyses were limited to genetic, dietary and demographic characteristics.

Conclusions

Bivariate analyses suggest that low SES increased the risk of both caries and obesity and could be responsible for their apparent coexistence in childhood, while multiple regression analyses suggest that SES plays a role in caries development that is independent of the role played by obesity. Public health measures designed to improve both dietary education and access to appropriate foodstuffs could decrease the dual disease burdens of childhood.

Acknowledgments

This study was supported by the ATPM/CDC (TS-0652), the NIDCR (RO1-DE09551 and RO1-DE12101) and GCRCP (M01-RR00059). Portions of the results of this study were presented at the 82nd General Session of the IADR in Baltimore, MD on March 10, 2005. The contents are the responsibility of the authors and do not necessarily reflect the official views of the granting organizations.

References

1. U S Department of Health and Human Services. Oral health in America: a report of the Surgeon General—executive summary. Rockville, MD: US Department of Health and Human Services, National Institute of Dental and Craniofacial Research, National Institutes of Health; 2000.
2. Glick M. A job well done, but still a long way to go. J Am Dent Assoc 2005;136:1506–8.
3. Hedley AA, Ogden CL, Johnson CL, Carroll MD, Curtin LR, Flegat KM. Prevalence of overweight and obesity among US children, adolescents, and adults, 1999–2002. J Am Med Assoc 2004;291:2847–50.
4. Ogden CL, Flegal KM, Carroll MD, Johnson CL. Prevalence and trends in overweight among US children and adolescents, 1999–2000. J Am Med Assoc 2002;288:1728–32.
5. Reifsnider E, Mobley C, Mendez DB. Childhood obesity and early childhood caries in a WIC population. J Multicultural Nurs Health 2004;10:24–31.
6. Tuomi T. Pilot study on obesity in caries prediction. Community Dent Oral Epidemiol 1989;17:289–91.
7. Willerhausen B, Haas G, Krummenauer F, Hohenfellner K. Relationship between high weight and caries frequency in German elementary school children. Eur J Med Res 2004;9:400–4.
8. Campain AC, Morgan MV, Evans RW, Ugoni A, Adams, GG, Conn JA et al. Sugar-starch combinations in food and the relationship to dental caries in low-risk adolescents. Eur J Oral Sci 2003;111:316–25.
9. Lingstrom P, van Houte J, Kashket S. Food starches and dental caries. Crit Rev Oral Biol Med 2000;11:366–80.
10. Gustaffson BE, Quensel CE, Lanke LS, Lundqvist C, Grahnén H, Bonow BE et al. The Vipeholm Dental Caries Study. Acta Odontol Scand 1954;11:232–364.

11. Woodward M, Walker ARP. Sugar consumption and dental caries: evidence from 90 countries. Br Dent J 1994;176:297–302.
12. Marshall TA, Levy SM, Broffitt B, Warren JJ, Eichenberger-Gilmore JM, Burns TL et al. Dental caries and beverage consumption in young children. Pediatrics 2003;112:e184–91.
13. Marshall TA, Broffitt B, Eichenberger-Gilmore J, Warren JJ, Cunningham MA, Levy SM. The roles of meal, snack and daily total food and beverage exposures on caries experience in young children. J Public Health Dent 2005;65:166–73.
14. Vargas CM, Crall JJ, Schneider DA. Sociodemographic distribution of pediatric dental caries: NHANES III, 1988–1994. J Am Dent Assoc 1998;129:1229–38.
15. Drewnowski A, Specter SE. Poverty and obesity: the role of energy density and energy costs. Am J Clin Nutr 2004;79:6–16.
16. Wardle J, Steptoe A. Socioeconomic differences in attitudes and beliefs about healthy lifestyles. J Epidemiol Community Health 2003;57:440–3.
17. Carlos Poston WS II, Foreyt JP. Obesity is an environmental issue. Atherosclerosis 1999;146:201–9.
18. Nicklas TA, Baranowski T, Cullen KW, Berenson G. Eating patterns, dietary quality and obesity. J Am Coll Nutr 2001;20:599–608.
19. Mattess RD, Hollis J, Hayes D, Stunkard AJ. Appetite: measurement and manipulation misgivings. J Am Diet Assoc 2005;105:S87–97.
20. Cordain L, Eaton SB, Sebastian A, Mann N, Lindeberg S, Watkins BA et al. Origins and evolution of the western diet: health implications for the 21st century. Am J Clin Nutr 2005;81:341–54.
21. Kaiser LI, Melgar-Quiñonez HR, Lamp CL, Johns MC, Sutherlin JM, Harwood JO. Food security and nutritional outcomes of preschool-age Mexican-American children. J Am Diet Assoc 2002;102:924–9.
22. Ludwig DS, Peterson KE, Gortmaker SL. Relationship between consumption of sugar-sweetened drinks and childhood obesity: a prospective, observational analysis. Lancet 2001;357:505–8.
23. Berkey CS, Rockett HRH, Field AE, Gillman MW, Colditz GA. Sugar-added beverages and adolescent weight change. Obes Res 2004;12:778–88.
24. Wang Y. Cross-national comparison of childhood obesity: the epidemic and relationship between obesity and socioeconomic status. Int J Epidemiol 2001;30:1129–36.
25. Danielzik S, Czerwinski-Mast M, Langnäse K, Dilba B, Müller MJ. Parental overweight, socioeconomic status and high birth weight are the major determinants of overweight and obesity in 5–7 y-old children: baseline data of the Kiel Obesity Prevention Study (KOPS). Int J Obes 2004;28:1494–592.
26. Drenowski A, Darmon N. The economics of obesity: dietary energy density and energy cost. Am J Clin Nutr 2005;82:265S–73S.
27. Warren JJ, Levy SM, Kanellis MJ. Dental caries in the primary dentition: assessing prevalence of cavitated and noncavitated lesions. J Public Health Dent 2002;62:109–14.
28. Warren JJ, Levy SM, Kanellis MJ. Prevalence of dental fluorosis in the primary dentition. J Public Health Dent 2001;61:87–91.
29. Eichenberger Gilmore JM, Hong L, Broffitt B, Levy SM. Longitudinal patterns of vitamin and mineral supplement use in young white children. J Am Diet Assoc 2005;105:763–72.
30. Marshall TA, Eichenberger-Gilmore JM, Broffitt B, Stumbo PJ, Levy SM. Diet quality in young children is influenced by beverage consumption. J Am Coll Nutr 2005;24:65–75.
31. Levy SM, Warren JJ, Davis CS, Kirchner HL, Kanellis MJ, Wefel JS. Patterns of fluoride intake from birth to 36 months. J Public Health Dent 2001;61:70–7.
32. Levy SM, Warren JJ, Broffitt B. Patterns of fluoride intake from 36 to 72 months of age. J Public Health Dent 2003;63:211–20.
33. Marshall TA, Levy SM, Warren JJ, Broffitt B, Eichenberger-Gilmore JM, Stumbo PJ. Associations between intakes of fluoride from beverages during infancy and dental fluorosis of primary teeth. J Am Coll Nutr 2004;23:108–16.
34. Centers for Disease Control and Prevention, U.S. Department of Health and Human Services. National Center for Health Statistics Clinical Growth Charts. 2005. Available at: http://www.cdc.gov/nchs/about/major/nhanes/growthcharts/clinical_charts.htm (Accessed December 2005).
35. Whitaker RC, Wright JA, Pepe MS, Seidel KD, Dietz WH. Predicting obesity in young adulthood from childhood and parental obesity. N Engl J Med 1997;337:869–73.
36. Clement K. Genetics of human obesity. Proc Nutr Soc 2005;64:133–42.
37. Wardle J. Understanding the aetiology of childhood obesity: implications for treatment. Proc Nutr Soc 2005;64:73–9.
38. Mamun AA, Lawlor DA, O'Callaghan MJ, Williams GM, Najman JM. Family and early life factors associated with changes in overweight status between ages 5 and 14 years: findings from the Mater University Study of Pregnancy and its outcomes. Int J Obes Relat Metab Disord 2005;29:475–82.
39. Berkowitz RI, Stallings VA, Maislin G, Stunkard AJ. Growth of children at high risk of obesity during the first 6 y of life: implications for prevention. Am J Clin Nutr 2005;81:140–6.
40. Perez MA, Latorre MRDO, Sheiham A, Peres KGA, Barros FC, Hernandez PG et al. Social and biological early life influences on severity of dental caries in children aged 6 years. Community Dent Oral Epidemiol 2005;33:53–63.
41. Antunes JLF, Narvai PC, Nugent ZJ. Measuring inequalities in the distribution of dental caries. Community Dent Oral Epidemiol 2004;32:41–8.
42. Anderson PM, Butcher KF, Levine PB. Maternal employment and overweight children. J Health Econ 2003;22:477–504.
43. Moreno LA, Tomas C, Gonzalez-Gross M, Bueno G, Perez-Gonzalez JM, Bueno M. Micro-environmental and socio-demographic determinants of childhood obesity. Int J Obes Relat Metab Disord 2004;28: S16–20.
44. Willems S, Vanobbergen J, Martens L, Maeseneer JD. The independent impact of household-and neighborhood-based social determinants on early childhood caries: A cross-sectional study of inner-city children. Fam Community Health 2005;28:168–75.

45. Heller KE, Burt BA, Eklund SA. Sugared soda consumption and dental caries n the United States. J Dent Res 2001;80:1949–53.
46. Skinner JD, Carruth BR, Moran III J, Houck K, Coletta F. Fruit juice intake is not related to children's growth. Pediatrics 1999;103:58–64.
47. Newby PK, Peterson KE, Berkey CS, Leppert J, Willett WC, Colditz GA. Beverage consumption is not associated with changes in weight and body mass index and low-income preschool children in North Dakota. J Am Diet Assoc 2004;104:1086–94.
48. Troiano RP, Briefel RR, Carroll MD, Bialostosky K. Energy and fat intakes of children and adolescents in the United States: data from the National Health and Nutrition Examination Surveys. Am J Clin Nutr 2000;72:1343S–53S.
49. McGloin AF, Livingstone MB, Greene LC, Webb SE, Gibson JM, Jebb SA et al. Energy and fat intake in obese and lean children at varying risk of obesity. Int J Obes Relat Metab Disord 2002;26:200–7.
50. Grant AM, Ferguson EL, Toafa V, Henry TE, Guthrie BE. Dietary factors are not associated with high levels of obesity in New Zealand Pacific preschool children. J Nutr 2004;134:2561–5.
51. Magarey AM, Daniels LA, Boulton TJC, Cockington RA. Does fat intake predict adiposity in healthy children and adolescents aged 2–15 y? A longitudinal analysis. Eur J Clin Nutr 2001;55:471–81.
52. Vereecken CA, Keukelier E, Maes L. Influence of mother's educational level on food parenting practices and food habits of young children. Appetite 2004;43:93–103.