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Robert J. Schroth, Christopher Lavelle, Robert Tate, Sharon Bruce, Ronald J. Billings and Michael E.K. Moffatt

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Prenatal Vitamin D and Dental Caries in Infants

AUTHORS: Robert J. Schroth, DMD, MSc, PhD,[a,b,c] Christopher Lavelle, DDS, PhD, DSc, MBA, FRCPath,[d] Robert Tate, PhD,[e] Sharon Bruce, BN, MA, PhD,[e] Ronald J. Billings, DDS, MSD,[f] and Michael E.K. Moffatt, MD, MSc, FRCPC[c,e]

Departments of [a]Preventive Dental Science, Faculty of Dentistry, [b]Pediatrics and Child Health, Faculty of Medicine, [d]Oral Biology, Faculty of Dentistry, [e]Community Health Sciences, Faculty of Medicine, University of Manitoba, Winnipeg, Manitoba, Canada; [c]The Manitoba Institute of Child Health, Winnipeg, Manitoba, Canada; [f]Division of Community Dentistry and Oral Disease Prevention, Eastman Institute for Oral Health, School of Medicine and Dentistry, University of Rochester, Rochester, New York

KEY WORDS
early childhood caries, enamel hypoplasia, infant, vitamin D

ABBREVIATIONS
DT—decayed teeth
ECC—early childhood caries
S-ECC—severe early childhood caries
25OHD—25-hydroxyvitamin D

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Address correspondence to Robert J. Schroth, DMD, MSc, PhD, Department of Preventive Dental Science (Faculty of Dentistry), Department of Pediatrics and Child Health (Faculty of Medicine), University of Manitoba, 507–715 McDermot Ave, Winnipeg, Manitoba, R3E 3P4. E-mail: umschrot@cc.umanitoba.ca

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WHAT'S KNOWN ON THIS SUBJECT: Many young children are at risk for caries, which is the most common chronic disease of childhood. As primary teeth begin to develop in utero, prenatal influences are believed to affect the integrity of enamel and subsequent resistance to decay.

WHAT THIS STUDY ADDS: This study shows, for the first time, that maternal prenatal 25-hydroxyvitamin D levels may have an influence on the primary dentition and the development of early childhood caries. Specifically, lower levels are associated with increased risk of caries in infants.

abstract

OBJECTIVES: Inadequate maternal vitamin D (assessed by using 25-hydroxyvitamin D [25OHD]) levels during pregnancy may affect tooth calcification, predisposing enamel hypoplasia and early childhood caries (ECC). The purpose of this study was to determine the relationship between prenatal 25OHD concentrations and dental caries among offspring during the first year of life.

METHODS: This prospective cohort study recruited expectant mothers from an economically disadvantaged urban area. A prenatal questionnaire was completed and serum sample drawn for 25OHD. Dental examinations were completed at 1 year of age while the parent/caregiver completed a questionnaire. The examiner was blinded to mothers' 25OHD levels. A P value $\leq .05$ was considered significant.

RESULTS: Overall, 207 women were enrolled (mean age: 19 $\pm$ 5 years). The mean 25OHD level was 48 $\pm$ 24 nmol/L, and 33% had deficient levels. Enamel hypoplasia was identified in 22% of infants; 23% had cavitated ECC, and 36% had ECC when white spot lesions were included in the assessment. Mothers of children with ECC had significantly lower 25OHD levels than those whose children were caries-free (41 $\pm$ 20 vs 52 $\pm$ 27 nmol/L; $P = .05$). Univariate Poisson regression analysis for the amount of untreated decay revealed an inverse relationship with maternal 25OHD. Logistic regression revealed that enamel hypoplasia ($P < .001$), infant age ($P = .002$), and lower prenatal 25OHD levels ($P = .02$) were significantly associated with ECC.

CONCLUSIONS: This study found that maternal prenatal 25OHD levels may have an influence on the primary dentition and the development of ECC. *Pediatrics* 2014;133:e1277–e1284

Tooth decay in infants and preschool-aged children is called early childhood caries (ECC). Although dental caries is the most common chronic disease of childhood,[1] it is declining in the general population. This finding, however, is not the case for certain groups such as Aboriginal children. For many at-risk groups, the rampant extent of decay exhibited is called severe early childhood caries (S-ECC).

ECC and S-ECC are multifactorial in origin and are influenced by biomedical factors (microorganisms, diet, and tooth resistance) and the social determinants of health.[2] One proposed explanation for the burden of S-ECC in some children is hypoplasia-associated S-ECC.[3] Enamel hypoplasia results from defective amelogenesis and is clinically identified by the absence of enamel and by pitting, grooves, or irregularities of enamel.[4,5] These defects increase the risk of early colonization by cariogenic bacteria, resulting in caries.[3,6,7] Therefore, the period when enamel forms is crucial to understanding the significance of enamel hypoplasia and risk for ECC. The primary maxillary anterior teeth begin to calcify during the second trimester (specifically, 13–17 weeks) and continue until 3 months' postnatal.[8] It is therefore important to investigate possible factors that can disrupt enamel formation and increase the risk of caries.

Enamel defects have been correlated with factors ranging from genetic disorders to problems during prenatal and early postnatal periods.[9] Vitamin D deficiency in utero is believed to be associated with enamel hypoplasia because of the metabolic insult to ameloblasts.[10–12] Vitamin D plays a central role in calcium and phosphorus homeostasis, which is needed for the calcification of hard tissues.[13]

The pioneering efforts of Mellanby[14,15] gave credence to the belief that the critical period for influencing the development of the primary dentition is in utero. Because the duration of primary tooth calcification is short and begins during the second trimester, prenatal nutrition has a tremendous influence on the formation of dental tissues.[16]

Although some clinicians are unfamiliar with research on vitamin D in caries and enamel hypoplasia, several historical studies have reported that vitamin D supplementation may prevent caries in children.[17–20] A recent meta-analysis of these studies confirmed the early findings.[21] New research suggests that vitamin D plays a role in the human immune response[22] and may reduce host resistance to cariogenic bacteria.

The purpose of the present study was to test the hypothesis of an association between lower maternal prenatal 25-hydroxyvitamin D (25OHD) status and the presence of ECC in the infant.

METHODS

A prospective cohort study was designed to investigate the association between prenatal vitamin D concentrations and dental caries in infants in a vulnerable urban population. Participants were recruited during the second or early third trimester after providing written informed consent. The target population was expectant women presenting for prenatal care in Winnipeg, Canada (latitude 49°53′N).

A serum sample was collected as part of a prenatal visit during the second or early third trimester, as the primary maxillary incisors begin to calcify during weeks 13 to 17 in utero and continue to do so throughout pregnancy. Serum analysis was conducted at Winnipeg's Health Sciences Centre. Samples were analyzed for levels of 25OHD, total calcium, inorganic phosphorus, and alkaline phosphatase (elevated levels indicate vitamin D insufficiency[14]). 25OHD, a reliable measure of overall vitamin D status,[23] was assessed via radioimmunoassay by using a DiaSorin kit (DiaSorin, Inc, Stillwater, MN). 25OHD concentrations <35 nmol/L were considered deficient and those ≥ 75 nmol/L were optimal.

Participants completed a questionnaire proctored by the principal investigator (Dr Schroth) or clinic staff. This instrument was based on a tool that assessed nutritional deficiencies in northern Manitoba.[24] The questionnaire was modified with input from researchers and clinicians, including a dietitian. Information was collected on demographic characteristics (eg, age, ethnicity, education level), pregnancy (eg, prenatal health status, parity, use of prenatal vitamins), health conditions, nutrition (eg, intake of milk, dairy, fish, eggs, meat), and awareness of ECC (eg, heard of ECC, older children had ECC, what causes ECC). Exposure to sunlight (eg, time spent in sunshine in summer), family composition (eg, relational status, family size), finances, and employment were also assessed.

The final component of the study was an assessment of the primary dentition, with the examiner (Dr Schroth) blinded to the prenatal 25OHD level of each infant's mother. Infants' teeth were assessed for caries by using established recommendations.[25] ECC and S-ECC were defined according to recognized definitions.[25] Incipient and noncavitated caries of enamel (white spot lesions) were recorded. Individual cumulative totals of the number of decayed, extracted, and filled primary teeth and cumulative totals of the number of decayed primary teeth (decayed teeth [dt] score) were determined. Developmental defects of enamel were assessed according to an established index for recording enamel defects such as hypoplasia and opacities.[5]

A follow-up questionnaire collecting information on demographic characteristics (eg, child's gender, age), household finances, birth weight, prematurity, feeding practices (eg, breastfeeding, bottle-feeding, introduction of solids), and infant health status was administered at the time of the infant's

examination. Caregivers were also asked about the age of the eruption of the first tooth, oral hygiene practices, and whether their child had visited the dentist.

The estimated sample size was reviewed by a biostatistician and validated by using PASS version 6.0 (NCSS, Kaysville, UT) based on prevalence data of prenatal vitamin D status in Manitoba.[24] The minimum sample size was doubled to allow for some loss while maintaining an adequate sample size.

Clinical and questionnaire data were entered into a Microsoft Office Access database (Microsoft Corporation, Redmond, WA) and analyzed by using NCSS version 2007 (Kaysville, UT) and SPSS version 17.0 (IBM SPSS Statistics, IBM Corporation, Armonk, NY). Analysis included descriptive statistics (frequencies and mean ± SD values). Bivariate analysis included χ^2 analysis, *t* tests, correlation, Poisson regression, and analysis of variance. Multiple logistic regression for ECC and Poisson regression for dt scores were used. Poisson regression is appropriate for count data, such as caries scores in infants.

Separate regression models for ECC were fit to identify important independent variables within sets of 5 themes, including serum metabolites (eg, 25OHD, calcium, alkaline phosphatase, phosphorus), factors influencing vitamin D status (eg, milk intake, margarine use, prenatal vitamin use, season, vitamin D drops), infant feeding practices (eg, bottle-feeding, breastfeeding, use of sippy cup), socioeconomic factors (eg, income, employment status), and dental status and dental behaviors (eg, siblings with ECC, tooth brushing, dental problem, age at dental examination, believing ECC is preventable). A final model was constructed, including independent variables significantly associated or approximating the threshold of significance with ECC in these separate models, in addition to variables routinely reported to be associated with ECC in the literature. For continuous variables, odds ratios and confidence intervals were calculated to reflect a change in 1 SD unit of the variable. For example, the odds ratio for 25OHD reflected a 1 SD unit change. A *P* value ≤.05 was considered significant.

RESULTS

A total of 207 women were enrolled (mean age: 19 ± 5 years). The majority (82%) were recruited from the Health Sciences Centre, and 93% resided in Winnipeg. Although 71% reportedly took vitamins during pregnancy, only 37% did so daily. Characteristics of participants and their offspring are given in Table 1.

Complete laboratory results were available for 200 participants (Table 2). The mean 25OHD level was 48 ± 24 nmol/L (median: 43 nmol/L); 65 (32.5%) participants had deficient concentrations (<35 nmol/L), and 24 (12%) had optimal levels (≥75 nmol/L). Participants undergoing blood draws during winter months (November–April) had significantly lower levels than those sampled during summer periods (May–October): 38 ± 22 versus 55 ± 24 nmol/L ($P < .001$). When stratified according to season, there was no significant difference in 25OHD concentrations between those who spent time outside in the sunshine and those who did not (Table 1).

Despite losses to follow-up, 64% ($n = 133$) of the cohort returned for the infant follow-up visit. There were no differences in age ($P = .24$), level of education ($P = .74$), or ethnic heritage ($P = .24$) between women lost to follow-up and those remaining in the study. Furthermore, there was no difference in the 25OHD levels between these 2 groups (50 ± 26 vs 45 ± 20 nmol/L; $P = .08$). A total of 135 infants (2 sets of twins) with a mean age of 16 ± 7 months (median: 13 months) returned. Overall, 56% of infants were male.

Enamel hypoplasia was identified in 22% of the cohort (29 of 134), the main forms being pits and missing enamel. Thirty-one infants (23%) had ECC when caries was restricted to cavitated enamel lesions. However, when white spot lesions of enamel were included, 49 infants (36%) had ECC. The mean dt score was 1.2 ± 2.1 (range: 0–10), whereas the mean score for the number of decayed, extracted, and filled teeth was 1.5 ± 2.8 (range: 0–17). When white spot incipiencies were included, the dt score was 3.4 ± 2.0.

Table 3 shows the relationship between mean 25OHD levels and ECC. Mothers of infants with ECC, based on the presence of cavitated caries lesions, had significantly lower prenatal concentrations of 25OHD than those whose children were caries-free ($P = .05$). However, when white spot lesions were included, there was no difference between groups. No significant associations were found between mothers' 25OHD concentrations and ECC in their infants when the deficient (<35 nmol/L) or optimal (≥75 nmol/L) thresholds were applied ($P = .36$ and $P = .38$, respectively).

Poisson regression revealed a significant inverse relationship between the average number of decayed teeth (dt score) and prenatal 25OHD levels ($P = .0002$) (Fig 1). Infants of mothers with lower 25OHD concentrations during pregnancy had significantly higher dt scores. The *t* test analyses were undertaken to assess the relationship between the number of dt scores with 25OHD thresholds for deficient and optimal concentrations. There was no significant difference in the mean number of primary teeth with decay among infants of mothers with 25OHD concentrations ≤35 nmol/L or >35 nmol/L (Table 4). Interestingly, infants of mothers who had optimal 25OHD levels (≥75 nmol/L) had a statistically lower mean dt score than those with mothers who had levels below this threshold ($P = .03$).

Mothers of infants with ECC were significantly more likely to be Aboriginal

TABLE 1 Maternal and Infant Characteristics and Associations With Maternal Prenatal 25OHD Levels

Variable	Total No. in Cohort[a]	Prenatal 25OHD Levels, Mean ± SD (nmol/L)	*P*
Maternal characteristics			
Mean age, y	19 ± 5	–	–
Resided in Winnipeg			.42
Yes	190 (93)	48 ± 25	
No	15 (7)	50 ± 23	
Canadian Aboriginal (First Nations, Métis, or Inuit)			<.001
Yes	186 (90)	46 ± 22	
No	20 (10)	69 ± 33	
Self-rated prenatal health status[b]			.03
Good	130 (64)	52 ± 24[c]	
Average	70 (34)	42 ± 24	
Poor	5 (2)	46 ± 19	
Primigravid			.87
Yes	125 (61)	48 ± 24	
No	81 (39)	49 ± 26	
Drink milk[b]			<.001
Often (daily)	103 (50)	56 ± 26[d]	
Sometimes (>1 time per week)	68 (33)	42 ± 21	
Rarely (<1 time per week)	20 (10)	34 ± 16	
Never	15 (7)	43 ± 23	
Daily vitamin use			<.001
Yes	74 (37)	57 ± 26	
No	125 (63)	44 ± 22	
Identified food(s) containing vitamin D[b]			.50
Yes	44 (22)	52 ± 32	
No	28 (14)	49 ± 25	
Do not know	130 (64)	47 ± 21	
Education level			.02
<High school	190 (92)	47 ± 24	
≥High school	16 (8)	62 ± 31	
Annual income, $			.30
≤18 000	196 (95)	48 ± 23	
>18 000	10 (5)	61 ± 40	
Had heard of ECC or antecedent term(s)			.07
Yes	159 (77)	47 ± 24	
No	47 (23)	53 ± 25	
Self-rating of dental health[b]			.01
Good	79 (38)	55 ± 29[e]	
Fair	100 (49)	44 ± 20	
Poor	26 (13)	47 ± 21	
Sun exposure (May–October)			.23
Spent time outside in sunshine	9 (7)	64 ± 34	
Did not spend time outside in sunshine	117 (93)	54 ± 23	
Infant characteristics			
Gender			.24
Male	75 (56)	52 ± 28	
Female	60 (44)	47 ± 24	
Premature			.43
Yes	17 (13)	54 ± 33	
No	117 (87)	49 ± 25	
Low birth weight			.96
Yes	6 (5)	49 ± 25	
No	124 (95)	49 ± 26	
Mean birth weight, g	3490 ± 561	/	/
Breastfed			.15
Yes	97 (74)	51 ± 25	
No	35 (26)	44 ± 29	
Bottle-fed			.29
Yes	130 (96)	49 ± 25	
No	5 (4)	83 ± 54	

(*P* = .02), rate their own health as average or poor (*P* = .01), have other children with ECC (*P* < .005), and consume milk less frequently during pregnancy (*P* = .01). Furthermore, they were more likely to use food banks and have low incomes (*P* < .005).

The χ^2 analysis revealed that infants with enamel hypoplasia were significantly more likely to have ECC (73% vs 27%; *P* < .001). Children with ECC were significantly older than those who were caries-free (19 ± 10 vs 14 ± 4 months; *P* = .001). ECC was not significantly associated with bottle-feeding (*P* = .86) or breastfeeding (*P* = .35). Among the 21 infants who were reported to have stopped bottle-feeding, there was no difference between the presence and absence of ECC and weaning age (*P* = .051). However, those who were still using sippy cups were significantly less likely to have ECC (*P* = .001).

A significantly smaller proportion of the 117 children whose teeth were being cleaned had ECC (*P* = .02). However, there was no significant difference between groups in the mean age when caregivers began to clean their infants' teeth (*P* = .07).

Regression analyses were performed to assess relationships between independent variables and the dependent outcomes of ECC and dt scores. These models addressed the themes of prenatal serum metabolite concentrations, factors influencing vitamin D attainment, infant feeding practices, family characteristics and finances, enamel hypoplasia and family dental history and awareness (models not shown). An overall final logistic regression model for ECC was constructed incorporating 12 different variables (Table 5). Some were significantly associated with ECC in earlier models or approximated the threshold of significance, whereas others were either significant at the bivariate level or were commonly identifiable contributors to ECC risk in the literature. Some other variables that influence vitamin D status were also included. Results revealed

TABLE 1 Continued

Variable	Total No. in Cohort[a]	Prenatal 25OHD Levels, Mean ± SD (nmol/L)	*P*
Mean age at eruption of first tooth, mo	6 ± 2	–	–
Health rating by caregiver[b]			.90
Very good	75 (56)	50 ± 24	
Good	51 (38)	49 ± 30	
Fair	8 (6)	46 ± 21	

[a] Unless otherwise noted, data are no. in cohort (% if applicable).
[b] Analysis of variance.
[c] Significantly differs from average.
[d] Significantly differs from sometimes and rarely.
[e] Significantly differs from fair.

TABLE 2 Prenatal Serum Concentrations of 25OHD and Related Metabolites

Assay	Range of Normal[a] Values	*N*	Mean ± SD	Range
25OHD, nmol/L	35–105 (winter), 37–200 (summer)	200	48 ± 24	5–145
25OHD deficiency, nmol/L				
<35		65	25 ± 6	5–34
≥35		135	59 ± 22	35–145
Calcium, mmol/L	2.10–2.60	198	2.25 ± 0.10	2.01–2.57
Phosphate, mmol/L	1.29–2.26 (<17 y) 0.81–1.45 (>16 y)	200	1.15 ± 0.19	0.69–2.28
Alkaline phosphatase, U/L	59–422 (≤17 y) 30–120 (>17 y)	200	98 ± 52	34–372

[a] Department of Biochemistry and Genetics Laboratory reference values.

that the presence of enamel hypoplasia ($P = .001$) and the age of infants at the time of the dental examination ($P = .01$) were significantly associated with ECC, with those aged ≥14 months at the time of their examination being more likely to have ECC. Furthermore, 25OHD levels during pregnancy were found to be significantly associated with ECC ($P = .05$). Backward logistic regression analysis was also performed, with the final iteration revealing that enamel hypoplasia ($P < .001$), infant age ($P = .001$), and lower 25OHD levels ($P = .02$) were significantly and independently associated with ECC after controlling for income and employment status, infant feeding methods, season, and infant oral hygiene practice.

Poisson regression for the dt score was performed including the same independent variables that appeared in the expanded logistic regression model for ECC. Similarly, results revealed that infant age, the presence of enamel hypoplasia, and maternal 25OHD levels during pregnancy were significantly associated with dt score (Table 6). Lower 25OHD levels and lower ratings of childhood health were associated with higher dt scores ($P = .04$).

TABLE 3 Relationship Between Oral Health Outcomes and Maternal 25OHD

Caries Status	Maternal 25OHD			*P*
	N	Mean ± SD	Median	
ECC (cavitated lesions)				.05
Yes	30	41 ± 20	39	
No	103	52 ± 27	47	
ECC (including white spot lesions)				.18
Yes	48	46 ± 24	41	
No	85	52 ± 28	46	

Based on *t* test analysis.

DISCUSSION

Although some studies have reported associations between vitamin D status and dental caries,[10–12,26] no previous study, to the best of our knowledge, has prospectively examined the relationship between prenatal 25OHD levels during periods of tooth development and caries in offspring. Many women had suboptimal 25OHD levels; nearly 90% had levels below the threshold for adequacy. Suboptimal levels are associated with increased risk for many chronic diseases, including osteoporosis, cardiovascular disease, and periodontal disease.[27–29] Unfortunately, most women in this study would need to take >2000 IU of vitamin D daily to raise their levels to 80 nmol/L.[30] Daily doses of 400 IU of vitamin D_3 for 8 weeks results in an increase of only 11 nmol/L.[29]

Mothers of children with ECC had significantly lower levels of 25OHD than mothers of caries-free children. In addition, there was an inverse relationship between prenatal 25OHD and the dt score, with lower concentrations predicting higher scores of decayed primary teeth. Infants whose mothers had optimal prenatal 25OHD concentrations (≥75 nmol/L) had significantly lower dt scores.

Several early studies identified a connection between vitamin D–fortified diets and sun exposure, a lower incidence and extent of caries, and a decrease in the prevalence of enamel hypoplasia in permanent teeth.[31,32] A recent meta-analysis revealed that supplementation with vitamin D_2, vitamin D_3, or ultraviolet light lowered the risk for caries.[21] It has also been suggested that 25OHD concentrations between 75 and 100 nmol/L offer protection against caries.[33]

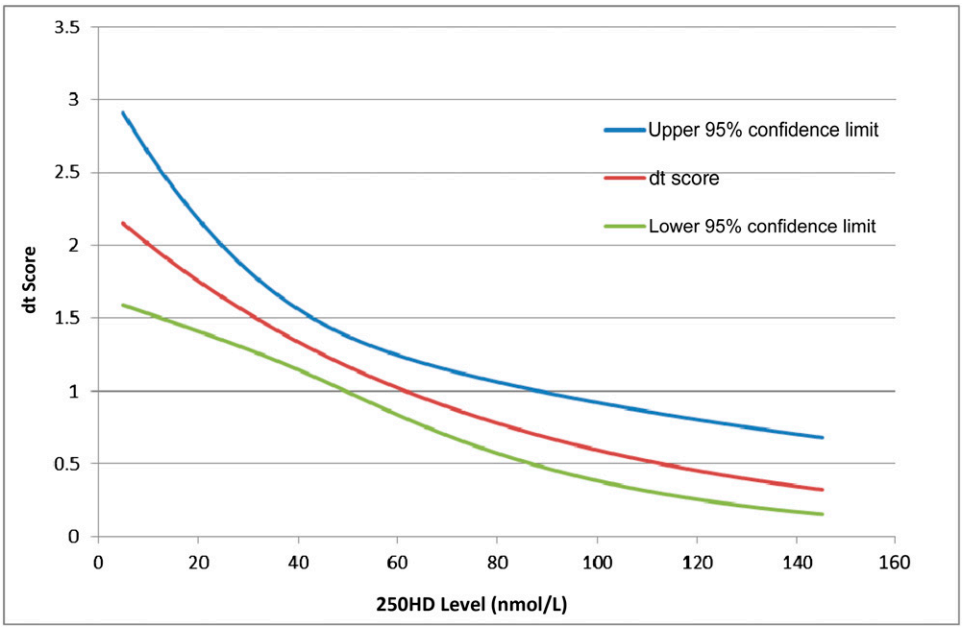

FIGURE 1
Predicted number of decayed primary teeth (dt score) according to 25OHD level.

A recent case-control study reported significantly lower 25OHD levels in children with S-ECC.[34] More recently, we have also reported similar findings in a larger sample of an association between S-ECC and 25OHD, even after controlling for season, milk intake, use of vitamins, and household income.[35] Although these 2 studies do not establish causation, they provide further evidence of an association between caries in early life and lower 25OHD concentrations.

This study highlights the incidence of ECC in a sample predominantly comprising Canadian Aboriginal children. Nearly one-quarter had ECC when cavitated lesions were considered and more than one-third when incipient lesions were included, far higher than urban-dwelling children of similar ages.[36] This finding is comparable to the 30.4% of Manitoba infants <24 months of age reported to have ECC.[37] Unfortunately, 23% of the infants in this study met the criteria for S-ECC.[25]

Regression modeling was necessary to control for the influence of confounders to determine whether prenatal 25OHD levels were associated with ECC. Because finances, poverty, and employment can influence the risk of caries, the final model incorporated several related variables. Age is a recognized predictor of ECC, and it was therefore also included in the model.[37] Those children examined at ≥14 months of age were at increased odds of having ECC. The model also accounted for infant feeding practices, oral hygiene, season, and milk intake. After controlling for these influences, 3 variables were significantly and independently associated with ECC: enamel hypoplasia, infant age (≥14 months), and 25OHD levels. Enamel hypoplasia was a strong predictor of ECC in our cohort (odds ratio >8), providing further credence to the theory of hypoplasia-associated S-ECC.[3,7]

The Poisson regression model for the untreated primary tooth decay score (dt) included the same independent variables incorporated in the expanded logistic regression model for ECC. Enamel hypoplasia, infant age, and 25OHD levels were again identified as independent predictors of caries scores. This blinded prospective study suggests that infants of mothers with lower 25OHD levels are

TABLE 4 Relationship Between dt Score and Maternal 25OHD Levels in Pregnancy

25OHD	dt Score		*P*
	N	Mean ± SD (Range)	
25OHD threshold, deficiency			.20
<35 nmol/L	44	1.6 ± 2.3 (0–10)	
≥35 nmol/L	88	1.1 ± 1.9 (0–9)	
25OHD threshold, Institute of Medicine			.32
<50 nmol/L	57	1.0 ± 1.9 (0–10)	
≥50 nmol/L	75	1.4 ± 2.1 (0–9)	
25OHD threshold, optimal			.03[a]
≥75 nmol/L	19	0.6 ± 1.2 (0–4)	
<75 nmol/L	113	1.4 ± 2.2 (0–10)	

[a] Aspin-Welch unequal-variance test.

TABLE 5 Logistic Regression for ECC (Excluding White Spot Lesions): Final Expanded Model

Variable	Regression Coefficient (b) (SE)	Adjusted Odds Ratio (95% CI)	*P*
Low annual income (reference: >$18 000)	−2.47 (1.49)	0.085 (0.005–1.57)	.1
Child health (reference: less than very good to good)	−0.61 (0.60)	0.55 (0.17–1.76)	.31
Infant's teeth being cleaned or brushed (reference: no)	1.29 (1.04)	3.63 (0.47–28.07)	.22
Drink milk (reference: not often)	−0.36 (0.60)	0.70 (0.21–2.29)	.55
Enamel hypoplasia (reference: no)	2.18 (0.67)	8.89 (2.40–32.87)	.001
No one with full-time employment in household (reference: no)	0.99 (0.91)	2.70 (0.45–16.24)	.28
Government assistance (reference: no)	−0.48 (0.60)	0.62 (0.19–1.99)	.42
Infant age at time of dental examination (reference: ≥14 mo)	−1.60 (0.62)	0.20 (0.06–0.68)	.01
Infant feeding (bottle) (reference: mixed)	0.25 (0.64)	1.28 (0.36–4.51)	.70
Infant feeding (breast) (reference: mixed)	−0.14 (1.62)	0.87 (0.04–20.63)	.93
Season (reference: summer)	−0.40 (0.62)	0.67 (0.20–2.27)	.52
25OHD level[a]	−0.029 (0.015)	2.02 (1.00–4.08)	.05

ECC reference = yes; R^2 = 32.9%. CI, confidence interval.

[a] SD in sample = 24.44.

TABLE 6 Poisson Regression for dt (Caries Tooth Score)

Variable	Regression Coefficient	±95% Confidence Interval	*P*
Intercept	1.68		
Low annual income (reference: <$18 000)	−0.28	0.73	.45
Child health (reference: less than very good to good)	−0.35	0.33	.04
Infant's teeth being cleaned or brushed (reference: no)	−0.13	0.51	.60
Drink milk (reference: not often)	0.054	0.36	.77
Enamel hypoplasia (reference: no)	1.02	0.37	<.001
No one with full-time employment in household (reference: no)	0.39	0.59	.20
Government assistance (reference: no)	0.13	0.38	.50
Infant age at time of dental examination (reference: ≥14 mo)	−1.03	0.38	<.001
Infant feeding (bottle-fed) (reference: mixed)	0.031	0.37	.87
Infant feeding (breastfed) (reference: mixed)	−0.53	0.81	.20
Season (reference: summer)	−0.32	0.42	.13
25OHD level	−0.013	0.0085	.002

significantly more likely to develop ECC. The exact mechanism is unclear, but it is likely that lower levels of vitamin D during tooth development result in enamel that is less resistant to caries.

Naturally, there were some limitations to the present study. Attrition of the cohort was expected, considering the life challenges faced by many of its participants. The cohort size restricted our ability to develop complex multivariate models. Although there were several losses to follow-up, there was no significant difference in 25OHD levels between those who remained in the study and those lost to follow-up. The questionnaires were also limited. In hindsight, they did not fully explore certain potential confounders that may have had an influence on infant oral health status, particularly caries risk.

The generalizability of our findings may also be limited. This study was not a random sample but rather one of convenience. However, the study provides insight into the nutritional status of expectant women and the oral health of their infants. We purposely targeted a high-risk population for both low vitamin D level and ECC, allowing this study to be generalizable to this urban Aboriginal population.

Overall, the study was of moderate size and deliberately involved a high-risk population of mostly urban Aboriginal subjects, with limited education levels and incomes. The prospective design allowed the natural history of caries to be observed and permitted multiple outcomes to be assessed for a single exposure (ie, 25OHD status). In addition, a temporal sequence was established, spanning pregnancy through infancy. Cohort studies also have the advantage of reduced bias. Another notable strength was that dental assessments were made while researchers were blinded to maternal 25OHD levels.

Findings from this study may have implications for early childhood oral health policy. Attempts to improve nutrition during tooth formation in utero and early childhood should be examined as a potential strategy to reduce the risk of caries. Prevention efforts should begin during pregnancy by bolstering maternal nutrition, either through improved dietary intake or supplementation with vitamin D.

CONCLUSIONS

This study shows, for the first time, that prenatal 25OHD levels may have an influence on the primary dentition and the development of ECC. Specifically, lower levels were associated with increased risk for dental caries in infants. Prenatal 25OHD levels, enamel hypoplasia, and infant age were independent predictors for caries.

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Robert J. Schroth, Christopher Lavelle, Robert Tate, Sharon Bruce, Ronald J. Billings and Michael E.K. Moffatt

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