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# Association of Maternal Vitamin D Deficiency with Early Childhood Caries

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**Abstract**

Alaska Native (AN) children experience one of the highest reported rates of severe early childhood caries (S-ECC). Serum vitamin D concentrations in AN childbearing women in the Yukon Kuskokwim Delta (YKD) region have decreased since the 1960s to currently low levels, related to a decrease in traditional marine diet. Recent studies suggest an association between prenatal vitamin D (25(OH)D) concentrations in mothers and S-ECC in their infants. We used independent *t* tests to analyze the influence of prenatal 25(OH)D levels in YKD AN mothers on S-ECC in their children using data collected in the Maternal Organics Monitoring Study (MOMS). Maternal 25(OH)D levels were assessed at prenatal visits and in cord blood. We queried electronic dental records to assess early childhood caries (ECC) status using highest decayed, missing, filled, primary teeth (dmft) scores at 12 to 59 mo of age. We examined prenatal and cord blood for 76 and 57 mother/infant pairs, respectively. Children 12 to 35 mo of age with "deficient" cord blood (25(OH)D <30 nmol/L) had a mean dmft score twice as high as children who were "nondeficient" at birth (9.3 vs. 4.7; $P = 0.002$). There was no significant difference in mean dmft scores for children aged 36 to 59 mo with deficient versus nondeficient cord blood 25(OH)D (10.9 vs. 8.7 $P = 0.14$). There was no significant difference in mean dmft scores for children aged 12 to 35 mo whose mothers had "sufficient" versus "insufficient" 25(OH)D during prenatal visits (9.0 vs. 7.4; $P = 0.48$). In this small sample, children with deficient vitamin D levels in cord blood had a dmft score at 12 to 35 mo 2-fold higher than children with nondeficient cord blood. Maternal 25(OH)D may influence the primary dentition, and improving vitamin D status in pregnant women might affect ECC rates in their infants.

**Keywords:** caries detection/diagnosis/prevention, child dentistry, dental public health, Alaska natives

## Introduction

Alaska Native (AN) children experience one of the highest reported rates of early childhood caries (ECC) in infants and preschool children (Centers for Disease Control and Prevention 2011). A more severe form of ECC, affecting children under 3 y of age, is referred to as severe ECC (S-ECC) (Drury et al. 1999). Recent studies suggest a possible association between maternal prenatal 25-hydroxyvitamin D (25(OH)D) levels and ECC (Schroth et al. 2014); however, there is no information about the impact of prenatal vitamin D status on dental health for AN children. AN children have experienced increased rates of early rickets since the 1990s (Singleton et al. 2015). Low blood vitamin D concentration is a concern for people living in circumpolar regions where traditional food consumption, a dietary source of vitamin D, is decreasing (Kuhnlein et al. 2004; Bersamin et al. 2006; Egeland et al. 2011; Sharma et al. 2011; Luick et al. 2014; Fohner et al. 2015). Recent studies have demonstrated decreasing vitamin D concentrations in childbearing-aged AN women (O'Brien et al. 2017).

ECC is influenced by both biomedical and environmental factors (U.S. Department of Health and Human Services, National Institute of Dental and Craniofacial Research 2000). One proposed explanation for the burden of ECC in some children is enamel hypoplasia resulting from defective amelogenesis (Caufield et al. 2012). Primary maxillary anterior teeth begin to calcify during the second trimester (~16 wk) and continue until 3 mo postnatally. Vitamin D plays a central role in calcium and phosphorous homeostasis needed for calcification of hard tissues, and vitamin D deficiency in utero is believed to be associated with enamel hypoplasia because of the insult to ameloblasts (Nikiforuk and Fraser 1981). Enamel hypoplasia increases the risk for caries as these defects are often difficult to clean and become readily colonized with cariogenic bacteria (Li et al. 1994; Oliveira et al. 2006). Furthermore, the missing or impaired enamel thickness means that the caries process can develop more rapidly as the underlying dentin is not as resistant to caries attack as intact enamel.

An overview of literature reviews and meta-analyses examining the relationship between vitamin D and 137 clinical outcomes concluded that the available literature supports a probable association between vitamin D concentrations and

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only a few select outcomes, including dental caries in children (Theodoratou et al. 2014). Investigators evaluating the effects of early vitamin D deficiency on growth, bone density, and dental health in later childhood found that children with vitamin D–deficient rickets had a greater risk of bone fracture and dental enamel degradation than healthy children (Zerofsky et al. 2015). Investigations have implicated higher doses of vitamin D with better dental health outcomes and decreased fracture risk (Bischoff-Ferrari 2014) and have found a negative association of vitamin D–rich enhanced dairy product consumption and dental caries incidence (Dror and Allen 2014). Studies from the 1930s (Specker and Tsang 1986) reported that vitamin D supplementation (from plant and animal sources and UV exposure) is associated with a lower relative risk for caries in children, and a contemporary meta-analysis of these studies confirmed these findings (Hujoel 2013). A recent case control study reported an association between S-ECC and 25(OH)D in children (Schroth et al. 2013). Studies from Canada and the United Kingdom reported associations between vitamin D and caries in larger samples of children (Dudding et al. 2015; Schroth et al. 2016). Finally, a recent cohort study investigated vitamin D concentrations' association with ECC within a vulnerable Canadian urban population of expectant women presenting for maternal care. The study found that maternal prenatal 25(OH)D levels may influence the primary dentition and subsequent development of ECC (Schroth et al. 2014).

The Maternal Organics Monitoring study (MOMS) is a prospective, ongoing cohort study examining diet and pollutant factors among mothers and their children in the rural Yukon Kuskokwim Delta (YKD) region in southwestern Alaska. Aggregating levels of nutrients from subsistence foods in maternal blood, the study seeks to understand if overall benefits of subsistence diets outweigh risks of exposure to environmental pollutants (Anwar et al. 2016). In the MOMS, 28% of maternal bloods drawn at prenatal visits and 91% of cord bloods had 25(OH)D levels that were insufficient (<50 nmol/L). The purpose of this study was to evaluate the association of prenatal and birth 25(OH)D concentrations in MOMS participants with development of ECC in their offspring.

## Materials and Methods

### Patient Population

This study focused on AN children living in the remote YKD region, which encompasses 75,000 square miles of coastal wetlands and tundra in southwest Alaska. The region's population of approximately 25,000 comprises primarily Yup'ik Eskimos who live in 52 small communities and the regional hub town. Health care is provided through the Yukon Kuskokwim Health Corporation (YKHC) at the YKD Regional Hospital (YKDRH), as well as by community health aides at primary care clinics (Singleton et al. 2000). Communities are connected by air, water, and snowmobile, with no road access to the remainder of Alaska. One-third of the communities do not have piped water. Among those communities with piped water, none are currently fluoridating water.

### Study Approval

The original MOMS and this current data analysis were both approved by the Alaska Area Institutional Review Board and by the Alaska Native Tribal Health Consortium and YKHC. Written informed consent was obtained from women participating in the MOMS, and the study conformed to STROBE guidelines.

### Study Design

Serum samples for 158 pregnant YKD women were collected at the time of their enrollment (6 to 38 wk of gestation) into MOMS (2010 to 2013). The 25(OH)D levels were performed at the Division of Laboratory Science, National Center for Environmental Health by radioimmunoassay (RIA). Since primary maxillary anterior teeth do not begin to calcify until the second trimester, we used only prenatal blood samples drawn at 16 wk of gestation or later and cord blood drawn at birth for analysis. Vitamin D levels are grouped by the National Academy of Medicine into 3 categories: sufficient (≥50 nmol/L), insufficient (<50 to ≥30 nmol/L), and deficient (<30 nmol/L) (Institute of Medicine Committee et al. 2010). Various covariates for the mothers and resulting children (i.e., birth weight, gestational age, and breastfeeding) were also collected.

As this study was investigating ECC, our outcome variable was children's decayed, missing, and filled teeth (dmft) scores. Most children have 20 primary teeth, which begin to erupt at 6 mo and begin to exfoliate by 72 mo. All 20 teeth are typically present between 24 and 60 mo. Scores for dmft range between 0 (caries free; i.e., no primary teeth decayed, missing due to caries, or filled) and 20 (all primary teeth affected by caries).

The YKHC dental system uses proprietary software, Clinical Product Suite by Quality Systems, Inc. (QSI), for its electronic dental records. QSI programmers created a table ("dfm_data") for oral health surveillance use, which documents the status for each of the 20 primary teeth, as well as the patient identifiers and date of the examination. An already existing patient information table provides information on year of birth and community of residence for each patient, as well as a table, already existing for billing purposes, that contains patient ID, dates of dental services, and dental service codes. Total decayed and filled teeth (dft) and dmft scores were created using the "dmf_data" table. For an individual dft score, the number of teeth that had either untreated decay or fillings was summed for a total score of 0 to 20. Total dmft scores were created in a similar fashion, except that the total score also contained the number of missing teeth due to caries, for a range of 0 to 20. In the dmft score, "missing" is never attributed to nonerupted teeth, and the software allows the dentist to specify teeth missing for noncarious reasons. A YKHC dentist on the research team validated the dmft and dft scores for 50 randomly selected patients by comparing "dmf_data" table data with patient electronic dental records. The YKHC dentists receive training on orientation on the standardized protocols for charting dmft and follow the American Dental Association classification system (Young et al. 2015).

In our study, dft and dmft scores were available for any child who had received a comprehensive dental examination through the YKHC dental system, the primary source of dental care in the region. We linked children's MOMS identification numbers to the electronic oral health surveillance system to obtain dmft scores. We only included dmft scores for children greater than 12 mo of age. If a child had more than 1 comprehensive dental exam and subsequently more than 1 dmft score between 12 and 59 mo, we included only their highest dmft score in the analysis for the age groups analyzed (12 to 35 mo and 36 to 59 mo).

### *Data Analysis*

We compared mean dmft scores for children by cord blood 25(OH)D level of deficiency (<30 nmol/L) and by maternal prenatal 25(OH)D level of insufficiency (<50 nmol/L). Since few children in our study had cord blood vitamin D levels that were sufficient (≥50 nmol/L), we compared mean dmft scores for those who were nondeficient (≥30 nmol/L) with those who were deficient (<30 nmol/L). Conversely, few women had deficient prenatal 25(OH)D levels, so we compared mean dmft scores for children whose mothers' prenatal 25(OH)D levels were sufficient (≥50 nmol/L) to those who were insufficient (<50 nmol/L). We performed independent *t* tests to determine significant differences in dmft scores between sufficiency groups. We also examined infant birth weight and gestational age as factors related to ECC development and performed a multivariable linear regression to examine the impact of these covariates on dmft score. We performed a Spearman (nonparametric correlation) for dmft versus 25(OH)D values for infants 12 to 35 mo and 36 to 59 mo to evaluate the relation between vitamin D deficiency and dmft. A *P* value ≤0.05 was significant. Data analyses were performed using the statistical software SAS 9.4 (SAS Institute). We confirm that we are in compliance with the STROBE checklist for cohort studies.

## Results

We analyzed 76 prenatal mother/infant pairs with prenatal blood ≥16 wk of gestation and 57 infants with cord blood from the MOMS. The mean (SD) maternal age at the time of blood draw was 25.7 (5.3) y (range, 17 to 41 y) (Table 1). Women were 16 to 38 wk of gestation at the time of blood draw. Seventy percent of the women breastfed at birth; 54% of the women did not use tobacco prenatally; 33% smoked, and 11% used chewing tobacco. The mean (SD) 25(OH)D concentration among prenatal women was 69.2 (21.5) nmol/L (range, 22.2 to 114.5 nmol/L). Overall, 16% of women had insufficient prenatal 25(OH)D concentrations (<50 nmol/L) and 2% had deficient prenatal 25(OH)D concentrations (<30 nmol/L).

Fifty-seven mother-infant pairs had both cord blood available for testing and dental records. The mean (SD) birth weight among the 57 infants was 3,615 (558) g (range, 2,254 to 4,758 g) (Table 1). Five percent ($n$ = 3) of the infants were <2,500 g. The mean (SD) 25(OH)D concentration in the cord blood was 31.5 (13.4) nmol/L (range, 6.9 to 63.8 nmol/L); 28 (49%) of cord blood 25(OH)D concentrations were vitamin D deficient (<30 nmol/L). Overall, 52 (91%) children had ECC, and only 5 were caries free. The difference in mean (SD) cord blood 25(OH)D of children with no caries ($n$ = 5) versus any caries ($n$ = 52) did not reach significance (40.0 [16.1] nmol/L and 30.6 [13.0] nmol/L, respectively, $t$ = 1.5, $P$ = 0.07). The mean dft and dmft scores for 12- to 35-mo-old children ($n$ = 43) were 6.6 and 7.2, respectively. Six children (14%) had extractions, and 19 (44%) had already received treatment under general anesthesia (GA) before 36 mo of age. In the multivariable analysis, there was no relationship between gestational age and dmft or birth weight and dmft. We did not identify significant differences in mothers or children with deficient versus nondeficient cord blood 25(OH)D except for a difference in breastfeeding rate (Table 2); however, in a binomial regression analysis, breastfeeding was not significantly related to dmft. There was no significant difference in the mean age in days at dental exam for the children with deficient (871.3 d of age) versus nondeficient (818.6 d of age; $P$ = 0.34) cord blood 25(OH)D. There were no significant differences in the numbers of anterior teeth with caries between children 12 to 35 mo with deficient (3.5) and nondeficient (2.7; $P$ = 0.357) cord blood 25(OH)D levels.

Prenatal 25(OH)D levels were higher than matched cord blood 25(OH)D levels; however, the 25(OH)D levels for matched prenatal and cord blood samples were significantly associated ($r$ = 0.42; $P$ = 0.0002). Matched prenatal and cord blood 25(OH)D pairs were most highly correlated for prenatal samples taken at late in pregnancy ≥28 wk of gestation ($r$ = 0.63; $P$ = 0.02). We chose to analyze prenatal blood in women ≥16 wk to correlate with the timing of calcification of primary maxillary anterior teeth. Matched prenatal and cord blood 25(OH)D pairs were highly correlated for prenatal samples taken at ≥16 wk but not for samples taken at <16 wk ($r$ = 0.54; $P$ = 0002 and $r$ = 0.32; $P$ = 0.077, respectively; Table 3).

We compared the mean dmft score for children whose cord blood 25(OH)D levels were "deficient versus nondeficient" (above and below 30 nmol/L; Table 4). Children 12 to 35 mo of age with "deficient" cord blood vitamin D (25(OH)D <30 nmol/L) had a mean dmft score twice as high as children who were "nondeficient" (9.3 vs. 4.7; $P$ = 0.002). There was a significant negative correlation between cord blood 25(OH)D levels and dmft at 12 to 35 mo ($R$ = −0.37, $P$ = 0.016, $n$ = 43). There was no significant difference in dmft score of children 36 to 59 mo with deficient versus nondeficient cord blood 25(OH)D (10.9 vs. 8.7; $P$ = 0.14).

Too few women ($n$ = 2) had deficient prenatal 25(OH)D levels to examine the children's resulting dmft scores above and below deficiency (30 nmol/L), so we analyzed dmft scores for children whose mothers' prenatal 25(OH)D levels were above and below sufficiency (50 nmol/L). We found no significant difference in mean dmft scores (9.0 vs. 7.4; $P$ = 0.48) for children 12 to 35 mo of age whose mothers' prenatal 25(OH)D levels were above and below sufficiency (50 nmol/L; Table 5). In addition, we found no significant difference in mean dmft scores for children 36 to 59 mo of age whose mothers' prenatal 25(OH)D levels were above and below sufficiency (50 nmol/L).

**Table 1.** Characteristics of Prenatal Women and Their Infants in the Maternal Organic Monitoring Study (2009 to 2013).[a]

| Characteristic | Value |
|---|---|
| Maternal characteristics (*n* = 76) | |
| Prenatal vitamin D Levels, nmol/L | |
| Mean (SD) | 69.3 (21.7) |
| Sufficient (≥50 nmol/L) | 41 (82) |
| Insufficient (≥30 and <50 nmol/L) | 8 (16) |
| Deficient (<30 nmol/L) | 1 (2) |
| Maternal age, y | |
| Mean (SD) | 25.7 (5.3) |
| <20 y | 9 (12) |
| 20 to 34 y | 62 (82) |
| 35+ y | 5 (6) |
| Breastfeeding | |
| Yes | 53 (70) |
| No | 12 (16) |
| Unknown | 11 (14) |
| Smoking | |
| Yes | 18 (35) |
| No | 31 (62) |
| Unknown | 1 (2) |
| Infant characteristics (*n* = 57) | |
| Vitamin D levels, nmol/L | |
| Mean (SD) | 31.5 (13.4) |
| Sufficient (≥50 nmol/L) | 6 (11) |
| Insufficient (≥30 and <50 nmol/L) | 23 (40) |
| Deficient (<30 nmol/L) | 28 (49) |
| Birth weight, g | |
| Mean (SD) | 3615 (558) |
| <2,500 g | 3 (5) |
| 2,500 to 3,499 g | 19 (33) |
| 3,500+ g | 35 (61) |
| Mean dmft score for infants with vitamin D cord blood levels by age group, mean (SD) | |
| <36 mo (*n* = 45) | 7.1 (5.3) |
| 12 to 23 mo (*n* = 20) | 5.8 (4.8) |
| 12 to 35 mo (*n* = 43) | 7.2 (5.2) |
| 24 to 35 mo (*n* = 30) | 7.9 (5.0) |
| 36 to 59 mo (*n* = 48) | 9.9 (5.1) |
| Mean dmft score for infants with prenatal vitamin D levels by age group, mean (SD) | |
| <36 mo (*n* = 34) | 7.8 (5.1) |
| 12 to 23 mo (*n* = 19) | 7.1 (5.2) |
| 12 to 35 mo (*n* = 33) | 7.7 (5.1) |
| 24 to 35 mo (*n* = 24) | 7.8 (4.3) |
| 36 to 59 mo (*n* = 32) | 10.8 (5.4) |

dmft, decayed, missing, filled, primary teeth.
[a]Values are presented as number (%) unless otherwise indicated.

**Table 2.** Characteristics of Mothers and Their Offspring for Children with Deficient (<30 nmol/L) and Nondeficient (25(OH)D) Vitamin D Levels.

| Characteristic | Deficient (*n* = 23) | Nondeficient (*n* = 20) | *P* Value |
|---|---|---|---|
| Gestational age, mean (SD) | 39.3 (1.5) | 39.3 (2.4) | 0.97 |
| Birth weight, mean (SD) | 3643.0 (514.1) | 3544.3 (526.6) | 0.54 |
| Maternal age, mean (SD) | 24.9 (4.8) | 25.3 (5.3) | 0.4 |
| Age in days at dental exam, mean (SD) | 890.2 (159.2) | 830.0 (187.0) | 0.81 |
| Breastfed, *n* (%) | 15 (68.2) | 18 (100.0) | 0.008 |
| Smoked, *n* (%) | 7 (30.4) | 8 (40.0) | 0.54 |

## Discussion

In this small cohort of AN mothers and their offspring, there was an association between dmft and vitamin D–deficient cord blood for children <36 mo of age. Our primary finding is that deficient 25(OH)D levels (<30 nmol/L) in cord blood are associated with ECC. Among children 12 to 35 mo of age, those with deficient concentrations of 25(OH)D had dmft scores

**Table 3.** Correlation of Matched 25(OH)D Samples Taken from Cord Blood and Prenatal Samples Drawn at Gestation ≥16 wk and <16 wk.

| Variable | *n* | Mean, mmol/L | SD | Minimum | Maximum | *r* | *P* Value |
|---|---|---|---|---|---|---|---|
| Prenatal 25(OH)D (gestation ≥16 wk) | 43 | 68.3 | 25.9 | 16.6 | 124.5 | 0.54 | 0.0002 |
| Cord 25(OH)D | 43 | 31.7 | 14.3 | 10.2 | 71.6 | | |
| Prenatal 25(OH)D (gestation <16 wk) | 32 | 60.1 | 17.3 | 23.4 | 93.3 | 0.32 | 0.077 |
| Cord 25(OH)D | 32 | 29.4 | 12.5 | 6.9 | 63.8 | | |

*r* is a Spearman correlation coefficient.

**Table 4.** Comparison of Mean Decayed, Missing, and Filled Teeth (dmft) Scores for Children above and below 36 mo of Age with "Deficient" versus "Not Deficient" Cord Blood Vitamin D (25(OH)D) Concentrations.

| | Cord Blood 25(OH)D Level, Mean (SE) | | |
|---|---|---|---|
| Age Group | Deficient (25(OH)D <30 nmol/L) | Not Deficient (25(OH)D ≥30 nmol/L) | *P* Value |
| 12 to 35 mo (*n* = 43) | 9.3 (1.1) | 4.7 (0.9) | 0.002 |
| 36 to 59 mo (*n* = 48) | 10.9 (1.0) | 8.7 (1.1) | 0.140 |

**Table 5.** Comparison of Mean Decayed, Missing, and Filled (dmft) Scores for Children above and below 36 mo of Age Whose Mothers Had "Insufficient" versus "Sufficient" Vitamin D (25(OH)D) Concentrations at a Prenatal Blood Draw ≥16 wk of Gestation.

| | Prenatal Blood Vitamin D Level, dmft Score, Mean (SE) | | |
|---|---|---|---|
| Age Group | Insufficient (25(OH)D <50 nmol/L) | Sufficient (25(OH)D ≥50 nmol/L and <75nmol/L) | *P* Value |
| 12 to 35 mo (*n* = 33) | 9.0 (2.5) | 7.4 (1.0) | 0.48 |
| ≥36 to 59 mo (*n* = 32) | 14.4 (1.0) | 10.1 (1.1) | 0.12 |

twice as high as those with nondeficient concentrations. There were not adequate maternal prenatal blood samples in the deficient range (<30 nmol/L) to evaluate an association between deficient prenatal vitamin D levels and ECC for children 12 to 35 mo of age, and we found no significant association between vitamin D–insufficient (<50 nmol/L) prenatal blood levels and ECC. Our findings suggest that low 25(OH)D levels in the deficient (<30 nmol/L) but not insufficient (<50 nmol/L) range are associated with ECC and that cord blood levels correlate best with ECC.

Maternal 25(OH)D diffuses across the placental barrier during pregnancy (Institute of Medicine Committee et al. 2010), and studies show that cord blood 25(OH)D concentrations are 75% to 90% of maternal concentrations at delivery with correlations increasing as week of gestation of maternal 25(OH)D measurement increases (Bodnar et al. 2007; Keim et al. 2014; Saraf et al. 2016). Many factors, including season and vitamin D supplementation, affect vitamin D status during pregnancy (El Hayek et al. 2010; Hossain et al. 2011). Like Keim et al. (2014), we found that prenatal and cord blood 25(OH)D levels were moderately correlated and that these correlations increased as gestation of the prenatal 25(OH)D level increased. Cord blood 25(OH)D is a better measure of fetal 25(OH)D status in late gestation and may be more relevant to pediatric outcomes than prenatal 25(OH)D (Keim et al. 2014).

Our association between vitamin-deficient 25(OH)D concentrations in cord blood and dmft in children 12 to 35 mo helps to confirm findings in a prospective cohort study of Canadian urban women (Schroth et al. 2014). In that Canadian cohort, mothers of infants with ECC at a mean of 16 mo had lower 25(OH)D levels in the second and third trimesters ($P = 0.05$), and regression analysis revealed that there was a significant negative relationship between average number of decayed teeth and prenatal 25(OH)D levels. However, like our study, the investigators did not show a significant association between deficient 25(OH)D levels in the prenatal blood samples and ECC in their infants. In our small cohort, we did see an association between vitamin D–deficient cord blood 25(OH)D level and dmft in children <36 mo. Perhaps this is because cord blood is a better measure of fetal 25(OH)D status than prenatal concentrations. Maternal 25(OH)D explains only a fraction of the variability in cord blood 25(OH)D, and other unknown factors must contribute to fetal vitamin D.

A growing body of literature suggests that there is an association between 25(OH)D and caries in children (Hujoel 2013; Schroth et al. 2013; Schroth et al. 2014; Dudding et al. 2015; Schroth et al. 2016). Our current study provides additional evidence for this association by demonstrating that vitamin D deficiency in prenatal women has a potential impact on caries risk in young children. The hypothesis that prenatal vitamin D deficiency affects S-ECC is biologically plausible because of the likely association between vitamin D deficiency in utero and enamel hypoplasia, leading to higher risk for early caries (Nikiforuk and Fraser 1981; Caufield et al. 2012). However, there are other potential causes for this relationship, including a difference in health behaviors and child oral hygiene in mothers of infants with deficient cord blood 25(OH) levels. We did not identify significant differences in mothers or children with deficient versus nondeficient 25(OH)D except for higher breastfeeding rates in children with nondeficient vitamin D.

Although breastfeeding was not significantly related to dmft, the sample size was small, and a more thorough assessment that includes breastfeeding and other confounding factors would require a larger cohort study.

Since other factors such as dental hygiene, intake of sugar-sweetened beverages, and access to running water and fluoride exposure affect dental caries as children get older, we were not surprised to see the strong association between cord blood 25(OH)D and dmft in children <36 mo disappear as these children aged. The association between higher dmft and insufficient prenatal 25(OH)D was not significant, and this association requires further study with a larger cohort. Recent evaluation of prenatal vitamin D concentrations in women from the YKD region has shown a higher prevalence of vitamin D deficiency than in MOMS. Among 211 early prenatal 25(OH)D concentrations in YKD women during 2016 to 2017, we found that 13.7% were vitamin D deficient (<30 nmol/L) and 53% were vitamin D insufficient (<50 nmol/L). In 2016, YKDRH initiated routine prenatal vitamin D supplementation with 1,000 IU vitamin D to prevent early rickets. We plan to evaluate the association between prenatal 25(OH)D concentrations and offspring ECC in this larger contemporary cohort.

This study had some limitations. First, this was a retrospective study on an existing small cohort; however, the 2-fold difference in dmft scores for 12- to 35-mo-olds with deficient versus nondeficient cord blood was highly significant in the age group most likely affected by maternal vitamin D deficiency. Second, age is a potential confounder since dmft increases with age of the child; however, in this cohort, children with sufficient 25(OH)D cord blood were not significantly different in age from children with deficient cord blood levels. Third, we were not able to adjust for key confounding factors such as dental hygiene, sugar-sweetened beverage use, or access to running water, which affect dental caries. Fourth, since so few children were caries free, we were unable to contrast 25(OH)D levels between those with ECC and those without ECC. Last, this is a secondary data analysis of a study not designed for this purpose, and we plan to conduct a larger cohort study designed to evaluate potential confounders. However, despite these limitations, we saw a strong association between deficient cord blood 25(OH)D and dmft in a small cohort of children <36 mo, in whom maternal factors such as vitamin D deficiency would be most important.

## Conclusion

Prenatal vitamin D levels may influence the primary dentition and the development of ECC. Improving vitamin D status in pregnant women might affect ECC rates in their infants.

### Author Contributions

R. Singleton, contributed to conception, design, data analysis, and interpretation, drafted the manuscript; G. Day, contributed to conception, design, data acquisition and analysis, drafted and critically revised the manuscript; T. Thomas, J. Berner, contributed to conception, design, data interpretation, critically revised the manuscript; R. Schroth, contributed to design, data analysis, and interpretation, critically revised the manuscript; J. Klejka, contributed to design, and data interpretation, critically revised the manuscript; D. Lenaker, contributed to design, data acquisition, and interpretation, critically revised the manuscript. All authors gave final approval and agree to be accountable for all aspects of the work.

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### Disclaimer

The findings and conclusions in this article are those of the author(s) and do not necessarily represent the official position of the tribal organizations.

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