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Association of Maternal and Neo Natal Nutrition with Dentaldevelompment in Childhood

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I. INTRODUCTION

VITAMIN D

Enamel abnormalities have been linked to everything from genetic disorders to issues during pregnancy and the first few months after birth. 9 Because of the metabolic stress to ameloblasts, vitamin D deficiency in utero is thought to be linked to enamel hypoplasia. The equilibrium of calcium and phosphorus, which is necessary for the calcification of hard tissues, is mostly regulated by vitamin D.

Mellanby's pioneering work solidified the idea that the foetus is the crucial time for influencing the primary dentition's development. Prenatal nutrition has a significant impact on the development of dental tissues because main tooth calcification has a brief time span and starts in the second trimester.

Several historical studies have suggested that vitamin D administration may prevent dental caries in children, despite the fact that some doctors are unaware of the study on vitamin D and dental caries and enamel hypoplasia. The preliminary conclusions were supported by a recent meta-analysis of these research. According to recent studies, vitamin D may influence how the human immune system reacts and may weaken the host's defences against cariogenic bacteria. The current study's goal was to evaluate the theory that lower maternal prenatal 25hydroxy vitamin D(25OHD)status and the existence of ECC in infants are related.

CALCIUM

How is it possible for a child who is otherwise healthy, who has only been blessed with one year in this life, and who has never once had candy or cake, to suffer from rotten teeth? Why are dental decay rates high in our modern, technologically advanced culture but very low in ancient hunter-gather societies, according to archaeological evidence? The role of nutrition and its impact on the intricate aetiology of dental caries play a part in the solutions to these concerns.

Infant dental caries are a mysterious and widespread condition. They involve a multifaceted syndrome that affects a sizable portion of the world's population, regardless of gender, age, or ethnicity; it does, however, tend to impact individuals with a poor socioeconomic standing to a higher extent (1). One of the most prevalent illnesses in kids is this one. It affects more than 50%. of all children aged 5 to 9 and more than 75 percent of all teenagers by the age of 18. By the age of two, 60% of all kids have at least one filled primary teeth (4). At a crucial juncture in a child's emotional and psychological development, it can be painful and uncomfortable for its victims and necessitates a significant amount of physical assistance. The most perplexing aspect of this illness is that it affects kids before they have had a chance to develop bad flossing habits or an excessive sweet tooth—patterns of behaviour that are more clearly connected to caries in adults.

OBESITY AND SKELETAL MATURITY

In fact, the World Health Organization has referred to obesity as "a global pandemic disease" (WHO, 2003] due to the steady rise in overweight and obesity among youngsters over the past few years, Overweight and obesity are currently the most prevalent medical disorders among children, according to the American Academy of Pediatrics Committee on Nutrition (Krebs and Jacobson, 2003).According to Binkin et al. (2009), the prevalence of obesity in children aged 6 to 9 living

in Central-Northern Italy was 16.6%.The heart there are short-term and long-term health hazards associated with childhood obesity and overweight cardiovascular consequences (high blood pressure, high cholesterol, was 8.9% and 9.0%, respectively. By contrast, the prevalence of obesity in children aged 8 to 9 living Both short-term and long-term health hazards are associated with childhood overweight and obesity: effects on Dyslipidemia, endocrine issues (such as hyperinsulinism, insulin resistance, impaired glucose tolerance, and type Il diabetes), adult overweight and obesity, pulmonary issues (such as asthma, obstructive sleep apnea, and Pickwickian syndrome), and orthopaedic issues [Lloyd et al., 2011; Saha et al., 2011: WHO, 2003]. In girls, obesity can appear in the early phases of puberty and can significantly speed up linear growth (Slyper, 2006, Ahmed et al., 2009); in boys, there can be significant changes in the timing of puberty (accelerated or delayed) [Slyper, 2006; Kleber et al., 2011]. Obesity can cause early craniofacial growth (increased mandibular length, prognathic jaws, and decreased upper anterior face height) and an increase in the average height during childhood, according to Kleber et al. (2011) and Ohrm et al. (2002). With the help of the Body Mass Index (BMI) and Dual Energy X-ray Absorptiometry, this cross-sectional study sought to evaluate the skeletal maturation through the cervical vertebral maturation (CVM) and dental age in normal weight, pre-obesity, and obese patients.

II. OBJECTIVES

VITAMIN D

Maternal vitamin D levels, which are measured by 25-hydroxyvitamin D [25OHD] levels, may be insufficient during pregnancy, which could impact tooth calcification and increase the risk of early childhood caries and enamel hypoplasia (ECC). This study's goal was to ascertain the connection between prenatal 25OHD concentrations and dental caries in young children throughout the first year of life.

CALCIUM

The complicated aetiology of crossing causes that characterises early childhood dental caries is what defines it. The most obviously modifiable element of this etiology is diet, especially when combined with adequate mouth hygiene.

Ensuring that the pregnant, nursing, and finally weaning mother's diet contains an adequate amount of calcium phosphate helps to reduce susceptibility to dental caries by removing a significant contributor—calcium phosphate deficiency to the complicated diagram that is dental caries. This is currently being advanced in a novel paradigm of dentistry that looks at the mineral content of saliva in addition to bacteria counts and employs a combination of bacterial management and mineral availability to re-mineralize early lesions and stop more severe decay before it arises.

OBESITY AND SKELETAL MATURITY

Obesity in children and skeletal-dental development abstract This cross-sectional study's Objective was to investigate the skeletal maturation of normal weight, pre-obesity, and obese patients utilising the cervical vertebral maturation (CVM), dental age, and dual energy X-ray Absorptiometry (DXA).

ANALYSIS OF THE ORIGINAL ARTICLE

VITAMIN D

METHODS

To examine the relationship between prenatal vitamin D concentrations and infantile dental caries in a vulnerable urban population, a prospective cohort research was created. After giving written informed agreement, participants were chosen for the study during the second or early third trimester. The intended audience consisted of pregnant women seeking prenatal treatment in Winnipeg, Canada (latitude 49°539N). Since the primary maxillary incisors start to erupt from weeks 13 to 17 in utero and continue to do so throughout pregnancy, a blood sample was taken as part of a prenatal visit during the second or early third trimester. The Health Sciences Centre in Winnipeg performed the serum analysis.

Alkaline phosphatase, total calcium, inorganic phosphorus, and 25OHD levels in the samples were all measured (elevated levels indicate vitamin D insufficiency14). A DiaSorin kit (DiaSorin, Inc., Stillwater, MN) was used to perform a radioimmunoassay to determine 25OHD, a valid indicator of vitamin D status overall23. 25OHD values below 35 nmol/L were deemed inadequate, and those above 75 nmol/L were deemed ideal.

Participants filled out a questionnaire under the supervision of the clinic staff or the lead researcher (Dr.Schroth). A technique that evaluated nutritional deficits in northern Manitoba served as the foundation for this instrument. With the help of researchers and physicians, including a dietician, the questionnaire was adjusted. Data was gathered on demographic factors (such as age, ethnicity, and level of education), pregnancies (such as parity and use of prenatal vitamins), health conditions, nutrition (such as consumption of milk, dairy, fish, eggs, and meat), and awareness of ECC (such as

knowledge that ECC exists, that ECC has affected older children, and what causes ECC). Additionally evaluated were family composition (such as relational status and family size), money, employment, and sunlight exposure (such as the amount of time spent in the sun throughout the summer).

The primary dentition was evaluated as the last part of the trial, with the examiner (Dr.Schroth) being blinded to the prenatal 25OHD level of each infant's mother. Using accepted guidelines, caries in infants' teeth was evaluated. According to accepted definitions, 25 ECC and S-ECC were defined. Enamel caries in its early stages and noncavitated lesions (white spots) were noted. The number of decaying primary teeth (decayed teeth [dt] score) and the number of decayed primary teeth (individual cumulative totals of the number of decayed, removed, and filled primary teeth) were calculated. An established index for recording enamel defects including hypoplasia and opacities was used to evaluate developmental problems of the enamel.

At the time of the infant's examination, a follow-up questionnaire was given to collect data on demographic factors (such as the child's gender and age), household finances, birth weight, prematurity, feeding practises (such as breastfeeding, bottle-feeding, and the introduction of solid foods), and infant health status. Additionally, questions regarding oral hygiene routines, the age at when the first tooth began to erupt, and dental visits were asked to the caregivers.

Based on prevalence data for prenatal vitamin D status in Manitoba, the estimated sample size was reviewed by a biostatistician and validated using PASS version 6.0 (NCSS, Kaysville, UT). To allow for some loss while retaining a sufficient sample size, the minimum sample size was increased.

Using NCSS version 2007 (Kaysville, UT) and SPSS version 17.0 (IBM SPSS Statistics, IBM Corporation, Armonk, NY), clinical and survey data were input into a Microsoft Office Access database (Microsoft Corporation, Redmond, WA) and analysed. Descriptive statistics, such as frequencies and mean +/- SD values, were included in the analysis. X 2 analysis, t tests, correlation, Poisson regression, and analysis of variance were all examples of bivariate analysis. We utilised Poisson regression for dt scores and multiple logistic regression for ECC. Poisson regression is suitable for count data, including newborn caries scores.

To identify significant independent variables within sets of five themes, separate regression models for ECC were fitted. These models included serum metabolites (such as 25OHD, calcium, alkaline phosphatase, and phosphorus), factors affecting vitamin D status (such as milk intake, margarine use, prenatal vitamin use, season, and vitamin D drops), infant feeding practises (such as bottle-feeding, breastfeeding, and use of a sippy cup), socioeconomic factors (such as income, employment status)In addition to variables that are frequently reported to be linked with ECC in the literature, a final model was built that included independent variables that were significantly or roughly approximating the threshold of significance associated with ECC in these distinct models. Odds ratios and confidence intervals for continuous variables were computed to account for changes in the variable of 1 SD unit. For instance, a change of 1 SD unit was reflected in the odds ratio for 25OHD. A P value of less than or equal to .05 was deemed significant.

OBESITY AND SKELETAL MATURITY METHODS

At the Department of Neuroscience, Human Nutrition Unit, University of Rome "Tor Vergata," 107 healthy patients between the ages of 6 and 12 (mean age 8.771.79) underwent Anthropometric measurements, BMI calculation, and DXA exam. At the Paediatric Dentistry Unit ofPTV Hospital, University of Rome "Tor Vergata," skeletal and dental age were evaluated. According to FM% McCarthy cut-offs classification and BMI, the participants were categorised as underweight, normal weight, pre-obesity, and obese.The Fisher's exact test was run between the gender categorical variable and the McCarthy cut-offs classification as well as between the McCarthy and BMI classifications. The Student's t test was also applied for each group of children (using McCarthy and BMI classifications) between Chronological and skeletal-dental age. Additionally, the Pearson correlationcoefficient was Performed to assess the relationship between skeletal and dental age. A substantial level of alpha = 0.05 was taken into consideration for all evaluations.

The Student's t test was also applied for each group of children (using McCarthy and BMI classifications) between chronological and skeletal-dental age; the Fisher's exact test was performed between the gender categorical variable and McCarthy cut-offs classification, and between McCarthy and BMI classifications. Besides, to

evaluate the association between skeletal and dental age, a Pearson correlation coefficient was calculated. In all the assessments a significant level of alpha = 0.05 was considered.

CALCIUM

A COMPLEX ETIOLOGY BY RISK FACTORS AND THEIRINTERSECTION

The main characteristic that sets early childhood dental caries apart is its complicated aetiology. With so many factors at play, it is challenging to identify specific, tangible characteristics that alone are responsible for the disease's development and swiftly fast progression. Instead, the convergence of numerous factors is what defines dental caries. Separate factors have been found which, taken separately, would put a person at risk, but only when those things are together specific causes of dental caries risk factors.

Environmental pollutants, illness, medicine, prenatal and postnatal diet, as well as genetic predispositions,are some of the risks that can contribute to dental caries development. Sometimes, genetics can explain a tendency as the result of Amelogenesis, the process of creating enamel, happens predictably in every tooth after another generation. Size, shape, colour, and even a substance's vulnerability to caries,enamel on teeth can be handed down from parents to children.

There may be numerous examples with no genetic trait at all, thus just because something has a genetic propensity does not indicate that other variables cannot cause it to manifest. Factors include Poverty, which has repeatedly been connected as a primary indication, can only provide a potential genesis. Due to the fact that a lack of resources is not the cause of poverty,inherently connected to sickness. Beyond genetics, poverty is a sign that suggests other factors.There are social and behavioural trends that do contribute to this mysterious sickness.

Evidence is accumulating that can shed light on earlier markers, such as economic position, and link it more directly to nutritional standing despite the difficultiesin assigning causality to one main cause. The significance of examining nutrition's role as one because of its close ties to human behaviour, dental caries has an influential influence on how it develops.It turns out to be the part of the disease's aetiology that is easiest to change because of for that reason, the focus of this chapter is on this aspect of the illness and how it affects both preventing and ending.

The proper disseminationof this information to improve research and education could be accomplished with the aid of public health professionals.The availability of alternatives to the present dentistry model, which involves surgical therapy, would increase created and disseminated.The significance of sufficient calcium levels in the potential usage of calcium phosphate solutions alongwith a preventive diet methods for early detection, eradication, and potential remineralization of bacteria. The early caries lesions could be used as a different approach to treating caries.

CARIES DEVLOPMENT AND ROLE OF STREPTOCOCCUS MUTANTS.

Dental caries is a contagious condition. It frequently spreads from child to caregiver. Streptococcus mutans, a dangerous pathogen, is usually to blame. This acid-producing

bacterium eventually exposes the internal structure of the tooth to disease as it eats away at the surface enamel. Not only is this S. mutans bacteria responsible, but Divvy up the blame. These bacteria's capacity to colonise the body is influenced by several factors their host.

Many people who harbour the bacteria may unknowingly manage to keep it under controllable levels in their mouths. Not all children under the care of individuals with high levels of this bacterium become infected. Factors such salivary flow, pH, and the state of the immune system, tooth development, and morphology all have an impact on the teeth will be attacked by the pathogenic germs. S. mutans is particularly harmful because it has the rare capacity to colonise flat surfaces and in perfect climatic conditions.

conditions multiply quite quickly. It consumes sugars and carbs, and as a waste product, it secretes acids. These acids cause the enamel to become less mineralized, which finally causes tooth decay.

Nutrition has traditionally been disregarded or barely mentioned as a part in this process. Physical touch has been pointed out as the main cause of food's principal responsibility for the emergence of dental caries. The S. mutans receive their nutrition directly from sugary or carbohydrate sources, allowing them to happily reproduce and flourish in their acidic environment. Although well acknowledged, this fact offers little to explain the enormous variation in data among dental caries victims. Carie susceptibility has frequently been associated with breastfeeding patterns, which has prompted dentists to advise early weaning. Previously was formerly known as "baby bottle

rot," but it is now known as "Early Childhood Caries" (ECC). This term change reflects a difference in how the disease's progression is now understood. Data supporting assertions that nursing was informal in nature had been overgeneralized, and more in-depth investigation has revealed that it possesses noncariogenic qualities (3). However, when considering the function that adequate levels of calcium phosphate play as a variable in this complex situation, the need of breast milk as a key food source for infants becomes clear.

SUSCEPTIBILITY

Good eating habits in childhood and adolescence are critical for reducing the chance of developing caries later in life, according to recognised recommendations (4,5). The rationale for these recommendations is that "ideal dietary habits for dental health are congruent with dietary recommendations for systemic health, growth, and development." They stress the role that nutrition plays in reducing the risk of caries (4). demonstrating that the emergence of caries may be a sign of general malnutrition. Despite the fact that this chapter has primarily focused on the function of calcium phosphate, it should be noted that overall nutritional health also contributes to the development of caries. Diet should be viewed as a dual risk factor because it provides S. mutans with an on-site food source.

The teeth are at double risk from malnutrition. First, it impairs the immune system, making the body more vulnerable to bacterial invasion and depriving it of the necessary lymphocytes for defence. Children with HIV exhibit an extremely high risk of dental caries, which rises in correlation with mild to severe immune suppression (6). Nutritional deficiencies have the additional effect of depriving the body of nutrients necessary for healthy tooth formation and re-mineralization, such as calcium phosphate and zinc. This results in a weak immune system. They play a crucial role in determining the structure of the teeth. Without appropriate levels of these essential components, the body lacks the building blocks needed to produce

TEETH'S HISTORY OF DEVELOPMENT

Although it happens so early in pregnancy that many women may not be aware of their pregnancy or have not yet changed their long-standing consumption patterns, the nourishment the growing embryo receives from its mother is essential for the formation of teeth. Early childhood development is crucial for tooth development;at which tooth production may be halted or altered by environmental influences. The disturbance of healthy tooth mineralization can be caused by illnesses, environmental toxins, drugs, and nutrition (7). Although the processes underlying this effect are not well understood, calcium phosphate may play a critical role in controlling tooth development (8). In addition to these other issues, a lack of calcium during critical periods may be to blame for the enamel's fissures and pits as well as the thin and flaky surfaces that provide bacteria easy access. When this happens, the once-strong and frequently brittle enamel surface gets so extreme that teeth might practically fall out of the mouth.Similar to trees, teeth have rings that show their developmental history. The enamel stria, a line, represents the tooth's growth patterns (9). 1 The neonatal line, a pronounced striation, is an indication that the mineralization process was interrupted at birth. This is one of the most obvious signs that dietary factors have an impact on tooth growth. This line demonstrates a significant environment shift. From being automated by the umbilical cord to demanding the joint mother and newborn cooperation, infant feeding methods undergo a significant transformation.

This entry in the tooth formation record could mean that the infant's nutritional demands were not supplied during the first few days of life because the mother's delayed nursing.The structure of the tooth is intricate. Connective tissues, nerves, and the vasculature are all present in this complex, mineral-rich cellular structure. A protein-based structural matrix is created during tooth development in utero and is later calcified. Since minerals are the building blocks of teeth, they must be present both during tooth formation and as a constant supply throughout life.

Enamel, dentin, and pulp are the three main components of a tooth. Calcium phosphate makes up the majority of both the enamel and dentin. This is why it seems logical that sufficient amounts of calcium phosphate would be needed when teeth are developing.

A STRUCTURAL COMPARISON OF CORPOREAL MINERAL RESERVES: TOOTH IS TO BONE

Although bone is far more complicated in character than teeth, a comparison is necessary to completely comprehend the function that both teeth and bone perform as corporeal mineral stores for substances like calcium phosphate. These reservoirs served as the evolutionary safety nets required to get our ancestors through calcium shortages.

Dentin, which makes up the internal structure of the tooth, shares the most molecular

similarities with the bone in the rest of your body. The similarities between the structure and susceptibility to disease in teeth and bone are best illustrated by looking at bone anatomy. Additionally, it illustrates the ongoing remineralization

Osteoblasts, osteocytes, and osteoclasts are three different types of cells that create and maintain bone structure. These cells perform a variety of functions, including generating new bone, preserving bone health, and obliterating worn-out, stressed bone. All of these activities include the movement of minerals in and out of the body's circulation, most significantly calcium phosphate. Minerals from the blood are used to strengthen the collagen and protein matrix during the formation of new bone. Minerals are released into the blood stream when bone is damaged.

Remodeling is constantly taking place in normal bone. In order to maintain healthy bone, a balanced process that occurs throughout life involves the breakdown of old bone tissue and the synthesis of new bone. If there are sufficient calcium levels in the blood, this equilibrium will remain. Calcium is needed by the osteoblasts for this remodelling PROCESS. The exchange of calcium through the blood is a continuous, balanced process as long as adequate blood calcium levels are maintained; as blood levels decrease, the balance is lost and the osteoblasts are unable to perform their function, leaving bones weak, unmineralized, and vulnerable to illness.

The body's calcium levels are controlled by the parathyroid glands. These tiny glands, which are contained within the thyroid gland's tissue, are able to pick up changes in the blood's calcium levels. In response to a dip in levels, they release parathyroid hormone (PTH). As a result, more calcium is absorbed from food in the intestines, more calcium is released from the bones, and more calcium is reabsorbed through the kidneys. When levels become too high, PTH secretion is reduced, and calcium levels stabilise once more (10). By using the bones and teeth as calcium reservoirs, this method enables the body to draw calcium from these sources when it is calcium depleted. If eating habits have persistent deficits, over time.

REINSTATEMENT OF MINERALS AND STRUCTURAL REPAIR

Re-mineralization is crucial for dental health, structural healing, and the maintenance of both teeth and bones. The dynamic dance of demineralization and re-mineralization is ongoing in the teeth. The pH of the mouth, the amount of minerals present in the saliva, and the concentrations of those minerals in the blood all interact to create conditions that either promote mineral loss or may allow minerals to reatach themselves to the tooth's surface (12). Through remineralization, tiny carious lesions may thus go unnoticed by the patient or the dentist and heal on their own.

When insufficient blood levels of amorphous calcium necessitate additions from these mineral stores within the body, teeth demineralize similarly to bone. The intricate structure of the tooth is made to tolerate fluctuations in calcium availability. A result of infrequent nutritional stability in hunter-gatherer tribes is the re-mineralization process. This integrated repair system enables for remineralization when the body has access to sufficient mineral stores. Calcium levels in the blood and saliva could be raised through regular monitoring of tooth health in order to support this natural remineralization process...

HISORICAL CONSUMPTION PATTENS AND ITS IMPLICATION

Compared to modern diets, historically, hunter-gatherer societies consumed diets that were significantly higher in calcium (13). Given that hunter-gatherers lacked access to dairy products, it is most likely that the majority of their calcium intake came from animal bones and potentially soil components, as anthropologists continue to record clay ingestion on numerous continents (11).With the spread of western practises, dental caries rates all over the world increased. Dental caries affects more than 95% of the population in the US alone (12). Diets in modern life are inadequate for our bodies' cellular needs; they are reduced by rising consumption of nutritionally deficient snacks and a dearth of nutrient-rich foods that are sources of essential mineralsThis cuisine is new to humans; it first became widely popular after the industrial revolution, which took place 200 years ago (13). Numerous chronic diseases have surfaced as a result of the quick change in dietary habits. Changing dietary patterns over time can be associated with a number of chronic diseases, including bone and periodontal disease. Lack of essential nutrients including calcium phosphate, vitamin D, and zinc may have contributed to the emergence of osteoporosis and rickets. Rickets and osteoporosis, two common bone illnesses with frequent effects on oral health due to insufficient vitamin D and calcium intake (14). Early childhood dental caries are also significantly influenced by similar eating patterns, and in many cases, the development of bone disease follows dental caries,

although being less well documented than osteoporosis and rickets despite the enormous population involved. Infant dental offices and rickets, which causes the bones to soften, are frequently experienced by patients at the same time. Dental caries could be categorically described as teeth rickets in some instances. Softening of the dental surface and even tooth cracking are signs of severe dental caries. Rickets develops when there are insufficient mineral levels available to help firm the collagen and protein matrix intended only as a building block within bone and not as a fundamental structure. This is because bone and teeth share structural similarities and similar mineral requirements. These methods make it simple to draw a connection between dental and bone health. The crucial component that they both share, calcium phosphate, makes it easiest to draw a connection between the remarkable similarity between bone loss and dental caries. Our bodies' cellular level evolutionary biology, which provides the building blocks for our bodies, has not yet fully adapted to the rapid cultural evolution that has so drastically changed our food in the previous 100 years. With the ultimate goal of improving overall nutrition and thereby increasing calcium consumption among people of all ages, helping to prevent bone diseases in general and dental caries in particular, this discrepancy in biological and cultural evolution may shed light on the significant role that public health can play in behaviour modification. This might pave the way for a departure from the surgical model of dentistry that has guided contemporary dentistry from its inception.

CALCIUM DEFICENCY

Guidelines for calcium consumption have been developed by the National Institutes of Health (NIH) to ensure appropriate immunological, bone, and dental health. They claim that while calcium is best obtained via calcium-rich meals, supplements can also be helpful in achieving ideal calcium levels. A coordinated public health plan is required, they continue, "to guarantee that the American population is consuming the recommended amount of calcium." The obvious significance of calcium in the role of dental caries prevention is highlighted by the national institute of health's call to action. The effects of calcium phosphate on the immune system and the lack of mineral availability for healthy tooth development have all been discussed in this chapter in relation to how calcium phosphate influences the development of baby dental caries. It has also demonstrated how the body obtains calcium from the teeth when there is a calcium shortage. As a result of this procedure, S. mutans can enter the body more easily. Because of this, prevention is the greatest method for treating infant dental caries. This must take the shape of dietary modifications that encourage calcium-rich foods as early feeding options for infants and toddlers and enhance calcium consumption in pregnant and nursing women. Since an accidental overdose would have to include abnormally high calcium levels, calcium supplementation is typically considered safe and unlikely to offer any risks of harmful health effects to practically all members of the general population. The UL is 2500 mg/d for children and adults 1 years of age and older, including those who are pregnant or nursing. it is challenging to make generalisations about individual calcium needs because genetic factors, dietary inconsistencies, and other influencing nutrients have a significant impact on calcium requirements for the individual. However, it is well known that the average person does not consume enough calcium. The following percentages of Americans do not consume the required amounts of calcium, according to the Continuing Survey of Food Intakes of Individuals (CSFII 1994–1996).

Ages 6 to 11: 58% girls and 44% boys.
Ages 12 to 19: 87% girls and 64% boys.

Ages 20 and up: 78% of women and 55% of men (18).
2If 78% of women are not getting enough calcium, their developing foetuses and nursing children will be negatively impacted and may even become deficient in calcium themselves. The sorts of foods from which one can obtain calcium are shown in Table 1, although the amounts required for even tiny amounts of calcium are frequently much higher than the reality of current consumption habits.

A NEW PREVENTION AND TREATMENT

The prevention of tooth caries by maintaining adequate calcium levels ought to be prioritised. Currently prevention is predominately emphasized through oral hygiene and sugar avoidance. The only mineral that is recommended for supplementation is fluoride. The majority of dentists and dental institutions still do not include proper calcium levels in their guidelines for preventative tactics.

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Table 1
Calcium Content of Common Foods

Food	*Serving size*	*Calcium content*
Milk[†]	1 cup	300 mg
White beans	1/2 cup	113 mg
Broccoli (cooked)	1/2 cup	35 mg
Broccoli raw	1 cup	35 mg
Cheddar cheese	1.5 oz	300 mg
Low-fat yogurt	8 oz	300–415 mg
Spinach cooked[‡]	1/2 cup	120 mg
Spinach raw[‡]	11/2 cup	120 mg
Calcium-fortified orange juice	1 cup	300 mg
Orange	1 medium	50 mg
Sardines or salmon with bones	20 sardines	50 mg
Sweet potatoes	1/2 cup mashed	44 mg

Notes: [†]Low-fat milk has comparable or greater calcium levels than whole milk.
[‡]The calcium from spinach is essentially nonbioavailable.
This table was adapted from the American Academy of Pediatrics, Calcium Requirements for Infants Children and Adolescents.

The use of topical fluorides in dentifrices, rinses, and gels as well as dietary fluoride supplements in children were all brought about by the epidemiological observation that fluoride in the diet causes tooth decayThis was a significant public health initiative with broad implications. The majority of teeth are made of the calcium phosphate salt apatite, which fluoride substitutes with carbohydrates in addition to acting as a bactericide. Fluoride-rich teeth are significantly less soluble and consequently less likely to demineralize. Given our history of dental practise, this was a relatively recent finding that has significantly changed the rate of dental caries. It is described in all dental literature pertaining to the management and prevention of caries. Despite the effectiveness of fluoride, the use of calcium as a preventative and therapeutic strategy has not yet permeated dental procedures from institutions like the National Institute of Health. Graham Mount, DDS, and Rory Hume, DDS, from the UCLA school of dentistry, claim that the present therapeutic objective is "to change the local biochemistry so that the patient is no longer losing tooth mineral, so that the condition is then cured and the patient is healed." This is reasonable, moral, suitable, and doable. This model has only started to take off. The dentists Hume and Mount foresee the expansion of this dental paradigm, in which surgical treatment will moreappropriately be referred to as repair, to incorporate bacterial tests and mineral balance monitoring in the future. In this way bacterial levels can be monitored along with calcium phosphate levels in the saliva. The use of various bactericides, including natural ones like xylitol, which starves the bacteria population, can then be used to reduce the amount of bacteria. Then, calcium levels can be increased through supplemental intake and on-site administration. With the aid of these technologies, dentists will be able to detect diseases at the molecular level before irreparable harm (like dental cavities) is done. If the disease is already there, they will then treat it so that it can be cured. They also believe that as we learn more about how remineralization might be used to restore early dental caries lesions, better remineralizing therapies will be created.

CALCIUM PHOSPHATE AND ITS ROLE IN TREATMENT

Amorphous calcium phosphate treatments that are given directly to the teeth are just lately the subject of research. By increasing the amount of bioavailable calcium in the saliva and directly on the teeth's surface, this treatment aims to create an environment where teeth are more likely to remineralize surface caries lesions. This remedy is a cream that is applied directly to the teeth with a toothbrush or put in a dental cup. This "has a remarkable potential to stabilise calcium phosphate in solution and dramatically raise the level of calcium phosphate in dental plaque," when casein phosphopeptides (CPP), components derived from milk, are added. However, it does highlight the need for further research into the various stages at which it is possible to promote remineralization and subsequently repair in the teeth using solutions such as the amorphous calcium phosphate. At the moment, this treatment is not being evaluated as a replacement for the surgical model of dentistry.

The efficiency of such a paradigm in the face of severe late-stage caries lesions is still mostly unknown.A Russian-based study that looked at deep caries lesions and came to the conclusion that there was a favourable effect on these deep lesions seemed to be alone in this investigation.They demonstrated that “tri-calcium phosphate normalised pulp function, promoted remineralization of dentin in the bottom of carious cavity, and were effective in treating deep caries.This highlights the need for more research but is not sufficient to support this paradigm in the treatment of late stage caries lesions Calcium phosphate is effective in repairing early caries lesions, according to several studies. This suggests that calcium treatment could be a significant

substitute for drilling and filling the lesions, which would harm the tooth structure and make it more susceptible to bacterial invasion in the future if the lesions can be found when they are still minor.The maintenance of original tooth structure is crucial since it is widely recognised that no restorative material can ever fully replace native tooth structure over the long run.It is clear that demineralized tooth structure can be partially repaired and remineralized.Therefore, just because dentin and enamel have lost calcium and phosphate ions as a result of acid damage does not mean that they should be removed.Several random clinical experiments using mineralized mouthwash and chewing gum as well as ones that specifically looked at the interaction between milk casein calcium phosphate and fluoride to assist boost total mineral content have demonstrated the effectiveness of re-mineralization.Early lesions have been reported to heal pretty well when treated with calcium. It has been demonstrated that it works even better when paired with fluoride.This method appears to be quite effective for treating early caries lesions, potentially avoiding the need for early drilling of teeth. It is yet unknown how much re-mineralization is possible and what exactly has to be done to promote it.

This therapy approach is crucial for treating early childhood tooth decay since it is far less invasive and may be less harmful psychologically to the growing young mind.When a child is this young, the only options for reconstructive dentistry are sedation, which is frequently performed in a hospital and can last up to 2 hours of general anaesthesia with unknown effects on the developing mind, or forced restraint during the procedure, which the child is frequently quite terrified of.Any solution that may be used to prevent this invasive treatment on a youngster who is still developing merits research, without a doubt.Young cells in other parts of the body have demonstrated a considerably larger capacity for repair than do adult cells, thus early repair in children theoretically might have much greater ramifications and possibly progress more successfully.Since calcium phosphate therapy for dental lesions is a relatively young and developing industry, research on its effectiveness in children is currently lacking.Consumers can now enjoy calcium in nearly every brand of toothpaste they purchase, since many creams are now sold straight to dentists with the goal of usage as an at-home prescription therapy.Calcium phosphate is the principal component of toothpastes that reduce tooth sensitivity.The majority of calcium treatments utilised nowadays come in the form of inexpensive toothpastes that contain little quantities of calcium.

VITAMIND

Result

There were 207 women registered in total (meanage:196 5years).The bulk(82%)of candidates came from the field of health sciences and 93% of them were residentsof Winnipegeven though 71%claimed to take supplements,only37% of pregnant women exercised every day.

For 200 subjects, complete laboratory findings were available (Table 2). The median 25OHD level was 43 nmol/L, and 65 participants (32.5%) had deficient concentrations. The mean 25OHD level was 48 6 24 nmol/L.nmol/L), and 24 (12%) had ideal concentrations. ($75 nmol/L). Participants travelling through blood tests during the winter (November-April) saw a marked decline levels than those recorded from May to October throughout the summer: 38 6 22compared to 55 6 24 nmol/L (P , .001). When Seasonally categorised, there was no discernible variation in 25OHD levels between individuals who spents pending time in the fresh air and those who omitted.

In spite of follow-up losses, 64% (n = 133) of the cohort showed up for the newborn follow-up appointment. There were no variations age (P =.24), educational attainment (P =.74), and gender ethnic background (P =.24) between lost to follow-up were women, and those staying put in the study. Furthermore, Between these 2 groups, the 25OHD levels were the same (50 6 26 vs.P =.08; 45 6 20 nmol/L. totaling 135two sets of twin babies with a mean age of16.5 months (median: 13 months)returned. In general, 56% of newborns were male.

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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)			.25
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 ± 35	
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	

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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.

Enamel hypoplasia was found in 22% of the cohort (29 of 134), with pits and absent enamel being the most common manifestations.31 infants (or 23%) had ECC at the time.Caries was only found in cavitated teeth. Enamel blemishes although, when white spot lesions of the enamel were also present.36% of newborns, 49, had ECC. Dt's mean1.2 6 2.1 was the score (range: 0–10), while the average score for the quantity of extracted, decaying, and filled was 1.5 6 2.8 teeth (range: 0–17).The dt score was 3.4 6 2.0 when white spot incipiencies were taken into account.

Based on the existence of cavitated caries lesions, mothers of infants with ECC had significantly lower prenatal amounts of 25OHD than mothers of infants without ECC.Children had no cavities (P =.05.White spot lesions, however, were Between groups, there was no distinction. There are no real connections. Between moms' 25OHD were discovered.ECC and concentrations in their infants in the event of a deficit (,35 nmol/L) or optimum thresholds ($75 nmol/L) were (P=.36 and P =.38, respectively) were used.

An important inverse link between the average number of decaying teeth (dt score) and levels of prenatal 25OHD (P =.0002). Babies whose mothers have lower 25OHD pregnant women's concentrations had dt scores that are much higher. t testing analyses were conducted to evaluate the relation between the dt number scores with 25OHD criteria for determining de-optimum and efficient concentrations.

The mean number of primary teeth with decay showed no discernible difference. Among children whose mothers have 25OHDlevels of #35 nmol/L or.35nmol/L (Table 4). Surprisingly, children of mothers with the best 25OHD levels($75 nmol/L) had a statistically less significant mean dt score compared to those whose moms had levels below this cutoff(P = .03).

Aboriginal women were much more likely to be the mothers of infants with ECC. having previous children with ECC (P =.005), rating their own health as average or poor (P =.01), rating their own health as average or bad (P =.02), and drinking less milk when pregnant (P=.01)..01). Additionally, they had a higher likelihood to depend on food banks and earn little money(P , .005).

According to the x2 analysis, babies with enamel hypoplasia had a considerably higher chance of developing ECC (73% vs 27%; P,.001). Children with ECC were notably more older than individuals without caries (19)P =.001) (610 vs. 14 64 months). In ECC, neither breastfeeding nor bottle feeding were connected in a significant way (P =.86)..35). In the group of 21 infants who were reportedly stopped using bottles to feed infants, No

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distinction could be made between the Weaning age, ECC, and presence or absence (P =.051). Nonetheless, those who were still utilising Sippy cups, and significantly lower chance of having ECC (P .001).

ECC was present in a considerably lesser percentage of the 117 kids whose teeth were being cleaned (P =.02). However, There was no discernible variation when comparing groups based on mean ages Caretakers started to wash their newborns'molars (P =.07).Anal regression IN order to evaluate the links between independent factors and the dependent results of ECC and dt scores. These models addressed the prenatal serum metabolite themes concentrations, and variables affecting vitamin D-level achievement, baby feeding techniques, family characteristics and finances, enamel family dental history, hypoplasia, and awareness (models not shown)

A comprehensive final logistic regression model for ECC was built using 12 different variables . Several were significantcantly connected to ECC in the past models, or approximations, of the whilst others were either not significant or were they significant at the bivariate level generally recognizable ECC contributors the literature on risk Additional elements that affect vitamin D levels likewise included.

Results showed that the age of babies at the time of the dental examination (P =.01) and the existence of enamel hypoplasia (P =.001)substantial correlations with ECC,those who are 14 months or older at the length of their examination was longer probable ECC presence. Additionally, 25OHD levels were observed to increase during pregnancy be strongly connected to ECC.(P = .05). Logistic regression going backward additionally, analysis was done, with the Final revision showing that the enamel infant age (P =.01), hypoplasia (P =.001), and Lower 25OHD levels (P =.02) and higher.001) were both substantially and separately

after adjusting for income and work status, infant feeding practises, season, and other factors practise of baby oral hygiene. For the dt score, Poisson regression was used. Including the identical independent variables that were used inthe more comprehensive logistic regression modelto ECC. Similar findings showed thatbaby age, the occurrence of hypoplasia in the enamel, and maternal 25OHD levels pregnant women were substantially more connected to dt score (Table 6).Lower grades and lower 25OHD levels greater dt scores were linked to better measures of children's health (P =.04).

III. DISCUSSION

Although some research has suggested a link between vitamin D status and dental caries,10–12,26 no prior studies have examined to the best of our knowledge, research has evaluated the link between prenatal 25OHD levels prospectively during the growth of the teethand birth defects in progeny. Many females has poor levels of 25OHD; virtually90% had amounts below the cutoff for adequacy.

Low levels raise the chance of developing a variety of chronic illnesses, such as osteoporosis, cardiovascular disease, and periodontal disease. 27–29 Unfortunately, this study's majority of women would require taking. Daily 2000 IU of vitamin D to boost their levels to80 nmol/L.30 daily 400 IU dosages of8 weeks of vitamin D3 use leads in an only a rise of 11 nmol/L.29.

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.

Compared to moms of children without caries, mothers of children with ECC had considerably lower levels of 25OHD. Additionally, there was a negative correlation between prenatal 25OHD and the dt score, with lower amounts indicating higher scores for primary teeth with

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decay. Those kids whose moms had optimum 25OHD concentrations during pregnancy ($75 nmol/L) had far less bdt results.

Early research found a link between vitamin D-fortified foods and sun exposure, as well as a decrease in the frequency and severity of caries.a reduction in the frequency of enduring enamel hypoplasiateeth.31,32An updated meta-analysis highlighted that the addition of UV light, vitamin D2, or vitamin D3.Caries risk was minimised by light.21 Additionally, it has been suggested concentrations of 25 OHD between 75 and100 nmol/L provide protection against caries.

Children with S-ECC.34 had considerably reduced 25OHD levels, according to a recent case-control study. In recent times, web have also reported comparable results in a larger sample of S-ECC and 25OHD associations, even after adjusting for season, milk consumption, and usage vitamins, as well as household income .Despite the fact that these 2 studies do not prove a connection, they offer more proof of a connection between early childhood caries and decreased 25OHD

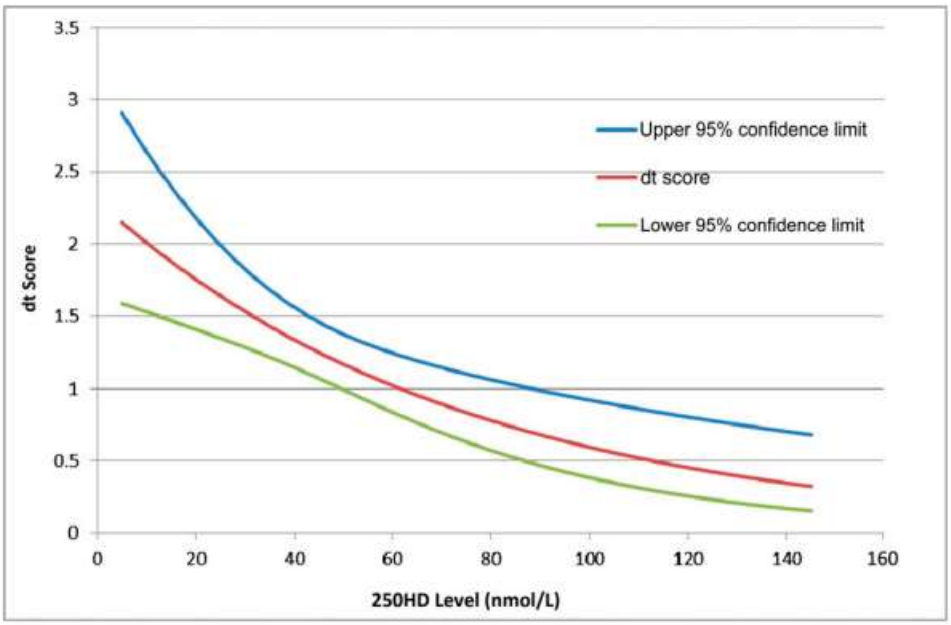

FIGURE 1
Predicted number of decayed primary teeth (dt score) according to 25OHD level.

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.

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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

This study emphasize the prevalence of ECC in a group primarily made up of Canadian Aboriginal kids. When cavitated lesions were taken into account, nearly one-quarter of the children had ECC, and when incipient lesions were taken into account, the number increased to more than one-third. 36This result is comparable to the 30.4% of Manitoba infants who were 24 months old and had ECC37, which was reported .Regrettably, 23% of the babies in this study met the S-ECC criteria.

To adjust for the impact of confounders and see if prenatal 25OHD levels were related to ECC, regression modelling was required. The final model included a number of related factors because income, poverty, and employment can all affect the chance of developing caries. Age was also included in the model because it is a known predictor of ECC. Children that were tested at 14 months of age had a higher likelihood of developing ECC. The model also took into account milk consumption, dental hygiene, season, and newborn feeding habits. After adjusting for these factors, enamel hypoplasia, infant age (≥14 months), and 25OHD levels were found to be significantly and independently linked with ECC. In our group, enamel hypoplasia was a potent predictor of ECC (odds ratio,.8), lending support to the idea of hypoplasiaassociated S-ECC. 3,7.

The same independent variables were included in the Poisson regression model for the untreated primary tooth decay score (dt). added to the expanding logistic ECC regression model. Age

at first birthday, 25OHD levels, and enamel hypoplasia were once more recognised as reliable independent predictors of caries scores According to this blinded prospective study, babies whose mothers had lower 25OHD levels are noticeably more likely to have ECC. Although the specific process is unknown, it is probable that reduced vitamin D levels during tooth development lead to enamel that is less resistant to caries.

There were, of course, certain restrictions on this study. Given the difficulties many of its members were experiencing in their personal lives, attrition within the cohort was anticipated. Our capacity to create intricate multivariate models was constrained by the cohort size. Despite a number of losses to follow-up, there was no appreciable difference in 25OHD levels between the research participants and those who were lost to follow-up. There were fewer questions as well. In retrospect, they should have done a better job of fully examining several potential variables that would have affected newborn oral health status, notably caries risk.

The findings' generalizability might potentially be constrained. This investigation was not a random sample, but rather convenience. Nevertheless, the study sheds light on the nutritional state. dental health and that of pregnant mother of their young. We did so on purpose. a group at high risk for both ECC and low vitamin D levels, enabling this generalizable study to this urban Indigenous people.

The study was somewhat sized overall and purposefully included a high-risk sample of predominantly urban Aboriginal respondents with low incomes and educational levels. The prospective strategy made it possible to observe caries' natural course and evaluate a number of outcomes in response to a single exposure (ie, 25OHD status). A temporal order was also established, covering pregnancy through infancy. Reduced bias is another benefit of cohort studies. Another noteworthy strength was that dental evaluations were performed without knowledge of maternal 25OHD levels by the researchers.

The results of this study may have an impact on early childhood dental health policies. As a potential tactic to lower the risk of caries, efforts to increase nutrition during tooth creation in utero and in early infancy should be looked into. Preventive measures should start during pregnancy by improving maternal nutrition, either through better food or vitamin D supplements

OBESITY AND SKELETAL MATURITY

Resources and techniques

After receiving written informed consent from their parents or legal guardians, 107 healthy patients between the ages of 6 and 12 (mean age 8.771.79) underwent anthropometric measurements, BMI calculations, and DXA exams at the Department of Neuroscience, Human Nutrition Unit, University of Rome "Tor Vergata," as well as the assessment of skeletal-dental age at the Paediatric Dentistry Unit of PTV Hospital, University of Rome "Tor Vergata." The existence of endometabolic and systemic illnesses was a requirement for group exclusion. 1) Anthropometric measurements: height (cm) was measured using a stadiometer to the nearest 0.1 cm, and body weight (kg) was measured to the nearest 0.1 kg using a scale (Invernizzi, Rome, Italy) (Invernizzi, Rome, Italy). 2)The recommended formula was used to compute the BMI.

3) DXA test A fan beam scanner, the DXA (Lunar model DPX-1Q Lunar Corp., Madison, WI) was used to analyse the body composition. The DXA exam measures bone mineral density (BMD), bone content, body fat mass (FM), and body fat free mass (FFM) (BMC). Within and between subjects, the coefficient of variation (CV% 100x SD/mean) varied from 1% to 5%. Bone measurements have a coefficient of variation that is less than 1%. This method results in an effective radiation dosage of roughly 0.01 mSv.

4) Skeletal age. The determination of the skeletal age was performed through the assessmentofemploying lateral cephalometric radiographs and the formulas created by Caldas et al. [2010], the cervical vertebral maturation (CVM) was calculated, giving the measurements of the third (C3) and fourth (C4) cervical vertebrae.Age of the female cervical vertebrae is calculated as follows: 1.3523+ 6.7691 x AH3/AP3+8.6408xAH4/AP4. Age of the cervical vertebrae in men is calculated as follows: 1.4892+11.3736x AH3/AP3 + 4.8726 x AH4/AP4 (AH= anterior vertebral body height; AP = anterior-posterior vertebral body length; C3 = C4 = 5) The Demirjiyan and Goldstein method [Demirjiyan and Goldstein, 1976) and panoramic radiographs were used to assess the dental age.

Criteria for classifying subjects

According to Mc Carthy's age-sex-specific centile curves, the patients were categorised as pre obese/obese. The 95^{th} and 85^{th} centiles were chosen to define the lower bounds of pre-obesity and obese, whereas the 2^{nd} centile was

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chosen to define the upper limit of underweight [McCarthy et al., 2006]. The individuals were split into four categories according to FM% McCarthy classification: Group A underweight (FM%2nd centile), Group B normal weight (2nd centile 95th centile). B) Using growth charts particular to the Italian population, the individuals were classified as pre obese/obese based on their age and sex-specific BMIs (BMI classification) [Cacciari et al., 2006]. The cut off points for evaluating participants who were underweight, overweight, or obese were the third, the 75th, and the 95th centiles. The subjects BMI classifications reveal that were separated into four groups: Group E, normal weight (3rd centile-75th centile), Group F, preobese (75th centile-95th centile), and Group H, obesity/RMI-95th

Statistic evaluation

The SPSS software was used to conduct the analysis (version 16; SPSS Inc., Chicago IL,USA). The One-Way-Anova test was used to analyse the assessment of differences between the two groups based on the means of continuous variables. With the exception of Groups A E, the Student's t test was also used for each group of kids between the ages of skeletal-dental age and chronological age using McCarthy and BMI classifications. The Nonparametric Mann Whitney test was used to determine whether there were any differences between the means of chronological and skeletal-dental ages for Groups A through E. McCarthy cut-offs categorization and the gender categorical variable were tested using the Fisher's exact test, as were McCarthy and BMI.classifications. Besides, to evaluate the association between skeletal and dental age, a Pearson correlation coefficient was calculated.In all the assessments a significant level of alpha = 0.05 was considered

IV. RESULT

The sample of 107 kids, aged 6 to 12 (mean age 8.771.79), is made up of 57 girls (53%) and 50 boys (47%). There were no statistically significant differences in the proportion of men/women among Groups A, B, C, and D, according to the Fisher's exact test (p=0.41). The Anova analyses revealed a statistically significant difference between Groups A, B, C, and D as well as between skeletal (p=0.03) and dental age (p=0.02), although the difference between Groups for chronological age is not statistically significant (p=0.22). Nearly the highest (Pearson correlation coefficient=0.994) and statistically significant association is between dental and skeletal age.

The FM% McCarthy categorization states that the skeletal-dental age always rises relative to chronological age as the FM% increases, moving from children of normal weight to those with obesity. For children of normal weight (Group B), pre-obese children (Group C), and obese children (Group D), this increase in skeletal-dental age equates to 6 months, 16 months, and 17 months, respectively (Group D).

		MEAN	SD	P-VALUE (ONE-WAY ANOVA TEST)
Chronological Age	Group A	9.30	1.72	0.22
	Group B	9.52	1.66	
	Group C	8.58	1.63	
	Group D	9.35	1.66	
Skeletal Age	Group A	8.50	1.83	0.03
	Group B	9.97	1.83	
	Group C	9.93	1.85	
	Group D	10.74	1.85	
Dental Age	Group A	8.34	1.77	0.02
	Group B	10.01	1.83	
	Group C	9.93	1.88	
	Group D	10.74	1.78	

TABLE 1 Mean and Standard Deviation (SD) of chronological, skeletal and dental age in McCarthy classification.

		MEAN	SD	P-VALUE (ONE-WAY ANOVA TEST)
Chronological Age	Group E	9.8	1.8	0.16
	Group F	9.0	1.6	
	Group G	9.2	1.4	
	Group H	9.9	2.1	
Skeletal Age	Group E	10.0	2.1	0.12
	Group F	10.0	1.8	
	Group G	10.6	1.6	
	Group H	11.3	2.3	
Dental Age	Group E	10.0	2.1	0.08
	Group F	10.0	1.8	
	Group G	10.6	1.6	
	Group H	11.3	2.1	

TABLE 2 Mean and Standard Deviation (SD) of chronological, skeletal and dental age in BMI classification.

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	BMI (kg/m²)	FM (%)	FFM (kg)	FM (kg)
Skeletal age	0.503[1]	0.409[1]	0.757[1]	0.580[1]
Dental age	0.516[1]	0.415[1]	0.762[1]	0.558[1]
BMD trunk (g/m2)	0.691[1]	0.514[1]	0.824[1]	0.690[1]
BMD ribs (g/m2)	0.752[1]	0.633[1]	0.770[1]	0.747[1]
BMD pelvis (g/m2)	0.567[1]	0.354[1]	0.786[1]	0.561[1]
BMD vertebral column (g/m2)	0.596[1]	0.479[1]	0.676[1]	0.604[1]
BMD whole-body (g/m2)	0.619[1]	0.406[1]	0.782[1]	0.603[1]
BMC trunk (g)	0.786[1]	0.643[1]	0.889[1]	0.801[1]
BMC ribs (g)	0.847[1]	0.757[1]	0.800[1]	0.853[1]
BMC pelvis (g)	0.626[1]	0.455[1]	0.888[1]	0.653[1]
BMC vertebral column (g)	0.643[1]	0.515[1]	0.766[1]	0.651[1]
BMC whole-body (g)	0.714[1]	0.521[1]	0.937[1]	0.722[1]

TABLE 3 Spearman's correlation coefficients ([1]p≤0.001), between skeletal-dental age, BMI, FFM, FM and bone mass (BMD, BMC).

Regarding the delay, a discrepancy between chronological age and skeletal-dental age is present in 60% of underweight children, 4.2% of children of normal weight, and not at all in children who are pre-obese or obese. On average, Group A children have skeletal-dental ages that are 12 months younger than their chronological ages. The difference between chronological and skeletal-dental age is statistically significant for pre-obese (p=0.01) and obese (p0.001) children, but not for underweight (p=0.46) or normal weight children (p=0.33). If we take into account Groups A, B, C, and D, these differences are statistically significant (p0.001).

The Fisher's exact test was performed to compare the FM% McCarthy Groups A, B, C, and D to the BMI Groups E, F, G, and H, and it revealed statistically significant differences between the two classifications (p 0.001).

For chronological, skeletal, and dental age, the Anova test did not reveal any statistically significant differences among Groups E, F, G, and H.

It is possible to see that within the group of children with normal weight (Group F) that there are statistically significant differences between chronological age and skeletal-dental age (p=0.01); the skeletal-dental age, in fact, is greater than the chronological age by a value of 11 months. This is done by applying the same inferential analysis and by taking into account the BMI classification
Additionally, the skeletal-dental age for the group of obese children (Group H) is greater than 17 months, but this information is not statistically significant (p=0.09).An evaluation of the association between the indicators was done in order to comprehend the connection between bone mineralization and body composition

V. DISCUSSION

Studies that examined the relationship between juvenile obesity and skeletal maturation and dental age used the BMI index and frequently found conflicting findings [Akridge et al., 2007; Basaran et al., 2007; Chen et al., 2010; Eid et al., 2002; Hilgers et al., 2006].

According to Mei et al. (2002), the BMI index is the most widely used index to categorise overweight-obesity. It is primarily used in aepidemiological studies because it is simple to calculate, non-invasive, inexpensive, and supports a quick data comparison. The sole drawback is that it cannot differentiate between FM and FFM, making it impossible to determine the body composition.Instead, DXA is a technique for determining body composition, measuring the FM and the FFM; however, because of its complexity and expense, this technique is only used in research settings [Mei et al., 2002].There are statistically significant variations between BMI and FM% (p 0.001) when comparing BMI and DXA data.According to the FM% (McCarthy cut-offs) categorization, it is shown that the skeletal-dental age is older than the chronological age as FM% rises, or as children go from normal weight to obese weight. For pre-obesity (p=0.01) and obese (p0.001) patients, the difference between chronological age and skeletal-dental age is indeed statistically significant.The BMI categorization does not show this tendency; in the group of obese participants, the skeletal-dental age is older than the chronological age, but this difference is not statistically significant (p=0.09); however, this difference appears to be significant when analysing Group F (p=0.01).When compared to DXA, the BMI incorrectly identified the adiposity status of a paediatric group, which may be the cause of these conflicting results.According to Akridge et al. [2007], there are no statistically significant

differences between normal weight and overweight subjects and obese subjects in terms of skeletal-dental maturation, however it is noted that the skeletal age of the obese participants is accelerating.According to several authors (Eid et al., 2002; Hilgers et al., 2006), overweight and obese patients exhibit a faster and statistically significant dental development when compared to subjects who are not overweight or obese.The findings of this study support previous research [Akridge et al., 2007; Chen etal., 2010; Basaran et al., 2007] that found a link between dental and cervical vertebral maturity. Instead, according to other writers [Demirijan et al., 1985], skeletal and dental development are under the control of many systems.Our findings demonstrated for the first time a link between the acceleration of skeletal-dental ageing and the DXA-measured body fat percentage. Additionally, the data show that bone mineralization strongly correlates with the FFM% rather than with BMI or FM%The adipose tissue, on the other hand, produces metabolically active molecules, such as the adipokines (leptin, adiponectin, proinflammatory cytokines) playing a role in the regulation of the bone metabolism, contrary to some authors' reports [Wang et al., 2005] that the FFM is an important predictive factor for the BMD.If obesity is linked to rapid skeletal-dental maturation, paediatric dental and orthodontic treatment timing may differ for these kids.In fact, when it comes to paediatric dental planning, the early eruption of permanent teeth at a time when the kids would not be able to practise good oral hygiene could lead to a rise in dental caries [Akridge et al., 2007; Costacurta et al., 2011; Hilgers et al., 2006]Additionally, orthodontic diagnosis, treatment planning, and treatment outcomes may be affected by an acceleration of skeletal-dental maturation in obese patients. When preparing for orthognathic surgery and orthodontic growth modification, the amount of possible remaining facial development is crucial [Akridge et al., 2007According to Neeley [Neeley and Gonzales, 2007], "the orthodontic therapy can be affected by obesity," given the likelihood that obese patients will exhibit irregular pubertal development as a result of the hormonal changes associated with obesity, a different bone metabolism (resulting in changes in growth and development or tooth movement), and specific craniofacial features (increased mandibular length, shorter upper face height, flatter or more concave profile.

VITAMIN D
CONCLUSION

This study provides the first evidence that prenatal 25OHD levels may affect the primary dentition and the growth of the ECC. Particularly, lower levels were linked to a higher incidence of dental cavities in young children. Age of the infant, enamel hypoplasia, and prenatal 25OHD levels were independent predictors of caries.

CALCIUM
CONCLUSION

The future of dentistry appears to include calcium, which could be particularly essential in treating early lesions in youngsters. The prevention of dental cavities before the need for such a therapy is much more crucial.Utilizing the available information on calcium's role in the prevention of early childhood dental caries is definitely essential.It is the responsibility of the public health professional to help parents, doctors, and dental practitioners understand the significance of prenatal and early childhood nutrition.

The entire people must have access to information about optimal calcium levels and assistance with supplemental requirements.Early childhood dental caries, the most common disease in children, might be greatly reduced, if not virtually totally averted, with the right educational initiatives. Stepping into the modern dental environment, a new model is immediately apparent.In order to foster an environment that favours re-mineralization in the early phases of caries formation, this approach would concentrate on early identification of high bacteria levels and use a mix of nutritional support for the body and teeth in addition to bacteria management.Through the management of nutrition and bacterial growth, youngsters may be able to grin brightly throughout adulthood.

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The results highlighted a relation between skeletaldental age acceleration and body fat percentage measured by DXA.

VITAMIN D
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