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Localized Enamel Hypoplastic Spots on the Labial Surfaces of the Primary Cuspids

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LOCALIZED ENAMEL HYPOPLASTIC SPOTS ON THE LABIAL SURFACES OF THE PRIMARY CUSPIDS

by

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VITA

The author, Shu-fei Wang, is the daughter of Chin-Tang Wang and I-Chen Kuo Wang. She was born on March 18, 1960, in Taipei, Taiwan, Republic of China.

Her high school education was completed in 1978 at the Taipei First Municipal Girls High School, Taipei, Taiwan, R.O.C.. In July, 1978, Dr. Wang entered the School of Dentistry, National Taiwan University, and received the degree of Bachelor of Dental Surgeon (B.D.S.) in June, 1984. From 1984 to 1988, Dr. Wang practiced as a general dentist in the World Dental Clinic as partnership in Taipei.

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She was also accepted for the Mott/Hurley fellowship for the post-graduate training in Pediatric Dentistry from Loyola University of Chicago, 1989. Dr. Wang presented the research abstract "Primary Enamel Dysplasia in Hispanic Children" in the 43rd Annual Session of Academy of Pediatric Dentistry, Boston, Massachusetts. She is expected to complete the Master of Science in Oral Biology in May, 1991.

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INTRODUCTION

The isolated enamel hypoplastic spots often seen on the labial surfaces of primary cuspids provokes frequent interest among the dentists who treat children. This interest arises from their amazingly similar and special characteristics, i.e., these areas of hypoplasia are:

(1) non-circumferential,
(2) located only on the labial surfaces of primary cuspids,
(3) predominately found on the mesiogingival quadrant,
(4) small, usually 1-2 mm in diameter, rarely up to 5 mm,
(5) flat to concave (bottom may be smooth to pitted),
(6) roughly circular or oblong,
(7) usually solitary but may be multiple on an individual tooth surface,
(8) white, yellow, or brown, and may be opaque,
(9) barely visible to well-defined,
(10) found in both symmetrical and non-symmetrical patterns even on any individual child,

(11) found in enamel tissue formed during the perinatal period.

Not only pediatric dentists are interested in this localized hypoplastic defect but also the anthropologists who are attracted by the durability of dental tissue and their ability to record health stress arising from a child's environment during growth and development. Because the developing tooth germ is sensitive to a wide range of systemic disturbances and is unable to recover once it is damaged, the tooth enamel can act as a repository of information on systemic insults received during development.

This defect has been continuously reported in prehistoric and modern population. But, the reported prevalence has been varied. Perinatal trauma, localized hypoxia, premature birth, neonatal nutritional disturbances (hypocalcemia), viral or bacterial infection, and the maternal health have been investigated, however, the etiology of this phenomenon remains unknown.

The enamel defects of the primary teeth may have the potential for identification of children who have undergone certain systemic insults during prenatal and early postnatal life. The purpose of this study is to determine the prevalence of these isolated primary cuspid hypoplastic enamel defects in a Hispanic population. Additional purposes are to relate the occurrence of this defect to a limited number of perinatal occurrences.

CHAPTER II

REVIEW OF THE RELATED LITERATURE

A. Amelogenesis

The formation of enamel posed unique questions in hard tissue biology. The information available about the activities of ameloblasts and their related cells would determine the theories of defect formation. However, amelogenesis was much more complicated than the earlier researchers ever imagined.[1]

Prior to 1940, enamel formation was considered to have two stages: apposition and calcification. In 1940, Diamond and Weinmann[1,2] divided the amelogenesis as a two-phase development consisting of a formative phase, in which the complete organic matrix containing all the mineral matter in a colloidal form was deposited, and a maturation or calcification phase, which was a crystallization of the colloidal calcium salts and later included an influx of mineral matter. Maturation was described as starting at the surface of the cuspal enamel and proceeding cervically in a

plane approximately at right angles to the Striae of Retzius. Deakins[1,3], supported the concept of a withdrawal of organic matter and water during the maturation phase, resulting in a very low content of organic matrix in mature enamel.

Witkop and Sauk[4], 1976, divided the enamel formation into three processes: (1) formation and secretion of an organic matrix, (2) mineralization of the matrix, and (3) maturation of enamel.

Suckling[5,6], used sheep as an experimental animal, producing enamel defects in the central incisors. He summarized the amelogenesis as follows: the main phases of ameloblast activity, (secretory, maturation, and regression) were accompanied by changes in the macroscopic appearance of the enamel. The maturation phase could be divided into an early and a late stage, based on changes in appearance and hardness values. In the early stage the enamel was dull, white, and relatively soft. In the late-maturation phase, the deeper enamel was well-mineralized, but the thin surface layer was still incompletely mineralized, producing an overall diffuse opaque appearance but broken by five or six horizontal transverse lines of translucent mature enamel. With further development, the diffuse opacity disappeared and all the enamel became translucent.

Robinson[7,8], described the four developmental stages according the appearance and chemical composition of human deciduous incisors as: (1) partially mineralized matrix was

secreted and some extracellular breakdown occurred; (2) selective replacement of matrix proteins by tissue fluid began; (3) almost all of the matrix protein was replaced by tissue fluid and an influx of calcium phosphate occurred; (4) the enamel became almost fully mineralized, mature and hard.

Deutsch and Pe'er[9], using Robinson's technique[7], studied the development of human fetal teeth and confirmed the presence of at least two different types of protein, amelogenins and enamelins from the amino-acid composition of the soft, forming enamel. The amelogenins were the predominant proteins in forming enamel, while enamelins represented only a small fraction of the total protein at this stage. During maturation, the amelogenin proteins were selectively lost, resulting in an increased enamelin concentration in the mature tissue. They also estimated the average rate of enamel formed per day along the tooth axis was about 0.023 mm per day. The rate at which the maturing enamel spread from the incisal tip toward the cervical margin was faster than the rate of enamel growth, being 0.04-0.05 mm per day.

Suga[10,11], proposed that the progressive mineralization pattern was completely different between the matrix formation and maturation stages. These patterns of mineralization were almost the same in all the animals examined. He divided amelogenesis into a primary phase corresponding to the matrix formation stage, and secondary, tertiary, and quaternary phases which correspond to the maturation stage of

amelogenesis. The increase in the secondary mineralization took place first slightly, from the surface toward the inner layer, and then heavily, from the inner layer toward the surface. The narrow outermost layer mineralized very slowly during the middle and late stages of maturation, but finally achieved the highest mineralization of the entire enamel layer.

B. Classifications of Enamel Defects

The definitions given by the Commission on Oral Health, Research and Epidemiology of the FDI for enamel opacities (qualitative defect in enamel, abnormality in translucency of enamel), hypoplasia (quantitative defect in enamel, reduced the thickness of enamel), discolored enamel (abnormal appearance in enamel), and developmental defects of enamel (disturbances in hard tissue matrices and their mineralization during odotogenesis) were based solely on simple descriptive criteria.[12] No reference was made to any causative factors involved or to the associated histopathology.

The modifications recently introduced by Suckling[1], 1989, were hypoplasia, demarcated opacities, and diffuse opacities. Hypoplasia could occur in the form of pits, grooves, or larger areas of missing enamel. The enamel of reduced thickness may be translucent or opaque. Diffuse opacities was of normal enamel thickness. It could have a linear, patchy, or

continuous distribution, but there was no clear boundary with the adjacent normal enamel. Part or all of the tooth surface could be affected. Demarcated opacities involved an alteration in the translucency of the enamel, variable in degree. The defective enamel was of normal thickness with a smooth surface. It had a clear boundary with the adjacent normal enamel and could be white, cream, yellow, or brown.[1,13]

C. Characteristics of Enamel Hypoplasia

Enamel hypoplasia is a defect in the enamel due to disturbance of ameloblastic function during amelogenesis. Apart from different types of genetically based enamel hypoplasias, amelogenesis imperfecta, which were very rare in contemporary peoples (1:8-15,000)[4,14] and perhaps more so among prehistoric samples,[15,16] two basic types of enamel hypoplasia have been observed.

Type one was caused by systemic effects followed a chronologic linear distribution pattern affecting many teeth forming simultaneously. This was a linear hypoplasia which followed the lines of tooth formation. The width of the defective enamel band was indicative of the duration of the insult.[17] The other type was gross, non-linear in distribution and usually occurred on the affected tooth individually. It was attributed to localized effects such as direct trauma (e.g., endotracheal intubation[18]) or indirect

trauma, (e.g., so called "Turner's teeth"[19,20]), or of idiopathic origin.[20]

Two localized types of enamel hypoplasia affecting the primary dentitions have been described in the literature. The first was a transverse encircling groove often on maxillary primary anterior teeth. The second affected only labial surfaces of primary canines and was isolated in appearance.[21]

D. Pathogenesis of Enamel Hypoplasia

The literature on the pathogenesis of the enamel hypoplasia is confusing. It is probable that systemic disturbances and local factors both contribute to the etiology[14,22]. However, it is difficult to show a predictable cause-and-effect association between the possible factors and enamel hypoplasia. According to Spouge[23], the formation of enamel involved a rhythmic sequence of cellular activity, interspersed with resting phases. Selective involvement of only those ameloblasts that were currently active at the time of a particular disturbance may account for the variability in development of the lesions. Some of the affected ameloblasts may die and stop secreting enamel, whereas other may recover and continue to secrete normal enamel over the defective spots, which could also help to explain the variability of the enamel lesions.

Small and Murray[24], 1978, and Pinborg[25], 1982, have

reviewed the literature on the etiology of developmental defects, and have identified over 90 factors to be responsible for enamel defects. However, a critical review showed that only a few of these have been associated with enamel defects in primary teeth.[26]

<A> Localized Trauma to the Primary Tooth Buds

Laryngoscopy and endotracheal intubation applied during the neonatal period may contribute to the unilateral localization of hypoplasia in the primary maxillary anterior region.[18,27,28]

Moylan et al.[18] studied 158 children who had required oral or nasal tracheal tubes as neonates. Twenty-eight of 158 (17.7%) had 90 defective teeth. Seventy-seven defective teeth were maxillary incisors (85.6%). The right central or lateral incisors were involved 1.7 times as much hypoplasia as the left. Seow[27] reported 85% of low birth-weight infants who had prolonged endotracheal intubation had defects of maxillary anterior teeth. Comparing with only 21.7% of the control neonates showing defects, the difference was statistically significant ($P<0.001$). However, Seow found that the defects occurred on the left side was twice that on the right. Kopra et al.[29] also reported maxillary incisor hypoplasia and a higher involvement of the left central incisor of neonates who had been intubated. Differences in individual techniques of

laryngoscopy may account for these apparently inconsistent data.

Suckling[5,6], 1980, Using sheep as an experimental model, was able to reproduce all the common enamel defects seen in children. Sudden severe physical trauma caused either hypoplastic grooves and missing enamel, or demarcated yellow or white opacities, depending on the phase of ameloblast activity: secretory, early, or late maturation, respectively.

<B> Systemic Disturbances

(1) Ingestion of Chemicals

* Fluorides:

It has long been established that excessive consumption of fluorides while teeth were developing caused hypoplastic enamel defects in permanent teeth, but such defects in primary teeth were thought to be rare.[30] Several recent studies have shown various degrees of dental fluorosis in deciduous teeth of children born and reared in medium- and high- fluoride area.[31,32,33]

An increasing number of reports indicated that the prevalence of fluorosis may be increasing among children in fluoridated and non-fluoridated communities. Reasons for the increases may relate to misuse of dietary fluoride supplements, ingestion of fluoride toothpastes, or increasing

amounts of fluoride in foods or the atmosphere.[34]

* Tetracycline:

Hypoplasia of enamel of the primary dentition associated with use of tetracycline therapy was first reported by Wallman and Hilton[35]. The dose, time of administration, and type of drug all influence the nature and extent of the discoloration.[14,19,20]

(2) Prematurity/Low Birth Weight

The transition trauma from intra-uterine to extra-uterine life can be observed histologically in the tooth as a neonatal line commonly present even in clinically normal people, but exaggerated in proportion to severity of systemic disturbances experienced in the perinatal period.

Seow et al.[36] found enamel hypoplasia in all of 15 prematurely born children with neonatal rickets. In another study, Seow et al.[37] found enamel hypoplasia and opacities to be more frequent in prematurely born children, increasing with decreasing birth weight. However, the very-low-birth-weight group included children intubated for respiratory distress, in whom local trauma might have been a factor.

Enamel hypoplasia and discoloration were more common in low-birth-weight (<2500 g) Japanese children, regardless of whether the intra-uterine period was less than or more than 34 weeks[38]. The data were, however, complicated by neonatal

problems such as hyperbilirubinemia, respiratory distress, and hypocalcemia, found in different subjects. Johnsen et al.[39] also found higher frequencies of enamel hypoplasia in the primary incisors of low-birth-weight infants compared with control.

Tsang et al.[40,41] reported that prematurely born infants had a substantial rate of developmental defects of enamel; they also tended to have low calcium stores and disturbed calcium metabolism, with the lowest-birth-weight children most severely affected. Disturbance in calcium homeostasis seemed to be a factor of importance in the etiology of enamel defects, and a number of illness underlying prematurity include hypocalcemia as a feature.[26]

(3) Malnutrition

Several investigators have surveyed the prevalence of hypoplasia in primary incisors of children in Guatemalan villages, presumably suffering from malnutrition. Hypoplasia was found to range from 22 to 43% by Sweeney[42,43] and 18 to 62% by Infante and Gillespie[44] in different villages. They also reported a high correlation with the incidence of weanling diarrhea. Diarrhea may conceivably have acted through malabsorption of fat-soluble vitamin D with consequent failure to absorb calcium. According to Nikiforuk and Fraser[17,22], hypocalcemia may be a common feature of these two systemic

conditions, malnutrition and diarrhea, and may indeed underlie enamel hypoplasia associated with a variety of other factors.

(4) Neonatal Hypocalcemia

Nikiforuk and Fraser[17,22] evaluated the teeth of children with disturbances of calcium and phosphate homeostasis. Extensive interglobular dentin but no enamel hypoplasia was noted in the primary and permanent teeth of 25 patients with X-linked hypophosphatemia. These patients were normocalcemic but very hypophosphatemic. Severe enamel hypoplasia of the permanent teeth, with moderate interglobular dentin, was observed in seven children with vitamin-D-dependent rickets; the children were also hypocalcemic and hypophosphatemic. Severe enamel hypoplasia of both primary and permanent teeth but no interglobular dentin was seen in 15 of 21 children with hypoparathyroidism, who were hypocalcemic and hyperphosphatemic. The authors hypothesized that enamel hypoplasia in these conditions was caused by hypocalcemia, and interglobular dentin by hypophosphatemia.

(5) Hemolytic Disease/Hyperbilirubinemia

Erythroblastosis fetalis, caused by RH factor incompatability between the fetus and the mother, might be manifested in the intrinsic staining of the primary teeth by

deposition of bilirubin in the teeth during fetal development. Enamel hypoplasia was also reported occurring in some cases. This usually involved incisal edges of the anterior primary teeth and the middle portion of primary canines and molars.[14,20]

(6) Maternal Diabetes

Infants of diabetic mothers exhibited an excess of symmetrical hypoplastic defects and opacities in primary teeth, and the excess may be less in offspring of women whose diabetes was well-controlled.[26]

(7) Viral Infections

Evans appeared to have been the first to report disturbances of tooth enamel formation in Maternal Rubella Syndrome children, quoting an incidence of 20%, whereas Gregg found defects in 50%. In the research conducted in a pediatric hospital, Hall found children with Rubella Embryopathy had a prevalence of surface defects of 81.8%[45].

Like rubella, cytomegalovirus (CMV) infections active during gestation or latent at time of birth can affect the primary dentition. Stagno et al. reported that enamel defects in 40% of 25 children with the more severe congenital CMV infection, compared with 5% in 93 with asymptomatic CMV infections.[26]

E. Primary Cuspids Hypoplasia

Part I. Characteristics

This defect was first reported by Jorgenson[46], 1956, who examined primary cuspids from both modern and medieval Danes. He observed that the labial surfaces of upper and lower primary canines displayed local enamel hypoplasia with frequent occurrence. These defects were considerably variable in size. The solitary type was most frequent, but a multiple type was also found. The hypoplastic areas were quite irregular in outline. They were always found within or on the boundary of the mesiogingival quadrant of the labial surface, but without any definite relation to the anatomic surface structures. Also, these areas manifested in a graduated scale of enamel deficiences, ranging from barely visible through the discolored and well-defined. The defects were confined to the neonatal and/or early postnatal zone.

Skinner[47], an archaeologist, described the defect as a roughly circular hypoplastic enamel patch approximately 1-2 mm in diameter with flat bottom extending partially or completely through the enamel. They were found on the labial surfaces of primary canines at approximately the junction of the gingival and middle thirds of the tooth.

Part II. Prevalence

Jorgenson reported that the hypoplastic defects occurred in 21% to 28% of both modern and medieval Danish primary canine teeth (870 teeth).[46, 58] The defect was reported by Skinner[48] to occur in 29% of 83 primary canine teeth of a cadaver sample from Calcutta, but in more than 50% of canine teeth selected from an Upper Paleolithic sample from Western Europe (20 teeth) and in a late Neolithic sample from the Middle East (34 teeth).

Recently, Badger[49], a U.S. Army Pediatric Dentist, found that 22% of all primary canines had at least one hypoplastic area, and that 44-46% of the 55 children he examined had a hypoplastic defect on at least one primary canine. He reported equal occurrence in males (25) and females(30). Brown and Smith[50] found 16.5% of all the primary cuspids examined had defects and an overall prevalence of 36% of the 112 children examined at Indiana University, School of Dentistry, and a slightly higher incidence in males. In addition, they found this lesion affected the mandibular canine twice as frequently as the maxillary canine. Silberman et al.[51] found that the prevalence of hypoplasia on the labial surfaces of mandibular primary canines in the Mississippi Head Start population is 37.1% for all black children and 34.5% for all children (344 black and 37 white). The racial differences were highly significant ($p<.001$). Nation et al.[52] studied the

developmental enamel defects of all the primary teeth in a group of Californian children. Of the 300 children examined, black children showed the highest rate of defects (60%), followed by Caucasians (38%), and Hispanics (18%). The racial differences were statistically significant. Of a total of 250 teeth with defects the majority were mandibular canines. Interestingly, the proportion of children with enamel hypoplasia affecting just the primary canine was 18%, a figure that increased only to 20.7% when the entire primary dentition was included.

Skinner[48], 1986, studied the prevalence of this defect in the 2,380 children who were scanned by two dental assistants in Burnaby, Canada, and reported an incidence of less than 1%. Males and females appeared equally likely to show the condition. The incidence was relatively more frequent among Indo-Asian and Black children.

Skinner[21], 1989, reported 33 children (2.4%) having affected primary canines from 1,350 kindergarten-aged children examined by dental assistants in Vancouver, Canada. He further noted that almost all of the affected children came from low-income families, often of East Asian or Chinese origin, in which there was a degree of milk avoidance and reduced breastfeeding.

Noonan[53] in 1965 reported on the examinations of 311 Caucasian children from a non-fluoridated community (Speedway, Indiana) and found 154 children with at least one facial

hypoplastic cuspid --- a prevalence rate of 49.5%. Noonan, also in 1969, reported 200 black children in Louisville, Kentucky, (a fluoridated community) and found 58.5% children had at least one defect. Both studies showed no statistical difference between right and left side, however, more than twice the number of defects appeared in the mandibular arch.

Duncan et al.[51,54], during screening examinations of 371 mississippi Head Start children (3-5 years of age), found that the prevalence rate of dental caries associated with primary canine hypoplasia was 26.0%. They concluded that the presence of primary canine hypoplasia resulted in an increased potential for the tooth becoming carious. They reported that fluoridation appeared to have no effect on the prevalence of this lesion or its subsequent cariogenisity. Johnsen et al.[55] found that 6.5% of the children they examined in fluoridated Ohio communities had carious lesions in hypoplastic defects. In a later study, Johnson et al.[56] reported similar proportions (5-7%) of hypoplastic defects that had become carious in optimally fluoridated, and fluoride-deficient Ohio communities.

Part III. Etiology

According to Lunt and Law primary cuspids began to calcify prenatally at approximately 17 to 18 weeks in utero and that, by birth, approximately one-third of this enamel has

been formed. Enamel formation was reported to be completed at approximately nine months of age.[57,58]. Badger[49], assuming a constant rate of calcification, estimated that the calcification of the cervical third of primary cuspid enamel would begin at 4 months of age. He suggested that disturbance or injury at this time to the ameloblasts might result in enamel hypoplastic defects.[49]

Skinner[47], 1986, postulated the cause of this localized cuspid enamel hypoplasia to be local trauma and related it to the extreme labial position of the developing canine crown, the unusual thinness of the overlying alveolar bone, and the stage of development of the crown. In a separate report in 1986, Skinner with Hung[48] postulated a combined systemic event (hypocalcemia causing alveolar fenestration) and a local traumatic event as the possible etiology of this lesion. Skinner[21] in a later report, 1989, stated that this defect appeared to be caused by minor physical trauma to the face approximately 6 months after birth when normal motor development led to the handling and mouthing of objects which damaged the developing tooth crown through deficient cortical bone over the canine crypt. He attributed the reduced cortical bone in the face of the infant to nutritional factors, involving calcium deficiency, of the mother and /or developing infant.

CHAPTER III

MATERIALS AND METHODS

The sample population of this study consisted of 83 healthy Hispanic children selected at random from Hispanic children brought to a dental health care program[a] in urban Chicago, Illinois, for a dental evaluation. All children enrolled in this program came from communities with poor standards of living. To verify Hispanic lineage, children's grandparents and parents must be of Hispanic heritage.

Only children with all four primary cuspids were selected for this study. Ages ranged from two to nine years old. There were 42 females and 41 males.

All examinations were performed by the author. These children were examined using : an overhead dental light, a mouth mirror, and an explorer. All examinations were conducted with the child in a reclined dental chair.

The evaluations of the presence of the hypoplastic defects were conducted in the following sequences : first, the

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[a]Infant Welfare Society, Chicago, Illinois.

author debrided all the tooth surfaces to be examined using a prophylaxis cup and a slurry of pumice, the teeth were then rinsed, and carefully dried with either compressed air or cotton rolls.

The isolated hypoplastic defects on the labial surfaces of cuspids were categorized as : normal (0), defect (1), defect with caries (2), defect with restoration (3), carious lesion (4), restoration (5). The defects (1) were further categorized as: discolored (1a), pitted (1b), or dished-out (1c). Discolored defects (1a) included white, yellow, and brown opaque areas with an intact enamel surface. Pitted defects (1b) included single and multiple lesions. Dished-out defects (1c) were defined as roughly circular or oblong in shape and their size ranged from 1.0 to 5.0 mm in diameter with a flat or concave appearance. (see **Figure 1-4**)

The teeth were noted as maxillary or mandibular. Right and left were also indicated. The labial surface of each tooth was divided into cervical, incisal and middle thirds, and mesial, distal and middle thirds, to provide additional documentation about the location (see **Fig 5**).

The results of each examination were recorded on the child's Examination Sheet (**Fig 5**). All parents were interviewed in an effort to find an explanation for the defect. To standardize the interview, a health questionnaire in English or Spanish (**Table 1 and 2**) was used. A Spanish-English (bilingual) dental assistant aided in the interpreting

and recording of all questionnaire data.

The data were analyzed by descriptive statistics to find out the prevalence of the hypoplastic defects in this Hispanic population, and by X^2-test to find the relationship between the enamel defects and the perinatal occurrence.

Figure 1. Normal Mandibular Primary Cuspid and a Maxillary Primary Cuspid with Discolored Defect

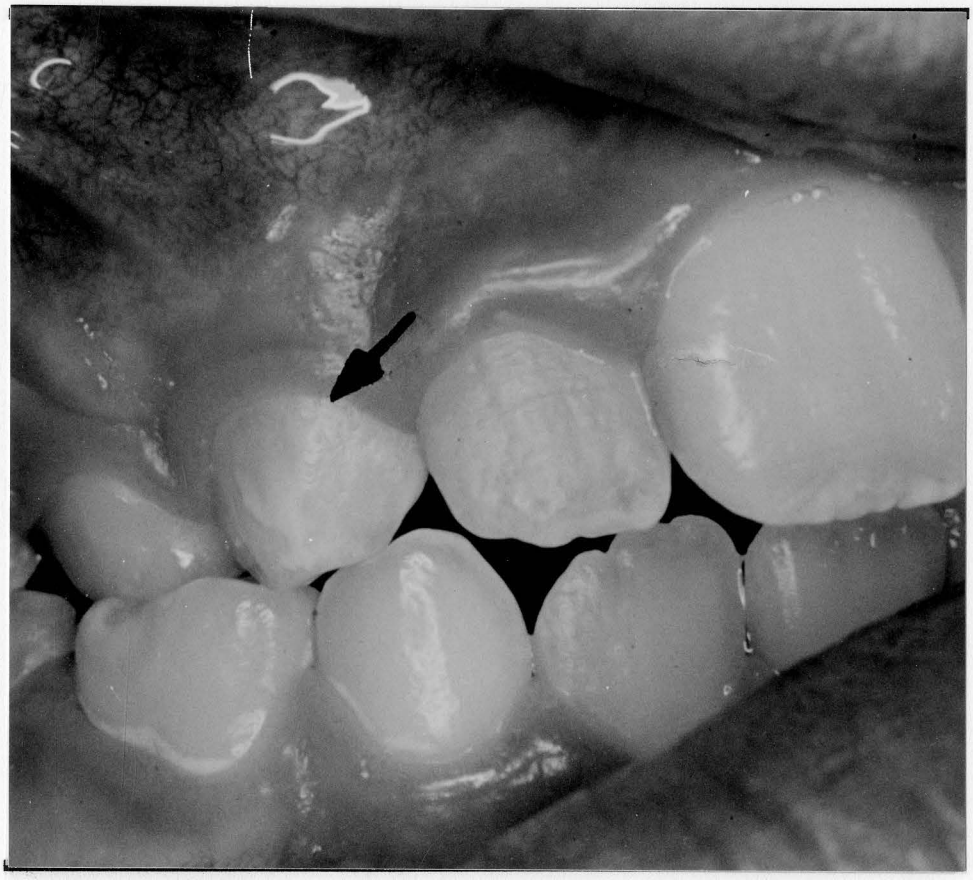


Figure 2. Normal Maxillary Primary Cuspid and a Mandibualr Primary Cuspid with a Solitary Dished-out Defect

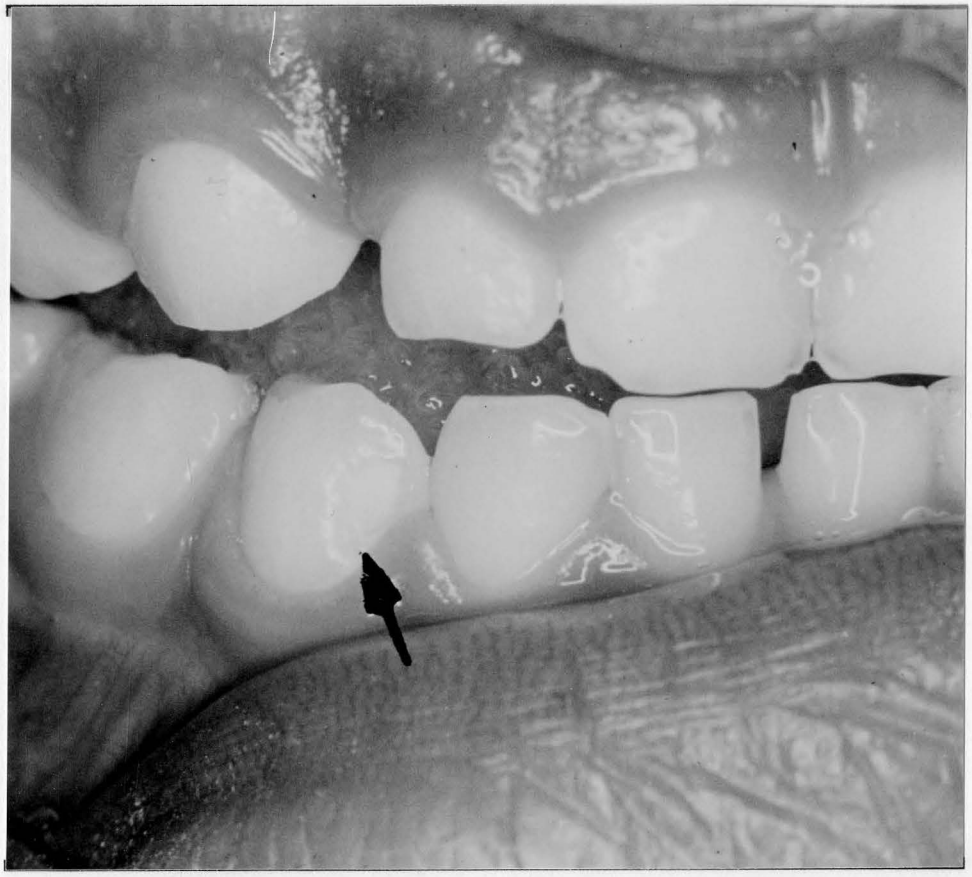


Figure 3. Normal Maxillary Primary Cuspid and a Mandibular Primary Cuspid with Multiple Dished-out Defects

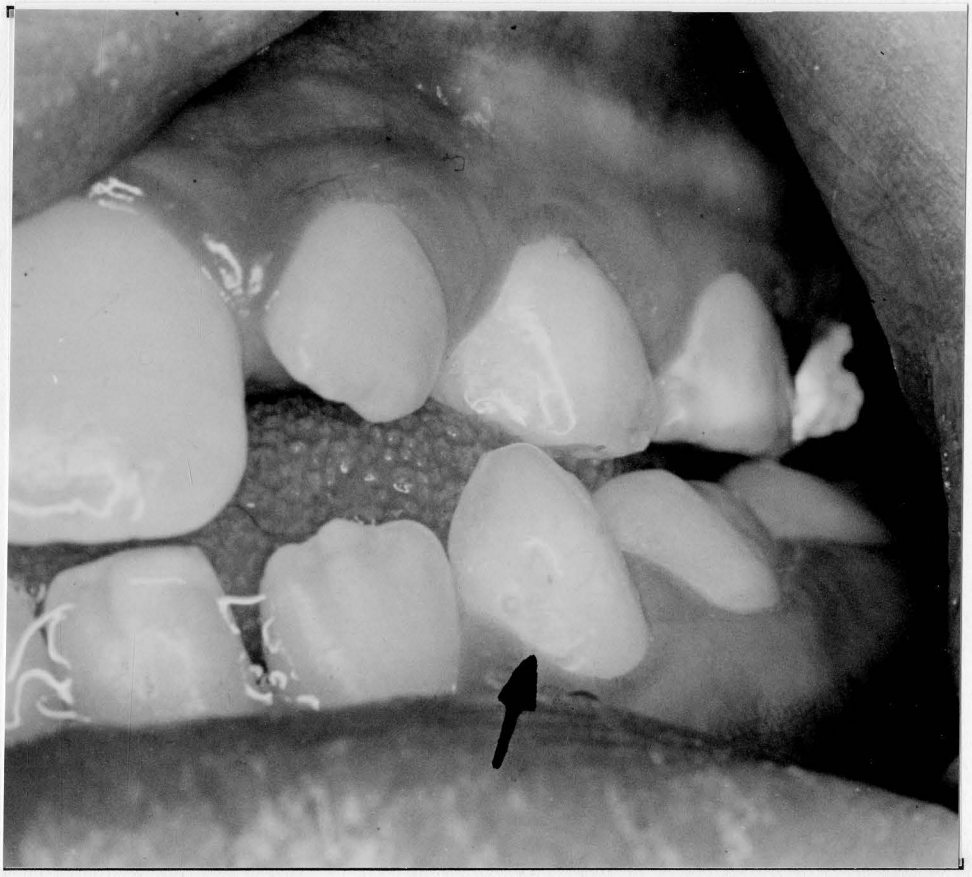


Figure 4. Normal Maxillary Primary Cuspid and a Mandibular Primary Cuspid with Defect with Caries

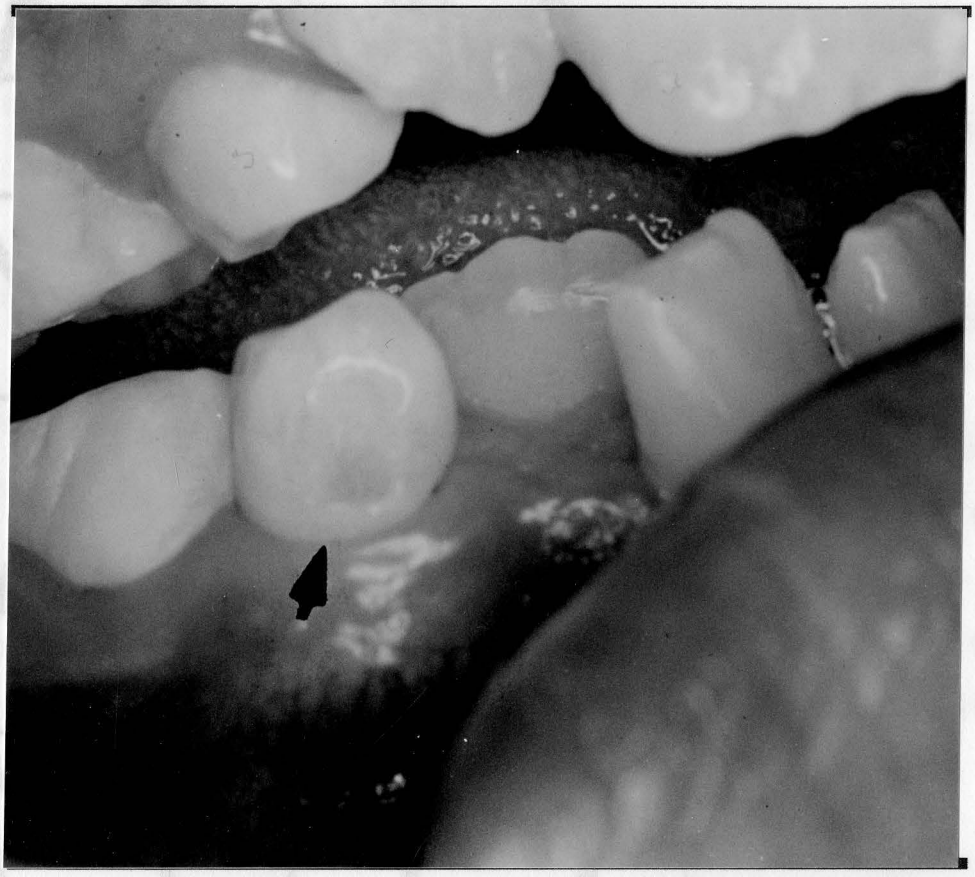
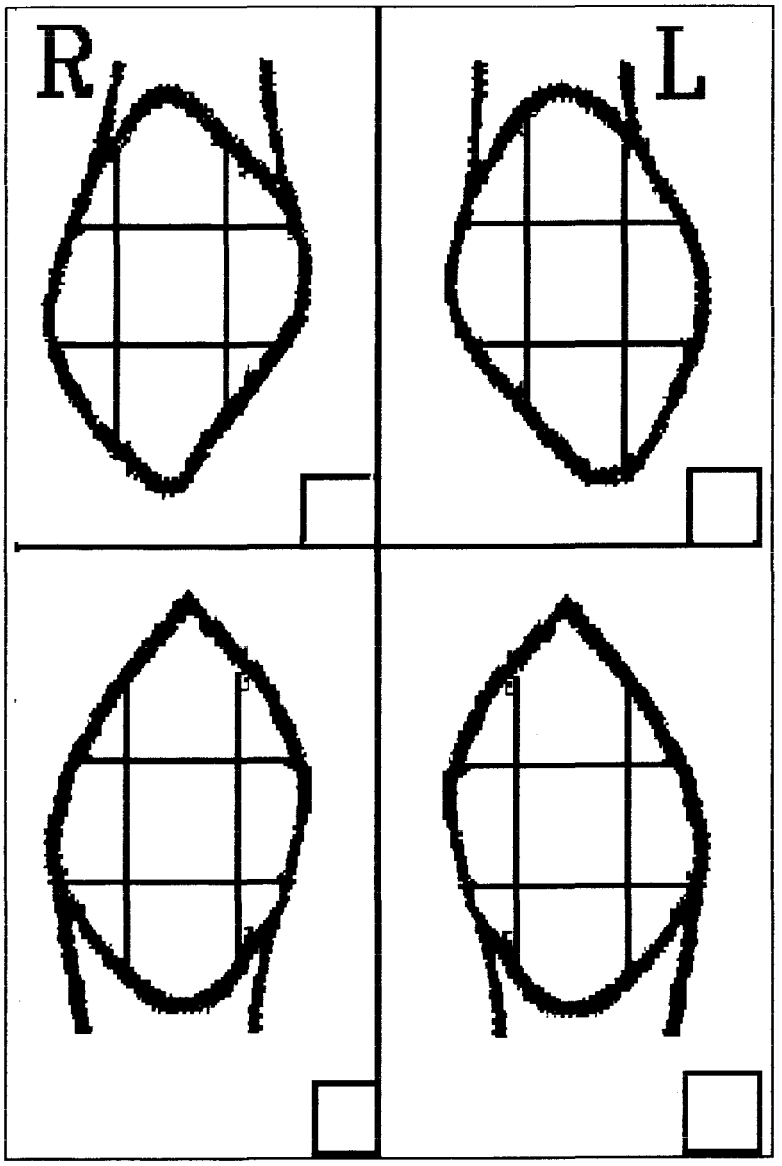


Figure 5. Examination Sheet



Present features:

0 = Normal

1 = Defects

1a = Discolored

1b = Pitted

1c = Dished-out

2 = Defects with caries

3 = Defects with Restoration

4 = Carious Lesion

5 = Restoration

Table 1. English Questionnaire

HEALTH QUESTIONNAIRE

Child's Name: _____ **Sex: Male** _____ **Female** _____
Date of Birth: _____ **Birthplace:** _____
Registration number in Infant Welfare Society _____

Please circle one

YES NO 1. Are both you and your spouse Hispanics?
YES NO 2. Are both parents of the child's mother Hispanics?
YES NO 3. Are both parents of the child's father Hispanics?
4. How old was the child's mother when she had this child? _____
YES NO 5. Did the mother have any health problems during this pregnancy that required medical attention? If yes, what was it? Please explain: _____
YES NO 6. Was your child born prematurely? If yes, how many weeks early? _____.
YES NO 7. Was your child born in a normal delivery? If not, please circle one: Forceps / C-section / Breech?
YES NO 8. Was your child breast fed? If yes, for how long? _____.
YES NO 9. Did your child have Chickenpox or Measles during the first year of life?
YES NO 10. During pregnancy and the first year after the child was born, did the family drink Chicago water? If not, please explain: _____.

Signature _____ **Relationship to child** _____

Table 2. Spanish Questionnaire

CUESTIONARIO DE SALUD

Nombre del paciente: _____ **Sexo: Hombre**___ **Mujer** ___
Fecha de Nacimiento: _____ **Lugar de Nacimiento** _____
Número de Registro en la Clinica de Infant Welfare Society _____

Por favor circule:

Sí No 1. ¿Son Ud. y su esposo/a hispanos?
Sí No 2. ¿Son los dos padres de la madré del niño/a hispanos?
Sí No 3. ¿Son los dos padres del papá del niño/a hispanos?
4. ¿Qué edad tenía la madré del paciente cuando nació el niño/a? _____.
Sí No 5. ¿La madré del paciente tuvo algún problema o complicación durante su embarazo que requirió atencion médica? Si hubo, ¿Cuál fue? Explique _____.
Sí No 6. ¿El nino/a nacio prematuro? Si fue prematuro, ¿Cuántas semanas? _____.
Sí No 7. ¿Su bebe nacio de parto normal? Si No, circule uno : Forceps/Cesarea/El niño nació de pie.
Sí No 8. ¿Se le dio pecho al bebé? ¿Por cuánto tiempo? _____.
Sí No 9. ¿El paciente tuvo sarampión o varicela durante su primer año de vida?
Sí No 10. ¿Durante el embarazo de la madre y el primer año de vida del paciente, la familia tomo agua de la ciudad de Chicago? Si la respuesta es No, Explique _____.

FIRMA _____ **Parentesco con el paciente** _____

CHAPTER IV

RESULTS

In this study, 83 Hispanic children were examined in the Infant Welfare Society of Chicago. Within this Hispanic population 59% of the children (49 affected/83 examined) had one or more of localized enamel hypoplastic defects on their primary cuspid teeth.

Of the 332 cuspids examined, 31.3% (104) manifested this localized defect. This was almost 2 (1.96) per affected child. (**Table 3**, Raw Data) These cuspid tooth defects included discolored (29 cuspids), pitted (6 cuspids), dished-out (54 cuspids), defect with caries (9 cuspids), defect with restoration (0 cuspids), carious lesion (1 cuspid), and restoration (5 cuspids).

Sex of the child was not related to the presence of the defects. There were 25 affected females of 42 examined and 24 affected males of 41 examined (**Table 4**).

The defects appeared more on the mandibular arch (36.1%, 60 affected/166 cuspids) than on the maxillary arch (26.5%, 44

affected/166 cuspids). Statistical analysis showed this to be a significant difference (**Table 5**, p=0.026).

The left side (33.7% = 56 affected/166 cuspids) showed significantly higher incidence of the defect than the right side (28.9% = 48 affected/166 cuspids) (**Table 6**, p=0.005). Mandibular left primary cuspids showed the highest incidence (**Table 7**, 32%).

In this study, no significant relationship was found between the presence of the defects and the following:

(1) Age of the mother at time of birth (**Table 8**),

(2) Maternal Complications during pregnancy (**Table 9**),

Eleven mothers reported complications. They included: anemia (6), hypertension (1), diabetics (2), bifid rib (1), and brain tumor (1).

(3) Prematurity (**Table 10**),

(4) Complications during Delivery (**Table 11**),

Thirteen children born by C-section. Nine of the children born by C-section presented with one or more defects.

(4) Breast Feeding (**Table 12**),

Time varied from 1 to 18 months in durations.

(5) Exanthematous Diseases (**Table 13**),

(6) Fluoridation (**Table 14**).

Table 3. Raw Data (Symbolic Designations)

NUM = number of patient
DEF = defect present per patient
1 = at least one cuspid present
0 = no defect showed on all cuspids
UR = upper right cuspid
UL = upper left cuspid
LR = lower right cuspid
LL = lower left cuspid
1 = defect present on this cuspid
0 = defect absent on this cuspid
AGE = age of this patient
SEX = sex of this patient
1 = male
0 = female
MOM = age of the mother when delivering the patient
COM = complication
1 = present
0 = absent
PRE = prematurity
1 = yes
0 = no
DEL = method of delivery
1 = normal
0 = complicated
BRE = breast feeding
1 = yes
0 = no
EXA = exanthematous diseases
1 = yes
0 = no
FLU = fluoridated water
1 = yes
0 = no
way = method of delivery,
1 = forceps
2 = C-section
3 = breech
b-d = duration of breast feeding
p-d = duration of prematurity

Table 3. Raw Data (I)

NUM	1	2	3	4	5	6	7	8	9	10	11	12	13	14	15
DEF	1	1	1	1	1	1	1	0	1	0	0	1	1	0	1
UR	0	1	1	1	1	0	0	0	0	0	0	1	0	0	0
UL	0	0	0	0	1	0	0	0	1	0	0	1	0	0	0
LR	0	0	0	0	0	1	0	0	1	0	0	0	1	0	1
LL	1	0	1	0	0	1	1	0	1	0	0	0	1	0	0
AGE	8	9	8	6	7	7	7	5	8	8	5	8	8	2	4
SEX	1	1	0	0	0	1	0	1	1	1	0	0	1	0	1
MOM	22	23	23	19	27	24	29	25	25	17	19	22	22	32	28
COM	0	1	0	0	0	0	0	0	0	0	0	0	1	1	1
PRE	0	0	0	0	0	0	0	0	0	0	0	0	0	0	1
DEL	1	1	1	0	1	1	0	1	1	1	1	1	1	0	1
BRE	1	0	0	0	1	1	1	0	1	1	0	0	1	0	1
EXA	0	0	0	1	0	0	0	0	0	1	0	0	1	0	0
FLU	0	1	1	1	0	1	1	1	1	1	1	1	1	1	1
way				3			2							2	
b-d	3M				8M	6M	8M		1M	7M			2M		3M
p-d															1W

Table 3. Raw Data (II)

NUM	16	17	18	19	20	21	22	23	24	25	26	27	28	29	30
DEF	0	1	1	1	1	0	1	1	0	0	1	1	0	1	0
UR	0	1	1	1	1	0	1	0	0	0	1	1	0	1	0
UL	0	1	1	1	1	0	1	1	0	0	1	1	0	0	0
LR	0	0	1	1	0	0	1	0	0	0	1	1	0	0	0
LL	0	1	1	1	1	0	1	1	0	0	0	1	0	1	0
AGE	3	6	8	8	5	8	9	6	5	4	3	5	3	7	6
SEX	1	0	0	0	0	0	0	0	0	0	1	1	1	0	1
MOM	26	25	26	17	21	32	29	32	34	35	30	17	24	29	18
COM	0	0	1	0	0	0	0	0	0	0	1	0	0	0	0
PRE	0	0	0	0	0	0	0	0	0	0	0	0	0	0	0
DEL	1	1	0	1	1	1	1	1	1	1	0	1	1	0	0
BRE	1	1	1	0	0	0	1	1	1	1	0	1	0	0	0
EXA	0	0	0	0	0	0	1	0	0	1	0	0	0	0	0
FLU	1	0	0	1	1	1	1	0	0	0	1	0	1	1	1
way			2								2			2	2
b-d	4M	12M	6M				3M	2M	6M	2M		3M			
p-d															

Table 3. Raw Data (III)

NUM	31	32	33	34	35	36	37	38	39	40	41	42	43	44	45
DEF	1	1	1	0	0	0	0	1	0	1	0	0	0	0	0
UR	0	0	0	0	0	0	0	1	0	0	0	0	0	0	0
UL	0	0	0	0	0	0	0	1	0	0	0	0	0	0	0
LR	1	1	0	0	0	0	0	1	0	1	0	0	0	0	0
LL	1	1	1	0	0	0	0	1	0	1	0	0	0	0	0
AGE	7	7	6	8	8	8	6	6	3	7	2	3	5	7	8
SEX	1	1	1	1	0	1	1	1	1	1	0	0	0	1	1
MOM	24	24	41	22	41	27	28	27	25	21	19	33	24	20	19
COM	0	0	0	1	1	0	0	0	0	0	0	1	0	0	0
PRE	0	0	0	0	1	0	0	0	0	0	0	1	0	1	0
DEL	0	1	1	0	0	1	1	1	1	1	1	1	1	1	1
BRE	0	1	1	0	1	1	0	0	1	1	1	0	1	1	1
EXA	1	0	1	0	0	0	0	0	0	0	0	0	1	0	0
FLU	1	1	1	1	1	0	1	1	1	1	1	0	1	0	0
way	2			2	2										
b-d		5M	3M		7M	6M			8M	18M	13M		4M	6M	12M
p-d					2W							6W			

Table 3. Raw Data (IV)

NUM	46	47	48	49	50	51	52	53	54	55	56	57	58	59	60
DEF	0	0	1	0	1	1	0	1	0	0	0	0	0	0	1
UR	0	0	0	0	0	0	0	1	0	0	0	0	0	0	0
UL	0	0	0	0	0	1	0	1	0	0	0	0	0	0	0
LR	0	0	1	0	1	1	0	0	0	0	0	0	0	0	1
LL	0	0	0	0	1	1	0	0	0	0	0	0	0	0	1
AGE	4	6	5	4	6	6	4	2	5	7	2	2	6	8	7
SEX	1	1	0	0	1	1	0	0	0	1	0	0	1	0	1
MOM	26	24	17	24	36	24	24	32	41	21	24	24	20	20	22
COM	0	0	0	0	0	0	0	0	0	0	0	0	0	0	0
PRE	0	0	0	0	0	0	0	0	0	0	0	0	1	0	0
DEL	1	1	1	1	1	1	1	1	1	1	1	1	1	1	1
BRE	0	1	1	1	1	1	0	0	1	1	1	1	1	1	1
EXA	0	0	0	0	0	1	0	1	0	0	0	0	0	0	0
FLU	1	0	1	0	1	1	1	1	0	0	1	1	1	1	1
way															
b-d			2M	12M	12M	6M			12M	12M	4M	4M	2M	12M	3M
p-d													4W		

Table 3. Raw Data (V)

NUM	61	62	63	64	65	66	67	68	69	70	71	72	73	74	75
DEF	1	1	1	1	1	1	1	1	1	1	1	0	1	1	1
UR	0	0	0	0	1	0	1	0	0	0	1	0	0	1	0
UL	0	0	0	1	0	1	1	0	1	0	1	0	0	1	0
LR	1	1	1	0	0	1	1	1	0	0	0	0	1	0	0
LL	1	1	0	0	0	1	1	1	0	1	1	0	1	0	1
AGE	4	4	3	4	8	4	7	4	8	6	4	9	5	5	6
SEX	1	1	0	0	0	1	1	0	0	1	1	0	0	0	1
MOM	26	26	27	18	28	22	22	19	24	24	28	38	28	33	19
COM	0	0	0	0	0	0	0	0	0	0	0	0	0	0	1
PRE	0	0	0	0	0	0	1	0	0	0	0	0	0	0	0
DEL	1	1	1	1	1	1	0	1	1	1	0	1	1	1	0
BRE	1	1	0	1	1	1	1	1	1	0	0	0	0	1	1
EXA	0	0	0	0	0	1	0	0	0	0	0	0	0	0	1
FLU	1	1	1	1	1	0	0	1	1	1	1	1	1	1	0
way							2				2				2
b-d	1M	1M		1M	11M	3M	3M	8M	2W					1M	3M
p-d															

Table 3. Raw Data (VI)

NUM	76	77	78	79	80	81	82	83
DEF	1	0	1	1	0	1	0	1
UR	0	0	1	1	0	0	0	0
UL	1	0	1	1	0	0	0	0
LR	1	0	1	0	0	0	0	1
LL	0	0	1	0	0	1	0	0
AGE	5	3	3	4	3	6	7	4
SEX	0	1	1	0	0	0	1	0
MOM	22	27	28	19	20	21	19	21
COM	0	0	1	0	0	0	0	0
PRE	0	0	1	0	0	0	0	0
DEL	1	1	0	1	1	1	1	1
BRE	1	1	1	1	1	0	0	1
EXA	0	0	0	0	0	0	0	0
FLU	0	1	1	1	1	1	1	1
way			2					
b-d	18M	2M	12M	8M	2M			3M
p-d			4W					

Table 4. Defects vs. Sex

	Unaffected	Affected	Total
Female	17	25	42
Male	17	24	41
Total	34	49	83

$p = 0.927$ NS $\quad X^2 = 0.01$

Table 5. Defects vs. Maxillary Arch and Mandibular Arch

		Maxillary Arch: Unaffected	Maxillary Arch: Affected	Total
Mand. Arch	Unaffected	84	22	106
	Affected	38	22	60
	Total	122	44	166

$p = 0.026$ Significant $X^2 = 4.98$

Table 6. Defects vs. Right Side and Left Sides

		Right Side: Unaffected	Right Side: Affected	Total
Left Side	Unaffected	86	24	110
	Affected	32	24	56
	Total	118	48	166

$p < 0.05$ Significant $X^2 = 7.99$

Table 7. Prevalence Of Affected Cuspids

Affected Cuspids	Female	Male	Total
R Maxillary	14	7	21(20%)
L Maxillary	14	9	23(22%)
L Mandibular	12	21	33(32%)
R Mandibular	9	18	27(26%)
Total	49	55	104(100%)

Table 8. Defects vs. Age Of Mother When She Had This Child

Mother's Age	Unaffected	Affected	Total
15-24	19	27	46
25-34	11	20	31
35-44	4	2	6
Total	34	49	83

$p = 0.363$ NS $\quad X^2 = 2.03$

Table 9. Defects vs. Complication During Maternal Pregnancy

	Unaffected	Affected	Total
No Complication	30	42	72
With Complication	4	7	11
Total	34	49	83

$p = 0.739$ NS $X^2 = 0.11$

Table 10. Defects vs. Prematurely Born

	Unaffected	Affected	Total
No Prematurity	30	46	76
Prematurity	4	3	7
Total	34	49	83

$p = 0.363$ NS $\quad X^2 = 0.83$

Table 11. Defects vs. Complicated Delivery

	Unaffected	Affected	Total
Normal	30	39	69
C-section or Breech	4	10	14
Total	34	49	83

$p = 0.301$ NS $\quad X^2 = 1.07$

Table 12. Defects vs. Breast Feeding

	Unaffected	Affected	Total
No Breast Feeding	13	16	29
Breast Feeding	21	33	54
Total	34	49	83

$p = 0.6$ NS $\quad X^2 = 0.28$

Table 13. Defects vs. Having Chickenpox Or Measles

	Unaffected	Affected	Total
Absent	31	40	71
Present	3	9	12
Total	34	49	83

$p = 0.224$ NS $X^2 = 1.48$

Table 14. Defects vs. Having Benefit From Fluoridated Water

	Unaffected	Affected	Total
Non-fluoridated	10	10	20
Fluoridated	24	39	63
Total	34	49	83

$p = 0.346$ NS $\quad X^2 = 0.89$

CHAPTER V

DISCUSSION

The uniqueness of the "spots" of primary cuspid enamel hypoplasia should make them relatively easy to identify, study, and compare findings of different investigators. However, definition(s), background of examiners and examination conditions appear to be variables that bias findings. Hence for this study, the writer must clarify the following:

(1) Definition of isolated enamel hypoplasia of primary cuspids:

The characteristics describing this unique defect of primary enamel and used in its definition for the purpose of this study are based on the earliest description by Jorgenson[46, 58]. Clinical observations by the writer and by Noonan[53] (the primary advisor in this research) completed the definition (see Page 1, Introduction). This definition covers

the morphology, the description, the location, and the distribution. The parameters of the definition are carefully specified to eliminate any confusion as to what the writer determined to be primary cuspid enamel hypoplasia.

(2) Defects with carious lesions and restorations:

Isolated carious defects and restored defects were included in the study. It was assumed that the isolated areas of caries and restorations in the mesiogingival section of the labial surfaces of these cuspids originated from defects. Most of them clearly showed defects as well. This is a confusing point in the literature as some researchers included the restorations, others excluded them. This would affect number of teeth and number of children as well that is the entire population sample.

(3) Examination Method:

Examination procedures were standardized in this study --- the teeth were cleaned, dried thoroughly, and then examined by using the explorer and mouth mirror under direct dental light. Earlier studies were conducted under a variety of conditions and often were not clear in this point. However, it is the writer's experience that the defect is often covered either by plaque or materia alba and that accurate observation

is hindered by the presence of moisture on the tooth surface and by poor lighting.

(4) All Four Primary Cuspids Present and Fully Erupted:

The defects appear most frequently on the cervical third of the clinical crown, hence, it is important to examine the fully erupt primary cuspid. Also, in order to statistically compare distribution and prevalence of the defects, only samples with all four primary cuspids present should be included in the research. The children in Badger's[49] sample varied in age from one year six months to eleven years six months --- an age range from incompleted eruption through exfoliation of cuspids. This may be an additional factor in explaining the differences among studies.

The prevalence of the defect (59%) in this study was the highest among the reported prevalences reviewed by the writer (**Table XV**). It was similar with studies conducted by Jorgensen[46, 58], Noonan[53], Badger[49], Brown and Smith[50], and Duncan[51] --- falling in their range of 36% to 59%.

It is to be noted that:

(1) Jorgensen[46, 58], as early as 1956, reported hypoplastic defects in primary canine teeth of modern (21%) and medieval (28%) Danes, finding striking similarities between the two. He did not report the number of children in his study.

(2) In 1965, Noonan found that the prevalence of the defect in

the teeth of 311 Caucasian children (23.2%) in a non-fluoridated community and later in 1969, 200 Black children (33.4%) in a fluoridated community, were not only similar to each other but were similar to the two groups of Danish children.

(3) Badger, 1985, reported similar prevalence (22%) of defects on the primary cuspids among a group of 55 children on a U.S. Army base.

Only Skinner[48,21] greatly differed. In 1986, he reported that less than 1% of a group of 2,380 Canadian children had one or more defects, and later in 1989, a prevalence of only 2.4% among another group (1,350) of Canadian children. The findings are extremely different and not truly explainable. They may be related to background of investigators (anthropologist vs. dentist) and of examiners (dental assistants vs. dentist), definition of defects, and conditions for examination.

The results in this study showed an equal distribution between males and females. However, more defects were found on mandibular cuspids (36.1%) than maxillary cuspids (26.5%). Both findings were consistent with the findings of other researchers[46, 58,48,49,50,51,53]. The greater number of mandibular defects was statistically significant and suggests either a circulation factor or a postural relationship that may dispose to local trauma. The finding that girls vs. boys display the same prevalence suggests a wider range of ages when the defect

may be initiated since the developmental ages of primary cuspids for boys vs. girls varies.

The higher incidence on the left side was contrary to some studies, i.e., Brown and Smith[50] showed right mandibular cuspids had the highest incidence, and Skinner[21] reported that the right side of the upper jaw was affected eight times and the left side only once, and similar to Jorgenson's samples of Medieval Danes which showed a highest prevalence of left lower cuspids. This difference between presence of defects on the right vs. the left side suggests local trauma as a factor. An evaluation of dominance (right vs. left hand) may be important to exploring this factor.

This study seems to confirm that racial differences and socioeconomic status are not factors in occurrences and distribution of the defects. Racial difference was reported by Duncan[51] (Black more than White) and Skinner[48,21] (Indo-Asia and Black more than White). However, the studies conducted by Noonan[53] and the present study, compared 311 White, 200 Black, and 83 Hispanic children under similar examination conditions, defect definition and criteria. These results were shown to have a non-significant difference by X^2 analysis (**Table 16**). Jorgenson's Danish population was also similar.

No differences among children with and without the defects were observed in terms of reported perinatal occurrences. In each perinatal factor, the Chi-square test showed no significant differences of prevalence of the

defects. These factors (see **Table 1**) included age of the mother at time of birth, pregnancy complications, prematurity, delivery pressure and complication, breast feeding, exanthematous diseases, and availability of fluoride. This finding was consistent with the results of the health questionnaires in studies conducted by Noonan[53] and Skinner[21].

It must be noted that the designations of fluoridated water vs. nonfluoridated water for the children were based on knowledge from the questionnaires from this Hispanic population. It was assumed that the children who were born and raised in Chicago had received the benefit of systemic fluorides and children born and raised in Mexico until at least one year of age had not benefitted from a fluoridated water supply.

The hypothesis that pressure from the birth canal compressed the labially-positioned primary cuspid through thin labial bone and caused this localized defect, may be ruled out by the presence of defects on the C-section born children (**Table 11**). The prevalence and appearance among C-section born children was similar to that of the over all population sample.

The hypothesis that localized heat generated from breast feeding transverse thin labial bone and caused the defects was also ruled out by the presence of defect on the bottle-fed children (**Table 12**). The prevalence and appearance among breast-fed children was also similar to that of the over all

population.

Prematurity often causes generalized or localized enamel hypoplasia on anterior primary incisors. It is usually circumferential. Prematurity did not cause a significant higher incidence of this localized primary cuspid defect in this population (**Table 10**).

It was obvious that the etiology of this localized hypoplastic enamel defect on the primary cuspids remains unknown. Skinner[21] suggested that the disturbances in calcium balance within the labial bone combined with the direct injury to ameloblasts are capable of eliciting enamel defects in primary teeth. He also hypothesized that this defect is not produced until after birth --- at an average age of about 6 months. He said that at 5 months there is a pronounced mouthing and chewing of bottles, toys, and anything a baby can hold. Immature and uncoordinated movements could result in a "bumping" of the face and gum tissues. This "bumping" in the susceptible infant could be a localized trauma to ameloblasts in their secretory phase, and result in a dysplasia of enamel similar to the enamel hypoplasia produced by Suckling on experimental animals.

This hypothesis that the defects may be caused by immature motor movements of objects held by infant seems reasonable. Differences between right side and left side, and maxillary vs. mandibulary arches, and dominance of right vs. left hand may lead to better analysis of this hypothesis.

Table 15. Comparison Of The Mean Prevalence

Noonan, 1965	Caucasian (Speedway)	49.5%
Noonan, 1969	Black (Louisville)	58.5%
Badger, 1985	------	45%
Brown & Smith, 1986	------	35.7%
Skinner, 1986	White (Canada)	< 1%
	Indo-Asian or Black	38.5%
Nation, 1987	------	18%
Duncan, 1988	Black (Head Start)	37%
Skinner, 1989	------ (Canada)	2.4%
Wang, 1990	Hispanics (Chicago)	59%

Table 16. Defects vs. Racial Difference

Race	Unaffected	Affected	Total
White	157	154	311
Black	83	117	200
Hispanic	34	49	83
Total	274	320	594

$p > 0.05$ NS $X^2 = 4.99$

CHAPTER VI

SUMMARY

The purpose of this study was to determine the prevalence of localized enamel hypoplastic spots on the labial surfaces of primary cuspids in a Hispanic population, and their relationship to certain perinatal occurrences.

Eighty-three Hispanic children with all primary cuspids present were selected from an urban pediatrics program. Heritage and the health history was determined by the questionnaire. After prophylaxis, examination was performed with mouth mirror, explorer and dental light.

The results were similar to a Danish population (Jorgenson, 1956), a black population (Noonan, 1969), and Caucasian populations (Noonan, 1965; Badger, 1985). Defects affected one half of the children but appeared in only one of four cuspids. It occured more in the mandible than maxilla, and with similar occurrence in boys and girls. Birth pressures and other evaluated perinatal occurrences showed no direct correlation to the defects.

CHAPTER VII

CONCLUSION

Definition of characteristic localized enamel defects of primary cuspids is key to standardizing investigation of this entity. Hence, in this study, by definition:

Localized primary cuspid enamel hypoplasia is:

(1) non-circumferential,

(2) located only on the labial surfaces of primary cuspids,

(3) predominately found on the mesiogingival quadrant,

(4) small, usually 1-2 mm in diameter, rarely up to 5 mm,

(5) flat to concave (bottom may be smooth to pitted),

(6) roughly circular or oblong,

(7) usually solitary but may be multiple on an individual tooth surface,

(8) white, yellow, or brown, and may be opaque,

(9) barely visible to well-defined,

(10) found in both symmetrical and non-symmetrical patterns even on any individual child,

(11) found in enamel tissue formed during the postnatal period.

Procedures in the identification of localized primary cuspid enamel hypoplasia should be standardized in order to compare the results among different studies. This should include cleaning, drying, and examination of the teeth under direct dental light while using an explorer by a single trained examiner. Findings within this Hispanic population

Within an inner city Hispanic population:

(1) The defects occur equally in boys and girls.

(2) The prevalence of the defect is higher on the mandibular cuspids than the maxillary cuspids.

(3) There are no racial or socioeconomic differences.

(4) Age of the mother at time of birth of the child, pregnancy complications, prematurity, delivery pressure and complications, breast feeding, exanthematous diseases, and availability of fluoride --- all do not correlate with or suggest a cause of this defect.

Enamel defects of the primary teeth continue to have the potential for identification of children who have undergone certain systemic insults during prenatal and early postnatal life. The role of hypocalcemia of expectant mothers and of infants and the occurrence of localized trauma due to uncoordinated motor movement during the first year of life may be etiologies of this localized hypoplastic enamel defect on the labial surface of primary cuspids.

BIBLIOGRAPHY

1. Suckling, G.W.: Developmental defects of enamel - historical and present-day perspectives of their pathogenesis. Adv Dent Res, 3:87-94, September, 1989.

2. Diamond, M. and Weinmann, J.P.: The enamel of human teeth. New York: Columbia University Press, 1940.

3. Deakins, M.: Changes in the ash, water, and organic content of pig enamel during calcification. J Dent Res, 21:429-435, 1942.

4. Stewart, R.E. and Prescott, G.H.: Oral Facial Genetics. St. Louis: The C.V. Mosby Company, 1976, 151-226.

5. Suckling, G.W.: Mineralization of the enamel of bovine permanent central incisor teeth using microhardness and histological techniques. Calcif Tissue Int, 28:121-129, 1979.

6. Suckling, G.W.: Defects of enamel in sheep resulting from trauma during tooth development. J Dent Res, 59:1541-1548, 1980.

7. Robinson, C., Fuchs, P., Deutsch, D., and Weatherfell, J.A.: Four chemically distinct stages in developing enamel from bovine incisor teeth. Caries Res, 12:1-11, 1978.

8. Robinson, C., Briggs, H.D., Atkinson, P.J., and Weatherell, J.A.: Chemical changes during formation and maturation of human deciduous enamel. Arch Oral Biol, 26:1027-1033, 1981.

9. Deutsch, D. and Pe'er, E.: Development of enamel in human fetal teeth. J Dent Res, 61:1543-1551, 1982.

10. Suga, S.: Progressive mineralization pattern of developing enamel during the maturation stage. J Dent Res, 61:1532-1542, 1982.

11. Suga, S.: Enamel hypomineralization viewed from the pattern of progressive mineralization of human and monkey developing enamel. Adv Dent Res, 3:188-198, September, 1989.

12. FDI Technical Report No. 15: An epidemiological index of developmental defects of dental enamel (DDE index). Int Dent J, 32:159-167, June, 1982.

13. Clarkson J.: Review of terminology, classifications, and indices of developmental defects of enamel. Adv Dent Res, 3:104-109, September, 1989.

14. Stewart, R.E., Witkop, C.J., and Bixler, D.: Pediatric dentistry. St. Louis: The C. V. Mosby Company, 1982, 87-117.

15. Cook, D.C.: Hereditary enamel hypoplasia in a prehistoric Indian child. J Dent Res, 9:1522, 1980.

16. Goodman, A.H.: Dental enamel hypoplasia in prehistoric populations. Adv Dent Res, 3:265-271, September, 1989.

17. Nikiforuk, G. and Fraser, D.: The etiology of enamel hypoplasia: a unifying concept. J Pediatr, 98:888-893, 1981.

18. Moylan, F.M.B., Seldin, E.B., Shannon, D.C., and Todres, I.D.: Defective primary dentition in survivors of neonatal mechanical ventilation. J Pediatr, 96:106-108, 1980.

19. Shafer, W.G., Hine, M.K., and Levy, B.M.: A Textbook of Oral Pathology. Philadelphia: WB Saunders Company, 1974, 53-58.

20. McDonald, R.E. and Avery, D.R.: Dentistry for the Child and Adolescent. St. Louis: The C. V. Mosby Company, 1987, 64, 127-137.

21. Skinner, M.F. and Hung, J.T.W.: Social and biological correlates of localized enamel hypoplasia of the human deciduous canine tooth. Am J Phy Anthrop, 79:159-175,1989.

22. Fraser, D. and Nikiforuk, G.: The etiology of enamel hypoplasia in children --- a unifying concept. Int Asso Dent Child, 13:1-11, January, 1982.

23. Spouge, J.D.: Oral Pathology. St.Louis: The C.V. Mosby Company, 1973, 145-165.

24. Small, B.W. and Murray, J.J.: Enamel opacities: prevalence, classifications and etiological considerations. J Dent Res, 6:33-42, 1978.

25. Pindborg, J.J.: Etiology of developmental enamel defects not related to fluorosis. Int Dent J, 32:123-134, 1982.

26. Bhat, M. and Nelson, K.B.: Developmental enamel defects in primary teeth in children with cerebral palsy, mental retardation, or hearing defects: a review. Adv Dent Res, 3:132-142, September, 1989.

27. Seow, W.K., Brown, J.P., Tudehope, D.I., and O'Callaghan, M.: Developmental defects in the primary dentition of low birth-weight infants: adverse effects of laryngoscopy and prolonged endotracheal intubation. Pediatr Dent, 6:28-31, January, 1984.

28. Angelos, G.M., Smith, D.R., Jorgenson, R., and Sweeney, E.A.: Oral complications associated with neonatal oral tracheal intubation: a critical review. Pediatr Dent, 11:133-140, June, 1989.

29. Kopra, D.E., Creighton, P.R., Buckwald, S., Kopra, L.B., and Carter, J.M.: The oral effects of neonatal intubation. J Dent Res, 67:165, 1988.

30. Russell, A.L.: The differential diagnosis of fluoride and non-fluoride enamel opacities. Public Health Dent, 21:143-146, 1961.

31. Thylstrup, A.: Distribution of dental fluorosis in the primary dentition. Community Dent Oral Epidemiol, 6:329-337, 1978.

32. Thylstrup, A., Fejerslov, O., and Mosha, H.J.: A polarized light and microradiographic study of enamel in human primary teeth from a high fluoride area. Arch Oral Biol, 23:373-380, 1978.

33. Larsen, M.J., Richards, A., and Fejerskov, O.: Development of dental fluorosis according to age at start of fluoride administration. Caries Res, 19:519-527, 1985.

34. Horowitz, H.S.: Fluoride and enamel defects. Adv Dent Res, 3:143-146, September, 1989.

35. Wallman, I.S. and Hilton, R.B.: Teeth pigmented by tetracycline. Lancet, 1:827-829, 1962.

36. Seow, W.K., Humphrys, C, and Tudehope, D.I.: Increased prevalence of developmental dental defects in low birth-weight, prematurely born children: a controlled study. Pediatr Dent, 9:221-225, September, 1987.

37. Seow, W.K., Brown, J.P., Tudehope, D.I., and O'Callaghan, M.: Dental defects in the deciduous dentition of premature infants with low birth weight and neonatal rickets. Pediatr Dent, 6:88-92, June, 1984.

38. Funakoshi, Y., Kushida, Y., and Hieda, T.: Dental observations of low birth weight infants. Pediatr Dent, 3:21-25, January, 1981.

39. Johnsen, D., Krejci, C., Hack, M., and Fanaroff, A.: Distribution of enamel defects and the association with respiratory distress in very low birth weight infants. J Dent Res, 63:59-64, 1984.

40. Tsang, R.C. and Oh, W.: Neonatal hypocalcemia in low birth weight infants. Pediatr, 82:423-425, 1973.

41. Tsang, R.C., Light, I.J., Sutherland, J.M., and kleinman, L.I.: Possible pathogenic factors in neonatal hypocalcemia of prematurity. J Pediatr, 82:423-429, 1973.

42. Sweeney, E.A., Carrera, J., Urrutia, J., and Mata, L.: Factors associated with linear hypoplasia of human incisors. J Dent Res, 48:1275-1279, 1969.

43. Sweeney, E.A., Saffir, A.J., and Leon, D.C.: Linear hypoplasia of deciduous incisor teeth in malnourished children. Am J Clin Nutr, 24:29-31, 1971.

44. Infante, P.F. and Gillespie, G.M.: An epidemiologic study of linear enamel hypoplasia of deciduous anterior teeth in Guatemalan children. Arch Oral Biol, 19:1055-1061, 1974.

45. Hall, R.K.: The prevalence of developmental defects of tooth enamel (DDE) in a pediatric hospital department of dentistry population (part I). Adv Dent Res, 3:114-119, September, 1989.

46. Jorgenson, K.D.: The deciduous dentition. A descriptive and comparative anatomical study. Acta Odont Scand, 14 (Suppl. 20):165-176, 1956.

47. Skinner, M.F.: An enigmatic hypoplastic defect of the deciduous canine. Am J Phys Anthropol, 69:59-69, January, 1986.

48. Skinner, M.F. and Hung, J.T.W.: Localized enamel hypoplasia of the primary canine. J Dent Child, 53:197-200, May-June, 1986.

49. Badger, G.R.: Incidence of enamel hypoplasia in primary canines. J Dent Child, 52:57-58, January-February, 1985.

50. Brown, J.D. and Smith, C.E.: Facial surface hypoplasia in primary cuspids. J Ind Dent Assoc, 65:13-14, July-August, 1986.

51. Silberman, S.L., Duncan, W.K., Trubman, A., and Meydrech, E.F.: Primary canine hypoplasia in Head Start children. J Public Health Dent, 49:15-18, Winter, 1989.

52. Nation W.A., Matsson, L., and Peterson, J.E.: Developmental enamel defects of the primary dentition in a group of Californian children. J Dent Child, 54:330-334, September-October, 1987.

53. Noonan, G.R.: A study of prevalence of isolated hypoplastic enamel spots on the labial surface of primary cuspids. unpublished.

54. Duncan, W.K., Silberman, S.L., and Trubman, A.: Labial hypoplasia of primary canines in black Head Start children. J Dent Child, 55:423-426, November-December, 1988.

55. Johnsen, D.C., Schultz, D.W., Schubot, D.B., and Easley, M.: Caries patterns in Head Start children in a fluoridated community. J Public Health Dent, 44:61-66, 1984.

56. Johnsen, D.C., Bhat, M., Kim, M.T., and et al: Caries levels and patterns in Head Start children in fluoridated and non-fluoridated urban, and non-urban sites in Ohio, USA. Community Dent Oral Epidemiol, 14:206-210, 1986.

57. Logan, W.H.G. and Kronfeld, R.: Development of the human jaws and surrounding structures from birth to the age of fifteen years. J Am Dent Assoc, 20:379-427, March, 1933.

58. Lunt, R.C. and Law, D.B.: A review of the chronology of calcification of deciduous teeth. J Am Dent Assoc, 89:599-606, May, 1974.

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