

THE RELATIONSHIP BETWEEN BIRTH WEIGHT AND ENAMEL DEFECT
PROBABILITY IN CHILDREN WITH PRENATAL MALNUTRITION

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CHAPTER 1

INTRODUCTION

1.1 Background

The growth and development of the teeth is a part of the whole body growth, and it might be affected by the interaction of genetic and environmental factors. The growth and development of the teeth is a complex and long lasting process, and it is a sensitive period against intrinsic as well as extrinsic factors of disturbances.^{1,2} The disturbances might happen in various phase/stage of teeth development with various intensity and period. This might result as the defect of teeth development i.e. enamel or dentin defects according to the disturbed tissues. Enamel defect means incompleted enamel that was caused by systemic environmental factors during prenatal or postnatal period. The critical growth and development of deciduous teeth occurs in perinatal period, while the critical phase of permanent teeth is in the postnatal period.^{2,4} Enamel defects include enamel hypoplasia and hypocalcification. Hypoplasia is caused by the disturbance of enamel matrix, while hypocalcification is caused by the disturbance disturbance of mineralization, and enamel maturation. According to ADA (Australian Dental Association), there are several factors that are related to the defects such as maternal factors, drugs during pregnancy, premature low birth weight, infection, malnutrition, and trauma.⁵ Prenatal factors that might cause defect of the teeth development are mostly maternal such as severe infection during pregnancy, malnutrition of the mother, mother's metabolic disorders, besides child's factor such as low birth weight.^{2,3,6-8}

Systemic condition such as Intrauterine Growth Retardation (IUGR) means the disturbances that occur in prenatal period that might cause intra uterine malnutrition of the fetus that might result as baby with prenatal malnutrition or might be call as Small For Gestational Age/ SGA, i.e. birth weight of lower than the normal -2 SD.⁹ It might result as a disruption of the development that cause defect of the organ development especially deciduous teeth. A good growth of fetus shows the mean weight gain in each phase/stage according to gestational age. Birth weight and gestational age indicate the growth of fetus; SGA neonates have birth weight of -2 SD beyond the normals (<2500 g).

The aim of this study was to find out the relationship between birth weight and enamel defect probability in children with prenatal malnutrition.

CHAPTER 2

1 Enamel Defect

Teeth development begins as early 4-5 weeks intrauterine, and in 2 months gestation deciduous dentition begins to develop. Prenatal period is the critical stage for any disturbances. Teeth are formed by mineralization of protein matrix and the process begins at about 4 months gestation and completed after birth. Enamel is hard tissue in the body that is not remodeled, changes in its structure could be happened in developing period. Enamel formation or Amelogenesis is very sensitive to any disturbances. Changes in its structure become a defect, it could be happened both in deciduous and permanent dentition. Genetic and environmental factor play the role in this condition. Environmental factor Could cause the defect, that are; prenatal infection, nutritional disturbances, systemic illness. Prematurity and low birth weight infant have been associated with defect in both permanent and deciduous teeth^(1,10,11,12)

Enamel defect means incompleting enamel, could be manifest as enamel hypoplasia and enamel hypocalcification, depend on disturbances and stage of growth and development of the teeth.

There are more 120 Risk factors have been linked to enamel defects, the most common are; Severely nutritional deficiency in pregnancy mother, Prematurity, Birth difficulties especially with hypoxia and disorders of the blood, trauma, and certain medications which given in pregnancy. For deciduous teeth enamel defects may result from disturbances in prenatal period, and for permanent teeth enamel defects may result from disturbances in postnatal period^(5,10-2)

Several nutrients are important for amelogenesis and osteogenesis; calcium, Phosphorus, Magnesium Fluoride, and some vitamins such, vitamin A, C, D, K. Deficiency in calcium can disturb amelogenesis and osteogenesis, 73 % children with calcium deficiency show enamel hypoplasia (14). Deficiency in vitamin C can cause disturbances in amelogenesis because vitamin C is important for collagen building in matrix formed.¹⁴

2.2 Clinical manifestation

Enamel Hypoplasia in deficiencies in enamel thickness resulting from disturbances in secretory phases of amelogenesis. The manifestation could be mild to severe, mild hypoplasia shows pit or groove on teeth surface, severe hypoplasia shows thin enamel or no enamel, and teeth looks small. This condition could easily become caries due to plaque attachment.

Enamel Hypocalcification is deficiencies in mineralization during mineralization phase, there are opaque, white, or yellow area of enamel on smooth surfacace.^{2,5,8}

Clinical manifestation due to the cause of disrturbances. Systemic disturbances show that the defect is generally, bilaterally. Local disturbances such as used of laryngoscope in managing baby's hypoxia, birth trauma show that the defect is locally, unilaterally and only 1 or more teeth affected.



Figure 2.1. Mild Enamel Hypoplasia¹²

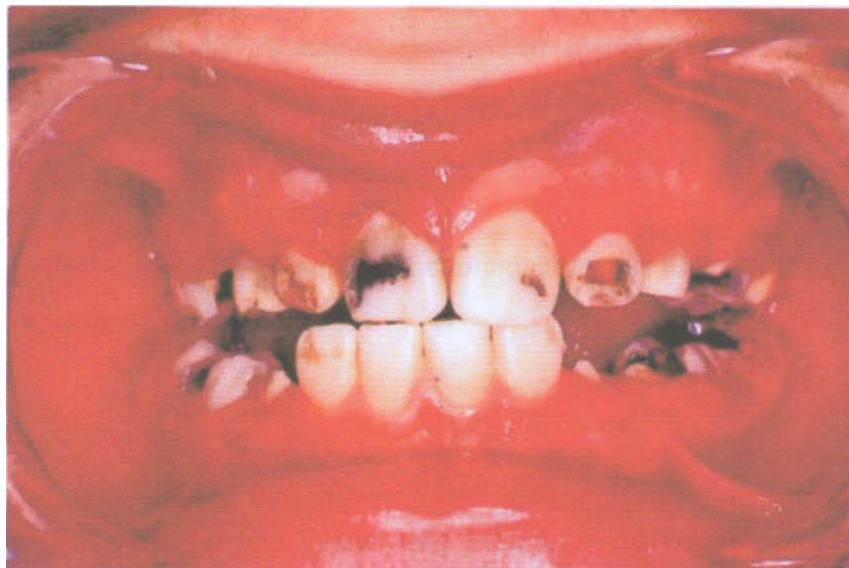


Figure 2.2. Moderate Enamel Hypoplasia³



Figure 2.3. Enamel Hypocalcification

2.3 Mechanism of Enamel Defect

Amelogenesis contains of matrix secretion and maturation of the matrix. Ameloblast is very sensitive to any disturbances resulting from genetic and environmental factor. Disturbances in secretion phase can disturb elongation of enamel crystal, it will reduction protein secretion resulting insufficiency of crystal elongation. This condition resulting reduction of the matrix cause thin enamel formed, part of enamel formed, or no enamel formed. This condition call enamel Hypoplasia.

If there is malnutrition during amelogenesis, there is reduction of ameloblast and resulting reduction of matrix forming and hypoplasia formed. Maturation of the enamel shown by reduction of water and calcium deposition. If there are nutritional/calcium deficiency, may resulting hypocalcification.

The size of defect depend on, instensity of etiological factor, time of disturbances, and period during crown formation.¹⁰⁻³

2.4 Prenatal Malnutrition

Growth and development of the baby is consist of 3 trimester, hyperplasia period (0-16 week intra uterin), hyperplasia and hypertrophy period (16-32 week) and hypertrophy period (32-born) (Fanarof, 12). In the first trimester there in increasing in the amount of the cell than cell size. In second trimester there is increasing in amount and size of the cell and in the third trimester there is increasing in fatt. Baby's growth shown by increasing of weight,

average 5 gram/days, and 30-35 gram/day in 34 week gestation. In the third trimester there is nutrition and hormonal influence rather than genetic.^{9,15,16.}

The etiology of prenatal mal nutrition divided on 3 groups; maternal factor, foetus factor, and placenta. Maternal factor such as; maternal TORCH infection, diabetes, hypertension preelampsia, maternal malnutrition, maternal infections in long period, age of the mother more 35 years, all that condition might be caused prenatal malnutrition. Foetus factor such as chromosom anomaly, syndromes also could be caused prenatal malnutrition. All that condition resulting failure of the placenta to transfer nutrition to the foetus, so prenatal malnutrition happened. Placenta's factor such, anomaly of the placenta. Placental infark and Placentitis could be caused prenatal malnutrition.^{9,15,16,18}

Birth weight show fetal growth rate, and the baby with mal nutrition shows birth weight lower or, much lower than normal commonly below 2500 gram. According to Fanaroff baby with malnutrition is shown that their birth weight is below than 2 SD normal birth weight. It could be Low birthweight /LBW (below 2500 gram), very low birth weight /VLBW (1500-2000 gram) and Very-very low birth weight /VVLBW (below 1500 gram). Birth weight could be as indicator of the condition of baby's nutrition, and also indicate if there is deviation of baby's growth.^{9,15-6}

The babys with this condition *needs specific management* due to their complication. The VVLBW is needed good peinatology management to make this babys survive without sequalae. Nowadays there are good management of perinatology, so, LBW, VLBW, VVLBW babys can live survive .

CHAPTER 3

3.1 Subject and Method

Subject were 150 children born with prenatal malnutrition aged 4-48 months and 300 normal children as control. We had complete data of mother's pregnancy (mother's age at birth, disease during pregnancy, and smoking habits) and children's birth including birth weight, gestational age, birth length, head circumference, hypoxia, and gender.

This was a clinical epidemiology study. Enamel defects were examined using DDE scoring modification and FDI index and determinate; mild defect score <12 , severe defect; >12 ^b and Interviews were done to collect data on birth weight and length, head circumference, gestational age, gender, and hypoxia in neonates. Enamel defects were recorded three times with 2 months intervals. We recorded the defects of deciduous teeth enamel and confounding variable factors of the child such as gender/sex, gestational age, birth weight and length, head circumference, and hypoxia. All data were analysed using multivariate analysis.

3.2 Operational Definition

1. Children with prenatal malnutrition means a neonate with birth weight beyond - 2SD of intrauterine growth curve. Birth weight usually below 2500 gram, some children are 2600 gram
2. The defect of deciduous teeth including hypoplasia and hypocalcification and the severity was measured using DDE index and FDI modification and determinate with mild defect with score <12 dan severe with score >12 ¹⁷

Table 3.1. Score Of Severity Enamel Defect¹⁷

Category	DDE score	
Normal	0	No defects
Mild	$>0-11,9$	
Severe	≥ 12	

Confounding variable Children's factors: birth weight, birth length, head circumference, sex, and hypoxia during delivery

CHAPTER 4

4.1 Result and Discussion

Table 4.1. Birth weight and the Probability Defect Value of Deciduous Teeth

Birth weight (grams)	The probability defect value of deciduous teeth		
	Normal	Mild	Severe
900	.0009109	.0147886	.9843004
1200	.0039606	.0379203	.9581191
1300	.0064134	.0514948	.9420918
1400	.0103168	.0694669	.9202163
1500	.0164453	.0928612	.8906935
1600	.0258915	.1226055	.8515031
1700	.0400949	.1592217	.8006834
1750	.0495104	.1800486	.7704411
1800	.0607676	.2023698	.7368626
1840	.0712445	.2211308	.7076246
1900	.0896395	.2503417	.6600187
1950	.107584	.2751451	.6172709
2000	.1279921	.2997624	.5722455

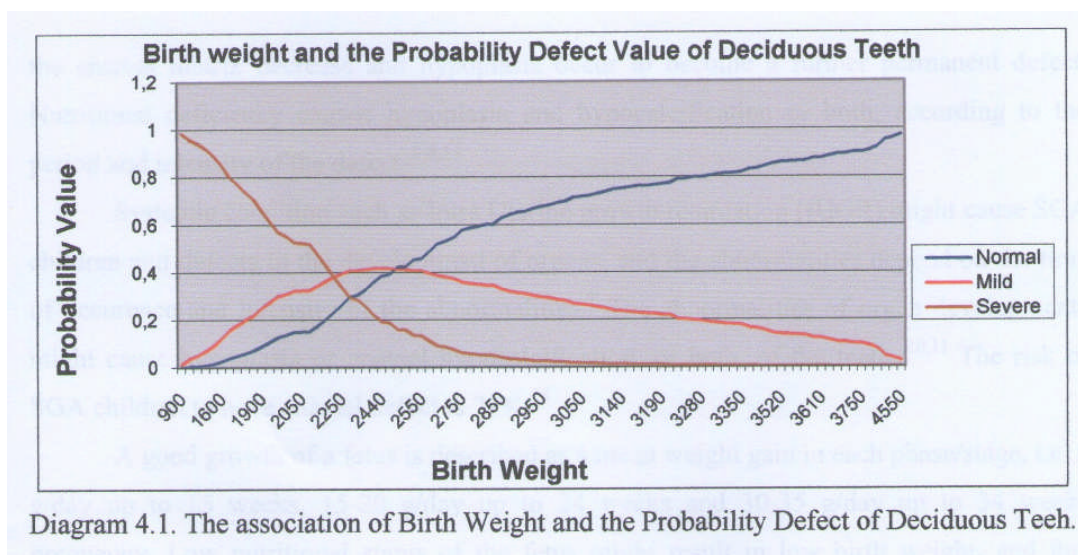


Figure 1 shows that the lower the birth weight, the more the probability of the severity. The higher the birth-weight the lower the probability of severity. Prenatal malnutritional. Children had less nutritional intakes that occur during the first trimester of pregnancy, and further during the second or third trimester that might occur in a short till a long time.

A healthy pregnant woman with adequate nutritional status will deliver a healthy neonate with normal birth weight, while a sick or unhealthy pregnant woman with low socio-economic condition will deliver a low birth weight neonate. About 70% of the low birth weights are SGA babies with birth weight lower than -2SD normal weight or the 10th percentile of Lubchenco intra uterine growth curve (<2500 g), or even has a birth weight of 2600 grams.^{9,15-6}

This condition could be caused by systemic condition such intra uterine growth retardation (IUGR) as a result of maternal factors such as severe infection during pregnancy, preeclampsia hypertension, maternal diabetes, smoking, alcohol, and mother's age at delivery of >35 years.^{9,11} Other causes of IUGR are placental abnormalities, and child's factors such as genetic abnormalities, syndromes, multiple gestation, that might cause intrauterine malnutrition.^{9,15-6,19}

Prenatal nutrition has an important role in matrices construction and enamel mineralisation of deciduous teeth, while postnatal nutrition has a role in matrices and mineralisation of permanent teeth.^{2,5,14} Vitamin A, C, D, K, and minerals (calcium, phosphor, magnesium, fluor) are the nutrition needed in constructing the teeth. Nutritional deficiency during bellstage might result in decreased activities of ameloblast in matrix secretion, so that

the enamel matrix decrease and hypoplasia occur to become a further permanent defect. Nutritional deficiency caused hypoplasia and hypocalcification or both, according to the period and intensity of the defect.^{5-8,14}

Systemic condition such as Intra Uterine growth retardation (IUGR) might cause SGA children and defects in the development of organs, and the abnormalities depend on the time of occurrence and intensity of the abnormalities.⁹ The abnormalities of organ developments might cause hypoplasia or enamel hypocalcification, or both, of the teeth.^{10,"} The risk of SGA children to have enamel defect is 79%.¹⁸

A good growth of a fetus is described as a mean weight gain in each phase/stage, i.e. 5 g/day up to 15 weeks, 15-20 g/day up to 24 weeks and 30-35 g/day up to 34 weeks pregnancy. Low nutritional status of the fetus might result in low birth weight, and this might cause a defect of the organ growth and development might be including the teeth.¹⁸

Birth weight and gestational age reflect the Fetal Growth Rate that means the birth weight reflects the nutritional status of the fetus, the better the nutritional status, the better the birth weight, and that means the intrauterine growth and development restriction did not occur or only in a mild intensity.¹⁴ Birth weight is the reflection of the intrauterine growth and development, so that the decrease of birth weight due to malnutrition might be a risk to become a growth defect.^{9,15-6,19} Mal nutrition is caused by lack of absorption of the placenta.

The above figure shows the association between birth weight and the severity of the defect of deciduous teeth, that is the lower the birth weight in small for gestational age children the more the probability of the severity of the defect.

CHAPTER 5

5.1 Conclusion

Birth weight in children with prenatal malnutrition reflect the severity of the defect of deciduous teeth, the lower the birth weight, the more severe the probability defect of the enamel.

5.2 Acknowledgement

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REFERENCES

1. Moyers RE. 1988. Handbook of Orthodontics. 4th ed. Chicago: Yearbook Medical Publisher Inc.
2. Stewart RE, Witkop CJ, Bixler D. The dentition. In: Stewart RE, Barber TK, Troutman KC, Wei SHY. Pediatric dentistry, scientific foundation and clinical practice. Editors. ST. Louis: The C.V. Mosby Co., 1982. p. 87-94.
3. Laskaris G. Color atlas of oral diseases in children and adolescents. Stuttgart: Thieme, 2000. p. 20-3.
4. Magnusson BO. Pedodontic: a systematic approach. Munksgaard: PJ. Schmidt Vojens, 1981. p. 79-85.
5. Australian Dental Association. Tooth enamel defects. Mi-tec Medical Publishing, 2007 Jan; 1.
6. Seow WK. Effect of preterm birth on oral growth and development. Australian Dental Journal. 1997;42(2):85-91.
7. McDonald RE, Avery DR. Dentistry for the child and adolescent. 6th Ed. St Louis: CV. Mosby Year-Book Inc, 1994. p. 53-9.
8. Welburry RR. Pediatric dentistry. 2nd Ed. Oxford University; 2001. P. 294,394-5.
9. Fanaroff AA, Martin RJ. Neonatal-perinatal medicine: diseases of the fetus and infant. 7th Ed. St Louis: Mosby Inc, 2002.
10. Lunardelli MS, Peres MA,; Breast-Feeding and other Mother -Child Factors, associated with developmental Enamel Defects Of Brazillian Children, Journal of Dentistry for Children - 73:2,2006 p70-6
11. Simmer JP Dental Formation and its impact on clinical Dentistry. J. Dent ed 2001 65(9): 896-9
12. Seow WK, Brown JP, Tudehope DA, O'Callaghan M. Dental defects in deciduous dentition of premature infants with low birth weight and neonatal rickets. Pediatrics Dentistry. June 1984;6(2)
13. Seow WK. Oral Complications of premature birth Austr dent j 1986;23-9
14. Wei SHY, Anderson TA. Nutrition and dental health. In: Stewart RE, Barber TK, Troutman KC, Wei SHY. Editors. Pediatric dentistry, scientific foundation and clinical practice. ST. Louis: The C.V. Mosby Co., 1982. p. 561-3.
15. Avery GB, Fletcher MA, MacDonald MG. Neonatology pathophysiology management of the newborn. 5th Ed. Philadelphia: Lippincott Williams & Wilkins; 1999.

16. Thureen PJ, Anderson MS, Hay WW. The small for gestational age infant. Neoreviews. 2001;2:e139-48.
17. Willyanti ;Skor Prediksi tingkat Keparahan Defek Email Gigi Sulung Pada Anak Dengan Kecil Masa Kehamilan Unpad Press,2009,p 98-99
18. Sjarif Willyanti³Oewen Roosye, Effendi Sjarif H, Sutrisna Bambang. Enamel defect of deciduous teeth in small gestational age children. Dental Journal 2010;43 june 2010 p:915.
19. Cunningham FG, Leveno KJ, Bloom SL, Hauth JC, Gilstrap L, Wenstrom KD. Williams obstetrics. 22nd Ed. New York: Me.Graw Hill, 2005. p. 744-64.