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ORIGINAL ARTICLE

Enamel Hypoplasia and its Correlation with Dental Caries In School Children of Bagalkot, Karnataka

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ABSTRACT

Aim: To study the prevalence and correlation of Enamel Hypoplasia and Dental caries among school children aged 6-15 years in Bagalkot, Karnataka.

Materials and Methods: 5500 school children aged 6 -15 years from different schools in Bagalkot were examined with mouth mirror and probe under natural light. The findings were entered into standard examination forms.

Results: Out of 5500 school children between the age of 6 – 15 years, 507 (9%) had carious teeth with hypoplasia with a mean of 3.1 carious teeth per child. In 5500 children 1987 (41%) had carious teeth without enamel hypoplasia with a mean of 1.39 decayed teeth per child and this shows that the prevalence of caries in hypoplastic teeth was evident and was statistically significant.

Keywords: Enamel hypoplasia, Dental caries, School children, Fluorosis.

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INTRODUCTION

Enamel is a unique mineralized tissue in its method of development, structure and chemical nature. It is the only mineralized tissue of ectodermal origin in vertebrates. Mature enamel, containing very little organic matrix, is the most highly mineralized and hardest tissue in the body. Enamel is so stable that it can resist heavy occlusal forces and various noxious chemicals. However, enamel is not without its weaknesses.

Developmental defects of enamel (Enamel Hypoplasia's) present a wide range of features. The defects may affect a circumscribed area of one surface of the enamel or, at the other extreme; they may be wide spread, affecting all surfaces of the enamel throughout its full thickness. Similarly the condition may be localized or generalized and may be symmetrical or asymmetrical across the midline of the dentition.

A large number of causes have been described for enamel defects, both environmental and genetic. Enamel hypoplasia may be inherited as primary defects of enamel or may be acquired as a result of childhood medical problems such as infections, metabolic derangements, premature birth and nutritional disorders. Ameloblasts, the specialized end cells that form enamel are very sensitive to insults and the locations of enamel defects on the teeth provide permanent records of injury during particular periods of development. The abnormal discoloration and tooth morphology associated with enamel hypoplasia may compromise esthetics and predispose the affected teeth to dental caries.

An increase in caries incidence is not necessarily a related phenomenon and on occasions, example, with fluorosis, the affected teeth are more resistant to the disease. The treatment that is required is directed towards improving the cosmetic appearance of the involved tooth or teeth and in some instances in preventing loss of tooth substance through caries, attrition, abrasion or erosion. Dental caries ranks among the most significant of human diseases in dental literature simply because of its frequency of occurrence. In India over 95% of population is affected, ranking dental caries first among the chronic diseases affecting human kind in terms of

the number of people involved.

The prevalence of enamel hypoplasia in many indigenous populations has been documented and is reported to be high. This reflects the high levels of infant perinatal morbidity in these communities. High prevalence's are observed in many prehistoric populations around the world which suggests high levels of biologic stress.

In India, there are several areas of high fluoride content. Fluorides as proven can cause fluorosis, which affects the enamel. A little study has been done on enamel hypoplasia and absolutely no literature on correlating this with dental caries. Bagalkot, a town which is also a district head quarters in northern Karnataka is a known area of fluoride belt (1). Fluorosis is quite rampant here with ground water levels of fluoride ranging between 0.5 to 4ppm in different areas of demographic areas of bagalkot and subsequently, enamel hypoplasia related to fluorosis is common.

The aim of this study was to examine the prevalence and etiological factors associated with enamel hypoplasia and its correlation with dental caries in 5500 school children, 6 -15 years of age in the schools of Bagalkot district, Karnataka, South India.

Table 1: Modified DDE INDEX for use in General purpose epidemiology studies

Demarcated opacities	Code
Normal	0
White/Cream	1
Yellow/ Brown	2
Diffuse Opacities	
Diffuse- Lines	3
Diffuse- Patchy	4
Diffuse-Confluent	5
Confluent/Patchy+ Staining +Loss of enamel	6
Hypoplasia	**Code**
Pits	7
Missing Enamel	8
Any other defects	9
Combinations	**Code**
Demarcated and Diffuse	A
Demarcated and Hypoplasia	B
Diffuse and Hypoplasia	C
All three defects	D

Table 2: Prevalence of Enamel Hypoplasia

Enamel Hypoplasia	Number affected	Per-centage
Teeth: N- 16,470	7,137	43%
Patients: N – 5,500	705	13%

Table 3: Prevalence of Dental caries

Dental caries	Number affected	Per-centage
Teeth: N -13,963	1576	10%
Patients: N – 5,500	507	9%

MATERIALS AND METHODS

This epidemiological study group consisted of 5500 school children aged 6 to 15 years residing in different schools in Bagalkot. The children were examined by the examiner under natural light. A mouth mirror and a probe were used to determine the presence of enamel hypoplasia and dental caries. All clinically visible surfaces were examined and the findings entered into a standard examination forms. A modified DDE (Developmental Defects of Enamel) index was used to classify the enamel defects (2). Enamel hypoplasia was considered present if a tooth surface showed enamel to be either pitted, grooved or missing. Enamel opacity was considered to be a distinct change in translucency of enamel. Dental caries was diagnosed and classified using WHO criteria (3).

RESULTS

Prevalence of enamel hypoplasia and Dental caries

In our study on 5500 school children of different age groups ranging from 6 years to15 years, 705 (13%) students had enamel hypoplasia (Table 1 and 2). In these 705 subjects, out of 16,470 teeth, 7137 (43%) had enamel hypoplasia. Mean number of hypoplastic teeth per child was 1.3 (range 0 – 28).

For the convenience of the study the subjects were divided into group A (Table 4) consisting of children 6 to 10 years and group B from 11 to 15 years (Table 5). From the Table 6 it was shown that none of the age groups or sex showed any significance. Also the 'Z' value comparing only enamel hypoplasia with different age groups show that there was no statistical significance shown between males and females of different age group. But group B showed a very low statistical significance (p – 0.05) as compared with the group A.

Relation between different types of enamel hypoplasia's with dental caries

Enamel defects were noted according to the modified DDE index (2). The diffuse patchy type of enamel hypoplasia was the more commonly seen defect in 305 patients (43%) with the maximum number of teeth involved (Table 8 and 9). Dental caries was seen in hypoplastic teeth which suggest that caries may be seen in patients with enamel hypoplasia. Five hundred and seven (9%) of children had at least one decayed tooth with a mean of 3.1 (range 0 -10).

In 5500 subjects, 1987 (41%) had dental caries without enamel hypoplasia and out of 1, 18,099 teeth 13,963 (12%) had at least one decayed tooth with a mean of 8.4 (range 0 -12). There was an association which was statistically significant (chi^2 = 651.75, df = 1, p Value=.01) (Table 3).

Relationship of Fluorosis and Dental caries

Among the 705 children with enamel

Table 4: Association of enamel hypoplasia and caries

Teeth	With caries	Without caries	Total
With hypoplasia	1576	5561	7137
Without Hypoplasia	13,963	104136	118099
Total	**15539**	**109697**	**125236**

Chi^2 = 651.750 ; df = 1; p Value =0.01

Table 5: Total number of children with different age groups showing correlation of Enamel hypoplasia with Dental caries (Group A)

Age	Total no of positive cases		Enamel hypoplasia+ caries		Caries on non-Hypoplastic teeth		Only enamel hypoplasia	
	Male	Female	Male	Female	Male	Female	Male	female
6 years	48	43	16	16	13	17	19	10
7 years	28	35	10	11	12	16	6	8
8 years	50	87	16	34	25	35	9	18
9 years	47	36	18	10	20	18	9	8
10 years	39	37	9	a10	22	17	8	10
Total	212	238	69	81	92	103	51	54
	450		150		195		105	

hypoplasia, 230 patients had fluorosis with 314 teeth affected. Dental caries was observed only in 97 (42%) students having 217 (7%) teeth affected. A high value of chi^2 (656.066) and p Value is less than 0.001. These high values of chi^2 assure the statement that the proportion of carious teeth among fluorosis teeth is almost negligible (Table 10).

Table 6: Total number of children with different age groups showing correlation of Enamel hypoplasia with Dental caries (Group B)

Age	Total no of positive cases		Enamel hypoplasia+ caries		Caries on non-Hypoplastic teeth		Only enamel hypoplasia	
	Male	Female	Male	Female	Male	Female	Male	female
11 years	38	22	10	5	17	8	11	9
12 years	63	41	22	13	21	8	20	20
13 years	14	31	5	8	5	9	4	14
14 years	11	10	6	5	3	1	2	4
15 years	17	8	6	3	4	3	7	2
Total	143	112	49	34	50	29	44	49
	255		83		79		93	

Table 7: Table of "Z" value comparing Enamel hypoplasia with different age group

Age	'Z' values	Significance	P Value
6 years	1.6688	Not Significant	0.05
7 years	0.1355	Not Significant	0.05
8 years	0.3810	Not Significant	0.05
9 years	0.3438	Not Significant	0.05
10 years	0.6677	Not Significant	0.05
11 years	0.9472	Not Significant	0.05
12years	1.7450	Not Significant	0.05
13 years	1.0517	Not Significant	0.05
14 years	1.1054	Not Significant	0.05
15 years	0.7860	Not Significant	0.05
Group A	0.3424	Not Significant	0.05
Group B	**2.1372**	**Significant**	**0.05**

Relation of Enamel Hypoplasia with student's Medical History and Location type

The influence of various prenatal (maternal) health factors such as pregnancy age, number of previous pregnancies and perinatal infections on the development of enamel hypoplasia was studied by comparing the percentage of hypoplastic teeth in children born of mothers with different frequencies of each condition.

As shown in table 5, the highest percentage of hypoplastic teeth was seen in children born of mothers below 16 years of age at the time of pregnancy. The next in order of frequency was seen in children born of mothers with multiple pregnancies. Prematurity of birth has been associated with enamel hypoplasia of the primary dentition. Studies of children with birth weights between 2500gm and 300gm have shown the prevalence of enamel hypoplasia to be around 20%. Our study shows 8% of low birth weight children. The probable reason could be deficiency of bone mineral as a result of metabolic derangements and inadequate mineral supply as also noted by Seow, W.K. and Humphry S.C. (4).

Our study population consisted of mainly two groups; rural and periurban. The rural groups of students were almost considered to be low socio- economic status. Osuji et al(1998) (5) have suggested that diffuse mottling may be more prevalent in children from high socio-economic groups but no figure were available in ours and their studies for the prevalence of such defects according to social class or location type.

Although chronic malnutrition in underdeveloped countries has been associated with increased caries rates, the mechanisms involved are unclear. In relation to caries, the effect of malnutrition may be mediated through alterations of salivary composition and volume. Malnutrition may also increase caries susceptibility by decreasing tooth resistance through the formation of enamel defects, such as linear enamel hypoplasia or perhaps increasing enamel solubility (6).

Table 8: Table showing the medical history and socioeconomic status of parents and children

Sr No.	Medical history	Total No of patients	Total no of teeth hypoplasia
1	Age at pregnancy		
	Less than 16 years	3	42
	More than 30 years	34	198
2	No of previous pregnancy		
	3-4 children	38	273
	More than 5 or 5 children	33	182
3	Maternal infection	9	53
4	Low SES (I.E.< 11,350)	23	466
5	Infections in child	45	248
6	Low birth weight of child <2.5kg/ 2500Gms	22	454
	Total	**207**	**1916**
	Percentage	**76%**	**48%**

Table 9: Total no of patients and teeth with different Grades of Enamel Hypoplasia in relation to Caries

Type of Defect	Teeth	Patients
Diffuse lines	854	188
Diffuse Patchy	1376	305
Diffuse Confluent	704	119
Opacity, Staining and loss of Enamel	189	47
Hypoplastic pits, grooves and loss of Enamel	42	12
Enamel hypoplasia with caries	231	135
Enamel Hypoplasia without caries	1012	360
Percentage	3%	19%

Distribution of enamel hypoplasia in deciduous and permanent dentition:

In permanent dentition maxillary and mandibular central incisors and molars showed the highest prevalence of enamel hypoplasia, while maxillary and mandibular canine showed the least. Where as in deciduous, maxillary and mandibular deciduous molars showed the highest frequency with the central incisor showing the least.

Table 10: Total No. of patients and teeth showing different grades of fluorosis and its relation with caries Total No. of patients with fluorosis – 230

Fluorosis Grade	Teeth	Patients
Very mild	1353	106
Mild	926	68
Moderate	764	39
Severe	373	17
Total	**3146**	**230**
Total No. of Flurosed teeth affected with caries	**217**	**97**

DISCUSSION

Studies of developmental defects of enamel are undertaken for a variety of reasons. Some are designed to monitor public health measures, especially the use of fluoride either in water supply or as tablets. Other studies are designed to clarify the mechanism of formation of these defects or to determine the relative importance of many etiological factors. Our present project was under taken to study the prevalence and association between enamel hypoplasia and dental caries in school children aged 6 -15 years.

The discussion is formulated under the following headings:

Table 11: Association of Fluorosis with caries

Fluorosis	With caries	Without caries	Total
With fluorosis	217	2712	2429
Without fluorosis	4115	9426	13541
Total	**4332**	**12138**	**15970**

Chi^2 value = 656.066 P value= <.001

Enamel Hypoplasia

Age

In group A, 8 year old children showed the highest prevalence of enamel hypoplasia where as in group B the highest prevalence was in 12 years old and this group was proved to be statistically significant. The children in the group A may have had less exposure to some etiological factors than those in group B as also observed Sendano.H.O (7) and others due age factor.

G.M.Gillepsie (8) mentioned in a study on enamel hypoplasia and dental caries found that, caries superimposed on hypoplastic teeth becomes more extensive as the age advances. M.V.Golpaygani in a recent study has shown that hypoplastic permanent teeth were more sensitive to dental caries process when compared with primary teeth (9). But in our study the enamel hypoplasia decreased with advanced age probably due to prophylactic measures or change in dietary habits undertaken by the growing children.

Number of teeth involved

In deciduous dentition, the molars showed the highest number of hypoplastic teeth, while the mandibular central incisors, showed the least number. In case of permanent dentition, the maxillary central incisors showed the highest prevalence followed by the mandibular and maxillary molars. The least number of hypoplastic teeth involved was the mandibular canines. Our studies showed similar findings as observed by Jackson and James (10), Li. Y. et al (11) and M.V. Gopaygani (9) in permanent dentition.

Types of defects

The prevalence of all types of enamel defects among the children in Bagalkot was not statistically significant. It is not appropriate to compare the results of our study directly with other epidemiological studies, because

of the differences in criteria and classification methods. The majority of early investigations used essentially the classification of AL- Alousi *et al* (12), modification of it, or developed their own classification.

Results obtained using the same DDE index (2); still do not provide clear comparisons because of the complexity of the index itself and other experimental factors such as, use of different light sources, examining teeth and tooth surface examined and fluoride concentration in the drinking water consumed by the population studied.

The tooth defect pattern shown in table 9 using modified DDE index suggest that there is a critical time period in which teeth are more susceptible to develop enamel defects. According to the table 9 the type of hypoplastic lesion most significantly present was "diffuse patchy", and pit and groove formed the least pattern.

Dental developmental chronology of the human dentition by Lunt and Law (13) suggest that one can anticipate different enamel defect patterns on the tooth surfaces. Theoretically, if a disturbance occurs at the 13th week in uterus, a hypoplastic lesion would appear on both maxillary and mandibular central incisors. Similarly, if a disturbance occurs during 6th month after birth, only canines, 1st molars and secondary molars would be affected.

The readings from our tables 5 and 6 support the above theory in deciduous dentition where as permanent dentition was also included in our study, the defects of enamel being probably due to disturbances at the calcification stage of permanent teeth as was seen in our study.

Medical History and Location type

The influence of various prenatal (maternal) health factors such as pregnancy age, number of previous pregnancies and peri-natal infections on the development of enamel hypoplasia was studied by comparing the percentage of hypoplastic teeth in children born of mothers with different frequencies of each condition.

As shown in table 8, the highest percentage of hypoplastic teeth was seen in children born of mothers below 16 years of age at the time of pregnancy. The next in order of frequency was seen in children born of mothers with multiple pregnancies. Prematurity of birth has been associated with enamel hypoplasia of the primary dentition. Studies of children with birth weights between 2500gm and 300gm have shown the prevalence of enamel hypoplasia to be around 20%. Our study shows 8% of low birth weight children. The probable reason could be deficiency of bone mineral as a result of metabolic derangements and inadequate mineral supply as also noted by Seow, W.K and Humphry S.C. (4).

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Although chronic malnutrition in underdeveloped countries has been associated with increased caries rates, the mechanisms involved are unclear. In relation to caries, the effect of malnutrition may be mediated through alterations of salivary composition and volume. Malnutrition may also increase caries susceptibility by decreasing tooth resistance through the formation of enamel defects, such as linear enamel hypoplasia or perhaps increasing enamel solubility. (9, 14, 15). Rough surfaces promote plaque formation by *Streptococcus mutans* adhesion and colony forming, and may lead to quick progression of the disease. Less mineralized enamel is more susceptible to caries. In a symposium on early childhood caries, Seow(1998) (16)called attention to the fact that enamel defects are an important but currently little investigated area in caries research (17).

Fluoride Belt

Bagalkot district which is situated in the northern part of Karnataka is considered to be a fluoride belt area, according to the survey conducted by the Mines and Geology department and central ground water Board (1). Flouride levels in ground water of various places in bagalkot district differed between 0.5 to as high as 4ppm. But the study was conducted in and around bagalkot city where the ground water fluoride level were between 1 to 1.2ppm.

Out of the 705 positive cases that were given the questionnaire, 273 cases responded. The maximum number of children showed that most of them were brought up in the fluoride belt area at least up to the age of 2 years (33%). This showed that there was high significance of fluoride in causing the enamel defects. But caries was less common in these areas irrespective of age, sex or ethnic group. A number of previous authors like Clarkson and O'Mullane (1989) (18), Cutress et. Al (1985) (19) and Betty de Liefde and Graham P.H. (1985) (20) also showed the similar results as that of our study.

Dental Caries

To determine if there was an association between enamel hypoplasia and dental decay, hypoplastic lesions presenting with and without dental caries were compared. The results according to table 7 indicate that dental caries occurred in teeth with enamel hypoplasia. This shows that the prevalence of caries in hypoplastic teeth was evident. This association was statistically significant which was also observed by other authors (9, 17, 21, 6).
In fluorosis the teeth are less susceptible to caries; however, some others believe the caries incidence to be above normal in hypoplastic teeth. An important area in caries etiology which is currently not well emphasized is the area of tooth defects. For some time after eruption, newly exposed enamel surface undergo the final stages of post eruptive maturation and hardening when ions such as fluoride are incorporated.

Epidemiological studies suggest that this period immediately after eruption and prior to final maturation is when the tooth is most susceptible to caries. Thus in many children a combination of recently erupted immature enamel in an environment of cariogenic flora with frequent ingestion of fermentable carbohydrates would render teeth particularly susceptible to caries.

In addition to lack of maturation the presence of developmental structural defects in enamel may increase the caries risk. Developmental defects of enamel may be manifested as partial or total loss of enamel (hypoplasia) or a change in translucency (opacity). Generalized enamel defects in the primary dentition have been associated with a variety of causes ranging from hereditary diseases to acquired prenatal, peri-natal and postnatal conditions such as birth prematurity and low birth weight infections, malnutritions, metabolic disorders and chemical toxicity. In addition, local trauma and infections are responsible for many localized defects (9,4).

In the primary dentition enamel defects are common. The overall prevalence ranging from 13 – 39% in normal full term infants to over 62% in those born preterm with very low birth weight. Surface irregularities such as pits and grooves predispose to plaque retentions, increased mutans streptococci colonization and possibly decreased clearance of carbohydrates.

In spite of significant research advances in dental development and enamel formation in the recent years, the pathogenetic mechanisms of developmental enamel defects remain poorly understood. It is likely that advances in cell and molecular biology will rapidly improve our understanding of this complex field in the future.

Finally, properly controlled long term clinical studies of children with systemic and local enamel anomalies are urgently required to identify the clinical complications associated with this common clinical entity.

CONCLUSION

The study revealed the following results.

- The school children of Bagalkot had moderate prevalence of hypoplasia.
- There was an association between enamel hypoplasia and dental caries which was statistically significant.
- The prevalence of hypoplastic teeth decreased as the age advanced.
- The maxillary and mandibular central incisors and molars showed maximum number of enamel hypoplasia and the least was seen in the maxillary and mandibular canines of permanent dentition.
- In deciduous, maxillary and mandibular molars showed the highest frequency of enamel hypoplasia with mandibular central incisors showing the least.
- Dental caries was more commonly prevalent in teeth without hypoplasia and it reduced as the age advanced.
- High prevalence of fluorosis and medical morbidity in the prenatal, peri-natal and infancy may be important etiological factors in the pathogenesis of enamel hypoplasia, although the relative importance of these factors is difficult to determine.

REFERENCES

1. Mines and Geology Department and Central ground water board, Banglore. Areas affected with increased fluoride concentration in villages of Karnataka. 1995.
2. World Health Organization. Oral Health Surveys. Basic Methods 2nd Ed. Geneva: WHO 1977.
3. Federation Dentaire Internationale. Commission on oral health research and epidemiology: A review of developmental defects of enamel (DDE Index). *Int Dent J* 1992;**42**:411-426.
4. Seow WK, Humpry SC. Increased prevalence of developmental dental defects in low birth weight, prematurely born children; a controlled study. *Pediatr Dent* 1987;**9**:221-223.
5. Osuji OO, Leake JL, Chipman ML, Nikiforuk G. Risk factors for dental fluorosis in a fluoridated community. *J Dent Res* 1988;**67**(12): 1488-1492.
6. Pascoe L, Seow WK. Enamel hypoplasia and dental caries in Australian Aboriginal Children: Prevalence and correlation between the two diseases. *Pediatr Dent* 1994;**16**:193-199.
7. Sedano HO. Clinical orodental abnormalities in Mexican children. *Oral Surg Oral Med Oral pathol*; 1989;**68**:300-301.
8. Gillespie GM, Peter IF. Enamel hypoplasia in relation to caries in Guatemalan children. *JDent* 1977;**5**:493-498.
9. Golpaygani MV, Mehrdad K, Mehrdad A, Ansari G. An Evaluation of the Rate of Dental Caries among Hypoplastic and Normal Teeth; A case control Study. *Res J Biol Sci* 2009;**4**(4): 537-541.
10. Jackson D, James PMC. Fluoridation in Anglesey. *Brit Dent J* 1975;**138**:165-171.
11. Li Y, Navia JM, BianYJ. Prevalence and distribution of developmental enamel defects in primary dentition of Chinese children, 3-5 years old. *Community Dent Oral Epidemiol* 1995;**23**:72-79.
12. Al-Alousi W, Jackson D. Enamel mottling in a fluoride and in a non- fluoride community. *Brit Dent J* 1975;9-15,56-60.
13. Lunt RC, Law DB. A review of the chronology of teeth eruption. *J Am Dent Assoc* 1974;**89**:872-879.
14. Seow WK. Enamel Hypoplasia in the primary dentition; a review. *J Dent Child*; 1991;**11-12**:441- 452.
15. Wilfred AN, Matsson I. Developmental enamel defects of the primary dentition in a group of Californian children. *J Dent Child* 1987;**9-10**:330-334.
16. Seow WK. Biological mechanism of early childhood caries. *Community Dent Oral Epidemiol* 1998;**26**:suppl,18-27.
17. Gravina DBL, Cruvinel VRN, Azevedo TDPL, Toledo OA, Bezerra ACB. Prevalence of dental caries in children born prematurely or at full term. *Braz Oral Res* 2006;**20**(4):353-357.
18. Clarkson J, O'Mullane D. Prevalence of enamel defects/fluorosis in fluoridated and non- fluoridated areas in Ireland. *Community Dent Oral Epidemiol* 1992;**20**:196-199.
19. Cutress TW. Defects of tooth enamel in children in fluoridated and non fluoridated water areas of Auckland. *New Zealand Dent J* 1985;**81**:12-19.
20. Betty-de-Liefde, Herbison PG. Prevalence of developmental defects of enamel and dental caries in New Zealand children receiving differing fluoride supplementation. *Community Dent Oral Epidemiol* 1985;**13**:164-167.
21. Hong L, Levy SM, Warren JJ, Broffitt B. Association between Enamel Hypoplasia and Dental Caries in Primary Second Molars: A Cohort Study. *Caries Res* 2009;**43**:345-353.