# The Prevalence of the Developmental Defects of Enamel in a Group of 8–15 Years Old Indian Children with Developmental Disturbances

CHHAVI JINDAL, SANGEETA PALASKAR, SHIKHA KLER


## ABSTRACT

**Aims:** To find the overall prevalence of the developmental defects of enamel among the total number of disabled children in the Panchkula district of Haryana, India.
To compare the prevalence of this dental developmental anomaly with the various types of disabilities like mental handicap, locomotor handicap, hearing impairment, partial sight and multiple handicaps.

**Material and Methods:** A total of 996 subjects (499 controls and 496 disabled children) were examined for the developmental defects of enamel by using a Modified DDE index. The 496 disabled children included 189 with mental retardation, 203 with locomotor handicaps, 39 with hearing impairment, 31 with partial sight and 34 with multiple handicaps.

**Statistical Analysis:** The data which was obtained was analyzed by using the SPSS package version 13. The differences were tested for statistical significance by using the Pearson's Chi-Square test.

**Results:** The percent prevalence of the developmental defects of enamel among the disabled group was 40.9% and in the controls it was 5.4%. The percent prevalence of the various developmental defects of enamel in decreasing order, among the various disabled groups was found to be as follows: 73.5% in the group with multiple handicaps, 56.4% in the group with the hearing impairment, 39.4% in the group with the locomotor handicaps, 37.6% in the group with the mental handicap and 16.1% in the group with the partial sight.

**Conclusions:** Overall, a high prevalence rate of the developmental defects of enamel was observed in this study in the disabled children. This reflects the association of various systemic disturbances with the development of the tooth.


**Key Words:** Developmental, Defects, Disabled, Enamel, Enamel hypoplasia

## INTRODUCTION

The developmental defects of enamel are one of the most frequently observed developmental abnormalities of the human dentition. These may be the defects of enamel matrix formation or mineralization or maturation with reduced or altered amounts of enamel which is caused by an insult to the ameloblast cells [1].

It has become evident that systemic or local environmental stresses or genetic factors or a combination of these are responsible for disrupting the metabolism of the ameloblasts which results in the tooth defects. Hence, the tooth enamel often acts as a repository of information on the systemic insults which are received during the development [2]. Such influences may begin before or after birth so that the deciduous or permanent or both teeth may be involved. Usually, it is the permanent teeth that are influenced and, in all instances only those that are not completely formed at the time of the disturbance are affected.

The recognition and identification of the dental anomalies are of great importance for a timely and accurate diagnosis of the numerous genetic abnormalities of the craniofacial region. The developmental defects in enamel especially in the primary teeth, may become useful as biological markers for the timing and in some cases, the nature of the insult to the tooth germ [3].

Although studies which are related to the increased prevalence of dental caries, poorer oral hygiene and a greater prevalence and the increased severity of periodontal diseases in disabled children have been well documented [4,5,6], the data on the prevalence of the dental developmental anomalies in these special group of children is scarce. Hence, an attempt has been made to find out the prevalence of the developmental defects of enamel in disabled children.

## MATERIALS AND METHODS

The prevalence of enamel hypoplasia was studied in a sample of 995 subjects who were aged 8-15 years, including 496 subjects with developmental disturbances (189 with mental retardation, 203 with locomotor handicaps, 39 with hearing impairment, 31 with partial sight and 34 with multiple handicaps) and a control group of 499 school children who were free from any of the above mentioned developmental disturbances. The disabled children included those students with special needs, who were studying in formal schools along with normal children under the scheme of 'Integrated Education for Disabled children' (IED), which was implemented by the Ministry of Human Resource Development (Department of Secondary and Higher Education). Before examination, a written consent was taken from the heads of the respective institutions.

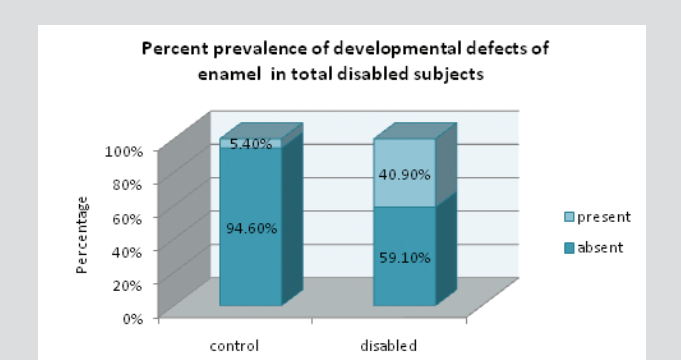
## EXAMINATION PROCEDURE

All the children were examined in their respective schools while they were seated on ordinary chairs, unless they were confined to wheel chairs. The oral examination was done under natural light by using a standard mouth mirror and a probe. All the teeth were screened for the developmental enamel defects of the teeth.

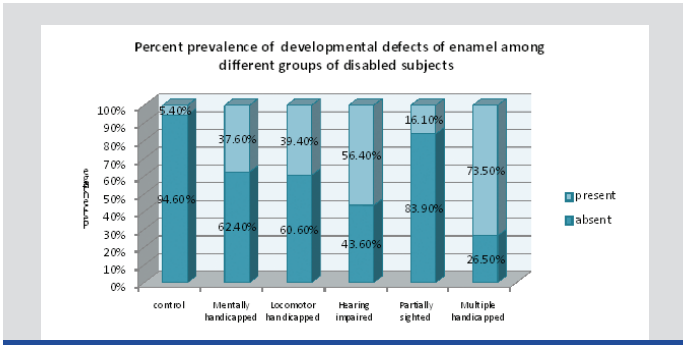
The developmental enamel lesions were diagnosed without drying or cleaning the teeth prior to the examination. The type and the

| Code | Type of defect | Definition |
|---|---|---|
| 0 | Normal | |
| 1 | Demarcated opacities [Table/Fig-1] | Opacity (5) is defined as the qualitative defect of the enamel identified visually as an abnormality in the translucency of the enamel. It is characterized by a white or discoloured (cream or yellow) area but in all cases enamel surface is smooth and the thickness of enamel is normal, except in some instances when associated with hypoplasia. Patchy, irregular, cloudy areas of opacity lacking well defined margins. |
| 2 | Diffuse opacities [Table/Fig-2] | distinct opacity with well defined margins |
| 3 | Hypoplasia [Table/Fig-3] | Hypoplasia (5) is defined as quantitative defect of enamel visually and morphologically identified as involving the surface of enamel (an external defect) and associated with reduced thickness of enamel. The defective enamel may occur as (a) shallow or deep pits arranged horizontally in a linear fashion across the tooth surface or generally distributed over the whole or part of the enamel surface; (b) the defective enamel may occur as small or large, wide or narrow grooves; (c) in some instances there may be partial or complete absence of enamel over small or considerable areas of dentine. |
| 4 | Other defects (Fig. 4) | If any defect does not fall into these categories, they were scored as others. |

**[Table/Fig-1]:** Modified DDE index




**[Table/Fig-2]:** Percent prevalence of developmental defects of enamel in total disabled subjects




**[Table/Fig-3]:** Percent prevalence of developmental defects of enamel among different groups of disabled subjects

localization of the developmental defects of enamel were classified according to the modified DDE index [7] which was introduced by Clarkson J and O'Mullane D [7].

The data which was obtained was analyzed by using the SPSS package, version 13. The differences were tested for statistical significance by using the Pearson's Chi-Square test.

## RESULTS

A total of 995 subjects were examined for the developmental defects of enamel, of which 499 were controls and 496 were disabled children. Out of the 499 controls, 303 (60.7%) were males and 196 (39.3%) were females. Among the 496 disabled children, 322 (64.9%) were males and 174 (35.1%) were females. Of the total of the 496 disabled subjects who were examined, 189 were mentally handicapped (107 males, 82 females); 203 were locomotor handicapped (151 males, 52 females); 39 were hearing impaired (22 males, 17 females); 31 were partially sighted (20 males, 11 females) and 34 were multiple handicapped (22 males, 12 females). [Table/Fig- 2 and 3] represents the percentage prevalence of the developmental disturbances of enamel in the different groups of subjects.

By comparing the prevalence of the developmental defects of enamel among the different groups by using Chi square tests, it was found that the "p" value was less than .001 i.e this was highly significant for the comparisons among the control and the mentally handicapped, the control and the locomotor handicapped, the control and the hearing impaired and the control and the multiple handicapped groups [Table/Fig- 3]. The "p" value was (0.01-0.05) i.e. significant among the control and the partially sighted groups [Table/Fig- 3].

The distribution of the total sample size according to the types of the developmental defects of enamel has been shown in [Tables/Fig-4 and 5].

The incisors were the most common teeth which were affected by the enamel hypoplasia in the subjects among the different groups followed by the molars, the canines and the premolars [Table/Fig-6].

## DISCUSSION

In the present study, an attempt has been made to compare the prevalence of the developmental defects of enamel among the various types of disabilities like mental handicap, locomotor handicap, hearing impairment, partial sight and multiple handicaps and to find the overall prevalence of this dental developmental anomaly among the total number of disabled children in the Panchkula district.

The ameloblasts are very sensitive to a wide range of systemic and genetic disturbances and are unable to recover once they are damaged. Hence, the tooth enamel often acts a repository of information on the systemic insults which are received during the development and these present as the developmental defects of enamel. All the subjects who were examined were in the 8 to 15 years age group. A similar age group was recommended by Clarkson JJ and O'Mullane DM [7], stating that the children who were aged 8 and 15 years gave a range of ages which were sufficiently wide to determine the prevalence of the defects on the early and late erupting teeth and the changes over time.

In the present study, the percent prevalence of the developmental defects of enamel among the normal children was 5.4% [Table/Fig-2]. This was comparable with the studies of Yonezu T et al (8) and Goodman AH et al (9), who reported the populations with the lowest enamel hypoplasia from Japan (2%) and Mexico (6%) respectively. The slight deviation in the figures could be attributed to the racial differences and the diversity of the methodological procedures which were used.

In general, a higher prevalence of enamel hypoplasia has been reported among malnourished children [10,11], very low birth weight children [12] and in patients with sensori-neurological defects [13].

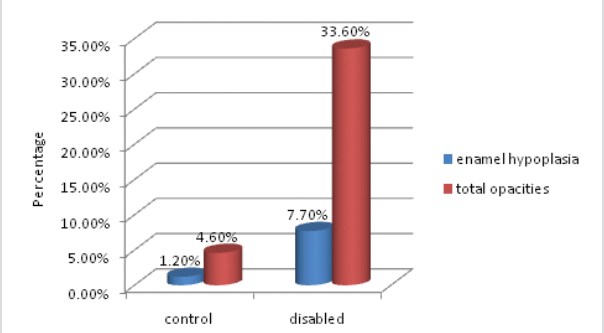
| | | Control | Disabled | Mentally Handicapped | Locomotor Handicapped | Hearing impaired | Partially Sighted | Multiple Handicapped |
|---|---|---|---|---|---|---|---|---|
| Normal | Count | | 293 | 118 | 123 | 17 | 26 | 9 |
| | % within abnormality | 94.6% | 59.1% | 62.4% | 60.6% | 43.6% | 83.9% | 26.5% |
| Demarcated opacities | Count | 4 | 62 | 18 | 25 | 10 | 1 | 8 |
| | % within abnormality | .8% | 12.5% | 9.5% | 12.3% | 25.6% | 3.2% | 23.5% |
| Diffuse opacities | Count | 15 | 73 | 27 | 24 | 8 | 3 | 11 |
| | % within abnormality | 3.0% | 14.7% | 14.3% | 11.8% | 18.9% | 9.7% | 32.4% |
| Hypoplasia | Count | 6 | 38 | 16 | 15 | 2 | 1 | 4 |
| | % within abnormality | 1.2% | 7.7% | 8.5% | 7.4% | 5.1% | 3.2% | 11.8% |
| Other defects | Count | 2 | 30 | 10 | 16 | 2 | 0 | 2 |
| | % within abnormality | .4% | 6% | 5.3% | 7.9% | 5.1% | .0% | 5.9% |
| Total | Count | 499 | 496 | 189 | 203 | 39 | 31 | 34 |
| | % | 100.0% | 100.0% | 100.0% | 100.0% | 100.0% | 100.0% | 100.0% |

**[Table/Fig-4]:** Distribution of total sample according to the type of developmental defects of enamel

The present study gave a percent prevalence of the 40% developmental defects of enamel among the total number of disabled children who were examined [Table/Fig-2]. These findings were comparable with those of Dummer PMH et al [14] and Kanchanakamol et al [15], who reported a prevalence rate of 48.9% and 32% respectively in malnourished children. Chaves AMB et al [16], Pascoe L and Seow WK [17], Fyffe HE et al [18] and Lai PY et al [19] observed a prevalence rate of 78.9%, 64.8%, 50% and 96% for enamel hypoplasia in their respective studies, which were quite higher than those of present study.

These differing prevalence figures for the developmental defects of enamel could be attributed to the differences in the populations which were studied and the diversity of the methodological procedures which were used. Furthermore, as the teeth lacked previous cleaning in some studies, this led to the underestimation of the prevalence rates. Also, according to Seow WK [20], the rapid development of caries in the teeth which were affected by the enamel defects made the diagnosis of a pre existing defect more difficult.

The percent prevalence of the dental developmental defects of enamel among the mentally handicapped was 37.6% in the present study [Table/Fig-3], which was similar to the results of Martinez A et al [21], where 37.06% of the children with neurological disorders and mental retardation had the developmental defects of enamel. This could be attributed to the fact that several systemic disturbances which affect the neurological development also alter the development of the tooth germ. Because enamel cannot be recovered once it is damaged, it provides information on the timing and the nature of the insults which potentially affect other ectodermally derived structures like the brain.




**[Table/Fig-5]:** Distribution of subjects according to opacities and hypoplasia type of developmental defects of enamel

39.4% of the locomotor handicapped subjects showed the developmental defects of enamel in the present study [Table/Fig- 3]. The results were comparable to those of Korchagina VV and Diakova SV [22], who found a 44.5+/- 3.5% incidence of enamel hypoplasia among children with the congenital and hereditary developmental defects of the CNS and the locomotor system.

A 56.4% prevalence of the developmental defects of enamel was found in the present study in the hearing impaired disabled subjects [Table/Fig- 3]. Murray GS and Johnsen DC [23] have reported 11 children with enamel defects out of 18 children who were examined for hearing defects. It may be pointed out that the cochlea and dental enamel develop over the same periods in the foetal life.

Although McMillan RS et al [24] described the relationship of the enamel hypoplasia to the cerebral and the ocular disorders, however the present study, we could find only a 16.1% prevalence of the developmental defects of enamel among the partially sighted disabled subjects [Table/Fig- 3].

A high (73.5%) prevalence of the developmental enamel defects in the multiple handicapped disabled subjects [Table/Fig- 3] showed more chances of ameloblastic damage as multiple defects in various tissues were present in these subjects.

Statistical comparisons of the mentally handicapped, the locomotor handicapped, the hearing impaired and the multiple handicapped subjects with the controls were highly significant in the present study [Table/Fig- 3], thus laying an emphasis on the delicate nature of the ameloblasts which could be affected by genetic and sensori-neural disturbances.

But our findings were in contrast to those of Warnakulasuriya KAAS [25] and Li Y et al [1], who found the prevalence of the localised hypoplasia to be higher than that of the opacities i.e 11.9% and 7.3% and 22.2% and 1.6% in the respective studies. The lower prevalence of the opacities which were reported might be due to

| | | Control | Disabled | Mentally handicapped | Locomotor handicapped | Hearing impaired | Partially sighted | Multiple handicapped |
|---|---|---|---|---|---|---|---|---|
| Incisors | Max | 19 | 102 | 35 | 41 | 13 | 5 | 8 |
| | mand | 14 | 64 | 17 | 28 | 4 | 4 | 11 |
| Canines | Max | 6 | 4 | 1 | 3 | 0 | 0 | 0 |
| | mand | 11 | 10 | 3 | 4 | 3 | 0 | 0 |
| Premolars | Max | 7 | 6 | 1 | 4 | 0 | 0 | 1 |
| | mand | 7 | 6 | 2 | 3 | 1 | 0 | 0 |
| Molars | Max | 10 | 17 | 5 | 6 | 5 | 0 | 1 |
| | mand | 8 | 41 | 23 | 10 | 5 | 0 | 3 |

**[Table/Fig: 6]:** Distribution of subjects according to the teeth affected by developmental defects of enamel

the poor illumination and the lack of the examination facilities which were needed to detect the enamel lesions. The higher prevalence of hypoplastic teeth may be due to untreated infections of the primary predecessors and a high incidence of childhood illnesses such as diarrhoea, which may affect tooth formation.

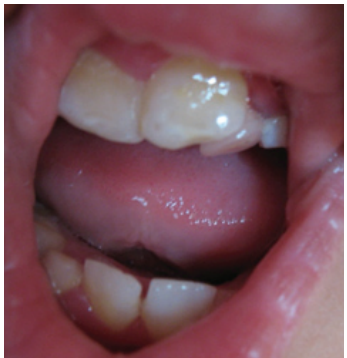
In the present study, the demarcated opacities were highest in the hearing impaired group (25.6%), followed by the multiple handicapped group (23.5%), the locomotor handicapped group (12.3%) and the mentally handicapped group (9.5%) and they were least in the partially sighted group (3.2%). The diffuse opacities were maximum in the multiple handicapped group (32.4%), followed by the hearing impaired group (18.9%), the mentally handicapped group (14.3%) and the locomotor handicapped group (11.8%) and they were least in the partially sighted group (9.7%). Hypoplasia was found to be highest in the multiple handicapped group (11.8%), followed by the mentally handicapped group (8.5%), the locomotor handicapped group (7.4%) and the hearing impaired group (5.1%) and they were least in the partially sighted group (3.2%). Other defects were found to be more in the locomotor handicapped group (7.9%), followed by the multiple handicapped group (5.9%) and the mentally handicapped group (5.3%) and they were least in the hearing impaired group (5.1%) [Table/Fig-4].

Comparison with other studies is a little difficult because of the lack of common terminologies and classifications. The formation of enamel involves a rythmic sequence of cellular activity, interspersed with resting phases. The selective involvement of only those ameloblasts that were currently active at the time of a particular disturbance, may account for the variability in the development of the enamel hypoplasia. Some of the affected ameloblasts may die and stop secreting enamel, whereas others may recover and continue to secrete normal enamel over the defective spots, which could also help in explaining the variability of the enamel lesions.

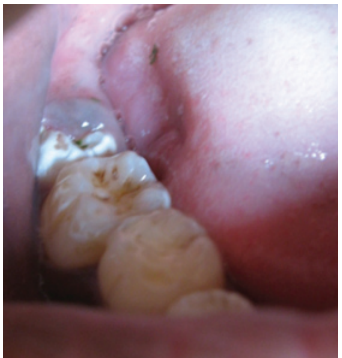
The possible explanation for this variation in the types of enamel defects could be that the demarcated opacities result from either a sudden severe disturbance to a discrete number of cells during their maturation state or from a less severe but longer lasting disturbance during their secretory phase. The diffuse opacities result from a chronic, less severe insult during the secretory and / or the post secretory phases, thus causing a delay in the completion of the mineralisation process.

The teeth which were examined in the present study presented more qualitative i.e opacities in both the control and the disabled groups; 4.6% and 33.6% respectively than the quantitative defects i.e hypoplasia which accounted only for 1.2% and 7.7% respectively [Table/Fig- 5]. These findings were consistent with those of Chaves AMB et al (16), where 13.9% were qualitative defects against 11.2% quantitative defcts and with those of Lunardelli SE and Peres MA [26] where the opacities (qualitative defects) were 23% and hypoplasia (quantitative defects) was 11.1%. This might be attributed to the fact that the teeth suffered injury during the calcification and the maturation of the enamel, rather than during the cell differentiation and the matrix secretion.

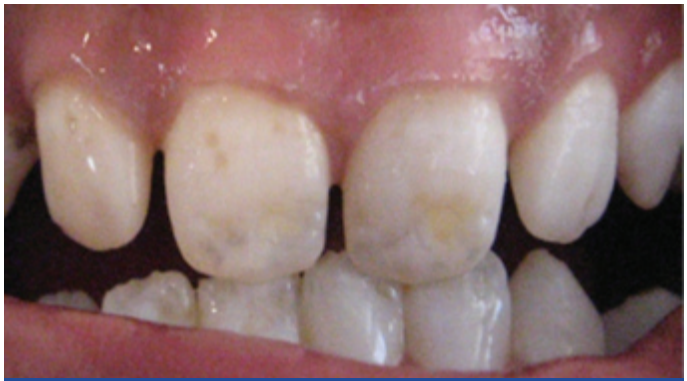
In the current study, among the disabled group, the developmental defects in the enamel were found to be highest in the maxillary incisors, followed by the mandibular incisors, the mandibular molars, the maxillary molars, the mandibular canines and the maxillary and the mandibular premolars. The least affected were the maxillary canines [Table/Fig- 6]. A higher incidence of the enamel defects in the upper incisors than in the lower ones was observed in the present study, which was in agreement with the results of Li Y et al [1], Pascoe L and Seow WK [17], Rugg-Gunn AJ et al [27] and Chaves AMB et al [16]. These findings can be explained on the basis of the observations made by Suga et al (28), who suggested that the difference in the enamel thickness could be the reason for this. Suga et al [28] speculated that the ameloblasts which were responsible for



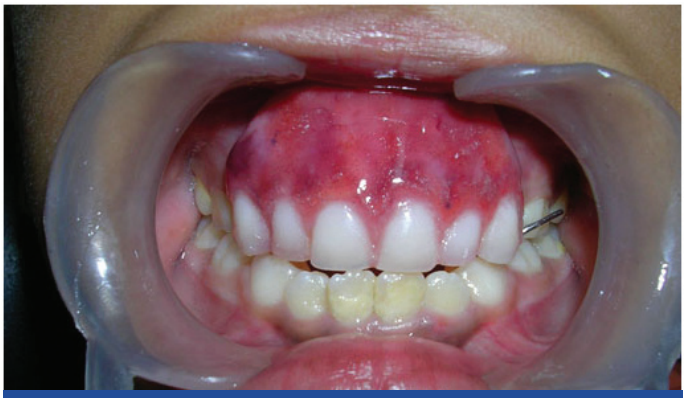
**[Table/Fig-7]:** Demarcated opacities present on both maxillary incisors



**[Table/Fig-8]:** Diffuse opacities in mandibular right first and second molars



**[Table/Fig-9]:** Enamel Hypoplasia affecting both maxillary central incisors, right maxillary lateral incisor & right mandibular lateral incisors



**[Table/Fig-10]:** Other defects in right and left maxillary premolars

the thick enamel were more susceptible to the systemic disorders than the ameloblasts which were associated with the thin enamel. The diffusion of calcium ions from the ameloblasts into the matrix and the removal of organic substances from the matrix are slower in the thick enamel than in the thin enamel. Therefore, the teeth are exposed to the systemic injuries for a longer period of time.

The analysis revealed a significant difference between the disabled children and the control group. The developmental defects of enamel belong to a group of non specific abnormalities and may be present in a number of syndromes. When associated with congenital malformations, they may point to the disorders of various tissues and systems and may occasionally present as the leading symptoms of these states, thus being the key elements in their diagnosis for genetic counselling.

It is essential to understand the various aspects of development because only then we can have a sound knowledge about developmental disturbances. Although they were neglected for long, these disabled children are now considered as an integral part of the society and are rightly known as “children with special abilities”.

This study not only reflects the dental abnormalities in disabled children but also inspires us to take appropriate preventive measures for the betterment of this group of the population, which can be achieved by:

- Making provisions for these special children to seek dental and medical aids.
- Conducting free dental checkup camps.
- Periodic follow up.
- Convincing various health providing organizations about the treatment needs of these special children.

## REFERENCES


[1] Li Y, Navia JM, Bian JY. Prevalence and distribution of developmental enamel defects in primary dentition of Chinese children 3-5 years old. *Community Dent Oral Epidemiol 1995*; 23:72-9.

[2] Bhat M and Nelson KB. Developmental enamel defects in primary teeth in children with cerebral palsy, mental retardation, or hearing defects: a review. *Adv Dent Res 1989*; 3(2):132-42.

[3] Jukic J, Skrinjaric I, Glavina D, Ulovec Z. The prevalence of oral and dental anomalies in children with developmental disturbances. *Acta Stomat Croat 2002*; 36:79-83.

[4] Kendall NP. Oral health of a group of non-institutionalized mentally handicapped adults in the UK. *Community Dent Oral Epidemiol 1991*; 19:357-9.

[5] Namal N, Vehit HE, Koksal S. Do autistic children have higher levels of caries? A cross-sectional study in Turkish children. *J Indian Soc Pedod Prev Dent June 2007*; 97-102.

[6] Shaw L, Maclaurin ET, Foster TD. Dental study of handicapped children attending special schools in Birmingham, UK. *Community Dent Oral Epidemiol 1986*; 14:24-7.

[7] Clarkson J and O’Mullane D. A Modified DDE Index for Use in Epidemiological Studies of Enamel Defects. *J Dent Res 1989*; 68(3):445-50.

[8] Yonezu T, Hayashi Y, Sasaki J, Machida Y. Prevalence of congenital dental anomalies of deciduous dentition in Japanese children. *Bull Tokyo Dent Coll 1997*; 38:27-32.

[9] Goodman AH, Allen LH, Hernandez GP, Amador A, Arriola LU, Chavez A et al. Prevalence and age at development of enamel hypoplasia in Mexican children. *Am J Phys Anthropol 1987*; 72:7-19.

[10] Niswander JD, Sujaku C. Congenital anomalies of teeth in the Japanese children. *American Journal of Physical Anthropology 1963; 21*: 569–74.

[11] Rao D, Amitha H, Munshi AK. Oral hygiene statuts of disabled children and adolescents attending special schools of South Canara, India. *Hong Kong Dent J 2005*; 2(2):107-12.

[12] Fejerskov O, Thylstrup A and Larsen MJ. Clinical and structural features and possible pathogenic mechanisms of dental fluorosis. *Scand J Dent Res 1977*; 85:510-34.

[13] Aldred MJ, Savarirayan R, Crawford PJM. Amelogenesis imperfecta: a classification and catalogue for the 21st century. *Oral Diseases 2003*; 9:19-23.

[14] Dummer PMH, Kingdon A, Kingdon R. Prevalence of enamel developmental defects in a group of 11- and 12-year-old children in South Wales. *Cummunity Dent Oral Epidemiol 1986*; 14:119-22.

[15] Kanchanakamol U, Tuongratanaphan S, Tuongratanaphan S, Lertpoonvilaikul W, Chittaisong C, Pattanaporn K et al. Prevalence of developmental enamel defects and dental caries in rural pre-school Thai children. *Community Dental Health 1996*; 13:204-7.

[16] Chaves AMB, Rosenblatt A and Oliveira OFB. Enamel defects and its relation to life course events in primary dentition of Brazilian children: A longitudinal study. *Community Dental Health 2007*; 24:31-6.

[17] Pascoe L and Seow WK. Enamel hypoplasia and dental caries in Australian aboriginal children: prevalence and correlation between two diseases. *Pediatric Dentistry 1994*; 16:193-9.

[18] Fyffe HE, Deery C and Pitts NB. Developmental defects of enamel in regularly attending adolescent dental patients in Scotland; prevalence and patient awareness. *Community Dental Health 1996*; 13:76-80.

[19] Lai PY, Seow WK, Tudehope DI, Rogers Y. Enamel hypoplasia and dental caries in very-low birthweight children: a case-controlled, longitudinal study. American *Academy of Pediatric Dentistry 1997*; 19(1):42-9.

[20] Seow WK. Clinical diagnosis of enamel defects: pitfalls and practical guidelines. *International Dentistry Journal 1997*; 47:173-82.

[21] De Coster PJ, Marks LA, Martens LC, Huysseune A. Dental agenesis: genetic and clinical perspectives. *J Oral Pathol Med 2009*; 38:1-17.

[22] Korchagina VV, Diakova SV. Dental enamel hypoplasia in children with combined congenital and hereditary defects in the development of the CNS and the locomoter system (infantile cerebral palsy, spinal cord hernias and myopathies). *Stomatologiia (Mosk) 1997*; 76:60-4.

[23] Murray GS and Johnsen DC. Hearing deficits correlated with timing of systemic disturbances as indicated primary incisor defects. *Ear and Hearing 1985*; 6:255-9.

[24] McMillan RS, PH, Kashgarian M. Relation of human abnormalities of structure and function to abnormalities of the dentition. I. Relation of hypoplaisa of enamel to cerebral and ocular disorders. *The Journal of the American Dental Association 1961*; 63:38-48.

[25] Warnakulasuriya KAAS. Prevalence of selected developmental dental anomalies in children, in Sri Lanka. *Journal of Dentistry for Children 1989*; 137-9.

[26] Lunardeeli SE, Peres MA. Prevalence and distribution of developmental enamel defects in the primary dentition of pre-school children. *Braz Oral Res 2005*; 19(2):144-9.

[27] Rugg-Gunn AJ, Al-Mohammadi SM, Butler TJ. Malnutrition and developmental defects of enamel in 2- to 6-year old Saudi boys. *Caries Res 1998*; 32(3):181-92.

[28] Suga S, Brown H, Coote G, Den Besten P, Fearnhead R, Gedalia I et al. Workshop on “Factors that influence the form and distribution of defects”. *Advances in Dental Research 1989*; 3:99-100.

**AUTHOR(S):**
1. Dr Chhavi Jindal
2. Dr Sangeeta Palaskar
3. Dr Shikha Kler

**PARTICULARS OF CONTRIBUTORS:**
1. Deptt. Of Oral & Maxillofacial Pathology, National Dental College & Hospital, Derabassi, Mohali, Punjab, India
2. Professor & HOD, Deptt. Of Oral & Maxillofacial Pathology, Sinhgad Dental College & Hospital, Pune
3. Senior Lecturer, Deptt. Of Oral & Maxillofacial Pathology, DAV(C) Dental College & Hospital, Yamunanagar, Haryana, India.

**NAME, ADDRESS, TELEPHONE, E-MAIL ID OF THE CORRESPONDING AUTHOR:**
Dr. Chhavi Jindal
Senior Lecturer, Deptt. Of Oral & Maxillofacial Pathology,
National Dental College & Hospital, Derabassi,
Mohali, Punjab, India
Phone: 094171-01415
E-mail address ichhavi25@gmail.com

**DECLARATION ON COMPETING INTERESTS:**
No competing Interests.

Date of Submission: **Feb 19, 2011**
Date of Peer Review: **Mar 16, 2011**
Date of Acceptance: **Mar 21, 2011**
Date of Publishing: **Jun 13, 2011**