Review Article

Aetiological Factors for Developmental Defects of Enamel

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Abstract

Developmental defects of enamel remain as a permanent record of a disturbance during amelogenesis. They may present in different forms, some of which may be perceived by an individual as being disfiguring and so requiring treatment to improve the appearance of the teeth. The aim of this review is to address the aetiological factors for DDE because the knowledge is essential for clinicians when explaining and discussing the presence of DDE with patients and their parents. The possible aetiological factors for enamel defects in permanent teeth can be broadly divided into two main categories: those with a localized distribution and those with a generalized distribution. Amongst the causative agents of localized defects of enamel are trauma, localized infection and irradiation. Amongst the causative agents of generalized defects of enamel are genetic disorders and systemic disturbances including intoxications, perinatal and postnatal problems, malnutrition, infectious diseases and a range of other medical conditions. Most of the available data on the aetiology of enamel defects have been gained from animal studies and case reports of children with systemic disorders. The lack of robust data makes the results of these studies inconclusive.

Keywords: Developmental defects of enamel; Aetiological factors

Abbreviations

DDE: Developmental Defects of Enamel

Introduction

Tooth enamel is formed during only a certain period of the tooth development and is irreplaceable. Ameloblasts, which are secretory cells that produce dental enamel, are particularly sensitive to changes in their environment during the long process of enamel production. Dysfunction of ameloblasts may occur resulting in changes in the appearance of the enamel in the permanent dentition. These Developmental Defects of Enamel (DDE) may range from slight abnormalities of the tooth's colour to a complete absence of the enamel.

Impacts of DDE

Effects of DDE may include tooth sensitivity or an increased risk of caries. Treatment of DDE attempts to improve the function and appearance of the affected teeth [1]. There is evidence that teeth with DDE have 10 times greater treatment need than normal teeth [2]. Apart from financial considerations of dental treatment, there is also the social cost including children's absence from school and parents' absence from work to attend multiple appointments. An affected individual may also experience low self-esteem or stigma because they perceive DDE as being disfiguring [3,4]. The knowledge of aetiological factors for DDE is essential for clinicians when explaining and discussing the presence of DDE with patients and their parents. Targeting risk factors could also assist in implementation of community strategies to limit the occurrence of DDE.

Terminology of DDE

An early report of enamel defects, according to Sarnat and Schour [5], appeared over 200 years ago when rickets, measles and scurvy were said to be associated with 'erosion' of the teeth. The term 'mottled enamel' was adopted by Black and McKay [6] to describe the appearance of teeth which they considered to represent an endemic form of the defect; it was not until 1931 that fluoride was identified as the causative agent of this defect [7]. The examples of the terminology that have been used in published studies to describe developmental defects of enamel are shown in Table 1 [5,6,8-20]. Some are simple descriptive clinical terms, while others are linked with the causative agent, or the histopathology of the defect. However, the majority of these terms are non-specific and frequently ambiguous. The terminology needs to be uniform to suit the requirements of the various investigators. Owing to the efforts of a working group of the Commission on Oral Health, Research and Epidemiology of the

Table 1: Examples of the terminology that have been used in some published studies to describe DDE.

Terminology	Author
Chalky enamel	Gottlieb [8]
Mottling	Black and McKay [6], Dean [9]
Chronologic enamel aplasia	Sarnat and Schour [5], [10]
Hypoplasia, hypocalcification	Weinmann [11]
Opaque hypoplasia	Hurme [12]
Fluorosed and idiopathic opacities	Zimmermann [13]
Hypoplasia, mottling, pigmentation	Hewat and Eastcott [14]
Fluorosed and non-endemic mottling	Jackson [15]
Internal and external hypoplasia	Andreasen and Ravn [16]
Opacities, hypoplasia	Young [17]
Opacities, pits, grooves	Suckling [18]
Cheese molar	Weerheijm [19]
Molar incisor hypomineralization (MIH)	Weerheijm [20]

International Dental Federation (FDI), a standardized terminology, which accompanies the FDI (DDE) Index, has been established [21,22]. Based on the quality and quantity of affected enamel, DDE can be classified into three main types: demarcated opacities diffuse opacities and hypoplasia [22].

Pathology of DDE

The stage of amelogenesis at which time the dysfunction occurs, the severity of the insult leading to temporary, or permanent inactivity of the cells, the duration of the insult, the phase of ameloblast activity during the relevant period, and the specific agent involved, may affect the final appearance of the defect [23,24]. For example, damage of secretory ameloblasts results in pathologically thin enamel. However, interference during the maturation stage can lead to defects which present as bands, or patches of chalky opaque porous enamel [25]. This is due to failure of the degradation and removal of some amino acid fractions; therefore, relatively large residues of organic material remain between the crystals. The presence of an organic 'contaminant' can retard or arrest further growth of the apatite crystals that are already present [26,27]. Moreover, Hall [28] claimed that some ameloblasts might subsequently recover from the insult during the maturation phase, which may account for the varying colour and degree of mineralization of the opacities that are known to occur. Different agents might initiate common pathways which lead to enamel defects with a similar appearance. Furthermore, based upon work on sheep, Suckling [23] suggested that the pathogenesis of each type of DDE was different and therefore should be considered separately.

Pathology of demarcated opacities

The demarcated opacity is a defect which involves an alteration in the translucency and has a distinct and clear boundary with the adjacent normal enamel, Figure 1. The colour can be white, cream, yellow or brown. Demarcated opacities have been produced experimentally in sheep. This defect followed trauma to cells in either the early or late maturation phase [29,30], and after parasitic infection in the secretory or early maturation phases [31]. Suckling claimed that yellow demarcated opacities resulted from an insult causing death of the ameloblasts early in their maturation stage while white demarcated opacities were found in sheep incisors following

disturbances in the secretory, plus the early- and the late-maturation phases. Suckling also claimed that the yellow demarcated opacities that she induced often had a white opaque margin, which had a higher hardness value than normal enamel. This leads to the assumption that some maturation cells as well as secretory cells have the ability to recover [32].

However, work on the teeth of humans and monkeys by Suga [33], suggested that ameloblasts were very sensitive to disorders at an early stage of maturation. Hence, if a cell is damaged by a systemic or local disorder at this stage, it cannot easily recover from dysfunction during the long period of maturation. Therefore, he hypothesized that demarcated opacities were due to a disturbance in the process of matrix degradation, which originally occurs during the matrix formation stage, to provide suitable physiochemical conditions for the commencement of maturation.

In respect of the severity and duration of the insult, Suckling [23] presumed that demarcated opacities were caused by a less severe, but longer lasting disturbance than that responsible for causing hypoplasia. Birgitta and Jörgen [24] observed that the homologous teeth of children with demarcated opacities were affected to varying extents. However, they found it difficult to make any assumption about the severity of the insult because the damaging agent seemed to have been rather nonspecific in most of the children. Hence, they assumed that two or more interacting factors were required to produce the defects [24].

Pathology of diffuse opacities

Diffuse opacities also involve alterations in the translucency of enamel and are white in colour, Figure 2. They can have a linear, patchy or confluent distribution but there is no clear boundary with the adjacent normal enamel. Histologically diffuse opacities are subsurface-hypo mineralized defects covered by a well-mineralized outer enamel surface. It is believed that these defects result from a long, continuous low-grade insult. They have been produced in sheep by a daily low dose of fluoride for a period of six months [34]. Diffuse opacities have been associated with an arrest in enamel maturation characterized by delayed breakdown of amelogenins [35], which may become entrapped in the detective enamel. Moreover, data from a

Figure 1: Cream demarcated opacities on the upper left central incisor.

Figure 2: Diffuse opacities.

Figure 3: Hypoplasia on the incisal edges of the four incisors.

sheep study showed that the defects increased in severity when fluoride was also given during the secretory phase [34]. However, if the insult was confined to the secretory phase, then normal maturation occurred, resulting in translucent enamel even if the matrix was abnormal [36].

The effect of increasing the intake of fluoride during tooth development upon the appearance of the enamel has been well-documented. Among the many hypotheses that have been proposed for the mechanism by which excess fluoride affects degradation and removal of enamel matrix proteins, three are favored by most researchers: (i) fluoride might directly affect ameloblasts [37]; (ii) proteins may be more tightly bound to fluoridated hydroxyapatite and, thus, proteinolysis might be more difficult [38]; and (iii) fluoride might inhibit enamel proteinases [39]. However, there is no conclusive proof that fluoride alone, either in the form of an excessive intake, or of an abnormal metabolic process in the presence of a low or normal intake, is responsible for all the enamel changes of diffuse opacities. Other systemic factors operating over a long period of tooth development such as malnutrition [40], chronic illness (diabetes insipidus) [41], or high altitude [42] may mimic or augment the effects of fluoride. If the defects are true fluorosis, then it is an apt reminder that biological factors can enhance cellular responses to low concentrations of fluoride in the environment [23].

Pathology of hypoplasia

Hypoplasia is a defect associated with a reduced quantity and hence thickness of enamel, Figure 3. Sarnat and Schour [5,10] believed that the morphologic unit of enamel hypoplasia was the enamel pit, which resulted from a cessation of ameloblastic activity. In a clinical investigation of anxious patients, whose childhood medical histories were available, it was concluded that a narrow zone of defect indicated a disturbance of short duration or an acute disease, while a wide zone indicated a disturbance of long duration or chronic disease [5,10].

More recently enamel hypoplasia has been produced in sheep by physical trauma [29,30], by systemic illness induced by intestinal parasites [31,43], and by a daily high dose of fluoride for a short period [44,45]. These findings indicate that hypoplastic defects are formed during the secretory phase of amelogenesis. The duration

of the insult is relatively short, and it is the severity that determines the extent of the defect and the translucency of the partially formed enamel. The aetiology does not seem to be of major importance, since local and systemic factors result in defects with a similar appearance and physical characteristics.

The chronological course of tooth development and the occurrence of DDE

The formation of the crowns of the 28 permanent teeth commences at around birth and is completed at around age 8 years [46]. Only dysfunction of ameloblasts during this period of time may result in the occurrence of DDE. Furthermore, enamel, once formed, has limited capacity for alteration. Therefore, enamel can serve as a kymograph which permits the dating of events that occurred during amelogenesis with a high degree of accuracy [5,10].

Location of DDE

The location of DDE depends on the stage of enamel production and on the time of the insult or injury to the ameloblasts. As enamel production commences at varying times in different tooth types, so the location of DDE will vary in different homologous pairs of teeth. All the teeth at the same stage of development may be affected, with homologous pairs of teeth having similar types of DDE in similar locations. This type of distribution is referred to as generalized DDE and may be caused by systemic factors. When only one or several adjacent teeth exhibit the same type of DDE, the defect causing event is probably localized.

Aetiology of DDE

Developmental defects of enamel with a similar appearance are not necessarily caused by similar aetiological agents. Conversely, the same aetiological factors can produce different defects at different stages of tooth development. Enamel defects may also result from a combination of factors. It has been proposed that there are well over 90 different factors that may be responsible for causing developmental defects of enamel [47,48]. Most of the available data on the aetiology of enamel defects have been gained from animal studies and case reports of children with systemic disorders; however, sound evidence for their involvement are equivocal. Only a few of these factors have been confirmed as being directly responsible for causing developmental defects. The possible aetiological factors for DDE in permanent teeth can be broadly divided into two main categories: those with a localized distribution and those with a generalized distribution.

Possible aetiological factors for DDE with a localized distribution

When one, or several, adjacent teeth in a horizontal, or vertical relationship exhibit enamel defect, the defect causing event was probably of a localized, rather than a generalized nature [49]. The localized disturbance, experienced by the individual, affected only a small group of ameloblasts. Amongst the causative agents of localized defects of enamel are trauma, localized infection and irradiation.

Trauma

Trauma in the form of a fall, or a blow to a primary tooth, causing intrusion or lateral luxation, are probably the most common causes of localized enamel defects in the succedaneous teeth [50-53]. The damage might happen either at the time of injury, by direct impact

of the root apex of the primary incisor on the developing permanent tooth, or at a later stage, as a consequence of post-traumatic complications. If the trauma to primary incisors leads to pulp necrosis then there is a greater likelihood of enamel defects occurring in the permanent successors [54].

Regrettably, another cause of trauma is surgery. In an Australian study, Williamson [55] demonstrated that exodontias could be a factor in the production of enamel defects in developing premolars. Children with cleft palates have significantly higher prevalence of enamel defects in primary as well as in permanent teeth [56]. These defects predominantly occur adjacent to the site of the surgical repair of the lip and palate. Therefore, the defects may have resulted from trauma during surgery [56,57]. However, the effect of other postnatal environmental factors such as nutrition and infection should not be excluded [57].

Localized infection

In a cohort of Chinese children, higher prevalence's of demarcated opacities and hypoplasia were found in permanent teeth whose primary predecessor teeth had experienced caries than in those

Table 2: Hereditary conditions that have been reported to be associated with DDE.

Hereditary condition	Author
Auto-immune polyendocrinopathy-candidiasis-ectodermal dystrophy	Lukinmaa [104]
Candidasisendocrinopathy syndrome	Porter [105]
Cleidocranaldysostosis	Fukuta [106]
Coeliac disease	Aine [90,91]
Congenital adrenal hyperplasia	Hallett and Hall [107]
Congenital contractual arachnodactyly	Ayers and Drummond [108]
Congenital unilateral facial hypoplasia	Gibbard and Winter [109]
Ectodermal dysplasias	Sastry [110]
Ehlers-Danlos syndrome	Letournean [111]
Epidermolysisbullosa	Wright [85]
Focal dermal hypoplasia	Al-Ghamdi and Crawford [112]
Heimler's syndrome	Pollak [113]
Hypoparathyroidism	Nikiforuk and Fraser [102,103]
Ichthyosis vulgaris	Pindborg [114]
Lacrimo-ariculo-dento-digital syndrome	Hollister [115]
Morguio syndrome	Barker and Welbury [116]
Mucopolysaccharidosis	Gorlin [117]
Oculodentodigital dysplasia	Frasson [118]
Orodigitofacialdysostosis	Gorlin [117]
Prader-Willi syndrome	Jablonski [119]
Pseudohypoparathyroidism	Jensen [86]
Seckel syndrome	Seymen [120]
Tricho-dento-osseous syndrome	Spangler [87]
Tuberous sclerosis	Hoff [98]
Vitamin D resistant rickets	Nikiforuk and Fraser [102,103]
William's syndrome	Hertzberg [121]
22q11 deletion syndrome	Klingberg [122]

without caries [58]. Caries extending into the pulp of primary teeth often results in necrosis of the pulp tissue and periapical lesions. In an animal study, Valderhaug [59] found that the chronic periapical periodontitis of primary tooth could destroy the cortical plate of bone surrounding the permanent tooth germ and affect the dental organ in various ways. Inter-radicular infection of a primary tooth can, in extreme cases, cause arrest of the developing tooth germ [60]. In a study conducted by McCormick and Filostrat [61], over 25% of abscessed primary teeth were associated with enamel defects, which varied from opacities to hypoplasia, in the successional permanent teeth. This phenomenon has also been observed by other researchers [62-66].

Irradiation

Since anti-neoplastic therapy affects all cells, it is not surprising that developmental defects of enamel have been documented after oncology therapy [67-72]. In the study conducted by Pajari and Lanning [73], it was found that the scattered irradiation, of 0.72-1.44Gy, to the dental arches during central nervous system irradiation caused an increase in the number of enamel opacities in children with acute lymphoblastic leukemia. In another study, Duggal and his colleagues [74] reported a significantly higher prevalence of all types of enamel defects and fewer teeth without enamel defects in the long term survivors of childhood cancer as compared with their siblings.

Possible aetiological factors for DDE with a generalized distribution

Developmental defects with generalized type of distribution may be caused by genetic disorders, or by environmental factors.

Genetic disorders

Amelogenesis Imperfecta (AI) has been defined as a heterogeneous group of genetic disorders affecting the enamel of the teeth by causing various degrees of hypoplasia, hypo mineralization or, a combination of the two [75,76]. The condition often results from a single gene defect, either as an X-linked, autosomal dominant or autosomal recessive trait [77-79]. Several classifications have evolved since 1945, based on the phenotype and mode of inheritance [75,80-83]. Nevertheless, some authors consider that AI may be present as part of hereditarily determined syndrome complexes, such as epidermolysis bullosa [84,85], pseudohypoparathyroidism [86], and tricho-dento-osseous syndrome [87]. Genetically determined enamel defects are less common than those resulting from acquired causes; for example, AI has been estimated to vary in the general population from about 1:14,000 [88] to less than 1:800 [89].

Some genetically-determined syndromes and disorders, as well as some acquired diseases, are frequently accompanied by enamel defects. The Finnish researcher Aine [90,91] found that more than 90% of patients with coeliac disease had enamel defects. She also claimed that the so-called coeliac-type enamel defect, with associated gastrointestinal symptoms, could lead to the diagnosis of coeliac disease [92]. Confirming Aine's results, Farmakis [93] and colleagues and Avşar and colleagues [94] also found higher percentage of enamel defects among celiac disease patients compared with the healthy controls. Other researchers have, however, failed to find any association between enamel defects and coeliac disease [95-97]. In tuberous sclerosis, pit-shaped enamel defects were said

to be pathogenomonic of the syndrome [98]; however, this has been questioned [99,100] because other researchers have found only a few cases of enamel pitting among tuberous sclerosis families [101]. By studying children with hereditary vitamin D resistant rickets, X-linked hypophosphatemia and hypoparathyroidism, it has been hypothesized that the enamel hypoplasia was caused by hypocalcemia. This observation led to formulation of a unifying hypothesis that enamel hypoplasias are caused specifically by hypocalcemia [102,103]. Other inherited disorders which have been reported, in the literature, to possibly be accompanied by enamel defects are shown in Table 2 [85-87,90,91,98,102-122].

Environmental factors

A considerable number of environmental factors have been reported to be capable of causing enamel defects. These systemic disturbances consist of intoxications, perinatal and postnatal problems, malnutrition, infectious diseases and a range of other medical conditions.

Intoxications

In the group of intoxications, the one responsible for affecting most people is probably fluoride. The relationship between DDE and fluoride consumption has been established for over 80 years and is well documented [7,9]. The prevalence of enamel defects increases with increasing levels of fluoride in the drinking water [123]. Fluorotic defects can vary from minor white striations to small or extensive areas of opaque enamel [124,125]. However, fluorotic defects do not have unique characteristics which allow them to be differentiated from defects caused by other factors [126,127]. The level of strontium in the drinking water has been shown to be associated with the frequency and severity of enamel mottling similar to that of fluorosis [128]. It seems that, the less severe the defects, the greater the problem of positive diagnosis for fluorosis [129,130].

Recently, Finnish studies have focused on dioxins as being a causative agent for developmental defects of permanent first molars [131,132]. Prolonged breast-feeding might increase mineralization defects in teeth because of environmental contaminants such as dioxins or dioxin-like compounds in breast milk [131,132] interfering with enamel maturation [133]. However, a Swedish study which identified similar levels of dioxins in breast milk failed to reproduce the findings [134]. Wozniak [135] reported that elevated levels of chemical compounds, e.g. fluorine, ammonia and sulphur, in the atmosphere increased the incidence of diffuse white/creamy mottling and lines in the 14 to 15 years old schoolchildren. Other intoxications include hypervitaminosis D [114], chronic lead poisoning [136], diphosphonate [137], and polychlorinated biphenyl poisoning [138].

Perinatal and postnatal problems

Improvements in the medical care of neonates and infants have improved the survival rates of low birth-weight and premature babies. However, they are prone to suffer from many serious illnesses, which in turn may cause the development of enamel defects through a central metabolic mechanism [139,140]. Children with low birth-weights, i.e. 2000g or below, have also been shown to have a much higher prevalence of enamel opacities in the first permanent molars and lateral incisors than children who had a normal birth-weight [141].

Medical problems at the time of delivery such as breech presentation, caesarean section and labour in excess of 20 hours, as well as a poor respiratory response in the postnatal period have been said to be associated with enamel hypoplasia of the primary dentition [142]. In a Dutch study of potential aetiological factors responsible for mineralization defects of permanent first molars, the authors noted a high frequency of medical problems at the time of delivery and childhood respiratory diseases [143]. According to their findings, it was suggested that oxygen deprivation influenced the mineralization of enamel matrix. However, Beentjes and his co-workers [144] found that there was no relation between enamel defects and complications during pregnancy and birth.

Malnutrition

Rugg-Gunn and his co-workers [40] reported that boys of 14 years classed as malnourished, by height for age percentage of the median of the reference population, had a higher prevalence of enamel defects than those classed as well-nourished. Moreover, rickets due to vitamin D deficiency has been reported as a cause of enamel hypoplasia [10,145,146]. Animal experiments have also demonstrated that a lack of vitamins A and D may cause enamel hypoplasia [147,148]. However, nutrition was not observed to be a significant aetiological factor in well-nourished New Zealand children [149]. Which may be why it was proposed that nutrition only became important when the level of malnutrition was reasonably high [40].

Infectious diseases and other medical conditions

Infectious diseases during early childhood, such as chicken-pox, measles, mumps, scarlet fever [150], tuberculosis [100], pneumonia, diphtheria, whooping cough [151], otitis media [152], and bulbar polio with encephalitis [153], have long been blamed for the presence of enamel defects. A number of other medical conditions such as gastro-intestinal disturbances [154], cyanotic congenital heart disease [155], neurological disorders [156], and renal disorder [157] have also been implicated as being aetiological agents. However, except for chicken-pox, Suckling and her co-workers [52] failed to find positive and strong associations between enamel defects and children experiencing one or more of these diseases in spite of extensive statistical testing.

Jackson [15] considered that the exanthematous fevers were a major cause of non-endemic mottling of the permanent first molars but only a contributory factor in other teeth. However, Wilson and Cleaton-Jones [158] found no association between the presence of enamel mottling and the occurrence of childhood fever in subjects from a fluoride-deficient area. In the study conducted by Suckling and Pearc [149], trauma and 'serious illness' were the only two aetiological factors which were statistically significant in a cohort of 243 children. 'Serious illness' in their study included urinary tract infections, convulsions and pneumonia. In one Australian study [159], infection was reported to be related to demarcated enamel defects. Swedish researchers suggested that respiratory diseases, especially asthma, were possible aetiological factors responsible for demarcated opacities in first molars; however, only four of the 516 subjects suffered from asthma [134]. The limited studies that involved human populations were predominantly retrospective; moreover, their conclusions can be criticized because only a small number of

subjects had developmental defects with an associated aetiology. Hence, the lack of robust data makes the results of these studies inconclusive.

One notable exception involved a group of New Zealand children who participated in the Dunedin Multidisciplinary Child Developmental Study [160]. This longitudinal study correlated the medical and dental histories of 696 children born in 1972 with the presences of enamel defects using the FDI (DDE) Index [21]. It was found that, apart from exposure to fluoride, chicken-pox before the age of 3 years and trauma to the primary teeth were the only other significant factors. However, the severity of systematic infections in these children is likely to have been reduced by immunization, the use of antibiotics and readily available medical facilities [52]. Nevertheless, this study demonstrates the difficulty of establishing the aetiology of enamel defects, even when medical and dental histories are available and when a study is prospective. Thus, the paucity of reliable information on the possible causal factors in the occurrence of DDE suggests a need for further investigations into the causes of the defects.

Conclusion

Because of the nature of DDE, the difficulty of methodology establishing the aetiology of enamel defects, lack of robust data, and the limitation of this paper, i.e. non-systematic review, the results of the reviewed studies are inconclusive. To obtain reliable data for aetiological factors of DDE, a cohort observational epidemiologic study in an area with high prevalence of DDE is needed. A long-term prospective study of a large group of young subjects using the same strict diagnostic criteria for DDE, and with frequent updates to health-related events in their early childhood, is the only realistic way to gather the necessary data on the aetiological factors of DDE.

Acknowledgement

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