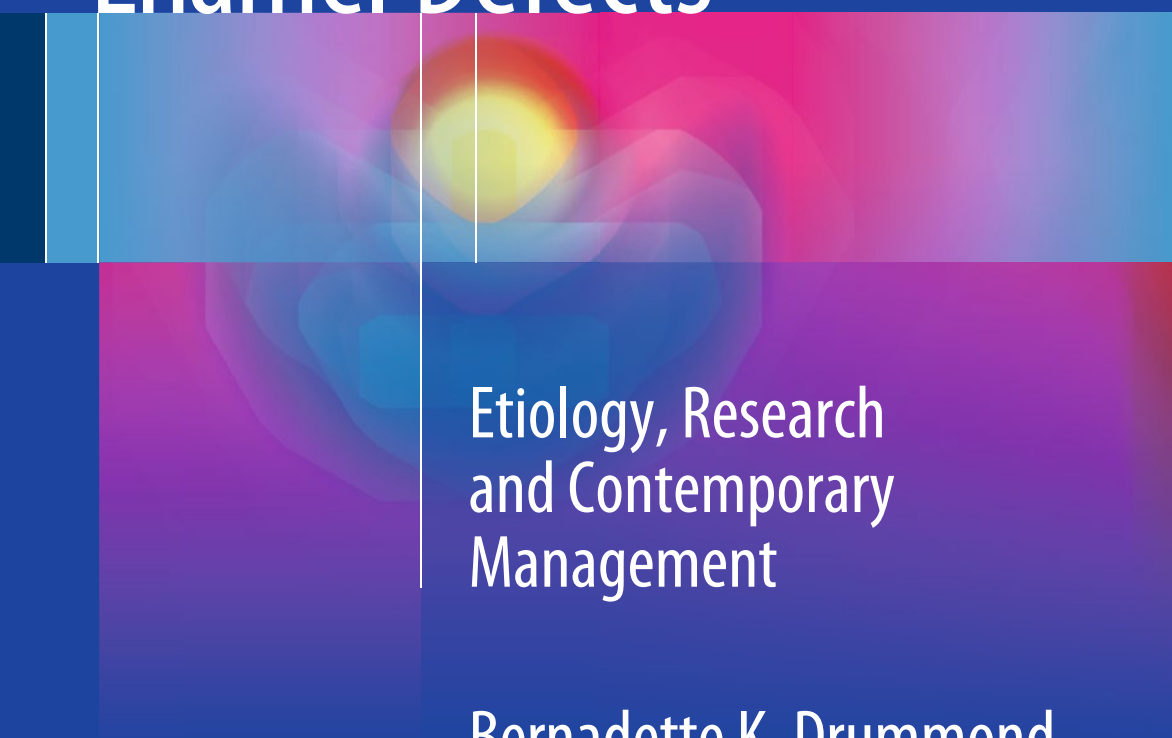
Planning and Care for Children and Adolescents with Dental Enamel Defects



Etiology, Research and Contemporary Management

Bernadette K. Drummond
Nicola Kilpatrick *Editors*

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This book is dedicated to our PhD supervisors, John Murray, John McCabe and Martin Curzon with advice from Colin Robinson and John Weatherell who inspired us both to constantly question, reflect and explore our clinical experiences. Together with our many colleagues, they have encouraged us to strive for excellence in both the management of and outcomes for children, adolescents and their families.

Preface

The prevalence of developmental defects of enamel (DDE) is reportedly increasing worldwide. It is difficult to assess this accurately, as in the past the greater prevalence of dental caries may have had a significant impact on the diagnosis of DDE by masking its presence. Clinically, defects vary greatly in their appearance in terms of size, color, and shape. DDE may affect both primary and permanent dentitions and can be either generalized across one or both dentitions or localized to specific teeth. The structure of enamel in affected teeth differs, such that there may be a quantitative reduction in enamel (known as hypoplasia) or a qualitative defect (known as hypomineralization), though often defects comprise a combination of the two. Despite the growing burden associated with treating affected individuals, there remains a lack of understanding of the etiology of DDE and evidence of clinical outcomes to support management.

When clinicians are faced with a child who has hypomineralized and/or hypoplastic teeth, it can be difficult to plan care for many reasons. Often, the child is young and has had little or no experience of dentistry. Apart from the difficulties for the child, this creates stress for the parents and puts pressure on clinicians trying to decide on the best way to manage the young patient. Affected teeth are difficult to anesthetize, and treatment can be uncomfortable. It is also clear that individuals with teeth affected by DDE experience significantly more restorative procedures throughout childhood and adolescence and have higher levels of dental anxiety. Poor esthetics coupled with increased sensitivity may further exacerbate the child's anxiety and have a real impact on their quality of life. Parents report that children eat slowly or refuse some foods, although they often do not complain directly of pain. Children note that they cannot eat ice cream or that people, including teachers and peers, comment on their chalky or discolored anterior teeth.

The protein content in hypomineralized enamel is significantly higher than in otherwise healthy enamel, which in turn alters its structure and interferes with the adhesion of conventional resin-based dental restorative materials. When a clinician looks at affected teeth for the first time, they should be aware that the child is likely to require a lifetime of care, even if the teeth remain caries free. This can be an overwhelming prospect when considering the best approach to early management so as to prepare the individual for the longer-term definitive solutions, which often cannot be finally decided until the child is in late adolescence or early adulthood. When severe defects are localized to one or only a few posterior teeth, it may be

appropriate to extract these compromised teeth. However, it is important to consider the impact of such extractions on the developing occlusion in order to optimize definitive occlusal outcomes. Finally, there are financial considerations to be taken into account both immediately and in the long term, which also will impact on the management choices that families will be able to make.

With all this in mind, the aims of this book are to summarize the current understanding of DDE in the primary and permanent dentitions, to discuss its impact on children and adolescents, and to provide guidance on treatment planning and management. The prevalence and etiology of DDE are addressed in Chaps. 1, 2, and 3. Chapters 4 and 5 review the contemporary understanding of the genetic influences on DDE and the potential associations of DDE with systemic conditions and syndromes. Dental professionals can play an important role in the diagnosis of systemic conditions if they record patterns of defects and take careful histories of other related health signs and symptoms. Chapter 6 is devoted to presenting information on the structure and composition of defective enamel. This is to encourage clinicians to think about how affected teeth behave in the oral environment and consider the impact of this on restorative materials and treatment techniques. Affected teeth pose particular challenges in relation to resin bonding, and clinicians have to consider where to place restoration margins and whether/how to pretreat the enamel to improve adhesion.

Chapter 7 summarizes the current knowledge of the impact of DDE from the patient's perspective. There is emerging awareness of the impact of DDE on children's and adolescents' quality of life not only in terms of the psychosocial influences of altered appearance but also as a result of a young person's experience of complex and often repeated dental interventions. This chapter highlights the importance of giving careful consideration to planning not only the obvious clinical treatment needs but also the longer-term treatment needs in the context of the broader psychosocial demands and individual/family expectations.

In planning a definitive outcome, clinicians have to consider the implications of growth on the developing occlusion, particularly where teeth have a very poor prognosis and extractions are being considered. Chapter 8 reviews dental and occlusal development. While specialist orthodontic input is ideal, this chapter also acknowledges that this is not always possible and gives an overview of the aspects of occlusion that should be considered when making decisions surrounding the choice and timing of extractions. The first chapter looking at the management of DDE (Chap. 9) is devoted to managing sensitivity, improving enamel mineralization, and preventing dental caries and erosion in affected teeth. Two Chaps. (10 and 11) address the immediate and intermediate restorative management of primary and permanent teeth with DDE. This includes the management of permanent teeth through adolescence, while planning the permanent restoration options. All these chapters give options and recommendations that are supported, where available, with contemporary evidence. The final chapter (Chap. 12) offers an interesting glimpse into cutting-edge research, in which techniques to regenerate enamel and develop enamel–like restorative materials offer hope for the future. As access to research reporting becomes increasingly available to all clinicians, this chapter is a guide on what to continue to search for in the future.

This book is intended to provide contemporary information on both DDE and its impact during childhood. Each of the chapters covering specific aspects of DDE has been written to stand alone. However, where appropriate, the reader is directed to further linked information in other chapters. Although guidance is offered on various management options from both preventive and restorative perspectives, it is not intended to suggest that these are the only options. Clinicians are encouraged to use the information to consider the best pathway for each individual patient after taking account of the child's and family's perspectives, the developing occlusion, the clinician's own skills, and the severity of the presenting anomaly.

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Dental Enamel Defects in the Primary Dentition: Prevalence and Etiology

1

W. Kim Seow

Abstract

The enamel of the primary dentition undergoes development from approximately the 13th week of gestation to around 3 years of age when the second primary molars erupt into the oral cavity. During this period, the developing primary dentition can be affected by many systemic and local environment insults that lead to changes in the quality and quantity of the enamel. The systemic influences that can cause abnormalities in the primary enamel range from pregnancy conditions such as pre-eclampsia to neonatal disruptions such as preterm births and postnatal infections such as rubella and chickenpox. The prevalence of enamel defects has been reported to range from approximately 30 % in the general population in USA, Britain, and Australia to over 70 % in preterm children and indigenous populations worldwide. The high prevalence of enamel hypoplasia reflects the vulnerability of developing teeth to environmental changes and suggests that complications of enamel defects, such as increased caries susceptibility, are common in the primary dentition.

The formation of enamel extends over a considerable period of time in the life of an individual. In the primary dentition, enamel formation commences at the tips of the incisors at approximately 15–19 weeks in utero and ends with the emergence of the second primary molars between 25 and 33 months of age [1]. During this long period of enamel development, many conditions can adversely affect the enamel-forming cells causing abnormalities to be produced. As enamel does not remodel, any aberrations occurring during formation may be permanently recorded on the surface as visible defects. Such developmental defects of enamel have major clinical significance. Some defects in the anterior teeth can affect aesthetics, cause severe tooth sensitivity, and impair masticatory function [2]. Importantly, enamel

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defects in the primary dentition are now increasingly recognized as a major risk factor for early childhood caries (ECC) [3].

Presentations of Developmental Defects of Enamel (DDE)

The formation of enamel or amelogenesis begins during the late bell stage of tooth development when specialized enamel-forming cells, the ameloblasts, are differentiated from the inner enamel epithelium [4]. The initial stages of enamel formation are characterized by the secretion of specific proteins by the ameloblasts such as amelogenin and ameloblastin to form an enamel matrix which is later mineralized [5, 6]. After the laying down of the enamel matrix, the ameloblasts regulate the removal of water and proteins from the enamel matrix and promote the ingress of minerals [7]. The cellular and biochemical events that occur during amelogenesis are complex and can be adversely affected by genetic changes as well as by systemic and local environmental conditions.

Abnormalities that originate during the formation of enamel are commonly referred to as developmental defects of enamel (DDE) [8]. The presentation and severity of DDE are dependent on the stage of enamel development at the time of the insult, as well as the extent and duration of the adverse condition [9]. Quantitative deficiencies of DDE, which usually arise from disruptions of matrix formation, are known as enamel hypoplasia and may be expressed as pits, grooves, and thin or missing enamel [8]. In contrast, qualitative enamel deficiencies are usually associated with altered enamel mineralization and may be expressed as changes in the translucency or opacity of the enamel that may be diffuse or demarcated, and colored white, yellow, or brown [8]. Generally, it is thought that the more immature the stage of enamel formation, the more vulnerable to damage. Disturbances which occur during the secretory stages of enamel formation are generally thought to reduce the quantity of enamel formed, and this is usually expressed as enamel hypoplasia. In contrast, disturbances at the final stages are usually associated with altered mineralization of the enamel which manifests clinically as opacities. As the various primary teeth in a child's mouth may be at different stages of enamel formation at the time of an adverse condition, a spectrum of DDE, ranging from mild opacities to severe enamel hypoplasia, can result from a single insult.

As DDE are permanent records of enamel changes, the location of a defect on the enamel surface can suggest the timing of the events that disrupted enamel formation. Although exact timing is often difficult to identify, knowledge of the chronology of development of the primary tooth crowns may be useful in estimating the approximate times of the insults (Table 1.1). Table 1.1 shows that the primary teeth usually commence mineralization in utero, starting with primary central incisors at approximately 15–19 weeks, canines at 16–22 weeks, first molars at 19–22 weeks, and second molars at 20–22 weeks [1]. At birth, the amount of enamel formed is approximately three-quarters, two-thirds, and one-third of the primary central incisor, lateral incisor, and canine crowns respectively. In addition, at birth, the first molar cusps are usually formed, but not coalesced, and the second molar cusp tips are commencing initial mineralization. Table 1.1 also shows that crown formation is

Table 1.1 Chronology of tooth crown formation of the primary dentition

Primary tooth	Tooth formation commences	Amount of crown formed at birth	Crown completed
Incisors	15–19 weeks in utero	Central incisors: three-quarters Lateral incisors: two-thirds	1.5–3 months
First molar	16–22 weeks in utero	Cusps formed, not coalesced	5.5–6 months
Canine	19–22 weeks in utero	One-third of cusp	9–10 months
Second molar	20–22 weeks in utero	Tips of cusps	10–11 months

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complete in the primary incisors by 3 months, the first molars by 6 months, the canines by 10 months, and the second primary molars by 12 months. After complete crown formation, the newly formed enamel undergoes a process of maturation and hardening that continues post-eruption [4].

The enamel formed prenatally may be demarcated from that formed postnatally by an area of altered enamel known as the neonatal line. This band of abnormal enamel has a disorganized prism alignment and contains more organic material compared to the adjacent enamel [10]. As the entire primary dentition commences calcification before birth, the neonatal line is usually present in all primary teeth [11]. It may widen to a clinically visible area of abnormal enamel if a child experiences adverse neonatal conditions such as fetal distress and difficult birth delivery. Many DDE associated with perinatal illnesses are thus located at the neonatal line.

Prevalence

Several indices have been utilized to record DDE, the most popular being a descriptive index, the Developmental Defects of Enamel (DDE) index which does not attempt to identify the etiology [8]. There have been several modifications of this index, including simplified versions that can be applied for screening purposes. Many authors use the simplified modifications of the index to record DDE in the primary dentition for practical reasons [8]. Variations in prevalence of DDE reported in different populations are likely to be the result of differing criteria being used to define enamel defects. In addition, there are population variations resulting from differences in general health, and background fluoride exposure levels may also contribute to differences in the reported prevalence of DDE. Furthermore, in many studies, the recording of DDE in primary teeth several years post-eruption may be complicated by difficulties in diagnosing DDE once the lesions have become carious or have been restored [12]. Finally, differing field conditions during examination for DDE, such as variations in lighting and whether the teeth are dried prior to recording of the defects, may also contribute to differences in reported prevalence.

As there are few high-quality studies, the true prevalence of DDE in the primary dentition is unclear. Most prevalence studies on the primary dentition have been based on convenient samples from the general or special population groups such as medically compromised children. Table 1.2 lists selected reports of prevalence of DDE in the primary dentition published since 1996. There are significant variations