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Review article

Enamel Hypoplasia and Dental Caries in Children: A Review Study

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Abstract
Enamel hypoplasia and dental caries significantly impact children's oral health, with enamel defects increasing susceptibility to bacterial colonization and decay. This review explores the prevalence, causes, and consequences of enamel hypoplasia, particularly molar-incisor hypomineralization (MIH), emphasizing its association with systemic, genetic, and environmental factors. Dental caries remains a major public health concern, especially in low- and middle-income countries, where inadequate fluoride exposure, poor oral hygiene, and socio-economic disparities exacerbate the condition. Given the strong correlation between enamel hypoplasia and caries, early detection, preventive measures, and appropriate clinical management are essential. Public health interventions, policy development, and further research are crucial in mitigating the burden of these conditions and improving children's oral health outcomes. Accordingly, the main aim of this review is to review recent findings on enamel hypoplasia and dental caries in children, highlighting their prevalence, causes, consequences, and the need for early detection, prevention, and public health interventions.
Keywords. Enamel Hypoplasia, Dental Caries, Hypomineralization, Oral Health.

Introduction

Disturbances in hard tissue matrices and their mineralization that occur during odontogenesis are known as developmental enamel defects (DDE) [1]. The intricate process of enamel morphogenesis begins with the secretion of the enamel matrix protein and progresses through mineralization and maturation. The procedure starts at the crowns' cusps and incisal portions and moves on to the teeth's cervical regions. A variety of macroscopic and structural alterations may arise from disturbances in the various phases of enamel formation [2].

Hypoplasia is a quantitative defect caused by defective enamel matrix development that manifests as decreased enamel thickness. When a normally produced organic enamel matrix undergoes deficient calcification, the result is enamel that is qualitatively defective hypomineralization (MIH). Clinically, MIH enamel has normal thickness but altered translucency, seen as defined opacities or diffuse white, yellow, or brown opacities. Whereas delimited opacities have visible borders with the nearby normal enamel, diffuse opacities are dispersed over the enamel surface without a noticeable border [3-6].

Conversely, cavities, also referred to as dental caries, are a prevalent issue among kids. Globally, the age-standardized prevalence of untreated dental caries is roughly 29% for permanent teeth and 8% for deciduous teeth [7]. These days, it is widely acknowledged that chronic illnesses are closely related to poor tooth health. Situations involving severe systemic ailments and oral diseases share numerous similarities [8]. Consequently, common risk factors affect the mouth, the body's first line of defense. There are notable regional differences that have important public health implications, such as those among South Asians and in India [9,10]. Numerous genetic, systemic, environmental, and local variables are among the potential causes of DDE [2-4,11].

There is a recognizable pattern to dental caries in newborns and toddlers. Very young children who suffer dental caries have been referred to by a variety of names and terms [12]. Initially, this illness was defined in terms of its genesis, emphasizing the improper application of nursing procedures. The terms listed below can be used interchangeably: Early childhood dental decay, comforter caries, nursing caries, maxillary anterior caries, rampant caries, infant bottle-fed tooth decay, early childhood tooth decay, and many more [13,14]. The causes of dental caries in preschool-aged children are indicated by some of these phrases [14].

Methods and data sources

Understanding the most recent findings on enamel hypoplasia and dental caries in children requires reviewing earlier research on the topic of the current study. The use of systematic methodologies was necessary to guarantee a thorough and trustworthy review. We looked through the following electronic databases: Embase, Web of Science Core Collection, Google Scholar, PubMed, and Scopus with the following keywords: Oral health; Dental caries, Enamel; Developmental defects of enamel and hypoplasia.

The bibliographies of relevant articles were manually reviewed to locate additional papers not in the electronic databases. The following criteria for inclusion were used to screen studies: Research with a case control, cross-sectional, or cohort design, the investigators were able to assess whether there were any time

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trends in the occurrence of DDEs by using a larger search scale for articles published between 1980 and 2025. Additionally, studies involving human subjects were included. Lastly, studies involving animal subjects were eliminated.

Findings from investigated articles

Enamel hypoplasia in children

In children, enamel hypoplasia develops in the primary canines from the neonatal to infantile stage and in the fetal stage to the early period of birth or six months after birth for molars of the second primary. In many instances, it starts at the matrix generation stage. There has been evidence linking the development of hypoplasia to severe hyperemesis gravidarum, anemia, attempted threatening abortion, and medications used to manage these symptoms [15]. Additionally, although many children also develop enamel hypoplasias as a result of sickness during infancy, most frequently from infections, metabolic imbalances, premature delivery, or starvation, primary enamel defects may be inherited [16]. It shows up as a lack of enamel thickness and is quantitative [17].

In clinical settings, enamel abnormalities may manifest as uneven and retentive surfaces, which raises the possibility of bacterial adherence and colonization [18, 19, 20, 21]. DDE may, therefore, put young children at risk for early childhood caries (ECC) and severe early childhood caries (S-ECC) [22, 23]. Furthermore, enamel hypoplasia is highly prevalent in other indigenous children with ECC, according to the findings of earlier investigations [24]. The ability to detect these DDE before they develop caries is crucial since a significant percentage of indigenous and Alaska Native (AI) children evaluated at Indian Health Service (IHS) dental clinics present with caries, possibly as a result of developmental abnormalities.

With almost 840 million prevalent and incident cases in 2015–2016, the global prevalence of enamel defects (EDs), also known as molar incisor MIH (EDs of the first permanent molars and incisors), has been estimated to be 13% [25]. Hypomineralized EDs of the primary molars are present in 0% to 41% of people worldwide, with a 7% pooled prevalence at the individual level and 0% to 30% at the tooth level, with a 4% pooled prevalence [26].

Conversely, compared to enamel defects linked to opacities, enamel hypoplasia resulting from enamel defects has a higher propensity to dental caries. According to clinical characteristics, MIH, another enamel defect (qualitative defect) that arises during the development period, can range from post-eruptive disintegration to the mild form (opacity). It might be challenging to distinguish between post-eruptive enamel degradation and enamel hypoplasia in these situations. Oral health may be significantly impacted by either type of enamel deficiency (ED) [27, 28].

Therefore, both temporary and permanent teeth as well as dentitions, may have enamel abnormalities. Teeth become more sensitive due to surface defects, such as enamel hypoplasia and MIH with post-eruptive enamel breakdown, which affect patients' daily routines and quality of life [29]. These defects also increase the risk of caries and increase pain.

In addition to systemic and environmental factors like fluoride intake, medications, nutritional deficiencies, prenatal infections, chicken pox or other early childhood diseases, and low vitamin D serum levels in both mother and child, genetic and hereditary factors like amelogenesis imperfecta may also be involved [28, 30–37]. According to studies, maxillary incisors were the primary teeth most impacted by hypoplasia, and low-birth-weight preterm children had a higher prevalence of hypoplasia than normal-birth-weight controls [37].

It is important to note that enamel MIH is a qualitative defect that involves diffuse, demarcated, or white, yellow, or brown discoloration that gets worse as the color darkens, while enamel hypoplasia is a quantitative defect that is characterized by enamel deficiency [38, 39]. When anomalies arise during the secretory and maturation phases, respectively, enamel hypoplasia and MIH result [40]. Etiologically, enamel hypoplasia can be divided into two groups: those that cause flaws that impact most or all of the teeth, and those that cause localized issues that are limited to one or a few teeth (such as trauma, infections, ankylosis, and radiation). Such extensive problems can be inherited or caused by environmental factors [41].

Dental caries in children

Dental caries is a complex and multifaceted issue brought on by an imbalance in the caries balance, which is the dynamic process of continuous mineralization and demineralization taking place on the tooth surface [42, 43]. 123 risk factors for dental caries were found in a thorough study by Kirthinga et al. that examined research from 1981 to January 2019 [44]. The physical, biological, environmental, behavioral, and lifestyle factors that increase the risk of dental caries include excessive levels of cariogenic bacteria, inadequate salivary flow, inadequate fluoride exposure, poor oral hygiene, inappropriate baby feeding practices, and poverty [45].

In low- and middle-income nations, the prevalence of dental caries is quickly increasing, with children living in disadvantaged neighborhoods being particularly affected [46, 47]. The prevalence of dental caries varies by nation in Africa. 64% Timor-leste [48], 78% Eritrea [49], 78% Tripoli Libya [50], 68.8% São Tomé Island [51], 49.7% Ghana [52], 20% Tunisea [53], 24.1% Nigeria [54], and 37.5%, 43.3% in Kenya [55, 56].

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Additionally, research in Ethiopia shows that the rates are as follows: 71.3% in the country's community survey [57], 21.8% in Bahir Dar [58], 47.4% in Addis Ababa [59], 41.5% in Gondar Town [60], and 48.5% in Finote Selam [61].

On the other hand, dental caries affects a significant percentage of schoolchildren worldwide, negatively affecting both their physical and mental health. Children who have dental caries may have trouble sleeping, eating, participating in social activities, and attending school, which can negatively impact their social development and sense of self [62, 63, 64]. Additionally, parents bear a heavy financial burden due to dental caries, especially in high-income nations where dental care is expensive. Beyond personal misery, oral health problems have an impact on overall health, increasing the risk of infective arthritis, pneumonia, and complications from diabetes [65].

Association between enamel hypoplasia and dental caries

Clinically speaking, developing enamel abnormalities significantly affect dental health by changing occlusal functions, impairing aesthetics, and increasing tooth sensitivity. Furthermore, several studies have indicated that some forms of DED may be significant risk factors for dental caries and erosions of the hard tissues of the teeth [67, 68]. Because the uneven, retentive surfaces of enamel hypoplasia and delineated opacities cause plaque accumulation and increased acid solubility of the afflicted enamel, respectively, it has been often observed that these conditions increase the experience of dental caries [66-68]. Enamel hypoplasia has clinical importance concerning malocclusion, increased risk of dental cavities, poor aesthetics, and tooth sensitivity. As a result, enamel protection as well as efficient preventive care and monitoring are necessary [69, 70].

The age difference between the eight-year-olds and the fifteen-year-olds, according to a study by Clarkson and O'Mullane [4], gave rise to a sample size big enough to identify patterns in the incidence of anomalies on both early and late emerging teeth. Jindal also discovered that enamel developmental defects were significantly more common in children with impairments. This illustrates the connection between tooth formation and numerous systemic illnesses [71]. A study in Himachal revealed that enamel developmental abnormalities were common in both primary and permanent teeth [72]. In his 1991 study, Goodman also found a connection between inadequate nutrition and enamel hypoplasia [73].

However, in 2000, the Federation Dentaire Internationale (FDI) and the World Health Organization (WHO) declared that the age of 12 is particularly significant for caries and enamel hypoplasia [74]. Furthermore, 517 children's caries prevalence and enamel abnormalities were examined by Montero et al. [75]. The prevalence of enamel abnormalities was 49%, the mean DMFT was 3, and 38% of the youngsters had carious teeth. Half of the lesions were linear opacities. Caries and enamel flaws were found to be positively correlated. The average age of the children who took part in a study by Disha et al. [29] was 10 years old, and the average age of the children who had hypoplasia was 10.4 years old. Compared to those aged 10–12, the prevalence of enamel hypoplasia was lower in those aged 8–9. Additionally, Fotedar S. et al. [76] noted this. Sixty percent of the youngsters in the sample were 11 years old and exhibited the severe form of hypoplasia. Similar findings were found in other research [76, 77, 78], where the older age groups chosen showed a higher prevalence of more diffuse forms of hypoplasia. This might have happened as a result of prolonged high exposure to etiological causes. These findings were in some ways in line with earlier research that indicated enamel flaws raise the likelihood of dental cavities in the teeth that are impacted [16, 23, 75, 79, 80]. Multivariable regression analysis results showed that, except for age 5–9 occurrence, the between-subject effect from enamel hypoplasia was consistently significant. This suggests that individuals with hypoplasia are more likely to have more caries-affected surfaces and to be at higher risk for caries between the ages of five and nine.

Another significant study by Idiculla et al. [81] discovered that 705 (13%) of 5500 schoolchildren in various age groups, from 6 to 15, showed enamel hypoplasia. Enamel hypoplasia was seen in 7137 (43%) of the 16,470 teeth in these 705 patients. There were 1.3 hypoplastic teeth on average per child (range 0–28). Additionally, the modified DDE index was used to identify enamel flaws. The most prevalent abnormality affecting the greatest number of teeth (305 patients, 43%), was diffuse patchy enamel hypoplasia.

Dental caries was observed in hypoplastic teeth, indicating that people with enamel hypoplasia may experience caries. With a mean score of 3.1 (range 0–10), 577 (9%) of the youngsters had at least one decaying tooth. Out of 1, 18,099 teeth, 13,963 (12%) had at least one decaying tooth with a mean of 8.4 (range 0–12), and 1987 (41%) of the 5500 individuals had dental caries without enamel hypoplasia. A statistically significant correlation was found (chi2 = 651.75, df = 1, p Value =.01). Similarly, 18.2% (185) of the participants in research by Fotedar et al. [76] showed enamel opacities. The prevalence was 17.9% in 89 subjects at age 12 and 18.6% in 96 subjects at age 15. Of the 1765 teeth analyzed in these 185 patients, 210 (11.8%) had enamel hypoplasia. Of these 210 hypoplastic teeth, 91 (10.9%) were impacted when the patient was 12 years old, and 119 (12.7%) were impacted when the patient was 15 years old. At the ages of 12 and 15, the average number of teeth with enamel abnormalities per child was 0.233 and 0.252, respectively. Males in both age groups had a noticeably greater prevalence of enamel hypoplasia.

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Conclusion

Therefore, enamel hypoplasia, a developmental defect, compromises enamel integrity, making teeth more vulnerable to bacterial colonization and decay. Various systemic, genetic, and environmental factors contribute to its occurrence, including preterm birth, nutritional deficiencies, and childhood illnesses. The high prevalence of enamel defects, particularly molar-incisor hypomineralization (MIH), underscores the need for early detection and intervention to prevent further complications, such as increased tooth sensitivity, aesthetic concerns, and a higher risk of dental caries. Dental caries remains a global public health challenge, disproportionately affecting children from low- and middle-income countries. The interplay between cariogenic bacteria, inadequate fluoride exposure, poor oral hygiene, and socio-economic factors exacerbates the incidence of caries in children. Studies indicate a strong association between enamel hypoplasia and dental caries, as compromised enamel structure facilitates plaque accumulation and acid penetration, accelerating the demineralization process. Given these findings, comprehensive oral health strategies should focus on early identification, preventive measures, and appropriate treatment to mitigate the risks associated with enamel defects and dental caries in children.

Dependently, early detection of enamel defects and dental caries should be prioritized through routine dental screenings and advanced diagnostic tools. Preventive strategies must include promoting maternal and child nutrition, ensuring adequate fluoride exposure, and encouraging proper oral hygiene practices. Public health interventions should focus on increasing awareness, reducing sugar consumption, and enhancing access to preventive dental care. Clinical management requires appropriate restorative treatments, frequent monitoring of affected children, and the use of minimally invasive dentistry techniques. Further research and policy development should aim to establish standardized guidelines, conduct longitudinal studies, and integrate oral health into general child healthcare programs. Implementing these measures will help reduce the burden of enamel hypoplasia and dental caries in children, ultimately improving their overall health and quality of life.

Author Contributions

Jbireal M J; Writing, resources and supervision, Almahrog A. and Graf YT; review and editing. All authors have read and agreed to the published version of the manuscript.

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Conflicts of Interest

The authors declare no conflicts of interest.

References

1. Fédération Dentaire Internationale Working Group: A review of developmental defects of the enamel dental index (DDE Index). Commission on Oral Health, Research & Epidemiology. Int Dent J 1992; 42(6): 411-26.
2. Seow, W. K. Developmental defects of enamel and dentine: Challenges for basic science research and clinical management. Aust. Dent. J. 2014;59(1 Suppl.), 143–154.
3. Anthonappa NM, Robert PP. Enamel defects in the permanent dentition: Prevalence and etiology. In: Drummond BK, Kilpatrick N, eds. Book Planning and Care for Children and Adolescents with Den tal Enamel Defects Etiology, Research and Contemporary Management. 1st ed. Berlin and Heidelberg, Germany: Springer-Verlag; 2015:15–30.
4. Clarkson J, O'Mullane D. A modified DDE index for use in epidemiological studies of enamel defects. J Dent Res. 1989; 68:445–450.
5. Crombie F, Manton D, Kilpatrick N. Aetiology of molar–incisor hypo mineralization: A critical review. Int J Paediatr Dent. 2009;19:73–83.
6. Wong HM. Aetiological factors for developmental defects of enamel. Austin J Anat. 2014;1:1003.
7. Collaborators GOD, Bernabe E, Marcenes W. Global, regional, and national levels and trends in burden of oral conditions from 1990 to 2017: a systematic analysis for the global burden of disease 2017 study. J Dental Res. 2020;99: 362-73.
8. Mohammed S, Zahmoul A, Jbireal JM. An Overview on the Relation Between Blood Disorders, Periodontitis and Dental Caries. Alqalam Journal of Medical and Applied Sciences. 2025;8(1):36-42.
9. Janakiram C, Antony B, Joseph J, Ramanarayanan V. Prevalence of dental caries in India among the WHO index age groups: a meta-analysis. J Clin Diagnos Res. 2018;12.
10. Miglani S. Burden of dental caries in India: current scenario and future strategies. Int J Clin Pediatr Dentist. 2020;13:155.
11. Kühnisch J, Thiering E, Heitmüller D, Tiesler CM, Grallert H, Heinrich-Weltzien R, et al. Genome-wide association study (GWAS) for molar-incisor hypomineralization (MIH). Clin Oral Investig. 2014;18(2):677-82.
12. Tinanoff N. Introduction to the Early Childhood Caries Conference: Initial description and current understanding. Community Dent Oral Epidemiol 1998;26:5-7.

Received*: 01-01-2025 -* ***Accepted****: 26-02-2025 -* ***Published****: 03-03-2025*

13. Dilley GJ, Dilley DH, Machen JB. Prolonged nursing habit: A profile of patients and their families. ASDC J Dent Child 1980;47:102-8.
14. Ismail AI, Sohn W. A systematic review of clinical diagnostic criteria of early childhood caries. J Public Health Dent 1999;59:171-91.
15. Nozaka K, Sato T, Mukaida T, Shimazu A, Hasegawa J, Amari E. Shoni Shikagaku Zasshi,1990:28(3):561 578.
16. Pascoe L, Seow WK: Enamel hypoplasia and dental caries in Australian aboriginal children: prevalence and correlation between the two diseases. Pediatr Dent. 1994, 16:193-9.
17. Robles MJ, Ruiz M, Bravo-Perez M, González E, Peñalver MA: Prevalence of enamel defects in primary and permanent teeth in a group of schoolchildren from Granada (Spain). Med Oral Patol Oral Cir Bucal. 2013;18:e187-93.
18. Lai PY, Seow WK, Tudehope DI, Rogers Y. Enamel hypoplasia and dental caries in very-low birthweight children: a case-controlled, longitudinal study. Pediatr Dent. 1997 Jan-Feb;19(1):42-9.
19. Seow WK. Clinical diagnosis of enamel defects: pitfalls and practical guidelines. Int Dent J 1997;47(3):173-82.
20. Seow WK, Clifford H, Battistutta D, Morawska A, Holcombe T. Case-control study of early childhood caries in Australia. Caries Res. 2009;43(1):25-35.
21. Li Y, Navia JM, Caufield PW. Colonization by mutans streptococci in the mouths of 3- and 4-year-old Chinese children with or without enamel hypoplasia. Arch Oral Biol 1994;39(12):1057-62.
22. Targino AG, Rosenblatt A, Oliveira AF, Chaves AM, Santos VE. The relationship of enamel defects and caries: a cohort study. Oral Dis. 2011 May;17(4):420-6.
23. Oliveira AF, Chaves AM, Rosenblatt A. The influence of enamel defects on the development of early childhood caries in a population with low socioeconomic status: a longitudinal study. Caries Res 2006;40(4):296-302.
24. Irvine J, Holve S, Krol D, Schroth R. Early childhood caries in indigenous communities: a joint statement with the American Academy of Pediatrics. Paediatr Child Health 2011;16(6):351-7.
25. Schwendicke F, Elhennawy K, Reda S, Bekes K, Manton DJ, Krois J. Global burden of molar incisor hypomineralization. J Dent 2018; 68:10–18.
26. McCarra C, Olegario IC, O'Connell AC, Leith R. Prevalence of hypomineralised second primary molars (HSPM): a systematic review and meta-analysis. Int J Paediatr Dent 2021; 32:367 382.
27. Jälevik B, Klingberg GA. Dental treatment, dental fear and behaviour management problems in children with severe enamel hypomineralization of their permanent first molars. International journal of paediatric dentistry. 2002 Jan;12(1):24-32.
28. Seow WK, Ford D, Kazoullis S, Newman B, Holcombe T. Comparison of enamel defects in the primary and permanent dentitions of children from a low-fluoride District in Australia. Pediatric dentistry. 2011 Jun 15;33(3):207-12.
29. Disha, V.; Zaimi, M.; Petrela, E.; Aliaj, F. An Investigation into the Prevalence of Enamel Hypoplasia in an Urban Area Based on the Types and Affected Teeth. Children 2024, 11, 474.
30. Arrow, P. Risk factors in the occurrence of enamel defects of the first permanent molars among schoolchildren in Western Australia. Community Dent. Oral Epidemiol. 2009, 37, 405–415.
31. Børsting T, Schuller A, van Dommelen P, Stafne SN, Skeie MS, Skaare AB, et al. Maternal vitamin D status in pregnancy and molar incisor hypomineralisation and hypomineralised second primary molars in the offspring at 7–9 years of age: A longitudinal study. Eur. Arch. Paediatr. Dent. 2022; 23, 557–566.
32. Fagrell T, Ludvigsson J, Ullbro C, Lundin SÅ, Koch G. Aetiology of severe demarcated enamel opacities—An evaluation based on prospective medical and social data from 17,000 children. Swed. Dent. J. 2011; 35, 57–67.
33. Ford D, Seow WK, Kazoullis S, Holcombe T, Newman BA. controlled study of risk factors for enamel hypoplasia in the permanent teeth. Pediatr. Dent. 2009; 31, 382–388.
34. Kühnisch J, Thiering E, Kratzsch J, Heinrich-Weltzien R, Hickel R, Heinrich J. Elevated serum 25(OH)-vitamin D levels are negatively correlated with molar-incisor hypomineralization. J. Dent. Res. 2015, 94, 381–387.
35. Lygidakis NA, Garot E, Somani C, Taylor GD, Rouas P, Wong FSL. Best clinical practice guidance for clinicians dealing with children presenting with molar-incisor-hypomineralisation (MIH): An updated European Academy of Paediatric Dentistry policy document. Eur. Arch. Paediatr. Dent. 2022; 23, 3–21.
36. Tapias-Ledesma MA, Jiménez R, Lamas F, González A, Carrasco P, De Miguel AG. Factors associated with first molar dental enamel defects: A multivariate epidemiological approach. J. Dent. Child. 2003; 70, 215–220.
37. Franco KMD, Peres SR, Moura-Ribeiro MVL. Prenatal and neonatal variables associated with enamel hypoplasia in deciduous teeth in low-birth-weight preterm infants. J. Appl. Oral. Sci. 2007; 15, 518–523.
38. Salanitri S, Seow W. Developmental enamel defects in the primary dentition: aetiology and clinical management. *Aust. Dent. J.* 2013;58, 133–140.
39. Da Costa-Silva C M, Ambrosano G M, Jeremias F, De Souza J F, Mialhe F L. Increase in severity of molar–incisor hypomineralization and its relationship with the colour of enamel opacity: a prospective cohort study. *Int. J. Paediatr. Dent.* 2011;21, 333–341.
40. Robinson C. Enamel maturation: A brief background with implications for some enamel dysplasias. *Front. Physiol.* 2014;5, 388.
41. Kanchan T, Machado M, Rao A, Krishan K, Garg A K. Enamel hypoplasia and its role in identification of individuals: A review of literature. Indian Journal of Dentistry, 2015;6(2), 99.
42. Featherstone JD. The caries balance: the basis for caries management by risk assessment. Oral Health Prevent Dentist. 2004;2:259-64.
43. Siqueira W, Custodio W, McDonald E. New insights into the composition and functions of the acquired enamel pellicle. J Dent Res. 2012;91:1110-8.
44. Kirthiga M, Murugan M, Saikia A, Kirubakaran R. Risk Factors for Early Childhood Caries: A Systematic Review and Meta-Analysis of Case Control and Cohort Studies. Pediatr Dent. 2019 Mar 15;41(2):95-112.

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45. Selwitz RH, Ismail AI, Pitts NBJTL. Dent Caries. Lancet. 2007;369(9555):51–9.
46. Ramesh S, Sundari S. Dental caries and oral hygiene practices among school children in Chennai, India: a cross sectional study.
47. Woan J, Lin J, CJJoA H, Auerswald. The health status of street children and youth in low-and middle-income countries: a systematic review of the literature. 2013;53(3):314–321.
48. Calache H, Christian B, Mamerto M, Kangutkar T, Hall M. An epidemiological study of dental caries and associated risk factors among primary school children in the Aileu Municipality, Timor-Leste. Rural Remote Health. 2019 Oct;19(4):5322.
49. Andegiorgish AK, Weldemariam BW, Kifle MM, Mebrahtu FG, Zewde HK, Tewelde MG, Hussen MA, Tsegay WK. Prevalence of dental caries and associated factors among 12 years old students in Eritrea. BMC Oral Health. 2017 Dec 29;17(1):169.
50. Alraqiq H, Eddali A, Boufis R. Prevalence of dental caries and associated factors among school-aged children in Tripoli, Libya: a cross-sectional study. BMC oral health. 2021 Apr 30;21(1):224.
51. Que L, Jia M, You Z, Jiang LC, Yang CG, Quaresma AAD, das Neves EMAA. Prevalence of dental caries in the first permanent molar and associated risk factors among sixth-grade students in São Tomé Island. BMC Oral Health. 2021 Sep 28;21(1):483.
52. Blankson PK, Amoah G, Thadani M, Newman-Nartey M, Amarquaye G, Hewlett S, Ampofo P, Sackeyfio J. Prevalence of oral conditions and associated factors among schoolchildren in Accra, Ghana: a cross-sectional study. Int Dent J. 2022 Feb;72(1):93-99.
53. Chouchene F, Masmoudi F, Baaziz A, Maatouk F, Ghedira H. Early Childhood Caries Prevalence and Associated Risk Factors in Monastir, Tunisia: A Cross-Sectional Study. Front Public Health. 2022 Feb 25;10:821128.
54. Eigbobo JO, Alade GJSMJ. Dental caries experience in primary school pupils in Port Harcourt. Nigeria. 2017;20(4):179.
55. Kassim B, Noor M. Chindia, oral health status among kenyans in a rural arid setting: dental caries experience and knowledge on its causes. 2006;83(2):100–5.
56. Gathecha G, Makokha A, Wanzala P, Omolo J, Smith P. Dental caries and oral health practices among 12-year-old children in Nairobi West and Mathira West Districts, Kenya. Pan Afr Med J. 2012;12:42.
57. Bogale B, Engida F, Hanlon C, Prince MJ, Gallagher JE. Dental caries experience and associated factors in adults: a cross-sectional community survey within Ethiopia. BMC Public Health. 2021 Jan 21;21(1):180.
58. Mulu W, Demilie T, Yimer M, Meshesha K, Abera B. Dental caries and associated factors among primary school children in Bahir Dar city: a cross-sectional study. BMC Res Notes. 2014 Dec 23;7:949.
59. Berhane H, Worku A. Oral Health of Young Adolescents in Addis Ababa—A Community-Based Study. Open Journal of Preventive Medicine. 2014;4, 640-648.
60. Tefera AT, Girma B, Adane A, Muche A, Ayele TA, Getahun KA, Aniley Z, Ali S, Handebo S. The prevalence of dental caries and associated factors among students living with disability in the Amhara region, Ethiopia. Clin Exp Dent Res. 2022 Dec;8(6):1505-1515.
61. Teshome A, Yitayeh A, Gizachew MJA. Prevalence of dental caries and associated factors among Finote Selam primary school students aged 12–20 years, Finote Selam town. Ethiopia. 2016;12(14):15–7.
62. World health organization. Sugars and dental caries. WHO: Geneva Swezerland; 2017.
63. Cvikl B, Haubenberger-Praml G, Drabo P, Hagmann M, Gruber R, Moritz A, Nell A. Migration background is associated with caries in Viennese school children, even if parents have received a higher education. BMC Oral Health. 2014 May 9;14:51.
64. da Fonseca MA, Avenetti DJDC. Social determinants pediatric oral health. 2017;61(3):519–32.
65. Yirsaw AN, Bogale EK, Tefera M, Belay MA, Alemu AT, Bogale SK, Getachew E, Andarge GA, Seid K, Lakew G. Prevalence of dental caries and associated factors among primary school children in Ethiopia: systematic review and meta-analysis. BMC Oral Health. 2024 Jul 10;24(1):774.
66. Farsi N. Developmental enamel defects and their association with dental caries in preschoolers in Jeddah, Saudi Arabia. Oral Health Prev Dent. 2010;8(1):85–92.
67. Vargas-Ferreira F, Salas MM, Nascimento GG, Tarquinio SB, Faggion CM Jr, Peres MA, Thomson WM, Demarco FF. Association between developmental defects of enamel and dental caries: A systematic review and meta-analysis. J Dent. 2015 Jun;43(6):619-28.
68. Hong L, Levy SM, Warren JJ, Broffitt B. Association between enam el hypoplasia and dental caries in primary second molars: A cohort study. Caries Res. 2009;43(5):345–352.
69. Salanitri S, Seow W. Developmental enamel defects in the primary dentition: aetiology and clinical management. Australian Journal. 2013:58(2):133-140.
70. Seow WK. Enamel hypoplasia in the primary dentition: a review. ASDC J Dent Child,1991:58(6):441-452.
71. Jindal C, Palaskar S, Kler S. The prevalence of the developmental defects of enamel in a group of 8-15 years old Indian children with developmental disturbances. J Clin Diagn Res. 2011, 5:669-74.
72. Chauhan D, Chauhan T. Prevalence of developmental defects of enamel in mixed and permanent dentition of 9 and 12 year old children of Himachal Pradesh, India: a cross sectional study. Int J Health Allied Sci. 2013, 2:185.
73. Goodman A, Jerome R. Dental enamel hypoplasias as indicators of nutritional status. Adv Dent Anthrop. 1991; 1:991.
74. WHO Assignment report, WHO/Geneva/Swiss 1998.
75. Montero MJ, Douglass JM, Mathieu GM. Prevalence of dental caries and enamel defects in Connecticut Head Start children. Pediatr Dent. 2003 May-Jun;25(3):235-9.
76. Fotedar S, Sogi GM, Sharma KR. Enamel hypoplasia and its correlation with dental caries in 12 and 15 years old school children in Shimla, India. J. Indian Assoc. Public Health Dent. 2014; 12, 18–22.

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77. Tomczyk J, Tomczyk-Gruca M, Zalewska M. Frequency and chronological distribution of linear enamel hypoplasia (LEH) in the Late Neolithic and Early Bronze Age population from ˙ Zerniki Górne (Poland)—Preliminary report. Anthropol. Rev. 2012; 75:61–73.
78. Idiculla JJ, Brave VR, Puranik RS, Vanaki S. Enamel hypoplasia and its correlation with dental caries in school children of Bagalkot, Karnataka. J. Oral Health Community Dent. 2011; 5:31–36.
79. Li Y, Navia JM, Bian JY. Caries experience in deciduous dentition of rural Chinese children 3–5 years old in relation to the presence or absence of enamel hypoplasia. Caries Res 1996; 30: 8–15.
80. Milgrom P, Riedy CA, Weinstein P, Tanner AC, Manibusan LB: Dental caries and its relationship to bacterial infection hypoplasia, diet and oral hygiene in 6- to 36-month-old children. Community Dent Oral Epidemiol 2000; 28: 295–306.
81. Idiculla JJ, Brave VR, Ouranik RS, Vanaki S. Enamel Hpoplasia and its Correlation with Dental Caries In School Children of Bagalkot, Karnataka. J Oral Health Comm Dent. 2011;5(1)31-36.

المستخلص

يؤثر نقص تنسج المينا وتسوس الأسنان بشكل كبير على صحة الفم لدى الأطفال، حيث تزيد عيوب المينا من قابلية استعمار البكتيريا والتسوس. يستكشف هذا الاستعراض انتشار وأسباب وعواقب نقص تنسج المينا، وخاصة نقص تمعدن الأضراس والقواطع، مع التركيز على ارتباطه بالعوامل الجهازية والوراثية والبيئية. يظل تسوس الأسنان مصدر قلق رئيسي للصحة العامة، وخاصة في البلدان ذات الدخل المنخفض والمتوسط، حيث يؤدي التعرض غير الكافي للفلورايد، وسوء نظافة الفم، والتفاوتات الاجتماعية والاقتصادية إلى تفاقم الحالة. ونظرًا للارتباط القوي بين نقص تنسج المينا وتسوس الأسنان، فإن الاكتشاف المبكر، والتدابير الوقائية، والإدارة السريرية المناسبة ضرورية. تعد تدخلات الصحة العامة، وتطوير السياسات، والمزيد من البحث أمرًا بالغ الأهمية في تخفيف عبء هذه الحالات وتحسين نتائج صحة الفم لدى الأطفال. وعليه، فإن الهدف الرئيسي من هذه المراجعة هو مراجعة النتائج الأخيرة بشأن نقص تنسج مينا الأسنان وتسوس الأسنان لدى الأطفال، مع تسليط الضوء على انتشارها وأسبابها وعواقبها والحاجة إلى الكشف المبكر عنها والوقاية منها والتدخلات الصحية العامة.

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