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Influence of Developmental Disturbances and Post-Eruptive Changes in Tooth Morphology: A Narrative Review

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ABSTRACT

Tooth morphology is shaped by a complex interplay of genetic, environmental, systemic, and local factors acting during development and throughout post-eruptive life. This narrative review synthesizes current evidence on factors influencing tooth development and post-eruptive changes, with emphasis on clinical and morphological implications. During odontogenesis, disturbances in morphodifferentiation, matrix formation, and mineralization may result in anomalies of size, shape, and structure. Traumatic injuries to primary teeth, particularly at younger ages, can disrupt the developing permanent successors, producing defects ranging from enamel hypoplasia and opacities to crown malformations. Local infections, surgical interventions, and perinatal trauma further contribute to developmental disturbances, often in a site-specific pattern depending on the timing and severity of insult. Following eruption, teeth are subjected to continuous functional and environmental challenges. Post-developmental anomalies, such as attrition, abrasion, and erosion, lead to irreversible loss of hard tissue and progressive modification of crown morphology. These processes are frequently multifactorial and may be compounded by restorative procedures or cultural practices involving intentional crown alteration. Together, developmental disturbances and post-eruptive modifications significantly influence dental form and structural integrity across the lifespan. Understanding the mechanisms, timing, and clinical presentation of these changes is essential for accurate diagnosis, treatment planning, and forensic interpretation. This review highlights the dynamic nature of dental morphology and underscores the importance of integrating developmental history with clinical findings in comprehensive dental assessment.

Keywords: Tooth development, Dental abnormalities, Tooth wear, Tooth morphology, Systemic effects.

1. Introduction

Environmental influences during the prenatal, perinatal, and postnatal stages play a critical role in shaping human dentition. Careful evaluation of the affected tooth and the location of structural disturbances may provide valuable information regarding the timing of developmental disturbances (1). For example, calcification of the crowns of deciduous incisors and first and second molars begins at approximately 12.5

weeks of gestation, whereas deciduous canines begin calcification around 20 weeks of gestation (2). Crown formation of deciduous teeth is generally completed by about 11 months postnatally (3). Therefore, any disruption occurring during these defined developmental windows may be permanently recorded in the tooth structure.

Such insults may manifest as structural lesions localized to specific regions of the crown, corresponding

to the stage of development at which the disturbance occurred (4,5). These defects may result from systemic conditions, such as maternal illness, nutritional deficiencies, metabolic disorders, perinatal complications, or from localized factors including trauma, infection, or pressure effects. Depending on the nature and timing of the insult, the resulting defect may affect a single tooth or multiple teeth, and may present unilaterally or bilaterally. Alteration of the micromorphology and ultrastructure of developing enamel and dentin can be due to local or systemic factors, producing permanent developmental disturbances that serve as chronological markers of early life events.

This narrative review was conducted through a structured search of the PubMed, Scopus, and Google Scholar databases. Literature published in English up to June 2026 was considered. Search terms included combinations of “tooth development”, “tooth morphology”, “developmental disturbances”, “dental anomalies”, “enamel defects”, “odontogenesis”, and “tooth wear”. The objective was to identify studies addressing developmental and post-eruptive factors influencing tooth morphology, including genetic, systemic, environmental, traumatic, and cultural influences. Priority was given to peer-reviewed articles, reviews, and landmark studies that contributed substantially to understanding the aetiology, clinical presentation, and forensic significance of morphological variations. As this manuscript was designed as a narrative review, formal systematic review procedures, such as predefined study screening, quantitative selection processes, and risk-of-bias assessment, were not undertaken.

The purpose of this narrative review is to provide an integrated overview of developmental disturbances and post-eruptive factors that influence tooth morphology. By bringing together evidence from developmental biology, clinical dentistry, and forensic sciences, this review aims to highlight the multifactorial nature of dental morphological variation and its significance in diagnosis, treatment planning, and personal identification.

2. Local Factors

Traumatic injuries to the deciduous dentition are of particular concern because of the close anatomical relationship between deciduous tooth roots and the

developing permanent tooth germs (6). Displacement injuries, such as intrusion and avulsion, may transmit force directly to the underlying permanent successor. Clinical evidence suggests a strong association between the severity of trauma to primary teeth and the extent of developmental disturbances observed in their permanent successors. Furthermore, the younger the child at the time of injury, the greater the risk and severity of subsequent defects, reflecting the vulnerability of teeth during early stages of morphodifferentiation and amelogenesis (2,7). Traumatic injury to primary teeth may also result in dilaceration of the developing permanent successor. Dilaceration refers to an abnormal angulation or sharp bend in the crown or root of a tooth, typically arising from mechanical displacement of the developing tooth germ (8). The severity and location of the curvature depend on the stage of development at the time of injury. Clinically, dilaceration may complicate eruption, endodontic treatment, and extraction procedures. From a forensic perspective, its distinct radiographic presentation may serve as a valuable identifying feature.

Disturbance to the developing tooth germ may manifest in a variety of morphological anomalies. Alterations in crown shape, such as peg-shaped lateral incisors, have been reported following injury during critical stages of odontogenesis (9). More complex developmental disturbances, including fusion or gemination, may arise when trauma interferes with the normal proliferation and differentiation of the dental lamina (10). Additional sequelae, such as talon cusp formation (11,12) and dens evaginatus or evaginated odontome (13), have also been associated with disruption of normal morphogenetic processes. These findings demonstrate that traumatic injury may affect not only enamel formation, but also the overall morphology of the developing crown.

Dens invaginatus, also known as dens in dente, is a developmental anomaly characterized by an infolding of enamel and dentin into the tooth structure during morphogenesis. This results in the formation of a deep invagination that may extend into the pulp cavity, creating a complex internal anatomy (14). The condition most commonly affects maxillary lateral incisors and may present unilaterally or bilaterally. Clinically, dens invaginatus is significant due to the increased risk of early caries development and pulpal pathology, as the invaginated area can facilitate bacterial ingress. Its

characteristic radiographic appearance and morphological variability make it clinically significant and potentially useful in forensic identification.

Developmental dental anomalies may be localized affecting single or multiple teeth or may present in a more generalized distribution depending on the nature and timing of the insult (15). Direct trauma to ameloblasts during the formation of the matrix, such as from a fall or blow to the deciduous predecessor, can produce sharply demarcated enamel defects in the corresponding permanent tooth. The clinical presentation depends largely on the stage of amelogenesis at the time of injury. Trauma occurring during the secretory (matrix formation) phase typically results in enamel hypoplasia due to disruption of enamel matrix deposition. In contrast, injury during the maturation phase interferes with mineralization, producing demarcated opacities without quantitative enamel loss (16).

Infective processes originating from a necrotic or chronically infected deciduous tooth represent another significant local etiological factor. Microbial invasion and associated inflammatory responses may extend to the follicle of the developing permanent tooth, leading to a spectrum of enamel defects (17). The resulting abnormalities range from mild opacities to pronounced hypoplastic defects, sometimes referred to clinically as Turner's hypoplasia.

Iatrogenic factors must also be considered. Surgical procedures involving the maxillofacial region—particularly in children with cleft lip and palate—may disturb adjacent developing tooth germs. Extractions, alveolar bone grafting, and reconstructive surgeries have been associated with enamel defects and morphological alterations, typically localized to teeth adjacent to the surgical site (18). Perinatal trauma represents an additional localized risk. Preterm infants often require laryngoscopy and endotracheal intubation to manage respiratory distress. Mechanical pressure from instrumentation may injure the developing enamel organ of the maxillary deciduous incisors, increasing the likelihood of enamel hypoplasia or other defects (16).

While individual developmental disturbances may arise from diverse local or systemic causes, their effects on tooth morphology frequently reflect the stage of odontogenesis affected. Disturbances occurring during morphodifferentiation are more likely to result in anomalies of tooth size, shape, or number, whereas

insults during matrix formation and mineralization predominantly produce structural defects, such as enamel hypoplasia and opacities. Although considerable evidence supports the influence of genetic and environmental factors on dental development, the relative contribution of each factor remains difficult to determine, because multiple influences often interact simultaneously. This complexity highlights the need for multidisciplinary approaches integrating developmental biology, clinical dentistry, and forensic sciences to improve understanding of dental morphological variation.

Collectively, these findings highlight the sensitivity of developing teeth to local trauma and environmental disturbances. The nature, severity, and timing of the insult determine the type and extent of the resulting defect, emphasizing the importance of early diagnosis, careful management of deciduous tooth injuries, and consideration of developmental history in both clinical and forensic evaluations. While local factors primarily exert their effects through direct injury or disruption of developing tooth structures, systemic factors may influence odontogenesis more broadly through genetic, metabolic, and environmental pathways.

3. Systemic Factors

Numerous developmental disturbances arise from complex interactions among genetic, epigenetic, and environmental factors during odontogenesis (19,20). Various developmental regulatory genes are actively involved in the process of odontogenesis (20–22). The frequent co-occurrence of anomalies affecting tooth morphology, size, and number reflects the shared signalling pathways involved during initiation and morphogenesis (22). One of the most frequently reported anomalies involves variations in the size of the maxillary lateral incisors and second premolars (23). Down syndrome is a genetic condition resulting from the trisomy of chromosome 21, which also presents various oral malformations. The prevalent defects in individuals with Down syndrome include anterior open bite, periodontal issues, and congenitally absent teeth (24). Microdontia, tooth agenesis, prognathic mandible, narrow maxilla, taurodontism, altered deciduous and permanent dentition eruption, and teeth malalignment are observable (25). The dentition often exhibits reduced intercuspal distances and less complex occlusal morphology, characterized by shallower pits and

fissures. The occlusal surfaces of the molars exhibit wrinkling, potentially attributable to a developmental abnormality (24,25).

Turner syndrome is a genetic condition characterized by females possessing normal growth hormones, but missing one X chromosome, which impacts their physical development. This impacts the crown dimensions of their teeth, with molars and premolars exhibiting reduced crown and root morphometrics compared to standard sizes (26). In such instances, permanent maxillary first molars in 45,X females may have reduced crown size with high prevalence associated with the three-cusp pattern as compared to normal controls (26). The deficiency of sex chromosome material disrupts morphology of the lingual cingulum region (Carabelli's trait, the distolingual cusp) of permanent maxillary first molar during tooth crown development, while exerting a lesser impact on Carabelli's trait and the distolingual cusp morphology of permanent maxillary second molar (27).

Klinefelter syndrome (KS), characterized by the presence of an additional X chromosome (47,XXY) (28). Taurodontism results in teeth with enlarged pulp chambers and apically displaced pulpal floors, producing crowns that appear proportionately larger relative to the roots (29). This characteristic is found in both deciduous and permanent teeth, in one or several molars within the same quadrant, and may present unilaterally or bilaterally (29).

Macrodontia refers to teeth that are larger than normal in size. This condition may affect a single tooth or multiple teeth and can be classified as true generalized, relative generalized, or localized macrodontia. It is relatively rare and may be associated with systemic conditions, such as pituitary gigantism, or may occur as an isolated finding (30). Clinically, macrodontia can lead to crowding, malocclusion, and aesthetic concerns. Its presence contributes to variations in dental morphology that may aid in individual identification.

Genetic disturbances affecting enamel and dentin formation may result in conditions, such as amelogenesis imperfecta and dentinogenesis imperfecta. Amelogenesis imperfecta comprises a group of hereditary disorders characterized by defects in enamel formation, which may manifest as hypoplastic, hypomineralized, or hypomature enamel (31). Clinically, affected teeth may appear discolored, fragile,

and prone to rapid wear. Dentinogenesis imperfecta, on the other hand, primarily affects dentin structure, leading to opalescent teeth with altered translucency, bulbous crowns, and early obliteration of the pulp chambers (32). Both conditions affect primary and permanent dentitions and demonstrate significant variability in clinical presentation. Their distinct morphological characteristics make them important considerations in both clinical diagnosis and forensic identification.

Among people of Asian descent, cleft lip and palate abnormality are more prevalent than in people of other origins (33). Cleft lip and palate abnormality are characterized by a complex set of factors that influence their development. These factors include both environmental and genetic influences. Dental abnormalities were shown to be more prevalent on the side of the mouth that was impacted by the cleft. The presence of a peg or conical shaped lateral incisor (34) was the aberration in tooth shape that occurred the most frequently (95%) among all the cases described.

Evidence from twin and family studies suggests that although genetic factors contribute substantially to dental development, environmental influences remain important determinants of variation in occlusal characteristics and tooth morphology (35-37). Systemic factors, such as low birth weight, prematurity, and nutritional deficiencies, have been associated with the development of enamel abnormalities (36-38). Dental developmental anomalies that arise during amelogenesis are irreversible due to the fact that dental enamel lacks the potential to undergo remodeling. As a result, the faults that arise during its creation are permanently inscribed on the tooth surface (39). In preterm infants, enamel hypoplasia is attributed to inadequate delivery of calcium and phosphorus mineral and the failure of gastrointestinal tract to absorb these minerals (40). Nutritional deficiencies resulting from starvation lead to various anomalies in tooth structure.

Deficiencies in protein and vitamins significantly affect the formation of dental structures, leading to various abnormalities (41). Vitamin D insufficiency may result from insufficient sunshine exposure and inadequate consumption of vitamin-rich foods. This may also result from any inherited condition related to absorption or metabolic conversion, as well as maternal levels of 25-hydroxy vitamin D (42,43). Phenytoin, carbamazepine, and oxcarbamazepine can lead to

increased elimination of the vitamin from the body (44). Vitamin D deficiency can also significantly disrupt normal tooth mineralization, leading to hypomineralized and faulty tooth structure, which increases susceptibility to fracture and disease. Vitamin D deficiency may manifest as characteristic band-like enamel hypoplasia, reflecting periods of disrupted mineralization during tooth development. The location of these defects may correspond to the period during which the deficiency occurred (45). Vitamin A insufficiency leads to compromised tooth development and enamel hypoplasia. The defining characteristic of enamel hypoplasia is the presence of hypoplastic pits or grooves in the enamel, typically exhibiting a horizontal or linear configuration (41). Developmental dental anomalies have also been reported among children undergoing cancer therapy. Patients aged 6 years or younger who had chemotherapy exhibited a greater incidence of abnormalities (46). The most prevalent defects in these patients manifest as agenesis, microdontia, and discernible hypoplasia ridges, frequently accompanied by concurrent disruptions in dentine development and root deformities. Duggal et al. demonstrated a significant frequency of enamel abnormalities across all teeth in cancer survivor individuals (47). A diminished proportion of individuals exhibited enamel abnormalities in certain teeth rather than in all. Leukaemia therapy, being the most prevalent childhood

malignancy, exerts a widespread influence on amelogenesis. Treatment for acute lymphoblastic leukaemia has been associated with an increased prevalence of developmental dental anomalies (48).

Despite substantial advances in understanding the genetic and environmental determinants of dental development, important knowledge gaps remain. Many reported associations are derived from observational studies, making it difficult to establish causal relationships or determine the relative contribution of individual factors. In addition, considerable variation exists in the clinical expression of developmental disturbances, even among individuals exposed to similar risk factors. Future studies integrating genetic, environmental, and developmental data may provide a more comprehensive understanding of the mechanisms underlying dental morphological variation.

Local and systemic influences may disrupt normal odontogenesis and result in a wide spectrum of dental morphological anomalies. These disturbances can affect tooth size, shape, number, and structure, potentially compromising function, aesthetics, and long-term oral health. At the same time, many developmental anomalies create distinctive morphological features that may assist in personal identification when documented in ante-mortem dental records. Therefore, recognition of these variations is important in both clinical practice and forensic investigations.

Table 1: Summary of major developmental disturbances on tooth morphology

Developmental factor	Examples	Principal morphological outcome	Clinical/Forensic significance
Local trauma	Intrusion, avulsion, displacement injuries to primary teeth	Dilaceration, enamel hypoplasia, crown malformations, opacities	May complicate eruption and treatment; distinctive radiographic features aid identification
Local infection	Chronic infection of primary teeth	Turner's hypoplasia, localized enamel defects	Provides evidence of previous developmental insult and assists diagnosis
Surgical/iatrogenic factors	Cleft-related surgery, bone grafting, extractions	Enamel defects and localized developmental disturbances	Important consideration during treatment planning
Genetic syndromes	Down syndrome, Turner syndrome, Klinefelter syndrome	Altered tooth size, shape, eruption pattern, taurodontism	Distinctive dental features may support syndrome recognition and identification
Nutritional and metabolic disturbances	Vitamin deficiencies, prematurity, low birth weight	Enamel hypoplasia, Hypomineralization, chronological defects	Reflects systemic health disturbances during tooth development

Developmental disorders of dental tissues	Amelogenesis imperfecta, dentinogenesis imperfecta	Structural defects affecting enamel and dentine morphology	Characteristic appearances assist diagnosis and forensic comparison
Post-eruptive influences	Attrition, abrasion, erosion	Progressive alteration of crown morphology and occlusal anatomy	May provide information regarding age, habits, and environmental exposures

4. Anomalies of Tooth Number

Developmental disturbances may also affect the number of teeth, resulting in conditions, such as hypodontia, oligodontia, anodontia, and hyperdontia. Hypodontia, defined as the congenital absence of one to six teeth excluding third molars, represents the most common form and frequently involves maxillary lateral incisors and second premolars. More severe forms include oligodontia (absence of more than six teeth) and anodontia (complete absence of teeth), often associated with genetic syndromes or mutations affecting early tooth initiation. In contrast, hyperdontia refers to the presence of supernumerary teeth, which may occur in both primary and permanent dentitions. The most common type is mesiodens, typically located in the maxillary anterior region. These anomalies may disrupt normal eruption patterns, cause crowding or impaction, and hold significant clinical and forensic relevance due to their distinctiveness.

The aetiology of hyperdontia remains incompletely understood, although hyperactivity of the dental lamina is the most widely accepted explanation. Supernumerary teeth may vary considerably in size and morphology, ranging from supplemental teeth that closely resemble normal dentition to rudimentary forms, such as conical, tuberculate, or molariform variants. In addition to causing crowding and delayed eruption, hyperdontia may complicate orthodontic treatment and surgical management. From a forensic perspective, the presence, position, and morphology of supernumerary teeth represent distinctive characteristics that may assist in comparative dental identification.

5. Forensic Significance of Developmental Dental Anomalies

Developmental dental anomalies possess considerable forensic value, because many are permanent, distinctive, and readily identifiable on clinical records and radiographs. Variations, such as dilaceration, dens invaginatus, taurodontism, peg-

shaped lateral incisors, supernumerary teeth, and congenital tooth absence may serve as individualizing characteristics during comparative identification. Unlike soft tissue features, these anomalies are resistant to post-mortem degradation and are often documented in routine dental examinations, radiographs, and treatment records.

The forensic significance of such anomalies increases when multiple unusual features occur in the same individual. For example, a combination of hypodontia, peg-shaped lateral incisors, and taurodontism may provide a highly distinctive dental profile. Similarly, developmental anomalies resulting from childhood trauma or systemic disturbances can leave permanent markers within the dentition that reflect specific developmental events. When ante-mortem and post-mortem dental records are compared, these unique morphological characteristics may provide valuable supporting evidence for establishing identity.

6. Changes in Teeth after Development

Following eruption into the oral cavity, teeth are continuously exposed to a dynamic environment that influences their morphology and crown dimensions. Unlike developmental alterations that occur during odontogenesis, post-developmental changes arise from functional use, pathological processes, therapeutic interventions, and, in some populations, intentional cultural modification. These alterations may significantly modify occlusal anatomy, surface texture, and overall crown morphology, with implications for clinical dentistry and forensic interpretation.

Tooth wear represents one of the most common post-eruptive changes and is defined as the irreversible loss of dental hard tissue from causes other than dental caries (49). It is typically multifactorial, resulting from the interplay of mechanical and chemical processes. Abrasion refers to pathological wear caused by external mechanical agents, such as tooth brushing technique or abrasive particles in dentifrices (50). The pattern is often

localized and corresponds to the site of the abrasive challenge. Attrition, in contrast, results from tooth-tooth contact, commonly associated with mastication or parafunctional behaviors, such as bruxism. Clinically, attrition produces matching wear facets on opposing teeth and may progressively reduce cusp height and occlusal relief (51).

Erosion involves the chemical dissolution of dental hard tissues caused by acids not derived from oral bacteria. Extrinsic acids, including carbonated beverages and citrus drinks, and intrinsic acids, particularly gastric hydrochloric acid in gastroesophageal reflux disease, contribute significantly to this process. A characteristic “scooped-out” appearance may develop, particularly where dentine is exposed, as dentine demineralizes more rapidly than enamel (52). In advanced stages, erosion may lead to substantial loss of enamel thickness and alteration of crown contour. Importantly, these processes rarely act in isolation; erosion may soften enamel, rendering it more susceptible to mechanical wear, thereby compounding tissue loss (53).

Beyond wear, post-developmental changes also include modifications resulting from restorative and endodontic procedures. Dental restorations, crowns, veneers, and orthodontic adjustments can alter crown dimensions and occlusal relationships (54). Over time, secondary dentine deposition and pulpal changes may also occur in response to functional or pathological stimuli, subtly modifying internal tooth morphology.

In addition, extreme environmental and cultural influences may produce distinctive patterns of crown alteration. Anthropological evidence documents deliberate tooth modification practices in certain aboriginal and traditional populations, such as tooth ablation, crown chipping, filing, inlay placement, dyeing, and other forms of intentional mutilation... etc. (55,56). These practices can result in characteristic crown reshaping that is culturally specific and may serve as markers of identity, status, or rite of passage.

The form and extent of intentional dental modification vary considerably among populations and historical periods. In some communities, these practices were associated with social identity, tribal affiliation, aesthetics, or rites of passage. Because the resulting

alterations are often highly characteristic and culturally patterned, they provide valuable anthropological and forensic information. Recognition of intentional modification is therefore important when interpreting unusual crown morphology and distinguishing culturally induced changes from pathological or developmental conditions.

Collectively, post-eruptive changes reflect the cumulative influence of function, environmental exposure, disease processes, therapeutic interventions, and cultural practices. Their multifactorial nature often complicates etiological diagnosis, as multiple mechanisms frequently operate concurrently. Careful morphological assessment, supported by clinical and contextual information, is therefore essential for accurate interpretation in both clinical and forensic settings (57).

7. Conclusions

Tooth morphology is influenced by a wide range of developmental and post-eruptive factors that act throughout life. Local trauma, infections, genetic conditions, nutritional deficiencies, and systemic disorders may disrupt odontogenesis, resulting in permanent alterations in tooth size, shape, number, and structure. Following eruption, functional wear, environmental influences, restorative procedures, and cultural practices continue to modify dental morphology and crown architecture.

Understanding the timing, mechanisms, and manifestations of these changes is essential for accurate diagnosis, treatment planning, and forensic interpretation. Developmental disturbances frequently leave permanent markers within the dentition that may provide valuable insights into an individual’s biological history and serve as distinguishing features during personal identification. Future research integrating developmental biology, advanced imaging technologies, and forensic applications may further enhance understanding of dental morphological variation and its clinical significance.

Conflict of Interests

The authors have no conflict of interests to declare.

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