Scientific Article

Comparison of Enamel Defects in the Primary and Permanent Dentitions of Children from a Low-fluoride District in Australia

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Abstract: ***Purpose:*** *The purpose of this study was to compare developmental defects of enamel (DDE) in the primary and permanent dentitions of children from a low-fluoride district.* ***Methods:*** *A total of 517 healthy schoolchildren were examined using the modified DDE criteria.* ***Results:*** *The prevalence of DDE in the primary and permanent dentition was 25% and 58%, respectively (P<.001). The mean number of teeth with enamel opacity per subject was approximately threefold compared to that affected by enamel hypoplasia (3.1±3.8 vs 0.8±1.4, P<.001 in the primary dentition and 3.6±4.7 vs 1.2±2.2, P<.001 in the permanent dentition). Demarcated opacities (83%) were predominant compared to diffuse opacities (17%), while missing enamel was the most common type of enamel hypoplasia (50%), followed by grooves (31%) and enamel pits (19%) (P=.04). In the permanent dentition, diffuse and demarcated opacities were equally frequent, while enamel grooves were the commonest type of hypoplasia (52%), followed by missing enamel (35%) and enamel pits (5%; P<.001).* ***Conclusions:*** *In a low-fluoride community, developmental defects of enamel were twice as common in the permanent dentition vs the primary dentition. In the primary dentition, the predominant defects were demarcated opacities and missing enamel, while in the permanent dentition, the defects were more variable. (Pediatr Dent 2011;33:207-12) Received November 3, 2009 Last Revision May 31, 2010 | Accepted July 2, 2010*

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Abnormalities of tooth enamel that arise from injury to the enamel organ are referred to as developmental defects of the enamel (DDE) and may be classified broadly as enamel hypoplasia or enamel opacities.[1,2] These developmental defects can have a significant impact on oral health, such as compromised esthetics, tooth sensitivity, and altered occlusal functions.[3-7] In addition, enamel defects are now increasingly recognized as risk indicators for dental caries and erosion in children.[3-8] Most studies report DDE prevalence in developed countries to be in the range of 5% to 49% and 9% to 68% for any enamel defect in the primary and permanent dentition, respectively.[9-14] In Australia, prevalence studies of DDE were mainly performed in fluoridated communities; these generally reported on defects present in permanent molars and incisors.[15,16]

In a recent study by Arrow in Western Australia, an area with artificially fluoridated water, the prevalence of enamel defects in the first permanent molars was found to be over 50%.[15] There have been, however, no large scale reports on the prevalence of DDE in healthy children from nonfluoridated communities in Australia, although data are available for preterm and other medically compromised children, as well as for indigenous communities.[7,17-19] Furthermore, in other countries where primary and permanent dentitions have been individually studied, there is a paucity of reports which examine the prevalence and presentations of DDE in the primary and permanent teeth of children from the same community. Comparing clinical presentations of the defects provides insight into the responses of ameloblasts to environmental insults in the primary and permanent dentitions[2,20] and facilitates the identification of etiological agents.

Therefore, the purpose of this study was to examine a group of healthy Australian children residing in a nonfluoridated community in order to compare and contrast the prevalence and presentations of developmental defects of the enamel in the primary and permanent dentitions.

Methods

This study was approved by the institutional ethics committees of the University of Queensland and Queensland Health The subjects were randomly recruited from several schools within a status health service in the state of Queensland, Australia.[21] As with other parts of the state, the community water supplies in the district were regularly monitored by the local government, and fluoride levels in the natural water

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were reported in health department publications to be approximately 0.1 ppm at the time of writing.[22] Furthermore, the free public oral health programs do not routinely prescribe fluoride supplements to the schoolchildren.

Schoolchildren in the first and the last years of elementary schools who were due to have their routine dental examinations under the free governmental oral health programs were selected for study. Signed, informed consent was obtained from the parents/guardians of children who participated in the study. Children with physical and mental handicaps or a history of serious illness and chronic medical conditions, such as cardiac disease, were excluded from the study.

The clinical examinations were conducted by 4 examining dentists in school dental clinics. Prior to commencement of the study, the examiners were trained in the use of the DDE index using color photographs that demonstrate a range of typical enamel defects. Intra- and interexaminer consistency testing was performed by each examiner repeating the dental examinations of 5 children who were not in the study on 3 separate occasions approximately a week apart. The Kappa statistic was computed from the results.[23] The teeth were cleaned with a toothbrush and lightly dried with air from the triple syringe prior to examination.

Visible surfaces of all teeth were examined and scored for enamel defects according to the criteria of the modified DDE index.[24] The DDE is a popular index for diagnosing developmental defects of enamel, as it distinguishes between defects that are observed as changes in the translucency of enamel (opacity), and defects that are visible as deficiencies in the quantity of enamel (enamel hypoplasia).[24] Enamel opacities can be further categorized as demarcated (Figure 1) if the borders of the lesion are well defined and diffuse if the lesion had no distinct borders[1] (Figure 2). Similarly, enamel hypoplasia can be classified as grooves (Figure 3), pits, or missing enamel (Figure 4).[24]

In the present study, if a tooth showed both enamel opacity and hypoplasia, it was classified as having enamel hypoplasia. Similarly, if a subject showed teeth with enamel hypoplasia and teeth with opacities, he/she was designated as having enamel hypoplasia. All visible surfaces of each tooth were examined and the results entered into a standard database. Enamel defects were differentiated from carious lesions by their clinical appearance and locations (usually not related to gingival margins or occlusal fissures). Statistical analyses were performed using the Fisher's exact test and student's *t* test, where appropriate, employing an alpha value of 0.05.

Results

The Kappa statistic for inter- and intraexaminer consistency ranged from 0.75 to 0.85. This represented the spread of consistency among examiners and within each examiner in their individual scores for all types of enamel defects. Consent rate for the study was over 87%. Medical histories revealed that all children in the study were healthy. Most reported daily tooth-brushing using age-appropriate fluoridated toothpaste. A total of 5 children were diagnosed with amelogenesis imperfecta based on their family histories and the clinical and radiographic appearance of the teeth of the subjects and their family members.[25] Of these: 3 exhibited the hypocalcified autosomal dominant variant; 1 exhibited the hypoplastic (smooth) X-linked dominant variant; and 1 exhibited the hypoplastic (pitted) autosomal dominant variant. These children were not included in the present study.

Overall prevalence. Of the 517 children examined, there were 163 (79 girls and 84 boys) with a mean age of 6.3 years who had a full primary dentition and 354 (215 girls and 139 boys) with a mean age of 13.5 years who had a full permanent dentition (excluding the third molars). As shown in Table 1, there were 244 subjects (47%) with at least 1 tooth with DDE, comprising 40 subjects with primary dentitions (25% of children with primary dentitions) and 204 with permanent dentitions (58% of children with permanent dentitions). The difference in overall prevalence between the primary and permanent dentition was statistically significant ($P<.001$). There were no significant differences in the mean numbers of affected teeth between primary and permanent dentitions (3.8±3.7 vs 4.5±5.1, $P>0.1$). In addition, there were no

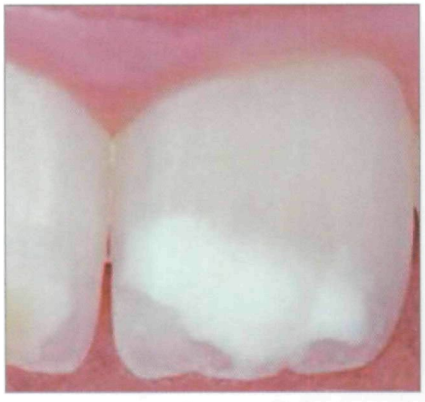


Figure 1. Demarcated opacity on the facial surface of a permanent maxillary central incisor.

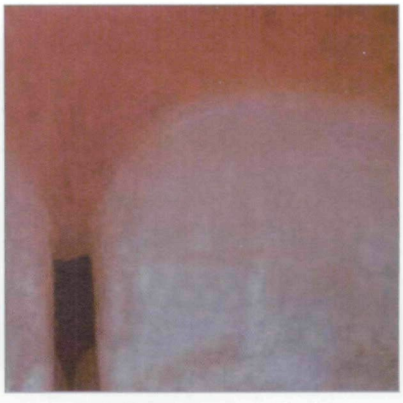


Figure 2. Diffuse opacity on the facial surface of a permanent maxillary central incisor.

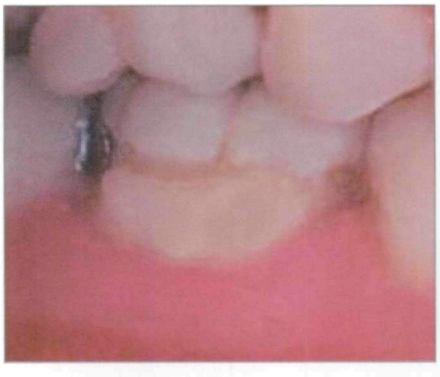


Figure 3. Enamel hypoplasia expressed as a horizontal groove on the buccal surface of a permanent first mandibular molar.

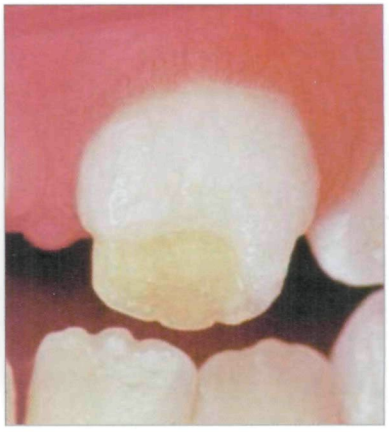


Figure 4. Enamel hypoplasia expressed as missing enamel on the facial surface of a permanent maxillary central incisor.

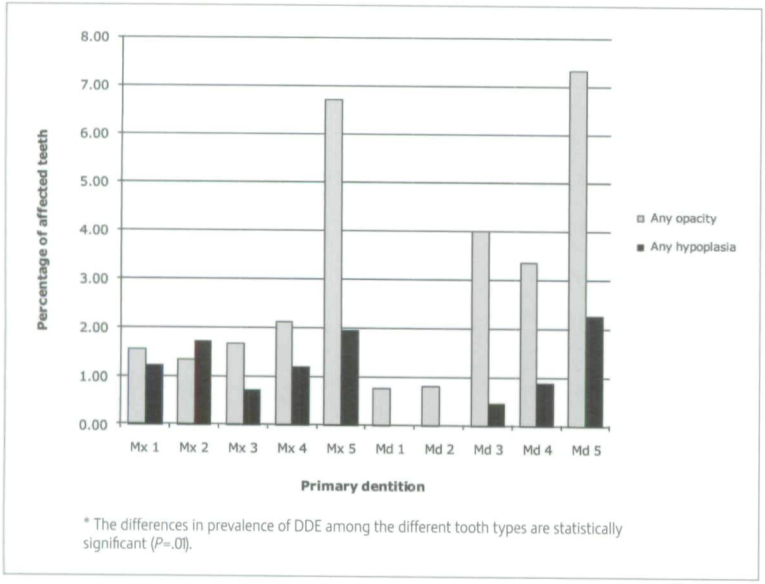
gender differences in the prevalence of enamel opacity or enamel hypoplasia in either the primary or permanent dentitions ($P>.10$).

Enamel opacities. As shown in Table 1, 169 children (33% of the total) had at least 1 tooth with enamel opacity. Of these, 24 subjects had primary dentitions (15% of all children with primary dentitions), and 145 subjects had permanent dentitions (41% of all subjects with permanent dentitions). The difference in prevalence between the primary and permanent dentition was significant ($P=.01$). Table 1 also shows that children showing demarcated opacities constituted 83% of those with opacities in the primary dentition compared to 51% of children with opacities in the permanent dentition ($P<.001$). In contrast, children with diffuse opacities constituted only 17% of those with opacities in the primary dentition and 49% of those with opacities in the permanent dentition ($P<.001$). The mean number of teeth affected by opacities per subject in the primary dentition was not significantly different compared to that of the permanent dentition (3.1±3.8 vs 3.6±4.7, $P>.10$; Table 1).

Enamel hypoplasia. Table 1 also shows the prevalence and presentations of enamel hypoplasia in children with primary and permanent dentitions. In total, 75 of 517 children (15%) showed enamel hypoplasia on at least 1 tooth. In the primary dentition, missing enamel was the most prevalent form of enamel hypoplasia and was seen in 8 of 16 children with enamel hypoplasia (50%). The next most common hypoplastic defect was grooves, observed in 5 children (31%), followed by enamel pits, observed in 3 subjects (19%). By contrast, in the permanent dentition, the most common hypoplastic defects were, in descending order: (1) enamel grooves, observed in 33 children (56%); (2) missing enamel, observed in 23 children (39%); and (3) enamel pits, observed in 3 children (5%). These differences in presentations between the primary and permanent dentitions were statistically significant ($P=.04$). The difference in the mean number of affected hypoplastic teeth between the permanent and primary dentitions, however, was not significant (0.8±1.4 vs 1.2±2.2, $P>.10$).

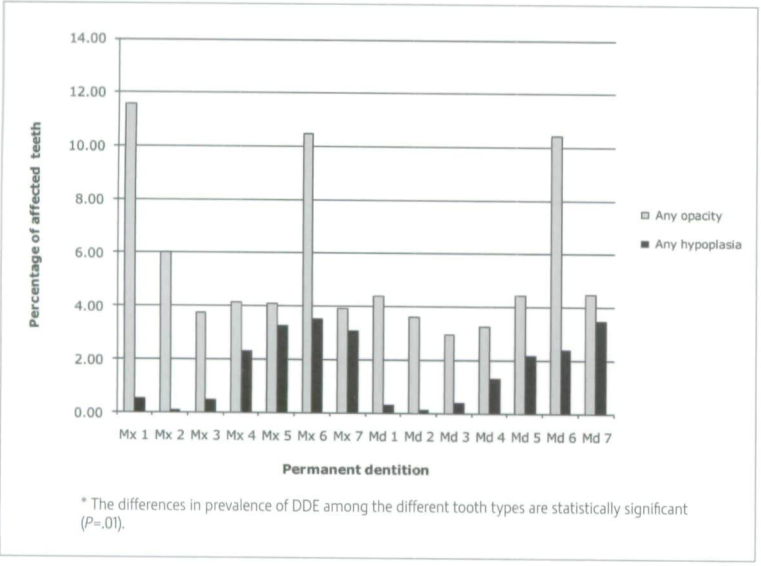
DDE prevalence by tooth type. Figure 5 shows the prevalence of enamel opacity and enamel hypoplasia in the primary dentition. Of the 3,260 primary teeth examined in the study, 152 (5%) had some form of DDE. As shown in Figure 5, in the primary dentition defects were most prevalent, in descending order, in the: (1) mandibular second molar (Md5); (2) maxillary second molar (Mx5); (3) mandibular canine (Md3); (4) mandibular first molar (Md4); (5) maxillary first molar (Mx4); (6) maxillary lateral (Mx2) and central incisors (Mx1); and (7) maxillary canine (Mx3). Only a very small percentage of the mandibular central and lateral incisors exhibited DDE, and these were limited to enamel opacities. The differences in prevalence among the various types of primary teeth were statistically significant ($P=.001$).

Figure 6 shows the prevalence of enamel opacity and enamel hypoplasia in the permanent dentition. Of the 9,912 teeth examined, 918 (9%) had some form of DDE. Permanent dentition defects were most prevalent, in descending order, on the: (1) maxillary first molars (Mx6); (2) mandibular first molars (Md6); (3) maxillary central incisors (Mx1); (4) mandibular second molars (Md7); (5) maxillary second premolars (Mx5); (6) maxillary second molars (Mx7); (7) maxillary lateral incisors (Mx2); (8) maxillary first premolars (Mx4); (9) mandibular second premolars (Md5); (10) mandibular central incisors (Md1); (11) mandibular lateral incisors (Md2); (12) mandibular first premolars (Md4); and (13) maxillary canines (Mx3). The mandibular canines were least affected. The differences in prevalence among the various types of permanent teeth were statistically significant ($P=.001$).



* The differences in prevalence of DDE among the different tooth types are statistically significant ($P=.01$).

Figure 5. Prevalence of developmental defects of enamel in the primary dentition by tooth type.*



* The differences in prevalence of DDE among the different tooth types are statistically significant ($P=.01$).

Figure 6. Prevalence of developmental defects of enamel in the permanent dentition by tooth type.*

Discussion

The results of the present study show that the prevalence rates of enamel opacities and hypoplasias in children from a low-fluoride urban community from Australia lie within the ranges previously reported in children from other developed countries[9,12,26-28] but are well below those observed in indigenous communities in the same country.[7,29] In addition, the present finding that the primary second molars, permanent first molars, and permanent central incisors are most commonly affected by DDE is consistent with most reports in the current literature.[11-14,28,30-32] The increased DDE risk of these teeth is probably related to a critical period of amelogenesis during the ages of 0- to 2-years-old, when the child is particularly vulnerable to a range of common systemic conditions that can affect enamel development.

The relatively high rates of enamel defects demonstrate the susceptibility of enamel to damage during development, and their clinical presentations suggest that both systemic conditions and local trauma are possible etiological factors in the present cohort of children.[2] As the present study children are generally healthy, the etiologic agents associated with the enamel defects are likely to be common childhood illnesses such as respiratory infections and chickenpox.[6,10] Other systemic insults that have been identified in other reports but are not likely to be found in the present healthy cohort include: chromosomosal disorders (eg, Down syndrome); metabolic conditions (eg, premature birth); endocrine disorders (eg, parathyroid conditions); nutritional deficiencies (eg, vitamin D deficiency); and chemical toxicity (eg, tetracyclines and anticancer medications).[6]

Enamel changes resulting from excessive systemic fluoride have been well reported as diffuse opacities in mild cases to extensive enamel hypoplasia and mottling in the severe forms.[20,33,34] In communities with high levels of natural fluoride and artificial fluoridation, direct correlations of fluoride levels in the water supply with prevalence and severity of enamel defects have been documented.[35,36] Previous investigations on fluorosis have focused mainly on the permanent dentition and reported that diffuse opacities are the most common form of enamel defects found in communities which have been exposed to the optimum ranges of fluoride in the drinking water.[27,33,34,36]

By contrast, the primary dentition is generally thought to be less affected by fluorosis, as most of the primary teeth develop prenatally when the fetus is partially protected from excessive fluoride levels.[20,37] The second primary molars which are largely formed post-natally, however, may be susceptible to excessive fluoride, and typical fluorotic diffuse opacities have been described in primary second molars of children who have been exposed to higher than optimal levels of fluoride from various sources.[20]

It is interesting to note that, in the present study, despite the lack of fluoridation in community water supplies, the prevalence rate of diffuse opacities of approximately 49% in permanent dentition is similar to those observed in communities with levels of optimally fluoridated water of approximately 0.7 to 1 ppm.[35,36,38] These findings suggest that the children in the present study

Table 1. ENAMEL OPACITIES AND HYPOPLASIA IN SUBJECTS WITH PRIMARY AND PERMANENT DENTITIONS

Subjects (N)	Primary	Permanent	Total	*P*-value (primary vs permanent; Fisher's exact test)*
	163	354	517	
Mean age±(SD) ys	6.3±0.7	13.5±1.3	9.9±1.3	
Enamel opacity				
Subjects with at least 1 tooth with opacity N (%)	24 (15)	145 (41)	**169 (33)**	<.001
No. of teeth per subject with at least 1 tooth with any opacity Mean±(SD)	3.1±3.8	3.6±4.7	**3.4±3.4**	NS
Subjects with at least 1 tooth with demarcated opacity N (%)	20 (83)	74 (51)	**94 (56)**	.02
No. of teeth per subject with demarcated opacity Mean±(SD)	1.9±2.2	1.3±1.9	**1.5±1.9**	NS
Subjects with at least 1 tooth with diffuse opacity N (%)	4 (17)	71 (49)	75 (44)	.001
No. of teeth per subject with diffuse opacity Mean±(SD)	1.9±7.2	2.3±2.3	**1.6±1.5**	NS
Enamel hypoplasia				
Subjects with at least 1 tooth with any enamel hypoplasia N (%)	16 (10)	59 (17)	75 (15)	.04
No. of teeth per subject with enamel hypoplasia Mean±(SD)	0.8±1.4	1.2±2.2	**0.8±1.8**	NS
Subjects with at least 1 tooth with enamel grooves N (%)	5 (31)	33 (56)	**38 (48)**	.01
No. of teeth with enamel hypoplasia per subject Mean±(SD)	0.3±1.1	0.8±2.2	**0.4±1.4**	NS
Subjects with at least 1 tooth with enamel pits N (%)	3 (19)	3 (5)	6 (7)	NS
No. of teeth per subject with enamel pits Mean±(SD)	0.1±0.4	0.1±0.3	**0.1±0.3**	NS
Subjects with missing enamel N (%)	8 (50)	23 (39)	**31 (38)**	NS
No. of teeth per subject with missing enamel Mean±(SD)	0.5±1.1	0.2±0.6	**0.3±1.0**	NS
Total subjects with all enamel defects N (%)	**40 (25)**	**204 (58)**	244 (47)	**<.001**
No. of teeth per subject with any enamel defect Mean±(SD)	**3.8±3.7**	**4.5±5.1**	**4.2±5.1**	**<.001**
No. of enamel opacities per subject vs number of hypoplasia per subject	*P*<.001 t=7.25 df=324	*P*<.001 t=8.20 df=706	*P*=.001 t=5.37 df=1,032	

*NS=nonsignificant.

may have consumed other sources of systemic fluoride, such as toothpaste or foods and beverages manufactured using fluoridated water.[39] On the other hand, as the ameloblasts can respond to different types of injury in similar ways, it is also possible that the diffuse opacities in the permanent dentition of the present cohort of children could have resulted from other types of systemic insults, such as infections or the antibiotics used to treat the infections (eg, amoxicillin).[40,41] In other permanent teeth, the relatively high prevalence of enamel grooves, which are usually associated with episodic acute systemic illnesses also may provide indirect evidence for the significant impact of non-fluoride causes of enamel defects.[2]

In contrast to the permanent dentition, the primary dentition in the present study showed a relatively low prevalence of diffuse opacities, and the predominant enamel defect is the demarcated opacity. Demarcated opacities can be produced experimentally in sheep through the application of local trauma.[2] Furthermore, the location of these defects on primary canine facial surfaces, where the facial cortical bone is thinnest,[12] has led to the general belief that demarcated opacities in primary teeth result from common oral trauma associated with falls or biting on hard objects before the teeth erupt.[42] Other evidence for a local etiology for enamel defects in the primary dentition can be obtained from reports of preterm children where trauma from laryngoscopy and endotracheal intubation is associated with enamel defects of the primary maxillary incisors.[43] In the permanent dentition, demarcated opacities—which constitute approximately half of all opacities in the present study also are generally thought to result from local injury such as trauma or infection to a developing tooth.[27]

This study is limited by its cross-sectional nature and the problem of accurate detail in the recall of medical histories of the individual children. On the other hand, it provides the first available prevalence data for enamel defects for both primary and permanent dentitions in a low-fluoride community in Australia before the commencement of water fluoridation. Thus, the present data can be usefully compared with post-fluoridation results to determine changes in the prevalence of enamel hypoplasia that are associated with the fluoridation of community water supplies which has commenced in the present community since the collection of the research data.

The present finding that 25% of children with primary dentitions and 58% with permanent dentitions have an average of approximately 4 DDE teeth each demonstrates the potential impact of DDE on the oral health of children. In addition to their well-known complications of compromised esthetics, sensitivity, and tooth structure loss, enamel hypoplasia is increasingly recognized as a significant risk factor for early childhood caries, particularly in children from low socioeconomic areas and indigenous communities.[5,7]

The association between enamel defects and caries suggests that the high prevalence of DDE is likely to contribute to the relatively high caries rates reported in this community vs other parts of Australia.[21] In addition, the presence of enamel defects also poses an increased risk to erosion lesions and may explain the higher rates of erosion noted in communities with enamel defects.[4] The relatively high prevalence rates of DDE and the potential of DDE to affect the oral health suggest that DDE be included as an indicator of dental health status in oral epidemiological studies of children.

Conclusions

Based on this study's results, the following conclusions can be made:

1. In a low-fluoride community in Australia, developmental defects of enamel (DDE) were observed in 25% of children in the primary dentition and 58% in the permanent dentition. Approximately 4 affected teeth were observed in each child.
2. Most DDE in both primary and permanent dentitions presented as opacities.
3. The demarcated opacity was most common in the primary dentition, whereas demarcated and diffuse opacities were equally represented in the permanent dentition.
4. Missing enamel was the most common hypoplastic defect in the primary dentition, whereas grooves were most prevalent in the permanent dentition.

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