Enamel defects and dental caries among children attending primary schools in Poznań, Poland


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A − research concept and design; B − collection and/or assembly of data; C − data analysis and interpretation; D − writing the article; E − critical revision of the article; F − final approval of the article

Abstract

Background. Both positive and negative associations between developmental enamel defects (DED) and dental caries have been reported in the literature.

Objectives. The aim of this study was to assess the prevalence of DED of permanent dentition and its association with dental caries in schoolchildren living in Poznań (Wielkopolskie Voivodeship, Poland).

Material and methods. A total of 2,522 6th grade children and 3,112 1st grade children were examined. Developmental enamel defects were described using the modified Developmental Defects of Enamel Index. Dental caries experience was assessed in accordance with the number of decayed, missing and filled teeth (DMFT).

Results. The study revealed 475 children (9.6%) to have at least 1 enamel defect of permanent dentition. In 6th-graders, statistical analysis confirmed significant differences between DMFT, DT (decayed teeth) and FT (filled teeth) numbers of various DED groups with subjects affected by diffuse opacities having generally the lowest caries indices and subjects with enamel hypoplasia and/or demarcated opacities having the highest caries indices. In both age groups, dental caries prevalence was statistically significantly higher in subjects with hypoplasia and/or demarcated opacities as compared to subjects without DED or with diffuse opacities (p < 0.05).

Conclusions. The prevalence of DED in the examined population was low and comparable to those reported in regions without fluoridated water. The study confirmed that children affected by diffuse enamel opacities were less susceptible to dental caries, while demarcated opacities and hypoplasia should be considered important dental caries risk factors.

Key words: dental caries, tooth abnormalities, enamel hypoplasia, dental fluorosis
Introduction

Scientific research has demonstrated that ameloblasts, which are secretory cells that produce tooth enamel, are highly sensitive to changes in their environment. Since enamel is formed only during a certain period of tooth development, dysfunction of ameloblasts may lead to permanent morphological consequences, namely, developmental enamel defects (DED). Defective formation of the enamel matrix results in hypoplasia, a quantitative defect, presented as a reduced thickness of enamel. Defective calcification of an otherwise normal fully developed organic enamel matrix produces qualitatively defective enamel (hypomineralization). Clinically, enamel with hypomineralization has normal thickness, but changed translucency, which is presented as white, yellow, or brown diffuse, or demarcated opacities. Diffuse opacities spread over the enamel surface without a clearly defined margin, while demarcated opacities have distinct boundaries with the adjacent normal enamel.1-4

A very wide spectrum of etiological factors, including genetic, systemic, local, and environmental factors, may lead to the development of DED. Defects caused by genetic factors form a separate entity, usually affecting both the primary and permanent teeth. They are less common than those resulting from acquired causes. As far as environmental factors are concerned, fluoride exposure has been reported to be the main determinant of the prevalence of DED in the given population.1,8 Several studies have also shown that teeth are very sensitive to the effects of dioxins, which arrest the degradation and removal of enamel matrix proteins.2,5 Other etiological factors of DED include various metabolic disturbances, prematurity, irradiation, fever, infections, nutritional deficiencies, and direct traumas to cells, which lead to unfavorable changes in the environment of enamel-forming cells.1,3,5

Developmental enamel defects with a similar appearance are not necessarily caused by similar agents and the same insult can produce different defects depending on the stage of tooth development.1 However, some types of DED are usually linked to specific etiological factors. The etiology of the diffuse opacities is generally thought to be associated with excessive fluoride exposure during tooth development.2,7 Demarcated opacities and hypoplasia are related to systemic conditions influencing teeth development, such as infections, antibiotic therapy, nutritional deficiencies, low birth weight, and exposure to dioxins.3,4,8,9 Also, the common causes of localized hypomineralization or hypoplasia in permanent dentition are chronic periapical infections and traumatic injuries within deciduous predecessors.10,11

Developmental enamel defects have a significant impact on oral health, compromising esthetics, increasing tooth sensitivity and altering occlusal functions. In addition, many studies have highlighted the possibility of some types of DED being important risk factors for caries and erosions of the hard dental tissues.12-16 Enamel hypoplasia and demarcated opacities have been frequently reported to increase dental caries experience, due to the irregular, retentive surfaces leading to plaque accumulation and higher acid solubility of the affected enamel, respectively.12,13,16

Data concerning prevalence of enamel defects in Poland is sparse, since children are not routinely screened for DED in Polish national epidemiological surveys.

The aim of the present study was to assess the prevalence of various types of DED in permanent dentition and its association with dental caries in the population of Polish children living in the city of Poznań (Wielkopolskie Voivodeship, Poland).

Material and methods

The Ethical Committee of Poznan University of Medical Sciences granted its approval for this study (Resolution No. 466/10). The study involved the assessment of DED prevalence and dental caries experience in the city of Poznań (Wielkopolskie Voivodeship, Poland) and was a part of dental screening financed from the Poznań City Council’s budget. Prior to the study, the parents of all children had received a letter about the examination and had been asked to give consent for their children participation in the investigation.

Schoolchildren of grades 1 and 6 (5–8-year-olds and 11–15-year-olds) were examined for DED and dental caries. Of the overall number of 8,165 1st- and 6th-grade children attending 81 public primary schools in Poznań, Poland, 5,634 children from 75 schools could enter the study. The remaining pupils were either not present on the day of the examination, or the school director or parents had not signed the consent for examination.

A total of 2,522 6th-grade children (1,343 girls and 1,179 boys) and 3,112 1st-grade children (1,572 girls and 1,540 boys) were examined, with 705 excluded, as they did not have permanent 1st molars fully erupted, leading to the final sample size of 4,929 children.

The mean age of the younger subjects was 6.9 ±0.4 years (± standard deviation [SD]; range 5–8 years), while the mean age of the older subjects was 12.02 ±0.22 years (± SD; range 11–15 years).

According to the information from the Sanitary Inspection, the natural level of fluoride in the tap water in Poznań in 1995–2005, measured at various points of the water mains, ranged from 0.3 to 0.9 mg/L.

In Poland, parents and their children are subject to compulsory national health insurance. Professional oral health care for children is covered by this insurance. Since 2003 all primary schoolchildren have been included in the school program of supervised brushing with 1.25% fluoride gel (6 times per year).

Dental examinations were conducted in classrooms at the beginning of 2010 by 7 calibrated dentists from the
Department of Pediatric Dentistry of Poznan University of Medical Sciences. Subjects were examined under the following conditions: the child was seated in a chair, and an examiner stood in front of the chair with a headlamp, a mouth mirror and a WHO probe.

Developmental enamel defects were examined using the modified Developmental Defects of Enamel Index for screening surveys, recording the categories of demarcated and diffuse opacities and hypoplasia. Teeth were inspected wet without previous professional cleaning. The status of the subject was defined according to the most severe defect seen in the subject. If a subject showed teeth with diffuse and demarcated opacities, they were designated as having demarcated opacities. Having teeth with opacities and teeth with hypoplasia was designated as having enamel hypoplasia. The prevalence of DED was determined by the inclusion of any individual who has been found to have at least 1 tooth affected by the condition.

The dental caries experience was assessed in accordance with the number of decayed (DT), missing (MT) and filled teeth (FT) calculated for all permanent teeth as the number decayed, missing and filled teeth (DMFT). Caries prevalence was calculated as a percentage of individuals with DMFT >0.

Caries diagnosis was carried out by visual and tactile examination, using an artificial light, a mouth mirror and a blunt dental probe. Active caries was recorded as present when the respective lesion showed an unmistakable cavity, undermined enamel or a detectably softened area. A probe was used to confirm the visual evidence of caries. Areas with visual evidence of demineralization, presenting no soft surface, were considered sound. A dental explorer was used for detecting the cavities on the proximal surfaces. Apart from that, if caries in dentine was visualized as a loss of translucency producing a shadow in a calculus-free and stain-free proximal surface, it was recorded as proximal decay, too. A tooth filled due to decay was recorded when a tooth had at least 1 permanent restoration placed to treat caries. Fissure sealants were not included in the FT component of the DMFT. The MT component was recorded when a tooth had been extracted due to caries complications (verified by interview).

Prior to the examination, a calibration exercise was conducted between examiners on a group of 30 patients, apart from the main study. The inter- and intra-examiner concordances assessed with Cohen’s kappa coefficient were all above 0.75.

Data analysis was performed with STATISTICA v. 12 (StatSoft Inc., Tulsa, USA) for Windows 10, assuming p < 0.05 as the level of statistical significance.

Statistical significance for differences between proportions was assessed using the χ² test.

After performing the Shapiro-Wilk normality test, a non-parametric Kruskal-Wallis analysis of variance (ANOVA) was used to assess any differences in the mean DMFT, and its DT, MT and FT components of different DED groups. If a statistical difference was detected, post hoc non-parametric multiple comparisons of mean ranks procedures were used to compare individual pairs of means. In order to compare the risk of having caries between different DED groups, the odds ratio (OR) was calculated.

### Results

Table 1 shows the prevalence of various types of DED as well as the effects of gender and age on the prevalence of enamel defects.

Out of the 4,929 children in the study, 475 children (9.6%) had at least 1 enamel defect of permanent dentition. Overall, the prevalence of DED in the older group was statistically significantly higher as compared to prevalence of DED in younger children (p = 0.0000). The presence of DED was not significantly associated with gender (p > 0.05).

No cases of generalized enamel defects, suggestive of genetically determined amelogenesis imperfecta, were detected in this study.

The prevalence of enamel hypoplasia was statistically significantly lower as compared to the prevalence of both types of opacities (p < 0.001). The percentage of subjects having diffuse and demarcated opacity was similar (3.7 vs 4.0, respectively).

Table 2 summarizes dental caries indices in 2 age groups according to different DED variables. Statistically significant differences were detected in both age groups. In the younger group, Kruskall-Wallis ANOVA detected statistically significant differences between DMFT, DT and FT.

<table>
<thead>
<tr>
<th>Type of DED</th>
<th>Hypoplasia</th>
<th>Demarcated opacity</th>
<th>Diffuse opacity</th>
<th>Without DED</th>
<th>χ² test (subjects with DED vs subjects without DED)</th>
</tr>
</thead>
<tbody>
<tr>
<td>Subjects</td>
<td>n</td>
<td>%</td>
<td>n</td>
<td>%</td>
<td>n</td>
</tr>
<tr>
<td>1st-graders</td>
<td>46</td>
<td>1.9</td>
<td>64</td>
<td>2.7</td>
<td>73</td>
</tr>
<tr>
<td>6th-graders</td>
<td>48</td>
<td>1.9</td>
<td>135</td>
<td>5.4</td>
<td>109</td>
</tr>
<tr>
<td>Females</td>
<td>48</td>
<td>1.9</td>
<td>110</td>
<td>4.3</td>
<td>102</td>
</tr>
<tr>
<td>Males</td>
<td>46</td>
<td>2.0</td>
<td>89</td>
<td>3.8</td>
<td>80</td>
</tr>
<tr>
<td>Total</td>
<td>94*</td>
<td>1.9</td>
<td>199</td>
<td>4.0</td>
<td>182</td>
</tr>
</tbody>
</table>

DED – developmental enamel defects; *p = 0.0000 as compared to subjects with demarcated opacity or diffuse opacity.
numbers in groups affected by different types of DED \((p < 0.0001; p = 0.0001; p = 0.0181\), respectively). Post hoc non-parametric multiple comparisons of mean ranks confirmed this statistically significant difference only for DMFT of subjects affected by demarcated opacities and/or hypoplasia, as compared to subjects without DED. In 6th-graders, post hoc tests confirmed statistically significant differences between DMFT, DT and FT numbers of various DED groups with subjects affected by diffuse opacities having generally the lowest caries indices and subjects with enamel hypoplasia and/or demarcated opacity having the highest caries indices.

In both age groups, dental caries prevalence was statistically significantly higher in subjects with hypoplasia and/or demarcated opacity as compared to subjects without DED or with diffuse opacities \((p < 0.05)\).

Estimation of the OR showed that children with demarcated or hypoplastic defects had several folds higher risk of having caries compared with those without DED \((OR = 2.83\) for 1st-graders, \(OR = 2.51\) for 6th-graders) or those who had diffuse opacities \((OR = 2.31\) for 1st-graders and \(OR = 4.24\) for 6th-graders). Older subjects without DED had a higher risk of having caries as compared to those with diffuse opacities \((OR = 1.69)\).

**Discussion**

The overall prevalence of DED in permanent dentition (9.6% of the affected subjects) is at the lower end of the findings from other studies carried out in areas with and without fluoridated drinking water. Similar rates of prevalence were observed in children residing in low fluoride area of Naples (Italy) and Campeche (Mexico) \((9.8%\) and \(7.5%\), respectively), while much higher figures were reported during a multicenter epidemiological study \((from 43\% in Greece to 70\% in the Netherlands) and in a population of 14-year-old boys in Saudi Arabia \((75\%)\).\(^{17-20}\)

Examinations of 1st-grade and 6th-grade children in the Śrem Commune (Wielkopolskie Voivodeship, Poland), where the level of fluoride in the drinking water ranges from 0.1 to 0.4 mg/L, revealed 25.7% of the subjects to have at least 1 permanent 1st molar or incisor affected by DED.\(^{21}\)

Prevalence rates of DED in areas with water fluoridation ranged from 26.1% for residents of the city of Araquara, Brazil, up to 92.1% for children in Hong Kong.\(^{22-23}\)

Researchers examining fluoridated communities reported a higher prevalence of diffuse defects, which should be attributed to the higher fluoride exposure of children during critical periods of enamel formation. In the study by Arrow et al. (2008), 47% of 7-year-old schoolchildren from the fluoridated region of Western Australia had permanent 1st molars affected by white diffuse opacities and in the study by Milsom et al. (1996), 40% of 8–9-year-olds from fluoridated Cheshire (UK) had diffuse defects on the permanent incisors.\(^{24-25}\)

In Poland, drinking water is not artificially fluoridated. In most of the country, fluoride concentration in drinking water is below 0.3 mg/L, although in some localities it exceeds 1.0 mg/L.\(^{26}\) Fluoride content in the water of the city of Poznań in 1995–2005 ranged from 0.3 to 0.9 mg/L,

**Table 2. Dental caries prevalence (%) of permanent dentition, DMFT numbers in various DED groups of younger and older subjects**

<table>
<thead>
<tr>
<th></th>
<th>Subjects without DED</th>
<th>Subjects with demarcated opacities or hypoplasia</th>
<th>Subjects with diffuse opacities</th>
<th>p-value (non-parametric Kruskal-Wallis ANOVA)</th>
</tr>
</thead>
<tbody>
<tr>
<td>1st-grade children</td>
<td></td>
<td></td>
<td></td>
<td></td>
</tr>
<tr>
<td>N</td>
<td>2,224</td>
<td>110</td>
<td>73</td>
<td></td>
</tr>
<tr>
<td>DMFT</td>
<td>0.17 ±0.59</td>
<td>0.38 ±0.80(^a)</td>
<td>0.18 ±0.51</td>
<td>0.0000</td>
</tr>
<tr>
<td>DT</td>
<td>0.11 ±0.45</td>
<td>0.28 ±0.73</td>
<td>0.03 ±0.16</td>
<td>0.0001</td>
</tr>
<tr>
<td>MT</td>
<td>0.00 ±0.00</td>
<td>0.00 ±0.00</td>
<td>0.00 ±0.00</td>
<td>NA</td>
</tr>
<tr>
<td>FT</td>
<td>0.06 ±0.35</td>
<td>0.10 ±0.41</td>
<td>0.15 ±0.49</td>
<td>0.0181</td>
</tr>
<tr>
<td>Caries prevalence</td>
<td>10.30</td>
<td>24.55(^b)</td>
<td>12.33</td>
<td>NA</td>
</tr>
<tr>
<td>6th-grade children</td>
<td></td>
<td></td>
<td></td>
<td></td>
</tr>
<tr>
<td>N</td>
<td>2,230</td>
<td>183</td>
<td>109</td>
<td></td>
</tr>
<tr>
<td>DMFT</td>
<td>1.50 ±1.78</td>
<td>2.15 ±1.88(^c)</td>
<td>1.04 ±1.47(^d)</td>
<td>0.0000</td>
</tr>
<tr>
<td>DT</td>
<td>0.56 ±1.17</td>
<td>0.67 ±1.25</td>
<td>0.14 ±0.37(^e)</td>
<td>0.0002</td>
</tr>
<tr>
<td>MT</td>
<td>0.02 ±0.16</td>
<td>0.03 ±0.21</td>
<td>0.02 ±0.19</td>
<td>0.4352</td>
</tr>
<tr>
<td>FT</td>
<td>0.91 ±1.36</td>
<td>1.45 ±1.65(^f)</td>
<td>0.88 ±1.43</td>
<td>0.0000</td>
</tr>
<tr>
<td>Caries prevalence</td>
<td>57.94(^g)</td>
<td>77.60(^h)</td>
<td>44.95</td>
<td>NA</td>
</tr>
</tbody>
</table>

ANOVA – analysis of variance; OR – odds ratio; DED – developmental enamel defects; N – number of subjects; DMFT – number of decayed, missing and filled teeth; DT – number of decayed teeth; MT – number of missing teeth; FT – number of filled teeth; \(^*p = 0.0371\) as compared to subjects without DED; \(^a,p = 0.0000\) as compared to subjects without DED \((OR = 2.83)\), \(p = 0.0418\) as compared to subjects with diffuse opacities \((OR = 2.31)\); \(^b\) \(p = 0.0000\) as compared to subjects without DED and subjects with diffuse opacities \((OR = 2.31)\); \(^c\) \(p = 0.0216\) as compared to subjects without DED, \(p = 0.0000\) as compared to subjects with demarcated opacities or hypoplasia; \(^d\) \(p = 0.0087\) as compared to subjects without DED, \(p = 0.0048\) as compared to subjects with demarcated opacities or hypoplasia; \(^e\) \(p = 0.0000\) as compared to subjects without DED, \(p = 0.0015\) as compared to subjects with diffuse opacities; \(^f\) \(p = 0.0000\) as compared to subjects without DED \((OR = 2.51)\) and subjects with diffuse opacities \((OR = 4.24)\); \(^g\) \(p = 0.0075\) \((OR = 1.69)\) as compared to subjects with diffuse opacities; NA – not applicable; OR – odds ratio.
which is close to WHO recommendations on the optimal fluoride level in the drinking water (0.5–1.0 mg/L). Apart from drinking water, the most common source of fluoride in Poland is toothpaste with an age-related concentrations between 250 and 1500 mg/L. Dietary fluoride supplements are available only on prescription and intended for use by children living in nonfluoridated areas.

In the present study, the prevalence of diffuse opacities (3.7%) was lower as compared to those reported in the drinking water of other communities with similar fluoride content, which suggests that total fluoride exposure of most examined children did not exceed limits above which fluorosis may develop. However, children in this study were examined at schools, under headlamp lighting and without previous cleaning of the teeth, which could significantly affect the number of detected anomalies. It must be remembered that wide variations of reported values of DED prevalence in the literature might be attributed to the differences in the study conditions, as well as the use of various terminologies and diagnostic criteria to describe the enamel defects. The diagnosis of DED can be influenced by the type of light source used for the examination, brushing or drying of teeth before the examination, additional photographs taken, as well as examination of only a particular group of teeth or the whole dentition.15,18

As far as the environmental risk factors of demarcated opacities are concerned, studies of Alaluusua et al. have shown that polychlorinated dibenzo-p-dioxins and dibenzofurans in mother's milk may cause hypomineralization defects in the child's permanent 1st molar teeth, while other studies did not confirm this association. Nevertheless, Jaraczewska et al. proved that the levels of dioxin-like compounds in the human milk samples from Wielkopolskie Voivodeship are lower than those reported in other European countries.29

Regarding gender, studies are in conflict as to whether boys or girls are more affected by DED.13,16,30,31 Our present study failed to demonstrate any significant difference in the prevalence of DED between girls and boys.

Differences in the prevalence of DED in both age groups could be explained from the biological point of view: given that as age increases, more permanent teeth erupt, the probability of finding teeth with DED also increases. On the other hand, in the older group, some enamel defects in the child's permanent 1st molar teeth, while other studies did not confirm this association. Nevertheless, Jaraczewska et al. proved that the levels of dioxin-like compounds in the human milk samples from Wielkopolskie Voivodeship are lower than those reported in other European countries.29

Conclusions

The prevalence of DED in examined population was comparable to those reported in regions without fluoridated water. The study confirmed that children affected by diffuse enamel opacities were less susceptible to dental caries, while demarcated opacities and hypoplasia should be considered important dental caries risk factors. The findings emphasize the need for continuous promotion of the proper use of fluorides, as well as intensification of dental caries prevention in patients affected by caries-prone enamel defects.

References


