Relationship Between Developmental Enamel Defects and Caries in 18-Year-Old Adolescents

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Research Article

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Abstract

Objective: The aim of the cross-sectional study was to assess the potential relationship between developmental defects of the enamel (DDE) and caries in adolescents aged 18 year.

Materials and methods: A total of 1,611 individuals aged 18 years were selected based on randomisation to form a nationally representative sample from all provinces of Poland. Dental evaluation was performed by 22 trained and calibrated dentists. DDE and caries were assessed using the modified DDE index, MIH Treatment Need Index, FDI and WHO criteria. T-test was used for comparing group means. A simple and multiple logistic regression were used to assess the relationship between DDE and caries ($p < 0.05$).

Results: The prevalence of DDE was 13.7%. Demarcated opacities (DEO) were the most frequent (9.65%); 4% had diffuse opacities (DIO), 1.5% had hypoplasia and 0.6% - MIH. The prevalence of caries was 93.2%, with mean DMFT of 6.50±4.22. The DMFT and DMFS values were: 7.52±4.77 and 11.31±8.94 in the group of patients with DEO; 7.85±4.74 and 11.88±9.83 with DIO and 7.56 ±4.57 and 10.96±8.77 with enamel hypoplasia, respectively. There was an association between the presence of caries and DDE ($p < 0.001$): DEO ($p = 0.001$) and DIO ($p = 0.038$), and between DDE and DMFT ($p < 0.001$).

Conclusions: Caries and DDE are prevalent and influenced the dental health of 18-year-old adolescents. The presence of DDE had significant association with caries severity in 18-year-old adolescents. Adolescents with DDE had a higher probability to develop carious lesions.

Clinical Relevance: DDE is associated with dental caries.

Introduction

Enamel morphogenesis is a complex process, which starts with enamel matrix protein secretion, followed by mineralisation and maturation. The process begins at the cusps and incisal parts of the crowns, progressing towards the cervical areas of the teeth. Disturbances in the different stages of enamel formation may result in a range of macroscopic and structural changes [1].

Defective formation of the enamel matrix leads to hypoplasia, a quantitative defect, which is clinically manifested by generalised enamel thinning or pitting defects, grooves or local loss of enamel. Defective calcification leads to hypomineralisation, a qualitative enamel density defect presenting *in vivo* as changes in colour and translucency of the enamel in the form of either demarcated opacities, with clearly defined margins, or diffuse opacities, without clear borders1. Developmental Defects of Enamel (DDE) are clinically manifested as white/cream enamel opacity (demarcated opacities (DEO), diffuse opacities (DIO), hypoplasia (Hypo)), associated with opacity or their combination [1–3].

The possible aetiology of DDE includes a number of genetic, systemic, environmental and local factors [1–6]. Fluoride level in the water in Poland is below 0.5 ppm F/l. The risk of fluorosis also seems to be low [7]. Studies demonstrated a relationship between a history of certain systemic diseases in early childhood (anemia, rubella, rickets, tetany, kidney and liver diseases, allergy, diarrheas, as well as administration of tetracycline and iron supplements), and an increased incidence of DDE in permanent dentition1. The influence of local and sociodemographic factors is also suggested [4].

Epidemiological data shows high rates and severity of caries in the country. Over 16 years (2001 vs 2017), the prevalence of caries among 18-year-olds decreased only by 4.2% (from 97.4–93.2%), and the mean DMFT decreased by 0.8 (from 7.3 to 6.5) [8].

A number of studies point to the possible relationship between DDE and the prevalence of dental caries [9–20]. Developmental enamel defects may play an important role in increasing the susceptibility to caries development. Increased rates of caries in teeth presenting with opacity and hypoplasia are due to increased microporosity and poor mineralisation, as well as increased accumulation of dental plaque [21].

Psychological well-being and Oral Health Related Quality of Life (OHRQoL), may be associated with aesthetic perception of teeth affected by the abnormal discoloration, tooth morphology, hypersensitivity and dental pain associated with developmental enamel defects [21, 22]. Some adolescents of Sujak et al. [21] study were dissatisfied with the condition of teeth affected by DDE. Among subjects who expressed dissatisfaction, 18.8% reported covering their mouths when smiling, 8.7% avoided going out with friends and 39.1% had consulted their dentists [21]. Results of studies indicated that the presence of DDE may cause negative impacts on a one's perception of oral health and on their daily performance [22, 23].The patients can suffer with pain, difficulties in eating, tooth brushing, or in anaesthetising. Dental fear and anxiety, as well as dental behaviour problems as a consequence of DDE, have been reported [23].

There is limited information about DDE in adolescents. The majority of studies concerning enamel defects have investigated children up to 12–14 years of age only [9–12, 16–18, 24]. Therefore very little information is available about the extent and severity of DDE in the full permanent dentition taking into account longitudinal changes with time in the epidemiological profile of DDE. This indicates that older age groups should be investigated as well. Comparing to healthy individuals, adolescents affected by DDE would need intensified professional prevention and treatment.

The aim of the cross-sectional study was to assess the potential relationship between different types of developmental defects of the enamel (DDE) and caries on a national sample of adolescents aged 18-year-old living in all sixteen provinces of Poland. The outcome variable were DDE and DMFT.

Materials And Methods

This was cross-sectional study involving questionnaire and clinical examinations. Adolescents aged 18 years (both genders) were selected based on three-layered randomisation to form a representative sample of population and enrolled in an epidemiological cross-sectional study conducted from October to November 2017 as part of monitoring of the health status of the Polish population [4, 8].

Study Population

In all sixteen provinces of Poland, administrative divisions of second level (counties) and third level (communes that are classified as urban or rural) were randomly chosen. The population study group was defined by a three-stage cluster sampling procedure: selection of states (the first large cluster), then selection of samples of schools (second-level cluster), followed by samples of groups (third-level cluster) and finally samples of 18-year-old adolescents. A minimum sample size as 1210 subjects was calculated based on the total number of 18- year-old adolescents living in Poland and their developmental enamel defects prevalence (15%±2.% margin of error at 95% confidence level). The attendance in the survey was voluntary, however the approval from the concerned school authorities was obtained. The inclusion criteria were: age, written consent for participation in the study and a completed questionnaire containing questions about socioeconomic factors (place of residence, level of parental education, economic status), as well as the presence at school on the day of study. Adolescents with disability and illnesses were excluded from the study. Adolescents who were currently using or had previously used permanent orthodontic appliances were excluded as well.

The Questionnaire

The survey was conducted with the use of a questionnaire including questions about sex, place of residence (urban/rural), mothers' education level, family economic status. The questionnaire was designed by the principal investigator (DO-K) and partly based on the previously used questionnaires in Polish national oral health surveys continued since 1999, follows the WHO recommendations [25]. Self-assessment of the socio-economic status (SES) included mother's education and the economic status. Based on classification of education, the authors divided to four categories: basic/primary, vocational, secondary and post-secondary, and higher. Based on respondent's subjective evaluation, the family economic status was divided into three levels high, middle and low levels.

Clinical Examination

Clinical examination of teeth was performed in artificial light (Dental 3W Mobile Portable Surgical Exam Light Medical Examination Lamp JSF-JC02; Illumination: 16000 Lux) using plane mouth mirrors (Hinte; ISO 9001:2008, CE MARK) and CPI (Community Periodontal Index) probe (Hinte; CE and ISO 9001:2015) in accordance with the WHO standard for epidemiological surveys (0.5 mm ball tip) [25]. Prior to the clinical examination the participants brushed their teeth. Caries was assessed using WHO criteria for the permanent dentition (DMF/T and DMF/S) [25]. Developmental enamel defects involving the labial/buccal surface of the teeth, based on the appearance and extension were assessed according to the modified Developmental Defects of Enamel (mDDE) index [26–30]. DDE was classified as demarcated white, yellow or brown opacities, diffuse opacities, and hypoplasia. Enamel opacity was considered to be a distinct change in translucency of enamel. A diagnosis of enamel hypoplasia was made when there was evidence of deficiency in enamel formation seen clinically as localized or generalized pits and grooves on the surfaces of teeth. Demarcated enamel opacities, surface breakdowns of hypomineralised enamel and atypical fillings and tooth extractions due to molar-incisor hypomineralisation (MIH) were diagnosed according to the criteria of the EAPD and the new 4-step MIH Treatment Need Index (MIH-TNI) [26, 29, 30]. DDE/MIH-associated defects were not scored in the DMF/T and DMF/S indexes. Hypomineralised lesions with a diameter < 1 mm were not recorded. Other enamel defects were excluded.

Dental evaluation was performed by 22 trained and calibrated dentists. Before the study, theoretical and clinical training and calibration focussed on caries lesions, DDE and MIH was undertaken with all 22 dentist and supervised by an experienced dentist (DO-K). The theoretical training provided information about study design, indices and diagnostic principles. This training was followed by a clinical course in which each of the investigators of sixteen teams from each province of Poland, consisting of two dentists, specialists in paediatric dentistry, with many years of experience, examined 18-year-olds under supervision of an experienced dentist (DO-K). Each paediatric dentist (examiner) independently examined the same group of ten patients. To determine the reliability of all investigators inter- and intra-examiner Kappa values were calculated; they were found to be in a good to excellent order of magnitude. Approximately 5% of the children were randomly selected for re-examination to continuously monitor the intra- and interexaminer reliabilities during the surveys. Mean inter-rater reliability between the reference examiner and other examiners was 0.898 (range from 0.857 to 1.000) for carious lesions, 0.949 (range from 0.872 to 1.000) for DDE, whereas mean intra-examiners reliability was 0.988 (range from 0.963 to 0.999) and 0.986 (range from 0.952 to 1.000) respectively.

The majority of the participants had municipal drinking water with a low level of fluoride (< 0.5 mg/ml) [7]. Furthermore, drinking water was sampled in all regions to assess fluoride content. An ion-selective electrode (Orion 9609) was used to determine water content of fluoride in the residential areas of participants by adding standard NaF solutions (fluoride standard 100 ppm F by Orion) twice. The detection rate of this method is 0.02mg/l. The relative standard deviation did not exceed 7%. The average reading was calculated.

The adolescents were asked for aesthetic and treatment problems caused by their enamel developmental defects.

Statistical analysis

Statistical analysis was performed using Statistica 12.0 (StatSoft) software ($p < 0.05$). T-test was used for comparing group means. The descriptive statistical analysis of the DDE and caries data included the determination of prevalence rates according to cut-off (DMFT/DMFS = 0; DDE = 0). Adolescents with at least one DDE were categorised to group DDE ≥ 1; otherwise, subjects with no DDE were scored as free of DDE. The components of the caries appearance and DDE were determined separately and mean values (standard deviation) were calculated. Relationships between pairs of selected variables were evaluated using Spearman's rank correlation coefficient. A simple and multiple logistic regression were used to assess the relationship between DDE, which was treated as independent variables and dental caries which were treated as binomial dependent variable. The results were presented in the form of odds ratios (OR - odds ratio) for simple logistic regression, and adjusted odds ratios (AOR) for multiple logistic regression as well confidence intervals (CI at 95% confidence level) for OR. Socioeconomic factors were considered as confounders in AOR calculation. Significance level for all the analyses was set at 0.05.

Results

A total of 1,611 adolescents aged 18 years participated in the study, exceeding a minimum representative sample size. A total of 1,800 adolescents were invited to participate in the study; however, 2.9% (n = 53) were absent at the time of dental examination, 5.6% (n = 100) refused to be examined and 2.0% (n = 36) did not complete the questionnaire. The number of individuals in each province ranged from 99 to 110 in urban and rural areas. Socio-demographic data are presented in Table 1.

Table 1
Socio-demographic characteristics of participants

Parameters Socio-demographic factors		Total N / % (95% CI)	Patients with DDE and caries N / % (95% CI)	Patients with DDE and no caries N / % (95% CI)
Gender	female	847/52.6% (50.1–55.0)	96/11.3% (9.3–13.7)	6/0.7% (0.2–1.5)
	male	764/47.4% (45.0-49.9)	110/14.4% (12.0-17.1)	8/1.0% (0.5–2.1)
	p		0.130	0.511
Place of residence	urban area	797/49.5% (47.0-51.9)	110/13.8% (11.5–16.4)	9/1.1% (0.5–2.1)
	rural area	814/50.5% (48.1–53.0)	96/11.8% (9.7–14.2)	5/0.6% (0.2–1.4)
	p		0.230	0.274
Mother's education	basic/primary	47/3.3%	8/17.0% (7.7–30.8)	1/2.1% (0.0-11.3)
	vocational	418/29.0%	54/12.9% (9.9–16.5)	2/0.5% (0.0-1.7)
	secondary/ postsecondary	532/36.9%	76/14.3% (11.4–17.6)	4/0.8% (0.2–1.9)
	higher	446/30.9%	49/11.0% (8.2–14.3)	5/1.1% (0.3–2.6)
	p		0.383	0.555
Socioeconomic status	low	47/2.9%	4/8.5% (2.4–20.4)	0/0% (0.0-2.2)

*chi-square test; p < 0.05

Parameters Socio-demographic factors		Total N / % (95% CI)	Patients with DDE and caries N / % (95% CI)	Patients with DDE and no caries N / % (95% CI)
	average	896/55.4%	111/12.4% (10.3–14.7)	5/0.6% (0.2–1.3)
	high	382/23.7%	48/12.6% (9.4–16.3)	3/0.8% (0.2–2.3)
	p		0.720	0.769
*chi-square test; $p < 0.05$				

The prevalence of DDE was 13.7% (220 patients), with an average of 0.85±3.34 teeth affected (Table 2). The most common type of DDE was demarcated (155/1611; 9.65%) and diffuse opacities (65/1611; 4.0%), followed by enamel hypoplasia (25/1611; 1.5%). MIH was diagnosed in 0.6% patients (10/1611).

Table 2
The prevalence of DDE in 18-year-old adolescents

Parameters	Participants n/N (%) (95% CI)	Teeth mean ± SD
m-DDE Index > 0	220/1611 (13.7%) (12.0-15.4)	0.85 ± 3.34
• demarcated opacity	155/220 (70.4%) (64.0-76.4)	0.50 ± 2.32
• diffuse opacity	65/220 (29.5%) (23.6–36.1)	0.32 ± 2.31
• enamel hypoplasia	25/220 (11.4%) (7.5–16.3)	0.03 ± 0.26
MIH	10/220 (4.5%) (2.2–8.2)	0.02 ± 0.32
n- patients with DDE; N-total study group; CI- confidence interval		

Prevalence of different types of DDE were significantly different ($p<0.05$). However mean number of teeth affected by these types of DDE was very low and the differences between various types of DDE were not significant ($p>0.05$). Table 3 shows comparisons of patients with DDE and caries vs. DDE only.

Table 3
Comparisons of patients with DDE and caries vs. DDE only

Parameters	Patients with DDE and caries n/N (%) (95% CI)	Patients with DDE only n/N (%) (95% CI)	p (for comparisons patients with DDE and caries vs. DDE only
m-DDE Index > 0	206/1611 (12.8%) (11.2–14.5)	14/1611 (0.9%) (0.5–1.5)	< 0.001*
demarcated opacity	144/1611 (8.9%) (7.6–10.4)	11/1611 (0.7%) (0.3–1.2)	< 0.001*
diffuse opacity	57/1611 (3.5%) (2.7–4.6)	3/1611 (0.2%) (0.0-0.5)	< 0.001*
enamel hypoplasia	23/1611 (1.4%) (0.9–2.1)	1/1611 (0.1%) (0.0-0.4)	< 0.001*
MIH	1/1611 (0.1%) (0.0-0.4)	0/1611 (0.0%) (0.0-0.2)	0.317
* chi-square test; p < 0.05			

The studied sample of adolescents was not exposed to confounding factors such as fluorine in the public water. The fluoride level in the public water was below 0.5 ppm at the time when the participants of the present study were born and living before 6-year-old**6**. Fluorine levels in drinking water sampled in the residential areas of participants measured by an ion-selective electrode (Orion 9609) were average 0.24 ± 0.22 ppm (mgF).

The prevalence of caries was 93.2% (1,501 patients), with 6.50 ± 4.22 teeth affected (i.e. 23.4% of teeth present in the oral cavity). The components of the DMF index as related to DDE are shown in the Table 4.

Table 4
Mean values of DMFT/DMFS scores and their components as related to DDE

Parameters	Developmental enamel defects		
	Present	Absent	p based on Mann–Whitney *U* test
	mean ± SD		
DMFT	7.66 ± 4.68	6.35 ± 4.12	< 0.001*
DT	2.58 ± 2.88	1.99 ± 2.78	< 0.001*
MT	0.13 ± 0.46	0.14 ± 0.52	0.943
FT	4.95 ± 3.79	4.22 ± 3.44	0.011*
DMFS	11.59 ± 9.15	9.51 ± 8.07	0.001*
DS	3.47 ± 4.43	2.98 ± 4.96	0.001*
MS	0.64 ± 2.30	0.69 ± 2.57	0.945
FS	7.48 ± 6.53	5.84 ± 5.38	0.001*

*Mann–Whitney *U* test; $p < 0.05$

DMFT = decayed, missing, filled primary teeth; DMFS = decayed, missing, filled surfaces; DT(S) = decayed tooth (surface), MT(S) missing tooth (surface), FT(S) filled tooth (surface); SD = standard deviation

As many as 56.9% of respondents were dissatisfied with their teeth, and every tenth respondent admitted that he avoided a smile due to his teeth. Dental diseases in 7.0% of participants were the cause of problems with eating hard food, and in 3.2% with chewing.

Logistic regression analysis did not confirm the association DDE with caries prevalence (Table 5). When DMFT > 0, there was no significant association with DDE. However, significant differences were found between the means of DMFT / DMFS (between DDE vs. without DDE; with demarcated vs. control; diffuse opacities vs. control).

Table 5
A relationship between DDE and caries

	DMFT > 0	DMFT	DMFS
	N (%)	**Mean ± SD**	
Total study group	1501/1611 (93.2%)	6.50 ± 4.22	9.74 ± 8.23
Patients with DDE	206/220 (93.6%)	7.66 ± 4.68	11.59 ± 9.15
Patients without DDE (control)	1255/1348 (93.1%)	6.35 ± 4.12	9.51 ± 8.07
p (vs control)		< 0.001*	0.001*
OR (95% CI)	1.09 (0.61–1.95), p = 0.770		
AOR (95% CI)	1.14 (0.61–2.14), p = 0.672		
• **demarcated opacities**	155/168 (92.3%)	7.52 ± 4.77	11.31 ± 8.94
p (vs control)		0.001*	0.010*
OR (95% CI)	0.88 (0.48–1.62), p = 0.688		
AOR	1.04 (0.53–2.06), p = 0.903		
• **diffuse opacities**	62/65 (95.4%)	7.85 ± 4.74	11.88 ± 9.83
p (vs control)		0.005*	0.026*
OR (95% CI)	1.53 (0.47–4.97), p = 0.478		
AOR (95% CI)	1.20 (0.37–3.95), p = 0.759		
• **enamel hypoplasia**	24/25 (96.0%)	7.56 ± 4.57	10.96 ± 8.77
p (vs control)		0.155	0.384
OR (95% CI)	1.77 (0.24–13.29), p = 0.575		
AOR (95% CI)	1.49 (0.20-11.27), p = 0.698		
• **patients with MIH**	10/10 (100.0%)	7.10 ± 2.18	11.30 ± 5.14
p (vs control)		0.565	0.484
OR (95% CI)	1.56 (0.09–26.90) p = 0.758		
AOR (95% CI)	1.52 (0.07–22.31) p = 0.874		

p- values for the differences and OR and AOR vs control (individuals without DDE); OR – odds ratio based on simple logistic regression; AOR – adjusted odds ratio based on multiple logistic regression

*statistical significance p < 0.05

Odds ratio of the chance of occurrence of dental caries in all types of DDE was not significantly different in comparison to control (i.e. individuals without DDE). However, the differences were significant between means of DMFT for patients with DDE vs. control; patients with demarcated opacities vs. control and for patients with diffuse opacities vs. control. The same differences were significant for DMFS. Spearman’s correlation did not confirm a relationship between socio-economic status, parents’ education and DDE. Table 6 shows the Spearman correlation coefficients between DMFT and DDE (a division into various defects, their number or the presence or absence(0/1)).

Table 6
Spearman's correlation between DMFT and DDE (a division into various defects, their number or the presence or absence(0/1))

	D (cavitated)	M (missing due to caries)	F (filled)	DMFT
diffuse opacity (number of teeth)	0.051*	-0.011	0.042	0.058*
demarcated opacity (number of teeth)	0.082*	0.012	0.045	0.077*
hypoplasia (number of teeth)	0.029	-0.040	0.015	0.028
MIH (number of teeth)	0.028	-0.001	-0.005	0.022
diffuse opacity (0/1)	0.052*	-0.011	0.043	0.059*
demarcated opacity (0/1)	0.080*	0.012	0.044	0.076*
hypoplasia (0/1)	0.029	-0.040	0.015	0.028
MIH (0/1)	0.028	-0.001	-0.005	0.022
*statistical significance $p < 0.05$				

Due to the large sample size, although the correlations were weak, they turned out to be statistically significant. The number of teeth with DDE did not increase the correlation with DMFT as it was similarly strong.

Discussion

This is, to our knowledge, the first study investigating the prevalence of DDE since 1990 in a nationally representative sample of 18-year-olds living in all sixteen provinces of Poland. The study was conducted as part of the Ministry of Health national programme– the only nationwide programme assessing oral health. Even though the programme was launched in 1999, it was in 2017 that it was extended with an additional examination of DDE. Reliable data or studies on larger populations are not available.

Retrospective determination of the aetiology of enamel defects is difficult. The presence of demarcated opacity and hypoplasia in the form of isolated, sporadically located lesions indicates local causes.

Diffuse opacities are usually found on the teeth with simultaneous enamel secretion and maturation, pointing to environmental aetiology and are related to systemic causes. Traumatic damage or periapical inflammatory lesions in the primary tooth or its early extraction may disrupt normal matrix deposition or enamel mineralisation and, consequently, lead to enamel defect (a demarcated spot and/or hypoplasia) in the permanent tooth.. The distribution of defects among Polish adolescents aged 18 years old was purpose of our previous paper [4]. The data from previous monitoring studies allow to conclude on the consequences of dental caries as a possible causal relationship with DDE. The 18-year-old adolescents in the study were aged 3 years in 2002, when the prevalence of caries among Polish children at that age was 56.2%, with a mean of 2.9 teeth affected [31]. In the same period (2002), caries was found in 86.9% of 6-year-olds, with 5.9 primary teeth involved [32].

A number of studies have shown a positive relationship between DDE and the severity of caries [9–20, 33]. The results of our study are consistent with previous studies that suggested that DDE increased the risk of dental caries, since the influence of enamel defects in the development of caries was observed [14, 33–35]. Fotedar et al.[14] also demonstrated a significant association between caries and enamel opacity among 12- and 15-year-olds from India. The relationship between DDE and the severity of dental caries was also confirmed in our study, which demonstrated significantly increased caries severity (expressed as DMFT and DMFS) in patients with DDE (7.66 ± 4.68 vs 6.35 ± 4.12 and 11.59 ± 9.15 vs 9.51 ± 8.07, $p < 0.001$). However, the severity of caries was increased in individuals presenting with qualitative enamel defects (demarcated and diffuse opacity) rather than those with quantitative defects (hypoplasia). Nevertheless, no significantly increased risk of caries among Polish adolescents in the presence of developmental enamel defects was shown, which may be due to the high prevalence of caries and a several-year residence time of teeth in the oral cavity. Some of these subjects will have their original DDE obliterated by caries, restoration and extraction.

In relation to enamel defects, all the types of defects can be associated with dental caries. Enamel hypoplasia is more susceptible to dental caries [15]. In opposite, individuals with diffuse opacities decurrently of greater fluoride exposure are less prone to exhibit dental caries [31]. The term ‘diffuse opacities’ is used interchangeably with dental fluorosis when is caused by an excessive intake of fluoride. The amount of fluoride contained in drinking water should be considered, especially if 0.70 mg/l or more of fluoride is present in the water [16, 23]. The studied sample of adolescents was not exposed to fluorine in the public water. The fluoride level in the public water was below 0.5 ppm at the time when the participants of the present study were born. The majority of the participants had municipal drinking water with a low level of fluoride (< 0.5 mg/ml). The low prevalence of diffuse opacities among participants in this study might be due to consumption of low level of fluoride in the public water. Another explanation might be the effect of remineralization. Compared with demarcated opacity, it is easier for diffuse opacities to be remineralized, similarly to assumption of Wong et al. [36] study.

The results of our study, however, demonstrated that the type of DDE could not explain the heterogeneity, probably because the aetiology of these defects share some similar influencing factors. It is possible that the longitudinal observation of DDE differ among populations with different prevalence of DDE. When analysis was performed for the different types of DDE, it was found higher prevalence of DEO compared to DIO. Reduction of frequency of diffuse opacities has been showed, similarly to Wong et al. [36] study. Demarcated opacities have distinct boundaries separating them from normal enamel which are thus more unlikely to disappear from mechanical and chemical reasons.

A question should be asked whether these are demarcated defects that promote the development of caries or whether dental caries develops due to the presence of causative factors of this disease in individuals with dental caries in primary dentition. A positive association between enamel defects and dental caries was identified in meta-analysis of Vargas-Ferreira et al. [9]. Individuals with DDE had higher pooled odds of having dental caries experience [OR 2.21 (95%CI 1.3; 3.54)]. A higher chance of dental caries should be expected among individuals with enamel defects.

It is beyond doubt that the risk of differential misdiagnosis due to the lack of precise data on different fluorine sources in childhood, is an important limitation of epidemiological studies assessing the incidence of enamel opacities classified as dental fluorosis.

It's important to emphasize that DDE and carious lesions were distinguished, diagnosed and recorded based on locations and surface features, followed diagnostic criteria and recommendations [25–30]. Training and calibration of all examiners in the present study resulted a good intra- and inter-examiner reliability. Dini et al. [18] demonstrated a two times lower risk of dental caries in children with diffuse enamel opacities compared to children with no or demarcated opacities.

The half of adolescents worry about their teeth. These findings are not in accordance with Sujak et al.[21] who suggested that very few subjects were concerned about the appearance of their teeth, or were not aware of their teeth being different. In the Vargas-Ferreira et al. [9] study children with DDE did not indicate any decrease in self-perception. However, this condition was associated with an impact on the functional limitation domain.

The strength of this study was large number of 18- year-old adolescents. A population-based sample was used, contrary to a clinical convenience sample, in some studies. The presence of controlling for confounding such as socioeconomic factors to find its influence on findings was taken into account and analysed, in contrast to some other studies. When considering the methodology of the present study, it should be mentioned that the recording of carious lesions and DDE followed the most recently published recommendations [26, 28–30]. The trained group of dental examiners showed good intra- and inter-examiner reliability values and good capacity to identify DDE and to discern the different types of DDE. Furthermore, restorations subsequent to carious lesions were delineated from DDE-related restorations, i.e. atypical restorations due to DDE were not scored as caries-associated restorations and, hence, were not part of the F-component and the DMF index. Therefore it was possible to distinguish between DMF and DDE-related lesions correctly. Restorations subsequent to carious lesions were delineated from DDE-related restorations, i.e. atypical restorations due to DDE were not scored as caries-associated restorations and, hence, were not part of the F-component and the DMF index. Therefore it was possible to distinguish between DMF and DDE-related lesions correctly.

It is important to mention that the cross-sectional observational epidemiological design of the study does not allow to establish the temporal causal relationship between DDE and caries. Further investigations using longitudinal design are needed to confirm these findings.

This study has several limitations that need to be taken into account for an adequate interpretation of the results, acknowledging the age of the subjects. Adolescents were recruited from the public high schools only. Only those who signed consent for participation were included to the study, what may cause selection bias. The examinations regarding dental caries should be separated from DDE to avoid observational bias, however, this was not possible in the present study. Some of the subjects will have their original DDE obliterated by caries, restoration and extraction. Due to the difficulty to differentiate between molars and incisors hypomineralisation and caries, misclassification bias could be taken into account. On the other hand, the tooth- and surface-related recording of MIH-related defects and restorations—which has been used in caries epidemiological trials for decades—is another step forward helping to determine the extent and severity of MIH precisely [19]. In addition, another limitation of the study refers to the lack of analysis according access to oral health services, dietary and oral hygiene habits aspects, data on sources of fluoride exposure other than water fluoridation. These factors may be considered as potential effect modifiers that may lead to a weak association between dental caries and enamel defects. Also, the reasons for missing teeth were not recorded; hence, some teeth that were missing due to caries were designated as non-carious. Diagnosis was based on visual and tactile examinations under artificial lighting conditions only. Radiographs were not taken, thus small caries lesions might have been underestimated nor recorded, what is a common problem in cohort studies. Some studies have investigated teeth with natural lighting, others with a flashlight for illumination and have used sterile gauze to remove debris. It is important to mention that in the present study all permanent teeth have been evaluated, while most studies assessed only index teeth. In addition - DMFT was used instead of more detailed index like ICDAS II. Different indices and criteria, examination variability, methods of recording, and varying age groups in various DDE studies, may limited the comparisons of the results. Thus, a standardised index should be used in future studies. Finally, because dental caries and DDE may share key risk factors, such as a disadvantaged background, a common risk approach should be more rational [37].

Conclusions

The results of this study indicated association between enamel defects and severity of caries. The presence of qualitative (opacity) DDE has an impact on the severity of caries in 18- year-old adolescents. Finally, it can be concluded that a significant relationship existed between DDE and dental caries in 18-year-old adolescents, with presence of DDE associated with increased caries experience. Further studies are however required to explore the relationship in this study population.

Declarations

Authors' contributions: DOK: had primary responsibility for protocol development, patient screening, enrolment, outcome assessment, supervision of the design and execution of the study, performing of the final data analyses, writing the manuscript and making the critical revisions; ATS: had primary responsibility for protocol development, patient screening, enrolment, outcome assessment, preliminary data analysis and writing the manuscript; NK: participated in the development of the analytical framework of the study, performed the final data analyses, contributed to the writing of the manuscript and making the critical revisions; DG: participated in the development of the protocol and analytical framework of the study, performed the final data analyses and contributed to the writing of the manuscript; UK: participated in the development of the protocol and analytical framework of the study, contributed to the writing of the manuscript and making the critical revisions. All the authors approved the final version of the manuscript

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Conflict of interest

No conflict of interest

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Ethical approval The study was approved by the Bioethics Committee of the Medical University of Warsaw (No KB/134/2017). The study has been conducted in accordance with the World Medical Association Declaration of Helsinki (version, 2008).

Informed consent Informed written consent was obtained from all individuals participating in the study.

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