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Abstract

Background: Developmental hypomineralised lesions of enamel (DHL) may represent a significant caries-risk factor. The present study´s aim is to determine the association between carious lesion severity and DHL in 6 to 12-year-old schoolchildren from Melbourne, Australia.

Methods: The sample was derived from randomly selected schools in inner Melbourne. A full dental examination was performed at the school. Socio-demographic data, caries experience (DMFT/dmft/ICDAS II) and the consequences of untreated carious lesions (PUFA/pufa) were measured. DHL, molar incisor hypomineralisation (MIH) and hypomineralised second primary molar (HSPM) presence was assessed using the European Academy of Paediatric Dentistry (EAPD) criteria.

Results: Of the children examined (n=327), 26.9% had DHL. The prevalence of MIH and HSPM was 14.7% and 8%, respectively. Almost 20% of children had severe carious lesions (ICDAS 5 & 6) in at least one permanent or primary tooth. Ordinal regression analyses indicated that DHL (OR=2.17; 95%CI: 1.35-3.49) and being born overseas (OR=2.59, 95%CI: 1.66-4.06) increased the likelihood of severe carious lesions.

Conclusions: One of four children had DHL. DHL-affected children had an increased likelihood of presenting untreated severe carious lesions compared to DHL-free children.

Abbreviations and acronyms:

DHL – demarcated hypomineralised lesions of enamel
MIH – molar incisor hypomineralisation
HSPM – hypomineralised second primary molars
OR – odds ratio
DMFT/dmft – decayed missing filled teeth
ICDAS – international caries detection and assessment system.
PUFA/pufa – pulp, ulceration, fistula, abscess index.
EAPD – European Academy of Paediatric Dentistry

Introduction

Dental caries is the most frequently diagnosed oral health condition amongst children around the world.[1] Despite the preventable nature of dental caries, untreated carious lesions remain the most prevalent chronic disease, affecting millions of children worldwide.[1] Dental caries affects children's general development, well-being and quality of life.[2]

The aetiology of dental caries is multi-factorial, involving complex interactions between the commensal oral microbiome and associated risk or protective factors, such as diet, behaviour, oral hygiene, genetics, host-susceptibility characteristics and socio-demographic factors.[3,4] Demarcated hypomineralised lesions of enamel (DHL) are developmental qualitative defects of tooth enamel, clinically characterised by well-defined areas of hypomineralisation. These defects are a relevant, but under-recognised risk factor for carious lesion development, and they may have an impact on caries experience, carious lesion severity and treatment burden, in both primary and permanent dentitions.[5,6]

Two distinct prevalent presentations of DHL have been recognised: a) molar incisor hypomineralisation (MIH) defined as "hypomineralisation of systemic origin affecting one to four first permanent molars, frequently associated with affected incisors", and b) hypomineralised second primary molars (HSPM) when MIH-like defects are present in second primary molars.[7,8] However, any tooth may be affected by DHL.[9] The proportion of caries experience that may be associated with DHL has been largely underestimated in the literature and a limited number of reports have highlighted this association by controlling for other confounding factors.[10] Recording the severity of carious lesions related to DHL would identify the treatment needs of those children who present atypical carious lesions, whose preventive and clinical management include more expensive and complex measures compared to children having 'classical' carious lesions.

The present cross-sectional study was conducted in Melbourne, Australia, where the DMFT/dmft has been historically rated as low. The latest Australian National Oral Health Survey indicates a dmft of 2.58 amongst 6 year-old children and a DMFT of 1.34 amongst 12 year-old Australian children.[11] Developmental DHL may act as a significant risk factor in low to medium caries-risk communities,[10] thus the aim of the present study is to determine the association between carious lesion severity and DHL in 6 to 12 year-old schoolchildren from Melbourne, Australia.

Materials and methods

Ethical considerations and sample

Ethical approval was obtained from the HREC of The University of Melbourne (HREC No 1340244). Permission from The Department of Education and Training (DET), State of Victoria, Evaluation and Analytics Branch and from The Catholic Education Office Melbourne was granted. The study was conducted in inner metropolitan Melbourne, the capital city of the State of Victoria, and 2nd largest city in Australia. At June 2015, there were an estimated 4.53 million people residing in Melbourne.[12] The randomized cluster sample was derived from catholic and state primary schools located in the eight most central Local Government Areas (LGAs). The concentration of fluoride in the reticulated water supply in Melbourne ranges from 0.8 to 0.9 ppm.[13]

Proportional and comparative sample size calculations were performed using G*Power Version 3.1.9.2.[14] The estimated prevalence of the main response variable (i.e. DHL prevalence) is based on the available data reported in the literature. The approximate percentage of children with MIH in Australia is based on 22%.[15] The estimated proportion of children with HSPM is 14%.[16] The anticipated level of confidence was set at the 95%. Therefore, the minimum participant requirements for conducting a cross-sectional prevalence study on MIH (22%) and HSPM (14%) with a proportion error estimation of 5% of the real value at 95% level of confidence are N = 264 and 186, respectively. A design effect (deff) of 1.2 was estimated for this sample giving a sample size of N = 317 children for MIH and N = 223 children for HSPM. The deff was estimated as the clusters (schools) selected in Melbourne were likely to be similar. The highest number was selected as the required sample size to determine the prevalence of children with DHL in general (N = 317). To allow the calculated final sample size of 317 to be obtained, all students from grade 1 to 6 in each selected school were invited to

participate (N = 1585). The sample size determined for the prevalence of children with DHL was also adequate for the comparative and predictive analyses of the caries experience between the children affected and non-affected by hypomineralisation.

Sampling procedures

To achieve the aims of the present study, taking into consideration funding and time availability, participating primary schools included schools with fewer than 300 students enrolled. Students from Grades 1 to 6 were invited to participate, which matched the age range selected for the present study (6 to 12 years-old). A total of 41 state primary schools were identified from the list available from DET in the eight LGAs. Thirty-five schools were selected randomly using Microsoft® Excel® (version 14.0, Microsoft Corporation, WA, USA) and invited to participate; two catholic schools and nine public schools accepted the invitation. Once permission had been obtained from school principals, parents were invited to return a signed consent and complete a brief socio-demographic questionnaire.

Calibration

Before initiation of the study, a calibration exercise in diagnosing developmental defects of enamel (DDE) and carious lesions was carried out for researcher (KG) with the aid of experienced trained examiners at the Melbourne Dental School. A total of 20 images were analysed in two attempts. Kappa values ranged from 72.2% to 94.4% representing "substantial" and "almost perfect" agreement for carious lesions and DHL scores.[17] Immediately after the completion of the study, reproducibility of the scores for DDE and carious lesions was tested using forty training exercises derived from images recorded in the present study, with kappa values ranging from 90 to 94%. After four weeks of collecting data, the intra-examiner coefficient values indicated "almost perfect" agreement (87%).[17]

Dental examination

Data collection was conducted between 2014 and 2015. The clinical dental examination lasted approximately 10 minutes. This examination was performed in an available room at the school and children were seated on a school chair after brushing their teeth before the examination. The teeth were dried using sterile cotton rolls and examined with the

aid of an external light source (Light-Tech®, Light-Tach Inc., FL, USA) and disposable examination kits. The buccal, occlusal and lingual / palatal surfaces of all the teeth were evaluated by one examiner (KG). Data for the permanent and primary dentition, if present, were recorded on audio and subsequently transferred to the field record sheet on the same day of examination.

Study variables

Socio-demographic factors included: sex, age groups (6-7, 8-9 and 10-12 years old) and type of school (proxy measure to socio-economics). The latter was classified according to the Index of Community Socio-Educational Advantage (ICSEA).[18] Three groups with a similar number of students were created, a) ICSEA index values lower than 1025, b) ICSEA index value from 1025 to 1055, and c) ICSEA index values higher than 1055. Place of birth was a proxy measure to identify the participants' migration status, defined as a) Australia, and b) overseas. A summary of previous dental visits were classified as a) routine check-ups and b) emergency treatment.

Teeth were assessed using the ICDAS II scoring criteria. Later, the codes were merged to facilitate analysis. These included Code 0 (sound surface), Code A, Code B and Code C representing initial carious lesions (ICDAS Code 2), established carious lesions (ICDAS Code 3 and Code 4) and severe carious lesions (ICDAS Code 5 and Code 6), respectively. ICDAS Code 1 was excluded from the present study analysis due to difficulty of recording this code accurately without access to compressed air.[19] The PUFA/pufa index was included to classify the clinical impact of untreated carious lesions.[20] Developmental enamel defect diagnosis was based on an existing index.[21] A two-digit code was recorded; the first digit indicated the type and severity of DHL ranging from 'sound = 0' to 'missing due to DHL = 9. The second digit was related to the extent of the lesion, 1= 'less than 1/3 of the surface', 2= 'between 1/3 and 2/3' and 3= 'greater than 2/3 of the tooth surface'. The severity of DHL was categorised as "mild" when only the presence of intact enamel opacity was detected, and "moderate/severe" when enamel breakdown was present, whether involving enamel only or enamel and dentine. Additionally, teeth with atypical restorations (i.e. the size and shape of restorations do not conform to the usual pattern of classical carious lesion), atypical carious lesions (i.e. the profile the carious lesions do not mirror the caries distribution in the child's mouth) and missing due to DHL were diagnosed as

moderately/severely affected. Enamel defects less than two millimetres in diameter were not recorded. At the child level, the most severe hypomineralised lesion and carious lesion encountered determined his/her classification according to dental caries severity and hypomineralisation severity.

Statistical analysis

Data analyses were computed using SPSS version 22 (IBM, NY, USA). Descriptive statistics of the prevalence and distribution of the data were determined. Chi-square (χ^2) statistics were used for testing differences between categorical groups in relation to the categorical independent variables. Kruskal-Wallis tests (*H*) were used to determine statistically significant differences between the number of the caries-affected permanent and primary teeth (i.e. DMFT, dmft, PUFA, pufa) in relation to the independent variables. Binary logistic regression models were used to determine the relative importance of the independent variables on DHL and dental caries occurrence. Adjusted Odds Ratios (OR) with 95% test-based confidence intervals (CI) were described. Finally, an ordinal logistic regression was also conducted to test the association between the independent variables and the ordinal dependent variable (merged ICDAS II: Codes 0, A, B and C). The assumptions for binary and ordinal logistic regression were tested and met. For example, the variance inflation factors (VIF) were examined to assess multicollinearity between independent variables, values ranged from 1 to 1.5 indicating low multicollinearity. The test for parallel lines was conducted to evaluate the proportional odds assumption; results demonstrated that this test was not significant ($p > 0.05$), concluding that the assumption holds. The results were considered significant at an alpha level < 0.05.

Results

A total of 371 from 1585 six to twelve-year-old children had their parents/guardian consent to participate in this study, and 327 children were examined, the remaining children (N = 44) were not present at the school on the day of examination or did not want to participate in the study. More than half of the children examined were male (52.9%, N = 173). Approximately one third of the study sample was distributed in each school type and by age groups. The majority of children were born in Australia (59.5%,

N = 194). The great majority of participants had visited a dental practitioner previously for routine check-ups (86.1%, N = 241)

The DMFT and dmft of the total sample was 0.68 and 1.66, respectively. Participants born overseas had significantly higher likelihood of DMFT higher than zero (OR 2.58, 95% CI 1.42 - 4.67, p = 0.002) and dmft higher than zero (OR = 1.93, 95% CI 1.14 – 3.27, p = 0.01) when compared with children born in Australia (Table 1). The PUFA + pufa prevalence was 10.7% (N = 35). Children born overseas had significantly higher odds for a PUFA + pufa prevalence higher than zero (OR = 4.95, 95% CI 1.94 - 12.65, p = 0.001) than Australia-born children (Table 1).

The prevalence of DHL was 26.9% (N = 88). Seventy (21.4%) children had DHL in permanent teeth and 28 (8.6%) children had DHL in primary teeth. Of the total, 48 children (14.7%) had MIH and 26 (8%) had HSPM. Children with HSPM demonstrated an increased risk for MIH (OR = 2.90, 95% CI = 1.18 - 7.11, p = 0.02).

Demarcated hypomineralised lesions of enamel were detected in all permanent and primary tooth groups. A total of 137 (3.4% tooth prevalence) permanent teeth were diagnosed with DHL. The majority of hypomineralised permanent teeth exhibited creamy/white demarcated lesions without post-eruptive breakdown (PEB) (51.1%, N = 70), followed by atypical restorations (20.4%, N = 28) and yellow/brown lesions with no PEB (15.3%, N = 21). Atypical carious lesions affected 16.1% of hypomineralised permanent teeth. On the other hand, 65 primary teeth (2.0% tooth prevalence) had clinical evidence of DHL. Of those, the highest proportion showed atypical carious lesions (24.6%, N = 16), followed by creamy/white lesions without PEB (21.5%, N = 14) and atypical restorations (20%, N = 13).

A higher DMFT was expressed in DHL-affected children compared to those who were not affected (1.08 vs. 0.53 teeth, $H(1) = 16.32$, $p < 0.001$), particularly in the DT component (0.66 vs. 0.43 teeth, $H(1) = 9.19$, $p = 0.002$) and FT component (0.39 vs. 0.09 teeth, $H(1) = 17.15$, $p < 0.001$). The dmft did not differ significantly between groups. A higher PUFA experience was present in the DHL-affected group compared to non-affected children (0.02 vs. 0 teeth, $H(1) = 5.45$, $p = 0.02$) (Table 2).

Teeth affected by DHL had a higher proportion of ICDAS II Codes B and C compared to unaffected teeth ($\chi^2(3) = 145.95$, $p < 0.001$) (Fig. 1). Carious lesions coded as B

affected 17.6% (N =36) of hypomineralised teeth. The majority of atypical carious lesions were coded as B (Fig. 2).

The ordinal logistic regression model was significant for carious lesion severity (Table 3) and the proportion of the variance explained by the independent variables was 12% ($\chi^2(7) = 38.34$, $p < 0.001$, Nagelkerke's pseudo R^2 12%). Almost 50% (N = 158) of children had carious lesions and 20% (N = 64) of the total children were classified as Code C. In the model, thresholds represent the response variable (ICDAS II merged codes) in the ordered logistic regression; participants that had a value of 2.45 or greater on the dependent variable were classified as Code C, giving that all the independent variables had the reference value. The ordered value for DHL-affected children for being classified into an increased ICDAS severity was 0.77 higher than non-affected children when the other variables in the model were held constant, with an OR of 2.17 (95% CI 1.35-3.49, p = 0.001). For place of birth, the ordered value for children born overseas being categorised by a high ICDAS score was 0.95 greater than children born in Australia with an OR of 2.59 (28.8% vs. 13.4%, 95% CI 1.66-4.06, $p < 0.001$).

Discussion

In the present study, almost 50% of children had at least one carious lesion (ICDAS > 0) and nearly half were coded as severe. Unfortunately, current preventive and oral health promotion strategies have been unsuccessful. Improving oral health literacy will increase awareness regarding the importance of early diagnosis and management.[22] Informing the community regarding the oral health benefits available for Victorian children (e.g. Child Dental Benefits Schedule. CDBS) has the potential to improve access to dental services in infancy.[23] These measures should help to reduce the disease prevalence in schoolchildren, as initial carious lesions are reversible if risk factors decrease. A few international studies have reported dental caries prevalence categorised by ICDAS codes, but these studies did not classify the participants according to the highest code encountered.[24,25] Therefore, comparisons with the present study´s findings are implausible. The importance of the present information is that amongst this group of children (20%), the carious lesions have remained untreated to a stage that can negatively influence their general health and perceived quality of life.[26] In particular, the caries experience and severity of children who migrated to Australia is high and this was corroborated by the present findings.[27] In Melbourne, a recent cross-sectional study demonstrated that 15% of 1 to 4 year-old children from Iraqi, Pakistani and Lebanese

backgrounds had cavitated carious lesions; related to parental education, length of residence in Australia and sugary drinks consumption; however, the study did not compare the oral health status of participants with locally born children.[28] Inequalities were found in the present study, therefore, immigrant families may be less aware of how to access available dental services, supported by a previous study showing that these families use public dental services at a lower rate.[29] Oral health systems may have failed to inform immigrant families about the dental services available and the importance of preventive and restorative treatment, especially in primary teeth. Thus, secondary prevention and restorative treatment needs remain unmet. Findings from the present study may be useful to guide health authorities to target resource distribution, to reduce disparity and improve the oral health literacy of their clients.

The overall prevalence of DHL in the present study was 27%. Of the participants, 21.4% and 8.6% had DHL in permanent and primary teeth, respectively. The prevalence was higher than most epidemiological surveys examining the mixed dentition, possibly due to differing diagnostic criteria.[30,31] Studies on DDE often use the mDDE index, which does not include PEB for demarcated lesions, possibly confounded by hypoplasia. This data supports the recommendation that all teeth with DHL should be recorded in future epidemiological studies using appropriate diagnostic criteria. The majority of DHL were mild lesions (i.e. intact CW or YB lesions) that occupied less than one-third of the tooth surface, which may not require any further treatment.[32] However, approximately one quarter of hypomineralised permanent and primary teeth had YB lesions. A strong association between dark brown enamel opacities and PEB has been postulated from laboratory study results,[33] as dark opacities have poorer mechanical properties caused by deficient mineral density, increased porosity and presence of organic content when compared to normal enamel or white DHL.[34] Atypical restorations were the next most prevalent type of defect for both dentitions. Australian oral health system has greater resources (including clinical guidelines) which make hypomineralised teeth more likely to be restored compared with other countries.[35]

The prevalence of MIH was 14.7%, similar to the estimated world average of 16%.[36] The prevalence of HSPM was 8.0%, also consistent with previously published epidemiological investigations in Germany (4.0%), Nigeria (4.6%), India (5.6%), Iraq (6.6%) and the Netherlands (4.9% and 9.0%).[9,37-40] Interestingly, a recent study from Melbourne has reported a higher prevalence rate of 14%.[16] This study examined

children younger than 5 years-of-age, unlike the present study, which sampled 6 to 12 year-old schoolchildren. Differences in samples may explain the variations in the reported prevalence since in the present study children with exfoliated SPMs were common. Nearly 30% of HSPM-affected children also had hypomineralised FPMs. Comparable co-morbidities have been published in Iraq, Nigeria, India and the Netherlands.[37-40] The concomitance of HSPM and MIH suggests common causative factors acting within a particular overlapping developmental period, affecting SPMs, FPMs and PIs simultaneously.[37] The clinical manifestations of HSPM appeared to be of greater severity compared to MIH characteristics; Mittal and collaborators (2015) published similar findings.[38] These differences are mainly based on the time period that these two index teeth (FPMs and SPMs) have been exposed to the oral environment.[41] Hypomineralised SPMs have received a greater exposure to a high caries risk environment (if present), masticatory and occlusal load, therefore, this may explain the higher frequency of PEB and atypical carious lesions.[38,40]

Thirteen per cent of hypomineralised teeth had atypical carious lesions which were coded as ICDAS B and C. The presence of DHL exacerbates caries risk, as teeth affected are more susceptible to develop carious lesions. Furthermore, hypomineralised enamel, once affected by caries, can lead to a rapid progression of the carious lesions due it's altered structure; demonstrated in the ordinal regression model controlling for other socio-demographic factors. Therefore, it is likely that these lesions progress more rapidly into severe carious lesions because of the presence of unfavourable oral health conditions and limited access to oral health care in those of low SES.

Conclusion:

Demarcated hypomineralised lesions of enamel affected approximately one in five children, and MIH (14%) and HSPM (8%) prevalence rates were consistent with many previously published studies. Demarcated hypomineralised lesions of enamel had a great impact on the caries severity, as DHL-affected children had an increased likelihood of presenting untreated severe atypical carious lesions. The general caries experience was low, but a high burden of disease was demonstrated amongst children with DHL.

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Table 1. Binary regression models for caries experience and PUFA+pufa prevalence by socio-demographics

Variables	DMFT = 0 N = 238 (72.8%)	DMFT > 0 N = 89 (27.2%)	AOR	95% CI	dmft = 0 N = 181 (55.4%)	dmft > 0 N = 146 (44.6%)	AOR	95% CI	PUFA + pufa = 0 N = 292 (89.3%)	PUFA + pufa > 0 N = 35 (10.7%)	AOR	95% CI
Sex												
Female	106 (68.8)	48 (31.2)	1		90 (58.4)	64 (41.6)	1		138 (89.6)	16 (10.4)	1	
Male	132 (76.3)	41 (23.7)	0.79	0.44-1.42	91 (52.6)	82 (47.4)	1.30	0.78-2.16	154 (89.0)	19 (11.0)	1.27	0.50-3.18
Age group												
6-7	92 (82.9)	19 (17.1)	1		68 (69.4)	30 (30.6)	1		94 (95.9)	4 (4.1)	1	
8-9	85 (72.0)	33 (28.0)	2.97*	1.33-6.62	55 (46.6)	63 (53.4)	2.11*	1.10-4.05	99 (83.9)	19 (16.1)	9.92*	1.15-85.48
10-12	61 (62.2)	37 (37.8)	4.08**	1.82-9.15	58 (52.3)	53 (47.7)	2.95**	1.57-5.55	99 (89.2)	12 (10.8)	21.30*	2.64-172.13
Type of school												
ICSEA > 1055	90 (81.8)	20 (18.2)	1		62 (56.4)	48 (43.6)	1		99 (90.0)	11 (10.0)	1	
ICSEA 1025-1055	84 (73.0)	31 (27.0)	1.90	0.88-4.09	72 (62.6)	43 (37.4)	1.55	0.80-2.98	106 (92.2)	9 (7.8)	1.71	0.54-5.40
ICSEA < 1025	64 (62.7)	38 (37.3)	1.71	0.84-3.47	47 (46.1)	55 (53.9)	0.86	0.47-1.54	87 (85.3)	15 (14.7)	1.10	0.36-3.28
Place of birth												
Australia	155 (79.9)	39 (20.1)	1		120 (61.9)	74 (38.1)	1		183 (94.3)	11 (5.7)	1	
Overseas	82 (62.1)	50 (37.9)	2.58*	1.42-4.67	60 (45.5)	72 (54.5)	1.93*	1.14-3.27	108 (81.8)	24 (18.2)	4.95**	1.94-12.65
Reason for dental visits												
Routine check-up	182 (95.5)	59 (24.5)	1		145 (60.2)	96 (39.8)	1		222 (92.1)	19 (7.9)	1	
Emergency	26 (66.7)	13 (33.3)	1.67	0.76-3.64	17 (43.6)	22 (56.4)	1.68	0.82-3.44	34 (87.2)	5 (12.8)	1.27	0.41-3.88
Model	Figures may not add due to missing values Model significant $\chi2(7) = 33.29$, $p<0.001$, Nagelkerke R^2:17%				Figures may not add due to missing values Model significant $\chi2(7) = 24.87$, $p = 0.001$, Nagelkerke R^2:11%				Figures may not add due to missing values Model significant $\chi^2(7) = 28.55$, $p<0.001$, Nagelkerke R^2: 22%			

AOR, Adjusted Odds Ratio. CI, Confidence Interval

*p < 0.05, ** p < 0.001

Table 2. Bivariate analyses for the caries experience between DHL affected and non-affected children

Caries experience	**DHL affected N = 88**	**DHL non-affected N = 239**	**All children N = 327**
DMFT mean (s.d.)**	1.08 (1.86)	0.53 (1.29)	0.68 (1.49)
DT mean (s.d.)*	0.66 (1.51)	0.43 (1.20)	0.49 (1.29)
MT mean (s.d.)	0.03 (0.24)	0.01 (0.09)	0.02 (0.15)
FT mean (s.d.)**	0.39 (0.98)	0.09 (0.48)	0.17 (0.66)
dmft mean (s.d.)	1.88 (2.73)	1.59 (2.53)	1.66 (2.58)
dt mean (s.d.)	1.32 (2.15)	1.05 (2.01)	1.12 (2.05)
mt mean (s.d.)	0.15 (0.65)	0.13 (0.73)	0.13 (0.71)
ft mean (s.d.)	0.41 (0.92)	0.40 (1.09)	0.41 (1.05)
Pulpal involvement			
PUFA mean (s.d.)*	0.02 (0.15)	0	0.01 (0.08)
pufa mean (s.d.)	0.26 (0.78)	0.18 (0.97)	0.20 (0.92)

** $p < 0.001$ and * $p < 0.05$ by Kruskal-Wallis analysis

Table 3. Ordinal regression model for ICDAS II merged codes, socio-demographics and DHL presence.

Variables	ICDAS merged codes N (%)				Parameter Estimates	AOR	95% CI
	Code 0 N(%) = 169 (51.7)	Code A N(%) = 10 (3.1)	Code B N(%) = 84 (25.7)	Code C N(%) = 64 (19.6)			
	Threshold 0.96		Threshold 1.09	Threshold 2.45			
Sex							
Male N(%)	91 (52.6)	5 (2.9)	35 (20.2)	42 (24.3)		1	
Female N(%)	78 (50.6)	5 (3.2)	49 (31.8)	22 (14.3)	-0.31	0.73	0.47-1.13
Age group							
10-12 N(%)	55 (56.1)	5 (5.1)	23 (23.5)	15 (15.3)		1	
8-9 N(%)	58 (49.2)	1 (0.8)	34 (28.8)	25 (21.2)	0.35	1.42	0.82-2.46
6-7 N(%)	56 (50.5)	4 (3.6)	27 (24.3)	24 (21.6)	0.43	1.53	0.90-2.59
SES							
High N(%)	60 (54.5)	6 (5.5)	22 (20.0)	22 (20.0)		1	
Middle N(%)	66 (57.4)	3 (2.6)	30 (26.1)	16 (13.9)	0.02	1.01	0.60-1.74
Low N(%)	43 (42.2)	1 (1.0)	32 (31.4)	26 (25.5)	0.52	1.69	0.98-2.91
Place of birth							
Australia N(%)	120 (61.5)	5 (2.6)	44 (22.6)	26 (13.3)		1	

Overseas N(%)	49 (37.4)	5 (3.8)	30 (30.5)	37 (28.2)	0.95	2.59	1.66-4.06**
DHL Presence							
No N(%)	135 (56.5)	7 (2.9)	58 (24.3)	39 (16.3)		1	
Yes N(%)	34 (38.6)	3 (3.4)	26 (29.5)	25 (28.4)	0.77	2.17	1.35-3.49**

Model significant $\chi^2(7) = 38.34$, $p < 0.001$, Nagelkerke R^2: 12%. Test of parallel lines $\chi^2(14) = 7{,}61$, $p = 0.91$

AOR, Adjusted Odds Ratio. CI, Confidence Interval

**$p < 0.001$

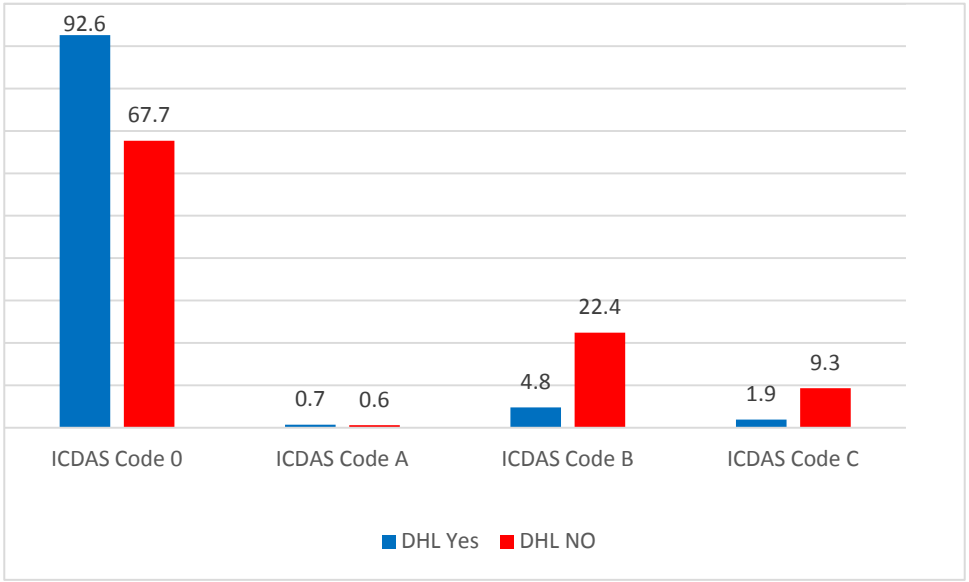

Figure 1. Distribution (%) of DHL-affected and unaffected teeth by ICDAS II scores.
DHL - affected (N = 161), Non - affected (N = 7138)
Statistical difference was found by ($\chi^2(3) = 145.95$, $p < 0.001$)

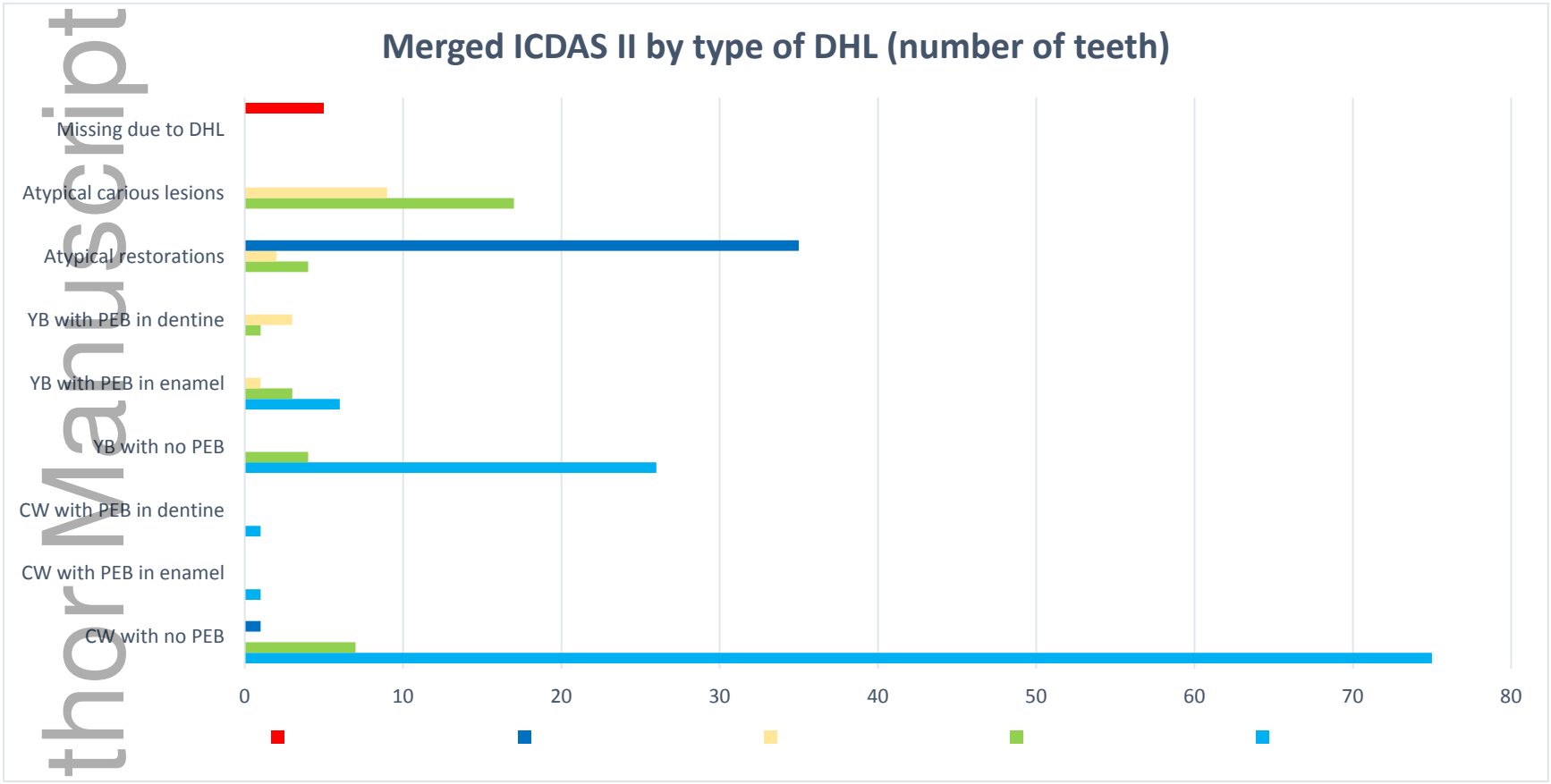

Figure 2. DHL demarcated hypomineralised lesion of enamel, CW creamy/white demarcated lesions, YB yellow/brown demarcated lesions, PEB post-eruptive enamel breakdown.