



Minerva Access is the Institutional Repository of The University of Melbourne

Author/s:

Owen, ML;Ghanim, A;Elsby, D;Manton, DJ

Title:

Hypomineralized second primary molars: prevalence, defect characteristics and relationship with dental caries in Melbourne preschool children

Date:

2018-03-01

Citation:

Owen, M. L., Ghanim, A., Elsby, D. & Manton, D. J. (2018). Hypomineralized second primary molars: prevalence, defect characteristics and relationship with dental caries in Melbourne preschool children. *Australian Dental Journal*, 63 (1), pp.72-80. <https://doi.org/10.1111/adj.12567>.

Persistent Link:

<https://hdl.handle.net/11343/293769>

Title Page:

Hypomineralised second primary molars: prevalence, defect characteristics and relationship with dental caries in Melbourne preschool children.

ML Owen, A Ghanim, D Elsby, DJ Manton

Melbourne Dental School, The University of Melbourne, Carlton, Vic., Australia

Contact: Professor David Manton, Melbourne Dental School, 720 Swanston St, Carlton, VIC 3053, 9341 1493

This is the author manuscript accepted for publication and has undergone full peer review but has not been through the copyediting, typesetting, pagination and proofreading process, which may lead to differences between this version and the [Version of Record](#). Please cite this article as [doi: 10.1111/adj.12567](https://doi.org/10.1111/adj.12567)

This article is protected by copyright. All rights reserved

Article type : Scientific Article

Corresponding Author mail id:- djmanton@unimelb.edu.au

Hypomineralised second primary molars: prevalence, defect characteristics and relationship with dental caries in Melbourne preschool children

Abstract

Background: Dental caries and enamel defects (DDE) are prevalent amongst children. The presence of DDEs, especially enamel hypomineralisation, may increase caries experience in those at increased caries-risk. The reported prevalence of hypomineralised second primary molars (HSPM) is 2.7% - 21.8%, although the occurrence in Australian children remains unknown. These HSPM represent a potential predictive factor for molar-incisor hypomineralisation (MIH).

Methods: In total, 623 three to five-year-old children from 30 randomly selected kindergartens participated. The HSPM were recorded using an index combining the EAPD MIH Judgment Criteria and modified DDE Index. Caries was recorded using International Caries Detection and Assessment System criteria.

Results: In total, 144 HSPM were observed in 88/623 (14.1%) children, a tooth-level prevalence of 5.8%. The prevalence of dentinal carious lesions was 13.2%, and caries prevalence ($d_{2-6mft} > 0$) was 36.4%. Cavitated carious lesions affected 30.7% of HSPM.

Conclusions: The relationship between an increase in HSPM lesion extent and increasing number of HSPM per child was statistically significant. A positive association between HSPM severity and extent at tooth-level existed ($p < 0.05$). There was a positive relationship between the extent of HSPM and carious lesion severity ($p < 0.05$). In this population, children with HSPM did not have overall greater caries experience than unaffected children.

Key words: Australian preschool children, demarcated hypomineralised lesion of enamel, developmental enamel defects, early childhood caries, hypomineralised second primary molars

Abbreviations and acronyms: AC – atypical caries, AE – atypical extraction, AR – atypical restoration, EAPD – European Academy of Paediatric Dentistry, ECC – early childhood caries, DDE – developmental defect of enamel, DHLE - demarcated hypomineralised lesions of enamel, dmft – primary decayed, missing (due to decay) and filled teeth, FPM – first permanent molar, HSPM - hypomineralised second primary molars, ICDAS II – International Caries Detection and Assessment System II, LGA – local government area, mDDE – modified Developmental Defect of Enamel Index, PEB – post-eruptive breakdown, SPM – second primary molar/s

Main Text

Introduction

Dental caries and developmental enamel defects (DDE) are the most common oral health conditions affecting children worldwide.^{1,2} Early childhood caries (ECC) remains a prevalent chronic disease in Australian preschool children, which may lead to discomfort and pain that compromises the child's ability to eat, sleep and overall quality of life.^{3,4} The prevalence of demarcated, hypomineralised enamel lesions (DHLE) in second primary molars (HSPM) ranges from 2.7% to 21.8%.⁵⁻¹³ The presence of DHLE may increase caries experience in individuals with increased caries-risk, and occurrence in Australian children is unknown.^{6-8,14-16}

Worldwide knowledge of DHLE involves first permanent molars predominantly, and there is a scarcity of information regarding the primary dentition. The majority of epidemiological surveys regarding primary teeth have focussed on the prevalence of enamel hypoplasia, neglecting hypomineralisation.^{6,8} The literature has often grouped diffuse and demarcated opacities together and failed to differentiate between enamel hypoplasia and hypomineralisation lesions with post-eruptive enamel loss, which has likely led to under-reporting of enamel hypomineralisation in primary teeth.^{6,8} Fortunately, comparable studies

dedicated to HSPM prevalence using EAPD criteria are improving the comparison and amalgamation of research data.⁵⁻¹³

A prevalent spectrum of enamel defects in permanent teeth, termed Molar-Incisor Hypomineralisation (MIH), affects approximately one in five Australian children.¹⁷ The second primary molars (SPMs) and first permanent molars (FPMs) share a period of amelogenesis, therefore individuals affected by MIH may be at increased risk of HSPM.^{6,8,10,18} Hypomineralised second primary molars are not only considered a similar and related phenomenon to MIH, but represent a unique predictive factor for MIH.^{5,6,8-10,19}

Both MIH and HSPM enamel lesions are classified as demarcated creamy-white or yellow-brown opacities, which may be associated with post-eruptive breakdown (PEB). Hypomineralised second primary molars appear susceptible to similar problems experienced by MIH-affected teeth, including increased susceptibility to carious lesion development, sensitivity, and the increased need for restorations and extractions.^{5-8,19} The likelihood of HSPM developing carious lesions appears to be modulated by overall caries risk and severity of the enamel hypomineralisation, although this relationship remains poorly understood.^{6,8} Clinical challenges for affected individuals and clinicians are evident, however, costs related to management of HSPM are poorly understood, and in all likelihood, under-estimated. Therefore, an improved understanding of the role of these enamel defects in ECC is needed.

The aim of this study was to determine in Melbourne preschool children: (1) the prevalence of HSPM; (2) the relationship between DHLE severity, extent and number of affected HSPM; (3) the prevalence of early childhood caries (ECC); (4) the relationship between ECC and HSPM.

Materials and Methods

Sampling procedure

The study was conducted in inner metropolitan Melbourne, the 2nd largest city in Australia. At June 2013, there were an estimated 4.35 million people residing in Greater Melbourne (Australian Bureau of Statistics. Regional Population Growth, Australia. Released 03 April

2014 edn, accessed 10 March 2015). The randomised cluster sample was derived from the eight most central Local Government Areas (LGAs), which included the City of Melbourne, and the surrounding LGAs, to therefore enable equal representation in all directions from the Central Business District.

The Australian Bureau of Statistics Census of Population and Housing 2011: SEIFA, which combines recognised factors in determining advantage or disadvantage; including income, education and skills, employment, housing costs, health and geographic isolation was utilised as a socio-economic indicator of the study population.²⁰ The average SEIFA of the study population was 1024, which equates to the children attending an early childhood centre in the most advantaged 20% of all LGAs in Australia.²⁰ However, within the LGAs in the present study there were still children who resided in large public housing estates. The reticulated water supply to this area has 1.0 ppm F.

Approximately 23,000 children aged 3-5 years resided in the eight LGAs selected for this study. Sample size calculations, based on Confidence Interval (CI) at 95% $Z = 1.96$, caries prevalence of 34% and estimate of precision of 5% gave a sample size of 345. Note that for an estimated HSPM prevalence of 5% (and precision of 2.5%), a sample size of 292 was determined, so the larger sample size was used. Therefore, a conservative design effect of 1.8 was estimated for this sample, giving a final sample size of 621. The final total of children examined was 623.

Ethical approval for a larger project, including this study, was obtained from The Department of Education and Early Childhood Development, State of Victoria, Evaluation and Analytics Branch, and The University of Melbourne Human Research and Ethics Committee. The larger project included the prevalence of ECC and enamel defects in the entire dentition and salivary biomarkers for ECC.

Study setting and examination criteria

A total of 283 early childhood centres, from the target area included childcare centres, council administrated kindergartens and independent kindergartens. The early childhood centres were selected randomly from each respective LGA using Microsoft® Excel® (version 14.0 – 2010 spreadsheet, Microsoft Corp., WA, USA). The number of centres sampled was in proportion to the number of early childhood centres in each LGA. Once consent had been obtained from an early childhood centre, parents/guardians wanting to participate completed

a written consent form, and medical and socio-demographic questionnaire.

Training and calibration exercises in detection and diagnosis of dental caries using International Caries Detection and Assessment System (ICDAS II) scoring criteria and enamel defects using the modified EAPD evaluation criteria were conducted by an experienced trained examiner.^{19,21} A set of 20 photographs were examined three times for each DDE and dental caries. Using Kappa statistic, the intra-examiner agreements for DDE and dental caries scores were 0.94 and 0.96, respectively. The inter-examiner agreements for DDE were 0.97 and 0.96 for dental caries.

Data collection was conducted from May to December, 2013.

The assessment of consented child participants included a dental examination of approximately ten minutes in duration. The teeth were brushed with a new children's soft bristled toothbrush. The children were examined in a lap-to-lap position. The buccal, occlusal and lingual / palatal surfaces of the teeth were dried with a cotton wool roll and evaluated by visual examination using a DenLite[®] illuminated mirror system (Welch Allyn Inc., NY, USA), and the prevalence and extent of enamel defects and carious lesions recorded. Where children demonstrated carious lesions on exposed tooth surfaces, or if approximal lesions were suspected, fibre-optic trans-illumination of all contact point areas was conducted to improve the reliability of the dental caries diagnosis using the Raddi plus LED attachment[®] (SDI Ltd., Bayswater, Vic, Australia). Enamel defects less than two millimetres in diameter were not recorded.

Where the size of form of dental carious lesion/s in SPMs was incongruent with the child's caries-risk, especially where other corresponding SPM were affected by DHLE, this was termed atypical caries (AC). Where the size and shape of a restoration did not conform to the overall caries-risk context, and other corresponding molars were affected by DHLE, it was assumed that the restoration was the direct result of HSPM, termed atypical restoration (AR). In certain instances, the demarcated opacity was evident at the carious lesion or restoration border; AR did not include those consistent with traditional carious lesion management, especially where corresponding molars were not affected by HSPM. The data were recorded onto two specially prepared dental record charts by a trained research assistant.²² A tooth with 'opacities only' was defined as mild HSPM, whilst those with PEB or AR were termed moderate/severe HSPM.

A SLR digital camera (Canon® 40 D body with Canon® EF 100 mm f/2.8 USM Macro Lens and Canon® MacroRing Lite MR-14EX external flash; Canon Corp., Tokyo, Japan) was used to record images to demonstrate the types of EDs observed and for calibration purposes; children were unidentifiable from such images (Fig.1).

Statistical analysis

All information gained from the questionnaire, DDE and ICDAS II coding was entered into Microsoft® Excel®. All of the data were then transferred to the program IBM® SPSS® version 21 (IBM Inc. NY, USA). The data were cleaned by labelling the variables and running the frequency of each on SPSS® to ensure all values corresponded with those entered on the spreadsheet.

A descriptive analysis of the prevalence and distribution of the clinical recordings was performed. Pearson's Chi-square, Spearman's Correlation or Fisher Exact tests were utilised for nominal or ordinal variables. Odds ratios (OR) at the 95% confidence intervals (CI) were calculated to determine the difference in HSPM distribution between age groups. Continuous variables were compared using one-way analysis of variance (ANOVA) tests. An alpha level (p) of < 0.05 was considered statistically significant.

Results

A total of 33 of 58 early childhood centres invited to participate in the research project agreed to participate. However, due to inadequate interest from parents/guardians at three centres, 30 centres were included in the sample. A total of 705 of 1,352 three to five-year-old children had their parents/guardians consent for them to participate in the study, and 623 children were present for the examination, a consent rate of 52% and participation rate of 46%. Of the 623 participants, 353 (56.7%) were aged four years, followed by the three-year old group of 142 (22.8%) and the five-year old children 128 (20.5%). Male participants comprised 327 (52.5%) and female participants 296 (47.5%) of the study population. All primary teeth were erupted, other than in four children with congenitally missing primary incisor teeth (12,432 primary teeth examined).

HSPM was diagnosed in 88 children with 144 teeth affected by HSPM, a prevalence of 14.1% and a tooth-level prevalence of 5.8%, with maxillary and mandibular teeth affected in near

equal numbers. Most children had one HSPM 59.1% (N = 52), with 23.9% affected twice (N = 21), 11.4% thrice (N = 10), and 5.7% (N = 5), had four HSPM. In order of decreasing prevalence these were demarcated creamy-white opacity 42.4% (N = 61), demarcated yellow-brown opacity 26.4% (N = 38), demarcated yellow-brown opacity with PEB 16.7% (N = 24), demarcated creamy-white opacity with PEB 11.8% (N = 17), and ARs 2.8% (N = 4).

The mean number of HSPM per affected child according to age, for three, four and five year-olds was 1.77 ± 1.11 (95% CI 1.28 - 2.26); 1.57 ± 0.84 (95% CI 1.33 - 1.80) and 1.69 ± 0.75 (95% CI 1.45 - 1.83), respectively. The differences between age groups were not statistically significant ($p > 0.05$).

There was a statistically significant increase in the extent of the DHLE affecting the SPM/s and increasing number of HSPM affected per child, ($\chi^2(5) = 6.3, p = 0.01$) (Table 1). In children with four HSPM, 20% of the lesions involved most of the tooth surface, compared to 13.5% of cases with one HSPM.

A statistically significant relationship between HSPM lesion severity and HSPM lesion extent at the tooth level was determined (Spearman Rank Correlation 0.499; $\chi^2(10), p < 0.001$), and is demonstrated in Fig. 2.

When reporting traditional WHO definition of carious lesions, i.e. dentinal lesions ($d_{4-6}mft > 0$), prevalence was 13.2%; when enamel lesions with surface integrity change ($d_{3-6}mft > 0$) were included, prevalence increased to 19.4%, whilst the inclusion of early enamel lesions ($d_{2-6}mft > 0$) increased prevalence to 36.4%. The mean caries experience at both tooth level ($d_{4-6}mft$) was lowest for three year-old children (0.63 ± 1.81), highest in the four-year group (0.98 ± 2.12), which was similar to the five-year old children (0.94 ± 1.71). However, the differences in mean $d_{4-6}mft$ according to age within HSPM affected and non-HSPM affected groups were not statistically significant. The children affected by enamel hypomineralisation had a lower prevalence of dental carious lesions (36.4%), compared to unaffected individuals (37.4%), although the differences were not statistically significant.

Overall, no significant difference in caries experience and severity in the SPM existed between those children with and without HSPM (Fig. 3).

Similarly, at the tooth-surface level HSPM molars (N = 144) were not significantly more likely to have carious lesions (ICDAS II Codes 2–6 or ICDAS II Codes 4–6) than SPMs without DHLE (N = 2348).

For affected HSPM, the frequency distribution and multivariate comparisons between HSPM severity in relation to carious lesion severity are illustrated in Fig. 4.

Tooth-level analysis of teeth affected by HSPM (N = 144) determined that the majority of HSPM (64.6%, N = 93) were sound (ICDAS II Code 0), indicating that 35.4% (N = 51) of HSPM were affected by dental caries (d₂₋₆). In total 20.8% (N = 30) of HSPM were affected by early enamel lesions, whilst only 7.6% (N = 11) had cavitated dentinal carious lesions (ICDAS II Codes 4–6). For HSPM with mild DHLE one-third had carious lesions (including white spots), whilst 40% of teeth with moderate/severe DHLE had carious lesions. Although a greater proportion of HSPM with moderate/severe DHLE had ICDAS II Codes 4–6 than those with mild DHLE (11.1% versus 6.1%), the relationship between carious lesion severity and HSPM severity did not reach statistical significance.

For the children with carious lesions in their HSPM (N = 33) the distribution of HSPM by carious lesion severity and the involved tooth surface area is tabulated (Table 2).

The majority of children had a combination of carious lesions (ICDAS II Codes 2 or 3) and DHLE affecting less than one-third of the tooth surface (54.5%, N = 18). Amongst the children with ICDAS II Code 4–6 lesions in their HSPM, 66.7% had DHLE involving greater than two-thirds of the tooth surface. A statistically significant relationship existed between the amount of tooth surface area in the SPM affected by the DHLE and the carious lesion severity in the HSPM ($\chi^2 (8) = 16.8, p < 0.05$).

Discussion

The present study is cross-sectional in nature and was conducted on children from the suburbs of inner Melbourne. As part of a broader epidemiological survey of dental caries and DDEs in the primary dentition, an HSPM prevalence of 14.1% was determined; comparable with studies worldwide.^{5,6,8-13} No statistically significant relationship between age or gender was determined for HSPM.

The low caries prevalence of children was not unexpected, considering the well-established socio-demographic gradient of dental caries.^{1,2,23,24} The examination of preschool children with very little caries experience shortly after complete eruption of their primary dentition, facilitated complete and accurate HSPM diagnosis. Very few SPMs were missing, and none of these extractions were attributed to HSPM, which enabled comprehensive assessment of almost all the SPMs of the participants.

Most DDE studies in primary teeth predate the inception of the EAPD judgment criteria for MIH in 2003, which led to improved recognition of DHLE.²⁵⁻³⁴ The ability to record PEB is essential for DHLE diagnosis, and the EAPD criteria help ensure that this characteristic is distinguished from other types of tooth surface loss.¹⁹ The present study was the first to apply the novel twelve-point scoring system, which represent an amalgamation of the mDDE index and EAPD judgement criteria to the entire primary dentition.²² Demarcated hypomineralised lesions of enamel and DHLE with PEB are each distinguished by colour, and therefore sub-categorised as either creamy-white or yellow-brown. The presence of AC, AR and AE due to DHLE were applied to HSPM using the same principles as the EAPD criteria for MIH.¹⁹ No missing teeth in the present study were attributed to HSPM. A tooth with demarcated hypomineralised opacities only was considered less severely affected than a tooth with PEB, AR or AC. Demarcated hypomineralised lesions without PEB comprised more than two-thirds of DHLE defects amongst HSPM, and 42.4% of HSPM had creamy-white opacities in the present study. Regarding HSPM teeth with moderate/severe defects, yellow-brown lesions with PEB were most prevalent, comprising 16.6%, whilst creamy-white lesions with PEB comprised 11.8%; 2.8% of all HSPM defects were ARs.

Used widely in epidemiological research throughout the world, ICDAS II is gradually becoming the reference standard for carious lesion detection and assessment in both the primary and permanent dentition.^{21,35-39} The ability to record carious lesions in their early stages makes ICDAS II more sensitive than the WHO dmft index.⁴⁰ A key advantage of ICDAS II is the ability to accurately record both the presence of carious lesions and their severity, measures that can be analysed relative to features of the DDE.^{21,26,40} By converting the ICDAS II codes to d₄₋₆mft, comparison can be made with other studies using the WHO criteria without discarding the important early enamel lesions from the dataset.

There was a trend between a greater number of moderate/severe HSPM defects and increasing number of HSPMs affected. Therefore, children with one or two affected HSPM

were more likely to have mild lesions without PEB, whilst participants with three or four affected SPMs experienced HSPM moderate/severe lesions (with PEB or AR) more frequently. Other HSPM epidemiological studies have not provided findings that either support or refute this trend. However, regarding MIH research, a significant positive relationship between the number of hypomineralised FPMs and the MIH-defect severity has been observed previously.⁴¹⁻⁴⁴

The relationship between more extensive HSPM lesions and a greater number of HSPM in each affected child was statistically significant. In children with only one HSPM, 71.2% had DHLE that covered less than one-third of the tooth surface, compared with 40.0% in cases with four molars affected. For the most extensive DHLE in SPMs, 13.5% of children with one HSPM were affected, compared to 20.0% of individuals with four HSPM, a previously unreported relationship, however, an identical relationship has been determined in MIH research.^{5,6,9,41}

For HSPM a statistically significant relationship between the DHLE severity in terms of clinical presentation and the surface area extension was found. The most minimal defect extension was most frequent in creamy-white DHLE than other DHLE types, being observed in greater than 90% of these defects. By comparison, DHLE involving greater than two-thirds of the tooth surface were rarely seen in creamy-white opacities without PEB (1.6%); being over three times as frequent for yellow-brown opacities without PEB, and over sixteen times more common for opacities with PEB. Overall, the most extensive DHLE in SPMs were eleven times more frequent in teeth with moderate/severe DHLE, compared to HSPM with mild DHLE only. Although this association has not been reported for HSPM in the literature, an identical significant relationship was reported for MIH-affected teeth and therefore future epidemiological investigation for HPSM is required.⁴¹

Although HSPM with severe rather than mild DHLE were affected by carious lesions 1.8 times more frequently, the differences between caries-risk according to defect severity were not statistically significant. Therefore, in this low caries prevalence population, DHLE cannot be considered a significant caries risk factor. The present study findings conflict with the vast majority of research in populations with higher caries prevalence, which have determined that EDs predispose affected teeth to development of carious lesions in those at increased caries-risk.^{8, 45-50} By comparison, regarding HSPM in a higher caries-risk population, Ghanim et al determined a significant association between carious lesion severity and HSPM severity.⁸

The difference in findings may be attributed in part to the low caries experience in the present study, and the resultant small number of HSPM with cavitated lesions.⁸

The depth and location of the PEB, whether it exposed enamel or dentine and the individuals' caries-risk may be more crucial to the prognosis the HSPM, than PEB itself. Although not recorded in the index, photographs of HSPM in the present study predominantly demonstrate PEB exposing enamel only. However, one may assume greater instances of PEB into dentine and carious lesion development would occur in older children, after prolonged function and exposure to more periods of caries risk. The nature of DHLE pathogenesis over time, and how this may modulate caries-risk in a population such as the present study is unknown. One cannot assume that caries-risk for HSPM remains static, and an improved understanding of which teeth and children are at greatest risk for defect 'progression' is needed.

Interestingly, in the present study population, the defect extent rather than defect severity was more critical in terms of carious lesion development. This may be due to the areas of the tooth affected in more extensive lesions, such as cuspal tips. Consequently, a more moderate/severe DHLE of minimal extent appears to be at lower risk for dentinal caries, than HSPM lesions of greater extent, all moderated by the individual caries-risk.

The potential manner in which caries-risk may modulate HSPM prognosis in primary teeth requires further investigation. Research in populations of different caries experience in both the preschool age group and into the mixed dentition may provide a more nuanced understanding of HSPM behaviour, and thereby assist development of evidence-based management guidelines, which can be tailored to the individual child.

Conclusions

The prevalence of HSPM was 14.1%. Creamy-white opacities represented 42.4% of HSPM lesions, with PEB affecting 28.5%. Atypical restorations were found in 2.8% of HSPM. More than half of all children with HSPM had only one affected tooth. More severe and extensive lesions correlated with increased numbers of HSPM. Children with HSPM did not have greater carious experience. A correlation between greater HSPM defect extent and increased carious lesion severity existed.

Reference

1. Tinanoff N, Reisine S. Update on early childhood caries since the Surgeon General's Report. *Acad Ped* 2009;9:396-403.
2. AAPD. Policy on Early Childhood Caries (ECC): Classification, Consequences and Preventive Strategies. *Pediatr Dent* 2014;37:50-52.
3. Armfield JM, Spencer AJ. Quarter of a century of change: caries experience in Australian children, 1977–2002. *Aust Dent J* 2008;53:151-159.
4. Sheiham A. Dental caries affects body weight, growth and quality of life in pre-school children. *Br Dent J* 2006; 201:625-626.
5. Elfrink M, Schuller A, Weerheijm K, Veerkamp J. Hypomineralized second primary molars: prevalence data in Dutch 5-year-olds. *Caries Res* 2008;42:282-285.
6. Elfrink M, Ten Cate J, Jaddoe V, Hofman A, Moll H, Veerkamp J. Deciduous molar hypomineralization and molar incisor hypomineralization. *J Dental Res* 2012;91:551-555.
7. Weerheijm KL, Elfrink ME, Kilpatrick N. Molar Incisor Hypomineralization and Hypomineralized Second Primary Molars: Diagnosis, Prevalence, and Etiology. *Planning and Care for Children and Adolescents with Dental Enamel Defects*. Heidelberg: Springer, 2015:31-44.
8. Ghanim A, Manton D, Marino R, Morgan M, Bailey D. Prevalence of demarcated hypomineralisation defects in second primary molars in Iraqi children. *Int J Paediatr Dent* 2013;23:48-55.
9. Costa-Silva CM, Simpson de Paula J, Ambrosano GM, Mialhe FL. Influence of deciduous molar hypomineralization on the development of molar-incisor hypomineralization. *Braz J Oral Sci* 2013;12:335-338.
10. Kühnisch J, Heitmüller D, Thiering E, Brockow I, Hoffmann U, Neumann C, Heinrich-Weltzien R, Bauer CP, von Berg A, Koletzko S, Garcia-Godoy F, Hickel R, Heinrich J. Proportion and extent of manifestation of molar-incisor-hypomineralizations according to different phenotypes. *J Public Health Dent* 2014;74:42-49.
11. Mittal R, Chandak S, Chandwani M, Singh P, Pimpale J. Assessment of association between molar incisor hypomineralization and hypomineralized second primary molar. *J Int Soc Prev & Community Dent* 2016;6:34-39.
12. Negre-Barber A, Montiel-Company JM, Boronat-Catalá M, Almerich-Silla JM. Hypomineralized Second Primary Molars as Predictor of Molar Incisor Hypomineralization. *Sci Rep* 2016;6:1-6.
13. Oyedele TA, Folayan MO, Oziegbe EO. Hypomineralised second primary molars:

prevalence, pattern and associated co morbidities in 8-to 10-year-old children in Ile-Ife, Nigeria. *BMC Oral Health* 2016;16:1-7.

14. Weerheijm K, Jälevik B, Alaluusua S. Molar–incisor hypomineralisation. *Caries Res* 2001;35:390-391.
15. Jälevik B, Klingberg G. Dental treatment, dental fear and behaviour management problems in children with severe enamel hypomineralization of their permanent first molars. *Int J Paediatr Dent* 2002;12:24-32.
16. Jeremias F, De Souza JF, Costa Silva CM, Cordeiro R, Zuanon ÂCC, Santos-Pinto L. Dental caries experience and Molar-Incisor Hypomineralization. *Acta Odontol Scand* 2013;1-7.
17. Arrow P. Prevalence of developmental enamel defects of the first permanent molars among school children in Western Australia. *Aust Dent J* 2008;53:250-259.
18. Brook A. Multilevel complex interactions between genetic, epigenetic and environmental factors in the aetiology of anomalies of dental development. *Arch Oral Biol* 2009;54:3-17.
19. Weerheijm KL, Duggal M, Mejare I, Papagiannoulis L, Koch G, Martens LC, Hallonstein A. Judgement criteria for molar incisor hypomineralisation (MIH) in epidemiologic studies: a summary of the European meeting on MIH held in Athens, 2003. *Eur J Paediatr Dent* 2003;4:110-113.
20. Australian Bureau of Statistics. Socio-economic Indexes for Areas (SEIFA), 2011. <http://www.abs.gov.au>. Accessed 20 March 2015.
21. Banting D, Eggertsson H, Ekstrand K, Zandoná AF, Ismail AI, Longbottom C, Pitts NB, Reich E, Ricketts D, Selwitz R, Sohn W, Topping GV, Zero D. International Caries Detection and Assessment System (ICDAS II). Criteria Manual Appendix. Workshop held in Baltimore, Maryland, March 12th - 14th 2005.
22. Ghanim A, Elfrink M, Weerheijm K, Marino R, Manton D. A practical method for use in epidemiological studies on enamel hypomineralisation. *Eur Arch Paediatr* 2015;16: 235-246.
23. Milen A. Role of social class in caries occurrence in primary teeth. *Int J Epi* 1987;16:252-256.
24. Harris R, Nicoll AD, Adair PM, Pine CM. Risk factors for dental caries in young children: a systematic review of the literature. *Community Dent Health* 2004;21:71-85.

25. Li Y, Navia JM, Bian JY. Prevalence and distribution of developmental enamel defects in primary dentition of Chinese children 3–5 years old. *Community Dent Oral Epidemiol* 1995;23:72-79.
26. Montero MJ, Douglass J, Mathieu G. Prevalence of dental caries and enamel defects in Connecticut Head Start children. *Pediatr Dent* 2003;25:235-256.
27. Murray J, Shaw L. Classification and prevalence of enamel opacities in the human deciduous and permanent dentitions. *Arch Oral Biol* 1979;24:7-13.
28. Nation W, Matsson L, Peterson J. Developmental enamel defects of the primary dentition in a group of Californian children. *ASDC J Dent Child* 1986;54:330-334.
29. Pascoe L, Kim Seow W. Enamel hypoplasia and dental caries in Australian aboriginal children: prevalence and correlation between the two diseases. *Pediatr Dent* 1994;16:193-193.
30. Rugg-Gunn A, Al-Mohammadi S, Butler T. Malnutrition and developmental defects of enamel in 2-to 6-year-old Saudi boys. *Caries Res* 1998;32:181-192.
31. Seow WK, Amaratunge A, Bennett R, Bronsch D, Lai P. Dental health of aboriginal pre-school children in Brisbane, Australia. *Community Dent Oral Epidemiol* 1996;24:187-190.
32. Slayton RL, Warren J, Kanellis M, Levy S, Islam M. Prevalence of enamel hypoplasia and isolated opacities in the primary dentition. *Pediatr Dent* 2001;23:32-43.
33. Weeks K, Milsom K, Lennon M. Enamel defects in 4-to 5-year-old children in fluoridated and non-fluoridated parts of Cheshire, UK. *Caries Res* 1993;27:317-320.
34. Kanchanakamol U, Tuongratanaphan S, Lertpoonvilaikul W, Chittaisong C, Pattanaporn K, Navia JM, Davies GN. Prevalence of developmental enamel defects and dental caries in rural pre-school Thai children. *Community Dent Health* 1996;13:204-207.
35. Pitts N. "ICDAS"-an international system for caries detection and assessment being developed to facilitate caries epidemiology, research and appropriate clinical management. *Community Dent Health* 2004;21:193-198.
36. Shoaib L, Deery C, Ricketts D, Nugent Z. Validity and reproducibility of ICDAS II in primary teeth. *Caries Res* 2009;43:442-448.
37. Ismail A, Sohn W, Tellez M, Amaya A, Sen A, Hasson H, Pitts NB. The International caries detection and assessment system (ICDAS): an integrated system for measuring dental caries. *Community Dent Oral Epidemiol* 2007;35:170-178.

38. Jablonski-Momeni A, Stachniss V, Ricketts D, Heinzl-Gutenbrunner M, Pieper K. Reproducibility and accuracy of the ICDAS-II for detection of occlusal caries in vitro. *Caries Res* 2008;42:79-87.
39. Honkala E, Runnel R, Honkala S, Olak J, Vahlberg T, Mare S, Kauko KM. Measuring dental caries in the mixed dentition by ICDAS. *Int J Dent* 2011; Article ID 150424, 2011 6 pages.
40. Banting D, Eggertsson H, Ekstrand KR, Ferreira Zandoná A, Ismail AI, Longbottom C, Pitts NB, Reich E, Ricketts D, Selwitz R, Sohn W, Topping GV, Zero D. Rationale and evidence for the international caries detection and assessment system (ICDAS II). *Int Caries Detection and Assessment System Coordinating Committee* 2012;1001:1-43.
<https://www.icdas.org/uploads/Rationale%20and%20Evidence%20ICDAS%20II%20September%2011-1.pdf>
41. Ghanim A, Manton D, Marino R, Morgan M, Bailey D. Molar-incisor hypomineralisation: prevalence and defect characteristics in Iraqi children. *Int J Pediatr Dent* 2011;11:413-421.
42. Lygidakis N, Dimou G, Briseniou E. Molar-incisor-hypomineralisation (MIH). Retrospective clinical study in Greek children. I. Prevalence and defect characteristics. *Eur Arch Paediatr Dent* 2008;9:200-206.
43. Wogelius P, Haubek D, Nechifor A, Nørgaard M, Tvedebrink T, Poulsen S. Association between use of asthma drugs and prevalence of demarcated opacities in permanent first molars in 6-to-8-year-old Danish children. *Community Dent Oral Epidemiol* 2010;38:145-151.
44. Soviero V, Haubek D, Trindade C, Da Matta T, Poulsen S. Prevalence and distribution of demarcated opacities and their sequelae in permanent 1st molars and incisors in 7 to 13-year-old Brazilian children. *Acta Odontol Scand* 2009;67:170-175.
45. Rodrigues C, Sheiham A. The relationships between dietary guidelines, sugar intake and caries in primary teeth in low income Brazilian 3 year-olds: a longitudinal study. *Int J Paediatr Dent* 2000;10:47-55.
46. Matee M, Hof M, Maselle S, Mikx F, van Palenstein-Helderman W. Nursing caries, linear hypoplasia, and nursing and weaning habits in Tanzanian infants. *Community Dent Oral Epidemiol* 1994;22:289-293.

47. Elfrink M, Veerkamp J, Kalsbeek H. Caries pattern in primary molars in Dutch 5-year-old children. *Eur Arch Paediatr Dent* 2006;7:236.
48. Seow W, Amaratunge A, Sim R, Wan A. Prevalence of caries in urban Australian aborigines aged 1-3.5 years. *Pediatr Dent* 1998;21:91-96.
49. Li Y, Navia J, Bian J. Caries experience in deciduous dentition of rural Chinese children 3-5 years old in relation to the presence or absence of enamel hypoplasia. *Caries Res* 1996;8-15.
50. Milgrom P, Riedy C, Weinstein P, Tanner A, Manibusan L, Bruss J. Dental caries and its relationship to bacterial infection, hypoplasia, diet, and oral hygiene in 6 to 36 month-old children. *Community Dent Oral Epidemiol* 2000;28:295-306.

Tables

Table 1. HSPM lesion extension in relation to the number of HSPM teeth for each affected child

Number of HSPM affected per child**	HSPM lesion extension*			Total N (%)
	< 1/3 N (%)	< 2/3 N (%)	> 2/3 N (%)	
One	37 (71.2)	8 (15.4)	7 (13.5)	52 (59.1)
Two	15 (71.4)	4 (19.0)	2 (9.5)	21 (23.9)
Three	1 (10.0)	6 (60.0)	3 (30.0)	10 (11.4)
Four	2 (40.0)	2 (40.0)	1 (20.0)	5 (15.9)
Total	55 (65.5)	19 (20.3)	14 (14.3)	88

HSPM: hypomineralised second primary molar/s

* HSPM lesion extension defines the proportion of the tooth surface affected by the lesion

** Statistically significant difference ($\chi^2(5) = 6.3, p = 0.01$)

Table 2. Distribution of HSPM by dental caries severity and the involved tooth surface area at tooth-level analysis

	HSPM – lesion extension*			Total
	<1/3 N (%)	<2/3 N (%)	>2/3 N (%)	
ICDAS II caries code**				
Distinct visual change in enamel (ICDAS II Code 2)	14 (73.7)	2 (28.6%)	3 (42.9)	19 (57.6)
Initial breakdown in enamel (ICDAS II Code 3)	4 (21.1)	4 (57.1%)	0	8 (24.2)
Dentinal shadow (ICDAS II Code 4)	1 (5.3)	0	1 (14.3)	1 (3.0)
Distinct cavity with visible dentine (ICDAS II Code 5)	0	1 (14.3)	2 (28.6)	4 (12.1)
Extensive cavity with visible dentine (ICDAS II Code 6)	0	0	1 (14.3)	1 (3.0)
Total	19 (57.6)	7 (21.2)	7 (21.2)	33

HSPM: hypomineralised second primary molar/s

ICDAS-II: International Caries Detection and Assessment System II

* HSPM lesion extension defines the proportion of the tooth surface affected by demarcated hypomineralised lesion/s of enamel

** Statistically significant increase in the severity of caries lesion per tooth surface with increasing amount of demarcated hypomineralised lesion/s of enamel extension ($\chi^2(8) = 16.8, p < 0.05$).

Figure Legends (Figures attached separately)

Figure 1. Clinical examples illustrating the different clinical presentations of DHLE. A, creamy-yellow; B, yellow; C, yellow-brown; D, creamy-yellow with PEB; E, brown with PEB; F, atypical caries; G, atypical restoration

DHLE: demarcated hypomineralised lesion of enamel

PEB: post-eruptive breakdown

Figure 2. Distribution of HSPM by lesion type and its extension at tooth-level analysis

DHLE: Demarcated hypomineralised lesion of enamel

HSPM: hypomineralised second primary molar/s

PEB: post-eruptive breakdown

* HSPM lesion extension defines the proportion of the tooth surface affected by the lesion

** Significant association between HSPM lesion severity and HSPM lesion extension (Spearman Rank Correlation 0,499; χ^2 (10), $p < 0.001$)

Figure 3. Proportion of HSPM affected and non-HSPM affected children by ICDAS II dental caries severity in their second primary molars

HSPM: hypomineralised second primary molar

ICDAS-II: International Caries Detection and Assessment System II

N (total) = 197 (number of children with carious lesions in their SPMs in the study population); N denotes the number of children with carious lesions of different ICDAS II severity in their SPMs according to whether they are HSPM affected or non-HSPM affected

% denotes the proportion of children which have carious lesions in their SPMs according to HSPM presence or absence in their dentition.

Figure. 4. Frequency distribution and multivariate comparisons between HSPM lesion severity in relation to ICDAS II dental caries severity at tooth level

HSPM: hypomineralised second primary molar

DHM: Demarcated hypomineralised lesion of enamel

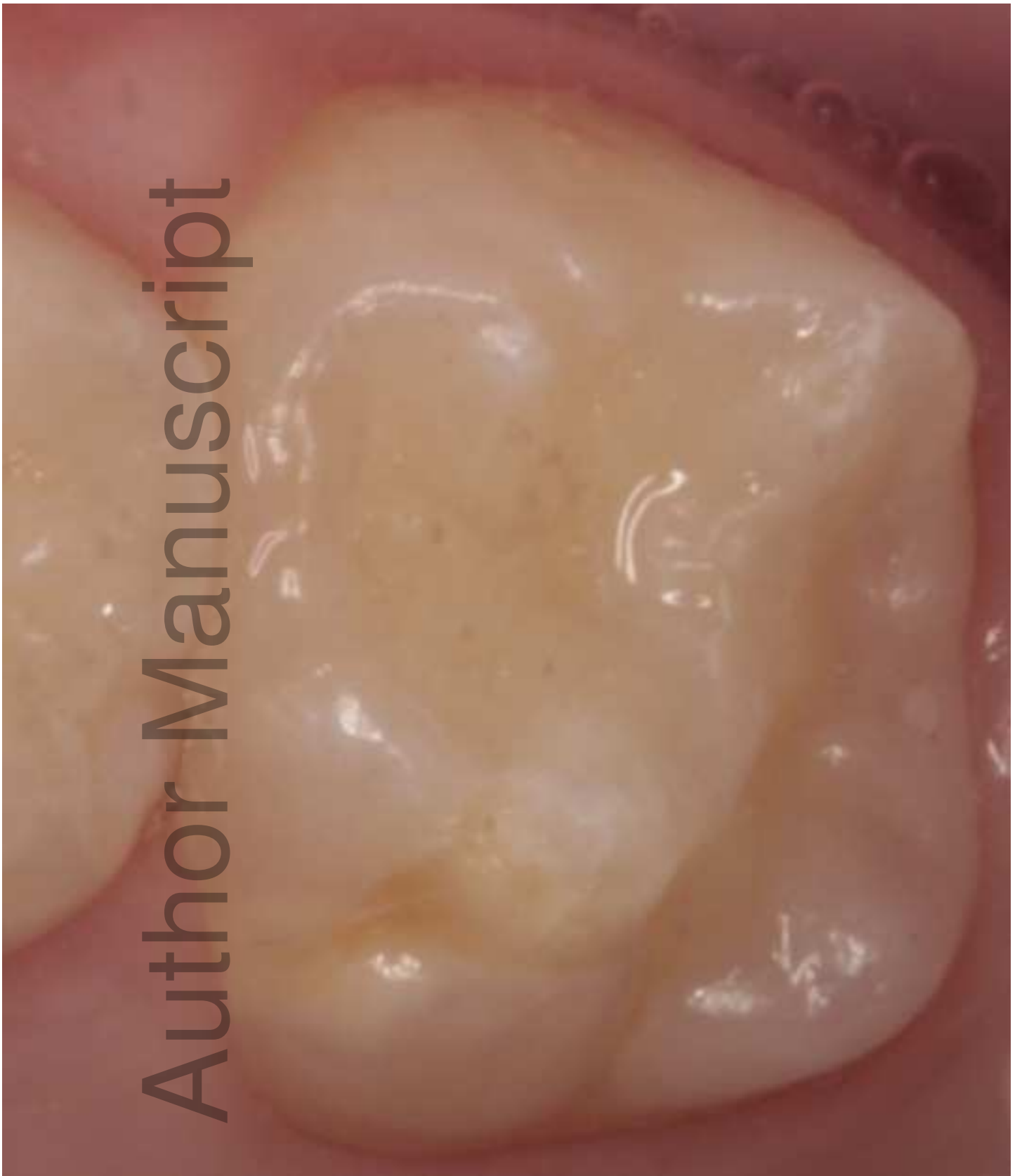
ICDAS-II: International Caries Detection and Assessment System II

N (total) = 144; N denotes the total number of HSPM affected teeth with different ICDAS II dental caries severity codes according to whether they have mild or moderate/severe DHLE in their affected SPMs

% denotes the proportion of teeth with either mild or moderate/severe HSPM lesions according to their ICDAS II dental caries status



adj_12567_f1-1.tif



adj_12567_f1-2.jpg



adj_12567_f1-3.jpg

Author Manuscript



adj_12567_f1-4.tif

Author Manuscript



adj_12567_f1-5.tif

Author Manuscript



adj_12567_f1-6.tif



adj_12567_f1-7.jpg

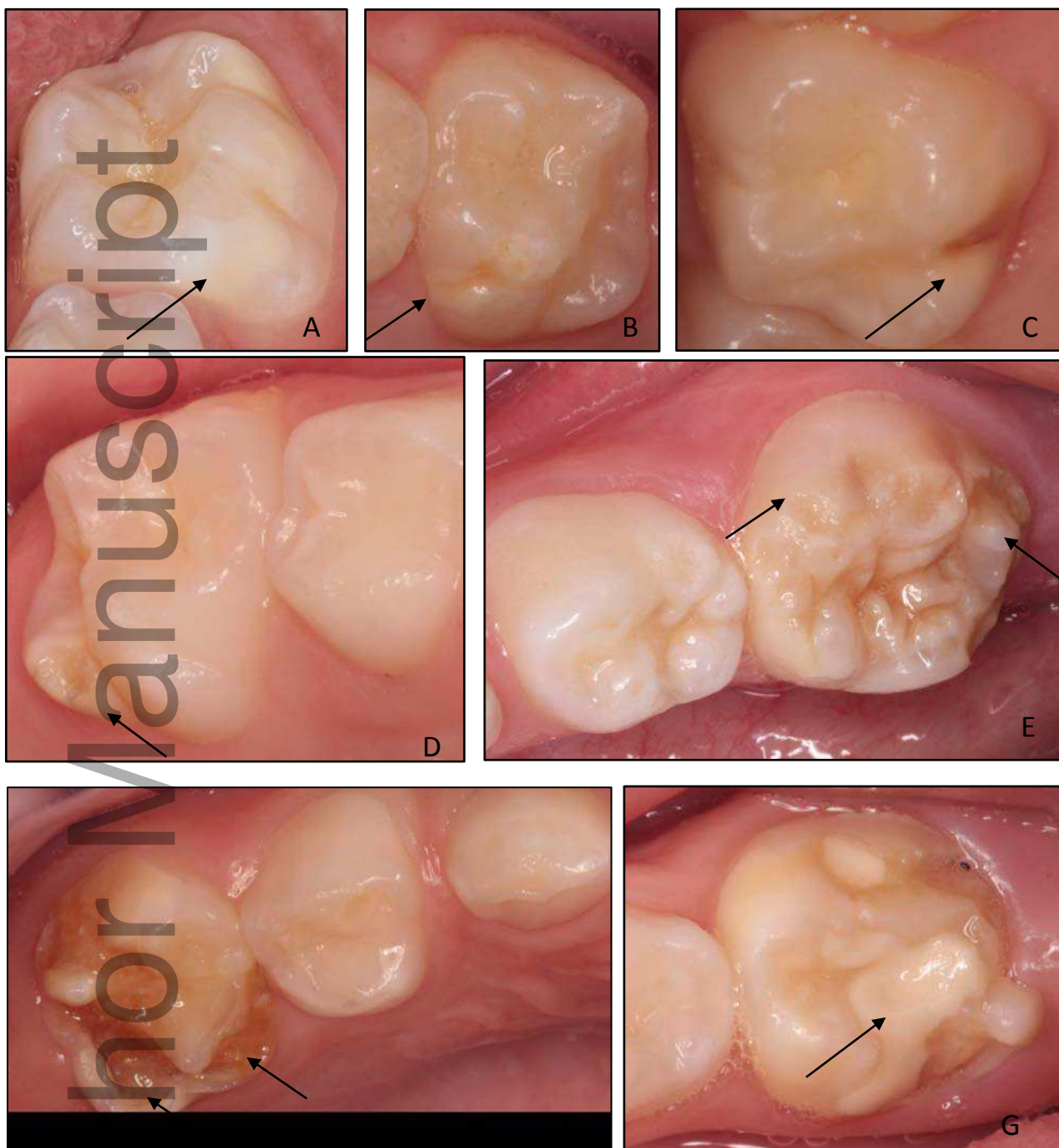


Figure 1. Clinical examples illustrating the different clinical presentations of DHLE.

A, creamy-yellow; B, yellow; C, yellow-brown; D, creamy-yellow with PEB; E, brown with PEB; F, atypical caries; G, atypical restoration

DHLE: demarcated hypomineralised lesion of enamel

PEB: post-eruptive breakdown

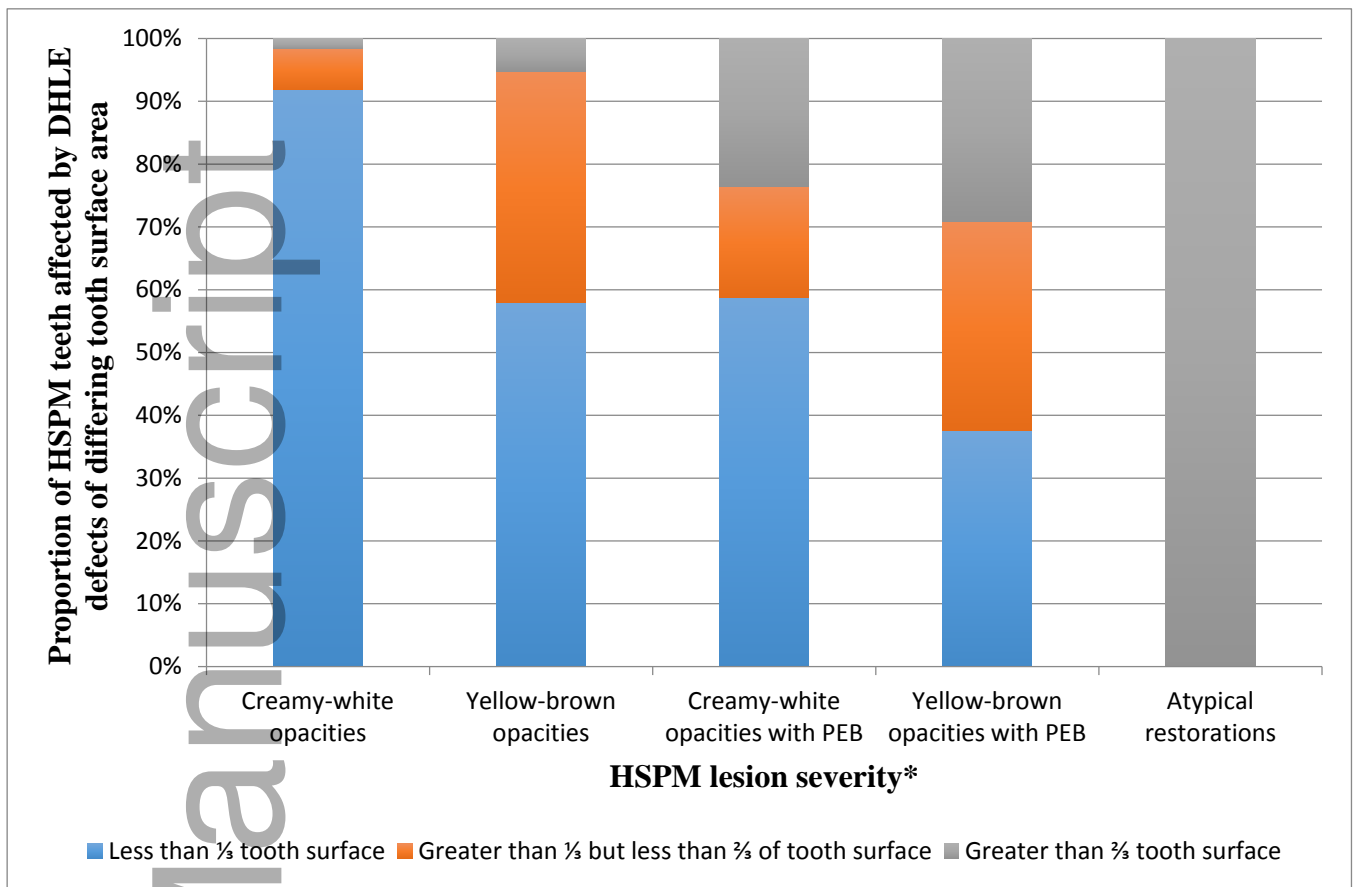


Figure 2. Distribution of HSPM by lesion type and its extension at tooth-level analysis

DHLE: Demarcated hypomineralised lesion of enamel

HSPM: hypomineralised second primary molar/s

PEB: post-eruptive breakdown

* HSPM lesion extension defines the proportion of the tooth surface affected by the lesion

** Significant association between HSPM lesion severity and HSPM lesion extension (Spearman

Rank Correlation 0,499; χ^2 (10), $p < 0.001$)

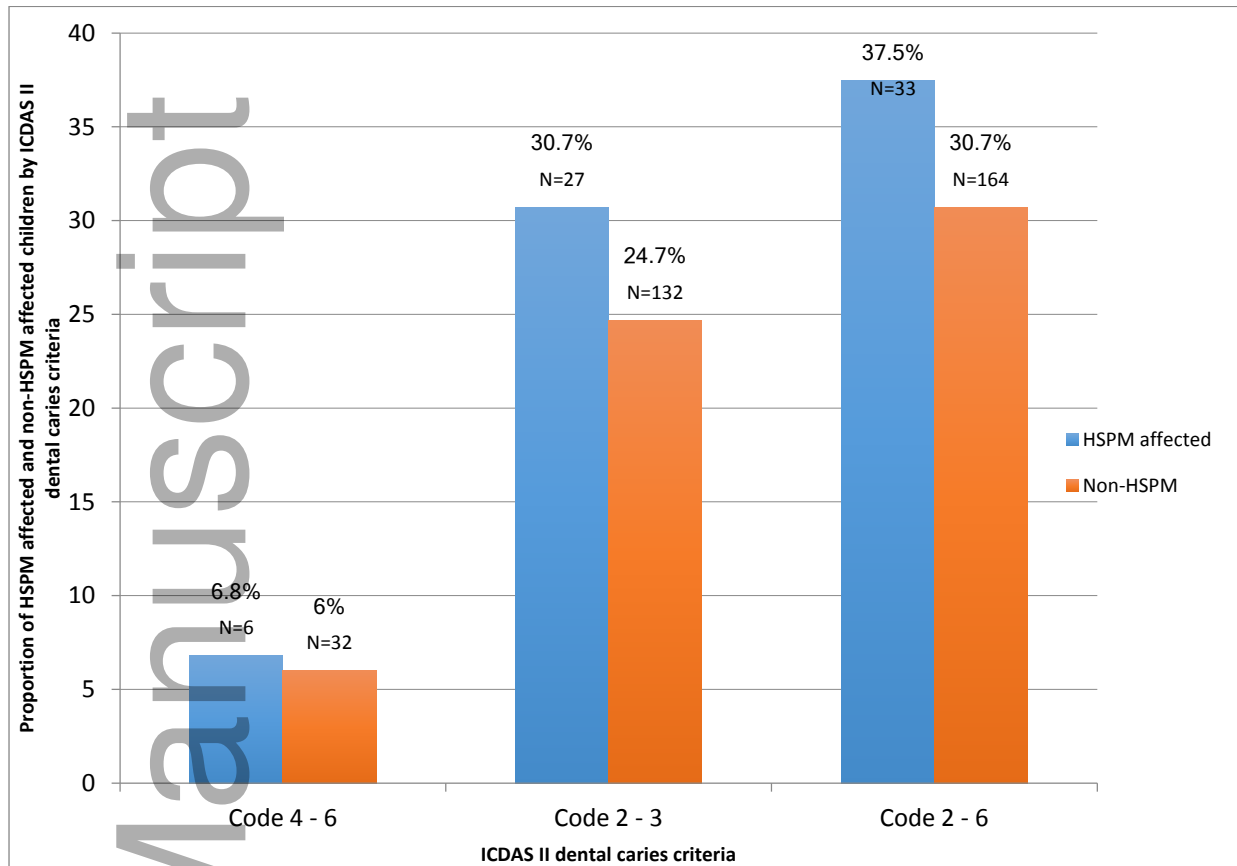


Figure 3. Proportion of HSPM affected and non-HSPM affected children by ICDAS II dental caries severity in their second primary molars

HSPM: hypomineralised second primary molar

ICDAS-II: International Caries Detection and Assessment System II

N (total) = 197 (number of children with carious lesions in their SPMs in the study population); N denotes the number of children with carious lesions of different ICDAS II severity in their SPMs according to whether they are HSPM affected or non-HSPM affected

% denotes the proportion of children which have carious lesions in their SPMs according to HSPM presence or absence in their dentition.

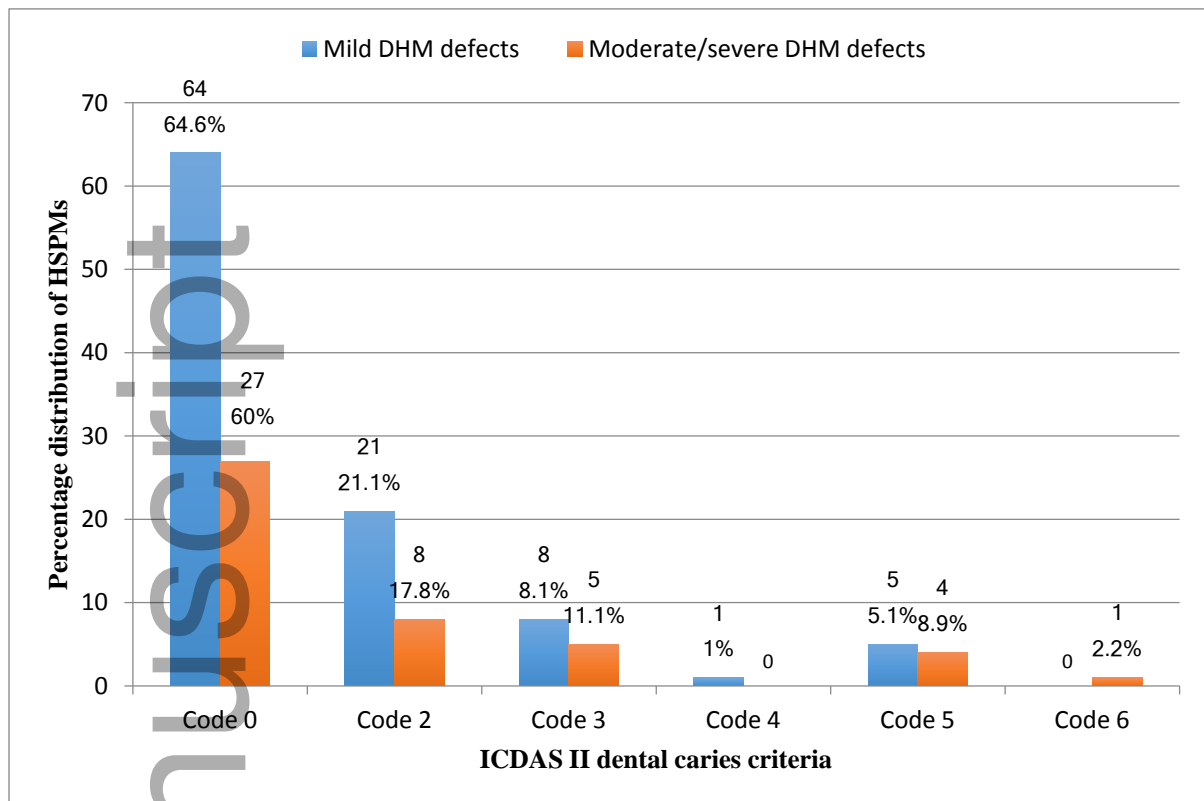


Figure 4. Frequency distribution and multivariate comparisons between HSPM lesion severity in relation to ICDAS II dental caries severity at tooth level

HSPM: hypomineralised second primary molar

DHM: Demarcated hypomineralised lesion of enamel

ICDAS-II: International Caries Detection and Assessment System II

N (total) = 144; N denotes the total number of HSPM affected teeth with different ICDAS II dental caries severity codes according to whether they have mild or moderate/severe DHLE in their affected SPMs

% denotes the proportion of teeth with either mild or moderate/severe HSPM lesions according to their ICDAS II dental caries status