DOI: 10.1111/j.1365-263X.2011.01143.x

Molar-incisor hypomineralisation: prevalence and defect characteristics in Iraqi children

AGHAREED GHANIM, MICHAEL MORGAN, RODRIGO MARIÑO, DENISE BAILEY & DAVID MANTON

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

International Journal of Paediatric Dentistry 2011; 21: 413–421

Background. Little prevalence data relating to molar incisor hypomineralisation (MIH) exist for Middle East populations.
Aim. To evaluate the prevalence and the clinical features of MIH in school-aged children residing in Mosul City, Iraq.
Design. A cluster sample of 823 7- to 9-year-old children had their first permanent molars and incisors (index teeth) evaluated using the European Academy of Paediatric Dentistry (EAPD) criteria for MIH. The examinations were conducted at schools by a calibrated examiner.
Results. Of the children examined, 177 (21.5%) had hypomineralisation defects in at least one index tooth, 153 (18.6%) had at least one affected first molar or first molars and incisors and were considered as having MIH. The most commonly affected teeth were maxillary molars. Demarcated creamy white opacities were the most frequent lesion type. Dental restorations and tooth extraction because of MIH were uncommon. Children with three or more affected teeth were 3.7 times more likely to have enamel breakdown when compared with those children having only one or two affected teeth.
Conclusions. Molar incisor hypomineralisation was common amongst Iraqi children. Demarcated opacities were more prevalent than breakdown. The severity of the lesions increased with the number of affected teeth. The more severe the defect, the greater the involved tooth surface area.

Introduction

Over the past two decades, a congenital defect of enamel mineralisation commonly referred to as molar incisor hypomineralisation (MIH) has been of increasing concern to clinicians worldwide. The defect involves hypomineralisation of one to four permanent first molars and is associated frequently with similarly affected permanent incisors[1].

Clinically, the defect presents as opaque lesions varying in colour from white to yellow or brown, with a sharp demarcation between the affected and sound enamel. In severe cases, post-eruptive enamel breakdown (PEB) can occur so rapidly that it appears clinically as if the enamel has not formed at all. When PEB occurs because of chewing forces, it is more conspicuous in the first permanent molars than in the incisors[1].

It has been postulated that MIH is a consequence of a variety of environmental factors acting systemically, which disturb the ameloblasts during their enamel production phase. However, the possibility of a genetic component in the development of MIH has not been excluded[2,3].

Recent studies have emphasised that the presence of MIH can produce a number of problems for the patient including dental pain, disfigurement, encouragement of rapid plaque retention, and enhancement of caries development[4,5]. The need for orthodontic treatment intervention as a consequence of tooth extraction caused by MIH has also been reported[6,7]. Moreover, substantial challenges to dental care are caused by MIH because of the lack of appropriate restorative management and difficulties with pain control[8].

A wide variation in the reported MIH-prevalence data exists. The majority of the data originate in European countries with a

Correspondence to:
Aghareed Ghanim, Melbourne Dental School, The University of Melbourne, Parkville, Vic. 3010 Australia.
E-mail: dentagh@gmail.com

prevalence rate varying from 3.6% to 37.5%[9,10]. Outside Europe, data from Western Australia give a prevalence of demarcated opacities as 22%[11], whereas data from Hong Kong indicate the lowest prevalence of MIH in the literature (2.8%)[12]. The highest recorded prevalence reported was observed amongst Brazilian subjects (40.2%)[13]. This broad range in the reported data may be attributed to the actual differences in MIH prevalence between different regions, masking of MIH by other conditions, possible prevalence variations by age cohorts, and/or to the different clinical examination protocols used to identify this condition[14]. Surprisingly, even following the establishment of the European Academy of Paediatric Dentistry (EAPD) evaluation criteria for MIH, a definitive global prevalence value table is not available. Data from North American and Middle Eastern populations, for instance, are lacking.

Iraq is one of the Middle East countries that have experienced a long series of military conflicts in the recent past resulting in the deterioration of public health in general and in child health in particular[15]. A recent survey amongst Iraqi dental academics investigating their perception of the existence and prevalence of MIH in Iraqi children highlighted the need to map the true prevalence of MIH amongst Iraqi children and to compare it with international data[16]. Because of these reasons and the clinical importance of MIH, this study aims to assess the prevalence of MIH in a representative group of primary school children resident in Mosul City, Iraq as well as to describe its distribution in first permanent molars and incisors and to determine the defect severity in relation to the number and type of the affected teeth.

Methods

Characteristics of the project location

Mosul is the second largest city in Iraq after the capital Baghdad and is located in the north part of the country on both sides of the Tigris River. Its 1,800,000 inhabitants are provided with a communal water supply from the Tigris River having a fluoride concentration 0.19 ± 0.07 ppm[17].

Study design and sampling procedure

Ethical approval was obtained from the Human Research Ethics Committee of the University of Melbourne and the Dental College of Mosul University. Permission from the Education Department of the Neinavha Governorate was also obtained to conduct a cross-sectional survey amongst 7- to 9-year-old children attending public primary schools in Mosul City Centre for the academic year 2009–2010. Cohorts of children born in 2000, 2001, and 2002 were recruited. Fifty-two schools were selected randomly from the schools list (total n = 102), equally distributed along the right and left sides of the Tigris River and containing several classes with second-, third-, and fourth-grade pupils. Directors of the selected schools were contacted by the researcher (AG) and invited to have their schools included in the study. Using a random starting point, children within schools were selected from a list of child names, and every fourth child was sampled. The total number of potential subjects was 1000 with equal numbers of both genders and age groups proposed to be examined. A package containing an information brochure explaining the aims, characteristics, and importance of the study was given by AG to the selected children to pass on to their guardians. A consent form asking guardians to give permission for their children to participate in the study was also included.

Calibration

Before initiation of the study, a calibration exercise was carried out for the examiner (AG) with the aid of experienced trained examiners at the Melbourne Dental School. A set of 20 photographs including both MIH and other enamel developmental defects was scored and the exercise repeated twice. Using kappa statistics, the inter- and intra-examiner agreements for MIH were reported as good (0.64) and excellent (0.80), respectively.

Inclusion and exclusion criteria of the study population

Inclusion criteria:

- Children (born in 2000, 2001, and 2002) of lifelong residents in Mosul City having at least one first permanent molar erupted or partially erupted and present at school on the day of examination.

Exclusion criteria:

- Children having amelogenesis imperfecta, tetracycline staining, or undergoing orthodontic treatment at the time of assessment.
- Children in whom the crowns of the first permanent molars were completely broken down where the potential cause of breakdown was impossible to determine.

Study setting

Prior to the clinical examination, the participating children were given a toothbrush and fluoridated toothpaste to brush their teeth thoroughly at the school under the supervision of the examiner. The clinical examination was performed in a suitable school environment with the aid of an assistant to record the observations on a pre-tested data sheet provided for that purpose. Children were asked to lie flat on a school table or desk. The teeth were dried using sterile cotton rolls and examined with the aid of a portable light source (Denlite, Miltex, PA, USA) and a disposable mirror head. A ball-ended explorer was drawn across the tooth surface to detect any surface continuity interruption. To inform parents about their child's oral health and indicate the need for treatment at the completion of the dental examination, each child was given a letter outlining their dental health status.

Survey instrument and examination criteria

The buccal/labial, occlusal/incisal, and lingual/palatal surfaces of the index teeth (permanent first molars and incisors) were examined. Because there was no available national system for the purpose of recording demarcated hypomineralisation defects, a 10-point scoring system based on the EAPD evaluation criteria[18] (as illustrated in Table 1) was used. To avoid confusion, the term molar hypomineralisation (MH) was used when referring to demarcated hypomineralisation defects affecting first molars. The term incisor hypomineralisation (IH) was used to refer to demarcated hypomineralisation defects affecting incisor teeth only, whereas the term MIH was used to refer to at least one affected first molar or a combination of affected first molars and incisors. Teeth that were erupted less than one third of the crown height were considered as unerupted. Demarcated hypomineralisation-related enamel defects were distinguished from diffuse opacities and hypoplasia following FDI 1992 definitions[19].

Table 1. Criteria for scoring molar incisor hypomineralisation according to European Academy of Paediatric Dentistry recommendations.

Code	Criteria
0	Enamel defect free
1	White/creamy demarcated opacities, no PEB
1a	White/creamy demarcated opacities, with PEB
2	Yellow/brown demarcated opacities, no PEB
2a	Yellow/brown demarcated opacities, with PEB
3	Atypical restoration
4	Missing because of MIH
5	Partially erupted (i.e., less than one-third of the crown high) with evidence of MIH
6	Unerupted/partially erupted with no evidence of MIH
7	Diffuse opacities (not MIH)
8	Hypoplasia (not MIH)
9	Combined lesion (diffuse opacities/hypoplasia with MIH)
10	Demarcated opacities in incisors only

MIH, molar incisor hypomineralisation; PEB, post-eruptive enamel breakdown.

Severity gradation

Although demarcated hypomineralisation severity assessment was not included in the recognised evaluation criteria, a record was made of the clinical presentation (opacities and PEB) of lesions in addition to indicating the tooth surface area involved. A tooth with opacities only was considered less severely affected than a tooth with PEB or an atypical restoration. The extent of the defect in a tooth was measured by the surface area of the enamel affected as follows: '<1/3 of the tooth surface involved'; 'At least 1/3 but <2/3'; and 'At least 2/3 of a tooth surface involved'[19].

When more than one defect was present on a surface, the combined size of the defects was recorded. When uncertainty existed regarding rating the lesion severity, the less severe rating was recorded. A defect of <2 mm in diameter was considered as sound[20].

Analysis

The collected data were analysed using the SPSS package version 17.0 (SPSS Inc, Chicago, IL, USA). A descriptive analysis of the prevalence and distribution of the clinical recordings was performed. Pearson's Chi-square or Fisher-Exact tests were utilised for nominal or ordinal variables. Odds ratios (OR) at 95% confidence interval (CI) were calculated to determine the difference in the defect severity between genders. Continuous variables were compared using one-way analysis of variance (ANOVA) test. Results at an alpha level <0.05 were considered statistically significant. To evaluate precisely the distribution and the type of affected teeth in those with demarcated hypomineralisation defects, sub-groups of children with all four first molars and 12 index teeth erupted were selected for further analysis.

Results

Distribution of the sample by socio-demographic factors

Of the 1000 potential subjects approached, 823 children agreed to participate, a response rate of 82.3%. The highest rate of response was in the 9-year-old group (91.8%), followed by the 8-year-old group (84.6%), whereas the 7-year-old group had the lowest rate of response (70.5%). Boys participated more than girls in all age groups (57.2% *vs* 42.8%). The vast majority (88.1%) of the children, boys more than girls (423 *vs* 302), had their four first molars erupted, whereas 64.7% of children had their 12 index teeth erupted.

Distribution and prevalence of the demarcated hypomineralisation defect by gender and age groups

The distribution pattern and prevalence of demarcated hypomineralisation defects are summarised in Table 2. The majority of 7- to 9-year-old children were not affected (626; 76.1%), with 197 (23.9%) having at least one permanent index tooth with an enamel developmental defect. Of these 197 subjects, 20 had diffuse opacities (4.1%) or hypoplasia (5.6%) or both (0.5%) without demarcated opacities. No cases with combined demarcated and nondemarcated lesions were observed. The remaining 177 subjects (89.8%) had a demarcated hypomineralisation lesion in at least one of their index teeth; of those, 24 subjects had demarcated hypomineralisation lesions in incisor teeth only. The prevalence of IH was 2.9%. Seventy-nine (9.6%) subjects had MH, and 74 (9.0%) had both MH and IH. According to the definition of MIH[1], 153 cases had

Table 2. Distribution and prevalence of demarcated hypomineralisation lesions in the permanent index teeth by age and gender.

Hypomineralisation-affected teeth	Total *N* (%) *n* = 823	Female *N* (%) *n* = 352	Male *N* (%) *n* = 471	7-year-old *N* (%) *n* = 235	8-year-old *N* (%) *n* = 282	9-year-old *N* (%) *n* = 306
Single incisor	11 (1.3)	5 (1.4)	6 (1.2)	3 (1.2)	4 (1.4)	4 (1.3)
More than one incisor	13 (1.6)	5 (1.4)	8 (1.6)	3 (1.2)	3 (1.0)	7 (2.2)
Single molar	40 (4.9)	19 (5.3)	21 (4.4)	11 (4.6)	12 (4.2)	17 (5.5)
More than one molar	39 (4.7)	14 (3.9)	25 (5.3)	16 (6.8)	10 (3.5)	13 (4.2)
Molars & incisors	74 (9.0)	24 (6.8)	50 (10.6)	15 (6.3)	29 (10.2)	30 (9.8)
Total children with MIH	153 (18.6)	57 (16.1)	96 (20.3)	42 (17.8)	51 (18.0)	60 (19.6)
Total children with MIH & incisor hypomineralisation	177 (21.5)	67 (19.0)	110 (23.3)	46 (19.5)	58 (20.5)	71 (23.2)

MIH, molar incisor hypomineralisation.

demarcated hypomineralisation lesions in at least one first molar tooth or first molars and incisors; hence the overall prevalence of MIH in the study population was 18.6%. Multiple molar involvement rather than a molar tooth involvement was seen more often in boys than girls giving rise to a higher total hypomineralisation defect prevalence in boys than girls (20.3% *vs* 16.1%, respectively). This difference, however, was not statistically significant. No significant difference was revealed in the defect prevalence between the different age single categories.

Distribution of the demarcated hypomineralisation defect by its type and tooth surface

Demarcated opacities were the most frequent lesion type in both molars and incisors. Overall, there were 461 teeth affected by demarcated hypomineralisation lesions including 279 (60.5%) first molars and 182 (39.5%) incisors (figure not shown). Tooth 16 was the molar most affected with 'demarcated creamy-white opacity', and when combined with tooth 26, they were the most common molars identified with 'demarcated yellow-brown opacity'. Opacities were observed more frequently in central incisors than in the lateral incisors in both arches. Post-eruptive breakdown was the second most common finding; of 75 of teeth with PEB, only eight incisors were involved, with permanent molars making up the remainder. The occlusal surface of the first molar was the site most commonly affected by PEB (40 lesions), 25 lesions were located on the buccal and 11 on the lingual/palatal surfaces. In incisors, such enamel defects were mainly localised labially. Significant differences existed between opacities and PEB on both labial and lingual/palatal surfaces ($\chi^2_1 = 72.5$, $P < 0.001$, $\chi^2_1 = 6.4$, $P < 0.01$, respectively).

Only ten teeth (mainly molars) had atypical restorations. Three molars, in two boys, one upper right molar and two lower left molars were considered to have been extracted because of hypomineralisation. Only 13 of the total affected index teeth were identified as 'partially erupted with MIH'.

Mean number of affected teeth with demarcated hypomineralisation lesion in sub-groups of children

In children with all first molars erupted, the mean number of affected molars per child was 1.8 compared with 1.6 in the group of children with all index teeth erupted. The mean number of affected incisors increased concomitantly with the increase in the number of affected molars (Table 3). Statistically significant differences were observed between the mean number of affected incisors compared with the number of affected molars in the group of subjects having all permanent molars erupted as well as in the group of subjects with their all index teeth erupted ($F_{3,141} = 14.8$, $P < 0.001$), ($F_{3,78} = 6.6$, $P < 0.001$), respectively.

MIH-defect severity in relation to the number of affected index teeth and gender

The distribution of opacities and PEB amongst subjects in relation to varying number of affected teeth and gender is presented in Table 4. In children with both MIH and IH defects, those with one or two affected teeth opacities were more frequently reported than PEB, whereas children with three or more affected teeth had significantly more PEB defects ($\chi^2_1 = 12.1$, $P < 0.001$). These children were over three times more likely to have PEB than children having only one or two MIH-affected teeth. When MIH and MH-affected children, however, were included in the calculation, the likelihood of PEB amongst children with three or more affected

Table 3. Distribution pattern of the affected first permanent molars and incisors in sub-groups of children.

Number of affected first permanent molar	Mean number of affected incisors in children with all index teeth erupted (SD)*	Mean number of affected incisors in children with all first molars erupted (SD)*
1 molar	0.7 (1.0)	0.6 (0.9)
2 molars	0.9 (1.2)	0.7 (1.0)
3 molars	1.8 (1.3)	1.4 (1.5)
4 molars	3.0 (1.4)	2.7 (1.7)

*SD, standard deviation.

Table 4. Distribution of post-eruptive enamel breakdown and opacities in relation to the number of the affected permanent index teeth and genders.

	Post-eruptive enamel breakdown *N* (%)	Opacity only *N* (%)	Total *N* (%)	Odds ratio	Confidence interval 95%
Cases with 3 or more MIH + IH-affected teeth	31 (60.8)*	37 (29.4)	68 (38.4)	3.38	1.14–4.56
Cases with 1–2 MIH + IH-affected teeth	20 (39.2)*	89 (70.6)	109 (61.6)		
All cases with MIH + IH	51 (28.8)	126 (71.2)	177		
Cases with 3 or more MIH + MH-affected teeth	18 (36.7)	13 (12.5)	31 (20.3)	4.13	2.32–5.72
Cases with 1–2 affected MIH + MH-affected teeth	31 (63.3)	91 (87.5)	122 (79.7)		
All cases with MIH + MH	49 (32.0)	104 (68.0)	153		
Male	35 (68.6)	75 (59.5)	110 (62.1)	1.86	1.36–2.56
Female	16 (31.4)	51 (40.5)	67 (37.9)		

*Statistically significant difference $P < 0.001$.
IH, incisor hypomineralisation; MH, molar hypomineralisation; MIH, molar incisor hypomineralisation.

teeth was even higher (OR 4.13). Boys were more likely to have PEB than girls (OR 1.86).

Relationship between MIH defect-type and its extension

Analysis of a possible link between MIH-defect severity in terms of clinical presentation and defect extent revealed a statistically significant relationship ($\chi^2_6 = 55.9$, $P < 0.001$) (Table 5). The frequency of demarcated creamy-white opacities decreased markedly with the increase of the surface area involved. The number of more severe MIH defects increased with an increase in lesion size. The PEB and atypical restorations that represent the most severe MIH clinical presentation were associated with larger lesions (OR 1.64; 95% CI 0.07–6.14).

Table 5. Distribution of molar incisor hypomineralisation (MIH) defect by its type and the involved tooth surface area.

	MIH–lesion extension*			
Lesion type	<1/3 *N* (%)	<2/3 *N* (%)	>2/3 *N* (%)	Total *N* (%)
Demarcated creamy-white opacity	28 (50.0)	13 (21.3)	4 (6.7)	45 (25.4)
Demarcated yellow-brown opacity	20 (35.7)	37 (60.7)	19 (31.0)	76 (42.9)
Enamel breakdown	8 (14.3)	10 (16.4)	32 (53.3)	50 (28.2)
Atypical restoration	0 (0)	1 (1.6)	5 (8.3)	6 (3.4)
Total	56 (31.6)	61 (34.5)	60 (33.9)	177

*Statistically significant difference $P < 0.001$.

MH-defect extension in relation to the number of the affected first permanent molar teeth

There was a clear increase in the extent of the defect area with the increasing number of affected molars per person (Fig. 1). In cases with all four molars affected, up to 70% of the tooth surfaces were involved compared with <20% in cases with only one molar affected (χ^2_6=19.8, $P < 0.01$).

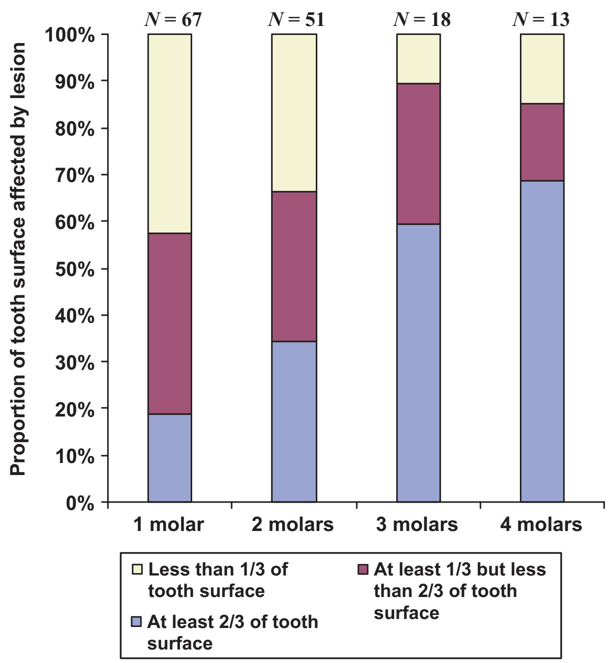

Fig. 1. Proportion of tooth surface affected by hypomineralised lesion according to number (*N*) of first molar tooth with defects.

Discussion

This is the first published study investigating the prevalence of MIH in Iraq, and one of a few relating to the Middle East. Given the high rate of recruitment (82.3%), the examined population may be considered as representative for the region, and the prevalence of 18.6% is therefore a plausible figure for MIH in the community. Given the unique health circumstances of the examined birth cohorts, it was expected that the prevalence figure would be higher than the current estimate. Nevertheless, the present prevalence was comparable to those reported in other studies[4,21–23], which further adds to the ambiguity of MIH pathogenesis. The comparable prevalence estimates encourage speculation that there are shared or common MIH-causative factors. More standardised prevalence studies are, however, needed, in particular in the Middle East where there is a paucity of data, to provide more relevant comparative data. On the other hand, although prevalence estimates were within reported ranges, the present findings contradict previous conclusions regarding the influence of age[9,24] and gender[10,11,24] on MIH prevalence. This supports the theory that MIH is a developmental defect that occurs once the threshold level for the insult required to disturb enamel formation at a critical stage is reached[25].

An age-related increase in the severity level of the hypomineralised defect highlights the dynamic properties of the defect. It is established that MIH enamel possesses inferior structural properties rendering the affected tooth highly susceptible to caries[26,27], however, in terms of caries severity as caries in enamel only or dentinal caries, it is not known how different individual levels of caries-risk affect the behaviour of MIH-affected teeth post-eruption. This reinforces the requirement for further research to elucidate this question and to determine the impact of individual oral health status on MIH.

Although no significant difference was determined in the distribution of the demarcated defect between the maxilla and the mandible, hypomineralised defects were more prevalent and severe in the maxilla than the mandible in accordance with previous studies[4,11].

The finding that the prevalence of PEB increased with the increased number of affected molars indicated that permanent molars are the teeth that best reflect the severity of the defects. Having compared subjects with different numbers of affected teeth, we found that with increasing numbers of molars affected, the greater the mean number of affected incisors as well as the greater the probability of more severe clinical presentation, confirming the findings of others[10,13,28]. This presumably indicates that the insult concurrently affects molars and incisors when the aetiological insult is severe. On the other hand, in children who had no molars affected, 2.9% had demarcated opacities in one or more incisors. It may be that these opacities have been caused by the same aetiological factors as the molar opacities and that this could underestimate the real distribution of the MIH defects. Alternatively, these defects could be caused by a different aetiological factor, such as trauma to the primary incisors. In this study, however, the prevalence of MIH was determined on the basis of having at least one first molar affected. Hence, we believe that the term idiopathic molar hypomineralisation[29] would be a better term when referring to this condition. Long-term prospective epidemiological studies are required to assess the predisposition of incisors to isolated involvement and to establish a reference aetiological factor databank for dental clinicians to achieve a better assessment for the risk factor/s and appropriate intervention strategies[8,16].

Creamy white opacities were considered amongst mild-MIH defect[21]. In agreement with the previous findings, in this study, the creamy white opacities were the most frequent type of defect[10,30]. Nonetheless, when considering the tooth surface area involved, 68.4% of the defects affected more than one third of the tooth surface. Having a large tooth surface area affected may pose an increased risk of PEB in the immediate future that in turn drives higher treatment needs. It is cleared that MIH is an existing dental problem in Mosul City; however, the present survey does not represent the

Iraqi community as a whole. Therefore, to better investigate the condition, a National Oral Health Survey including MIH as a component is needed. The humanitarian situation that resulted from the unique social circumstances the Iraqi community has experienced during the lives of the current subjects could influence the current prevalence of MIH. An attempt to identify whether certain health conditions existed and influenced the prevalence of MIH is required. To be able to identify children at risk for MIH and because those children with MIH have significant treatment needs, it is important to map the prevalence in the Middle East region, using representative populations and valid, reliable assessment criteria.

Conclusions

Molar incisor hypomineralisation occurs in Iraqi children with a prevalence rate of 18.6%. Creamy white demarcated opacities represented over 48% of the total MIH defects. The hypomineralised defect was more prevalent and severe in maxillary teeth than mandibular teeth. The severity of defects increased with age, and the prevalence was greater in boys compared with girls. The number of affected molars was directly related to the mean number of affected incisors, and the more severe the lesion the greater was the involved tooth surface area.

What this paper adds

- This study enriches the available international MIH records by providing a reference database for the prevalence of this condition in a Middle East population.
- It is the first study that gives Iraqi data concerning MIH.

Why this paper is important to paediatric dentists

- Paediatric dentists will encounter children with MIH commonly.
- Paediatric dentists who encounter children with incisors affected by MIH should expect more severely affected molars.

Acknowledgements

The authors thank the children and their parents for permitting their children to participate in the study and also acknowledge the schools principals for their support in carrying out this study. Special thanks to Professor Tahani Alsandook (The Dean of Mosul Dental College) and Dr Ali Alnuaimi for their support and assistance when collecting data. The support of the Cooperative Research Centre for Oral Health, the University of Melbourne and the Education Department of the Neinavha Governorate is acknowledged with gratitude.

References

1 Weerheijm KL. Molar incisor hypomineralization (MIH): clinical presentation, aetiology and management. *Dent Update* 2004; **31**: 9–12.
2 Crombie F, Manton D, Kilpatrick N. Aetiology of molar–incisor hypomineralisation: a critical review. *Int J Paediatr Dent* 2009; **19**: 73–83.
3 Brook AH, Smith JM. The aetiology of developmental defects of enamel: a prevalence and family study in East London, U.K. *Connect Tissue Res* 1998; **39**: 151–156.
4 Leppäniemi A, Lukinmaa PL, Alaluusua S. Nonfluoride hypomineralizations in the permanent first molars and their impact on the treatment need. *Caries Res* 2001; **35**: 36–40.
5 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.
6 Albadri S, Zaitoun H, McDonnell ST, Davidson LE. Extraction of first permanent molar teeth: results from three dental hospitals. *Br Dent J* 2007; **203**: E14. discussion 408–409.
7 Ong DC, Bleakley JE. Compromised first permanent molars: an orthodontic perspective. *Aust Dent J* 2010; **55**: 2–14.
8 Crombie F, Manton DJ, Kilpatrick N. Molar incisor hypomineralization: a survey of members of the Australian and New Zealand Society of Paediatric Dentistry. *Aust Dent J* 2008; **53**: 160–166.
9 Koch G, Hallonsten A-L, Ludvigsson N, Hansson B-O, Holst A, Ullbro C. Epidemiological study of idiopathic enamel hypomineralisation in permanent teeth of Swedish children. *Community Dent Oral Epidemiol* 1987; **15**: 279–285.
10 Wogelius P, Haubek D, Poulsen S. Prevalence and distribution of demarcated opacities in permanent 1st molars and incisors in 6 to 8-years-old Danish children. *Acta Odontol Scand* 2008; **66**: 58–64.
11 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.

12 Cho SY, Ki Y, Chu V. Molar incisor hypomineralization in Hong Kong Chinese children. *Int J Paediatr Dent* 2008; **18**: 348–352.
13 Soviero V, Haubek D, Trindade C, Matta TD, 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.
14 Weerheijm KL, Mejare I. Molar-incisor-Hypomineralisation: a questionnaire inventory of its occurrence in member countries of the European Academy of Paediatric Dentistry (EAPD). *Int J Paediatr Dent* 2003; **13**: 411–416.
15 Kreisel W. *Health Situation in Iraq*. Brussels: WHO/EMRO, Regional Health Statistical Base, 2001.
16 Ghanim A, Morgan M, Mariño R, Manton D, Bailey D. Perception of molar-incisor hypomineralisation (MIH) by Iraqi Dental Academics. *Int J Paediatr Dent* 2011; 10.1111/j.1365-263X.2011.01118.x.
17 Al-Dahan ZA, Al-Rawi BA. Determination of fluoride, zinc and lead ions concentrations in primary teeth and drinking water and dental caries experience. *Al–Rafidain Dent J* 2006; **6**: 23S–29S.
18 Weerheijm K, Duggal M, Mejàre I *et al.* 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.
19 Federation Dentaire International (FDI) Commission on Oral Health, Research and Epidemiology. A review of the development defects of enamel index (DDE Index). *Int Dent J* 1992; **42**: 411–426.
20 Calderara PC, Gerthoux PM, Mocarelli P, Lukinmaa PL, Tramacere PL, Alaluusua S. The prevalence of molar incisor hypomineralisation (MIH) in a group of Italian school children. *Eur J Paed Dent* 2005; **2**: 79–83.
21 da Costa-Silva CM, Jeremias F, de Souza JF, Cordeiro Rde C, Santos-Pinto L, Zuanon AC. Molar incisor hypomineralization: prevalence, severity and clinical consequences in Brazilian children. *Int J Paediatr Dent* 2010; **20**: 426–434.
22 Jälevik B, Klingberg G, Barregård L, Norén JG. The prevalence of demarcated opacities in permanent first molars in a group of Swedish children. *Act Odont Scand* 2001; **59**: 255–260.
23 Zawaideh FI, Al-Jundi SH, Al-Jaljoli MH. Molar incisor hypomineralisation: prevalence in Jordanian children and clinical characteristics. *Eur Arch Paediatr Dent* 2011; **12**: 31–36.
24 Kukleva MP, Petrova SG, Kondeva VK, Nihtyanova TI. Molar incisor hypomineralisation in 7-to-14 year old children in Plovdiv, Bulgaria – an epidemiologic study. *Folia Med (Plovdiv)* 2008; **50**: 71–75.
25 Whatling R, Fearne JM. Molar incisor hypomineralization: a study of aetiological factors in a group of UK children. *Int J Paed Dent* 2008; **18**: 155–162.
26 Mahoney EK, Rohanizadeh R, Ismail FSM, Kilpatrick NM, Swain MV. Mechanical properties and micro-structure of hypomineralised enamel of permanent teeth. *Biomaterials J* 2004; **25**: 5091–5100.
27 Xie ZH, Kilpatrick NM, Swain MV, Munroe PR, Hoffman M. Transmission electron microscope characterization of molar-incisor-hypomineralisation. *Mater Sci: Mater Med J* 2008; **19**: 3187–3192.
28 Lygidakis NA, 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.
29 Mangum JE, Crombie FA, Kilpatrick N, Manton DJ, Hubbard MJ. Surface integrity governs the proteome of hypomineralized enamel. *J Dent Res* 2010; **89**: 1160–1165.
30 Suckling GW, Brown RH, Herbison GP. The prevalence of developmental defects of enamel in 696 nine-year-old New Zealand children participating in a health and development study. *Community Dent Health* 1985; **2**: 303–313.