Original Article

Prevalence and etiology of molar-incisor hypomineralization (MIH) in the city of Istanbul

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Abstract *Background/purpose:* Molar-Incisor-Hypomineralisation (MIH) is the term used to depict a condition in which one or more of the permanent molar teeth and usually no less than one incisor tooth is hypomineralised and the prevalence rates vary from 2.4 to 40.2%. The aim of this study was to assess the prevalence and the risk factors of MIH in children in Istanbul, Turkey.

Materials and methods: A total of 1511 (760 M, 751 F), 8- to 11-year-old children were examined who had their first permanent molar and incisors evaluated using the EAPD criteria for MIH. Hypomineralized molars and incisors were recorded based on developmental defects of enamel index. The potential aetiological factors were retrieved through personal interview and etiological questions were asked to the parents. Statistical analysis was performed with a chi-Square test.

Results: MIH was observed in 215 (14.2%; 102 male, 113 female) children. The sample (1511 children) comprised 71 (9.9%) 8 year-olds with MIH and 144 (18.2%) 11 year-olds with MIH. A significant difference was found between 8 (9.9%) and 11-year-old (18.2%) children with MIH ($p \leq 0.001$). Complications during the mother's pregnancy, birth prematurity, average breast feeding period, diarrhea frequency, digestive system diseases, asthma, frequent high fever, ear infection, renal failure, rubeola, chickenpox and parotitis were found to be significantly associated with MIH ($p < 0.001$).

Conclusion: There are many events that can cause MIH which we cannot control or predict. Therefore, longitudinal studies with large sample size are needed so as to determine how various likely etiological factors described affect the etiological role.

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Introduction

A damage in the enamel organ during amelogenesis may create some changes in the tooth enamel.[1] These changes, called developmental defects of enamel (DDE), are noticeable, as the tooth enamel looses its usual translucent character. The 15th gestational week is the beginning of the amelogenesis stage for the primary teeth, which concludes its growth a year after birth (second primary molar).[1,2] 10%—49% of healthy children in advanced countries have DDE in primary dentition and 9%—63% of them have DDE in permanent teeth, as studies reported.[1]

Enamel defects are linked to many hereditary, acquired, systemic and local aetiological factors.[1,3—7] Genetic variation can affect the caries susceptibility, size, shade, shape, and enamel microhardness, as genetics rigidly determines the formation of enamel during amelogenesis stage.[8] Maturation stage of development is affected and may determine the different kinds of enamel defects. Occurring in the secretory phase of amelogenesis, the reduction of enamel thickness results in the dental defect called enamel hypoplasia. Enamel hypomineralization, a condition causing enamel opacities can be the result of the affected ameloblasts in the late amelogenesis stage of mineralization or maturation.[3,9] Environmental and systemic factors may affect amelogenesis, even though it is controlled by the genes. When the defects occurs in the secretory stage, the outcome is generally enamel hypoplasia, while the defects in the maturation stage results in enamel hypomineralisation.[8]

"Molar incisor hypomineralisation" (MIH) is a dental defect caused by a common pattern of enamel hypomineralization affecting molar and incisor teeth.[3,8,10—15] A number of terminologies (hypomineralised first permanent molars (FPMs), idiopathic enamel hypomineralisation, nonfluoride hypomineralisation, internal enamel hypoplasia, and cheese molars) have been used to define MIH.[6,11,16,17] Mostly linked with affected incisors, if one or more permanent first molars are disturbed in the initial phase of enamel maturation causing a specific abnormality, it is called MIH even though the cause of MIH has been associated with numerous pre-and perinatal conditions.[3,14,18—23] A number of medical factors such as prenatal, perinatal, and postnatal illnesses, low birth weight, antibiotic consumption, and exposure to toxins during breast feeding are possible contributors of MIH; yet the aetiology of MIH is not proven.[8,14,20,21,24,25]

Scientifically, in addition to asymmetric severity, the colour shade of demarcated opacities varies from white to yellow/brownish and there is a clear distinction between the affected and sound enamel.[3,6,8,13,16,19,25—29] MIH may affect enamel in scope from mild to severe. This scope of post-eruptive enamel loss is not regular, though this may be explained by post-eruptive enamel breakdown in first permanent molars, caused by the occlusal load of mastication.[8,16,18,25,29,30] In comparison to anterior teeth, molars are more likely to have post-eruptive breakdown.[6,25,27,29,30]

There is an enormous variation reported in the prevalence rate of MIH in various countries. Most of the literature based in Europe report that the prevalence rates vary from 2.4 to 40.2%[11,14,19,20,23,26—28,30—41] and there are fewer studies on primary teeth.[11,16,20,42,43]

The purpose of this study was to estimate the prevalence and analyze the potential etiological factors of MIH in school-aged children in Istanbul, Turkey.

Materials and methods

The study received approval from the Ethics Committee of Istanbul University, Medical Faculty (No: 2009/2973-113) and was carried out in agreement with the Declaration of Helsinki principles. The written authorization of consent was provided by every parent or caretaker in the name of both themselves and their children.

Subjects examination

For balanced distribution, 10 urban areas were selected as layered in accordance with the population density from the 39th province of Istanbul. Ten of the 524 elementary schools affiliated to Republic of Turkey Ministry of National Education in these regions were numbered and selected randomly with draw. Meaningful sampling size were found at least 1424 samples that statistically calculated with a margin of error of 2.5%, a confidence level of 95%, and a significance level of 0.05, a power level 90%.

The study population comprised of 1511 children (8 and 11 years old) had their FPMs and incisors (index teeth) analyzed within the scope of the European Academy of Paediatric Dentistry (EAPD) criteria for MIH.[32,36] The optimal age of the 8-year-old group of children whose teeth were still in place to observe the initial state of the defect and 11-year-old children in order to understand the extent to which the factors affect the severity of the defect were included in the study. Two calibrated pediatric dentist (MK, EBT) carried out dental examinations. Intra-examiner calibration was performed by repeating examinations of 100 children after an interval of 1 week. Kappa values for intra-examiner consistency were 95.5% for teeth with MIH.

The examination of children for MIH was performed in a school with normal day lighting. The children sat upright on a chair during examination. The dentists got the teeth wet and used a dental mirror and dental probe to get rid of food debris if needed. Every tooth (4 FPM and 8 incisors) was evaluated in accordance with EAPD criteria to diagnose

Figure 1 The hypomineralized permanent incisors and molar.

MIH, there should be at least one affected first permanent molar and incisor (Fig. 1).

Evaluation criteria A) questionnaire

The potential aetiological factors were assessed with a questionnaire comprised of 19 questions and were sent to parents/caretakers in addition to the study consent form. A total of 1511 questionnaires were completed and returned. The questionnaire contained following sections:

(1) Demographic data (child's age, gender, place of birth),
(2) Whether the mother was healthy or taking any medicines during pregnancy, how was the baby delivered, whether there were any complications during or before childbirth and child's birth weight.
(3) Average breastfeeding period
(4) Child's medical history of first four years (diarrhea, digestive system diseases, asthma, pneumonia, respiratory tract infections, tonsillitis, pharyngitis, frequent high fever ($\geq 39\,°C$), otitis media, chronic renal failure, urinary infections and childhood diseases as a rubella, scarlet fever, chickenpox).

Evaluation criteria B) clinical examination

Demarcated opacity
A defect that changes the translucency of the enamel, variable in degree. The defective enamel is of normal thickness with a smooth surface and can be white, yellow or brown opacities. The demarcated opacity is not caused by caries, ingestion of excess fluoride during tooth development or amelogenesis imperfecta etc., and/or

Post-eruptive enamel breakdown
A defect that indicates mild to severe structure enamel loss after eruption of the tooth, e.g. hypomineralisation related attrition. Enamel loss due to erosion was excluded, and/or

Atypical restoration
The size and form of the restoration do not match the present caries distribution in the child's mouth. E.g. amalgam, composite, glass ionomer and crown restorations.

Extracted molar due to MIH
While the presence of major opacities or atypical restorations in other permanent first molars, missing of permanent first molar suggests the possibility of MIH induced attraction. The extraction of incisors in MIH is not a common occurrence.

Unerupted
The first permanent molar or the incisor to be examined are not yet erupted.

Statistical analysis

The data accumulated were evaluated statistically using the MedCalc 11.6.0.0 version and the data were subjected to Chi-square test and multivariable regression analysis after Benferroni corrections. A descriptive analysis of the prevalence and distribution of the clinical recordings were performed. The 95% confidence intervals were calculated for prevalence. A p value of <0.05 was considered statistically significant.

Results

A total of 1511 (760 M, 751 F), 8- to 11-year-old (719 of 8 years old; 792 of 11 years old) children were examined. MIH was observed in 215 (14.2%; 102 male, 113 female) children. The sample (1511 children) comprised 71 (9.9%) 8 year-olds with MIH and 144 (18.2%) 11 year-olds with MIH. No significant difference was found between gender and MIH. However, a significant difference was found between 8 (9.9%) and 11-year-old (18.2%) children with MIH ($p \leq 0.001$). There was a higher prevalence in the older age group (Table 1).

When detailing the distribution of defects according to age and gender, no significant difference was found between gender and defects whereas a significancy was found between age and defect presence ($p \leq 0.001$) (Table 2).

Among the affected teeth, the majority of the defects presented were demarcated white/creamy and yellow/brown type opacities. When the distribution of defect scores evaluated according to tooth numbers in affected group, it was noted that dental restorations were not common and tooth loss was only on permanent molars. As a treatment option, glass ionomer and crown restorations were not applied in affected children. Also there were no incisors' missing because of MIH presence (Table 3).

Table 1 MIH distribution according to age and gender.

		MIH+		MIH−		Total		χ^2	p values
		n	(%)	n	(%)	n	(%)		
Gender	Male	102	(13.4)	658	(86.6)	760	(100)	0.818	0.366
	Female	113	(15.0)	638	(85.0)	751	(100)		
Age	8	71	(9.9)	648	(90.1)	719	(100)	21.309	≤0.001[a]
	11	144	(18.2)	648	(81.8)	792	(100)		

[a] Chi-square test. MIH+: Affected. MIH−: Unaffected.

Results of etiological factors are given in Table 4. Complications during the mother's pregnancy, birth prematurity, average breast feeding period, diarrhea frequency, digestive system diseases, asthma, frequent high fever, ear infection, renal failure, rubeola, chickenpox and parotitis were found to be significantly associated with MIH (p < 0.001). Similarly, birth weight, pneumonia, lower respiratory tract and throat infections, urinary tract infections rubella and scarlet fever were not linked with MIH etiology (p > 0.001) (Table 4), Logistic regression analysis was applied by Forward LR method. Dependent variable MIH (+), (−), independent variables were taken from p < 0.05 among the variables in Table 4 and the analysis was completed in step 6. According to these results birth prematurity, diarrhea frequency, digestive system diseases, renal failure, rubeola and chickenpox were found to be significantly associated with MIH (Table 5).

Discussion

The results of this study demonstrate that MIH prevalence is 14.2% in Istanbul, Turkey for children living in Istanbul, therefore it may be concluded that it is a common condition. The different studies show different results for prevalence rate of MIH. In Table 6, different country and city's scores regarding MIH is summarised. The differences between results may be because of the methodological differences (different birth cohorts in different studies, recruitment of population or convenience samples), irregular methods of clinical examination and differences in the ethnicity and age groups. The percentages and methodological informations for MIH examinations were nearly similar to previously published systematic review studies.[44]

Another important fact is that in the previous studies which were carried out before the EAPD policy document's publication, numerous phenotypes of MIH were included.[45]

The study conducted in India showed that the prevalence of MIH was 0.48%.[10] In another study from Hong Kong, the prevalence of MIH was 2.8%.[26] These rates are lower than other studies. The reason for this might be the different ethnic and age groups that had been examined and the time it had been conducted. On the other hand, this study was on the same page with other studies in gender respect as girls and boys were affected almost equally.[13,19–22,25,26,28,42,46–50] In this present study, there was not an significant difference between males and females with MIH.

In Brazil, there was a 40.2% prevalence between the ages of 7 and 13 in a study conducted by Soviero et al.[27] This study represents the highest prevelance in the studies that have been conducted in the literature and demonstrates how one should take into account distribution and the degree of the defect for the different regions of Brazil.[27,51] The reason for this is because of the use of different indices and criteria, examination variability, methods of recording, and different age groups.

The optimal age of the 8-year-old group of children whose teeth were still in place to observe the initial state of the defect and 11-year-old children in order to understand the extent to which the factors affect the severity of the defect were included in the study. A significant difference was found between 8 (9.9%) and 11-year-old (18.2%) children with MIH in the present study. It maybe relevant delayed eruption. In every child, all maxillary and mandibular permanent incisors and molars can not be completed at 8 years of age. It can be important for MIH diagnosis at older ages.

Table 2 Distribution of defects according to age and gender.

Defect presence?	Gender						Age					
	Male		Female		Total		8		11		Total	
	n	(%)	n	(%)	n	(%)	n	(%)	n	(%)	n	(%)
No defect	595	(78.3)	577	(76.8)	1172	(77.6)	612	(85.1)	560	(70.7)	1172	(77.6)
Only molar teeth defect	27	(3.6)	29	(3.9)	56	(3.7)	12	(1.7)	44	(5.6)	56	(3.7)
Only incisor teeth defect	36	(4.7)	32	(4.3)	68	(4.5)	24	(3.3)	44	(5.6)	68	(4.5)
MIH+	102	(13.4)	113	(15.0)	215	(14.2)	71	(9.9)	144	(18.2)	215	(14.2)
Total	760	(100)	751	(100)	1511	(100)	719	(100)	792	(100)	1511	(100)
χ^2	1.092						47.846					
p values	0.779						≤0.001[a]					

[a] Chi-square test. p≤0.001 MIH+: Affected.

Table 3 Distribution of defect scores according to tooth numbers in affected group.

Tooth number Defect score	16		26		36		46		11		12		21		22		31		32		41		42	
	n	(%)	n	(%)	n	(%)	n	(%)	n	(%)	n	(%)	n	(%)	n	(%)	n	(%)	n	(%)	n	(%)	n	(%)
Enamel defect free	64	(29.8)	66	(30.7)	48	(22.3)	57	(26.5)	89	(41.4)	176	(81.9)	96	(44.7)	182	(84.6)	141	(65.6)	169	(78.6)	153	(71.2)	180	(83.7)
White/creamy opacities	55	(25.6)	46	(21.4)	48	(22.3)	44	(20.5)	75	(34.8)	26	(12.1)	78	(36.3)	22	(10.2)	56	(26)	31	(14.5)	43	(20)	22	(10.2)
Yellow/brown opacities	9	(4)	9	(4.2)	5	(2.3)	4	(1.9)	40	(18.6)	11	(5)	28	(13)	9	(4.2)	15	(7.0)	13	(6.0)	16	(7.4)	11	(5.2)
Slight tooth structure loss	18	(8.4)	18	(8.4)	13	(6.0)	17	(7.9)	6	(2.8)	1	(0.5)	8	(3.7)	1	(0.5)	3	(1.4)	2	(0.9)	2	(0.9)	2	(0.9)
Mild tooth structure loss	10	(4.7)	15	(7.0)	17	(7.9)	10	(4.7)	1	(0.5)	1	(0.5)	1	(0.5)	1	(0.5)								
Severe tooth structure loss	20	(9.3)	24	(11)	33	(15.4)	30	(14.0)	1	(0.5)														
Amalgam restorations	27	(12.6)	21	(9.8)	33	(15.4)	35	(16.3)																
Composite restorations	9	(4.2)	12	(5.6)	13	(6.1)	11	(5)	3	(1.4)			4	(1.8)							1	(0.5)		
Glass ionomer restorations	0	0	0	0	0	0	0	0																
Crown restorations	0	0	0	0	0	0	0	0																
Missing because of MIH	3	(1.4)	4	(1.9)	5	(2.3)	7	(3.2)																
TOTAL	215	(100)	215	(100)	215	(100)	215	(100)	215	(100)	215	(100)	215	(100)	215	(100)	215	(100)	215	(100)	215	(100)	215	(100)

In 2013, Sonmez et al. examined the large group, 4049 children between 7 and 12 years old (2029 girls, 2020 boys) in Ankara, Turkey. 7.7% of the group showed MIH. In children younger than 4 years old, prematurity (7%), gastrointestinal problems (3.9%), pneumonia (6.3%), frequent fever (26.1%), measles (14.7%), and chickenpox (29.3%) were linked to MIH.[8] The same year, another study was conducted in Samsun which is located north of Turkey. Tunc et al. examined 105 children (59 girls, 46 boys) aged between 7 and 11 years with severe MIH, and 105 healthy children in the same age and sex groups. There was not any important difference between MIH and healthy groups but demonstrated that MIH patients' dental development was more advanced than the healthy group.[18] Another study in Turkey, Istanbul was reported by Kuscu et al., in 2009. The study aimed at discovering the prevelance of MIH in children from a region which has the most industrialization and pollution, and from an island with green energy. There were 153 children examined for MIH prevalence (109 from industrialized area, 44 from island with green-energy). There was 9.1% prevalence in the island, while 9.2% was in the industrialized area. The study claimed there is no relation between environmental factors and MIH prevalence.[52] Our study was also conducted in Istanbul, the largest and most cosmopolitan city of Turkey, has a population of 15 millon. Our study has 1511 samples and reflects the biggest population for MIH incidence in Istanbul.

Some of the recent studies focused on the possible determinants such as medical problems in the period of prenatal, perinatal and postnatal; oxygen shortage in ameloblasts, dioxin in breast milk, respiratory infections, medicine use of the child or their treatment with antibiotics and exposure to environmental pollution during the first years of life.[24,25,43,52] The causes of MIH are a mix of these factors and/or the factor is present during the enamel formation.[53] On the other hand, recent genetic studies report that MIH is a multifactorial disturbance. The maturation period of tooth enamel that is commonly affected by MIH corresponds to the last trimester of pregnancy to the third year of a child's life, and it is possible that genetic variation may somehow interact with environmental factors.[3] The hypothesis is that genetic variation in ENAM and AMELX genes may cause localized form of enamel hypomineralization.[3,9] Genetic factors which affect only dental enamel may play a role in enamel defects, or a more general systemic syndrome may cause them.[1,9]

There is no clear association between antibiotic use and MIH. It cannot be confirmed if the disease or the drug is the cause of association, as antibiotics are mostly used with upper respiratory infections.[24] Enamel hypomineralisation may be caused by some factors which can also influence the enamel formation as demonstrated by animal experiments on high fever, dioxin exposure and the use of antibiotics (amoxicillin). Elfrink et al., reported that use of antibiotics, anti-allergic or anti-asthmatic medicines during pregnancy were not associated to Deciduous Molar Hypomineralisation-(Hypomineralised Second Primary Molars) (DMH —HSPM). Also Elfrink et al. reported that the enamel hypomineralisations in HSPM are similar to those observed in MIH in the permanent dentition.[43] Kuscu et al.'s findings are parallel to that view as there was no clinically visible link between MIH and amoxicillin in their animal study. In contrast, Laisi et al.

Table 4 Distribution of etiological factors in affected and unaffected children.

Etiological factors		MIH+ n	MIH+ (%)	MIH− n	MIH− (%)	Total n	Total (%)	χ^2	p values
Complications during pregnancy	Yes	13	(26.0)	37	(74.0)	50	(100)	5.87	0.015[a]
	No	202	(13.8)	1259	(86.2)	1461	(100)		
Birth prematurity	Yes	16	(29.6)	38	(70.4)	54	(100)	10.88	0.001[a]
	No	199	(13.7)	1258	(86.3)	1457	(100)		
Birth Weight	<1500 g	4	(14.8)	23	(85.2)	27	(100)	0.369	0.832
	1500−2500 g	75	(13.5)	480	(86.5)	555	(100)		
	>2500 g	136	(14.6)	793	(85.4)	929	(100)		
Average breast feeding period	None	25	(25.5)	73	(74.5)	98	(100)	11.282	0.004[a]
	<12 months	114	(13.0)	762	(87.0)	876	(100)		
	≥12 months	76	(14.2)	461	(85.8)	537	(100)		
Diarrhea frequency	Yes	37	(33.3)	74	(66.7)	111	(100)	35.827	<0.001[a]
	No	178	(12.7)	1222	(87.3)	1400	(100)		
Digestive system diseases	Yes	30	(46.9)	34	(53.1)	64	(100)	58.361	<0.001[a]
	No	185	(12.8)	1262	(87.2)	1447	(100)		
Asthma	Yes	22	(30.6)	50	(69.4)	72	(100)	16.512	<0.001[a]
	No	193	(13.4)	1246	(86.6)	1439	(100)		
Pneumonia	Yes	28	(18.1)	127	(81.9)	155	(100)	2.082	0.149
	No	187	(13.8)	1169	(86.2)	1356	(100)		
Lower respiratory tract diseases	Yes	52	(12.5)	363	(87.5)	415	(100)	1.353	0.245
	No	163	(14.9)	933	(85.1)	1096	(100)		
Throat infections	Yes	83	(15.2)	463	(84.8)	546	(100)	0.662	0.416
	No	132	(13.7)	833	(86.3)	965	(100)		
Frequent high fever	Yes	33	(20.6)	127	(79.4)	160	(100)	5.998	0.014[a]
	No	182	(13.5)	1169	(86.5)	1351	(100)		
Ear infection	Yes	32	(20.9)	121	(79.1)	153	(100)	6.236	0.013[a]
	No	183	(13.5)	1175	(86.5)	1358	(100)		
Renal failure	Yes	27	(39.7)	41	(60.3)	68	(100)	37.869	≤0.001[a]
	No	188	(13.0)	1255	(87.0)	1443	(100)		
Urinary tract infection	Yes	30	(18.2)	135	(81.8)	165	(100)	2.371	0.124
	No	185	(13.7)	1161	(86.3)	1346	(100)		
Rubeola	Yes	28	(45.9)	33	(54.1)	61	(100)	52.249	≤0.001[a]
	No	187	(12.9)	1263	(87.1)	1450	(100)		
Chickenpox	Yes	97	(31.6)	210	(68.4)	307	(100)	95.217	≤0.001[a]
	No	118	(9.8)	1086	(90.2)	1204	(100)		
Rubella	Yes	6	(22.2)	21	(77.8)	27	(100)	1.439	0.230
	No	209	(14.1)	1275	(85.9)	1484	(100)		
Parotitis	Yes	24	(35.3)	44	(64.7)	68	(100)	25.889	≤0.001[a]
	No	191	(13.2)	1252	(86.8)	1443	(100)		
Scarlet fever	Yes	6	(26.1)	17	(73.9)	23	(100)	2.691	0.101
	No	209	(14.0)	1279	(86.0)	1488	(100)		

[a] *Chi-square test, p<0.05.* MIH+: Affected. MIH−: Unaffected.

indicated that one of the factors leading MIH is the early use of amoxicillin.[54] In the present study, we revealed that it is unclear if the MIH connection was a result of the disease, or the drugs used for treatment of the disease.

In theory, ameloblastic activity during enamel mineralization can be highly affected when health conditions such as asthma or adenoid infections were reported. These diseases influence it directly or indirectly with their symptoms including hypoxia, hypocalcaemia, fever, and/or malnutrition. Hypoventilation in many respiratory diseases such as asthma or adenoid infections may cause respiratory acidosis and abnormal oxygen levels, which affect the enamel matrix pH as demonstrated by the experiments. These conditions lead to enamel hypomineralization since

they obstruct the action of the proteolytic enzymes and the development of the crystal hydroxyapatite. Ameoloblasts can be affected similarly which can explain the risk factor of asthma to MIH.[6] In this study, asthma, high fever, ear infection, renal failure, rubeola, chickenpox and parotitis were found to be extremely associated with MIH.

Allazzam et al. reported that MIH was statistically associated with adenoiditis, fever, frequent tonsillitis, asthma, frequent antibiotics intake, child's positive medical history. No association is found between MIH and histories of birth prematurity, birth complications, low birth weight, or breast feeding duration.[6] On the contrary, Elfrink et al. found that children with normal birth weight appears to have less risk for enamel defects in the primary

Table 5 Variables in the equation.

		B	S.E.	Wald	df	Sig.	Exp(B)
Step 1	Rubeola	1447	,156	85,599	1	,000	4251
	Constant	−1496	,078	365,882	1	,000	,224
Step 2	Digestive system	1642	,277	35,125	1	,000	5168
	Chickenpox	1391	,160	75,865	1	,000	4019
	Constant	−,797	,140	32,498	1	,000	,451
Step 3	Digestive system	1664	,279	35,481	1	,000	5279
	Renal failure	1332	,278	22,942	1	,000	3788
	Chickenpox	1337	,162	68,170	1	,000	3808
	Constant	−,225	,184	1483	1	,223	,799
Step 4	Digestşve system	1591	,282	31,918	1	,000	4908
	Renal failure	1352	,280	23,266	1	,000	3865
	Rubeola	1300	,292	19,862	1	,000	3668
	Chikenpox	1231	,166	55,301	1	,000	3425
	Constant	,297	,221	1806	1	,179	1345
Step 5	Diarrhea	,907	,251	13,013	1	,000	2477
	Digestive system	1400	,293	22,827	1	,000	4057
	Renal failure	1218	,290	17,654	1	,000	3380
	Rubeola	1330	,293	20,621	1	,000	3781
	Chikenpox	1225	,167	53,558	1	,000	3405
	Constant	,534	,235	5147	1	,023	1706
Step 6	Birth prematurity	,802	,335	5753	1	,016	2231
	Diarrhea	,910	,252	13,047	1	,000	2484
	Digestive system	1373	,295	21,621	1	,000	3946
	Renal failure	1197	,289	17,101	1	,000	3309
	Rubeola	1302	,292	19,807	1	,000	3676
	Chickenpox	1229	,168	53,751	1	,000	3416
	Constant	,863	,273	9968	1	,002	2370

dentition than the children with low birth weight, as it is more possible for them to have connection with other likely causes associated with maternal health status for enamel defects.[43] In this present study, asthma, high fever, and some of medical infections were found to be significantly associated with MIH. However, low birth weight, urinary infections were not associated. This was also in agreement with what Allazam et al. found in their studies. They discovered no association between MIH and breast feeding duration and preterm birth. The reason for these difference may be different age groups in different populations.

In a review published in 2009, Crombie et al. chose 53 articles to review from 1123 articles which they examined in the database. These included different possible aetiological factors, some of which were arranged as a group for convenience. They showed that the data were moderate if polychlorinated biphenyl/dioxin exposure affect MIH; nutrition, birth and neonatal factors, and acute or chronic childhood illness/treatment role's evidence was not strong and the role of fluoride or breastfeeding's evidence was also very weak.[55] They concluded that current evidence for the aetiology of MIH is not enough, and strategies are advised to enhance the strength of future studies.

Similar to the present study, Mittal et al., reported MIH prevalence and defect aspects of MIH for school children (1792, 6—9 year old) in Northern India region in 2014. A prevalence of 6.31% was reported. They also found that FPMs were more commonly influenced than permanent incisors and white/creamy opacity without post-eruptive breakdown was the most common lesion in accordance with the present study.[16]

MIH teeth are more likely to develop caries as a result of the enamel hypomineralisation, greater porosity of the tooth structure and, as a consequence, its lower mechanical resistance.[43,53,56] Just like the child having caries, MIH causes hot and cold sensitivity for the affected teeth, even without caries.[30,57] Defective molar teeth may be more in need of dental treatment as they are more prone to plaque accumulation and dental caries. Alteration of the prismatic morphology in the porous enamel causes difficulty in bonding to enamel, which leads to repeated treatment with constant loss of fillings.[6,51] On the other hand, a reduction in mineral density at microscopic level was observed by X-ray microtomography.[24] More dental treatments are performed on the children with affected molars than those with no affected molars and an important amount of teeth ultimately necessitate extraction.[6] Because of the MIH, tooth extraction in the molar teeth was rarely observed in this study.

Although these studies had some limitations such as bias of parents' answers, the present study demonstrates that MIH is a difficulty in existence. Special attention should be given to determine if particular health conditions are present and affected the prevalence of MIH. It is important that children with MIH should be diagnosed and monitored since they need to have special treatment. Health professionals and administrators should be aware that regional difference is a factor of MIH prevalence. There are many events that can cause MIH which we cannot control or predict.

Table 6   Summary of published data from different countries on MIH prevalence.

Reference number	Authors	Year	Country/City	Number of subjects	Age range of subjects	Prevalence
31	Leppaniemi et al.	2001	Finland/Helsinki	488	7—13	19.3%
58	Weerheijm et al.	2001	The Netherlands	497	11	9.7%
28	Jalevik et al.	2001	Sweden/Kallered, Molndal	516	7—8	18.4%
33	Dietrich et al.	2003	Germany/Dresden	2408	10—17	5.6%
34	Kosem et al.	2004	Slovenia	2339	12—18	14%
19	Calderara et al.	2005	Italy/Lissone	227	7—8	13.7%
59	Fteita et al.	2006	Libya/Benghazi	378	7—9	2.9%
12	Jasulaityte et al.	2007	Lithuania/Kaunas	1277	7—9	9.7%
60	Muratbegovic et al.	2007	Bosnia Herzegovina	560	12	12.3%
61	Lygidakis et al.	2008	Greece/Athens	3518	5.5—12	10.2%
26	Cho et al.	2008	China/Hongkong	2635	11—14	2.8%
62	Kemoli	2008	Kenya/Machakos	3591	6—8	13.7%
63	Kukleva et al.	2008	Bulgaria/Plovdiv	2960	7—14	3.58%
52	Kuscu et al.	2009	Turkey/Bozcaada Turkey/Kocaeli	153	7—10	9.1% 9.2%
27	Soviero et al.	2009	Brazil/Rio de Janeiro	249	7—13	40.2%
51	Da Costa-Silva et al.	2010	Brazil/Botelhos	918	6—12	19.8%
64	Biondi et al.	2011	Argentina/Buenos Aires	1098	11	15.9%
65	Zawaideh et al.	2011	Jordan/Amman, Irbid, Al-Karak	3241	8	17.6%
66	Balmer et al.	2012	UK/Great Britain	3233	12	15.9%
37	Condò et al.	2012	Italy/Rome	1500	4—14	7.3%
7	Ahmadi et al.	2012	Iran/Zahadan	433	7—9	12.7%
20	Martinez Gomez et al.	2012	Spain/Barcelona	505	6—14	17.8%
30	Elfrink et al.	2012	The Netherlands/Rotterdam	6161	6	8.7%
2	Ghanim et al.	2013	Iraqi/Mosul	823	7—9	18.6%
21	Souza et al.	2013	Brazil/Araquara	1511	7—12	12.3%
67	Jeremias et al.	2013	Brazil/Araraquara	1157	6—12	12.3%
38	Groselj and Jan	2013	Slovenia	558	6—11.5	21.4%
8	Sonmez et al.	2013	Turkey/Ankara	4049	7—12	7.7%
56	Ghanim et al.	2014	Iran/Shiraz	810	9—11	20.2%
16	Mittal et al.	2014	India/Chandigarh	1792	6—9	6.31%
6	Allazzam et al.	2014	Saudi Arabia/Jeddah	267	8—12	8.6%
39	Garcia-Margarit et al.	2014	Spain/Valencia	840	8	21.8%
68	Jankovic et al.	2014	Bosnia Herzegovina	141	8	12.8%
22	Pitiphat et al.	2014	Thailand/Khon Kaen	860	6—7	20%
15	Bhaskar and Hegde	2014	India/Udaipur	1173	8—13	9.46%
29	Ng et al.	2014	Singapore/Island	1083	7	12.5%
13	Shrestha et al.	2014	Nepal/Kavre	749	7—12	13.7
14	Petrou et al.	2014	Germany/Dusseldorf, Greifswald, Hamburg, Heidelberg	2395	8	10.1%
45	Kuhnisch et al.	2014	Munich/Germany	693	9—13	9.4%
25	Oyedele et al.	2015	Nigeria/Ile Ife	469	8—10	17.7%
42	Temilola et al.	2015	Nigeria/Ile Ife	236	8—10	9.7%
46	Kirthiga et al.	2015	India(Davangere	2000	11—16	8.9%
40	Kevrekidou et al.	2015	Greece/Thessaloniki	2335	8—14	21%
69	De Lima Mde et al.	2015	Brazil/Piaui	594	11—14	18.4%
17	Mittal et al.	2015	India/Uttar Pradesh	978	6—8	5.09%
70	Hussein et al.	2015	Malaysia/Selangor	154	7—12	16.9%
50	Krishnan et al.	2015	India/Salem	5000	9—14	7.3%
71	Hysi et al.	2016	Albania/Tirana	1575	8—10	14%
41	Schmalfuss et al.	2016	Norway/Tromso and Balsfjord	794	16	13.9%
49	Mishra et al.	2016	India/Uttar Pradesh	1369	8—12	13.9%
72	Tourino et al.	2016	Brazil/Lavras	1181	8—9	20.4%
48	Wuollet et al.	2016	Finland/Lammi and Jalasjarvi	287	7—12	11.5%
47	Yannam et a.	2016	India/Chennai	2864	8—12	9.7%
10	Subramaniam et al.	2016	India/Bengaluru	2500	7—9	0.48%
11	Mittal et al.	2016	India/Nagpur	1109	3—12	7.11%

Therefore, longitudinal studies with large sample size are needed so as to determine how various likely etiological factors described affect the etiological role. These studies would be more valuable when the biological mechanisms which cause this enamel defect are determined.

Conflicts of interest

The authors have no conflicts of interest relevant to this article.

Acknowledgements

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