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Molar-Incisor Hypomineralisation – a review of its public health aspects

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Abstract

Since Molar-Incisor Hypomineralisation (MIH) was defined by Weerheijm et al (2001), it has been the focus of considerable research attention. In assessing whether MIH is a dental public health problem, the prevalence, aetiology, treatment needs, and impacts of the condition need to be considered. MIH has been found to be a common dental anomaly with significant short and long-term impacts to the individual and treating dentist that justifies it being considered a dental public health problem. The research to date has made it difficult to interpret the extent of its public health significance due to the lack of standardisation of diagnostic criteria, the different age ranges of examined children, and differences in sampling methods. The modified version of the EAPD criteria (Elfrink et al, 2015) shows promise for the fields of MIH epidemiological and aetiological research. It would be helpful for future service planning and improved clinical outcomes to have a standardised criterion for future national oral health studies and routinely collected data from the Community Oral Health Service. It is clear that there are considerable impacts on children with moderate to severe MIH, their families and treating clinicians; however, there has been very little investigation of either the psychological impacts or the costs (both in treatment and in time off work and school). Dental public health and paediatric dentists should work together on the many aspects of MIH to undertake research on its prevalence, aetiology, and clinical outcomes. This research would help improve awareness of this complex condition among patients, their families, dental professionals and policy-makers.

Introduction

Molar-Incisor Hypomineralisation (MIH) has been the focus of considerable research attention in recent years, mostly from paediatric dentists. Weerheijm et al (2001) suggested the term Molar-Incisor Hypomineralisation (MIH) for a specific pattern of enamel defects defined as "Hypomineralisation of systemic origin of one to four permanent first molars frequently associated with affected incisors". Prior to this naming of MIH as a specific clinical entity, many names had been used in the literature, such as hypomineralised first molars, cheese molars, idiopathic enamel hypomineralisation, and non-fluoride hypomineralisation (Weerheijm et al, 2015).

MIH is a qualitative defect of enamel that can range clinically from localised chalky white defects, to yellow/brown defects, and has a clear demarcation between the affected and normal enamel. Examples of MIH are shown in Figures 1, 2 and 3. Severe cases will feature post-eruptive breakdown or atypical restorations. An association between its colour and the degree of porosity or protein content has been established, with yellow–brown defects having lower hardness and higher protein content than white defects (Jälevik and Norén, 2000; Farah et al, 2010a,b). The opacities are usually limited to the incisal (or cuspal) one-third of the crown, rarely affecting the cervical enamel (Jälevik and Norén, 2000; Farah et al, 2010c). Farah et al (2010c) found that the enamel of MIH affected teeth is not thinner than that of unaffected teeth suggesting that the aetiological insult occurs during the maturation phase of tooth development. Ameloblasts have been shown in animal models to be very sensitive to environmental

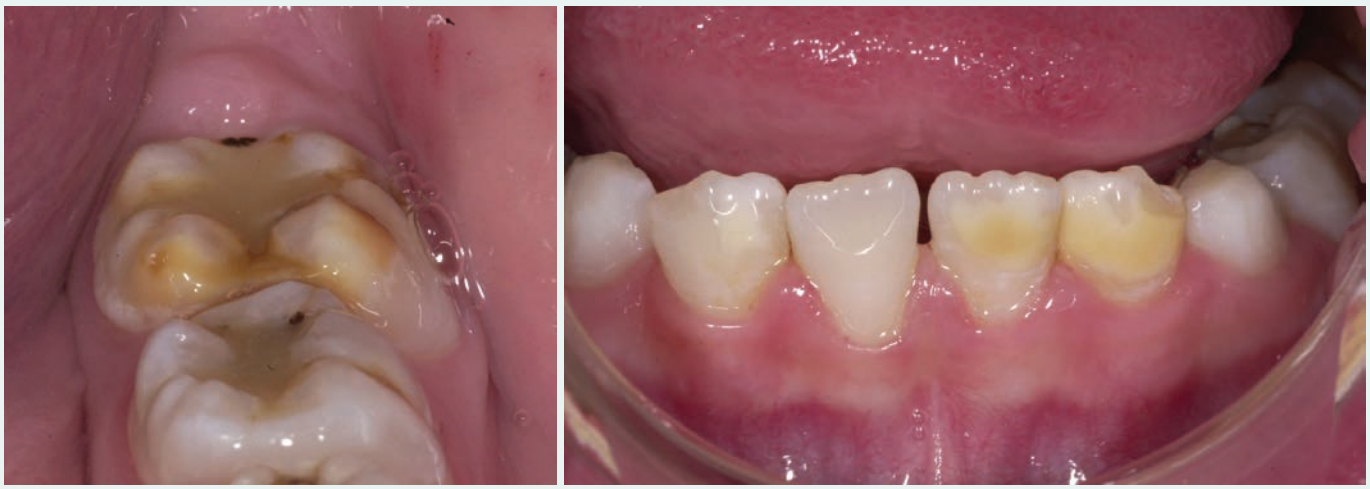

Figure 1. An example of MIH affecting permanent first molars and mandibular permanent incisors (Photos courtesy of Dr Erin Mahoney).

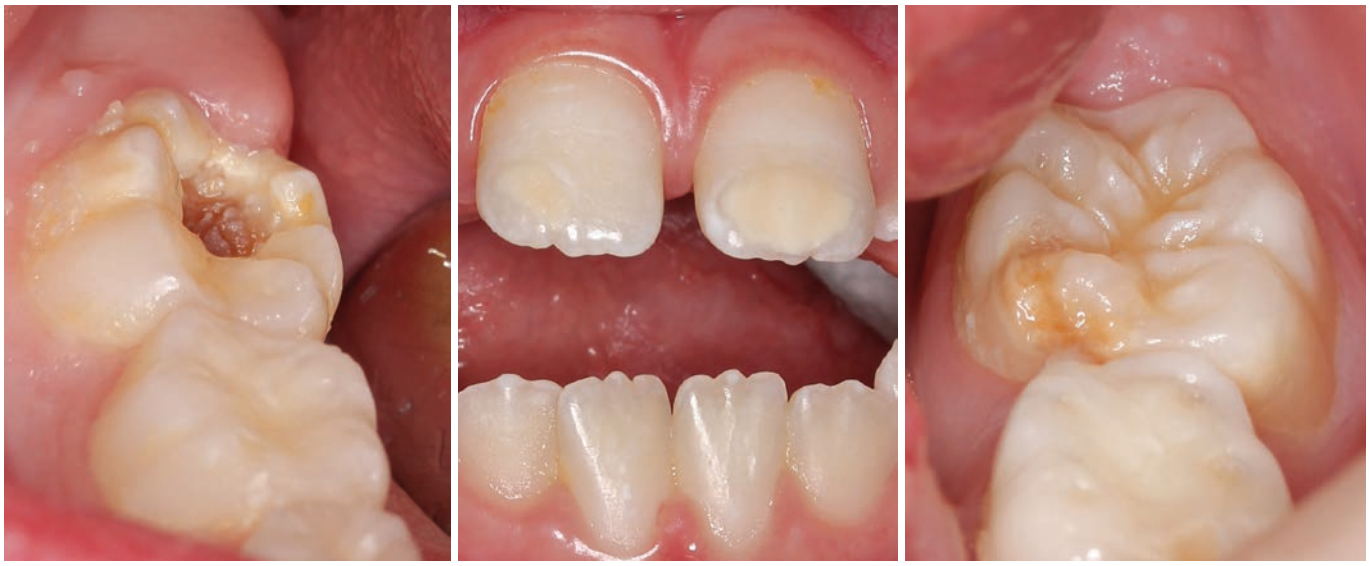

Figure 2. An example of MIH affecting maxillary central incisors and mandibular permanent first molars in a 7 year-old child. The child's lower right mandibular permanent first molar has had post-eruptive breakdown and rapid caries causing pain.

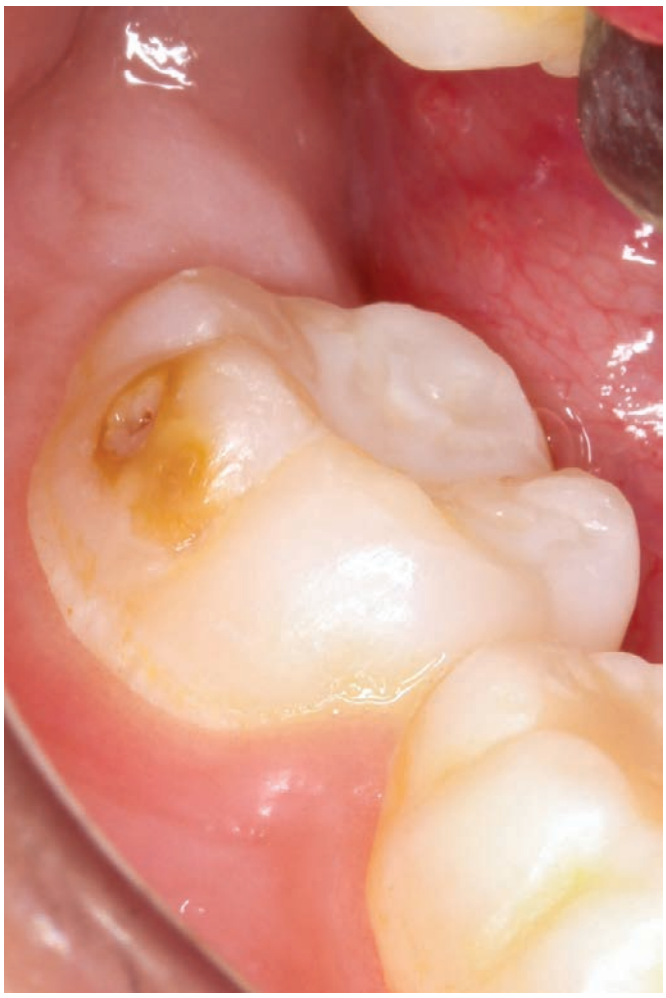

Figure 3. An example of a MIH demarcated brown enamel defect with post-eruptive breakdown.

disturbances in the early stages of enamel maturation (Suga, 1989).

Even though the four permanent first molars in a given individual have been subjected to the same systemic insult, the resulting severity and pattern of enamel defects is often asymmetrical (Weerheijm, 2004). It has been suggested that MIH may be a spectrum of severity ranging from small white enamel defects affecting just the permanent first molars to the more severely affected brown enamel defects on multiple permanent first molars and some permanent incisors (Chawla et al, 2008; Oliver et al, 2014). The number of permanent first molars affected can range from one to four. The probability of the permanent incisors being affected appears to be greater in cases with higher numbers of permanent first molars involved, and when those permanent first molars have more severe brown enamel lesions (Weerheijm, 2003; Ghanim et al, 2011; Oliver et al, 2014).

MIH has been recently linked to similar enamel defects in the second primary molars, with the later referred to as Hypomineralised Second Primary Molars (HSPM). A population-based, prospective cohort study by Elfrink et al (2012) found those with HSPM to be four times more likely to have MIH. Surveys of representative samples of 6-9 year-old children in India and Spain have also shown higher odds of MIH among those with HSPM (Mittal and Sharma, 2015a; Negre-Barber et al, 2016). HSPM can therefore be considered a predictive marker for MIH, and may help with its early diagnosis, although the absence of HPSMs does not preclude a child from having MIH.

The tips of the permanent canines and cusps of second molars have also been observed to have similar demarcated opacities (Weerheijm, 2003; Lygidakis et al, 2010). The prevalence of enamel defects on canines is often not reported, due to the fact that these teeth do not erupt until around age 11 years and most studies concerning MIH have observed younger participants. A study of Norwegian 16-year-olds found that one in four participants with MIH had at least one affected canine,

which was significantly more frequent than in the non-MIH participants (Schmalfuss et al, 2015).

The term "enamel hypoplasia" has sometimes been incorrectly used when there has been post-eruptive breakdown. Enamel hypoplasia is a quantitative defect of the enamel resulting from the disturbance of the ameloblasts in the secretory phase of enamel formation. By contrast, enamel hypomineralisation is a qualitative defect of the enamel resulting from a disturbance during the maturation phase of enamel formation, which causes the enamel to be weaker. This weak enamel can break down easily under masticatory forces, a process is known as post-eruptive breakdown. The two entities can be difficult to distinguish clinically (Ghanim et al, 2015; Weerheijm et al, 2015).

MIH creates challenging problems for both the affected child and the treating dentist. Teeth with MIH defects are often hypersensitive, difficult to anaesthetise, more susceptible to rapidly progressing dental caries, and they can be difficult to restore due to loss of tooth substance and the young age of the patient. Moreover, demarcated opacities on the permanent incisors can result in aesthetic problems. The treatment ranges (depending on the symptoms and severity of the case) from topical fluoride varnish, preventive coatings, adhesive restorations or stainless steel crowns, to extractions; in severely affected molars, extraction may be the treatment of choice to prevent long-term treatment burdens and costs. If extraction is required, it is ideally timed with the calcification of the bifurcation in the lower second permanent molar and in consultation with an orthodontist or paediatric dentist (Jälevik and Klingberg, 2002; Weerheijm, 2004; Mejàre et al, 2005; Oliver et al, 2014).

In considering whether an issue is a public health problem, the criteria of Burt and Eklund (2005) can be useful: first, it needs to be a widespread actual or potential cause of morbidity or mortality; and second, there should be a perception on the part of the public, government, or health authorities that the condition is a public health problem. When considering whether MIH is a dental public health problem, its prevalence and impact of the condition need to be analysed (Marshman et al, 2009). Given the significant long-term burden of MIH to the individual (both functionally and aesthetically), and the potential high cost of treating (both to the families and the State), it is important in planning care delivery to assess the prevalence, treatment needs, and impacts of the condition. Research into the prevalence of MIH is important in establishing evidence of the scale of MIH as a dental public health problem and to help secure resources to help treat affected individuals. Moreover, research into its aetiology is an important part of the process for determining appropriate preventive strategies.

Diagnostic criteria

One of the difficulties in MIH research is that there have been many different examination protocols, diagnostic criteria, case definitions and reporting used in the past. Some studies have excluded carious, extracted or restored permanent first molars, and this has likely meant that the prevalence of MIH has been underestimated (Crombie at al, 2009). The misdiagnosis of post-eruptive breakdown as "hypoplasia" may also have meant that MIH is under-recorded (Weerheijm et al, 2015). Ideally, research in MIH and HSPM needs to be standardised to allow comparisons to be made among studies (Elfrink et al, 2015).

Many different indices have been used for collecting MIH data. The modified Dental Defects of Enamel (mDDE) index has been used in many MIH studies; it classifies the enamel defects as either demarcated opacities, diffuse opacities, or hypoplasia (Clarkson and O'Mullane, 1989). Further adaptations to address limitations of the mDDE index were developed by Jälevik et al (2001a), who subdivided the demarcated lesions into mild, moderate and severe categories. Teeth with PEB or an existing restoration are considered to be in the severe category. However, the mDDE index was still considered to be time-consuming to use and to have limitations for MIH research. Accordingly, the European Academy of Paediatric Dentistry (EAPD) judgement criteria were developed by Weerheijm et al (2003) and then modified by Lygidakis et al (2010) to standardise the diagnosis and characteristics to be used in epidemiological studies. The EAPD criteria recommend that teeth be examined wet after brushing, at around the age of eight years old. The four permanent first molars and eight permanent incisors are examined for: absence or presence of demarcated opacities; post-eruptive enamel breakdown; atypical restorations; extraction of molars due to MIH; and the failure of eruption of a molar or an incisor.

Since the EAPD judgement criteria were developed in 2003, there has been a marked increase in clinical and research interest in MIH. However, that research continues to be criticised for a lack of standardisation which may have contributed to the large variations in estimated prevalence rates among studies (Crombie et al, 2009; Jälevik, 2010; Elfrink et al, 2015; Ghanim et al, 2015). Elfrink et al (2015) have recently recommended another modified version of the EAPD criteria, developed at the 12th EAPD Congress in Poland in 2014. They recommended the inclusion of at least 300 children for prevalence studies and 1000 children for aetiological factor studies. It is also recommended for HSPM studies that the children be 5 years old and, for MIH studies, they be 8 years old. Importantly, they recommended that the examiners be calibrated and use a standardised recording form as described by Ghanim et al (2015). They argued that doing this would improve the grade of evidence and enable meta-analyses to compare studies. The standardised forms published by Ghanim et al (2015) have integrated elements of the EAPD criteria and the mDDE index. They have developed two forms: the short form, designed for simple screening surveys, which only grades the index teeth for MIH and HSPM; and the long form, designed for longitudinal research, and which includes all teeth and a severity grading score for MIH/HSPM teeth. The new standardised EAPD criteria show promise for MIH research, but they require validation and reliability testing in different populations (Ghanim et al, 2015).

Table 1. A summary of the MIH prevalence studies with representative samples

Investigators	Country	Year	Sample size	Age (years)	Score criteria	Prevalence (%)	95% Confidence Interval
Kirthiga et al (2015)	India	2013	2000	11-16	EAPD 2003	8.9	7.1, 10.1
Weerheijm et al (2001)	The Netherlands	1999	497	11	Weerheijm 2001	9.7	7.1, 12.3
Muratbegovic et al (2007)	Bosnia & Herzegovina	2004	560	12	EAPD 2003	12.3	9.6, 15.0
Jasulaityte et al (2008)	The Netherlands	2003	442	9	MIH 2001	14.3	11.0, 17.6
Zawaideh et al (2011)	Jordan	2009	3241	7-9	EAPD 2003	17.6	16.3, 18.9
Garcia-Margarit et al (2014)	Spain	2009	840	8	EAPD 2003	21.8	19.0, 24.6

Another recently developed index, the Molar Hypomineralised Severity Index (MHSI), has been developed based on clinical characteristics and the EAPD judgement criteria (Chawla et al, 2008). It was validated in a practice-based study of paediatric dental specialists in Melbourne, Australia (Oliver et al, 2014), but to date has not been used elsewhere.

Prevalence

Reported prevalence estimates for MIH differ depending on the region and population studied (Weerheijm, 2004). A review of the literature on the prevalence of MIH and HSPM by Elfrink et al (2015) found a wide range in reported prevalence (MIH 2.9 to 44.0%; HSPM 0–21.8%). This may represent real differences among regions, or it could be due to a lack of standardisation of the diagnostic criteria, different ages of children at examination, differences in sampling, and different birth cohorts (Weerheijm and Mejàre, 2003). Very few studies have used representative samples protocol. In the studies that have done so (Table 1), a higher prevalence has been reported from those using younger participants; this may be because, in some of the older children, the more severely affected permanent molars have already been extracted by the time of data collection. Some of the studies reporting higher MIH prevalence have included small enamel defects, while others have excluded defects smaller than 2 mm. Data from studies using non-representative samples (Table 2) should be interpreted with caution. For example, Balmer et al (2005) reported on small samples of children attending two orthodontic clinics (in Sydney and Leeds) over a two-week period and found very high rates of MIH (44% and 40% respectively). These should not be interpreted as prevalence estimates: it is likely that any such clinical sample will feature higher proportions of children with the condition because dealing with the consequences of major enamel defects is one of the indications for orthodontic treatment.

Most of the MIH prevalence studies have been from Europe, with none from North America and very few from the Middle East (Ghanim et al, 2011; Meligy et al, 2014). This raises the question of why dental researchers have not reported on MIH in North America; is it less prevalent in North America and therefore not a concern? Closer to home in New Zealand, Mahoney and Morrison (2011) reported the prevalence of MIH to be 15.7%, surveying 756 children from different socioeconomic areas in the Wellington region. This means that one in seven children may have this condition, and dental professionals in New Zealand will have to deal with this problem on a regular basis.

There has been some speculation that the prevalence of MIH is increasing. However, this could be from improved awareness and recognition of the condition, and an ongoing decrease in the prevalence of dental caries in the permanent dentition making MIH more noticeable (Weerheijm et al, 2015). More research with standardised research criteria and representative samples is needed to know whether there has actually been an increase in MIH prevalence. Such work should be conducted using standard oral epidemiological techniques. Reports from clinical samples are not helpful in this respect.

Aetiology

Since the description of MIH as a clinical entity by Weerheijm et al (2001), there have been many studies of its aetiology. However, despite that work, the cause of MIH is currently unclear. Given the development of the enamel in the permanent first molars and second primary molars, the timing of any causative environmental disruption can be estimated to be between the 18th week of pregnancy and around 3-5 years of age (Weerheijm et al, 2015). However, the most vulnerable period is thought to be from birth until 6-7 months of age (Fagrell et al, 2013).

Most of the studies investigating the aetiology of MIH to date have been retrospective using questionnaires or interviews, and a limitation of this method is recall bias from information provided by the parents for information of their children's early years (Crombie et al, 2009). Another limitation of clinical studies in MIH is the small numbers of participants; hence, the recommendation by Elfrink et al (2015) to have 1000 participants for studies on the aetiological factors of MIH. Larger prospective studies starting with pregnant mothers and following to the eruption of the permanent first molars are required for more understanding of the factors involved (Weerheijm, 2003).

The literature mentions many possible aetiological factors as the cause for MIH, and these can occur in the pre-, peri-, or postnatal periods. Some possible factors reported are respiratory tract problems, dioxins, pregnancy complications, perinatal complications, low birth weight, oxygen starvation, calcium and phosphate metabolic disorders, frequent childhood diseases and high

Table 2. A summary of the MIH prevalence studies with non-representative samples

Investigators	Country	Year Examined	Sample type	Sample size	Age (years)	Score criteria	Prevalence (%)
Cho et al (2008)	China/Hong Kong	2006	Retrospective school dental clinic notes	2635	11-14	EAPD 2003	2.8
Fteita et al (2006)	Libya	2004	School cluster sample	378	7-8	MIH 2001	2.9
Koch et al (1987)	Sweden	1979, 1983	Birth year group cohorts	2252	9-13	Koch et al 1987	4.4-15.4
Dietrich et al (2003)	Germany	2002	School cluster sample	2408	10-17	mDDE	5.6
Preusser et al (2007)	Germany	Unknown	School cross-sectional sample	1002	6-12	Koch et al 1987	5.9
Mittal et al (2014)	India	2009-2010	School cluster sample	1792	6-9	EAPD 2003	6.3
Condo et al (2012)	Italy	1996-2011	Dental clinic random selection	1500	4-15	EAPD 2003	7.3
Sonmez et al (2013)	Turkey	Unknown	School cluster sample	4049	7-12	EAPD 2003	7.7
Allazzam et al (2014)	Saudi Arabia	2011	Dental clinic cross-sectional	267	8-12	EAPD 2003	8.6
Elfrink et al (2012)	The Netherlands	2008-2011	Birth cohort	6161	6	EAPD	8.7
Parikh et al (2012)	India	Unknown	School and dental clinic cluster sample	1366	8-12	EAPD 2003	9.2
Bhaskar and Hedge (2014)	India	2011-2012	Dental clinic cross-sectional	1173	8-13	EAPD 2003	9.5
Jasulaityte et al (2007)	Lithuania	2004	School cluster sample	1277	6-9	EAPD 2003	9.7
Yannam et al (2016)	India	2012	Randomised school cluster sample	2864	8-12	EAPD 2003	9.7
Petrou et al (2014)	Germany	2011-2012	School cluster sample	2395	8	EAPD 2003	9.9
Lygidakis et al (2008)	Greece	2003-2005	Retrospective dental clinic notes	3518	5-12	EAPD 2003	10.2
Mittal and Sharma (2015b)	India	2012-2013	School cluster sample	1240	8-12	EAPD 2003	10.5
Ng et al (2015)	Singapore	2011	School cluster sample	1083	7.7	EAPD 2003	12.5
Calderara et al (2005)	Italy	2002	School cluster sample	227	7-8	MIH 2001	13.7
Kemoli (2008)	Kenya	2006	School cluster sample	3591	6-8	Kemoli 2008	13.7
Schmalfuss et al (2015)	Norway	2010-2011	School cluster sample	794	15-17	EAPD 2003	13.9
Zagdwon et al (2002)	United Kingdom	Unknown	School cluster sample	307	7	mDDE	14.5
Kusku et al (2008)	Turkey	2007	Dental clinic cross-sectional	147	7-9	EAPD 2003	14.9
Mahoney and Morrison (2011)	New Zealand	2008, 2011	School cluster sample	756	7-10	mDDE	15.7
Gurrusquieta et al (2017)	Mexico	Unknown	School cluster sample	1156	6-12	EAPD 2003	15.8
Balmer et al (2012)	Great Britain	2008-2009	School stratified sample	3233	12	mDDE	15.9
Biondi et al (2011)	Argentina	2010	Dental clinic cross-sectional	1098	7-17	DDE	15.9
Alaluusua et al (1996)	Finland	1993-1994	Birth cohort	102	6-7	Alaluusua 1996	17.0
Martinez Gomez et al (2012)	Spain	2008-2009	Dental clinic random sample	505	6-14	EAPD 2003	17.8
Jalevik et al (2001)	Sweden	1998	School cluster sample	516	7-8	mDDE	18.4
Ghanim et al (2011)	Iraq	2009-2010	School cluster sample	823	7-9	EAPD 2003	18.6
Leppaniemi et al (2001)	Finland	1996	Dental clinic cross-sectional	488	7-13	Alaluusua 1996	19.3
Da Costa-Silva et al (2010)	Brazil	2008	School cluster sample	918	6-12	EAPD 2003	19.8
Ghanim et al (2014)	Iran	Unknown	Randomised school cluster sample	810	9-11	EAPD 2003	20.2
Arrow (2008)	Australia	2006-2007	School cluster sample	511	7.1	mDDE	22.0
Negre-Barber et al (2016)	Spain	2013	Birth cohort	414	8-9	EAPD 2003	24.2
Wogelius et al (2008)	Denmark	2005	Dental clinic cross-sectional	745	6-8	EAPD 2003	37.3
Balmer et al (2005)	United Kingdom	Unknown	Orthodontic clinic cross-sectional	25	8-18	mDDE	40.0
Soviero et al (2009)	Brazil	2006	School cross-sectional sample	249	7-13	EAPD 2003	40.2
Balmer et al (2005)	Australia	Unknown	Orthodontic clinic cross-sectional	25	8-16	mDDE	44.0

fever (Jälevik et al, 2001b; Beentjes et al, 2002; Weerheijm, 2004; Lygidakis et al, 2008; Whatling and Fearne, 2008; Alaluusua, 2010). The use of antibiotics (in particular amoxicillin) has also been mentioned, but it is difficult to determine whether the association with MIH is caused by the antibiotic or the illness itself (Jälevik et al, 2001b; Weerheijm, 2004; Whatling and Fearne, 2008; Laisi et al, 2009; Alaluusua, 2010). Exposure to high levels of dioxins or polychlorinated biphenyls (PCBs) has been associated with opacities and/or hypoplasia via the mother's breast milk (Alaluusua et al, 1996). Other studies have not found a link between breastfeeding duration and MIH (Jälevik et al, 2001b; Leppäniemi et al, 2001; Whatling and Fearne, 2008). In fact, studies of developing nations' populations report that breastfeeding has a protective role against enamel defects and suggest that nutrition may be a more important factor (Agarwal et al, 2003; Crombie et al, 2009). Fluoride does not appear to be a factor in the causation of MIH (Whatling and Fearne, 2008; Crombie et al, 2009; Alaluusua, 2010; Balmer et al, 2012).

An alternative aetiology has been proposed by Viera and Kup (2016) who suggested that MIH may be a genetic condition involving the genes which guide enamel formation. They justify their theory by the geographic prevalence variation and lack of clear associations of environmental risk factors and MIH. A study of DNA samples from MIH cases and control cases found that several genes that are involved with enamel formation were also associated with MIH (Jeremias et al, 2013). More prospective cohort and laboratory studies will help us understand this better and answer questions such as why MIH is not common on other teeth (Whatling and Fearne, 2008; Alaluusua, 2010).

Very few studies have reported on ethnic differences in the aetiology of MIH. In Singapore, Ng et al (2015) found that a significantly higher proportion of Malay children compared to Chinese children had MIH. A Wellington study (Mahoney and Morrison, 2009; Mahoney and Morrison, 2011) found no statistically significant ethnic differences in MIH prevalence in New Zealand. It also found no difference by school area deprivation status. This contrasts with a study by Balmer et al (2005), which showed a significant association between higher socioeconomic status and MIH prevalence in Northern England.

Three systematic reviews into the aetiology of MIH (Crombie et al, 2009; Alaluusua, 2010; Silva et al, 2016) have all concluded that none of the research so far has identified clear risk factors, but all agree that the aetiology is likely to be multifactorial. Silva et al (2016) reported in a review that childhood illness is likely to play a role in MIH, but further prospective studies that account for confounding are needed, together with clear definitions for exposures. The authors surmised that it is likely that the cause of MIH is multifactorial, with genetic and epigenetic influences involved.

Psychosocial impacts

The importance of psychosocial research is becoming more appreciated in the dental literature and it is a relatively new area of enquiry, especially in relation to children and young people. Over recent times, there has been an increase in reporting on the relationship between oral health status and children's Oral Health Related Quality of Life (OHRQoL) (Do et al, 2016; Thomson, 2016). However, there has been very little research on the psychosocial impacts of MIH to date. The psychosocial impacts of oral health can be researched by using quantitative and qualitative measures. The quantitative measures are normally by OHRQoL questionnaires, such as the Child Perceptions Questionnaire (CPQ), and Child Oral Health Impact Profile (COHIP). Qualitative methods such as focus groups, interviews, and written diaries could be used to gain more insight into the impacts of MIH (Marshman and Rodd, 2015). A follow-up study in Western Australia found that child OHRQoL was not associated with the presence of enamel defects, but this could have been due to the short-form $CPQ_{11\text{-}14}$ scale used not being discriminative enough for use with enamel defects (Arrow, 2017).

Most of the research on the psychosocial impacts of developmental defects of enamel has been in respect of dental fluorosis. One study by Marshman et al (2009) used in-depth interviews to explore the appearance-related impacts of developmental defects of enamel in the permanent incisors. There were twenty-one participants, of whom five had demarcated opacities and eleven had diffuse opacities. They found that there was a range of impacts for young people, but these were associated with defining aspects of sense of self, rather than age, gender or severity of enamel defect.

The impact of repeated treatment for MIH can also have an effect on the child. Jälevik and Klingberg (2002) found that children with severe MIH had treatment on the first permanent molars nearly ten times as often as a comparison group without MIH. The children with MIH also had more fear and anxiety about dental treatment. The authors recommended early treatment planning, good local anaesthetic and to the consideration of sedation. A follow-up study by Jälevik and Klingberg (2012) to assess the long-term outcomes of children who had severe MIH at age 9 years were studied again at age 18 years. They found that participants with severe MIH had poorer oral health and had undergone treatment on their permanent fist molars more than four times as often as the control group by age 18 years. Interestingly, although the severe MIH group had significantly more behaviour management problems, there was no difference in dental fear scores between the two groups, but this could be due to the small sample size.

Another impact to consider with MIH treatment is the time spent away from school and work for dental appointments by the child and accompanying family members. In some cases, multiple restorative appointments are needed, or a general anaesthetic may be required because of the treatment complexity and/or age of the child. These impacts should be included in any future longitudinal MIH research, along with information on the condition's psychosocial impacts (Marshman and Rodd, 2015).

Conclusions

From a the dental public health perspective, it is important to establish how prevalent the condition is,

its burden to the individual, whether the condition can be prevented, and the financial cost of the condition to the health system. While MIH is less prevalent than dental caries—with approximately two in five year-8 New Zealand school children being affected by dental caries, and one in seven children being affected by MIH—MIH is still a significant oral health issue for our children (Mahoney and Morrison, 2011; Ministry of Health, 2014). However, we still do not know how many of these children are then going on to require treatment and what the cost of this treatment is to the individual and the healthcare system.

It is clear that there are large impacts on the children affected by severe MIH, with hypersensitivity, invasive treatments, aesthetic concerns and dental anxiety. In severe MIH cases, there is often a need for general anaesthetics for extractions or stainless steel crowns, and orthodontic treatment. However, very little research has investigated the psychosocial aspects for children affected by MIH. Again, there is a need for prospective cohort studies that include some OHRQoL questions, together with more in depth qualitative investigations to help answer these questions.

The role of research into the prevalence and aetiology of MIH is of importance to help address the oral health of the child population. From a public health point of view, it would be helpful to include a standardised score criterion in future national oral health studies and routinely collected data by New Zealand Community Oral Health Service to measure changes over time. Identifying trends and possible preventive strategies is an important role of public health dentistry. Having high quality standardised research is important for this. Prospective cohort research following children prenatally until the eruption of the permanent first molars and permanent incisors could help identify the predisposing and aetiological factors of MIH and could help with limiting or even preventing its occurrence. As with dental caries, the adage that prevention is better than treatment applies to MIH.

Children who are identified as at risk of MIH can be monitored closely around the time of permanent first molar eruption to allow early detection and preventive measures. Using HSPMs as a predictive risk indicator to enhance monitoring around 6 years of age could be included into the New Zealand Community Oral Health Service's clinical guidelines.

It is important for dental public health and paediatric dental specialists to work together on the many aspects of MIH to improve research, clinical outcomes and education for patients, their families, dental professionals and policy-makers for this condition.

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