Prevalence of enamel defects and MIH in non-fluoridated and fluoridated communities

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ABSTRACT. Aim This was to study the prevalence of enamel defects and molar incisor hypomineralisation (MIH) in children attending Leeds Dental Institute (UK) and Westmead Dental Hospital, Sydney (Australia).

Methods Prospective dental examinations were carried out on 25 children referred to two orthodontic departments. A questionnaire was completed to obtain background information and about previous fluoride (F) exposure followed by an oral examination. First permanent molars and permanent incisors were examined for presence, type and severity of enamel defects using the modified DDE screening index. χ² tests were used to compare results.

Results Data for 24 children in Sydney and 20 in Leeds presented with at least one enamel defect. Of 300 teeth examined, 155 in Sydney and 82 in Leeds had a defect (p < 0.005). Severity of enamel defects was higher in Sydney. The children presenting with any type of enamel defect in at least one incisor or molar were 21 in Sydney and 10 in Leeds. However, if only demarcated defects were considered, the number in Sydney dropped to 11 and in Leeds remained at 10.

Conclusions There was a higher prevalence of enamel defects in those children living in F Sydney than in non-F Leeds, but the prevalence of MIH was the same supporting the view that F is not associated with the aetiology of MIH.

KEYWORDS: Enamel defects, Fluoride, MIH, Hypocalcification.

Introduction
Molar Incisor Hypomineralisation (MIH) is used to describe a specific pattern of enamel defects, consisting of asymmetric, well demarcated defects affecting the enamel of the first permanent molars and is associated with similar defects in permanent incisors and canines [Weerheijm et al., 2003]. Clinically the defects appear as white, yellow or brown and are susceptible to post eruptive breakdown, reflecting the hypomineralised nature. Most prevalence studies have been carried out in Northern Europe and rates of between 3.6 and 25% were reported [Weerheijm, 2004]. Given the essentially chronological nature of the defects it would seem that a specific agent or agents must be responsible. Several aetiologies have been suggested including environmental change, dioxine exposure, childhood illness, antibiotics, vaccines and low birth weight [Weerheijm, 2004].

The role of fluoride (F) in the aetiology of MIH is unclear. The relationship between F and enamel defects generally has been well documented [Dean et al., 1942; Alaluusua et al., 1999; Evans and Stamm, 1991; Whelton et al., 2004], however, these defects present very differently to MIH. They are generalised, diffuse and only affect the subsurface layer. In spite of the fact that the two conditions appear clinically and histologically distinct, it may be that environmental F can contribute to MIH by lowering the threshold at which MIH occurs or actually raising the resistance of enamel to MIH.

The aims of this study, therefore, were to investigate the prevalence of enamel defects and molar incisor hypomineralisation (MIH) in children living in a F community Sydney (Australia) and a non F community Leeds (UK).

Materials and methods
Sample selection. This was drawn from all new, previously unassessed children referred to the orthodontic departments at Leeds Dental Institute (Leeds, UK) and Westmead Centre for Oral Health (Sydney, Australia). All consenting children attending within a two week period were asked to participate and...
had to be between the ages of 8 and 16 years, had no relevant medical history and were lifetime residents.

**Data collection.** Demographic baseline information was collected of age, sex, postcode and length of time at their present address. Families were also asked to complete a questionnaire to establish previous and current exposure and use regarding F toothpastes, mouth rinses, systemic supplements and professional applications.

**Enamel defects.** The prevalence of enamel defects was determined at a clinical examination when all first permanent molars and all permanent incisors were assessed and recorded using the modified developmental defects of enamel (mDDE) screening index [Clarkson and O’Mullane, 1989]. The examiner was trained and calibrated in its use and a Kappa score for intra-examiner reproducibility of 0.76 was achieved. All examinations were performed visually in the dental chair and with a Darray dental light.

**Statistics.** Data was entered into SPSS statistical package. Chi squared tests were used to analyse differences between the populations.

**Results**

**Sample composition.** The sample composition was 50 patients, 25 (9 males and 16 females) from Leeds (mean age 11.3±2.41) and 25 (7 males and 18 females) from Sydney (mean age 11.9±2.31). No child invited to take part in the study refused and there were no significant differences between the compositions of the two groups.

**Use of fluoride.** Two children in the Sydney group did not know if their toothpaste had F in it; all other children in the study used F toothpaste. Mean age at which patients began using a F toothpaste was slightly older in Sydney (3.74 years) compared with Leeds (2.8 years) but the difference was not statistically significant (p = 0.185). No children in the study had used systemic F supplements; 5 children in Leeds currently used a F mouth rinse compared to only 1 in Sydney. Only one child in Leeds had had a professional F application.

**Clinical examination results.** The results of the clinical examination are summarised in table 1. A total of 600 teeth were examined in 50 patients. There were slightly more children from Sydney with an enamel defect, but this difference was not significant; however this difference became more apparent if the number of teeth with a defect was counted. In this case there was almost double the number of teeth with an enamel defect in the Sydney group than in Leeds. In addition, the average number of teeth with a defect per person was much higher in the Sydney group which was significant statistically (p < 0.05).

Figure 1 shows that Sydney children not only presented with a higher number of defects, but also of greater severity: 11.5% of the defects detected in Sydney covered at least 2/3 of the tooth (mDDE code 3) compared with just 2.4% of the defects found in Leeds. Similarly, the number of defects affecting at least 1/3 to 2/3 of the tooth (mDDE code 2) was greater in Sydney (32.4% of the total as compared with 24.4% in Leeds). Also, the Leeds group had a higher proportion of defects covering less than 1/3 of the tooth (73.2%) compared to the Sydney group (56.1%).

Differences in the groups were still apparent in considering children with defects in both their molars and their incisors: 21 of the Sydney children had a defect in both one molar and one incisor, compared

<table>
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<th>Table 1 - Frequency of severity of enamel defects in two groups of children living in Sydney (Australia) and Leeds (UK).</th>
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<td>Sydney</td>
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<tr>
<td>Total Patients</td>
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<td>Patients with a Defect</td>
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<td>Total teeth</td>
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<td>No. of teeth with defect</td>
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<td>Average defects per person ± standard deviation</td>
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FIG. 1 - Frequency of severity of enamel defects in two groups of children living in Sydney (Australia) and Leeds (UK).
with only 10 children in Leeds. However, if diffuse opacities were omitted and only demarcated opacities considered then the numbers in the two groups became almost identical.

A demarcated defect in both an incisor and a molar occurred in 11 of the Sydney patients and 10 of the Leeds patients (Fig. 2).

**Discussion**

Australia has used water F for over 50 years and at the time of the study nearly two thirds of its population resided in an area with adjusted levels of water F [Spencer et al., 1996]. In keeping with the World Health Organisation [WHO, 1986] guidelines and the Australian Drinking Water Guidelines [NHMRC/ARMCANZ, 1996], the F levels in the public water supply for Sydney were adjusted and kept in the range of 0.9-1.5 mg/l. Current available information shows that between 1992 and 1998, the F levels in Sydney’s drinking water ranged from 0.96-1.16 mg/l compared with F levels of <0.1ppm for Leeds.

As the F water levels are higher in Sydney, it is not surprising that the children had much higher levels of dental defects. This is consistent with many other studies which have shown similar differences [Whelton et al., 2004]. Indeed almost all of the children in the Sydney group had at least one defect. The high levels of diffuse defects in this study may well have reflected a selected sample of children who were from highly motivated families, those who had been referred for orthodontic treatment and may have had higher levels of F supplements. But the questionnaire results indicated very low use of supplements (other than toothpaste), probably reflecting the low level of caries risk in both groups, and no significant differences between groups. These specific populations were chosen because it was accessible through dental hospitals without having the bias that may occur in children referred to a paediatric dental unit (who might, by definition, have higher levels of enamel defects).

Although the study sample was small, there were significant differences in the number of affected teeth in the two cities and these differences remained when considering the number of children with a defect in both an incisor and at least one molar. A critical component of the definition for MIH, however, is that the defects are well demarcated. If this was also applied, then the number of children satisfying the diagnostic criteria for MIH was almost exactly the same in each group.

These findings, that a prevalence of MIH seemed unaffected by environmental F levels, were similar to that of Koch [2003], who reported on a retrospective survey carried out in a F area of Sweden in 1981. Prevalence of MIH in life-long residents was compared with residents who had not been born in the area and in both populations the prevalence was about 6%. Koch concluded that F was not implicated in the aetiology of MIH.

Levels of MIH found in this study were higher than in previous studies in spite of applying the strict criteria that there had to be incisor involvement as well as the demarcated appearance. It is difficult to explain the reasons for this, although the specific population (one with an orthodontic need) has not been studied for MIH previously. It may be that enamel defects in first permanent molars lead to their own orthodontic problems, whilst defects in incisors exacerbate aesthetic issues for the children. Both these factors may contribute to a higher prevalence in this particular population. It is interesting to note the similarity between prevalence levels of MIH in two cities so far apart geographically. Most other MIH prevalence studies have been conducted almost exclusively in Europe.

**Conclusion**

There was a higher prevalence of enamel defects in those children living in fluoridated Sydney than in non-fluoridated Leeds, but the prevalence of MIH was the same supporting the view that fluoride is not associated with the aetiology of MIH.
Acknowledgements

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