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Keywords Children; Dental caries; Developmental defects of enamel; Epidemiology; Molar incisor hypomineralisation.

Abstract

Aim To assess the prevalence of molar incisor hypomineralisation (MIH) in children in Slovenia and explore the relationship between MIH defects and caries in the primary and permanent dentition, and tooth- and surface-specific associations between MIH defects and caries on first permanent molars (FPM).

Materials and methods The study population was comprised of 558 children aged 6.0–11.5 years. The prevalence of developmental defects of enamel (DDE) on FPM and permanent incisors was assessed through clinical examination by a calibrated examiner using the modified DDE Index of the FDI. Also recorded were atypical fillings, post-eruptive enamel breakdowns, and extractions because of MIH. Dental caries was assessed using the WHO criteria.

Results At least one FPM with MIH defects was found in 21.4% of children. Children with MIH had significantly higher (p<0.05) caries experience in their permanent teeth. In primary teeth, higher caries experience was not statistically significant. MIH defects in FPM were associated with higher caries scores at the tooth (p<0.01), and at the surface (p<0.05) level.

Conclusion MIH is common in Slovenia. Children with MIH are more prone to dental caries development on their permanent teeth. On FPM teeth and surfaces affected with MIH defects, even with only mild demarcated opacities with apparently intact enamel, an increased caries experience is present.

Introduction

Molar incisor hypomineralisation (MIH) is defined as hypomineralisation of systemic origin of one to four first permanent molars (FPM), frequently associated with affected permanent incisors (PI) [Weerheijm et al., 2001]. Clinically, these developmental defects of enamel (DDE) vary from white to yellow or brownish demarcated opacities to severely hypomineralised broken enamel.

The prevalence of MIH appears to have increased in recent years and is considered to be a clinical problem [Weerheijm, 2003]. In different parts of the world MIH has a prevalence ranging from 2.4 to 40.2% [Jälevik, 2010]. As noted, the great differences in MIH prevalence observed could be caused, not only by different local environmental circumstances and hereditary factors, but also by different methods of recording and different criteria for MIH [Jälevik, 2010]. In a previous study in Slovenia [Jan and Vrbič, 2000], the percentage of buccal surfaces of two index FPM affected with demarcated opacities from children in the general population was shown to be 5.3%. Histologically, the hypomineralised enamel demonstrates areas of porosity of varying degree [Jälevik, 2001] with less organised enamel structure with voids [Mahoney et al., 2004]. Differences in chemical composition with lower mineral content [Jälevik, 2001; Mahoney et al., 2004] contribute to its smaller mechanical resistance [Mahoney et al., 2004], which facilitates structural loss.

DDE have been speculated to be associated with the risk of dental caries in the affected permanent dentition [Ellwood and O’Mullane, 1994]. In FPM with severe MIH defects and unprotected dentine, fast dental caries development was observed [Weerheijm, 2003] and the treatment need was significantly increased [Leppäniemi et al., 2001; Jälevik and Klingberg, 2002; Kotsanos et al., 2005; Ghanim et al., 2012a]. MIH defects have been reported to be associated with a greater prevalence of dental decay in permanent teeth [Leppäniemi et al., 2001; Jälevik and Klingberg, 2002; Muratbegovic et al., 2007; Cho et al., 2008; Costa-Silva et al., 2010; Mahoney and Morrison, 2011; Ghanim et al., 2012a and 2012b], but not in all studies [Heitmüller et al., 2012].

Dental caries forms through a complex interaction over time between acid-producing bacteria and fermentable carbohydrates, and is also affected by many host factors including teeth and saliva [Selwitz et al., 2007]. Morphological and chemical characteristics of MIH defects contribute to higher local acid solubility and permeability [Jälevik, 2001] and faster penetration of oral bacteria [Fagrell et al., 2008]. Thus, affected teeth could be more susceptible to caries attack. Even FPM with apparently intact enamel opacities can be very sensitive to thermal, chemical and mechanical stimuli, which can impair general oral hygiene habits [Weerheijm et al., 2001]. Furthermore, low salivary flow rates and low saliva pH have been observed to be more common in the MIH affected children [Ghanim et al., 2012b], another factor that could possibly contribute
to higher general caries risk. It would be important to find out if an increased general caries susceptibility is present in children with MIH, and if there is an association between MIH defects and caries at the surface level, as different caries preventive measures would be efficient.

The goal of the present study was to investigate the prevalence of MIH in children in Slovenia. We used the modified DDE Index and additionally recorded other types of MIH defects. Furthermore, we aimed to explore a possible relationship between MIH defects and the caries experience pattern in the primary and permanent dentition. In addition, tooth- and surface-specific associations on FPM between caries and MIH defects were explored.

Materials and methods

The study was carried out in three primary schools from three different regions in Slovenia. Schools with their own dental offices provided the same conditions for dental examination and children were enrolled in similar preventive care programmes, fluoride regimens and oral health care. The levels of fluoride in their water supplies were below 0.1 mg/L. Altogether, 613 children between 6.0 and 11.5 years of age were invited to participate, of those 91% (558 children) responded and agreed. As only children with all FPM erupted were included, the study population therefore comprised a total of 478 children (212 girls, 266 boys, mean age 9.1±1.4 years, range 6.0–11.5 years). All the parents gave written informed consent and the Ethics Committee at the Ministry of Health in Slovenia approved the study.

Children were examined in a dental chair under artificial light by one calibrated dentist (M.G.) using a standard mouth mirror and a dental probe. Prior to examination, the children brushed their teeth under teachers’ supervision. DDE on FPM and PI, and dental caries on all primary and permanent teeth present were recorded. The teeth were not dried prior to examining for DDE, but were air dried prior to examining for dental caries. The type and the extent of the DDE were assessed using the modified DDE Index for use in screening surveys [FDI, 1992] on buccal, occlusal, and lingual surfaces on FPM, and on buccal surfaces on PI. Demarcated opacities, diffuse opacities, hypoplasia, and combinations of these defects were recorded. Additionally recorded were atypical fillings, post-eruptive enamel breakdowns, and extractions because of MIH. Criteria for atypical fillings on FPM were opacities remaining next to the filling and an unusual location and shape of the filling, often including buccal or lingual smooth surface and/or cusp capping. Criteria for atypical fillings on PI were buccal fillings that could not be related to trauma [Weerheijm et al., 2003]. All types of DDE were grouped into any type of DDE. Demarcated opacities, atypical fillings, post-eruptive enamel breakdowns, and extractions because of MIH defects were grouped into MIH defects according to the EAPD criteria [Weerheijm et al., 2003]. Dental caries, missing teeth, and fillings were assessed using the WHO criteria [WHO, 1997]. No radiographs were taken. Questionnaires completed by parents and data from the patients’ dental records provided information on demographics, oral hygiene habits, and fluoride exposure. Altogether 91% parents completed the questionnaires.

Prior to the study, the principal investigator (M.G.) was calibrated in the recording of DDE and dental caries against an experienced epidemiologist (J.J.). The calibration was performed on the basis of the examination of 30 children.

Statistical analysis

All the data were analysed using the SPSS 12.0 statistical software package for Windows (SPSS Inc., Chicago, Ill, USA). Differences in the categorical data were tested using the Chi-square test, differences in the continuous data distributed normally by the Student’s t-test, and differences in the continuous data not distributed normally by the Mann–Whitney U test. A p-value of <0.05 was considered statistically significant; all tests were two–tailed. Only children with all FPM and all PI erupted were included in statistical analysis when analyzing the prevalence of DDE on both FPM and PI.

Results

Inter-examiner reliabilities were calculated to weighted kappa values of k=0.87 and k=0.89 for DDE and dental caries respectively, and for intra-examiner reproducibility they were 0.94 and 0.98, respectively.

Among the 478 children examined with all FPM erupted, 308 had all FPM and all PI erupted. Altogether, 1912 FPM and 2464 PI were examined for DDE.

Table 1 presents the prevalence of different types of DDE on FPM. Buccal FPM surfaces had significantly (p=0.000) more MIH defects and more of any DDE defects than occlusal and lingual surfaces. Upper FPM were affected with any DDE significantly more often (p=0.006) than lower FPM. Among FPM with DDE, 25.7% of MIH defects, 43.1% of diffuse opacities, and none of the enamel hypoplasia extended over more than one third of the surface. Among FPM with MIH defects, 13.7% had DDE extending over more than one third of the surface and 28.4% had DDE on more than one surface. No FPM were extracted because of dental caries or MIH.

Table 2 shows the prevalence of different types of DDE on PI. Upper PI were significantly (p=0.000) more often affected than lower PI, and central PI were significantly (p=0.031) more often affected than lateral PI. Among PI with DDE, 11.4% of MIH defects, 24.8% of diffuse opacities, and only one enamel hypoplasia extended over more than one third of the surface. Only 0.5% of PI had dental caries and/or fillings. There were no statistically significant differences in the prevalence of different types of DDE on FPM and PI between left and right, between genders, and between birth cohorts. Among the 308 children, at least
one FPM with MIH defects was found in 21.4%. These children were diagnosed as children with MIH according to the EAPD criteria [Weerheijm et al., 2003]. Of the examined children, 10.4% (48.6% of children with MIH) had at least one FPM and at least one PI with MIH defects; 12.3% of children (57.5% of children with MIH) had more than one FPM with MIH defects. The mean number of affected FPM per child with MIH defects was 1.8±0.9. Children with MIH had on average 0.8±1.0 PI affected at the same time. Among the 478 children with all FPM erupted, 64.7% had sound permanent teeth and 65.1% had sound FPM. Mean DMFT/number of permanent teeth was 0.05±0.09, mean DMFT in FPM was 0.69±1.11, and mean DMFS in FPM was 0.93±1.77. Dental caries and/or fillings were present on 17.2% of FPM, 6.2% had caries lesions present, and 12.2% had fillings. The most often affected were the occlusal FPM surfaces (12.6%), and then the buccal (5.6%) and lingual (3.3%) surfaces. There were no statistically significant differences between genders. Among the 454 children with at least one primary tooth present, 23.1% had sound primary teeth. The mean dmft/number of primary teeth was 0.32±0.28. No significant differences in the frequency of toothbrushing, use of fluoride toothpaste, and fluoride supplements were present in children with and without MIH. Table 3 presents caries experience among children with and without MIH. There were no statistically significant differences in the age distribution between 105 children with MIH and 373 children without MIH, hence DMFT and dmft scores between the groups could be compared. Children with MIH had significantly

### Table 1

<table>
<thead>
<tr>
<th>Type of DDE</th>
<th>FPM affected (%)</th>
<th>Upper FPM affected (%)</th>
<th>Lower FPM affected (%)</th>
<th>Buccal FPM surfaces affected (%)</th>
<th>Occlusal FPM surfaces affected (%)</th>
<th>Lingual FPM surfaces affected (%)</th>
</tr>
</thead>
<tbody>
<tr>
<td>MIH defects</td>
<td>9.6</td>
<td>10.9</td>
<td>8.3</td>
<td>6.3</td>
<td>3.7</td>
<td>2.8</td>
</tr>
<tr>
<td>Any DDE</td>
<td>13.5</td>
<td>15.7</td>
<td>11.3</td>
<td>9.3</td>
<td>4.3</td>
<td>4.5</td>
</tr>
<tr>
<td>Demarcated opacities</td>
<td>9.3</td>
<td>10.8</td>
<td>7.8</td>
<td>6.0</td>
<td>3.2</td>
<td>2.5</td>
</tr>
<tr>
<td>Diffuse opacities</td>
<td>4.2</td>
<td>5.2</td>
<td>3.1</td>
<td>3.0</td>
<td>0.6</td>
<td>1.7</td>
</tr>
<tr>
<td>Enamel hypoplasia</td>
<td>0.3</td>
<td>0.1</td>
<td>0.4</td>
<td>0.2</td>
<td>0.0</td>
<td>&lt;0.1</td>
</tr>
<tr>
<td>Atypical fillings</td>
<td>0.5</td>
<td>0.4</td>
<td>0.6</td>
<td>0.3</td>
<td>0.4</td>
<td>0.2</td>
</tr>
<tr>
<td>Post-eruptive enamel breakdown</td>
<td>&lt;0.1</td>
<td>0.0</td>
<td>0.1</td>
<td>&lt;0.1</td>
<td>&lt;0.1</td>
<td>&lt;0.1</td>
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</table>

### Table 2

<table>
<thead>
<tr>
<th>Type of DDE</th>
<th>PI affected (%)</th>
<th>Upper PI affected (%)</th>
<th>Lower PI affected (%)</th>
<th>Central PI affected (%)</th>
<th>Lateral PI affected (%)</th>
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<tbody>
<tr>
<td>MIH defects</td>
<td>5.0</td>
<td>7.1</td>
<td>2.9</td>
<td>5.8</td>
<td>3.9</td>
</tr>
<tr>
<td>Any DDE</td>
<td>12.3</td>
<td>17.6</td>
<td>7.1</td>
<td>15.3</td>
<td>9.4</td>
</tr>
<tr>
<td>Demarcated opacities</td>
<td>4.8</td>
<td>7.0</td>
<td>2.7</td>
<td>5.7</td>
<td>4.0</td>
</tr>
<tr>
<td>Diffuse opacities</td>
<td>6.7</td>
<td>10.7</td>
<td>2.7</td>
<td>8.5</td>
<td>4.9</td>
</tr>
<tr>
<td>Enamel hypoplasia</td>
<td>1.7</td>
<td>1.5</td>
<td>1.8</td>
<td>2.7</td>
<td>0.6</td>
</tr>
</tbody>
</table>

### Table 3

<table>
<thead>
<tr>
<th></th>
<th>With MIH defects</th>
<th>Without any DDE</th>
<th>*p-value</th>
</tr>
</thead>
<tbody>
<tr>
<td>DMFS in FPM, mean±SD</td>
<td>1.31±0.04</td>
<td>0.83±0.17</td>
<td>0.012</td>
</tr>
<tr>
<td>DMFT in FPM, mean±SD</td>
<td>0.92±0.126</td>
<td>0.62±0.106</td>
<td>0.016</td>
</tr>
<tr>
<td>DMFT in permanent teeth, mean±SD</td>
<td>0.95±0.128</td>
<td>0.66±0.114</td>
<td>0.023</td>
</tr>
<tr>
<td>dmft in primary teeth, mean±SD</td>
<td>3.53±3.24</td>
<td>2.83±2.76</td>
<td>0.076</td>
</tr>
</tbody>
</table>

* p-value is from the Mann–Whitney U test

### Table 4

<table>
<thead>
<tr>
<th></th>
<th>With MIH defects</th>
<th>Without any DDE</th>
<th>*p-value</th>
</tr>
</thead>
<tbody>
<tr>
<td>Buccal FPM surfaces with caries and/or fillings, % (n)</td>
<td>24.6 (45/183)</td>
<td>16.0 (265/1654)</td>
<td>0.005</td>
</tr>
<tr>
<td>Occlusal FPM surfaces with caries and/or fillings, % (n)</td>
<td>9.9 (12/121)</td>
<td>5.3 (92/1734)</td>
<td>0.041</td>
</tr>
<tr>
<td>Lingual FPM surfaces with caries and/or fillings, % (n)</td>
<td>26.8 (19/71)</td>
<td>12.0 (219/1830)</td>
<td>0.001</td>
</tr>
</tbody>
</table>

* p-value is from the Chi-square test

**TABLE 1** Percent distribution of first permanent molars (FPM) (n=1912) and their surfaces according to the presence of different types of developmental defects of enamel (DDE).

**TABLE 2** Percent distribution of permanent incisors (PI) (n=2464) according to the presence of different types of DDE.

**TABLE 3** Mean±SD number of decayed, extracted, and filled surfaces (DMFS) and teeth (DMFT) among children with (n=105) and without (n=373) MIH in first permanent molars (FPM), all permanent teeth, and all primary teeth present.

**TABLE 4** Association of MIH defects with dental caries experience on first permanent molars (FPM) and their individual surfaces.
higher caries experience in their FPM, at the tooth and at the surface level. They also had significantly higher caries experience in all their permanent teeth present. Among children with MIH, 43.8% had dental caries and/or fillings on FPM, 43.8% had dental caries and/or fillings on their permanent teeth present, and 76.2% had dental caries and/or fillings on their primary teeth present, compared with 32.4%, 33.2%, and 72.1% among children without MIH, respectively. Also, higher caries experience in their primary teeth was present, but the difference was not statistically significant. The bivariate relationship between MIH defects and caries experience of FPM is presented in Table 4. Caries was significantly associated with MIH defects at both the tooth and surface level. Of the MIH affected FPM, 8.7% had caries, and 19.1% had fillings. Of the FPM not affected with MIH defects, 5.6% had caries, and only 11.4% had fillings. Using Chi-square analyses, MIH defects extending over more than one third of the surface were statistically significantly associated with MIH defects at both the tooth and surface level. They also had significantly higher caries experience in their permanent teeth compared to children without MIH defects. These findings are in accordance with the study among English children where those with one or more demarcated opacities had significantly more dental caries and fillings on permanent teeth, and fewer children were caries free than children without demarcated opacities [Ellwood and O’Mullane, 1994]. The present observation also confirmed the reports of previous studies performed in patients with severe MIH cases and low caries activity [Leppäniemi et al., 2001; Jälevik and Klingberg, 2002 and 2012]. In the present study the DMFT values of the FPM were lower as in the previous studies with severe MIH cases, where MIH children were referred to a paediatric specialist. Nevertheless, a significant association with the DMFT values in permanent teeth was observed. Our results of a significant association between MIH defects and general caries experience in permanent teeth are in line with the results of the studies performed in general populations [Muratbegovic et al., 2007; Cho et al., 2008; Costa-Silva et al., 2010; Mahoney and Morrison, 2011; Ghanim et al., 2012b]. However, Heitmüller and coworkers [2012] observed no relationship between the presence of MIH affected FPM and caries experience in permanent teeth of 10-year-olds. Their mean DMFT values were very low. Moreover, specific association between MIH defects...
in FPM and higher caries experience, at the tooth and at the surface level was revealed, confirming the association between hypomineralisation of individual FPM and their dental caries status [Ghanim et al., 2012a]. Children with and without MIH defects were enrolled in similar preventive care programmes, and no significant differences in the frequency of toothbrushing, use of fluoride toothpaste, and fluoride supplements were present. Salivary risk factors were not evaluated, although it was proposed they might be associated with higher caries risk in MIH affected children [Ghanim et al., 2012b]. Evidence suggests that histologically and chemically, the defective enamel sites might be a locus of higher susceptibility to dental caries development than the normal enamel [Jälevik, 2001; Fagrell et al., 2008]. Our results, where a surface-specific association between caries and MIH defects was observed, proved that the defective enamel surface can be more prone to dental caries development. The results suggest that higher local caries susceptibility of the MIH defects could be more important than higher general caries susceptibility in MIH affected children. This observation is consistent with the results of the longitudinal study of Jälevik and Klingberg [2012], where similar increment in the DMFT values showed that children in the MIH group were not more prone to caries than controls. Demarcated opacities with apparently intact enamel were the most prevalent defects in the present study, and even though the MIH defects were not severe, their association with caries experience at the surface level was observed. These findings show that surfaces affected with MIH defects would need early intervention measures as they are at greater risk for dental caries development. Also, higher caries experience in primary teeth of children with MIH defects was present, but the difference was not statistically significant. Similar results were reported by previous studies where caries experience of the primary dentition expressed by dmft was not significantly higher in the MIH group, and differences arose only later when the permanent first molars erupted [Jälevik and Klingberg, 2002; Kotsanos et al., 2005; Mahoney and Morrison, 2011].

In the study, an association between larger extensions of MIH defects on FPM surfaces and presence of carious lesions was observed. The findings are consistent with those of Ghanim and coworkers [2012b] that the extent of MIH defects is associated with dental caries severity. A limitation of the present study is, however, that the severity of the carious lesions was not recorded. In the longitudinal study of Costa-Silva and coworkers [2011] the relationship between MIH severity and the increase in dental caries activity was observed. In the present study, only one case of post-eruptive breakdown was observed. The explanation for our observation could be that children in our study had relatively low caries experience, especially in their permanent teeth. It is also possible that post-eruptive breakdowns were restored with atypical fillings, or could be masked and diagnosed as caries.

Conclusion
The results of the present study showed that every fifth child had at least one FPM with MIH defects. Demarcated opacities with apparently intact enamel were the most prevalent. Children with MIH had significantly higher caries experience in their permanent teeth. Moreover, specific associations between MIH defects in FPM and higher caries experience, at the tooth and at the surface level was revealed.

Acknowledgements
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References