

## THE SYNDROME MIH SYSTEMIC IMPACT IN CHILDREN AND ADOLESCENTS: RELEVANCE AREA

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### ABSTRACT

The aim of our study is to monitor permanent incisors and molars syndrome of hypomineralization in (MIH) in a pediatric population in Iași, using evaluation procedures and criteria established by EAPD. Study group consisted of 334 children (169 girls and 165 boys), divided into two - 43 children with developmental defects of enamel and 291 children without developmental defects of enamel. The prevalence of developmental enamel defects was 10.28% for permanent incisors and 4.26% to permanent molars; MIH syndrome prevalence in the studied casuistry was 14.54%. We notice the presence of multiple lesions 3.3% - opacity, hypoplasia or stains, and in 64.4% of cases the defect extension less than one third of the tooth surface. The risk of being affected more molars grow at a rate of 47% where it affects more incisors.

**Keywords:** syndrome of molar incisor hypomineralization (MIH), enamel defect index, child.

### INTRODUCTION

At the sixth Congress of the European Academy of Paediatric Dentistry (EAPD) theme focused on mineralization of enamel defects (opacity, hypomineralization, dyschromia), which involves permanent molars receive and studies were performed on syndrome of molar incisor hypomineralization (MIH).

In subsequent studies published in the literature was observed that the prevalence of MIH syndrome in the paediatric population is increasing in Europe and require larger studies to assess the risk of damage in different areas, comparative and representative.

#### Aim of the study

The aim of our study is to monitor

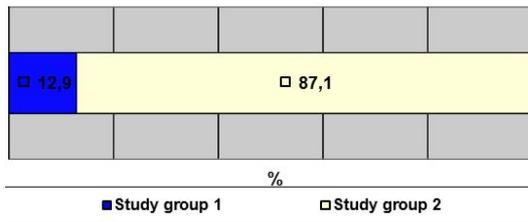
permanent incisors and molars syndrome of hypomineralization in (MIH) in a paediatric population in Iași, using evaluation procedures and criteria established by EAPD.

### MATERIAL AND METHODS

#### Study group

There were investigated 334 children: 169 girls (50.6%) and 165 boys (49.4%). Depending on the presence of enamel defects in the development group was divided into two sublots (Fig. 1), to carry out the terms of a comparison:

- Study lot 1-43 children with developmental defects of enamel (12.9%);
- Study lot 2-291 children without developmental defects of enamel (87.1%).



**Fig. 1. Distribution in study groups**

**Methods**

The children were evaluated in natural light without prior drying or washing of the teeth. Ideally, teeth should be dry at the time of examination, cleaning pigment and bacterial plaque as the real surface of the tooth exposed so you can see hidden defects that can escape from a routine clinical examination. Clinical examination was performed with current dental instruments, with detailed examination of the affected areas. Natural or artificial light was used according to the examination requirements. Tooth surfaces were visually inspected, and

the suspects were explored with the probe to determine the contours and surface defects.

At the sixth Congress of the European Academy of Paediatric Dentistry (EAPD) theme focused on mineralization of enamel defects involving permanent molars receive and studies were made on the prevalence of incisor-molar hypomineralization syndrome (MIH).

In previous studies we used different criteria that have made it difficult to compare prevalence figures. On the other hand it has been suggested by scientists that MIH syndrome prevalence is increasing and that would be beneficial to collect more information on the distribution of MIH in the paediatric population in Europe. In this direction, prevalence studies comparable and representative are needed urgently.

Data was used for the systematic enamel defect index (EDI) presented in Athens (2003) presented in table I.

Demarcated opacity = demarcated defect involving an alteration of enamel translucency. Defective enamel is presented with normal thickness with a smooth surface and can be white, yellow or brown
Posteruptiv disposal of enamel (PDE) = fault surface indicating deficiency after tooth eruption. The loss of substance on the surface of enamel after tooth eruption. The loss is often associated with pre-existing demarcated opacity.
Atypical restoration = the size and shape restorations is not in accordance with the picture of the decay time. In most cases, there must be restoration extended to palatal or buccal smooth. At the edge restorations can frequently be noted opacity. The incisors can be observed atypical restoration cannot be related to an injury.
Molar extracted due to first permanent molar MIH syndrome = absence must be made in relation to other teeth arch. Extraction due to MIH syndrome suspects are opacities or atypical restoration to receive permanent molars others combined with the absence of first permanent molar. Also, the absence of molars and permanent teeth combined with integrity in the rest, but with demarcated opacities on the incisors, is suspected of MIH. It is not likely to be extracted due to MIH incisors.
Uneruptive permanent teeth = first permanent molars or permanent examined did not erupt yet.

*Note: If a large cavity lesions with demarcated opacities in the border areas of non-cavity or caries, these teeth should be appreciated that MIH. Other changes in dental enamel, such as amelogenesis imperfecta, white spot lesions, tetracycline stains, erosion, fluorosis, white ridges and cuspal edges should be excluded from the types of enamel defects outlined above.*

**Table I. Definitions of evaluation criteria used in the MIH diagnosis for prevalence studies**

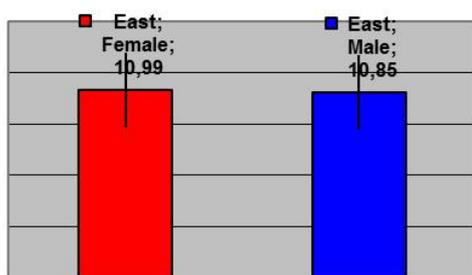
**Statistical methods**

Processing of primary data by centralizing and systematizing that group has resulted in the primary indicators that are presented as absolute values. Based on primary indicators by different statistical methods of comparison, abstraction and generalization derived indicators were obtained using EpiInfo and Excel statistical functions. We have applied tests of significance; the generally accepted threshold is 95%, for Student t-test quantitative values and qualitative test for variables  $\chi^2$  and relative risk (RR). Parameters were used for correlation coefficient of correlation (Pearson).

**RESULTS**

Distribution of children by age was determined from the first examination, without significant differences in gender statistically (t-Student = 0.46, GL = 332,  $p > 0.05$ ) and is as (fig. 2):

- the average age of children at baseline was  $10.92 \pm 2.79$  years, ranging between 6 and 16;
- gender, average age of female cases was



**Fig. 2. The average age of children (CI95%) at study entry**

Koch et al., in 1987, examined the children in Sweden aged 8 and 13 years and have found a variation in prevalence between 3.6 - 15.4%. In them, maxillary incisors were severely affected and 60% of them had all four molars receive regular affected.

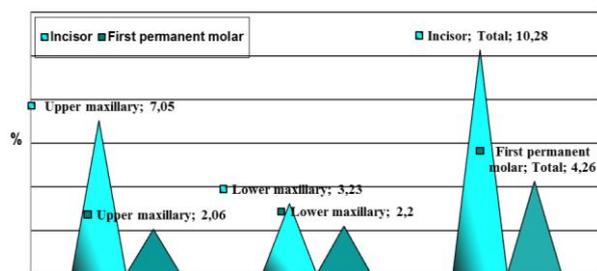
$10.99 \pm 2.78$  years varying from 6 to 15 years (CI95% confidence interval: 8.21 to 13.77 years);

- the average age in males was  $10.85 \pm 2.81$  years varying from 6 to 16 years (CI95% confidence interval: 8.04 to 13.66 years).

The prevalence of enamel defects in development was 10.28% for permanent incisors and 4.26% for first permanent molars (Fig. 3).

This study reveals a prevalence of 14.54% of MIH syndrome.

Currently there are very few data available on prevalence of MIH syndrome, even though studies have been made in this regard. A survey conducted by members of the European Academy of Preventive dentistry shows that paediatric dentists in Europe are aware of MIH's severity, and most consider it a clinical problem. Available data on the prevalence, especially in Northern Europe, fluctuating between 3.6 - 25%, and our study has shown a prevalence of 14.54%, a value high enough, but explained by the low concentration of fluoride in the we studied (prevalence decreases in areas with fluoridated water).



**Fig. 3. The prevalence of enamel defects in development of incisors and molars to receive permanent**

Lappaniemi et al., in 2001, found a prevalence of 19.3% in Finnish children aged between 7 and 13 years, and Alaluusua et al. (1996-1999) showed a percentage of 25% and a prevalence of 17% in Finnish children whose mothers were encouraged to breastfeed

naturally more than 8 months. Compared with these results, Weerheijm et al. (2001) found a small percentage of 9.7% to a group of Germans children for 11 years, which MIH syndrome present two or more affected molar ratio of 79%.

Therefore, EDI is a tool suitable for studies in our population, with a simple classification based on descriptive criteria, which gives flexibility for data, being adapted to research. Applying the  $\chi^2$  test, in our study to obtain the value of  $p < 0.001$  and relative risk (RR), the ratio of the rate of occurrence of enamel defects on permanent incisors to receive permanent molars, 2.41 (1.59 <RR <3.67). Statistical significance indicates that significant defects on the two types of teeth do not exclude the occurrence of a incisor defect is a risk factor of approximately 2.4 times higher for the first

occurrence of the defect and permanent molar.

It is observed that as long as there are opacities on incisors in eruption, there is a risk of developing defect on first permanent molars.

But trying to determine the dependence of the two types of dental using correlation coefficient (r) of enamel defects on permanent incisors and permanent molars in the lower maxillary presented will receive a direct correlation  $r = 0.22$ , a value statistically insignificant.

Permanent incisors and molars correlation between the maxillary permanent receive  $r = -0.50$  was, which highlights the indirect correlation, which points out that the prevalence of damage on permanent molars are more likely to get affected if incisors have a lower prevalence (Table II).

Correlation incisor – first permanent molar	Correlation coefficient	IC95%
Upper maxillary	- 0,50	-0,55 – -0,45
Lower maxillary	+ 0,22	+0,11 – + 0,33
Upper and lower maxillary	- 0,21	-0,12 – -0,33

**Table II. Correlation between different teeth in children affected**

First permanent molar ratio damage to incisor is about 1/4 (Fig. 4). Our results are consistent with his research Weerheijm et al. (2001), which defined the phenomenon as a permanent first molars hypomineralization (one to four teeth) with affected incisors and suggested to name the hypomineralization molar-incisor syndrome. It was observed in our study that the MIH lesions from the first permanent molar are often associated with maxillary incisor lesions and rarely in that jaw (Fig. 4).

These associations indicate a systemic problem in the first year of life of the child, ie the period they are mineralized crowns of permanent molars and incisors receive. Because chewing forces do not act on opacities incisors, their enamel is not so easily disintegrate after eruption. Pearson correlation outlining that

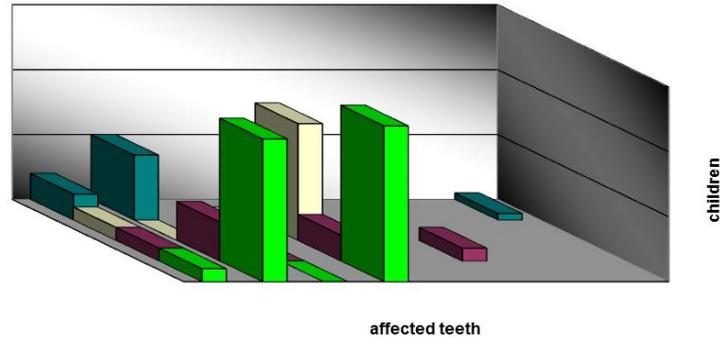
when incisors are affected more, increase the risk of being affected more molars in 47% ( $R^2 = 0.2203$ ,  $r = +.47$ ).

Combining suggests that when there MIH syndrome, we have a specific influence in developing enamel for a limited period of time (Table III).

Table III shows the chronological development of permanent molars and incisors first. Enamel formation is a delicate process that can share teaching in several parts: secretory phase - when partially mineralized enamel is thick and maturation phase.

Later, organic matter and water in the enamel are removed to allow additional influx of minerals.

A disturbing factor appeared in the maturation phase will translate clinically as an opacity of enamel.



**Fig. 4. Distribution of enamel defects of the upper and lower maxillary**

Teeth	Beginning of mineralization		Complete mineralization of coronar part		Eruption	
	Upper maxillary	Lower maxillary	Upper maxillary	Lower maxillary	Upper maxillary	Lower maxillary
I1	3 months	3 months	4½ years	3½ years	7¼ years	6¼ years
I2	11 months	3 months	5½ years	4 years	8 years	7½ years
M1	32 iu weeks	32 iu weeks	4¼ years	3¾ years	6¼ years	6 years

**Table III. Chronological development of permanent molars and incisors**



(a) 11: minimum opacities of the face and incisal edge; 21: demarcated opacities of the face palatal; 32: The minimum opacity distal level; (b) 16: hypoplasia of the distal part of the occlusal ridge transversal fissures; (c) 26: opacity with medial filling; (d) 46: discreet opacity, barely visible in the form of white dots on the ridges.

**CLINICAL CASES**

**Case 1:** P.B., 7 years old, male.

Features captured in the history: asthma at age 1-2 years, numerous ear infections early

in life, scarlet fever at age 2 ½ years.

**Case 2:** A.L., 9 years old, female.

Features highlighted in the history: trauma during the first years of life, subsequent

scarring in the lower lip; repeated scarlet fever during the first four years of life, recurrent purulent angina with high fever for several weeks; lack of vaccines for childhood

diseases, patients suffering from rubella, measles and mumps with fever marked the first four years of life.



**(a) 36: marked opacities and hypoplasia in the distal occlusal relief;**

**(b) 46: occlusal opacities;**

**(c) 32, 42 and 41: buccal opacities.**

**Teeth 11 and 21 - due to developmental defects during early childhood trauma.**

## DISCUSSIONS

The literature listed several possible causes of MIH's such as climate change. Other authors have suggested that respiratory diseases and decreased oxygen level would induce ameloblastic disease. Similarly, a decrease of oxygen associated with weight loss, calcium and phosphate metabolism problems and more childhood diseases can be causes of MIH's. Also, during childhood vaccines have been suggested as possible causes, but no data at this time to certify it.

In research conducted in Hong Kong in 2008 SHIM-Yin Yung KI, MIH syndrome prevalence was 2.8% at a mean age of 12 years. Demarcated opacities was noted that the incisors are more frequently encountered than the molar defects [16]. Hypomineralization of teeth, there were 27

maxillary molars with dental fillings receive, 25 mandibular molars receive maxillary lateral incisor and one [16].

As in our study of children's medical history had a role in MIH syndrome.

MIH syndrome prevalence in Chinese children in Hong Kong was 2.8% lower than our values.

Studies were performed on MIH syndrome spread in European countries, where they reported percentages between 3.6% and 19.3% [4]. According to a study by Weerheijm and employees, affecting maxillary molars receive was about equal to that of mandibular molars. Boys and girls were almost equally affected [7, 9, 14, 16] as noted in our study.

A great impact on the treatment needs of molars with MIH syndrome was reported in areas with low prevalence of dental caries

[16]. In this study, sealing and dental restorations were 38% and 47% to receive permanent molars hypomineralization [9]. In a recent study, Möller and Jälevik and dental occlusion followed developments in 27 children with MIH syndrome, which many receive permanent molars were extracted before eruption of second permanent molar. They noted a good space closure after extraction orthodontic additional treatments.

Remember the 2005's studies of Mejare and collaborators who showed that the average number of permanent molars and incisors affected receive per subject was 3.7 (SD = 0.71) [17]. The study was conducted at the Department of Paediatric Dentistry of "Eastman Dental Institute Stockholm". In 2005 we formulated a questionnaire that was issued by the European Academy of Paediatric Dentistry European countries MIH syndrome. Large variations in reported prevalence of MIH syndrome (06/03/25%) may reflect real differences between regions and countries. In conclusion MIH syndrome aetiology is still unknown. Possible causes have been suggested such as environmental changes over time [13], infections such as respiratory diseases during childhood [6,10] or dioxin in breast milk [2, 3] and no genetic influence cannot be excluded.

Another study of 2009 [S. Lais, A. Ess and colleagues] MIH syndrome associated with use of antibiotics (amoxicillin, penicillin V,

cephalosporins, macrolides and sulfonamides) child receives in the first year of life [11]. One possible explanation is that amoxicillin interfere with the ameloblastic function and anticipate initiating and accelerating amelogenesis could explain the production of enamel hypomineralization MIH syndrome cases.

MIH syndrome causes many problems for children and adolescents. Teeth are very sensitive and require treatment, especially when affected incisors appearing aesthetic problem.

Study results show that amoxicillin used early is among the underlying factors of MIH syndrome. Another causal factor included erythromycin [22].

## CONCLUSIONS

The prevalence of enamel defects in development was 10.28% for permanent incisors and molars receive 4.26% of permanent MIH syndrome prevalence in the studied casuistry was 14.54%.

We notice the presence of multiple lesions 3.3% - opacity, hypoplasia or stains, and in 64.4% of cases an extension of the defect less than one third of the tooth surface, to 35.6% of injury cases ranging from 1/3 and 2/3 of it.

The risk of being affected more molars grow at a rate of 47% where it affects more incisors.

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