

## RISK FACTORS INVOLVEMENT IN ENAMEL DENTAL DISPLASIA

Marinela Păsăreanu, Corina Florea

“Gr.T.Popa” University of Medicine and Pharmacy Iași, Pedodontics Department

**Abstract.** The extended tooth formative period offers extremely large possibilities for various etiological factors to act. Hereditary, congenitally or acquired factors finally justify the existence of an equally large variety of clinical forms, with psychological and functional impact, sometimes a very serious one on patient. Our goal was to correlate developing defects frequencies to the sex and age of the examined children and to some potential risk factors used in the reference literature: delivery, birth weight, infant feeding between 0-4 months of age and the social-economical conditions. The study was made on 600 subjects of 8-11 year age and the index used to systematize the data, was SCOTS index. The obtained results have pointed out that there are not statistically significant differences in enamel defect distribution versus children sex or age, but there is a strongly significant prevalence of enamel defects using the logistic model of regression of some potentially risk factors such as prematurity, birth weight, nutrition, or family income level.

**Key words:** primary and mixed dentition, SCOTS index, enamel dysplasia, risk factors

**Rezumat.** Extinderea perioadei formative a organului dentar pe parcursul unei etape lungi oferă posibilități extrem de largi pentru intervenția unor factori etiologici variați: ereditari, congenitali, dobândiți, ceea ce justifică în final varietatea tot atât de mare a formelor clinice, cu impact psihologic și funcțional uneori foarte grav asupra pacientului. Scopul cercetării noastre a fost corelarea prevalenței defectelor de dezvoltare ale smalțului (după indicele SCOTS) cu sexul, vârsta copiilor examinați și o serie de factori potențiali de risc citați în literatura de specialitate: tipul de naștere, greutatea la naștere, tipul de alimentație între 0-4 luni și condițiile socio-economice. Cercetarea s-a făcut pe un număr de 600 subiecți cu vârsta între 8-11 ani. Copiii au fost examinați în condiții standard, în lumină naturală, fără spălarea în prealabil a dinților. Rezultatele obținute au relevat că nu există diferențe statistice semnificative de distribuție la copiii cu defecte de smalț în funcție de sex și vârstă, însă am găsit o prevalență puternic semnificativă a defectelor de smalț folosind modelul logistic de regresie a câtorva factori potențiali de risc: prematuritatea, greutatea scăzută la naștere, tipul de alimentație (0+4 luni) și condițiile socio-economice.

**Cuvinte cheie:** dentiție temporară și mixtă, indice SCOTS, displazia de smalț, factori de risc

### INTRODUCTION

The extended tooth formative period offers extremely large possibilities for various etiological factors to act. Hereditary, congenitally and acquired factors finally justify the reality of an equally large variety of clinical forms,

with psychological and functional impact on patient, sometimes a very serious one. Even though the structural dental abnormalities (dysplasia) have a quite low frequency, the gravity of these cases requests systematical and extensive studies in order to evaluate

its size, characteristics and correlations with some potential risk factors.

The goal of our study was to establish the epidemiological particularities of the enamel dental dysplasia in children communities and to correlate these results with some potentially risk factors (delivery, birth weight, infant feeding between 0-4 month of age and socio-economical conditions).

#### MATERIAL AND METHOD

600 schoolchildren aged between 8 and 11 years old have been examined in standard conditions without prior washing and drying of the teeth (1).

The SCOTS index (2) have been used for developmental enamel defects and data were analysed with EPI INFO 6.0

and SPSS 6.0 for 95% confidence interval.

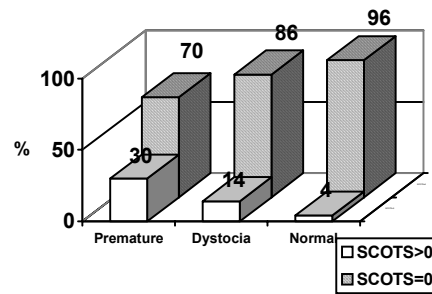
#### RESULTS AND DISCUSSION

The distribution of the enamel defects according to SCOTS index (table 1) indicates a frequency of 6.7% for the entire group of schoolchildren (SCOTS-0). Diffuse opacities (SCOTS-2) have an index of 1.7%, the limited ones 1.2% (SCOTS-1) while hypoplasia has only a value of 2.7% (SCOTS-3). The others codes are less represented except the code 9, because the primary upper incisors are physiologically lost between 6 and 7 years old and this code includes the absent teeth and those that cannot be examined.

**Table 1. Enamel defects distribution according to SCOTS index**

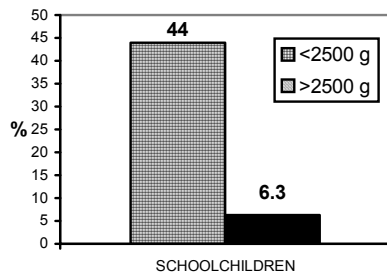
	Enamel defects code-SCOTS										No. of schoolchildren
	0	1	2	3	4	5	6	7	8	9	
%	93.3	1.2	1.7	2.7	0	0	1	0.2	0	0	600

In our study the frequency of the enamel defects was correlated with sex and age and some potentially risk factors such as the type of delivery, birth weight, infant feeding between 0-4 months and socio-economical conditions. The correlation between the dysplasia frequency and the type of delivery shows values of 30% for the premature born infants, 14% for abnormal birth and 4% for infants delivered normally (fig. 1).



**Fig. 1 Enamel defects (SCOTS) according to type of birth at schoolchildren**

The children with low birth weight (under 2.500 g) had a much higher frequency of dysplasia (44%) than those with a normal birth weight (6.3%). We can notice here that the low birth weight seems to be an important risk factor according to the significance level of the difference ( $p < 0.0001$ ) (fig. 2). The primary teeth have a long pre- and postnatal development period and the incisors form before the 10<sup>th</sup> intrauterine week and continue for few months after birth. Any kind of distress during these teeth are developing will result in enamel defects. The low birth weights (under 2.500 g) and the premature delivery are the indicators for fetal malnutrition and they are associated to enamel defects for temporary teeth.



**Fig. 2 Enamel defects frequency according to birth weight**

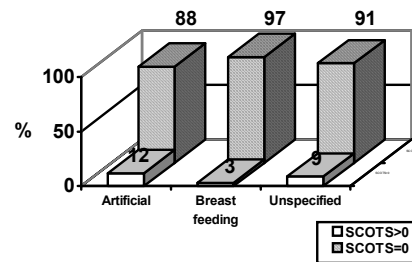
In our study, the prematurely born infants have three times more enamel defects than the children with normal gestational age. The exact pathogenically

mechanism that can explain how the prematurity and low birth weight affect the enamel development and the mineralisation is unknown yet, but elements as respiratory stress, apnea, hypoglycemia, heart disabilities and infections have been associated with a high prevalence of hypoplastic enamel.

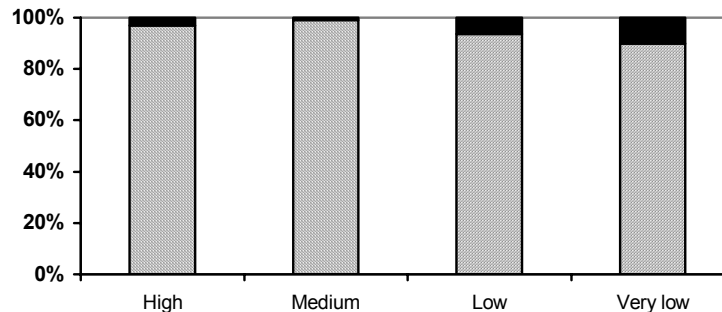
Infant feeding: The nutrition in the first 4 months of life seems to be another risk factor statistically confirmed ( $p < 0.001$ ).

Schoolchildren, who had a formula feeding showed a level of dysplasia of 12% but to this percentage also contributed the children with an unspecified type of feeding (fig. 3).

Also, it can be noticed an increase of the enamel defects frequency for the children with low (8%) and very low socio-economical conditions (13%) ( $p < 0.001$ ). The low conditions included those cases with a poor family income and very low conditions meaning institutionalized children (fig. 4).



**Fig. 3 Enamel defects according to 0-4 month nutrition diet**



**Fig. 4 Prevalence of the enamel defects (SCOTS) according to socio-economical condition**

Although the logistic regression confirmed the statistically significance for all four factors as well as the risk for developing such type of dysplasia, we were not able to use it for prediction, because the structure of the group was not representative for the child population of our region; but this will be the goal of another study.

Many researchers were preoccupied by the high prevalence of the enamel hypoplasia revealed by SCOTS index. Thus, Sarnat showed that there is a high correlation between the child diseases and the hypoplastic enamel development (3) and Sweeney found in 1971 a significantly high prevalence of the hypoplastic enamel for malnourished children (4). Nikiforuk and Fraser in 1981 suggested that enamel hypoplasia could be the result of a hypocalcemia due to the phospho-calcium equilibrium (5). Even though the precise cause and its effect on the ameloblast metabolism as not elucidated, it was obvious that the disturbed local or systemically environment, hereditary factors or any of those combinations are responsible

for development of the enamel or dentin defects (6). Fearne and coworkers in 1990 suggested that the low birth weight and premature infants have statistically significant hypoplasia prevalence than for the normal birth weight children, so the pre- and neonatal conditions could play an important role in development of this defect (7).

Our results are the same with those reported by Li Y., Navia M.J. and Bian J. Their study was made on an infant population from the country, with different socio-economical and nutritional levels in China (6).

A study made by Skinner and Hung in 1989 reports that natural feeding between 0 and 4 months after or even more is strongly associated with a low prevalence of the enamel defects (8).

#### CONCLUSIONS

Sex and distribution of enamel developmental defects show no statistical significance.

A high significant frequency of the enamel defects was correlated with some potentially risk factors: prematurity, low birth weight, type of feeding (0-4

month) and socio-economical conditions when the logistic model of regression was used.

The logistic regression showed predictability for our lot that can be used for risk evaluation in different regions, but in this case it has to be used representative groups for that area.

It is necessary a complete clinical and paraclinical evaluation of this disease and of the risk factors involved for a real clinically and therapeutically prognoses and for optimal preventive and interceptive methods.

#### REFERENCES

1. Păsăreanu M. – *The gravity of the developmental enamel defects in school children*. Journ. Med. Prev., 8(3), 39-43, 2000.
2. Pitts N.B., Stephen K.W. – *Scots public health index for developmental defects of enamel*. Journal of Dental Research, 70, 683, 1991.
3. Sarnat B.G., Stephen K.W. – *Enamel hypoplasia (chronologic enamel aplasia) in relation to systemic disease a chronological morphologic and etiologic classification*. J.Am. Dent.Assoc., 29, 67-75, 1982.
4. Sweeney E.A., Saffir A.J., Leon R. - *Linear hypoplasia of deciduous incisor teeth in malnourished children*. Am.J.Clin.Nutr., 24, 29-31, 1971.
5. Nikiforuk G., Fraser D. – *The etiology of enamel hypoplasia: a unifying concept*. J. Pediatric, 98, 888-93, 1981.
6. Li Y., Navia J.M., Bian J.J. – *Prevalence and distribution of developmental enamel defects in primary dentition of Chinese children 3-5 years*. Community Dentistry and Oral Epidemiology, 23(2), 72-9, 1995.
7. Fearne J.M., Elliot J.C., Wong F.S. – *Deciduous enamel defects in low birth-weight children: correlated X-ray, microtomographic and backscattered electron imaging study of hypoplasia and hypomineralisation*. Anatomy and Embriology, 189(5), 375-81, 1994.
8. Skinner M.F., Hungji T. – *Social and biological correlates of localized enamel hypoplasia of the human deciduous canine tooth*. Am.J. Phys. Antropol., 79, 159-75, 1989.