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To cite this article: Maria Joana Castro, José João Mendes, Susana Vinga & David Casimiro de Andrade (2021) Differential diagnosis of developmental defects of enamel: a review, Annals of Medicine, 53:sup1, S37-S37, DOI: [10.1080/07853890.2021.1897305](https://doi.org/10.1080/07853890.2021.1897305)

To link to this article: <https://doi.org/10.1080/07853890.2021.1897305>



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Published online: 28 Sep 2021.



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# Differential diagnosis of developmental defects of enamel: a review

Maria Joana Castro<sup>a</sup>, José João Mendes<sup>b</sup>, Susana Vinga<sup>c</sup> and David Casimiro de Andrade<sup>a</sup>

<sup>a</sup>Faculdade de Medicina Dentária da Universidade do Porto (FMUDP), Porto, Portugal; <sup>b</sup>Centro de Investigação Interdisciplinar Egas Moniz (CiiEM), Instituto Universitário Egas Moniz (IUEM), Caparica, Portugal; <sup>c</sup>Instituto Superior Técnico da Universidade de Lisboa (IST-UL), Lisboa, Portugal

## ABSTRACT

**Introduction:** Developmental defects of enamel (D.D.E.) are a change resulting from any disturbance during tooth formation. They may manifest as quantitative defect, hypoplasia, presenting deficit of enamel thickness, or qualitative defect, hypomineralization, which expresses itself as diffuse or unmarked opacity of enamel [1,2]. Their approach and treatment imply a correct diagnosis, so it is essential to distinguish between them and know how to discern from other clinical entities.

**Materials and methods:** PubMed search with MeSH "enamel hypoplasia" and "differential diagnosis" with the limits: abstract available, humans and 2000-2019. After selecting 19 articles, other PubMed searches were made with: "molar incisor hypomineralization", "dental fluorosis", "amelogenesis imperfecta", "white spot lesions", "dental erosion" and "attrition", combined with "differential diagnosis" and same limits. 49 articles were selected. The exclusion criteria were non-existent full-article and articles repeated.

**Results:** D.D.E. manifest in 3 forms either isolated or in combination: Demarcated opacity: white or discoloured area of enamel, well demarcated from the sound smooth enamel of normal thickness; Diffuse opacity: abnormality translucence of normal thickness enamel without defined margins that manifests as patchy, irregular or cloudy areas that follow the pattern of the perikymata; Hypoplasia: areas of reduced thickness of enamel that can take the form of pits, grooves until complete absence of it [1]. D.D.E. may affect one tooth, a set or all of them [3]. They may affect temporary or permanent teeth or both [2-6]. According D.D.E.'s manifestation and associated aetiology we can distinguish different clinical entities: hypoplasia [2,7-10]; diffuse opacity [7,9]; demarcated opacity [7]; fluorosis [2,8,11]; hypomineralization of 2nd primary molar [7,9]; molar-incisor hypomineralization [5,7-11]; amelogenesis imperfecta [5,7-10]; and D.D.E. associated with inherited systemic disorders [3,4,6]. The D.D.E. result as pre-eruptive lesion to the tooth [11]. Thus, if the lesion occurs post-eruptively, we should admit other clinical entities such as white spot lesion [2,7-9,11] or tooth wear (erosion or attrition) [12].

**Discussion and conclusions:** D.D.E. exhibit different topographical forms and each call for specific therapeutic approach. Thus, its diagnosis is essential to establish the proper treatment. Furthermore, they may have a significant impact on oral health. They are risk factors of dental caries [1,4,6,8] and erosion [5,9]. They may alter occlusal functions [5], cause of hypersensitivity [5,6,8,9] and compromise aesthetics [5,6,9]. The patient's cooperation may be impaired because of difficulties to anesthetize [8,9] and repeated adhesion failure of restorative materials [5,8].

CONTACT Maria Joana Castro  maria\_joanacastro@hotmail.com

## Acknowledgements

The authors declare that there is no conflict of interest.

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DOI: 10.1080/07853890.2021.1897305

## Effect of an antioxidant on the microtensile bond strength ( $\mu$ TBS) of restored teeth after dental bleaching

Inês Caetano Santos<sup>a</sup>, Rodrigo Abreu<sup>b</sup>, Luís Proença<sup>a</sup>, António H. S. Delgado<sup>a</sup>, Mário Polido<sup>a</sup> and José João Mendes<sup>a</sup>

<sup>a</sup>Centro de Investigação Interdisciplinar Egas Moniz (CiiEM), Egas Moniz Cooperativa de Ensino Superior, Caparica, Portugal; <sup>b</sup>Instituto Universitário Egas Moniz (IUEM), Egas Moniz Cooperativa de Ensino Superior, Caparica, Almada, Portugal

### ABSTRACT

**Introduction:** Dental bleaching is regarded as a safe medical treatment for those who want to achieve a brighter smile. Bleaching is a chemical process in which oxidation occurs, affecting the way teeth absorb or reflect light [1]. Oxidative ability of bleaching agents and the presence of free hydroxyl radicals in the apatite modifies the mineral and protein composition of the enamel increasing its solubility. This renders the enamel surface adverse to the best bonding conditions [2,3]. Some strategies, including antioxidants such as sodium ascorbate, are capable of re-establishing the bond strength to nearest normal values, showing to be an effective method when used immediately after dental bleaching treatment [4,5]. The purpose of this study is to evaluate the effect of an antioxidant on the microtensile bond strength of bleached teeth that were subsequently restored, comparing different waiting times for the procedure.

**Materials and methods:** Thirty human permanent molars were sectioned into halves ( $n=60$ ), and randomly distributed between three groups: control (CG), bleaching (G1) and bleaching + sodium ascorbate (G2). Groups G1 and G2 were bleached 2 h/day for a 7-day period. After bleaching, G2 received a 10% sodium ascorbate gel for 50 min. In each group, samples were divided in equal parts where one part ( $n=5$ ) was immediately restored with an adhesive system and a resin composite (T0) and the other half ( $n=5$ ) was stored, in artificial saliva at 37 °C, and restored after 7 days (T1). After 24 h, samples were sectioned into microspecimens ( $\sim 1\text{mm}^2$ ) and tested at a crosshead head-speed of 0.5 mm/min. Data analysis was performed using a factorial ANOVA, at a significance level of 5%.

**Results:** Groups in which sodium ascorbate was applied presented mean  $\mu$ TBS values (G2T0:  $22.1 \pm 3.3$  MPa; G2T1:  $24.5 \pm 2.9$  MPa) significantly higher than the bleaching only and immediately restored group (G1T0,  $10.9 \pm 3.3$  MPa) ( $p < .001$ ). The group in which teeth were bleached and restored after 7 days (G1T1,  $19.4 \pm 2.6$  MPa) showed significantly higher values than the group immediately restored after bleaching (G1T0) ( $p = .006$ ). No significant differences were found between the groups in which sodium ascorbate was applied (G2) and the group in which teeth were bleached and restored after 7 days (G1T1) ( $p > .05$ ).

**Discussion and conclusions:** Sodium ascorbate is capable of capturing free oxygen radicals retained on the enamel. These promote decomposition of hydrogen peroxide, which in turn prevents the complete polymerisation of the adhesive system thus affecting bond strength [2,5]. The application of sodium ascorbate is an effective alternative to improve bond strength when compared to a waiting period before the restorative procedure.

**CONTACT** Inês Caetano Santos  [inescaetanasantos@gmail.com](mailto:inescaetanasantos@gmail.com)

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DOI: 10.1080/07853890.2021.1897310