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Diagnostic guide enabling distinction between taphonomic stains and enamel hypomineralisation in an archaeological context

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ABSTRACT

Objective: Molar Incisor Hypomineralisation (MIH) is a structural anomaly that affects the quality of tooth enamel and has important consequences for oral health. The developmentally hypomineralised enamel has normal thickness and can range in colour from white to yellow or brown with or without surface breakdown. The possibility of finding MIH in 'ancient populations' could downplay several current aetiological hypotheses (e.g., dioxin derivatives, bisphenols, antibiotics) without excluding the possible multifactorial aspect of the anomaly. In an archaeological context, chemical elements contained in the burial ground can stain teeth yellow or brown and therefore might create a taphonomic bias. The purpose of the present study is to test a proposed diagnostic guide enabling determination of the pathological or taphonomic cause of enamel discolouration and defects that resemble MIH present on 'ancient teeth'.
Design: Two sample groups including MIH discoloration (n = 12 teeth) from living patients, taphonomic discoloration (n = 9 teeth) and unknown discoloration (n = 2 teeth) from medieval specimens were tested. Three non-destructive methods—Raman spectroscopy, X-ray micro-computed tomography and X-ray fluorescence were utilised.
Results: Hypomineralised enamel has decreased mineral density ($p < 0.0001$) and increased phosphate/β-carbonate ratio ($p < 0.01$) compared to normal enamel whereas relative concentrations of manganese, copper, iron and lead are similar. In taphonomic discoloration, relative concentrations of these elements are significantly different ($p < 0.05$) to normal enamel whereas mineral density and Raman spectra profile are comparable.
Conclusions: Enamel hypomineralisation can be distinguished from taphonomic staining in archaeological teeth.

1. Introduction

Developmental defects of enamel (DDE) have been described for many decades (Bhussry, 1958; Kostlan & Plackova, 1962; Suckling, 1989). These enamel defects are simplistically classified as quantitative (hypoplasia) and qualitative defects (hypomineralisation). Hypomineralisation is defined as an abnormality in the mineral content and translucency of the enamel and also denominated as opacity of the enamel (Jalevik & Noren, 2000).

A specific pattern of developmental hypomineralisation of enamel on first permanent molars and often incisors was defined as *Molar Incisor Hypomineralisation* (MIH) in 2001 (Weerheijm, Jalevik, & Alaluusua, 2001). MIH is a demarcated qualitative defect of enamel of systemic origin, affecting one or more of the first permanent molars, often affecting the permanent incisors (Weerheijm et al., 2003). MIH is derived from an alteration in ameloblast function during the enamel maturation phase (Jalevik, Dietz, & Noren, 2005). Ameloblast metabolism related to MIH is thought to be more sensitive to change during the perinatal period (Fagrell, Salmon, Melin, & Noren, 2013). The affected teeth show clearly demarcated opacities of enamel of normal thickness with a smooth surface which can range from white to yellow or brown in colour

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(Weerheijm et al., 2003). Enamel of normal appearance in hypomineralised tooth has normal mineral density (Crombie et al., 2013).

Internationally, current MIH prevalence ranges between 3 and 44% with an average of 15%, which, considering its clinical, aesthetic and functional consequences, make it a major public health problem (Elfrink, Ghanim, Manton, & Weerheijm, 2015). The aetiological factors of MIH are not clear, however, it is likely to be multifactorial in origin involving child health (Crombie, Manton, & Kilpatrick, 2009). To date, few prospective studies addressing multiple risk factors have been published (Fagrell, Ludvigsson, Ullbro, Lundin, & Koch, 2011; Kuhnisch, Mach et al., 2014). This lack of studies relates to the need for longitudinal and often expensive studies, and the potential for loss of follow-up of participants is great, increasing sample size and decreasing representativeness. The most current hypotheses relate to *peripartum* problems, childhood diseases, dioxins (through breastfeeding), antibiotics during the first years of life of the child and ingestion of bisphenol A (e.g., through baby bottles and food packaging). Some currently postulated risk factors are innovations of the past century (e.g.,

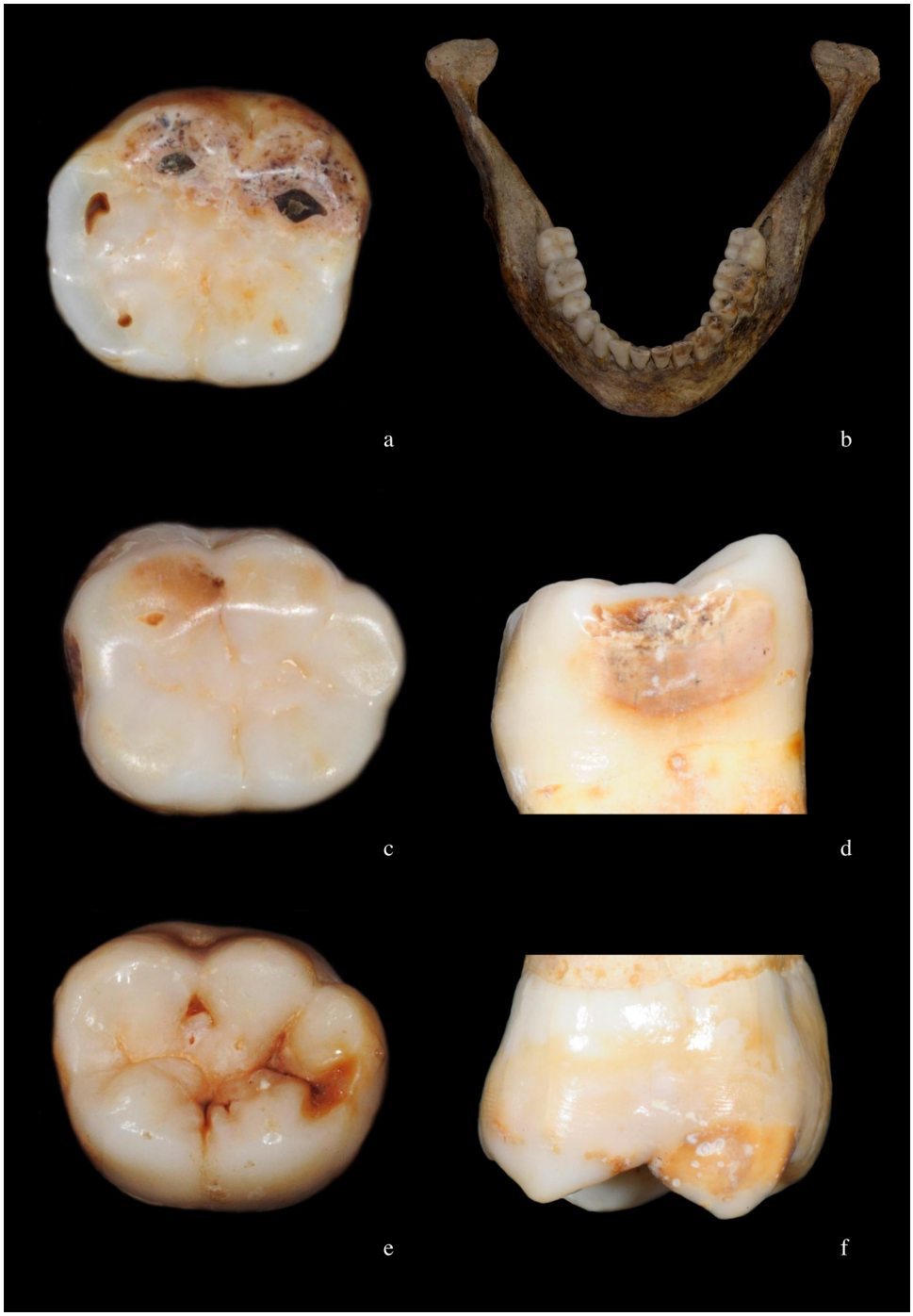

Fig. 1. (a) Taphonomic discoloration and wear on tooth 36 from (b) (Sains-en-Gohelle, France, 7–16th centuries), (b) Mandible and teeth with taphonomic discolorations (Sains-en-Gohelle, France, 7–16th centuries), (c) Wear on tooth 46 (Sains-en-Gohelle, France, 7–16th centuries), (d) White spot lesion stained by taphonomy on tooth 46 (Sains-en-Gohelle, France, 7–16th centuries), (e-f) Discolorations resembling demarcated hypomineralised lesion of enamel on tooth 46 (e) and 26 (f) (Sains-en-Gohelle, France, 7–16th centuries).