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Stress near the start of life? Localised enamel hypoplasia of the primary canine in late prehistoric mainland Southeast Asia

Siân E. Halcrow*, Nancy Tayles

Department of Anatomy and Structural Biology, Otago School of Medical Sciences, P.O. Box 913, University of Otago, Dunedin, New Zealand

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Abstract

Localised hypoplasia of the primary canine (LHPC) is characterised by roughly circular defective areas of thinned or missing enamel on the labial surface. This defect is rarely reported in bioarchaeological research. Using samples from late prehistoric mainland Southeast Asia, this paper documents the prevalence of LHPC to produce baseline data for this defect. The samples are from seven archaeological sites in Thailand and collectively span from *ca*. 4000 to 1500 BP. In the combined samples, 32/79 (40.5%) of individuals and 47/199 (23.6%) of teeth had LHPC. The high occurrence of LHPC may suggest there was poor maternal and infant health. There is also a high occurrence of caries associated with LHPC, which has implications for the assessment of deciduous dental health. This paper stresses the importance of the collection of dental enamel defect data from deciduous teeth including LHPC in bioarchaeological research.

Keywords: Dental defects; Deciduous teeth; Physiological stress; Thailand

1. Introduction

Dental enamel defects, particularly linear enamel hypoplasia (LEH), are important indicators of population health in bioarchaeological research. Investigating health using archaeological teeth is advantageous because they are resistant to diagenesis and do not remodel, thus providing robust, permanent markers of stress. Deciduous dental enamel defects can give useful insights into maternal and early infant health. One of these, LHPC, is reported in living populations to be associated with poor maternal health and low socio-economic status (SES) (Skinner and Hung, 1989). Localised hypoplasia of the primary canine, when reported in bioarchaeological studies, accounts for a disproportionate number of defects in deciduous teeth (Lukacs, 1991; Skinner, 1986; Skinner and Hung, 1989; Skinner and Newell, 2003: 61), but is not often reported. Recent archaeological work in Southeast Asia now provides an adequate sample of subadults to investigate LHPC in these populations (Bayard and Solheim, n.d.; Higham, 2002; Higham and Kijngam, 1984; Higham and Thosarat, 2004a,b; Pietrusewsky and Douglas, 2002a,b). The purpose of this paper is to illustrate the value of incorporating LHPC in research on the health of prehistoric populations by reviewing the aetiology of this defect and to provide baseline prevalence data for LHPC in select Southeast Asian archaeological samples (Fig. 1).

2. Deciduous dental enamel defects

Deciduous enamel defects include those, such as LHPC, that affect the quantity of enamel due to a disruption in the deposition of enamel matrix during formation, and defects in the quality of the enamel resulting from disruptions and deficiencies in mineralisation and include opacities and discolourations (Hillson, 1996: 165). It is argued that deciduous dental enamel defects are good indicators of permutations in amelogenesis from the second trimester to the end of the first year of life (Goodman and Rose, 1990a). The importance of looking at deciduous teeth and the defects specific to these teeth is becoming recognised both in archaeological and modern human

* Corresponding author. Tel.:+64 3 479 5265; fax: +64 3 479 7254
E-mail address: sian.halcrow@anatomy.otago.ac.nz (S.E. Halcrow).

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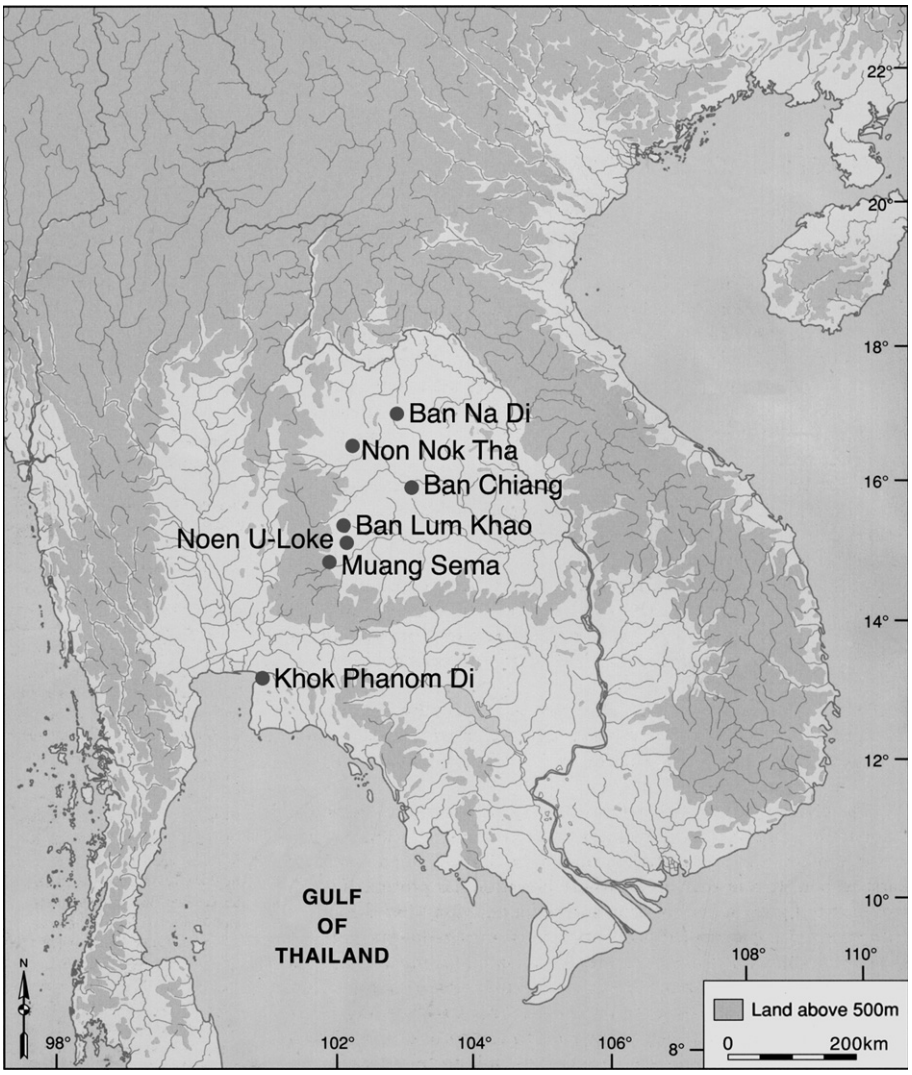

Fig. 1. Locations of the sites in Southeast Asia.

and non-human primate studies (e.g., Guatelli-Steinberg and Lukacs, 1999; Lukacs, 1999; Skinner and Newell, 2003), but still few bioarchaeological studies collect these data or report on these defects, compared with the vast number that investigate LEH in permanent teeth.

2.1. Localised hypoplasia of the primary canine

Localised hypoplasia of the primary canine is characterised by a roughly circular area of defective or thinned enamel in the form of a flattened or concave pit generally on the midlabial surface of the crown (Skinner and Newell, 2003: 61). Hillson and Bond (1997) refer to these as ''plane form'' hypoplastic lesions. In the clinical literature they are also referred to as primary canine hypoplasia (Duncan et al., 1994; Silberman et al., 1989, 1991) and facial surface hypoplasia (Brown and Smith, 1986).

Skinner and Hung (1989) used histological examination of longitudinal sections of canines with LHPC to identify formation timing with accumulated enamel prism length and crown extension methods. They found that, contrary to previous ideas about LHPC, they do not form until around 6 months of age. In some instances they also observed some cervically placed ameloblasts that had recovered, suggesting that the age of formation of LHPC may have been underestimated (Skinner and Hung, 1989).

Localised hypoplasia of the primary canine was originally described by Jørgensen (1956). He reported the appearance of LHPC in approximately one quarter of both modern and mediaeval Danes and attributed them to genetic factors. However, despite the reported high prevalence of this defect, its aetiology is not well understood (Lukacs, 1991). Some interest in the study of LHPC has arisen because of its perceived potential for investigating the theory of the innate vulnerability of males to stress in both human and non-human primates (Guatelli-Steinberg and Lukacs, 1999). Guatelli-Steinberg and Lukacs (1999) make the assumption in this research that LHPC form prenatally, despite Skinner and Hung's (1989) research. Given that LHPC commonly occur on the midlabial surface or cervical portion of the tooth and that only the cuspal 1/3 of the tooth is formed *in utero* (Massler et al., 1941), these