*Slov. Antropol., 21(1):1-5, 2018*

# AGE AT DEVELOPMENT OF LOCALISED HYPOPLASIA OF PRIMARY CANINES (LHPC) IN CHILDREN IN THE NECROPOLIS OF GREAT MORAVIA IN ZNOJMO-HRADIŠTĚ (9TH–10TH C. AD, CZECH REPUBLIC)

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**Abstract:** The aim of the study was to estimate the pre-, peri-, or postnatal origin of localized hypoplasia of primary canine (LHPC) and determine the most common age of death of individuals with LHPC. We also evaluated the total defect formation time and determine the average value in the months ahead. The examined file was 31 non-adults in the necropolis of Great Moravia in Znojmo-Hradiště (9th and the first half of the 10th century), who had retained primary canines with LHPC. 80.0% of individuals showed postnatal hypoplastic defect, 13.3% prenatal and 6.7% (n=1) displayed both postnatal and perinatal LHPC, too. The incidence of localised hypoplasia was the same in the upper and lower teeth. In maxillary canines, the incidence of postnatal hypoplasia was very high (90.9%), in the mandibular canines also significantly prevailed but was slightly lower (81.8%). On average, therefore, the incidence of LHPC of postnatal origin was 86.4% for the examined canines. More than half of the examined individuals with LHPC died at the age of 2–5 years. Average time of formation of hypoplastic defects in the primary canines comes in our group at 4.9 months, which is comparable with literature data of 5.2 months.

**Key words:** localised hypoplasia, deciduous canines, Slavic population, non-adults, enamel formation, Central Europe

## Introduction

This ancient centre and the direct predecessor of the medieval and present town of Znojmo was the heavily fortified Great Moravian stronghold of St. Hippolytus – Pöltenberg in the 9th to 11th centuries (Kalousek 1955, Klíma 1989). A rich central burial ground/necropolis from the 9th and first half of the 10th centuries was discovered. Seven years of research have so far brought a thorough investigation, versatile documentation and a collection of nearly 600 rich skeleton graves, which covered about one third of the assumed/anticipated area of the necropolis.

Localised enamel hypoplasia of the human primary canine (LHPC) is an area of defective enamel formation on the labial surface of the crowns of primary canines that was first reported by Jorgenson (1956). The size of such a defective area is usually 1–2 mm with a flat base. Most cases of LHPC are solitary, but multiple lesions in one tooth have been observed in both prehistoric and modern populations. Genetic and environmental factors are considered to be important in the aetiology of the lesion (Jorgenson 1956, Taji et al. 2000, Mukhopadhyay et al. 2014) as well as minor trauma to the developing canine tooth bud, e.g. sharp objects placed in the month of neonates (Skinner 1986). The location of the lesion is mostly at the mid-crown level. At birth, the incisal one third of the crown is formed and mineralization of all primary canines lasts until approximately nine months after birth (Mukhopadhyay et al. 2014). These facts show that the defects are initiated shortly after birth (Taji et al. 2000). At birth the cortical bone protecting the primary canine is absent or thin (temporary

fenestration as a result of osteopenia). Due to the protruded position of the low canines during their formation, along with the compression of the lower labial wall during birth, the prevalence of hypoplasia is higher (Nation, Matsson and Peterson 1987, Skinner and Newell 2000). However, there are studies reporting more common maxillary defects than mandibular ones (Shkrum and Wheeler 2010). A study of concordance in monozygotic and dizygotic twins has showed that the genetic factor is not the sole one (Taji et al. 2000) and probably several factors contribute to this process. Similarly, it is improbable that the environmental factor is the sole reason for the formation of a hypoplastic lesion, for were that the case, a study on Australian Aboriginals would have certainly showed a higher prevalence of hypoplasia because they live in harsher environmental conditions. Several studies (Lukacs 1991, Skinner and Hung 1986, Skinner 1986, Nation, Matsson and Peterson 1987, Seow 1991) confirmed a localized trauma as an aetiological factor of LHPC. Other up-to-date factors reported in the literature are a systemic effect on the ameloblasts, abrupt death of ameloblasts as a consequence of oxygen and nutritional deficiency and some instruments in the oral cavity such as laryngoscopy, intubation, etc., poor maternal health and low socio-economic status (Seow 1997, Aine et al. 2000, Skinner and Hung 1989, Taji et al. 2000).

**Materials and Methods**

We examined primary teeth of all non-adults from the burial site Znojmo-Hradiště excavated up to 2008. The studied collection of non-adults from the central necropolis and the settlement environment was, for a dental study, divided into the following groups: a) with LHPC (31 individuals), b) where LHPC was not detected (37 individuals) and c) where primary canines were missing or cannot be evaluated (35 individuals).

The dental examination was carried out closely by one examiner to eliminate inter-observer error, using the oblique light, zoom and a stereo-microscope. The teeth were rotated at various angles to highlight areas of deficient or defective enamel. The lack of continuity of the surface enamel was recorded as hypoplasia, according to the DDE index (the standardised code from the epidemiological index of developmental defects D-hypoplasia – pits and G-hypoplasia – missing enamel) (FDI Commission on Oral Health 1992, 423). We noted the location on the type of canine – maxillary or mandibular and left or right site. We then pinpointed the shape of hypoplastic defect – round, oval, triangular or irregular following Taji et al. (2000) and localization on labial surface of canine. This surface we have divided in thirds vertically – cervical, middle and incisal in the incisocervical plane, and horizontally – mesial, middle and distal in the mesiodistal plane following Wheeler (1969). Based on the location of the defect on the tooth surfaces, the lesions were divided into prenatal, perinatal, postnatal, or a combination of these according to the stage of mineralization.

Measureable primary canines (without abrasion, loose) with LHPC were photographed at 10-12× magnification under stereomicroscope. In the Quick Photo Camera, the teeth were measured from the cusp of the canine to the start of the enamel defect and the distance of the cusp of the canine and the cement-enamel border. According to literature (Mukhopadhyay et al. 2014), the initiation of calcification of primary canine starts at five months in utero, beginning at the cusp tip and mineralization of crowns of all primary canines is not complete until about nine months after birth. At birth, the incisal one third of the crown is formed, another two thirds of the maxillary canine crown 9.2 months, resp. mandibular canine crown 11.5 months after birth (Skinner 1986). Thus, the location of the defect may provide valuable clues about the onset of the lesion. Hilson (1996) provides more precise data for each type of tooth. For primary canine states before the birth of mineralization of 30%, both upper and lower teeth. So, we set border of 30% on the length of the labial surface of the crown (Fig. 1), and then borders of the perinatal period, 14 days before the birth and 14 days after birth, according the data from literature (Skinner 1986) an intermediate formation rate of enamel for the deciduous canine is 0.33 mm per month. If the distance of the defect start exceeded the border of perinatal period, the LHPC was determined to be postnatal, if the start lay between these borders was perinatal, and if the start of the defect fell below border of the perinatal

period, the LHPC was determined to be prenatal. Based on an intermediate formation rate of enamel for the deciduous canine 0.33 mm per month, we tried to determine more accurately by measuring the age of the individual at the time of the defect formation start and the time during which the defect is formed. Only canines that were not abraded (n=22 teeth of 15 individuals; Table 1) could be used for this measurement. For others, the period of birth was estimated only by assessment or orientational measurement in the Sigma Scan Pro program. For those canines, where the origin of localized hypoplasia was determined by both methods (by measurement and assessment), the results were mostly identical.

All individuals with LHPC were buried in the back position with the only exception slightly curled left. Grave goods were present in almost all individuals with LHPC graves, we cannot therefore say that were poor graves. Commonly occurring shards of container, chicken eggs shells, animal bones, seeds, iron knives and earrings, mostly bronze, but also lead and occurred even one silver earring.

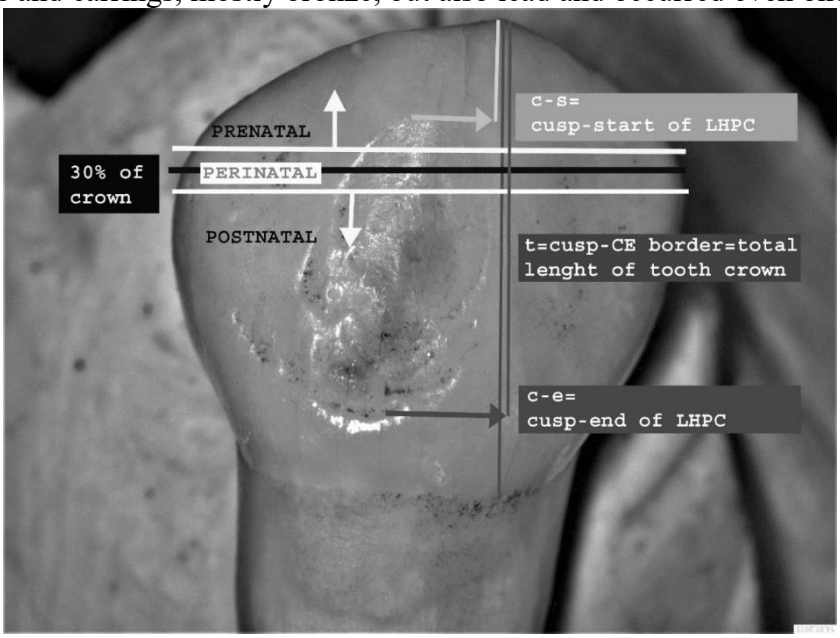

Fig. 1: Measure schema of LHPC

**Results**

The primary canine sample consisted of n=191 teeth, n=86 upper and n=105 lower primary canines. The canines of 45.59% individuals (n=31) displayed at least one hypoplastic defect. Approximately one eighth of individuals carried two or three hypoplastic defects on their canines, and two individuals (2.94%) had all their canines affected by hypoplasia. Most of the individuals were 2–5 years old, and more than quarter of them had multiple hypoplasia on the canines.

Based on the location of the defect on the measured tooth surface, the most frequent defect on both primary canines lower and upper originated in the postnatal period of life (86.4%). The table 1 shows the one individual has postnatal defect on one of the canines and perinatal defect on the other canines. The 90.9% of maxillary defects were of postnatal origin, only one defect was of prenatal origin. There are also dominating postnatal defects on the lower canines but not as significantly as in the maxillary teeth (81.8%). One defect originated in prenatal period of life and one in perinatal period. This defect could be the result of pressure on the lower jaw during childbirth, but two other individuals with a prenatal defect could be born prematurely.

More than half of these 15 individuals are children aged 2 to 5 years (n=8). Hypoplasias are predominantly postnatal in origin (n=12), only one 3-year-old and 6- to 7-year-old individual has LHPC of prenatal origin and one 7-year-old has both postnatal defect and perinatal defect. The remaining seven individuals are aged 6–11 years. Five of them have LHPC of postnatal origin, one prenatal, and one of the other, which also carries a defect of postnatal origin, carries on another canine

a defect of perinatal origin. We found that half (50.0%) of postnatal LHPC began to form in the first quarter of the first year of life (Fig. 2). Another six ones to six months of age, that is, during the first half of the year, 83.3% of all LHPCs were formed. In two prenatals, defects based on the origin of the defect a month before birth – it is question whether individuals born prematurely and was the reason of pressure during childbirth. The perinatal defect occurred less than a week before birth, therefore probably a LHPC originated at birth.

Table 1: Number of individuals with LHPC and number of teeth (maxillary, mandibular and total) with LHPC according to period of origin

| **Period of origin of LHPC** | **Number of individuals (%)** | **Number of maxillary teeth (%)** | **Number of mandibular teeth (%)** | **Number of teeth total (%)** |
|---|---|---|---|---|
| Postnatal | 12 (80.0) | 10 (90.9) | 9 (81.8) | 19 (86.4) |
| Perinatal | 0 (0.0) | 0 (0.0) | 1 (9.1) | 1 (4.5) |
| Perinatal + postnatal | 1 (6.7) | 0 (0.0) | 0 (0.0) | 0 (0.0) |
| Prenatal | 2 (13.3) | 1 (9.1) | 1 (9.1) | 2 (9.1) |
| Prenatal + postnatal | 0 (0.0) | 0 (0.0) | 0 (0.0) | 0 (0.0) |
| **Total** | **15 (100.0)** | **11 (100.0)** | **11 (100.0)** | **22 (100.0)** |

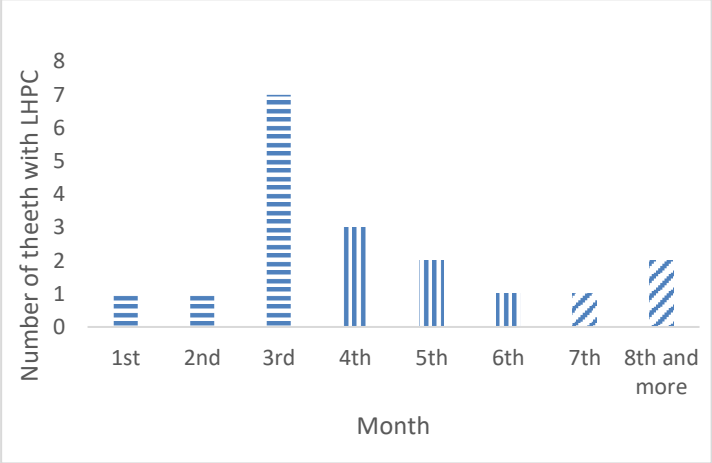


Fig. 2: LHPC formation rate in months after birth

We also tried to measure how long period of the life the hypoplastic defect was formed. The vast majority LHPC is relatively large defects, thus formed over its entire length several months. 15 teeth had a defect, which formed for more than a quarter of a year, only seven teeth had a defect of smaller extent, which formed within a quarter (three months). Skinner (1986) gives an average period of defect formation 5.2 months. In our set, this average time was 4.9 months. The hypothesis that those who died at an early age will have the greatest defects has not been confirmed. The largest defects had two 3-year-old individuals, but these were followed by individuals aged 7–9 years (five individuals). For the remaining 16 individuals, the LHPC was assessed only by macroscopy and the time of formation was estimated. The set contains the majority of individuals under the age of five (a total of 11 aged 1.5–5 years) and five individuals aged six to eight years. We evaluated a total of 28 primary canines with LHPC of these individuals. Apart from one defect, which we consider to be perinatal, others appear to be postnatal. A small perinatal defect was on a mandibular canine of a 3-year-old boy; on the other canine he had hypoplasia of postnatal origin, also of a small scale.

**Conclusion**

From the results of our measurement, perhaps the cause of the occurrence of these LHPCs could be considered above all the poor living conditions in the infant age (insufficient nutrition) and the related early deaths in the early childhood, as well as manipulation with various objects in the oral cavity due to the onset of defect formation a few weeks after birth, but such conclusions would have to be documented primarily by a larger number of individuals.

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