Experiencing Childhood at Roonka:

An Analysis of Enamel Hypoplasia in the Permanent Dentition of Australian Aboriginal Hunter-Gatherers

THE UNIVERSITY OF AUCKLAND
Te Whare Wānanga o Tāmaki Makaurau
NEW ZEALAND

CAITLIN BONHAM SMITH AND JUDITH LITTLETON

Introduction

Little is known about childhood in prehistoric Australian Aboriginal societies. Enamel hypoplasia provides a window into the life-history of these children (Littleton, 2005). DEH is a record of a critical period of disruption to growth in childhood, resulting from a trade-off within the body between resources allocated to growth (in this case dental formation) and resources needed to overcome a period of adversity where the body is investing in the immune system (Goodman and Rose, 1990). We explore the life-histories of children through the analysis of enamel hypoplasia amongst human remains from Roonka, a large burial place situated along the Murray River in South Australia (8,000 – 200BP).

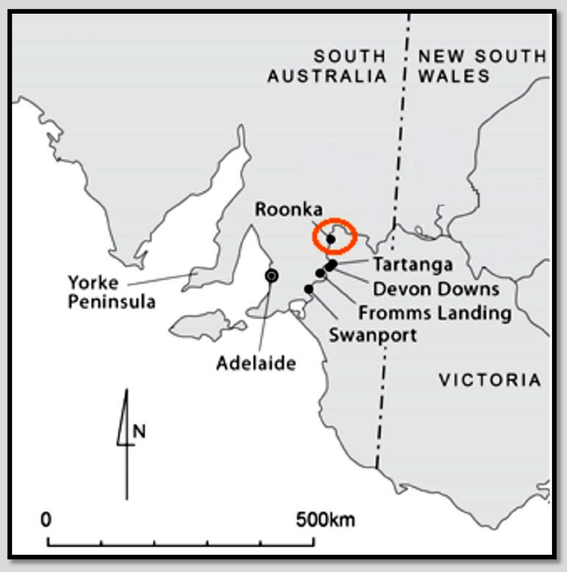

Figure 1 – Map of South Australia showing the position of Roonka along the Murray River

Figure 2 – Looking out at Roonka over the Murray River, photo by author

Methods

Dental enamel hypoplasia (DEH) was recorded in all individuals with teeth using two methods (n=130) (figure 3). Hypoplasia was assessed macroscopically across the dentition in order to understand variation in the number, types, and timing of defects. The location of the defect was recorded according to the figure 4 (below). This method was originally used by Hillson (1996) and modified by Littleton (2005) for use with Australian Aboriginal populations. The aim of this method is to capture the general patterns of defects in the enamel over all of the dentition, and thus gain a better understanding of the timing of challenges to child growth.

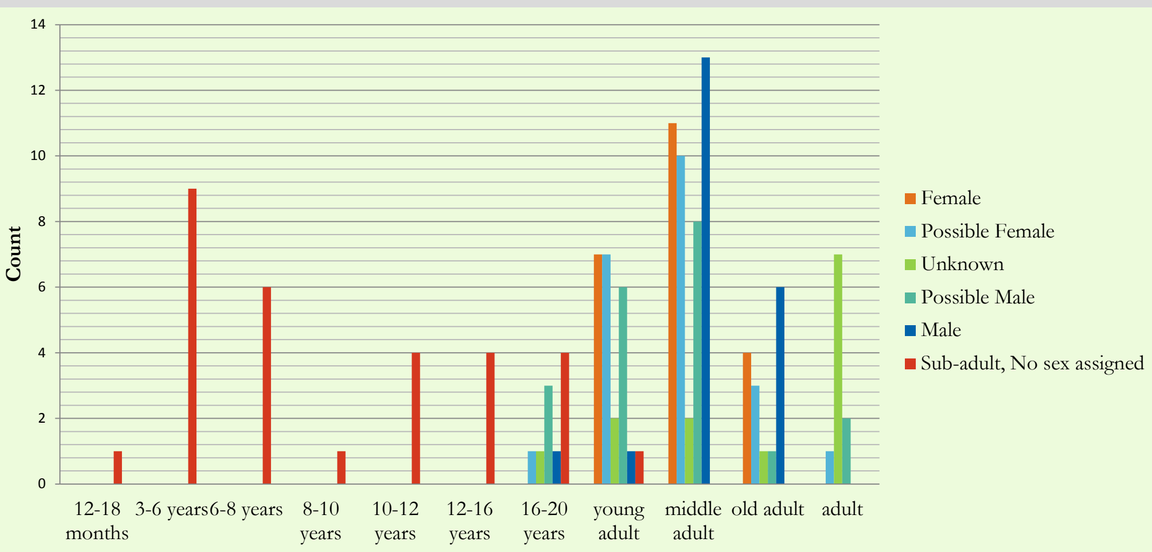

Figure 3– age and sex distribution of the individuals analysed

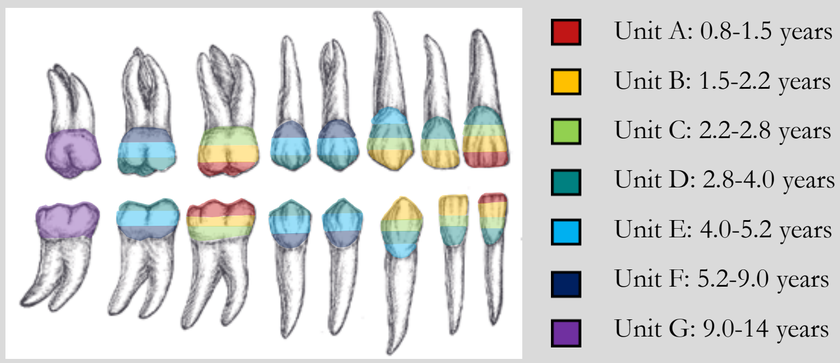

Figure 4– diagram of enamel units used for recording the position on an enamel defect. Modified from Littleton (2005). The units represent ages at which the enamel was formed. Therefore a defect that is located in Unit A represented a growth disruption between 0.8 and 1.5 years.

Additionally, lower canines (n=66) were observed under a scanning electron microscope in order to obtain finer detail in the timing of defects using a method outlined in (McFarlane, 2014; McFarlane et al., 2014). The bias resulting from attrition (defects lost) was controlled for by observing the number of disruption events (maximum number of defects across an enamel unit) in individuals with 5 or more enamel units observable.

When are disruptions to growth occurring in childhood at Roonka?

DEH is ubiquitous at Roonka, with all but 4 individuals having at least 1 defect when attrition is controlled. The Roonka sample demonstrates a peak prevalence at unit D (c. 2.8-4.0 years). Corresponding with the peak prevalence, the largest increase in age of onset occurred in the 6th to 7th deciles when recorded microscopically, corresponding to units C and D (c. 2.2-4 years) (figure 6).

The prevalence at Roonka does not drop off in the later forming units (E, F, and G). A greater number of defects in later forming units at Roonka suggests that for individuals with defects in F and G (c. 5.2 – 9 years and 9-14 years) stressors impacted growth for a prolonged period of time, a pattern which is not seen in a comparative post-contact Australian Aboriginal population (Littleton, 2005). As seen in table 1, while the late occurring defects are present, the number of defects is highly variable.

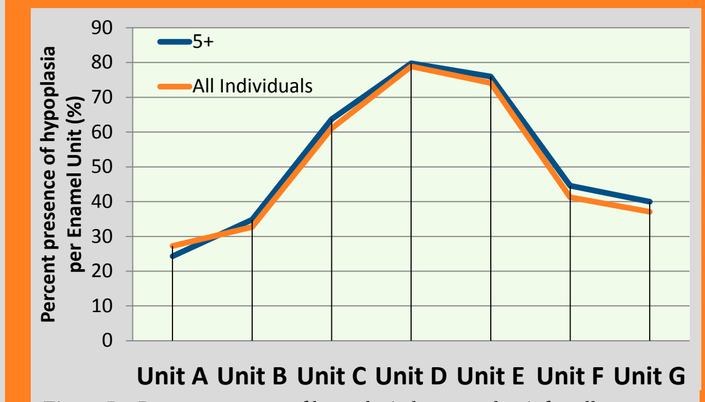

Figure 5 – Percent presence of hypoplasia by enamel unit for all individuals (n=130) and individuals with 5+ units present, controlling for attrition

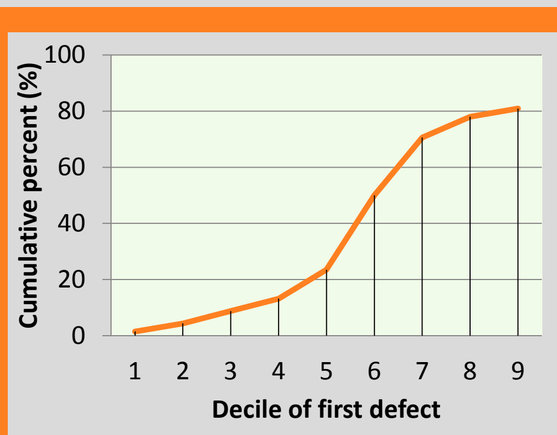

Figure 6 – Decile in which the first defect occurred as recorded from SEM images

Unit	Number of Units Observable	Mean Disruption Events	Median Disruption Events	Std. Deviation	CV (Coefficient of Variation)
A	44	0.318	0	0.561	176.40%
B	102	0.363	0	0.541	149.20%
C	114	0.754	1	0.698	92.60%
D	110	1.245	1	0.848	68.10%
E	113	1.088	1	0.84	77.20%
F	115	0.496	0	0.654	131.90%
G	90	0.378	0	0.51	135.00%

Table 1– A table of descriptive statistics by enamel unit for the number of disruption events (defects) showing the collapse in variation (CV) in the middle occurring units

There is high variability in the number of disruption events experienced (CV 73.3%) which may reflect variation in individual frailty or differences in exposure. If this variability is observed through childhood (by enamel unit), it becomes clear that this variability collapses in unit D (c. 2.8-4.0 years), which is the unit where nearly everyone has at least 1 defect. Transitions to solid or harder foods diets are associated with increased physiological stress resulting from shifts in nutrient intake, and therefore may be reflected in patterns of DEH (Pechenkina et al., 2002). Ethnohistoric accounts of childhood near Roonka describe a transition from close maternal contact and soft food to harder foods and more sociality (Eyre, 1845). Further evidence of this transition was found in microwear data, showing significant shifts in diet occurred between young children (0-3 years), juveniles (4-12 years), and adolescents (12-16 years) at Roonka (Burrows, 2015). The uniformity of DEH across the individuals at Roonka at this age indicates that it is a shared transition.

Relatively high child mortality between 3 and 6 years, largest gain in age at onset of DEH in units C and D, and highest percent prevalence of DEH in unit D all suggest this is not an easy transition for some children at Roonka. This data illuminates how childhood life experiences are shaped by the basics of growth and development (distribution of DEH by age unit) but also the social construction of childhood (variation by age unit). Further research is centred upon investigating the survival costs of this transition and whether this variability is structured chronologically or whether it points to a diversity of living conditions for children who are buried at the same place.

Acknowledgements

Thank you to Keryn Walshe and the South Australia museum for allowing us access to this remarkable and beautifully curated collection. I would like to acknowledge the First People of the River Murray and Mallee, who approved this project, and whose questions continue to shape our research. This research is funded by a Marsden Grant, and would not be possible without that support.

References

Burrows L. 2015. Sub-Adult Life History at Roonka Flat: A Dental Microwear Approach to Understanding Traditional Australian Aboriginal Aging Processes, unpublished thesis. The University of Auckland.

Eyre EJ. 1845. Journals of Expeditions into Central Australia and Overland from Adelaide to King George's Sound. London: T and W Boone.

Floyd B, Littleton J. 2006. Linear enamel hypoplasia and growth in an Australian aboriginal community: Not so small, but not so healthy either. Annals of Human Biology 33(4):424-443.

Goodman A, Rose J. 1990. Assessment of systemic physiological perturbations from dental enamel hypoplasias and associated histological structures. Yrbk Physical Anthropology 33:59–110.

Hillson S. 1996. Dental anthropology. Cambridge: Cambridge.

King T, Hillson S, Humphrey L. 2002. A detailed study of enamel hypoplasia in a post-medieval adolescent of known age and sex. Archives of Oral Biology 47(1):29-39.

Littleton J. 2005. Invisible Impacts But Long-Term Consequences: Hypoplasia and Contact in Central Australia. American Journal of Physical Anthropology 126: 295-304.

McFarlane GP. 2014. Estimating the percentage of crown height lost in mandibular canines. In: Anonymous 16th International Symposium on Dental Morphology and 1st Congress of the International Association for Paleodontology. Zagreb, Croatia.

Pechenkina EA, Benfer RA, Zhijun W. 2002. Diet and health changes at the end of the Chinese Neolithic: The Yangshao/Longshan transition in Shaanxi province. American Journal of Physical Anthropology 117(1):15-36.