Experiencing Childhood at Roonka:

A Bioarchaeological Analysis of Enamel Hypoplasia in the Dentition of Australian Aboriginal Hunter-Gatherers

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Introduction

Dental defects on the surface of teeth, called enamel hypoplasia, provide a detailed record of disruptions to childhood development resulting from stressors such as illness or under nutrition (Goodman and Rose, 1990; Hillson, 1996). **These defects result 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 (investment in the immune system).** The resultant defects remain on the tooth permanently as a record of a critical period of disruption in childhood.

Little is known about childhood in prehistoric Australian Aboriginal societies. Enamel hypoplasia provides a window into the life-history of these children (Littleton, 2005). Did children experience multiple stressful periods as their teeth formed or were they buffered from these challenges by biological or cultural confounders? I explore these questions through the analysis of enamel hypoplasia amongst human remains from Roonka, an archaeological site situated along the Murray River in South Australia.

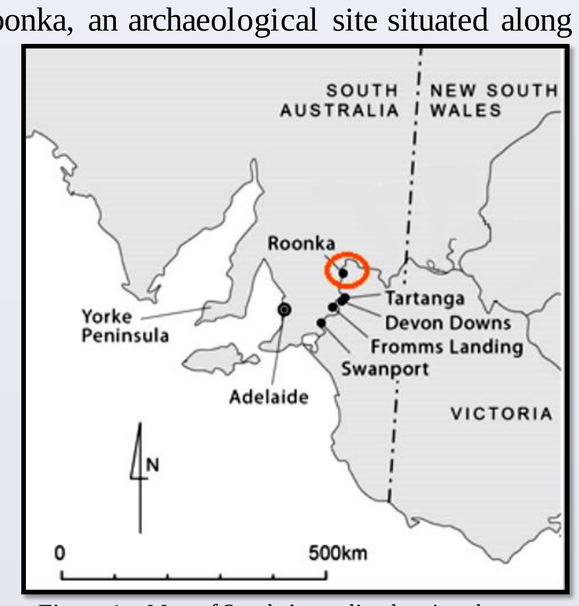

Figure 1 – Map of South Australia showing the position of Roonka along the Murray River

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

Previous Work on Childhood Lifehistory

Webb (1984) reported unusually high frequencies of enamel hypoplasia along the Murray River. He proposed that this pattern was the result of these populations both increasing in size and becoming less mobile. The consequence of these changes was increased physiological stress due to famine and disease, leaving a record of childhood growth disruptions (Webb, 1984; Lourandos and Ross, 1994). I will test this hypothesis using the two models described below.

Model 1: Stress > adaptation	Model 2: biocultural adaptation to stress
Evidence: • High reported frequencies of enamel hypoplasia in the Murray region (Webb, 1984). • Large number of burials along the Murray, supporting high population numbers (Pardoe, 1988). • An overall increase in the number of archaeological sites in the last 3,000 years (Lourandos and Ross, 1994).	**Evidence:** • Ethnographic information indicated that children along the Murray were 'spoiled with food' until they transitioned to foraging for themselves (Eyre, 1845). • This is supported by an analysis of striations on teeth at Roonka, which clearly demonstrated this transition around age 6 (Burrows, 2015).
Potential Process: There is a reliance on a staple crop, and people become less mobile. Population levels increase. The staple crop becomes less common, and food resources are short. The energy requirements needed to grow and fuel the immune system cannot be met. Children are exposed to illness, and the body must allocate resources to immune function and away from growth. The stress affects all children the same way, resulting is systematic and homogenous timing of growth disruptions.	**Potential Process:** People are mobile, but do tend to stay within tribal boundaries. There is a staple crop, but reliance on it is seasonal, or only in hard times. Children are protected from physiological stress at the earliest ages (and from weaning) by a social structure that favors only having one young child in a family at a time, and 'spoiling' that child with food. Children are less at risk of illness at a young age, because they are staying close to home. Around age 6 to 7 years there is a transition from dependent childhood to independent childhood.
Expected Outcome: Highly uniform timing of defects + overall high frequencies	**Expected Outcome:** middle to high frequencies + a lot of variation in the timing of defects

Table 1- Models of physiological stress based on previous work

Methods

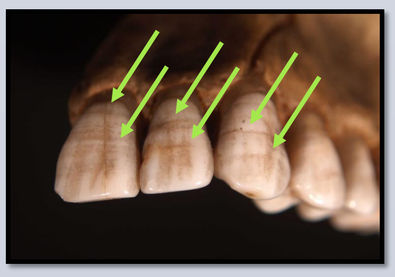

Figure 3 – Enamel hypoplasia, photo by author

All of the individuals with teeth (n=133) were scored for enamel hypoplasia (Figure 3). Each tooth is observed for the number and type of defects, and the location of the defect on the tooth surface. The location of the defect is critical, because it indicates the age at the onset of the event that caused the growth disruption. This provides a measure of the timing of growth disruption (stress) in childhood.

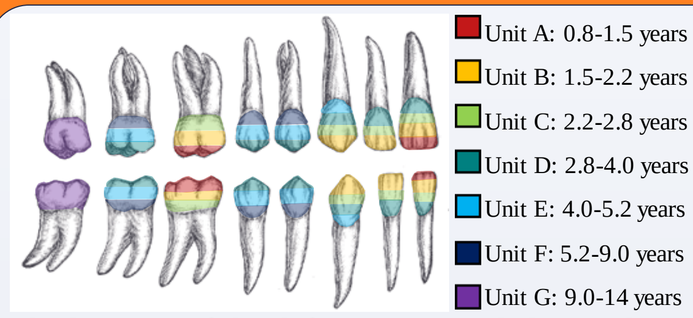

Figure 4 – diagram of enamel units used for recording the position on an enamel defect. Modified from Littleton (2005). The units represent ages at which the enamel was formed. Therefore a defect that is located in Unit A represented a growth disruption between 0.8 and 1.5 years.

The location of the defect was recorded according to the figure 4 (above). 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.

Results and Discussion

Webb's (1984) data showed a clear peak in the frequency of enamel hypoplasia at 3.5 years. Similarly, the Roonka children showed a rise in the mean number of defects around 2.8 to 4 years (Figure 6, 7). However, unlike Webb's sample, the frequency of defects is maintained throughout childhood. Interestingly, preliminary data shows that the Roonka sample exhibited higher overall frequencies of hypoplasia than Webb's Central Murray sample. Specifically the 5.2-9.0, and the 9.0-14 year units displayed higher frequencies. This would indicate that while elsewhere along the Murray physiological stress was alleviated by 9 years of age, at Roonka, young people are continuing to experience growth disruptions (table 2).

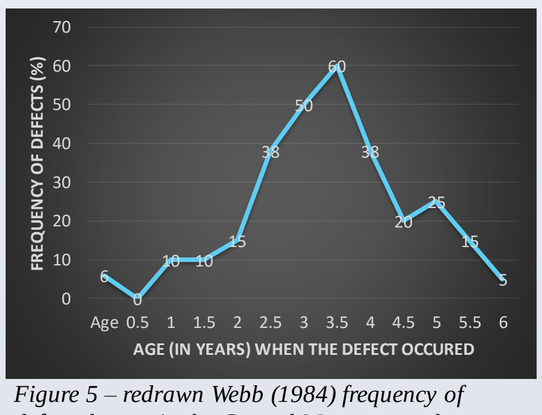

Figure 5 – redrawn Webb (1984) frequency of defects by age in the Central Murray sample

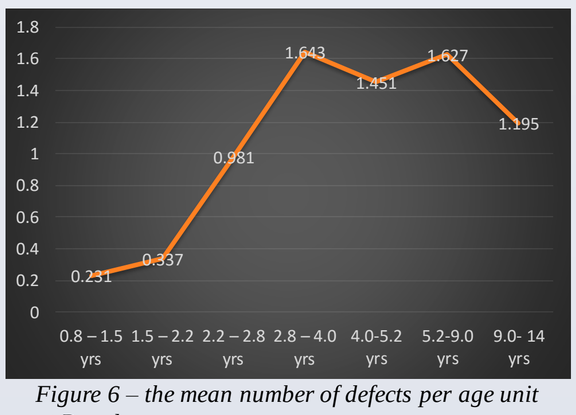

Figure 6 – the mean number of defects per age unit at Roonka

However, contrary to expectations predicted by the first model, children do not have multiple early occurring defects that are often associated with the weaning process (around 2 years of age). It is possible that the peak in the mean number of defects occurs at the tail in of the weaning process. There is ethnographic evidence that mothers were supplementing food with breastmilk until age four (Eyre, 1845). Furthermore, as shown in table 2, the amount of variation in the timing of the defects changes dramatically by age unit. At the youngest ages there is a lot of variation in the number of defects. This variation decreases until the peak at 2.8-4 years (red arrow), then increases again (blue arrow). Therefore using the models discussed earlier, it appears that the children at Roonka were actually experiencing a mixture of these two processes. At earlier and later ages, the number of defects is more variable, indicating differing success in buffering. Meanwhile, between 2.8 and 5.2 years the number of defects was much more homogeneous, indicating that nearly all children experienced at least 1 growth disruption at this time. This could indicate that cultural adaptations are successfully mitigating stress in the youngest at Roonka, and then that protection becomes variable once the child has to supplement their energy resources themselves. What is clear from the early stages of this research is that investigation into childhood life history is crucial to our understanding of pre-contact Australian Aboriginal health, subsistence strategy, and social organization.

Age when defect formed	0.8 – 1.5 yrs	1.5 – 2.2 yrs	2.2 – 2.8 yrs	2.8 – 4.0 yrs	4.0-5.2 yrs	5.2-9.0 yrs	9.0-14 yrs
Mean Number of Defects	.231	.337	.981	1.643	1.451	1.627	1.195
Std. Deviation	.4846	.5828	1.1880	1.7005	1.6628	2.4043	1.9334
Coefficient of Variation (%)	210.0	172.9	121.2	103.5	114.6	174.8	162.9

Table 2 – mean number of defects, standard deviation, and coefficient of variation for age units from Roonka. Red arrow indicates decreasing variation in the mean number of defects, and the blue arrow indicates increasing variation. The unit from 2.8-4.0 years shows the smallest amount of variation in the number of defects.

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