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 (i.e., 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 – 20,800BP).

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 decades when recorded microscopically, corresponding to units C and D (c. 2.2-4.4 years) (figure 6).

The prevalence at Roonka does not drop off in the later forming units (F, G, and F). 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.

The timing of these disruption events reflects life histories of children at Roonka. 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 (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 in prehistoric Australian Aboriginal societies indicate DEH (Pechenkina et al., 2002). Ethnohistoric accounts of childhood in prehistoric Australian Aboriginal societies indicate that this variability is a shared transition.

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.

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 (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 in prehistoric Australian Aboriginal societies indicate that this variability 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 baselines 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.

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References


Burrows, 2015. The uniformity of DEH across the individuals at Roonka at this age indicates that it is a shared transition.

Additional values in (1000BP).

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

Figure 3 – diagram of enamel units used for recording the position on an enamel defect. Modified from Littleton (2005). The letters represent enamel units as defined by Burrows (2015). Therefore a defect that is located in Unit A represents a growth disruption between 0.8 and 1.5 years.

Figure 4 – age and sex distribution of the individuals analyzed

Figure 5 – Percent presence of hypoplasia by enamel unit for all individuals (n=110) and individuals with >7 units present, controlling for attrition

Figure 6 – Decile in which the first defect occurred as recorded on SEM image

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 collapsing units

<table>
<thead>
<tr>
<th>Unit</th>
<th>Number of Units Observable</th>
<th>Mean Disruption Events</th>
<th>Median Disruption Events</th>
<th>Std. Deviation</th>
<th>CV (Coefficients of Variation)</th>
</tr>
</thead>
<tbody>
<tr>
<td>A</td>
<td>44</td>
<td>0.518</td>
<td>0</td>
<td>0.561</td>
<td>176.4%</td>
</tr>
<tr>
<td>B</td>
<td>102</td>
<td>0.563</td>
<td>1</td>
<td>0.695</td>
<td>52.6%</td>
</tr>
<tr>
<td>C</td>
<td>114</td>
<td>0.751</td>
<td>1</td>
<td>0.966</td>
<td>68.1%</td>
</tr>
<tr>
<td>D</td>
<td>110</td>
<td>1.245</td>
<td>1</td>
<td>0.846</td>
<td>68.1%</td>
</tr>
<tr>
<td>E</td>
<td>113</td>
<td>1.088</td>
<td>1</td>
<td>0.84</td>
<td>77.2%</td>
</tr>
<tr>
<td>F</td>
<td>115</td>
<td>0.496</td>
<td>0</td>
<td>0.654</td>
<td>120.3%</td>
</tr>
<tr>
<td>G</td>
<td>90</td>
<td>0.370</td>
<td>0</td>
<td>0.55</td>
<td>118.0%</td>
</tr>
</tbody>
</table>

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 (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 in prehistoric Australian Aboriginal societies indicate that this variability is a shared transition.

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