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 undernutrition (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 (Littletton, 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.

### 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 mobility, stress, illness 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

**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).

**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 in systematic and homogenous timing of growth disruptions.

**Expected Outcome:** Highly uniform timing of defects + overall high frequencies

### Model 2: biocultural adaptation to stress

**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:** 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 ‘spilling’ 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:** Middle to high frequencies + a lot of variation in the timing of defects

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### Table 1 - Models of physiological stress based on previous work

<table>
<thead>
<tr>
<th>Model</th>
<th>Description</th>
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</thead>
<tbody>
<tr>
<td>Stress &gt; adaptation</td>
<td>High uniform timing of defects + overall high frequencies</td>
</tr>
<tr>
<td>Biocultural adaptation to stress</td>
<td>Middle to high frequencies + a lot of variation in the timing of defects</td>
</tr>
</tbody>
</table>

### Reference


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