Childhood in colonial Otago, New Zealand – integrating isotopic and dental evidence for growth disturbance and oral health

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Experiences of childhood in colonial New Zealand are difficult to reconstruct from the historical record alone. Many of those who came to the colony were illiterate, and the Victorian tendency to avoid discussion of pregnancy and breastfeeding practices restricts our understanding of this important period. Bioarchaeological investigation, however, has the potential to illuminate the life stories of these first-generation Pākehā (European) settlers. Here we use isotopic evidence combined with dental pathology from children interred in a historic cemetery from Otago, New Zealand, to examine colonial childhood. We show how weaning practices in the colony differ from those experienced by their emigrant parents, highlight periods of illness likely associated with the weaning process, and bring to light the potential problems caused by maternal Vitamin D deficiency in the colony.

Keywords: Colonial New Zealand; carbon isotopes; nitrogen isotopes; linear enamel hypoplasia; Vitamin D deficiency.

Introduction

Children are an important cohort of society in their own right but, for the bioarchaeologist, also provide valuable insight into population health (Mays et al. 2017). As the most vulnerable members of the population, infants and children bear the brunt of the environmental stresses that societies may be experiencing, in particular inadequate nutrition and disease (Halcrow 2020). The transition from breastfeeding through the weaning process and into childhood is an especially vulnerable time, with infant feeding practices having a significant impact on survival. Inadequate nutrition and exposure to pathogens in supplementary food and water during weaning, and

throughout infancy and childhood have been linked with both developmental delays and the risk of chronic diseases, such as heart disease, in adulthood (Barker 2012; Waterland and Michels 2007). By studying childhood diet and health in the archaeological record we can, therefore, gain insights into survivorship and morbidity amongst the children of a population, and the health of the population as a whole (e.g. Gowland 2015).

Conditions in 19th century Britain and Ireland were certainly detrimental to the health of many. Wages were low and nutritional status was generally poor (Griffin 2018). Movement from rural areas to the burgeoning towns of the Industrial Revolution led to overcrowded, squalid living conditions in towns and cities, facilitating the transmission of infectious diseases (Wohl 2009). Smoke and particulates from the factories led to an increase in respiratory issues, and child labour was common (Moseley 2008; Perkin 1993). In the early 1800s, it was estimated that more than half of children born into families of the UK's urban poor died before they reached 5 years of age (Kay 1832). However, life in rural areas was not necessarily an improvement in comparison to urban environments. Rural populations were vulnerable to crop failures, poor drinking water and issues surrounding low income and associated poverty (Bogin 1999; Gowland et al. 2018).

For many, hope came in the form of subsidised travel to the newly established colony of New Zealand. From the 1840s onwards, the New Zealand Company advertised New Zealand as a place where resources were plentiful, and the poor conditions of the Industrial Revolution could be escaped. It was particularly lauded as a healthy place to raise children with commentators remarking on "the chubby children… and the very hearty appearance of their parents" (Swainson 1840: 56), and the "glow of health exhibited on the cheeks of the children of Europeans" in New Zealand (Lang 1839: 54). The New Zealand Company vision was that settlements in the colony would be a slice of life in England, transplanted to a new land, but with an abundance of resources

available to all. This was a place where the urban and rural poor could escape the cycle of poverty, and wealthier emigrants could raise their families to truly make their mark in society.

However, the biological realities of childhood for the Pākehā (European) settlers are largely unknown. Statistical records highlight that the birthrate was high during the colonial period in New Zealand, as was the infant mortality rate. Between 1861–1899, around 8% (or one in every eleven) Pākehā children died before reaching the age of one (Clarke 2012). In Otago, our area of study, there was growing concern over the numbers of 'destitute children' during the 1860s, leading to the establishment of the Otago Benevolent Institution which provided relief for infants and children abandoned, neglected and orphaned by their caregivers in the city of Dunedin and the wider Otago region (Angus 1984). Newspapers mention both the 'pitiable' children of Dunedin whose working mothers left them to their own devices during the day (Otago Daily Times, 1879), and the sensationalised stories of sin-ridden mothers who allowed their young families to become destitute (Bruce Herald, 1870). Between the years of 1869 and 1874, 216 children were admitted to the Otago Benevolent Institution, for the most part these were the offspring of prostitutes, women in prison and of 'seduced' women in domestic service (Angus 1984).

Generally, though, in keeping with the Victorian idea that children should be 'seen and not heard', the everyday details of children's lives are rarely mentioned in archival records. Bioarchaeological analysis of infants and children recently excavated at the site of St. John's, Milton in south Otago (Petchey et al. 2017) provides an opportunity to gain new insight into the youngest members of colonial society. Historical records associated with St. John's Milton suggest that, like the rest of New Zealand, the settlement of Milton had a high infant mortality rate, and skeletal evidence attests that life there was certainly not easy (Buckley et al. 2020). The aim of this

paper is to investigate the individual biological experiences of infants and children in the St John's sample through the integration of isotopic and osteological evidence of diet and health.

A combination of incremental isotopic and palaeopathological data provides the bioarchaeologist with a powerful tool for examining childhood history in the past (Walter et al. 2020; King et al. 2018; Garland et al. 2018; Eerkens et al. 2017). Chemical evidence for dietary changes during the weaning process and childhood can be obtained through isotopic analysis of tissues which form early in the life course (e.g. Craig-Atkins, Towers, and Beaumont 2018; Beaumont et al. 2015). For example, in both adults and children teeth retain a dietary signature from early life and hair, which forms up to time of death, may give insight into early life experiences for those who did not survive beyond early childhood (Beaumont et al. 2013). Isotopic indicators of dietary change can be compared with estimated time(s) of growth disturbance in childhood from determining the position, and therefore formation time, of dental enamel defects on an individual's teeth.

As a result of the strong genetic control over formation of teeth, the enamel of the tooth crown is still formed during times of physiological stress, but the secretion of the dental matrix is reduced. The thinning or absence of enamel, a dental enamel defect (DED), occurs when the secretory phase of amelogenesis is disrupted (Goodman and Armelagos 1985; Goodman 1998). Maintaining these organs at the expense of calcified tissues is preferential for immediate survival and therefore the presence of dental enamel defects can indicate that some type of systemic physiological disruption occurred during the formation of the tooth (Goodman and Martin 2002; Goodman 1991). A dual isotopic and DED approach allows us to address potential environmental factors, such as inadequate nutrition and infectious and metabolic diseases, that may have affected the health of mothers and their children, disrupting normal growth.

Often in bioarchaeology the early-forming tissues of adults (i.e. teeth) are studied in order to understand childhood experiences. In this study, we compare data from adult dentition (i.e. those who survived childhood) with records from the deciduous teeth and hair of those who died during childhood (i.e. the non-survivors). This more nuanced analysis allows us to assess differences between childhoods experienced in the UK (pre-emigration) and NZ (as first-generation colonists), applying a life course approach (Agarwal 2016) towards early childhood stressors. We aim to build an understanding of childhood experiences and their impact on the individual, expanding these individual stories to consider how the new environment may have affected population health in the fledgling colony.

Weaning behaviour and child health in Industrial Revolution Britain and colonial New Zealand - what do we know?

Weaning of infants is a process beginning with the introduction of non-breastmilk foods and ending when breastfeeding has completely ceased (Tomori, Palmquist, and Quinn 2018; Stuart-Macadam and Dettwyler 1995). Weaning practices are almost never uniform throughout a population as multiple factors affect decision making about when to wean and how long the weaning process should be (Lewis 2007; Dettwyler 1995; Sellen and Smay 2001). Britain during the Industrial Revolution was a time of changing norms surrounding breastfeeding and weaning practices, and those norms varied substantially between social classes, geographical locations in the British Isles and on the individual level. Today the WHO defines initiation of weaning prior to 6 months and weaning completion prior to two years of age as 'early' and likely detrimental to the health of the infant (World Health Organisation 2007). However, from the beginning of the 19th century women, particularly of the urban lower classes, were more likely to be employed in

industry than in the centuries prior, leading to increased likelihood of early weaning. During this period women commonly chose, or were forced to, spend little to no time breastfeeding and weaned quickly in order to return to earning (Wickes 1953; Henderson, Lee-Thorp, and Loe 2014; Newman and Gowland 2017; Perkin 1993). In the nineteenth century other factors may also have resulted in an accelerated weaning process. Poor living conditions for the underclasses lead to nutrient deficiencies and illness in mothers, often resulting in an inability to produce enough milk, or production of nutrient-deficient milk (Cheadle 1889). Infants were given pap or panada (soaked-cereal based weaning foods) or broths *in lieu* of or alongside breastmilk. However, most of these supplementary foods were nutritionally inadequate and may have resulted in deficiencies if not supplemented (Fildes 1986).

There was also variation in breastfeeding and weaning practices in different areas. If a mother could avoid returning to work, breastfeeding for more than 6 months was more normal. Rural mothers in England, as well as the Scottish and Irish were noted for this practice of longer breastfeeding, contrasting with the urban English poor (Fildes 1995). This practice was advantageous in many ways. Not only did it aid infant survival but may also have provided a contraceptive effect, allowing the mother to avoid another costly pregnancy and child in the family (Martin 2018).

Written sources describing the weaning behaviour of the upper middle and upper classes in Britain and Ireland suggest that the popularity of breastfeeding varied depending on whether or not it was perceived as fashionable at the time. In the 1840s–1850s it was recommended that the weaning process begin at 6 months and last to 12–18 months of age (Bull, 1849 cit. in Wickes 1953). However, the rich could afford wet-nurses allowing them to avoid the inconvenience of breastfeeding themselves (Perkin 1993). This practice allowed the mother to return to being

reproductively and socially active, and focus on producing more children, particularly male heirs (Fildes 1986). Though wet nursing became less fashionable in Britain towards the end of the nineteenth century, in New Zealand it remained popular among the colonial elites, and advertisements for wet nurses can be found in colonial newspapers right up to the turn of the century (Otago Daily Times, 1865; Star, 1880; Press, 1899).

Published sources from New Zealand from 1860s to 1890s align well with infant feeding recommendations from Britain and the USA. Indeed, many newspaper articles regarding weaning and infant health from the time are reprints of articles from British or US newspapers (e.g. Otago Witness, 1878; Globe, 1876). This printed advice suggests that the start of weaning was generally based upon the appearance (or cutting) of the front teeth (Fildes 1986; Otago Witness, 1878), with the weaning process ideally involving a gradual introduction of supplementary food. However, the timing of cessation of weaning was not agreed upon, with some arguing that children should be allowed no breastmilk beyond the age of 8 months (New Zealand Herald, 1886), and others claiming breastmilk in moderation should still be given up to 1.5 years (Otago Witness, 1878). What few personal accounts there are of European women breastfeeding in New Zealand are also contradictory. Some describe weaning only when poor health of the mother prevented extended breastfeeding (Caldwell, 1860 cit. in Porter and MacDonald 1996). Other letters of the time hint that in the New Zealand upper class, early weaning was common. Lely Richmond (1863), for example, commended her sister for none of her children having "depended for any long period wholly upon you for sustenance", while Lizzie Heath (1868) claimed it was common to have children “one every year”, implying early weaning in order for women to become reproductively active again (both cit. in Porter and MacDonald 1996). However, this archival evidence is related

to the upper-middle-class women of New Zealand, rather than the everyday colonists who made up the bulk of society.

In reality, breastfeeding and weaning practices likely varied substantially between individuals, even from the same place, time and social class. Study of individual weaning trajectories and disease experiences in the past, however, can help inform our interpretation of maternal, infant and population health, as well as the availability of weaning foods. By comparing individuals who were born in the UK with those who were born as some of the first European New Zealanders, we will be able to understand the differences (or lack thereof) between weaning decision-making 'at home' and in the new colony.

Archaeological proxies for weaning practices and impact on infant/child health

There are multiple lines of archaeological evidence which can be used to interpret breastfeeding and weaning practices in the past, and/or their outcomes in terms of health. These include chemical analysis of tissues, DED and tooth wear, and palaeodemographic techniques (Mays et al. 2017). In this study we focus on the primary data produced through bioarchaeological methods; isotopic analysis and the macroscopic study of oral health indicators such as DED and dental caries. DED are enamel defects linked to multiple aetiologies, including inadequate nutrition and disease, during the time of enamel formation (Smith 2018; Hillson 2014). Dental caries may be linked to diet, in particular the introduction of carbohydrate-rich weaning foods (Bonsall, Ogden, and Mays 2016), although the formation of carious lesions is also multifactorial and may be linked to fluoride levels, oral microbiome, hormonal balance, oral hygiene etc. (Hillson 2008a, 2008b; Kinaston et al. 2019; Lukacs and Largaespada 2006). Additionally, because the weaning period is a time of

increased vulnerability to nutritional deficiencies, we also explore dental and skeletal indicators of diseases related to undernutrition.

Isotopic analysis has long been used to study population-scale weaning timing (see Tsutaya and Yoneda 2015 for a review), but the recent advent of incremental isotopic techniques mean that it can now be used to interpret weaning trajectories in individuals (Beaumont et al. 2013; Beaumont and Montgomery 2015; Henderson, Lee-Thorp, and Loe 2014). Breastfeeding and the weaning process correlate with characteristic changes to the isotopic composition of tissues forming at this time. While breastfeeding an infant, their $\delta^{13}C$ values are elevated by 1 per mil (‰), and $\delta^{15}N$ values are elevated by 3–5‰ relative to the mother. During this period the infant is consuming their mother's protein, effectively placing them a trophic level 'above' them in terms of isotopic values. As the child undergoes weaning, these values gradually decrease until (at the point of complete weaning) they are similar to adult values in the population – providing they are being weaned onto a diet similar to that of the adults (Millard 2000).

Thus, by analysing serial sections of tissues that grow over known time periods, we can establish when weaning is likely to have commenced and when breastmilk ceased to be an important part of the childhood diet. In this study we use incremental analysis of both dentition (as per Beaumont et al. 2013) and hair (as per Fuller et al. 2006) for this purpose .

A 1mm increment of deciduous dentine will represent approximately 3 months of life (± 0.5 months), with formation times of each increment broadly known (Beaumont and Montgomery 2015; AlQahtani, Hector, and Liversidge 2010). Hair, on the other hand, forms up to time of death at a rate of 1cm per month (O'Connell et al. 2001). Therefore, by sampling in 1cm sections we can gain insight into diet at a monthly time-resolution prior to death.

Alongside the study of isotopic changes to tissue composition, we may also see DED that may have formed as a result of factors associated with the weaning process or later childhood (as per review by Katzenberg, Herring, and Saunders 1996). These may include inadequate nutrition, the introduction of pathogens with supplementary food and water exacerbated by the loss of immunologic components of breast milk (Katzenberg, Herring, and Saunders 1996; Goodman and Rose 1990). DED are associated with physiological perturbations that disrupt the secretory phase of amelogenesis (Kinaston et al. 2019). Macroscopically, DED can be expressed in a number of ways, but is typically observed as linear, plane-form or cuspal enamel hypoplasia and, in the deciduous dentition, circular carious lesions (i.e. the development of carious lesions on a pre-existing hypoplastic defect) (reviewed in Kinaston et al. 2019). Localised hypoplasia of the primary canines (LHPC) is characterized by a circular defect of missing enamel on the labial surface of this tooth type. LHPC is thought to result from prenatal/postnatal ill-health causing the temporary fenestration of the cortical bone around the tooth crypt combined with localised trauma to the developing tooth crown (see reviews in Stojanowski and Carver 2011; Halcrow and Tayles 2008; Skinner, Rodrigues, and Byra 2014). Macroscopic analyses, also known as the field method of recording, are used in this study because of the exceptionally fragile condition of the SJM teeth. Microscopic analyses have been suggested as identifying more depressed perikymata (Cares Henriquez and Oxenham 2019) and therefore more periods of stress. However, microscopic techniques rely on moulding the teeth, which was not feasible for the SJM sample.

DED have typically been referred to as non-specific indicators of physiological stress because the aetiology of enamel defects is so varied (Goodman and Rose 1990; Hillson 2014). However, recent critique of the use of the term ‘stress’ by Edinborough and Rando (2020 and articles within the special issue) has highlighted the general misuse of the term in bioarchaeology,

because it is so loosely defined. There is also uncertainty surrounding how 'stress' influences skeletal and dental growth, confusion added by the multiple aetiologies of DED, and issues understanding the relationship between stress and adaptation – a problem raised by the osteological paradox (Wood et al. 1992). In our study, where possible, we have attempted to match the estimated age of the duration of the weaning process with the estimated timing of the formation of the dental defect to identify whether there is a possible causal effect between the two.

Finally, the dietary changes associated with weaning may also result in visible dental or skeletal pathology. In the dentition, for example, deciduous caries may be present. Though caries is a multifactorial disease, it is often linked to diet (reviewed in Kinaston et al. 2019), and may potentially indicate that children were fed carbohydrate-rich weaning foods (as per Bonsall, Ogden, and Mays 2016). In 19th century New Zealand deciduous caries may also relate to sugar consumption during childhood.

Nutritional deficiencies associated with dietary change may also result in characteristic bony lesions or dental pathologies. Although most nutritional deficiencies do not leave diagnostic skeletal lesions, vitamin D deficiency, vitamin C deficiency, and acquired anemias (iron/vitamin B12 deficiency) are notable exceptions to this (Brickley and Mays 2019; Brickley 2018). Before the introduction of supplementary foods, breastmilk is the sole source of the majority of vitamins and trace minerals, with the exception of vitamin D which must be synthesized in the infant's skin through exposure to sunlight (World Health Organization, 2009). After approximately six months, the infant stores of iron acquired *in-utero* begin to deplete and vitamin C contained in breastmilk is no longer able to meet their metabolic needs. Therefore, the presence of skeletal lesions associated with scurvy and anaemia in infants and young children (<2 years) may be suggestive of

environmental factors, such as nutritionally insufficient supplementary food and the introduction of pathogens, especially parasites and bacteria, during the weaning process (Halcrow et al. 2018).

Materials

The individuals studied in this project derive from the St. John's Milton Anglican burial ground, located in Otago, New Zealand (Petchey et al. 2017). These individuals represent the first organised European settlement of the Tokomairiro plains, south of the city of Dunedin (Figure 1), and formed an early rural settlement, farming the plains and providing food for the colony (Sumpter and Lewis 1949). The burial ground was in use from first European settlement in 1850 to 1925, but the majority of burials date to the 1870s (Findlay 2016). All adults from the site are colonists who were born elsewhere, while all children were born in the colony and are first-generation Pākehā New Zealanders (King et al. 2020).

[Figure 1 near here]

Skeletal preservation at the site was highly variable. In fact, the only well-preserved elements of infants/children were their dentitions and hair; bones did not survive well in the burial environment (Buckley et al., 2020). Age at death of infants and children was therefore estimated using only dental formation stages (Moorrees, Fanning, and Hunt 1963b, 1963a). For adults, a multifactorial age estimation approach was used, combining observation of late fusing epiphyses (Schaefer, Black, and Scheuer 2009), pubic symphysis/auricular surface morphology (Brooks and Suchey 1990; Lovejoy et al. 1985), and dental wear (Smith 1984). Adult sex was estimated using the morphology of the pelvis and cranium (Phenice 1969; Walker in Buikstra and Ubelaker 1994; Acsádi and Nemeskéri 1970).

Enamel defects were assessed in individuals with available erupted dentition (*n* = 7 adults and *n* = 9 non-adults). All adult individuals with preserved dentition have previously been analysed using incremental isotopic techniques (King et al. 2020, *n* = 7). In this study all infants and children with preserved incrementally-forming tissues (i.e. either teeth or hair) were sampled. Unfortunately, the dentine of most non-adult individuals in the site was not well enough preserved to yield good quality collagen. As a result, only B8 (represented by a deciduous canine) yielded incremental dentine data. All other individuals analysed for carbon and nitrogen stable isotopes values (*n* = 8) are represented by hair samples. For individuals who died during the breastfeeding or weaning period, incremental hair results provide information regarding breastfeeding and weaning practices in the colony. For children who died at older ages, incremental hair analysis provides information regarding post-weaning childhood diet close to the time of death. Full isotopic sampling information is given in Table 1.

Individual	**Age at death**	**Tissue sampled (FDA tooth #)**	**Number of increments**
B2	neonate	hair	1
B8	18 months	tooth – max Rdc (53)	9
B16	2 years	hair	11
		tooth max Rdm1 (54)	*no yield*
B18	18 months	hair	4
		tooth mand Rdm1 (84)	*no yield*

B19	1 year	hair	7
		tooth max dm1 (54)	*no yield*
B20a	1.3 – 1.5 years	hair	2
		tooth – max Ldi1 (61)	*no yield*
B20b	3 years	hair	7
		tooth – max Rdm1 (54)	*no yield*
B27	15 months	hair	1
		tooth max Ldm1 (64)	*no yield*

Table 1. Individuals sampled/examined for each analysis undertaken in this study, and tissues used. Samples in italics did not yield good quality collagen.

Methods

Isotopic analysis:

The incremental isotopic analysis of dentine was conducted using established isotopic protocols (Beaumont et al. 2013; supplementary methods). In this study isotopic records from hair are also assessed, as some children were young enough at time of death to retain weaning signals in their hair. Hair was prepared as per O'Connell et al. (2001). Collagen and hair samples were analysed using a Costech elemental analyser connected to a Thermo Delta V Advantage isotope ratio mass spectrometer at the Stable Isotope Biogeochemistry Laboratory (SIBL), Durham University. Carbon and nitrogen isotope results are reported in standard delta (δ) notation as $\delta^{13}C$ and $\delta^{15}N$.

Isotopic accuracy was monitored through repeat measurements of international standards (e.g. USGS 40, USGS 24, IAEA 600, IAEA N1, IAEA N2). Analytical error was established by running repeat analyses and is < 0.2‰ for both isotopic systems.

Increments were assigned ages using the methods of Beaumont and Montgomery (2015). We acknowledge that linear sections of dentine cut across lines of dental formation, and therefore note that all assigned ages have associated errors, which differ from increment to increment (Tsutaya 2020). We do not use these ages as definitive, but rather as a way of comparing experience between individuals. In order to graphically present results dentine increment values were corrected for the hair-collagen offset: approximately +1.4‰ for $\delta^{13}C$, and +2‰ for $\delta^{15}N$ (Caut, Angulo, and Courchamp 2009; Drucker et al. 2008).

Enamel hypoplasia recording:

Research has shown that the anterior dentition is more sensitive to linear enamel hypoplasia development than the posterior teeth (Goodman and Armelagos 1985). However, the inclusion of the posterior teeth allows for the assessment of types of enamel hypoplasia that also affect molars (e.g. cuspal hypoplasia) and ensures teeth from different developmental life stages are included (Towle and Irish 2020). Therefore, in this study all available tooth crowns were assessed for linear enamel hypoplasia (LEH), plane-form and cuspal enamel hypoplasia, as well as circular caries and LHPC in the subadult dentition, as outlined in Kinaston et al. (2019), Ogden et al. (2007) and Cook and Buikstra (1979) and Halcrow and Tayles (2008), respectively. Full DED recording methods used are provided in the Supplementary Methods.

To estimate the general time of defect development for linear enamel hypoplasia of the permanent dentition, the distance between cemento-enamel junction (CEJ) and the defect was

measured for the six anterior mandibular and maxillary teeth using Mitutoyo needle-pointed digital calipers to 0.10 mm accuracy. The crown height was also measured from the occlusal surface of the tooth to the CEJ. The average crown height for each tooth type could not be estimated for the SJM assemblage because there were no adult individuals with unworn anterior teeth. As a result, we used the wear grade (from Smith 1984) to estimate the percentage of wear on crown height (Grade 1: 2.5%, Grade 2: 5%. Grade 3: 10%, Grade 4: 15%, Grade 5: 20%). Wear percentages were incorporated with the crown height to estimate the age of formation of the linear enamel hypoplasia defect using the LEH Chronology Calculator for northern Europeans (Cares Henriquez and Oxenham 2019). Assessing the timing of development for plane-form and cuspal enamel hypoplasia was not possible because of the diffuse nature of these defects on the crown surfaces (Hillson and Bond 1997). Pinpointing LHPC developmental timing is also difficult as it is thought to be related to prenatal ill-health resulting in the fenestration of the alveolar bone surrounding the tooth coupled with localized post-natal trauma to the tooth crown (see review by Halcrow and Tayles 2008).

Palaeopathology

All infant/child remains were macroscopically examined and any abnormalities were recorded after Ortner (2003: 49). Observation was limited by preservation issues; with the exception of a single foetal cranial fragment, all individuals were represented by dentition only. Caries were recorded on the deciduous and permanent dentitions of the non-adult individuals. Caries were recorded by location on the tooth (occlusal, interproximal, root surface, etc.) if the lesion had irregular border, was discoloured, and had necrotic dentin (Hillson 2008a).

Results

Isotopic analysis:

All isotopic results are presented in Supplementary Table 1. One individual in the sample has the complete weaning process visible isotopically, B8. The lowering of both $\delta^{15}N$ and $\delta^{13}C$ values at the start of B19's profile may also be indicative of a weaning process ceasing around 6 months of age, but there are too few datapoints prior to this age to be certain. All other individuals do not present typical weaning patterns (i.e. concurrent $\delta^{15}N$ and $\delta^{13}C$ value changes), and it is reasonable to assume that all were weaned prior to their first datapoint. Almost all individuals whose isotopic records represent the post-weaning period display a level of isotopic variability during childhood: for example, B16's $\delta^{15}N$ values decrease by around 2‰ between 14 and 20 months of age, while $\delta^{13}C$ values remain consistent. This potentially reflects fluctuations in meat consumption in early childhood and/or changing nitrogen balance during periods of growth (as per Henderson, Lee-Thorp, and Loe 2014).

[Figure 2 near here]

Figure 3 plots all childhood dietary isotope values from individuals at St John's Milton together. It includes both those who died during childhood and the adults (previously published in King et al. (2020)) of the population. Table 2 gives summary information for both children and adults.

[Figure 3 near here]

Individual	SJM child/ adult	End of weaning	Evidence for post-weaning dietary change?
B8	Child	17 months	Not visible

B16	Child	<13 months	2‰ decrease in $\delta^{15}N$ values from 14–20 months
B18	Child	<14 months	2‰ increase in $\delta^{15}N$ and 1‰ in $\delta^{13}C$ values from 14–18 months
B19	Child	6 months ?	No
B20a	Child	Unknown	Not visible
B20b	Child	<2.4 years	No
B6	Adult	<1.5 years?	approx. 1‰ fluctuations in $\delta^{15}N$ and $\delta^{13}C$ values between 2–3.5 years
B11	Adult	3.3 years	1‰ fluctuation in $\delta^{15}N$ values between 2–4years
B21	Adult	20 months	Gradual decrease in $\delta^{15}N$ post-weaning
B29	Adult	<10 months	<1‰ increase in $\delta^{15}N$ values between 1–3.5 years of age

Table 2. Information on age of possible weaning start and end for all individuals analysed.

Dental Enamel Defects

Only LEH (no cuspal or plane-form hypoplasia) was observed in the adult dentitions from the SJM assemblage, and this was mostly present on the anterior dentition (incisors and canines) (Supplementary Table 2, Fig 4). Only B23 had LEH on a mandibular first molar. The total number of anterior teeth available for analysis, the tooth affected by LEH and the timing of the defect development for each individual is presented in Table 3.

Burial	**TOT**	**AT (FDI)**	**Wear grade**	**Wear %**	**LEH 1 (yrs)**	**LEH 2 (yrs)**	**LEH 3 (yrs)**	**Isotopic interpretation of weaning period**
B4	3	32	3	10	2.0	2.6	3.2	Not assessed
B6	10	33	3	10	4.3			<1.5 years – <1.5 years?
B11	11	33	5	20	1.9	2.7	3.1	<9 months – 3.3 years
B11	11	32	4	15	2.0			
B11	11	42	4	15	2.0			
B11	11	41	5	20	2.1			
B11	11	31	3	10	2.1			
B11	11	21	4	15	2.3			
B11	11	11	4	15	2.4			
B11	11	43	4	15	2.4	2.9	3.2	
B11	11	12	4	15	2.8			
B11	11	23	4	15	3.1			

B13	1	43	5	20	2.3			Not assessed
B21	9	32	3	10	1.7	3.0		< 14 months - 20 months
B21	9	42	4	15	2.1	3.0		
B21	9	12	4	15	2.8			
B21	9	13	5	20	2.9	4.6		
B21	9	11	5	20	3.3			
B21	9	43	4	15	3.4	4.8	5.5	
B23	7	42	4	15	1.8			Not assessed
B23	7	41	3	10	1.8			
B23	7	32	3	10	1.9			
B23	7	31	4	15	2.1			
B23	7	12	4	15	3.2			
B23	7	43	4	15	3.5			
B23	7	33	3	10	3.6			

Table 3. Total observable anterior teeth, LEH affected teeth, LEH chronology and estimated weaning period for the SJM adults with LEH. TOT = total observable anterior teeth; AT = tooth affected by LEH (using FDI system); Wear grade from Smith (1984) and wear % described in text; LEH 1, 2, 3 = estimated age of formation in years for each observed LEH using the method outlined in Cares Henriquez and Oxenham (2019). Weaning period estimated from incremental isotope analyses.

[Figure 4 near here]

Three adult individuals, burials B4, B6 and B13, displayed LEH on only one tooth (although B4 had three LEH present on a single tooth). For burials B4 and B13, these individuals had three or fewer anterior teeth to assess, so it is unknown if other teeth may have had LEH. B6 was the only individual who had only one LEH but multiple anterior teeth (n = 10). With the exception of B6, the timing of the first LEH formation for all the SJM adult individuals ranged between 1.7 and 2.3 years. For Burial 4there were three distinct periods of LEH formation between approximately 2 - 3.2 years of age. For burial 11, there were possibly three matched events, the earliest occurring between the age of 1.9–2.1 years, another around 2.3–2.4 years, and a final event around 2.7–3.2 years. Burials 21 and 23 had an early period of LEH formation from matching LEH events, between approximately 1.7–2.1 years and then later periods ranging between 2.8–3.4 and 3.2–3.6 years, respectively. Burial 21 displayed a final period of LEH formation between approximately 4.6–5.5 years old.

For the subadults, only one individual had erupted permanent dentition (B5, aged 6–8 years) and none of these four permanent mandibular incisors had enamel hypoplasia. Nine subadult individuals had a total of 92 deciduous teeth that could be assessed and only two of these individuals had evidence for linear enamel hypoplasia (B20A and B27) (Supplementary Table 3; Table 4 and Table 5). B20A had two LEH present on the right mandibular canine (FDI 83). B27 had LEH in the form of linear row of pits on the cervical part of the crown of both maxillary canines (FDI 53 and 63) (Figure 5), but poor preservation meant it was not possible to assess the distance from the CEJ or determine the extent of tooth formation.

[Figure 5 and Table 4 near here]

Table 4. Prevalence rates by age of dental enamel defects, LEH, circular caries, planar defects and LHPC on deciduous teeth for the SJM non-adult assemblage

		overall DED				LEH				Circular caries				U-shaped plane-form defect				LHPC			
Age	N* ind	A	O	%	A/N (%)	A	O	%	A/N (%)	A	O	%	A/N (%)	A	O	%	A/N (%)	A	O**	%	A/N (%)
0.7 - 1.9 years	6	10	67	14.9	3/6 (50.0%)	3	67	4.5	2/6 (33.3%)	2	67	3.0	1/6 (16.7%)	2	67	3.0	1/6 (16.7%)	3	17	17.6	2/6 (33.3%)
2 - 8 years	3	0	25	0.0	0/3 (0%)	0	25	0.0	0/3 (0%)	0	25	0.0	0/3 (0%)	0	25	0.0	0/3 (0%)	0	6	0.0	0/3 (0%)
Total	9	10	92	10.9	3/9 (33.3%)	3	92	3.3	2/9 (22.2%)	2	92	2.2	1/9 (11.1%)	2	92	2.2	1/9 (11.1%)	3	23	13.0	2/9 (22.2%)

N*- Total number of individuals assessed. Note that burials 1, 3a, and 6 have been removed from the total counts as they only had unerupted teeth present
O-total teeth observed
O**-total primary canines observed
A- total teeth affected by each condition
A/N- Total affected individuals/total number of individuals
DED- Dental enamel defect
LEH- Linear enamel hypoplasia
LHPC- Localised hypoplasia of the primary canine

Circular carious lesions were located on bands of hypomineralised enamel of the cervical half of the central maxillary incisor crowns (FDI 51, 61) of burial 27 (Figure 6), suggesting a possible growth disruption between 30 fetal weeks and birth (AlQahtani, Hector, and Liversidge 2010) that led to caries susceptibility during the post-natal period (Duray 1990).

[Figure 6 near here]

Burial	**Age at death**	**DED**	**DED timing**	**Isotopic interpretation of weaning period**
B3B	1–1.5 years	None		Not assessed
B5	6–8 years	None		Not assessed
B8	18 months	None		6–7 months – 17 months
B16	2 years	None		<13 months – 20 months
B18	1-2 years	None		<14 months – <14 months
B19	1 year	LHPC	6 months ?	6–7 months – >9 months
B20A	1.3–1.5 years	LEH	~ 4.5 – 10.5 months	Unknown
B20B	3 years	None		< 2.4 years – < 2.4 years
B27	15 months	CC, USPH; LHPC	12 foetal weeks to birth; 6 months ?	Not assessed

Table 5. SJM non-adult burials assessed for DED, DED timing and estimated weaning period. DED = dental enamel defect, LEH = linear enamel hypoplasia, CC = circular carious lesions, USPH = U-shaped plane hypoplasia; LHPC = localised hypoplasia of the primary canine. DED timing estimated from AlQahtani et al. (2010) and weaning period estimated from incremental isotope analyses

Burials B19 and B27 had LHPC of their mandibular canines; tooth 83 was affected in Burial B19 and teeth 73 and 83 in Burial B27 (Figure 5). While the cusps of deciduous canines develop *in utero*, most of the tooth crown develops post birth (AlQahtani, Hector, and Liversidge 2010). Therefore, the position of these defects in the cervical part of the crown suggests the defect may have developed around 6 months of post-natal life, taking into account the aetiology of LHPC is thought to be a combination of prenatal ill-health and post-natal localised trauma (Skinner and Newell 2000; Skinner and Hung 1989; Halcrow and Tayles 2008).

As well as having circular carious lesions associated with LEH, B27 also exhibited U-shaped central notching defects, a plane-form hypoplasia, in the occlusal 3rd of the crowns of the deciduous mandibular central incisors (FDI 71, 81; Figure 7). This indicates a failure of enamel secretion occurring between 12 and 30 e weeks (AlQahtani, Hector, and Liversidge 2010; Sadler 2015) the potential aetiology of which is discussed below.

[Figure 7 here]

Other dental pathology

Three of the children had carious lesions of the deciduous dentition, including two individuals under two years of age (B16 and B27) (Table 6). Burial 27 was the infant with the circular carious lesions on both central maxillary incisors and B16 had an occlusal molar carious lesion. Burial 20B had four molar carious lesions that were either massive (n=2) or located on the occlusal surface of the crowns (n=2).

Age	# of individuals	Caries			
		A	O	%	n/individual (%)

0.7 – 1.9 years	6	3	67	4.5	2/6 (33.3%)
2 – 8 years	3	4	25	16.0	1/3 (33.3%)
Total	9	7	92	7.6	3/9 (33.3%)

Table 6. Deciduous carious lesions present in the sample. Note, that burials 1, 3a, and 6 have been removed from the total counts as they only had unerupted teeth present that were not developed enough to assess for EH. O = total deciduous teeth observed; A = total deciduous teeth affected by caries. All carious lesions were observed on the molars, with the exception of two circular carious lesions present on the central maxillary incisors of B27 (age 1–1.5 years).

Discussion

Interpreting weaning behaviour

Most incremental isotopic studies of weaning behaviour, regardless of archaeological context, have noted that intra-population variation in weaning time is the norm (Henderson, Lee-Thorp, and Loe 2014; Beaumont et al. 2015; King et al. 2018; Cocozza et al. 2021; Craig-Atkins, Towers, and Beaumont 2018). These results from colonial New Zealand are no exception to this pattern. Individuals with weaning behaviour visible isotopically all seem to have followed different weaning trajectories.

In many ways this is unsurprising, we know that weaning behaviour varied wildly even within the UK based on factors such as social class, working hours of the mother, place of residence, and whether breastfeeding was being used to mitigate poverty through further pregnancy (Nitsch, Humphrey, and Hedges 2011; Newman and Gowland 2017; Fildes 1998). The Milton settlers came from throughout the UK, excepting one individual (B4) who hailed from

continental Europe, born into families from different social classes, and with parents of different occupations (Findlay 2016). Variation in weaning behaviour may therefore reflect complex socioeconomic differences between individuals.

At least in part, weaning variability may reflect class differences both at home and in the new colony. Dry-feeding was relatively common in the nineteenth century and involved use of supplementary foods from close to birth, with little or no breastfeeding by the mother. This practice was both the fashion among the wealthy and the economical choice for the extremely poor (Nitsch, Humphrey, and Hedges 2011; Fildes 1986, 1995). Based on isotopic evidence, B29 may have been dry fed, as she seems to have been weaned prior to 10 months of age. We suggest she may have come from a wealthy background, as she had the most ornate coffin furniture in the sample implying wealth in later life (King et al. 2020). Interestingly, B29 was the only individual who did not exhibit LEH on any of her 7 observable teeth, 3 of which were anterior. It remains unknown if this lack of LEH is a sampling bias, or a reflection of a more privileged socioeconomic upbringing that buffered her from the environmental conditions that the other individuals may have experienced. Those with evidence for later weaning, conversely, may have come from family's deliberately prolonging the weaning process in order to avoid the financial burden of further pregnancy, or families who could not afford breastmilk substitutes (Nitsch, Humphrey, and Hedges 2011; Fildes 1998).

Comparing LEH timing with isotopic weaning profiles

Comparing LEH timing with isotopic evidence for weaning duration was only possible for adult burials B6, B11 and B21. For B6, the one LEH present was estimated to have formed around 4.3 years of age, many years after the projected end of weaning around 1.5 years of age. The fact that,

for B6, only one tooth had LEH out of 10 observable anterior teeth may indicate that the LEH could be a result of a localised event such as trauma to the tooth during formation (Towle and Irish 2020).

Burial 11 had multiple LEH present that were estimated to occur throughout the age ranges of 1.9–3.1 years. This range corresponds with the weaning period estimated from the isotope analysis of sometime before 9 months until approximately 3.3 years old. For B11, it is possible the LEH formation was associated with environmental factors associated with weaning, such as nutritionally-poor supplementary foods, inadequate amounts of food or pathogens in introduced food or water. Although B4, B13 and B23 did not provide isotope results, these individuals displayed a similar age range of early LEH development that ranged between 1.8 and 3.2 years old, suggesting that they had similar health experiences to B11 potentially including weaning during a similar life stage. For B21, isotopic results suggest a weaning period before 14 months up to around 20 months. This individual experienced three periods of LEH formation, one ranging between 1.7 and 2.1 years (at the end of the weaning period), the next a little later in childhood between the ages of 2.8 and 3.4 years, and the final between the ages of 4.6 and 5.5 years. Although we cannot know exactly what caused the LEH over this individual's early life, it is apparent that they were affected by multiple systemic insults that were severe enough to disrupt their tooth formation, such as inadequate nutrition or disease.

In contrast to the adults of SJM, most of the children (n = 6/9) from the SJM assemblage did not have any form of DED. This could be interpreted as them not experiencing conditions that cause growth disruptions, making it more likely that they died of acute causes, such as gastrointestinal disease. One of the exceptions to this is B20A, who had two LEH present on the lower third of a deciduous mandibular canine, indicating growth disruption between approximately

4.5 and 10.5 months old (AlQahtani, Hector, and Liversidge 2010). The hair isotopic record of this individual was not long enough to estimate weaning time, but if their weaning duration was similar to the other SJM children, then this defect would likely have occurred during this life stage. B27 had distinctive pitting in the cervical region of both maxillary canines indicating a serious growth disruption between approximately 7.5 months and 1 year of age, potentially associated with the initiation of weaning. Burial 27 also had LHPC, circular carious lesions and plane-form defects, discussed further below

Two infants, B19 and B27, had LHPC. Various interpretations of the aetiology of this enamel defect have been proposed including maternal ill-health and poor diet affecting bone formation over the tooth crypt *in utero* coupled with localized trauma to the tooth crown at the age of when babies have developed the co-ordination to bring hard objects into their mouths (~6 months post-birth) (Skinner and Newell 2000; Skinner and Hung 1989). Interestingly, for B19, the possible formation time of the LHPC (~6 months) corresponds with the beginning of the weaning period identified by the isotopic analyses. The beginning of weaning, when infants are moving food and objects to their face, has been associated with the tooth trauma thought to cause LHPC. Regardless of the exact aetiology of the LHPC in Burials 19 and 27, the defects also likely represent periods of maternal ill-health during pregnancy that affected infant tooth development after birth.

Childhood diet vs. adult diet in Milton

Generally speaking, individuals who did not survive childhood have post-weaning $\delta^{15}N$ values that are lower and $\delta^{13}C$ values that are more positive than average adult values at SJM. This may represent dietary differences between adults and children at the site. This pattern is consistent with

children consuming more low-trophic level C_4 resources than adults, for example maize. Although maize was not commonly eaten in the colony, corn flour was advertised as a flavoursome and cheaper alternative to wheaten flours, particularly considered ideal for children and invalids (Witness 1889). This isotopic difference may therefore reflect the substitution of corn flour into the paps and panadas for infants and children. Reliance on these plant-based carbohydrate-rich gruels may be partially responsible for the lower $\delta^{15}N$ values of the children in Milton. Support for this interpretation comes in the form of deciduous caries in some of the individuals – sometimes considered evidence for the consumption of starchy or sugary weaning foods, although the multifactorial nature of caries is important to recognize and therefore the observed carious lesions may be related to other factors such as fluoride levels and oral hygiene.

There are alternative dietary explanations for this pattern – children may have consumed more freshwater fish (with its more positive $\delta^{13}C$ values) and less meat overall (with associated lower $\delta^{15}N$ values) than adults: or, this pattern may not be dietary-related at all. It has been interpreted by others as a product of differences in nutrient requirements between children and adults, and the nitrogen deficit present in growing children, (e.g. Henderson, Lee-Thorp, and Loe 2014), though this has not been borne out in all studies (e.g. Waters-Rist and Katzenberg 2009).

Bioarcheological evidence for nutritional deficiencies or acute metabolic disease in the SJM sample

Some hair profiles do exhibit the 'spikes' in $\delta^{15}N$ values that have been associated with physiological stress (Reitsema 2013; Fuller et al. 2005). For example, B18's values rise by 1.5‰ between 15 and 18 months of age and B20A's values rise 0.5‰ in the month before their death. These spikes could represent episodes of severe and acute nutritional insufficiency or metabolic

disease during childhood for these individuals. However, isotopic changes may be due to numerous factors and this interpretation remains tentative.

One individual, B27, has clear dental evidence for some form of deficiency or metabolic insult during development. The unusual notching defects (a plane-form enamel hypoplasia) observed in their mandibular deciduous incisors, in addition to the LEH, LHPC and circular carious lesions observed for this individual, are generally suggestive of maternally transferred nutritional deficiency in the second and third trimester. Notch-shaped enamel defects can also be associated with congenital syphilis ("Hutchinson's incisors"); but these are only known to occur in permanent teeth and typically affect the maxillary incisors (Fiumara and Lessell 1983; Hillson, Grigson, and Bond 1998). DED of both the deciduous molars and incisors can also be a hallmark of a number of mineralisation disorders, including congenital vitamin D deficiency (Purvis et al. 1973; Reed et al. 2017). Vitamin D deficiency causes hypocalcaemia and hypophosphatemia (Foster et al. 2014) leading to issues in developing dentition.

The appearance of the notched-shaped hypoplasia observed in B27 is similar to that described in an early 20th century medical text describing dental lesions associated with vitamin D deficiency: *"There may be only a pitting, producing a honeycombed appearance of the enamel, or the enamel covering is slight, and the cutting edge of the tooth presents sharp points, giving a characteristic appearance to the tooth"* (Dick, 1992 cit. in Gowland et al. 2018: 56). It is therefore possible that the mother of B27 suffered from vitamin D deficiency during her pregnancy, particularly given that this condition is seasonally endemic in Otago today (Wheeler et al. 2018). Other studies of 16th–18th century English skeletal assemblages have also found cuspal and plane-form enamel hypoplasia that are likely also related to vitamin D deficiency (Gowland 2018; Ogden, Pinhasi, and White 2007), indicating that vitamin D deficiency was endemic in both the

home nations of the colonists, and the environment they immigrated to. The poor prenatal health of B27 may have led to continued sickness and ill-heath during infancy, as shown by the later-forming LEH and LHPC, which may have contributed to their early death.

Conclusions

Overall, the isotopic and paleopathological results paint a picture of colonial childhood that was not without its trials. Previous study of the sample has noted that infant mortality in the population was high (Buckley et al. 2020). The lack of DED in most children suggests that this high mortality rate was associated with acute episodes rather than the chronic stresses and recovery periods which result in visible DED. However, to fully understand life as a colonial child we need to go beyond general population trends. Here, we have been able to take a life-history approach to assess the individual experiences of childhood in St. John's Milton. For example, there are three children with recorded DED who did experience growth disruption during childhood, and for Burial 20A (and possibly B19 and B27) this was likely associated with the transition from breastmilk to supplementary foods. In all populations the weaning period is a vulnerable time for children, and colonial Milton was not an exception to this.

In addition to experiencing problems post-birth, one individual (B27) has evidence for maternal ill-health affecting their development in utero. The notching defects on the incisors of this individual suggest that vitamin D deficiency may have posed a problem for the population. Vitamin D not only has an important role in the formation of bone and teeth but also has essential regulatory roles in a wide range of physiological functions, including various immune mechanisms (Snoddy, Buckley, and Halcrow 2016). As such, vitamin D deficiency in the children of Milton may be representative of a larger problem with negative implications for the health of the wider

community. Future work on this site will apply new histological and radiographic methods for assessing markers of vitamin D deficiency in teeth (D'Ortenzio et al. 2018; Brickley, Kahlon, and D'Ortenzio 2020) to explore this hypothesis further (as per pilot data in Snoddy et al. 2020).

The study of adult dentition allows us to assess the experiences of childhood in the adults who grew up outside of New Zealand in the sample. Isotopic results suggest that the adult colonists experienced quite diverse infant-feeding practices, in contrast to the more uniform weaning behaviour seen in the children of colonial Milton. A number of the adults analysed had more than one LEH in their dentitions (and on individual teeth) indicating multiple events that caused growth disruption throughout their childhoods. Importantly, the presence of the LEH indicates that they survived whatever caused the growth disruption and lived well into adulthood, but how these earlier events affected adult health remains unknown. The adult LEH timings align with the isotopically observed weaning period in two individuals, highlighting again the vulnerability of this period regardless of where childhood is experienced.

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References

Acsádi, G, and J Nemeskéri. 1970. *History of Human Life and Mortality*. Budapest: Akadémiai Kiadó.

Agarwal, Sabrina C. 2016. "Bone Morphologies and Histories: Life Course Approaches in Bioarchaeology." *American Journal of Physical Anthropology* 159 (January): S130–49. https://doi.org/10.1002/ajpa.22905.

AlQahtani, S. J., M. P. Hector, and H. M. Liversidge. 2010. "Brief Communication: The London Atlas of Human Tooth Development and Eruption." *American Journal of Physical Anthropology* 142 (3): 481–90. https://doi.org/10.1002/ajpa.21258.

Angus, John. 1984. *A History of the Otago Hospital Board and Its Predecessors*. Dunedin: John McIndoe Ltd.

Barker, David JP. 2012. "Developmental Origins of Chronic Disease." *Public Health* 126 (3): 185–89. https://doi.org/10.1016/j.puhe.2011.11.014.

Beaumont, Julia, Jonny Geber, Natasha Powers, Andrew Wilson, Julia Lee-Thorp, and Janet Montgomery. 2013. "Victims and Survivors: Stable Isotopes Used to Identify Migrants from the Great Irish Famine to 19th Century London." *American Journal of Physical Anthropology* 150 (1): 87–98. https://doi.org/10.1002/ajpa.22179.

Beaumont, Julia, Andrew Gledhill, Julia Lee-Thorp, and Janet Montgomery. 2013. "Childhood Diet: A Closer Examination of the Evidence from Dental Tissues Using Stable Isotope

Analysis of Incremental Human Dentine." *Archaeometry* 55 (2): 277–95. https://doi.org/10.1111/j.1475-4754.2012.00682.x.

Beaumont, Julia, and Janet Montgomery. 2015. "Oral Histories: A Simple Method of Assigning Chronological Age to Isotopic Values from Human Dentine Collagen." *Annals of Human Biology* 42 (4): 405–12. https://doi.org/10.3109/03014460.2015.1045027.

Beaumont, Julia, Janet Montgomery, Jo Buckberry, and Mandy Jay. 2015. "Infant Mortality and Isotopic Complexity: New Approaches to Stress, Maternal Health, and Weaning." *American Journal of Physical Anthropology* 157 (3): 441–57. https://doi.org/10.1002/ajpa.22736.

Bogin, Barry. 1999. *Patterns of Human Growth*. Cambridge, UK: Cambridge University Press.

Bonsall, L., A. R. Ogden, and S. Mays. 2016. "A Case of Early Childhood Caries from Late Roman Ancaster, England." *International Journal of Osteoarchaeology* 26 (3): 555–60. https://doi.org/10.1002/oa.2441.

Brickley, Megan B. 2018. "Cribra Orbitalia and Porotic Hyperostosis: A Biological Approach to Diagnosis." *American Journal of Physical Anthropology* 167 (4): 896–902. https://doi.org/10.1002/ajpa.23701.

Brickley, Megan B, Bonnie Kahlon, and Lori D'Ortenzio. 2020. "Using Teeth as Tools: Investigating the Mother–Infant Dyad and Developmental Origins of Health and Disease Hypothesis Using Vitamin D Deficiency." *American Journal of Physical Anthropology* 171 (2): 342–53. https://doi.org/10.1002/ajpa.23947.

Brickley, Megan B, and Simon Mays. 2019. "Metabolic Disease." In *Ortner's Identification of Pathological Conditions in Human Skeletal Remains*, edited by Jane E Buikstra, 531–66. London: Academic Press.

Brooks, S., and J. M. Suchey. 1990. "Skeletal Age Determination Based on the Os Pubis: A

Comparison of the Acsádi-Nemeskéri and Suchey-Brooks Methods." *Human Evolution* 5 (3): 227–38. https://doi.org/10.1007/BF02437238.

Buckley, Hallie R, Phil Roberts, Rebecca L Kinaston, Peter Petchey, Charlotte L King, Kathryn Domett, Anne Marie E Snoddy, and Elizabeth A Matisoo-Smith. 2020. "Living and Dying on the Edge of the Empire: A Bioarchaeological Examination of Otago's Early European Settlers." *Journal of the Royal Society of New Zealand* Early View.

Buikstra, Jane E, and Douglas H Ubelaker. 1994. *Standards for Data Collection from Human Skeletal Remains*. Lafayette, AK: Arkansas Archaeological Survey.

Cares Henriquez, Alejandra, and Marc F. Oxenham. 2019. "New Distance-Based Exponential Regression Method and Equations for Estimating the Chronology of Linear Enamel Hypoplasia (LEH) Defects on the Anterior Dentition." *American Journal of Physical Anthropology* 168 (3): 510–20. https://doi.org/10.1002/ajpa.23764.

Caut, Stéphane, Elena Angulo, and Franck Courchamp. 2009. "Variation in Discrimination Factors (Δ15N and Δ13C): The Effect of Diet Isotopic Values and Applications for Diet Reconstruction." *Journal of Applied Ecology* 46 (2): 443–53. https://doi.org/10.1111/j.1365-2664.2009.01620.x.

Cheadle, Walter B. 1889. *On the Principles and Exact Conditions to Be Observed in the Artificial Feeding of Infants :The Properties of Artificial Foods and the Diseases Which Arise from Faults of Diet in Early Life*. 4th ed. London: Smith, Elder and Co.

Clarke, Alison. 2012. *Born to a Changing World*. Wellington: Bridget Williams Books.

Cocozza, Carlo, Ricardo Fernandes, Alice Ughi, Marcus Groß, and Michelle M. Alexander. 2021. "Investigating Infant Feeding Strategies at Roman Bainesse through Bayesian Modelling of Incremental Dentine Isotopic Data." *International Journal of*

Osteoarchaeology, 0–2. https://doi.org/10.1002/oa.2962.

Cook, Della Collins, and Jane E. Buikstra. 1979. "Health and Differential Survival in Prehistoric Populations: Prenatal Dental Defects." *American Journal of Physical Anthropology* 51 (4): 649–64. https://doi.org/10.1002/ajpa.1330510415.

Craig-Atkins, Elizabeth, Jacqueline Towers, and Julia Beaumont. 2018. "The Role of Infant Life Histories in the Construction of Identities in Death: An Incremental Isotope Study of Dietary and Physiological Status among Children Afforded Differential Burial." *American Journal of Physical Anthropology* 167 (3): 644–55. https://doi.org/10.1002/ajpa.23691.

D'Ortenzio, Lori, Isabelle Ribot, Bonnie Kahlon, Benoit Bertrand, Emmy Bocaege, Emeline Raguin, Annabelle Schattmann, and Megan Brickley. 2018. "The Rachitic Tooth: The Use of Radiographs as a Screening Technique." *International Journal of Paleopathology* 23 (December): 32–42. https://doi.org/10.1016/j.ijpp.2017.10.001.

Dettwyler, Katherine A. 1995. "A Time to Wean : The Hominid Blueprint for the Natural Age of Weaning in Modern Human Populations." In *Breastfeeding: Biocultural Perspectives*, edited by Patricia Stuart-Macadam and Katherine A. Dettwyler, 39–73. New York: Routledge.

Drucker, Dorothée G., Anne Bridault, Keith A. Hobson, Elwira Szumae, and Bocherens Hervé. 2008. "Can Carbon-13 in Large Herbivores Reflect the Canopy Effect in Temperate and Boreal Ecosystems? Evidence from Modern and Ancient Ungulates." *Palaeogeography, Palaeoclimatology, Palaeoecology* 266 (1–2): 69–82. https://doi.org/10.1016/J.PALAEO.2008.03.020.

Duray, Stephen M. 1990. "Deciduous Enamel Defects and Caries Susceptibility in a Prehistoric Ohio Population." *American Journal of Physical Anthropology* 81 (1): 27–34.

https://doi.org/10.1002/ajpa.1330810105.

Edinborough, M, and C Rando. 2020. “Stressed Out: Reconsidering Stress in the Study of Archaeological Human Remains.” *Journal of Archaeological Science* 121: 105197.

Eerkens, Jelmer W., Bryna Hull, Jena Goodman, Angela Evoy, Joshua D. Kapp, Sidra Hussain, and Richard E. Green. 2017. “Stable C and N Isotope Analysis of Hair Suggest Undernourishment as a Factor in the Death of a Mummified Girl from Late 19th Century San Francisco, CA.” *PLoS ONE* 12 (9). https://doi.org/10.1371/journal.pone.0184921.

Fildes, Valerie A. 1986. *Breasts, Bottles and Babies*. Edinburgh: Edinburgh University Press.

———. 1995. “The Culture and Biology of Breastfeeding: An Historical Review of Western Europe.” In *Breastfeeding Biocultural Perspectives*, edited by Patricia Stuart-Macadam and Katherine A. Dettwyler, 101–44. New York: Aldine De Gruyter.

———. 1998. “Infant Feeding Practices and Infant Mortality in England, 1900–1919.” *Continuity and Change* 13 (2): 251–80.

Findlay, Robert. 2016. *A History of the St. John’s Burial Ground. Back Road, Tokomairiro, Otago*. Auckland: Robert M Findlay.

Fiumara, Nicholas J, and Simmons Lessell. 1983. “The Stigmata of Late Congenital Syphilis.” *Sexually Transmitted Diseases* 10 (3): 126–29. https://doi.org/10.1097/00007435-198307000-00005.

Foster, B. L., M. S. Ramnitz, R. I. Gafni, A. B. Burke, A. M. Boyce, J. S. Lee, J. T. Wright, S. O. Akintoye, M. J. Somerman, and M. T. Collins. 2014. “Rare Bone Diseases and Their Dental, Oral, and Craniofacial Manifestations.” *Journal of Dental Research* 93 (7_suppl): 7S-19S. https://doi.org/10.1177/0022034514529150.

Fuller, Benjamin T., J.L. Fuller, D.A. Harris, and R.E.M. Hedges. 2006. “Detection of

Breastfeeding and Weaning in Modern Human Infants with Carbon and Nitrogen Stable Isotope Ratios." *American Journal of Physical Anthropology* 129 (2): 279–93. https://doi.org/10.1002/ajpa.20249.

Fuller, Benjamin T., James L. Fuller, Nancy E. Sage, David A. Harris, Tamsin C. O'Connell, and Robert E. M. Hedges. 2005. "Nitrogen Balance and Δ15N: Why You're Not What You Eat during Nutritional Stress." *Rapid Communications in Mass Spectrometry* 19 (18): 2497–2506. https://doi.org/10.1002/rcm.2090.

Garland, Carey, Laurie Reitsema, Clark Spencer Larsen, and David Hurst Thomas. 2018. "Early Life Stress at Mission Santa Catalina de Guale: An Integrative Analysis of Enamel Defects and Dentin Incremental Isotope Variation in Malnutrition." *Bioarchaeology International* 2 (2): 75–94. https://doi.org/10.5744/bi.2018.1019.

Goodman, AH. 1998. "Developmental Defects of Dental Enamel." In *The Cambridge Encyclopedia of Human Growth and Development*, edited by SJ Ulijaszek, FE Johnston, and MA Preece, Cambridge, 210–11. Cambridge.

Goodman, AH, and D Martin. 2002. "Reconstructing Health Profiles from Skeletal Remains." In *The Backbone of History: Health and Nutrition in the Western Hemisphere*, edited by R Steckel and JC Rose, 11–60. Cambridge: Cambridge University Press.

Goodman, Alan H. 1991. "Stress, Adaptation, and Enamel Developmental Defects." In *Human Paleopathology: Current Synthesis and Future Options*, edited by DJ Ortner and Arthur C. Aufderheide, 280–87. Washington D.C: Smithsonian Institution Press.

Goodman, Alan H, and George J. Armelagos. 1985. "Factors Affecting the Distribution of Enamel Hypoplasias within the Human Permanent Dentition." *American Journal of Physical Anthropology* 68 (4): 479–93. https://doi.org/10.1002/ajpa.1330680404.

Goodman, Alan H, and Jerome C Rose. 1990. “Dental Enamel Hypoplasias as Indicators of Nutritional Status.” In *Advances in Dental Anthropology*, edited by MA Kelley and CS Larsen, 279–93. New York: Wiley-Liss.

Gowland, Rebecca L., Anwen Caffell, Sophie Newman, Alysa Levene, and Malin Holst. 2018. “Broken Childhoods: Rural and Urban Non-Adult Health during the Industrial Revolution in Northern England (Eighteenth-Nineteenth Centuries).” *Bioarchaeology International* 2 (1): 44–62.

Gowland, Rebecca L. 2015. “Entangled Lives: Implications of the Developmental Origins of Health and Disease Hypothesis for Bioarchaeology and the Life Course.” *American Journal of Physical Anthropology* 158 (4): 530–40. https://doi.org/10.1002/ajpa.22820.

———. 2018. “‘A Mass of Crooked Alphabets’: The Construction and Othering of Working Class Bodies in Industrial England.” In *Bioarchaeological Analyses and Bodies*, 147–63. Springer International Publishing. https://doi.org/10.1007/978-3-319-71114-0_8.

Griffin, Emma. 2018. *A Short History of the British Industrial Revolution*. London: MacMillan.

Halcrow, Siân E. 2020. “Infants in the Bioarchaeological Past: Who Cares?” In *The Mother-Infant Nexus in Anthropology. Small Beginnings, Significant Outcomes*, edited by Rebecca L Gowland and Siân E Halcrow, 19–38. Cham, Switzerland: Springer. https://doi.org/10.1007/978-3-030-27393-4_2.

Halcrow, Siân E, Charlotte L King, Andrew R Millard, Anne Marie E Snoddy, Rachel M Scott, Gail E. Elliott, Hallie R Buckley, Vivien G Standen, and Bernardo T Arriaza. 2018. “Out of the Mouths of Babes and Sucklings: Breastfeeding and Weaning in the Past.” In *Breastfeeding: New Anthropological Approaches*, edited by C. Tomori, A. Palmquist, and E.A. Quinn, 155–69. New York: Routledge.

Halcrow, Siân E, and Nancy Tayles. 2008. “Stress near the Start of Life? Localised Enamel Hypoplasia of the Primary Canine in Late Prehistoric Mainland Southeast Asia.” *Journal of Archaeological Science* 35 (8): 2215–22. https://doi.org/10.1016/j.jas.2008.02.002.

Henderson, Rowena C., Julia Lee-Thorp, and Louise Loe. 2014. “Early Life Histories of the London Poor Using Δ13C and Δ15N Stable Isotope Incremental Dentine Sampling.” *American Journal of Physical Anthropology* 154 (4): 585–93. https://doi.org/10.1002/ajpa.22554.

Hillson, Simon. 2008a. “Dental Pathology.” In *Biological Anthropology of the Human Skeleton*, edited by M. Anne Katzenberg and Shelley R. Saunders, 301– 340. New York: Wiley-Liss.

———. 2008b. “The Current State of Dental Decay.” In *Technique and Application in Dental Anthropology*, edited by Joel D. Irish and K Nelsen, 111–35. Cambridge: Cambridge University Press.

———. 2014. *Tooth Development in Human Evolution and Bioarchaeology*. Cambridge, UK: Cambridge University Press.

Hillson, Simon, and Sandra Bond. 1997. “Relationship of Enamel Hypoplasia to the Pattern of Tooth Crown Growth: A Discussion.” *American Journal of Physical Anthropology* 104 (1): 89–103. https://doi.org/10.1002/(SICI)1096-8644(199709)104:1<89::AID-AJPA6>3.0.CO;2-8.

Hillson, Simon, Caroline Grigson, and Sandra Bond. 1998. “Dental Defects of Congenital Syphilis.” *American Journal of Physical Anthropology* 107 (1): 25–40. https://doi.org/10.1002/(SICI)1096-8644(199809)107:1<25::AID-AJPA3>3.0.CO;2-C.

Katzenberg, M. Anne, D. Ann Herring, and Shelley R. Saunders. 1996. “Weaning and Infant Mortality: Evaluating the Skeletal Evidence.” *American Journal of Physical Anthropology*

101 (S23): 177–99. https://doi.org/10.1002/(SICI)1096-8644(1996)23+<177::AID-AJPA7>3.0.CO;2-2.

Kay, James Phillips. 1832. *The Moral and Physical Condition of the Working Classes Employed in the Cotton Manufacture in Manchester*. London: James Ridgeway.

Kinaston, RL, A Willis, Justyna J. Miszkiewicz, M Tromp, and M Oxenham. 2019. "The Dentition: Development, Disturbance, Diet and Chemistry." In *Ortner's Identification of Pathological Condition in Human Skeletal Remains*, 749–97. London: Elsevier.

King, Charlotte L., Siân E. Halcrow, Andrew R. Millard, Darren R. Gröcke, Vivien G. Standen, Marco Portilla, and Bernardo T. Arriaza. 2018. "Let's Talk about Stress, Baby! Infant-Feeding Practices and Stress in the Ancient Atacama Desert, Northern Chile." *American Journal of Physical Anthropology* 166 (1): 139–55. https://doi.org/10.1002/ajpa.23411.

King, Charlotte L, Halle R Buckley, Peter Petchey, Rebecca L Kinaston, Andrew R Millard, Jana Zech, Patrick Roberts, Elizabeth Matisoo-Smith, Geoff M Nowell, and Darren R Gröcke. 2020. "A Multi-Isotope, Multi-Tissue Study of Colonial Origins and Diet in New Zealand." *American Journal of Physical Anthropology* 172 (4): 605–20.

King, Charlotte L, Anne Marie E Snoddy, Andrew R Millard, Darren R Gröcke, Vivien G Standen, Bernardo T Arriaza, and Siân E Halcrow. 2018. "A Multifaceted Approach towards Interpreting Early Life Experience and Infant Feeding Practices in the Ancient Atacama Desert, Northern Chile." *International Journal of Osteoarchaeology* 28 (5): 599–612. https://doi.org/10.1002/oa.2671.

Lang, John Dunmore. 1839. *On the Prospect Which New Zealand Affords for the Establishment of a British Colony, Or Four Letters, to The Right Hon. Earl Durham, Governor of the New Zealand Land Company Etc. on the Colonization of That Island, and of the Present*

Condition and Prospec. London.

Lewis, Mary E. 2007. *The Bioarchaeology of Children : Perspectives from Biological and Forensic Anthropology*. Cambridge, UK: Cambridge University Press.

Lovejoy, C. Owen, Richard S. Meindl, Thomas R. Pryzbeck, and Robert P. Mensforth. 1985. "Chronological Metamorphosis of the Auricular Surface of the Ilium: A New Method for the Determination of Adult Skeletal Age at Death." *American Journal of Physical Anthropology* 68 (1): 15–28. https://doi.org/10.1002/ajpa.1330680103.

Lukacs, John R., and Leah L. Largaespada. 2006. "Explaining Sex Differences in Dental Caries Prevalence: Saliva, Hormones, and 'Life-History' Etiologies." *American Journal of Human Biology* 18 (4): 540–55. https://doi.org/10.1002/ajhb.20530.

Martin, Melanie. 2018. "Mixed-Feeding in Humans: Evolution and Current Implications." In *Breastfeeding New Anthropological Approaches,* edited by Cecília Tomori, Aunchalee EL Palmquist, and EA Quinn, 140–54. New York, NY: Routledge.

Mays, Simon, Rebecca L Gowland, Siân E Halcrow, and Eileen Murphy. 2017. "Child Bioarchaeology: Perspectives on the Past 10 Years." *Childhood in the Past* 10 (1): 38–56. https://doi.org/10.1080/17585716.2017.1301066.

Millard, Andrew R. 2000. "A Model for the Effect of Weaning on Nitrogen Isotope Ratios in Humans." In *Perspectives in Amino Acid and Protein Geochemistry,* edited by GA Goodfriend, MJ Collins, M Fogel, SA Macko, and JF Wehmiller, 51–59. Oxford: Oxford University Press.

Moorrees, CFA, Elizabeth A. Fanning, and Edward E. Hunt. 1963a. "Formation and Resorption of Three Deciduous Teeth in Children." *American Journal of Physical Anthropology* 21 (2): 205–13. https://doi.org/10.1002/ajpa.1330210212.

Moorrees, CFA, Elizabeth A Fanning, and Edward E Hunt. 1963b. “Age Variation of Formation Stages for Ten Permanent Teeth.” *Journal of Dental Research* 42 (6): 1490–1502.

Moseley, Stephen. 2008. *The Chimney of the World: A History of Smoke Pollution in Victorian and Edwardian Manchester*. Abingdon, Oxon: Routledge.

Newman, Sophie L, and Rebecca L Gowland. 2017. “Dedicated Followers of Fashion? Bioarchaeological Perspectives on Socio-Economic Status, Inequality, and Health in Urban Children from the Industrial Revolution (18th-19th C), England.” *International Journal of Osteoarchaeology* 27 (2): 217–29. https://doi.org/10.1002/oa.2531.

Nitsch, Erika K., Louise T. Humphrey, and Robert E.M. Hedges. 2011. “Using Stable Isotope Analysis to Examine the Effect of Economic Change on Breastfeeding Practices in Spitalfields, London, UK.” *American Journal of Physical Anthropology* 146 (4): 619–28. https://doi.org/10.1002/ajpa.21623.

O’Connell, T.C., R.E.M. Hedges, M.A. Healey, and A.H.R.W. Simpson. 2001. “Isotopic Comparison of Hair, Nail and Bone: Modern Analyses.” *Journal of Archaeological Science* 28 (11): 1247–55. https://doi.org/10.1006/JASC.2001.0698.

Ogden, A.R., R. Pinhasi, and W.J. White. 2007. “Gross Enamel Hypoplasia in Molars from Subadults in a 16th–18th Century London Graveyard.” *American Journal of Physical Anthropology* 133 (3): 957–66. https://doi.org/10.1002/ajpa.20608.

Organization, World Health. 2009. *Infant and Young Children Feeding*. Geneva: World Health Organisation.

Ortner, Donald J. 2003. *Identification of Pathological Conditions in Human Skeletal Remains. Identification of Pathological Conditions in Human Skeletal Remains*. Elsevier Inc. https://doi.org/10.1016/B978-0-12-528628-2.X5037-6.

Perkin, J. 1993. *Victorian Women*. London: John Murray.

Petchey, Peter, Hallie R Buckley, Rebecca Kinaston, and Baylee Smith. 2017. "A Nineteenth Century Settlers' Graveyard: Preliminary Report on the Excavation of St. John's Cemetery, Back Road, Milton, Otago." *Archaeology in New Zealand* 60 (1): 21–31.

Phenice, T. W. 1969. "A Newly Developed Visual Method of Sexing the Os Pubis." *American Journal of Physical Anthropology* 30 (2): 297–301. https://doi.org/10.1002/ajpa.1330300214.

Porter, Frances, and Charlotte (Eds.) MacDonald. 1996. *"My Hand Will Write What My Heart Dictates". The Unsettled Lives of Women in Nineteenth-Century New Zealand as Revealed to Sisters, Family and Friends.* Auckland, NZ: Auckland University Press.

Purvis, Richard J., G. S. Mackay, Forrester Cockburn, W. J.Mc K. Barrie, E. M. Wilkinson, Neville R. Belton, and John O. Forfar. 1973. "Enamel Hypoplasia of the Teeth Associated with Neonatal Tetany: A Manifestation of Maternal Vitamin-D Deficiency." *The Lancet* 302 (7833): 811–14. https://doi.org/10.1016/S0140-6736(73)90857-X.

Reed, Susan G., Delia Voronca, Jeanette S. Wingate, Mallika Murali, Andrew B. Lawson, Thomas C. Hulsey, Myla D. Ebeling, Bruce W. Hollis, and Carol L. Wagner. 2017. "Prenatal Vitamin D and Enamel Hypoplasia in Human Primary Maxillary Central Incisors: A Pilot Study." *Pediatric Dental Journal* 27 (1): 21–28. https://doi.org/10.1016/j.pdj.2016.08.001.

Reitsema, Laurie J. 2013. "Beyond Diet Reconstruction: Stable Isotope Applications to Human Physiology, Health, and Nutrition." *American Journal of Human Biology* 25 (4): 445–56. https://doi.org/10.1002/ajhb.22398.

Sadler, TW. 2015. *Langman's Medical Embryology*. Philadelphia: Lippincott Williams &

Wilkins.

Schaefer, M, S Black, and L Scheuer. 2009. *Juvenile Osteology: A Laboratory and Field Manual.* London: Academic Press.

Sellen, Daniel W., and Diana B. Smay. 2001. "Relationship between Subsistence and Age at Weaning in 'Preindustrial' Societies." *Human Nature* 12 (1): 47–87. https://doi.org/10.1007/s12110-001-1013-y.

Skinner, Mark F., Antonia T. Rodrigues, and Chris Byra. 2014. "Developing a Pig Model for Crypt Fenestration-Induced Localized Hypoplastic Enamel Defects in Humans." *American Journal of Physical Anthropology* 154 (2): 239–50. https://doi.org/10.1002/ajpa.22497.

Skinner, Mark F, and J. T. W. Hung. 1989. "Social and Biological Correlates of Localized Enamel Hypoplasia of the Human Deciduous Canine Tooth." *American Journal of Physical Anthropology* 79 (2): 159–75. https://doi.org/10.1002/ajpa.1330790204.

Skinner, Mark F, and E Newell. 2000. "A Re-Evaluation of Localized Hypoplasia of the Primary Canine as a Marker of Craniofacial Osteopenia in European Upper Paleolithic Infants." *Acta Universitatis Carolinae. Medica* 41 (1–4): 41–58.

Smith, B. Holly. 1984. "Patterns of Molar Wear in Hunter–Gatherers and Agriculturalists." *American Journal of Physical Anthropology* 63 (1): 39–56. https://doi.org/10.1002/ajpa.1330630107.

Smith, Tanya M. 2018. *The Tales Teeth Tell: Development, Evolution, Behavior*. Cambridge, MA: The MIT Press.

Snoddy, Anne Marie E, Hallie R Buckley, and Siân E Halcrow. 2016. "More than Metabolic: Considering the Broader Paleoepidemiological Impact of Vitamin D Deficiency in Bioarchaeology." *American Journal of Physical Anthropology* 160 (2): 183–96.

https://doi.org/10.1002/ajpa.22968.

Snoddy, Anne Marie E, Hallie R Buckley, Charlotte L King, Rebecca L Kinaston, Geoff Nowell, Darren R Gröcke, Warwick J Duncan, and Peter Petchey. 2020. "'Captain of All These Men of Death': An Integrated Case-Study of Tuberculosis in 19th Century Otago, New Zealand." *Bioarchaeology International* 3 (4): 217–37.

Stojanowski, Christopher M., and Charisse L. Carver. 2011. "Inference of Emergent Cattle Pastoralism in the Southern Sahara Desert Based on Localized Hypoplasia of the Primary Canine." *International Journal of Paleopathology* 1 (2): 89–97. https://doi.org/10.1016/j.ijpp.2011.06.001.

Stuart-Macadam, Patricia, and Katherine A. Dettwyler. 1995. *Breastfeeding : Biocultural Perspectives*. New York: Routledge.

Sumpter, Denis J, and Jack J Lewis. 1949. *Faith and Toil. The Story of Tokomairiro, Otago*. Dunedin: Otago Centennial Historical Publications.

Swainson, William. 1840. *Observations on the Climate of New Zealand: Principally with Reference to Its Sanative Character*. London: Smith, Elder and Co.

Tomori, Cecilia, Auchalee EL Palmquist, and EA Quinn. 2018. *Breastfeeding, New Anthropological Approaches*. Abingdon, Oxon: Routledge.

Towle, Ian, and Joel D. Irish. 2020. "Recording and Interpreting Enamel Hypoplasia in Samples from Archaeological and Palaeoanthropological Contexts." *Journal of Archaeological Science* 114 (February): 105077. https://doi.org/10.1016/j.jas.2020.105077.

Tsutaya, Takumi. 2020. "Blurred Time Resolution of Tooth Dentin Serial Sections." *American Journal of Physical Anthropology*, September, ajpa.24113. https://doi.org/10.1002/ajpa.24113.

Tsutaya, Takumi, and Minoru Yoneda. 2015. “Reconstruction of Breastfeeding and Weaning Practices Using Stable Isotope and Trace Element Analyses: A Review.” *American Journal of Physical Anthropology* 156 (February): 2–21. https://doi.org/10.1002/ajpa.22657.

Walter, Brittany S., Sharon N. DeWitte, Tosha Dupras, and Julia Beaumont. 2020. “Assessment of Nutritional Stress in Famine Burials Using Stable Isotope Analysis.” *American Journal of Physical Anthropology,* April. https://doi.org/10.1002/ajpa.24054.

Waterland, Robert A., and Karin B. Michels. 2007. “Epigenetic Epidemiology of the Developmental Origins Hypothesis.” *Annual Review of Nutrition* 27 (1): 363–88. https://doi.org/10.1146/annurev.nutr.27.061406.093705.

Waters-Rist, Andrea L, and Mary Anne Katzenberg. 2009. “The Effect of Growth on Stable Nitrogen Isotope Ratios in Subadult Bone Collagen.” *International Journal of Osteoarchaeology* 20 (May 2008): 172–91. https://doi.org/10.1002/oa.

Wheeler, Benjamin, Barry Taylor, Michel de Lange, Michelle Harper, Shirley Jones, Adel Mekhail, and Lisa Houghton. 2018. “A Longitudinal Study of 25-Hydroxy Vitamin D and Parathyroid Hormone Status throughout Pregnancy and Exclusive Lactation in New Zealand Mothers and Their Infants at 45° S.” *Nutrients* 10 (1): 86. https://doi.org/10.3390/nu10010086.

WHO. 2007. “Planning Guide for National Implementation of the Global Strategy for Infant and Young Child Feeding,” 1–46.

Wickes, Ian G. 1953. “A History of Infant Feeding Part III: Eighteenth and Nineteenth Century Writers.” *Archives of Disease in Childhood* 28 (140): 332–40.

Witness, Otago. 1889. “Advertisements.” *Otago Witness, Issue 1950, 4 April 1889*, 1889.

Wohl, Anthony S. 2009. *The Eternal Slum: Housing and Social Policy in Victorian London*.

New Brunswick, NJ: Transaction Publishers.

Wood, James W., George R. Milner, Henry C. Harpending, Kenneth M. Weiss, Mark N. Cohen, Leslie E. Eisenberg, Dale L. Hutchinson, et al. 1992. “The Osteological Paradox: Problems of Inferring Prehistoric Health from Skeletal Samples [and Comments and Reply].” *Current Anthropology* 33 (4): 343–70. https://doi.org/10.1086/204084.

Figure 1. Map showing location of the study site (grey circle) with reference to major New Zealand cities (black circles), from King et al. (2020).

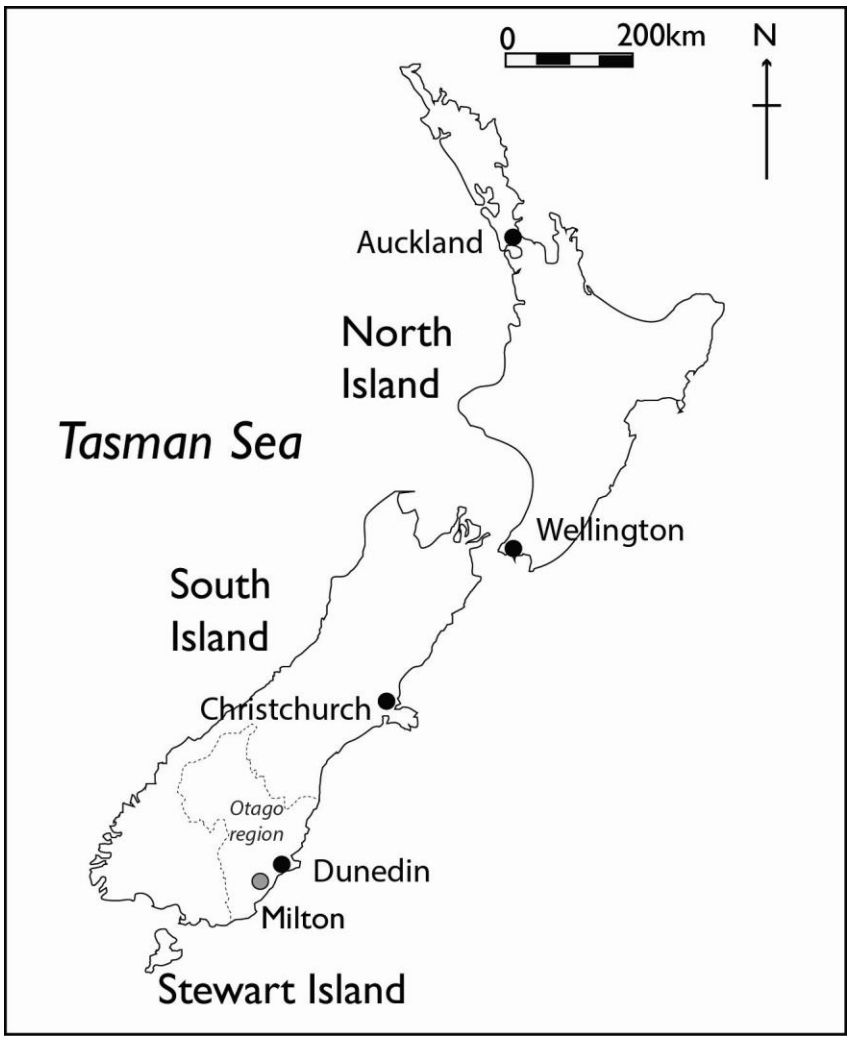

Figure 2. Individual incremental isotopic plots showing early childhood carbon and nitrogen isotope values for the infants and children at the site. Note that B8 values derive from dentine, while all other plots are hair values.

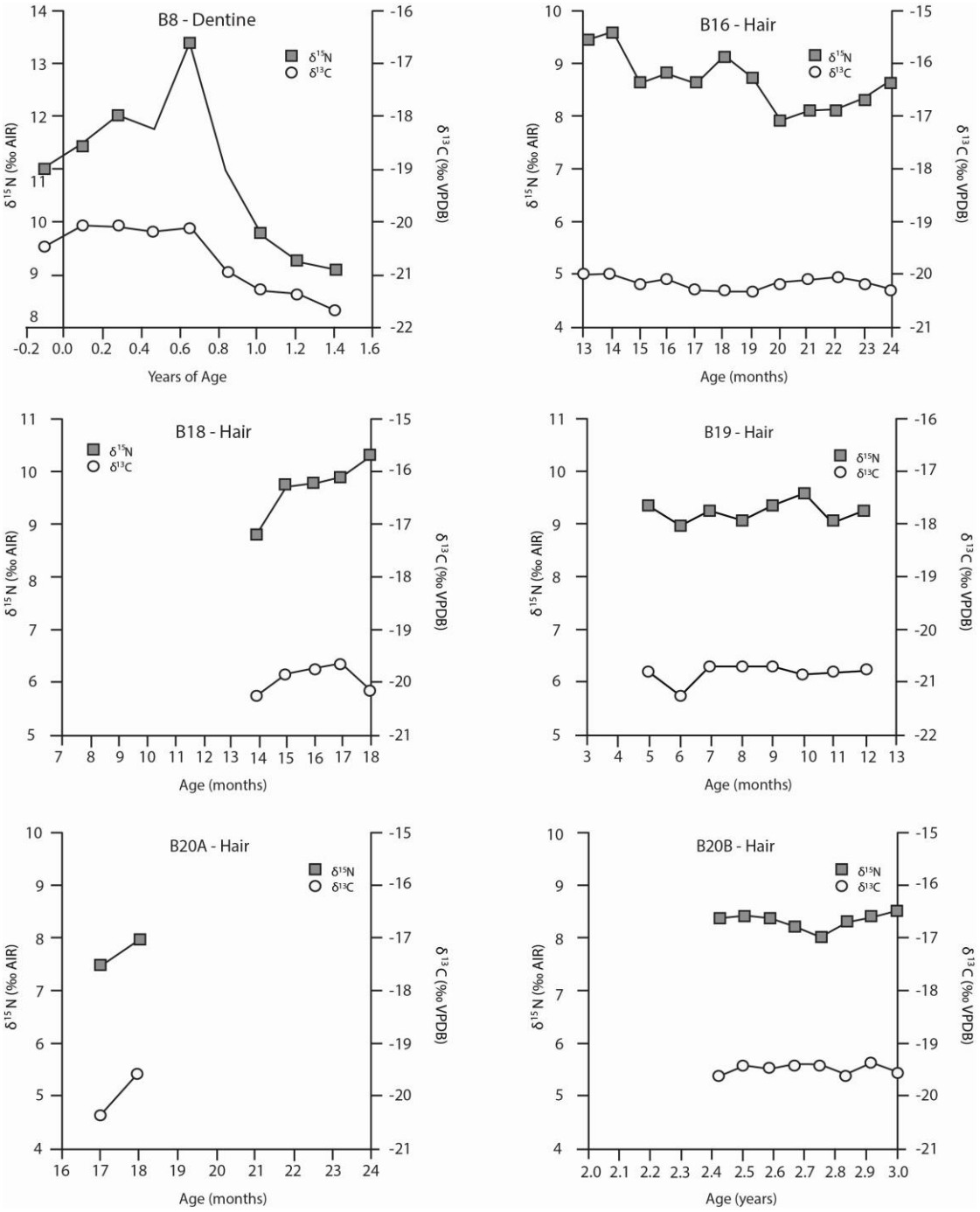

Figure 3. All isotopic profiles up to 5 years of age (60 months), δ^{15}N values = left graph, δ^{13}C values = right graph. All dentine values have been corrected for the hair-collagen offset. Shaded rectangles represent mean adult bone collagen values corrected for the hair-collagen offset (δ^{15}N = 10.8, δ^{13}C =-19.6) ± 1SD .

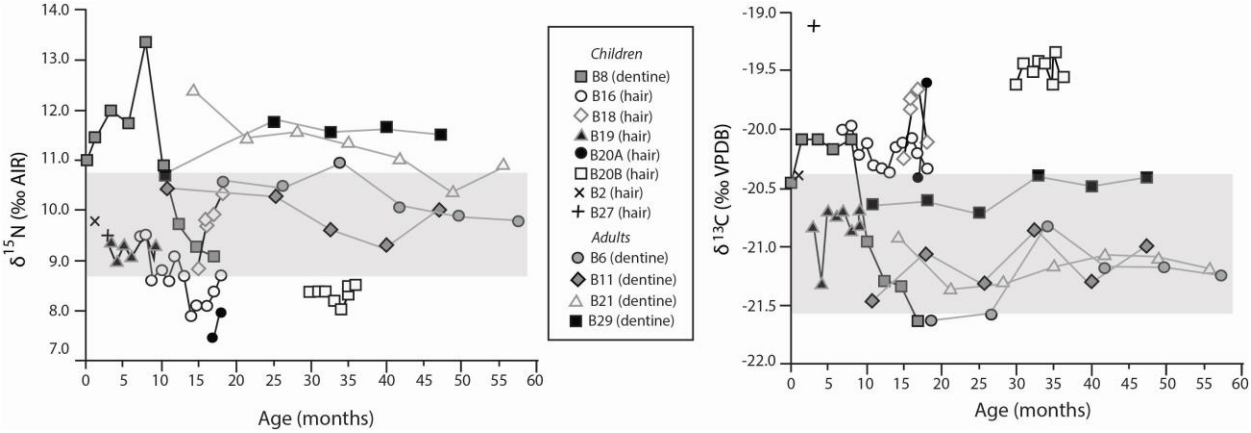

Figure 4. Example of LEH defects, here shown with white arrows on the dentition of B11

Figure 5. Burial 27, deciduous canines. Squiggly arrow points to the pitted LEH in the cervical region of the right and left maxillary canines. Arrow heads denote the LHPC of the right and left mandibular canines.

Figure 6. Central maxillary incisors of B27, with arrows showing hypomineralised bands with circular carious lesions

Figure 7. Deciduous mandibular central incisors of B27 showing central notching defects in the crowns.

Disclosure Statement

No potential competing interest was reported by the authors

Supplementary methods

Isotopic analysis - dentition

Teeth were half sectioned and enamel removed from one half using a diamond cutting wheel and dental drill. Dentine was then demineralised in 0.5M HCl until able to be bent. The dentine collagen pseudomorph was rinsed in DI H2O before sectioning into 1mm increments using a sterilised surgical scalpel and metal ruler. Increments were placed into pre-weighed microcentrifuge tubes, 1ml pH 3 HCl solution was then added and increments were gelatinised at 70^{o}C for 48 hrs. Once gelatinised, increments were frozen at -30^{o}C before lyophilising.

Isotopic analysis - hair

Approximately 10 strands of hair were separated from any poorly- preserved remains of skulls using bamboo tools to avoid damage. These were gently pre-cleaned using DI H2O, then sectioned into 1cm increments and placed in microcentrifuge tubes. Each increment was then solvent washed in 2:1 methanol: chloroform solution. This solution was added to each microtube, lids were closed and the samples sonicated for 20 minutes. The solvent was then pipetted out, and replaced with clean solvent. This process was repeated until the solvent remained clear after sonication.

Dental pathology recording

All deciduous and permanent teeth were examined under incandescent and fluorescent light using 1.75x magnification. The crowns of the deciduous and permanent teeth were visually assessed and a soft-tipped wooden tool was used to confirm defects. All available tooth crowns were assessed if they were fully formed, even if the tooth may not have erupted, or was erupting, at the time of death. Teeth were not assessed for EH if they were damaged postmortem, had massive carries that destroyed the crown or had substantial calculus deposits that obscured the crown surfaces. Linear enamel hypoplasia was identified as present by a continuous line or groove, or row of pits, across at least 50% of the crown's surface. Teeth were also assessed for plane-form and cuspal enamel hypoplasia, circular caries and LHPC as outlined in

Ogden et al. (2007) and Cook and Buikstra (Cook and Buikstra, 1979) and Halcrow and Tayles (2008), respectively.

Previous studies have used the criteria that at least two anterior teeth from the same individual must display a linear enamel hypoplasia that formed during a similar chronological time to differentiate systemic stress from 'localised' stress events that could be caused by trauma to the tooth (e.g. King et al., 2005; Ward et al., 2020). However, because of the multiple aetiologies of EH and the small sample size of the SJM assemblage, we report all instances of observed EH, including plane-form defects and localised hypoplasia of the primary canine (LHPC). For LEH, we note if there were only a limited number of anterior teeth available for analysis. In the adult SJM assemblage, all individuals had evidence for antemortem tooth loss (AMTL) of the posterior teeth. Overall, 35 of a total of 93 (37.6%) observable posterior tooth sockets showed that the tooth had been lost before death (Buckley et al., in review) and this loss may have biased recording of cuspal and plane-form defects of this tooth type.

		Overall teeth			Anterior teeth (incisors and canines)			
Sex	Age	A	O	%	A	O	%	A/N (%)
Male*	Mid	18	57	31.6	18	25	72.0	**4/4 (100%)**
Female	Mid	1	23	4.3	1	13	7.7	**1/2 (50%)**
Unknown	Unknown	8	10	80	7	7	100.0	**1/1 (100%)**
Total		27	90	30.0	26	45	57.8	**6/7 (85.7%)**

Supplementary Table 1. Prevalence of LEH in the overall dentition and anterior dentition in the adult SJM assemblage. *Four teeth with massive caries could not be recorded for LEH. O-total teeth observed. A- total teeth affected by LEH. A/N (%)- Total affected individuals/total number of individuals.

Individual	Tissue sampled	Increment #	Age represented	%N	d15N	%C	d13C	C:Natomic
SJM B2	Hair	1	Close to birth	12.15	9.79	39.30	-20.39	3.8
SJM B8	Dentine	1	-0.1 years	14.46	11.0	42.81	-20.5	3.5
		2	0.09 years	15.06	11.5	42.83	-20.1	3.3
		3	0.28 years	18.65	12.0	51.37	-20.1	3.2
		4	0.47 years	10.31	11.8	28.93	-20.2	3.3
		5	0.66 years	14.11	13.4	41.75	-20.1	3.5
		6	0.84 years	14.23	10.9	41.75	-20.9	3.4
		7	1.03 years	13.73	9.7	41.41	-21.3	3.5
		8	1.21 years	13.52	9.2	40.05	-21.3	3.5
		9	1.40 years	12.42	9.1	41.31	-21.6	3.9
SJM 16	Hair	1	24 months	14.05	8.66	47.21	-20.31	3.9
		2	23 months	13.31	8.35	41.55	-20.20	3.6
		3	22 months	10.66	8.11	34.67	-20.09	3.8
		4	21 months	13.33	8.09	42.48	-20.11	3.7
		5	20 months	13.90	7.91	43.87	-20.16	3.7
		6	19 months	13.68	8.67	46.65	-20.36	4.0
		7	18 months	13.18	9.12	45.75	-20.34	4.0
		8	17 months	13.74	8.59	42.61	-20.29	3.6
		9	16 months	13.78	8.80	45.88	-20.11	3.9
		10	15 months	13.35	8.63	40.93	-20.21	3.6
		11	14 months	13.27	9.59	48.40	-19.98	4.3
		12	13 months	13.21	9.49	45.68	-20.01	4.0

SJM 18	Hair	1	18 months	13.04	10.28	42.55	-20.12	3.8
		2	17 months	13.49	9.92	41.85	-19.67	3.6
		3	16 months	14.19	9.74	42.30	-19.80	3.5
SJM B19	Hair	1	12 months	13.74	9.17	44.10	-20.76	3.7
		2	11 months	13.29	9.59	44.28	-20.85	3.9
		3	10 months	13.64	9.36	39.70	-20.71	3.4
		4	9 months	13.33	9.08	40.38	-20.73	3.5
		5	8 months	12.97	9.32	42.26	-20.70	3.8
		6	7 months	12.93	8.98	39.10	-21.29	3.5
		7	6 months	12.82	9.36	44.80	-20.85	4.1
B20a	Hair	1	18 months	13.35	7.95	40.12	-19.60	3.5
		2	17 months	12.64	7.49	41.54	-20.41	3.8
SJM B20b	Hair	1	3 years	12.79	8.50	41.00	-19.58	3.7
		2	2.9 years	13.22	8.37	43.20	-19.49	3.8
		3	2.83 years	11.90	8.04	38.41	-19.43	3.8
		4	2.75 years	13.42	8.20	42.12	-19.43	3.7
		5	2.67 years	12.06	8.38	38.53	-19.49	3.7
		6	2.58 years	12.89	8.39	36.85	-19.45	3.3
		7	2.5 years	12.54	8.38	35.73	-19.63	3.3
SJM B27	Hair	1	15 months	13.53	9.48	40.54	-19.12	3.5

Supplementary Table 1: All isotopic data associated with the infants/children from St. John's Milton.