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Poundbury Camp in Context – a new perspective on the lives of children from urban and rural Roman England

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ABSTRACT

Objectives

Our current understanding of child morbidity in Roman England is dominated by studies of single sites/regions. Much of the data are derived from 3rd-5th century AD Poundbury Camp, Dorchester, Dorset, considered an unusual site due to high levels of non-adult morbidity. We have little understanding of children in rural areas, and whether Poundbury Camp was representative of Romano-British childhood.

Materials and Methods

The study provides the first large scale analysis of child health in urban and rural Roman England, adding to the previously published intra-site analysis of non-adult palaeopathology at Poundbury Camp. Age-at-death and pathology prevalence rates were reassessed for 952 non-adults (0-17 years) from five major urban, six minor urban and four rural sites (1st-5th century AD). The data were compared to the results from 364 non-adults from Poundbury Camp.

Results

Rural sites demonstrated higher levels of infant burials, and greater prevalence of cribra orbitalia in the 1.1-2.5 years (TPR 64.3%), and 6.6-10.5 year cohorts (TPR 66.7%). Endocranial lesions were more frequent in the minor urban sample (TPR 15.9%). Three new cases of tuberculosis were identified in urban contexts. Vitamin D deficiency was most prevalent at Poundbury Camp (CPR 18.8%), vitamin C deficiency was identified more frequently in rural settlements (CPR 5.9%).

Discussion

The Poundbury Camp data on morbidity and mortality are not representative of patterns in Roman England and other major urban sites. Rural children suffered from a distinct pattern of diseases of deprivation, prompting reconsideration of how Romano-British land management affected those at the bottom of the social hierarchy.
The study of child skeletal remains in bioarchaeology has become an increasingly popular way to measure the impact of environmental stress in the past (Perry, 2005; Lewis, 2007; Halcrow and Tayles, 2008; 2011; Gowland, 2015). In England, while more comprehensive studies of children living in medieval and post-medieval societies emerge (Lewis, 2016; Newman and Gowland, 2016), our understanding of child health during the Roman period, a time of major social upheaval, is still little understood. The studies that exist are coloured by reliance on a limited number of skeletal assemblages (Redfern, 2007; 2008; Lewis, 2010; 2011; 2012; Redfern et al., 2012). In contrast to later periods, documentary evidence relating specifically to Roman Britain is scarce, and any historical evidence for weaning, child rearing, education, health, and employment of children comes mainly from the Mediterranean. Arguments surrounding the extent to which Roman culture was adopted by native populations are still on-going, with ideas of ‘Romanisation’, whether considered imperially-imposed acculturation or native emulation, gradually being replaced by more complex models such as creolisation (Webster, 2001; Mattingly, 2006).

Further, extensive studies that integrate skeletal evidence with archaeological and environmental data are limited in Roman archaeology due to cremation as the predominant burial rite during the 1st and 2nd centuries AD. The shift in funerary rites from mostly cremation to inhumation during the 3rd century AD means that we are often, but not exclusively, limited to the later periods of Roman rule (ca. AD 250-410) for osteological studies. Although the majority of our skeletal data are derived from urban sites, many questions still remain about the extent of urbanisation, and the impact it had on the health of the native population. Some argue that the Romans brought about improvements in sanitation and living conditions (Mattingly, 2006: 323), with planned Roman cities boasting marbled surfaces and flowing water, providing extensive facilities for the comfort and health of the inhabitants (Morley, 2005). Diametrically opposed is the view that urbanisation widened the divide between the rich and the poor, with many suffering hardships of poverty, social unrest, and subservience to the conquering population. Redfern and Roberts (2005) concluded that the pathology and mortality levels in seven urban sites in Roman Britain pointed to unsanitary and squalid living conditions. They cite environmental evidence from Poultry Lane in London as indicating household refuse built-up in yards, wells were contaminated by the close proximity of pigs and chickens, and outdoor latrines were dug into kitchen spaces. These conditions, in combination with an increased migrant population and the introduction of pubic bath houses, led to a decline in the sanitary conditions in these crowded, built-up areas in England (Redfern, 2008). Studies comparing the prevalence of pathology in urban
and rural settlements in late Roman England have demonstrated that living in the countryside also negatively affected its residents (Pitts and Griffin, 2012; Redfern et al., 2015). While important in their contribution, this evidence is limited in its scope by reliance on previously published data, the lumping of non-adults into a single age bracket (Redfern and Roberts, 2005; Pitts and Griffin, 2012), a focus only on adult remains (Roberts and Cox, 2003; Griffin and Pitts, 2011) or the study if a single site or region (Lewis, 2011; Redfern et al., 2015).

The children excavated from the late Romano-British cemetery of Poundbury Camp, Dorset (3-5th century AD) represent the largest single sample of child skeletons from this period (n=364). They were first analysed in 1993 when some unusual cases of infection and trauma were identified (Molleson and Cox, 1988; Farwell and Molleson, 1993; Molleson, 1989; 1992a,b). As is the case with many major Romano-British sites, the Poundbury Camp osteological report pre-dated major advances in child palaeopathology. Re-analysis of the child remains revealed new evidence for rickets, scurvy and trauma (Lewis, 2010), as well as identifying the first cases of non-adult tuberculosis in Roman England (Lewis 2010; 2011). Thalassaemia was also identified for the first time in the palaeopathological record, through characteristic rib lesions, uncovering ill-health in children born to migrant parents (Lewis, 2012). The prevalence of pathology at this site was surprising in its similarity to that seen in post-medieval samples, suggesting the severity of the impact of urbanisation, cultural practices and perhaps conquered existence was radically affecting child health and survival. Lewis (2011) stated that without comparatively analysed child data from other urban and rural sites, the extent to which this pattern reflected the experience of children under Roman rule in England could not be assessed. There are reasons to believe that Poundbury Camp was exceptional in its extent of pathology. For example, the inhabitants of the civitas capital of Durnovaria buried their dead at Poundbury Camp in a managed ‘Christian-style’, witness to a ‘Romanised’ elite at the site (Sparey-Green, 2004). Incoming Christian beliefs may have profoundly shaped daily conduct at the site, dictated dietary practices for women and children, and impacted on their nutritional status and overall well-being (Cool, 2006; Lewis, 2010). In addition, the foundation of churches would have dictated land distribution and management, as well as the lifestyles of the local population throughout western areas of Roman Britain (Jones, 1982; Frend, 1992; Faith, 1997:16-8; Whittaker and Garnsey, 1997:301; Esmonde Cleary, 2004). This study aims to rectify this deficiency in our knowledge about the true nature of the lifestyle and lived reality of the children of Roman England by presenting new data on the analysis of 953 individuals from 15 urban and rural sites in Roman England. Through this unique dataset, we will explore the extent to which the
late Romano-British villa economy affected the peasantry, and whether the urban environment was in fact as taxing on child health as the Poundbury Camp data would suggest.

MATERIALS AND METHODS

Demographic and palaeopathological data for the 364 Poundbury Camp (3-5th century) non-adults were compared to 953 non-adults examined from 15 urban and rural early and late Romano-British sites from across England, dating from the 1st-5th century AD (Table 1, Fig.1). In accordance with Pitts and Griffin (2012) sites were divided into major urban (coloniae, civitates), minor urban (nucleated/small towns) and rural settlements (villages, farmsteads, villa estates). ‘Major urban’ sites were defined as large legal and administrative planned settlements, including a grid street layout, public buildings, a forum and a spiritual focus (Wacher, 1974; Burnham and Wacher, 1990; Millett, 1990; Laurence et al., 2011). The characteristics of ‘minor urban’ (un-)walled settlements spark ongoing debates. It is generally agreed however that these display some urban aspects, such as evidence for town planning and a market to facilitate local trade, albeit on a smaller scale (Hingley, 1989; Burnham, 1993; 1995; Millett, 1995; Wilson, 2011). Rural sites were defined as undefended farmsteads, villages or villa estates with a predominantly agricultural focus (McCarthy, 2013). Depending on their location, rural sites would have exhibited varying economic and socio-cultural dependence on nearby towns (Laurence et al., 2011; White, 2014). However, their agricultural focus still rendered them as rural in character and urbanisation of the countryside as seen in Italy was not apparent in Roman Britain (Laurence, 2011). Current models on life in the Romano-British countryside consider villa economies as estates managed by landowners, with a peasant population that cultivates the land as tenants or freeholders, living either on the estate itself, or the surrounding villages (Taylor, 2001; Mattingly, 2006; McCarthy, 2013; Breeze, 2014). The terms ‘urban’ and ‘rural’ describe a type of settlement rather than geographic location, however there is growing awareness of the difficulty in classifying the type of Romano-British settlements (Mattingly, 1997; Millett, 1999; Burnham et al., 2001; Millett, 2001; Pearce, 2008; Rogers, 2011). Not all urban cemeteries would have contained those living and dying exclusively within these large settlements, as many individuals may have been derived from the “urban periphery” (Goodman, 2007:1-2), or represent rural migrants (Griffin and Pitts, 2012; Redfern et al., 2015). [Figure 1 here; Table 1 here].
Age-at-death was estimated using Moorrees et al.’s (1963a,b) standards for deciduous and permanent tooth formation using Smith’s (1991) tabulation. When the dentition was absent, age was assigned based on maximum diaphyseal length and epiphyseal fusion (Scheuer and Black, 2000). Perinatal ageing used Scheuer et al.’s (1980) diaphyseal length regression formulae which was considered the most appropriate standard as they are derived from English neonates. Non-adults were then assigned to one of seven age groups: perinate (<42 weeks gestation), 0.0-1.0 years, 1.1-2.5 years, 2.6-6.5 years, 6.6-10.5 years, 10.6-14.5 years and 14.6-17.0 years. The cut-off point of 17.0 years was determined by the completion of the root of the third molar (Rc), providing a mean age of 16.9 years old (Moorrees et al., 1963a). Age groups correspond with important developmental milestones in childhood such as infancy or adolescence, allow for direct comparison with the Poundbury Camp data, and limit observer bias.

In order to assess levels of stress caused by infections of a non-specific nature and/or localised trauma, sub-periosteal new bone formation and endocranial lesions were recorded. Sub-periosteal new bone formation was recorded as healed or active in accordance with Ortner (2003). Lesions were evaluated in consideration with any other pathological lesions present in any one individual to prevent over-recording (Lewis and Roberts, 1997; Weston, 2008). As we are yet to be able to differentiate a traumatic or infectious origin of sub-periosteal new bone formation from healthy regular bone growth in the youngest individuals (Lewis, 2007), perinates and infants were removed from analysis for new bone formation. Endocranial lesions were recorded according to Lewis (2004). Fine deposits of fibre bone around the cruciate eminence of the occipital bone were discounted when seen in the infants as they may result from normal cranial development (Lewis, 2004). The overall disease state of the individual was taken into account, as endocranial lesions that were observed in diagnoses for tuberculosis, and vitamin D or C deficiencies, form part of a wider systemic infectious or metabolic response.

As evidence for non-adult tuberculosis (TB) in the Roman period has only recently been identified (Lewis, 2012; Clough and Boyle, 2010), special attention was paid to the presence or absence of this condition in the re-analysed material. Tuberculosis was diagnosed in the presence of lytic lesions in the spine in combination with visceral rib lesions (Pfeiffer, 1984; 1991; Roberts and Buikstra, 2003), supported by the presence of widespread new bone formation (Santos and Roberts, 2001). Pott’s disease (spinal collapse) was considered pathognomonic (Resnick and Kransdorf, 2005:758-63).
Acquired haemopoietic conditions such as iron deficiency anaemia were assessed through the presence of cribra orbitalia and porotic hyperostosis, scored according to Stuart-Macadam (1991). Macroscopic assessment of cribra orbitalia is influenced by preservation and the porous nature of non-adult bone which may mimic or obscure subtle lesions. Therefore, Grades 1 (capillary-like impressions), and 2 (scattered fine foramina) were discounted to prevent over-recording. Given recent research into the aetiology of cribra orbitalia, lesions are interpreted as a general indicator of poor nutritional status, as the result of a lack of fresh vegetables and meat, a high pathogen load, parasitic infections, or diarrhoeal disease (Holland and O’Brien, 1997; McIlvaine, 2013; Mahmud et al., 2013). Healed lesions were distinguished from active lesions following Mensforth and colleagues (1978). In addition, evidence for the genetic anaemia thalassaemia, was assessed based on the presence of costal osteomas or ‘rib-within-a-rib’ (Lawson et al., 1981a,b), osteopenia and generalised cortical thickening, in addition to cribra orbitalia and porotic hyperostosis (Ortner, 2003:364-6, Tyler et al., 2006, Lagia et al., 2007; Lewis, 2011).

Vitamin D deficiency (rickets and osteomalacia) was identified following the criteria of Ortner and Mays (1998), Ortner (2003) and Brickley and Ives (2008). The presence of rickets in archaeological populations allows us to make inferences on dress, childcare practices, climate and environmental pollution (Lewis, 2002:55; 2010; Mays et al., 2006; Brickley and Ives, 2008: 263; Pettifor, 2014). More recently, Snoddy et al. (2016) considered the extra-skeletal effects of vitamin D deficiency, which increase the risk for autoimmune and infectious diseases, demonstrating that the effects of rickets within a population extend beyond brittle bones. Vitamin C deficiency (scurvy) was scored according to Brickley and Ives (2006) and the ‘Ortner criteria’ for lesions on the skull and metaphyseal areas of the long bones (Ortner and Ericksen, 1997; Ortner et al., 1999; 2001; Crandall and Klaus, 2014; Stark, 2014). Stark (2014) describes the presence of scurvy as a vehicle for exploring food insecurity, preferential feeding and subsistence economy. Co-occurrence of metabolic disease is likely, and non-adults may suffer from both vitamin C and D deficiency, or one of the former coupled with a haematopoietic condition (Brickley and Ives, 2008). However, since vitamin D and C interaction is inhibitory, but the haemorrhaging process unaffected, scurvy is likely to mask the expression of vitamin D deficiency (Schattmann et al., 2016). In order to differentiate co-occurrence, complete skeletons are necessary which the archaeological record cannot always provide. It was decided that individuals that exhibited inconclusive lesions attributable to either or both metabolic diseases were grouped to avoid skewing of the metabolic disease data. Discussion of individuals with potential co-occurrence is not included.
here. All individuals with a diagnosis of trauma, metabolic disease, or tuberculosis were sought to be examined radiographically, which was not possible for the Cannington and Trentholme Drive archives.

In order for a systemic pathological condition to be considered absent and an individual to be included in the true and crude prevalence rate calculations, the tibiae, cranial vault and thoracic spine needed to be observable. Differences between the four groups were assessed using the 4x2 chi-square statistic at 99.5% confidence (p<0.005) when percentages demonstrated big differences between the groups. Differences in the frequency of skeletal pathologies and age-at-death across the site types were used to assess the association between lesion presence or absence and survival into later childhood. Young non-adult, and particularly infant bone has complex properties which include rapid remodelling and masking of pathologies, and insufficient time for a disease to act on the skeleton before death. Rather than comparing infants to older non-adults with all skeletal pathologies merged, a broader age group of 0-5 years was used to buffer against these limitations, comparing lesion frequency in young non-adults aged 0-5 years to older non-adults aged 6-17 years. Differences in the frequencies of pathology according to age-at-death were assessed using Fisher’s exact test set at 99.5% confidence (p<0.005).

RESULTS

Age-at-death

The age-at-death distribution in the Romano-British samples is provided in Table 2 (Fig 2). The number of perinates was low at Poundbury Camp compared to other sites, but did not differ statistically ($\chi^2=4.78$, d.f.=3). However, the number of infant remains (under 1 year old) was significantly higher for rural sites at 31.3% ($\chi^2=17.32$, p<0.005, d.f.=3). The rural sample has high numbers of intramural infant burials. All 48 burials from Catshore and Bradley Hill in Somerset were recovered from within the settlement boundaries, resulting in high numbers of intramural infant burials in the rural cohort. There is a dip in the number of 6.6-10.5-year olds in the rural sample to 5.2%, although the rate is not significantly lower than at Poundbury Camp and other urban settlements ($\chi^2=7.76$, d.f.=3). Interestingly, the age-at-death distribution at Poundbury Camp is different to the rest of the urban sites in the study and does not directly compare to other major urban sites. [Table 2 here; Figure 2 here].

Pathology and age-at-death
The major urban, minor urban and rural groups show greater lesion frequency in older non-adults, whereas Poundbury Camp is characterised by an inverse pattern. Statistically significant differences in the distribution of skeletal pathologies and age-at-death were found in the non-adults from minor urban ($X^2=16.94, p<0.001, d.f.=2$) and rural sites ($X^2=14.69, p<0.001, d.f.=2$) (Table 3). Young non-adults as non-survivors exhibited significantly lower lesion frequencies than older non-adults at these sites. [Table 3 here].

**Indicators of non-specific stress**

The prevalence of endocranial lesions was significantly higher in the minor urban cohort at 15.9% compared to Poundbury Camp and all other sites types ($X^2=21.96, p<0.001$, d.f.=3) (Table 4). There was no statistical difference in the distribution of sub-periosteal new bone formation between all four samples ($X^2=7.2, d.f.=3$). Rural non-adults exhibited equal frequencies of active and healed lesions at 2.1%, whereas active new bone formation was more frequently observed at Poundbury Camp (2.1%, healed 0.7%) and both major urban (active 4.9%, healed 3.8%) and minor urban sites (active 3.9%, healed 2.0%) (Table 5). [Table 4 and 5 here].

**Tuberculosis**

At Poundbury Camp, 165 individuals were assessed for tuberculosis, with a total of 10 (6.1%) suspected cases. These include three cases (1.8%) of pulmonary infection, five individuals (3.0%) with lesions possibly indicative of tuberculosis and with pulmonary disease as a differential diagnosis, and two children (1.2%) aged 10.6-14.5 years old with lesions strongly suggestive of tuberculosis (Table 6). Six new cases of probable and possible tuberculosis were identified in the current study (Table 7), with all three probable cases from major and minor urban sites (Figs. 3-5). [Figure 3, 4, 5 here]. There are still only few cases of probable and possible non-adult tuberculosis to make meaningful statistical comparisons between the sites types. However, a trend is apparent in higher rates of pulmonary infection in major urban sites, compared to Poundbury Camp, as well as minor urban and rural sites (Table 6). [Table 6 and 7 here].

**Haematopoietic conditions**
The true prevalence of cribra orbitalia was statistically significantly higher at Poundbury Camp compared to both urban and rural sites types, at 38.5% ($\chi^2=29.88$, $p<0.005$, d.f.=3) (Table 8). Within the 1.1-2.5 and 6.6-10.5 year age groups, rural non-adults display higher rates at TPR 64.3% and TPR 66.7% respectively (Fig. 6). Small sample sizes in the rural age cohorts (14 and 6 respectively) prevent meaningful testing for statistical significance. At Poundbury Camp, a total of 11 infants were reported with active cribra orbitalia, whereas only one infant with active lesions was identified in the current study, from rural Bradley Hill. In the older children, the occurrence of active lesions was similar in the rural sites and Poundbury Camp at 24.8% and 21.0%, respectively, compared to between 10-15% in the urban site types (Table 7). The distribution of porotic hyperostosis is statistically significant ($\chi^2=19.73$, $p<0.005$, d.f.=3), with the highest rates observed at Poundbury Camp (7.2%) and the rural sites (6.2%), and low rates in the major urban (1.4%) and minor urban sample (2.8%) (Table 8).

One probable and two possible cases of thalassaemia were identified at Poundbury Camp (Lewis 2012). Two further possible cases were identified in the major urban sites of Gloucester and one probable case at the Colchester *colonia* (Rohnbogner, 2016). All identified individuals are below the age of 24 months. Relative frequencies of possible cases of thalassaemia are at CPR 1.1% at Poundbury Camp (n=3/276) and CPR 1.0% in the major urban sample (n=3/314). [Table 8 here; Figure 6 here].

**Metabolic disease**

The prevalence of lesions indicative of vitamin D deficiency was reported to be 4.8% at Poundbury Camp, and recorded in 89.2% (n=33) of the children under 2.5 years (Lewis 2011) (Table 9). Particularly in the infants, there is a trend for higher rates observed at Poundbury Camp (CPR=18.8%) compared to other Romano-British settlement types, although not significant ($\chi^2=6.49$, d.f.=3). Vitamin D deficiency was reported similarly in 1.1-2.5 year olds from *Durnovaria* and major urban and rural sites (Fig. 7). In the combined rural cemeteries, scurvy was identified in 5.9% of the non-adults, compared to 4.8% at Poundbury Camp, although this difference was not significant. Interestingly, all rural children aged to 2.6 years and older with lesions indicative of scurvy stem from the Cannington cemetery in Somerset. Infantile scurvy was reported at similar rates in major urban and rural sites, as well as Poundbury Camp. In non-adults aged 1.1-2.5 years and older, there is a trend for higher rates of vitamin C deficiency on rural sites, closely followed by the rates of scurvy reported from Roman Dorchester (Table 9, Fig. 8). Across all sites, there were individuals...
that displayed lesions that may have arisen due to metabolic disease or nutritional shortages, however these were either distributed inconclusively, or the skeleton was not sufficiently preserved to make a conclusive diagnosis. [Table 9 here; Figure 7 and 8 here].

DISCUSSION

The study presents the first large-scale analysis of non-adult palaeopathological data from Roman Britain. By contextualising the findings from previously published Poundbury Camp, new insights can be gleaned into the lives and deaths of children across Britannia, rather than the Durnovaria civitas alone. Some of the key findings remind us that Poundbury Camp remains an exceptional site. Yet there are some similarities in lesion frequencies across the sites, particularly between Poundbury Camp and the rural cohort of this study that urge us to consider equally compromised living conditions across Roman Britain.

Significantly higher rates of infant burials on rural sites are apparent. High numbers of infant burials are frequently observed in Roman Britain and have sparked ongoing debates about infanticide (Gowland and Chamberlain, 2002; Gilmore and Halcrow, 2014), or exposure (Gowland et al., 2014; Millett and Gowland, 2015). Low numbers of perinates at Poundbury Camp were observed, and when compared to the number of older infants, would suggest that exogenous factors were having little impact on the survival of babies born in to major urban settlements (Frenzen and Hogan, 1982; Scott and Duncan, 1999). This seems extremely unlikely given the potential hazards of overcrowding, poor hygiene and infections (Rawson, 2003:121; Roberts and Cox, 2003:123-30). It is feasible that differential burial rites reserved for the youngest impacted on the relative frequencies of infant burials between the site types. Indeed, intramural burial, exclusion from the formal cemetery, clustering in a dedicated area of the cemetery, or burial in a separate site altogether, have all been observed with infant burials in Roman Britain (Philpott, 1991; Scott, 1991; Pearce 1999; 2001; Esmonde Cleary, 2000; Wileman, 2005; Gowland et al., 2014). The observed patterns may be a reflection of cultural practices, rather than of mother and infant health, forcing us to approach infant mortality with caution.

At the other end of the age spectrum, the proportionately lower numbers of 6.6-10.5-year olds living and dying in the countryside, may indicate the migration of these older children, either voluntarily or forced, to commence their working lives. While challenging to prove isotopically in regions where the urban and rural geology may be similar, we know that this type of migration occurred elsewhere in the Roman Empire (Prowse et al., 2007; Killgrove and Montgomery, 2016).
Tuberculosis remains the quintessential urban disease of Roman Britain, although non-adult TB is no longer confined to Roman Dorchester and was also found in minor urban towns. The findings support previous research on the unsanitary and crowded urban environment (Roberts and Cox, 2005; Hall, 2005; Lewis 2011). However, non-adult TB that we can discern osteologically is merely the tip of the iceberg (Lewis, 2007). It is not until the post-primary/secondary phase of TB that skeletal changes occur, which requires either re-infection, or re-activation of the latent primary tuberculosis due to immune system suppression (Nelson and Wells, 2004; Roberts and Manchester, 2004:187). A compromised immune system aggravates the susceptibility to TB infection, and clinical studies attest a link between measles and whooping cough with increased TB risk (Nelson and Wells, 2004). Poor nutritional status also affects the severity and onset of the disease (Pfeiffer, 1984). Non-adults in major and minor urban environments not only experienced an increased risk of infection, but were also exposed to more prominent immunosuppressive factors than their rural peers. The trend for higher rates of pulmonary infection in major urban sites supports this argument, and is a witness to air pollution promoting respiratory ailments (Roberts and Buikstra, 2003; Roberts, 2007). In the absence of spinal involvement, the one possible case of rural TB is likely to be a respiratory infection. The adolescent from Cannington, Somerset, suggests that rural dwellings were poorly ventilated, and agrarian lifestyles would have forced close contact with animals and associated risks of infection.

The majority of individuals with new bone formation, particularly from Poundbury Camp and the urban sites, exhibited active new bone formation. Active lesions at the time of death may be a witness to ongoing, even chronic, health insults in these urban environments, perhaps stemming from generally poor health (Wood et al., 1992). However, the prevalence of sub-periosteal new bone formation as an indicator of non-specific stress contributes little to the debate due to the variety of causative agents such as generalised infection, neoplastic disease or trauma (Weston, 2012; DeWitte, 2014; Klaus, 2014). An additional limitation are the unique properties of non-adult bone. The non-adult periosteum is more prone to inflammation and tearing, and yet bone heals and remodels quicker than its adult equivalent, causing older lesions to be overlooked as a result of complete healing (Wenaden et al., 2005; Lewis 2007:133). The overall rate of skeletal pathologies in younger and older childhood may be a more viable indicator of general stress and health insults, which may combat some of these limitations. Skeletal pathology and age-at-death would suggest that growing up at Poundbury Camp proved most challenging in the early years, which is a stark reminder of the squalid nature of the settlement and maladaptive child rearing practices that may have been
followed at the site (Roberts and Cox, 2003; Lewis, 2010). More children with skeletal pathologies survived past the age of five years old in the non-Poundbury sites. Increased resilience to stressors is commonly observed in those growing up in rural settings (Wells and Evans, 2003), and the evidence suggests that the more rustic environments of Roman Britain may have proved less taxing on young children. However in the first instance, we do have to consider that some of the younger non-adults may not have suffered from a disease for long enough to manifest skeletally. Nevertheless, the inverse relationship of pathology and age-at-death observed at Poundbury Camp compared to the remainder of sites highlights the complex and unique nature of the burial assemblage at Roman Dorchester.

Lewis (2010) interpreted the prevalence of cribra orbitalia at Poundbury Camp as a witness of poor weaning foods, and unsanitary living conditions which prompted diarrhoeal diseases. The overall TPR of cribra orbitalia was significantly elevated at Poundbury Camp, a finding first reported by Stuart-Macadam (1991), forming the basis for Stuart-Macadam’s recording scheme. However, we have to be cautious not to let these findings colour our understanding of the true extent of haematopoietic conditions in Roman Britain. To date, Poundbury Camp yielded the highest number of non-adults with probable thalassaemia, alongside one case from Roman Colchester (Rohnbogner, 2016), and two possible cases from Roman Gloucester where preservation prevents from making conclusive diagnoses. Although we now know that the disease is not exclusive to Roman Dorchester, high rates of cribra orbitalia at Poundbury Camp could be related to high levels of genetic anaemia, rather than lifestyle and environment (Lewis, 2012).

As suggested by Lewis (2010), women in Roman Dorchester who breastfed may have been under nutritional and environmental stress themselves (Katzenberg et al., 1996; Robit et al., 2013). Fasting may have been undertaken as an early Christian ritual, resulting in high rates of cribra orbitalia in young children, and particularly infants at Poundbury Camp (Lewis, 2010). Although the issue is complex, maternal mal- or undernutrition may be traced via haematopoietic and metabolic shortcomings in infants and weanlings, and may have influenced the differing relationship of skeletal pathology and survivorship at Poundbury Camp, compared to other site types (Fildes, 1986; Temkin, 1991; Garnsey, 1999; Rawson, 2003a; Gowland, 2015). The majority of individuals across all sites types with porotic hyperostosis (67.6%), rickets (89.2%) and scurvy (64.1%) were younger than 2.5 years old. Formation of cranial porotic lesions in this age group may be related to maternal diets low in vitamins B6/B12 and iron, yielding insufficient levels of the micronutrients in breast milk (Kumar et al., 2008; Allen, 2012). The adverse effect of low maternal calcium intake is
largely kept at bay by increased intestinal absorption of calcium during pregnancy, and depletion of skeletal calcium stores during breastfeeding (Kovacs, 2005). However, mothers that were extremely low in vitamin D could pass the deficiency on during pregnancy, and subsequently via breast milk with decreased vitamin D content (Wagner et al., 2008). In most cases, rickets as calcium deficiency is normally seen after two years in response to inadequate weaning and supplementary feeding (Thacher, 2006). Although rare, scurvy may be transferred from mother to child following severe malnourishment during pregnancy (Crandall, 2014; Robbins Schug and Blevins, 2016). The possibility of depleted ascorbic acid stores in breastfeeding mothers also have to be considered, potentially resulting in infants and young children receiving vitamin C deficient breast milk, which would have its most marked impact prior to supplementation (Salmennperä, 1984; Emmett and Rogers, 1997). Infantile scurvy was reported at comparable rates on all sites which indicates shortcomings in maternal health, weaning practices and associated foods across all settlement types. Additionally, high rates of scurvy in children aged 1.1-2.5 years old from rural sites and in the ‘Christian’ cohort at Poundbury Camp are apparent. The bulk of scorbutic children at Roman Dorchester stems from the ‘non-pagan’ or ‘Christian’ cohort, perhaps a result of ‘Romanisation’ and devotion to a higher power that demanded significant dietary and lifestyle changes particularly for women and children (Rutgers et al., 2009; Lewis 2010). An early Christian following in southwestern parts of later Roman Britain may have promoted an ascetic lifestyle for women (Cool, 2006), in turn affecting infants and weanlings.

Apart from physiological characteristics of the mothers, strategies during transitional feeding would have impacted on the wellbeing of weanlings (Fildes, 1986; Katzenberg et al., 1996; Robit et al., 2013). Generally, a lack of fresh fruits and vegetables in the weaning diet is not surprising, given Soranus’ recommendation of cereal-based weaning foods which have been isotopically validated (Temkin, 1991; Powell et al., 2014). However, it was anticipated that rural children would have been weaned on vitamin C rich foods. Mothers in the countryside may have ceased breastfeeding earlier than around the isotopically ascribed 3-year benchmark (Fuller et al., 2006; Nehlich et a., 2011; Redfern et al., 2012; Powell et al., 2014), leaving the child more susceptible to nutritional deficiencies including scurvy.

In modern populations the prevalence of rickets is highest in children aged 3-18 months, as vitamin D stores are diminished six months after birth and need to be replenished via sunlight (Pettifor and Daniels, 1997; Foote and Marriott, 2003). As recommended by Soranus, swaddling and wrapping infants in clothes once they sit up, may have been a universal practice across Roman Britain and excessively shielded children from the sun
(Temkin, 1991:84-7,116). However, the clinical literature supports that swaddling has a marginal impact on vitamin D deficiency and promotes child wellbeing (Kutluk et al., 2002; van Sleuwen et al., 2007), and even fully clothed infants only need two hours of sunlight per week to maintain healthy bone structure (Pettifor and Daniels, 1997:665). In the crowded living quarters children may have been kept indoors during their younger years, due to lack of outdoor space and accidents on the busy roads and alleyways could be avoided. Parents and primary caregivers may have exhibited similar behaviours in rural settlements. Mothers working in the field would have covered their children and carried them in a sling, shielding away sunlight.

Some of the rachitic children may have been generally poorly (Snoddy et al., 2016). By keeping them indoors to recover and rest, vitamin D deficiency may have ensued, possibly exacerbated by low calcium levels in the diet, prolonged breastfeeding, and/or gastrointestinal maladies limiting calcium absorption, i.e. weanling’s diarrhoea (Pettifor and Daniels, 1997; Buckley, 2000; Foote and Marriott, 2003). Rachitic infants were most frequently reported in Roman Dorchester and minor urban sites, yet 1.1-2.5 year olds exhibited similar frequencies among major urban, rural and both ‘pagan’ and ‘Christian’ Poundbury Camp children. The minor urban rate is elevated in infancy, and then drops to the lowest rate in 1.1-2.5 year olds. Either rachitic children in minor urban sites were less likely to live past infancy, or poverty and biased resource allocation impacted on children more severely after the first year of life. It appears that the distribution of vitamin D deficiency at Poundbury Camp is neither the norm, nor representative for Roman Britain, and that the disease itself may not be traced back to dress or cultural habits alone. However, co-occurrence of vitamin C/D deficiency causes rickets to be macroscopically masked by scorbutic lesions (Schattmann et al., 2016), and rachitic lesions are inhibited by starvation, i.e. severe protein-calorie deficiency (Adams and Berridge, 1969; Salimpour, 1975). It therefore has to be assumed that the actual rate of rickets was higher in Roman Britain due to limitations in recognising the disease osteologically, and the time elapsing between onset of deficiency and skeletal lesions.

A significantly higher prevalence of healed cribrotic lesions was reported at Poundbury Camp, however there was also a trend for higher rates of active cribra orbitalia in the rural children. The distribution of both cribra orbitalia and porotic hyperostosis is comparable at Poundbury Camp and the rural cohort, urging us to consider similarities in cultural practices and the living environment between Roman Dorchester and the countryside of southwestern Roman Britain. We may have to consider whether rural individuals were
included in the Poundbury Camp cemetery, either by migration or living on rural sites in the hinterland or suburbium (Goodman, 2007:76-8; Laurence et al., 2011:288; Pearce, 2015). Results confirm elevated stress in the rural population as suggested by Griffin and Pitts (2012) and Redfern et al. (2015). We assume that a substantial portion of cribrotic lesions resulted from iron-deficiency anaemia and megaloblastic anaemia. Iron and vitamin B6/B12 are mainly obtained from the same food sources, i.e. red meat, pork, poultry, seafood, oatmeal and some vegetables (Baker et al. 2010; NHS 2015a). Previously published literature on dietary variability in Roman Britain attests that the consumption of plant foods, meat and fish was not uniform across social strata (King, 1984; 1999; 2001; Molleson, 1992; van der Veen, 2008; van der Veen et al., 2007; 2008; Cummings, 2009; Müldner, 2013). Although trace element analysis has revealed that those buried at Poundbury Camp ate a varied diet of meat, fish and plants, this may have been status dependent (Molleson, 1992).

Rural children may have experienced the same health insults as those at Poundbury Camp, possibly at a more pressing scale, causing them to develop even higher rates of orbital lesions in response to nutritional stress, infections, and high bacterial or parasitic pathogen loads (Stuart-Macadam, 1991; Holland and O’Brien, 1997; Wapler et al., 2004; Djuric et al., 2008; Walker et al., 2009; Oxenham and Cavill, 2010). If animal products were more difficult to access for lower status individuals as attested by zooarchaeological and isotopic studies (van der Veen et al., 2008; Cummings, 2009; Kilgrove and Tykot, 2012; Cheung et al., 2012; Müldner, 2013), the higher incidence of cribra orbitalia in rural children may indicate their lower status. Oatmeal itself was mainly reserved for animal fodder and only eaten occasionally (Cool, 2006:71; Britton and Huntley, 2011). Even among those consuming oatmeal, if the child suffered from a parasitic infection or diarrhoeal disease, the loss of nutrients would have presented a risk for iron and vitamin B6/B12 deficiencies when meat and fish were scarce (Holland and O’Brien, 1997; Facchini et al., 2004; Mahmud et al., 2013). Porotic lesions on the ectocranium were most common at Poundbury Camp and among rural non-adults, although prevalence is low overall. Redfern et al. (2015) observed porotic hyperostosis in 7.0% of individuals of all ages from rural sites in Roman Dorset, whereas no marrow hypertrophy in non-Poundbury Camp urban sites were observed. Migration into Roman Dorchester by rural individuals may have elevated the rates in the Poundbury Camp cemetery, or alternatively, rural dead from outlying villages and farmsteads may have been buried in the cemetery.

Contrary to what we would expect, endocranial lesions were lowest at Poundbury Camp and other major urban sites. More children in the rural cohort were affected, with
significantly higher numbers in the minor urban cohort. Perhaps the distribution demonstrates that settlements with an agricultural focus yielded conditions likely to promote inflammation in children. Lewis (2016) observed higher rates of endocranial lesions in rural 10.0-13.9 year olds from early medieval England. Areas of rural poverty existed in the medieval period, and perhaps similar health insults were prompted by the social stratification of Romano-British society, causing greater levels of stress, inflammation and infection in rural children. Recent findings on health in the Roman *suburbium* demonstrate that life outside of the urban centres was taxing (Killgrove and Tykot, 2012), and Redfern and colleagues (2015) demonstrated that compromised health in rural settings was a reality across Roman Dorset. The greater numbers of inflammatory responses as measured by endocranial lesions may have also arisen due to vitamin deficiencies, trauma or rapid new bone growth during early childhood (Lewis, 2004; Zahareas, 2011). Vitamin C deficiency was prevalent among the rural cohort, and we would expect to see trauma in children as a result of agrarian lifestyles and close contact with livestock. Perhaps a number of those identified with endocranial lesions in the rural sample were suffering from the onset of metabolic disease, or cranial trauma which had not produced additional skeletal lesions at the time of death, or cannot be identified due to preservation. A greater dispersal of vitamin C deficiency across older age groups was seen at Poundbury Camp and the rural sites. All of the rural children with possible scurvy aged 2.6 years and older originate from Cannington, Somerset, and all but one case of scurvy recorded in the rural children were active at the time of death (92.3%), suggesting a chronic state of deficiency. It is important not to confuse the mal- or undernutrition seen with starvation (Mays, 2014). The NHS recommends a daily intake of 40mg of vitamin C per day for adults (NHS, 2015b). More is required for children and pregnant/lactating women (Brickley and Ives, 2008:48; National Institutes of Health, 2011), yet clinical symptoms only start to develop once less than 10mg are available per day (Hodges et al., 1971; Stark, 2014). The minimum intake is covered by small amounts of fruits and vegetables, such as a cup of leek or pear (USDA National Nutrient Database, 2013). Pimentel (2003) stated that clinical symptoms will improve within two weeks after daily ingestion of 200mg of ascorbic acid, although skeletal lesions may take years to remodel entirely (Parfitt, 2002; 2004). Rural children did not experience starvation but rather a restricted diet, as some low levels of ascorbic acid were present to initiate bony changes (Crandall et al., 2012; Stark, 2014).

It may be suggested that the underlying social and economic causes resulting in infantile scurvy at Poundbury Camp and the rural sites were not the same. To date, the high levels of scurvy seen at Poundbury Camp are interpreted as the result of, perhaps even
voluntary, dietary restrictions that accompanied a new belief system (Cool, 2006). Rural infants may have developed scurvy due to withholding of resources, where access to foods was regulated by a market economy (Pitts, 2008). Given the context, the health implications of biased food allocation and resource distribution between urban and rural communities need to be considered (Whittaker and Garnsey, 1997:284; Redfern et al., 2015), as stress perpetuated by political and economic factors (Klaus, 2012; Crandall, 2014). If the population in the countryside was mainly concerned with having to provide for the urban population and army, a substantial portion of produce would have been reserved for trade and taxes (Scheidel and von Reden, 2002). Particularly at Cannington, Somerset, scurvy in the children aged 2.6 years and older may be linked with possible site characteristics of quarrying and large-scale organised exploitation of the land for farming (Rahtz, 2000:393,423), taking its toll on the mothers and children and potentially generating an influx of low-grade workers (McCarthy, 2013:102).

Peasant families would have lived in oppressive conditions compromising their diet, mental and physical wellbeing, a by-product of manorial or otherwise exploitative landownership and tenancy (de la Bédoyère, 1993:86; Jones 1996:208). Land would have been distributed to peasant farmers in late Roman Britain, based on the renewal of an annual lease. By the 4th century however, tenants became legally tied to the estate and land tenancy became hereditary. This made the process of taxation easier to oversee whilst also relieving the landowner of having to provide food and accommodation to the workers (de la Bédoyère, 1993:86). Bonded workers would therefore not only have been customary to late Romano-British villae, but tenancy affected the farmers in villages outside of villa estates (de la Bédoyère, 1993:74-5; Jones 1996:208-15). The malnutrition observed in rural Romano-British children may be a result of oppressive landownership, affecting food security and distribution in the countryside (Crandall, 2014). Social change and status differences inherently define the foods people have access to, and differences may have been pronounced in Roman Britain, affecting those at the bottom of the social ladder most profoundly (Armelagos et al., 2014). In theory, the state of deprivation on rural sites would also impact on the adults, particularly the females who will perpetuate a cycle of deprivation and depressed health status over several generations (Gowland, 2015). Although there is increasing awareness of marked ill-health beyond the city walls, both at Rome (Cucina et al., 2006) and in Roman Britain (Redfern et al., 2015), more research incorporating adult and non-adult osteological and isotopic data is desired to evaluate the full extent of health implications rural inhabitants may have experienced under Roman rule.
CONCLUSION

First and foremost, Poundbury Camp was the archetypal site that presented palaeopathologists with detailed data on child health, allowing us to question previous assumptions about urban living in the Roman period. The site’s contribution to current knowledge on childhood in Roman Britain, particularly in major urban settlements, is invaluable. Yet, Romano-British bioarchaeology is faced with a lack of comparative data to contextualise the high levels of ill-health witnessed in the Poundbury Camp non-adults. The problem is particularly pronounced with a view to rural settlements, as children of the countryside remain notoriously understudied. Naturally, the study relies on the children that died and were buried at major urban, minor urban and rural sites. As with any study that explores morbidity and mortality in the past, it is influenced by burial practices, preservation, excavation and the movement of people (Wood et al., 1992; 2002). Despite its limitations it presents the most comprehensive overview of non-adult health in Roman Britain to date.

The research discussed here provides new perspectives and increases our breadth of understanding of rural lifeways, adding to the debate on everyday life in the towns and countryside of Roman Britain. The results demonstrate that ill-health in Romano-British children was shaped by settlement type, and further highlight the somewhat unusual nature of Poundbury Camp as a major urban cemetery, particularly with view to the association of skeletal pathology and age-at-death. An overarching observation is that health was generally poor across Roman Britain, with children of all site types affected by metabolic disease, haematopoietic disturbances and infection. Some similarity is apparent between Roman Dorchester and other major urban sites, particularly in the diseases that relate to living environment, such as tuberculosis and vitamin D deficiency. Unexpectedly, the children from major urban cemeteries did not consistently show the highest lesion frequencies, and childhood pulmonary infection is no longer an exclusively urban disease. Overall, vitamin C deficiency and cribra orbitalia affected rural children at higher rates. Upon considering socio-economic and political factors that would have shaped land management and tenancy in southern and western England, we can glean how deficiency diseases would have been sustained by these children. Rural poverty affected those growing up in the countryside to a similar extent as non-local cultural habits may have done at Poundbury Camp, brandishing the site as unique among its other major urban counterparts.
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Table 8. Number and percent of haematopoietic lesions
Table 9. Number and percent of vitamin D and C deficiency
Table 1. Study samples by settlement type

<table>
<thead>
<tr>
<th>Site</th>
<th>Date (AD)</th>
<th>Type</th>
<th>Number of non-adults</th>
<th>Site Reference</th>
</tr>
</thead>
<tbody>
<tr>
<td>Winchester (North, West, East)</td>
<td>1-4&lt;sup&gt;th&lt;/sup&gt; century</td>
<td>Major Urban</td>
<td>166</td>
<td>Ottaway et al. (2012)</td>
</tr>
<tr>
<td>Kingsholm, Gloucester</td>
<td>2-4&lt;sup&gt;th&lt;/sup&gt; century</td>
<td>Major Urban</td>
<td>17</td>
<td>Hurst (1985), (1986)</td>
</tr>
<tr>
<td>Gambier-Parry Lodge, Gloucester</td>
<td>2-4&lt;sup&gt;th&lt;/sup&gt; century</td>
<td>Major Urban</td>
<td>12</td>
<td>Heighway (1980); Mullin (2006)</td>
</tr>
<tr>
<td>Trentholme Drive, York</td>
<td>3-4&lt;sup&gt;th&lt;/sup&gt; century</td>
<td>Major Urban</td>
<td>24</td>
<td>Wenham (1968); Ottaway (2009)</td>
</tr>
<tr>
<td>Bath Gate, Cirencester</td>
<td>4&lt;sup&gt;th&lt;/sup&gt; century</td>
<td>Major Urban</td>
<td>64</td>
<td>Viner and Leech (1982)</td>
</tr>
<tr>
<td>Butt Road, Colchester</td>
<td>4-5&lt;sup&gt;th&lt;/sup&gt; century</td>
<td>Major Urban</td>
<td>109</td>
<td>Crummy and Crossan (1993)</td>
</tr>
<tr>
<td><strong>Major urban total</strong></td>
<td></td>
<td></td>
<td><strong>392</strong></td>
<td></td>
</tr>
<tr>
<td>Baldock, Hertfordshire</td>
<td>2-4&lt;sup&gt;th&lt;/sup&gt; century</td>
<td>Minor Urban</td>
<td>83</td>
<td>Stead and Rigby (1980); Burleigh</td>
</tr>
<tr>
<td></td>
<td></td>
<td></td>
<td></td>
<td>and Fitzpatrick-Matthews (2010)</td>
</tr>
<tr>
<td>Queenford Farm/Mill, Oxfordshire</td>
<td>3-4&lt;sup&gt;th&lt;/sup&gt; century</td>
<td>Minor Urban</td>
<td>60</td>
<td>Durham and Rowley (1972); Chambers</td>
</tr>
<tr>
<td></td>
<td></td>
<td></td>
<td></td>
<td>(1987)</td>
</tr>
<tr>
<td>Ancaster, Lincolnshire</td>
<td>3-4&lt;sup&gt;th&lt;/sup&gt; century</td>
<td>Minor Urban</td>
<td>81</td>
<td>Todd (1975); Cox (1989)</td>
</tr>
<tr>
<td>Great Casterton, Rutland</td>
<td>3-4&lt;sup&gt;th&lt;/sup&gt; century</td>
<td>Minor Urban</td>
<td>38</td>
<td>McConnell et al. (2012)</td>
</tr>
<tr>
<td>Ashton, Northamptonshire</td>
<td>4&lt;sup&gt;th&lt;/sup&gt; century</td>
<td>Minor Urban</td>
<td>60</td>
<td>Dix (1983)</td>
</tr>
<tr>
<td>Dunstable, Bedfordshire</td>
<td>3-5&lt;sup&gt;th&lt;/sup&gt; century</td>
<td>Minor Urban</td>
<td>27</td>
<td>Matthews (1981)</td>
</tr>
<tr>
<td><strong>Minor urban total</strong></td>
<td></td>
<td></td>
<td><strong>349</strong></td>
<td></td>
</tr>
<tr>
<td>Owlslebury, Hampshire</td>
<td>1-4&lt;sup&gt;th&lt;/sup&gt; century</td>
<td>Rural</td>
<td>16</td>
<td>Collis (1968), (1977)</td>
</tr>
<tr>
<td>Cannington, Somerset</td>
<td>3-4&lt;sup&gt;th&lt;/sup&gt; century</td>
<td>Rural</td>
<td>148</td>
<td>Rahtz et al. (2000)</td>
</tr>
<tr>
<td>C catsgore, Somerset</td>
<td>2-5&lt;sup&gt;th&lt;/sup&gt; century</td>
<td>Rural</td>
<td>19</td>
<td>Leech (1982)</td>
</tr>
<tr>
<td>Bradley Hill, Somerset*</td>
<td>4-5&lt;sup&gt;th&lt;/sup&gt; century</td>
<td>Rural</td>
<td>29</td>
<td>Leech et al. (1981); Gerrard (2011)</td>
</tr>
<tr>
<td><strong>Rural total</strong></td>
<td></td>
<td></td>
<td><strong>212</strong></td>
<td></td>
</tr>
<tr>
<td><strong>Total sample</strong></td>
<td></td>
<td></td>
<td><strong>953</strong></td>
<td></td>
</tr>
</tbody>
</table>

*non-adult burials were recovered from within the settlement boundaries associated with a 4<sup>th</sup> century AD building (Leech et al., 1981), rather than from the 5<sup>th</sup> century AD cemetery (Gerrard, 2011)
Table 2. Age-at-death of non-adults from Roman England

<table>
<thead>
<tr>
<th>Age (yrs)</th>
<th>Poundbury Camp</th>
<th>Major urban</th>
<th>Minor urban</th>
<th>Rural</th>
</tr>
</thead>
<tbody>
<tr>
<td></td>
<td>n</td>
<td>%</td>
<td>n</td>
<td>%</td>
</tr>
<tr>
<td>Perinate</td>
<td>55</td>
<td>15.1</td>
<td>81</td>
<td>20.6</td>
</tr>
<tr>
<td>0.0-1.0</td>
<td>87</td>
<td>23.9</td>
<td>65</td>
<td>16.6</td>
</tr>
<tr>
<td>1.1-2.5</td>
<td>46</td>
<td>12.6</td>
<td>42</td>
<td>10.7</td>
</tr>
<tr>
<td>2.6-6.5</td>
<td>55</td>
<td>15.1</td>
<td>62</td>
<td>15.8</td>
</tr>
<tr>
<td>6.6-10.5</td>
<td>41</td>
<td>11.3</td>
<td>44</td>
<td>11.2</td>
</tr>
<tr>
<td>10.6-14.5</td>
<td>34</td>
<td>9.3</td>
<td>48</td>
<td>12.2</td>
</tr>
<tr>
<td>14.6-17.0</td>
<td>13</td>
<td>3.6</td>
<td>28</td>
<td>7.1</td>
</tr>
<tr>
<td>Non-adult</td>
<td>33</td>
<td>9.1</td>
<td>22</td>
<td>5.6</td>
</tr>
<tr>
<td><strong>Total</strong></td>
<td><strong>364</strong></td>
<td></td>
<td><strong>392</strong></td>
<td></td>
</tr>
</tbody>
</table>

% rounded percentage of site total for each age group

Table 3. Differences in frequency of skeletal pathology and age-at-death

<table>
<thead>
<tr>
<th>Site type</th>
<th>0-5 years</th>
<th>6-17 years</th>
<th>X²</th>
</tr>
</thead>
<tbody>
<tr>
<td><strong>Poundbury Camp</strong></td>
<td>242</td>
<td>92</td>
<td>0.71</td>
</tr>
<tr>
<td>n skeletal pathology</td>
<td>60</td>
<td>19</td>
<td></td>
</tr>
<tr>
<td>% skeletal pathology</td>
<td>24.8</td>
<td>20.7</td>
<td></td>
</tr>
<tr>
<td><strong>Major urban</strong></td>
<td>234</td>
<td>136</td>
<td>10.26</td>
</tr>
<tr>
<td>n skeletal pathology</td>
<td>50</td>
<td>50</td>
<td></td>
</tr>
<tr>
<td>% skeletal pathology</td>
<td>21.4</td>
<td>36.8</td>
<td></td>
</tr>
<tr>
<td><strong>Minor urban</strong></td>
<td>256</td>
<td>85</td>
<td>16.94*</td>
</tr>
<tr>
<td>n skeletal pathology</td>
<td>70</td>
<td>44</td>
<td></td>
</tr>
<tr>
<td>% skeletal pathology</td>
<td>27.3</td>
<td>51.8</td>
<td></td>
</tr>
<tr>
<td><strong>Rural</strong></td>
<td>162</td>
<td>49</td>
<td>14.69*</td>
</tr>
<tr>
<td>n skeletal pathology</td>
<td>47</td>
<td>29</td>
<td></td>
</tr>
<tr>
<td>% skeletal pathology</td>
<td>29.0</td>
<td>59.2</td>
<td></td>
</tr>
</tbody>
</table>

*p<0.001, d.f.=2
Table 4. Number and percent for endocranial lesions in non-adults with crania

<table>
<thead>
<tr>
<th>Age (yrs)</th>
<th>Observed</th>
<th>Affected n/%</th>
<th>Observed</th>
<th>Affected n/%</th>
<th>Observed</th>
<th>Affected n/%</th>
<th>Observed</th>
<th>Affected n/%</th>
</tr>
</thead>
<tbody>
<tr>
<td>Perinate</td>
<td>33</td>
<td>1/3.0</td>
<td>61</td>
<td>5/8.2</td>
<td>43</td>
<td>1/2.3</td>
<td>21</td>
<td>1/4.8</td>
</tr>
<tr>
<td>0-1.0</td>
<td>50</td>
<td>3/6.0</td>
<td>4</td>
<td>0</td>
<td>19</td>
<td>3/15.8</td>
<td>9</td>
<td>0</td>
</tr>
<tr>
<td>1.1-2.5</td>
<td>37</td>
<td>4/10.8</td>
<td>37</td>
<td>4/10.8</td>
<td>54</td>
<td>11/20.4</td>
<td>23</td>
<td>3/13.0</td>
</tr>
<tr>
<td>2.6-6.5</td>
<td>47</td>
<td>2/4.3</td>
<td>48</td>
<td>0</td>
<td>57</td>
<td>8/14.0</td>
<td>22</td>
<td>3/13.6</td>
</tr>
<tr>
<td>6.6-10.5</td>
<td>34</td>
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<td>5/21.7</td>
<td>7</td>
<td>1/14.3</td>
</tr>
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<td>1/3.4</td>
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<td>23</td>
<td>1/4.4</td>
<td>17</td>
<td>2/11.8</td>
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<td>22</td>
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<td>8</td>
<td>2/25.0</td>
<td>13</td>
<td>1/7.7</td>
</tr>
<tr>
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<td>2/50</td>
<td>11</td>
<td>0</td>
<td>5</td>
<td>0</td>
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<td>0</td>
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<tr>
<td>Total</td>
<td>247</td>
<td>14/5.7</td>
<td>196</td>
<td>7/3.6</td>
<td>192</td>
<td>30/15.9</td>
<td>92</td>
<td>10/10.9</td>
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% rounded percentage of site total for each age group

Table 5. Number and percent crude prevalence rates of new bone formation

<table>
<thead>
<tr>
<th>Age (yrs)</th>
<th>Observed</th>
<th>Affected n/%</th>
<th>Observed</th>
<th>Affected n/%</th>
<th>Observed</th>
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<td>-</td>
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<td>0</td>
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<td>0</td>
</tr>
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<td>27</td>
<td>2/7.4</td>
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<td>2/4.1</td>
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<td>46</td>
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<td>7/11.3</td>
<td>25</td>
<td>1/4.0</td>
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<td>1/3.1</td>
<td>37</td>
<td>5/13.5</td>
<td>25</td>
<td>2/8.0</td>
<td>7</td>
<td>1/14.3</td>
</tr>
<tr>
<td>10.6-14.5</td>
<td>30</td>
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<td>5/13.9</td>
<td>27</td>
<td>2/7.4</td>
<td>17</td>
<td>1/5.9</td>
</tr>
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<td>14.6-17.0</td>
<td>11</td>
<td>2/18.2</td>
<td>22</td>
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<td>13</td>
<td>2/15.4</td>
</tr>
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<td>0</td>
</tr>
<tr>
<td>Total</td>
<td>143</td>
<td>4/2.8</td>
<td>182</td>
<td>18/9.9</td>
<td>204</td>
<td>14/6.9</td>
<td>95</td>
<td>5/5.3</td>
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</table>

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<th>Status</th>
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<th>Affected n/%</th>
<th>Observed</th>
<th>Affected n/%</th>
<th>Observed</th>
<th>Affected n/%</th>
<th>Observed</th>
<th>Affected n/%</th>
</tr>
</thead>
<tbody>
<tr>
<td>Active</td>
<td>143</td>
<td>3/2.1</td>
<td>182</td>
<td>9/4.9</td>
<td>204</td>
<td>8/3.9</td>
<td>95</td>
<td>2/2.1</td>
</tr>
<tr>
<td>Healed</td>
<td>143</td>
<td>1/0.7</td>
<td>182</td>
<td>7/3.8</td>
<td>204</td>
<td>4/2.0</td>
<td>95</td>
<td>2/2.1</td>
</tr>
</tbody>
</table>

% rounded percentage of site total for each age group
Table 6. Summary of probable or possible tuberculosis and pulmonary infection

<table>
<thead>
<tr>
<th></th>
<th>Total</th>
<th>Probable tuberculosis</th>
<th>Possible tuberculosis</th>
<th>Pulmonary infection</th>
</tr>
</thead>
<tbody>
<tr>
<td></td>
<td>Observed</td>
<td>Affected n/%</td>
<td>Affected n/%</td>
<td>Affected n/%</td>
</tr>
<tr>
<td>Poundbury Camp</td>
<td>165</td>
<td>2/1.2</td>
<td>5/3.0</td>
<td>3/1.8</td>
</tr>
<tr>
<td>Major urban</td>
<td>161</td>
<td>2/1.2</td>
<td>1/0.6</td>
<td>8/5.0</td>
</tr>
<tr>
<td>Minor urban</td>
<td>157</td>
<td>1/0.6</td>
<td>1/0.6</td>
<td>2/1.3</td>
</tr>
<tr>
<td>Rural</td>
<td>84</td>
<td>0</td>
<td>1/1.2</td>
<td>0</td>
</tr>
<tr>
<td>Total</td>
<td>567</td>
<td>5/0.9</td>
<td>8/1.4</td>
<td>13/2.3</td>
</tr>
</tbody>
</table>

% rounded percentage of site total
Table 7. Probable and possible tuberculosis in the non-Poundbury non-adults

<table>
<thead>
<tr>
<th>Site/skeleton</th>
<th>Age range</th>
<th>Bones present</th>
<th>Lesions observed</th>
<th>Diagnosis</th>
</tr>
</thead>
<tbody>
<tr>
<td>Butt Road, Colchester/376</td>
<td>10.6-14.5</td>
<td>Skull, teeth, ribs, spine, pelvis, long bones, hands/feet</td>
<td>Lytic lesions and minimal new bone formation on visceral aspect of ribs. Lytic foci on vertebral bodies of L1-L5. S1 with lytic lesion on anterior aspect of body. MC1 with possible dactylitis. Iliac blades with resorptive foci on cortex both medio-anteriorly and adjacent to auricular surface bilaterally with localised new bone formation.</td>
<td>Probable pulmonary TB, with possible gastro-intestinal origin</td>
</tr>
<tr>
<td>Butt Road, Colchester/672</td>
<td>2.6-6.5</td>
<td>Skull, teeth, ribs, spine, pelvis, long bones</td>
<td>Localised healed new bone formation on pleural aspect of rib heads, some with lytic foci in new bone deposits. One right rib head with large lytic lesion on vertebral aspect with sclerotic margins and minimal new bone formation. Pott’s disease affecting T1-T3 with fusion evident.</td>
<td>Probable pulmonary TB</td>
</tr>
<tr>
<td>Ashton/126</td>
<td>2.6-6.5</td>
<td>Skull, teeth, ribs, spine, pelvis, long bones, hands/feet</td>
<td>Parietal endocranial lesions. Healing new bone formation on the tibial shafts bilaterally. Widespread active new bone formation on medial aspects of iliac blades. Destructive resorption on sacral bodies and L5. S1 with large lytic lesion on left lateral portion of body. Large lytic focus on superior aspect of L5 vertebral body.</td>
<td>Probable pulmonary TB, with possible gastro-intestinal origin</td>
</tr>
<tr>
<td>Trentholme Drive, York/31</td>
<td>6.6-10.5</td>
<td>Mandible, teeth, ribs, spine, pelvis, long bones, hands/feet</td>
<td>Left ribs with prominent new bone formation on visceral aspect with localised lytic foci in new bone deposits. Widespread new bone formation on tibial shaft and calcanei. Right fibula with suppurative osteomyelitis.</td>
<td>Possible pulmonary TB/pulmonary infection</td>
</tr>
<tr>
<td>Ancaster/55</td>
<td>2.5-6.5</td>
<td>Skull, teeth, ribs, spine, pelvis, long bones, hands/feet</td>
<td>Parietal endocranial lesions. Ribs with new bone formation on pleural aspect, with localised lytic lesion. Ulnae, femora, tibiae and fibulae with widespread new bone formation.</td>
<td>Possible pulmonary TB/pulmonary infection</td>
</tr>
<tr>
<td>Cannington/51b</td>
<td>14.5-17.0</td>
<td>Skull, teeth, ribs, spine, pelvis, long bones, hands/feet</td>
<td>Parietal endocranial lesions. Ribs with new bone formation and lytic lesions within new bone deposits. Widespread active new bone formation on femoral and tibial shafts.</td>
<td>Possible pulmonary TB/pulmonary infection</td>
</tr>
</tbody>
</table>
Table 8. Number and percent true prevalence rates for cribra orbitalia and porotic hyperostosis

<table>
<thead>
<tr>
<th></th>
<th>Poundbury Camp</th>
<th>Major urban</th>
<th>Minor urban</th>
<th>Rural</th>
</tr>
</thead>
<tbody>
<tr>
<td><strong>Cribra orbitalia</strong></td>
<td></td>
<td></td>
<td></td>
<td></td>
</tr>
<tr>
<td>Age (yrs)</td>
<td>Observed</td>
<td>Affected n/%</td>
<td>Observed</td>
<td>Affected n/%</td>
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<tr>
<td>Perinate</td>
<td>26 0</td>
<td>54 0</td>
<td>37 0</td>
<td>19 0</td>
</tr>
<tr>
<td>0.0-1.0</td>
<td>36 2/5.6</td>
<td>33 0</td>
<td>26 0</td>
<td>26 1/3.8</td>
</tr>
<tr>
<td>1.1-2.5</td>
<td>45 14/31.1</td>
<td>30 4/13.3</td>
<td>37 6/16.2</td>
<td>14 9/64.3</td>
</tr>
<tr>
<td>2.6-6.5</td>
<td>56 19/33.9</td>
<td>32 10/31.3</td>
<td>42 19/45.2</td>
<td>13 6/46.2</td>
</tr>
<tr>
<td>6.6-10.5</td>
<td>52 21/40.4</td>
<td>24 5/20.8</td>
<td>18 7/38.9</td>
<td>6 4/66.7</td>
</tr>
<tr>
<td>10.6-14.5</td>
<td>44 17/38.6</td>
<td>30 10/33.3</td>
<td>20 6/30.0</td>
<td>12 3/25.0</td>
</tr>
<tr>
<td>14.6-17.0</td>
<td>11 3/27.3</td>
<td>18 6/33.3</td>
<td>6 2/33.3</td>
<td>11 5/45.5</td>
</tr>
<tr>
<td>Non-adult</td>
<td>1 1/100</td>
<td>5 1/20.0</td>
<td>0 0</td>
<td>0 0</td>
</tr>
<tr>
<td>Total</td>
<td>200 77/38.5</td>
<td>223 36/16.1</td>
<td>186 40/21.5</td>
<td>101 28/27.7</td>
</tr>
<tr>
<td><strong>Active</strong></td>
<td>200 42/21.0</td>
<td>223 24/10.8</td>
<td>186 29/15.6</td>
<td>101 25/24.8</td>
</tr>
<tr>
<td><strong>Healed</strong></td>
<td>200 25/12.5</td>
<td>223 12/5.4</td>
<td>186 10/5.4</td>
<td>101 1/0.9</td>
</tr>
<tr>
<td><strong>Porotic hyperostosis</strong></td>
<td></td>
<td></td>
<td></td>
<td></td>
</tr>
<tr>
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</tr>
<tr>
<td>Perinate</td>
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<td>51 0</td>
<td>37 0</td>
<td>20 0</td>
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<tr>
<td>0.0-1.0</td>
<td>78 9/11.5</td>
<td>42 0</td>
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<tr>
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<td>50 2/4.0</td>
<td>18 4/22.2</td>
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<td>54 0</td>
<td>20 0</td>
</tr>
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<td>6.6-10.5</td>
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<td>22 0</td>
<td>6 0</td>
</tr>
<tr>
<td>10.6-14.5</td>
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<td>41 1/2.4</td>
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<td>15 0</td>
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<td>20 0</td>
<td>8 0</td>
<td>13 2/15.4</td>
</tr>
<tr>
<td>Non-adult</td>
<td>15 2/13.3</td>
<td>11 1/9.1</td>
<td>5 0</td>
<td>1 1/100</td>
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<td>278 4/1.4</td>
<td>235 3/2.8</td>
<td>130 8/6.2</td>
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</table>

% rounded percentage of site total for each age group
Table 9. Number and percent prevalence rates for vitamin D and C deficiency

<table>
<thead>
<tr>
<th>Age (yrs)</th>
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<th>Minor urban</th>
<th>Rural</th>
</tr>
</thead>
<tbody>
<tr>
<td></td>
<td>Observed</td>
<td>Affected n/%</td>
<td>Observed</td>
<td>Affected n/%</td>
</tr>
<tr>
<td>Perinate</td>
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<td>0</td>
<td>81</td>
<td>0</td>
</tr>
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<td>0.0-1.0</td>
<td>48</td>
<td>9/18.8</td>
<td>58</td>
<td>4/6.9</td>
</tr>
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<td>1.1-2.5</td>
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<td>3/8.1</td>
<td>40</td>
<td>3/7.5</td>
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<td>60</td>
<td>1/1.7</td>
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<td>0</td>
<td>44</td>
<td>0</td>
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<td>47</td>
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<td>28</td>
<td>1/3.6</td>
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<td>Non-adult</td>
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<td>20</td>
<td>0</td>
</tr>
<tr>
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<td>378</td>
<td>9/2.4</td>
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Vitamin C deficiency

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<th>Observed</th>
<th>Affected n/%</th>
<th>Observed</th>
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<td>48</td>
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<td>81</td>
<td>0</td>
<td>66</td>
<td>0</td>
<td>41</td>
<td>0</td>
</tr>
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<td>0.0-1.0</td>
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<td>3/6.3</td>
<td>58</td>
<td>4/6.9</td>
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<td>1.1-2.5</td>
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<td>4/10.8</td>
<td>40</td>
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<td>56</td>
<td>1/1.8</td>
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<td>3/12.5</td>
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<td>2/6.5</td>
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<td>1/3.3</td>
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<td>28</td>
<td>0</td>
<td>11</td>
<td>1/9.1</td>
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<tr>
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<td>1/3.4</td>
<td>47</td>
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<td>28</td>
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<td>20</td>
<td>2/10.0</td>
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<tr>
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<td>28</td>
<td>1/3.6</td>
<td>11</td>
<td>0</td>
<td>14</td>
<td>0</td>
</tr>
<tr>
<td>Non-adult</td>
<td>5</td>
<td>1/20.0</td>
<td>20</td>
<td>0</td>
<td>7</td>
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<tr>
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<td>12/4.8</td>
<td>378</td>
<td>7/1.9</td>
<td>327</td>
<td>8/2.4</td>
<td>202</td>
<td>12/5.9</td>
</tr>
</tbody>
</table>

% rounded percentage of site total for each age group
Figure 1. Location of sites (black: major urban; dark grey: minor urban; light grey: rural).

Figure 2. Age-at-death at Poundbury Camp and non-Poundbury sites.

Figure 3. Probable tuberculosis. a, new bone formation on the medial aspect of the right ilium; b, left rib inferio-medial with resorption and lytic lesion, also new bone formation on visceral aspect; c, S1 with lytic focus on the inferior margin of vertebral body; d, T5 left supero-lateral: resorptive focus on anterior vertebral body (arrow); e, MC1 with probable dactylitis. From Butt Road, Colchester, skeleton 376 (with kind permission from Colchester and Ipswich Museums).

Figure 4. Probable tuberculosis. Left lateral (a) and right lateral (c): destruction of the vertebral body of T2, and fusion of T2 and T3 at the left pedicle and lamina indicative of Pott’s Disease; b, right rib inferiorly with new bone formation and lytic foci in sub-periosteal new bone; d, right rib inferiorly with large circular lytic lesion on the vertebral aspect of head. From Butt Road, Colchester, skeleton 672 (with kind permission from Colchester and Ipswich Museums).

Figure 5. Probable tuberculosis. a, Type 4 endocranial lesion on parietal fragment; b, widespread periosteal new bone formation on medial aspect of right ilium; c, L5 superiorly with eroded and resorptive destruction of vertebral body (arrows); d, S1 superiorly with circular lytic lesion (arrow); e, S1 anteriorly with destruction of the anterior margins of sacral body. From Ashton, skeleton 126 (with kind permission from Vivacity Peterborough Museum and Art Gallery).

Figure 6. True prevalence rates of cribra orbitalia by age group.

Figure 7. Vitamin D deficiency in Romano-British non-adults.

Figure 8. Vitamin C deficiency by age group.
Figure 1. Location of sites (black: major urban; dark grey: minor urban; light grey: rural).
Figure 2. Age-at-death at Poundbury Camp and non-Poundbury sites.
Figure 3. Probable tuberculosis. a, new bone formation on the medial aspect of the right ilium; b, left rib inferio-medial with resorption and lytic lesion, also new bone formation on visceral aspect; c, S1 with lytic focus on the inferior margin of vertebral body; d, T5 left supero-lateral: resorptive focus on anterior vertebral body (arrow); e, MC1 with probable dactylitis. From Butt Road, Colchester, skeleton 376 (with kind permission from Colchester and Ipswich Museums).
Figure 4. Probable tuberculosis. Left lateral (a) and right lateral (c): destruction of the vertebral body of T2, and fusion of T2 and T3 at the left pedicle and lamina indicative of Pott’s Disease; b, right rib inferiorly with new bone formation and lytic foci in sub-periosteal new bone; d, right rib inferiorly with large circular lytic lesion on the vertebral aspect of head. From Butt Road, Colchester, skeleton 672 (with kind permission from Colchester and Ipswich Museums).
Figure 5. Probable tuberculosis. a, Type 4 endocranial lesion on parietal fragment; b, widespread periosteal new bone formation on medial aspect of right ilium; c, L5 superiorly with eroded and resorptive destruction of vertebral body (arrows); d, S1 superiorly with circular lytic lesion (arrow); e, S1 anteriorly with destruction of the anterior margins of sacral body. From Ashton, skeleton 126 (with kind permission from Vivacity Peterborough Museum and Art Gallery).
Figure 6. True prevalence rates of cribra orbitalia by age group.
Figure 7. Vitamin D deficiency in Romano-British non-adults.
Figure 8. Vitamin C deficiency by age group.