A novel investigation into migrant and local health-statuses in the past: a case study from Roman Britain

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Abstract

Migration continues to be a central theme in archaeology, and bioarchaeology has made significant contributions towards understanding the disease and demographic consequences of migration in different periods and places. These studies have been enhanced by stable isotope studies of mobility and diet, which have revealed further complexities.

This study integrates osteological, palaeopathological and stable isotope evidence to investigate the interrelationship between migrant and local population disease frequencies in Roman Britain. Previous analyses have identified migrants from across the Roman Empire, along with increases in the prevalence rates of infectious and metabolic diseases, poor dental health and non-specific indicators of stress. This study aims to explore the extent to which migrants and people born in Britain differed in terms of mortality risk and the frequencies of disease variables. Osteological and dental data from 151 individuals excavated from 24 Romano-British cemetery sites with mobility isotope data were statistically analysed. The results reveal significant differences between migrant and local populations for periosteal new bone formation, rib lesions, residual rickets, and dental health variables. When data were pooled for both sexes, a statistically significant difference in mortality between the two groups was also observed.

Overall, the results of this study suggest that migrants transformed patterns of disease in the Romano-British period, and combined with the changes to settlement patterns and environment, created new disease risks for both groups. The results also show that many of the key bioarchaeological indicators of change following the Roman conquest may actually reveal more about disease and health experienced in the wider Empire.

Migration remains a prominent theme in archaeology, and human skeletal remains have proven fundamental to such studies (Baker and Tsuda 2015:3; Burmeister 2000; Hakenbeck 2008; Murphy and Klaus 2017). Clinical and epidemiological studies have shown that the interaction between migrant and local (also known as host) populations can result in profound and sometimes devastating impacts in terms of disease frequency (Ahonen et al. 2007; Bhopal 2014; Mascie-Taylor and Krzyżanowska 2017). Recent advances in biomolecular techniques have revolutionised our understanding of migration in the past (Baker and Tsuda 2015: 4-5; van Dommelen 2014) (e.g. Stantis et al. 2015). Stable isotope data from teeth have provided important information concerning childhood origin, as well as dietary staples, which may point to an 'exotic' provenance (Eckardt 2010). This study integrates the osteological, palaeopathological and stable isotope evidence to examine the differences in disease and mortality risk between incomers and the local population during the Roman occupation of Britain in AD 43-410.

The Roman conquest of Britain resulted in a distinct and transformative shift in the archaeological record, including new settlement patterns, material culture and funerary rites (Mattingly 2006). Migration in Roman military and urban populations has been explored extensively using mobility isotope studies by a number of authors (e.g., Eckardt 2010; Eckardt et al. 2014), resulting in a substantial published dataset (see also, Martiniano et al. 2016). Bioarchaeological studies have observed disease differences between pre-Roman (late Iron Age) and Roman populations, and also between urban and rural communities (Bonsall 2013; Redfern and DeWitte 2011a; Redfern et al. 2015; Rohnbogner 2015). Regional and national datasets have established that mortality risk increases for males and subadults, with increases in the frequency of infectious and metabolic diseases, dental disease and indicators of stress post-Conquest (Redfern and DeWitte 2011b; Redfern et al. 2012).

Using Roman Britain as a case-study, our research builds on these studies by investigating the extent to which these patterns of disease (see Temple and Goodman 2014) and mortality risks were created and shaped by people who had travelled within the region or country where they died, and those who came to Britain from abroad. It also examines whether the clinical trend for differences in health patterns, particularly disease frequencies and mortality rates between migrant and non-migrant (or host) populations can be established in an archaeologically derived sample of human remains.

Materials and Methods

Osteological data

The sample comprised a total of 24 cemetery sites from Dorset, Hampshire, Yorkshire, and the Greater London area (Figure 1) for which isotope values had been published, and a human bone report or archived data were accessible (Tables 1 and 2); the dataset used in the study is published as a Supplementary Table 1). These sites were predominantly urban and/or military in nature, as many sites, such as York and Gloucester were urban settlements established by the military, who also had a permanent base there (see Wacher 2016). The archaeological and stable isotope evidence revealed that these settlements had been inhabited by both migrants and people whose childhoods were spent in Britain. Ancestry evidence for the Roman period, gleaned from ancient DNA and forensic methods, suggests that, in some instances, British-born people had non-White European heritage (Leach et al. 2009; Redfern et al. 2016, 2017). Only a portion of each of the cemetery sites had been sampled for stable isotope ratios (e.g. Chenery et al. 2011).

Demographic analyses of these cemeteries revealed a male bias, which is often the case in Roman cemeteries, particularly those with a military connection (Pearce 2011), and this has influenced the sex ratio of the overall sample (Table 3). A total of 151 individuals were included, ranging in age from infancy to older adult. Eighty-one were adult male (≥15 years old), 47 adult female, 3 adults of unknown sex, and 20 subadults aged between 10 months and 15 years old (Table 3, see also Supplementary Table 1). The decision to lower the subadult/adult threshold to 15 years old was undertaken because in a Roman life course perspective, this was the age when girls and some boys were recognised as being socially adult (Harlow and Laurence 2002: 54-78).

Using previously published isotope data, an inventory of context numbers was created and cross-referenced to the human bone reports. The majority of skeletons had been analysed after the introduction of recording standards (Brickley and McKinley 2004; Buikstra and Ubelaker 1994) meaning that, for the most part, the same methods to determine sex, estimate age-at-death and identify diseases had been used. However, we recognise that inter-observer error in observations and differences in recording methods presents an inescapable bias (see, Roberts and Cox 2003: 26-30). For the older reports, individuals were included only when the methods used to record the human remains were described and considered to be adequate. Additionally, the osteological recording had to be sufficiently detailed to establish the presence/absence of bones in order to accurately calculate prevalence rates. Bone completeness and preservation varied greatly between cemetery sites due to a variety of taphonomic factors, including differences in soil types across Britain (UK Soil Observatory 2016).

A re-evaluation of diagnoses of pathological lesions was undertaken for each individual based on the descriptions of the bone and dental lesions present in the reports. This was deemed necessary because of recent developments in the diagnostic criteria relating to a range of metabolic and infectious diseases. The selected conditions (Table 4) were only recorded if the requisite bones or teeth necessary for a diagnosis were present, and were scored as present (1) or absent (0). If the requisite bones or teeth were not present or unobservable due to taphonomic damage this was scored as a (9); for example, rickets and osteomalacia were scored as (9) if long bones were absent due to post-mortem taphonomic damage or truncation, as these bones are critical to making a diagnosis (Brickley and Ives 2008: 97-100, 109-111) (See Supplementary Table 1). Each individual had been previously assessed for age and sex, and for age-at-death a mid-point value was produced from the age range provided in order to facilitate statistical analyses (See Supplementary Table 1); for those individuals with open-ended age estimates of >45 or >60, the values 50.5 and 60, respectively, were used.

Stable and radiogenic isotope evidence for mobility

The study's dataset had been published from 1998 onwards, with lead, oxygen and strontium isotopes from dental enamel used to explore mobility (Richards and Montgomery 2012). These isotope systems represent different environmental parameters to which an individual was exposed, and specifically in the Roman period, anthropogenic pollutants (lead), thus providing information about childhood residential origins (Evans et al. 2012; Montgomery 2002). By comparing data from an individual with the values prevailing in the burial location, it is possible to reliably assert that the person had migrated to that locale before death (Montgomery 2010).

As the field of stable isotope analysis has witnessed rapid development since the 1990s, it was imperative that the published values were scrutinised and re-evaluated to establish a person's origin. The isotope values were interrogated by the third author who then assigned the individual to one of three groups: regional local (values consistent with the region where the person was buried), British (excludes regional locals, values consistent with the British Isles), and non-British (values not consistent with the British Isles) (Table 2; see also Supplementary Table 1). Individuals were assigned local status if both their strontium and oxygen isotope ratios were consistent with origins in the biosphere overlying the geology of the cemetery according to the strontium isotope tables in Evans et al. (2012) *and* with the 2sd oxygen isotope range of humans from either western (Winchester, Gloucester and Poundbury Camp) or eastern (London and Yorkshire) England. If one of these isotope ratios precluded local origins but did not preclude origins in Britain these individuals were deemed to be consistent with origins in Britain. If *either* the strontium or oxygen isotope ratio fell outside the ranges for Britain, *or* the individual had previously been convincingly identified as of non-British origin based on published isotope evidence for exposure to non-British lead (Montgomery et al. 2010; Shaw et al. 2016), or carbon isotope evidence for the consumption of a C_4 diet (Müldner et al. 2011; Eckardt et al. 2015), the individual was deemed to be of non-British origin. This method may return false positives, i.e. some individuals may be assigned local status when they were not, but should not produce false negatives, i.e. it was highly unlikely that individuals were incorrectly assigned non-local status. For some individuals (see Table 2), their borderline/undiagnostic values meant that it was not absolutely clear-cut which group individuals belonged to, and these were identified as 'possible' locals (regional or British) or 'possible' non-British.

Statistical analyses

Differences between the three groups (regional local, British, and non-British) with respect to the frequencies of a range of disease variables (cribra orbitalia, porotic hyperostosis, periosteal new bone formation, rib lesions, enamel hypopolastic defects, periodontal disease, dental caries, and calculus) were assessed using Chi-square analyses. The second author ran the statistical analyses excluding the 'possible' isotope groupings for cribra orbitalia, porotic hyperostosis, periosteal new bone formation and rib lesions. These results were found to be similar to those obtained by including 'possible' individuals. Therefore, these individuals were included where appropriate in the three groupings for all statistical analyses (e.g. all those scored as "possibly not British" were included in the "not British" group). Residual rickets and tuberculosis were both rarely observed pathologies; thus, we used Fisher's exact tests to assess differences in the presence of these diseases. Chi-square tests were also used to assess differences in the sex distributions between the three groups. These statistical tests are appropriate given that we are assessing the associations among and between categorical variables (most of which are binary, i.e. the sex and disease variables).

Differences in survival among the three groups were assessed using Kaplan-Meier survival analysis with a log rank test and using pooled data on age from all three groups. Analysis was performed using SPSS version 23. Kaplan-Meier analyses were performed separately for samples including: individuals of all ages; adults only (≥15 years old); female adults; and male adults.

The differences in risks of mortality between all paired combinations of groups (regional local vs. British; regional local vs. non-British; British vs. non-British) were assessed by pooling the adult (≥15 years old) data to estimate the Gompertz hazard of adult mortality and by modeling group as a covariate affecting the baseline Gompertz hazard. The Gompertz hazard is a two-parameter, parsimonious model of adult mortality: $h(a) = \alpha e^{\beta a}$. For each paired combination of groups, all individuals in one group were assigned a covariate score of 0, and all members of the other group were assigned a score of 1, and the group covariate was modeled using a proportional hazards specification. Table 6 specifies which group, from each pair, was assigned the covariate value of 1 for analysis. For example, to evaluate differences in mortality between regional locals and non-British people, the regional locals were coded as "0" and the non-British individuals were coded as "1"; in this case, a positive estimate for the parameter representing the effect of the covariate would suggest non-British people were at an increased risk of death compared to regional locals, while a negative estimate would suggest that non-British people were at a decreased risk of death. Hazards models such as this can be applied to relatively small samples, as they smooth random variation in mortality data (Gage 1988). Parameters were estimated using maximum likelihood analysis with the program *mle* (Holman 2005). The fit of the full model with the regional group covariate compared to a reduced model in which the value of the parameter representing the regional covariate is set equal to 0 was assessed using a likelihood ratio test (LRT). The LRT tests the null hypothesis that the regional group was not associated with elevated nor decreased risks of death. The LRT was computed as follows: LRT = $-2[\ln(L_{reduced}) - \ln(L_{full})]$, where LRT approximates a χ^2 distribution with df=1. Similarly to the survival analyses described above, the hazards model was applied separately to samples including: all adults; adult females, and adult males.

Though we are wary of relying too heavily on *p*-values to evaluate our findings, given the problems associated with conventional null hypothesis significance testing (see for example, Lang et al. 1998; Rothman 1998; Goodman 1999; Cohen 2011; Trafimow and Marks 2015) and our relatively small sample sizes, we do consider *p*-values equal to or less than 0.05 to suggest a significant difference and values of 0.1 or less to indicate a trend worthy of discussion and potential future study.

Results

The results of the survival and hazards analyses are provided in Tables 5 and 6. Kaplan-Meier survival analyses (Table 5) using a sample that includes subadults (<15 years old) does not reveal significant differences among the three groups. However, using a sample that includes just the adults (males, females, and adults of "indeterminate" sex ≥15 years old), reveals a difference in survivorship between those from Britain and people who had migrated to it (*P*=0.088). The estimated mean survival age is lowest for the non-British people, though there is considerable overlap in the 95% confidence intervals for all groups. When adult males and females are assessed separately, the results are not statistically significant. Mean survival age for males is similar across all three groups. For females, regional locals had the highest mean survival age, and non-Britons had the lowest; however, the 95% confidence intervals for all groups overlap considerably.

For the comparison of regional local vs. non-British using a pooled sex sample (regional local = 0, non-British = 1), the positive value of the group covariate indicates higher risks of mortality for non-British people compared to the regional locals (*P*=0.03). A similar pattern was observed when analyses were restricted to females (*P*= 0.06), but no differences in mortality among the groups were found for males (Table 6). No significant differences in any of the samples were observed between regional locals and British, or between British and non-British people for disease variables and indicators of stress. Overall, these results suggest that, at least among females, non-British people fared relatively poorly with respect to risk of mortality compared to regional locals (Table 6). We note that the small sample sizes might be influencing these analyses to an unknown degree.

Statistically significant differences in disease variables among the regional locals, British, or non-British people were observed for periosteal new bone formation, rib lesions, and residual rickets (frequencies are shown in Tables 7 and 8, and the results of statistical analyses are shown in Table 9). For each of these variables, regional locals have the lowest frequencies of palaeopathology. For residual rickets, regional and British individuals have similarly low frequencies. However, for periosteal new bone formation and rib lesions, the British are similar to non-Britons in having relatively high frequencies compared to regional locals. No significant differences in tuberculosis frequencies are observed, though we note that very few people in general had signs of this disease in their skeleton (Table 7 and 8). However, it is interesting to note that non-British people were more likely to show evidence for tuberculosis (Table 8).

Substantial differences among the regional groups are also observed for dental health pathologies (periodontal disease, carious lesions, and dental calculus) (Table 10). In general, Britons had the highest frequencies of all dental health pathologies. Periodontal disease frequencies were lowest among non-Britons, but for both carious lesions and dental calculus, regional locals and non-Britons had similar relatively low frequencies (compared to British locals).

As expected, the sex distributions varied across the three groups (Tables 3 and 9, P= 0.075). The non-British sample contains the highest proportion of females and the British sample has the lowest. As shown in Table 9, the difference in sex distribution between the British and non-British samples is statistically significant (P = 0.03).

Discussion

The most important finding of this analysis is that the disparity between late Iron Age and Romano-British patterns of disease, particularly for periosteal new bone formation, rib lesions and residual rickets appears to be primarily driven by migrants from elsewhere in the Empire. This explanation has been one of several alternatives posited within the literature (amongst others, Redfern and DeWitte 2011a; Roberts and Cox 2003), and our research has demonstrated that this hypothesis should come to the fore in future interpretations. This result has implications for how living environments (e.g. urban centres) and the consequences of Roman colonisation are interpreted and understood (Gowland and Redfern 2010), and prompts us to re-evaluate the conclusion that Roman colonisation saw a general increase in disease. The result is not a cause-and-effect outcome of conquest and colonisation; instead, these data reflect greater variation in disease states in correlation with the increasingly diverse population composition. This is not to say that the urban centres did not negatively impact their inhabitants, but rather that these centres altered disease patterns bi-directionally between different communities. The results indicate a mixture of time spent in Britain and elsewhere in the Empire, with the negative long-term consequences of people's childhood environments being 'seen' in Britain, because this is where these migrants died.

Each new community and settlement created by migrants from within and outside of Britain, as well as local indigenous people, would have created heterogeneous disease environments, because each person would have had a unique migration experience and individual history of disease. The ability for an individual to create changes at the population level is frequently seen in migrant health studies; for example, it only takes one person carrying an infectious disease for many thousands to become infected (Ahonen et al. 2007; Mascie-Taylor and Krzyżanowska 2017; Odone et al. 2015). Such a situation may explain why patterns of disease in Roman Britain have been found to substantially differ between settlement types, but also between those supposedly of the 'same' type, such as urban settlements (Bonsall 2013; Rohnbogner and Lewis 2016; Redfern et al. 2015).

Differences in disease frequencies between people whose childhoods were spent in Britain versus those who grew up elsewhere may be evidenced by variations in the frequency of non-specific indicators of stress (e.g., enamel hypoplastic defects) (Goodman and Martin 2002). Our finding that non-British migrants had the highest rates of enamel hypoplastic defects (though not statistically significant in our analyses), is notable as this is one of the most frequently observed differences between pre- and post-Conquest populations (e.g. Peck 2009; Redfern 2007; Roberts and Cox 2003). It is problematic to interpret this disease variable as evidence of the detrimental impact of new settlement types and increased childhood adversity in Britain (Redfern 2007; Redfern and DeWitte 2011a; Redfern et al. 2012, 2015; Redfern and Roberts 2005; Rohnbogner 2015). Instead, as Gowland and Redfern (2010; Redfern and Gowland 2011) proposed, enamel hypoplastic defects record childhood insults to health experienced elsewhere in the Empire, compounded by different living environments and childcare practices, the negative impacts of which are seen in the high frequencies of enamel hypoplastic defects reported from other locales in the Empire, especially Italy (amongst others, Caldarini et al. 2006; Henneberg and Henneberg 2005; Paine et al. 2009; Gowland and Garnsey 2010). Crucially, these findings underline the point that the Roman Empire encompassed great diversity in terms of economic development, compounded by stark environmental and sociocultural differences between the Mediterranean heartland, which had large established urban settlements for many hundreds of years compared to the peripheral territories (Garnsey and Saller 1987; Laurence and Berry 1998; Mattingly 2010; Nevett and Perkins 2000; Woolf 1998).

The higher frequency of periosteal new bone formation in migrants compared to regional local and British individuals may reflect a response to being confronted with new environmental conditions. The transformation of Britain's wider and local living environments, food-ways and population under Roman rule may also explain why a high frequency of periosteal new bone formation was observed in British-locals. For example, the increase in rib lesions hints at a higher prevalence of specific infectious disease, exposure to pollution through work, and greater indoor pollution, probably exacerbated by a decreased local air quality due to people living in larger communities than ever before (Roberts et al. 1994, 1998; Roberts and Lewis 2002).

Each disease variable studied provides its own perspective on migrant health and the impact on the indigenous population through the creation of these new communities and living environments. One of the key changes post-conquest is the decline in dental health for both sexes and all age-groups (Redfern 2007; Roberts and Cox 2003). The marked increase in carious lesions and dental calculus follows a post-Conquest shift in dietary patterns from the over-whelming terrestrial and low sugar food-ways of the late Iron Age communities, which display little intra- or inter-community variation, to the post-Conquest diet with increased levels of marine resources, more sugar (e.g. dried fruits, with honey frequently used as a sweetener) and greater variation between age, sex and status groups, as well as between settlement types (e.g. rural-urban diets Cool 2006) (Bonsall 2014; King 1999a, 1999b, 2001; Locker 2007; Peck 2009; Redfern et al. 2012; van der Veen et al. 2008).

Another interesting finding is the relationship between migrant status and residual rickets. This metabolic disease is caused by a deficiency of vitamin D, usually sustained because of a lack of exposure to sunlight (Brickley and Ives 2008; Brickley et al. 2010: 78-81). Research has demonstrated that being vitamin D deficient increases the likelihood of developing poor general health, as it negatively impacts on a person's immune system (Brickley et al. 2014; Snoddy et al. 2016). The clinical literature has also associated high lead exposure with the development of rickets in children (Caffey 1938; Gordon and Whitehead 1949), and this period saw an increase in the use of lead (e.g. water-pipes and especially the consumption of bio-available lead compounds), a trend evidenced by a rise in lead levels in human remains (Aufderheide et al. 1992; Budd et al. 2004; Lessler 1983; Montgomery et al. 2010; Nriuagu 1983; Retief 2005; Scarborough 1984).

The limb and spinal deformities caused by vitamin D deficiency were known to the Romans, being described in two medical texts (e.g. Soranus 1991: Book II) (Rajakumar 2003). Ancient historians have proposed that even sun-rich cities such as Rome were prime centres for the conditions which create rickets, because of dietary insufficiencies produced by the population's over-reliance on cereals and cultural practices such as sun avoidance and clothing (Gowland and Redfern 2010; Minozzi et al. 2012; Molto 2000; Soliman El-Banna et al. 2014).

Prior to this study, the presence of rickets in Roman Britain was understood to be another indicator of significant post-Conquest change, and the rare cases of this disease suggest that cultural buffering may have influenced this result (Gowland and Redfern 2010; Redfern and DeWitte 2011a, 2011b; Redfern and Gowland 2011; Roberts and Cox 2003; Rohnbogner 2015). However, our results suggest that the increase in prevalence of rickets reflects childhoods spent elsewhere in the Empire. For example, the burial of a 14 year old girl from Roman London whose isotope results were consistent with a childhood spent in the southern Mediterranean, and who had lived in London for at least four years before her death (Arthur et al. 2016; Redfern et al. 2016), exhibited mild bowing deformities consistent with rickets in younger childhood (Brickley and Ives 2008: 97-100; Brickley et al. 2010; Redfern et al. 2017).

There is a marked increase in the number of individuals with tuberculosis from the late Iron Age (Redfern and DeWitte 2011a; Roberts and Cox 2003: 120), but there was no statistical difference between the groups for tuberculosis, although non-British people showed a greater prevalence of the disease along with rib lesions (Kelley and Micozzi 1984;

Mariotti et al. 2015; Roberts et al. 1994; Santos and Roberts 2001). There are many reported cases of tuberculosis from across the Roman Empire, including Rome (amongst others, Canci et al. 2006; Hajdu et al. 2012; Hlavenková et al. 2015; Rubini et al. 2014). The Empire produced and enabled multiple causes and pathways of infection: urban settlements, many of which had poor sanitation, with homes densely packed together; long- and short-distance population movement (e.g. trade), including the forced or free migration of vulnerable people (e.g. migrants and the enslaved) to new environments, many of which were experiencing socio-economic stress (i.e. structural violence); and congenital infection due to maternal infection during pregnancy (Eddy 2015; Farmer 2004a, 2004b; Mittal et al. 2014).

One of the more surprising findings of our study was that female migrants had a higher mortality risk, despite having the biological advantage of an enhanced immune system (Gubbels-Bupp 2015; Jaillon et al. 2017). Earlier work in Dorset, demonstrated that it was adult males rather than females who had a higher mortality risk (Redfern and DeWitte 2011b), but this work was unable to distinguish between migrants and locals, as only a few individuals in the region had been analysed for mobility (Richards et al. 1998). We suggest that our new result provides a more nuanced insight into intra-sex differences in health, because only female migrants show a higher prevalence of pathologies. There is increasing evidence for female mobility in the Roman Empire, as part of the military community, as slaves, or due to family circumstances, occupations, and economic activities (e.g. merchants) (Allason-Jones 2005; Allison 2006; Becker 2006; Greene 2012, 2013; Hemelrijk 2015; Kleijwegt 2012; Saller 1998). Within Roman society, females were seen as 'less' than males and their social, economic and political freedoms were curtailed, resulted in embedded structural inequalities (Redfern forthcoming). For many females (free or enslaved) their lives were tied to the domestic sphere (Allason-Jones 2005; Dixon 2001; Evans Grubb 2002; Hemelrijk 2015; Saller 1998), which increased the likelihood of developing diseases associated with pollution (i.e. rib lesions), because of living and working in homes with poor ventilation (Roberts and Lewis 2002). Such a life-style could also limit their access to sunlight, thus increasing their risk of developing vitamin D deficiency (Brickley and Ives 2008: 77-82; Brickley et al. 2014), particularly in Britain where during the autumn and winter months, the sunlight is not strong enough for the body to metabolise the necessary quantities of vitamin D to ensure good health (Diffey 2013). This would have been a greater risk for those individuals from the Mediterranean and the near East with darker skin pigmentation (Chandler 2001; Eckardt et al. 2014; Leach et al. 2009). Isotopic data from across the Empire have established that in many locales, including Britain, female diets were less diverse and more cereal-based than adult males (Powell et al. 2014) (see also, Prowse et al. 2004, 2005). Such a diet would additionally increase the risk of females developing an iron deficiency, because of the iron inhibiting chelates found in cereals (Stuart-Macadam and Kent 1992: 83, 137).

Unfortunately, due to the small sample size and because the majority of these samples have not been precisely dated (either stratigraphically or using radiocarbon), we are unable to test whether there are temporal changes throughout the Roman period in Britain. The predominance of cremation burial during the earlier period of Roman occupation also creates a bias towards skeletal samples dating to later Roman Britain. Nor were we able to investigate detailed differences in health between rural and urban settlements due to a current dearth in isotopically-tested samples from rural sites. It is likely that this situation will improve in the near future with an increasing research focus on Roman rural settlements (Smith et al. 2016).

Conclusions

This analysis is the first bioarchaeological study to unite osteological and isotope data to investigate how migrant and local population influenced each other's patterns of disease. For Roman Britain, the changes in disease frequencies traditionally used as evidence for the transformation in living environment and culture instead relate to childhoods and lives spent outside of the province. Childhoods across the Empire were often compromised by disease, and in some instances by childcare practices.

Although urban centres must have played an important role in changing patterns of disease in Roman Britain, it was the composition of the people living within these and other settlements across Britain which really produced a transformation in the bioarchaeological record. The unique, heterogeneous life-ways of migrants, fundamentally altered disease transmission and frequencies, changed mortality rates and in conjunction with the introduction of new food-and life-ways, transformed disease patterns from the late Iron Age. The development of new settlements, inhabited by internal migrants and those from overseas, created new environments which posed health challenges for all, despite many growing-up in some of the most urbanised places in Europe. Therefore, many of the key indicators of changing disease frequencies associated with the Roman conquest and colonisation of Britain actually reveal more about the increased heterogeneity of communities in Britain.

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Figure captions

Figure 1. Map showing the location of the towns selected for analysis in Roman Britain. Each site report listed in Table 1 has a detailed location map. Drawn by J. Davis from a base-map made available at http://www.d-maps.com/carte.php?num_car=5585&lang=en

Table 1. List of sites used in the study

City/ County	Site name	Sample size	Reference
Dorset	Poundbury Camp, Dorchester	3	Richards et al. (1998)
Gloucestershire	London Road, Gloucester	13	Simmonds et al. (2008)
Hampshire	Winchester	40	Booth et al. (2010); Eckardt et al. (2009)
Lancashire	Hollow Banks, Scorton	10	Eckardt et al. (2015)
London	1-4 Giltspur Street	2	Shaw et al. (2016)
	49-55 Mansell Street	3	Shaw et al. (2016)
	65-73 Mansell Street	1	Shaw et al. (2016)
	60 London Wall	2	Shaw et al. (2016)
	Broadgate	1	Shaw et al. (2016)
	Cotts House	1	Shaw et al. (2016)
	Great Dover Street	2	Shaw et al. (2016)
	Harper Road	1	Shaw et al. (2016)
	Hooper Street	4	Shaw et al. (2016)
	Lant Street	19	Redfern et al. (2016)
	Spitalfields Market	5	Bell, pers comm; Montgomery et al. (2010)
	St Bartholomew's Hospital	1	Shaw et al. (2016)
Yorkshire	Bainesse Farm, Catterick	20	Chenery et al. (2011)
	Dere Street, Catterick	2	Chenery et al. (2011)
	Honeypot Road, Catterick	2	Chenery et al. (2011)
	Trentholme Drive, York	9	Leach et al. (2009)
	3 Driffield Terrace, York	4	Leach et al. (2009); Montgomery et al. (2011)
	6 Driffield Terrace, York	18	Leach et al. (2009)

Table 2. Stable isotope data organised by location, site and individual (data from sources listed in Table 1)

County	Site name and modern city/town	Context (or grave) number	Sex	Local status (9 = no data, 1 = regional local, 1.5 = possible regional local, 2 = British, 3 = inconsistent with Britain, 3.5 = possible inconsistent with Britain)	Regional local (1 = local to region, 2 = British and non-local)	British (1 = local to region or Britain, 2 = non-British)
Dorset	Poundbury Camp, Dorchester	235	Female	3.5	2	2
		862	Female	3.5	2	2
		1255	Subadult	3.5	2	2
Gloucestershire	London Road, Gloucester	1103	Female	1	1	1
		1127	Male	1	1	1
		1131	Female	2	2	1
		1181	Female	1	1	1
		1216	Male	2	2	1
		1238	Male	2	2	1
		1328	Male	1	1	1
		1364	Female	2	2	1
		1518	Male	2	2	1
		1539	Female	1	1	1
		1541	Male	2	2	1
		1544	Male	1	1	1

		1553	Male	1	1	1
Hampshire	Winchester	12	Male	1	1	1
		84	Female	1	1	1
		118	Subadult	1	1	1
		119	Female	3	2	2
		212	Female	1	1	1
		271	Female	3	2	2
		281	Male	2	2	1
		435	Female	1	1	1
		489	Male	2	2	1
		566	Male	2	2	1
		661	Female	1	1	1
		683	Male	2	2	1
		776	Male	2	2	1
		806	Female	1.5	1	1
		812	Male	1	1	1
		861	Male	2	2	1
		862	Male	1	1	1
		874	Subadult	1	1	1
		926	Subadult	1	1	1
		932	Male	1	1	1
		1026	Subadult	1	1	1
		1084	Female	1	1	1
		1091	Female	1	1	1
		1094	Female	2	2	1
		1114	Female	1	1	1
		1119	Male	3	2	2
		1133	Subadult	1	1	1
		1134	Female	1	1	1
		1197	Female	2	2	1
		1207	Female	1	1	1
		1227	Female	1	1	1
		1244	Subadult	1	1	1
		1271	Male	1	1	1
		1277	Male	2	2	1

		1289	Male	1	1	1
		1517	Male	1	1	1
		1697	Male	1	1	1
		1761	Subadult	1	1	1
		1870	Subadult	1	1	1
		1894	Male	2	2	1
Lancashire	Hollow Banks, Scorton	502 (grave 1)	Unknown	3	2	2
		511 (grave 13)	Male	1	1	1
		523 (grave 2)	Male	3	2	2
		529 (grave 5)	Unknown	3	2	2
		535 (grave 6)	Male	3	2	2
		541 (grave 10)	Unknown	3	2	2
		565 (grave 11)	Female	3	2	2
		594 (grave 12)	Male	1	1	1
		600 (grave 7)	Male	3	2	2
London	1-4 Giltspur Street	599	Female	1	1	1
		709	Female	1	1	1
	49-55 Mansell Street	163	Female	1	1	1
		390	Female	2	2	1
		724	Male	1	1	1
	65-73 Mansell Street	37	Male	1	1	1
	60 London Wall	695.5	Male	9		
		803.6	Male	1	1	1
	Broadgate	400	Subadult	3	2	2
	Cotts House	30	Male	1	1	1
	Great Dover Street	150	Subadult	1	1	1
	Great Dover Street	325	Female	3	2	2
	Harper Road	311	Female	1	1	1
	Hooper Street	518	Female	1	1	1
		652	Male	1	1	1
		1407	Female	1	1	1
		1673	Female	1	1	1
	Lant Street	13	Female	2	2	1
		27	Male	2	2	1

		58	Female	2	2	1
		103	Subadult	3	2	2
		128	Male	9		
		154	Male	3.5	2	2
		157	Female	3.5	2	2
		208	Female	3	2	2
		253	Female	9		
		321	Male	3.5	2	2
		339	Subadult	3	2	2
		358	Male	9		
		369	Subadult	9		
		379	Male	2	2	1
		385	Subadult	3	2	2
		407	Male	9		
		434	Female	3.5	2	2
		440	Male	9		
		465	Male	3.5	2	2
	Spitalfields Market	1988	Female	3	2	2
		15903	Female	3	2	2
		23873	Subadult	1	1	1
		34209	Male	1	1	1
		34245	Male	3	2	2
	St Bartholomew's Hospital	182	Female	1	1	1
Yorkshire	Bainesse Farm, Catterick	255	Female	1	1	1
		277	Male	2	2	1
		324	Male	2	2	1
		422	Female	1	1	1
		475	Male	1	1	1
		632	Subadult	1	1	1
		678	Male	1	1	1
		679	Subadult	2	2	1
		709	Male	1	1	1
		746	Male	1	1	1
		756	Female	1	1	1
		801	Female	2	2	1

	812	Subadult	1	1	1
Catterick Bridge, Catterick	37	Male	1	1	1
	77	Female	1	1	1
	136	Male	1	1	1
	166	Male	1	1	1
	389	Subadult	1	1	1
	484	Subadult	1	1	1
Dere Street, Catterick	P IV 9	Male	1	1	1
Honeypot Road, Catterick	941	Male	1	1	1
	942	Male	1	1	1
Trentholme Drive, York	4	Male	1	1	1
	153	Female	1	1	1
	157	Male	1	1	1
	173	Male	1	1	1
	411	Male	2	2	1
	466	Male	1	1	1
	513	Female	2	2	1
	608	Male	2	2	1
	708	Male	2	2	1
3 Driffield Terrace, York	16	Male	1	1	1
	37	Male	1	1	1
6 Driffield Terrace, York	1	Male	1	1	1
	2	Male	2	2	1
	4	Male	1	1	1
	6	Male	1	1	1
	7	Male	1	1	1
	8	Male	2	2	1
	9	Male	3	2	2
	12	Male	1	1	1
	14	Male	2	2	1
	15	Male	2	2	1
	17	Male	1	1	1
	18	Male	2	2	1
	19	Male	2	2	1
	20	Male	2	2	1
	21	Male	2	2	1

22	Male	2	2	1
23	Male	2	2	1
24	Male	3	2	2

Table 3: Sample sizes for each group. The ‘regional local ‘sample includes ’possible’ regional locals, and the ‘non-British’ sample includes those with values that are possibly inconsistent with Britain.

	Adult Male	Adult Female	Adult Indeterminate Sex	Subadult	Total
Regional Local	42 (50.6%)	27 (32.53%)	0 (0%)	14 (16.87%)	83
British	29 (74.36%)	9 (23.08%)	0 (0%)	1 (2.56%)	39
Non-British	10 (34.48%)	11 (37.93%)	3 (10.34%)	5 (17.24%)	29

Table 4: The categories of diseases included in the study.

Category	Diseases	Reference
Indicators of Stress	Cribra orbitalia, porotic hyperostosis, enamel hypoplastic defects and periosteal new bone formation	Goodman and Martin (2002); Hillson (2005); Humphrey and King (2000); King et al. (2005); Stuart-Macadam and Kent (1992); Rivera and Mirazón Lahr (2017); Walker et al. (2009); Weston (2008)
Metabolic diseases	Scurvy, osteomalacia, rickets and residual rickets	Brickley and Ives (2008); Brickley et al. (2010, 2014)
Specific infectious disease	Tuberculosis	Lewis (2011); Mariotti et al. (2015); Roberts and Buikstra (2008); Sandgren et al. (2014)
Non-specific infectious disease	Rib lesions	Kelley and Micozzi (1984); Roberts et al. (1994, 1998); Weston (2008, 2012)
Dental health	Periodontal disease, carious lesions, dental calculus	Hillson (2005)

Table 5: Kaplan-Meier survival analysis results.

Samples		Mean survival time	95% CI	Mantel-Cox *p*-value
All ages	**Regional local (n = 67)**	30.58	26.77 - 34.40	0.25
	British (n = 31)	34.40	30.17 - 38.63	
	Non-British (n = 28)	29.23	24.89 - 33.57	
Adults (includes "indeterminate")	**Regional local (n = 53)**	36.93	33.93 - 39.92	0.088
	British (n = 30)	35.1	30.96 - 39.24	
	Non-British (n = 25)	31.46	27.42 - 35.49	
Males	**Regional local (n = 33)**	36.23	32.47 - 39.98	0.925
	British (n = 24)	35.23	30.78 - 39.68	
	Non-British (n = 9)	35.39	28.07 - 42.71	
Females	**Regional local (n = 20)**	38.08	33.03 - 43.12	0.435
	British (n = 6)	34.58	23.06 - 46.11	
	Non-British (n = 11)	32.36	27.22 - 37.51	

Table 6: Maximum likelihood estimates (with standard error in parentheses) of the effect of the covariate (individuals in the group indicated with an * in the first column were assigned a covariate score of 1) on the Gompertz model and the results of the likelihood ratio tests.

	Samples	**Covariate Effect**	**-2LLR**	***p***
Pooled sexes	Regional local (n = 53) vs. British* (n = 30)	0.105 (0.21)	0.21	0.65
	Regional local (n = 53) vs. non-British* (n = 25)	0.55 (0.25)	4.57	0.03
	British (n = 30) vs. non-British* (n = 25)	0.41 (0.28)	2.10	0.15
Males	Regional local (n = 33) vs. British* (n = 24)	0.066 (0.25)	0.06	0.81
	Regional local (n = 33) vs. non-British* (n = 9)	0.11 (0.41)	0.08	0.78
	Britain local (n = 24) vs. non-British* (n = 9)	0.033 (0.39)	0.007	0.93
Females	Regional local (n = 20) vs. British* (n = 6)	0.097 (0.39)	0.043	0.84
	Regional local (n = 20) vs. non-British* (n = 11)	0.79 (0.41)	3.52	0.06
	British (n = 6) vs. non-British* (n = 11)	0.64 (0.83)	1.31	0.25

Table 7: Disease and dental health frequencies by stable isotope grouping. % = Percentage of regional samples with or without a disease/lesion.

	Cribra orbitalia		Porotic hyperostosis		Periosteal new bone formation		Rib lesions		Enamel hypoplastic defects	
	Absent	**Present**	**Absent**	**Present**	**Absent**	**Present**	**Absent**	**Present**	**Absent**	**Present**
Regional Local	18 (48.6%)	19 (51.4%)	61 (84.7%)	11 (15.3%)	55 (71.4%)	22 (28.6%)	67 (95.7%)	3 (4.3%)	54 (68.4%)	25 (31.6%)
British	15 (57.7%)	11 (42.3%)	30 (85.7%)	5 (14.3%)	19 (51.4%)	18 (48.6%)	24 (72.7%)	9 (27.3%)	26 (68.4%)	12 (31.6%)
Non-British	9 (60%)	6 (40%)	22 (81.5%)	5 (18.5%)	15 (51.7%)	14 (48.3%)	22 (75.9%)	7 (24.1%)	16 (55.2%)	13 (44.8%)

Table 8: Specific diseases by stable isotope grouping

	Residual Rickets		Tuberculosis	
	Absent	**Present**	**Absent**	**Present**
Regional Local	74 (98.7%)	1 (1.3%)	80 (98.8%)	1 (1.2%)
British	34 (97.1%)	1 (2.9%)	39 (100%)	0 (0%)
Non-British	25 (86.2%)	4 (13.8%)	27 (96.4%)	1 (3.6%)

Table 9: Results of comparisons of diseases and sex distributions. Unless otherwise indicated, p-values for Chi-square tests are shown. The p-values for Fisher's exact tests (for residual rickets and tuberculosis, both of which were rarely observed) are indicated by an asterisk *.

Variable	**Comparison**	***p*-value**
Cribra orbitalia	Regional × British × non-British	0.67
Porotic hyperostosis	Regional × British × non-British	0.89
Periosteal new bone formation	Regional × British × non-British	0.05
Rib lesions	Regional × British × non-British	0.002
Enamel hypoplastic defects	Regional × British × non-British	0.41
	Regional/British × non-British	0.18
Residual rickets	Regional × British × non-British	0.017*
Tuberculosis	Regional × non-British	0.45*
Periodontal disease	Regional × British × non-British	0.028
	Regional × British	0.28
	Regional × non-British	0.05
	British × non-British	0.01
Carious lesions	Regional × British × non-British	0.096
	Regional × British	0.04
Dental calculus	Regional × British × non-British	0.187
	Regional × British	0.08
Sex	Regional × British × non-British	0.075
	Regional × British	0.1
	Regional × non-British	0.28
	British × non-British	0.03

Table 10 : Dental health frequencies given by stable isotope groupings

	Periodontal disease		Carious lesions		Dental calculus	
	Absent	**Present**	**Absent**	**Present**	**Absent**	**Present**
Regional Local	15 (26.3%)	42 (73.7%)	43 (54.5%)	36 (45.6%)	27 (34.2%)	52 (65.8%)
British	5 (16.1%)	26 (83.9%)	13 (34.2%)	25 (65.8%)	7 (18.4%)	31 (81.6%)
Non-British	12 (48%)	13 (52%)	16 (55.2%)	13 (44.8%)	10 (34.5%)	19 (65.5%)

Map of Britain showing the location of the sites used in the study. Drawn by J. Davis based on http://d-maps.com ©Museum of London

189x234mm (300 x 300 DPI)

City/ County	Site name	Context (or grave) number
Dorset	Poundbury Camp, Dorchester	235
		862
		1255
Gloucestershire	London Road, Gloucester	1103
		1127
		1131
		1181
		1216
		1238
		1328
		1364
		1518
		1539
		1541
		1544
		1553
Hampshire	Winchester	12
		84
		118
		119
		212
		271
		281
		435
		489
		566
		661
		683
		776
		806
		812
		861
		862
		874
		926
		932
		1026
		1084
		1091
		1094
		1114
		1119
		1133

		1134
		1197
		1207
		1227
		1244
		1271
		1277
		1289
		1517
		1697
		1761
		1870
		1894
Lancashire	Hollow Banks, Scorton	502 (grave 1)
		511 (grave 13)
		523 (grave 2)
		529 (grave 5)
		535 (grave 6)
		541 (grave 10)
		565 (grave 11)
		594 (grave 12)
		600 (grave 7)
London	1-4 Giltspur Street	599
	1-4 Giltspur Street	709
	49-55 Mansell Street	163
	49-55 Mansell Street	390
	49-55 Mansell Street	724
	60 London Wall	695.5
	60 London Wall	803.6
	65-73 Mansell Street	37
	Broadgate	400
	Cotts House	30
	Great Dover Street	150
	Great Dover Street	325
	Harper Road	311
	Hooper Street	518
	Hooper Street	652
	Hooper Street	1407
	Hooper Street	1673
	Lant Street	13
	Lant Street	27
	Lant Street	58
	Lant Street	103
	Lant Street	128
	Lant Street	154
	Lant Street	157

	Lant Street	208
	Lant Street	253
	Lant Street	321
	Lant Street	339
	Lant Street	358
	Lant Street	369
	Lant Street	379
	Lant Street	385
	Lant Street	407
	Lant Street	434
	Lant Street	440
	Lant Street	465
	Spitalfields Market	1988
	Spitalfields Market	15903
	Spitalfields Market	23873
	Spitalfields Market	34209
	Spitalfields Market	34245
	St Bartholomew's Hospital	182
Yorkshire	Bainesse Farm, Catterick	255
		277
		324
		422
		475
		632
		678
		679
		709
		746
		756
		801
		812
	Catterick Bridge, Catterick	37
		77
		136
		166
		389
		484
	Dere Street, Catterick	P IV 9
	Honeypot Road, Catterick	941
		942
Yorkshire	Trentholme Drive, York	4
		153
		157
		173
		411
		466
		513

	608
	708
3 Driffield Terrace, York	16
	37
6 Driffield Terrace, York	1
	2
	4
	6
	7
	8
	9
	12
	14
	15
	17
	18
	19
	20
	21
	22
	23
	24

Sex (F: female; M: Male; S: subadult; U: unknown)	Age midpoint	Cribra orbitalia
F	40.5	0
F	40.5	9
S	10	1
F	30.5	9
M	21.5	0
F	21.5	9
F	30.5	0
M	40.5	1
M	40.5	9
M	21.5	1
F	21.5	0
M	30.5	0
F	30.5	1
M	21.5	1
M	30.5	0
M	30.5	0
M	50.5	0
F	adult	1
S	0.9	9
F	30.5	9
F	60	9
F	30.5	9
M	50.5	9
F	50.5	9
M	50.5	9
M	30.5	9
F	50.5	9
M	50.5	0
M	adult	0
F	60	9
M	50.5	9
M	60	9
M	40.5	9
S	9	9
S	13	9
M	21.5	0
S	5.5	1
F	30.5	9
F	21.5	9
F	adult	1
F	30.5	9
M	50.5	9
S	6.5	9

F	40.5	9
F	60	9
F	adult	9
F	40.5	9
S	13	1
M	50.5	9
M	40.5	9
M	40.5	1
M	60	9
M	40.5	9
S	6	9
S	9.5	9
M	21.5	9
U	21	9
M	30	9
Male	30	9
U	30	9
M	30	0
U	30	9
F	21	9
M	30	9
M	30	0
F	40.5	1
F	40.5	9
F	adult	0
F	40.5	0
M	50.5	1
M	40.5	1
M	30.5	1
M	50.5	9
S	8	1
M	21.5	1
S	7	9
F	21.5	9
F	x	1
F	40.5	1
M	adult	0
F	adult	9
F	adult	1
F	32	0
M	40.5	0
F	32	1
S	16	1
M	37	1
M	22	1
F	27	0

F	42	9
F	37	1
M	42	0
S	15	9
M	Adult	0
S	15	0
M	Adult	0
S	14	9
M	27	9
F	40.5	0
M	37	1
M	42	1
F	40.5	9
F	21.5	0
S	5	9
M	30.5	0
M	50.5	1
F	21.5	9
F	37	9
M	40	9
M	22.5	9
F	50.5	9
M	22.5	9
S	5	9
M	42.5	9
S	13.5	9
M	45	9
M	50.5	9
F	27.5	9
F	adult	9
S	5	9
M	Adult	9
F	27.5	9
M	22.5	1
M	25	9
S	4.5	9
S	2.25	9
M	40	9
M	22.5	0
M	adult	0
M	Adult	1
F	Adult	1
M	Adult	0
M	Adult	1
M	Adult	1
M	Adult	1
F	Adult	1

M	Adult	0
M	Adult	1
M	40.5	0
M	30.5	0
M	x	9
M	40.5	0
M	40.5	1
M	30.5	0
M	40.5	0
M	40.5	0
M	Adult	0
M	Adult	0
M	30.5	1
M	21.5	0
M	40.5	0
M	19	1
M	30.5	0
M	30.5	0
M	40.5	1
M	30.5	1
M	21.5	0
M	21.5	0

Porotic hyperostosis	Periosteal new bone formation	Rib lesions	Scurvy	Osteomalacia
9	1	1	0	0
0	1	1	0	0
0	0	1	0	9
0	1	0	0	0
0	0	0	0	0
0	1	0	0	0
0	1	1	0	0
0	1	1	0	0
0	0	0	0	0
0	1	0	0	0
0	0	0	0	0
0	1	0	0	0
0	1	0	0	0
0	1	0	0	0
0	1	0	0	0
0	1	0	0	0
0	1	0	0	0
0	1	9	0	0
0	0	0	0	0
0	1	0	0	0
0	0	0	0	0
0	0	0	0	0
0	0	0	0	0
0	0	0	0	0
0	1	0	0	0
0	0	0	0	0
0	0	0	0	0
0	0	0	0	0
0	0	9	0	0
0	0	0	0	0
0	1	9	0	0
0	0	0	0	0
9	1	0	0	0
0	0	0	0	0
0	0	9	0	0
0	0	0	0	0
0	0	0	0	0
0	0	0	0	0
0	0	0	0	0
0	0	9	0	0
0	0	0	0	0
0	0	0	0	0
0	0	0	0	0

1	1	1	0	0
0	0	0	0	0
0	0	9	0	0
0	0	0	0	0
0	0	0	0	0
0	0	0	0	0
0	0	9	0	0
1	0	0	0	0
0	1	0	0	0
0	0	0	0	0
0	0	9	0	0
0	0	0	0	0
0	0	0	0	0
0	0	0	0	0
0	0	0	0	0
0	1	0	0	0
9	0	0	0	9
0	1	0	0	0
0	0	0	0	0
0	0	0	0	0
0	1	0	0	0
0	1	0	0	0
1	1	0	0	0
0	0	0	0	0
0	0	0	0	0
0	1	0	0	0
0	0	0	0	0
1	0	9	0	9
1	0	9	0	9
0	0	0	0	0
1	0	0	0	0
1	1	0	0	0
0	0	0	0	0
0	0	0	0	0
0	0	0	0	0
1	0	0	0	0
0	0	0	0	0
0	0	0	0	0
0	0	0	0	0
0	1	0	0	0
1	1	1	0	0
0	1	1	0	0
1	1	1	0	9
0	0	1	0	0
1	1	0	0	0
0	0	0	0	0

0	0	1	0	0
1	1	1	0	0
0	1	0	0	0
0	0	0	0	9
1	1	0	0	0
0	0	0	0	9
0	0	0	0	0
0	1	0	0	9
0	0	0	0	0
1	1	1	0	0
1	1	0	0	0
1	1	0	0	0
0	0	0	0	0
0	0	0	0	0
0	0	0	0	0
0	0	0	0	0
0	0	0	0	0
0	0	0	0	0
9	0	0	0	0
9	0	0	0	0
9	0	0	0	0
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Rickets	Residual rickets	Tuberculosis	Periodontal disease	Carious lesions	Calculus
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Enamel hypoplastic defects
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