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Scurvy at the agricultural transition in the Atacama desert (ca 3600–3200 BP): nutritional stress at the maternal-foetal interface?☆

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ABSTRACT

Studies of contemporary populations have demonstrated an association between decreased dietary diversity due to resource scarcity or underutilization and an increase in diseases related to poor micronutrient intake. With a reduction of dietary diversity, it is often the women and children in a population who are the first to suffer the effects of poor micronutrient status. Scurvy, a disease of prolonged vitamin C deficiency, is a micronutrient malnutrition disorder associated with resource scarcity, low dietary diversity, and/or dependence on high carbohydrate staple-foods. The aim of this paper is to assess the potential impact of nutritional transition on the prevalence of diseases of nutritional insufficiency in an archaeological sample. Here, we report palaeopathological findings from an Early Formative Period transitional site located in coastal Northern Chile (Quiani-7). The subadult cohort from this site is composed of four perinates who exhibit a number of non-specific skeletal changes suggestive of a systemic pathological condition. One of these is associated with an adult female exhibiting diagnostic skeletal lesions of scurvy. We argue that the lesions exhibited by these perinates may represent maternal transmission of vitamin C deficiency but acknowledge that there are difficulties in applying current diagnostic criteria for scurvy to individuals this young.

1. Introduction

Studies of contemporary human groups have shown a direct relationship between the subsistence strategy employed and the general health status of individuals within that population (Johns and Eyzaguirre, 2007; Amuna and Zotor, 2008). Often statistically significant differences can be seen in age-at-death, infant and maternal mortality, and infectious disease prevalence in populations experiencing a major shift in available and/or preferred dietary staples (Popkin, 1993). Different subsistence economies (i.e. hunter-gather societies, horticulturalism, and intensive agriculture) carry their own environmental advantages and disadvantages. However, it is generally accepted among epidemiologists that the decrease in dietary variety and increased reliance on micronutrient-poor staple crops (e.g. rice, wheat, maize) that characterize transitional agricultural societies has a general negative impact on human health in many populations (Tontisirin et al., 2002; Eckhardt, 2006). Periods of resource scarcity, such as famine due to climatic events or war, can also lead to low dietary diversity and an increase in micronutrient malnutrition disorders (WHO, 1999; Webb and Thorne-Lyman, 2005).

The epidemiological relationship between agricultural intensification, dietary uniformity, and negative changes in health outcomes appears to be mirrored in ancient human groups. Prolific bioarchaeological evidence exists for an increase in skeletal indicators of physiological stress and infectious disease, as well as infant mortality in ancient populations undergoing subsistence transition (see Goodman et al., 1984; Larsen, 1995; Starling and Stock, 2007; Temple, 2010; Papathanasiou, 2011). Likewise, historic populations suffering from periods of resource scarcity have been shown to exhibit an increased prevalence in skeletal manifestations of physiological stress and disease (Geber and Murphy, 2012; Geber, 2014; Yaussy et al., 2016). However, little palaeopathological work has been published to date on the impact of resource availability, subsistence strategy, and agricultural transition on human health in the ancient Atacama Desert of Northern Chile, one of the most arid environments in the world and a unique location for assessing the impact of the adoption of agriculture (de Bryson et al.,

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2001; Alfonso et al., 2007; Rivera, 2008; Watson et al., 2010). Coastal communities were likely partially buffered from environmental resource scarcity due to the exploitation of rich marine resources, providing a more broad-spectrum diet. However, populations living through a subsistence transition would potentially still have been at risk for nutritional deficiencies as they began to rely more heavily on supplementary crops for caloric sufficiency. We hypothesise that resource vulnerability during the earliest stages of the agricultural transition would lead to an increased prevalence in skeletal lesions suggestive of disease of poor nutritional intake or dietary diversity. Here we present a case-series of individuals from the early transitional site of Quiani-7, in coastal northern Chile, who exhibit lesions of abnormal porosity and subperiosteal new bone. We present a differential diagnosis of these lesions and discuss our findings in terms of the effects of resource scarcity on maternal and foetal health and potential implications for subsistence strategy and nutritional status in the Early Formative Period Atacama. This study also provides further support for consideration of foetal and perinatal remains as sensitive indicators of population-level nutritional stress.

1.1. Dietary diversity and vitamin C deficiency

As discussed above, clinical research suggests that micronutrient malnutrition is a major factor in the prevalence of non-communicable diseases (NCDs), such as metabolic disease, in populations undergoing subsistence transition (Eckhardt, 2006). Micronutrients (vitamins, trace minerals, and electrolytes) play essential physiological roles in immunoregulation, hormone synthesis, and bone homeostasis (Kapil and Bhavna, 2002; Maggini et al., 2007; Allgrove, 2011). However, unfortified staple crops are typically micronutrient poor and heavy dependence on these foods for caloric sufficiency has been associated with an increased prevalence of micronutrient malnutrition disorders such as iron deficiency anaemia and scurvy (WHO, 1999; Zimmermann and Hurrell, 2007; , 245–246). The World Health Organization estimates that two billion individuals suffer from micronutrient malnutrition today, with populations suffering from resource scarcity and/or low dietary diversity being particularly susceptible negative health effects in the form of NCDs (Tulchinsky, 2010).

Vitamin C deficiency and sub-clinical vitamin C insufficiency are micronutrient malnutrition disorders that are strongly associated with low dietary diversity in populations experiencing famine and/or dependency upon high carbohydrate staple crops (WHO, 1999; Iannotti and Lesorogol, 2014). Vitamin C (ascorbic acid) is a six-ringed lactose synthesised in the liver of all mammals with the exception of primates, guinea pigs, and some fruit bats, which require exogenous sources (Du et al., 2012, 444). Photosynthetic organisms have the ability to synthesise ascorbate (Smirnoff and Wheeler, 2000), and the richest natural sources of vitamin C are fruits and dark green vegetables, particularly citruses, asparagus, broccoli, and kale, although potatoes, kumara (sweet potato), and some other tubers contain enough ascorbic acid to prevent manifestations of deficiency if consumed in sufficient quantities (Levine et al., 1999, 1417). The absolute minimum daily requirement of vitamin C necessary to prevent overt deficiency in adults is 10 mg, although the RDA is 60 mg with an increased need during illness, pregnancy, and lactation (Hirschmann and Raugi, 1999, 899; Levine et al., 1999; Pimentel, 2003, 331). High infectious disease burden or parasitic load in particular can lead to a greater metabolic requirement of vitamin C (Hunt et al., 1994; Isamah and Asagba, 2003).

Ascorbic acid acts as an electron donor (reducing agent) in the human body and all of its known physiological functions stem from this property (Levine and Padayatty, 2014, 400). The ascorbate radical is a powerful antioxidant, preventing tissue damage by pairing with other free radicals, and there is extensive evidence for a protective role of vitamin C against diseases that result from oxidative damage to DNA (Padayatty et al., 2003). Vitamin C also functions as a co-factor for numerous enzymes involved in a wide range of physiological processes, including neuroendocrine and peptide hormone synthesis, the production of amino acids involved in ATP generation, and the synthesis of isoenzymes required for the stabilisation of the collagen triple helix (Padayatty and Levine, 2001, 353). The unmineralised bone matrix and the basement membrane of blood vessels are largely composed of collagen (Robey and Boskey, 2008; Popovich et al., 2009, 411; Saladin, 2014, 750–752). In the absence of sufficient vitamin C for stabilisation of the collagen structure (somatic stores of < 350 mg), abnormal skeletal development and subperiosteal reaction to chronic, low-grade haemorrhage from weakened blood vessels can lead to diagnostic lesions for this disease in skeletal remains (Ortner and Ericksen, 1997; Ortner et al., 1999; Brickley and Ives, 2008; Popovich et al., 2009). Because of this, vitamin C deficiency can potentially be diagnosed in human skeletal remains, allowing bioarchaeologists to infer possible periods of resource scarcity or low dietary diversity in the past (Buckley et al., 2014; Halcrow et al., 2014).

1.2. The early formative period in the atacama desert

The Atacama Desert is located on the Western coast of South America between 18°N and 27°S in what is now Northern Chile and Southern Peru (Arriaza et al., 2008; Brown and Ortner, 2011). The Atacama is one of the world's most arid environments due to the moisture blocking effects of the Andes to the east and the drying action of anticyclonic air masses from the Pacific High Pressure belt (Núñez et al., 2002). Average rainfall here is < 2 mm per year, although there is some regional and seasonal variability in both precipitation and evaporation rates (Houston, 2006a, 2006b). River valleys fed by the seasonal melting of Andean snows create oases along much of the western coast which allow for patches of higher than normal biodiversity and provide the natural resources necessary for human occupation in an otherwise barren environment (Cereceda et al., 2008a, 2008b).

The Formative Period in Andean archaeology is loosely defined as an overarching chronological phase of pre-polity cultural traditions characterised by the production of cultivars, animal domesticates, and early ceramics (Muñoz 2004, 2011; Núñez and Santoro, 2011; Muñoz and Fernández, 2014; Andrade et al., 2015). Within the Northern Atacama the Formative Period is often divided chronologically into early (ca. 3500–2500 BP) and late (2500–1500 BP) phases (Rivera 1991, 21; Rivera, 2008). The Early Formative Period is characterised by a dramatic shift in subsistence practices and cultural identity (de Bryson et al., 2001). The pre-Formative Archaic Period was dominated for over 5000 years by the Chinchorro Cultural Complex (ca 9000–3500 BP), a sedentary hunter-gatherer society comprised of familial groups occupying isolated coastal estuaries (Arriaza, 1995). They employed advanced maritime technology to exploit the intertidal and pelagic zones of the highly biodiverse Pacific coast. Faunal and palaeobotanical remains from Chinchorro middens and habitation sites indicate a high protein diet rich in marine foods such as bony fishes, molluscs, marine mammals, and kelp (, 250–251; , 40–41; ; Castro et al., 2016). Dietary $\delta^{15}N$ isotopic evidence from human remains at Chinchorro cemetery sites is also suggestive of heavy consumption of marine resources (Aufderheide et al., 1993; Andrade et al., 2015; King et al., in review); exploited terrestrial resources likely included sea grass, various reeds, and fruit bearing shrubs (Arriaza et al., 2001; Muñoz, 2004, 2011; Reinhard et al., 2011; , 43).

Marine resources continued to be heavily used during the Early Formative Period, particularly on the coast (Andrade et al., 2015; , 14; Bonilla et al., 2016). However this period also saw the introduction of supplemental cultivars such as squash, beans, and maize, possibly via movement of highland populations to the coast, bringing an influx of Andean domesticates (, 21; , 5; , 548; Muñoz, 2011; Núñez and Santoro, 2011). Because of this, analysis of malnutrition-related NCDs, such as scurvy, in human remains from these sites has the potential to provide important information about the impact of subsistence transitions and

Fig. 1. Map of the Northern Atacama showing the location of Quiani-7 (after King et al., in review).

the movement towards intensive agriculture on ancient human health in the Atacama.

2. Materials and methods

2.1. The site: quiani-7

Quiani-7 is a coastal site located eight kilometres south of the modern port city of Arica (Fig. 1). The cemetery itself is one of a cluster of mortuary sites positioned on a rocky terrace ~20 m above sea level on the south side of a massive coastal bluff. It was first excavated in 1974 after the burials were disturbed by road construction (Dauelsberg, 1974), and subsequently by Standen and Focacci in 1984 (Standen pers. com.). Radiocarbon dating of desiccated human muscle places the use of the cemetery in the Early Formative Period (3600–3200 cal BP; Dauelsberg, 1974; , 97). This site is thought to provide the earliest evidence in the region of a clear shift in subsistence economy and cultural practices from the maritime-based hunter-gatherer economy and elaborate mortuary practices of the Archaic Chinchorro Cultural Complex (Dauelsberg, 1974). There is archaeological evidence of continued reliance upon marine resources in the form of harpoons, cactus fishhooks, mollusc shells, and sea lion and porpoise bones (Bird, 1943; Dauelsberg, 1974). However, palaeobotanical evidence suggests that maritime subsistence strategies were supplemented here by domesticates such as squash (*Cucurbita* spp.) (Dauelsberg, 1974). In contrast to Chinchorro cemetery sites in the Arica region, there is no evidence of artificial mummification at Quiani-7, although the hyperarid environment has led to the natural mummification of several sets of human remains. All individuals from this period, including infants, were interred in a lateral, flexed position and wrapped in textile or animal skin mats (Dauelsberg, 1974). Associated grave goods included lapis lazuli and animal bone beads, woollen and plant fibre textiles, baskets, and occupational paraphernalia such as fishhooks and harpoons (Dauelsberg, 1974, Standen pers. com.).

All individuals analysed for this study were recovered during the 1984 excavation. Twelve graves in total containing twelve individuals were recovered. Although this sample is small, most individuals are at least 75% complete and all are exquisitely preserved (Table 1), allowing for more detailed lesion observation than is usual for many archaeological collections.

2.2. Age and sex estimation

A demographic breakdown of the Quiani-7 sample is provided in Table 1. Adult age was estimated using standard methods outlined in Buikstra and Ubelaker (1994) with the most widely accepted methods (e.g. Brooks and Suchey, 1990; Meindl and Lovejoy, 1989) employed where possible according to skeletal completeness. Because age estimation methods from the adult skeleton are accompanied by relatively large margins of error, individuals were then placed into broad age-at-death categories of young (20–34), middle (35–49), and old adults (50+). Sex was estimated where possible using pelvic and cranial morphology (Table 1; Phenice, 1969; Acsádi and Nemeskéri, 1970). Subadult age was estimated by diaphyseal length (Fazekas and Kósa, 1978; Maresh, 1970), as dentition was absent or unobservable for most individuals. These diaphyseal length standards were developed from contemporary Hungarian and American samples and may be less accurate in estimating the biological age of ancient South American

Table 1
Demography and completeness of the Quiani-7 sample with age and sex estimation methods. NA = not applicable; pnw = prenatal weeks; m = postnatal months.

Individual	Completeness	Estimated Age	Method(s)	Estimated Sex	Method(s)
Quiani-7 TA	> 75%	20–34	Meindl and Lovejoy, 1989; Brooks and Suchey, 1990	Male	Phenice, 1969; Acsádi and Nemeskéri, 1970
Quiani-7 T9	50%	adult	NA: no skull, pelvis, or sternal ribs; all epiphyses fused	Unknown	NA
Quiani-7 T13	75%	20–34	Iscan et al., 1984, 1985	Unknown	NA
Quiani-7 T15	75%	32–36 pnw	Fazekas and Kósa, 1978	Unknown	NA
Quiani-7 T16	> 75%	50+	Meindl and Lovejoy, 1989; Brooks and Suchey, 1990	Female	Phenice, 1969
Quiani-7 T16a	50%	35–49	Meindl and Lovejoy, 1989	Female	Phenice, 1969
Quiani-7 T17	> 75%	20–34	Meindl and Lovejoy, 1989; Brooks and Suchey, 1990	Female	Phenice, 1969; Acsádi and Nemeskéri, 1970
Quiani-7 T17a	> 75%	40 pnw	Fazekas and Kósa, 1978	Unknown	NA
Quiani-7 T18	75%	38–40 pnw	Fazekas and Kósa, 1978	Unknown	NA
Quiani-7 T19	75%	1.5 m	Maresh 1970	Unknown	NA
Quiani-7 T21	50%	20–34	Meindl and Lovejoy, 1989; Brooks and Suchey, 1990	Male	Phenice, 1969
Quiani-7 T22	75%	35–49	Meindl and Lovejoy, 1985	Male	Phenice, 1969; Acsádi and Nemeskéri, 1970

individuals since growth is influenced by biological affinity (Lewis, 2007). The clinical definition of the perinatal period is 28 prenatal weeks to 28 postnatal days (Mosby, 2001). However, to mitigate the error that accompanies skeletal perinatal age estimation standards we define perinates as pre-term, full term, and post-full term individuals less than 50 developmental weeks (Halcrow et al., 2009, 397).

2.3. Lesion recording

All surfaces of all skeletal elements present were examined macroscopically and the type and distribution of any abnormal bony changes (e.g. abnormal proliferation or destruction, abnormalities of density, size, and shape) were recorded. Subsequent to macroscopic examination, analogue radiographs were taken in the antero-posterior view of all skeletal elements of any individuals exhibiting abnormal macroscopic pathology. A modified version of Brickley and Ives (2008) scoring system for differential diagnosis of metabolic bone disease was employed, with lesions classified as strongly diagnostic (S), diagnostic (D), or generally suggestive (G), with more than one 'D' feature being required for a diagnosis of scurvy. We have expanded this classification system to include additional features such as vascular impressions and new bone on the endocranial surface of the vault (Ingalls, 1936; Brown and Ortner, 2011; Table 2).

3. Results

Of all individuals analysed 41.6% (5/12) exhibited lesions suggestive of a systemic pathological process. One adult female (associated with perinate T17a) and all four perinates had abnormal skeletal changes in the form of apposition of active (woven) subperiosteal new bone and abnormal, fine porosity (< 1 mm across), at cranial vascular and entheseal sites (Figs. 2 a and b, 4 c). All perinates exhibit diffuse apposition of active subperiosteal new bone on the shafts of appendicular elements (Fig. 3b), thick plaques of active subperiosteal new bone on the postero-lateral shafts of the ribs (Fig. 4b), and low mineral density and cortical thinning in the post-cranial skeleton. Additionally, two perinates exhibited new bone on the endocranial surface of the vault (T17a, T18; Fig. 4a), two exhibited extensive porosity in regions of endochondral growth (T17a, T19; Fig. 3a), and two exhibited abnormal thinning and cortical porosity of the calvaria (T15, T19). Lesion distribution and prevalence for the adult and perinatal cohorts are outlined in Figs. 5a and b and Table 3. Detailed individual descriptions of lesion type and distribution are provided in the supplementary data.

4. Discussion

4.1. Differential diagnosis

Proposed underlying processes behind the skeletal changes we have observed are summarised as follows:

- Widespread, diffuse apposition of subperiosteal new bone throughout the postcranial skeleton: suggestive of a systemic inflammatory process OR within the range of normal appositional growth.
- Extensive porosity in regions of endochondral growth: suggestive of non-existent bone matrix formation and/or mineralisation OR within the range of normal endochrondral growth.
- Abnormally thin, porous calvaria (craniotabes): suggestive of poor bone matrix mineralisation.
- Layers of irregular, spiculated new bone on the ectocranial surface: suggestive of renewed mineralisation of osteoid following reintroduction of vitamin D, calcium, or phosphorous OR within the range of normal intramembranous growth.
- Discrete islands of subperiosteal new bone around cranial vascular foramina and entheseal sites: indicative of a localised inflammatory response, possibly to low-grade haemorrhage.
- Discrete islands of subperiosteal new bone on the endocranial surface: indicative of localised inflammatory response in the subperiosteal space of the dura mater.
- Discrete regions of fine (< 1 mm) abnormal porosity at cranial vascular and entheseal sites: indicative of capillary proliferation, possibly a hypervascular response to low-grade haemorrhage.
- Vascular impressions in regions of subperiosteal new bone: indicative of capillary proliferation, possibly a hypervascular response to low-grade haemorrhage or infection.

A number of different pathological and non-pathological conditions can cause these skeletal changes. We discuss possible alternative aetiologies below.

4.1.1. Growth: appositional, endochondral, and intramembranous ossification

Several of the features we have observed in our perinatal sample are not necessarily abnormal in foetal and infant life and caution is therefore needed in interpretation. Subperiosteal new bone in skeletal remains is often attributed to pathological inflammation of the overlying periosteum (periostitis) (Weston, 2012). However, the deposition of subperiosteal new bone is also part of the process of appositional bone growth (Scheuer and Black, 2000; Weston, 2012). Likewise, while endochrondral porosity can be indicative of poor bone matrix formation or mineralisation (Brickley and Ives, 2008), normal metaphyseal pitting occurs due to osteoclastic activity in the zone of ossification of the growth plate (, 26; Ortner et al., 2001). Finally, formation of the calvaria occurs via gradual ossification of a membranous sac resulting in layered, spiculated new bone on the ectocranial surface of the foetal and perinatal cranial vault (Scheuer and Black, 2000; , 134).

Others have discussed the challenges of differentiating normal vs. pathological changes in the perinatal and infant skeleton, particularly in regards to subperiosteal new bone (, 341; , 134–137; , 498). There appears to be an element of subjectivity in lesion interpretation during

Table 2
Lesions observed at Quiani-7 with possible aetiologies and diagnostic strength for scurvy. Features in bold are not included in Brickley and Ives (2008) diagnostic system. ♦Feature may also be expressed during normal foetal/perinatal growth. NA = not applicable.

Features Observed at Quiani-7	Diagnostic Strength for Scurvy	Possible Aetiologies	Comparative Source
Abnormal porosity in cortex of the calvaria – distinguishable from hyperplastic trabeculae	D	scurvy, rickets, trauma, infection	Ortner and Ericksen, 1998; Ortner et al., 1999; Ortner, 2003; Brickley and Ives, 2008
Hyperplastic trabeculae in the calvaria	NA	iron deficiency, megaloblastic, or hemolytic anaemia	; Walker et al., 2009; Oxenham and Cavill, 2010
Subperiosteal new bone in orbits	D	scurvy, trauma	Ortner and Ericksen, 1998; Brickley and Ives, 2006; Brickley and Ives 2008; Brickley and Ives, 2008; Usha et al., 2011
Subperiosteal new bone on endocranial surface of the vault♦	**G**	scurvy, trauma, infection	Ingalls, 1936; Lewis, 2004
Subperiosteal new bone on the lateral shafts of the ribs	NA	trauma, infection	Guttentag and Salwen, 1999; Lewis, 2007
Spiculated, irregular new bone on the ectocranial surface of the vault♦	D	scuvy, healing rickets	Ortner and Mays, 1998; Brickley et al., 2006; Brickley and Ives, 2008
Subperiosteal new bone and/or abnormal porosity on the maxillae: hard palate, infraorbital foramina, anterior surface, and/or posterior surface	D	scurvy, trauma, infection	Ortner and Ericksen, 1998; Ortner, 2003; Brickley and Ives 2006; Brickley and Ives, 2008
Subperiosteal new bone and/or abnormal porosity on interior mandible	D	scurvy, trauma, infection	Ortner et al., 1999; Brickley and Ives, 2008
Subperiosteal new bone and/or abnormal porosity on the greater wings of the sphenoid	D	scurvy	Ortner and Ericksen, 1998; Brickley and Ives, 2008; Geber and Murphy, 2012
Subperiosteal new bone on the shafts of long bones♦	D	scurvy, rickets, trauma, infection, neoplastic disease, autoimmune disease	Joffe 1961; Jaffe, 1972; Ortner 2003; Brickley and Ives, 2008; van der Merwe et al., 2010
Subperiosteal new bone and/or abnormal porosity in the supraspinous and/or infraspinous fossa	D	scurvy, trauma	Ortner et al., 1997; Ortner et al., 2001; Ortner, 2003; Brickley and Ives, 2006 Brickley and Ives, 2008
Vascular impressions on the endocranial surface of the vault	**G**	scurvy, trauma, infection	Lewis, 2004; Brown and Ortner, 2011
Vascular impressions on the ectocranial surface of the vault	**G**	scurvy, trauma, infection	Brown and Ortner, 2011
Abnormal porosity extending > 10 from the distal metaphyseal plate of long bones (juveniles)	**D**	scurvy, rickets	Ortner et al., 2001
Generalized osteopenia (radiographic)	G	scurvy, rickets	Joffe, 1961; Young et al., 1979; Brickley and Ives, 2008
Thin, irregular cortices (juveniles – radiographic)	D	scurvy, healing rickets	Young et al., 1979; Brickley and Ives, 2008

these developmental stages since “abnormality” can be a matter of degree rather than presence. Additional difficulties accompany this study due to the small size of our perinatal cohort, which makes population standards of normality in terms of perinatal subperiosteal bone apposition difficult to define. Comparison of the Quiani-7 cohort to an anatomical collection of perinatal individuals (Fig. 4b) highlights the fact that the subperiosteal new bone we have observed, although subjectively extensive, may be within the range of normal variation for this age group. However, discrete islands of subperiosteal new bone and fine, clustered porosity at cranial vascular and entheseal sites (e.g. Fig. 4c) are not consistent with normal development and the endochrondral porosity we have observed appears to be abnormal according to Ortner and colleagues’ definition (i.e. > 10 mm from the distal metaphyseal plate; 2001: 348). Likewise, endocranial new bone associated with normal growth is typically far milder in expression than that exhibited by T17a and T18, taking the form of thin layers of spiculated new bone radiating out from the cranial bosses rather than the thick, disorganized plaques with large vascular impressions we observed in these individuals. As such, we argue that while caution is necessary, there is room to allow the possibility of a pathological origin to some of the skeletal changes we have observed in the perinatal cohort.

4.1.2. Systemic infectious process

Congenital transmission of treponematosis and some viral infections have been known to cause diffuse apposition of subperiosteal new bone in the perinatal and infant skeleton (Jaffe, 1972; Alessandri et al., 1995; Ortner, 2003; Satti et al., 2010; Cantey et al., 2013). However, these viral diseases do not leave pathognomonic skeletal lesions and no specific indicators of treponemal disease, such as gummatous periostitis (in older subadults or adults), dental, or skeletal indications of congenital involvement are present in any of the individuals from this site. As discussed in section 4.1.1, there are additional limitations to attributing the subperiosteal new bone in our perinatal cohort to a pathological process given the age of these individuals.

4.1.3. Trauma

Subperiosteal haemorrhage from traumatic events, particularly child abuse, can result in an inflammatory and hypervascular response leading to regions of porous, subperiosteal new bone formation (Lewis, 2007, 178). We argue that trauma is an unlikely cause for the lesions we have observed given their consistent distribution between individuals at cranial entheseal sites, such as the interior mandible, which are unlikely to be the locations of external force. Trauma due to child abuse is also frequently accompanied by cranial and long bone fractures (Kemp et al., 2008; Offiah et al., 2009), which were absent in our perinatal cohort.

4.1.4. Haematopoietic neoplasm

Haematopoietic type neoplasms include cancers of blood (leukaemia) and plasma cells (myeloma) (Harris et al., 1999). Proliferative and destructive skeletal changes are common in both leukaemia and myeloma; however, myeloma is rare before the age of 40 with maximum frequency occurring between 50 and 70 years (Ortner, 2003, 377) and can be reasonably excluded from our diagnosis. Leukaemia is a relatively common paediatric cancer and may result in diffuse apposition of subperiosteal new bone in the appendicular skeleton, as well as lytic activity around vascular foramina and long bone metaphyses (Ortner, 2003, 376). However, lytic lesions were absent in all the Quiani-7 individuals, and the presence of diffuse subperiosteal new

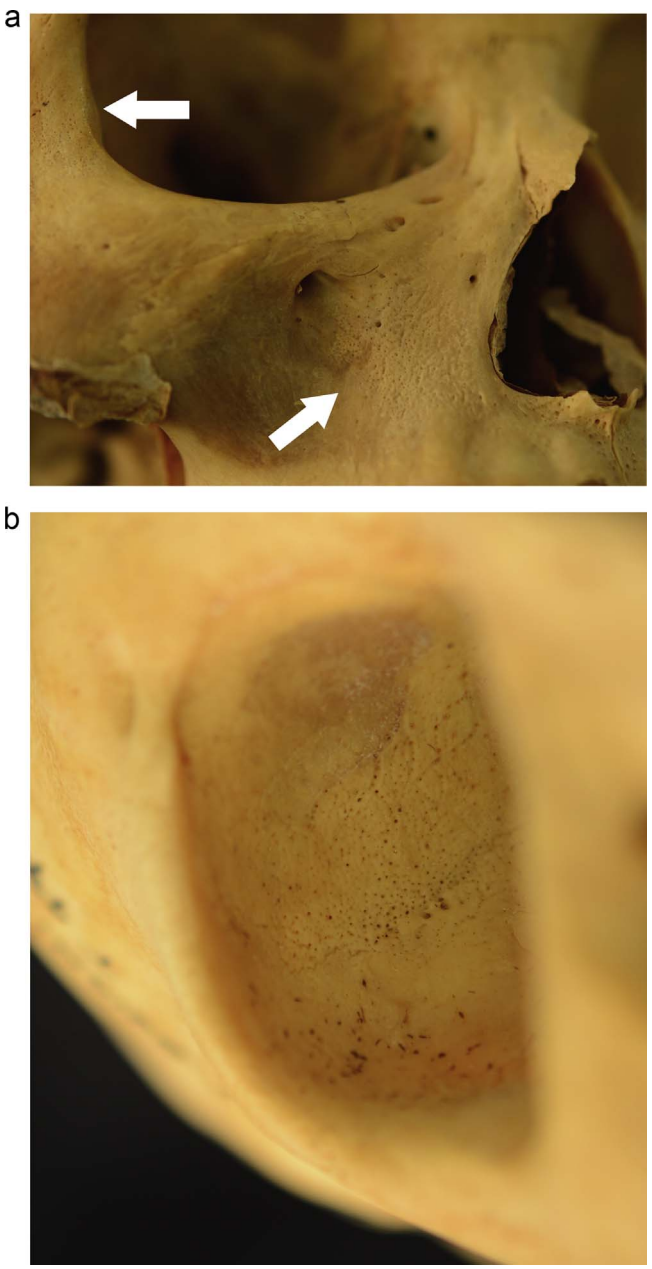

Fig. 2. a) Maxilla and zygomatic bone (T17) exhibiting abnormal islands of subperiosteal new bone around the infraorbital foramen and on the frontal process of the zygomatic b) Right orbital roof (T17) exhibiting porous subperiosteal new bone.

bone is not a strong enough feature to justify diagnosis. Furthermore, the presence of paediatric leukaemia in our perinatal cohort is clinically unlikely.

4.1.5. Caffey's disease (Infantile cortical hyperostosis)

Infantile cortical hyperostosis (ICH) is a condition of early postnatal life characterised by unevenly distributed soft tissue swelling, heavy apposition of subperiosteal new bone and cortical thickening of the mandible and long bones (Resnick and Niwayama, 1995; Kamoun-Goldrat and le Merrer, 2008). Post-cranial lesions are typically asymmetric (, 144–145). Although familial forms exist, the majority of cases are idiopathic and do not show a predilection for a particular ethnic group or geographic region (Glorieux, 2005; , 143–144). ICH typically begins to manifest around nine postnatal weeks and most cases will resolve spontaneously by five months (, 4436; , 144). This condition is clinically uncommon, occurring in 3:1000 US infants, and while foetal

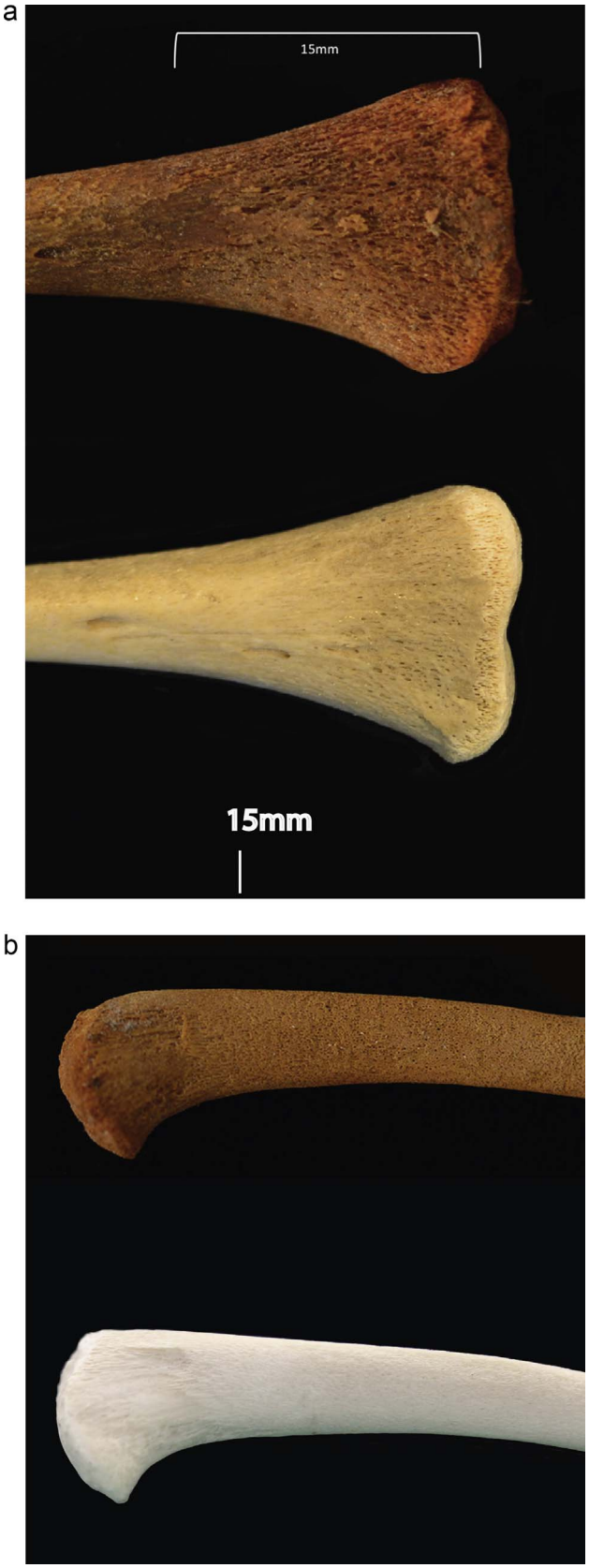

Fig. 3. a) (top) Distal femur (T18) exhibiting heavy porosity extending ~15 mm up the diaphysis from the distal metaphyseal plate (bottom) comparative image from a 38–40 week foetus (University of Otago, Department of Anatomy) b) (top) Medial tibia (T17a) exhibiting diffuse apposition of active, porous subperiosteal new bone (bottom) comparative image from a 40 week foetus (University of Otago, Department of Anatomy).

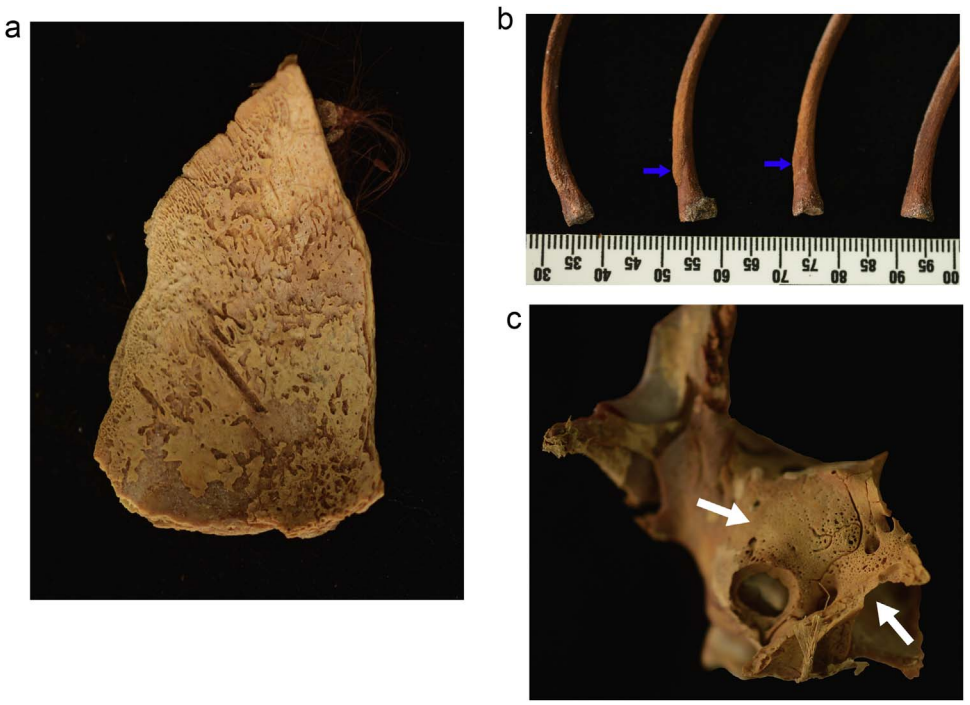

Fig. 4. a) Parietal fragment (T18) exhibiting diffuse apposition of subperiosteal new bone with vascular impressions on the endocranial surface b) Right ribs (T17a) exhibiting heavy plaques of subperiosteal new bone on the postero-lateral surfaces of the shafts (blue arrows) c) posterior aspect of left maxillae and sphenoid (T16) exhibiting porous subperiosteal new bone with vascular impressions (arrows). (For interpretation of the references to colour in this figure legend, the reader is referred to the web version of this article).

and perinatal cases have been reported, they are extremely rare (Kamoun-Goldrat and le Merrer, 2008). Furthermore, the subperiosteal new bone formation associated with ICH is generally far heavier than what we have observed at Quiani-7, resulting in gross skeletal deformity (Resnick and Niwayama, 1995). It is therefore unlikely that ICH is responsible for the diffuse apposition of subperiosteal new bone in these individuals.

4.1.6. *Anaemia*

The term anaemia covers a variety of disorders of red blood cell

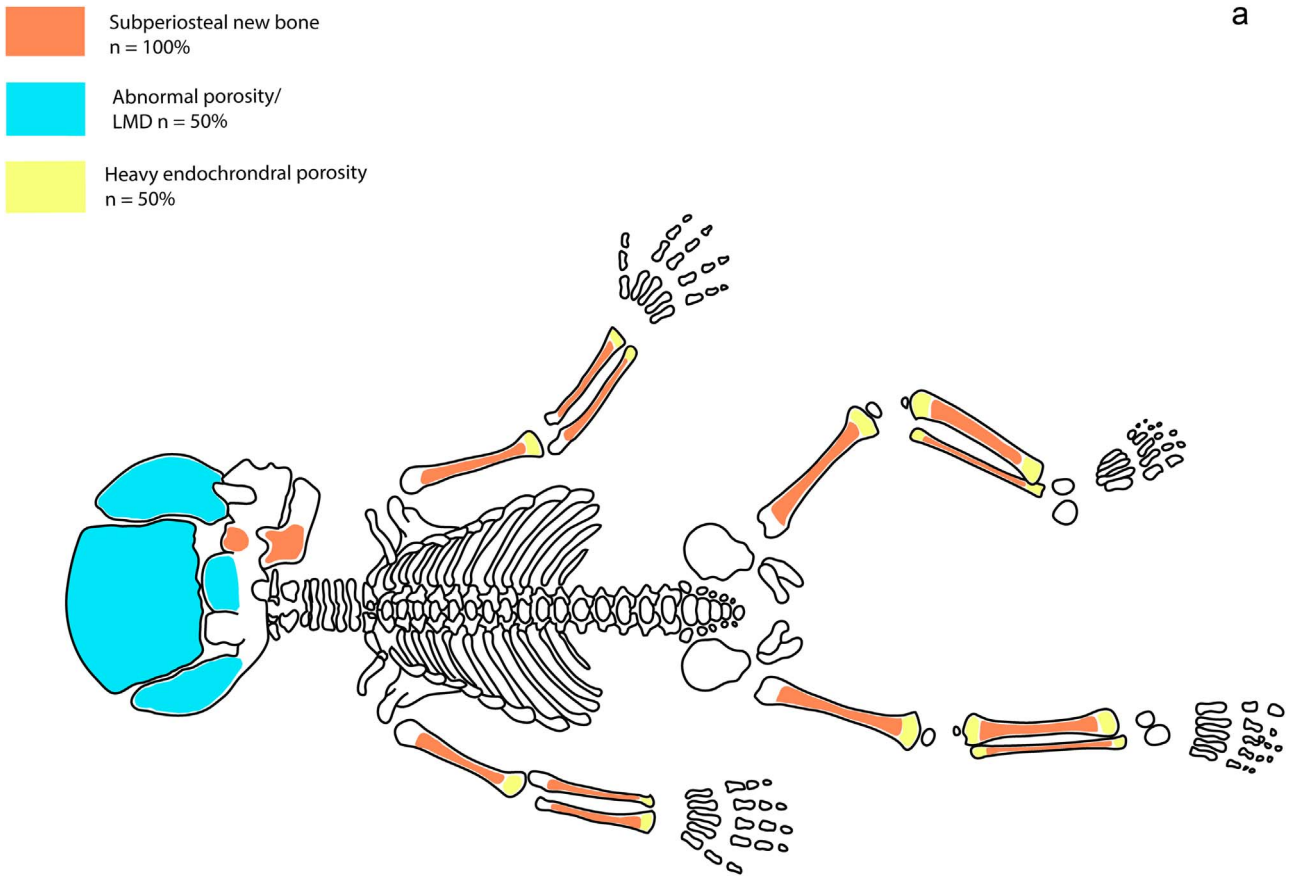

Fig. 5. a) Lesion distribution and prevalence for the subadult cohort. LMD = low mineral density b) Lesion distribution and prevalence for the adult cohort.

underproduction or dysfunction that negatively affect the ability of the vascular system to transport oxygen (Resnick, 1995, 2107). The skeletal changes of anaemias are related to the need for the body to expand haematopoietic centres, which leads to hyperplastic lesions of trabecular bone, such as porotic hyperostosis or cribra orbitalia (, 2108; , 364; Oxenham and Cavill, 2010). While these processes do result in regions of abnormally severe porosity, the lesions are characterised by a clear expansion of the trabeculae through the cortex, rather than cortical porosity alone (Ortner, 2003, 375). One of our individuals, T19, did exhibit a region of hyperplastic trabecular bone in a fragment of occipital. However, the majority of individuals did not exhibit such lesions, and regions of abnormal porosity that penetrated the cortex (as opposed to porous subperiosteal new bone) were typically found in elements with low trabecular content, such as the greater wings or pterygoid plates of the sphenoid. Because of this, we exclude anaemia from our diagnosis of all individuals other than T19 and argue that if this individual did suffer from anaemia, it was likely comorbid with another pathological condition.

4.1.7. Rickets

Rickets is a collective term that describes the juvenile manifestations of severe vitamin D, calcium, or phosphorous deficiency, although it can also be caused by genetic abnormalities, renal disease, or gastrointestinal malabsorption disorders (Carpenter, 2008; Lips et al., 2013). The histological characteristics of this disease are poor or non-existent mineralisation of osteoid and disorganisation of the columnar cartilage of the growth plate (, 331–332; Özkan, 2010, 138). Rickets can occur in perinates and young infants if the mother experienced a severe deficiency of calcium or vitamin D during pregnancy or lactation, and these cases are characterised by widespread osteopenia, craniotabes, and metaphyseal flaring and cupping (Oppenheimer and Snodgrass, 1980; Park et al., 1987). These lesions are consistent with some of those observed in our perinatal cohort. Furthermore, healing rickets can result in layers of spiculated, irregular new bone, which forms when osteoid resumes mineralisation (Brickley and Ives, 2008, 103), and this was observed in the calvaria of T17a, T18, and T19. However, there is considerable overlap between the manifestations of rickets and infantile scurvy (Brickley and Ives, 2008) and as previously mentioned, ectocranial new bone is also associated with ossification of the foetal skull. Additionally, comorbid cases of scurvy and rickets can be challenging to assess in skeletal remains as vitamin C deficiency inhibits osteoid formation while rickets inhibits osteoid mineralisation, impeding some of the classic manifestations of each of these conditions (e.g. accumulation of unmineralised osteoid and ossified haematomas) (Schattmann et al., 2016). However, a diagnosis of rickets is not consistent with the discrete islands of porosity and subperiosteal new bone found at cranial vascular and entheseal sites in the subadult cohort and the adult female T17. Therefore, while we cannot exclude the possibility of rickets in the perinates at this site (see section 4.2), it is unlikely to be the cause of the majority of lesions we have observed here.

4.1.8. Scurvy

As discussed above, scurvy is caused by severe, prolonged vitamin C deficiency. In adults physical effects typically begin to manifest after two to three months of an intake of less than 10 mg per day (WHO, 1999), although critically ill individuals and pregnant and lactating

Table 3
Individuals in the Quiani-7 sample with associated lesions and their collective diagnostic strength for scurvy. Bold text indicates a diagnostic or suggestive feature. *Indicates diagnosis should be considered with caution due to age of affected individual.

Individual	Lesions exhibited	Collective diagnostic strength for scurvy
Quiani-7 TA	Well-remodelled oblique fracture in the distal 1/4th of the right humerus, vertebral osteophytosis throughout spine, evidence of bilateral osteoarthritis in temporomandibular and acromioclavicular joints.	No diagnostic or suggestive features
Quiani-7 T9	No macroscopic pathology	No diagnostic or suggestive features
Quiani-7 T13	Evidence of osteoarthritis in zygapophyseal joints of the thoracic spine	No diagnostic or suggestive features
Quiani-7 T15	**Discrete islands of porous subperiosteal new bone on the greater wing on the sphenoid and lateral mandible (body and rami)**; thick plaques of subperiosteal new bone on the lateral shafts of all ribs; **diffuse appostion of porous subperiosteal new bone on the all surfaces of the shafts of all long bones** and the ilia; craniotabes**; possible osteopenia**.	Diagnostic*
Quiani-7 T16	Evidence of osteoarthritis in the right hip and right acromioclavicular joint	No diagnostic or suggestive features
Quiani-7 T16a	**Osteopenia**; evidence of osteoarthritis in left knee	Single suggestive feature; not diagnostic
Quiani-7 T17	**Discrete islands of fine, clustered porosity and porous subperiosteal new bone bilaterally on the anterior and posterior surfaces of the maxillae, on the posterior sphenoid, the orbital roofs, and interior mandible (rami).**	Diagnostic
Quiani-7 T17a	**Discrete islands of porous, subperiosteal new bone bilaterally in the orbital roofs, lateral mandible (rami and body), right greater wing of the sphenoid (left missing), supraspinous fossae of the scapulae, and on the endocranial squamous temporal, squamous occipital, and frontal**; thick plaques of subperiosteal new bone on the lateral shafts of all ribs; **diffuse appostion of porous subperiosteal new bone on the all surfaces of the shafts of all long bones; heavy endochrondral porosity**; **possible osteopenia.**	Diagnostic*
Quiani-7 T18	**Discrete islands of porous, subperiosteal new bone on the endocranial parietal and bilaterally on the lateral and interior mandible (rami), subscapular, infraspinous, and supraspinous fossae of the scapulae;** thick plaques of subperiosteal new bone on the lateral shafts of all ribs; **diffuse appostion of porous subperiosteal new bone on the all surfaces of the shafts of all long bones** and the ilia; **possible osteopenia.**	Diagnostic*
Quiani-7 T19	**Discrete islands of porous subperiosteal new bone bilaterally on the interior surfaces of greater wings of the sphenoid and the right orbial roof (left very incomplete)**; **heavy microporosity covering orbital surfaces of the maxillae**; thick plaques of subperiosteal new bone on the lateral shafts of all ribs; diffuse appostion of porous subperiosteal new bone on the all surfaces of the shafts of all long bones**; heavy endochrondral porosity**; craniotabes; porotic hyperostosis; **possible osteopenia**	Diagnostic*
Quiani-7 T21	Vertebral osteophytosis in the lumbar spine; bilateral remodelling periostistis on antero-medial shafts of the tibiae	No diagnostic or suggestive features
Quiani-7 T22	Mild vertebral osteophytosis in cervical spine (rest of column missing)	No diagnostic or suggestive features

women have a greater metabolic requirement (Hirschmann and Raugi, 1999; Long et al., 2003). Scurvy is clinically associated with a number of non-specific symptoms, such as fatigue and joint pain (Reuler et al., 1985; Pimentel, 2003); however, physical signs are directly related to defective collagen synthesis. In scorbutic individuals, compromised vascular integrity causes widespread, chronic, low-grade haemorrhage, which often results in petechia (pin-point cutaneous bruising), gingival bleeding, perivascular edema, and subperiosteal haematomas (see Hirschmann and Raugi, 1999; Olmedo et al., 2006; Popovich et al., 2009). The secondary effects of this chronic bleeding, particularly in the subperiosteal space of long bones, the cranial vault, orbits, small facial bones and the alveolar bone of the mandible and maxilla are assumed to lead to many of the skeletal lesions commonly employed by bioarchaeologists in the differential diagnosis of scurvy from ancient human remains (Table 2). Biomechanical stress of normal muscular actions, such as mastication or facial expression, on compromised blood vessels is thought to be the cause of these islands of subperiosteal inflammation (Ortner et al., 1997). Once sufficient levels of vitamin C for osteoid formation are restored, porous subperiosteal new bone forms at vascular and entheseal sites where low-grade haemorrhage from compromised blood vessels had occurred (Brickley and Ives, 2008, 56). Fine, abnormal porosity (< 1 mm across) within cortical bone may also form at these sites as capillaries proliferate as part of the inflammatory response to the presence of extravascular blood (Ortner et al., 2001; Klaus, 2014a).

Qualitative changes to bone can also occur as a direct effect of the inability of the body to form osteoid in a vitamin C deficient state. Both adults and juveniles often exhibit generalised osteopenia and an abnormally thin, irregular cortex when chronically deficient (Joffe, 1961; Young et al., 1979). In juveniles endochondral growth is also negatively affected by weakened bone matrix formation, leading to abnormally heavy porosity at metaphyseal sites (Ortner et al., 2001). Radiographically, alternating opaque ('white line of Frankel') and lucent ('scurvy lines') may also be visible in regions of endochondral growth, although these tend to resolve rapidly and are only visible in active cases of severe deficiency (Brickley and Ives, 2008, 62).

Scurvy appears to be the only common potential origin of the lesions of abnormal porosity and islands of subperiosteal new bone at entheseal and vascular sites exhibited by the adult female T17 (Table 3; Fig. 2a,b). Similar islands of subperiosteal new bone and porosity are also present in all four Quiani-7 perinates (Table 3; Fig. 4c). However, as discussed above, there are significant difficulties in differentiating the skeletal changes associated with normal growth from pathological lesions in individuals this young. For example, the extensive endochondral porosity and flaring that we observed in the perinatal cohort may be due to the absence of bone matrix formation, but these individuals may also represent the extreme end of a range of normal variation in skeletal changes associated with endochrondral growth. Likewise, the heavy apposition of diffuse subperiosteal new bone we have observed could be due to either pathological periostitis or appositional growth. All four perinates also exhibit what we believe may be generalised osteopenia, a non-specific feature of scurvy; however, female infants generally have lower bone mineral density than males and there are difficulties in comparing individuals of unknown sex (Rupich et al., 1996). If our lesion scoring system for scurvy is followed (e.g. Table 2), all of the Quiani-7 perinates technically exhibit multiple diagnostic features. However, given the overlap between some of these features and normal foetal growth processes, they do not carry the same diagnostic strength as they would in an older child or adult. Therefore, while scurvy is a potential cause of the skeletal changes observed in the Quiani-7 perinatal cohort, supported by the diagnostic features exhibited by the adult female T17, we stress that caution is needed in applying diagnostic standards developed on older individuals to foetal and infant remains.

Because many of the lesions we have observed involve the formation of subperiosteal new bone, which cannot occur until overt vitamin C deficiency has been at least partially overcome (e.g. , 341), we argue that T17 was in the healing stages at the time of her death. If the associated perinate T17a is the child of this individual and this nutritional deficiency was maternally transferred, the absence of a scurvy line/white line of Frankel is unremarkable, as lesion resolution is particularly rapid during foetal and infant life (Longaker et al., 1992; Broker and Reiter, 1994; , 39). Assuming the adult female T17 had overcome a period of extreme deficiency, the associated foetus T17a may well have already remodelled these indicators of active scurvy. We discuss the biosocial implications of these findings below.

4.2. Perinatal scurvy: metabolic bone disease of infancy?

Perinatal scurvy is rarely reported in the clinical literature (see Hirsch et al., 1976), and the strongest evidence for transmission of scurvy via the maternal-foetal interface comes from work with porcine and cavid (guinea pig) experimental models (Rivers et al., 1970; Wegger and Palludan, 1994). These studies found that the foetuses of vitamin C deficient mothers exhibit the same or similar lesion type and patterning as their adult counterparts (i.e. subperiosteal haemorrhage and defective bone matrix formation). An increased incidence of spontaneous abortion has also been observed in scorbutic animal models, suggesting that human maternal scurvy may carry an associated elevated risk of perinatal death (Wells, 1931; Wegger and Palludan, 1994). Intriguingly, Rivers and colleagues found haemorrhages in the foetuses of biochemically vitamin C deficient mothers who did not themselves exhibit signs of scurvy suggesting that these individuals may exhibit macroscopic lesions of scurvy before their mothers (Rivers et al., 1970, 219). All of the Quiani-7 subadults were perinatal and the sole source of vitamin C for these individuals would be maternal, either via the placenta or breast milk (, 190; , 203–204). Additionally, an increased maternal requirement for vitamin C during pregnancy and lactation means that mothers are a high-risk group for scurvy in populations suffering from vitamin C insufficiency (Hirschmann and Raugi, 1999, 899). The presence of multiple diagnostic lesions of scurvy in the adult female T17 provides additional supporting evidence for a maternal route of transmission of vitamin C deficiency in prenatal and early postnatal life at this site, and suggests that pregnant and lactating women and their offspring were more vulnerable to the effects of population-wide resource scarcity.

Cases of perinatal scurvy from Roman England, South Asia and the Pacific have been reported in the bioarchaeological literature (Lewis, 2010; Buckley et al., 2014; Robbins Schug and Blevins, 2016). This discrepancy between clinical and palaeopathological reporting may be because many of the diagnostic lesions used by bioarchaeologists, such as new bone formation and porosity at cranial vascular and entheseal sites, are subtle and likely to be missed entirely by radiographic assessment of live individuals. Other radiographically visible characteristics (e.g. osteopenia, craniotabes, and diffuse periostitis) are common to a number of metabolic bone diseases. As such, clinical cases of perinatal scurvy may be classified as general 'metabolic bone disease of infancy', particularly when they co-occur with known vitamin D or calcium deficiency (e.g., low serum vitamin D concentrations).

Micronutrient deficiencies rarely occur in isolation, and the possibility of multiple diseases of malnutrition as the cause of metabolic bone disease at Quiani-7 needs to be discussed. As previously mentioned, many of the lesions we observed (e.g. osteopenia and abnormally heavy and extensive endochondral porosity) are common to a number of metabolic bone disorders such as rickets. The Atacama is a UVB rich environment and material cultural evidence from the Early Formative period Arica region suggests that clothing allowed sufficient cutaneous exposure for adequate vitamin D synthesis (Bird, 1943; Dauelsberg, 1974). Vitamin D deficiency has been clinically observed in swaddled infants in UV rich environments, however, as breast milk alone may not provide adequate vitamin D even if the mother is not technically deficient (Hollis and Wagner, 2004; Robinson et al., 2006).

Maternal calcium deficiency is also thought to cause rickets and/or osteopenia in perinates and it is possible that low dietary intake of this nutrient contributed to metabolic bone disease at this site (Brooke and Lucas, 1985; Backström et al., 1996). Dietary phosphorous deficiency is another potential cause of metabolic bone disease but this is extremely rare and usually only observed in cases of near total starvation as phosphorous is widely bioavailable (Waldholtz and Andersen, 1988; Ruppe and Jan de Beur, 2008).

4.3. Subadults as a population proxy and implications for vitamin C nutritional status at quiani-7

The potential of subadult remains as sensitive indicators of population-wide health status has been discussed elsewhere (Buckley, 2000; Lewis et al., 2007; Halcrow and Tayles, 2011; Snoddy et al., 2016). Due to their rapid growth and development, subadults are more sensitive to environmental insults than adults and are likely to be the first individuals within a population to experience the negative effects of resource scarcity (Schell and Knutsen, 2002; Snoddy et al., 2016). Foetal, perinatal, and infant individuals can be particularly informative from a bioarchaeological perspective as they are subject to extremely rapid skeletal development and will manifest disorders of bone formation or homeostasis due to poor nutrition within a relatively short period of time (Halcrow et al., 2017). However, caution is needed when attempting to differentiate pathological lesions from features associated with normal appositional and endochondral growth during the foetal/perinatal period.

Isolated cases of scurvy within a larger subadult cohort could be argued to be due to feeding practices or to the presence of another illness that leads to a higher individual requirement of vitamin C. Given the high prevalence of lesions suggestive of scurvy in the perinates in our sample, it is not unreasonable to consider vitamin C deficiency as a possible indication of a period of population-wide resource scarcity and low dietary diversity at Quiani-7; however, a larger and more demographically representative subadult cohort containing individuals old enough to exhibit unambiguous lesions of the disease would be needed to strengthen this argument.

4.4. Scurvy at quiani-7: resource scarcity, subsistence transition, or both?

As previously discussed, micronutrient malnutrition disorders can either be associated with sufficient caloric intake accompanied by low dietary diversity (e.g., intensive agricultural economies) or by general resource scarcity such as famine conditions. The Early Formative Period in the Atacama represents a movement towards intensive agriculture; however, palaeobotanical and zooarchaeological evidence from Quiani-7 indicates a mixed-horticultural economy accompanied by heavy utilisation of natural resources (Dauelsberg, 1974). Substantial reliance upon high-carbohydrate staple crops such as maize likely did not occur in this region until the Middle-Horizon Period (ca 1500-1000 BP) (King et al., in review). Additionally, the presence of subperiosteal new bone formation in our sample implies that vitamin C sufficiency was at least partially restored before death, suggesting periodic low dietary diversity rather than a static state of poor nutrient intake. Because of this, we argue that subsistence transition alone was unlikely the cause of vitamin C deficiency at Quiani-7.

Major climatic events, such as drought or cyclones, can cause periods of resource scarcity either through crop failure or sudden poor availability of natural resources (Rosenzweig et al., 2001). The extreme arid environment of the Atacama makes this region ecologically unstable in the face of such events (Stenseth et al., 2002; Houston, 2006a,b) and populations undergoing subsistence transition here would be particularly vulnerable to the negative effects of climate changes. The El Niño/Southern Oscillation (ENSO) phenomena are paired climatic events (El Niño/La Niña) that have a significant environmental impact on the western coast of South America (Jaksic, 2001). The ENSO phenomenon describes linked cyclic periodic changes in ocean temperature and atmospheric current in the Pacific, occurring approximately every two to five years (Guilyardi, 2006, 330). During El Niño cycles, the warming of the Pacific Ocean causes a massive decrease in marine biodiversity along the South American coast, with large-scale die-offs of microorganisms, mollusks, and vertebrates, as well as drastic changes in the composition of dominant pelagic species (e.g. warm water vs. cold water fish) (Sandweiss et al., 2004; Tam et al., 2008; Taylor et al., 2008). In contemporary human groups, these environmental changes can have devastating effects on the economy of population centres that rely heavily on marine resources (Badjeck et al., 2010). Intriguingly, palaeoclimatic work suggests that ENSO events increased in frequency in the Atacama around 3700 cal BP, and it has been hypothesised that this may have been a factor in the collapse of the maritime Chinchorro cultural complex and the subsequent transition to agriculture (Williams et al., 2008). In environments where vitamin C-rich plant resources are scarce, fish and marine mammal liver can be an important complementary source of ascorbic acid, allowing for sufficient intake of this nutrient in regions of low terrestrial biodiversity (Geraci and Smith, 1979; Fediuk et al., 2002). Naturally occurring terrestrial sources of vitamin C are scarce in the coastal Atacama Desert and confined to small fruit-bearing shrubs such as *Prosopis spp*. (Rundel et al., 1991). If the people of Quiani-7 were still largely dependant upon marine resources for their vitamin C intake, is possible that El Niño events could have disrupted one of their primary sources of ascorbic acid.

La Niña (cooling) events could also lead to resource scarcity, contributing to micronutrient 'famine' periods. La Niña causes dryer than normal conditions in western South America and is known to cause crop failure in contemporary human populations (Meza, 2013). Although vitamin C content varies somewhat between species and according to storage time and preparation techniques, squash, which is known to have been consumed at this site, is a relatively rich source of ascorbic acid, and regular consumption of this crop would likely have provided sufficient quantities of vitamin C to avoid scurvy (Kim et al., 2012). If cultivars, rather than marine resources, were the primary source of vitamin C for the people of Quiani-7, crop failure due to La Niña events could have caused periodic resource scarcity which led to the skeletal manifestations of scurvy we have reported. Nitrogen and carbon isotopic studies of the individuals from this site could provide more information on marine and terrestrial resources exploited here, placing our palaeopathological findings in a broader environmental context.

Isotopic work previously conducted on dental calculus from several individuals from Quiani-7 suggests a heavy marine signature (Poulson et al., 2013). However, analysis of dietary isotopes from dental calculus is problematic as the isotopic signatures of calculus and bone collagen are not equivalent dietary proxies (Salazar-García et al., 2014). Nitrogen isotopic analysis conducted on hair from a single mummified individual from this site was also suggestive of heavy marine consumption (Bonilla et al., 2016). However, these results fall outside acceptable parameters for isotopic analysis of hair as outlined by O'Connell and Hedges (1999), indicating possible diagenetic influence. Additional isotopic analysis using bone collagen is therefore necessary for more reliable dietary reconstruction at this site. Nitrogen isotopic analysis of the perinatal individuals could also provide information about physiological stress and maternal health at Quiani-7, as has been demonstrated in an archaeological population from Vanuatu (Kinaston et al., 2009). Analysis of microfossils in dental calculus may provide more direct evidence of cultivars and natural plant species consumed at Quiani-7 and should also be considered in future bioarchaeological work on this site. Tromp et al. (2016) have recently employed microfossil analysis of dental calculus for dietary reconstruction in the Pacific.

5. Conclusion

This paper has presented palaeopathological evidence of skeletal manifestations of scurvy in an Early Formative Period site in the Atacama Desert. One adult female, associated with a perinate of approximately 40 weeks, exhibits diagnostic lesions of this disease. All four perinates from this site also exhibit lesions suggestive of a systemic pathological process, although we caution that arriving at a reliable diagnosis of scurvy is difficult in individuals this young due to the overlap of some diagnostic features with those associated with rapid skeletal growth. Pregnant and lactating women and their offspring are known high-risk groups for diseases of low dietary diversity, such as vitamin C deficiency, and thus can be considered sensitive indicators of population-level nutritional status. We have argued that these findings may be indicative of periodic resource scarcity among the people of Quiani-7 and that when palaeoclimatic and palaeodietary evidence from this period and region is considered, this is more likely attributable to synergy of climatic events and subsistence transition than subsistence transition alone. Future work at this site might focus on more rigorous dietary reconstruction through nitrogen and carbon isotopic analysis of bone collagen and microfossil analysis of dental calculus in order to better characterise the subsistence transition in the Early Formative Period Atacama.

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