doi 10.4436/JASS.92005

JASs Reports
Journal of Anthropological Sciences
Vol. 92 (2014), pp. 257-271

Evidence of rickets and/or scurvy in a complete Chalcolithic child skeleton from the El Portalón site (Sierra de Atapuerca, Spain)

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Summary - *A case of what are most likely metabolic diseases is identified in a child buried during Chalcolithic times in the El Portalón site (Sierra de Atapuerca, Burgos, Spain). The skeleton has been directly dated by C14 to between 5030 to 5020 Cal BP. Macroscopic analysis and a CT scan reveal a set of lesions both in the skull and the long bones, which indicate that this individual probably suffered from rickets and scurvy at different stages of his/her life. The lesions are bilateral and are characterized by abnormal porosity, new bone formation and deformation of long bones. The presence of non-specific stress indicators, such as enamel hypoplasias and Harris lines, allow us to establish two times of stress associated with these pathologies: one crisis during infancy (1-3 yrs) and subsequently a second crisis at the beginning of childhood (3-5 yrs). The etiology of both metabolic diseases could be associated with abnormal feeding during these stages of life and/or the living conditions of these populations, e.g., the preparation of food and/or the existence of infections caused by the transmission of pathogens and unhealthy hygiene. Evidence of metabolic diseases during the recent European Prehistory is rather unknown and very few cases have been reported. Thus, the child from El Portalón can add relevant information about the life and health conditions of these prehistoric populations.*

Keywords – *Paleopathology, Stress indicators, Bone porosity, Enamel hypoplasias, Harris lines, Recent Prehistory.*

Introduction

The presence of metabolic diseases, such as anemia, scurvy or rickets, has been a major cause of mortality and morbidity during infancy and childhood since ancient times (Ortner & Mays, 1998). These life stages are critical periods in which sub-adults adapt to environmental, biological and cultural stressors (Wheeler, 2012). Stressors that lead to dietary problems typically appear with the spread of agriculture. A sedentary lifestyle, domestication, animal pathogens and a low diversity of food had serious consequences on the health status of these people (Cohen & Armelagos, 1984). Metabolic diseases are not always easy to diagnose in archaeological

remains. The skeletal manifestations or lesions left from stressors are similar. For instance, the cribra orbitalia is found in metabolic disorders associated with anemia, scurvy and rickets. A widened metaphyses of long bones and prominent ribs are associated with both scurvy and rickets (Aufderheide *et al.*, 1998; Ortner, 2003). We also have to consider the fact that the skeletal elements necessary to make a proper diagnosis are usually not well preserved in prehistoric samples, which is the reason why most research on scurvy and rickets focuses on the study of medieval and contemporary (s. XVII-XIX) remains (Ortner & Mays, 1998; Ortner *et al.*, 2001; Mays *et al.*, 2006; Mays *et al.*, 2009; Brown & Ortner, 2011), with few rickets cases reported in older historic eras in populations of ancient Egypt (Ruffer, 1911 in Aufderheide *et al.*, 1998 and Ortner, 2003).

Nevertheless, to this day, very few studies specific to rickets or scurvy have been conducted on prehistoric populations. Among them, we can mention Živanović (1982), who reported a case of rickets in a Neolithic child found at the Padina site in Djerdap (Serbia), dated to 7000 BC. Unfortunately, neither a description nor differential diagnoses were presented for this case. More recently, Mays (2008) reported a case of scurvy in a sub-adult individual from the Bronze Age in Wiltshire, England, which was the earliest reported example of scurvy in the British Isles.

The discovery of a complete skeleton of a buried child from Chalcolithic times (between 4600 and 3950 BP in this region, Delibes & Fernández, 2000) in the Atapuerca hills on the North plateau of the Iberian Peninsula provides the material necessary to appropriately diagnose these diseases and gives us the opportunity to shed light on the nutritional and health status of these human populations during recent prehistory. This is done by following criteria described in classical literature (Ortner & Ericksen, 1997; Mays *et al.*, 2006) and applying new technologies, which are described below. Furthermore, we explore the skeletal lesions present in this child and discuss the possible causes behind the development of these pathologies.

Material and Methods

The complete skeleton studied here represents a sub-adult (Atp'12.1420) (Fig. 1) recovered during the 2012 field season at the El Portalón site, in the Sierra de Atapuerca (Burgos, Spain) (Carretero *et al.*, 2008). The skeleton has been radiocarbon dated to 4350 ± 30 BP (Cal BP 5030 to 5020) and presents a good state of preservation, despite the fact that the skull is deformed due to taphonomic causes.

Since dental development is not as affected by environmental factors as the skeletal age indicators (Konigsberg & Holman, 1999), the age at death of Atp'12.1420 was estimated using tooth mineralization stages (Moorrees *et al.*, 1963) according to the tables proposed by Anderson *et al.* (1976).

Stature is estimated by using the regression formulae based on long bones length (specifically, the combination of femur + tibia) proposed by Smith (2007). The skeletal and dental remains of Atp'12.1420 are also analyzed to assess the overall health status. Prolonged exposure to unhealthy environmental and physical conditions can leave its mark on bones and teeth; when these indicators are recognized, they provide information about physiological stressors or are a reflection of poor health issues, such as weaning, infections famine or seasonal fluctuation of food (Ribot & Roberts, 1996; Buckley, 2000).

Both the cranial and the postcranial skeleton were analyzed macroscopically and also by electron microscope in the search for deformities, abnormal porosity and reactive woven bone formation added to the outer cortex of normal bone. Abnormal porosity in the skull vault was determined by the presence of fine pores (1 mm or less in diameter) that penetrated the lamellar bone surface (Ortner & Ericksen, 1997). Porosity was evaluated in the long bones in terms of the distance between the subchondral bone adjacent to the growth plate and the most distant margin of porosity along the shaft (Ortner & Ericksen, 1997). The absence or presence of roughening of the bone underlying the epiphyseal growth plates were also taken into account (Mays *et al.*, 2006).

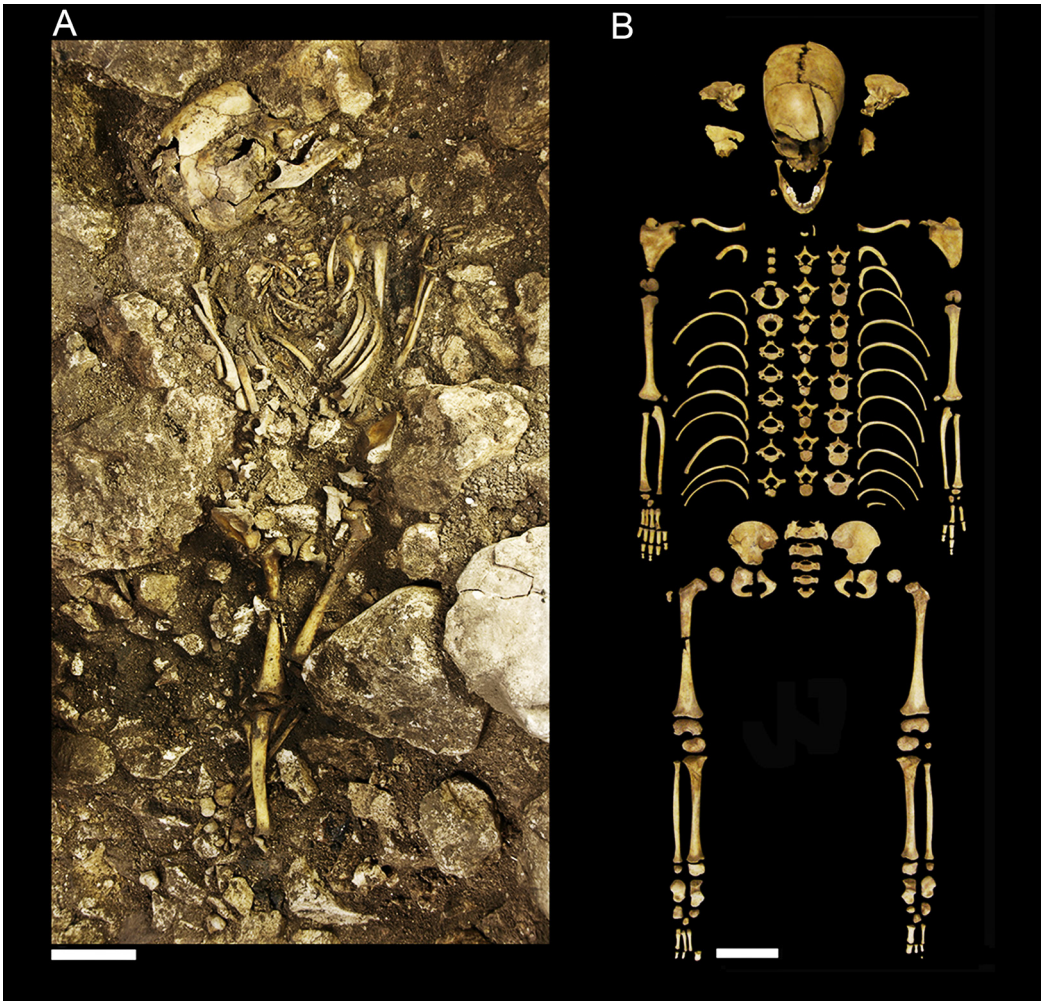

Fig. 1 - Skeletal remains of Atp'12.1420 during excavation in the archaeological site (A) and all recovered bones in the laboratory (B). Scale bars represents 10 cm. The colour version of this figure is available at the JASs website.

The maxilla, mandible and long bones were CT scanned (YXLON-Y CT-Compact) at the University of Burgos. An examination of these CT scans allow us to evaluate the bone structure and mineralization as well as to identify marrow hyperplasia, bending deformities, cupping of metaphyseal subchondral bone and fraying of bone beneath the epiphyseal plate (Thacher *et al.*, 2000; Pettifor, 2004). The CT images were imported to Mimics 10.0 software (Materialise, Leuven) for virtual reconstruction. Anterior–posterior radiographs of main long bones performed by CT scan were also used to identify Harris lines. Only lines transverse to the long axis of the bone with an extension of at least halfway across the bone width (Mays, 1985) were recorded. All long bones show Harris lines but the age of formation can only be estimated for the tibia using the Hummert & Van Gerven (1985) method for this bone. This method

requires, first, determinating the location of the primary center of ossification on the shaft, taking into consideration the fact that 43% of the tibia's shaft growth is distal. Once this is done, the distance between the primary ossification center and the Harris line was measured. In order to estimate the percentage of growth in the completed shaft at the time of line formation, the measured distance was divided by the diaphyseal length of the tibia, and the resulting percentage was compared with the table for tibial growth performed by Hummert & Van Gerven (1985) to calculate the age at which the line of growth arrest occurred.

Similarly, a dental study was performed while taking into account the presence or absence of caries, tooth wear, calculus and enamel hypoplasias both in deciduous and permanent teeth (Hillson, 2000). Enamel hypoplasias (EH) were observed using a JEOL JSM-6460LV scanning electron microscope with an accelerating voltage of 20kV and a magnification of 50x. Five permanent teeth were examined: upper and lower left central incisors (I^1- I_1), lower right lateral incisor (I_2), lower left canine (C) and lower right second molar (M_2). All forms of EH, including pits and furrows, were recorded. The duration of each enamel defect and the determination of the age at which defects occurred were calculated by adhering to the method described by King *et al.* (2005). Time of crown formation of each tooth analyzed was calculated assuming a perikymata periodicity of 9 days and by adding the averages calculated by Reid & Dean (2000) for the amount of appositional enamel present beneath the cusp tips. The age of occurrence of each EH was calculated by counting the number of perikymata present between the cusp tip and the enamel defect position and by adding the average age at which enamel becomes visible at the crown surface on each tooth. All calculations are shown in years.

Results

The state of root mineralization observed in the CT images allows us to estimate an age of 6.6 (SD 0.71) years if Atp'12.1420 were a male or 6.01 (SD 0.62) years if it had been a girl. These results are perfectly compatible with his or her state of tooth eruption.

The stature was estimated at 101.3 cm on average and is based on the length of the main lower limb long bones (femur and tibia). Compared to standards reported by Scheuer & Black (2000), the stature of this individual is below the normal range of variation for a child of 6-7 years.

Pathological lesions

Skull. The ectocranial surface of the skull vault shows a slight porosity (porotic hyperostosis, degree I Stuart-Macadam, 1982 in Ribot & Roberts, 1996), which extends bilaterally and symmetrically around all the cranial sutures (about 20 mm), as well as around the glabella in the frontal region. Both the external and the internal tables of the skull bones have a normal appearance. On the anterior area of the left orbit (Fig. 2A), there are foramina with a trabecular structure that penetrate the bone, which are lesions that are typical of orbital pitting (degree IV Stuart-Macadam, 1982 in Ribot & Roberts, 1996). The right orbit is not complete, but in the preserved area, there is a lesion similar to what is present in the left orbit. The external surface of the greater wings of the sphenoid bone displays an abnormal cortical porosity compared with other immature individuals of similar age (Fig. 2B).

Maxilla. The maxilla is complete and most of the teeth remain in position (emerged left and right permanent M^1 and left I^1 and the deciduous dm^1, dm^2 and deciduous canines). Except for the left dm^2, the remaining deciduous teeth show significant levels of dental wear, especially the right dm^1. The right dm^2 shows a cavity that does not penetrate the pulp (Fig. 2C). On the opposite side, the left dm^2 also presents a cavity in the interproximal and occlusal surfaces, which affects the pulp. The latter lesion is projected onto the cement-enamel junction of the root of the adjacent molar (left dm^1). A large lesion (8.6 mm BL and 4.9 mm MD) with destruction of the alveolar bone, together with periosteal remodeling, is observed in the tooth socket of this piece. Two linear EH were found by analyzing left I^1. The

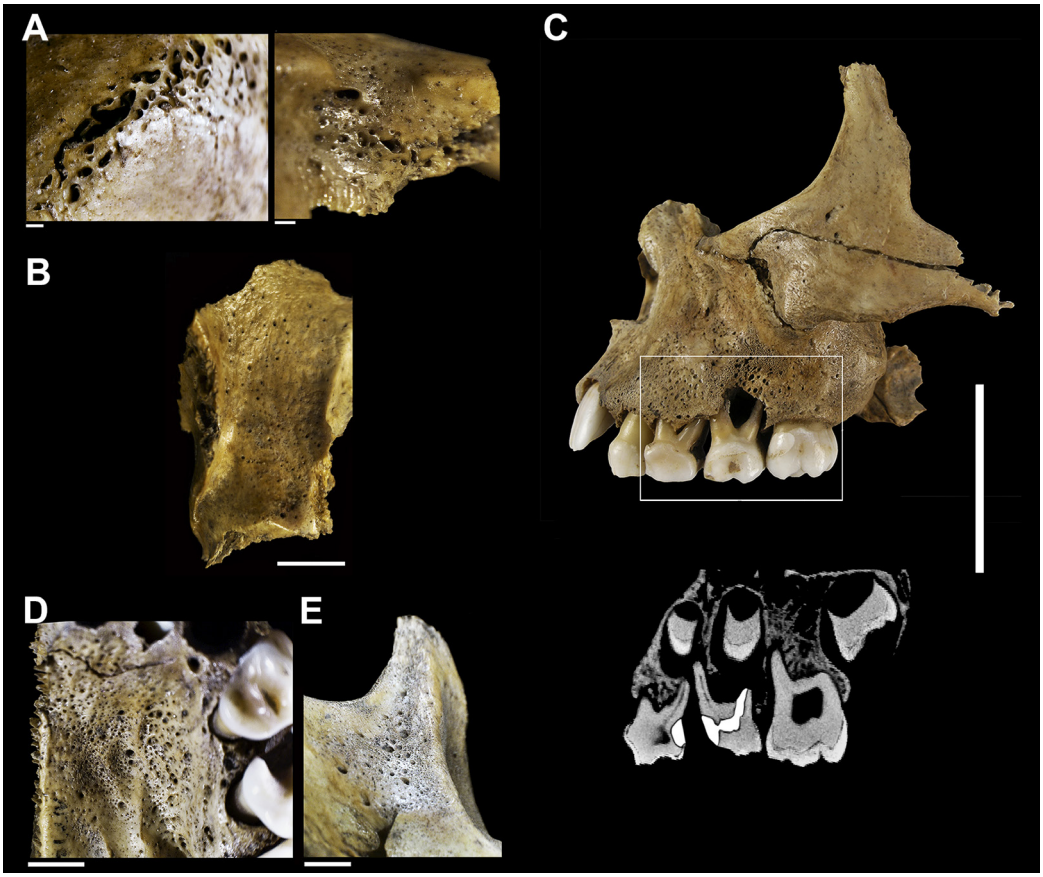

Fig. 2 - (A) Abnormal porosity linked to trabecular structure and associated with cribra orbitalia - from left to right - in anterior area of right orbit and in anterior area of left orbit. (B) Abnormal porosity along the outer surface of the left wing of the sphenoid bone. (D) Abnormal porosity on the palatal surface of the left side of maxilla. (E) Abnormal porosity on the left medial surface of the coronoid process of the mandible. (C) Lateral view of the left maxilla and CT-scan section of the same bone in a parasagittal plane through the level of the carious lesion of left dm^2 and dm^1 (shadow in white color). Scale bar represents 5 mm in A, D and E, 10 mm in B and 5 cm in C. The colour version of this figure is available at the JASs website.

periodicity of these enamel defects has been determined to have occurred around 1.5 and 3 years, respectively (Tab. 1). Furthermore, the maxilla displayed an increase in abnormal porosity in the posterior zygomatic process and from the alveolar region up to the nasal spine. This last case was observed together with new bone formation.

Similarly, abnormal porosity spread throughout the whole palate surface (Fig. 2D), albeit more developed on the left side. This palatal lesion extends beyond the alveolar process toward the transverse palatine sutures. Given the extension of the porosities beyond the alveolar region in both the unerupted and already erupted teeth, this porosity must be regarded as pathological and ruled out as evidence of normal growth from dental development (Ortner & Ericksen, 1997).

Mandible. The mandible is complete and it displays the same state of dental development as the maxilla (Ubelaker, 1978 in Scheuer & Black,

Table 1 - Number of nonspecific stress indicators (critical periods) detected in Atp'12 1420 distributed by age periods of one year.

STRESS INDICATOR		AGE PERIODS (IN YEARS)				
		1-2	2-3	3-4	4-5	5-6
ENAMEL HYPOPLASIAS (*)	Left I^1	1	1			
	Left I_1	2		2		
	Right I_2		1	1		
	Left lower Canine			2	4	
HARRIS LINES (**)	Right Tibia				2	3
	Left Tibia				3	1

(*) Age period for enamel hypoplasias was estimated following the method described by King et al. (2005).
(**) Age period for Harris lines was estimated following the method developed by Hummert & Van Gerven (1985).

2000). The occlusal surface of the deciduous teeth shows less wear than the maxillary teeth and there is no evidence of cavities. Enamel defects were found in three out of four mandibular teeth analyzed (left I_1, right I_2 and left C). Linear EH affected the left I_1 and right I_2, while pit-shaped defects affected only the lower canine.

Periodicity of mandibular EH defects covers a temporal range from 1 year to 5 years, with a peak between 3-4 years (Tab. 1). This spans a longer period of time than that observed for the upper dentition due to the inclusion of the canine in this analysis, which has a different time growth than the incisors (Hillson, 2000) (Fig. 3).

The mandibular coronoid processes display numerous foramina of different sizes that penetrate the cortex (see Fig. 2E). This lesion is manifested bilaterally and it is more developed on the left side. The porosity does not extend beyond the mandibular foramen, so, once again, it cannot be attributed to the normal growth process (Mays, 2008).

Postcranial Skeleton. The ribs exhibit normal curvature and the vertebral bodies do not present alterations either. Out of the upper limb long bones, both ulnas are the only elements that display an exaggerated posterior curvature of the proximal diaphysis (Fig. 4A). Both femora have normal curvature and there is no evidence of coxa vara or flattening of bone beneath the femoral head.

The tibia displays anterior bowing and the diaphysis of the fibula presents antero-posterior flattening. It is noteworthy that the middle third of the shaft of long bones, particularly that of the lower limbs, has numerous striations or small pores, in addition to the presence of new bone formation. All the long bone metaphyses are cup-shaped, but they do not show irregularities/roughening of the bone beneath the epiphyseal growth plates (Fig. 4B). By contrast, the distal epiphysis of the tibiae (Fig. 3B), distal femorae and distal right radius have Harris lines (HL). The age at formation of HL on the tibiae is from 4 to 6 years (Tab.1).

In sum, all pathological features detected in Atp'12.1420 (Tab. 2) can be attributed to two major stress crises based on dental and post-cranial evidence. The first crisis or infancy crisis happened at around 1.5-3 years old (EH in upper and lower first incisors) and a second or early childhood crisis at 3-5 years old (EH in lower dentition and Harris lines in the tibiae).

Discussion

The presence of cribra orbitalia and porotic hyperostosis can be associated with genetic or acquired anemia, such as iron-deficiency anemia (Ortner, 2003; Djuric *et al.*, 2008). Genetic anemias (e.g., thalassemia and sicklemia) are rare in comparison to the acquired anemias, but this is not to say that they did not occur in the past. In our case, a lack of facial deformities (Resnick, 1995) could rule out anemia of genetic origin. Moreover, even though genetic anemias are linked to rickets, symptoms usually manifest by 2 years of age and blood transfusions are needed for survival, so it is unlikely that children with genetic anemias would have survived beyond infancy in the past (Lewis, 2012). Despite the

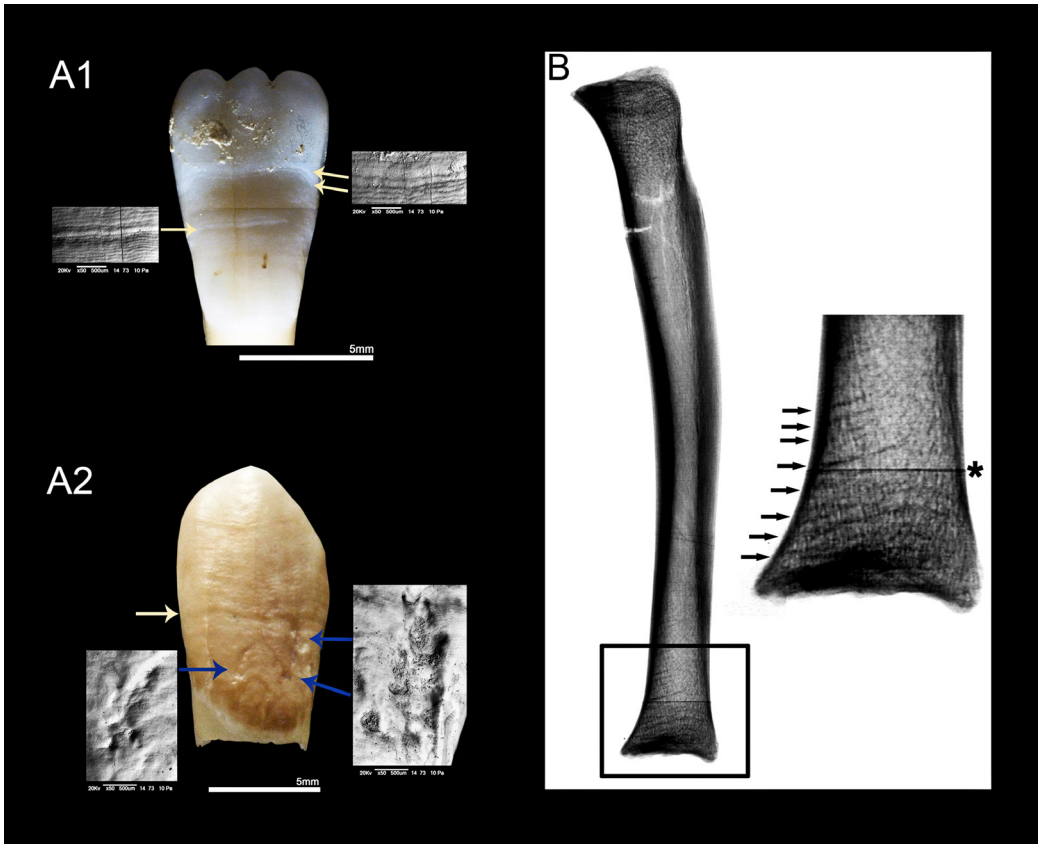

Fig. 3 - Non-specific stress indicators observed in Atp'12.1420 teeth. (A1) Linear enamel hypoplasias in lower permanent first left incisor indicated by arrows. (A2) Enamel hypoplasias in permanent lower left canine; linear defect (top to the left) and pit-shaped enamel defects (the other arrows). B: Lateral view of a radiography of the right tibia with an enlarged detail of the Harris lines in the distal epiphysis. The asterisk (*) indicates an artifact provoked by the x-ray. The colour version of this figure is available at the JASs website.

fact that these lesions (cribra orbitalia and porotic hyperostosis), in addition to EH and HL, are non-specific stress indicators (Ribot & Roberts, 1996), their presence can indicate several pathologies relevant to our case study: either rickets or scurvy (Stuart-Macadam, 1985; Ortner, 2003). It is during infancy when children grow quickly (Humphrey, 1998) and, therefore, it is during this period of life that these pathologies are more prevalent (Ortner, 2003). Nevertheless, a differential diagnosis of these two diseases must be based on as much pathological evidence as possible detected throughout the skeleton.

In our case and following what has already been pointed out by other authors (Aufderheide *et al.,* 1998; Ortner, 2003), the absence of marrow hyperplasia in the skull of Atp'12.1420 (absence defined as a lack of expansion of diploe at the expense of the outer table of the skull) most likely rules out a diagnosis of anemia, but, in contrast, the bowing observed in the shaft of some long bones could indicate a possible case of rickets (Ortner, 2003). Rickets is a metabolic disease caused by a deficiency of vitamin D (Jaffe, 1972) that produces a defect in bone mineralization during endochondral growth, which is why

Tab. 2 - Pathological features associated with rickets, scurvy or both that are present in Atp'12.1420.

RICKETS	BOTH PATHOLOGIES	SCURVY
Deformed arm bones [1]	Cranial vault porosity [1,2]	Greater wing of the sphenoid bone hyperostosis [2]
Deformed leg bones [1]	Orbital roof porosity [1,2]	Zygomatic porosity [2]
	Long-bone metaphyses flaring [3]	Maxilla porosity [2]
	Long-bone metaphyses porosity [1,2]	Infraorbital foramen porosity [2]
		Palate porosity [2]
		Coronoid process porosity[2]

[1] Following Ortner and Mays (1998). [2] Following Ortner et al. (2001). [3] Following Mays et al. (2006).

the most affected areas are the epiphyseal growth plates (Mays *et al.*, 2009). All Atp'12.1420 growth plates appear normal; therefore, we cannot rule out the possibility that this child was rickets-free well before its death. The child could have had rickets when he or she was still crawling and/or during the process of learning to walk. The posterior curvature of the ulna and the slight deformity of the lower limb are compatible with this interpretation, as in similar cases reported by Ortner & Mays (1998).

On the other hand, the bilateral abnormal porosity in some skull regions, the mandible, the maxilla, the hard palate and the greater wing of the sphenoid bones are not typical lesions of rickets (see Ortner, 2003). Instead, this specific distribution pattern of abnormal porosity fits much better with the lesions of scurvy (Ortner & Ericksen, 1997; Ortner *et al.*, 2001; Mays, 2008).

Scurvy is a deficiency in vitamin C (ascorbic acid), which humans are unable to synthesize and must obtain from dietary sources (Aufderheide & Rodríguez-Martin, 1998). A vitamin C deficiency causes severe impairment of collagen synthesis resulting in fragile blood vessels, hemorrhages and defective osteoid formation (Akikusa *et al.*, 2003). The porous lesions of Atp'12.1420 are probably the result of an inflammatory process and they are clearly associated with the anatomy of chewing, specifically with the *temporalis muscle* and the palatine artery (Brown & Ortner, 2011). Stress caused by muscle activity can lead to bleeding, since blood vessels are located nearer to the skin in these areas of the skull (Jaffe, 1972). This could also be true for the porous lesions present around the midshaft of long bones, which could be the result of vascular vulnerability and the subperiosteal hemorrhages between the cortex and periosteum (Ortner *et al.*, 2001).

Although both scurvy and rickets are metabolic diseases related to nutritional deficiencies, they can be discussed independently with respect to Atp'12.1420, since they apparently occurred at different times during the short lifespan of this individual. In this Chalcolithic child, the presence of two enamel hypoplasia crises (1-3 years and 3-5 years) and Harris Lines (around 4-6 years) reveals that this child experienced several growth disruption events.

Infancy crisis

As mentioned above, the case of rickets in Atp'12.1420 might have occurred at the end of the first year of life, while the child was still crawling or starting to walk. The presence of enamel hypoplasias in the incisors at around 1-3 years indicates some type of stress, and several authors have linked EH at this age with the introduction of foods other than breast milk in the diet (see Katzenberg *et al.*, 1996). Weaning is a gradual and variable process in current hunter-gatherer populations, and starts between 18-24 months of age on average (Tacher *et al.*, 2006). Studies

Fig. 4 - (A) Antero-medial view of the right ulna and X-rays of the same bone showing pronounced posterior curvature of the proximal diaphysis. B: Epiphyseal growth plates of some long bones from Atp'12.1420 showing the normal (non pathological) morphology that indicates absence of changes caused by active rickets. From top to bottom: proximal right humerus; proximal right fibula; distal left femur and distal left tibia. For the humerus, fibula and femur images, anterior is above and lateral is to the right. For the tibia, anterior is above and medial is to the right. Scale bar represents 5 cm. The colour version of this figure is available at the JASs website.

carried out on archaeological populations (Waters Rist *et al.*, 2011) show a similar pattern, with the initiation of the discontinuation of breast-feeding between 1.5 and 2 years old. Based on this information, the crisis of Atp'12.1420 (EH) occurred when all or most of his nutritional input was obtained through breast-feeding. Maternal milk contains low levels of vitamin D (Pettifor, 2004) and these levels can be reduced even more depending on the nutrition and the

health status of the mother. It is widely acknowledged that the health status of some prehistoric populations was negatively affected by the advent of agriculture (Cohen & Armelagos, 1984). This occurred as the result of the limited variability of food, and because the phytate content of the cereals inhibited the absorption of nutrients such as iron or calcium (Pettifor, 2004). Tacher (2006) demonstrated that Nigerian infants with rickets had mothers with lower breast-milk calcium concentration. While it is true that a poor quality of mother's milk plays an important role in the development of rickets, nutritional factors only come into play when ultraviolet radiation is insufficient (Henderson *et al.*, 1990). There are marked differences in the ability of humans to synthesize vitamin D according to latitude and season (Mays, 2008). At the latitude of the Sierra de Atapuerca (42ºN), present-day humans are unable to synthesize this vitamin between the months of November to February (Holick, 1990). Considering the fact that humans are capable of storing vitamin D in their bodies for extended periods of time (Robins, 1991), it is likely that the first EH crisis of Atp'12.1420 consisted of seasonal episodes and, if they were related to rickets because of a vitamin D deficiency, they were overcome when the child began walking and he/she was exposed to the sun in a more direct and habitual way.

The sequelae of rickets depend on the duration and severity of this condition (Ortner & Mays, 1998; Pettifor, 2004). For some researchers, such as Kreiter *et al.* (2000) and Holick (2005), one of the consequences of vitamin D deficiency is the delay in growth. Clearly these episodes of stress may have implications for a child's growth, to the point of slowing down the growth when there are deficiencies in feeding (Youmans, 1948). This defense mechanism allows them to reduce and delay their food requirements by redirecting nutrition from normal growth and development processes; hence the nutrients are used for daily survival. However, this hypothesis has been questioned (Rajah *et al.*, 2008; Pinhasi *et al.*, 2006). These authors have founded that a direct causal link between rickets and short stature does not exist and that the normal growth rate resumes once the disease has been overcome. In our case Atp`12.1420 shows a stature underneath that expected for their age.

Early childhood crisis

The physical growth of a child is the result of a complex interaction between genes and environmental factors (Tanner,1986). Furthermore, as part of these environmental factors, there are complicating and interacting factors presented by infectious diseases, inadequate nutrition (specifically a lack of key elements such as Vitamin D and protein), that appears to intensive shortly after the child has been weaned (Motarjemi *et al.*, 1993; Pinhasi *et al.*, 2006).

According to archaeological evidence unearthed during fieldwork at El Portalón, we can say that nutrition of these Chalcolithic and Bronze Age groups was based on the consumption of domesticated animals and their derivatives, along with various types of cereals. As a result, it is plausible that an inadequate intake of fruits and vegetables could have been the cause of scurvy for this child. Gastrointestinal disorders and parasitic factors may have also contributed to the scurvy condition (Fain, 2005).

In this regard, a very recent genetic study (Sverrisdóttir *et al.,* 2014) conducted on adult bones from El Portalón site of the same stratigraphical level as Atp'12.1420 has shown that this community either lacked the mutation that causes lactase persistence or the frequency was extremely low. It appears that individuals would have been able to consume fermented milk products, such as butter, yoghurt and cheese (fermentation converts much of the lactose into fats). This would be useful for survival during famine conditions or during seasonal shortages of food. Still, for some individuals consumption of fermented milk foods would have caused lactose intolerance symptoms, such as diarrhea; and this would have led to common nutritional deficiencies.

We should look beyond periods of famine for an answer of how or why scurvy might have been exhibited in this sample. Common culinary practices specific to cooking and food storage might be enough to cause the destruction of vitamin

C found in fresh meat (Akikusa *et al.*, 2003; Mays, 2008). In addition, ceramic vessels are the ideal substrate for the spread of bacteria and pathogens as a result of poor hygiene during the cooking of food (Motarjemi *et al.*, 1993). The presence of ill health caused by these pathogens, diarrhea and dysentery, clearly would exacerbate the loss of nutrients with serious consequences for childhood growth (Martorell *et al.*, 1975; Black *et al.*, 1984). They would also present a serious challenge for an immature immune system (Motarjemi *et al.*, 1993).

Several clinical studies have shown that infantile diarrhea can cause a failure to absorb ascorbic acid from the intestinal tract. This would not be clinically significant in acute diarrhea of a short duration, but if it is a protracted or chronic diarrhea, this would cause the development of scurvy in early childhood (Long *et al.*, 2007). This would be consistent with the presence of Harris Lines and enamel defects after three years of age observed in Atp'12.1420. The cause of these EH (after 3 years) cannot be associated with the weaning process, but rather, the most likely scenario is that they are related to the aforementioned increase in infectious diseases in early childhood. In addition, we must not forget that individuals with metabolic diseases are more likely to suffer from infectious diseases, since vitamin C is essential for fighting infections (Aufderheide & Rodriguez- Martin, 1998).

Life conditions and the archaeological record

The transition to agriculture had both a positive and a negative effect on human health. The negative effects include specific nutritional deficiencies and increase exposure to zoonosis pathogens (Cohen & Armelagos, 1984; Papathanasiou, 2005; Paine & Brenton, 2006; Eshed *et al.,* 2010). Therefore, it is difficult to imagine that metabolic diseases were not common during recent prehistory (few do). Nevertheless, surprisingly enough, there is only one reported case described exhaustively in recent literature (Mays, 2008). A review of the literature from the Iberian Peninsula indicates that most Holocene prehistoric burials contain bones of children, but once again, the information available about them is very limited (Ibañez, 2010). For example, besides the complete skeleton reported here, we have also found seven isolated bones (one right scapula; two right femora; two first upper incisors; and two fragments of distal metaphysis of the left femur) in the Chalcolithic level from Portalón. They represent seven other sub-adult individuals (Individual nº 1: prenatal. Individual nº2: six months old. Individual nº 3: one to two years; Individual nº 4: three to four years; Individual nº 5: six years old; Individual nº 6: eight and individual nº10: seven years) whose burials were altered by the Bronze Age people. As is the case in many sites, these isolated remains do not allow for intensive analysis. This adds even more value to Atp'12.1420.

Outside of the sphere of the Iberian Peninsula, the situation is relatively similar. Despite the high infant mortality rate (around 50%) expected in pre-industrial populations (Kamp, 2001), skeletal remains of sub-adults are scarce and not well preserved, and complete skeletons are extraordinary (Wood *et al.*, 1992). One reason for this lack of record could be attributed to differential preservation of sub-adult bones, but also to cultural beliefs that influence mortuary behavior (Bourbou, 2003). The studies of these remains are thus focused on the analysis of the dental pathologies (Goodman *et al.*, 1980) and, when preserved, on non-specific stress indicators on the postcranial bones (Lambert, 1993). Analysis of enamel Hypoplasias, cribra orbitalia or Harris lines reveal a high degree of physiological stress in these prehistoric populations (Robson, 2003) and, in most cases, are associated with deficiency processes due to weaning. But deaths usually attributed to weaning crises may conceal another type of more specific underlying pathology (Buckley, 2000). For example, when periostitis (non-specific stress indicators) appears in recovered isolated skeletal remains, it is usually related to an infectious process (Ribot & Roberts, 1996), but when more skeletal elements or complete skeletons can be studied, such as the one reported here, periostitis can be due to vascular vulnerability and the subperiosteal hemorrhages between the cortex and periosteum caused by scurvy (Ortner *et al.*, 2001).

Once the individuals surpass the critical stage of infancy, mortality decreases significantly (Wood *et al.*, 1992), but it is not rare to find skeletal remains of children over four years of age in archaeological sites. As pointed out by Bocquet-Appel & Masset (1997), mortality at these ages is an anomalous fact, probably related to catastrophic events such as famines or epidemics. In this sense, it is possible that constant famines caused by crop failure in prehistoric times (Robson, 2003) may have led to the development of metabolic diseases in children throughout the entire growth period, but both the scarcity of the archaeological record and the incompleteness of recovered skeletal remains makes it extremely difficult to render a conclusive diagnosis. In sum, a finding like Atp'12.1420 indicates that it is worth a through analysis of the few immature remains found in the recent archaeological record in order to gain more insight into possible causes of death and, therefore, the living conditions of those prehistoric populations.

Conclusions

The Chalcolithic sub-adult exhibited nonspecific stress indicators, such as enamel hypoplasias, cribra orbitalia and Harris lines, that indicate periods of stress during the infancy and early childhood. One crisis took place during infancy at 1-3 years (EH in upper and lower first incisor) and a second crisis occurred during early childhood at 3-5 years (EH in lower dentition and Harris lines in the tibiae).

A more comprehensive review of the remains of this child allows to relate different nonspecific stress indicators with specific diseases. In the crisis suffered during infancy, the curvature of limbs indicates that Atp'12.1420 could may have suffered a dietary deficiency episode leading to rickets exceeded at the time of his/her death. Subsequently in the crisis suffered during childhood, abnormal porosity of the skull suggests a possible case of scurvy.

It is very difficult to determine the exact causes of the skeletal lesions in this individual. It appears that they are the result of several metabolic disorders. Breastfeeding and the general health of the mother, nutritional availability after weaning, and multiple environmental stressors (bacteria, and parasites) probably played a role in the etiology of these lesions. Skeletal lesions in young children are rare in the archaeological record and each case helps to reveal life conditions. As more examples are found and reported on, the better we will be at interpreting the past we as attempt to reveal the human condition.

Acknowledgements

We are grateful for the support Professor Robert Paine has given us throughout the revision of the manuscript and for his ideas and feedback to improve this work, as well as by his effort with the English edition of the text. We have benefitted from fruitful discussions with our colleagues from the Centro UCM-ISCIII of Madrid and from the Laboratorio de Evolución Humana (L.E.H.) at the University of Burgos. We thank Laura Rodriguez and Elena Santos for her technical support with the CT-Scan and Rebeca García for her assistance with the Electron Microscope. Thanks to Amalia Pérez and Eneko Iriarte for their comments and suggestions. This research was supported by the Ministerio de Educación y Ciencia Projects No. CGL2009 12703-C03-03 and 2012-38434-C03-01 and by the Junta de Castilla y León Project No. BU005A09. Fieldwork at the Atapuerca sites is funded by the Junta de Castilla y León and the Fundación Atapuerca. Finally we thank Emiliano Bruner and the anonymous reviewers for their useful suggestions and comments.

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Associate Editor, Emiliano Bruner