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An Ectocranial Lesion on the Middle Pleistocene Human Cranium From Hulu Cave, Nanjing, China

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ABSTRACT The earlier Middle Pleistocene human partial cranium from Hulu Cave, Tangshan, Nanjing (Hulu 1) exhibits an ectocranial lesion which covers most of the anterior neurocranium, largely between the temporal lines and extending from the supratoral sulcus to the anterior parietal bone. The endocranial surfaces and the remainder of the cranium (upper facial skeleton, lateral frontal bone, posterior parietal bones, and mid-occipital bone) are normal. The healed lesion exhibits both resorption and the laying down of new bone. Differential diagnosis suggests that the lesion was caused by either trauma (broad compressive trauma, tensile trauma to the scalp, or partial scalp removal) or burning (with damage to scalp and superficial neurocranium). Dietary deficiencies, infection, and neoplastic disorders do not fit the lesion characteristics. The Hulu 1 specimen therefore joins a growing sample of Pleistocene *Homo* remains with nonfatal and nontrivial disorders. Am J Phys Anthropol 135:431–437, 2008.

As the human fossil record has increasingly been investigated from a paleopathological perspective, it has become apparent that pathological skeletal lesions were relatively common among Pleistocene human foraging populations. In addition to primary documentation and diagnosis of these lesions, there have been attempts to assess the degrees to which the severity and survival of these biological insults may relate to aspects of Pleistocene human behavior, including diet (both quality and quantity), mobility, technology, risk of trauma, interpersonal violence, survival from serious injuries or congenital disorders, and social caring (e.g., Walker et al., 1982; Trinkaus, 1983; Berger and Trinkaus, 1995; Tillier et al., 2001; Lebel and Trinkaus, 2002; Zollikofer et al., 2002; Bräuer et al., 2003; Guatelli-Steinberg et al., 2004; Lordkipanidze et al., 2005; Trinkaus et al., 2006). All of these assessments are dependent upon preservation, sample sizes, and diagnoses, all ongoing issues given the nature of the human fossil record.

As a contribution to this ongoing documentation and discussion we describe and assess a pathological lesion covering most of the anterior neurocranial vault of the Hulu 1 earlier Middle Pleistocene cranium. The lesion was mentioned by the Tangshan Archaeological Excavation Organized by the Nanjing Municipal Museum and Archaeology Department of Peking University (1996) and by Wang (2001), but this presentation builds primarily on the preliminary description of this lesion by Shang et al. (2002) based on a cast, photos and information provided by Wu X.

HULU 1: DISCOVERY, CONTEXT, AND GEOLOGICAL AGE

In 1992 scientists from the Nanjing Institute of Geology and Palaeontology (NGIP), Chinese Academy of Sciences investigated the karstic Hulu Cave (Huludong), near the town of Tangshan (32° 03′ N, 119° 03′ E) in the district of Nanjing in eastern China (Mu et al., 1993; Wu et al., 2002a). On the basis of the mammalian fossils discovered in the clay deposits of the Hulu Cave, it was suggested by Wu Q. of the Institute of Vertebrate Paleontology and Paleontology (IVPP) in Beijing that the site should be close in age to Zhoukoudian Locality 1 and that human remains may be preserved in Hulu Cave. As a result, the joint NIGP and IVPP Tangshan Cave Working Group were formed and investigated the cave intermittently during 1992 and 1993. It was during this period, in 1993, that the Hulu 1 cranium (also known as: Nanjing 1 and Tangshan 1) was discovered by a local laborer securely in the lower clay level (Wu et al., 2002a).

Uranium-series dates on speleotherms from Hulu Cave were run using thermal ionization mass spectrometry (TIMS), providing a range of dates in the Middle Pleistocene and an age for the Hulu 1 cranium of >500 ka BP (Chen et al., 1996, 1998; Wang et al., 2002). In addition, three TIMS U-series dates on a flowstone immediately overlying the level of the Hulu 1 cranium provided a mean minimum age for the cranium of 577 + 44/−34 ka BP (Zhao et al., 2001). This is inferred to mean that the flowstone formed during oxygen isotope stage (OIS) 14 or 15 with its mean age assigning it to the

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warmer OIS 15. Since the fossil is associated with a cold climate fauna (Xu et al., 1993; Liu et al., 1998) and the human fossil predates the formation of the flowstone, it is likely that Hulu 1 derives from OIS 16 (~620 ka BP) or slightly older deposits (Zhao et al., 2001). In addition, an amino acid racemization dating of fossil cervid teeth from the same stratigraphic level as Hulu 1 provided an age of about 624–638 ka BP (Liu et al., 2002). The fossil therefore securely derives from the first half of the Middle Pleistocene.

HULU 1: PRESERVATION

The Hulu 1 cranium preserves three pieces of cranial bone, a calvarial-facial piece (see Fig. 1), an occipital and posterior left parietal section, and a smaller right parietal piece (Wu et al., 2002b). The latter two pieces join along the lambdoid suture, but they are separate from the anterior cranial section.

The frontal bone is largely complete, especially on the left side. It retains all of the left squamous portion and supraorbital region, most of the left orbital roof, the interorbital portion, the right supraorbital torus to approximately midorbit, and the right squamous portion from the midline to the region of stephanion. There is a large bregmatic sutural bone, 22.5 mm anteroposterior by 13.5 mm transversely on the exocranial surface. The anteromedial right parietal bone extends ~29 mm from the coronal suture along the sagittal suture and then laterally to the region of the temporal line. The left anterior parietal piece is intact along the coronal suture from bregma to the region of pterion with ~30 mm of the anterior squamous suture and ~40 mm of the anterior sagittal suture. The left parietal bone is complete 10–14 mm more posteriorly endocranially than exocranially.

The facial skeleton retains all of the left nasal bone, the posterosuperior half of the right nasal bone, the complete left zygomatic bone, and the anterior facial surface of the left maxilla. The maxilla retains the bone between the nasomaxillary and zygomaticomaxillary sutures, the orbital margin, and the superior half of the lateral nasal aperture margin; its inferior horizontal break is just below the level of the inferior zygomatic bone. In addition, a portion of the left inferolateral orbital floor is preserved, and with it the anterosuperior and superior walls of the left maxillary sinus.

The preserved occipital bone retains the majority of the left nuchal plane from the midline to close to the left asterion, plus the medial half of the right nuchal plane. The nuchal torus (and associated superior nuchal line) is preserved similarly from left asterion to the mid-right side. The inferior portion of the occipital plane remains up to ~20 mm from the nuchal torus, along with a portion of the left lambdoid suture extending superomedially from asterion and a smaller portion of the right lambdoid suture above the lateral occipital break. To the left lambdoid suture is attached a portion of the posteroinferior left parietal bone, with the posterior curve of the temporal line and most of the angular torus. The right parietal piece, also posteroinferior, is smaller than the left one but preserves much of the same anatomical region. There is no contact between the posterior and anterior parietal sections, with the minimum gap between the portions of the left parietal bone estimated to be 15–20 mm (Wu et al., 2002b).

The retained internal and external neurocranial surfaces are intact with little abrasion or chemical erosion.

Fig. 1. Superior view of the Hulu (Nanjing) 1 anterior cranium, showing the pervasive lesion on the frontal squamous and adjacent anterior parietal bones.

The only damage to the preserved surfaces is along the various breaks.

The age-at-death of the individual was originally estimated by Wu et al. (2002b) to be <50 years and probably between 21 and 35 years based on the endocranial and exocranial patent nature of the coronal, anterior sagittal, lambdoid, and sphenofrontal sutures. Given the large range of variation in the obliteration of these sutures (Jackes, 2000), it is best to say that Hulu 1 was fully mature and not geriatric.

HULU 1: ECTOCRANIAL LESION

The bones of the facial skeleton, including the orbital walls and the maxillary sinus, are free of any pathological alterations, as are all of the preserved endocranial surfaces of the frontal bone, the parietal bones, and the occipital bone. The posterior parietal and occipital pieces are normal externally. There is no indication of a fracture to the facial or vault bones. The posterior and right side postmortem breaks through the lateral frontal bone and the anterior parietal bones, as well as the posterior parietal and occipital bones, show no expansion of the diploë and no involvement of the diploë or the internal table in any abnormalities. However, the external surface of the anterosuperior neurocranium exhibits a large and irregular area, a lesion which covers most of the frontal squamous and adjacent areas of the parietal bones (Figs. 1 and 2).

The lesion is a rough irregularity of the external table. Anteriorly, it begins ~20 mm above glabella on the midline and ~18 mm from the anterior margin of the middle of left supraorbital torus, at the level of supratoral sulcus. Posteriorly, it extends ~27 mm from the right coronal suture to the break on the right parietal bone. It continues around the right side of the bregmatic ossicle to the sagittal suture on the anteromedial corner of the left parietal bone and then to the postmortem break,

≥36 mm from the coronal suture. It is ~18 mm from the coronal suture on the left parietal bone lateral of the lesion around the bregmatic ossicle. As such, it involved most of the bregmatic ossicle.

The maximum dimensions of the primary portion of the lesion are ≥100 mm anteroposteriorly to the midline parietal break posterior of the ossicle and ~72 mm transversely. Given its approximately ellipsoid shape, the estimated surface area of the lesion is therefore ~56 cm^2. The primary central and roughened portion of the lesion is ~62 mm anteroposteriorly by ~55 mm transversely, but it is bordered on each side by raised, rounded, anteroposteriorly oriented margins, which are 12–15 mm wide on the right and 10–12 mm wide on the left.

On the left side, the raised and rounded margin extends to the temporal line, blending with it for ~54 mm from the posterior temporal crest to slightly posterior of the coronal suture. The margin on the right side is similar, but it does not appear to have approached the temporal line except near the coronal suture. It is unclear how far posterolaterally the lesion may have extended on the right side, since it was truncated by the break on either side of the coronal suture near the right stephanion, especially on the parietal bone. It therefore appears that the lesion was constrained on the left by the temporal muscle to the original temporal line, but the lesion extended through the temporal line (or altered the attachment of the temporal fascia) on the right side to an indeterminate amount. There is also a groove on the left parietal bone (Fig. 2: No. 4), extending posteriorly ~27 mm from the posterior edge of the lesion to the broken edge of the bone; it is not clear whether it is part of the lesion, but it may be a vascular sulcus secondary to or independent of the lesion.

The central portion of the lesion is an irregular and rugged area, with an anteroposteriorly oriented depression to the left of the midline and then a longitudinal and thin groove closer to the midline with radiating medial and lateral grooves coming from it. At the anterior end there is an arc of small, raised, rounded eminences of bone. These eminences continue onto a roughened area that arcs posteriorly to the right of the midline, with a series of cord-like structures that are 10–15 mm in length (Fig. 2, No. 2). The eminences, anteriorly and right lateral, as well as fine grooves in the left side depressed area, appear as though they follow lines radiating from a point in the middle of the lesion. None of the bone within the raised right, left, or posterior (parietal) margins of the lesion represents the original subpericranial surface; it has all been altered by a complex process of resorption (mostly to the left of the midline) and deposition (mostly along and to the right of the midline), with extra bone laid down around the margins.

There appears to be no antemortem porosity of the surface; what appears as small defects in the exocranial surface (see Fig. 1) are areas of postmortem mineral staining. Consequently, none of the bony lesion was active at the time of the individual's death.

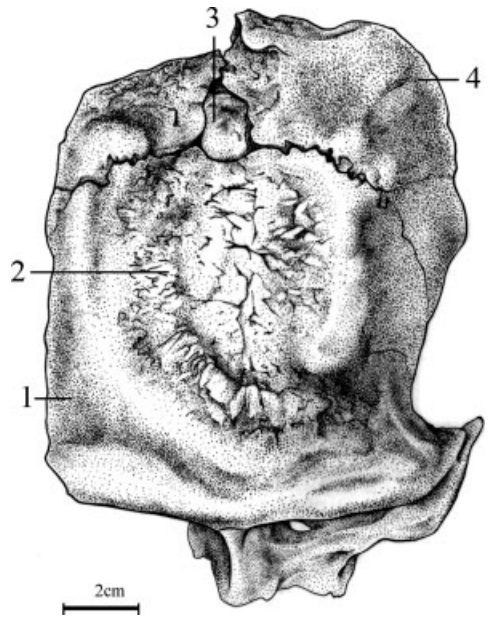

Fig. 2. Drawing of the Hulu (Nanjing) 1 anterior cranium in superior view. 1, anterolateral margin of the marginal swelling on the right side. 2, cord-like structures that arc anteriorly and laterally on the right side of the primary lesion area. 3, bregmatic ossicle covered and surrounded by the lesion on the posterior frontal bone and anteromedial parietal bones. 4, shallow groove extending posterolateral from the lesion area on the anterior left parietal bone.

DIFFERENTIAL DIAGNOSIS

A variety of abnormalities can produce pathological alteration of the cranial vault bones, including dietary deficiencies (anemia), infectious disease (syphilis and tuberculosis), tumors (osteoblastomas, meningiomas, and hemangiomas), periostitis, trauma, and burning (cf. Steinbock, 1976; Ortner and Putschar, 1981; Zimmerman and Kelley, 1982; Aufderheide and Rodríquez-Martin, 1998).

Anemia-related porotic hyperostosis is the result of compensatory expansion of the hematopoetic marrow in the diploic space, causing porosity of the external table, with expansion of the diploic honeycomb through the external table in extreme cases (Stuart-Macadam, 1992). The absence of diploic expansion in the broken edges of the parietal and frontal bones, porosity to the external table, or internal table involvement makes this an unlikely diagnosis.

Although tuberculosis can occasionally form frontal lesions, it is primarily a lytic process that results in multiple small areas of bone destruction (Aufderheide and Rodríquez-Martin, 1998). Syphilis can also cause cranial vault lesions, either localized or widespread across the neurocranial vault (Steinbock, 1976), but it is also primarily a lytic process and therefore does not fit the pattern of bone alteration present in Hulu 1. Moreover, syphilis and other treponemal infections can also produce facial bone alterations, and such lesions are absent from at least those facial regions preserved on Hulu 1. In addition, it is unclear whether mycobacterial or treponemal diseases were present in the Middle Pleistocene of the old world (Rothschild and Turnbull, 1987; Baker and Armelagos, 1988; Aufderheide and Rodríquez-Martin, 1998).

Tumors affect the external surfaces of the cranial vault bones, but they tend to be rare and are normally associated with diploic and endocranial alterations. Osteoblastomas occur extremely rarely on the periosteal surfaces of calvarial bones endocranially or exocranially, but the area of involvement on the bone is usually small

($\leq$2 cm in diameter) (Cervoni et al., 1997; Lin et al., 2005). Meningiomas are soft tissue neoplasms arising from mesothelial cells of the dura mater. They are therefore of intracranial origin, such that the tumor primarily affects the internal table by pressure atrophy or by direct invasion and erosion. In the rare cases in which they are sufficiently extensive, the meningioma may become intraosseus (or intradiploic), extend onto the external surface of the cranial vault (Cirak et al., 2000; Agrawal et al., 2007; Jovanovic et al., 2006), or into the paranasal sinuses (Swain et al., 2001), especially if untreated for extended periods of time (Michalik et al., 2006). In cases in which they are intradiploic, they may displace the external table outward, producing abnormalities of the external subpericranial bones. Hemangiomas are generally benign, solitary tumors formed by proliferating blood vessels, varying in size from small to moderately large (Politi et al., 2005). Very rarely (<1% of cases) they may be found in the cranium, including the parietal and frontal regions (Khanam et al., 2001; Paradowski et al., 2007). However, as with porotic hyperostosis, the lesion originates in the diploic space, and it can erode through either table, producing openings on the external surfaces.

Although it is possible that any of these neoplasms might produce the kinds of alterations evident on the external surface of the Hulu 1 frontal bone, none of them fits the overall Hulu 1 pattern. The area of involvement is too large to be likely to have been caused by an osteoblastoma. Both meningiomas and hemangiomas produce pronounced intradiploic changes if they affect the external table, and there is no evidence of diploic expansion and/or internal or external tabular bulges. The absence of endocranial alterations also contradicts the pattern of meningiomas. Moreover, all of these tumors occur very rarely, and although some prehistoric crania have been diagnosed with one or the other of them (cf. Aufderheide and Rodríquez-Martin, 1998), it would be truly exceptional to find one in a Middle Pleistocene cranium.

Periostitis tends to be a more of a descriptive term than a diagnosis, since it involves inflammation of the periosteal (or pericranial) tissues and associated subperiosteal bone alteration, and it can have a multitude of ultimate causes. Diagnosis of its cause usually requires more complete remains than are available for Hulu 1. It is possible that the frontoparietal lesion of Hulu 1 is the product of a localized periostitis, although it would not be possible to determine its ultimate etiology if it were.

The lesions on Hulu 1 could have resulted from traumatic injury to the anterior neurocranium. This could have been a broad superficial compressive injury to the majority of the area of the lesion, with resultant damage to the underlying scalp and pericranium. The tissue damage and resultant osseus reactions, including both new bone deposition and necrotic bone resorption, could account for the lesion on the frontal and parietal bones. However, one would have to invoke a broad enough impact area to affect much of the lesion area, limit its extent largely between the temporal lines, affect only the external table, and yet not fracture the bone itself. Superficial (i.e., external table) neurocranial vault lesions, at least among other Pleistocene humans, tend to be more localized, resulting in either a linear defect or a subcircular one less than $\sim$30 mm in diameter (cf. Keith, 1927; Weidenreich, 1943; Trinkaus 1983; Pérez et al., 1997; Kricun et al., 1999; Trinkaus et al., 2006). Yet, sufficient damage to the scalp could produce a serious hematoma deep to the galea aponeurotica (Cooling and Viccellio, 1991).

An alternative injury would be a tensile one in which the scalp is forcibly pulled away from the neurocranium but neither torn nor removed (a hair-pulling injury). In such cases, the pericranium is not directly impacted, but a large subgaleal hematoma can occur (Yip et al., 2003; Seifert and Püschel, 2006).

A subgaleal hematoma, whether from compressive or tensile trauma, can spread through the subaponeurotic tissues and hence along the pericranium, affecting the underlying bone. The extension of the galea aponeurotica and the subaponeurotic fibroadipose tissue laterally to the zygomatic arches superficial to the temporal fascia means that a hematoma should remain external to the temporal fascia, which would agree with the restriction of the Hulu 1 skeletal lesions largely medial to the temporal lines, especially on the left side. Less clear is the distinct limitation of the lesion at the supratoral sulcus, since the loose subaponeurotic tissue should extend to the orbits, assuming that its attachments in Middle Pleistocene *Homo* with large supraorbital tori were the same as in recent humans. Subgaleal hematomas in modern humans are known to expand into the orbits and produce ocular abnormalities (Pope-Pegram and Hamill, 1986; Yip et al., 2003; Seifert and Püschel, 2006).

A more extreme scenario would be scalping of the individual, accidental or intentional traumatic removal of a portion of the scalp. With the removal of a portion of the scalp, in the absence of cutmarks into the external table, the localized exocranial surface should have a series of pathological changes, including bone necrosis and the presence of inflammatory granulation tissue in the diploë which isolates the necrosis from the underlying normal skull tissue. When new bone regenerates from the remaining diploë, the necrotic outer table is shed (Hamperl and Laughlin, 1959; Smith, 2003). The completely healed bone surface is characteristically depressed and relatively smooth (Ortner and Putschar, 1981; Smith, 2003). Documented cases in which the individual survived for some period of time (Hamperl and Laughlin, 1959; Ortner and Putschar, 1981; Aufderheide and Rodríquez-Martin, 1998; Smith, 2003) result in both necrotic bone resorption of portions of the external table and the subsequent partial regrowth of the exocranial bone in the affected area. The lesion on Hulu 1 conforms to this general pattern in that it has a well-demarcated, generally smooth but unevenly remodeled surface with coarsely pitted or nodular bone. It resembles the lesion on a 20th century documented case [PMES 1.EB.1 (6) from the Pathology Museum of the Royal College of Surgeons of Edinburgh (Ortner and Putschar, 1981)] of a woman with industrial traumatic evulsion of the scalp from the parietal region; both of them have similar changes to the external table, although Hulu 1 lacks the marginal porosity of the recent individual, indicating the absence of associated hypervascularity and inflammation on Hulu 1. The extent of postnecrotic resorption in historically documented cases appears to be greater than what is likely to have occurred with Hulu 1, although long term survival and extensive healing might account for the differences between Hulu 1 and documented historical cases of *in vivo* scalping, both accidental and intentional.

Finally, the Hulu 1 frontal bone lesion fits the pattern of changes associated with a serious burn of the scalp (Law et al., 1992; Shen et al., 1995; Gümüş et al., 2006; Yeong et al., 2006). Such a burn can produce destruction of any hair, the scalp tissues, the external table, and even necrosis of the full calvarial thickness. Normal healing, assuming that the tissue damage is not too extensive, would then involve gradual replacement of the superficial tissues, resorption of any necrotic bone, and subsequent partial regrowth of the external calvarial bone. The extent of tissue regrowth and bone remodeling would, of course, be dependent on the extent and depth of the original burn. The Hulu 1 scar could conform to a healed burn if the tissue destruction was limited to the scalp (and any forehead hair), the underlying pericranium and possibly the external surface of the external frontal squamous table. The limitation of the lesion largely to the area between the temporal lines could mean that the burn was centrally located and/or that the lateral cranial vault was protected by the temporal muscles and fasciae.

From these considerations, the frontoparietal lesion of Hulu 1 could have been caused by localized periostitis of an indeterminate etiology, but it was more probably the result of localized trauma and/or scalping with secondary effects and healing or the product of a serious burn to the forehead. It is also possible that the bony scar is the result of trauma followed by periosteal inflammation and/or an extensive subgaleal hematoma. In either case, the nature of the bony scar indicates that the abnormality covered most of the anterosuperior cranial vault, but that it was completely healed by the time of the death of the individual.

DISCUSSION AND CONCLUSION

It is therefore apparent that the Hulu 1 individual sustained a traumatic alteration of the anterior scalp, a serious neurocranial burn some time before death, and/or (but less likely) a large scale periosteal reaction. The infliction appears to have been localized to the anterior external neurocranial vault, at least of the cranium, given the absence of any pathological changes in the facial skeleton, the endocranial neurocranium, or the posterior neurocranium. Moreover, any one of these diagnoses implies that it was not a systemic disorder but one due to an insult to the head.

As such, the lesion would have involved serious localized pain, and possibly a major level of hemorrhaging and/or subsequent sensitivity. If the lesion was due to some form of trauma, there is a variety of possible accidental or intentional causes, none of which can be identified from the lesion or its location. If the lesion was secondary to anterior cranial burning, it would have occurred close to the time of the earliest evidence for the use of fire in at least Eurasia; the oldest evidence for fire in east Asia is from the $\leq$500 ka BP site of Zhoukoudian Locality 1 (Weiner et al., 1998), although secure evidence for fire and hearths is present at the $\sim$800 ka BP site of Gesher Benot Ya'aqov in southwest Asia (Goren-Inbar et al., 2004; Alperson-Afil et al., 2007).

The Hulu 1 cranium therefore joins a growing series of Pleistocene human remains with nontrivial pathological alterations. There is a number of cases of such skeletal changes among Late Pleistocene archaic and modern humans (e.g., Trinkaus, 1983; Duday and Arensburg, 1991; Berger and Trinkaus, 1995; Kricun et al., 1999; Tillier, 1999; Schultz, 2006; Trinkaus et al., 2006). In addition, such cases are becoming increasingly documented for Early and Middle Pleistocene human remains. These earlier ones include probable dietary deficiencies in KNM-ER 1808 and Eliye Springs KNM-ES 11693, developmental abnormalities in Salé 1, Singa 1 and probably Berg Aukas 1, and an infectious disorder in Broken Hill 1 (Walker et al., 1982; Hublin, 1991; Montgomery et al., 1994; Spoor et al., 1998; Trinkaus et al., 1999; Bräuer et al., 2003). These are joined by traumatic (or probably traumatic) cranial lesions on Lantien (Gongwangling) 1, Ceprano 1, Zuttiyeh 1, at least eight of the Atapuerca-SH crania, and several of the Zhoukoudian Locality 1 remains (Keith, 1927; Weidenreich, 1943; Caspari, 1997; Pérez et al., 1997; Manzi et al., 2001) and by serious dentoalveolar abnormalities on Atapuerca-SH 700/721/888, Aubesier 11, Broken Hill 1, Dmanisi 3444/3900, and Ehringsdorf 6 (Carter, 1928; Vlček, 1993; Pérez et al., 1997; Lebel and Trinkaus, 2002; Lordkipanidze et al., 2005).

Together these remains document the probably high level of risk to which these pre-Late Pleistocene humans were subjected. These remains also document their ability to survive both minor and major abnormalities, since all of these lesions document some degree of survival.

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