Antemortem trauma and survival in the late Middle Pleistocene human cranium from Maba, South China

Xiu-Jie Wu[a,1], Lynne A. Schepartz[a,b], Wu Liu[a], and Erik Trinkaus[c,1]

[a]Key Laboratory of Evolutionary Systematics of Vertebrates, Institute of Vertebrate Paleontology and Paleoanthropology, Chinese Academy of Sciences, Beijing 100044, China; [b]School of Anatomical Sciences, University of the Witwatersrand Medical School, Johannesburg 2193, South Africa; and [c]Department of Anthropology, Washington University, St. Louis, MO 63130

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Paleopathological assessment of the late Middle Pleistocene archaic human cranium from Maba, South China, has documented a right frontal squamous exocranially concave and ridged lesion with endocranial protrusion. Differential diagnosis indicates that it resulted from localized blunt force trauma, due to an accident or, more probably, interhuman aggression. As such it joins a small sample of pre-last glacial maximum Pleistocene human remains with probable evidence of humanly induced trauma. Its remodeled condition also indicates survival of a serious pathological condition, a circumstance that is increasingly documented for archaic and modern *Homo* through the Pleistocene.

injury | fracture | skull | Asia

The identification of traumatic lesions in human fossils is of interest for assessing the relative risk of injury to different human groups, the anatomical distribution of trauma and its behavioral implications, the potential to identify interhuman aggression, and the abilities of Pleistocene humans to survive serious injury and posttraumatic disabilities (1–5). In this context, we report on a healed lesion in the late archaic East Asian Maba 1 partial cranium. Although not included in the original description of the specimen (6), the lesion was mentioned by Wu and Poirier (7) but only as an exocranial "scar" and "an unexplainable bulging" endocranially at the same location. Further paleopathological diagnosis and analysis of the lesion are therefore warranted.

Maba Site

The Maba 1 cranium was discovered in June 1958, in a karst cave at Lion Rock (or Lion Hill; Shizishan) (24° 40′ 27.3″ N, 113° 34′ 49.8″ E), Maba town, Qujiang district, Shaoguan city, Guangdong province, China (Figs. S1 and S2). Lion Rock is composed of two isolated mountains connected through subsurface streams: Lion Head and Lion Tail Mountains. The Maba human remains were found in the cave of Lion Head Mountain.

The cave entrance faces west and is at the same elevation as the current ground surface, 84 m above sea level. The cave contained three strata, primarily consisting of yellow brown clays filling a complex of fissures and openings. Maba 1 and a diversity of mammalian fossils were found in the second level. These deposits extended as much as 10 m above the first level and were ∼8 m in length, ∼7 m in width, and ∼6 m in total height given the sloping level (Fig. S3). In the northern portion of the second stratigraphic level, there is a deep and narrow crevice (Fig. S3) measuring 40–50 m long and 0.6–1.5 m wide. The Maba cranium and associated faunal remains were unearthed from this crevice at a depth of 1 m by farmers removing cave sediments for fertilizer (8).

The associated faunal remains (Table S1), and especially the presence of *Crocuta crocuta ultima* and *Cuon javanicus*, indicate a later Middle or Late Pleistocene age for the Maba cranium (9–11). A uranium series date on associated vertebrate teeth yielded an age of 129,000–135,000 y before present (yBP) (12), but it is not clear whether this determination accurately dates the cranium. U-series dating techniques for teeth and bone at the time did not always adequately account for the open nature of uranium systems and often underestimated their ages (13). More recent $^{230}Th/^{234}U$ dating of capping flowstone samples from Southern Branch Cave, another chamber in Lion Head Mountain, suggests that some of the Maba deposits may be as old as 237,000 yBP (14). There is, however, no firm correlation between those flowstones and the stratigraphic position of the Maba cranium. Even so, these two sets of dates and the faunal evidence suggest that Maba 1 dates to the later Middle Pleistocene [marine isotope stages (MIS) 7–6] or possibly the early Late Pleistocene (MIS 5e).

Maba Human Remains

When found, the Maba specimen was in several pieces and was reassembled with bridging portions filled in (6) (Fig. 1). The fossil is highly mineralized and is colored yellow-brown with some regions of black staining. It consists of ∼40% of the frontal bone including the right orbitonasal portion, 90% of the nasal bones, the greater part of the parietal bones (∼75% of the left and ∼70% of the right), four fragments of the right temporal squama, the right greater wing of the sphenoid, the right frontal process of maxilla, and the frontosphenoidal process of the right zygomatic bone (Fig. 1 and Fig. S4).

Most of the damage to the preserved portions involves clean fossilization breaks. There is some erosion of the right lateral external surface, and there are damaged areas more posteriorly on the right and left parietal bones (Fig. S5). Across the supraorbital region there are parallel grooves from gnawing by a relatively large rodent. This damage is probably from porcupines (*Hystrix*) given their tendency to collect and gnaw bones in caves (15, 16) and their presence in the faunal assemblage. The gnaw marks are on the anterolateral process of the zygomatic bone, the supraorbital torus, the right inferior edge of the right nasal bone, and the anterior portion of the frontal bone surrounding the glabellar region (Fig. S6). The gnawing removed most of the left supraorbital torus and exposed the frontal sinus over both orbits.

Despite the loss of most of the facial skeleton, the cranial base, and the occipital region, plus the supraorbital rodent gnawing and some surficial erosion, the bone of the Maba 1 cranium is in excellent condition with little erosion of the endocranial surface and only modest alteration of the subpericranial surface (Figs. S4 and S5).

Endocranially, the coronal and sagittal sutures are completely obliterated around the area of bregma; exocranially portions of the two sutures are visible but fused. Despite substantial

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[1]To whom correspondence may be addressed. E-mail: trinkaus@wustl.edu or wuxiujie@ivpp.ac.cn.

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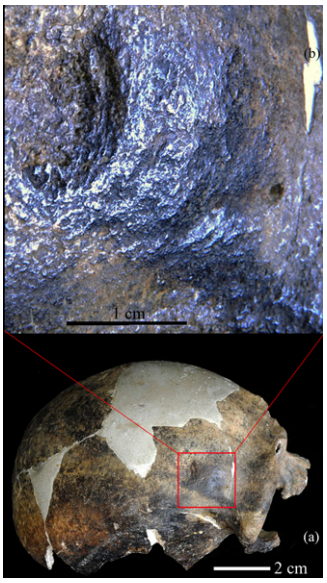

Fig. 1. Right superolateral view of the Maba cranium showing the position (*A*) and detail (*B*) of the depressed lesion.

Fig. 2. Detail of the Maba 1 frontal squamous lesion showing the concentric ridges that developed within the lesion.

variation in suture closure ages (17, 18), this fusion should indicate an age in the fourth to sixth decade postnatal. The older adult inferred age is supported by the relative thinness of the internal table and the expansion of fine diploic trabeculae (19, 20).

Morphologically Maba 1 has a thick and prominent supraorbital torus that arches over the circular orbits. The nasal bones are narrow, pinched, and strongly projecting. Because it is a rare cranial specimen of a late archaic human from mainland eastern Asia, Maba 1 has been assessed extensively from a comparative morphological perspective (e.g., refs 7 and 21–23). It was initially thought to show strong similarities to the Neandertals (6), and it does show the *forme en bombe* rounded neurocranial profile in *norma occipitalis* of the Neandertals (24). Little else aligns it with the Neandertals to the exclusion of other late archaic humans. More recent authors have stressed the forward positioning of the infraorbital surface, as inferred from the zygomatic and nasal bones, and related the specimen to the older East Asian Middle Pleistocene Dali cranium (e.g., refs. 7, 21, and 23). Maba 1 is therefore generally considered to be an East Asian late archaic human, part of the regional sequence of such humans in the later Middle and early Late Pleistocene.

Maba 1 Frontal Bone Lesion

On the right external table of the frontal bone, a semicircular, or lunate, lesion (Fig. 1*A*) is located 31.5 mm posterior to the lateral supratoral sulcus, 36.5 mm from the coronal suture, and 15.5 mm above the right temporal line. The area of the depressed portion of the vault lesion is ~308 mm^2, measuring ~14.0 mm in length and ~1.5 mm below the frontal external contour at its deepest point. The front of the lesion forms an irregularity of the external table, evident in its darker color. The center of the depression is rough. Several concentric waves within it created rounded edges, none of which is a complete circle (Fig. 1*B*). When the lesion is enlarged (Fig. 2), healing of the bone can be seen to have taken place surrounding the depressed area.

Depending on the position and orientation of the computerized tomography (CT) slice (Fig. 3), the external depression appears as either relatively flat or a distinct depression. More importantly, the CT slices show a slight thickening of the external table and of the adjacent diploic trabeculae at the point of the depression. The external table is relatively thick overall along the parasagittal contour through the lesion (Fig. 3*A*), and the resultant additional thickening is therefore modest. The depression has nonetheless changed the contour of the border between the external table and the underlying diploë.

Endocranially, as noted (7), there is an inward bulge of the internal table immediately underlying the exocranial depression. The endocranial surface of the bulge is smooth, rising from the normal surrounding surface. In cross-section, the bulge is evident as a distinct endocranial protrusion (Fig. 3 *B* and *C*), and the already-thin internal table exhibits further thinning at the point of maximum projection.

Although there is extensive exocranial remodeling of the bone in the depressed area, along the raised anterior margin and extending anteriorly from it, it is not clear whether the lesion was completely healed. Fine, slightly zig-zag, irregular cracks can be seen exocranially radiating from the depressed area, with at least one clear crack in each anatomical direction. These cracks have the appearance of radiating cracks from a traumatic impact to the human cranial vault (25); if so derived, it is unclear how they persisted while the depression, diploic space, and tables underwent extensive bone remodeling. Cracks are evident around the more medial postmortem damage to the posterior left parietal bone (Fig. S5), so it is possible that these cracks radiating from the lesion are from fossilization and resemble those of a traumatic impact given the changed topography and potentially compromised strength of the frontal bone around the lesion.

The area of the lesion does not show any evidence of intracranial or diploic infection, such as osteomyelitis. The roughened area anterior to the depression and ridge may represent new bone laid down in response to periostosis at the time of the insult, but, if so, it was extensively remodeled before death.

Differential Diagnosis

A variety of abnormalities can produce lesions of the cranial vault bones, including anemias, tumors, treponemal and mycobacterial infections, burning, and trauma (26).

Porotic hyperostosis of the cranial vault, from diploic hematopoietic marrow hypertrophy resulting in subpericranial exposure of the diploic trabeculae, is principally associated with megaloblastic anemia from dietary deficiencies or with congenital anemias, primarily in children (27). Maba 1 shows only normal, age-related diploic expansion and no penetration of the trabeculae into the external table.

Tumors can affect the external cranial vault bones, but they are rare and normally associated with diploic and endocranial

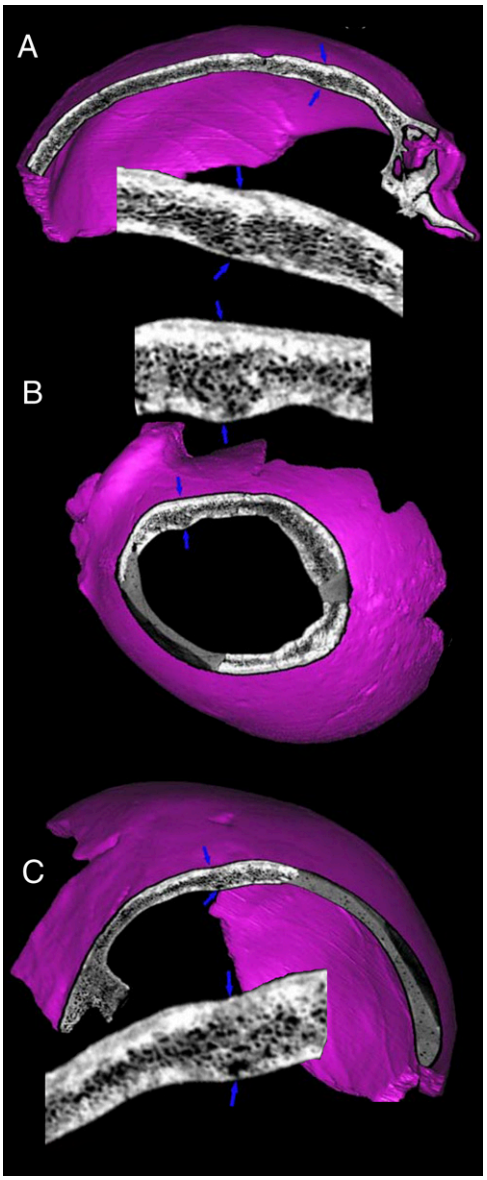

Fig. 3. CT reconstructions and cross-sections in the area of the lateral frontal lesion. (*A*–*C*) Parasagittal view (*A*), superolateral view (*B*), and coronal view (*C*). Each view is oriented such that the external table of the lesion is above. The coronal view is looking posteriorly, such that the right side of the cranium is on the left.

alterations. Osteoblastomas occur on the endocranial or exocranial surfaces, and the area of involvement is usually small (28, 29). Meningiomas arise from the dura mater, are intracranial in origin, and primarily affect the internal table by pressure atrophy or by direct invasion and erosion. Rarely a meningioma may become intradiploic, extend onto the external surface, and displace the external table outward (30–32). Hemangiomas are benign, solitary tumors formed by proliferating blood vessels (33) that are rarely found in the cranium (34, 35); the lesion originates in the diploë and can erode through either table. Metastatic neoplasms produce round vault perforations with dentate margins (36). None of these tumors produces the kinds of depressed lesion with endocranial bulging seen on the Maba 1 frontal.

Tuberculosis (mycobacterial) and syphilis (treponemal) infections can produce external frontal depressions (36, 37). The former arise from lytic processes and leave areas of bone destruction. The latter lesions are destructive (caries sicca) of the diploë and external table and frequently produce surficial sequestra.

Burning of the external cranium can produce extensive or localized lesions, as a result of destruction of hair, scalp, pericranium, and even the external table (38, 39). If severe, burning produces necrosis of the external bone, which heals through deposition of new bone and resorption of necrotic bone. It is unlikely to result in a normal cross-sectional distribution of the tables and diploë.

Finally, lesions such as the one on the Maba 1 frontal bone could result from a localized traumatic impact, one sufficient to impact the external table, produce the concentric ridges of raised bone, and create a depression of the exocranial bone that extended through the diploë and tables to generate an internal bulge (40). The surficial and cross-sectional appearance of the lesion suggests compressive stress on the external table resulting in the depression and tensile stress internally producing the bulge, with secondary remodeling of the external table. The concentric rings, and possibly the radiating cracks (if they are indeed antemortem, see above), suggest a localized blunt force trauma (40–42). It also resembles the size and form of cranial vault injuries from small and hard missiles (43). More generalized cranial trauma, from falls or head impacts on large hard objects, tend to produce large comminuted or linear fractures and/or compression of the cranial base (40, 44, 45).

Discussion

The lesion on the right frontal squamous of Maba 1 therefore appears most likely to have been the result of a localized, blunt force trauma, sufficiently strong to produce the concentric ridges, the external depression, and the internal bulge. At the same time, the bone was extensively remodeled, with rounding off of the external new bone and maintenance or reestablishment of the normal proportions between the tables and the diploë. Such remodeling minimally takes several months to develop (46) and could involve substantially longer periods (45), indicating the nonlethal nature of the trauma.

Cranial (and postcranial) lesions attributable to trauma are relatively common among Middle and Late Pleistocene humans, both archaic and modern (here restricted to those before the last glacial maximum) (Table S2). Some of them involve relatively minor to moderate changes that impacted the external table and, to variable extents, the underlying diploë [e.g., Atapuerca-SH crania 1–8; Biache 1; Casal de' Pazzi 1; Ceprano 1; La Chaise BD-17; Dolní Věstonice 3, 13, and 16; Ehringsdorf 2; Krapina 4 and 20; Mladeč 5; Pavlov 1; Shanidar 1 and 5; Swanscombe 1; Zhoukoudian skulls X and XII; Zuttiyeh 1; and possibly Ngandong 7 and Sangiran 38 (see Table S2 for details and references)]. Additional cases of more severe trauma involve the extensive remodeling of the external table of Cova Negra 1 and Hulu/Nanjing 1 (47, 48), the serious fracture of the external table with resorption of Krapina 34.7 (49), the slicing wound to Saint-Césaire 1 (3), and depressed fractures of the vault with thinning of the bone and endocranial protrusion on Dolní Věstonice 11/12 and Qafzeh 11 (50, 51) (Fig. S7). There are also several cases of healed facial injuries (Atapuerca-SH cranium 1, Dolní Věstonice 3, and Shanidar 1) and minor supraorbital trauma (Atapuerca-SH 764, Biache 2, Ceprano 1, Feldhofer 1, Šal'a 1, and Zuttiyeh 1) (Table S2); the injuries to at least Dolní Věstonice 3 and Shanidar 1 resulted in some loss of function (50, 52). Not included here are alterations of the cranium that cannot be distinguished from postmortem taphonomic changes (e.g., Gongwangling/Lantian 1, Saldanha 1, and several of the Zhoukoudian Locality 1 crania) (53–56).

Of these injuries, the ones that most closely parallel the Maba 1 lesion are those of Dolní Věstonice 11/12 and Qafzeh 11 (Fig. S7). Both of these individuals sustained impacts (on the midline frontal and the right lateral squamous, respectively) with exocranial depressions, endocranial bulges, and in Dolní Věstonice

11/12 a raised margin to the lesion. Unlike Maba 1 they had marked thinning of the bone, sufficient for the diploë to be absent in the central portions of their lesions. The lesion on Maba 1 therefore follows a pattern evident among other Middle and Late Pleistocene humans.

There are a variety of behavioral scenarios that can produce such trauma, the most commonly invoked one being interpersonal violence (3, 4, 57, 58). However, the only current case of a fossil human cranium, with traumatic injury unlikely to have been caused by any other mechanism than human technology and violent behavior, is the slicing injury to the Saint-Césaire 1 cranium (3). To this instance can be added the piercing rib injury of Shanidar 3 (52) and the upper thoracic fatal wound of Sunghir 1 (5).

It is possible that the Maba 1 frontal lesion, as well as those of Dolní Věstonice 11/12, Qafzeh 11, and other Pleistocene specimens, was the result of interhuman violence. If so, it is principally the evidence for a localized strong impact in Maba 1 that argues for a human agent rather than the generally dangerous circumstances of being a Pleistocene forager in concert with an abundance of large carnivores, technologies that required close encounters with substantial prey, and the use of diverse weaponry. It is not possible to assess whether the incident was accidental, intentional but the result of a short-term disagreement, or premeditated aggression (see discussion in ref. 5).

Cranial injuries from recent human interpersonal altercations are primarily facial (40) and otherwise mostly occur above the "hat brim line" (59, 60). Given the predominance of right-handedness, they are mostly on the left side (59, 61). The depressed injury of Maba 1, plus those of Dolní Věstonice 11/12, Qafzeh 11, and Shanidar 1 (and possibly Krapina 34.7 and Cova Negra 1), therefore generally conforms to the pattern of lesions from interindividual aggression among recent humans. However, most of them are on the right side, despite the predominance of right-handedness in Pleistocene *Homo* (62–64).

Pooling the major and minor sided cranial traumatic lesions (Table S2), 50% are on the right ($n = 56$). This right side frequency is significantly above what would be expected from an average of 12.7% recent human left-handedness ($P < 0.0001$), as well as the highest recent human percentage of left-handedness (27.5%; $P = 0.0002$), for throwing and hammering (48 samples) (65). Considering only lesions on the anterior half of the skull ($n = 41$) minimally changes the distribution (49% right) or the degree of difference ($P < 0.0001$ and $P = 0.0019$, respectively). Maba 1 and the other Pleistocene *Homo* injuries therefore do not fully follow the distribution of cranial injuries expected from violent interhuman altercations.

At the same time, the Maba 1 lesion, as with the other identified Pleistocene cranial lesions, was healed long before death. Whatever the ultimate cause of the lesion, it provides further evidence of long-term Pleistocene human survival of potentially debilitating conditions. In particular, closed-injury depressed cranial vault fractures are commonly, but not necessarily, associated with varying degrees of posttraumatic amnesia from trauma at the impact site and/or from contralateral cerebral injury (66, 67). In addition to severe craniofacial trauma, there are a number of Pleistocene human remains that provide evidence of long-term survival of serious developmental abnormalities, appendicular trauma with loss of function, or pronounced degenerative processes (see Table S2 for details and references). Although serious abnormalities have been documented for recent nonhuman primates (68) and at least one other Pleistocene mammal (*Ursus spelaeus*) (69), the growing focus on Pleistocene *Homo* lesions as paleobiological evidence indicates that such abnormalities and their survival were relatively common.

Conclusion

The antemortem lesion on the right frontal squamous of the late Middle Pleistocene Maba 1 cranium indicates that it was mostly likely caused by a localized impact, one sufficiently strong to produce both exocranial depression and remodeling and an endocranial protrusion. It is probable that it was the result of an interpersonal altercation, with blunt-force trauma, given its form, but accidental injury cannot be excluded. It may be the oldest such case known, depending upon one's behavioral interpretation of the earlier Zhoukoudian Locality 1 and Hulu/Nanjing lesions. Healed with only a suggestion of posttraumatic infection, the Maba 1 lesion joins a series of other craniofacial traumatic lesions of Pleistocene humans that provide evidence of apparently elevated levels of risk to injury and the ability to survive both major and minor conditions.

Materials and Methods

The Maba 1 cranium, in the Institute of Vertebrate Paleontology and Paleoanthropology, Chinese Academy of Sciences, was analyzed visually and using stereomicroscopy and CT. It was scanned in coronal orientation using a high-resolution industrial CT scanner (type 450kV-ICT) at the Institute of Vertebrate Paleontology and Paleoanthropology. The CT scan parameters were X-ray tube voltage 430 kV, X-ray tube current 9 mA, and 0.3 mm slice thickness. Two hundred ninety slices were obtained. The primary scanned slice data were processed with 2D reconstruction software made by the Institute of High Energy Physics, Chinese Academy of Sciences. The complete set of slice pixel matrix is 1,024 × 1,024, and color depth is 8 bits. The reconstruction diameter of each slice is 283 mm, and each pixel size is 0.276367 mm × 0.276367 mm. On a Dell Graphics Workstation, the 3D reconstructions were created by postprocessing the CT data and running Mimics 13.0 (Materialise NV) to extract the maximum information concerning internal cranial features, osseous distribution, and the lesion. Right- and left-side distributions of lesions were assessed using exact binomial tests in Stat-Xact 4.0.1 (70).

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