

# Pathological Alterations in the Archaic *Homo sapiens* Cranium From Eliye Springs, Kenya

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**ABSTRACT** This paper reports on the results of a first computerized tomography (CT)-based study of the Middle Pleistocene matrix-filled skull KNM-ES 11693 from Eliye Springs at Lake Turkana. Ectocranially, the hominid cranium exhibits a remarkable enlargement of the vault symmetrical to the sagittal suture and a porotic surface covering most of the vault. CT analysis further revealed a

strong thickening of the cranial vault as well as other relevant aspects. Differential diagnosis suggests that the changes of the Eliye Springs cranium were probably caused by chronic anemia in the childhood or youth of this individual. *Am J Phys Anthropol* 120:200–204, 2003.

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The nearly completely preserved fossil cranium KNM-ES 11693 (Fig. 1) was recovered from beach deposits at the western shore of Lake Turkana in 1982. Its heavy degree of mineralization, which is also shown by some faunal remains from the immediate vicinity, strongly indicates that the specimen is derived from later deposits of the Koobi Fora Formation which underlie the Holocene Galana Boi Beds (Bräuer and Leakey, 1986). Based on comparative analyses, the Eliye Springs cranium has been classified as a representative of late archaic *Homo sapiens* (Bräuer and Leakey, 1986; Bräuer, 1989). Recent revision of the origin of anatomically modern humans in Africa suggests that modern cranial morphology dates back to about 150,000 years BP, and near-modern or late archaic humans appeared at Lake Turkana and in other parts of the continent as early as about 300,000 years ago (Bräuer et al., 1997; Bräuer, 2001; McDermott et al., 1996). Thus, based on the morphological similarities to absolutely dated archaic *Homo sapiens* specimens, an age of about 200,000–300,000 years can be assumed for the Eliye Springs hominid. Besides its clear archaic features, the KNM-ES 11693 cranium, most likely of a robust male individual, also exhibits a peculiar large symmetrical enlargement of the frontal and parietal bones which differs in size and shape from sagittal keelings and prominences in other archaic *Homo sapiens* specimens like Kabwe (Zambia), Salé (Morocco), and Omo Kibish 2 (Bräuer and Mbua, 1992). Only in the context of a computerized tomography (CT)-based study of the Eliye Springs speci-

men did it become evident that the cranium has been affected by severe pathological alterations.

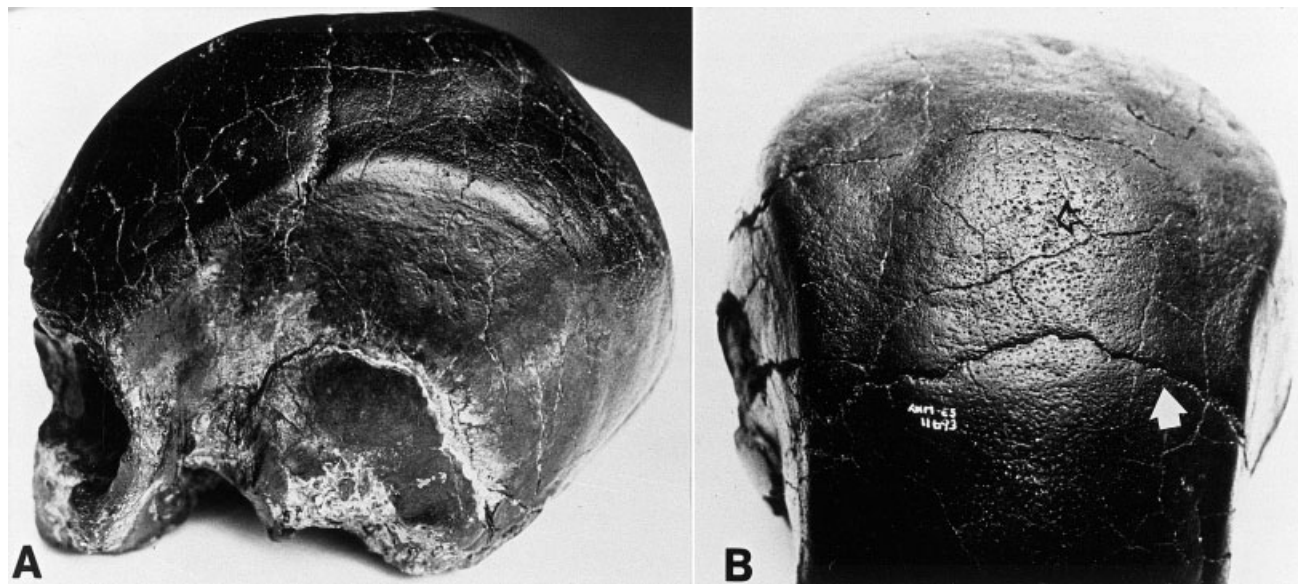
## ECTOCRANIAL ALTERATIONS

The external surface of the cranial vault shows a remarkable, irregular coarse-porotic morphology which symmetrically covers the frontal and both parietal bones as well as the superior part of the occipital squama between the temporal lines and the attachments of the occipitofrontalis muscles (Fig. 1). Behind the coronal suture, the porotic area has its widest extension. This is especially evident in the wide, symmetrical enlargement of the vault adjacent to the sagittal suture, which moreover has completely fused, whereas the coronal and lambdoidal sutures do not show any signs of ectocranial obliteration. On the frontal, the bulging begins above the supraglabellar flattening and broadens towards the coronal suture. According to these features, the thickening was caused by an extensive growth of the diploë, which enlarged toward the outer lamina. The irregular appearance of the external morphol-

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**Fig. 1.** **A:** Cranium KNM-ES 11693 shows strong enlargement on vault. **B:** Porosity on vault; black open arrow indicates fused sagittal suture and white arrow points to open coronal suture.

ogy of the protruding areas of the parietals also partially resembles remodeled (i.e., scarred bone) surfaces. A fine porosity is found on the greater wings of the sphenoid bones and the temporal squamae. The external surface of the vault is in such excellent condition that none of the above-mentioned alterations could be due to effects of geochemical changes (e.g., diagenesis).

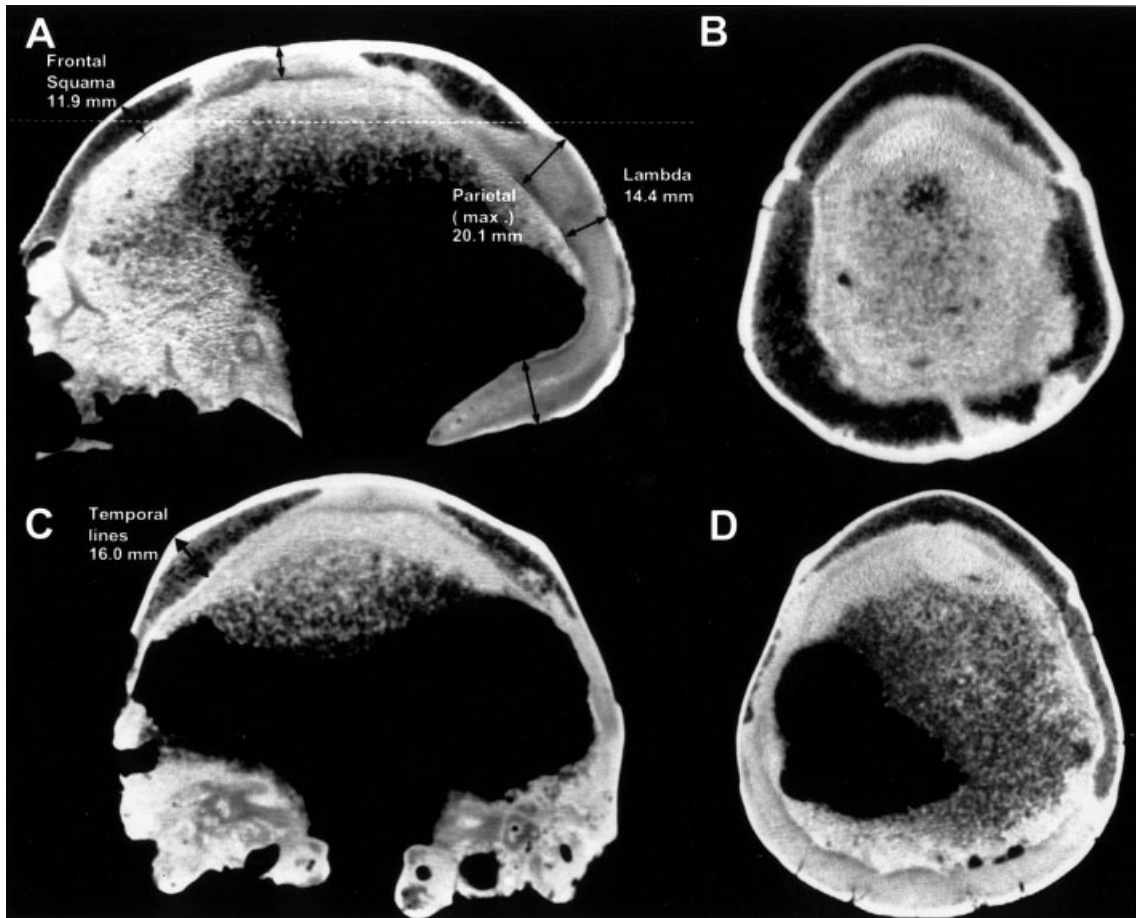
### CT ANALYSIS

The CT scanning of the complete matrix-filled cranium was carried out by Fred Spoor at the Diagnostic Centre in Nairobi. Two hundred and one coronal CT scans were made with a Siemens Somatom AR.SP scanner with an exposure of 249 mAs at 130-kV tube voltage and a slice thickness of 1 mm. Image computing was done using an SP 90 kernel and the extended Hounsfield scale (CT number scale). By linear interpolation, isotropic voxels were reconstructed with a size of 0.41 mm. Therefore, we could obtain slices in any direction. Due to the pixel size of  $0.41 \times 0.41$  mm and slice thickness of 1 mm, a maximum error of 1.1 mm ( $\sqrt{0.41^2 + 0.41^2 + 1^2}$ ) can be assumed. The results of CT revealed a moderate to strong thickening in the midsagittal plane, reaching up to 20 mm at the posterior part of the parietals (Fig. 2A). The widening of the cranial wall, however, is not restricted to the enlargement but also extensive in the adjoining areas, as can be seen on a coronal scan about 2 cm behind the coronal suture (Fig. 2C). An internal growth of the internal lamina leading to a reduction of the endocranial space is not visible on the CT scans. The thicknesses at the midfrontal squama and at lambda measure 11.9 mm and 14.4 mm, respectively. The inferior part of the occipital squama is not as thick as the midsagittal CT scan (Fig. 2A) seems to suggest, be-

cause the internal occipital crest is cut here. This is supported by parasagittal scans showing a normal thickness. Thus, the thickening of the skull is indeed limited to the vault. The border between the original endocranial surface of the internal lamina and the sandstone matrix cannot always be clearly seen in the CT scans. In addition to a diploic widening, the external lamina is also relatively thick especially along the parietals, with a value of approximately 2 mm measured at about 1 cm behind bregma (the total thickness at this point measures ca. 11.3 mm); there is no circumscribed rarefaction. The internal lamina exhibits a thickness of about 1 mm, as determined at several points. The seemingly partial solidification of the bone marrow spaces (Fig. 2A,D) is most likely not due to disease but to the fossilization process. The high density (mean, 2,100 Hounsfield units (H.U.)) of these homogeneously structured areas at the fronto-parietal transition, the posterior parietal region, and the occipital is nearly identical to the density of the matrix. In contrast, the original diploë, which is reliably observable in the left parasagittal region, gave values of around 1,300 H.U., and the external lamina of 2,900 H.U. Thus, it appears quite likely that at these homogeneous regions, the diploë is nearly completely saturated by sediment matrix. The bone trabeculae of the original diploë form the relatively enlarged, irregular modules of the red bone marrow (Fig. 2).

### DISCUSSION

In view of the various pathological alterations shown by the Eliye Springs cranium, it appears very likely that the extreme thickening is due to disease. Nevertheless, it has to be considered that the vault bones in archaic *Homo sapiens* and *Homo erectus*



**Fig. 2.** **A, C:** Midsagittal and coronal CT scans show thickening of KMN-ES 11693 vault. Diploic layer of posterior parietals and occipital is completely saturated by matrix. Thickness at various points is displayed, also showing that widening is not restricted to fronto-parietal enlargement. **B:** Transversal CT scan (level as indicated in A by dotted line) reveals a homogeneous thickening of diploic space. In left part of parietals, diploic layer displays incomplete saturation by matrix. **D:** Transversal scan taken above frontal sinus and parallel to orbito-meatal plane.

**TABLE 1.** Thickness of cranial vault (mm) in Eliye Springs compared to other fossil hominids<sup>1</sup>

Hominids	Bregma	Lambda
Eliye Springs	10.1	14.4
African <i>Homo erectus</i> (n = 4) <sup>2</sup>	8.0–9.5	8.0–11.0
African archaic <i>Homo sapiens</i> (n = 3) <sup>3</sup>	8.0–12.0	7.0–8.5
Chinese <i>Homo erectus</i> (n = 6) <sup>4</sup>	7.0–9.7	11.0
Chinese archaic <i>Homo sapiens</i> (n = 3) <sup>5</sup>	5.5–8.0	5.5–9.8
Indonesian <i>Homo erectus</i> (n = 6) <sup>6</sup>	7.5–10.0	9.5
Ngandong/Solo (n = 6) <sup>7</sup>	7.5–10.0	6.5–10.5

<sup>1</sup> Bräuer, personal observation\*; Clarke, 1985; Kennedy, 1991; Weidenreich, 1943; Wu and Poirier, 1995.

<sup>2</sup> KNM-ER 3883\*, ER 730\*, ER 1821\*, OH 12\*.

<sup>3</sup> Ndutu\*, Kabwe, Florisbad.

<sup>4</sup> Zhoukoudian 2, 3, 10, 11, 12, 1966\*.

<sup>5</sup> Jinniushan, Maba\*, Xujiayao\*.

<sup>6</sup> Trinil\*, Sangiran 2\*, 4\*, 10\*, 12\*, 17\*.

<sup>7</sup> Solo 1\*, 3\*, 5\*, 8\*, 9\*, 11\*.

crania were generally thicker than in recent humans. Table 1 (containing specimens which are not regarded as pathological) indicates that Eliye Springs wall thickness at lambda strongly exceeds that of all comparative specimens, which is also supported by the fact that the thickness of the ad-

joining posterior part of the parietals even measures ca. 20 mm.

A thickened cranial vault could have various causes, as described in pathological and paleopathological literature (Steinbock, 1976; Ortner and Putschar, 1981; Aufderheide and Rodriguez-Martin, 1998; Schultz, 1999, 2001). The external morphology of the vault and the results of CT characterize a process which might be responsible for the lesions observed in this skull. A bundle of healed or chronic diseases such as osteomyelitis, leukemia, rickets, and hypervitaminosis A can be excluded. A healed inflammation of the scalp is also unlikely, since the extent of thickening of the vault bones does not fit this diagnosis (Schultz, 1993a). Internal frontal hyperostosis (Morgagni-Stewart-Morel syndrome) is, in fact, characterized by an internal thickening and nodular hyperostosis which is only localized at the frontal and the anterior part of the parietals (Salmi et al., 1962; Antón, 1997). Thus, this diagnosis as well can be neglected.

Three more probable diagnoses remain. The first is otitis deformans or Paget's disease. However, the external morphology and the regular trabecular

structure of the vault bones do not agree with the characteristic features of this disease (Norman, 1991; Kanis, 1998; Resnick and Niwayama, 1981). The CT scans do not show the irregular and often asymmetric cotton-wool appearance of circumscribed parts or the whole skull typical of Paget's disease. Moreover, Paget's disease generally occurs in humans older than 40 years, whereas the Eliye Springs individual had probably died at an age between 25–40, as indicated by the closure of the basi-sphenoid synchondrosis and the open coronal and lambdoidal sutures.

A second possible diagnosis is a deficient parathyroid activity which, however, has only very rarely been diagnosed on the cranium (Ziegler, 1991). This disease is also associated with a thickening of the calvaria and increased bone density. However, the characteristic structures of patchy or circumscribed osteosclerosis (Delling et al., 1987) are not present in the Eliye Springs cranium. The dense areas of the diploic layer result from saturation by matrix (see above). Also, the bulging of the external surface of the cranial vault is not a feature which has been described as associated with parathyroid disorders (pseudohypoparathyroidism).

Third, it is possible that the changes observed in the Eliye Springs skull were caused by severe chronic anemia in the childhood or youth of this individual. In historic and prehistoric times, similar changes due to chronic anemia were relatively frequent (Steinbock, 1976; Schultz, 1986; 2001; Schultz et al., 2001; Stuart-Macadam, 1992). Also for the final Middle Pleistocene skull from Singa, Sudan, and the late Pleistocene cranium WLH 50, Australia, such diagnoses have been favored (Spoor et al., 1998; Webb, 1990). The nature and location of the porotic changes macroscopically observed on the external surface of the vault of the Eliye Springs skull are characteristic of structures found in cases of healed chronic anemia (e.g., Aufderheide and Rodriguez-Martin, 1998; Ortner and Putschar, 1981; Steinbock, 1976). Using microscopic techniques, a reliable diagnosis is relatively easy to obtain (e.g., Schultz, 1993a,b; 2001; 2002). However, in the case of the Eliye Springs skull, it was not possible to take samples for microscopic research. In nonfossilized skulls, the bone architecture of a thickened skull vault can be radiologically or microscopically studied. In these cases, the most characteristic features are the complete disintegration of the external lamina and the radial arrangement of the bone trabeculae in the external half of the diploë in the significantly thickened areas of the skull vault, which produce by their parallel orientation the typical "hair-on-end phenomenon" (El-Najjar et al., 1976; Steinbock, 1976; Stuart-Macadam, 1992; Schultz, 2001). Because of the postmortem fill of the modules of the red bone marrow of the skull vault (i.e., diploë) by the products of the fossilization process (e.g., crystals), CT scans could not prove structures characteristic of chronic anemia, such as the well-known

"hair-on-end phenomenon." In the Singa skull it was also not possible to determine the hair-on-end appearance (Spoor et al., 1998). In chronic anemia, the typical changes are, as a rule, expressed by a pronounced thickening and a coarsely porotic surface of the affected skull region, both of which are found in the areas of the parietal and frontal eminences, the lambdoid areas of the parietals, and the upper part of the occipital squama (e.g., Ortner and Putschar, 1981; Schultz, 2001; Schultz et al., 2001; Steinbock, 1976). Thus, the changes observed in the Eliye Springs skull agree with this description. The lack of a thinning of the external lamina could be due to the healing process, which produces by remodeling of the porotic external surface a secondary, relatively thick external lamina (Schultz, 2001), particularly, in Asian and African archaic *Homo sapiens* and *Homo erectus*, in which there is great variation in the thickness of this lamina (Bräuer, personal observation). A relatively thick external lamina occurs, e.g., in the archaic occipital specimen Eyasi 4 from Tanzania (Bräuer and Mabulla, 1996). In case of iron-deficiency anemia, the lack of a thinning of the lamina could have also resulted from the adult individual overcoming the disease due to reduced iron demands in adolescence.

If we accept the diagnosis of anemia, the enormous thickening of some regions of the vault bones makes it probable that this skull represents a case of chronic anemia (cf. Schultz, 2001; Schultz et al., 2001). In a florid process of porotic hyperostosis of the skull vault caused by anemia, within the porotic structure, there can be a few irregular, short clefts in the center of the hypertrophic regions, which sometimes give the external surface of the vault a star-like appearance. When the external surface is only porotic and relatively smooth, this can be interpreted as the result of a healing process (Resnick and Niwayama, 1981; Schultz, 2001). As a rule, such healing which can run over several years, and is characterized by an extended bone remodeling process which could change the external vault morphology as seen in the Eliye Springs skull. Thus, this individual probably suffered from anemia during its subadult years. Furthermore, in the skulls of subadults, the macroscopic alterations characteristic of such an advanced stage of porotic hyperostosis caused by anemia are very similar to changes due to scurvy or occasionally hemangioma (e.g., Ortner and Putschar, 1981; Schultz, 2001). As the remodeled vestiges of these diseases look, as a rule, macroscopically different (Schultz, 2001, 2002), and the individual from Eliye Springs died in adulthood, these diagnoses can be neglected. Also, healed inflammatory processes of the scalp and skull bones (e.g., periostitis, osteomyelitis) are not very probable, because of the morphology of the external surface of the skull bones and the distribution of changes of the vault. In the case of the Eliye Springs skull, the nature of the anemia cannot reliably be determined. However, most probable are causes such as nutri-

tional stress (e.g., deficiency diseases: iron, proteins; cf. El-Najjar et al., 1976; Schultz, 1982; Stuart-Macadam, 1992) or parasites (e.g., helminthic diseases, cf. Reinhard, 1992; malaria, cf. Schultz 1990). Also, genetic causes such as sickle-cell anemia (Faerman et al., 2000) cannot be excluded. Thus, up to now the Eliye Springs hominid probably represents the oldest discovered case of anemia.

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