Small anatomical variant has profound implications for evolution of human birth and brain development

Robert G. Tague[1]
Department of Geography and Anthropology, Louisiana State University, Baton Rouge, LA 70803

Humans differ from primates and other mammals in a number of anatomies, including having big brains and big babies. The evolutionary origin and adaptive significance of a big brain and the consequent difficulty of giving birth to a big baby have long been discussed. Falk et al. (1), in PNAS, report on a small anatomical difference of the cranium between humans and chimpanzees, and document the presence of this trait in early human ancestors. This trait may have profound importance for understanding evolution of birth and brain development. Falk et al. (1) report on the metopic suture (MS) in modern humans, two species of chimpanzees, and our hominid ancestors, *Australopithecus* and earlier *Homo*. MS is the joint between the two frontal bones of the cranium (Fig. 1). In the human fetus and young child, the confluence of MS with the coronal and sagittal sutures is the anterior fontanelle. This is the "soft spot" in a baby's cranium. Fusion of MS occurs in infancy (2–4), whereas the anterior fontanelle closes by 2 y of age (5). Fusion of MS begins near the nose and progresses toward the anterior fontanelle. As the cranium forms around the developing brain, premature closure of MS causes atypical cranial development (6).

Based on large samples of humans and chimpanzees, Falk et al. (1) report that MS fuses at a later age in humans than chimpanzees, with age based on eruption of deciduous and permanent molars. In contrast with humans (as described earlier), Falk et al. (1) report that MS fuses shortly after birth in chimpanzees. Moreover, Falk et al. (1) show that, among adults, an unfused or partially fused MS is seen in a higher proportion in humans than chimpanzees. The authors infer that this difference in persistent MS between adult humans and chimpanzees is causally related to the difference between these species in modal age of MS fusion in infancy. The authors suggest three not mutually exclusive reasons for the adaptive significance of later age of fusion of MS in humans compared with chimpanzees: (*i*) obstetrical dilemma, (*ii*) high rate of early postnatal brain growth, and (*iii*) reorganization of the brain's frontal cortex.

Obstetrical dilemma refers to competing selection pressures on birth canal size in humans: reduction in birth canal size is advantageous for bipedalism but disadvantageous for birth. In this context, an unfused MS and anterior fontanelle facilitate parturition because the fetus' cranial bones can shift position (i.e., mold) under the forces of uterine contraction and resistant bony pelvis. Molding changes the shape of the fetal head and provides a better "fit" in the mother's pelvis. The second explanation is postnatal brain growth. Humans have rapid brain growth in infancy, with our brain doubling in weight. Martin (7) showed this contrast in brain growth between humans and rhesus macaques. In humans, the velocity of brain growth is virtually unchanged from fetal development through infancy, whereas, in the monkey brain, growth decelerates near the time of birth and remains at that lower level through infancy. Because sutures fuse when cranial bones are in prolonged contact with one another (8, 9), the exuberant brain growth in human infants keeps the frontal bones separated from one another, and, correspondingly, the MS and anterior fontanelle patent. The third explanation is a partly regionally specific restatement of the second explanation. That is, late closure of MS is a result of growth and reorganization of the brain's frontal cortex. Humans have a larger frontal cortex than apes, although the proportionate size of our frontal cortex is similar to that of apes (10). The explanations of obstetrical dilemma and rapid growth of the brain have been advanced by others; I believe Falk et al. (1) are the first to propose reorganization of the frontal cortex as an explanation. I have previously assumed that rapid brain growth in the neonate is the probable explanation for MS and anterior fontanelle, and that cranial molding is an obstetrically fortuitous derivative. The suggestion by Falk et al. (1) that late closure of MS and anterior fontanelle is a result of reorganization of the frontal cortex is provocative.

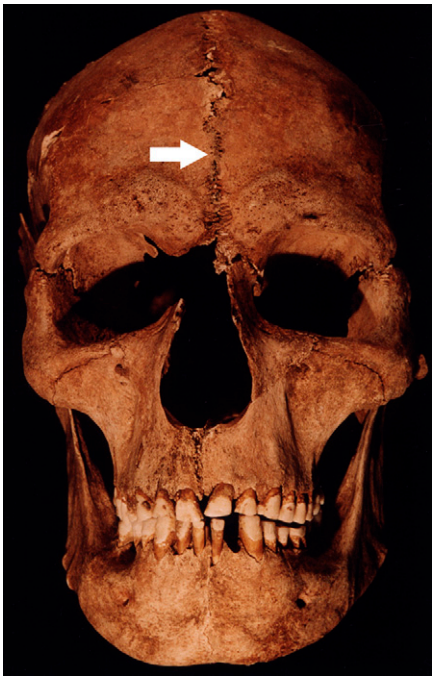

Fig. 1. Arrow indicates persistent MS in adult human (postmortem damage to nose and right orbit). Image courtesy of Dr. Clark Spencer Larsen.

The frontal cortex is involved in how humans are distinctly different from other mammals. A nonexhaustive list of traits associated with the frontal cortex includes language, memory, judgment, problem solving, socialization, and motor function. Interestingly, the white matter of our prefrontal cortex increases in volume faster than that of chimpanzees during infancy (11). Perhaps later fusion of MS in humans compared with chimpanzees is related not just to our larger brain or larger frontal cortex, but rather to our fast-developing prefrontal cortex.

The prefrontal cortex is related to abstract thinking, anticipation of outcomes from particular behaviors, motivation, and social behavior. Size may not be the principal distinguishing feature between humans and apes in the frontal and prefrontal cortices; rather, cytoarchitectonic differences between the species may be more important (10).

The comparison by Falk et al. (1) between humans and chimpanzees in age at fusion of MS and in prevalence of unfused and persistent MS in adults, with the attendant proximate and ultimate causes, is interesting enough, but that is not what distinguishes their report. The authors also document a persistent MS and remnant of anterior fontanelle in the famous Taung fossil specimen, which is an early hominid juvenile from South Africa. Taung is the

Author contributions: R.G.T. wrote the paper.
The author declares no conflict of interest.
See companion article on page 8467.
[1]E-mail: rtague@lsu.edu.

www.pnas.org/cgi/doi/10.1073/pnas.1205763109

first australopithecine discovered and described, and is the type specimen for *Australopithecus africanus* (12). Although MS in Taung was noted in 1925 (13), this anatomical feature was not further discussed or analyzed in the literature, despite a plethora of studies of Taung, until its rediscovery by Falk et al. (1). The authors also report persistent MS in other early hominids dated from between 3.0 and 1.5 Mya.

MS in smaller-brained *Australopithecus* (compared with *Homo*) could be an obstetrical adaptation. These early bipeds had a small pelvis, but also likely gave birth to small newborns; they would have had more obstetrical difficulty than apes, but less than humans (14, 15). However, the obstetrical dilemma argument is weakened by the observation that the squirrel monkey, *Saimiri sciureus*, has an even more confined birth canal relative to neonatal size than humans (15), but adults do not have a persistent MS (16).

If obstetrical dilemma is not the evolutionary reason for MS, brain growth or reorganization of frontal cortex remains the likely explanation. The endocranial volume (i.e., brain size) of adult *A. africanus* does exceed that of chimpanzees by approximately 22% [~458 mL vs. ~378 mL, respectively (17, 18)], but it is approximately 66% smaller than that of humans [~1,350 mL (19)]. Data are not available to evaluate growth velocity of the brain or frontal cortex in early hominid infants, but persistent MS in *Australopithecus* may imply a difference between them and apes in cognition. Of course, this implication is based on the untested assumption that persistent MS in juveniles and adults in early hominids and modern humans is associated with rate of brain growth in infancy or frontal cortex reorganization.

If obstetrical dilemma is not the evolutionary reason for MS, brain growth or reorganization of frontal cortex remains the likely explanation.

Falk et al. (1) provide a comprehensive analysis of an anatomical variant in humans and chimpanzees, document its presence in Taung and other hominid ancestors, and offer reasonable interpretations about its evolutionary significance. This study will engage the intellectual imagination of researchers, who will follow up with their studies to try to pinpoint which of the proffered explanations, or perhaps new explanation, is the most likely evolutionary cause for persistent MS. That Falk et al. (1) have written a manuscript that will likely lead to ramifying and multiplicative studies by others is a testament to its scientific noteworthiness.

ACKNOWLEDGMENTS. The author thanks Clark Larsen for providing the figure.

1. Falk D, Zollikofer CPE, Morimoto N, Ponce de León MS (2012) Metopic suture of Taung (*Australopithecus africanus*) and its implications for hominin brain evolution. *Proc Natl Acad Sci USA* 109:8467–8470.
2. Vu HL, Panchal J, Parker EE, Levine NS, Francel P (2001) The timing of physiologic closure of the metopic suture: A review of 159 patients using reconstructed 3D CT scans of the craniofacial region. *J Craniofac Surg* 12: 527–532.
3. Weinzweig J, et al. (2003) Metopic synostosis: Defining the temporal sequence of normal suture fusion and differentiating it from synostosis on the basis of computed tomography images. *Plast Reconstr Surg* 112: 1211–1218.
4. Sim SY, Yoon SH, Kim SY (2012) Quantitative analysis of developmental process of cranial suture in Korean infants. *J Korean Neurosurg Soc* 51:31–36.
5. Duc G, Largo RH (1986) Anterior fontanel: Size and closure in term and preterm infants. *Pediatrics* 78:904–908.
6. Aryan HE, et al. (2005) Surgical correction of metopic synostosis. *Childs Nerv Syst* 21:392–398.
7. Martin RD (1983) Human brain evolution in an ecological context. James Arthur lecture. *Am Mus Nat Hist* 52: 1–58.
8. Graham JM, Jr., Smith DW (1980) Metopic craniostenosis as a consequence of fetal head constraint: Two interesting experiments of nature. *Pediatrics* 65: 1000–1002.
9. Sanchez-Lara PA, et al.; National Birth Defects Prevention Study (2010) Fetal constraint as a potential risk factor for craniosynostosis. *Am J Med Genet A* 152A: 394–400.
10. Semendeferi K, Lu A, Schenker N, Damasio H (2002) Humans and great apes share a large frontal cortex. *Nat Neurosci* 5:272–276.
11. Sakai T, et al. (2011) Differential prefrontal white matter development in chimpanzees and humans. *Curr Biol* 21:1397–1402.
12. Dart RA (1925) *Australopithecus africanus*: The man-ape of South Africa. *Nature* 115:195–199.
13. Hrdlička A (1925) The Taungs ape. *Am J Phys Anthropol* 8:379–392.
14. Tague RG (1991) Commonalities in dimorphism and variability in the anthropoid pelvis, with implications for the fossil record. *J Hum Evol* 21:153–176.
15. Tague RG, Lovejoy CO (1998) AL 288-1—Lucy or Lucifer: Gender confusion in the Pliocene. *J Hum Evol* 35:75–94.
16. Dolan KJ (1971) Cranial suture closure in two species of South American monkeys. *Am J Phys Anthropol* 35: 109–117.
17. Neubauer S, Gunz P, Schwarz U, Hublin J-J, Boesch C (2012) Brief communication: Endocranial volumes in an ontogenetic sample of chimpanzees from the Taï Forest National Park, Ivory Coast. *Am J Phys Anthropol* 147:319–325.
18. Neubauer S, Gunz P, Weber GW, Hublin J-J (2012) Endocranial volume of *Australopithecus africanus*: New CT-based estimates and the effects of missing data and small sample size. *J Hum Evol* 62:498–510.
19. McHenry HM, Coffing K (2000) *Australopithecus* to *Homo*: Transformations in body and mind. *Annu Rev Anthropol* 29:125–146.