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LB1's virtual endocast, microcephaly, and hominin brain evolution

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ABSTRACT

Earlier observations of the virtual endocast of LB1, the type specimen for *Homo floresiensis*, are reviewed, extended, and interpreted. Seven derived features of LB1's cerebral cortex are detailed: a caudally-positioned occipital lobe, lack of a rostrally-located lunate sulcus, a caudally-expanded temporal lobe, advanced morphology of the lateral prefrontal cortex, shape of the rostral prefrontal cortex, enlarged gyri in the frontopolar region, and an expanded orbitofrontal cortex. These features indicate that LB1's brain was globally reorganized despite its ape-sized cranial capacity (417 cm^3). Neurological reorganization may thus form the basis for the cognitive abilities attributed to *H. floresiensis*. Because of its tiny cranial capacity, some workers think that LB1 represents a *Homo sapiens* individual that was afflicted with microcephaly, or some other pathology, rather than a new species of hominin. We respond to concerns about our earlier study of microcephalics compared with normal individuals, and reaffirm that LB1 did not suffer from this pathology. The intense controversy about LB1 reflects an older continuing dispute about the relative evolutionary importance of brain size versus neurological reorganization. LB1 may help resolve this debate and illuminate constraints that governed hominin brain evolution.

Introduction

Since the 2004 announcement of the new hominin species, *Homo floresiensis* (Brown et al., 2004; Morwood et al., 2004), controversy has surrounded the interpretation of its type specimen, LB1 (Argue et al., 2006). Here we review our earlier studies pertaining to this controversy and provide background for new material that is presented below. Using ratios constructed from gross measurements that capture overall shape of endocasts, our initial study (Falk et al., 2005a) revealed that LB1's virtual endocast has an unusual suite of characteristics, the combination of which sets it apart from all other known hominins. It resembles endocasts of *Homo erectus* in its relative height, the disparity between its maximum and frontal breadths, the relative widths of its caudal and ventral surfaces, and its long, low lateral profile (Falk et al., 2005a). The relative length of LB1's orbital surface (and certain segments thereof) sorts it with *Homo sapiens* (Falk et al., 2005a). LB1's small cranial capacity and brain size/body size ratio (relative brain size), on the other hand, sort it with apes and australopithecines (Falk et al., 2005a).

Our interpretation of LB1's endocast differs from those of workers who believe that LB1 represents a modern human who was afflicted with primary or secondary microcephaly rather than a new hominin species (Hall et al., 2004; Henneberg and Thorne, 2004; Weber et al., 2005; Jacob et al., 2006; Martin et al., 2006a,b; Richards, 2006; Martin, 2007; Rauch et al., 2008). Scientists agree, however, that microcephaly is not a simple or easily defined pathology. Primary microcephaly (also called 'true microcephaly,' 'primary autosomal recessive microcephaly,' or 'microcephaly vera,') is a genetically and clinically heterogeneous condition that, to date, has been associated with at least seven autosomal recessive loci and five associated genes, as well as various maladies that would once have been precluded from this diagnosis (Falk et al., 2007a). Affected individuals are frequently from consanguineous unions, and have been reported from many parts of the world.

To address the hypothesis that LB1 was a microcephalic *H. sapiens* rather than a member of a new species, we conducted an earlier comparative study of virtual endocast shape in 10 normal humans and nine extremely varied (heterogeneous) microcephalics who included individuals with different demographics and types of microcephaly, and had appropriately-sized braincase volumes (Falk et al., 2007a). The purpose of studying such a heterogeneous sample was to identify features that might be generally

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representative of microcephalics. Eight gross measurements that are traditionally used to capture brain shape were obtained electronically from the virtual endocasts, and used to generate ratios to characterize their shapes (see Falk et al., 2007a for landmarks and other details). Discriminant and canonical analyses were employed to study shape differences between the two groups and backward stepwise discriminant analysis was used to identify the most powerful discriminators. Two ratios that quantify cerebellar protrusion and relative frontal breadth, and capture shape features that are widely reported for microcephalics in the clinical literature (Hofman, 1984; Peiffer et al., 1999; Mochida and Walsh, 2001; Trimborn et al., 2004; Gilbert et al., 2005), mathematically sorted our samples of normal and microcephalic virtual endocasts. A classification function that incorporated these ratios was then used to classify LB1, a dwarf, and one microcephalic that allegedly resembles LB1 (Martin et al., 2006b) as either a normal or a microcephalic human (Falk et al., 2007a). LB1's relative frontal breadth and (lack of) cerebellar protrusion sorted it with normal rather than microcephalic *H. sapiens* (Falk et al., 2007a), which is consistent with our earlier findings (Falk et al., 2005a,b). On the other hand, the dwarf and microcephalic that was alleged to resemble LB1 were classified as microcephalics. The cranial capacity of the dwarf (752 cm^3) was somewhat larger than those of the microcephalics we studied and we believe it suffered from secondary microcephaly (Falk et al., 2007a).

Despite statistical results that were highly significant (even with our small sample sizes) and that supported the conclusion that LB1 was not a microcephalic (Falk et al., 2005b, 2006, 2007a,b), numerous workers continue to argue that LB1 was a pathological *H. sapiens* who suffered from microcephaly (Martin, 2007), Laron Syndrome (Hershkovitz et al., 2007, 2008), cretinism (Obendorf et al., 2008), or microcephalic osteodysplastic primordial dwarfism type II (MOPD II) (Hall et al., 2004; Rauch et al., 2008). An assumption that is at the heart of these various hypotheses is that LB1's ape-sized cranial capacity (417 cm^3) is too small to be from a normal hominin that lived 18,000 years ago (Martin, 2007). However, LB1's endocast reproduces a highly convoluted cerebral cortex with a unique combination of derived features, "which are consistent with capabilities for higher cognitive processing" (Falk et al., 2005a:242). Because these derived features occur in multiple areas across its surface, LB1's virtual endocast appears to represent an "epitome of neurological reorganization" (Falk et al., 2007b:42).

However, some workers have dismissed the concept of neurological reorganization as an "outlandish form of special pleading….[that] unavoidably requires the emergence of some entirely new principle in the development of the brain of the Flores hominid" (Martin, 2007:14), and one reviewer of the present paper repeatedly asserted that s/he knew of no study that correlates brain shape features with behavior. The following section provides background regarding neurological reorganization that addresses these assertions.

Historical background

Concerns about inferring cognitive abilities from the external morphology of brains or endocasts have a long tradition, partly because this endeavor was historically associated with phrenology, which was rightfully dismissed at the end of the 19th century as a pseudoscience (Gould, 1981). Further, although sulcal patterns have, traditionally, been of paleoanthropological interest (Dart, 1925, 1940, 1956; Smith, 1927), sulci usually do not correlate precisely with the borders of functionally-defined cytoarchitectonic fields (Zilles et al., 1997; Amunts et al., 1999). Despite these caveats, however, gross sulcal patterns have been associated with enlarged cortical representations (and related changes in cortical shape) that subserve functional (behavioral) specializations in mammals including carnivores (Welker and Campos, 1963) and primates (Falk, 1981, 1982), in a phenomenon that Harry Jerison has labeled the "principle of proper mass" (Jerison, 1973). For example, raccoons have greatly enlarged forepaw representations in their primary somatosensory cortices in which, remarkably, the various digit and palm pad areas are demarcated from one another by sulci, and this derived cortical morphology is attributed to the fact that raccoons use their forepaws to an unusual degree to explore their environments (Welker and Campos, 1963).

It is also well known that dramatic changes may occur in sensory and motor cortices during a human's lifetime as revealed by medical imaging studies of Braille readers and upper limb amputees, which demonstrate that the cerebral cortex can exhibit long-term adaptations, including enlargement or relocation of specific representations such as those for hands (Amunts et al., 1997). Further, gross cortical features entailing sulcal depths or patterns have been identified in people with exceptional abilities such as highly-trained musicians (Amunts et al., 1997; Schlaug, 2001; Bangert and Schlaug, 2006).

Rather than being "an outlandish form of special pleading …. [that] unavoidably requires the emergence of some entirely new principle" (Martin, 2007:14), the concept of neurological reorganization has enjoyed a long and respected tradition in paleoanthropology (Dart, 1925, 1940; Smith, 1927; Gould, 2001). Ralph Holloway, in particular, has championed the idea that endocasts may be used to detect cerebral "organizational change" that is "reflected in convolutional patterns, hemispheric asymmetries, and size-shape morphometric patterns as analyzed through multivariate statistical techniques" (Holloway, 1983b:215). Further, Holloway has expressed the view that "features of neural organization such as increased neuron size, dendritic branching and glial neural rations, and decreased neural density…. are better correlated with behavioural efficiency than cranial capacity per se" (Holloway, 1973:457). Holloway, in fact, has argued that neurological reorganization occurred during early hominin evolution with little, if any, concomitant increase in brain size (Holloway, 1983a).

Other studies that have demonstrated regularities in brain organization across placental mammals have shown that this phenomenon does not preclude species-specific adaptations in the brain (Finlay and Darlington, 1995), contrary to Martin (2007). In fact, one of the most authoritative discussions of cortical organization and evolution suggests "that the cortex is a veritable hotbed of evolutionary reorganization" (Preuss, 2001:140), and notes that "functional imaging studies in humans indicate that higher-order cognitive tasks engage multiple cortical areas dispersed across the cortical mantle, areas that are probably linked by direct corticocortical connections. The evolution of new cognitive abilities might involve the enhancement of existing links between areas, or even the establishment of links between previously unconnected areas" (Preuss, 2001:156) - or "rewiring" to put it metaphorically. Although the debate about brain size versus neurological reorganization has been polarized in the past (Falk and Gibson, 2001), it is now clear that both were important during hominin evolution, and that new information and approaches are helping to reconcile what Stephen Jay Gould called "a falsely perceived dichotomy":

> Moreover, the commingling of cellular with biometric studies, and of growths and sizes of parts and wholes with research on microarchitectural and cellular reorganization, testifies to the healing of past controversies, and to a coordinated approach using the most fruitful themes of both sides in a falsely perceived dichotomy (Gould, 2001:xvi).

Below, we review and extend our earlier observations about brain shape in microcephalics (Falk et al., 2007a), and discuss the

intersection of our findings regarding microcephalics with those from studies of mutations underlying certain neurological disorders that may have implications for the nature and timing of hominin brain evolution. We also extend our earlier observations of the virtual endocast of LB1 (Falk et al., 2005a). Others have questioned why we identified certain features of LB1's virtual endocast as derived rather than pathological, which we address in detail for the first time on a feature-by-feature basis. Although the phylogenetic origin of *Homo floresiensis* is not yet resolved, the virtual endocast of LB1 has important implications for understanding hominin brain evolution.

Materials and methods

This section reviews procedures that were developed in our earlier studies, and addresses some concerns that have been raised. Our initial sample included three-dimensional computed tomographic (3DCT) reconstructions of the internal braincases (virtual endocasts) that reproduce details of external brain morphology from LB1, an adult female chimpanzee, an adult female *H. erectus*, a contemporary woman, and a European microcephalic (Falk et al., 2005a). As detailed elsewhere (Falk et al., 2005a), the CT data were obtained, processed, and analyzed at Mallinckrodt Institute of Radiology. For comparative purposes, we also studied traditional endocasts from Sts 5 (*Australopithecus africanus*), KNM-WT 17000 (*Paranthropus aethiopicus*), 10 humans, 10 gorillas, 18 chimpanzees, an adult female pygmy, and five *H. erectus* (Falk et al., 2000, 2005a). Our observations confirmed that LB1's cranium was free of substantial distortion, as originally described (Brown et al., 2004), and the slight amount caused by pressure from sediments was adjusted when we interpolated missing areas during CT-reconstruction of the virtual endocast (Falk et al., 2005a). Elsewhere in this volume (Baab and McNulty, 2009), postmortem distortion is also shown to be of minimal concern with respect to 3D analyses of LB1's cranial asymmetries.

Linear measurements, ratios, and volumes were investigated to describe LB1's virtual endocast within a comparative context (see Falk et al., 2005a,b for data). Our discriminant analyses focused on ratios rather than absolute measurements to compensate for different brain sizes, and most of the linear measurements that we collected were gross ones that have been used for the better part of a century to capture aspects of overall brain shape in primates (e.g., see Falk and Clarke, 2007).

Our original study of LB1's endocast included only one endocast from a microcephalic who was 10 years old when he died (Falk et al., 2005a). Inclusion of this specimen was criticized because the calotte and base of the cast of the skull that we CT scanned to obtain a virtual endocast were of different colors and chemical compositions (Martin et al., 2006a,b). Despite the two parts having been cast with different materials, the CT data produced a virtual endocast with a volume of 276 cm^3 (Falk et al., 2005a), which is only 4 cm^3 larger than the capacity that was initially reported (Vogt, 1867; Falk et al., 2007a). Inclusion of this specimen was also criticized because of the young age of the individual (Martin et al., 2006a,b); however, analyses of a large heterogeneous sample of microcephalics suggest that brain size tends to decrease after about four years of age, which implies that braincases of microcephalics reach their maximum size (cranial capacity) by this age and, further, that the "fit" of microcephalic brains within their respective skulls becomes looser with advancing age (Hofman, 1984). A potential limitation with this analysis is that it used the traditional approach for anthropological study of brain growth using cross-sectional data rather than actual growth curves of individuals (Hofman, 1984).

An important objective of our earlier microcephalic study was to identify a range of cranial volumes for microcephalics that could reasonably be compared with LB1 (Falk et al., 2007a). Because brains of microcephalics appear to decrease in size with age, adult microcephalic brain weights depart further from the mean for normal humans than do brain weights of younger microcephalics (Hofman, 1984); thus, the mean of 400–500 g/cm^3 that is widely quoted in the clinical literature for microcephaly is 19–21 standard deviations (SDs) below the mean for normal adult males and 12–13 SDs below the mean for adult females (Hofman, 1984; Falk et al., 2007a). We, therefore, estimated the range for brain size in microcephalics by computing the mean for a combined sample of 25 male and female microcephalics aged 21 to 74 years (from data kindly provided by Michel Hofman), rather than using data for normal human adults to estimate the range of brain sizes in adult microcephalics, as other workers have done. (The same reasoning would apply if one were to use definitions of microcephaly based on occipitofrontal head circumference (Falk et al., 2007a).) The mean brain mass for the combined microcephalic sample is 365 g with a SD of 95, yielding an estimated range (mean $\pm$ 3 SDs) of 80–650 g (or cm^3) (Falk et al., 2007a). This average is somewhat less than those quoted for microcephalics in the literature, possibly because it was determined from a sample that includes numerous older specimens with brain masses that had decreased with age (Hofman, 1984). We believe 650 cm^3 is a reasonable upper limit for braincase (endocast) volume in primary microcephalics, although secondary microcephalics (i.e., individuals whose small brains are secondary to some other pathology) may have somewhat larger mean braincase volumes (Falk et al., 2007a).

To maximize the chances of capturing general brain shape features that might characterize microcephaly despite its variability and genetic complexity, we compiled a heterogeneous sample of microcephalics with appropriately-sized braincase volumes (Falk et al., 2007a). Although, like fossil hominins, complete microcephalic skulls are relatively rare, we were able to locate and measure virtual endocasts for nine microcephalics by processing three-dimensional computed-tomographic (3D-CT) scans (see Falk et al., 2007a for details). The specimens in our sample represent both sexes, range in age from the 10-year-old individual used in our initial study (Falk et al., 2005a) to mature adults, range in cranial capacity from 276–671 cm^3 (we included two specimens that were slightly above our estimated upper limit of 650 cm^3 to increase sample size), and come from the United States, Europe, South America, and Africa. The sample also includes both primary and secondary microcephalics (Falk et al., 2007a: Table 1), although incomplete records prevented us from estimating the percentages of each. For comparative purposes, we also processed and measured virtual endocasts from a mixed Euro-American and African American sample of ten normal humans that includes six females and four males ranging in age from about 18–45+ years (Falk et al., 2007a).

Below, we provide further evidence that refutes the suggestion that LB1 suffered from microcephaly and detail new information about derived features of LB1's cerebral cortex. Our references to specimens from the genus *Australopithecus* are based on reports from the literature and analyses of endocasts in DF's collection, including Taung (Falk and Clarke, 2007), Stw 505, Sts 5, Sts 60, and the No. 2 specimen from Sterkfontein. Our observations pertaining to *Paranthropus* are based on the literature and analyses of the following endocasts from DF's collection: SK 1585, KNM-WT 17000, OH5, KNM-ER 23000, and KNM-WT 17400 (Falk et al., 2000).

Results

Microcephaly

Since the announcement of the discovery of *H. floresiensis*, a number of workers have suggested that LB1 represents a member

of *H. sapiens* who was afflicted with pathological microcephaly (literally 'small head') rather than a new species of hominin (Henneberg and Thorne, 2004; Weber et al., 2005; Jacob et al., 2006; Martin et al, 2006a,b; Martin, 2007; Richards, 2006). Most of the conclusions that LB1 may have been a human microcephalic, however, were based on postcranial, dental, or cranial features other than the brain/endocast of LB1, microcephalics, and normal humans. Those who considered endocasts at all failed to quantify relevant neurological features in support of their interpretations, or did so only superficially. Although one study claimed to analyze 19 microcephalic endocasts, including one with six proportional measurements (ratios) that were supposedly nearly identical to LB1's (Weber et al., 2005), photographs of this endocast were not published and our repeated efforts to learn the identification number and repository of the specimen so that it could be included in a comparative analysis met with failure (Falk et al., 2005b).

The four of our nine microcephalics that were less than fully adult are distributed across the adult sample (Fig. 1), contrary to the suggestion that "inclusion of microcephalics that died young and hence presumably suffered from low-functioning syndromes is likely to bias any analysis towards greater distinction from normal humans" (Martin, 2007:18; see also Martin et al., 2006a,b). Further, by definition, "low-functioning microcephalics" suffer an early "death that typically occurs within the first several years of life" (Gilbert et al., 2005:4). Since our youngest specimen died at 10 years of age, our sample consists only of high-functioning individuals.

The same workers that suggest our sample of microcephalics is too heterogeneous with respect to age, assert that it is also too homogeneous because there are "more than 400 genetic syndromes associated with microcephaly" (Martin, 2007:15). A source for this observation (Gilbert et al., 2005), however, notes that this statistic applies only if microcephaly is "defined as an occipitofrontal circumference that is at or below −2 standard deviations (SD) at birth," and concludes that "a more restrictive definition of microcephaly has been proposed….The head size of long-term survivors in these severe cases typically ranges between −5 and −10 SD later in life" (Gilbert et al., 2005:4). Traditional definitions of microcephaly based on brain size in normal humans are also too broad when it comes to defining an appropriate sample of microcephalics with which to compare LB1; thus, a definition of microcephaly that includes individuals with brain sizes that "fall more than 3 SD below the mean for age and sex" would include adult females with brains of 1100 g and males with brains of 1300 g (Hofman, 1984:88), a far cry above the 417 cm^3 of LB1 (Falk et al., 2005a). (Although it is somewhat larger, cranial capacity in cubic centimeters is traditionally accepted as a proxy for brain mass in grams.) For these reasons, we believe it is better to define comparative samples of microcephalics that are to be compared to small-brained hominins such as LB1 using a range of variation estimated from microcephalics themselves (as we have done) rather than from normal *H. sapiens* (Falk et al., 2007a).

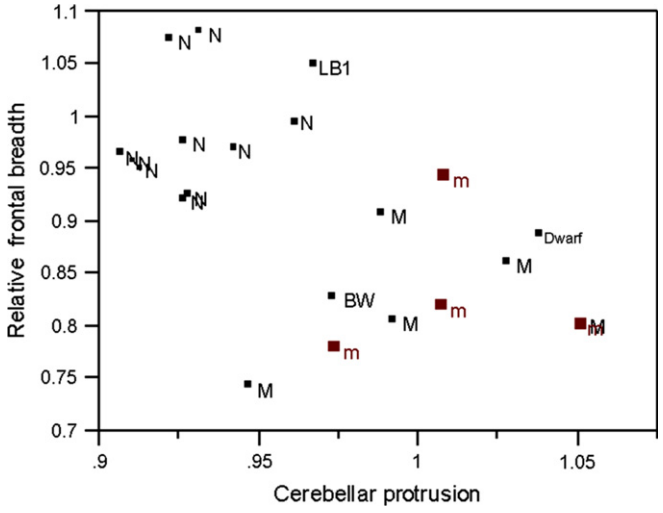

Figure 1. Scatter plot of relative frontal breadth on cerebellar protrusion. Discriminant analysis demonstrated that these two variables classified microcephalics (M, adults; m, less than fully adult) and normal humans (N) with 100% success. Notice that the four microcephalics that were less than fully adult are distributed across the adult sample, contrary to the suggestion that their inclusion might bias our analysis towards greater distinction from normal humans (Martin, 2007). The Basuto woman (BW, a microcephalic whose endocast is alleged to resemble LB1's), the dwarf, and LB1, which were not used to develop the classification functions, were classified, respectively, as two microcephalics and a normal human. Reproduced from Falk et al. (2007b).

Along similar lines, it has been suggested that we selectively excluded from our microcephalic study an endocast from a microcephalic specimen from the Hunterian Museum of the Royal College of Surgeons (London) (RCS) that we supposedly examined: "Falk et al. (2007a) …. excluded the RCS hemi-skull that they had also examined" (Martin, 2007:17). Martin, on the other hand, added this specimen to our plot of relative frontal breadth against cerebellar protrusion (Fig. 1) and claimed that with its addition, "the reported clear separation between microcephalic and normal humans is eliminated" (Martin, 2007:18). We have, however, never examined the RCS hemi-skull or its endocast although we have seen photographs of them. The reason we did not seek a copy of the half-endocast is because it appeared from the photograph of the hemi-skull that it had been cut off center, and the measurements required for our analyses depend on bilateral data (as clearly stated in Falk et al., 2007a:2517). Because little, if anything, is known about petalia asymmetries in skulls of microcephalics, we did not think it would be scientifically sound to correct the midline of a hemi-endocast produced from an irregularly-cut hemi-skull, estimate the bilateral dimensions from the corrected hemi-endocast, and use those estimates to classify the skull.

We think that use of measurements that incorporate uncorrected endocast midlines is even more problematic. According to Martin (2007:18), "In fact, the RCS hemi-skull was cut slightly lateral to the midline, this reducing its breadth by a small amount. As this reduces the breadth of the frontal lobes, the point for RCS should actually lie somewhat higher than indicated." What, we wonder, does such a reconstruction do to bilateral measurements obtained from the caudal end of the endocast? Further, how does one determine which hemispheres have the most projecting frontal, occipital, and cerebellar poles from such a hemicast, all of which are required to obtain the measurements necessary to place a specimen on Fig. 1 (Falk et al., 2007a)?

It has recently been suggested that, rather than being a distinct species, *H. floresiensis* suffered from microcephalic osteodysplastic primordial dwarfism type II (MOPD II), which is said to be characterized by, "an adult height of 100 cm, grossly normal intelligence despite severely restricted brain size, absence of a sloping microcephalic morphology, and a number of minor morphological features including facial asymmetry, small chin, abnormal teeth, and subtle bony anomalies of the hand and wrist" (Rauch et al., 2008:818–819). Although MOPD II patients share short stature with *H. floresiensis*, the resemblance stops there, as illustrated by comparing published images of both groups (Brown et al., 2004; Rauch et al., 2008). Relative head size (compared with stature) in MOPD II patients appears markedly enlarged compared with LB1, whose relative brain size scales like an australopithecine or ape (Falk et al., 2005a). Rather than having grossly normal intelligence, MOPD II patients usually have intelligence quotients in the 50–90 range, and "none have been able to live independently" (Hall et al.,

2004:63). Further, "no pregnancies have been documented in adult women with MOPD II" (Hall et al., 2004:61), which is relevant because, to date, *H. floresiensis* is represented by 12 individuals who were inhabitants on Flores between 95,000–17,000 ka and must, therefore, have been fertile. The other features listed by Rauch et al. (2008) are misleading. The slope of the forehead (indeed the profile of the entire face) does not appear similar in MOPD II patients and LB1, and rather than having supraorbital tori like *H. floresiensis*, the former are characterized by underdeveloped supraorbital ridges (Rauch et al., 2008). Despite lacking an external chin (a primitive feature for hominins), LB1 is prognathic; MOPD II patients appear retrognathic or micrognathic. The teeth of the latter are small and most often there is increased space between them, whereas *H. floresiensis* is megadont (Brown et al., 2004). Tooth roots of the former are often hypoplastic or absent (Hall et al., 2004), which is the opposite of *H. floresiensis*. Enlarged sella turcica, and premature closure of fontanelles and cranial sutures are often reported for MOPD II patients (Hall et al., 2004), but do not characterize *H. floresiensis* (Jungers et al., in preparation; Falk et al., 2009). Rather than having the robust long bones with normal cortical thickness of *H. floresiensis*, the bones of MOPD II patients are thin, and their feet are short rather than extraordinarily long as is the case for the former (Hall et al., 2004; Jungers et al., 2008). The suggestion that MOPD II patients share features of the wrist documented for *H. floresiensis* (Tocheri et al., 2007) is also belied by the literature (Hall et al., 2004).

To date, we are unaware of descriptions in the literature of microcephalic brains, including those of MOPD II patients (Hall et al., 2004; Rauch et al., 2008), that manifest anything like the suite of derived cortical features seen in LB1's virtual endocast (Falk et al., 2005a, 2007a,b). In particular, the degree of protrusion of the occipital lobe over the cerebellum, the expanded gyri in Brodmann's area 10, and the relatively great width and orbitofrontal expansion of LB1's frontal lobes (Falk et al., 2005a, 2007a) are antithetical to the features that typify brain shape, not just in our sample of microcephalics (Fig. 2), but also in microcephalic brains that are discussed, measured, and illustrated in the clinical literature (Hofman, 1984; Peiffer et al., 1999; Mochida and Walsh, 2001; Trimborn et al., 2004; Gilbert et al., 2005; Falk et al., 2007a).

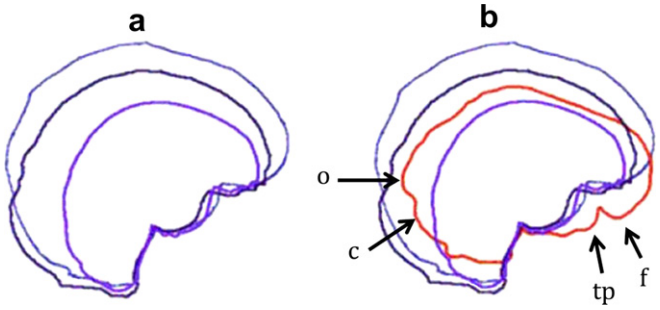

Figure 2. Right-lateral outlines of endocasts from microcephalics and LB1. (a) Superimposed outlines of three endocasts from our mixed sample of primary and secondary microcephalics, including the smallest (276 cm^3) and largest (671 cm^3) of the nine that we analyzed. The outlines were aligned along the ventral margins of their brainstems and the points that define the intersection of the right brainstem with the right temporal lobe were superimposed. These outlines demonstrate a generally similar brain shape in different-sized microcephalics despite the heterogeneity of our sample. (b) The same three outlines with the outline of LB1's 417 cm^3 endocast superimposed. Brain shape in LB1 exhibits a long-low profile that has an occipital lobe (o) that projects noticeably in a caudal direction relative to the cerebellum (c) in contrast to the microcephalics, and a derived ventral expansion of the orbital surface of the frontal lobes (f) that characterizes normal humans but not primary microcephalics. Notice that LB1's temporal pole (tp) also projects rostrally compared with those of the microcephalics. Modified after electronic Supporting Information for Falk et al. (2007a).

LB1's virtual endocast

LB1's braincase volume was determined electronically to be 417 cm^3 (Falk et al., 2005a), which is larger than an earlier estimate of 380 cm^3, as first measured with mustard seeds (Brown et al., 2004). The 37 cm^3 difference is attributable to variation in how cranial holes were plugged and thus to measurement error associated with the reconstructions. LB1's virtual endocast reproduces numerous sulci, gyri, and other details of the cerebral cortex, in keeping with the observation that skulls of smaller-brained species within a group of related species tend to produce relatively detailed endocasts (Radinsky, 1972). In overall shape, it manifests a modest left-frontal and extreme right-occipital petalia. Such "reversed" petalia patterns are correlated to some degree with left-handedness in living people (LeMay, 1977; LeMay et al., 1982), especially if the right-occipital petalias are particularly pronounced (Bear et al., 1986), as is the case with LB1.

We initially observed that LB1's endocast appeared globally derived because of an expanded posterior parietal association cortex, wide temporal lobes, and convoluted dorsal prefrontal cortex (Falk et al., 2005a). Subsequently, we observed that LB1's frontal lobes also reproduced a derived (expanded) orbital surface (Falk et al., 2007a). Here we provide information about why we now identify seven features of LB1's endocast as derived and nonpathological.

Caudally positioned occipital lobe In LB1, the occipital lobe extends caudally relative to the cerebellum, which is tucked forward underneath the occipital cortex (Falk et al., 2005a) (Fig. 3a). The relatively rostral position of the cerebellum is a derived feature compared with the usual position for apes and is believed to be associated with reorganization of the parieto-occipital association cortices that accompanied the assumption of erect posture (Smith, 1927; Dart, 1940). Although the derived condition is not fully developed in some (but not all) early hominin specimens (e.g., KNM-WT 17000, Sts 5), it is a classic one that characterizes humans (Smith, 1927).

Lack of a rostrally-located lunate sulcus Whether or not lunate sulci are located in a derived human-like caudal position or in a primitive ape-like rostral location in australopithecine endocasts, has been

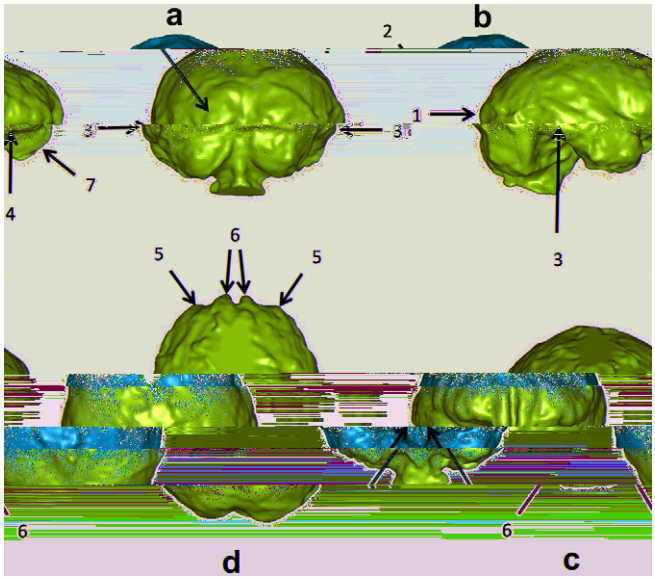

Figure 3. Virtual endocast of LB1. Views: a, right lateral; b, posterior; c, frontal; d, dorsal. Numbers for arrows correspond to the seven derived features discussed in the text.

argued since Dart first expressed the opinion that Taung reproduced a lunate sulcus in a human-like location (Dart, 1925), and this issue still remains unresolved (Holloway et al., 2004; Falk, 2007, in press). Both hemispheres of LB1's endocast lack a rostrally-located ape-like lunate sulcus, and the left hemisphere manifests a small crescent-shaped sulcus that we identified as a lunate sulcus in the derived, human, caudal location (Falk et al., 2005a) (Fig. 3b). Since we made this identification, however, new research suggests that the caudally-located crescent-shaped sulci that infrequently appear in human brains (Connolly, 1950) may *not* be homologous with lunate sulci of nonhuman primates (Allen et al., 2006). If so, this raises the question of whether or not humans have "true" lunate sulci that, by definition, approximate the rostral border of Brodmann's area 17 (primary visual cortex). This, in turn, raises an important question about whether or not lunate sulci migrated caudally during hominin brain evolution, as has been widely presumed (Dart, 1925; Smith, 1927; Holloway et al., 2004), or if lunate sulci simply disappeared as brains enlarged and became reorganized during hominin evolution. (For discussion of reorganization in the visual cortex, see Preuss et al., 1999; Preuss, 2001.) Ideally, one would investigate this problem by viewing a series of hominin endocasts from different parts of the fossil record. This is not easily accomplished because hominin endocasts usually fail to reproduce accurate sulcal patterns of the occipital lobe (Le Gros Clark et al., 1936; Connolly, 1950) and because larger primate endocasts reproduce poorer detail than smaller ones (Connolly, 1950; Radinsky, 1972). What we can say, however, is that LB1 does not have a lunate sulcus in the primitive ape-like location despite its ape-like brain size, and similar lack of a rostrally-located lunate sulcus in humans is associated with a derived enlargement of parieto-occipital association cortices (Smith, 1927; Dart, 1940).

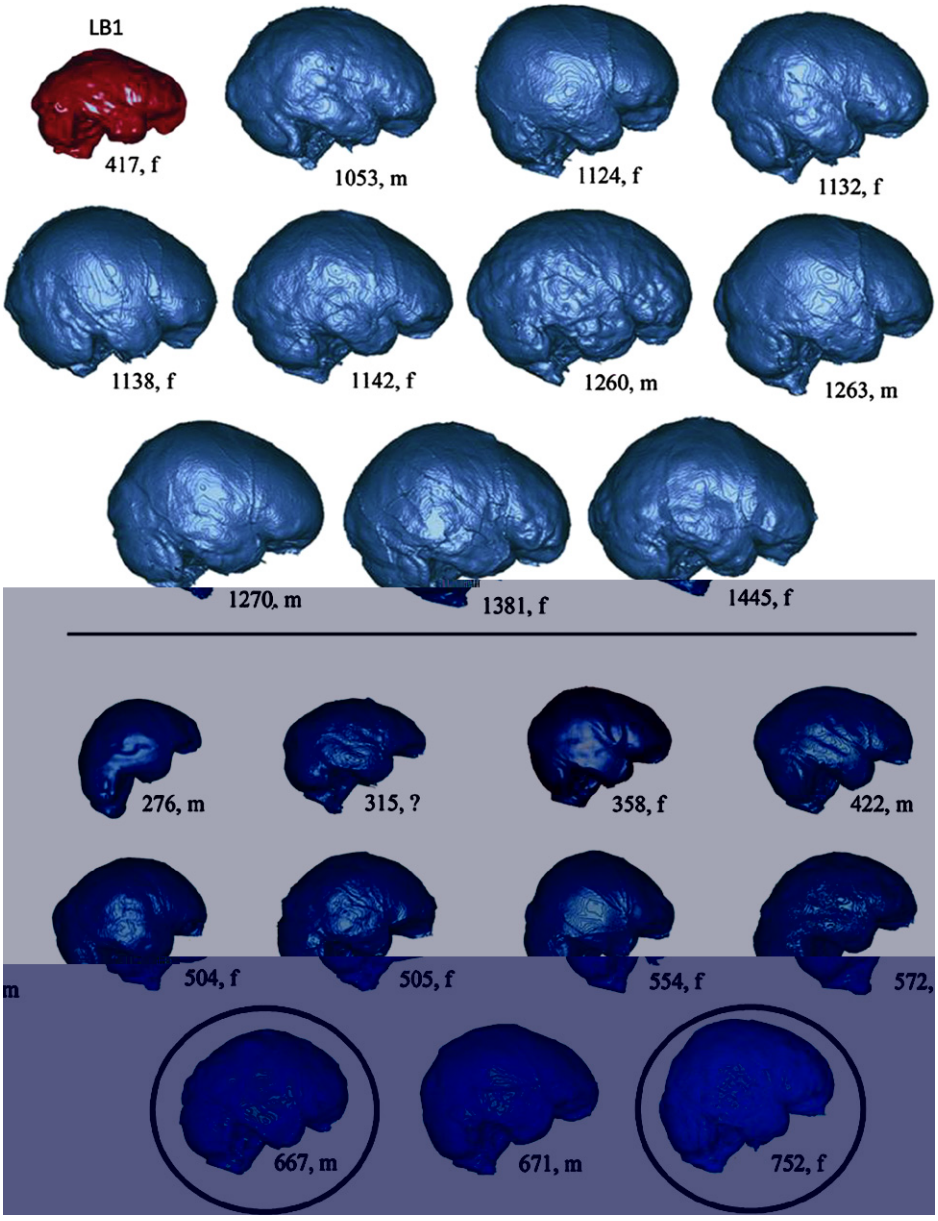

Figure 4. Comparisons of right lateral views of virtual endocasts from 10 normal humans (above) and 11 microcephalics (below). Discriminant and canonical analyses classify the virtual endocast of LB1 (upper left) with normal humans rather than a subset of nine microcephalics used to formulate a classification function (Falk et al., 2007a). Records associated with the specimens do not reveal the percentages of primary and secondary microcephalics in this sample, although the two circled specimens are known to have small brains that are secondary to other pathologies. See Falk et al. (2007a) for further details about specimens. Images are labeled with their cranial capacities (cm^3) and sex: f, female; m, male. Modified after Falk et al. (2007a).

et al., 2007a) (Fig. 4). Despite its tiny volume, the endocast of LB1 shares the derived expansion of the orbital surface of the frontal lobes with much larger-brained normal humans (Figs. 3a, 4) and *A. africanus*.

The ventral orbitofrontal cortex receives input from visual and sensory systems (e.g., taste and smell), influences motivational and emotional behavior in primates including humans, and is involved in regulation of appropriate social behavior (Rolls, 2004). Interestingly, human patients exhibiting socially inappropriate behavior associated with damage to this region have been demonstrated to have specific impairments in the identification of vocal and facial emotional expressions (Hornak et al., 1996; Rolls, 1999, 2004). By 2.5–3.0 Ma, derived expanded ventromedial orbitofrontal cortices had evolved in brains (endocasts) of *Australopithecus* (Smith, 1927; Dart, 1940; Falk et al., 2000), the genus which is thought to have given rise to *Homo* (Falk et al., 2000; González–José et al., 2008), while *Paranthropus* retained the primitive relatively-flattened orbital surfaces similar to those of extant African great apes (Falk et al., 2000).

Discussion

The seven derived features that span the surface of LB1's cerebral cortex from posterior to anterior do not appear to be pathological: an expanded occipital lobe that projects further caudally than the cerebellum has traditionally been recognized as a derived feature that separates most hominins from most great apes (Smith, 1927; Dart, 1940). LB1's caudally-located crescent-shaped sulcus is superficially similar to the so-called lunate sulcus that is sometimes seen as a normal variation in brains of *Homo sapiens* (Connolly, 1950; Allen et al., 2006), and is consistent with its lack of a primitive ape-like

rostrally-located lunate sulcus. Together, these features strongly suggest a derived parieto-occipital association cortex, which has been recognized classically as an important higher-order association region. (In addition to enhancing the ability to synthesize information from different senses, early workers attributed such reorganization to changes in the sizes and interconnections between the neurological substrates for hands and feet that accompanied selection for bipedalism (Smith, 1927; Dart, 1940).) Although LB1 stands apart from other hominins in the extent of its brachycephaly due to laterally expanded caudal portions of the temporal lobes, expansion of this part of the temporal lobe has long been recognized as a derived trait compared with apes (Smith, 1927; Dart, 1940) that occurred in ancestors of *H. sapiens* after their split from chimpanzees. LB1's pattern of gyri in the lateral prefrontal cortex, associated with lack of an ape-like orbitofrontal sulcus, and a more rostrally squared-off prefrontal cortex are also derived features compared with apes (Falk, 1983). Apes, and some fossil hominins, also manifest distinct gyri that appear similar to, but smaller than, those in LB1's frontopolar region (Brodmann's area 10). The expanded ventromedial surface of LB1's orbitofrontal cortex is a normal derived feature that appeared by 2.5–3.0 Ma in gracile australopithecines (Smith, 1927; Dart, 1940; Falk et al., 2000). Thus, although the combination of primitive and derived features seen in LB1's virtual endocast is unique, none of them appear pathological and the most dramatic features (expanded caudal temporal lobes and expanded prefrontal cortices) appear in just those parts of the brain that have recently been recognized as foci of differential selection during the course of human evolution based on studies utilizing modern imaging and cytoarchitectonic techniques (Semendeferi et al., 1997; Semendeferi and Damasio, 2000; Semendeferi, 2001; Preuss, 2001; Rilling and Seligman, 2002; Schoenemann et al., 2005). For these reasons, we believe that LB1 had a small, globally neurologically-reorganized brain that is consistent with the higher cognitive abilities that have been attributed to *H. floresiensis* (Brown et al., 2004; Morwood et al., 2004).

Hominin brain evolution: shift in the big picture

Until recently, received wisdom held that hominin brain size increased somewhat in australopithecines compared with their ape ancestors, "took off" in early *Homo* around two million years ago, continued to increase significantly (with or without spurts) until the relatively recent time of classic Neandertals, and thereafter decreased a bit and leveled off at its present world mean of around 1350 cm^3 (Falk, 2004). The discovery, however, of small-brained "transitional" hominins in Dmanisi, Republic of Georgia (Gabunia et al., 2000; Vekua et al., 2002), the redating of Java *H. erectus* sites (Huffman, 2001; Morwood et al., 2003), and the finding that some of the earlier fossil hominins had smaller cranial capacities than previously believed (Falk, 2004, 2007; Falk et al., 2000; Falk and Clarke, 2007), suggest that cranial capacity did not begin to accelerate dramatically in early *Homo* around 2.0 Ma, but began increasing in the *Australopithecus* ancestors of *Homo* long before then (Fig. 5) and continued increasing thereafter without obvious "punctuated" events (Leigh, 1992; see Falk, 2007 for details).

In addition to absolute brain size, it is important to examine trends in the evolution of brain size relative to body size (relative brain size). Over the years, various encephalization quotients (EQs) and other indices have been developed for estimating how much mean brain size of any given taxon departs from that expected for a mammal (or other baseline animal) of equivalent body size (Stephan et al., 1970; Stephan, 1972; Jerison, 1973, 1982, 2001). It is well known, for example, that humans have brains that are approximately three times the size predicted for an ape (or other nonhuman primate) of equivalent body size (Stephan et al., 1970; Passingham, 1973, 1975; Passingham and Ettlinger, 1974), which is indicated by an index (i) = 3 (Fig. 5). Predicting relative brain size for fossil hominins is trickier because of the twofold difficulty of (a) correctly identifying the taxa associated with hominin postcrania, and (b) using those postcrania to estimate body size. What are needed to formulate reasonable hypotheses about relative brain size in fossil hominins are relatively complete skeletons that have both skulls and postcrania, and these are few and far between. Despite some opinions to the contrary, the best estimate for relative brain size of australopithecines is similar to that expected for apes of equivalent body sizes (Fig. 5, i = 1). The best estimate for early *H. erectus* (~1.5 Ma) is gleaned from the relatively complete skeleton of the youth from Nariokotome in Kenya (KNM-WT 15000), which has a cranial capacity that is twice the volume expected for an ape of equivalent body size (Fig. 5, i = 2; see Falk, 2007 for details).

When it comes to relative brain size, LB1 is somewhat perplexing because it scales like an ape or australopithecine (i = 1 in Fig. 5) rather than, as far as one can tell, an early *H. erectus* (Falk et al., 2005a). The hypothesis that *H. floresiensis* might represent an endemically-dwarfed descendant of *H. erectus* (Brown et al., 2004) has been rejected by some workers because of LB1's relative brain size (Martin et al., 2006a). Although it is conceivable that an endemically-dwarfed descendant of a larger-bodied species would scale along the same brain size/body size curve (and thus have a relatively larger mean brain size because of its smaller body size) (Falk et al., 2005a), it is not yet known whether or not this occurs in insular dwarfs or if the situation is more complicated and possibly variable depending on taxa (Schauber and Falk, 2008).

As Fig. 5 illustrates, LB1 has important implications for the "falsely-perceived dichotomy" regarding the relative importance of brain size versus neurological reorganization during hominin brain evolution (Gould, 2001). LB1 reveals that significant cortical reorganization was sustained in ape-sized brains of at least one hominin species, which is in keeping with Holloway's long-held belief that cortical reorganization may take place in hominins without a concomitant increase in brain size (Holloway, 1983a). Whether the reorganization occurred initially in big- or small-brained ancestors of LB1 remains an open question. For now, Fig. 5 suggests that the "space" within which hominin brains evolved is delineated by (at least) two vectors: brain size (y axis) and cortical reorganization (x axis), and that either one may have been differentially important in any given species.

Although "thousands of mutations in many hundreds (or possibly even thousands) of genes might have contributed to the evolution of the human brain" (Gilbert et al., 2005:584), some workers hypothesize that two autosomal recessive genes that cause microcephaly when mutated were under pronounced selection (in their non-mutated states) in the last common ancestor of apes (microcephalin at the MCPH1 locus) and in hominins (ASPM [abnormal spindle-like microcephaly associated] at the MCPH5 locus) in conjunction with selection for increasing brain size (Zhang, 2003; Evans et al., 2004; Gilbert et al., 2005). Our findings are consistent with the hypothesis that genes associated with microcephaly may have been among those that contributed to aspects of hominin brain evolution because certain shape characteristics of primary microcephalic brains/endocasts resemble primitive traits of early hominins who are not believed to have contributed directly to the ancestry of *H. sapiens* (González-José et al., 2008). Thus, primary microcephalics have relatively pointed frontal lobes (when viewed dorsally) and apelike shapes of their orbital surfaces (Falk et al., 2007a) that resemble the condition of *Paranthropus* but not *A. africanus*, which manifests derived squared-off frontal lobes and ventrally expanded orbital surfaces similar to those of humans (Falk et al., 2000). These converging genetic and paleoneurological data raise the interesting possibility that some of the genes involved in microcephaly (e.g., ASPM) may

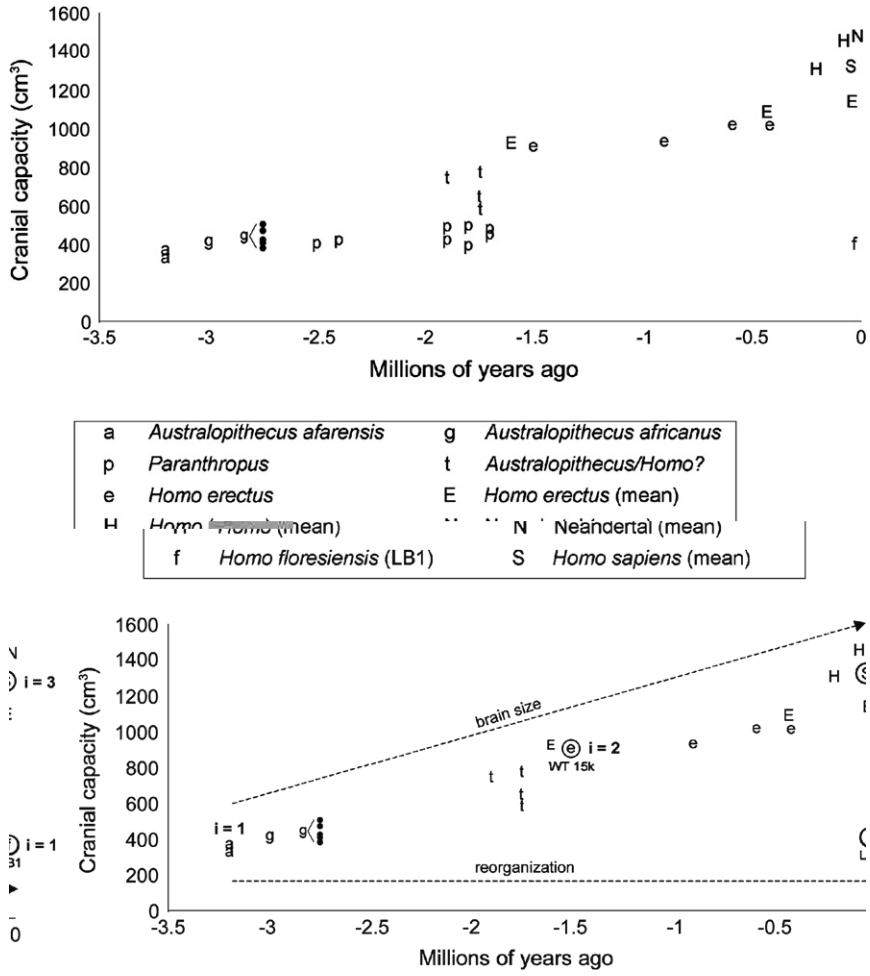

Figure 5. Cranial capacities of hominins plotted against time. The top plot contains capacities for *Paranthropus*, a genus that is not believed to be directly ancestral to humans (González-José et al., 2008). The trend for brain size increase appears flat until about 2.0 Ma, and then begins to increase in *Homo*. *Paranthropus* is excluded in the bottom plot, and the trend for brain size increase now appears to increase from before 3.0 Ma, due in part to inclusion of the recently described "transitional" specimens (t) from Dmanisi, Republic of Georgia (listed as *Australopithecus/Homo?*). The earliest australopithecines and relatively recent LB1 (*Homo floresiensis*) have brain sizes expected for apes of equivalent body sizes (index or i = 1); *Homo erectus* from Nariokotome (KNM-WT 15000) has a brain that is twice the size expected for similarly-sized apes (i = 2); and extant *Homo sapiens*' mean brain size is three times that expected for apes of equivalent body size (i = 3). Reproduced from Falk (2007).

have played a role in, not just brain size, but also the evolution of neurological reorganization of (at least) the frontal lobes in the *Australopithecus* → *Homo* lineage that led to humans (Falk et al., in preparation). We are currently exploring this hypothesis. Meanwhile, who knows what other combinations of brain size and neurological reorganization existed in, as yet, undiscovered hominins? As Fig. 5 suggests, the realm of possibilities is impressive.

Conclusions

Traditionally, announcements of new hominin species that challenge conventional scientific paradigms have been greeted with skepticism by scientists who question the new species' authenticity and, instead, attribute their remains to aberrant apes or pathological human beings. This happened with the discoveries of Neandertals (Gruber, 1948; Drell, 2000), *Homo* (*Pithecanthropus*) *erectus* (Dubois, 1896), and australopithecines (Dart, 1925; Findlay, 1972), all of which were eventually recognized as legitimate species. It has happened again in 2004 with the announcement of *H. floresiensis* (Brown et al., 2004; Morwood et al., 2004), whose type specimen (LB1) has been attributed to a pathological *H. sapiens* afflicted with various types of primary and secondary microcephaly, Laron Syndrome, and cretinism. Although it is beyond the scope of this paper, elsewhere each of these alternative hypotheses have (Falk et al., 2007a,b) or are (Falk et al., 2009; Jungers et al., in preparation) being answered with scientific data.

As discussed in this paper, we find nothing in LB1's endocast to suggest microcephaly. It has been suggested that we must prove that LB1 did not have any of 400 genetic syndromes that, allegedly, are associated with small brain sizes in *H. sapiens* (Martin, 2007; but see above). A general assertion of pathology, however, is not a testable null hypothesis. The burden of proof is on those who believe that LB1 was a microcephalic (or suffered from another type of pathology), and they should find a documentable representative of *H. sapiens* (living or dead) with a known pathology that reproduces a virtual endocast that appears like LB1's. So far, they have not done so, although it is not for lack of trying and making assertions that cannot be substantiated (Weber et al., 2005; Falk et al., 2005b).

The debate about LB1 seems to us to be particularly contentious, partly because it entails the long and passionate controversy about the relative importance of brain size versus neurological reorganization during hominin evolution (Falk and Gibson, 2001). As we have shown, LB1 suggests that cortical diversity and its associated neurological reorganization was an important factor in the "big picture" of hominin brain evolution that has previously been

under-appreciated. Indeed, our experience in the debate about *H. floresiensis* confirms the suggestion that some scientists find cortical diversity in general (and LB1 in particular) "inconvenient":

> The fact of cortical diversity is perhaps even more inconvenient for those anthropologists and paleontologists wanting to investigate brain evolution. To acknowledge the diversity of cerebral organization is to acknowledge that the issue of reorganization versus encephalization has been settled in favor of reorganization. There is no longer a good reason to consider encephalization as an index of some general functional capacity (intelligence) that is common to all mammals. We must face up to the fact that encephalization is largely uninterpretable in terms of cognitive or behavioral processes (Preuss, 2001:154).

It is important to acknowledge that, at the moment, only one endocast (LB1) is available for *H. floresiensis*. This is, however, not unusual for discoveries of new hominins. As detailed in this paper and elsewhere (Falk et al., 2005a,b; 2007a,b), we analyzed data from 3D-CT scans of LB1's skull to produce a virtual endocast (which is highly preferable to ones produced by traditional methods (Falk, 2004)), and used rigorous scientific methods to gather and analyze data from the endocast and to interpret them within appropriate comparative contexts. That is the best anyone can do with one specimen, and it is our fondest hope that more skulls of *H. floresiensis* will be discovered so that we can learn more about its brain. As has happened historically with the discovery of new hominin taxa, we expect that recovery of more specimens will eventually resolve the debate about *H. floresiensis*.

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