Supporting Information

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SI1: The LB1 Brachycephaly Paradox—Words and Numbers in Conflict

This section focuses primarily on contradictory characterizations of the cranial vault shape of LB1 in published reports.

The initial paper (1) attempting to establish the taxon *Homo floresiensis*, which was based on a hypodigm comprising only LB1 and LB2 (an isolated left P3), suggested a relationship to *Homo erectus*, the only fossil hominin taxon known from the region. A key part of the description included the statements that "the LB1 cranial vault is long and low." Data on LB1 cranial shape appear in table 1 of ref. 1, which gives a maximum cranial width (113 mm), length (143 mm), and cranial index of 79.1, approaching the upper limit of mesocephaly (brachycephaly > 80.0, dolichocephaly < 75.0).

Our group's direct measurements made on the LB1 cranium in 2005 show a cephalic index of 80.1 (113/141 mm), whereas the most recent measurements of maximum cranial length and maximum cranial width (2) yielded a cranial index value of 82.0 (114/139 mm). It follows, as we have long maintained, that LB1 is bracycephalic. At any time after 2004, anyone with a ruler and a copy of the original report that contained figure 1 (ref. 1, p. 1056) could have accomplished replication by measuring the length and breadth of the skull for themselves from the published photograph. Nonetheless, confusing statements about the cephalic index of the LB1 skull have continued.

2005. "In a principal component analysis, LB1 groups with *H. erectus* and is separate from the *Homo sapiens*, Sts 5 (Fig. S4), and the pygmy, based on the first principal component (weighted heavily on relative height and the disparity between maximum breadth and frontal breadth), and is separate from *H. erectus* and the microcephalic in the second principal component (weighted heavily on breadth relative to length) (Fig. 2A)" (3).

"Our data show that LB1's well-convoluted brain could not have been a miniaturized version of the brain of either *H. sapiens* or *H. erectus*." (3)

2007. "Despite LB1's having brain shape features that sort it with normal humans rather than microcephalics, other shape features and its small brain size are consistent with its assignment to a separate species." (4)

"As shown here, the frontal breadth relative to cerebellar width and lack of cerebellar protrusion of LB1's endocast classify it with 100% probability with normal *H. sapiens* rather than microcephalics. The relative length of its orbital surface also sorts LB1 with *H. sapiens* (1). On the other hand, LB1's endocast shows affinities with *Homo erectus* in its relative height, disparity between its maximum and frontal breadths, relative widths of its caudal and ventral surfaces and long, low lateral profile (1). Its tiny cranial capacity, relative brain size, and derived ventrally expanded orbital surface, however, show affinities with *Australopithecus africanus*." (4)

2013. ***Compare.*** "The LB1 cranial vault is long and low. In comparison with adult *H. erectus* (including specimens referred to as *Homo ergaster* and *Homo georgicus*) and *H. sapiens* the calvarium of LB1 is extremely small. Indices of cranial shape closely follow the pattern in *H. erectus* (Table 1)." (1)

"The initial proposal that *H. floresiensis* descended from *H. erectus* rested on craniofacial similarities—such as a low cranial vault. . ." and "the neurocranial shape of *H. floresiensis* closely resembles that of *H. erectus* s.l." (5)

Paradox. No matter how aesthetically imaged by 3D CT or "contextualized" via geometric morphometrics, the resemblances between LB1 and *H. erectus* are more verbal than dimensional, and brachycephaly remains an unusual attribute on which to claim affinity with *H. erectus.*

Resolution. Numerical data show unambiguously that the brain of LB1 is very small, its vault is low, its shape is brachycephalic, and its cerebellar region is small. All of these attributes, conflicting and confusing in any attempt to find its place among various hominin taxa, particularly *H. erectus*, are common attributes of individuals with Down syndrome (DS), who, as is the case with LB1, are found associated with other people who are unaffected, as appears to be the case in the Liang Bua Cave.

SI2: Flores Geology and Biogeography

More Ancient Ancestors, More Time for Contact and Colonization. The original time span for the evolution of a new hominin species on Flores was said to be about 1 Ma (6, 7). Taken at face value, such dates would have placed humans on Flores by the latter half of the Quaternary. To support the hypothesis that the new species represented by Liang Bua finds evolved its purportedly unique suite of morphological characteristics in isolation there, it would have been essential to have provided independent evidence (not merely to have postulated) that the island was reached in sufficient numbers to provide genetic variation adequate to establish a new species (through sufficiently intense selective pressure to transform a larger-bodied, larger-brained population, *H. erectus*, into a form more diminutive in all respects, with strangely altered craniofacial and body proportions), then to maintain it for some 40,000 generations without any augmentation of variability via gene flow, and to accomplish all this in isolation, as initially was asserted by the new species supporters. This hypothesis of island isolation now is contradicted by the contention that Flores was colonized by pre-erectus migrants (8, 9), presumably before arrival of the hypothesized *H. erectus* makers of the tools dated to about 1 Ma. These critical points, many of them made 8 y ago (10), never have been addressed by advocates of the new taxon.

In place of answers to the questions that we raised about in situ evolution of a new human species on Flores (10), others (8, 9) instead have substituted a new set of problems that seem equally intractable and at least as implausible. The first problem is that, quite simply, if the new species did not evolve in isolation on Flores from an *H. erectus* ancestor, then all of the rhetoric about island isolation logically should be dropped from further attempts to account for the paradoxical morphological pattern of the supposed new species based virtually entirely on the phenotype of the single specimen, LB1. Second, if one accepts the (unlikely) proposition that the hominins who colonized Flores originated at the phylogenetic level of australopithecines or early *Homo*, this would more than double the time span available for human populations to have reached the island. At that time, about 2 Ma, the closest humans to Flores, and the principal evidence of early *Homo* outside of Africa at about 1.7 Ma, are the Dmanisi remains in the Caucasus. Their postcranial features "include modern-human-like body proportions and lower limb morphology indicative of the capability for long-distance travel" (11), quite different from LB1 as described by Jungers and associates (12–14). Conventionally the Dmanisi fossils now are postulated to be early *Homo* (15), formally *H. erectus*, although with complex subspecies designations (15) supplanting tentatively earlier nomina such as *H. ergaster*, if not *H. georgicus* (16, 17), none of which are likely to be the earliest form of *Homo* sp. They also have cranial capacities markedly greater than LB1's

430 mL, ranging from 546 to 775 mL (15–17). Over a much longer span of time, there would have been more sea level fluctuations, some of them pronounced (18), and hence, many more possibilities for contact with and colonization of Flores from elsewhere. At this point, some qualification about the frequency, magnitude, and consequences of sea level fluctuations is in order.

Geological and Geographic Factors Affecting Human Dispersion: Lower Sea Levels and Diminishing Water Gaps. In the broader context of a 180-Ma history of sea level and ice volume changes, Miller et al. (19) noted the occurrence of a sea level peak in the early Pliocene. Subsequently, commencing about 2.55 Ma, large Northern Hemisphere ice sheets caused marked, periodic sea level fluctuations, noted previously (10), based on independent determinations (20, 21). During the last 780,000 y, these sea level changes were on the order of >100 m, paced by the ∼100,000-y eccentricity cycle (19); before that time, smaller sea level changes on the order of <60 m were paced primarily by the 41,000-y tilt cycle.

During the periods of heightened glaciation, within the chain of islands including Flores, water gaps were reduced or eliminated by lower sea levels. At glacial maxima, Bali was connected to Java but separated by the deep but narrow, <10 km, water gaps from islands to its east. The present day distance from Lombok to the little island (Nusa Penida) to the west is 20 km. Other estimates (22) (http://www.ifrao.com/the-first-mariners-project/) suggest that the Lombok-Bali distance at the lowest sea level was still about 20 km due to lower land location; uplift occurred later. Lombok Strait appears to have separated Bali from what may have been at times a contiguous land mass that united the now separate islands of Lombok, Sumbawa, Komodo, Flores, Solor, Adonara, and Lembata. Still needed is "…greater understanding of the geological evolution, especially changes in land-sea distributions and changes in the widths of marine barriers…" because "a major current debate centers on whether *Homo erectus* required the intellectual development for language and seamanship to cross from Asia to Flores by 800,000 years ago or was the dispersal of early humans affected by natural rafting or other means?" (22).

Given that the documented sea levels and water gaps (19, 23) are thought to have been challenges even for *H. erectus*, what are we to make of the speculation by Argue and others that the Flores hominins most closely related to *Homo habilis* or even australopithecines were able to make such crossings? Such speculations require the existence of as yet purely hypothetical pre-erectus ancestors of Flores hominin populations, but there is no fossil evidence for these outside of Africa, particularly in Asia; taxonomic status of the important specimens from Dmanisi (11) is a side issue. If the hypothetical Asian pre-erectus populations did exist, it is all but certain that they would have had less technological capability than later populations. However, somehow they supposedly made it to Flores when sea levels periodically were higher and gaps between islands were wider than during the subsequent period when *H. erectus* existed. Overall, an African common ancestor of the Flores hominins and *H. habilis* as required by Argue et al. (8) is a *deus ex machina* lacking independent evidence. This attempted explanation is another Flores paradox.

Multiple Contacts in the Context of Regional Continuity. Previously, Jacob et al. (10) had made the evident case for the likelihood of multiple contact and colonization events with Flores by humans emanating from larger land masses to the west and north: "Assuming that the earliest hominins reached Flores during the first intense glacial stage ∼750 ka, there could have been numerous hominin arrivals during later glacial stages with low sea levels, before the final higher sea levels at the beginning of the Holocene (10 ka) again might have constrained contacts." If the original tools on Flores more than 1 Ma or ∼840,000 y ago are taken to have represented contact and occupation by a population at the *H. erectus* level, and if it also is contended that ancestors of the new species on Flores had become reduced in brain size and body size before their arrival on Flores (therefore representing a species that had originated at an earlier, pre-erectus stage of evolution), then there had to have been more than one contact (by *H. erectus* and by the ancestors of the hypothetical pre-erectus new Flores species), which contradicts Baab and McNulty (24). One cannot have it both ways. In addition, there is ample evidence for at least one colonization of Flores event by *H. sapiens* populations at ∼40 ka, and it is far more probable that there were, as we have argued previously (10), multiple contacts by other *H. sapiens* populations within the time ranges posited for the Liang Bua Cave humans on Flores. "There are many theories about the migration of population groups into Wallacea, most of which assume two or more migration waves" (25). Recent research (22) persuasively makes the case for maritime colonization by populations conventionally designated as *H. erectus* well before 800,000 y ago. Our position also is compatible with that articulated from three decades ago (26–28), emphasizing lineage continuity and the artificiality of a species boundary between *H. erectus* and *H. sapiens*. In that context, multiple contacts among human populations on the Asian mainland, on major islands such as Java, and on the scatterings of smaller islands such as Palau and the chain that includes Flores would be seen as normal, expected, continuing occurrences rather than as unique events.

In sum, geological and biogeographical evidence provides no basis for asserting isolation of any human population on Flores sufficiently long for the evolution of a new hominin species (10). Neither is there evidence for the existence of antecedent populations exhibiting its allegedly unique features anywhere between a hypothetical origin in Africa and anywhere along any plausible route through the Sunda region, or anywhere else, for that matter. In the absence of persuasive evidence either for in situ evolution of a new hominin species on Flores or for the spread of a population with its features from elsewhere, it is essential to reexamine alternative hypotheses for the suite of features that characterize LB1, on which *H. floresiensis* is founded.

SI3: Core Hypothesis

LB1 Is Developmentally Abnormal. We make the point here that our group's evidence-based core hypothesis has remained constant throughout the duration of this controversy. Since 2004, we have held that the LB1 specimen's diagnostic characteristics represent developmental abnormality in an individual specimen of a regional *H. sapiens* population and not defining attributes of an entire new species that is posited to have evolved in isolation on Flores from a *H. erectus* ancestor or from some other unidentified hominin predecessor earlier and elsewhere.

Two of the authors (Eckhardt and Henneberg) were fortunate to have access to the Liang Bua skeletal specimens in February 2005, when we studied them with the permission of Teuku Jacob, Head, Laboratory of Bio-Paleoanthropology, Gadjah Mada University Faculty of Medicine, and Raden Soejono, Head of the Indonesian Centre for Archaeology and one of the coauthors of the original report published by *Nature* in October 2004.

As a result of our studies of the specimens in the Laboratory of Bio-Paleoanthropology, our working group submitted to *Nature* in March 2005 a very short manuscript that succinctly summarized several material errors of observation, description, and interpretation of the LB1 specimen. After consultation with Peter Brown, *Nature* declined to publish the submission. However, as a result of the review process, our unpublished manuscript and the referees' comments were made available to Michael Morwood and used without our permission or knowledge in a later book: *A New Human* (29).

The authors of *A New Human* openly describe this violation of the confidentiality that scientists assume to be part of a professionally ethical review process on pp 228 and 229 of their book: "By mid-March, Jacob and his team had submitted a brief paper to *Nature* titled 'Large Errors in the Depiction of Small Humans,' in which they correctly pointed out that the photograph of the femur describing LB1 was reversed, and the nearly complete femur was the left, rather than the right, as we described. None of their other criticisms had substance and some were misleading... They again concluded that LB1 was pathological.... One of the referees said that the paper of Jacob et al. had no real substance, that they were playing a game of 'gotcha,' and that *Nature* or any other reputable journal should be above this type of behavior. The other referee objected strongly to the way that the material had been seized before the excavation team had a chance to study it, and said that the paper should be rejected—which it was."

The effect of this rejection was to prevent, in the first months of this scientific controversy, the publication of evidence that cast doubt on the validity of the new species diagnosis. That delay was later remedied partly by our previous publication in 2006 (10) and more fully nearly a decade later. "Large errors in the depiction of small humans" now is available as originally submitted in 2005 at www.LiangBuaCave.org. Every point made by us in that paper now has been substantiated in the subsequent years by members of our research group, and many of them have been corroborated independently by other researchers.

SI4: Skeletal Signs of DS Observable in LB1 (Notes to Table 1)

Small Brain. The small cranium and enclosed brain have as a correlate cognitive reduction that occurs in 99.8% of people affected with DS. Approximately 20% of young children with DS have an occipitofrontal circumference (OFC) that is in the lower end of the normal range, but most affected individuals exhibit microcranium after midinfancy (30) as DS brain development slows with age relative to that of unaffected individuals. The extent of brain size reduction varies widely from slight to more than 6 SDs below population norms, with LB1 falling in the middle to lower end of these downward deviations from unaffected individuals. See the main text for full discussion of this point.

Brachycephaly. Brachycephaly is a common occurrence in DS. The original determination of cephalic index of LB1 (1) was 79.0% (113/143 mm). Our group's measurements produced a cephalic index of 80.1% (113/141 mm), whereas the most recent measurements of maximum cranial length and maximum cranial width (2) yielded a cranial index value of 82.0 (114/139 mm). It follows, as we have maintained since 2005, that LB1 is bracycephalic. We know of no australopithecine or early *Homo* (lower Pleistocene) that is brachycephalic. Brachycephaly also is not common among tropical modern humans.

Reportedly (2) all major cranial sutures of LB1 are obliterated, although statements are equivocal. This obliteration may indicate that LB1's skull suffered premature cranial synostosis that constrained LB1's head size at the level of a prepubertal child as is common in DS. After 3–5 mo of age, infants with DS exhibit shorter antero-posterior diameters (31) in relation to cranial width, with these proportions persisting through later life in about 75% of individuals. See the main text for fuller discussion of this point.

Atlanto-Occipital Deformity. Figure 8 from ref. 2 shows the atlas (LB1/3) positioned in front of the occipital condyles; the figure caption draws attention to "the concavities and roughened topology in their articular surfaces." Similarly, the text notes the roughened surface of the articular facet of the axis and corresponding condyle. The explanations offered by the authors posit unbalanced right and left neck muscles that are inserted on the asymmetrically deformed cranial surface, or alternatively, congenital torticollis and posterior deformational plagiocephaly. This attribution considerably confounds the complexity and directionality of outcomes in an interrelated set of developmental elements, as we explain later.

Regarding causation of atlanto-occipital deformity, much of the tentative evidence that does exist (32, 33) arises from limited clinical studies that tend to present the atlanto-axial pathologies as the more proximate or primary lesions, with torticollis, etc., as more secondary. For example, a serious form of atlanto-occipital disorder, hemiatlas, "may cause a rather severe and progressive torticollis" (34).

For more than half a century, craniocervical abnormalities in particular have been known to be among the diagnostic signs of DS (35), occurring in 10–20% of affected individuals (36–40), with consequences for potentially serious or fatal injury (41). An extensive review of the development of the atlas (42) provides a foundation for understanding the functional anatomy of this region and particularly its great variation in morphological expression (43). The generalized joint and ligamentous laxity associated with DS seems to play a role in the relatively common atlanto-axial subluxation, arguably expressed in LB1 (10). A study of congenital occipitoatlantal instability (COI) observes that the usual curved architecture of the occipital condyle that develops in normal controls over time does not occur in DS patients with COI (44).

Facial Asymmetry. Our research group (10) was, to our knowledge, the first to note and quantify the craniofacial asymmetry of LB1. These observations were denied, disputed, and discounted for several years. Some investigators (24) mismeasured the degree of palatal rotation in LB1, underestimating it by half; our observations conservatively stated at about 5° were confirmed, and exceeded, by a group working independently of us (2).

Most human faces are not perfectly symmetrical, but the extent of departure from bilateral symmetry has developmental implications and functional consequences. There is considerable discussion concerning aesthetic preferences for facial attractiveness, with many recent studies involving subjects across cultures and of varying ages suggesting partiality for faces that are average and regular, i.e., without unusual asymmetry (45), with preferences for symmetry possibly underlying preferences for averageness, because average faces are more symmetric than other faces (46) However, beyond aesthetic perceptions or preferences "...In the clinic gross degrees of asymmetric development of paired body parts are likely to present as an abnormality begging for causal and pathogenic explanations" (47). Thus, as with brain size and stature, facial asymmetry beyond the normal range is a general sign of developmental disorder (48), of which DS is one of the most serious common examples in living populations. It recently was demonstrated that DS individuals show more fluctuating asymmetry of facial prominences than their euploid siblings and that there is a clinical increase in DS facial developmental instability from the frontal prominence to the mandibular prominence (49). See the main text for full discussion of this point.

Small or Missing Skull Sinuses. For LB1, figure 2 of ref. 1 showing a midsagittal CT view of the cranium and mandible is uninformative on the extent of cranial and facial sinus development. Figure 2 in a later publication (50) with an antero-posterior CT section through the frontal-facial region shows a tiny right frontal sinus. The postmortem damage to the frontal, reaching deep into the supraorbital region of the frontal bone, did not expose any frontal sinuses; the entire area of the exposed subcortical bone is taken up by trabeculae of diploë. Figure 2 of ref. 50 otherwise is unclear and uninformative, providing relatively little more in the way of illustratively or descriptively useful information on sinus development in LB1. However, in a section titled "Infraorbital

and Paranasal Surfaces," the authors note that "The infraorbital surface of LB1 is extremely short vertically." Maxillary sinuses do not appear to have been illustrated for LB1 despite the large numbers of CT scans published in various papers, but such illustrations that do exist, e.g., figure 2 in ref. 1 and figures 1 and 2 in ref. 50, present sunken midfacial (infraorbital) regions flanking the piriform aperture; these seem to leave little space for much maxillary sinus development.

All of the above observations and inferences support the hypothesis of DS in LB1. Among the many serious health challenges faced by individuals with DS is chronic nasal drainage or rhinorrhea, which is a common clinical finding in children with DS (51). The underlying cause is deficient growth of the facial skeleton. Radiological studies (52) on individuals with DS document abnormal development in the frontal, maxillary, and sphenoid sinuses, including hypoplasia and nonpneumatization of the paranasal sinuses.

As in the case of the reduced brain size in DS adults, noted above and in the main text of this paper, virtually all regions of the DS syndrome skull are deficient in growth (53, 54), with the greatest effects observed in the structures that would be expected to show the most marked development after birth (ethmoid, nasals, mandible); hypoplastic or even absent paranasal sinuses also may contribute to midfacial hypoplasia. Beneath the modestly salient frontal bone, the underdeveloped maxillae of LB1 are primary contributors to the reduced midfacial skeleton situated superior to a disproportionately underdeveloped mandible (see information below on microgenia). Notably, the developmentally based progression of relative diminution from forehead through midface to mandible parallels the increased frequency of fluctuating asymmetry (FA) from the frontal eminence through the maxillary and median nasal prominences to the mandibular prominence already noted (49), and the parallel pattern of increasing asymmetry along the same superior to inferior clinal lines documented in LB1 (10, 55).

In normal human development, most of the sinuses begin to appear during the fourth and fifth months of fetal life, with the sphenoid sinus emerging after birth in the anterior region of the sphenoid and gradually extending posteriorly at a time when the maxillary sinuses usually are represented by a furrow in the nasal wall. DS skulls commonly show deficient development of the sphenoid, frontal, and maxillary sinuses. In a radiographic study of 29 DS skulls spanning an age range of 8–49 y, frontal sinuses were markedly deficient in development for 24 patients (83%), with the sphenoid sinuses being considerably reduced in size in 66% of patients and all of the other sinuses being underdeveloped (53).

Microgenia (Micrognathia). LB1 formally exhibits microgenia or micrognathia, an unusually small chin. However, this feature needs to be considered in the context that there exist at least three alternative explanations for the appearance of microgenia in this specimen: (*i*) as one of the supposedly primitive features of a totally new human species (1); (*ii*) as a regional feature common in Australomelanesian populations (10); and (*iii*) as a developmental consequence of DS affecting the morphology of LB1 (55). The second alternative has been confirmed objectively as true (56), and hence casts extreme doubt on the first alternative as, at best, an unnecessary speculation. Plainly, the normal mandibular phenotypes observable in Australomelanesian populations (Flores, Palau, and elsewhere) are known entities, whereas advocacy of the same features as unique to a hypothetical new species is not only objectively false—as with a great many of the unique features of LB1, they are variable in expression and frequency, thus at best unusual only individually or in combination—but also is the ultimate philosophical resort to an unknown entity. Just as clearly, the second and third alternatives are not mutually exclusive. The mandibles of LB1 and LB6, often said to be identical, quite evidently are not, by simple visual inspection (shown in figures 1–4 in ref. 57). This contrast is most readily apparent in ramus height, which is much shorter in LB6; in addition, the symphyseal region in LB6 is flatter and more nearly inclined toward the vertical than the more bulbous and receding anterior contour of LB1.

Pertaining to the second alternative, a mandible with a reduced bony chin is a regional characteristic found widely in normal members of Australomelanesian populations (10, 56, 58), a point that is ignored or denied consistently by advocates of the new species hypothesis; the extreme form of this misrepresentation is in figure 19 and its legend in ref. 57, which purports to disprove our documentation of the existence of a reduced (neutral or negative) chin in some Australomelanesians (frequencies of which obviously vary from population to population in the region) by showing a lateral radiograph on one unidentified Australomelanesian with a projecting bony mental tuberosity supposedly within a facial phenotype that has the "appearance of a receding chin given by external soft tissue." The argument fails logically, because other than in a typological framework, any one specimen cannot disprove by proxy the existence of traits found in individuals among multiple populations in an entire geographic region. Moreover, there is reason to be skeptical of this particular example because the soft tissue profile is so tenuous that Brown and Maeda needed to delineate its contours with a line that is drawn in, obscuring if not altering the appearance of the soft tissue itself. Even if we do not reject the augmented evidence provided by this single unidentified specimen, it is obvious logically that the existence of any individual Australomelanesian with some bony chin projection covered by soft tissue that gives it a receding appearance cannot establish the generality of that hypothetical or actual anatomical conformation. Among 76 adult Rampasasa, 93.4% exhibited neutral or negative chins externally, on the basis of their soft tissue configuration. In the entire sample, only eight subjects (15.35%) showed a positive bony chin that appeared neutral on the basis of its soft tissue covering, and no subject exhibited a negative soft tissue chin that concealed a salient underlying bony chin (56).

Independently supporting the observation that results showing that reduced or absent bony chins among the Rampasasa (56) do not stand in isolation are the mandibles recovered on Palau (58, 59). Two mandibular fragments (B:OR-14:8-122 and B:OR-14:8-771) are from recent adult modern humans (59). As noted by the authors of the Palau study, the former specimen lacks a vertical keel, distended inferior margin, T-shaped keel, T-shaped mental trigone, and associated mental fossae. The less complete second specimen lacks part of the symphyseal region but nonetheless also exhibits a highly reduced mental eminence.

With respect to the third alternative for a reduced chin structure, as shown in Table 1, microgenia is a feature that forms part of the facies commonly seen in individuals with DS regardless of the regional population from which they are derived. It should be noted, however, that although some "ethnic" (i.e., populational or regional) variations are known (60, 61), the facial morphology of DS shows some constant features (62–67), as is true also for other serious developmental disorders such as cleft lip and palate (61). In general, the extensive biomedical literature on abnormal variation presents an entirely different perspective from the typological view widely accepted in paleoanthropology. This point is important because, although perspectives may vary, there is no rational basis for believing anything other than that there is only one set of evolutionary developmental principles that apply to hominin populations past and present. Because there is no extensive biomedical literature on the occurrence of DS in Australomelanesian populations, and also recognizing that the differences of the LB1 mandible from that of LB6 combine a further degree of symphyseal reduction with greater evidence for tooth loss and periodontal disease, in

this regard, the LB1 mandible seems broadly consistent with a diagnosis of DS.

Taurodontism. Others (68) have proposed the presence in LB1 of "significant taurodontism; that is, the presence of an enlarged tooth body due to an apical displacement of the root bifurcation, is evident in these and other LB1 teeth." The teeth referred to are identified by Brown and colleagues as first lower premolars (P_3s), which Obendorf's group (68) hypothesize are retained first lower deciduous molars (dm_1s). Though there is no compelling basis to accept the interpretation of LB1 lower premolars as retained deciduous molars, such an interpretation is a possibility. Irrespective of this interpretation, we regard the presence of some degree of taurodontism in LB1 as sustainable. Its presence is, however, uncertain due to the poor quality of evidence that continues to be provided by Peter Brown and his supporters. The basis for our position here comprises the ambiguous images offered only on a website (http://pandora.nla.gov.au/pan/10345/20080516-0014/www-personal.une.edu.au/_pbrown3/Henneberg%20hobbit%20claim.html). The caption for one of these images (to which it is difficult to refer because no figure numbers are provided) states: "Cross section through the centre of the left side and right side LB1 teeth (based on CT scan data). Dark patch in the centre of the tooth crowns is the pulp cavity." In that image, what, by location, should be the left secondary maxillary premolar (P^4) shows a tooth body that appears deep, with roots that seem to divide relatively apically. We use the qualifiers "appears" and "seems" because the LB1 second upper premolars are known to be rotated ~90° in the tooth row. This rotation is another of the allegedly unique characters of a most unusual new species—but rotated teeth occur routinely in low frequencies in human populations, including the Rampasasa (10), so what appears in the image to be roots somewhat apically divided might possibly be a misleading double image due to overlap of the roots in a buccolingual plane. Until less equivocal evidence is provided by Brown and colleagues, reasonable uncertainty must remain. The next image also is given with no figure number but begins with the caption "3-D rendering of LB1 CT scan data (1 mm slice intervals), with exposed section through the left molars." There, the left lower third molar shows no apparent root division. A classic paper on taurodontism (69) notes "it may be stated categorically that the first molar in any molar series tends less than any of the other molars to the condition of taurodontism and that the second and third molars tend more to the condition of taurodontism." The poorly rendered image provided by Brown on his website is indeterminate, but nonetheless is consistent with the sequential progression described (69) for taurodontism. The CT images published elsewhere, especially figures 7*B* and 8 in ref. 70, document taurodontic appearance of left lower molars of LB1 in the sense that roots of the LB1 teeth are short, whereas the crowns are of substantial height in relation to the roots. DS patients have teeth with short roots (71, 72). We can make no more precise determinations than the ones provided here. Given the length of time that the Liang Bua Cave specimens have been available to Peter Brown and colleagues for study and the material resources available to them, there remains a paucity of reliable data that have been provided openly and objectively. Accurate assessment of the occurrence of taurodontism in LB1 and LB6 should be possible radiographically with little cost or effort.

Taurodontism, as already noted above, describes teeth with enlarged pulp chambers, apical displacement of the pulp cavity floor, and little or no constriction at the cementoenamel junction. In the first quantitative study of taurodontism (69), three discontinuous categories (hypotaurodontism, mesotaurodontism, and hypertaurodontism) were established. A subsequent paper (73) devised a nonarbitrary measurement system in which the height of the pulp chamber is expressed as a percentage of overall tooth height below the cementoenamel junction. The alteration in tooth morphology responsible for taurodontism is attributed (74) to failure of Hertwig's epithelial sheath diaphragm to invaginate at the usual horizontal level. Although usually considered to be a developmental abnormality, taurodontism also can have the functional effect of increasing the useful life of teeth that are subjected to heavily abrasive wear caused by a coarse diet; this factor could account for high frequencies of taurodontism in populations as geographically and genetically diverse as Eskimos and Australian Aborigines (75, 76).

The first descriptions of these variant tooth morphologies were made by Gorjanovic-Kramberger in 1908 (77), who reported relatively high frequencies of taurodontism in neandertal primary and secondary premolars and molars. Taurodont tooth frequencies that may signal differential adaptive responses in normal populations tend to range in the low single digits; for example, taurodont teeth occur in primary dentitions of Japanese children at 0.54%, whereas they occur in the permanent dentition of Israelis at 5.6% (78). In cases of disrupted development, taurodont frequencies are one or two orders of magnitude higher (79). One study (80) observed taurodontism in 12 of 33 (36.4%) DS extracted lower molars, and frequencies can reach 60% of DS patients. Supporting the inference that, in DS, taurodontism signals individual developmental disorder rather than exclusively a population-level selective response to heavy tooth wear, is the observation that taurodontism occurred in 40% of non-DS twins with cleft palate; the common element in cleft palate and DS is serious disruption of normal developmental processes, although the causes of cleft palate and DS are independent (81). Further, among 12 patients showing various combinations of X chromosomal aneuploidy, 11 had taurodont molars; the inference was that these extreme variants of pulp chamber shape signal a generalized amplified instability of development. Taurodontism is a dental anomaly commonly reflecting disrupted developmental homeostasis (82).

Taurodontism has not been found in dentitions of normal Australomelanesians, despite thorough study of those dentitions nearly a century ago (83). Thus, any presence of taurodontism in LB1 in the small sample from Liang Bua comprising one maxillary and two mandibular dentitions is more likely to be attributable to disrupted development than nonpathological regional variation.

Short Stature. See main text for full discussion.

Short Femora. See main text for full discussion.

Flat Feet. Elements of the LB1 bony foot have been described at length (14), with a subsequent paper (13) repeating some of the same data with a clearer statement on the existence in LB1 of flat feet and the occurrence of osteophytes. Flat foot, *pes planus*, is seen in a substantial majority of people with DS; resultant abnormal gaits commonly lead to the development of other movement problems.

Flaring Ilia. The LB1 pelvic girdle initially was described (1) as "represented by a right innominate [sic; = os coxae] with damage to the iliac crest and pubic region, and fragments of the sacrum and left innominate. The right innominate, which is undistorted, has a broad greater sciatic notch suggesting that LB1 is female… the iliac blade is relatively short and wide; however the ischial spine is not particularly pronounced. Compared with modern humans the LB1 ilium has marked lateral flare, and the blade would have projected more laterally from the body, relative to the plane of the acetabulum." A later study (14) concurred with the description made by Brown et al. (1) and elaborated further, stating that "if one articulates casts of the sacra of australopithecines such as AL 288-1 ("Lucy") or STS 14 (or a sacrum from a *very* small human [their emphasis]) with LB1/7 to establish anatomical planes,…the iliac blade flares strongly beyond

the margin of the acetabulum. . .This degree of iliac flaring is not a function of the sacrum with which it is paired; rather, it is intrinsic to the os coxae and resembles that seen in australopithecines. . .but not in modern humans." That particular inference, as with the overall assessment of LB1, is limited in its accuracy by the failure to give more than cursory consideration to possible developmental abnormalities.

For more than a decade, radiographic diagnosis of DS in infants has been based in part on anteroposterior radiographs. Pelvic bone abnormalities including widened, flared iliac wings with increased outward curvature have been found to be present in up to 80% of newborn infants with DS (84–87). Iliac wings in patients with DS were more divergent and tended to be oriented in a more coronal plane, as well as shorter than iliac wings in normal controls (88). The more dorsal angulation did not seem to result from outward rotation of the sacroiliac joint but rather was due to the intrinsic curvature of the iliac wings. Paralleling the close correspondence between the degree of flaring in LB1 and DS patients is the overall reduced size of the pelvis. Recall here the implied match of the LB1 pelvis with "a sacrum from a *very* [their emphasis] small human" quoted above (14). "In a radiological study of the pelvis in 66 adult subjects with Down's syndrome, 25 (38%) were found to have the classical pelvic shape associated with Down's syndrome in infancy and childhood, with the appearance of the pelvis and hip joints being highly variable, matching the variety of appearances seen in normal subjects, '*except that generally the pelvis was smaller than normal*' [our emphasis]" (88).

Short Digits. Some dimensions of manual and pedal phalanges have been published (14, 89, 90). In general, taking into account partial damage of some LB bones and uncertainties of their allocation to specific rays, dimensions of these bones seem smaller than the averages provided as a standard for people of European origin (90). This small size is especially true for the distal pollical and nonpollical phalanges [15.7 vs. 21.36 (−3.55 SDs) and 13.4 vs. 17.15 mm (−2.60 SDs)] for left hands of European females. No specific standards for Indonesians were available to us.

Periodontitis Plus Low Incidence of Caries. Periodontitis is very obvious in LB1 and described as such by the original finders (1). There is no periodontitis in LB6 (57). According to those granted access to the specimens (57, 70), LB1 is said to have had no caries at all. We have disputed it (91), but the qualified inferences we can make from published photographs indicate that the carious lesions would have been limited, affecting at most three teeth in the total known Liang Bua Cave sample comprising one maxilla and two mandibles.

In further keeping with DS and predisposing to periodontitis, the front teeth in the LB1 mandible are irregularly placed. The LB1 mandible is also characterized by two or three missing teeth and much greater irregularity in the placement of the remaining teeth. In LB1, the second premolars (P_4s) on both sides are missing. On the left, the alveolus is still open, implying that loss of the tooth might have occurred a short time before death or after death. On the right, the alveolar bone is completely healed, whereas there is interproximal wear on the flanking teeth (57). This combination of healed alveolus and interproximal wear means that the tooth in the position of P_2 was present for some time during the adult life of LB1, and then it was lost at a time before death long enough for the alveolus to heal completely. It has been suggested (68) that the tooth present in this position was a deciduous second molar that was retained for quite some time into adulthood, whereas the true adult P_2 was agenetic. An alternative interpretation is that the right lower second premolar became carious and was extracted or was lost some (substantial) time before death. As is well known, both first premolars in the LB1 mandible had morphologically unusual (but by no means unique) enlarged crowns and Tomes' roots (10).

Plagiocephaly. In some papers (92, 93), the term plagiocephaly has been used more or less interchangeably as both description and diagnosis, with insufficient discrimination between cause and effect. Such treatment is at substantial variance with the biomedical literature. Plagiocephaly is a broad term for cranial asymmetry. The condition can result from premature fusion of one or more cranial sutures (synostotic plagiocephaly) or may be due to an infant's skull being shaped by external forces (intrauterine constraint, twinning, or resting position), with this condition constituting what is called "deformational plagiocephaly" (DP); also associated with DP are isolated torticollis, hypotonia, and cervical spine abnormalities. "Positional plagiocephaly" is a more specific term used by pediatricians and other physicians to denote the effect of external forces related to positioning of the infant by caregivers during sleep or other activities (94). Failure to distinguish clearly among the various particular meanings of plagiocephaly (92, 93) leads inevitably to confusion among developmental causalities and consequences, which range across a spectrum from relatively mild to quite serious pathological consequences.

The common effect of the externally applied force (e.g., sleeping position in modern infants) is to produce a skull, which, when viewed from above, presents a rhomboid or parallelogram appearance that may extend to forward displacement of the ear on one side of the head of a living infant, corresponding to the location of the external auditory meatus on the skull. This shape is characterized by flattening on one side of the back of the head and a noticeably rounder shape on the other side.

In one description of LB1 (92), it is noted that "viewed from above or below, LB1's cranium shows 'parallelogram' skewing with distinct left occipital flattening and a slight anterior shift of the left face, as exemplified by the asymmetric dispositions of the mandibular fossae, external ear canals, temporal fossae, and anterior malar surfaces (Fig. 1 B-D)." However, comparison of that figure with their figure 4 (deformed head of a modern infant with PDP) shows that, despite the LB1 skull having multiple other objectively determined asymmetries (facial asymmetry, palatal rotation, etc., as detailed elsewhere), displacement of the external auditory meati is relatively slight; also see table 2 of ref. 92.

As far as implications, the insouiciant conviction by one paleoanthropological group about the benignity of plagiocephaly is not shared by biomedical scientists (94), who note "We do not yet understand nor have a sound basis for making hypotheses about the specific mechanisms that link DP with neurobehavioral development. The direction of basic causal pathways is yet to be even tentatively established (i.e., do motor and other neurodevelopmental deficits lead to or follow from skull deformation?). Although there is the basis for hypothesizing a positive linear relation between plagiocephaly and neurobehavioral outcome, hypotheses regarding how or why the two may be linked are extremely tentative."

As we have noted previously (95), the label plagiocephaly simply cannot be used as a catch-all cover for the panoply of pathologies that now are admitted, albeit hesitatingly and reluctantly by many paleoanthropologists, to be present in the LB1 specimen. The approach of Kaifu's group—suggesting environmentally induced plagiocephaly in place of more fundamental developmental disordering—requires investigators to ignore the fact that some undisputed abnormalities in LB1 are postcranial, as in the flat feet marked by osteophytes and the strikingly short femora (13). Even aside from its microcephalic brain and craniofacial asymmetry, the extent of pathologies documented in LB1—torticollis, atlanto-occipital abnormality, reversed petalia, flat feet with osteophytes, etc.—with or without any diagnosis of a specific syndrome, should disqualify LB1 as a holotype of a new hominin species. Regardless of taxonomic challenges, conceding the existence of multiple anomalies but maintaining that they may not have a common causal element is a violation of the Occam's Razor principle.

A case of nonsynostotic deformational plagiocephaly involving intentional cranial deformation was described from the Early Feudal Period in Armenia (96). Although the plagiocephaly in the early Armenian skull was severe, involving asymmetry of the face and the skull base, unlike the case in LB1, the asymmetrical atlas (figure 4 in ref. 96) does not appear to involve condylar surface flattening or irregularity (which also were not mentioned by the author).

Hypothyroidism. Thyroid gland dysfunction is common in DS, with nearly a third of DS patients (97) or more as adults (98) exhibiting hypothyroidism. In a series of papers, Obendorf and colleagues (68, 99, 100) hypothesized that LB1 and LB6 may be myxoedematous endemic cretins, with many signs of hypothyroidism. The diagnosis of cretinism has been disputed on several grounds (50), with many of Brown's arguments being based on a highly selective presentation of pertinent data. One example of this is "...the brain volume of LB1 has been estimated to range between 380 and 417 ml (Brown et al., 2004; Falk et al., 2005a; Holloway et al., 2006)." The 380-mL volume estimate was that of Brown et al. (1), to which no subsequent estimate ever came close; not cited at all was our higher estimate (10), which recently was confirmed to within less than 1% (101). A key part of the argument by Brown (50) was that, due to planar photographic distortion, Obendorf et al. (68) had mismeasured the fossa for the pituitary (a primary target for thyroid hormone). This statement is all but impossible to test because basic data provided by Brown cannot be credited due to the refusal to make specimens available for independent replication. On other grounds, attribution of hypothyroidism in DS cannot be refuted by measurements on the sella turcica, the skeletal seat of the pituitary gland that influences thyroid function, because in DS, the sella turcica is so highly variable, ranging from virtually normal through deviations of the anterior wall, or notch in the anterior wall, or cleft in the sella floor (102). That is, in DS, thyroid gland dysfunction may be present but not have a uniform correlate in pituitary structure. It is our belief that the meritorious core arguments by Obendorf and colleagues concern hypothyroidism and its effects on development, which is abnormal in LB1. It is, of course, possible that an individual with DS also was exposed to a low iodine concentration in their diet. Many DS patients do have congenital hypothyroidism; coupled with DS muscle hypotonia that is very common in DS, the effect is likely to produce the postcranial skeletal signs as described (99).

An ancillary observation (102) is of particular interest. In many cephalometric measurements, the sella location constitutes an important reference point for assessing cranial base angle, which is greater in patients with DS. Brown et al. (1, p 1056) noted that "The cranial base angle (basion-sella-foramen caecum) of 130° is relatively flexed in comparison with both *H. sapiens* (mean 137°–138° (35, 36), but a substantial scatter (1SD = 7.43°)—see Ross and Henneberg 1995, Ross et al. 2004 and Indonesian *H.erectus*. ... Other small-brained hominins, for instance STS 5 *Australopithecus africanus*, have the primitive less-flexed condition." The comparative data used by Brown et al. (1) seem oddly chosen. Even allowing for some variations in measurement of cranial base angles, a wide range of hominins, including some small brained ones, actually have cranial base angles (CBAs) very similar to those of modern humans (see tables 4 and 5 and figures 3, 6, and 7 in ref. 103), with all specimens measured using the same methods: 93 *H. sapiens* = 111.8°; STS5 = 114°, MLD37/38 = 110.5°, Kabwe = 128°). Other researchers (40), using a slightly different measurement protocol (defined as sella-nasion/basion-sella), determined the CBA to be 129.92° for 25 non-DS subjects and 140.31° for 25 DS patients. Thus, the cranial base morphology would be yet another impediment to the already implausible attempt to derive *H. floresiensis* specifically from an early pre-erectus African ancestor. In contrast, the cranial base angle of LB1 is compatible with a diagnosis of DS.

Anomalous Wrists. Several wrist bones of LB1 (capitate, scaphoid, and trapezoid, plus portions of the lunate and hamate, all from the left side) have been extensively described, if not always clearly (89, 104, 105). These descriptions have been treated in comparative context with bones of other hominoid primates, including some present and past hominins. More recently, several additional wrist bones (106, 107) have been described: LB20, a "mostly intact" right capitate, and LB 21 and LB22, left and right partial hamates. Orr and colleagues (106, 107) suggested that, based on their association with other remains assigned to LB6, these all represent parts of the same individual.

We cannot undertake an extended review of these studies in this paper, but a main point that must be made is that the comparisons repeatedly juxtapose images of individual specimens (e.g., LB1) with single images representing entire taxa (e.g., *Pan troglodytes* and *H. sapiens*). This approach obviously forces comparison of some individual specimens with idealized or composite types for different taxa. The fact that there is no visual indication of the extent of within taxon variation (normal or abnormal) thereby renders the exercises inherently typological and unconvincing. Added to this is the slightly varied but generally consistent failure to group the carpal bones of LB1 with any particular earlier hominin taxon; rather, the impression given is that the wrist bones of LB1 show primitive features. However, these traits are far more likely to convey information about atavistic development than about phylogenetic affinity.

In connection with our exploration of DS as a strong fit for the known skeletal characteristics of LB1, the extent to which the carpals of the LB20/LB21/LB22/LB6 hypothetical composite specimen do or do not duplicate the features of the LB1 carpals is a moot point and rather diagnostically uninformative. "Radiographic changes in the hands of patients with Down's syndrome are not specific. Pseudoepiphysis of the base of the second metacarpal is common. The little finger is short and curved with its tip directed toward the thumb (clinodactyly). This configuration is due principally to a short, and frequently wedge-shaped, middle phalanx. Clinodactyly occurs in many congenital disorders, particularly those associated with mental retardation, including other trisomies. ... Skeletal maturation may be accelerated, normal, or retarded" (108).

Arthropathy is common in DS patients, accompanying joint laxity in the hands, and may influence ulnar deviation, possibly influencing wrist bones from childhood; there also may be premature ossification of wrist bones with associated crowding and possible shape changes (109, 110).

Small Cerebellum. Vannucci et al. (111) found that two craniometric ratios used by Falk and colleagues (4) distinguished living microcephalics from normocephalic subjects, yet neither of those ratios was cited by Falk et al. (4). The reanalysis (111) extracted the pertinent values and showed that cerebellar protrusion of LB1 is outside the normocephalic range for a sample of 13- to 18-y-old subjects. Comparison of LB1 with the microcephalic endocast sample showed that cerebellar protrusion of LB1 was consistently within the microcephalic range, whereas the LB1 frontal breadth was near the upper limits of the microcephalic range due to a combination of wider frontal breadths and narrower cerebellar widths. As a result, LB1 expresses marked brachycephaly and cerebellar hypoplasia.

Aylward, et al. (112) demonstrated that 40 DS individuals had significantly smaller cerebellar volumes than matched controls, even after adjusting for total brain volume or total intracranial volume. Cerebellar volumes for DS subjects were 73% of normal controls, whereas brain volume was 85% of normal and intracranial volume was 87% of normal. There was no correlation of cerebellar volume with age. These findings confirm similar results obtained in earlier smaller-scale studies.

SI5: Developmental Genetics of DS

Among the 1 in every 691 live births that result in DS (113), there is wide variation in frequency among populations (114), from about 1/300 to 1/1,000 births, which themselves represent the marked reduction in survival from 0.45% of all conceptions (115). In 95% of cases, the DS phenotype arises from trisomy of the entire chromosome 21, whereas another 3–4% of cases are caused by an unbalanced translocation of a portion of chromosome 21 to another acrocentric chromosome (partial trisomy 21). About 25% of the unbalanced translocations are familial, with complex consequences for recurrence risk estimation and potential for false confirmation of the new species interpretation in the unlikely event that another individual specimen similar to LB1 is recovered from Liang Bua Cave. About 1% or 2% of DS phenotypes result from mosaicism (extra copies of chromosome 21 in only some cells). Less commonly, DS phenotypes result from partial monosomy of chromosome 21 (116).

On chromosome 21, the first region, from the centromere to ~31.2 Mb, produces a severe phenotype. This region covers the gene-poor section of HSA21, which contains ~50 genes. There are no cases with a deletion spanning the second region from 31.2 to 36 Mb, but a partial deletion produced a severe phenotype, indicating that this region, with a high gene density (~80 genes), contains a combination of loci that may not be tolerated in a monosomic state. The third region, from ~36 or 37.5 Mb to the telomere, contains a large number of genes, ~130, but its monosomy results in a milder phenotype (109). A 3.8- to 6.5-Mb region on 21q21.22 including about 30 genes has been identified as a DS critical region, although the genes included in it may not account for all DS phenotypes (117). Genetic polymorphisms in both Hsa21 and non-Hsa21 genes in combination with environmental influences account for the wide phenotypic variation of DS individuals (115). In sum, DS is genomically heterogeneous, with this underlying situation reflected through developmental processes into wide phenotypic diversity among individuals with DS.

SI6: Comparative Anthropometric Data on DS

For comparison with DS subjects, we used anthropometric data on adult North Americans reported by the Civilian American and European Surface Anthropometry Resource (CAESAR) (118). Measurements for this project were taken according to standard anthropometric and International Organization for Standardization procedures, whereas sampling ensured that they were representative for the entire population. Statistical parameters of distributions were reported separately for males and females and for each body side where appropriate. To make these data comparable with those of DS subjects (119), we averaged male and female means assuming equal numbers of both sexes and also averaged means for left and right sides of the body. Obtaining SDs corresponding to such averaged means would require complex calculations based on additional assumptions. We chose not to do that, and for comparison of averages of DS subjects (119) with CAESAR averaged data, we calculated simple ratios of DS averages to CAESAR averages (Tables S4 and S5).

In Table S5, male and female CAESAR means for both sides of the body (where bilaterally measured) were averaged, assuming equal representation of both sexes. Means comparable with trunk length and head and neck height measurements of DS patients were not directly reported in CAESAR. To obtain trunk length from CAESAR data, we subtracted mean trochanteric height from mean acromial height, whereas to obtain head and neck height, we subtracted acromial height from stature. In selecting these CAESAR means, we tried to be as close as possible to the measurement technique on DS subjects, in which study investigators used acromion and trochanterion to measure their DS patients (119). Comparison of DS patient averages with corresponding CAESAR averages shows that DS adults present a consistent set of morphological deviations from unaffected Americans: reduced stature, smaller head height, shorter limb segments, and longer trunks. Precise values of ratios may be affected by some discrepancies in measurement techniques, but the overall picture is clear and in agreement with other metric comparisons of DS patients to unaffected people.

SI7: Families with Multiple Members Exhibiting Down Syndrome Signs

For decades there have been families known to have multiple offspring presenting DS signs. In some cases, the occurrence of several affected individuals in a family can be due to a mother herself having a mild form of the syndrome and giving birth to several affected offspring (120). One family with a history of DS included a mother exhibiting a moderate intellectual disability, multiple spontaneous abortions, and an apparently balanced pericentric inversion of chromosome 21 (121). More complex situations abound. Another family had two children displaying some features of DS despite having apparently normal karyotypes, with a third child being cognitively impaired and having complete monosomy G; the mother had a deletion of one of the long arms of her G chromosomes (122). Ballantyne et al. (123) described a woman possessing a G group chromosome 21 with deleted short arms, who transmitted this chromosome to a cognitively impaired son plus a daughter with DS, as well as also having a chromosomally and phenotypically normal son and daughter. Three Kuwaiti families each had three sibs exhibiting regular trisomy 21, and nine families each had two or more sibs with trisomy 21 (124); the incidence of high levels of consanguinity (54.3% in Kuwait) is discussed as a potential but ill-understood contributing factor, as well might be the case for the Liang Bua Cave sample.

We note these examples, among many more that could be included, as pertinent to two statements commonly made about the Liang Bua Cave skeletons. The first is that discovery of one more skull could resolve the competing hypotheses of new species vs. abnormal development. In fact, logically this is far from the case. Given the numerous documented cases of familial DS, it is possible that additional skulls exhibiting signs similar (not necessarily identical) to LB1 could represent an example of this sort (note in this regard that the LB6 mandible, although similar in size to that of LB1, is not identical in its development or morphology). The second misconception—misrepresentation, really—is that our group's hypothesis holds that the Liang Bua Cave skeletons represent a population of abnormal people. Nowhere have we posited this, preferring to focus on the plain abnormalities of LB1 alone. The remaining specimens are too fragmentary to make a definitive determination possible at this point; in some cases, it is difficult to ascertain whether one is dealing with damage, injury, or some individual developmental detail. Selective publication of information by the supporters of *H. floresiensis* makes this a moot point. Multiple abnormal individuals in the same cave are neither highly likely nor make it possible to rule out without independent replication that thus far has been blocked by those who now control access to the specimens.

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Table S1. Ratios of right and left distances from the midline

Our measurement*	R/L% (absolute value)	Corresponding region (48)
Orbital lateral rim distance	6.90	Frontal
Infraorbital foramen distance	4.46	Maxillary
Piriform aperture breadth	8.33	Medial and lateral nasal
Maxilloalveolar breadth	8.53	Maxillary
Maxillary bicanine breadth	29.31	Maxillary
Average of IFD,MAB, MBB above	14.10	Maxillary
Mental foramen distance	39.73	Mandibular

*From ref. 10, text below figure S7. If one places our measurements in strict order, IFD is superior to PAB, corresponding in even closer detail to the cline observed by Starbuck et al. (48).

Table S2. Stature reconstructions of LB1 using different formulae

Formula	Reconstructed stature (mm)
365 + 2.98 (femur length)*	1,199
520 + 3.08 (tibia length)*	1,244
1.22 (femur length + tibia length) + 70.37†	1,332
2.15 (femur length) + 725.7†	1,328
2.39 (tibia length) + 814.5†	1,376

*Javanese females (125).
†"Mongoloid," i.e., US military personnel of Asian ancestry (126).

Table S3. LB1 stature (in meters) estimated from different skeletal elements using various formulae for males and females

LB1 elements	Length (mm)	Male (Europeans) (1)	Female (Europeans) (1)	Male (Asian) (2)	Female (White) (2)	Unisex (Aboriginal Australians) (3)
Humerus	234	1.40	1.43	1.49	1.40	1.28
Ulna	205	1.45	1.46	1.49	1.45	—
Femur	280	1.27	1.29	1.33	1.24*	1.06
Tibia	235	1.33	1.34	1.27*	1.06	—
Fibula	225	1.28	1.28	1.35	1.26	—
Ratio of average stature estimate from upper limb elements to that from lower limb elements		1.10	1.11	1.11	1.13	1.20

*These two estimates from ref. 4.

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Table S4. Comparisons of body dimensions of DS patients (119) with those of adult Americans (CAESAR averages) (118)

Dimension	DS mean	DS SD	CAESAR (US) sex and side averaged	DS/CAESAR
Stature	1.53	0.09	1.70	0.90
Head and neck	26.34	2.98	30.27	0.86
Trunk	56.37	3.64	49.10	1.15
Thigh	35.58	2.70	42.44	0.84
Shank	33.24	3.45	38.92	0.85
Foot	21.74	1.87	25.29	0.86
Arm	25.37	2.41	31.34	0.81
Forearm	21.50	1.97	25.13	0.86

Stature in meters; all other dimensions in centimeters as originally reported.

Table S5. Proportions of body element dimensions for DS patients and unaffected individuals (119) and DS/unaffected ratios

Proportion	DS patients	Unaffected persons	DS/ unaffected
Thigh/stature	0.233	0.240	0.97
Shank/ stature	0.217	0.219	0.99
Arm*/stature	0.166	0.178	0.93
Foot/thigh	0.611	0.583	1.05

*Length of humerus.