Evolutionary Anthropology 15:65–77 (2006)

ARTICLES

Human and NonHuman Primate Brains: Are They Allometrically Scaled Versions of the Same Design?

JAMES K. RILLING

Allometric analyses of brain structure sizes across the primate order demonstrate that human, ape, and other anthropoid brains are not simply allometrically scaled versions of the same generalized design. Both human and ape brains exhibit specializations with respect to other anthropoid brains. Ape specializations include elaboration of the cerebellum (all apes) and frontal lobes (great apes only), and probably connectivity between them. Human brain specializations include an overall larger proportion of neocortex, with disproportionate enlargement of prefrontal and temporal association cortices; an apparent increase in cerebellar connections with cerebral cortical association areas involved in cognition; and a probable augmentation of intracortical connectivity in prefrontal cortex.

Many anthropologists are interested in understanding the source of cognitive and behavioral differences between humans and nonhuman primates on one hand, and between apes and other primates on the other. Why is it that apes, as compared with other primates, excel at certain aspects of social cognition, such as self-awareness,[1] components of theory of mind,[2] and capacity for symbolic thought?[3–7] Why is it that humans excel in myriad cognitive domains, such as language, theory of mind, reciprocal exchange, manufacture and use of tools, cultural learning, mathematics, and artistic expression? To answer these questions in a proximate sense, we must look to the brain, noting similarities and differences in the brains of humans, other apes, and other nonhominoid primates. A sensible place to begin is by comparing neural systems with known involvement in thought and reasoning. This review will focus on two such brain regions and their component parts: the cerebellum and the cerebral cortex. Although the cerebellum has traditionally been regarded as a structure involved predominantly in motor function, recent evidence strongly indicates its involvement with cognition. Moreover, it is intimately connected with the cerebral cortex, the structure that dominates the primate brain.

Two primary data sets have been used for allometric comparisons of primate brains. The overwhelming majority of published studies are based on the postmortem data set of Stephan and colleagues[8,9] which consists of 27 anthropoid and 21 prosimian species. Most species have only one or two specimens. A more recently acquired magnetic resonance imaging (MRI) data set (http://lx50.fmridc.org/f/fmridc/77) has both advantages and disadvantages as compared with the postmortem data set.[10] The MRI data set has no prosimians and only 11 anthropoid species. Further, MRI does not allow access to cytoarchitectonic details that are microscopically observable in postmortem brains. However, in contrast to the postmortem data set, which lacks bonobo or orangutan specimens, the MRI data set includes all the great ape species, as well as a lesser ape (*Hylobates lar*). The MRI data set averages four specimens per species, which permits a reasonable estimate of intraspecific variation. The MRI data are from captive animals, whereas the postmortem data are typically from wild primates, meaning that the latter may be more representative of primate brain anatomy in species-typical environments. However, the MRI data may be superior in another sense, that they do not suffer postmortem fixation artifacts. Although attempts have been made to correct for shrinkage of brains caused by postmortem fixation, their accuracy is questionable due to the differential shrinkage of gray and white matter.[11] In this review, results from the two data sets are compared whenever possible. When analyses based on

James K. Rilling is an Assistant Professor of Anthropology at Emory University, with a secondary appointment in the Department of Psychiatry and Behavioral Sciences. Dr. Rilling and his colleagues use non-invasive functional brain imaging techniques to compare brain structure and function in monkeys, apes and humans, with the goal of identifying human brain specializations and informing our knowledge of human brain evolution. His lab also investigates the neural bases of human social behavior by measuring brain function with fMRI as subjects play interactive games with human partners.
Department of Anthropology, Department of Psychiatry and Behavioral Sciences, Yerkes Regional Primate Research Center, Emory University, Atlanta, GA 30322.
Email: jrillin@emory.edu.

Key words: comparative neuroanatomy; brain evolution; allometry; MRI; cerebellum; cerebral cortex; prefrontal cortex

DOI 10.1002/evan.00000
Published online in Wiley InterScience (www.interscience.wiley.com).

Box 1. Analytical Methods

The postmortem methods typically involve sacrificing animals, followed by fixation of the brain. Brains are sectioned and sections are magnified and projected onto photographic film paper having a known weight per square millimeter. Structure borders are delineated on filter paper, then cut out and weighed. The weight is divided by the conversion factor to calculate the area for each traced section. Areas are multiplied by the distance between sections and divided by the square of the magnification to arrive at a volume estimate. Between 60 and 80 sections are measured per brain. Structure volumes are corrected for shrinkage due to fixation by multiplying by the ratio of fresh to fixed brain volume. A second conversion corrects the structure volume to the volume of the species standard brain weight to obtain the most species-typical estimate possible using only one or two individual specimens. This is accomplished by multiplying by the ratio of the weight of the standard fresh brain to the weight of the individual fresh brain. No information on reliability is provided for the postmortem methodology.

MRI scans were acquired from living, anesthetized primates. Images were exported to a computer workstation where image analysis software was used to measure areas and volumes. All structures were defined manually, sometimes with the assistance of computer thresholding procedures that select brain tissue based on pixel signal intensity. To assess the accuracy of MRI whole brain volume estimates, two rhesus macaques were scanned *in vivo* and then sacrificed so that postmortem brains could be measured. The postmortem fresh brain volumes of 97 and 93 cc compare favorably with the respective MRI volumes of 97.4 and 90 cc. These data suggest that MRI provides a reasonable estimate of true brain volume. However, in some MRI scans the border between gray and white matter is not distinct; contrast and brightness must be manipulated to sharpen this boundary. This procedure has the drawback of moving the perceived gray matter-CSF border, which decreases brain volume estimates. To clarify gray-white matter boundaries, contrast and brightness were manipulated in all MRI scans. Using one rhesus macaque and one chimpanzee MRI scan, it was determined that contrast-enhanced MRI volumes underestimate unaltered MRI volumes by an average of 6.4%. Intrarater reliability of whole brain and cortex measurements is good. The mean coefficient of variation (CVs) for repeated brain volume estimates was 1%–6% ($n = 8$ brains). For repeated cortical gray matter estimates, the mean percentage CV was 1.4% (intrarater, $n = 6$ brains).

the two are in agreement, we have increased confidence that conclusions are not affected by weaknesses inherent in either. However, as will become apparent, there are some discrepancies between results from the two data sets. Therefore, methodological differences should also be considered (see Box 1).

Cross-species comparisons of brain structure volumes will employ the allometric method. However, allometric analyses that fit regression lines through species data points are flawed insofar as regression analyses assume independence of data points. In fact, species are not independent because of shared phylogenetic history. Alternative methods, such as independent contrasts, have been proposed to solve this problem. This method involves plotting contrasts in the X and Y variables between sister clades and fitting a regression line through these contrasts. However, interpretation of independent contrast plots is less intuitive than are standard allometric regression analyses, and the latter sometimes generate insights that cannot be gleaned from the former. Therefore, wherever possible, both types of analyses will be considered. Comparisons of slopes obtained using the two methods are presented in Table 1.

BRAIN SIZE

Across the primate order, brain size varies from less than two cc in the mouse lemur to more than 1,200 cc in humans.[8] The average brain sizes for strepsirhines, New World monkeys, Old World monkeys, lesser apes, great apes, and humans from the postmortem data set are presented in Figure 1. Primates with larger bodies are expected to have larger brains. Indeed, body weight explains 62% of variation in brain size across the primate species in the Stephan data set ($r = 0.79$). However, when the well-known nonlinear relationship between the two is considered by log transforming

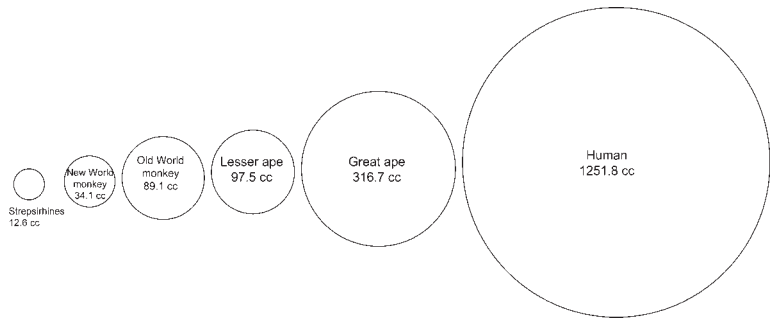

Figure 1. Comparison of average brain volume in strepsirhines, New World monkeys, Old World monkeys, lesser apes, great apes, and humans. Brain volume is proportional to circle area. Data are from the Stephan postmortem data set.

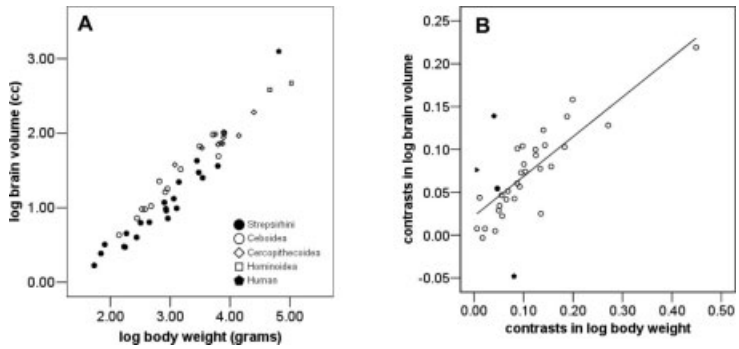

Figure 2. Brain size relative to body size for the postmortem sample. A) Logarithmic plot of brain volume against body weight for the entire sample of 48 primate species. The equation of the least squares regression line fit through the entire sample is Y = 0.80X − 1.18. B) Independent contrast regression of contrasts in log brain volume on contrasts in log body weight. The slope is 0.59 (0.50, 0.69). Filled pentagons: outliers involving humans (humans versus chimpanzees and the average of humans and chimpanzees versus gorillas); filled triangle: outlier for the contrast between *Daubentonia* and the average of *Avahi, Propithecus,* and *Indri;* filled circle: contrast between strepsirhines and haplorhines.

the data, body size explains an impressive 94% of variation in brain size ($r = 0.97$) (Fig. 2A). As has been noted repeatedly,[12] the slope of the log transformed line is significantly less than one (0.80), indicating a negatively allometric relationship in which increases in brain size fail to keep pace with increases in body size. This negative allometry implies that the ratio of brain size to body size predictably decreases as primates become larger.[13] Correcting for nonindependence of species data points due to shared phylogenetic history yields an even shallower slope.[14] For example, independent contrast analysis with the program CAIC yields a slope of 0.59 (0.50, 0.69) for the sample of species included in Figure 2A. This result illustrates how failing to control for phylogenic effects can seriously confound regression slopes.

Although absolute brain size may partially explain species differences in intelligence, the fact that elephant and whale brains are several times larger than human brains gives us pause and suggests the need to control for body size. As a result, there has been much interest in establishing a measure of relative brain size that removes the effect of body size, thereby revealing how much extra neural tissue a species possesses. A wide variety of methods has been proposed for calculating relative brain size.[9,15–18] However, it is often possible to appreciate differences in relative brain size through visual inspection of plots of brain volume against body weight without the use of formal statistics.[15] Plots of the postmortem (Fig. 2) and MRI data sets (Fig. 3) are revealing. Humans are obvious outliers in both (Figs. 2A, 3A). Furthermore, Figure 2A shows that strepsirhine primates have smaller brains for their body size than do anthropoid primates, as previously demonstrated by Armstrong.[19] Analysis of covariance (ANCOVA) can be used in formal testing for differences in regression equations among different clades. The full regression model in ANCOVA fits a separate line through each clade and tests for differences in slope and intercept. If there are no significant differences in slope, a reduced model fits parallel lines through each clade, allowing only the y-intercept or elevation to vary. For the Stephan data set in Figure 2A, there are no significant differences in slope among the clades ($F_{4,37} = 1.58$). However, humans lie at a higher elevation than do all other clades, while strepsirhines lie at a lower elevation than do all other clades ($F_{5,41} = 14.1$, all $p < 0.05$ for pair-wise comparisons). In other words, humans have larger brains for their body size and strepsirhines have smaller brains for their body size than each of the other clades. There are no significant differences in elevation among the other three clades.

The independent contrast regression for the entire sample is shown in Figure 2B. Large independent contrast residuals indicate larger than expected size change in one structure relative to another since the two clades diverged. We expect contrasts involving species with large brains for their body size to have large residuals. Three residuals from Figure 2B are outliers (standardized residual > 1.96). As expected, two involve humans (humans versus chimpanzees and gorillas versus the average of humans and chimpanzees). The third is for the contrast between *Daubentonia* and the average of *Avahi, Propithecus,* and *Indri,* reflecting the large relative brain size of *Daubentonia*.[9] The residual for the contrast between strepshirhine and haplorhine primates (dark circle in Figure 2B), while posi-

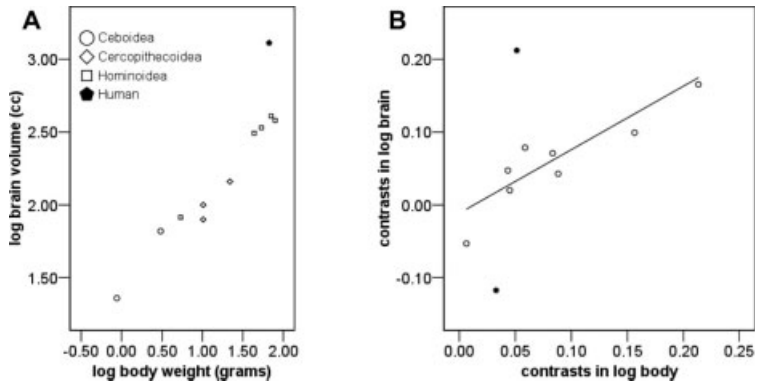

Figure 3. Brain size relative to body size for the MRI sample. A) Logarithmic plot of brain volume against body weight for 11 species of anthropoid primates. The equation of the least squares regression line fit through the entire sample is Y = 0.71X + 1.36. B) Independent contrast regression of contrasts in log brain volume on contrasts in log body weight. The slope is 0.70. Filled pentagons: outliers involving humans (humans versus chimpanzees and gorillas versus the average of chimpanzees, bonobos, and humans).

tive, is not an outlier. This is hard to reconcile with Figure 2A, which shows that where there is an overlap in body weight between anthropoids and strepsirhines, strepsirhines consistently have smaller brains, strongly implicating an increase in relative brain size with the evolution of anthropoids. In this case, the independent contrast method fails to detect the grade shift.

Turning to the MRI data, which includes only anthropoids, there is no significant difference in the slope of the various clades ($F_{2,4} = 2.04$), but humans lie at a higher elevation than the other three do and apes lie at a higher elevation than cercopithecoid monkeys do (Fig. 3A) ($F_{3,6} = 37.1$; $p < 0.05$ for pairwise comparisons). The independent contrast regression yields two outliers, both of which involve humans (Fig. 3B). After omitting the human data point because it is an outlier, the largest standardized residual (1.54) is for the *Hominoidea-Cercopithecoidea* contrast, consistent with the difference in elevation found with ANCOVA.

Thus, in contrast to the postmortem data, the MRI data suggest that apes have larger relative brain size than that of Old World monkeys. The discrepancy could be explained by the species composition of the two samples: The postmortem sample has more monkeys and the MRI sample has more apes. However, one cannot say which result is more accurate. Collectively, these data are therefore inconclusive on the issue of relative brain size differences between apes and other nonhuman anthropoids. However, humans have relatively larger brains than other primates do, and Figure 2A convincingly demonstrates that anthropoids have relatively larger brains than do strepsirhines.

BRAIN COMPOSITION

Differences in absolute and relative brain size are likely part of the explanation of cognitive differences among humans, apes, and other anthropoids. However, irrespective of these differences in brain size, it is important to ask whether all nonhuman primate brains are qualitatively similar. Are they scaled versions of the same design or are they fundamentally different in composition? In a principal components analysis of 131 mammalian species from the postmortem data set, Finlay and Darlington[20] found that the first principal component, essentially brain size, explained more than 96% of the variance in the size of 11 major brain structures: the olfactory bulb, paleocortex, medulla, mesencephalon, septum, hippocampus, schizocortex, cerebellum, diencephalon, striatum, and neocortex. Coupled with known neurodevelopmental mechanisms, this result led to the conclusion that intractable developmental programs force mammalian brain growth to follow predictable allometric trends in which individual brain structures enlarge mainly by concerted enlargement of the entire brain. In other words, all mammalian brains are basically smaller or larger versions of the same plan. Nevertheless, the magnitude of unexplained variability in the sample was such that two species with the same brain volume could differ by as much as 2.5 times in the size of a given structure, leaving substantial room for species-specific adaptation in brain design.

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In subsequent analyses of the same data, Barton and Harvey[21] emphasized the limitations of developmental constraints and the importance of mosaic brain evolution, showing that the primate neocortex is nearly five times larger than the neocortex of an insectivore of equivalent nonneocortical brain size. Further, they demonstrated significant correlations in evolutionary size change between brain structures that are components of the same functional system after controlling for changes in the size of other brain structures. They concluded that components of functional systems evolved together independently of evolutionary size change in other structures and the rest of the brain. This suggests that mammalian brains are not all simply smaller or larger versions of the same basic plan.

Like Finlay and Darlington, de Winter and Oxnard[22] used principal components to analyze the Stephan data set, but conducted their analysis on ratios rather than absolute volumes. They examined both structure size to medulla size and structure size to neocortex size. The first three principal components cleanly separated the three major orders, bats, insectivores and primates, further supporting the conclusion that mammalian brains are not all similarly designed. The analysis further divided the primate data into four groups: prosimians plus marmosets and tamarins; ceropithecids plus howlers and sakis; apes plus spider and woolly monkeys; and humans. The authors postulate that these four groups map onto different locomotor strategies. Further analysis revealed that the dispersion of these groups in the primate direction was produced by a concerted increase in the proportions, relative to the medulla, of the neocortex, striatum, cerebellum, and diencephalon. What is left unanswered by this informative analysis is whether the various primate groups lie along a single, predictable allometric trajectory. While it is clear from the analysis that human brains are more like those of nonhuman primates than those of other mammals, it is not clear whether human brains are simply enlarged versions of a generalized primate design.

The Cerebellum

Anatomy of the cerebellum

Much like the cerebrum, the cerebellum consists of cortical gray matter overlying white matter within which lie subcortical nuclei, known as cerebellar deep nuclei (Fig. 4). Inputs to the cerebellum arrive at the cerebellar

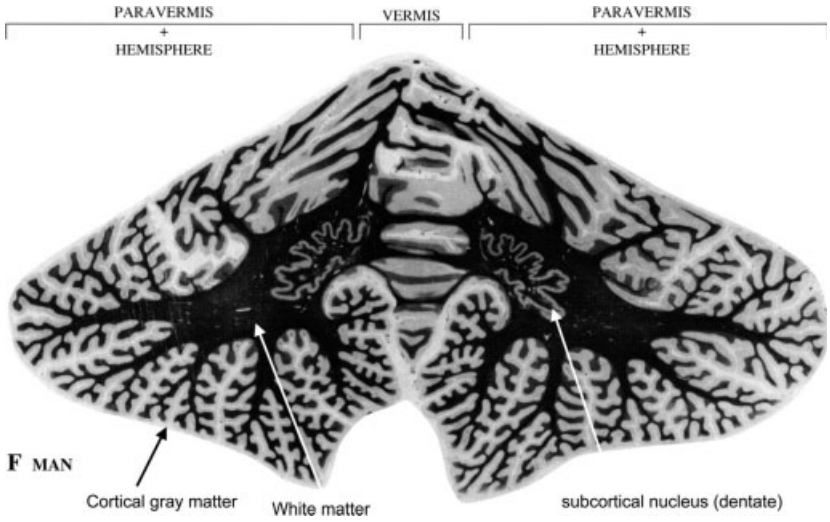

Figure 4. Cerebellar anatomy. Postmortem staining causes white matter to appear black and gray matter to appear light. Reprinted with permission from Altman and Bayer.[81]

cortex; outputs leave the cerebellum via the deep nuclei.

Anatomical and neuropsychological evidence shows that the cerebellum is involved not only in movement, but also cognition. The cerebellum is extensively interconnected with the cerebral cortex. Snider[23] proposed that the cerebellum is "the great modulator of neurologic function." That is, it improves the skilled performance of any cerebral area to which it is linked by two-way neural connections.[24] Connections with motor areas would increase the speed and skill of movement, while connections with cognitive areas would improve the speed and skill of thought.

Comparative cerebellar anatomy

Are there differences between the cerebella of humans, apes, and the rest of the anthropoids? Absolute cerebellum volume varies markedly within the MRI sample (Fig. 5A). Eighty-eight percent of the variability in (log) cerebellar size is explained by log body weight ($r = 0.94, p < 0.001$) (Fig. 5B). Much of the unexplained variance can be attributed to the human data point, which lies well above the regression line, indicating that the human cerebellum is larger than expected for a primate of our body weight. Given that the entire human brain is larger than expected for a primate of our body weight (Figs. 2A, 3A), one might ask whether the cerebellum is unusual in this regard or whether other human brain structures are also disproportionately large relative to body weight. In fact, the cerebellum is second only to the neocortex, albeit a distant second, in terms of its size relative to body size.[9]

The fact that the neocortex and cerebellum are the two structures that enlarged most relative to body size in humans, coupled with the existence of extensive connections between the two, suggests that they may have evolved in tandem as a coordinated system.[21] Using independent contrasts to eliminate the effect of common inheritance, Barton and Harvey[21] showed this to be true for primates. Cerebellar contrasts are significantly correlated with neocortex contrasts. This relationship is stronger than that of cerebellar contrasts and other major brain divisions, such as the medulla, mesencephlon, and diencephalon.

The ape cerebellum is not an allometrically enlarged anthropoid cerebellum

Despite this tendency for the cerebro-cerebellar system to evolve as a coordinated whole, there is evidence that the relationship between the two structures was altered with the evolution of hominoids.[25,26] When cerebellum is regressed on cerebral cortex for the MRI data set, a regression line through the apes lies above each of the five monkey species (Fig. 6A). Plotting individual subjects rather than species means gives a more complete picture of the data, although it exacerbates the problem of nonindependence of data points. When individual subject data are plotted (Fig. 6B), apes lie at a higher elevation than does any of the other three taxa ($p < 0.05$). Subsequent analyses of these data,

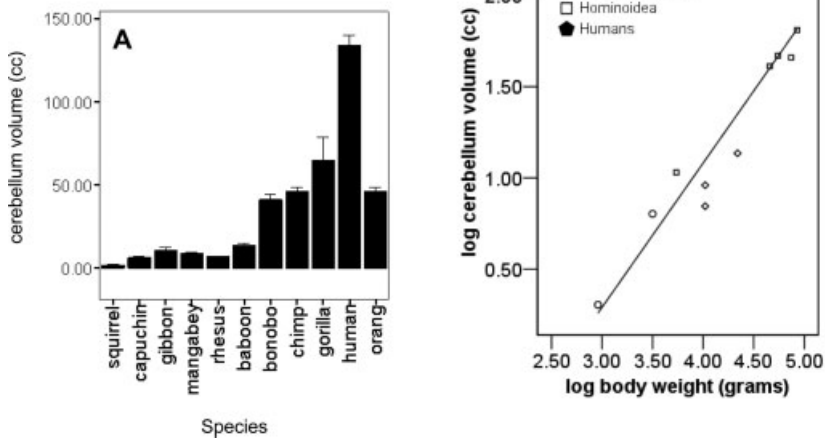

Figure 5. Comparison of cerebellum volume in anthropoid primates. A) Mean absolute cerebellum volume from MRI scans in 11 anthropoid primate species, in ascending order of body weight. Note that the gorilla body weight is low for the MRI data set because it is based on one small female and one subadult male. Error bars are +/− 1 se. B) Logarithmic plot of cerebellum volume against body weight, with least squares regression line fit through the entire sample.

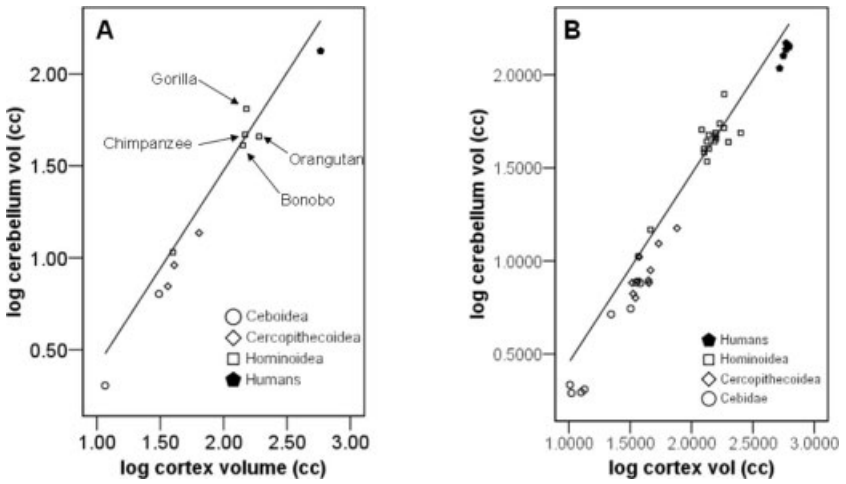

Figure 6. Comparison of cerebellum volume relative to cerebral cortical gray matter volume in anthropoid primates. Regression of log cerebellum volume on log cerebral cortical volume A) plotting species means, B) plotting individual specimens. Least squares regression lines are fit through the ape sample. Human data points are plotted but not included in either regression. Data are from Rilling and Insel.[10,25]

combined with additional postmortem data by MacLeod and colleagues,[27] show that this difference between apes and monkeys is concentrated in the cerebellar hemispheres as opposed to the medial aspect of the cerebellum, known as the vermis.

An independent contrast analysis of these data yields just one outlier, for the contrast between gorilla and the average of chimpanzees, bonobos, and humans. The outlier signifies that after gorillas diverged from the common ancestor of chimpanzees, bonobos, and humans, there was an atypical change in size of either the cerebral cortex or cerebellum in one of the two lineages. Either gorillas experienced a larger-than-expected increase in cerebellar size given their change in cerebral cortex size, or the common ancestor of chimps, bonobos, and humans experienced a larger-than-expected change in cerebral cortex volume given its change in cerebellum size. Data presented below support the latter possibility.

The contrast between apes and Old World monkeys is not an outlier (1.02), which might suggest that the apparent grade shift between apes and other anthropoids is an artifact introduced by failing to control for phylogenetic effects. However, a larger data set that combines postmortem and MRI data shows that lesser apes clearly have larger cerebella than do other anthropoids of similar brain size.[27] The same trend is evident in Figure 6B, albeit with a smaller sample and some overlap. Moreover, as we saw earlier for the haplorhine-strepsirhine comparison, independent contrast analyses do not always detect grade shifts. Overall, results indicate that the evolution of hominoids involved a larger increase in cerebellum size than expected for the increase in cerebral cortex size.

Further differences between apes and other nonhuman anthropoids are found in the cerebellar dentate nucleus, which projects to the cerebral cortex by way of the thalamus. Based on morphological, histological, embryological, histochemical, and pathological evidence, the dentate nucleus is thought to consist of an older dorsomedial part and a newer ventrolateral part.[28] Among anthropoid primates, the ventrolateral part is reportedly unique to humans and apes.[29] Leiner, Leiner, and Dow[29] argue that it sends output to nonmotor regions of the frontal lobe by way of the ventrolateral thalamus, suggesting a role in cognition. These are merely hypothesized connections based on neuropsychological and neurophysiological evidence. However if they are true, then the emergence of the ventrolateral dentate in hominoids could reflect a qualitative shift toward increased cerebellar involvement with cognition by virtue of connections with nonmotor frontal lobe regions.

What is the functional significance of the elaborated hominoid cerebellum? As suggested, it might improve the functioning of all cortical regions with which it is connected. One cortical target of lateral cerebellar output is motor and premotor cortex, by way of the thalamus, and it has been argued that apes have a greater complexity of movement than monkeys do.[27,30] Also compatible with augmentation of function in motor and premotor cortex is Ott's observation that, in contrast to apes and humans, baboons lack "presyntactical motor planning," the ability to modify the pace of current movements based on awareness of movements to follow.[31] Along the same lines, Byrne[32] has emphasized the ability of great apes to use on-line corrective guidance during the execution of complex foraging tasks. Other skills that apes excel at relative to other anthropoids are also likely to depend on motor and premotor cortex. For example, when reaching for an object, apes and humans exhibit more complex preshaping of their hands than do other anthropoids.[33,34] Beyond these differences in motor-related functions, some researchers believe that apes and other anthropoids also possess cognitive differences. In view of known connections between them, it is conceivable that cerebellar augmentation of prefrontal function could be involved in apes' putative capacity for self-awareness,[1] components of theory of mind,[2] and capacity for symbolic thought,[3,4] since in humans each of these abilities depends on prefrontal cortex.[35–38]

The human cerebellum is not an allometrically enlarged ape cerebellum

The human cerebellum is markedly larger than the ape cerebellum, even after adjusting for differences in body weight (Fig. 5B). In addition, Matano[39] showed that the ventrolateral portion of the dentate nucleus is more prominent in humans than in great apes. Despite this tendency for cerebellar elaboration in humans, it should be noted that humans actually

fall below the ape regression line of cerebellar volume against cerebral cortical volume (Fig. 6). This indicates that humans have small cerebella for their cortex size, humans have large cerebral cortices for their cerebellar size, or some combination of the two. Data presented in the next section support the second of these possibilities.

What could be the functional significance of enlarged cerebellar hemispheres and ventrolateral dentate in humans? One possibility is accurate overhand throwing, which likely represented a strong selective pressure throughout our hunting-and-gathering past.[40–42] Accurately throwing rocks and projectiles may have been crucial for hunting and scavenging prey, predator defense, and intergroup hostilities. Another possibility is that the enlarged human cerebellum supports fine motor coordination involved in the manufacture and use of tools. However, it is also clear that the cerebellum takes on a cognitive role in humans, being involved in a wide range of mental operations. For example, Leiner, Leiner, and Dow[43] have emphasized its role in language in view of neuropsychological evidence and hypothesized connections with the left inferior frontal cortex (that is, BA 44 or Broca's area), known for its role in speech production. In view of the wide range of mental operations the cerebellum has been linked with, one interesting possibility is that the human cerebellum is involved in a global augmentation of frontal lobe function that extends to cognitive domains beyond language. Consistent with this idea, a recent report based on fossil endocast analyses argued that cerebellar expansion in Late Pleistocene and Holocene humans may have resulted in a generalized increase in computational efficiency.[44]

Cerebral Cortex

The cerebral cortex is the layer of gray matter on the surface of the cerebral hemispheres. Cerebral cortex includes paleocortex (that is, olfactory cortex), hippocampus, schizocortex (cortex surrounding the hippocampus), and neocortex. Neocortex, defined by the presence of four to six cell layers, is unique to mammals and has expanded to occupy most of the cerebral cortex of primates. Within the neocortex, primary cortices are directly connected with peripheral sensory and motor pathways. These cortices pass information along to adjacent secondary sensory cortices that further refine the sensory information and relay it to multimodal association cortices. These cortices integrate information from two or more sensory modalities and coordinate this information with plans for action. Motor plans are relayed to the primary motor cortex, which issues motor commands to the spinal cord.

The postmortem and MRI data sets take slightly different measurements of the cerebral cortex. The postmortem data set reports neocortical volume, whereas the MRI data set gives the volume of the entire cerebral cortex minus the hippocampus. In practice, the distinction is trivial, since paleo and schizocortex are a tiny fraction of total cerebral cortex in primates. (For example, human neocortex = 1006.5 cc; human paleocortex = 6.0 cc; and human schizocortex = 6.1 cc).

The finding that the human cerebellum is smaller than expected for an ape of our cerebral cortex size could signify that humans have been downwardly displaced from the ape regression line and have a small cerebellum for our cortex size, that humans have been displaced horizontally (to the right) from the ape regression line because the cortex is disproportionately large relative to the cerebellum, or some combination of the two (see Fig. 6). To determine which is the case, cerebral cortex (combined volume of cerebral cortical gray matter plus the underlying white matter) and cerebellum are both separately regressed on the volume of the rest of the brain, excluding both cerebral cortex and cerebellum (Fig. 7).[45] For the MRI data set (Fig. 7), humans have a larger cerebral cortex (Fig. 7A) and an expected cerebellar size (Fig. 7B) relative to predictions based on the ape regression line. On the other hand, the postmortem data suggest that humans have both a larger-than-expected neocortex (9.0%) and a smaller-than-expected cerebellum (20%) (Figs. 7C, D), although in both cases the human data point lies well within the 95% confidence bands. The postmortem sample yields a much steeper slope for the regression of neocortex on the rest of the brain in hominoids (1.54) than does the MRI sample (0.91). The wide confidence bands in Figure 7C imply little confidence in the slope of the postmortem sample due to the small number of ape species on which the line is based. The MRI sample has both more ape species, and each species data point is based on averaging a larger number of individuals. So the MRI slope is more likely to be close to the true slope. Furthermore, independent contrast analyses yield a shallower slope for the postmortem data, also suggesting that the postmortem slope may be too steep. A shallower hominoid slope would give the human data point a larger positive residual and make the results more similar to the MRI data. Even if the postmortem slope were accurate, it implies a strong positive allometry between neocortex and the rest of the brain, so that larger brains have proportionately more neocortex. In this case, the human brain would be different from other ape brains, but the difference would be predictable.

Despite this discrepancy between the postmortem and MRI data sets for the standard regression analyses, independent contrast analyses of neocortex versus the rest of the brain for both data sets agree in generating a single outlier, which is for the contrast between gorilla and the average of humans and *Pan*. The same contrast is not an outlier for the independent contrast regression of cerebellum on the rest of the brain for either data set. Thus, both data sets indicate that after the divergence of gorillas from the common ancestor of chimpanzees and humans, the latter exhibited a larger-than-expected increase in cerebral cortex size given the change in the size of the rest of the brain. This is particularly interesting in light of ecological differences between the two clades, with gorillas subsisting on a more spatially and temporally predictable food source (leaves), which may require less cognitively demanding foraging strategies than those of chimpanzees or humans. Finally, nei-

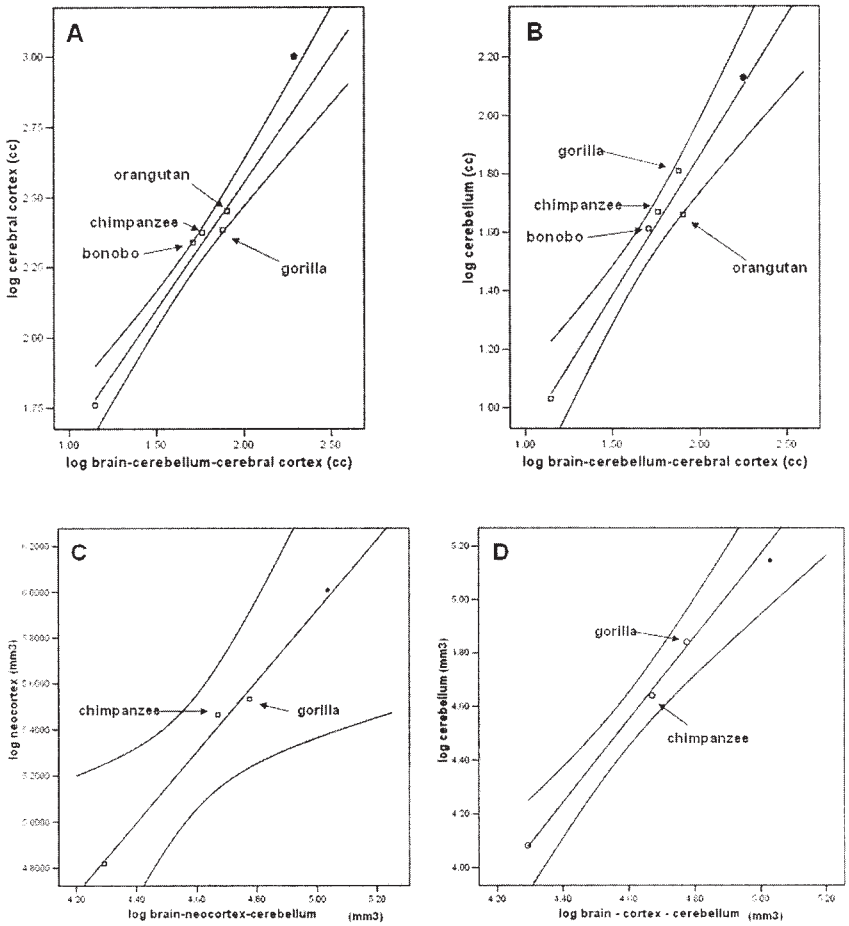

Figure 7. Comparison of cerebral cortical (gray plus underlying white matter) and cerebellar volume relative to the rest of the brain in apes and humans. A) and B): MRI sample. C) and D): Postmortem sample. In all graphs, a least-squares regression line is fit through the apes, excluding humans. Ninety-five percent confidence bands are included. Human = filled pentagon.

ther data set identifies the human versus *Pan* contrast as an outlier and, once again, the independent contrast analyses fail to identify an apparent departure from allometry.

Cortical Gyrification

In a series of objects of the same shape but differing in size, surface area scales to the two-thirds power of volume. So if primate brains were to maintain their shape with increases in size, cortical surface area would be expected to scale at the 0.67 power of brain volume.[46] Instead, the exponent of allometry relating cortical surface area and brain volume is 0.80[47] or 0.81 using the method of independent contrasts, indicating that the relationship is not subject to phylogenetic effects. In other words, cortical surface area scales with positive allometry on brain volume among anthropoid primates, indicating that the brain changes its shape in a predictable way.[46] The change in shape is reflected in an increase in gyrification, a measure of the degree of cortical folding in the brain.[46,48–50]

But why should the cortical surface grow out of proportion to the rest of the brain? As brain size increases among primates, cortical thickness increases minimally, whereas cortical surface area increases substantially, so that the cortex grows like an expanding sheet that covers the brain. One hypothesis is that, as this cortical sheet expands, it must fold in order to fit within the confines of a spherically shaped skull.[46,50] The result is that larger brains with more cortex have more cortical folds.

Outward folds of the cortex are known as gyri; the inward folds that divide them are known as sulci. One method of quantifying cortical gyrification is to compute a gyrification index (GI), defined as the ratio of the length of the total cortical surface (including cortex buried within cerebral sulci) to the length of the superficially exposed cortical surface (excluding cortex buried within sulci)[49] (Fig. 8A). This measurement can be calculated slice by slice across the entire brain.

As described, the average whole brain GI predictably increases with increasing brain size among anthropoids.[10,49] As expected, given our large brain, humans have a larger GI than do nonhuman primates at each slice along the rostral-caudal axis of the brain. However, the degree of gyrification in the rostral-most sections of the human brain is even greater than expected based on brain size (Fig. 8B).[10,49] These sections are in the prefrontal cortex. The result is not confounded by the nonindependence of species data points, given that the independent contrast slope for the nonhuman sample falls within the 95% confidence interval of standard regression slope.

The same result is obtained if prefrontal GI is regressed on prefrontal volumes.[51] Humans are a positive outlier. It is important to note that this observation constitutes a departure from allometry in humans. It suggests that natural selection uniquely modified the human brain to deviate from the rules of brain design that obtain among other primates. A unique evolutionary modification in the human prefrontal cortex is intriguing because this brain region is involved in many cognitive operations that are especially well-developed in humans, such as symbolic thinking, knowledge of appropriate social behavior, decision making, planning, cognitive control, and working memory.[35,52–55]

Although the significance of this disproportionate gyrification in human prefrontal cortex is unknown, various explanations have been proposed based on the assumption that, in addition to extrinsic factors like skull shape, factors intrinsic to the

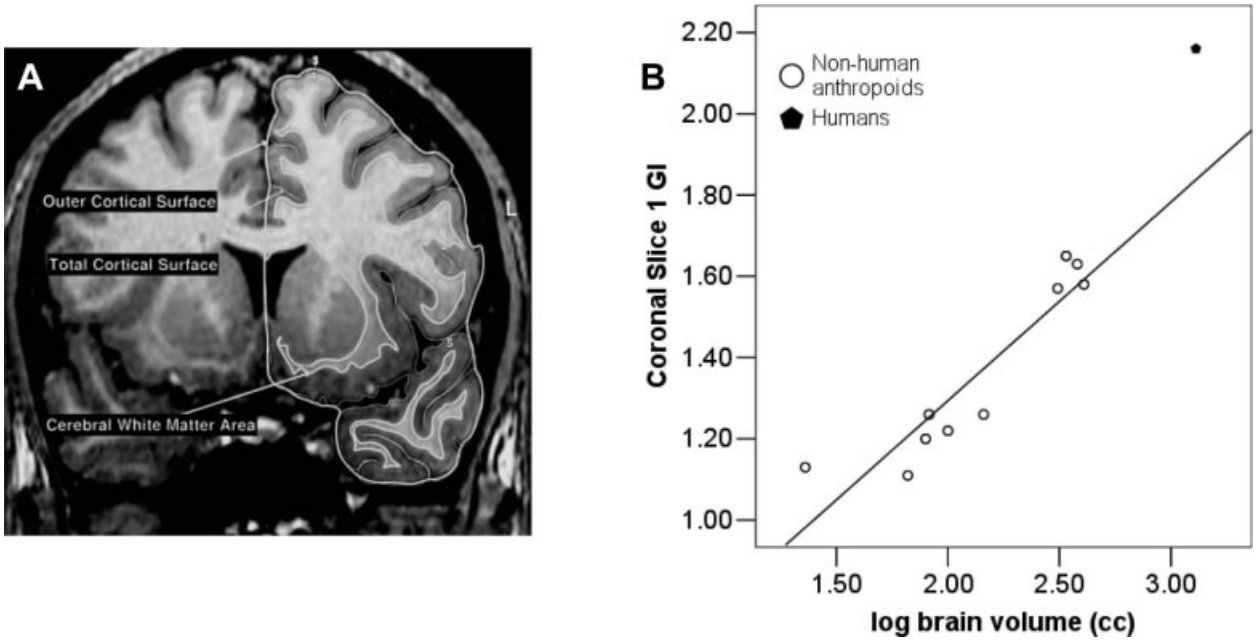

Figure 8. Gyrification in human cerebral cortex. A) coronal MRI section through human brain with tracing of total and outer cortical lengths that are used to calculate the gyrification index (GI). B) Plot of prefrontal GI against brain volume with least squares regression line fit through the nonhuman sample. Reprinted with permission from Rilling and Insel.[10]

cortex may also contribute to cortical folding. Two prominent models of gyrification argue that increases in gyrification reflect augmented intracortical connectivity.[56,57] If so, then human evolution involved selection for increased intracortical connectivity in the prefrontal cortex.

Frontal Cortex

In the field of comparative primate neuroanatomy, perhaps no question has engendered more interest than whether the human frontal lobes are larger than expected for a primate of our brain size. It is important to realize that, in terms of absolute size, the human frontal lobes dwarf those of other primates. This may be tremendously relevant to explaining cognitive differences among species.[58,59] Here, we ask whether the human brain has been reorganized away from the typical primate pattern so that the frontal lobe occupies a larger-than-expected proportion of brain volume.

Analyses of the MRI data set revealed that humans and great apes have a similar proportion of frontal cortex to total cortical volume, and that this proportion exceeds that in lesser apes and monkeys.[60] Allometric regression of frontal lobe volume on the volume of the rest of the hemisphere revealed that all 10 human data points actually lie below the best fit line through the nonhuman anthropoid sample. However, most of the data points fall within the 95% confidence bands around the regression line, leading Semendeferi and colleagues[60] to conclude only that the human frontal lobes are not larger than expected for a primate of our brain size. However, if the slope is accurate, then the fact that all 10 human points lie below the regression line (the probability of that happening by chance is vanishingly small) is strong evidence that our frontal lobes are actually smaller than expected for a primate of our brain size.

These findings for the frontal lobe as a whole do not preclude a possible specialization in human prefrontal cortex (PFC).[59,60] Given the difficulty of reliably defining the boundaries of prefrontal cortex from cortical surface landmarks, this question is better answered with histological materials that allow prefrontal cortex to be defined cytoarchitectonically. Indeed, allometric analyses based on prefrontal measurements from histological material[61,62] show the human PFC to be larger than expected for a primate of our total neocortex size.[35,63] Moreover, the slope of the regression line is greater than one, indicating that allometry alone will result in a greater proportion of PFC in larger brains. As Semendeferi and coworkers[60] point out, the magnitude of the departure from allometry may be exaggerated for the histological data set due to sampling biases when using only one or two hemispheres per species. Nevertheless, two separate histological data sets both find humans to have significantly more PFC than is predicted by nonhuman primate allometry.[35,61,62] This conclusion is also supported by the observation that primary motor (BA4) and premotor (BA6) cortex (nonprefrontal) occupy a much smaller proportion of the cortex in humans than in other primates.[35,61,62,64,65] If these areas are smaller than expected, but frontal cortex as a whole is as large as or only slightly smaller than predicted, then PFC should be larger than predicted. These results for prefrontal volume dovetail with the prefrontal gyrification data discussed in suggesting that the human prefrontal lobe has been uniquely modified.

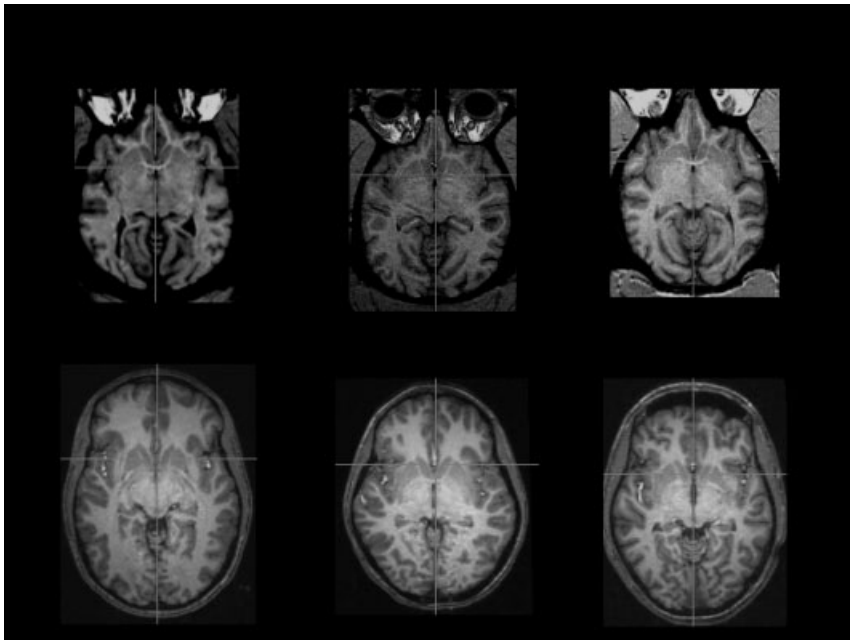

Figure 9. Orbitofrontal cortex in humans and chimpanzees. Horizontal MRI sections through the brains of three chimpanzees (top) and three humans (bottom) showing the difference in orbitofrontal white matter volume. Human images have been reduced to the size of chimpanzee images for the purpose of comparing the relative size of OFC white matter.

Evidence for specialization of human PFC also exists at the cellular level. The peak branching complexity of layer 3 pyramidal-cell basal dendrites is markedly higher in the human PFC than in that of macaques or marmosets, whereas the branching complexity in temporal and occipital cortex is similar in these species.[66] Since neurons receive input over their dendritic surface, the increased branching complexity in human PFC likely reflects integration of a larger number of inputs, bolstering the hypothesis of increased intracortical connectivity in human PFC.

If there has been augmentation of intracortical connectivity in human PFC, this should be reflected in the volume of prefrontal white matter. Using the MRI data set plus additional human scans, Schoenemann, Sheehan, and Glotzer[51] measured the volume of gray and white matter anterior to the genu of the corpus callosum, a portion of PFC, and found humans to have significantly more white matter than is predicted by the nonhuman primate regression of prefrontal white versus nonprefrontal cerebral volume. The human value was 41% larger than predicted. On the other hand, prefrontal gray matter volume did not differ from predictions. This observation was foreshadowed by a study that used a smaller sample.[67] The difference between human and ape brains in this regard can be appreciated in Figure 9, in which the amount of orbitofrontal white matter can be contrasted in three human and three chimpanzee brains.

It is also possible to compare specific cytoarchitectonic areas within the PFC to determine if the disproportionate expansion of human PFC is localized to a particular prefrontal region. For example, as a proportion of total brain volume, Brodmann's area 10 at the frontal pole of the brain is twice as large in humans as that in great apes,[68] though only 6% larger than allometric predictions for an ape brain of human size.[69] In contrast to area 10, the proportion of brain volume occupied by orbital area 13 is not larger in humans than in great apes.[70]

Occipital Cortex

Primary visual cortex has also been quantified and allometrically compared across primates. Human primary visual cortex (striate cortex) is considerably smaller than expected for a nonhuman primate of our brain[71,72] or neocortex size,[71] but about the size expected for a typical nonhuman primate of our body size.[63] If the frontal cortex as a whole and the primary visual cortex are both smaller than expected for a primate of our neocortical size, some other cortical region must be larger than predicted by nonhuman primate allometry. One such region is the temporal lobe.

Temporal Cortex

Two studies that have used the MRI data set to measure and allometrically compare temporal lobe volume across anthropoid primates (Fig. 10)[26,73] have found that human data points cluster above the ape regression line (Fig. 10B).[26,73] This suggests that humans have larger temporal lobes than expected for an ape of our brain size. Given that temporal lobe borders are difficult to define and each study defined them differently, the consistency of the results is compelling. The independent contrast slope for hominoids is not significantly different from the standard regression slope, suggesting that the results are not confounded by nonindependence of species data points. Again, the independent contrast regression yields a lone outlier for the contrast of gorilla and the average of chimpanzees, bonobos, and humans, but fails to identify the human-*Pan* contrast as an outlier. The expansion of the temporal lobe in humans is concentrated in temporal lobe white matter, which likely consists of axons that link temporal and frontal cortex.[73]

It is possible that this departure from allometry in the human temporal lobe relates to the evolution of the neural substrates for human language. The classic model of brain language function derived from neuropsychological patients emphasized the importance of the left inferior frontal cortex (Broca's area) for speech production and the left posterior, superior temporal lobe (Wernicke's area) for speech comprehension. In recent years it has become clear that, in addition to these areas, large portions of the lateral surface of the left frontal and temporal lobes are also involved in human language.[74–76]

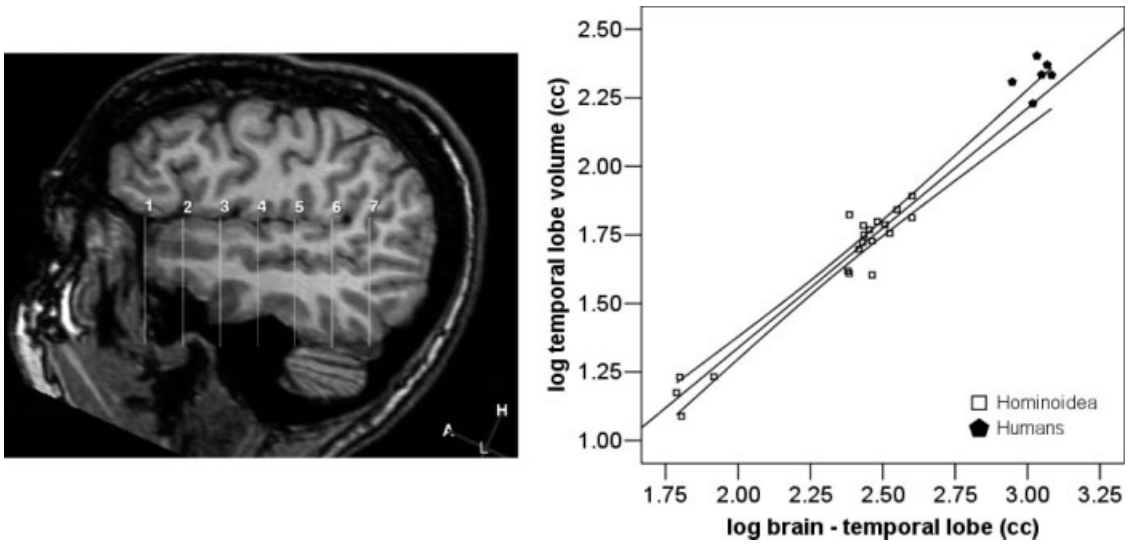

Figure 10. Allometric comparison of primate temporal lobe volumes. A) parasagittal MRI from a human subject showing location of temporal lobe sections. B) Plot of temporal lobe volume against the volume of the rest of the brain in apes and humans, with a least-squares regression line fit through the apes. Reprinted with permission from Rilling and Seligman.[73]

Therefore, disproportionate expansion of temporal and prefrontal cortex may have supported the evolution of human language. The case for reorganization of the human temporal lobes is supported by other evidence. In primates, object recognition takes place in a pathway known as the ventral visual object recognition stream, which runs along the inferior temporal lobe. However, this pathway is located more ventrally and caudally within the human temporal lobe than in that of monkeys, which Ungerleider, Courtney, and Haxby[77] suggest is a consequence of displacement by expansion of language-related cortex.

Thus, prefrontal and temporal cortices have apparently expanded beyond allometric predictions in the human brain, but primary visual and motor cortex are much smaller than expected. This is compatible with the suggestion that primary sensory and motor areas of the cortex have expanded minimally if at all in human evolution, while higher-order association cortices have expanded dramatically[64,78,79] since higher-order association cortices are found in prefrontal, temporal, and posterior parietal regions.

CONCLUSIONS

This discussion has made it clear that human, ape, and other anthropoid brains are not only quantitatively, but also qualitatively different. Both types of differences are likely relevant to explaining species differences in cognitive abilities. This review has identified specializations of human and ape brains, as well as general trends that hold across the order.

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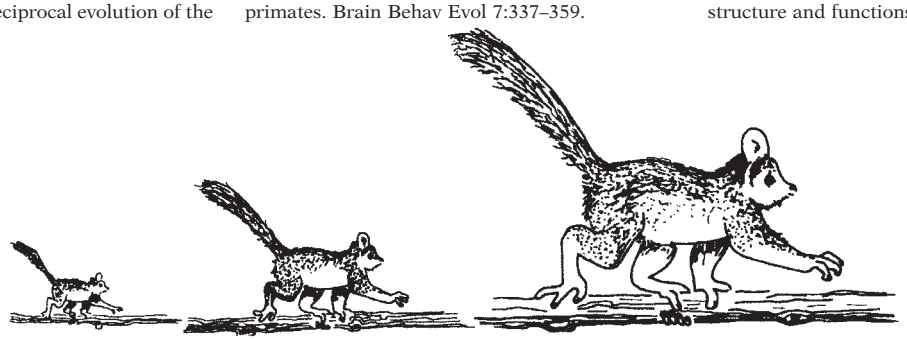

APPENDIX 1: COMPARISON OF SLOPES OBTAINED WITH STANDARD ALLOMETRIC REGRESSION ANALYSES AND INDEPENDENT CONTRAST ANALYSES

Regression	Dataset	Group	IC	Standard
Log brain vs. log body	postmortem	all	**0.59**	0.80 (0.74, 0.86)
		prosimian	**0.56**	0.66 (0.57, 0.75)
		ceboidea	0.78	0.76 (0.64, 0.89)
		cercopithecoidea	0.48	0.48 (.328, .632)
		hominoidea	0.53	.562 (−.565, 1.69)
	MRI	all	0.7	0.71 (.50, .91)
		ceboidea	I.D.	0.85
		cercopithecoidea	0.6	0.64 (−2.70, 3.97)
		hominoidea	**0.72**	0.60 (.50, .70)
Log cerebellum vs. log cerebral cortex	MRI	all	**0.93**	1.12 (0.98, 1.27)
		ceboidea	I.D.	1.17
		cercopithecoidea	1.05	1.01 (−1.89, 4.09)
		hominoidea	1.05	1.07 (0.44, 1.70)
Log cerebral cortex vs log brain-cortex-cerebellum	postmortem	all	1.33	1.32 (1.27, 1.37)
		ceboidea	1.24	1.25 (1.17, 1.33)
		cercopithecoidea	1.26	1.20 (0.98, 1.42)
		hominoidea	1.46	1.54 (−0.85, 3.93)
	MRI	all	1.04	1.04 (0.89, 1.20)
		ceboidea	I.D.	0.7
		cercopithecoidea	0.88	.83 (−3.95, 5.61)
		hominoidea	0.86	0.91 (0.64, 1.17)
Log cerebellum vs. log brain-cortex-cerebellum	postmortem	all	1.28	1.28 (1.22, 1.34)
		ceboidea	1.23	1.24 (1.15, 1.32)
		cercopithecoidea	1.2	1.26 (1.01, 1.51)
		hominoidea	1.55	1.55 (0.58, 2.52)
	MRI	all	0.99	1.14 (0.93, 1.36)
		ceboidea		0.84
		cercopithecoidea	1.02	0.98 (−2.71, 4.68)
		hominoidea	0.95	0.96 (.55, 1.36)
Log cortical gray vs. log cortical white	postmortem	all	1.24	1.25 (1.19, 1.32)
		ceboidea	1.25	1.28 (1.09, 1.46)
		cercopithecoidea	1.5	1.50 (0.28, 2.71)
		hominoidea	I.D.	1.4
	MRI	all	1.12	1.12 (1.06, 1.19)
		ceboidea		1.025
		cercopithecoidea	1.09	1.08 (0.53, 1.63)
		hominoidea	1.19	1.19 (0.86, 1.52)
Log temporal vs. log brain-temporal	postmortem	all	0.89	0.80 (0.69, 0.90)
		ceboidea		0.55
		cercopithecoidea	0.97	0.98 (.59, 1.36)
		hominoidea	0.8	0.85 (0.52, 1.17)

Independent contrast slopes in bold-face type are outside the 95% confidence interval of the standard regression slope.
I.D. = insufficient data to estimate the slope.