Evolution of the Size and Functional Areas of the Human Brain

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Abstract

The human brain is one of the most intricate, complicated, and impressive organs ever to have evolved. Understanding its evolution requires integrating knowledge from a variety of disciplines in the natural and social sciences. Four areas of research are particularly important to this endeavor. First, we need to understand basic principles of brain evolution that appear to operate across broad classes of organisms. Second, we need to understand the ways in which human brains differ from the brains of our closest living relatives. Third, clues from the fossil record may allow us to outline the manner in which these differences evolved. Finally, studies of brain structure/function relationships are critical for us to make behavioral sense of the evolutionary changes that occurred. This review highlights important questions and work in each of these areas.

Encephalization quotient (EQ): calculated as the ratio of a species' actual brain size to the size expected given its body weight

INTRODUCTION

The evolution of the human brain has been one of the most significant events in the evolution of life. Although the outline of how and why this happened is being filled in, many fundamental questions remain to be answered. The fossil record, in concert with a comparative neuroanatomical analysis of closely related species, shows that the hominid brain increased in size more than threefold over a period of approximately 2.5 million years. However, it has become increasingly clear that the human brain is not simply a large ape brain: Important qualitative and quantitative changes occurred as well. Some of these changes are a result of broad patterns of brain evolution that appear across species, either for developmental reasons or because of patterns of adaptation that are inherent in the nature of life. Some are presumably a result of direct selection for specific behavioral abilities of various kinds. Unraveling which adaptational explanations are possible or likely requires understanding as much as we can about how brain structure relates to behavioral variation. Unraveling the story of human evolution requires research in each of these areas: (*a*) general patterns of brain evolution, (*b*) comparative assessment of brain anatomy across species, (*c*) the fossil history of human-brain evolution, and (*d*) brain structure/function relationships.

This review focuses on trying to understand evolutionary changes in brain size as well as the proportions of different brain areas. It highlights conceptual areas and questions that are prone to misunderstanding, need particularly careful assessment, are of current controversy, or present important avenues for future research. It necessarily leaves out some research areas that are both important and interesting, such as deep cortical and brainstem nuclei, the evolution of specific neuron types and their specializations, and possible gene-expression changes.[1]

PATTERNS OF BRAIN EVOLUTION

Brain/Body Scaling

How should species be compared with respect to brain size? It has long been known that brain size scales with body size across broad groups of animals (Dubois 1913). For this reason, some measure of relative brain size has usually been favored in comparative studies. Empirically the relationship between brain and body size can be estimated using a function of the form $[\text{brain}] = c[\text{body}]^a$, where c and a are empirically derived constants. Because the brain/body relationship is nonlinear, a simple ratio of brain to body is problematic (e.g., small animals have larger ratios than larger animals on average). Encephalization quotients (EQs) are widely used measures of relative brain size that take this empirical relationship between brain and body size into account. They are simply the ratio of a species' actual brain size to the brain size expected for an animal of its body size (Jerison 1973). The expected brain size for a given species is usually derived using a regression (or other method, e.g., reduced major axis) of log brain to log body size for the comparison group of species, resulting in a formula of the type $[\log \text{brain}] = \log c + a[\log \text{body}]$. Jerison (1973) used mammals as the comparison group, but one can estimate EQs, for example, on the basis of primates only. One can also extend the concept to subcomponents of the brain and scale them either against

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[1] I encourage those interested in extended reviews of human brain evolution to consult Allman 1999, Deacon 1997, Falk 1992, Geary 2005, Holloway et al. 2004a, and Striedter 2005.

body size or brain size (see, e.g., Schoenemann 1997).

Because EQs are calculated on the basis of empirical estimates of brain/body-scaling relationships, they are sensitive to the particular sample used to derive *a* and *c* parameters. Jerison (1973) originally estimated the scaling parameter *a* (i.e., slope) for mammals as ~0.67, but Martin (1981) estimated it to be 0.76 using a larger sample. Using Jerison's (1973) equation, human EQs are ~7, whereas Martin's (1981) equation gives the values at ~5. Regardless of the slope estimate used, however, humans consistently have the highest values among mammals.

Figure 1 shows brain/body-size relationships for a sample of 52 primate species and illustrates different scaling estimates for separate primate subtaxa. Primates as a group tend to have larger brains than the average mammal, with EQs for anthropoids (all primates excluding prosimians) averaging ~2 (i.e., anthropoids have brains approximately twice the size of the average mammal of their body size). Even though absolute brain size is significantly larger in pongids (chimpanzee, bonobo, gorilla, orangutan) than in all other anthropoids except humans, they do not have substantially larger EQs, indicating their brains are scaling approximately similar to other anthropoids. Human brain sizes, by contrast, are not explained by brain/body scaling in either mammals or primates.

Although EQs are superficially appealing as a measure of comparative brain size, it is unclear exactly how to interpret EQ differences between species. The behavioral relevance of EQ has long been questioned (Holloway 1966), yet it is often incorrectly assumed that it must be the most behaviorally relevant variable of brain size—as if it were some coarse estimate of intelligence or other behavioral ability (e.g., Kappelman 1996). Others have uncritically assumed that increases in absolute brain size may not be meaningful if EQs remain the same (e.g., Wood & Collard 1999, Wynn 2002). Unfortunately, EQs are not so easily interpreted. If EQ really tells us something about intelligence or general behavioral complexity, what are we to make of the large whales, who have the lowest EQs of all mammals [e.g., humpback whales (*Megaptera nodosa*) have EQs of 0.18 (Schoenemann 1997)]? Humpback whales display a variety of complex behaviors, including structured vocal sequences (songs?) that last 5–25 min before repeating, and complex feeding techniques, including the use of bubble clouds to encircle prey (Rendell et al. 2001).

Other species comparisons further highlight the problem: Guinea pigs (*Cavia cutler*) have significantly higher EQs (0.95) than do elephants (*Loxodonta africana*, 0.63). This is true even though guinea pig brains weigh only ~3.3 g, whereas elephant brains can weigh over 5700 g (Schoenemann 1997). If EQ really tells us something important about behavior, this should be evident in a comparison of guinea pig versus elephant behavior, yet this does not appear to be the case. If anything, given the variety of complex social behaviors known in elephants (e.g., McComb et al. 2001), absolute brain size appears more behaviorally relevant in this comparison.

In fact, a number of studies suggest that—for some behavioral domains—absolute brain size is more relevant than EQ. This is true for measures of the ease of learning abstract rules as opposed to simple associations (Rumbaugh et al. 1996), as well as the speed of learning object-discrimination tasks (Riddell & Corl 1977). In addition, although relative brain size is often emphasized in brain/behavior studies, associations are invariably also significant if absolute brain volumes are used (e.g., Dunbar 1995, Reader & Laland 2002). Jerison (1973) recognized this when he suggested an alternative to EQ: an estimate of extra neurons, which is the absolute amount of neural tissue beyond that predicted empirically by body mass (e.g., two species with identical EQs but different body masses also differ in number of extra neurons). This measure is, however,

Pongids: larger-bodied apes, part of the taxonomic superfamily Hominoidea, which include common and bonobo chimpanzees, gorillas, and orangutans not commonly used in discussions of hominid brain evolution.

The incorrect assumption that absolute brain size is not particularly behaviorally relevant may stem from the fact that, because brain size scales with body size, it is assumed growth of the two must be tightly constrained together developmentally (e.g., Finlay et al. 2001). Such developmental constraints would require that a larger-bodied species have a larger brain, so simply comparing species in absolute brain size would improperly conflate brain size with body-size differences. The problem with this view is that brain/body scaling does not, in fact, necessarily imply developmental constraint. Although the correlation between brain and body size is high (typically $r > 0.95$), at least a tenfold range of variation in absolute brain size exists at a given body size in mammals (Finlay et al. 2001, Schoenemann 1997). Thus, any developmental constraint would not appear to be strong. In addition, it is not often recognized that brain/body scaling could be the result of body size constraining brain size, rather than the brain and body being tightly developmentally linked. Jerison's (1973) explanation for the brain/body relationship is that larger bodies need larger brains both to control greater muscle mass and to process greater amounts of sensory information. Other explanations emphasize the limiting role of metabolic resources in brain growth across species (Armstrong 1983, Martin 1981). In either case, absolutely larger brains might always be adaptive if they can be paid for metabolically (and hence ecologically). Because brains are metabolically expensive [i.e., having high metabolic rates per gram of tissue (Aiello & Wheeler 1995, Hofman 1983b)], brain sizes tend to vary at a given body size as a function of the usefulness of brains for a particular species niche. Larger animals generally have larger brains because they have more metabolic resources available to put toward brain development and maintenance, not because of developmental constraints (Schoenemann 2004).

Under this model, body size varies according to a variety of ecological constraints and tends to put upper limits on possible brain size, which in turn tend toward the high end, particularly in social species (see below). The human condition is explained by behavioral adaptations that lead to a relaxation of ecological constraints. This behavioral-selection/body-size-limiting model of brain-size evolution is more consistent with the wide range of brain sizes at a given body size in mammals, and it also explicitly does not assume that EQ is the only behaviorally relevant comparative measure. In addition, this model is consistent with the finding that two genetic loci known to be important to brain size development, ASPM and microcephalin, both show signatures of strong selection specifically at the evolutionary divergence of the line leading to pongids and hominids (away from all other primates) even though pongids do not show an increase in relative brain size over other primates (as noted above) (see Evans et al. 2004, Wang & Su 2004).

In summary, brain size does scale with body size, and EQ differences do suggest something about the relative importance brains have had during a species' evolutionary history, but they should not be used uncritically as proxies of species' behavioral capacities. That brain size scales with body size across mammals does not constitute strong evidence of developmental constraints tying the two together. Absolute brain size itself appears to be behaviorally relevant.

Patterns of Internal Brain Allometry

Larger brains have more neurons (Haug 1987), but for these neurons to remain equally well connected to each other (in the sense of a signal having the same average number of synapses to traverse to travel between any two neurons), the number of connections (axons) must increase much faster than the number of neurons (Ringo 1991). Hofman's (1985) data show that white matter increases faster than gray matter with increasing brain size. (White matter contains longer-distance axonal connections between cortical areas, whereas gray matter contains most neuronal cell bodies and dendritic connections.) However, the increase is not fast enough to maintain equal degrees of connectivity between neurons (Ringo 1991). As brain size increases, therefore, there is a concomitant increase in the separation between existing areas, leading to a strong correlation between brain volume and the number of distinguishable cortical areas across mammals (Changizi & Shimojo 2005). These scaling relationships predict ~150 distinct cortical areas in humans. Although the human cortex is not yet completely mapped, this predicted number is broadly consistent with what is thought to be the actual number (Van Essen et al. 1998).

HUMAN BRAINS IN COMPARATIVE PERSPECTIVE

Understanding human brain evolution requires the assessment of exactly how human brains differ with respect to the size of various components from those of other living animals, particularly our closest evolutionary relatives: the pongids. Empirically mapping all the possible differences across many species is an extraordinarily time-consuming task, and at present we are nowhere near a complete understanding of all the differences that may exist. Below I review the brain components that have been studied in enough detail to give at least preliminary assessments about the relative status of human brains. The neuroanatomical variables studied to date are either relatively easy to measure or have been thought to be particularly interesting behaviorally. Because conscious awareness is localized to areas of the cortex (the outermost layer of gray matter of the cerebral hemispheres), much comparative research has focused on cortical subdivisions. In some areas, only the size of an entire cortical lobe has been estimated in enough species to allow any comparison; in other areas, studies exist of more-localized (and presumably functionally specific) areas.

Because humans differ from other species on a number of interesting behavioral dimensions (e.g., communication, ability to harness technology, problem solving, complexity of social relationships; see below), and because the neural processing underlying these is often located in different brain regions, there is no general agreement about what components are most important to study a priori. For this reason, this review covers most of the components for which information is available. These include overall brain size, olfactory bulb, cerebellum, visual cortex, temporal lobe, and the overall frontal cortex and its components (primary motor, premotor, and prefrontal cortices). Because of space limitations, noncortical areas other than the cerebellum (e.g., deep cortical and brainstem nuclei) are not discussed here. Behavioral implications of anatomical differences are reviewed in the subsequent sections. Our knowledge of anatomical differences is further advanced than our knowledge of what these differences might mean behaviorally. In general, however, it is generally assumed, implicitly or explicitly, that more tissue translates into greater sophistication in neural processing in some way, which in turn suggests increased complexity of the behaviors mediated by that particular area (or areas). Actual direct tests of this assumption are relatively rare, however.

Brain Size

The most obvious evolutionary change during human evolution, as noted above, has been an increase in both absolute and relative brain size (Holloway 1995). Estimated brain sizes of our closest living relatives, the pongids (large-bodied apes), are as follows: common chimpanzee (*Pan troglodytes*), 337 (±16) cc; pygmy chimpanzee (*P. paniscus*), 311 (±11) cc; gorilla (*Gorilla gorilla*), 397 (±67) cc; and orangutan (*Pongo pygmaeus*), 407 (±29) cc (Rilling & Insel 1999). Modern human brain sizes vary widely, but average ~1330 cc (Dekaban 1978, Garby et al. 1993, Ho et al. 1980a, Pakkenberg

& Voigt 1964).[2] Modern human brains are 3.1 times larger than predicted on the basis of primate brain/body-size allometric scaling (Schoenemann 1997).

Olfactory bulb. The olfactory bulb is the first major processing area for the sense of smell. Stephan et al.'s (1981) data show that the olfactory bulb is only ∼30% as large as predicted for primate brains of our size (Schoenemann 1997). Because overall brain size is approximately three times larger in modern humans, this suggests the olfactory bulb has lagged behind overall brain-size evolution. This finding is consistent with the belief that olfaction is relatively poor in humans, comparatively. However, the human olfactory bulb is ∼1.6 times larger than expected for a primate of our body size (Schoenemann 1997). Exactly how one should interpret differences relative to body size versus differences relative to brain size is a major unresolved issue. It would appear that olfaction is not unimportant.

Cerebellum. The cerebellum plays a key role in modulating patterns of muscle movements and appears to play a role in timing generally. It may be involved in aspects of language processing as well (Gazzaniga et al. 1998). The human cerebellum is ∼2.9 times as large as expected for a primate of our body size (calculated from data in Stephan et al. 1981) and as such has increased only slightly more slowly than the brain as a whole. MacLeod et al. (2003) have shown that there is a grade shift in hominoids with respect to the size of the cerebellar hemispheres, with hominoids as a group (humans included) showing greatly enlarged cerebella compared with monkeys. The cerebellum's participation in language presumably explains why it has not lagged behind as has the olfactory bulb, for example.

Visual cortex. The visual cortex is so named because it is the site of the initial conscious processing of visual information. It is located in the occipital lobe, which is the most posterior portion of the cortex. The human primary visual cortex (the initial cortical area devoted to processing visual information) is only ∼60% the size it should be for a primate brain that size (Holloway 1992), but it is ∼1.5 times larger in absolute terms than it is in chimpanzees (Stephan et al. 1981). The human primary visual cortex is 5% larger than expected given a primate of our body size (Deacon 1997, Schoenemann 1997). It is not clear whether this says anything about relative visual processing abilities in humans. However, given that the human primary visual cortex is smaller in relative terms, but larger in absolute terms, it could be used to test which is more behaviorally relevant: If humans have behavioral advantages over apes in the visual domain (specifically in those known to be mediated by the primary visual cortex), it would suggest that absolute amounts of neural tissue would be more important than relative amounts, at least for visual processing. Such a study has not been done.

Preuss & Coleman (2002) have documented a variety of changes of the neurons in the primary visual cortex in humans, particularly a population of interneuronal connections in layer 4a that appears to have expanded significantly in human evolution. The exact behavioral significance is not known, but these changes emphasize that human-brain evolution involved more than simple changes in size (relative or absolute) of brain regions.

Temporal lobe. The temporal lobe plays a critical role in auditory information, as well as memory (through the hippocampal formation and associated areas), emotion (amygdala), and conceptual understanding (Carpenter & Sutin 1983). As a result, it also plays an important role in language processing. Rilling & Seligman (2002) report that humans have significantly larger overall volumes, white matter volumes, and surface areas of their temporal

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[2] Original data in grams was converted to estimated cc using the formula (brain volume in cc) = (brain mass in grams)/1.036. European-derived samples only.

lobes than predicted on the basis of ape scaling relationships. This suggests an elaboration in humans of the behaviors mediated in this lobe.

Frontal lobe. The frontal lobe consists of all cortical areas anterior to the central sulcus (which angles inferoanteriorly to superoposteriorly along the midlateral convexity of the cortex on both hemispheres).[3] It contains a number of different functional areas, including the primary motor area (also known as Brodmann's area 4 to neuroanatomists, located immediately anterior and adjacent to the central sulcus), which directly controls conscious muscle movements; the premotor area (known as Brodmann's area 6, located immediately anterior to the primary motor area), which plans complex muscle-movement sequences; and the prefrontal cortex (everything anterior to the premotor area), which mediates a number of higher cortical functions important for planning, language, and social interactions, as well as having a general executive oversight of other brain regions (see below). A number of studies have quantified the entire frontal cortex (which is relatively easy to delineate across species), without subdividing it into its functional subdivisions. Overall, the frontal lobe in humans appears to be as large as expected, given a primate brain of our size (Bush & Allman 2004, Semendeferi et al. 2002, von Bonin 1963). Semendeferi et al. (2002) report that human frontal cortex averaged 37.7 (±0.9)% of the entire brain, compared with 35.4 (±1.9)% for common chimpanzees, 34.7 (±0.6)% for bonobo, 36.0% for gorilla, and 37.6 (±1.1)% for orangutan.

However, recent data suggest a difference with respect to gray matter/white matter proportions of the frontal lobe, which suggest potentially important behavioral implications. Schenker et al. (2005) report that humans have significantly more white matter volume in areas close to the cortical surface than hominoid (pongids plus the smaller-bodied apes such as gibbons and siamangs) data predict. Calculating from their data (Schenker et al. 2005), human frontal cortical gray matter is 3.6 times larger than the average for their pongid sample, but human frontal gyral white matter is 4.7 times larger. This suggests a bias toward white matter expansion in humans, although the extent to which this is explained statistically by allometric-frontal/nonfrontal (or some other) scaling cannot be determined (e.g., total brain sizes are not reported). Because allometric explanations have neither straightforward behavioral implications nor developmental-constraint implications, as discussed above, it is not clear what an allometric explanation would mean in any case. However, it may reflect increased functional distinctions of areas within the frontal lobe. In any case, the frontal lobe is, at a minimum, more than three times larger than it is in pongids, and this likely has important behavioral implications of some kind. To understand what these might be, it is useful to look at specific subdivisions of the frontal lobe.

Primary motor and premotor areas. Although the sample sizes are small ($N = 7$), the primary motor area in humans appears to be only ~33% as large as predicted for a primate brain our size (Blinkov & Glezer 1968). This suggests our primary motor cortex has scaled approximately with absolute body size during human evolution (Deacon 1997). Given that this area mediates the direct conscious control of muscle movements, and given that humans do not seem particularly gifted or particularly poor comparatively with respect to muscle control, this approximate scaling with body size (but not brain size) argues against the importance of relative amounts of neural tissue for behavioral ability, at least for this area.

The human premotor area, just anterior to the primary motor area, also appears smaller than predicted given absolute brain size, although not to the same extent. The premotor

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[3] The surface of the cortex is not smooth but is folded back and forth upon itself, resulting in patterns of indentations referred to as sulci (singular, sulcus) separated by ridges referred to as gyri (singular, gyrus).

area is ∼60% as large as predicted for a primate brain our size (Blinkov & Glezer 1968).[4] This suggests the premotor area has not lagged as far behind as the primary motor area as brain size increased. Taken together, these two findings suggest an elaboration of motor planning but not an increase in motor control per se. No direct tests of these suggestions have been reported, however.

Prefrontal. If the entire frontal lobe is approximately as large as expected given overall human brain size, yet the two portions of the frontal lobe reviewed above (the primary motor and premotor areas) are significantly smaller, then the rest of the frontal lobe (i.e., the prefrontal) must necessarily be larger than expected (Preuss 2000). There is nevertheless currently some controversy over this point. Brodmann's original cytoarchitectural studies, which form the basis for much of our knowledge of cortical areas, strongly point to significantly larger prefrontal cortices in humans compared with other primates (Brodmann 1909). Allometrically, Brodmann's data suggest the human prefrontal is ∼2 times larger than predicted on the basis of the size of the rest of the brain (Deacon 1997). The suggestion of biased expansion is also supported by research quantifying the degree of folding in different parts of the cortex: Humans appear to have substantially more convoluted (and hence, a greater volume of) cortex in prefrontal regions (Armstrong et al. 1991, Rilling & Insel 1999).

Studies using magnetic resonance imaging (MRI) to quantify prefrontal cortex also support this contention. Because the posterior boundary of the prefrontal cortex does not follow obvious sulcal/gyral gross morphological features, it is not possible to exactly delineate it using structural MRI. However, a proxy for prefrontal cortex volume can be used: cortical volume anterior to the corpus callosum (the major tract of white matter connecting the two hemispheres). This proxy is commonly applied to both human (e.g., Raz et al. 2005, Sax et al. 1999) and nonhuman primate (Lyons et al. 2002; variant in McBride et al. 1999) studies. Estimated in this way, human prefrontal cortex was shown to be significantly larger than in pongids (Schoenemann et al. 2005b). Specifically, human values averaged 12.7% of total brain volume, compared with an average of 10.3% for the four pongid species. Nonprefrontal cerebral volume in humans averaged 3.7 times larger than the average of *P. paniscus* and *P. troglodytes*, but the prefrontal portion averaged 4.9 times larger.

Assessing both Semendeferi et al.'s (2002) data on the total frontal lobe and our own data, it appears that if the analysis is restricted to increasingly anterior regions of the frontal (keeping in mind that the prefrontal occupies the most-anterior portions of the frontal lobe), humans appear increasingly disproportionate. With respect to allometric scaling, total human frontal cortex is slightly smaller than predicted (Semendeferi et al. 2002), but the prefrontal (using our proxy) is slightly larger than predicted (Schoenemann et al. 2005b). Given that the prefrontal proxy used appears to underestimate human values much more so than other primates (Schoenemann et al. 2005a), this strongly suggests that human prefrontal is in fact larger, both as a percentage of total brain volume, as well as allometrically. Our data do not allow a clear confirmation of whether human prefrontal cortex is actually twice the predicted size, however.

In addition, the human difference appeared biased toward white matter rather than gray matter (Schoenemann et al. 2005b). Comparing humans with chimpanzees, prefrontal gray volumes averaged 4.8 times larger in humans, whereas nonprefrontal gray

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[4] Semendeferi et al. (2002) report that human precentral gyrus volumes (expressed as percent of total cortex; i.e., not allometrically) fall within the range of their hominoid sample. However, because the precentral gyrus does not contain all of the motor cortex, and only contains a small portion of the premotor cortex, it is not clear exactly what this suggests about either motor or premotor cortex size in humans.

volumes averaged 4.2 times larger. By contrast, prefrontal white volumes averaged 5.0 times larger in humans, whereas nonprefrontal white volumes averaged only 3.3 times larger. Furthermore, the human average value was significantly allometrically larger as well, although not to the extent found in Brodmann's original data. Sherwood et al. (2005) point out that prefrontal white volume is predicted by prefrontal gray volume in our dataset, suggesting that the difference appears to be in proportions of prefrontal versus nonprefrontal. Given that a critically important role of the prefrontal is to moderate activity in posterior cortical areas, this apparent shift in proportions likely has important behavioral implications, as discussed below.

Additional support for the biased expansion of the prefrontal comes from work morphing primate brains into human brains. Deformation maps describing the necessary transformations allow for detailed, global assessments of morphological differences. Comparisons between humans and bonobo (*P. paniscus*) (Zilles 2005), common chimpanzees (*P. troglodytes*) (Avants et al. 2005), and macaque monkeys (Van Essen 2005) have been reported. In all three cases, substantial increases in the prefrontal region were reported. Our own group found that the average common chimp–human difference was approximately twofold for some prefrontal areas (Avants et al. 2005). To date, these studies involve comparisons between only two species, so scaling trends across primates cannot be estimated. However, it should be possible to extend these analyses to multiple species comparisons, resulting in separate allometric-scaling estimates—and extent of human divergence, if any—for individual areas at high resolution. Furthermore, the morphing algorithms can be applied to collections of cell-stained brain sections, thereby combining the resolution of detailed cytoarchitectural analyses (studies of patterns of neurons in the cortex) with the ability of morphing algorithms to quantify changes in shape.

Within the prefrontal itself, studies suggest a mosaic of evolutionary changes. A cytoarchitectural study of Brodmann's area 13, a subdivision of the prefrontal that mediates aspects of social behavior (particularly emotional dimensions), suggests that this area lagged behind the expansion of the brain as a whole: It is only 1.5 times larger than the average pongid value (Semendeferi et al. 1998). By contrast, another subdivision of the prefrontal, Brodmann's area 10, which is known to mediate tasks involving planning and organization of thought and future behavior (Carpenter & Sutin 1983), is 6.6 times larger in humans than in pongids (Semendeferi et al. 2001). Holloway (2002) notes this is actually only slightly more than one would predict given how area 10 scales with brain size. This is because the relationship is strongly positively allometric (i.e., as brain size increases, area 10 seems to increase much faster).

How should we interpret these findings in subareas of the prefrontal? First, as discussed above, even if allometry statistically predicts some increase in humans, this does not license us to conclude that the increase is behaviorally irrelevant (Schoenemann et al. 2005a). Nor does the existence of allometric scaling constitute a demonstration of the existence of inherent developmental constraints. Larger brains may have larger prefrontal cortices (or subregions therein) because selection places greater demands on the oversight role of prefrontal areas as posterior regions become more complex. As discussed above, absolute amounts of cortical tissue have been shown to be correlated with a variety of behavioral dimensions. Thus, even though area 13 is relatively smaller than one would predict, it might nevertheless indicate an important behavioral change, particularly given the enhanced interactive sociality that has increasingly characterized the human condition (see below). Increases in area 10 almost surely suggest increased importance in the various dimensions of behavioral planning.

FOSSIL EVIDENCE OF HUMAN BRAIN EVOLUTION

What is known about the evolutionary history of these apparent differences in brain anatomy? The goal of research in this area is to determine exactly what can be inferred about the behavior of fossil hominids from imprints on the inside surface of their braincases. This is of central importance to understanding human brain evolution. Because of this intrinsic interest in the behavior of fossil hominids, a great deal of effort has been spent trying to extract maximal inference out of minimal data. Because brains do not fossilize, the richness of comparative data is always greater than the fossil data. The following subsections highlight these limitations and serve to illustrate the inherent difficulty of the task. At present, the fossil record is clearest for overall brain size as indexed by cranial capacity, although other suggestive clues have been found regarding possible early changes in different brain regions related to visual processing and language.

Cranial Capacity

Cranial capacity is by far the most well-attested change in human brain evolution in the fossil record. **Figure 2** plots cranial capacity estimates against estimated specimen age for published hominid fossils dating from 15 KYA to 4 MYA, a total of 145 individual specimens. It is apparent from **Figure 2** that changes in hominid brain size began sometime between 3 and 2 Mya.

Although it has been argued that brain size has undergone punctuational (i.e., not continuous and gradual) change at various points during hominid evolution (e.g., Hofman 1983a), **Figure 2** suggests the range of variation at any particular point in time (best exemplified in modern species) combined with the sparse sampling evident for particular time periods of our evolutionary history (e.g., between 1.0 and 0.5 Mya) indicate that it may be premature to assess the likelihood of punctuational events. Recent empirical analyses of cranial capacity changes over time support this notion (De Miguel & Henneberg 2001, Lee & Wolpoff 2003). Furthermore, it is not clear how important species designations (which are inherently problematic for fossils) are for understanding brain-size evolution. De Miguel & Henneberg (2001) showed that 90% of the variation in fossil hominid brain size can be explained simply by the age of fossils, leaving only 10% to be explained by species differences and/or measurement error. This suggests that most of the brain size increase during hominid evolution was not closely tied to speciation events.

Lunate Sulcus

The portion of the cortex that has received perhaps the greatest amount of attention in fossil specimens is the visual cortex. A sulcus known as the lunate marks the anterior boundary of the primary visual cortex in nonhuman primates, although it is often missing in humans (Allen et al. 2005a, Connolly 1950). In relative terms, an anterior position of the lunate (and therefore anterior extent of the primary visual cortex) is characteristic of the general pongid condition, whereas a posterior placement is characteristic of modern humans. As discussed above, the primary visual cortex is a relatively smaller portion of the human brain in comparison with chimpanzee brains, even though in absolute terms it is ~1.5 times larger than that of either chimpanzees or gorillas (Stephan et al. 1981).

The evolutionary history of this change has been the source of considerable controversy over the years: Did it occur early in hominid evolution (e.g., in early Australopithecines), thereby signaling some form of early reapportionment (Holloway prefers the term reorganization) of neural resources? Or was it simply the result of the primary visual cortex lagging behind as brain size increased during human evolution? Holloway and Falk have, over the years, disagreed on the possible positioning of the lunate sulcus on brain endocasts attributed to both *Australopithecus africanus* (Taung) and *A. afarensis* (AL 162–28, 3.2 Mya) (e.g., Falk 1987, Holloway 1995). Holloway believes the lunate either cannot be seen (Taung) or is likely in a modern human posterior position (AL 162–28) in early Australopithecines, long before substantial changes in absolute brain size occurred. Falk and others have argued that it likely occurred later, as a result of brain size increases in other cortical regions. Debates over the position of the lunate have not been resolved, in part because it is difficult to assess its location unequivocally on endocast specimens and because of disagreements about the proper orientation of the AL 162–28 specimen. Holloway and colleagues (Holloway et al. 2004b) have recently reported on another *A. africanus* specimen, STW 505, which they show appears to have a relatively posterior lunate.

In addition, Holloway et al. (2003) discuss the brains of two apparently normal chimpanzees that nevertheless have lunates in human-like posterior locations. They note this finding demonstrates the possibility of a posterior lunate in the absence of increased brain size, which means Holloway's long-held contention of a posterior shift in early small-brained hominids is certainly possible. However, these specimens also complicate behavioral interpretations of the apparent change. Holloway et al. (2003) argue that the reduction of the primary visual cortex has no behavioral implications. However, if a chimpanzee can have a human lunate pattern (presumably indicating a reduced primary visual cortex), this pattern is not a clear indicator of human-like behavior, and therefore the location of the lunate in fossil endocasts may not be behaviorally interpretable, rendering the lunate sulcus debate moot.

However, Holloway and colleagues (Holloway et al. 2004b, p. 6) argue that a posterior lunate in Australopithecines "indicates an expanded posterior parietal cerebral cortex [anterior to the primary visual cortex], and was most likely associated with enhanced social behavior including communication." Thus cortical expansion anterior to the lunate is argued to have behavioral implications, but reduction posterior to the lunate is argued to be behaviorally meaningless. If the size of the primary visual cortex really is irrelevant, why do humans have 1.5 times as much primary visual cortex, given the evolutionary costs of brain tissue (see below)? If Holloway and colleagues are right about the location of the lunate in AL 162–28 and STW 505 (which they seem to be), it is either behaviorally meaningless, or Australopithecines had reduced visual processing capabilities of some kind. Both these alternatives are problematic. Thus, at this point it is not clear how to interpret the position of the lunate in fossil specimens.

Broca's Area

Evidence relevant to the origin of language is intrinsically of great interest. There is some suggestion of the elaboration of Broca's area (a key cortical region that plays a central role in language processing, located in the left prefrontal portion of the inferior frontal lobe) in fossil hominids. Holloway (1983) noted that a Broca's cap (an endocranial bump over what would be Broca's area) is present—although inconsistently—in pongids. Thus, the presence of a Broca's cap does not definitively indicate the existence of language, unfortunately. Tobias (1975) and Holloway (1983) argue that Broca's cap becomes increasingly present in early *Homo*, however (see also Broadfield et al. 2001). Tobias (1983) argues this indicates early *Homo* had language. Both Falk (1983) and Holloway (1995) agree that the endocast of the early *Homo* specimen KNM-ER 1470 (1.8 Mya) looks more modern-human-like in the inferior frontal region. Although this does not prove that KNM-ER 1470 had a Broca's area or that it had language, it nevertheless suggests something important is occurring in this region. Broadfield et al. (2001) report that the specimen Sambungmacan 3 has asymmetrical Broca's caps, which they speculate may indicate a level of language ability beyond

Petalias: areas where the brain extends farther in some direction for one hemisphere over another

that found in earlier hominids. This specimen is assumed to be Middle Pleistocene *Homo erectus*, although it is unfortunately not well provenienced (Marquez et al. 2001). Thus, suggestive (but equivocal) evidence exists of possible language-related changes in the brains of early *Homo*, dating back to almost 2 Mya.

Asymmetry

Because important aspects of behavior are asymmetrically organized (e.g., key components of language are usually processed in the left hemisphere, which is usually also the dominant hemisphere for hand movements), it is of interest to determine whether cortical asymmetries can be found that predict these behavioral asymmetries, thereby possibly allowing us to infer behavior from fossil specimens. Research to date has centered on particular anatomical asymmetries known as petalias. Holloway & de la Coste-Lareymondie (1982) report that only modern and fossil hominids (including australopithecines, *H. erectus*, and Neanderthals) show a consistent, distinct right-frontal and left-occipital petalial pattern. Pongids also show petalias, but they did not display the same degree of consistency, particularly in the combination of right-frontal and left-occipital petalias. Only 25% of pongids assessed displayed this pattern, compared with 82% of hominids. Falk et al. (1990) demonstrated that Rhesus monkeys (*Macaca mulatta*) show a significant tendency toward right-frontal petalias (although not to the same consistency as found in modern and fossil hominids) but not for left-occipital petalias. Thus it appears that petalias in general are common in anthropoids, but the particular pattern of petalias and the degree of consistency appear unique to hominids.

Holloway & de la Coste-Lareymondie (1982) speculate this right-frontal/left-occipital petalial pattern may be related to right-handedness, language-related symbol manipulation, and spatio-visual integration. However, because the hominid pattern occurs in a fair number of pongid specimens, the functional significance of this finding is obviously not clear. It cannot be considered definitive evidence of any particular behavior in individual specimens.

Summary of the Fossil Evidence

It is clear that brain evolution started in earnest sometime between 2 and 3 Mya. Although punctuated models of brain size increase can be fit to the data, there is no compelling reason to assume anything other than a reasonably constant trend toward increasing brain size over time. Apart from cranial capacity, only suggestive, equivocal clues of possible behavioral patterns are evident in the fossil record of hominid brain evolution, mostly relating to the question of language evolution. Although definitive statements are not currently warranted, we do not presently know the limits of possible inferences about the behavior of fossil hominids from their endocranial remains. The intrinsic interest in reconstructing hominid behavior ensures that every possible avenue of inference will be explored in the future. It is certainly possible that additional associations between cranial form and brain anatomy (and ultimately behavior) will eventually be uncovered using modern morphometric methods.

EVOLUTION OF BRAIN AND BEHAVIOR

Explaining the anatomical changes in the human brain reviewed in the sections above remains a central question in human evolutionary studies. Exactly why these changes happened, and what they might mean behaviorally, is a question of fundamental importance. As with any evolutionary change, there are two basic kinds of explanation: adaptive change (the result of selection) and nonadaptive change (the result of genetic drift). Adaptive explanations are probably the strongest for questions of brain evolution as compared with almost any other biological characteristic. This is partly because of the pattern of changes and partly because of the apparent evolutionary costs involved in the changes, both of which strongly argue for adaptive explanations.

Although genetic drift can lead to the fixation of particular alleles at a single locus by chance, the odds that an evolutionary succession of alleles at a variety of loci (e.g., see Gilbert et al. 2005) leads progressively in a particular direction (e.g., larger brain size) is extraordinarily unlikely. In addition, these changes have occurred in the face of apparently strong evolutionary costs (Smith 1990). Costs in this context refer to correlated effects that—everything else being equal—ultimately translate into decreased numbers of offspring produced per unit time. These costs have to be paid every generation. First, as noted above, the brain is among the most metabolically expensive organs in the body [i.e., neural tissue has among the highest metabolic rates per gram (Aiello & Wheeler 1995, Hofman 1983b)]. Second, increasing brain size in primates is strongly correlated with longer gestation periods, an increased period of infant dependency, and delayed reproduction (Harvey & Clutton-Brock 1985), which all decrease the number of offspring an individual can produce per unit time. Third, there is an apparent trade-off in bipedal hominids between locomotor efficiency and ease of childbirth (Lovejoy 1975). Fourth, there is potentially a problem of cooling a larger brain (Falk 1990). If there were no counterbalancing advantages to larger amounts of brain tissue, individuals with smaller brains would necessarily have a selective advantage.

For adaptive evolutionary change to occur, reproductive benefits must have consistently (or at least on average) accrued to individuals—within successive populations connecting ancestral and descendent populations—who varied anatomically from the average of their populations. Of course, brain size may not have uniformly and gradually increased in every successive population. If so, there would have been some ancestral populations in which individuals with larger-than-average brain sizes did not have reproductive advantages. [This might explain the possible decrease in brain size from Neanderthal to modern humans (Schoenemann 2004).] However, on average over our evolutionary history, reproductive benefits needed to have been at least marginally greater for larger-brained individuals.

Brain Evolution Through Behavioral Selection

> *As the various mental faculties gradually developed themselves the brain would almost certainly become larger. No one, I presume, doubts that the large proportion which the size of man's brain bears to his body, compared to the same proportion in the gorilla or orang, is closely connected with his higher mental powers.*
>
> Darwin 1871, p. 145

Given that larger brain sizes must have provided individuals with reproductive advantages during significant portions of our evolutionary history, it is reasonable to ask both (*a*) what exactly the advantages were, and (*b*) whether these advantages still exist within modern humans (and/or other living primates). Although Gould (1981) suggested that variation in brain size is behaviorally meaningless in modern populations, it is not at all clear why we should expect brain size above some threshold to have absolutely no advantage. The simplest a priori model would posit a behavioral benefit (or usefulness), on average, to larger brains that would be present regardless of whether the particular benefit translated into reproductive advantages in any given environment. The behavioral benefit might be, for example, greater memory ability, planning ability, or linguistic ability. Whether such benefits were evolutionarily meaningful depends on the degree to which they helped pay (reproductively) for the evolutionary costs associated with larger brains. This, in turn, depends on the specific ecological and social conditions characteristic of a given

Genetic correlation: occurs when changes in the genetic effects on one trait have the effect of changing the other trait

Heritability: the proportion of phenotypic (observable) variance of a characteristic in a population that is explained by genetic variance in that population

population. An increasingly powerful linguistic processor is likely not worth the costs for a species that is not highly socially interactive, for example. More complex models regarding costs and benefits of increasing brain size are of course possible (e.g., wildly nonlinear relationships between behavioral abilities and brain size). However, parsimony requires investigating simpler explanations first.

For selection on some behavioral ability to cause evolutionary changes in brain anatomy, there must necessarily have been a genetic correlation between them. Failing this, selection on behavior would have no evolutionary effect on brain anatomy, and we would lack an explanation for evolutionary changes in the brain. Attempts to assess such brain/behavior correlations are therefore central for any model that posits the evolutionary importance of behavior in human brain evolution.

It is also not generally appreciated, however, that the genetic correlation between behavior and brain size does not actually have to be large to explain human brain-size evolution (Schoenemann et al. 2000). This is partly because the rate of change in brain size per generation is only ~8 mm^3 (assuming a 1000-cc increase in brain size over 2.5 million years and a 20-year average generation length) (see **Figure 2**), or approximately 0.0002 standard deviations of chimpanzee-brain size (see also Holloway et al. 2004a). The size of the genetic correlation needed to account for this amount of change per generation is partly a function of the strength of selection operating on the hypothesized correlated (directly selected) variable (e.g., some behavioral ability), as well as of the extent to which both brain size and the selected variable are genetically influenced, or heritable (Falconer 1981).[5] If we posit a small genetic correlation of only $r = 0.05$ and heritabilities (i.e., the proportion of phenotypic variation in a population explained by genetic variation) of 0.3, the selected individuals (i.e., those who contribute to the subsequent generation) only have to differ from their overall population average by less than 0.006 standard deviations on the hypothesized directly selected characteristic (Schoenemann et al. 2000). This is so small that it would be extremely difficult to demonstrate empirically. Although the strength of selection could have varied over time during our evolutionary history, these estimates clearly demonstrate that we do not need to hypothesize more than a weak genetic correlation to explain the dramatic change in brain size via selection on correlated behavioral characteristics.

This does not mean, however, that the genetic correlation must therefore be small and that there is no justification for assessing genetic correlations within humans without massive sample sizes. It simply shows that it need not be large. Because of the evolutionary costs described above, it is highly unlikely that the correlation was in fact zero (or negative), as that would require some unknown nonbehavioral explanation for increasing brain size.

Evidence of Genetic Correlations Between Brain and Behavior

There is substantial evidence that many features of brain anatomy are influenced by genetic factors. In a number of studies, researchers have reported heritability of brain size to be significant, with estimates ranging from 0.66 to 0.94 (reviewed in Winterer & Goldman 2003). Heritability estimates of the patterns of sulci and gyri on the cortical surface are generally significantly lower, although this may be an artifact of the difficulty of quantifying them (Winterer & Goldman 2003). Thompson et al. (2001) estimated independent heritabilities for each voxel (the smallest unit of volume in an MRI image) of

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[5] The strength of selection and the size of the genetic correlation interact to determine the likely amount of change per generation. Assuming reasonable heritabilities, the same amount of change can occur if either selection is strong and the genetic correlation is weak, or if selection is weak but the genetic correlation is strong.

cortical gray matter in a sample of monozygotic and dizygotic twins and found evidence for widespread genetic influence. Volumes of the left and right frontal lobes have estimated heritabilities of between 0.52 and 0.66 (Geschwind et al. 2002), and a number of Brodmann areas (including some prefrontal regions) appear to have at least moderate heritabilities (Wright et al. 2002). Thus, brain anatomy appears to be under genetic influence.

Similarly, cognitive abilities have been shown to have genetic influence. Although intelligence is a controversial concept in some areas of the social sciences, the consensus among those who research individual differences in cognitive ability is that genetic influences on general cognitive ability (*g*)[6] are substantial, although environmental influences are also clearly evident (Neisser et al. 1996). Genetic influences have also been demonstrated for other cognitive domains, such as verbal and spatial factors, although the degree to which these genetic factors are independent of *g* is not always clear (Plomin et al. 1997).

Although there is evidence of genetic influences on both brain and behavior, to what extent are these influences genetically correlated? A large number of studies reported finding phenotypic (not genetic) correlations between brain size and behavioral ability (usually *g*, or other IQ-related abilities). Obviously, our ancestors were not selected to do well on modern IQ tests, but some of the abilities tapped by these tests possibly were selected for. Studies using MRI to estimate actual brain size report correlations averaging $r = \sim 0.45$ (Rushton & Ankney 1996).

However, these are phenotypic correlations, not genetic correlations, and are therefore not necessarily evolutionarily relevant. Phenotypic correlations could be caused by a third variable affecting both the brain and behavior (or other hypothesized evolutionarily relevant correlate) in similar directions. Socioeconomic status, for example, is correlated with both IQ scores (Herrnstein & Murray 1994) and growth of the body (Bogin 1999), and one might therefore predict a spurious correlation between brain size and IQ on this basis. In addition, cross-assortative mating can result in phenotypic correlations between characteristics that actually have completely independent genetic influences (Jensen & Sinha 1990). If the correlation between brain size and some behavior is spurious, selection on that behavior cannot explain evolutionary changes in brain size. It is therefore critically important to assess the strength of the genetic correlation, not just the phenotypic correlation, between the brain and behavior.

One way to control for possible confounds is by assessing the strength of the association between brain size and behavior among siblings within families. Family members share essentially the same socioeconomic status (this can be directly assessed), and meiosis ensures siblings contain random associations of alleles at different loci, thereby eliminating cross-assortative mating effects. To date, only a few studies have used this methodology. Two within-family studies using head circumference as a proxy for brain size have reported equivocal results (Jensen 1994, Jensen & Johnson 1994; see discussion in Schoenemann et al. 2000). Only a few MRI studies have been reported to date. Our own group found essentially no correlation ($r = -0.05$, NS) within families between brain size and an estimate of *g* in 36 pairs of adult sisters (Schoenemann et al. 2000). Another study of male-sibling pairs reported a nonsignificant but positive within-family correlation of $r = 0.23$ between brain size and *g* (Gignac et al. 2003). In addition, a recent study using a genetically informative cohort of 24 monozygotic pairs, 31 dizygotic pairs, and 25 additional nontwin siblings estimated a genetic correlation between *g* and both gray and white matter volumes of $r = 0.29$ and

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[6] Performance on a wide variety of cognitive tests is correlated. *g* is a measure that statistically explains these correlations and as such is typically interpreted as a measure of general cognitive ability. IQ tests are good measures of *g*.

$r = 0.24$, respectively (both $p < 0.05$) (Posthuma et al. 2002). The genetic correlation for overall brain size was not reported, unfortunately, but was presumably similar (given that gray + white = total brain).

One unresolved issue is the direction of causality. It has long been known that environmental influences can affect brain volume, specifically cortical gray matter volume in rats (Diamond 1988). This means that genes with higher *g* could be causing individuals to experience more-stimulating and complex environmental situations, which may in turn cause developmental (not genetic) increases in their brain sizes and cortical gray volumes (Posthuma et al. 2003). If this were the sole reason for the brain size/*g* correlation, then selection for *g* is not likely the explanation for our increased brain sizes during our evolutionary history because it implies brain-size variation is solely a developmental response. However, some of the brain size/*g* correlation is likely explained through *g* influencing brain size, which means that the evolutionarily critical association—in which brain size causes (or allows for) changes in *g*—is likely smaller than the $r = 0.24$ to $r = 0.29$ genetic correlations suggested by Posthuma et al.'s (2002) study.

Taken together, these studies suggest the genetic correlation between brain size and *g* is not zero but is not as large as the phenotypic correlations typically reported for MRI studies of brain volume and *g* mentioned above. The proportion of genetic variance in *g* explained by genetic variance in brain volume appears substantially less than 8.5%. This is still large enough to explain the evolution of brain size in hominids, however.

Other behavioral measures. Although most research has focused on correlations with behaviors associated with general cognitive ability, natural selection may have acted on other unrelated behavioral dimensions as well as (or instead of) general cognitive ability. Associations between neuroanatomical variation and other behavioral dimensions that are conceivably evolutionarily relevant have also been found, including spatial ability, working memory, and the ability to extract relevant information from a distracting environment.

Spatial ability has been the focus of research partly because it was proposed to help explain sex differences in brain size, which average ~1 standard deviation (Ho et al. 1980b). Some but not all of this difference is explained by body weight (Ankney 1992, Falk et al. 1999) and/or fat-free weight (Schoenemann 2004). Because males and females differ on average in spatial ability, in Western populations at least (Halpern 1987), if spatial ability correlates strongly with brain size, the residual sex difference in brain size might therefore be explained. However, many studies, including the more recent genetically informative MRI-based ones, have failed to find a significant association between brain size and spatial abilities (Posthuma et al. 2003, Schoenemann et al. 2000, Wickett et al. 2000).

Spatial-ability differences still may explain differences in the shape of the corpus callosum, which in women appears to be larger in the posterior region (e.g., Davatzikos & Resnick 1998, de Lacoste-Utamsing & Holloway 1982). This portion of the corpus callosum, known as the splenium, connects areas of the parietal lobes known to mediate spatial tasks. Thus, Holloway and colleagues (Holloway et al. 1993) have suggested the anatomical difference in corpus callosum morphology may therefore be explained by sex differences in spatial abilities. Consistent with this suggestion, our own group has recently found that women with smaller (more male-like) splenia score better on a test of mental rotation–spatial ability (P.T. Schoenemann, A. Dubb, J. Hu, J. Lewis & J. Gee, unpublished manuscript).

Working-memory abilities (i.e., the ability to manipulate information in short-term memory to solve particular problems or goals) appear to be associated with dimensions of brain size ($r = 0.40$, $p < 0.05$ for gray matter; $r = 0.33$, $p < 0.05$ for white matter)

(Posthuma et al. 2003). Because the prefrontal cortex is known to be particularly important to the mediation of working memory (Goldman-Rakic 1996), Posthuma et al.'s finding is of particular interest in light of the possible biased increase in the prefrontal cortex during human evolution discussed above. Working memory abilities have been shown to correlate with the length of the main prefrontal sulcus (principal sulcus) across a variety of Old and New World monkeys, and this correlation completely explains the association between working-memory ability and cranial capacity across these species (Redmond 1999). This raises the question of whether the association found by Posthuma et al. (2003) in humans is actually more properly localized to the prefrontal cortex (which unfortunately was not separately delineated in their study).

The size of the prefrontal cortex in humans has been shown to correlate with the Stroop task within families (Schoenemann et al. 2000). This test measures the extent of linguistic interference in naming colors, when ink color and word name are mismatched (e.g., the word red written in blue ink). It is generally considered a test of the ability to extract (and focus on) the relevant information from an environment and is known to be mediated by prefrontal areas.

The prefrontal cortex also mediates a variety of additional particularly interesting behaviors, including planning (Damasio 1985), memory for serial order and temporal information (Fuster 1985), aspects of language (Deacon 1997), and social information processing (de Bruin 1990). The importance of serial-order memory is generally not appreciated in discussions of human behavioral evolution. One of the clearest behavioral advantages humans have over other organisms is the ability to reconstruct, understand, and utilize causal information. All human technological sophistication is dependent on this ability. In turn, causality is dependent on the ability to remember the serial order of past events. Without this, it is impossible to reconstruct (remember) what actions or behaviors lead to exactly which outcomes. Thus serial-order memory likely played a key role in human behavioral evolution. Although no studies have addressed whether serial-order memory is associated with prefrontal size (or size of some other region), this would seem to be a fruitful direction to pursue.

Brain Size and Conceptual Complexity

A general argument can be made that increasing brain size brought with it an increase in conceptual or semantic complexity (Gibson 2002). Jerison (1985, p. 30) suggested that "[g]rades of encephalization presumably correspond to grades of complexity of information processing. These, in turn, correspond in some way to the complexity of the reality created by the brain, which may be another way to describe intelligence."

A number of observations support this view. First, concepts are instantiated in the brain as webs or networks of activation between different areas (see Pulvermuller 2001). Most of our subjectively experienced concepts are actually complex combinations of sensory information processed in various ways by the different cortical centers. Taste, for example, is actually a complex interaction of olfactory (smell) and gustatory (taste) inputs (e.g., the flavor of a banana is largely olfactory). Similarly, the auditory perception of a phoneme can be altered if it is paired with a mismatched visual input (McGurk & MacDonald 1976). This means there must be networks connecting differing regions as well as areas that mediate the integration of this information.

To what extent is brain size relevant to conceptual complexity? Larger-brained species have more complicated networks of interconnection, thereby leading to greater potential conceptual complexity (Lieberman 2002). It has long been known that certain areas of the body (e.g., the lips and hands) are disproportionately represented in the somatosensory and primary motor areas of the brain and that these match differences in the degree of sensitivity and/or motor control for different parts of our body (Penfield & Rasmussen 1950). Thus, even within species, we have a clear association between the amount of cortical tissue and behavioral dimensions (Gibson 2002). Animals with specific behavioral specializations (e.g., bat echolocation) have correlated increases in areas of the brain known to mediate those behaviors (Krubitzer 1995).

We can then add to this the tendency for larger-brained animals to display greater degrees of cortical specialization: Individual areas tend to be more specific in function and less directly connected to other areas. This is critical to conceptual complexity because it increases the brain's potential to differentiate complex sensory information into diverse constituent parts, thereby helping to magnify subtle differences between different streams of sensory input. The argument can be summarized as follows: Increasing brain size leads to increasingly complex processing within areas, greater degrees of autonomy between areas, and greater complexity of the possible interactions between areas. This leads to a greater complexity of possible network-activation states, which is equivalent to a greater degree of conceptual subtlety and sophistication possible in the organism's representations of reality. Whatever else increasing brain size led to in hominid evolution, it is difficult to escape the conclusion that conceptual complexity increased substantially during this time. Furthermore, given the fundamentally socially interactive nature of humans, as well as the general association between degree of sociality and brain size (discussed below), this increase in conceptual complexity is likely directly relevant to the evolution of language.

Brain Evolution and Language

Although the exact evolutionary changes in the brain necessary to allow for language are not known, language clearly relies on a large number of neural resources. The importance for language of Broca's and Wernicke's areas has been known for more than a century, but it has become increasingly clear that language requires the cooperation of a wide range of cortical areas, including the cerebellum (Gazzaniga et al. 1998), right-hemisphere areas [important for processing logical inferences encoded in language (Beeman et al. 2000)], prefrontal cortex [important for higher-level language functioning (Novoa & Ardila 1987)], and areas of the frontal lobe outside Broca's area (Alexander et al. 1989). Functional brain imaging studies suggest the prefrontal cortex also plays a critical role in conceptual/semantic processing (Gabrieli et al. 1998). All of this indicates that language draws on a wide array of neural resources, which suggests that important features of brain evolution may be explained by the coevolution of language (Deacon 1997).

That language evolution is specifically relevant to brain-size evolution has been suggested many times (e.g., Dunbar 1996, Gibson 2002, Wang 1991, Washburn 1960). Darwin (1871, p. 57) himself argued that, although language use during human evolution likely had effects on the elaboration of the vocal organs, "the relation between the continued use of language and the development of the brain has no doubt been far more important." Brain size, in this view, is itself an index of language evolution. This suggests language has origins that are substantially older than the appearance of anatomically modern *H. sapiens*, which date to perhaps ∼160,000 years ago (White et al. 2003). As reviewed above, the trend toward increasing brain size began sometime before 2 Mya (**Figure 2**). Although there is widespread disagreement about how far back language extends in human evolution (for a review, see Schoenemann 2005), it is difficult to escape the conclusion that language likely played a major role in the evolution of the human brain. The evidence of the relationship between brain size and conceptual complexity at a minimum suggests that fundamental changes in human cognition critical to language evolution began prior to ∼2 Mya.

Sociality and Brain Evolution

Primates in general, and humans in particular, are socially interactive animals. Our ability to survive and reproduce is at least as dependent on successfully navigating social arrangements as it is navigating the physical environment (Holloway 1975, Humphrey 1984). Social interactions are intrinsically complicated, and the complexity increases with increasing social group size. Humphrey (1984) pointed out that the increasing complexity of the social world selects for increasing cognitive sophistication (social intelligence) in individuals, which in turn creates even more complex social interactions. This creates a cycle of ever-increasing social complexity, leading to ever-increasing intellect among individuals—what Humphrey refers to as an evolutionary ratchet. Because of the apparent benefits of being skilled at social manipulation in such an increasingly complex social existence, this has become known as the Machiavellian intelligence hypothesis.

It appears likely that selection for social abilities was an important influence on brain evolution. A number of comparative studies of primates have confirmed an association between measures of brain and/or neocortex size (both absolute and relative) and a variety of measures of social complexity, including mean social group size, social clique size, frequency of reported acts of deceptive behavior, amount of social play (but not nonsocial types of play), and the degree to which male-dominance rank fails to accurately predict mating success (see review in Dunbar 2003, and references therein). All of this is consistent with the idea that brain size is a factor in social ability, broadly defined, although there are glaring exceptions: Orangutans are relatively large-brained but relatively asocial. Clearly, brain size is not a perfect function of social complexity. However, there is no other known behavioral variable that correlates as highly with brain size across species.

Dunbar (1996) has further argued that human language represents a form of social grooming that allowed the increase in group size beyond that otherwise possible. Although there are a number of extrapolations needed to arrive at this conclusion, language clearly serves a highly social function in humans.

Although the comparative evidence that social complexity correlates with brain size is strong, the specific abilities crucial to social ability within humans (or any other species) are not clearly defined or understood. Some people are more social than others, and some social people are better at understanding and/or manipulating social interactions than others. However, there does not appear to have been much research into this question from a neurocognitive and/or neuroanatomical standpoint. Presumably social competence depends on a wide variety of abilities, including language, nonverbal-cue processing, memory (particularly of past interactions and the order of past events), and probably many other basic cognitive abilities. Interestingly, intact prefrontal cortex (unlike other cortical areas) appears to be crucial for the maintenance of high position in dominance hierarchies in monkeys (de Bruin 1990, Myers et al. 1973). Given that this area seems to have undergone disproportionate increase, as discussed above, this appears to be a promising avenue of investigation with respect to brain evolution. Other parts of the brain that appear to be important for social behavior include the amygdaloid nuclei and overlying temporal pole (tip of the temporal lobe) and the posterior medial orbital cortex (including Brodmann's area 13, located in the inferior prefrontal cortex) (Kling 1986). As reviewed above, Brodmann's area 13 shows a moderate increase in absolute terms, although it does lag behind the increase of the brain as a whole. Piecing together the effects the social environment had on modifying the brain during human evolution will likely continue to be the focus of significant research in the future.

Ecological Hypotheses

An alternative (but not mutually exclusive) hypothesis is that adaptation for ecological challenges influences brain evolution. Milton (1981) pointed out that different types of diet vary with respect to the cognitive demands they place on individuals. Fruit is patchily distributed in both time and space, whereas leaves are much less cognitively demanding to obtain. This suggests that species that specialize in fruit (or, more generally, any food source that is cognitively demanding to obtain) are expected to have larger brains than species that do not. Dunbar (1995) did not find a significant association between a measure of relative brain size (ratio of the neocortex to the rest of the brain) and percent of fruit in diet in anthropoid primates. However, Barton (1996) did find a significant association within diurnal haplorhines (specifically, diurnal monkeys and apes) between absolute and relative brain size and percent of fruit in the diet. This discrepancy is possibly a result of a difference in neuroanatomical variables used.

It is also possible that the causality runs the other way: Because larger brains are more metabolically expensive, some sort of dietary accommodation may be necessary to pay for it nutritionally. The expensive-tissue hypothesis (Aiello & Wheeler 1995) argues for a tradeoff between brain and gut size. If larger brains mean smaller guts, then one would predict a higher quality diet. This hypothesis is supported in primates (Fish & Lockwood 2003). It also fits the human case, in which brain size started increasing at about the same time as meat became increasingly important in hominid diets (as indexed by the initial appearance of stone tools).

Tools and Brain Evolution

Is it possible that the cognitive demands of tool making itself spurred brain evolution? Reader & Laland (2002) showed that frequency of tool use in primates is positively correlated with both absolute and relative brain volume. Although early hominid stone-tool industries are not highly complex technologically, they may have required cognitive abilities beyond that shown by apes, for example, in the sequencing of required actions (Toth & Schick 1993). A preliminary functional brain–imaging study of stone-tool manufacturing suggests the activation of cortical areas mediating spatial cognition, as well as motor, somatosensory, and cerebellar areas (as might be expected given the nature of the task), although prefrontal areas known to be relevant to planning were not significantly activated (Stout et al. 2000). If this finding can be replicated, given that spatial ability does not appear significantly correlated with brain size in modern humans (as discussed above), it may argue against early stone-tool manufacturing specifically spurring brain-size evolution (although the spatial abilities tested involved paper-and-pencil tests, rather than hands-on, three-dimensional manipulation as in the stone-tool study). Research on the possible importance of stone-tool manufacturing is clearly in its infancy, and future functional-imaging studies are needed to clarify the issue.

It has also been suggested that the development of accurate throwing might have spurred brain evolution. Calvin (1983) pointed out that human throwing accuracy requires timing abilities (for the release of the thrown object) that far exceed the probable timing accuracies of neurons (judging from measurements of the intrinsic variability in neuronal signals). He pointed out that increasingly accurate timers could be built by putting greater and greater numbers of even inaccurate neurons in parallel in a timing circuit. He also suggested that, given the cortical areas in the primary motor cortex controlling the mouth and tongue (involved in language) and the hand (involved in throwing) were reasonably close to each other, selection for throwing ability may have led to changes that preadapted the brain for language. This hypothesis has proven difficult to test, but it may be consistent with the tentative finding that the premotor cortex in humans may not have lagged as far behind as the primary motor cortex during human brain evolution. It is also consistent with the finding that sequential finger tapping is disrupted by concurrent speech (which depends on the left hemisphere in most individuals) only if the tapping is done with the right hand (which is also controlled by the left hemisphere) and not the left hand (which is controlled by the right hemisphere) (Ikeda 1987).

The Cognitive Reserve Hypothesis

An additional explanation for the increase in brain size in human evolution is that it may have allowed for an increase in longevity (Allen et al. 2005b, Humphrey 1999). The argument is that larger brains would buffer individuals against a variety of inevitable brain insults as individuals age, thereby increasing the useful cognitive lifespan. The results of a variety of clinical studies are consistent with the idea that larger brain size has a protective effect for a number of brain diseases and types of injury (reviewed in Allen et al. 2005b). Why (and whether) longevity would be evolutionarily adaptive in humans is unclear, although the survival of older, postreproductive individuals has been argued to be important (e.g., the grandmother hypothesis). The cognitive reserve hypothesis and the idea that more neural resources translate into better cognitive functioning of some kind are not mutually exclusive, of course.

Summary of the Evolution of Brain and Behavior

Explaining why the human brain changed as it did requires determining the behavioral implications of changing brain size and/or the proportions of various brain components. To be evolutionarily relevant, associations between the brain and behavior must be genetic correlations. These correlations can be quite small, however. Although both brain morphology and behavioral dimensions have been shown to be genetically influenced, genetic correlations between brain anatomy and behavior appear to be quite modest. The genetic correlation between overall brain size and general cognitive ability appears to be substantially smaller than overall phenotypic correlation. Associations between specific functional areas and specific behavioral abilities appear to be somewhat more robust.

A number of general behavioral models of brain evolution have been proposed that have theoretical and/or cross-species empirical support. These include the idea that brain size is associated with increased conceptual complexity, language ability, social ability, ecological challenges, the development of tools, and the need for a cognitive reserve. Direct tests of these hypotheses await future research.

CONCLUSION

Over the past 2 to 3 million years, our brain has changed in dramatic and behaviorally interesting ways. Although brain size and body size are correlated, absolute increases in neural tissue are likely behaviorally relevant, and the overemphasis on EQ needs to be tempered. There is substantial evidence that the human brain is also not simply a larger version of a generic primate brain, with some areas showing evidence of lagging behind (such as the olfactory bulb, primary visual cortex, primary motor and premotor areas) and some accounting for disproportionate increases (such as the prefrontal). We should expect to find clues about the details of the behavioral evolution of our species from these patterns. Given the evolutionary costs of neural tissue, disproportional increases (even in absolute terms) would not likely have occurred unless they conferred some sort of adaptive (reproductive) advantages, on average, to individuals in the successive populations. The advantages could be slight, however, making our task as scientists potentially difficult. General cognitive ability appears to show weak associations with brain size, and a number of behavioral dimensions appear to be associated with specific brain areas. Hypotheses involving conceptual complexity, social abilities, language, ecological challenges, tool use, and the cognitive reserve hypothesis all appear to have merit for explaining human brain evolution

Filling out the history of human brain evolution will continue to utilize an intensively interdisciplinary approach in which information, methods, and resources from a wide variety of fields will increasingly be marshaled to the task of squeezing every last possible bit of valid inference out of the data. Fundamentally, our arguments will always necessarily be statistical judgments. At present, we do not even know the limits of what we can and cannot know about this history. Discovering these limits is a central task for the future.

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LITERATURE CITED

Aiello LC, Wheeler P. 1995. The expensive tissue hypothesis: the brain and the digestive system in human and primate evolution. *Curr. Anthropol.* 36:199–221

Alexander MP, Benson DF, Stuss DT. 1989. Frontal lobes and language. *Brain Lang.* 37:656–91

Allen JS, Bruss J, Damasio H. 2005a. MRI analysis of the calcarine and lunate sulci in modern humans. *Am. J. Phys. Anthropol.* 126(S40):67

Allen JS, Bruss J, Damasio H. 2005b. The aging brain: the cognitive reserve hypothesis and hominid evolution. *Am. J. Hum. Biol.* 17:673–89

Allman JM. 1999. *Evolving Brains*. New York: W.H. Freeman

Ankney CD. 1992. Sex differences in relative brain size: the mismeasure of woman, too? *Intelligence* 16:329–36

Armstrong E. 1983. Relative brain size and metabolism in mammals. *Science* 220:1302–4

Armstrong E, Zilles K, Curtis M, Schleicher A. 1991. Cortical folding, the lunate sulcus and the evolution of the human brain. *J. Hum. Evol.* 20:341–48

Avants BB, Schoenemann PT, Gee JC. 2005. Lagrangian frame diffeomorphic image registration: morphometric comparison of human and chimpanzee cortex. *Med. Image Anal.* 10:397–412

Barton RA. 1996. Neocortex size and behavioral ecology in primates. *Proc. R. Soc. London Ser. B* 263:173–77

Beeman MJ, Bowden EM, Gernsbacher MA. 2000. Right and left hemisphere cooperation for drawing predictive and coherence inferences during normal story comprehension. *Brain Lang.* 71:310–36

Blinkov SM, Glezer II. 1968. *The Human Brain in Figures and Tables*. New York: Plenum. 482 pp.

Bogin B. 1999. *Patterns of Human Growth*. Cambridge, UK: Cambridge Univ. Press. 2nd ed.

Broadfield DC, Holloway RL, Mowbray K, Silvers A, Yuan MS, Márquez S. 2001. Endocast of Sambungmacan 3 (Sm 3): A new *Homo erectus* from Indonesia. *Anat. Rec.* 262:369–79

Brodmann K. 1909. *Vergleichende Lokalisationsiehre der Grosshirnrinde in ihren Prinzipien Dargestellt auf Grund des Zellenbaues*. Leipzig: Johann Ambrosius Barth Verlag

Bush EC, Allman JM. 2004. The scaling of frontal cortex in primates and carnivores. *Proc. Natl. Acad. Sci. USA* 101:3962–66

Calvin WH. 1983. A stone's throw and its launch window: timing precision and its implications for language and hominid brains. *J. Theor. Biol.* 104:121–35

Carpenter MB, Sutin J. 1983. *Human Neuroanatomy*. Baltimore, MD: Williams & Wilkins

Changizi MA, Shimojo S. 2005. Parcellation and area-area connectivity as a function of neocortex size. *Brain Behav. Evol.* 66:88–98

Connolly CJ. 1950. *External Morphology of the Primate Brain*. Springfield, IL: C.C. Thomas

Damasio AR. 1985. The frontal lobes. In *Clinical Neuropsychology*, ed. K Heilman, E Valenstein, p. 339–75. Oxford, UK: Oxford Univ. Press

Darwin C. 1871. *The Descent of Man and Selection in Relation to Sex*. Vol. 1. London: John Murray

Davatzikos C, Resnick SM. 1998. Sex differences in anatomic measures of interhemispheric connectivity: correlations with cognition in women but not men. *Cereb. Cortex* 8:635–40

Deacon TW. 1997. *The Symbolic Species: The Coevolution of Language and the Brain*. New York: W.W. Norton

de Bruin JPC. 1990. Social behavior and the prefrontal cortex. In *Progress in Brain Research*, ed. HBM Uylings, CG Van Eden, JPC de Bruin, MA Corner, MGP Feenstra, pp. 485–97. New York: Elsevier Science

Dekaban AS. 1978. Changes in brain weights during the span of human life: relation of brain weights to body heights and body weights. *Ann. Neurol.* 4:345–56

de Lacoste-Utamsing C, Holloway RL. 1982. Sexual dimorphism in the human corpus callosum. *Science* 216:1431–32

De Miguel C, Henneberg M. 2001. Variation in hominid brain size: How much is due to method? *Homo* 52:3–58

Diamond MC. 1988. *Enriching Heredity: The Impact of the Environment on the Anatomy of the Brain*. New York: Free Press

Dubois E. 1913. On the relation between quantity of brain and the size of the body in vertebrates. *Verh. Kon. Akad. Wetenschappen Amsterdam* 16:647

Dunbar R. 1996. *Grooming, Gossip and the Evolution of Language*. London: Faber & Faber

Dunbar RIM. 1995. Neocortex size and group size in primates: a test of the hypothesis. *J. Hum. Evol.* 28:287–96

Dunbar RIM. 2003. The social brain: mind, language, and society in evolutionary perspective. *Annu. Rev. Anthropol.* 32:163–81

Evans PD, Anderson JR, Vallender EJ, Gilbert SL, Malcom CM, et al. 2004. Adaptive evolution of ASPM, a major determinant of cerebral cortical size in humans. *Hum. Mol. Genet.* 13:489–94

Falconer DS. 1981. *Introduction to Quantitative Genetics*. New York: Longman. 2nd ed.

Falk D. 1983. Cerebral cortices of East African early hominids. *Science* 221:1072–74

Falk D. 1987. Hominid paleoneurology. *Annu. Rev. Anthropol.* 16:13–30

Falk D. 1990. Brain evolution in *Homo:* the "radiator" theory. *Behav. Brain Sci.* 13:333–81

Falk D. 1992. *Braindance: New Discoveries About Human Origins and Brain Evolution*. New York: Henry Holt

Falk D, Froese N, Sade DS, Dudek BC. 1999. Sex differences in brain/body relationships of Rhesus monkeys and humans. *J. Hum. Evol.* 36:233–38

Falk D, Hildebolt C, Cheverud J, Vannier M, Helmkamp RC, Konigsberg L. 1990. Cortical asymmetries in frontal lobes of Rhesus monkeys (*Macaca mulatta*). *Brain Res.* 512:40–45

Finlay BL, Darlington RB, Nicastro N. 2001. Developmental structure in brain evolution. *Behav. Brain Sci.* 24:263–308

Fish JL, Lockwood CA. 2003. Dietary constraints on encephalization in primates. *Am. J. Phys. Anthropol.* 120:171–81

Fuster JM. 1985. The prefrontal cortex, mediator of cross-temporal contingencies. *Hum. Neurobiol.* 4:169–79

Gabrieli JD, Poldrack RA, Desmond JE. 1998. The role of left prefrontal cortex in language and memory. *Proc. Natl. Acad. Sci. USA* 95:906–13

Garby L, Lammert O, Kock KF, Thobo-Carlsen B. 1993. Weights of brain, liver, kidneys, and spleen in healthy and apparently healthy adult Danish subjects. *Am. J. Hum. Biol.* 5:291–96

Gazzaniga MS, Ivry RB, Mangun GR. 1998. *Cognitive Neuroscience: The Biology of the Mind*. New York: W.W. Norton

Geary DC. 2005. *The Origin of Mind: Evolution of Brain, Cognition, and General Intelligence*. Washington, DC: Am. Psychol. Assoc.

Geschwind DH, Miller BL, DeCarli C, Carmelli D. 2002. Heritability of lobar brain volumes in twins supports genetic models of cerebral laterality and handedness. *Proc. Natl. Acad. Sci. USA* 99:3176–81

Gibson KR. 2002. Evolution of human intelligence: the roles of brain size and mental construction. *Brain Behav. Evol.* 59:10–20

Gignac G, Vernon PA, Wickett JC. 2003. Factors influencing the relationship between brain size and intelligence. In *The Scientific Study of General Intelligence: Tribute to Arthur R. Jensen*, ed. H Nyborg, pp. 93–106. London: Elsevier

Gilbert SL, Dobyns WB, Lahn BT. 2005. Genetic links between brain development and brain evolution. *Nat. Rev. Genet.* 6:581–90

Goldman-Rakic PS. 1996. The prefrontal landscape: implications of functional architecture for understanding human mentation and the central executive. *Philos. Trans. R. Soc. London Ser. B* 351:1445–53

Gould SJ. 1981. *The Mismeasure of Man*. New York: Norton. 352 pp.

Halpern DF. 1987. *Sex Differences in Cognitive Abilities*. Hillsdale, NJ: Erlbaum

Harvey PH, Clutton-Brock TH. 1985. Life history variation in primates. *Evolution* 39:559–81

Haug H. 1987. Brain sizes, surfaces, and neuronal sizes of the cortex cerebri: a stereological investigation of man and his variability and a comparison with some mammals (primates, whales, marsupials, insectivores, and one elephant). *Am. J. Anat.* 180:126–42

Herrnstein RJ, Murray C. 1994. *The Bell Curve*. New York: Free Press

Ho K, Roessmann U, Straumfjord JV, Monroe G. 1980a. Analysis of brain weight. I. Adult brain weight in relation to sex, race, and age. *Arch. Pathol. Lab. Med.* 104:635–39

Ho K, Roessmann U, Straumfjord JV, Monroe G. 1980b. Analysis of brain weight. II. Adult brain weight in relation to body height, weight, and surface area. *Arch. Pathol. Lab. Med.* 104:640–45

Hofman MA. 1983a. Encephalization in hominids: evidence for the model of punctuationalism. *Brain Behav. Evol.* 22:102–17

Hofman MA. 1983b. Energy metabolism, brain size, and longevity in mammals. *Q. Rev. Biol.* 58:495–512

Hofman MA. 1985. Size and shape of the cerebral cortex in mammals. I. The cortical surface. *Brain Behav. Evol.* 27:28–40

Holloway. 1966. Cranial capacity and neuron number: a critique and proposal. *Am. J. Phys. Anthropol.* 25:305–14

Holloway RL. 1975. *The Role of Human Social Behavior in the Evolution of the Brain*. New York: Am. Mus. Nat. Hist.

Holloway RL. 1983. Human paleontological evidence relevant to language behavior. *Hum. Neurobiol.* 2:105–14

Holloway RL. 1992. The failure of the gyrification index (GI) to account for volumetric reorganization in the evolution of the human brain. *J. Hum. Evol.* 22:163–70

Holloway RL. 1995. Toward a synthetic theory of human brain evolution. In *Origins of the Human Brain*, ed. J-P Changeux, J Chavaillon, pp. 42–54. Oxford: Clarendon Press

Holloway RL. 2002. Brief communication: How much larger is the relative volume of area 10 of the prefrontal cortex in humans? *Am. J. Phys. Anthropol.* 118:399–401

Holloway RL, Anderson PJ, Defendini R, Harper C. 1993. Sexual dimorphism of the human corpus callosum from three independent samples: relative size of the corpus callosum. *Am. J. Phys. Anthropol.* 92:481–98

Holloway RL, Broadfield DC, Yuan MS. 2003. Morphology and histology of chimpanzee primary visual striate cortex indicate that brain reorganization predated brain expansion in early hominid evolution. *Anat. Rec. A Discov. Mol. Cell Evol. Biol.* 273:594–602

Holloway RL, Broadfield DC, Yuan MS. 2004a. *The Human Fossil Record, Volume 3. Brain Endocasts: The Paleoneurological Evidence*. Hoboken, NJ: Wiley & Sons

Holloway RL, Clark RJ, Tobias PV. 2004b. Posterior lunate sulcus in *Australopithecus africanus*: Was Dart right? *C. R. Palevol.* 3:287–93

Holloway RL, de la Coste-Lareymondie MC. 1982. Brain endocast asymmetry in pongids and hominids: some preliminary findings on the paleontology of cerebral dominance. *Am. J. Phys. Anthropol.* 58:101–10

Humphrey N. 1984. The social function of intellect. In *Consciousness Regained*, ed. N. Humphrey, pp. 14–28. Oxford, UK: Oxford Univ. Press

Humphrey N. 1999. Why human grandmothers may need large brains. *Psycholoquy* 10:24. **http://psycprints.ecs.soton.ac.uk/archive/00000659/**

Ikeda K. 1987. Lateralized interference effects of concurrent verbal tasks on sequential finger tapping. *Neuropsychologia* 25:453–56

Jensen AR. 1994. Psychometric *g* related to differences in head size. *Pers. Individ. Dif.* 17:597–606

Jensen AR, Johnson FW. 1994. Race and sex differences in head size and IQ. *Intelligence* 18:309–33

Jensen AR, Sinha SN. 1990. Physical correlates of human intelligence. In *Biological Approaches to the Study of Human Intelligence*, ed. PA Vernon. Norwood, NJ: Ablex

Jerison HJ. 1973. *Evolution of the Brain and Intelligence*. New York: Academic

Jerison HJ. 1985. Animal intelligence as encephalization. *Philos. Trans. R. Soc. London Ser. B* 308:21–35

Kappelman J. 1996. The evolution of body mass and relative brain size in fossil hominids. *J. Hum. Evol.* 30:243–76

Kling AS. 1986. Neurological correlates of social behavior. *Ethol. Sociobiol.* 7:175–86

Krubitzer L. 1995. The organization of neocortex in mammals: Are species differences really so different? *Trends Neurosci.* 18:408–17

Lee SH, Wolpoff MH. 2003. The pattern of evolution in Pleistocene human brain size. *Paleobiology* 29:186–96

Lieberman P. 2002. On the nature and evolution of the neural bases of human language. *Yearb. Phys. Anthropol.* 45:36–62

Lovejoy CO. 1975. Biomechanical perspectives on the lower limb of early hominids. In *Primate Functional Morphology and Evolution*, ed. RH Tuttle, pp. 291–326. The Hague: Mouton

Lyons DM, Afarian H, Schatzberg AF, Sawyer-Glover A, Moseley ME. 2002. Experience-dependent asymmetric variation in primate prefrontal morphology. *Behav. Brain Res.* 136:51–59

MacLeod CE, Zilles K, Schleicher A, Rilling JK, Gibson KR. 2003. Expansion of the neocerebellum in Hominoidea. *J. Hum. Evol.* 44:401–29

Marquez S, Mowbray K, Sawyer GJ, Jacob T, Silvers A. 2001. New fossil hominid calvaria from Indonesia—Sambungmacan 3. *Anat. Rec.* 262:344–68

Martin RD. 1981. Relative brain size and basal metabolic rate in terrestrial vertebrates. *Nature* 293:57–60

McBride T, Arnold SE, Gur RC. 1999. A comparative volumetric analysis of the prefrontal cortex in human and baboon MRI. *Brain Behav. Evol.* 54:159–66

McComb K, Moss C, Durant SM, Baker L, Sayialel S. 2001. Matriarchs as repositories of social knowledge in African elephants. *Science* 292:491–94

McGurk H, MacDonald J. 1976. Hearing lips and seeing voices. *Nature* 264:746–48

Milton K. 1981. Distribution patterns of tropical plant foods as an evolutionary stimulus to primate mental development. *Am. Anthropol.* 83:534–48

Myers RE, Swett C, Miller M. 1973. Loss of social group affinity following prefrontal lesions in free-ranging macaques. *Brain Res.* 64:257–69

Neisser U, Boodoo G, Bouchard TJJ, Boykin AW, Brody N, et al. 1996. Intelligence: knowns and unknowns. *Am. Psychol.* 51:77–101

Novoa OP, Ardila A. 1987. Linguistic abilities in patients with prefrontal damage. *Brain Lang.* 30:206–25

Pakkenberg H, Voigt J. 1964. Brain weight of the Danes. *Acta Anatom.* 56:297–307

Penfield W, Rasmussen T. 1950. *Cerebral Cortex of Man: A Clinical Study of Localization of Function*. New York: Macmillan

Plomin R, DeFries JC, McClearn GE, Rutter M. 1997. *Behavioral Genetics*. New York: W.H. Freeman. 3rd ed.

Posthuma D, Baare WF, Pol HEH, Kahn RS, Boomsma DI, De Geus EJ. 2003. Genetic correlations between brain volumes and the WAIS-III dimensions of verbal comprehension, working memory, perceptual organization, and processing speed. *Twin Res.* 6:131–39

Posthuma D, De Geus EJ, Baare WF, Pol HEH, Kahn RS, Boomsma DI. 2002. The association between brain volume and intelligence is of genetic origin. *Nat. Neurosci.* 5:83–84

Preuss TM. 2000. What's human about the human brain? In *The New Cognitive Neurosciences*, ed. MS Gazzaniga, pp. 1219–34. 2nd ed. Cambridge, MA: Bradford Books/MIT Press

Preuss TM, Coleman GQ. 2002. Human-specific organization of primary visual cortex: alternating compartments of dense Cat-301 and calbindin immunoreactivity in layer 4A. *Cereb. Cortex* 12:671–91

Pulvermuller F. 2001. Brain reflections of words and their meaning. *Trends Cogn. Sci.* 5:517–24

Raz N, Lindenberger U, Rodrigue KM, Kennedy KM, Head D, et al. 2005. Regional brain changes in aging healthy adults: general trends, individual differences and modifiers. *Cereb. Cortex* 15:1676–89

Reader SM, Laland KN. 2002. Social intelligence, innovation, and enhanced brain size in primates. *Proc. Natl. Acad. Sci. USA* 99:4436–41

Redmond JCJ. 1999. Cranial capacity and performance on delay-response task correlated with principal sulcus length in monkeys. *Am. J. Phys. Anthropol.* 109:33–40

Rendell L, Whitehead H, Rendell L, Whitehead H. 2001. Culture in whales and dolphins. *Behav. Brain Sci.* 24:309–24; discussion 324–82

Riddell WI, Corl KG. 1977. Comparative investigation of the relationship between cerebral indices and learning abilities. *Brain Behav. Evol.* 14:385–98

Rilling JK, Insel TR. 1999. The primate neocortex in comparative perspective using magnetic resonance imaging. *J. Hum. Evol.* 37:191–223

Rilling JK, Seligman RA. 2002. A quantitative morphometric comparative analysis of the primate temporal lobe. *J. Hum. Evol.* 42:505–33

Ringo JL. 1991. Neuronal interconnection as a function of brain size. *Brain Behav. Evol.* 38:1–6

Rumbaugh DM, Savage-Rumbaugh ES, Wasburn DA. 1996. Toward a new outlook on primate learning and behavior: complex learning and emergent processes in comparative perspective. *Jpn. Psychol. Res.* 38:113–25

Rushton JP, Ankney CD. 1996. Brain size and cognitive ability: correlations with age, sex, social class, and race. *Psychon. Bull. Rev.* 3:21–36

Sax KW, Strakowski SM, Zimmerman ME, DelBello MP, Keck PEJ, Hawkins JM. 1999. Frontosubcortical neuroanatomy and the continuous performance test in mania. *Am. J. Psychiatry* 156:139–41

Schenker NM, Desgouttes AM, Semendeferi K. 2005. Neural connectivity and cortical substrates of cognition in hominoids. *J. Hum. Evol.* 49:547–69

Schoenemann PT. 1997. *An MRI study of the relationship between human neuroanatomy and behavioral ability*. PhD diss. Univ. of Calif., Berkeley

Schoenemann PT. 2004. Brain size scaling and body composition in mammals. *Brain Behav. Evol.* 63:47–60

Schoenemann PT. 2005. Conceptual complexity and the brain: understanding language origins. In *Language Acquisition, Change and Emergence: Essays in Evolutionary Linguistics*, ed. WS-Y Wang, JW Minett, pp. 47–94. Hong Kong: City Univ. of Hong Kong Press

Schoenemann PT, Budinger TF, Sarich VM, Wang WS. 2000. Brain size does not predict general cognitive ability within families. *Proc. Natl. Acad. Sci. USA* 97:4932–37

Schoenemann PT, Glotzer LD, Sheehan MJ. 2005a. Reply to "Is prefrontal white matter enlargement a human evolutionary specialization?" *Nat. Neurosci.* 8:538

Schoenemann PT, Sheehan MJ, Glotzer LD. 2005b. Prefrontal white matter volume is disproportionately larger in humans than in other primates. *Nat. Neurosci.* 8:242–52

Semendeferi K, Armstrong E, Schleicher A, Zilles K, Van Hoesen GW. 1998. Limbic frontal cortex in hominoids: a comparative study of area 13. *Am. J. Phys. Anthropol.* 106:129–55

Semendeferi K, Armstrong E, Schleicher A, Zilles K, Van Hoesen GW. 2001. Prefrontal cortex in humans and apes: a comparative study of area 10. *Am. J. Phys. Anthropol.* 114:224–41

Semendeferi K, Lu A, Schenker N, Damasio H. 2002. Humans and great apes share a large frontal cortex. *Nat. Neurosci.* 5:272–76

Sherwood CC, Holloway RL, Semendeferi K, Hof PR. 2005. Is prefrontal white matter enlargement a human evolutionary specialization? *Nat. Neurosci.* 8:537–38; author reply 538

Smith BH. 1990. The cost of a large brain. *Behav. Brain Sci.* 13:365–66

Stephan H, Frahm H, Baron G. 1981. New and revised data on volumes of brain structures in insectivores and primates. *Folia Primatologica* 35:1–29

Stout D, Toth N, Schick K. 2000. Stone tool-making and brain activation: position emission tomography (PET) studies. *J. Archaeol. Sci.* 27:1215–23

Striedter GF. 2005. *Principles of Brain Evolution*. Sunderland, MA: Sinauer Associates

Thompson PM, Cannon TD, Narr KL, van Erp T, Poutanen VP, et al. 2001. Genetic influences on brain structure. *Nat. Neurosci.* 4:1253–58

Tobias PV. 1975. Brain evolution in the Hominoidea. In *Primate Functional Morphology and Evolution*, ed. RH Tuttle, p. 353–92. The Hague: Mouton

Tobias PV. 1983. Recent advances in the evolution of the hominids with especial reference to brain and speech. In *Recent Advances in the Evolution of Primates*, ed. C Chagas, p. 85–140. Vatican City: Pontificia Acad. Sci.

Toth N, Schick K. 1993. Early stone industries and inferences regarding language and cognition. In *Tools, Language and Cognition in Human Evolution*, ed. KR Gibson, T Ingold, pp. 346–62. Cambridge, UK: Cambridge Univ. Press

Van Essen DC. 2005. Surface-based comparisons of macaque and human cortical organization. In *From Monkey Brain to Human Brain*, ed. S Dehaene, J-R Duhamel, MD Hauser, G Rizzolatti, pp. 3–19. Cambridge, MA: MIT Press

Van Essen DC, Drury HA, Joshi S, Miller MI. 1998. Functional and structural mapping of human cerebral cortex: Solutions are in the surfaces. *Proc. Natl. Acad. Sci. USA* 95:788–95

von Bonin G. 1963. *The Evolution of the Human Brain*. Chicago: Univ. Chicago Press

Wang WSY. 1991. Explorations in language evolution. In *Explorations in Language*, pp. 105–31. Taipei, Taiwan: Pyramid Press

Wang YQ, Su B. 2004. Molecular evolution of microcephalin, a gene determining human brain size. *Hum. Mol. Genet.* 13:1131–37

Washburn SL. 1960. Tools and evolution. *Sci. Am.* 203:63–75

White TD, Asfaw B, DeGusta D, Gilbert H, Richards GD, et al. 2003. Pleistocene *Homo sapiens* from Middle Awash, Ethiopia. *Nature* 423:742–47

Wickett JC, Vernon PA, Lee DH. 2000. Relationships between factors of intelligence and brain volume. *Pers. Individ. Dif.* 29:1095–22

Winterer G, Goldman D. 2003. Genetics of human prefrontal function. *Brain Res. Brain Res. Rev.* 43:134–63

Wood B, Collard M. 1999. The human genus. *Science* 284:65–71

Wright IC, Sham P, Murray RM, Weinberger DR, Bullmore ET. 2002. Genetic contributions to regional variability in human brain structure: methods and preliminary results. *Neuroimage* 17:256–71

Wynn T. 2002. Archaeology and cognitive function. *Behav. Brain Sci.* 25:389–438

Zilles K. 2005. Evolution of the human brain and comparative cyto- and receptor architecture. In *From Monkey Brain to Human Brain*, ed. S Dehaene, J-R Duhamel, MD Hauser, G Rizzolatti, pp. 41–56. Cambridge, MA: MIT Press

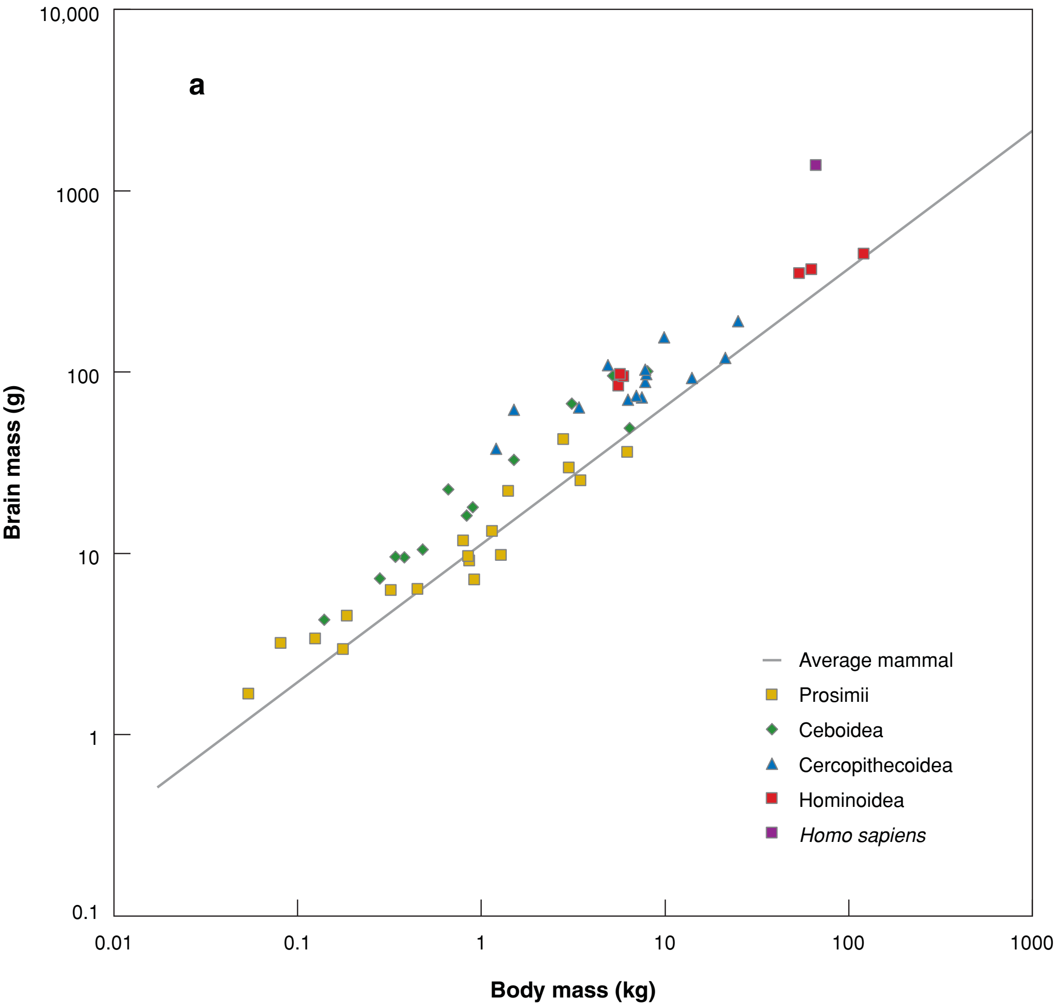

Figure 1

Brain/body relationships among primates. (*a*) Individual species plotted with Martin's (1981) estimate for the average mammal. (*b*) Least squares–regression estimates for various primate subtaxa. All primates (excluding humans): [log brain *g*] = 1.24 + 0.761 [log body kg], r^2 = 0.92, *N* = 51; Hominoidea (excluding humans): [log brain *g*] = 1.548 + 0.553 [log body kg], r^2 = 0.99, *N* = 6; Cercopithecoidea: [log brain *g*] = 1.54 + 0.477 [log body kg], r^2 = 0.87, *N* = 14; Ceboidea: [log brain *g*] = 1.35 + 0.765 [log body kg], r^2 = 0.94, *N* = 13; Prosimii: [log brain *g*] = 1.111 + 0.659 [log body kg], r^2 = 0.92, *N* = 18. Data extracted from literature sources (see Schoenemann 1997 for details); pongid data estimated from cranial capacities using [brain weight *g*] = [cranial capacity cc]/ 1.14 (following Kappelman 1996).

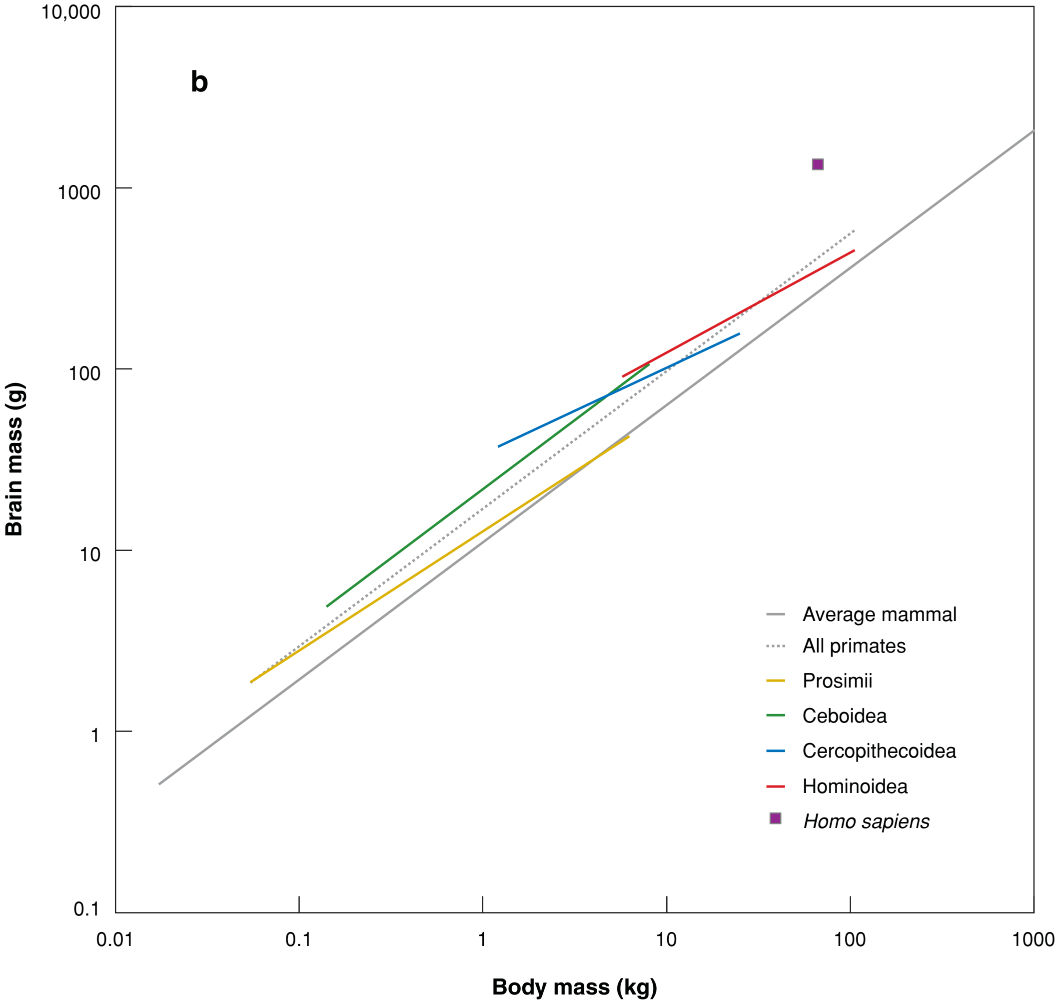

Figure 1

(*Continued*)

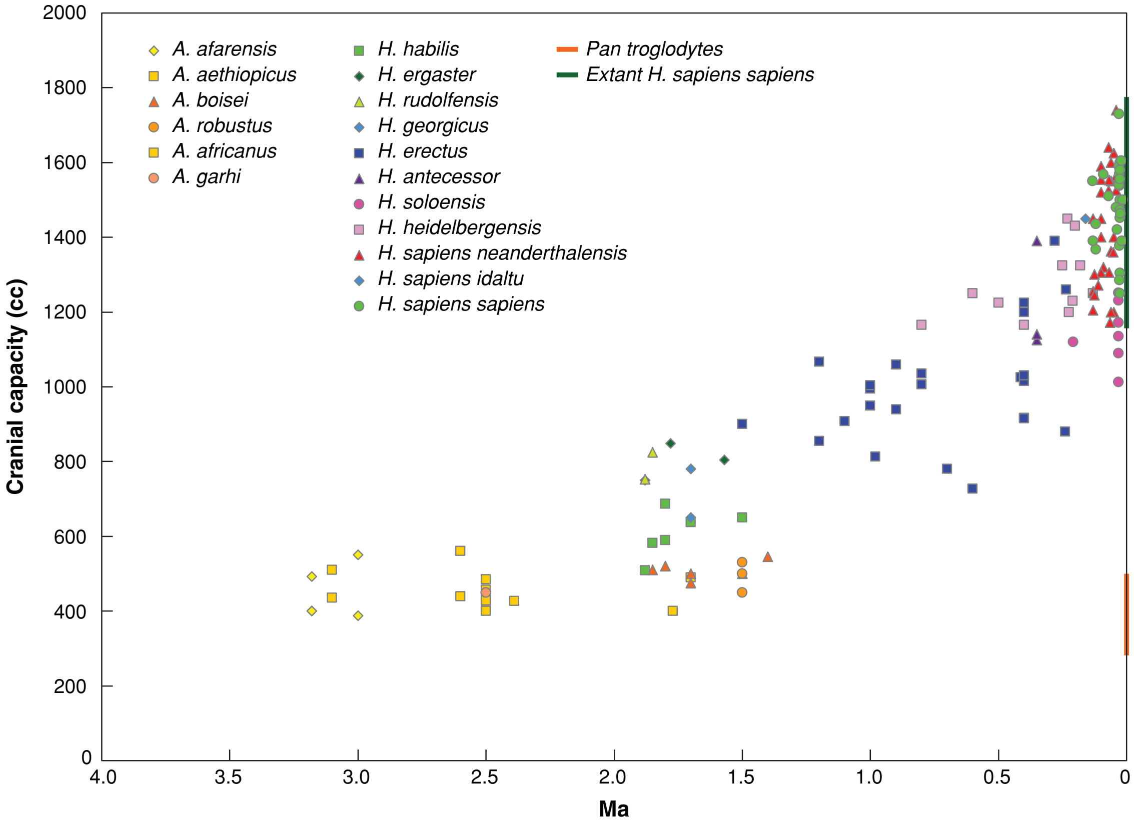

Figure 2

Cranial capacity in fossil hominids over time. Extant chimpanzees (*Pan troglodytes*) and humans (*Homo sapiens sapiens*) are included for comparison. Fossil data and species designations from Holloway et al. 2004a. *Pan* and *Homo* species data compiled from literature sources listed in Schoenemann (1997).

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