organizational scheme of the dolphin neocortex is unique and highly different from that in primates. These differences further support the notion that the same cognitive capacities in primates and dolphins are underwritten by different neurobiological 'themes,' resulting in convergent cognition." Her claim that similar cognitive abilities can arrive from divergent brain anatomy adds a level of complexity to evolutionary cognitive neuroscience, one that deserves emphasis.

The final chapter in this part examines human cranial capacity. Although one of the editors of this volume (J.P.K.) has argued against the race/IQ relationship on scientific grounds (e.g., the heterogeneity of race on a genetic level), Philippe Rushton and Davison Ankney provide a significant and important review of their own and others' research on the topic of brain and cognition as measured (mainly) via IQ and cranial capacity.

Does brain size matter in terms of cognition? This simple question is parsed throughout this section, as the question becomes increasingly complicated in terms of how and what we measure in the brain, brain size, and cognition.

4 The Evolution of Ontogeny and Human Cognitive Uniqueness: Selection for Extended Brain Development in the Hominid Line

Valerie E. Stone

Humans are remarkable among primates for both our large brains re tive to body size and our complex cognitive skills (Darwin, 1871; fo review, see Oxnard, 2004). Thus, any evolutionary approach to cog tive neuroscience should give an account of how humans came to ha these unique features. Several different fields can contribute to such account. Comparative neuroscience can give us information about s tematic variations in the size, development, and connectivity of differe brain regions across primate species. Archaeology can give us clues abo body size, brain size, and development for extinct hominid species. Ev lutionary psychology can provide insight into which cognitive mech nisms we might share with other mammals or primates and which a likely to be unique to our species. Cognitive neuroscience can investig the brain systems that underlie uniquely human cognitive abiliti through patient studies and neuroimaging. The challenge for evolutio ary cognitive neuroscience is to weave together these approaches in way that illuminates human cognition.

There are several accounts of which cognitive abilities are uniq to humans, with authors tending to put forward their favorite candid ability as *the* defining feature of humanity. However, it is likely that the are several overlapping abilities that uniquely define human cognitic There is a growing recognition that this is the case in discussions human uniqueness, with lists of these abilities including langua executive function, long-term memory and future planning, recursic complex categorization and problem solving, abstraction, and theo of mind (Byrne, 2001; Corballis, 2003; Dunbar, 1998; Hoffeck 2005; Pinker & Bloom, 1990; Stone, 2005; Stone & Gerrans, 200 Suddendorf, 1999, 2004; Tooby & DeVore, 1987). Depending on t writer, "uniquely human" can mean that humans are the only curre species that possesses a certain cognitive ability, or it can mean th

Homo sapiens sapiens is unique compared to our extinct hominid ancestors in possessing a certain cognitive ability. Claims about our abilities relative to those of extant primates can be tested empirically in the laboratory; claims about our abilities relative to those of other hominids are tested using inferences from artifacts such as tools and hunted animal bones associated with fossilized hominids. With each of these abilities, of course, certain aspects may be shared with other species, and other aspects may be unique (Stone, 2005; Suddendorf, 2004). Each unique ability builds on other cognitive abilities that we share with other primates. Some basic ability to associate a symbol with a meaning may well be an ability that we share with other primates (Snowdon, 2002). However, complex syntax and recursion are aspects of language that seem to be uniquely human (Corballis, 2003; Pinker & Bloom, 1990). Our ability to monitor others' eye gaze is a building block of theory of mind that we share with other primates; however, inferences about others' belief and knowledge, "theory of mind proper," appears to be unique to our species (for a review, see Stone, 2005). Nevertheless, there is a basic set of uniquely human cognitive abilities that seem to be uncontroversial: recursion, episodic memory and future planning, theory of mind, complex problem solving requiring high levels of executive function, and language that involves complex syntax (Byrne & Whiten, 1988; Corballis, 2003; Pinker & Bloom, 1990; Suddendorf, 1999; Tooby & DeVore, 1987). This list is certainly not exhaustive; it is merely a minimal set for which there is evidence.

Cognitive neuroscience has already given us information on the brain areas involved in these abilities in humans. The most complex levels of executive function seem to be mediated by lateral prefrontal cortex (Cummings, 1993; Knight & Grabowecky, 1995). We know that simpler aspects of executive function, such as basic working memory, are also mediated by prefrontal cortex in primates (Goldman-Rakic, Bourgeois, & Rakic, 1997). We know that the storage and retrieval of episodic memory and future planning depend on the frontal and temporal lobes, though memories may be stored throughout the cortex (Knight & Grabowecky, 1995; Rowe, Owen, Johnsrude, & Passingham, 2001; Shimamura, 2000; Shimamura, Janowsky & Squire, 1990; Tulving, 1995; Wood & Grafman, 2003). We know that language, syntax, and recursion also depend on the frontal and temporal lobes (Caplan et al., 2002; Cooke et al., 2001). Furthermore, humans are not merely designed to process information, but to act on that information. Cognitive abilities such as those in the list above are of no use without

the ability to execute sequences of action as the output of cognition. We also know from cognitive neuroscience that executing sequences of actions depends on frontal regions and striatum, possibly also parietal lobes, for their involvement in body and action representations (Cummings, 1993; Krams et al., 1998; Reed, Stone, & McGoldrick 2005; Rowe et al., 2001). In very rough terms, then, we can identify brain structures that subserve the uniquely human aspects of cognition and action: prefrontal cortex, temporal cortex, parietal cortex and striatum, perhaps frontal and temporal cortex particularly. By inference, these structures would have been under selection in the evolution of the primate and hominid line.

To create brains with more complex abilities, natural selection can act on two factors: the number of neurons and the connectivity of those neurons. Connectivity includes not only the "wiring diagram" but also which neurotransmitters are used where. Connectivity is probably the most important factor; however, it is also the most difficult to study, because we do not have a complete map of neural connections in the human brain, nor in most other primate brains. Neuroscience often discusses research on "the monkey brain" or "the primate brain," but such phrases almost always refer to rhesus macaques, and sometimes vervets or squirrel monkeys. Thus, our comparative knowledge of connectivity patterns within and between brain structures in primate and human brains is incomplete, limited to a tiny number of species. Number of neurons is a little easier to study. Having more neurons in a brain structure generally means either a larger structure or a more convoluted structure. Size or convolution of a particular structure is a much rougher measure of a structure's function than is connectivity, but it has the advantage of being information that is available for a greater number of primate species (Rilling & Insel, 1999; Semendeferi, Armstrong, Schleicher, Zilles, & Van Hoesen, 2001; Semendeferi & Damasio, 2000; Stephan, Frahm & Baron, 1981). Furthermore, we can also make inferences about size of brain structures from analyzing fossilized skulls of extinct hominid species (Falk, 1987). While acknowledging that size of brain structures is one of the roughest possible measures of function, I would nevertheless like to review comparative research on the size of frontal and temporal lobes, cortex, and striatum.

One study has looked at a link between function and size of brain structures comparatively. The executive brain is defined as the neocortex plus striatum (i.e., basal ganglia), to denote those parts of the brain involved in executing complex actions (Keverne, Martel, & Nevison,

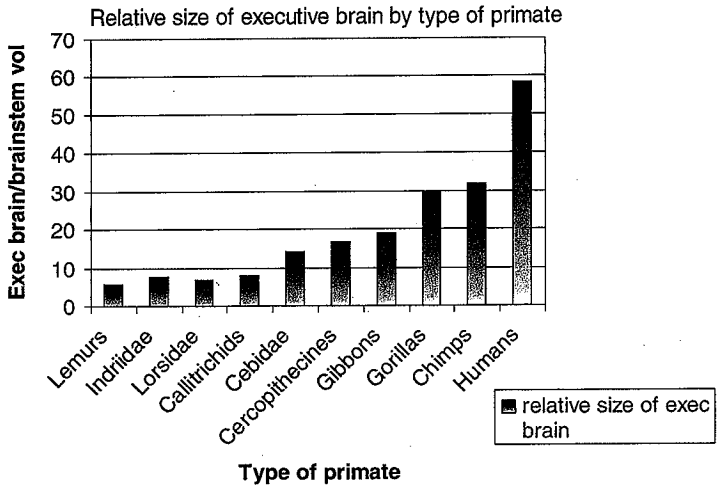


Figure 4.1
The executive brain is defined as the volume of neocortex plus striatum (Keverne et al., 1996; Reader & Laland, 2002). Taking a ratio of this volume to brainstem volume produces a measure of executive brain size that is corrected for the influence of body size and overall brain size (Barton, 1990; Reader & Laland, 1996). The relative size of the executive brain appears larger in apes than in other primates, and especially large in humans. (Data from Stephan, Frahm, & Baron, 1981.)

1996; Reader & Laland, 2002). Neocortex and striatum are closely linked genomically and neuroanatomically (Reader & Laland, 2002). (Note that the term executive has nothing to do with executive function or the frontal lobes in this context, but rather refers to the execution of action.) When corrected for body size, the executive brain is much larger in humans than in other primates (figure 4.1). In great apes and humans, compared to monkeys, one can see a greater capacity for perceiving and using innovative sequences of actions to solve complex problems (Byrne, 2001; Tooby & DeVore, 1987). This capacity includes both social problem solving, such as political maneuvering, social learning, and theory of mind, and physical problem solving, such as tool use and innovative strategies for foraging. Our own species has these abilities in the extreme, as demonstrated by the variety and flexibility of human cultures, tool manufacture and use, and the number of ecological niches in which we can forage successfully. Reader and Laland (2002) attempted

to index complex and flexible problem-solving skills in primates by counting reported instances of innovation, social learning, and tool use in 116 primate species. Executive brain size data were available for 32 species. Even with such an approximate measure, they demonstrated a significant relationship between the size of the executive brain (from Stephan et al., 1981), corrected for body size, and instances of problem solving. Thus, increases in the size of the executive brain over evolution seem to be associated with functional increases in intelligence.

Many analyses of the size of primate brain regions are based on a published data set of postmortem analyses of the brains of primates that either died naturally in captivity or were recovered from poachers (Stephan et al., 1981). An advantage of this data set is that it contains information on over 40 primate species. A disadvantage is that often a "species" is represented by one individual. Data on the size of brain regions in living primates, based on multiple individuals, would obviously be an improvement. New technologies have made this ethically possible. Recently, other researchers have begun using volumetric analysis of structural MRI scans to determine the size of various brain regions in living, sedated primates. Such data currently exist for only a few species, but it is to be hoped that in the future, they will become available on a larger number of species. Semendeferi and colleagues have scanned macaques, gibbons, orangutans, gorillas, chimps, bonobos, and humans to determine overall brain volume and the size of frontal, temporal, and parieto-occipital regions (Semendeferi & Damasio, 2000; Semendeferi et al., 2001) (figure 4.2). Insel and colleagues have analyzed the volume of brain regions and the degree of cortical convolution in 11 primate species (Rilling & Insel, 1999). These analyses show clear increases in the size of some cortical regions across apes and humans, and little increase in others. Although the executive brain overall clearly seems larger in humans, particular subdivisions of the cortex—temporal, frontal, and parietal lobes—show little evidence of disproportionate expansion specific to those regions over species that have diverged at various points over the past 18 million years. However, subdivisions of these areas may be important. Within the frontal lobes, the subdivisions of dorsolateral frontal, medial frontal, and orbitofrontal also do not show strong evidence of disproportionate size in humans (Semendeferi et al., 1997). However, the frontal pole, Brodmann's area 10, shows a dramatic increase in size in humans compared to our closest relatives (figure 4.3). The frontal pole is known to be involved in executive function, problem solving, future planning, and episodic memory, all things

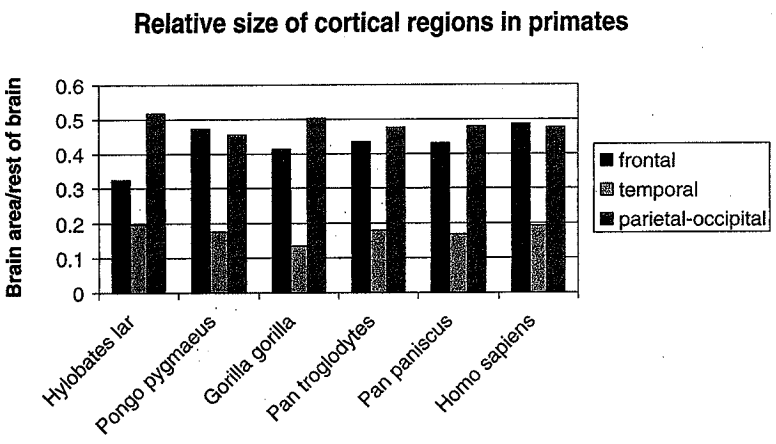


Figure 4.2
Size of three major divisions of cortex relative to whole brain size in primates most closely related to humans. With such gross divisions between cortical areas, no clear increase in size for any one area is evident for humans relative to non-human primate species. (Data from Semendeferi et al., 2000.)

that contribute to the cognitive uniqueness of humans (Braver & Bongiolatti, 2002; Lepage, Ghaffer, Nyberg, & Tulving, 2000; Tulving, 1995; Wood & Grafman, 2003).

If, as Reader and Laland (2002) propose, our complex cognitive abilities require larger cortical areas, then how has evolution produced these large brains? Selection would have had to occur in the genes regulating brain ontogeny. Prenatal neural development is the first aspect of ontogeny under genetic control. Finlay and Darlington (1995) have pointed out that there are strong developmental constraints on the size of different brain structures, such that the size of one structure is tightly correlated with the size of other structures, with approximately 96% of the variance in structure size accounted for by the size of other brain structures. This linkage between structure sizes appears to be strongly related to the length of time spent generating neurons prenatally, known as neurogenesis ($r = 0.94$; Finlay & Darlington, 1995). To the extent that a particular part of neocortex, such as the frontal pole, is larger in humans, it may also be the case that the rest of the neocortex and subcortical structures such as the thalamus are larger as well. Thus, one place that natural selection can act on the genome to produce bigger brain structures is on genes that regulate the extent of neurogenesis.

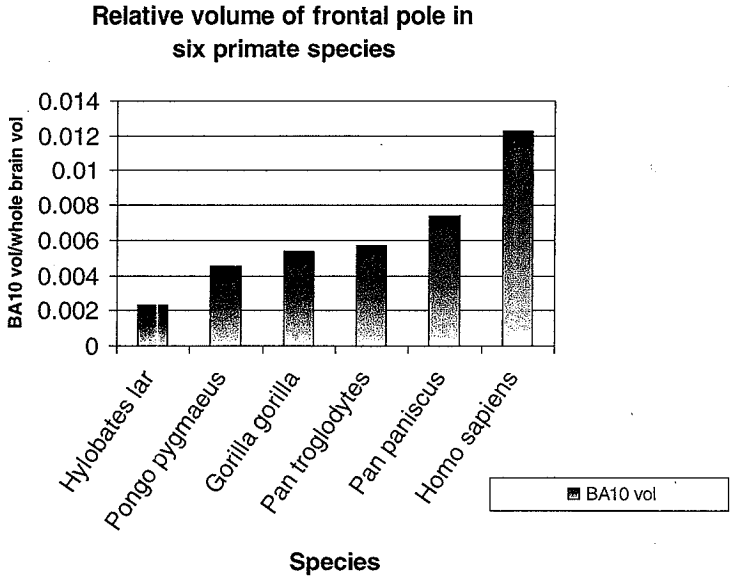


Figure 4.3
In contrast to the cortical areas in figure 4.2, the volume of the frontal pole relative to the whole brain appears to have undergone expansion in the primate line, with the frontal pole in humans disproportionately larger than in the primates most closely related to humans. (Data from Semendeferi et al., 2001.)

However, there would be no advantage conferred by genes for the extension of neurogenesis to create bigger brains unless those brains could develop and wire themselves up correctly.[1] Genes for the extension of neurogenesis would have had to be closely linked, in primate and hominid evolution, with genes for the extension of postnatal brain ontogeny. Neural development from the fetal stage to the adult stage is quite an extended process in humans. Prenatally, there is a period of neurogenesis, during which precursor cells divide and then later differentiate into neurons. Postnatally, neurons grow, dendritic branches extend, and synaptic density increases. After a plateau period of peak synaptic

1 Saying that a brain is wired "correctly" is shorthand for "in a way that would make the resulting organism inclusively fit"—that is, in a way that enables the developing organism to solve the adaptive problems it is confronted with at each developmental stage.

density, unused synapses are pruned over the course of late childhood and adolescence. Myelination of axons also continues throughout childhood and adolescence.

Large brains require longer maturation times than small ones (Passingham, 1985). In particular, the relatively large brains of great apes and humans require a longer period of "postnatal development" (also known as "experience") in order to become fully functioning adult brains (Allman & Hasenstaub, 1999; Smith & Tompkins, 1995). Early in infancy and childhood, neurons grow and form synapses. As childhood and adolescence progress, synapses decrease in density in a process known as pruning. Thus, experience is necessary for the correct wiring up of the human brain. Natural selection could have acted on genes regulating the overall extent of postnatal brain development in primates and hominids as part of the evolution of hominids' large brains.

Neural Development in Humans and Primates

As noted by Finlay and Darlington (1995), neurogenesis is one key way to affect the size of brain structures. The longer the period of prenatal neurogenesis for a structure, the more neurons are in that structure. One of their key points was that the extent of neurogenesis is very tightly linked across structures (Finlay, Darlington, & Nicastro, 2001). Extending this period of neurogenesis or increasing the rate of precursor cell division during neurogenesis is one of the primary developmental changes that natural selection could have acted on to produce large hominid brains (Finlay & Darlington, 1995; Finlay et al., 2001). Finlay and Darlington (1995) argue that if selection acted on genes regulating the extent of neurogenesis to enlarge any particular brain structure, then, because this process is so tightly linked across neurons in all areas, all brain structures would also have become larger. Neurogenesis also occurs in the adult brain, though as of now, the functional consequences of adult neurogenesis are unknown (Djavadian, 2004; Duman, 2004).

Certain stages of postnatal neural development are quite extended in humans. During the 1–3 years after birth, there is an initial period of cortical neuron growth and promiscuous synapse formation, in which it appears that any neurons that come into contact and share the same neurotransmitters will form a synapse (Huttenlocher & Dabholkar, 1997a). (This same developmental period extends only from 2–3 months in rhesus macaques; Goldman-Rakic et al., 1997.) Synaptic density peaks

latest in prefrontal cortex and temporal cortex (Giedd et al., 1999; Huttenlocher & Dabholkar, 1997a). Dendritic branches continue to grow and lengthen from birth up to 2 years in occipital cortex, and beyond that period in frontal cortex (Huttenlocher & Dabholkar, 1997a). There is a plateau period of peak synaptic density in the frontal lobes from 1–3 years to 7–8 years, the period of greatest plasticity in cognition (Huttenlocher & Dabholkar, 1997a). (The plateau of peak synaptic density lasts from 2–3 months to 3–4 years in macaques, and may not show such pronounced differences between frontal and occipital regions; Goldman-Rakic et al., 1997; Huttenlocher & Dabholkar, 1997a.)

Pruning is the next stage of synaptic development. From ages 7–8 years through adolescence and into young adulthood, to about age 20, there is a period of decrease in synaptic density in both frontal and posterior regions, down to a lower plateau that is maintained until after age 60 (Huttenlocher, 1979). Adult synaptic density is about 60% of that during the peak plateau period (Giedd et al., 1999; Goldman-Rakic et al., 1997; Huttenlocher & Dabholkar, 1997a). During late childhood and adolescence, while synaptic density is decreasing, it is thought that unused synaptic connections disappear, or are "pruned" (Changeux, 1993; Huttenlocher & Dabholkar, 1997a; Moody, 1998). Although skeletal growth in humans undergoes an adolescent growth spurt (Bogin, 1999), developmental changes in synaptic density appear to proceed gradually through this period (Giedd et al., 1999).

The cognitive consequences of synaptic pruning are speculative at this point. Synapses that are used more often remain, while those that are rarely used appear to be pruned (Johnston, 1995; Moody, 1998). It has been proposed that pruning increases the efficiency of information processing in the cortex (Goldman-Rakic et al., 1997; Moody, 1998; Pribram, 1997). Goldman-Rakic et al. (1997) and Huttenlocher and Dabholkar (1997b) both stress that it is important to separate learning from changes in synaptic density, and state that learning appears to result from changes in the strength of *existing* synaptic connections, not from the *formation or loss* of synaptic connections. Since learning continues throughout adulthood and changes in synaptic density appear to plateau at adult levels by about age 20, this seems to be an important possible distinction. However, refinement of a cognitive process or skill during development, which they both say is what synaptic pruning allows, would seem to be procedural learning. Furthermore, the discovery of adult neurogenesis and pruning of neurons in the hippocampus, with

some proposed role in learning and memory (Djavadian, 2004), means we should be cautious in asserting that learning can only occur through one kind of synaptic change.

Myelination, which allows neural signals to travel more rapidly, also proceeds in stages. It begins near birth in central subcortical white matter, spreads posteriorly, and only later anteriorly, not reaching prefrontal regions until after 6 months (Benes, 1997; Huttenlocher & Dabholkar, 1997b; Yakovlev & LeCours, 1967). Myelination of frontal cortex continues through childhood, adolescence, and into young adulthood (Benes, 1997; Yakovlev & LeCours, 1967). Myelination has traditionally been thought to be an excellent index of brain maturity. However, because some areas never fully myelinate even in the adult (e.g., callosal connections between frontal areas), some claim that myelination is less useful as a marker of brain maturation than changes in synaptic density (Goldman-Rakic et al., 1997).

This general pattern of brain ontogeny—neurogenesis, early, dense synapse formation followed by synaptic pruning, and sequential myelination of different regions—seems to be a general pattern of primate brain development, although the extent and timing of different phases of development differ between species. Primates have capitalized on the extension of development. For a species to have an adult brain of a particular size, that species must go through the necessary ontogenetic stages to wire up that brain appropriately, and for larger brains, these stages of brain development will take longer.

Late Development of Complex Skills

Although many cognitive abilities in humans first emerge during the period of peak synaptic density, many of our more complex cognitive functions do not reach adult levels of efficiency and competence until late adolescence or early adulthood, when synaptic pruning and myelination are complete (Huttenlocher & Dabholkar, 1997b; Pribram, 1997). For example, while by age 4 children may be able to produce and understand the syntax required by embedded sentences, the more subtle skills required for more complex syntax and conversation take longer to master (Bosacki, 2003; De Villiers & Pyers, 2002; Smith, Apperly, & White, 2003). Children at age 4 show some evidence of understanding the future (Suddendorf & Busby, 2003), and many adolescents can master frontal executive function tasks (Welsh, Pennington, & Groisser, 1991). However, adolescents in general are not known for

their ability to plan ahead and anticipate future consequences, a major function of planning and executive abilities. Development in these cognitive abilities proceeds throughout adolescence (Pribram, 1997). It takes about 19–21 years to develop a functioning adult *Homo sapiens sapiens* brain. Thus, extended childhood seems to allow for an extended period of plasticity in neural development, while extended adolescence allows for a longer period of synaptic pruning—fine-tuning of cortical skills and processes through experience—and an increase in processing speed through myelination.

Extension of Development During Hominid Evolution

Recent data on dental development indicate that this extension of childhood and adolescence is a recent evolutionary phenomenon in the hominid line (Dean et al., 2001). The cyclical deposition of enamel on teeth results in periodic markings in dental tissue, called striae of Retzius, and the surface ridges on the tooth formed by these striations are called perikymata. Counting perikymata allows an estimate of crown formation times and thus an estimate of the rate of development (Dean et al., 2001). Dental development rates in australopithecine species and *Paranthropus* overlap with those of great apes, whereas Neanderthals demonstrate crown formation times more closely resembling those of modern humans. One might expect that early species of *Homo*, *Homo habilis* and *Homo erectus*, would show an intermediate pattern of development, but the surprising fact is that crown formation times in these species overlap completely with those of australopithecines (Dean et al., 2001). Thus, the pattern of extended ontogeny appears to be a recent evolutionary change, occurring since *Homo erectus*, and coincides in evolutionary time with the expansion of brain size since *Homo erectus*. Archaic *Homo sapiens* species had cranial capacities of 1,000 cc or more, compared to ~800 for *Homo erectus*, and Neanderthals and early modern humans had cranial capacities of 1,200–1,500 cc (Falik, 1987; Foley, 1997; Smith, Gannon, & Smith, 1995). It is these larger-brained species that seem to have longer childhoods, based on dental data (Dean et al., 2001).

Complex Cognition and the Executive Brain

One would expect to see a close relationship between the time it takes the brain to develop and the size of the brain. For which structures should this relationship be strongest? Humans' most complex, cortically

mediated skills take the longest to develop; thus the size of brain structures subserving these abilities should be most closely related to maturation time. Long-lived organisms may need to be particularly adept at complex problem solving. The longer-lived a species is, the more the environment will change during its lifetime and the more the organism will have to be able to adapt flexibly to change (Allman & Hasenstaub, 1999). Thus, if it is our flexible problem-solving abilities (whether social or physical) and the motor behavior based on those abilities that need the most time to develop, one would expect the size of the executive brain to be most closely linked to the length of time it takes the brain to mature. Other researchers have demonstrated relationships between brain size and measures of maturation in primates (Allman & Hasenstaub, 1999; Allman, McLaughlin, & Hakeem, 1993; Sacher & Staffeldt, 1974; Smith, 1989), but none has investigated the maturation of the executive brain per se, or focused on linking maturational variables to key points in neural development. We can ask whether there is a relationship between the time it takes the brain to develop through childhood and the adolescence and the size of the adult executive brain across primate species. This is a methodologically difficult question to answer because of a lack of available data on brain development in most primate species, but we can get a rough answer by using existing data to approximate the necessary variables.

Measuring Time to Reach Adulthood

For humans and macaques, developmental milestones in life history correspond approximately to milestones in neural development. Peak synaptic density begins to decline at 7.5 years for humans and at 3 years for macaques (Goldman-Rakic et al., 1997; Huttenlocher & Dabholkar, 1997a), whereas the emergence of second incisors occurs at 7.1 years for humans and 2.6 years for macaques (Smith, Crummett & Brandt, 1994; Smith et al., 1995). The time to reach adulthood in neural development for humans and macaques—that is, to reach an adult plateau for synaptic density—is 19–21 years for humans and 4 years for rhesus macaques (Goldman-Rakic et al., 1997; Huttenlocher & Dabholkar, 1997a). Humans first reproduce at an average age of 18 in industrial cultures and 19 in hunter-gatherer cultures (Trinkaus & Tompkins, 1990). Rhesus macaques first reproduce at an average age of 3.7 years (Goldman-Rakic et al., 1997; Harvey & Clutton-Brock, 1985; Huttenlocher & Dabholkar, 1997a). Data on these milestones in neural development are available only for these two primate species. Thus, to essay whether the time to reach adulthood and the size of the executive brain are related, it is necessary to find a proxy variable that corresponds well to the age at which adult levels of synaptic density are reached. Given the correspondences already noted for humans and macaques in relation to average age at first reproduction, or generation time, this seems a reasonable proxy variable to use for time to reach adulthood at the neural level.[2] For data on average age at first breeding, I used published data sets on life history variables in primates and humans (Godfrey, Samonds, Junger, & Sutherland, 2001; Harvey & Clutton-Brock, 1985).

Measuring Executive Brain Size

Data were compiled from published sources on brain volume and volume of different brain structures for 47 species of primates, including *Homo sapiens sapiens* (Godfrey et al., 2001; Harvey & Clutton-Brock, 1985; Rilling & Insel, 1999; Semendeferi & Damasio, 2000; Semendeferi et al., 2001; Stephan et al., 1981). Not all data points were available for all species. Size of neocortex, striatum (basal ganglia), and brainstem were available for 30 species for which life history data were also available. Following Reader and Laland (2002), executive brain size was calculated as a measure of size of neocortex plus striatum.

Controlling for Body Size

Since brain size covaries strongly with body size, some measure of body size must be used as a reference variable to control for allometric effects

2 There are several possible measures of time to adulthood. Time to reach one's full growth in stature is one possibility but is problematic. Adolescents may reach their full adult stature well before they have reached adult levels of cognitive and social maturity. Also, full growth in stature can precede the attainment of full adult weight (Dainton & Macho, 1999). Time to sexual maturity is another possible index of time to adulthood but will underestimate the correct figure. Human females reach menarche at age 13 (industrial cultures) to age 16 (hunter-gatherer cultures) (Trinkaus & Tompkins, 1990), but may not be fully grown at that point. Furthermore, it is often several years after menarche that a first child would be born (5 years in industrial cultures, 3 in hunter-gatherer cultures) (Trinkaus & Tompkins, 1990). For an evolutionary analysis such as this one, only data for hunter-gatherers are relevant. In great apes and many primate species as well, there is a significant delay (1–4 years) between onset of menarche and birth of first offspring (Harvey & Clutton-Brock, 1985). Of these measures of adulthood, only generation time seems to correspond to the data on neural development.

on overall brain size. Brainstem volume is considered the most conservative way to control for this (Barton, 1999; Keverne et al., 1996; Reader & Laland, 2002). Stephan et al. (1981) present data on the size of the medulla and midbrain, but unfortunately not on the size of the pons; thus, medulla + midbrain was used as an estimate of brainstem size, as in Reader and Laland (2002). The ratio of executive brain volume to brainstem volume was calculated as:

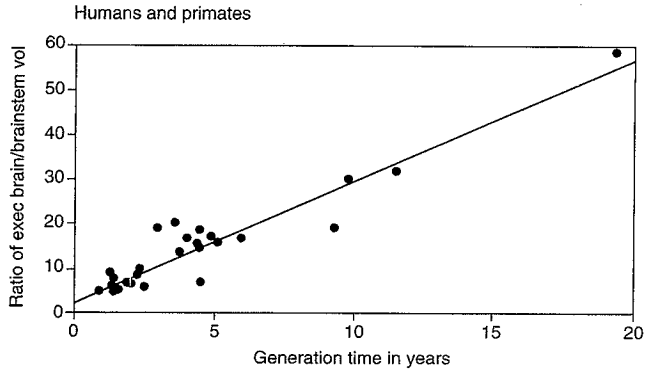
$$\frac{(\text{Volume of neocortex}) + (\text{Volume of striatum})}{(\text{Volume of medulla}) + (\text{Volume of midbrain})}$$

Predicting Executive Brain Size from Time to Reach Adulthood

I ran a linear regression of this ratio of executive brain/brainstem volume on generation time for 30 primate species, including humans. There was a strong linear relationship ($r = 0.95$, $r^2_{adj} = 0.90$, $F_{1,28} = 248.4$, $P < 0.0001$) (figure 4.4A). For primates, including humans, time to reach adulthood explains 90% of the variance in size of the executive brain. The linkage between time to reach adulthood and size of cortex is tight.

One possibility is that our species's data represent such an extreme outlier in executive brain size and time to reach adulthood that they inflate the strength of this linear relationship for primates. Figure 4.4A shows that the human data point lies far from those of other primates on both variables. Are humans typical primates in the relationship between development and brain size? To reduce the effect of the outlier status, I ran a log transformation on both variables, executive brain volume and generation time, and repeated the regression. The results of the analysis do not change substantively with this transformation ($r = 0.90$, $r^2_{adj} = 0.80$, $F_{1,28} = 118.0$, $P < 0.0001$).

To test whether we are typical primates for this developmental pattern, I ran the same regression analysis relating the ratio of executive brain/brainstem volume to time to reach adulthood for the remaining 29 primate species (excluding humans). There was still a strong relationship ($r = 0.88$, $r^2_{adj} = 0.77$, $F_{1,27} = 95.3$, $P < 0.0001$). The data point for humans lies almost exactly on the regression line for nonhuman primates (figure 4.4B). Studentized deleted residuals for the regression in figure 4.4A also show that humans are not significantly different from the value that would be predicted from the regression equation for nonhuman primates (for humans, studentized deleted residual $t_{27} = 1.62$, $P > 0.10$, N.S.; Stevens, 1984). We are indeed like other primates in this respect.



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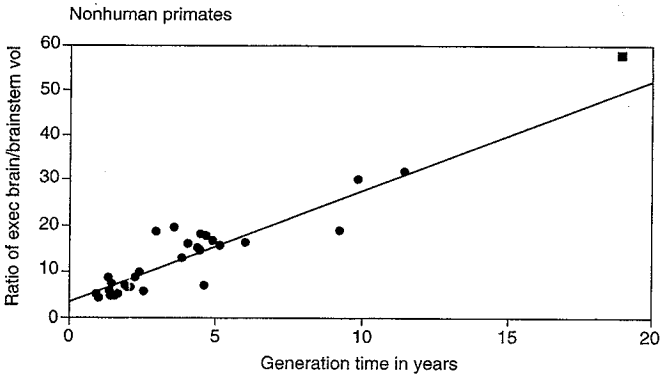

B

Figure 4.4
Regression lines predicting relative size of executive brain in primates an humans (A) and in nonhuman primates alone (B). The regression line looks muc the same in the two cases. When the regression line for nonhuman primates i extrapolated, human data are seen to lie very close to the line. Thus, in this devel opmental relationship, humans appear to show a typical primate pattern, just more extended one.

How Specific Is This Developmental Relationship to the Executive Brain?

Does the linear relationship between executive brain size and generation time exist simply because the executive brain is such a large part of total brain volume? This does not seem to be the case, since this linear relationship also holds true for smaller structures that are involved in executing action. To test the specificity of the relationship between specific brain structure sizes and time to reach adulthood, one can use the volume of several distinct brain structures to predict generation time in a multiple regression. The contribution of each structure to the relationship with generation time can then be determined. The major subdivisions of the brain reported in the data set of Stephan et al. (1981) are neocortex, striatum, brainstem, limbic system, cerebellum, hypothalamus, and thalamus. Accordingly, I calculated the ratio of the volume of each of the following structures to brainstem volume: executive brain, limbic system, cerebellum, hypothalamus, and thalamus. These values were entered into a simultaneous multiple regression predicting time to adulthood. Only relative size of executive brain and thalamus were significantly related to time to reach adulthood (table 4.1). Relative size of the limbic system appears particularly unrelated to time to adulthood. Just as olfactory and limbic structures show some differences in how they scale with other brain structures (Finlay et al., 2001), the size of the limbic system is unrelated to generation time (see table 4.1). One key fact about the executive brain is that the ultimate result is behavior, action. The basal ganglia

Table 4.1
Relationship of Brain Structures (Relative to Brainstem Size) to Generation Time

Variable Tested for Relationship to Generation time	Semipartial r	β	t	P
Executive brain vol./brainstem vol.*	0.21	0.862	4.24	0.001
Limbic system vol./brainstem vol.	0.05	0.056	1.01	0.33
Cerebellum vol./brainstem vol.	−0.04	−0.215	−0.87	0.40
Hypothalamus vol./brainstem vol.	0.04	0.061	0.84	0.42
Thalamus vol./brainstem vol.*	0.13	0.311	2.52	0.025

*$P < 0.05$.
Note: Structures involved in complex problem solving and execution of skilled action show a stronger relationship. Olfactory and limbic structures show no relationship. Limbic system size was calculated as size of hippocampus + ento- and perirhinal cortex + pre- and parasubicular cortices + amygdala + palaeocortex (Stephan et al., 1981).

are included because of their key role in generating action. However, the thalamus could be seen as important for executing skilled actions. Three different frontal-subcortical circuits connect different regions of frontal cortex (dorsolateral, orbitofrontal, and anterior cingulate) to particular parts of the basal ganglia and the thalamus (Cummings, 1993). Thus, the thalamus, in addition to being a sensory relay station for the cortex, is an important structure for behavior generated from frontal lobe computations. One could make an argument for extending the definition of the executive brain to include the thalamus. Maturation time appears to be related primarily to the size of the executive brain and its associated action-related structures.

How Specific Is the Relationship of Brain Size to Time to Reach Adulthood?

Generation time as a measure of time to adulthood was chosen because it is a rough match to time for synaptic density to reach adult levels in humans and macaques. Thus, it seemed the most appropriate choice for testing the relationship between the time for the brain to mature and the size of brain structures. However, many developmental and life history variables are closely related to each other (table 4.2), and thus it may be an extension of development per se that shows a close relationship to executive brain size, rather than only time to reach adulthood. Other developmental variables might also show strong relationships. The age of emergence of second incisors is roughly matched to the age at which synaptic density begins to decrease for humans and macaques (7.5 vs. 7.1, and 4.0 vs. 3.6, respectively). Although data on both age at second incisor emergence and executive brain size are available for only 10 species, the relationship of this developmental variable to the relative size of the executive brain still appears quite strong ($r = 0.92$, $r^2_{adj} = 0.82$, $F_{1,8} = 41.1$, $P < 0.0001$).

To determine if there is a general developmental factor, I chose a set of developmental variables, each coded in years, for which any pair of variables in the set had at least 15 observations in common. This set was gestation time, age at sexual maturity, generation time, and longevity, available for only 20 species. I ran a principal components factor analysis for a two-factor solution on these four variables to determine a primary factor for development. The first factor accounted for 88% of the variance. Generation time loaded most heavily on this factor, gestation time the least (factor loadings: gestation time, 0.87; age at sexual maturity, 0.97; generation time, 0.98; longevity, 0.93).

Table 4.2
Relationship between Variables Measuring Key Points in Development for Primates and Humans

Time Point	GST	LD	M1	M2	I2	M3	SM	GT
GST (gestation time)	1.0							
Tooth emergence: LD (last deciduous ["baby"] teeth)	0.81* (N = 21)	1.0						
M1 (first molars)	0.92* (N = 22)	0.96* (N = 21)	1.0					
M2 (second molars)	0.88* (N = 19)	0.96* (N = 19)	0.98* (N = 20)	1.0				
I2 (second incisors)	0.97* (N = 17)	0.84* (N = 17)	0.96* (N = 18)	0.92* (N = 18)	1.0			
M3 (third molars ["wisdom teeth"])	0.89* (N = 16)	0.96* (N = 14)	0.98* (N = 16)	0.99* (N = 15)	0.91* (N = 13)	1.0		
SM (age at sexual maturity)	0.80* (N = 37)	0.91* (N = 13)	0.92* (N = 14)	0.98* (N = 11)	0.84* (N = 9)	0.96* (N = 10)	1.0	
GT (generation time)	0.81* (N = 49)	0.94* (N = 19)	0.95* (N = 22)	0.96* (N = 17)	0.90* (N = 17)	0.95* (N = 16)	0.97* (N = 36)	1.0
LG (longevity)	0.65* (N = 41)	0.88* (N = 15)	0.90* (N = 15)	0.87* (N = 13)	0.84* (N = 12)	0.91* (N = 11)	0.87* (N = 32)	0.87* (N = 38)

*$P < 0.001$.
Note: Data from Godfrey et al. (2001); Harvey and Clutton-Brock (1985); Smith, Crummett, and Brandt (1994); and Smith, Gannon, and Smith (1995).

I then ran a regression of relative executive brain size (executive brain volume/brainstem volume) on this developmental factor to determine how well development in general explained executive brain size. As with generation time, the relationship was strongly linear ($r = 0.96$, $r^2_{adj} = 0.93$, $F_{1,18} = 227.1$, $P < 0.0001$). A general developmental factor that included more life history and developmental variables would undoubtedly be an even stronger predictor, but estimates of a developmental factor based on fewer than 20 observations would not likely be stable. Although time to reach adulthood is clearly an important developmental variable, strongly related to executive brain size, the relationship may be to length of development overall. Given the strong correlations between developmental variables, genes extending one phase of development may extend all phases.

Are Particular Subdivisions of the Executive Brain Important in Determining This Developmental Relationship?

The executive brain includes the entire neocortex. Cognitive neuroscience, of course, focuses on much more fine-grained distinctions between the functions of different cortical regions. In a perfect world, data on the size of many specific cortical regions would be available for a large number of primate species, and we could ask focused questions about how the size of different cortical regions might be related to development. Multiple regression analysis could be used to determine if certain subdivisions of neocortex contribute more to the developmental relationship with time to reach adulthood than do others. However, currently this can be done with only a few data points, and so it must be considered provisional and speculative at best. Size data for subdivisions of the neocortex are not available for many species. Although some excellent imaging work has been done in recent years to measure gyrification and the size of the whole brain and of the frontal, temporal, and parieto-occipital regions, this has only been done for 6–11 species (Rilling & Insel, 1999; Semendeferi & Damasio, 2000; Semendeferi et al., 2001). Of these, data on time to reach adulthood are available for only six species, and data on the general developmental factor calculated above are available for only five. Furthermore, brainstem size from Stephan et al. (1981) as a control for body size is available on only four of the six species. To be able to analyze data for even six species, a different way of controlling for overall size must be used here. Though

dividing by brainstem size is preferable, some authors argue for using brain region size as a proportion of total brain size (e.g., Barton & Dunbar, 1997; Clark, Mitra, & Wang, 2001). Using brainstem size is considered more conservative and avoids the problem of using the size of the structure itself as part of the control variable (Keverne et al., 1996; Oxnard, 2004). However, using proportion of total brain volume allows us to analyze six species instead of four. If one part of the cortex contributes more to the relationship with time to adulthood than do other parts of the cortex, a multiple regression using relative size of cortical areas to predict time to adulthood should show the relative contributions of these different areas.

From the data published by Semendeferi and Damasio (2000), I computed the ratio of each subdivision of the executive brain to the volume of the whole brain for six species of apes and humans. I ran a simultaneous multiple regression predicting time to adulthood (generation time) from the relative size of frontal, temporal, and parieto-occipital cortex. For these six species, this regression did not reach significance at the 0.05 level for predicting time to adulthood ($r = 0.97$, $r^2_{adj} = 0.86$, $F_{3,2} = 11.2$, $P = 0.083$). Within this regression, the beta weight for frontal volume showed a significant relationship to time to adulthood ($\beta = 1.86$, $P = 0.038$), with the beta weight for temporal lobe volume, approaching significance ($\beta = 0.74$, $P = 0.059$).

However, when a similar regression was run substituting relative frontal pole volume (BA10) for relative frontal lobe volume (from Semendeferi & Damasio, 2000; Semendeferi et al., 2001), the result was highly significant ($r = 0.99$, $r^2_{adj} = 0.99$, $F_{3,2} = 177.3$, $P = 0.006$; relative frontal pole volume, temporal lobe volume, and parieto-occipital volume predicting time to adulthood). Relative frontal pole and temporal lobe volume were significantly related to time to adulthood, parieto-occipital volume was not (table 4.3). These analyses are on a number of variables on a small number of species, and thus have few degrees of freedom and little variability to work with. They can say very little beyond being suggestive of fruitful lines of inquiry for the future if data on more species become available. Nevertheless, the suggestion is there in the data that the frontal and temporal lobes, and the frontal pole in particular, may be the outer limit for the relationship with time to reach adulthood. Because the frontal and temporal lobes are the latest to develop (Giedd et al., 1999; Huttenlocher & Dabholkar, 1997a), it makes sense that size of frontal lobes might have the strongest relationship to total length of time spent in development.

Table 4.3
Relationship of Cortical Areas (Relative to Total Brain Volume) to Generation Time

Variable Tested for Relationship to Generation Time	Semipartial r	β	t	P
BA10 vol./total brain vol.*	0.87	0.95	20.07	0.002
Temporal lobe vol./total brain vol.	0.25	0.25	5.74	0.03
Parieto-occipital vol./total brain vol.	0.021	0.023	0.48	0.68

*$P < 0.05$.
Note: Volume of frontal pole (Brodmann's area 10) shows a stronger relationship to time to reach adulthood than does volume of temporal lobes or parieto-occipital cortex. Both frontal pole and temporal lobe volume are significantly related to time to reach adulthood. Data from Semendeferi and Damasio (2000) and Semendeferi et al. (2001).

Size Isn't Everything

A structure can be complex and have more neurons without becoming much larger over the course of evolution if it becomes more convoluted. Rilling and Insel (1999) measured the degree of whole brain gyrification in 11 primate species. If size of cortex shows a significant relationship to time to reach adulthood, degree of gyrification should as well. For eight species for which data on both time to adulthood and degree of gyrification[3] were available, the regression predicting time to adulthood from degree of whole brain gyrification was highly significant ($r = 0.94$, $r^2_{adj} = 0.87$, $F_{1,6} = 49.1$, $P < 0.0001$). Even with so few species' data, the relationship was quite strong. Gyrification as an index of number of neurons might be as good as or better than structure size. Again, it is to be hoped that such data will become available on more species in the future.

Relationship Between Executive Brain Size and Development for Extinct Hominids

How do our ancestors fit into this developmental relationship? Time to reach adulthood and executive brain size can be estimated independently

3 The gyrification index in Rilling and Insel (1999) is already corrected for whole brain size, so it could be entered directly into the regression without having to take any kind of ratio.

for a handful of extinct hominids. We can then see whether these values fall close to the regression line derived from humans and primates. Since there is no way to get an estimate of brainstem size in hominids, there is no way to place hominids on a graph relating generation time and executive brain/brainstem ratio. However, we can use the regression relating the log transformation of executive brain volume and time to reach adulthood (generation time).

Although no fossilized brains are contained in the archaeological record, we do have several skulls that housed our ancestors' brains, and so we can measure cranial capacity for our ancestors. Cranial capacity is directly related to executive brain size, so that executive brain volume for our extinct ancestors can be estimated using regression techniques (figure 4.5). Furthermore, teeth fossilize and can give us an accurate estimate of development. Dental development rates for fossilized hominid teeth have been used to compare development in apes, australopithecines, *Homo erectus*, Neanderthals, and modern humans, and to derive estimates of the age at which first molars would have emerged for a few species of hominids (Dean et al., 2001; Dean, quoted in Pearson, 2001). As the age of emergence for first molars is strongly related to generation time (see table 4.2), this data can be used to estimate generation time for extinct hominids. When values for executive brain size and for time to reach adulthood are estimated for *Australopithecus afarensis*, Asian *Homo erectus*, and Neanderthals, the data are seen to lie very close to the regression line derived for primates and humans (see figure 4.5). These estimates are quite rough. However, although the extent of childhood and adolescence has changed over hominid evolution (Dean et al., 2001; Smith & Tompkins, 1995), the developmental pattern linking brain size and rate of development appears to have remained the same.

Discussion

Although humans have much larger executive brains and much longer development times than other primates, we are quite typical primates in the developmental pattern linking size of executive brain to length of childhood and adolescence. As shown in the preceding discussion, our time to reach adulthood and our executive brain size can be closely predicted by extrapolating the regression line for other primates. This appears to be true for extinct hominid species as well. If this developmental pattern is common to primates, then relatively small changes in genes regulating the extent of childhood and adolescence may have been

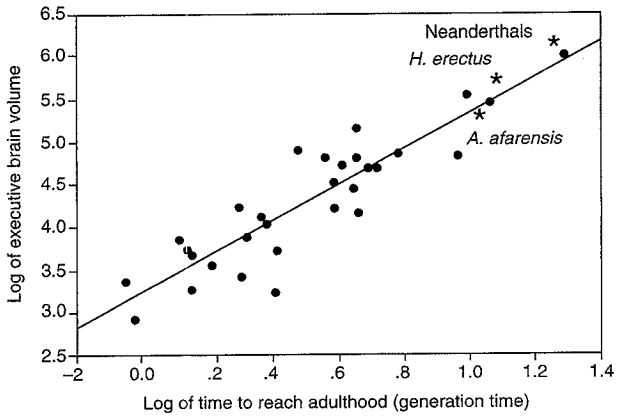

Figure 4.5
Placing extinct hominids on the regression line relating time to adulthood and executive brain size for primates and humans. *Australopithecus afarensis*, Asian *Homo erectus*, and Neanderthals all lie close to the line for primates and humans. Executive brain volume for extinct hominids was estimated using the regression equation: $\log_{10}$ executive brain volume = 2.707 + 1.063 × $\log_{10}$ cranial capacity, derived from primates and humans (N = 38 species). Time to reach adulthood was estimated using the regression equation: time to adulthood = 1.049 + 2.966 × age at first molar emergence, derived from primates and humans (N = 22 species). (Dental data and cranial capacity data from Dean et al., 2001; Falk, 1987; Foley, 1997; Harvey & Clutton-Brock, 1985; Smith, Crummett, & Brandt, 1994; Smith, Gannon, & Smith, 1995.)

key events in the speciation of *Homo sapiens sapiens*. Genes involved in regulating the timing of events in neural development may have been particularly important.

Finlay and Darlington (1995) demonstrated that the size of many brain structures is highly intercorrelated across mammalian species, and perhaps explained by some of the same underlying factors, such as extent of neurogenesis. The above analyses imply that in spite of the strong intercorrelations in size among structures, the size of only certain brain structures, the executive brain and thalamus, is related to time to mature to adulthood. Emphasizing the linkage between *sizes* of different brain structures may neglect key differences in developmental patterns between these structures.

Lieberman, McBratney, and Krovitz (2002) have shown that the size of frontal and temporal lobes may significantly differentiate *Homo sapiens sapiens* from our closest hominid relatives, archaic *Homo sapiens*

and Neanderthals. The key differences in skull morphology that significantly differentiate *Homo sapiens sapiens* from archaic *Homo sapiens* and Neanderthals are in the bones surrounding the temporal lobes, allowing for greater temporal lobe volume, and the high, domed forehead of our species, allowing more room for the frontal pole (Lieberman et al., 2002).[4] The size of these cortical structures, the frontal pole and temporal lobes, may show this relationship to time to reach adulthood most strongly. As these may be relatively late-developing structures, they place an outer limit on when neural maturation is complete (Giedd et al., 1999; Huttenlocher & Dabholkar, 1997a).

The picture of brain evolution that emerges from a comparative analysis of brain development, linked with insights from hominid archaeology, can inform cognitive neuroscience. Evolutionary perspectives on the human brain suggest lines of research relevant to understanding human cognitive uniqueness. Neuroimaging can produce more detailed information about the functions of the frontal pole. Neurological and developmental disorders that disproportionately affect the frontal and temporal lobes, such as schizophrenia or frontotemporal dementia, may provide a window into understanding unique aspects of human cognition. Conversely, the fact that these brain areas have expanded so recently in evolution may explain why they are vulnerable to these particularly human diseases.

Finally, not only our purely cognitive abilities but also our social capacities have made our brains unique. Raising slow-developing offspring is a task requiring significant parental and kin investment. If a large brain requires a long childhood and adolescence, then, as the time to reach adulthood grew longer in the hominid line, the amount of investment in offspring had to increase as well. Parents must invest more time and resources to raise altricial offspring successfully. The high investment required by our extended altritiality would call for multiadult cooperation—the typical mammalian pattern of investment only from the mother would no longer be sufficient to ensure the success of the offspring. Such investment could come from monogamy and paternal investment, but it could also come from looser social arrangements involving multiple adults, such as kin members or polyamorous mating arrangements

4 Although the data of Semendeferi and Damasio (2000) do not show that the temporal lobes of humans are disproportionately larger than would be expected for other apes based on whole brain size, the analyses of Lieberman et al. (2002) point to the temporal lobes significantly differentiating *Homo sapiens sapiens* from our closest hominid relatives.

(Beckerman & Valentine, 2002). Whatever the mating system involved, changes in the attachment system—that is, in the capacity of adults other than the mother to become attached to an infant—must have been among the changes that took place in hominid evolution as well. High parental investment tends not to occur in primates with large size differences between the sexes and high levels of male-male competition (Plavcan & Van Schaik, 1997). *Homo erectus*, subsequent species of *Homo*, and possibly *Australopithecus afarensis* as well are characterized by relatively minor size differences between the sexes (Plavcan & Van Schaik, 1997; Reno, Meindl, McCollum, & Lovejoy, 2003), consistent with a system of multiadult cooperation and high parental investment (Larsen, 2003). Extended development means that selection pressures on brain systems mediating adult care for children would have been significant in hominid evolution. Thus, clues to human uniqueness can also be sought in social neuroscience, in brain systems involving oxytocin and vasopressin, regulating social bonding. Both social neuroscience and cognitive neuroscience can contribute to an understanding of hominid brain evolution.

Ontogeny does not recapitulate phylogeny. However, to understand the ontogeny of the human brain, it is essential to consider phylogeny, because the course of ontogeny has undergone evolutionary change in the hominid line. The analyses presented here point to an important set of methods that can be used in evolutionary cognitive neuroscience. Our closest relatives over the past 6 million years are all extinct. We cannot analyze the brains of our extinct ancestors and cousins directly, but by using comparative analyses such as the ones described in this chapter, we can do so indirectly. Many variables are known to covary in primates. If one variable known for primates is also known for hominids, such as cranial capacity, regression analyses using interrelated variables allows the interpolation of data for extinct hominids. Comparative neuroanatomy and comparative studies on development can provide us with a rich database from which to draw inferences about what was being selected for in the brain over the course of primate and hominid evolution. Brains themselves do not fossilize, but knowing that general developmental pattern for primates and humans, we can begin to understand brain development in our extinct hominid ancestors.

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