M. A. Hofman and D. Falk (Eds.)
Progress in Brain Research, Vol. 195
ISSN: 0079-6123

CHAPTER 4

Embracing covariation in brain evolution: Large brains, extended development, and flexible primate social systems

Christine J. Charvet* and Barbara L. Finlay

Behavioral and Evolutionary Neuroscience Group, Department of Psychology, Cornell University, Ithaca NY, USA

Abstract: Brain size, body size, developmental length, life span, costs of raising offspring, behavioral complexity, and social structures are correlated in mammals due to intrinsic life-history requirements. Dissecting variation and direction of causation in this web of relationships often draw attention away from the factors that correlate with basic life parameters. We consider the "social brain hypothesis," which postulates that overall brain and the isocortex are selectively enlarged to confer social abilities in primates, as an example of this enterprise and pitfalls. We consider patterns of brain scaling, modularity, flexibility of brain organization, the "leverage," and direction of selection on proposed dimensions. We conclude that the evidence supporting *selective* changes in isocortex or brain size for the *isolated* ability to manage social relationships is poor. Strong covariation in size and developmental duration coupled with flexible brains allow organisms to adapt in variable social and ecological environments across the life span and in evolution.

Keywords: evolution; primate; cortex; social; variation.

Introduction

Beware Procrustes bearing Occam's Razor.[1]

Numerous attempts have been made to account for the high intelligence and impressively large isocortex of primates, and especially humans. Theories about the evolution of large brains can be broadly divided into two classes, organized by direction of causality. The first class focusses on the energetic requirements of large brain production, looking for an innovation allowing greater or more efficient energy acquisition or

*Corresponding author.
Tel.: +1-607-255-3996; Fax: (607) 255-8433
E-mail: charvetcj@gmail.com

[1]Attributed to Lise Menn, Department of Linguistics, University of Colorado. Procrustes is the Greek innkeeper of mythology who either sawed off the legs of his guests or stretched them on a rack to make them fit his beds. Occam's razor, of course, is the scientific principle of parsimony, to prefer the simplest theory from a set of contenders to explain some phenomenon.

DOI: 10.1016/B978-0-444-53860-4.00004-0

utilization, assuming the benefits of a large brain to be manifest (e.g., the expensive-tissue hypothesis, Aiello and Wheeler (1995); the radiator hypothesis, Falk (1990) and Falk and Gage (1998); the introduction of cooking, Carmody and Wrangham (2009); the conscription of alloparental care, Isler and Van Schaik (2009)). These theories have intrinsic bidirectional, "racheting," or exaptive aspects explored by their authors. In the case of the "expensive-tissue" hypothesis, reallocation of metabolic costs from gut to brain permits a larger brain to be supported, enhancing memory for the location and seasonality of high-quality foods, which then allow further increases in brain size, and so forth. In the case of fire use for food preparation, selection for the cognitive competency to produce fire not only allows more nutrients to be extracted from foodstuffs but also ritualizes eating as an intrinsically social activity. Biparental care for young allows more numerous and/or larger offspring but also immediately provides more opportunities for learning from conspecifics, typically over a longer period. We argue here that the multiple types and levels of causality embedded in most of these scenarios should be acknowledged at the outset.

The second class of theories about primate brain size and intelligence focusses on the selection for specific behavioral or cognitive competencies as the essential change in brain organization (e.g., the social brain hypothesis, Dunbar (1992); symbol-making, Deacon (1990); recursion in the language facility, Hauser et al. (2002); and many more, Finlay (2007) and Sherwood et al. (2008)). We examine a particular hypothesis, the social brain hypothesis, as emblematic of this approach, not as a theory *per se* deserving of special criticism! We will argue that a focus on brain size and a specific behavioral adaptation neglects to consider coordinated variations in developmental schedules, body, brain, and brain region size. This focus is so much an obligatory feature of the basic discriminative methods of analysis anthropologists and comparative anatomists have been using, that it rarely receives any scrutiny. These tactics simultaneously focus attention not only away from the wide variations in social systems within species but also away from the necessary coordination and covariance of changes in size (i.e., body, brain, brain region) and developmental duration across species. We conclude that brain or isocortex size is unlikely to have *selectively* expanded to manage social systems as many energetic, ecological, and behavioral factors together coordinate changes in development and brain structure. We argue that it is essential to consider the coordinated nature of variations in size and time between and within species to understand human brain evolution.

The social brain hypothesis

The social structure of primates is highly diverse. Some primates are monogamous, while other primates are polygamous or polyandrous (Smuts et al., 1986). Some primates live in groups forming simple or complex social hierarchies, while other primates are mainly solitary (Smuts et al., 1986). The finding that brain size and specifically the relative size of the isocortex positively correlate with group size has been used to argue that bigger groups require large brains and larger isocortices to manage social relationships (Dunbar, 1992, 1993, 2009; Dunbar and Shultz, 2007a,b; Lehmann and Dunbar, 2009; Shultz and Dunbar, 2007). In support of this hypothesis is the finding that the residuals of a phylogenetically controlled linear regression between brain and body size were found to positively correlate with aspects of social structure and behavior related to group size in primates (Pérez-Barbería et al., 2007). Moreover, the size of the isocortex, in particular, relative to the rest of the brain, was found to positively correlate with group size in primates (Fig. 1; Dunbar, 1992). Taken together, these correlative analyses have been used to argue that larger brains and proportionately enlarged isocortices endow

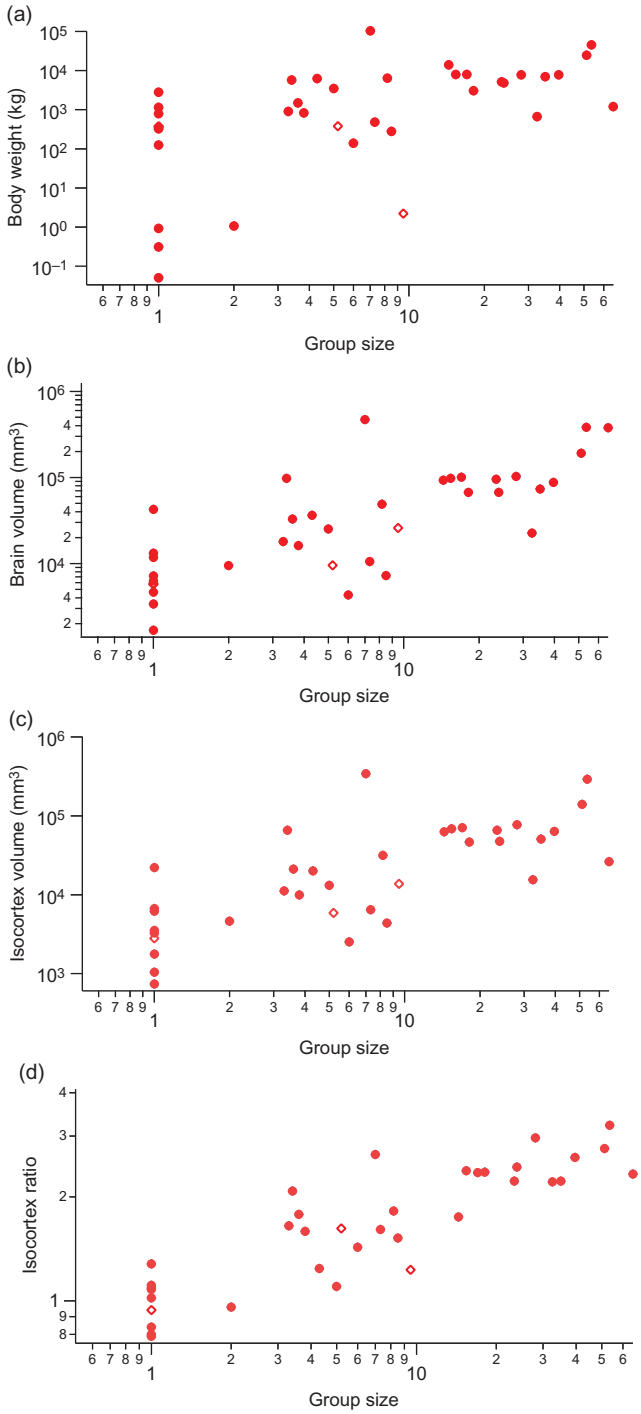

Fig. 1. Body weight (a), brain volume (b), isocortex volume (c), and the size of the isocortex relative to the rest of the brain (d) are regressed against group size in primates. Open diamonds are averages of species. Data are from Dunbar (1992); isocortex volume is gray matter with a white matter component as detailed in Stephan et al. (1981).

primates with increased cognitive "computational power" to manage larger number of possible social interactions and relationships (Dunbar, 1992, 1993, 2009). Although these analyses attempt to correct for possible confounds in cross-species variations in overall brain and body size, the analyses used to support the social brain hypothesis neglect to consider the behavioral setting of allometric and developmentally coordinated changes in brain region size: to consider the brain's intrinsic plasticity and its fundamental role in learning and adapting to both physical and social environments. In addition, the magnitude of the residualized change in brain size attributable to discriminable social factors or identified behavioral capacities of any kind in most cases is extremely small compared to the range over which brains vary. This notable and persistent discrepancy has caused speculation that there might be "two kinds" of size: shared and unshared (residualized) variation in brain size might be physically distinct in some way (Aboitiz, 1996). This is an interesting idea, but one that still has no empirical support.

Coordinated changes in time and size

Overall body size varies widely between primate species ranging from 56 g in gray mouse lemur (*Microcebus murinus)* to 105 kg in gorillas (*Gorilla gorilla*). The brain occupies 3% of the overall body volume in the gray mouse lemurs compared with 0.4% of the overall body in gorillas (Stephan et al., 1981). Well-known comparative analyses of brain and body size across mammalian species show that the overall brain size scales with a negative allometry when regressed against the body (Armstrong, 1982; Jerison, 1973; Jerison, 1979). That is, as bodies expand, brains get proportionately smaller.

The size of the isocortex also varies widely between primate species ranging from 0.74 cm^3 in the gray mouse lemur to approximately 1000 cm^3 in humans (Stephan et al., 1981). The size of the isocortex occupies only 44% of the overall brain in the gray mouse lemur compared to 80% of their entire brain in humans (*Homo sapiens*; Stephan et al., 1981). It might therefore seem reasonable to assume that the proportionally enlarged isocortex of humans sets us apart from other primates. However, it has long been established that the size of the brain of humans, and the isocortex particularly, is an allometrically scaled-up version of its close relatives (Hofman, 1989, See Chapter 18).

Mammalian and nonmammalian vertebrates exhibit a conserved pattern of brain scaling (Figs. 2 and 3; Darlington et al., 1999; Finlay et al., 2011; Finlay and Darlington, 1995; Reep et al., 2007; Yopak et al., 2010). Some brain regions (e.g., medulla) scale with a negative allometry when regressed against the rest of the brain. Other brain structures such as the isocortex scale with a positive allometry when regressed against the rest of the brain (Finlay and Darlington, 1995) so that as mammalian brain sizes expand, the more they come to be dominated by the volume of the isocortex (Fig. 4).

Isocortical subdivisions vary widely between primate species. For instance, the frontal cortex occupies 19% of the overall brain in a monk sakai (*Pithecia monachus*), whereas the frontal cortex occupies approximately 42% of the overall brain in humans (Smaers et al., 2010, 2011). Isocortical regions, such as "primary visual cortex" or "frontal cortex" (variously defined), also exhibit distinct allometric scaling with the rest of the isocortex (Fig. 3; Bush and Allman, 2004; Kaskan et al., 2005; Smaers et al., 2010, 2011). Other isocortical subdivisions (e.g., primary somatosensory isocortex) scale with a negative allometry when regressed against the rest of the isocortex (Fig. 3; Kaskan et al., 2005). Taken together, these findings demonstrate that as primate brains get bigger, the isocortex and, in particular, the frontal and visual cortices become disproportionately enlarged relative to the rest of the brain.

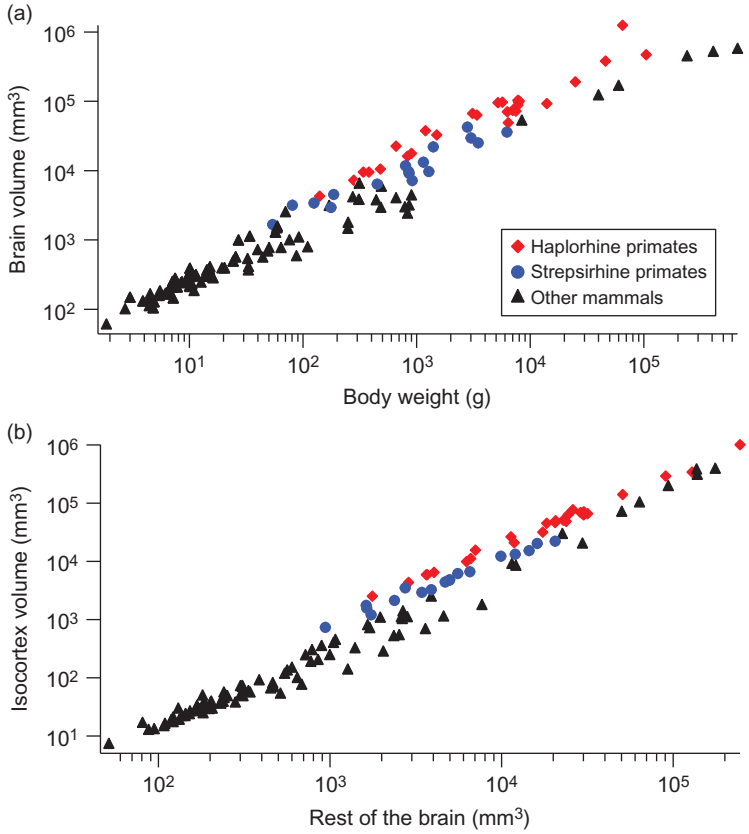

Fig. 2. Brain volume as a function of body weight (a) and isocortex volume as a function of the rest of the brain (b) in primates and other mammals. These data show that the overall brain and isocortex size is disproportionately expanded in primates relative to many other mammals, but that the size of the brain and isocortex strongly covaries within taxonomic groups. Data are from Stephan et al. (1981).

How might these coordinated changes in body, brain, and brain region size arise in development? Finlay and Darlington (1995) have argued that the conserved pattern of brain scaling is mirrored in a conserved pattern in the sequence of developmental events. Allometric variations in adult brain size arise because of the exponential increase in the progenitor pool population when developmental schedules lengthen. That is, progenitor cells that exit the cell cycle late in development benefit from an exponential multiplication of cells relative to progenitor cells that exit the cell cycle early in development (Finlay et al., 2001). The effect of stretching developmental schedules is that structures that are born late in development (e.g., isocortex) become proportionately enlarged relative to the overall brain.

We should emphasize here that we are not using this description of development as an argument for overwhelming "developmental constraint," brain or behavioral uniformity, but rather to lay out the reasonably simple basis for the nonlinear behavior of the vertebrate brain "Bauplan" as it enlarges. Within the context of this Bauplan, new phenotypes emerge, arising from such diverse developmental mechanisms as

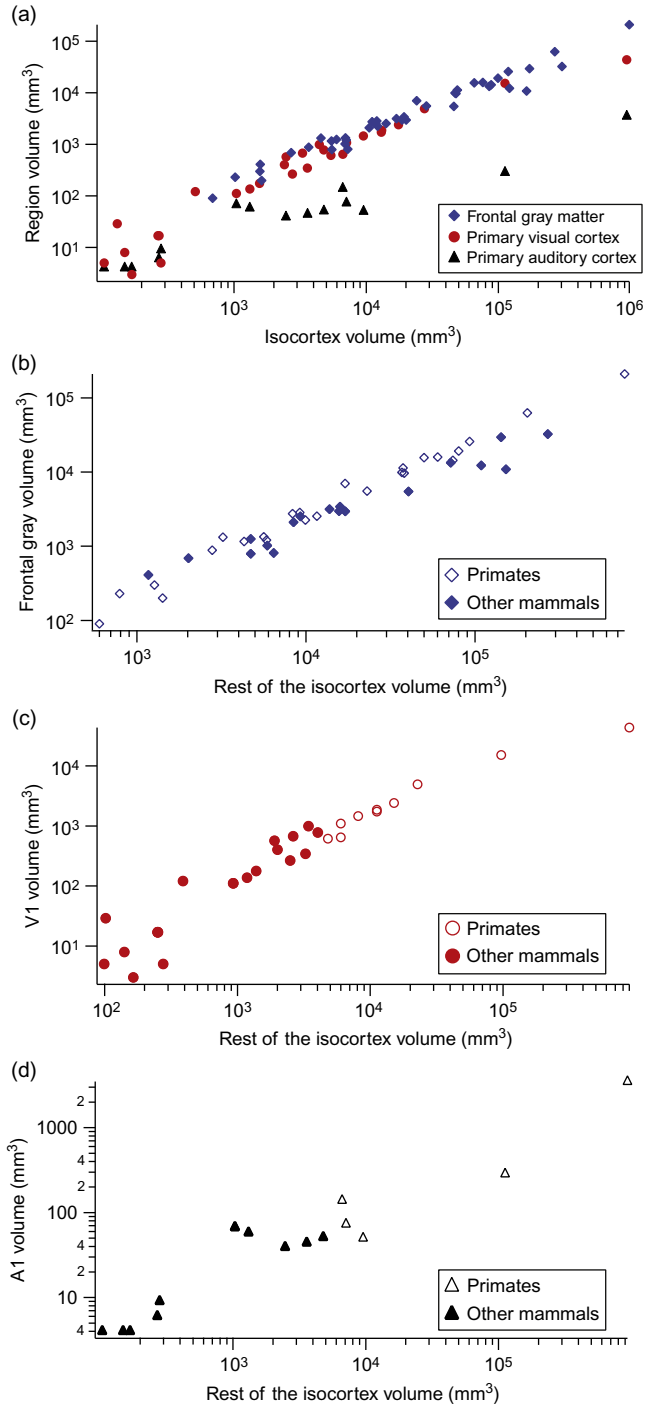

Fig. 3. Isocortical regions are plotted against the overall isocortex volume (a) or the rest of the isocortex volume (b–d) in several mammalian species. The frontal gray matter and the primary visual cortex (V1) expand faster than the size of the primary auditory cortex (A1) as overall brain size increases. These observations suggest that various isocortical regions expand with a distinct allometry. Data are from Kaskan et al. (2005) and Smaers et al. (2010).

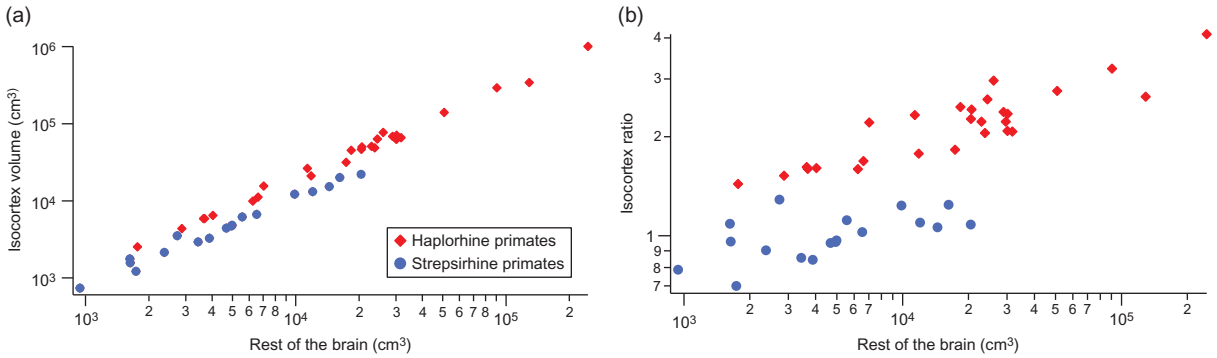

Fig. 4. Isocortex volume (a) and isocortex volume relative to the rest of the brain (b) are plotted against the rest of the brain (i.e., brain–isocortex) in haplorhine and strepsirhine primates. As brains expand, the isocortex becomes disproportionately enlarged in strepsirhine and haplorhine primates. This is evident from the observation that the relative size of the isocortex increases as the rest of the brain expands. Data are from Stephan et al. (1981).

changing rates of cell division (Charvet et al., 2011), early alterations in gene expression patterns that specify brain regions (McGowan et al., 2010; Menuet et al., 2007; Sylvester et al., 2010), heterochronic shifts of "clocks" of cell specification with respect to the establishment of precursor pools to change neuron proportions (Finlay, 2008; Dyer et al., 2009), and extended or limited receptor distributions altering motivational strengths (Young and Wang, 2004). Rather than ignoring "common variance" in brain size by examining residuals, we argue that the stability of this fundamental plan is so strong that it demands a functional explanation of its own.

Variation in size and time

Selective changes in brain region size between taxonomic groups are called grade shifts (Barton and Harvey, 2000). Primate suborders exhibit a number of grade shifts in brain and brain region size. Haplorhine primates (i.e., new-world and old-world monkeys and apes) exhibit a disproportionately enlarged brain and a disproportionately enlarged isocortex relative to that of strepsirhine primates (i.e., lemurs, lorises, galagos; Figs. 2 and 4; Barton and Harvey, 2000; Finlay et al., 1998; Stephan et al., 1981). Both haplorhine and strepsirhine primates exhibit a disproportionately enlarged isocortex relative to many other mammals. However, within each primate suborder, the size of the isocortex is extremely predictable when regressed against the rest of the brain (Fig. 2).

Comparative developmental studies in primates and other mammals show that the grade shifts just described in isocortex size may arise due to selective alterations in the timing of developmental schedules. Comparative analyses of isocortex generation and development showed that haplorhine primates (i.e., rhesus monkeys, humans) selectively delay isocortical neurogenesis compared to rodents (i.e., rats, mice, hamsters, spiny mice, guinea pigs; Clancy et al., 2000, 2001, 2007; Finlay et al., 1998). Relative delay in isocortical neurogenesis entails that the isocortical progenitor pool population will multiply exponentially relative to other nondelayed structures and the isocortex will expand in neuron number and size. Note that we have described at this point two separate, but logarithmically additive, ways of increasing relative cortex size. Increase in duration of development alone to produce a larger brain, with rate of cell production unchanged, automatically increases the relative proportion

of the isocortex. The primate isocortex expands disproportionately still more, adding to the fundamental nonlinearity of allometric scaling an increase in the dedicated precursor cell pool for the cortex, by delaying isocortical stem-cell cessation with respect to the schedule established in rodents and insectivores, and thereby producing a "grade shift."

Some intrinsic difficulties on the use of residuals and ratios in allometric studies

In a series of studies beginning in the early 1990s, Dunbar and his colleagues showed that the relative size of the isocortex or brain size and residuals derived from a linear regression of brain and body size positively correlate with group size or related measures of group size in primates (Dunbar, 1992, 2009; Dunbar and Shultz, 2007a,b; Pérez-Barbería et al., 2007). In parallel with statistical practices in the field, the first studies looked at basic regressions between two variables. The next set of studies used more elaborate multiple regression techniques and phylogenetic contrasts to eliminate the statistical problem of non-independence of taxonomic relationships. Recent studies attempted to determine aspects of temporal emergence of the correlated features examined using discretized variables in conjunction with extensive phylogenetic analyses (Pérez-Barbería et al., 2007). As statistical analyses flourish, it is rare to see any representation of primary data, and the basic "visual" sense of the strength of association, magnitude of results, or amount of variation has tended to fade. In this chapter, following the historical progression of the analyses described, we will plot the basic data relating brain, body, and group size in primates, then add in taxonomic variability, and finally consider the range of variation and a few of the measurement issues in group size, but will go back and plot the basic data on which these claims are established. We should emphasize that we do not contest that there is a relationship between relative brain size, and (possibly) relative isocortex volume and social complexity, generally speaking. The sophistication of the statistical analyses is undoubted. What we do contest are the basic assumptions of the techniques, the causal relationships implied, and the claim that the relationship between social competence and relative brain size, compared to any of a number of other measures of behavioral complexity, is unique.

First, we describe problems with statistical comparisons between groups involving basic allometric relationships between brain parts, to set out very basic issues, which antedate the social brain hypothesis. A number of studies examining the potential mechanisms underlying species-specific adaptations or developmental disorders have focused on the relative sizes of parts of the brain. The initial problem (not a problem of the social brain studies) is "relative to what"? For example, suppose it is shown that the relative size of the frontal cortex is greater in autistic individuals relative to healthy individuals (Carper and Courchesne, 2005; Courchesne et al., 2011), even correcting for a somewhat greater brain size in the autistic group by taking a ratio of frontal cortex to brain volume overall. If individual variation in humans follows primate brain allometry, increases in brain size will produce an even greater increase in the proportion of cortex, and frontal cortex will be a greater proportion still (Fig. 4). "Correcting" for brain size by taking a simple ratio of frontal cortex to brain size between two groups with differing brain sizes will invariably demonstrate relatively more frontal cortex in the group with the larger mean brain size, but this is simply a predictable outcome of the underlying allometry and no indication of any unusual hypertrophy or pathology of the frontal cortex. Although this problem plagues a number of comparative studies in which two species are compared, or brains with a developmental disorder that are compared to normal brains, fortunately, for studies of primate brain evolution, we have ample information to be able to predict the different allometries of various brain divisions.

The use of residuals derived from allometric equations relating the size of two structures is a common method to compare brain region size across species. In the case of the social brain hypothesis, the finding that residuals derived from the linear regression between brain and body size correlates with group size must account for grade shifts in brain size between haplorhine and strepsirhine primates, and when that is done, a significant statistical relationship remains (Pérez-Barbería et al., 2007). The brains of haplorhine primates are disproportionately enlarged relative to those of strepsirhine primates. However, brain and body size strongly covary within haplorhine and strepsirhine primates. Fitting a linear regression through brain and body size in primates (haplorhines and strepsirhine primates) would fit a linear regression with a different slope and intercept than those obtained by fitting two separate linear regressions through the brain and body of haplorhine and strepsirhine primates (Fig. 5). Returning to the basic data, we look at the amount of association between relative brain size and social group size in these two taxonomic groups. In the case of the social brain hypothesis, the brain versus body residual values obtained for both haplorhine and strepsirhine primates correlate more strongly with group size than does the brain versus body residual values derived from separate linear regressions for haplorhine versus strepsirhine primates (Pérez-Barbería et al., 2007). However, the correlation coefficients derived from the residuals of brain to body size in both scenarios are surprisingly low, and it would appear that something about the grade shift in relative brain and cortex size between these two taxa is accounting for most of the effect. Further analyses from the same laboratory group and others considering phylogenetic contrasts, other behavioral measures, and more elaborate statistics generally

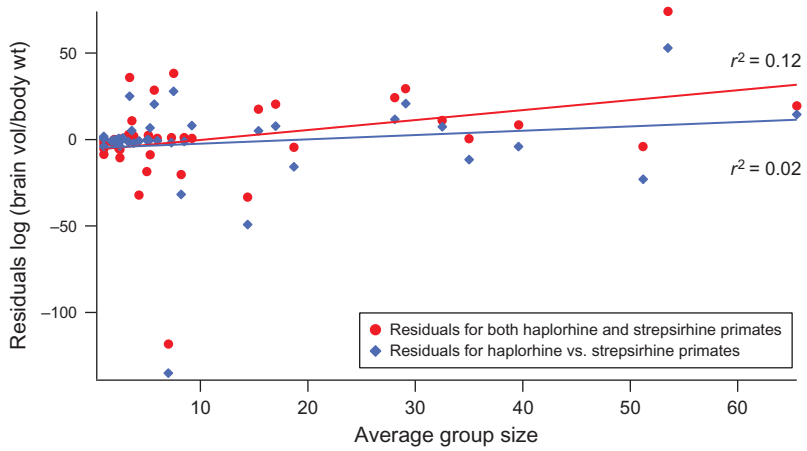

Fig. 5. Residuals derived from a linear regression through the brain size and body size are correlated against group size in primates. In one scenario, residuals are derived from a linear regression through the brain and body size of both haplorhine and strepsirhine primates (red line). In another scenario, residuals are derived from two separate linear regressions derived for haplorhine and strepsirhine primates (blue line). The correlation coefficient between group size and brain versus body size residual values derived from both haplorhine and strepsirhine primates is higher than the correlation coefficient between group size and brain versus body size residuals obtained for strepsirhine and haplorhine primates. Data are from Pérez-Barbería et al. (2007). Although the authors examined the geometric means rather than the arithmetic means of primate group size, the low correlation coefficients between group size and brain versus body size residuals in primates do not support the claim that group size correlates with brain size in primates.

demonstrate a statistically significant but clearly very small effect in residual change in brain size (Barton, 1993; Dunbar, 1993).

We return to the basic data in Fig. 1 of the regressions of primate body, brain isocortex volume, and "isocortex ratio" with group size (taken from Dunbar, 1992). The social brain hypothesis posits that isocortex (either volume or ratio) and group size are positively correlated (isocortex volume residualized with respect to both body size and brain volume). Of interest here is the relationship of body size to social group size, which opens the possibility of many other causal routes between brain size and group size than the ones mentioned here—for example, niche, range size, and type of food consumed. Further, consider other behaviors demonstrated to vary with brain size: "innovation" in the wild, successful invasion of new territories, residual mortality when corrected for body size, and laboratory measures of learning ability. These all correlate with each other and with relative brain size (González-Lagos et al., 2010; Lefebvre and Sol, 2008; Lefebvre et al., 2002; Reader et al., 2011). The strength of the association between isocortex size and group size that would be left after partialling out capability to innovate, or general learning ability, seems unlikely to be significant. Put another way, we would suggest that virtually any reasonable measure of cognitive or behavioral complexity—working memory grammatical sequence learning, innovation and so on—would show the same relationship to relative brain size.

Variation in social structure within a species

The size of social systems and social structures varies widely within a species. Humans form groups that are variable in size (Dunbar, 1993; Zhou et al., 2005). Some humans are polygamous or polyandrous, whereas others are monogamous. Some humans pair-bond with a single individual for life, while others pair-bond for short bouts. Given the variation in social structure within humans, it seems difficult to assign a specific sociality index or group size to humans. Indeed, Dunbar has refrained from estimating group size in humans (Dunbar, 1993). Instead, estimation of group size for humans is based on analyses of group size and brain size of nonhuman primates.

Strepsirhine primates are considered to be solitary or form small groups. In contrast, haplorhine primates are thought to aggregate in large groups. However, there is considerable variation within species in each of these taxonomic groups (Fig. 6; Smuts et al., 1986). Among strepsirhine primates, lemurs such as the white sifaka (*Propithecus verreauxi*) have been observed to be solitary but they may also form groups of up to 13 individuals (Smuts et al., 1986). Among haplorhine primates, rhesus monkeys form groups that range from nine to well over 100 individuals (Berman et al., 1997; Smuts et al., 1986). Gorillas are considered to form large and complex social systems but some members of these species are actually solitary (Smuts et al., 1986). Collectively, these observations suggest that the size of social system varies widely within a species and that estimates of mean group size neglect to consider the wide variation in the size of social systems within each primate species.

Food resource distribution contributes systematically to social organization in vertebrates generally, and mammals and primates specifically (Chapman, 1990a). Resource availability in part determines the decision of birds to contribute alloparental care to relatives, rather than to seek independent reproduction (Emlen, 1974). For instance, in primates, it has been reported that spider monkeys (*Ateles geoffroyi*) from Costa Rica vary from 1 to 35 individuals and 50% of the variance in mean subgroup size can be predicted from the size, density, and distribution of food patches (Chapman, 1990b). These observations suggest that primates are actually highly flexible in modifying the size of their social system in response to resource availability. In the case of humans (Betzig, 2009), it has been persuasively argued that, in cases where resources are physically

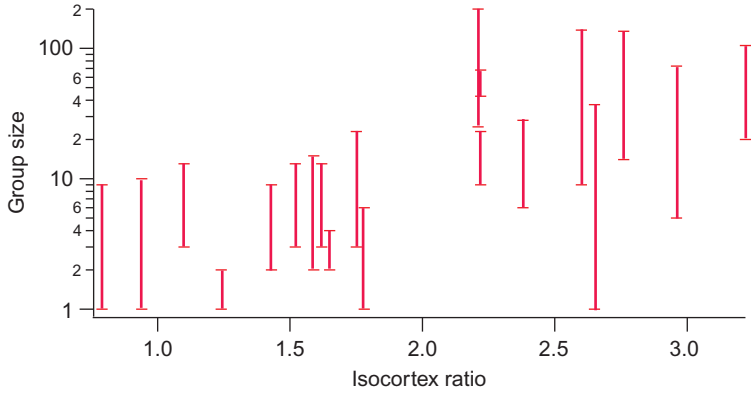

Fig. 6. The range of reported group size within primates is plotted against the relative size of the isocortex. These data show that group size varies extensively within primate species. Data on group size ranges are from Izawa (1976), Smuts et al. (1986), Koenig (1995), and Higham et al. (2009).

stationary, and can be monopolized, as in traditional agrarian societies, the resulting social structure can be characterized as "eusocial," with reproductive activity limited to a few individuals, who can control large harems, with explicitly designated nonreproductive castes.

The social brain hypothesis, quite obviously, does not argue for a complete absence of contribution of other factors to group size, but only for a constraint on maximal tolerable group size related to brain size. When the actual range of natural variation in primate societies is considered, however, the conceptualization of how any individual might be selected on to cope with a particular group size becomes suspect, and the kind of explanations offered seem more like general capacity arguments, rather than a numerical limit on the number of individuals to be remembered.

Constancy in size and time within a species: An unusual example from human pygmies

Developmental schedules in primates, and mammals generally, subsuming brain growth, body growth, maturational milestones, and life span are very highly intercorrelated. Moreover, the initial production of brain tissue is exceptionally predictable as a constant function per unit time (Passingham, 1985), and brain volume, both relative and absolute, is highly correlated with life span. For example, several studies have shown that variations in body size are associated with variations in postnatal growth, life-history schedules, and life expectancy within humans and across species (Charnov, 1991; Migliano et al., 2007; Nettle, 2010). One recent study showed that human pygmies from two different continents reach adult stature and sexual maturity and die earlier than taller individuals (Fig. 7; Migliano et al., 2007). These findings suggest that size, developmental schedules, as well as the overall life span length covary within a species. It is not clear what factors might have caused changes in size, developmental schedules, and the overall life span in pygmies, but there is no evidence that we know of to suggest that a simple social structure or a reduced social system in pygmies caused them to be smaller than taller individuals, or the reverse.

Constancy in brain architecture fosters variation in brain function

The plasticity of the isocortex described in present neuroscience work, and in functional imaging,

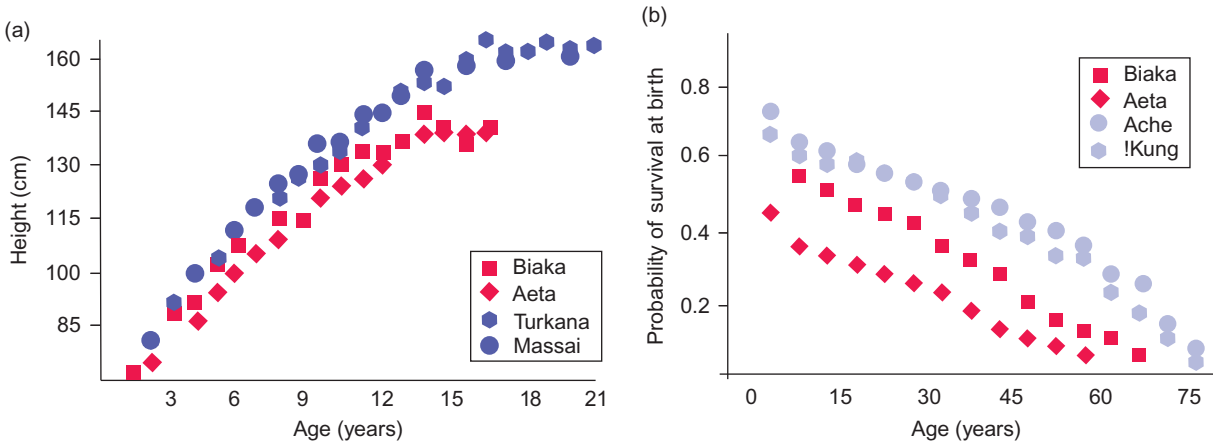

Fig. 7. The Biaka and Aeta are taller than Turkana, Massai, Ache, and !Kung. (a) Biaka and Aeta reach adult height later than Turkana and Massai. (b) Biaka and Aeta have a reduced survival probability at birth than Biaka and Aeta do. Data are from Migliano et al. (2007).

is increasingly at odds with the specificity and modularity of brain function that is often presumed in studies relating brain region size to specific cognitive abilities—this enterprise termed "neuroecology." There is tremendous plasticity in what sensory modalities a brain region may process. It is well known that the visual cortex or auditory cortex may reallocate function to process information from other modalities in the absence of visual input or auditory input. Experimental studies have found that early removal of large regions of the visual cortex, superior colliculus, and the brachium of the inferior colliculus of developing ferrets gives rise to a novel and functional visual pathway that projects through the medial geniculate nucleus to the primary auditory cortex (Roe et al., 1993; Sur et al., 1988). In naturally blind humans, functional magnetic resonance imaging studies show increased blood oxygen level-dependent (BOLD) signals in the visual cortex during tactile discrimination (Sadato et al., 1996; Sathian and Stilla, 2010), auditory-spatial tasks (Collignon et al., 2011), and sentence comprehension tasks (Bedny et al., 2011). Reallocation of function is not restricted to long-term deprivations of one sensory modality. Short-term loss of vision also leads to increased BOLD signal in the occipital cortex during tactile discrimination tasks (Kauffman et al., 2002; Merabet et al., 2008). Collectively, these observations suggest that there is tremendous flexibility in processing information from various modalities. We argue that this flexibility allows organisms to respond to changing social and environmental situations throughout the life span.

At the core of the social brain hypothesis is the assumption that primates with larger isocortices can manage social groups because they have more cognitive capacity for social information than primates that form smaller groups. Does this claim about specific intelligence track well onto specific abilities subserved by cortical areas in humans? Unfortunately, there is little connection between these literatures. In particular, it has been suggested that the size and activation of frontal and temporal isocortices correlate with basic measures of intelligence in humans. In support of this argument is the finding that the size of the isocortex and activation of the frontal and temporal cortices correlate with measures of intelligence and social cognitive performance

in humans (Jung and Haier, 2007; Powell et al., 2010). However, the isocortex obviously mediates more behaviors involved in social situations, and social behaviors are mediated by a multitude of brain regions (Anderson, 2010). Within the temporal cortex, Broca's area is a neural substrate for the perception and production of speech. However, Broca's area is also involved in other tasks such as imitation (Makuuchi, 2005), anticipation of movement, and imagery of motion (Binkofski et al. 2000). Therefore, the finding that activation and size of the isocortex is correlated with measures of intelligence or social cognitive performance does not, in and of itself, show that the expanded isocortex confers specific increased cognitive abilities to manage social situations.

Predictable relationships in brain architecture and brain size

Coordinated variations in developmental length, life span, and brain size appear to have evolved in a number of mammalian taxa. Instances of convergent evolution suggest that this covariation may be intrinsically linked. Similar to haplorhine primates, elephants and cetaceans exhibit an extended period of development, an extended life span, and a large brain (Armstrong, 1982; Haug, 1987; Hofman, 1983, 1993). The observation that elephants and cetaceans differ from their sister groups and most other mammals in being large and having prolonged developmental periods and extended life spans suggests that changes in time and size have evolved together multiple times.

Elephants and cetaceans are considered to be among the longest-lived mammals. Among cetaceans, whales such as the bowhead whale (*Balaena mysticetus*) have been estimated to live over 100 years (George et al., 1999). The Asian elephant (*Elephas maximus*) exhibits some of the longest recorded life spans of land animals, with an estimated maximum life span of approximately 65–86 years (Weigl, 2005; Wiese and Willis, 2004). Elephants and whales not only exhibit extended life spans, but they also exhibit extended periods of postnatal development. For instance, the Asian elephant reaches adult stature at around 17 years of age and it is estimated that the bowhead whales reach sexual maturity after 22 years of age (George et al., 1999). Bowhead whales are among the largest animals weighing 100,000 kg and adult Asian elephants are among the largest land animals weighing approximately 3000 kg. The finding that some elephants and cetaceans are among the largest and most long-lived animals suggests that these taxa expanded and prolonged the duration of developmental length and life span. Taken together, these observations suggest that time and size vary together. The coordinated variation in size and time may entail predictable consequences for behavior.

Some elephants, cetaceans, and primates are well known for their cooperation. We argue that an extended duration of postnatal development entails an extended period of postnatal parental care, which may foster affiliate behavior and cooperation directed toward juveniles or adult group members. In support of this argument is the observation that some primates and elephants not only receive parental care, but they also receive allomaternal or alloparental care (Lee, 1997; Rapaport and Haight, 1987; Riedman, 1982). Some elephants and primates display evidence of life-long affiliate and cooperative behavior toward kin (Langergraber et al., 2007) and nonkin (de Waal et al., 2008; Langergraber et al., 2007; Plotnik et al., 2011). Evidence that long-life histories covary with animal cooperation is also found among nonmammalian vertebrates such as corvids (Møller, 2006; Seed et al., 2008). Taken together, these findings suggest that species that exhibit long-life histories also exhibit evidence of animal cooperation and affiliate behavior. However, it is not clear if sociality may foster coordinated changes in the organism's overall size, brain size, or cortex size. It is possible that coordinated changes in developmental schedules foster

changes in social behavior, and the record of that learning process is the mature isocortex.

Causal scenarios, which depend on covariation, give development a central role

We now return to the bidirectional forms of brain change described in the introductory sections and develop some causal scenarios, which may link the morphological and general behavioral capacities under consideration here. Particularly, we are interested in those behaviors, which allow the brain to play a causal role in its own construction. In primates, big bodies and big brains take longer to make and require more resources. Those larger primate infants are necessarily going to have a longer developmental period to learn in, and because their larger brain size is likely to be associated with biparental or possibly alloparental care, more individuals populate its extended developmental period, both at any moment and over time. This extended learning period may enable these large infants to better develop the categorization skills to differentiate individuals and their motivations, learn elaborate methods of food processing, or learn the unique characteristics and affordances of foraging sites, depending on what the social and natural ecology presents. By its essential, covarying nature, however, a big-brained mammal has an extended developmental period, populated by at least one and often many caretakers, which will make social complexity in large-brained creatures a high probability. While we are uncertain whether group size *per se* is a good measure of a broader notion of social complexity, which must certainly be a multivariate entity, we suggest that the real mediating variable between brain size and behavioral complexity might be developmental time, and not simply the number of neurons available to discriminate individuals.

This view of brain evolution is quite distinct from the one that emerges from the differentiating, residual variance view of brain part evolution. In the view of brain evolution set out by Dunbar and colleagues, additional brain goes directly to improve capacity limitation on the ability to either remember or orchestrate the interactions of a set number of individuals, a direct mapping of a social problem defined numerically to a volume of committed tissue. In our view, the extended developmental schedule required to make a large brain and the size of the brain itself must be considered as one variable. The extreme conservation of this relationship across mammals suggests that there have been few advantages in attempts to decouple rate of production and size of the end product. The motivations each developing organism brings to the environment in combination with its plastic brain allow the information represented in the physical and social environment to construct the mature organism on which natural selection will act.

Acknowledgments

This work was supported by an NSF grant #0849612 to B. L. F. and by the Eunice Kennedy Shriver National Institute of Child Health and Human Development fellowship #F32HD067011 to C. J. C. The content is solely the responsibility of the authors and does not necessarily represent the official views of the Eunice Kennedy Shriver National Institute of Child Health and Human Development or the National Institutes of Health.

References

Aboitiz, F. (1996). Does bigger mean better? Evolutionary determinants of brain size and structure. *Brain, Behavior and Evolution*, *47*, 225–245.

Aiello, L. C., & Wheeler, P. (1995). The expensive-tissue hypothesis: The brain and the digestive system in human and primate evolution. *Current Anthropology*, *36*, 199–221.

Anderson, M. L. (2010). Neural reuse: A fundamental organizational principle of the brain. *The Behavioral and Brain Sciences*, *33*, 245–266 discussion 266–313.

Armstrong, E. (1982). A look at relative brain size in mammals. *Neuroscience Letters*, *34*, 101–104.

Barton, R. A. (1993). Independent contrasts analysis of neocortical size and socioecology in primates. *The Behavioral and Brain Sciences*, *16*, 694–695.

Barton, R. A., & Harvey, P. H. (2000). Mosaic evolution of brain structure in mammals. *Nature*, *405*, 1055–1058.

Bedny, M., Pascual-Leone, A., Dodell-Feder, D., Fedorenko, E., & Saxe, R. (2011). Language processing in the occipital cortex of congenitally blind adults. *Proceedings of the National Academy of Sciences of the United States of America*, *108*, 4429–4434.

Berman, C. M., Rasmussen, K. L. R., & Suomi, S. J. (1997). Group size, infant development and social network in free-ranging rhesus monkey. *Animal Behavior*, *53*, 405–421.

Betzig, L. (2009). But what is government itself but the greatest of all reflections on human nature? *Politics and the Life Sciences*, *28*, 102–105.

Binkofski, F., Amunts, K., Stephan, K. M., Posse, S., Schormann, T., Freund, H. J., et al. (2000). Broca's region subserves imagery of motion: a combined cytoarchitectonic and fMRI study. *Human Brain Mapping*, *11*, 273–285.

Bush, E. C., & Allman, J. M. (2004). The scaling of frontal cortex in primates and carnivores. *Proceedings of the National Academy of Sciences of the United States of America*, *101*, 3962–3966.

Carmody, R. N., & Wrangham, R. W. (2009). The energetic significance of cooking. *Journal of Human Evolution*, *57*, 379–391.

Carper, R. A., & Courchesne, E. (2005). Localized enlargement of the frontal cortex in early autism. *Biological Psychiatry*, *57*, 126–133.

Chapman, C. A. (1990a). Ecological constraints on group size in three species of neotropical primates. *Folia Primatologica: International Journal of Primatology*, *55*, 1–9.

Chapman, C. A. (1990b). Association patterns of spider monkeys: The influence of ecology and sex on social organization. *Behavioral Ecology and Sociobiology*, *26*, 409–414.

Charnov, E. L. (1991). Evolution of life history variation among female mammals. *Proceedings of the National Academy of Sciences of the United States of America*, *88*, 1134–1137.

Charvet, C. J., Striedter, G. F., & Finlay, B. L. (2011). Evo-devo and brain scaling: Candidate developmental mechanisms for variation and constancy in vertebrate brain evolution. *Brain, Behavior and Evolution*, *78*, 248–257.

Clancy, B., Darlington, R. B., & Finlay, B. L. (2000). The course of human events: Predicting the timing of primate neural development. *Developmental Sciences*, *3*, 57–66.

Clancy, B., Darlington, R. B., & Finlay, B. L. (2001). Translating developmental time across mammalian species. *Neuroscience*, *105*, 7–17.

Clancy, B., Kersh, B., Hyde, J., Anand, K. J. S., Darlington, R. B., & Finlay, B. L. (2007). Web-based method for translating neurodevelopment from laboratory species to humans. *Neuroinformatics*, *5*, 79–94.

Collignon, O., Vandewalle, G., Voss, P., Albouy, G., Charbonneau, G., Lassonde, M., et al. (2011). Functional specialization for auditory-spatial processing in the occipital cortex of congenitally blind humans. *Proceedings of the National Academy of Sciences of the United States of America*, *108*, 4435–4440.

Courchesne, E., Campbell, K., & Solso, S. (2011). Brain growth across the life span in autism: Age-specific changes in anatomical pathology. *Brain Research*, *22*, 138–145.

Darlington, R. B., Dunlop, S. A., & Finlay, B. L. (1999). Neural development in metatherian and eutherian mammals: Variation and constraint. *The Journal of Comparative Neurology*, *411*, 359–368.

de Waal, F. B., Leimgruber, K., & Greenberg, A. R. (2008). Giving is self-rewarding for monkeys. *Proceedings of the National Academy of Sciences of the United States of America*, *105*, 13685–13689.

Deacon, T. W. (1990). Rethinking mammalian brain evolution. *American Zoologist*, *30*, 629–705.

Dunbar, R. I. M. (1992). Neocortex size as a constraint on group size in primates. *Journal of Human Evolution*, *22*, 469–493.

Dunbar, R. I. M. (1993). Coevolution of neocortical size, group size, and language in humans. *The Behavioral and Brain Sciences*, *16*, 681–735.

Dunbar, R. I. (2009). The social brain hypothesis and its implications for social evolution. *Annals of Human Biology*, *36*, 562–572.

Dunbar, R. I. M., & Shultz, S. (2007a). Understanding primate brain evolution. *Philosophical Transactions of the Royal Society of London. Series B, Biological Sciences*, *362*, 649–658.

Dunbar, R. I., & Shultz, S. (2007b). Evolution in the social brain. *Science*, *317*, 1344–1347.

Dyer, M. A., Martins, R., da Silva Filho, M., Muniz, J. A., Silveira, L. C., Cepko, C. L., et al. (2009). Developmental sources of conservation and variation in the evolution of the primate eye. *Proceedings of the National Academy of Sciences of the United States of America*, *106*, 8963–8968.

Emlen, J. T. (1974). An urban bird community in Tucson, Arizona: Derivation, structure, regulation. *The Condor*, *76*, 184–197.

Falk, D. (1990). Brain evolution in Homo: The "radiator" theory. *The Behavioral and Brain Sciences*, *13*, 333–381.

Falk, D., & Gage, T. B. (1998). Radiators are cool: A response to Braga & Boesch's published paper and reply. *Journal of Human Evolution*, *35*, 307–312.

Finlay, B. L. (2007). E pluribus unum: Too many unique human capacities and too many theories. In S. Gangestad & J. Simpson (Eds.), *The evolution of mind: Fundamental*

questions and controversies (pp. 294–304). New York: Guilford Press.

Finlay, B. L. (2008). The developing and evolving retina: Using time to organize form. *Brain Research*, *1192*, 5–16.

Finlay, B. L., & Darlington, R. B. (1995). Linked regularities in the development and evolution of mammalian brains. *Science*, *268*, 1578–1584.

Finlay, B. L., Darlington, R. B., & Nicastro, N. (2001). Developmental structure in brain evolution. *The Behavioral and Brain Sciences*, *24*, 263–278.

Finlay, B. L., Hersman, M. N., & Darlington, R. B. (1998). Patterns of vertebrate neurogenesis and the paths of vertebrate evolution. *Brain, Behavior and Evolution*, *52*, 232–242.

Finlay, B. L., Hinz, F., & Darlington, R. B. (2011). Mapping behavioural evolution onto brain evolution: The strategic roles of conserved organization in individuals and species. *Philosophical Transactions of the Royal Society of London. Series B, Biological Sciences*, *366*(1574), 2111–2123.

George, J. C., Bada, J., Zeh, J., Scott, L., Brown, S. E., O'Hara, T., et al. (1999). Age and growth estimates of bowhead whales (Balaena mysticetus) via aspartic acid racemization. *Canadian Journal of Zoology*, *77*, 571–580.

González-Lagos, C., Sol, D., & Reader, S. M. (2010). Large-brained mammals live longer. *Journal of Evolutionary Biology*, *23*, 1064–1074.

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). *American Journal of Anatomy*, *180*, 126–142.

Hauser, M. D., Chomsky, N., & Fitch, W. T. (2002). The faculty of language: What is it, who has it, and how did it evolve? *Science*, *298*, 1569–1579.

Higham, J. P., Warren, Y., Adanu, J., Umaru, B. N., MacLarnon, A. M., Sommer, V., et al. (2009). Living on the edge: Life-history of olive baboons at Gashaka-Gumti National Park, Nigeria. *American Journal of Primatology*, *71*, 293–304.

Hofman, M. A. (1983). Energy metabolism, brain size and longevity in mammals. *The Quarterly Review of Biology*, *58*, 495–512.

Hofman, M. A. (1989). On the evolution and geometry of the brain in mammals. *Progress in Neurobiology*, *32*, 137–158.

Hofman, M. A. (1993). Encephalization and the evolution of longevity in mammals. *Journal of Evolutionary Biology*, *6*, 209–227.

Isler, K., & Van Schaik, C. P. (2009). Why are there so few smart mammals (but so many smart birds)? *Biology Letters*, *5*, 125–129.

Izawa, K. (1976). Group sizes and composition of monkeys in the upper Amazon basin. *Primates*, *17*, 367–399.

Jerison, H. J. (1973). *Evolution of the brain and intelligence.* New York: Academic Press.

Jerison, H. J. (1979). The evolution of diversity in brain size. In M. E. Hahnet al. (Ed.)*Development and evolution of brain size* (pp. 29–57). New York: Academic Press.

Jung, R. E., & Haier, R. J. (2007). The Parieto-Frontal Integration Theory (P-FIT) of intelligence: Converging neuroimaging evidence. *The Behavioral and Brain Sciences*, *30*, 135–154 discussion 154–187.

Kaskan, P. M., Franco, E. C., Yamada, E. S., Silveira, L. C., Darlington, R. B., Darlington, L. C., et al. (2005). Peripheral variability and central constancy in mammalian visual system evolution. *Proceedings of the Royal Society B: Biological Sciences*, *272*, 91–100.

Kauffman, T., Théoret, H., & Pascual-Leone, A. (2002). Braille character discrimination in blindfolded human subjects. *Neuroreport*, *13*, 571–574.

Koenig, A. (1995). Group size, composition, and reproductive success in wild common marmosets (*Callithrix jacchus*). *American Journal of Primatology*, *35*, 311–317.

Langergraber, K. E., Mitani, J. C., & Vigilant, L. (2007). The limited impact of kinship on cooperation in wild chimpanzees. *Proceedings of the National Academy of Sciences of the United States of America*, *104*, 7786–7790.

Lee, P. C. (1997). Allomothering among African elephants. *Animal Behavior*, *35*, 278–291.

Lefebvre, L., Nicolakakis, N., & Boire, D. (2002). Tools and brains in birds. *Behaviour*, *139*, 939–973.

Lefebvre, L., & Sol, D. (2008). Brains, lifestyles and cognition: Are there general trends? *Brain, Behavior and Evolution*, *72*, 135–144.

Lehmann, J., & Dunbar, R. I. (2009). Network cohesion, group size and neocortex size in female-bonded Old World primates. *Proceedings of the Royal Society B: Biological Sciences*, *276*, 4417–4422.

Makuuchi, M. (2005). Is Broca's area crucial for imitation? *Cerebral Cortex*, *15*, 563–570.

McGowan, L., Kuo, E., Martin, A., Monuki, E. S., & Striedter, G. (2010). Species differences in early patterning of the avian brain. *Evolution*, *65*, 907–911.

Menuet, A., Alunni, A., Joly, J. S., Jeffrey, W. R., & Rétaux, S. (2007). Expanded expression of Sonic Hedgehog in *Astyanax* cavefish: Multiple consequences on forebrain development and evolution. *Development*, *134*, 845–855.

Merabet, L. B., Hamilton, R., Schlaug, G., Swisher, J. D., Kiriakopoulos, E. T., Pitskel, N. B., et al. (2008). Rapid and reversible recruitment of early visual cortex for touch. *PLoS One*, *3*, e3046.

Migliano, A. B., Vinicius, L., & Lahr, M. M. (2007). Life history trade-offs explain the evolution of human pygmies. *Proceedings of the National Academy of Sciences of the United States of America*, *104*, 20216–20219.

Møller, A. P. (2006). Sociality, age at first reproduction and senescence: Comparative analyses of birds. *Journal of Evolutionary Biology*, *19*, 682–689.

Nettle, D. (2010). Dying young and living fast: Variation in life history across English neighborhoods. *Behavioral Ecology*, *21*, 387–395.

Passingham, R. E. (1985). Rates of brain development in mammals including man. *Brain, Behavior and Evolution*, *26*, 167–175.

Pérez-Barbería, F. J., Shultz, S., & Dunbar, R. I. (2007). Evidence for coevolution of sociality and relative brain size in three orders of mammals. *Evolution*, *61*, 2811–2821.

Plotnik, J. M., Lair, R., Suphachoksahakun, W., & de Waal, F. B. (2011). Elephants know when they need a helping trunk in a cooperative task. *Proceedings of the National Academy of Sciences of the United States of America*, *108*, 5116–5121.

Powell, J. L., Lewis, P. A., Dunbar, R. I., García-Fiñana, M., & Roberts, N. (2010). Orbital prefrontal cortex volume correlates with social cognitive competence. *Neuropsychologia*, *48*, 3554–3562.

Rapaport, L., & Haight, J. (1987). Some observations regarding allomaternal caretaking among captive Asian elephants (*Elephas maximus*). *Journal of Mammalogy*, *68*, 438–442.

Reader, S. M., Hager, Y., & Laland, K. N. (2011). The evolution of primate general and cultural intelligence. *Philosophical Transactions of the Royal Society of London. Series B, Biological Sciences*, *366*, 1017–1027.

Reep, R. L., Finlay, B. L., & Darlington, R. B. (2007). The limbic system in Mammalian brain evolution. *Brain, Behavior and Evolution*, *70*, 57–70.

Riedman, M. L. (1982). The evolution of alloparental care and adoption in mammals and birds. *The Quarterly Review of Biology*, *57*, 405–435.

Roe, A. W., Garraghty, P. E., Esguerra, M., & Sur, M. (1993). Experimentally induced visual projections to the auditory thalamus in ferrets: Evidence for a W cell pathway. *The Journal of Comparative Neurology*, *334*, 263–280.

Sadato, N., Pascual-Leone, A., Grafman, J., Ibanez, V., Deiber, M.-P., Dold, G., et al. (1996). Activation of the primary visual cortex by Braille reading in blind subjects. *Nature*, *380*, 526–528.

Sathian, K., & Stilla, R. (2010). Cross-modal plasticity of tactile perception in blindness. *Restorative Neurology and Neuroscience*, *28*, 271–281.

Seed, A. M., Clayton, N. S., & Emery, N. J. (2008). Cooperative problem solving in rooks (*Corvus frugilegus*). *Proceedings of the Royal Society B: Biological Sciences.*, *275*, 1421–1429.

Sherwood, C. C., Subiaul, F., & Zawidzki, T. W. (2008). A natural history of the human mind: Tracing evolutionary changes in brain and cognition. *Journal of Anatomy*, *212*, 426–454.

Shultz, S., & Dunbar, R. I. (2007). The evolution of the social brain: Anthropoid primates contrast with other vertebrates. *Proceedings of the Royal Society B: Biological Sciences*, *274*, 2429–2436.

Smaers, J. B., Schleicher, A., Zilles, K., & Vinicius, L. (2010). Frontal white matter volume is associated with brain enlargement and higher structural connectivity in anthropoid primates. *PLoS One*, *5*, e9123.

Smaers, J. B., Steele, J., Case, C. R., Cowper, A., Amunts, K., & Zilles, K. (2011). Cortex evolution: Human brains are the extreme of a lateralized ape trend. *Brain, Behavior and Evolution*, *77*, 67–78.

Smuts, B., Cheney, D., Seyfarth, R., Wrangham, R., & Struhsaker, T. (1986). *Primate societies.* Chicago: University of Chicago Press.

Stephan, H., Frahm, H., & Baron, G. (1981). New and revised data on volumes of brain structures on insectivores and primates. *Folia Primatologica: International Journal of Primatology*, *35*, 1–29.

Sur, M., Garraghty, P. E., & Roe, A. W. (1988). Experimentally induced visual projections into auditory thalamus and cortex. *Science*, *242*, 1437–1441.

Sylvester, J. B., Rich, C. A., Loh, Y. H., van Staaden, M. J., Fraser, G. J., & Streelman, J. T. (2010). Brain diversity evolves via differences in patterning. *Proceedings of the National Academy of Sciences of the United States of America*, *107*, 9718–9723.

Weigl, R. (2005). *Longevity of mammals in captivity; from the living collections of the world.* Stuttgart: Kleine Senckenberg-Reihe.

Wiese, R. J., & Willis, K. (2004). Calculation of longevity and life expectancy in captive elephants. *Zoo Biology*, *23*, 365–373.

Yopak, K. E., Lisney, T. J., Darlington, R. B., Collin, S. P., Montgomery, J. C., & Finlay, B. L. (2010). A conserved pattern of brain scaling from sharks to primates. *Proceedings of the National Academy of Sciences of the United States of America*, *107*, 12946–12951.

Young, L. J., & Wang, Z. (2004). The neurobiology of pair bonding. *Nature Neuroscience*, *7*, 1048–1054.

Zhou, W. X., Sornette, D., Hill, R. A., & Dunbar, R. I. (2005). Discrete hierarchical organization of social group sizes. *Proceedings of the Royal Society B: Biological Sciences*, *272*, 439–444.