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## CHAPTER 19

# Primate encephalization

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**Abstract:** Encephalization is a concept that implies an increase in brain or neocortex size relative to body size, size of lower brain areas, and/or evolutionary time. Here, I review 26 large-scale comparative studies that provide robust evidence for five lifestyle correlates of encephalization (group living, a large home range, a high-quality diet, a strong reliance on vision, arboreal and forest dwelling), six cognitive correlates (better performance in captive tests, more tactical deception, innovation, tool use, social learning, all subsumed in part by general intelligence), one life history correlate (a longer lifespan), two evolutionary correlates (a high rate of change in microcephaly genes, an increase in brain size over macroevolutionary time), as well as three trade-offs (a slower juvenile development, a higher metabolic rate, sexually selected dimorphism). Of the 26 different encephalization measures used in these studies, corrected neocortex size, either with a ratio or a residual, is the most popular structural correlate of the functional variables, while residual brain size is the measure associated with the greatest number of them. Controversies remain on corrected or absolute measures of neural structure size, concerted versus mosaic evolution of brain parts and specialized versus domain-general brain structures and cognitive processes.

Keywords: primate; encephalization; cognition; neocortex; brain.

## Introduction

Brain size shows a strong positive relationship with body size over a large set of animal species, but some species clearly have brains that are much larger than expected, given their body size. Behaviors that would be considered intelligent in

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Tel.: 1-514-398-6457; Fax: 1-514-398-65069 E-mail: louis.lefebvre@mcgill.ca humans have been observed in several of the larger-brained species in different animal classes. Over the past three million years, endocranial volume has increased dramatically in our hominin lineage, as has evidence for key cognitive innovations like biface tool manufacture and fire. Taken together, these three observations have given rise to the idea that something about brain enlargement, once the allometric effects of growth have been removed, has coevolved with cognition. Encephalization is the concept born from this idea. In comparative biology, the concept describes the difference between animals in the amount of neuronal mass or volume available beyond some value predicted by body size. In paleontology, it refers to increases in brain size observed over evolutionary time in some taxa. In neuroanatomy, it describes the relative increase in size of higher brain structures like the neocortex<sup>1</sup> and pallium in classes like birds and mammals compared to the subpallium in more "primitive" clades like fish or reptiles. Anatomical traits like folding are also sometimes described in similar terms, with highly gyrified brains seen as more encephalized than smoother ones. In this chapter, I first discuss some of the controversies surrounding the concept of encephalization, and then review 26 large-scale comparative studies that identify key correlates of primate encephalization.

# Problems with the concept of encephalization

Encephalization is not a simple descriptive concept. It is a relative one that implies a comparison over time, a comparison between taxa, and/or a comparison of some neural structures (so-called higher) with other neural structures (so-called lower) or with the whole body. It is also a concept that mixes two levels of explanation: the structural level of neurons, brains, and bodies and the functional level of information processing. Finally, as evidenced by the terms used in the first paragraph-"something about brain enlargement has coevolved with cognition"-the concept can be fuzzy. What exactly is the "something" in the brain that has coevolved with "cognition"? In fact, what is "cognition" and how can it be operationalized? Is the relative nature of encephalization justified and the removal of the allometric growth component in brain size necessary?

These questions are particularly relevant in primates because recent work in neuroanatomy (Burish et al., 2010; Gabi et al., 2010; Herculano-Houzel et al., 2007) and comparative psychology (Deaner et al., 2007) on this order has raised the possibility that absolute measures of brain and neocortex size may be more relevant than the traditional relative ones. Second, it is in primates that we have the widest array of operational measures of cognition on a wide sample of species, as well as attempts to synthesize this array into a common framework based on general intelligence (Deaner et al., 2006; Reader et al., 2011). The operational measures go from learning and problem-solving tests in captivity (Johnson et al., 2002; Riddell and Corl, 1977) to taxonomic counts of tactical deception (Byrne and Whiten, 1990), innovation, tool use, social learning (Reader and Laland, 2002), and extractive foraging (Reader et al., 2011) taken mostly from the wild. The literature on primates also includes good quantitative estimates of lifestyles where complex cognition might provide a selective advantage, for example, group living (Dunbar, 1998; Dunbar and Shultz, 2007a) or foraging for high-quality dispersed food such as fruit (Barton, 1996; Clutton-Brock and Harvey, 1980; Fish and Lockwood, 2003). Third, primates feature one of the best neuroanatomical databases (Isler et al., 2008; Stephan et al., 1981) to test structural (Barton and Harvey, 2000; Clark et al., 2001; Finlay et al., 2001; Yopak et al., 2010) and functional (Barton, 1998; Dunbar, 1998; Reader et al., 2011) hypotheses.

Based on current knowledge, the structural basis of encephalization in primates can be described as a series of nested scaling relationships that link numbers of neurons and glia, cortical white and gray matter mass, neocortical volume, whole brain, body, and spinal cord mass. The current consensus is that (1) numbers of neurons and glia scale isometrically (i.e., 1 to 1) with neocortex mass as well as brain mass,

<sup>&</sup>lt;sup>1</sup>I use the term "neocortex" throughout, rather than "isocortex," because it is the term most often used in the literature I cover here.

but cell density does not (Gabi et al., 2010; Herculano-Houzel et al., 2007); (2) neocortical gray matter scales isometrically (and white matter with positive allometry, i.e., slope>1) with mass of the rest of the brain (Barton and Harvey, 2000): (3) frontal white and grav matter have a positive allometric relationship with neocortical mass (Smaers et al., 2010), as does (4) brain mass with spinal cord neuron numbers (Burish et al., 2010); (5) brain mass has a negative allometric relationship (i.e., slope < 1) with body mass, with different slopes at different taxonomic levels (Isler et al., 2008; Pagel and Harvey, 1989). In primates (but not in other mammalian orders: rodents: Herculano-Houzel et al.. 2006: insectivores: Sarko et al., 2009), brain mass is therefore equivalent to neuron numbers due to the 1 to 1 relationship between them and the lack of relationship of mass and volume with cell density.

### Brain size versus control mechanisms

The main functional prediction that corresponds to these structural scaling rules is that cognitive benefits, accruing in certain ecological conditions and traded off against some costs, have coevolved with encephalization at one or more of the neuroanatomical scales mentioned above. One problem with this prediction is that two levels of explanation, as well as two traditions of empirical testing, are used. Knowing which brain area's size is best correlated with a measure of cognition and knowing what brain processes control this cognitive ability are distinct questions. Traditionally, the questions have been addressed with different techniques and, while their answers are mutually relevant, they involve different levels of explanation. To answer the mechanistic control question, one has to identify the key neuronal events that lead to performance differences in cognitive tasks. The events, which could be localized or distributed over many parts of the brain, involve neurotransmitters, receptors, enzymes that affect

neurotransmitter metabolization, as well as blood oxygenation changes that follow neuronal activity. The mapping of blood oxygenation changes that is achieved with magnetic resonance imaging goes some way toward linking neuronal events with the identification of brain areas involved in particular cognitive activities, but they do not answer the question of size differences between brain areas across different species. For example, a consensus seems to be developing among neuroscientists (Deary et al., 2010) that tasks with strong loadings on general intelligence involve a distributed network of at least 14 brain areas in humans (review by Jung and Hier, 2007 of 37 imaging studies involving 1557 subjects). Whether the activation of the 14 areas during general intelligence tasks translates into more neurons, and thus, a greater volume in each of the 14 brain parts is another matter. How the 14 brain parts would evolve to different sizes in different species due to the selective advantages of more versus less general intelligence is also unknown. Would it be through concerted evolution (Finlay et al., 2001) of the entire zone encompassing the 14 areas? Would it be via mosaic evolution (Barton and Harvey, 2000) of the 14-area network only? Would it be through 14 separate evolutionary events each affecting a different area? The distinction between size evolution and proximal control, along with the unresolved question of how the two levels are linked, needs to be taken into account in all our thinking about brain-intelligence coevolution.

Theories of encephalization originate from the simple observation that the brain as a whole, as well as areas that take up a large proportion of the brain such as the avian pallium and the mammalian neocortex, is many times larger in some species than in others. These empirical observations warrant scientific study, in the same way that research on body size has long been a legitimate field in ecology and evolution (the search topic "body size evolution" currently yields 7275 articles on the Web of Science). The fact that bodies are made up of many parts that

are selected for different purposes has not led critics to doubt the validity of the whole field, but this has sometimes happened for brain size. In particular, recent work on spatial memory and bird song has led to the belief in some circles that the idea of encephalization is meaningless because it refers to too broad an anatomical structure (Healy and Rowe, 2007). However, in the case of both spatial memory and bird song, strong mechanistic research programs had long identified key neural centers involved in the storage of specialized information about space and learned vocalizations. When Sherry and Vaccarino (1989) lesioned the hippocampus of chickadees and found that spatial memory of previously stored food was lost, their focus on that brain structure was based on decades of lesion (Morris et al., 1982) and electrophysiological research (e.g., place cells. O'Keefe and Dostrovsky, 1971). Similar work by Nottebohm et al. (1976) had also identified nuclei like HVC. RA (robust nucleus of the arcopallium), and area X as specialized centers for oscine song, as well as demonstrated neurogenesis coincident with song learning (Paton and Nottebohm, 1984). Sherry's serendipitous discovery (Sherry, 2011) that the chickadee hippocampus was much larger than that of the nonstoring canary whose brain atlas had been used to guide the lesion surgery of Sherry and Vaccarino (1989) was posterior to the strong research tradition identifying this brain structure as a key center for spatial memory. In a similar manner, the discovery by DeVoogd et al. (1993) that the size of nucleus HVC (but not area X) was proportional to the size of the song repertoire of oscine species came several years after Nottebohm's pioneering mechanistic work.

The study of encephalization does not have the same history or luck as that of spatial memory and bird song. The question "why is a corvid brain so large?" is much older than the data showing that crows can manufacture tools (Hunt, 1996) and magpies can recognize themselves in a mirror (Prior et al., 2008). The brain centers and neural events that are necessary for mirror image

recognition and tool manufacture in birds are also currently unknown. We are therefore obliged to use a top-down approach rather than the bottom-up program that spatial memory and bird song researchers were fortunate enough to have at their disposal. We start with the old observation of brain and body size covariation by Dubois and Lapicque (Gayon, 2000, for a historical perspective), and then seek cognitive and lifestyle correlates of this variation. If (1) these correlates are independent of each other, (2) each one is more strongly tied to size variation in one brain area than in others, and (3) structural studies of brain organization demonstrate strict mosaic evolution of areas on the basis of functional specialization, then the concept of encephalization loses much of its relevance because it is not specific enough. If instead the cognitive and lifestyle measures covary and size variation in brain areas is concerted with variation in the whole brain through common evo-devo processes, then the idea of encephalization is more useful.

Bearing these caveats in mind, what is the current state of the literature on species differences in structural and functional correlates of encephalization in primates? In this chapter, I deal only with large-scale (either all primates or all haplorrhines) comparative analyses that examine phylogenetically controlled correlates of encephalization. I do not include analyses that focus only on a single clade such as apes or New World or Old World monkeys; I also do not include analyses that add Homo sapiens to the primate database. Tables 1-4 summarize the evidence taken from 26 studies that focus on the whole brain or its major "higher" divisions, the telencephalon and the neocortex. Other brain areas that are usually not considered in encephalization research have also been subject to comparative analyses featuring correlations between cognitive functions and structure size (the cerebellum: Lewis and Barton, 2004; Dunbar and Shultz, 2007b; the amygdala: Lewis and Barton, 2006; the hippocampus: Lewis and Barton, 2006; Shultz and Dunbar, 2010a; the main

Encephalization measure	Lifestyle correlate						
	DQ	HR	HU	GS	VI		
Brain vol or mass				X●			
Res brain vol ag. body	••x	••	•	•XX	•••		
EQ							
Neonatal brain vol							
Tel vol				х			
Res tel vol ag. body				x			
Res tel vol ag. brain				х			
Res tel ag. ROB				•			
Neo vol				x			
Neo+striatum vol							
NV neo vol							
Res neo vol ag. body				х			
Res neo vol ag. brain	х	•	х	••x			
Res neo vol ag. ROB	•х	•	•	•••	••		
Res neo ag. medulla	•*	•	•	•			
Res NV neo ag. body		•		x			
Res NV neo ag. ROB		х		••			
Res NV neo ag. ROB ag.	•			•			
body							
Neo/brain				•			
Neo/ROB	х	х		•••			
Neo/ROB, ag. brain							
Neo+stri/bstem	х	х		•			
Neo+stri/bstem, ag. body							
Neo/ROB-cereb	х	х		х			
NV neo/ROB	х	xx		••			
NV neo/ROB-cereb	х	х		•			

Table 1. Lifestyle correlates of encephalization

# Encephalization abbreviations: vol, volume; res, residual or result of partial correlation; EQ, encephalization quotient; neo, neocortex; NV, nonvisual; ag. body, regressed against body mass; tel, telencephalon; ROB, rest of brain; stri, striatum; cereb, cerebellum; bstem, brainstem. DQ, diet quality: Barton (1996) and Fish and Lockwood (2003). HR, home range: Deaner et al. (2000) and Walker et al. (2006). HU, habitat use: Dunbar and Shultz (2007b).

GS, group size: Barton (1996), Deaner et al. (2000), Dunbar (1998), Joffe and Dunbar (1997), Lehmann and Dunbar (2009), Lindenfors (2005), Lindenfors et al. (2007), and Walker et al. (2006). VI, visual input: Barton (1998, 2004) and Kirk (2006).

•: Result, p < 0.05; •\*: result, p = 0.06; x: result, ns.

and accessory olfactory bulbs: Barton, 2006b; the striatum: Graham, 2011; the primary visual cortex: Barton, 1996), but they are not covered in the tables.

The 26 studies all yield linear associations between some structural measure of encephalization

Encephalization measure	Cogr	Cognitive correlate					
	TD	IN	TU	SL	G	TR	
Brain vol or mass						••	
Res brain vol ag. body						x	
EQ						x	
Neonatal brain vol Tel vol Res tel vol ag. body Res tel vol ag. brain Res tel ag. ROB							
Neo vol	•				•	••	
Neo+striatum vol NV neo vol		•	•	•		•	
Res neo vol ag. body Res neo vol ag. brain						х	
Res neo vol ag. ROB Res neo ag. medulla Res NV neo ag. body Res NV neo ag. ROB Res NV neo ag. ROB ag. body Nao(brain					x	х	
Neo/ROB	•				•	••*	
Neo/ROB, ag. brain							
Neo+stri/bstem Neo+stri/bstem, ag. body Neo/ROB-cereb NV neo/ROB		•	• •*	•	•		
NV neo/ROB-cereb							

Table 2. Cognitive correlates of encephalization

Encephalization abbreviations as in Table 1.

TD, tactical deception: Byrne and Corp (2004).

IN, TU, SL, innovation, tool use, social learning: Reader and Laland (2002).

G, general intelligence: Reader et al. (2011).

TR, test rankings: Deaner et al. (2007) and Shultz and Dunbar (2010a).

•: Result, *p* < 0.05; •\*: result, *p* = 0.06; x: result, ns.

(over two dozen different measures) and a cognitive variable, a trade-off, or a lifestyle. The studies suggest that more encephalized primate species or genera tend to (1) eat a higher quality diet, (2) have larger home ranges, (3) are arboreal and live in closed forests, (4) occur in larger groups, and

#### Table 3. Trade-offs of encephalization

#### Table 4. Evolutionary correlates of encephalization

	Trade-off		
Encephalization measure	JD	MR	SS
Brain vol or mass			
Res brain vol ag. body	••	••	•
EQ			
Neonatal brain vol			
Tel vol			
Res tel vol ag. body			
Res tel vol ag. brain			
Res tel ag. ROB			•
Neo vol			
Neo+striatum vol			
NV neo vol			
Res neo vol ag. body			
Res neo vol ag. brain	х	•	х
Res neo vol ag. ROB	•	•	х
Res neo ag. medulla	•	•*	
Res NV neo ag. body			
Res NV neo ag. ROB			
Res NV neo ag. ROB ag. body			
Neo/brain			
Neo/ROB			•
Neo/ROB, ag. brain			•
Neo+stri/bstem			
Neo+stri/bstem, ag. body			
Neo/ ROB-cereb			
NV neo/ROB	•		
NV neo/ROB-cereb	•		

Encephalization abbreviations as in Table 1.

JD, juvenile development: Barrickman et al. (2008) and Walker et al. (2006).

SD, sexual dimorphism: Lindenfors et al. (2007) and Schillaci (2006, 2008).

MR, metabolic rate: Isler and van Schaik (2006).

•: Result, *p*<0.05; •\*: result, *p*=0.06; x: result, ns.

(5) use more binocular visual input than less encephalized primates (Table 1). They also show (6) more tactical deception, (7) innovation, (8) tool use, (9) social learning, (10) general intelligence, and (11) better performance in captive tests, than do less encephalized primates (Table 2). Encephalization is traded off against (12) a slower juvenile development, (13) a higher metabolic rate, and (14) a greater degree of sexually selected dimorphism (Table 3). More encephalized primates have (15) a longer lifespan. Over

	Evolutionary correlate			
Encephalization measure	LS	MG	ET	
Brain vol or mass		х		
Res brain vol ag. body	••x	•	•	
EQ		х		
Neonatal brain vol		•		
Tel vol				
Res tel vol ag. body				
Res tel vol ag. brain				
Res tel ag. ROB				
Neo vol		х		
Neo+striatum vol				
NV neo vol				
Res neo vol ag. body				
Res neo vol ag. brain		Х		
Res neo vol ag. ROB		•	х	
Res neo ag. medulla		•		
Res NV neo ag. body				
Res NV neo ag. ROB				
Res NV neo ag. ROB ag. body				
Neo/brain				
Neo/ROB		•		
Neo/ROB, ag. brain				
Neo+stri/bstem	•			
Neo+stri/bstem, ag. body				
Neo/ROB-cereb	х			
NV neo/ROB	х			
NV neo/ROB-cereb	Х			

Encephalization abbreviations as in Table 1.

LS, life span: Barrickman et al. (2008); Walker et al. (2006).

MG, microcephaly genes: Ali and Meier (2008); Montgomery et al. (2011).

ET, evolutionary time: Shultz and Dunbar (2010b).

•: Result, *p*<0.05; •\*: result, *p*=0.06; x: result, ns.

evolutionary time, there has been (16) an increase in primate encephalization, along with (17) a high rate of change in some of the genes associated with whole brain and neocortex size (Table 4). In the following sections, I summarize the evidence linking one or more encephalization measure with the 17 functional variables. Many of the comparative studies test several neural measures. The tables include all tests of all neural measures, whether they lead to significant (indicated by a "•") or nonsignificant (indicated by an "x") results. The text in the sections leaves out many of these details, so as not to overburden the reading.

# Lifestyles associated with encephalization (Table 1)

Historically, studies on lifestyles favoring encephalization preceded those in which direct tests of cognitive measures were used. The comparative psychology work of Riddell and Corl (1977) was contemporaneous with the earlier ecological studies of Clutton-Brock and Harvey (1977) but was not as influential (currently 22 citations in the Web of Science, compared to 570). It is the discovery by Clutton-Brock and Harvey (1980) that frugivorous primates have a larger brain than do folivorous ones that gave an impetus to ecological studies of encephalization. Similar work on other orders suggested that dietary reliance on multiple foods (omnivory) that are hard to find (fruit, vertebrate prey) might select for enlarged brains in many mammals.

Many early studies of encephalization did not remove the potential pseudoreplication effects of common ancestry on their comparative data. The advent of phylogenetic corrections improved comparative work and provided a means to distinguish between phylogenetically confounded versus repeated independent coevolution of brain enlargement and cognition. Barton (1996) and Fish and Lockwood (2003) confirmed with phylogenetic controls the dietary trends reported by Clutton-Brock and Harvey (1980). They also used quantitative indices instead of categorical measures of diet: in Barton's case, the measure was percent fruit in the diet, while in Fish and Lockwood's, the diet quality index included fruit, meat, and leaves.

The abundance and the spatial and temporal distribution of fruit are, on average, more difficult to track than that of leaves. This is the cognitive challenge that is assumed to be behind the relatively large brain of frugivorous primates. Food that is patchy and whose ripeness has to be

tracked in space and time might also have to be searched for over a wide range. It is therefore logical that primates with larger brains should also have larger home ranges. Deaner et al. (2000) have confirmed with phylogenetic corrections and two different measures of relative neocortex size the earlier finding by Clutton-Brock and Harvey (1980) that home range is positively associated with residual brain size. Two other habitat use variables have been examined by Dunbar and Shultz (2007b), occurrence in open/ mixed versus closed forest habitats, as well as terrestrial versus arboreal locomotion between feeding and resting sites. In univariate analyses, arboreal and closed forest species showed a large residual brain and neocortex size.

Another key lifestyle variable hypothesized to be associated with encephalization is group living, which was first tested by Sawaguchi and Kudo (1990). The assumption here is that a larger brain or neocortex can process a larger amount of social information resulting from the alliances, networks, and dominance relationships that increase, presumably in a nonlinear manner, with the number of individuals in a primate group. Dunbar and others have confirmed with phylogenetic corrections that neocortex size is associated with several features of sociality: social group size (Barton, 1996; Dunbar, 1992), number of females in the group (Lindenfors, 2005), grooming clique size (Kudo and Dunbar, 2001), frequency of coalitions (Dunbar and Shultz, 2007a), and network connectivity (Lehmann and Dunbar, 2009). Lindenfors et al. (2007) suggest that female primates, but not males, show the relationship between neocortex size and sociality. Instead, sexual selection for large size in males is more strongly associated with the size of limbic structures involved in aggression (see the section "Trade-offs").

Barton's recent work has focused on correlates of specialized brain parts, in accordance with his views on mosaic evolution of functionally linked areas (Barton, 1999, 2006a, 2007; Barton and Harvey, 2000; Whiting and Barton, 2003).

Barton and colleagues (Lewis and Barton, 2004, 2006) have found that the size of the cerebellum, amygdala, and hypothalamus are associated with social play (as is the size of the striatum, Graham, 2011); the size of the main olfactory bulb with diet and diurnal versus nocturnal activity: and the size of the accessory olfactory bulb with social and mating systems (Barton, 2006b). More relevant to the question of encephalization are Barton's (1998) findings that the size of specialized visual areas (primary visual cortex, parvocellular and magnocellular layers of the lateral geniculate nucleus) and the number of neurons in them are positively correlated with allometrically corrected brain size. Barton (2004) also shows that degree of binocular convergence correlates both with the size of specialized visual areas in the brain, as well as relative size of the neocortex and whole brain. adding further support to the idea that vision was a major factor in primate encephalization.

Attempts to test lifestyle variables together, to see if one is a better predictor than the other or if they covary in ways that suggest they are not independent, have produced mixed results. Joffe and Dunbar (1997) have shown that visual areas of the cortex (striate cortex and lateral geniculate nucleus) have a poorer relationship with social group size than do nonvisual areas, suggesting that vision per se, contrary to Barton's ideas, did not play a major role in the encephalization of the social brain. Barton (1996) showed that social group size and percent fruit in the diet predict independent portions of the variance in relative neocortex size. Deaner et al. (2000) have found that either social variables such as group size or ecological variables such as home range are the only significant correlate of allometrically corrected brain size depending on the method used for the correction. Walker et al. (2006) report a similar result using stepwise regressions. When residual brain size is the encephalization measure, only home range size is significant, while group size and percent fruit in the diet are not. When the encephalization measures are neocortex ratios (calculated in five different ways),

only group size now enters the multiple regressions, with home range and percent fruit dropping out. Reader et al. (2011) report that lifestyle variables, whether social (group size) or dietary (percent fruit in diet, number of food categories in diet), load together on the second component of a PCA in which the main component regroups five cognitive measures. The correlations between the social and the dietary variables are weakly positive, varying between 0.14 and 0.25 (Reader et al., 2011). Finally, Dunbar and Shultz (2007b) conducted multivariate and path analyses on several lifestyle and life history variables that were significant predictors of residual whole brain and neocortex size in univariate analyses. In the final multivariate model, residual brain size was best predicted by diet and lifespan, while the effects of group size, home range size, and habitat use did not contribute significantly to the model. Residual neocortex size was best predicted by group size and lifespan, with the other lifestyle variables dropping out. In the path analysis, the relationship between group size and residual neocortex was direct and bidirectional, as was the relationship between residual brain size and lifespan. In contrast, the relationship between residual brain size and diet was indirect and included several unidirectional intermediates, with metabolic rate driving both diet and brain size. Intriguingly, body size was only indirectly driving brain size in the analysis, via its unidirectional effects on metabolic rate and lifespan.

# Cognitive correlates of encephalization (Table 2)

Frugivory and sociality seem to have a robust relationship with primate encephalization. However, these are lifestyles in which enhanced cognition might be an advantage, but they are not cognitive variables *per se*. If, for instance, one measured species differences in the number of conspecific faces primates can memorize, this would provide a direct test of the cognitive differences that are constrained by a smaller social brain. In the absence of such experiments, only direct quantitative estimates of cognitive abilities provide the missing link between lifestyles and encephalization.

Riddell and Corl (1977) were the first to show in a large data set that species differences in "cerebral development indices" correlated with performance on a variety of tasks. Deaner and colleagues (Deaner et al., 2006, 2007; Johnson et al., 2002), using phylogenetic corrections, generalized this finding by ranking with a Bayesian procedure the different primate genera on all available comparative tasks studied in captivity. Deaner et al. (2007) then compared these ranks to eight different measures of encephalization. Absolute (log-transformed) size of the whole brain and the neocortex were the only significant correlates of cognitive performance on independent contrasts between primate genera; neocortex ratio showed borderline significance, but other relative measures, whether corrected by body size or size of the rest of the brain, did not, Reader et al. (2011) assessed the relationship between the general intelligence factor they extracted from eight cognitive and lifestyle measures and the experimental data from captive studies described by Riddell and Corl and Deaner and colleagues. They found significant correlations in both cases. This supports the idea that cognitive tasks given in captivity are ecologically valid measures of cognitive differences found in the field, as well as the idea that measures taken both in the field and in captivity are to some extent controlled by general intelligence.

Historically, Byrne and Whiten (1990) were the first to examine taxonomic differences in the frequency of a cognitive ability, tactical deception, in the wild and in captivity. The idea here is that the number of times human observers witness the use of a particular type of cognition in particular species can serve as a quantitative measure of taxonomic variation in that ability. Byrne and Whiten were careful to correct their data for potential biases that might inflate observations in well-studied or more visible species. The anecdotal nature of their data, a method that had been more of less banned from comparative psychology since the days of E.L. Thorndike, was also extensively discussed (see peer commentaries included with Whiten and Byrne, 1988). Byrne and Whiten (1990) focused on cases of "Machiavellian" intelligence involving social manipulation and tactical deception. Byrne and Corp (2004) then showed that deception frequency per species, corrected for research effort, was positively correlated with both absolute and relative size of the neocortex.

The taxonomic count technique was then generalized by Reader and Laland (2002) to cases of innovation, tool use, and social learning. All three of these measures were shown to correlate with neocortex ratio. Recently, Reader et al. (2011) have reexamined the cognitive measures they used in their earlier article. They added a new measure, extractive foraging, as well as Byrne and Whiten's data on tactical deception frequency. These five cognitive measures were then submitted to PCA to see if they all loaded on one general factor or if the social (social learning, tactical deception) measures loaded on a separate factor from the nonsocial ones (tool use, extractive foraging). All five measures loaded strongly on a first component that explained 65% of the variance; this result is compatible with the idea that there is a general intelligence factor (g) behind the measures. More interestingly, the five cognitive measures all loaded together on the same factor even when three lifestyles variables (group size, percent fruit in diet, and number of food categories in diet) were added to the factor analysis. The lifestyle variables all loaded on a second, independent factor. What these data suggest is not only that general intelligence might be an important part of primate cognition but also that the distinction mentioned above between lifestyle correlates of brain size and cognitive measures per se might be more important than the off-cited difference between social and nonsocial intelligence

(see also Overington et al., 2008). Finally, the positive correlations between Reader et al.'s g and the results of the captive tests analyzed by Riddell and Corl (1977) and Deaner et al. (2006) argue against a common bias to taxonomic counts based on quantified anecdotes. Such a bias might lead to positive intercorrelations between taxonomic counts, but should not also produce correlations with the results of laboratory tests. These might instead be biased by species' responses to captivity and testing by humans, but the positive relationship with field counts argues against this possibility.

# Trade-offs (Table 3)

A solitary folivore is predicted to profit less from an enlarged neocortex than an omnivore living in a large group. Lifestyles, however, are not the only contexts that affect the evolution of complex cognition and large brains. Life history is also thought to be important, in particular the slow-fast continuum of developmental traits. Based on this continuum, more encephalized primates should have small litters, as well as long periods of gestation, lactation, and juvenile growth. These are costs, as they imply increased parental investment and a delay in reproductive maturity. However, the lengthened lifespan that also goes with the slow-fast continuum can compensate for the sexual maturation delay by increasing the duration of the reproductive period. Several researchers (e.g., Allman et al., 1993; Deaner et al., 2003; Hofman, 1983, 1993; Sacher and Staffeldt, 1974) have examined one or more of these life history traits, often with varying results (see Table 1 in Barrickman et al., 2008 for a review). Based on a large data set taken mostly from field studies, the analysis of Barrickman et al. (2008) supports most of the preslow-fast dictions from the view of encephalization. Taking into account body size and phylogeny, primate brain size is positively associated with length of the juvenile period and age at first reproduction. Gestation length is also associated with brain size, but time to weaning is not. The advantage that counterbalances these developmental costs is a lengthened life span (Table 4; Dunbar and Shultz, 2007b; Barrickman et al., 2008).

Many papers on primate (especially human) encephalization mention the metabolic costs that a large brain represents. These costs can be met in large-brained species either by increasing metabolic rate or by reducing the energetic costs associated with other organs, for instance digestive ones (Aiello and Wheeler, 1995). Both can be achieved via an improvement in diet quality, which increases caloric intake as well as digestibility. Isler and collaborators (Isler and van Schaik, 2006; Isler et al., 2008), using two different samples of primate data, have confirmed the predicted relationship between basal metabolic rate and brain size, with phylogeny and body size controlled for, as have Dunbar and Shultz (2007b).

As humans, we might think that an increase in cognitive efficiency is always a good thing, but in some primate species, variance in reproductive success might be more strongly affected by noncognitive factors, to the point of actually selecting against encephalization. The finding of Lindenfors et al. (2007) that limbic areas involved in aggression were associated with group size and dimorphism in male primates, but not in females, hints at such an effect. If a male, emigrating from its natal troop, competes with other males via intense individual aggression, traits such as body size, canine length, and fighting ability might be more important than cognitive abilities that would allow alliance management, tactical deception, large grooming networks, and kin recognition in circumstances where individual aggression is less important. From an "expensive tissue" perspective, this might also create trade-offs between structural investment in brain versus canine and muscle tissue. One operational measure of the intensity of male competition is sexual dimorphism, which can be quite large in some species, such as mandrills. As predicted, Schillaci (2006) reports a significant negative Author's personal copy

relationship between brain size and degree of dimorphism (Schillaci, 2006), as well as a relationship between brain size and mating system, with monogamous primates having larger brains than polygynous ones (Schillaci, 2008). Coherent with this view is the fact that the correlation between male rank and mating success in polygamous primates is negatively associated with neocortex size: the stronger the reproductive skew in favor of high ranking males, the smaller the neocortex ratio (Pawlowski et al., 1998).

A second possible trade-off involving sexual selection is suggested by Pitnick et al.'s (2006) finding that in bats, sperm competition has led to positive selection on testis size, with a structural trade-off on other "expensive tissues" negatively affecting brain size (see, however, Dechmann and Safi, 2009). Two studies (Lemaître et al., 2009; Schillaci, 2006) have now shown that, although the data confirm Pitnick et al.'s idea in echolocating bats, the prediction is not supported in primates. This negative result is coherent with the data on mating systems: sperm competition is low when only one male copulates with a female, whether the mating system is strict monogamy or strict polygyny. It is when females copulate with several males that sperm competition is highest, a system that is associated with intermediate-sized brains in Schillaci's (2008) analysis. The prediction on sperm competition thus leads the larger brains of the monogamous primates to cancel out the effect of the smaller brains of the polygynous ones in the statistical comparison with the intermediate-sized brains of the multimale/multifemale species.

# Are some encephalization measures better than others?

Authors of individual studies often argue that the encephalization measure they are using is the most appropriate one; they sometimes strengthen their argument by showing that alternative size measures show either a poorer or a nonsignificant correlation with the cognitive variable they are testing. Though this approach is defendable, I will concentrate here on the trends in the entire set of tables rather than on single cases. Comparisons of encephalization indices are tricky, especially if different measures are derived from different techniques. For example, correcting whole brain size by body size and neocortex size by medulla size might make the second index look better simply because its correction factor has less measurement error, individual variability, and noncognitive selection pressures favoring a larger or a smaller body. Figure 1 illustrates this point using data on 43 extant nonhuman primate species taken from Stephan et al. (1981).

Regressing log neocortex size against log size of the medulla yields almost the same trends as regressing log whole brain size (minus the medulla) against log medulla (Fig.1a), leading to very similar residuals (Fig. 1b). However, regressing log size of the brain (minus the medulla) against log body size leads to results that show much more variation (Fig. 1c). Phylogenetic corrections might change these results slightly, but not alter the overall, qualitative conclusions. If a neocortex index based on a brainstem control is a much better predictor of a given cognitive measure than is brain size regressed against body mass, this should thus not automatically be taken to mean that extra-cortical areas are not involved in a particular cognitive process. More work is clearly needed on phylogenetically corrected data to compare the different encephalization measures.

The first obvious trend in Tables 1–4 is the very large number of encephalization measures. For corrected neocortex size only, there are 14. Half of these corrections are done with residuals from regressions, while the other half are done with ratios. Some of the neocortex measures use the entire structure, while others subtract the primary visual areas from the rest of the neocortex, and others still add the striatum. It is the structure used as the independent variable in the regressions and the denominator in the ratios that varies the most: it goes from body mass to volume



Res (br - medulla) against medulla

Fig 1. (a) Ln volume of the neocortex (open circles) and ln volume of the brain minus the medulla (filled circles) plotted

of the whole brain, to that of the brain minus the neocortex or the brain minus the neocortex and the cerebellum, to the brainstem (mesencephalon plus medulla oblongata) to the medulla alone. However, before one concludes that some encephalization measures are better correlates of a particular functional variable than others, we need to know more about the indices themselves. Figure 2 illustrates this point: different ways of calculating relative size of the neocortex yield very different results. It matters little whether size of the neocortex is regressed against that of the whole brain or against that of the rest of the brain (see close relationship in Fig. 2a). However, other indices give discordant results: residual neocortex size regressed against the rest of the brain has a weak positive relationship with neocortex ratio over size of the whole brain (Fig. 2b), but a very poor relationship with neocortex ratio over size of the medulla (Fig. 2c). In turn, the relationship between the two neocortex ratios is strong, but nonlinear (Fig. 2d). What is clearly needed in the future is a comparative study that examines the similarities and differences between the different measures of encephalization, before they are used to test any functional predictions. Failing this, structural differences between encephalization measures may confound any apparent difference in the correlates of different functional variables.

Over all entries in the tables, measures of telencephalon size, whether absolute or corrected, are by far the least popular (six results) and least successful (more nonsignificant results, four, than significant ones, two). Residual brain size is much more successful: of the 24 results that use it, 18 show a significant relationship and only 6 a

against ln volume of the medulla. (b) Residual of neocortex volume regressed against medulla volume plotted against residual of brain minus medulla volume regressed against medulla volume. (c) Residual of brain minus medulla volume regressed against medulla volume plotted against residual of brain minus medulla volume regressed against body volume. Abbreviations as in Table 1.



Fig. 2. (a) Residual neocortex volume regressed against volume of the rest of the brain plotted against residual neocortex volume regressed against whole brain volume. (b) Residual neocortex volume regressed against volume of the rest of the brain plotted against neocortex ratio over the whole brain. (c) Residual neocortex volume regressed against volume of the rest of the brain plotted against neocortex ratio over the medulla. (d) Neocortex ratio over the whole brain plotted against neocortex ratio over the medulla. Abbreviations as in Table 1.

nonsignificant one. Neocortex size is the most popular: it appears in 91 results, 59 of which are significant. An obvious caveat on these numbers is that some very productive research groups have, over the years, contributed multiple papers with significant relationships between a particular functional predictor and a particular structure, inflating the trends in the tables.

Over the 17 functional predictors of encephalization in Tables 1–4 (column headings), residual brain size shows at least one significant association with 11 of them. Residual neocortex size regressed against the rest of the brain is significantly associated with eight functional predictors, while neocortex ratio is associated with six. Absolute neocortex volume, in one form or the other (alone or with the volume of the striatum added or the volume of the primary visual cortex removed), is significantly associated with all six cognitive variables in Table 2, but with none of the other predictors in Tables 1, 3, and 4.

The trends in Table 1–4 suggest that both the whole brain and the neocortex, but not the telencephalon, are relevant neuroanatomical levels to test predictors of encephalization. They also suggest that both corrected and absolute neocortex volumes are of interest. Are these results coherent with genetic and evo-devo approaches to encephalization? Several genes that, in their abnormal form cause human microcephaly, have recently been studied in hominid lineages (Evans et al., 2004; Kouprina et al., 2004) and in

comparative analyses on wider primate data sets (Ali and Meier, 2008; Montgomery et al., 2011; see Chapter 2 of this volume). Alternative evo-devo processes have also been invoked to account for either concerted evolution of all brain parts via differences in embryological neurogenesis ("late is large," Finlay et al., 2001) or mosaic coevolution of adaptively specialized sets of brain areas (Barton and Harvey, 2000; Whiting and Barton, 2003). Does the current consensus on microcephaly genes and concerted versus mosaic evolution allow us to decide between the whole brain and the neocortex as the most appropriate neuroanatomical level? Does it tell us whether absolute size of the neocortex is more relevant than corrected size?

The problem is that there is no consensus on these points, at least for the moment. Proponents of concerted and mosaic evolution focus on different parts of the variance in brain component size and use different methods to test their predictions. Of the four main papers on brain part evolution published in Science or Nature, Finlay and Darlington (1995) compare log-transformed absolute volumes of brain parts and find concerted evolution. Barton and Harvey (2000) partial out the size of the rest of the brain for each brain part and find mosaic evolution. Clark et al. (2001) transform each brain part into fractions of the total brain and find scalable taxon-specific cerebrotypes. de Winter and Oxnard (2001) use multivariate analysis on ratios of each brain part divided by the volume of the medulla, then again by the volume of the neocortex and find clusters of unrelated taxa that share similar niches. One or more of these conclusions might well be correct, but the differences in data transformations used in the studies might also constrain the realm of possible results that can be obtained.

An example of the different effects of data transformations is given in Figure 3.

The column on the left features the cerebellum, and the column on the right, the hippocampus, all plotted against the Finlay and Darlington (1995) measure of ln absolute brain part volume. The

first line (a and b) features the transformation used by Barton and Harvey (2000), the second line (c and d) the transformation used by Clark et al. (2001), and the third (e and f) and fourth (g and h) lines the transformations used by de Winter and Oxnard (2001). What the figure clearly shows is that the transformations treat the brain part data in very different ways. In half of the eight cases (a, b, c, and f), pairs of transformations are uncorrelated over the different primate species. In one case (e), they show a tight nonlinear positive relationship, while in the other three (d, g, and h), they show a loose negative linear one. From the top and bottom lines of the figure, one could conclude that the cerebellum and hippocampus show similar trends, while from the middle two lines, that they show divergent trends.

What is needed in this debate is more comparative work on embryological neurogenesis in different brain areas of different primate species, similar to what is being done in birds by Striedter and Charvet, (2008, 2009; Charvet and Striedter, 2009). If developmental schedules are concerted and conserved, this should be detectable in the growth trends of different brain areas in different species. If instead brain parts develop as taxonspecific, functionally related mosaic pieces, this should also be obvious in embryonic growth. If strict mosaic evolution prevails, then there is no reason to expect that whole brain size should be relevant functional predictions to about encephalization. In contrast, concerted evolution would imply that both whole brain and brain part size would correlate with cognitive measures.

As far as microcephaly genes are concerned, the three most relevant studies also show contradictory results (Table 4). Evans et al. (2004, 2006) had suggested that the microcephaly genes ASPM and MCPH1 have evolved at a faster rate in lineages leading from the last common ancestor of apes to modern humans than in other lineages, but the data from Montgomery et al. (2011) do not support this idea. Instead, they find adaptive variation across all primate lineages. Ali and Meier (2008) had also linked adaptive evolution

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Fig. 3. Left column: ln volume of the cerebellum plotted (a) against ln volume of the cerebellum with volume of the rest of the brain partialled out; (c) against volume of the cerebellum divided by volume of the whole brain; (e) against volume of the cerebellum divided by volume of the neocortex. Right column: ln volume of the hippocampus plotted (b) against ln volume of the hippocampus with volume of the rest of the brain partialled out; (d) against volume of the hippocampus divided by volume of the hippocampus divided by volume of the hippocampus divided by volume of the neocortex. Abbreviations as in Table 1.

of ASPM to changes in relative neocortex size, but Montgomery et al. (2011) again find little support for this in a wider sample of primates. The strongest relationship in Montgomery et al. (2011) is that between the rate of coding change the microcephalv genes ASPM and in CDK5RAP2 and the absolute size of neonatal primate brains. As Montgomery et al. conclude, this is the relationship that one would predict on causal bases, as microcephaly genes are involved in embryological neurogenesis.

If genes associated with encephalization seem to be selected in primates and if rates of genetic change correlate with brain size differences, is there evidence for brain size changes over macroevolutionary time? Shultz and Dunbar (2010b) have looked at this question in several mammalian lineages. They show that primates have the highest encephalization slope over time of the six mammalian orders they tested (Table 4). At the suborder level, anthropoids have the highest slope, with Strepsirrhines also showing a significant positive slope. Other mammalian orders, for instance, Insectivora, do not show this directional trend toward enlarged brains over evolutionary time.

# Conclusion

Primates are by far the best-studied clade in terms of brains and cognition, in part because of the interest in humans shown by neuropsychologists and paleoanthropologists. Robust evidence is now available for several lifestyle, cognitive, and life history correlates of brain and neocortex size. Promising new avenues of research are opening in molecular genetics, with the possibility that some brain regions might be differentially imprinted by the paternal or maternal genome (Keverne et al., 1996; Wilkinson et al., 2007). The main controversies in the field seem to be over the encephalization measures that should be used and whether encephalization results from concerted mosaic evolution. More or

embryological work is needed to resolve these issues, beyond the current correlational approaches.

With the current evidence, it is difficult to reject the concept of encephalization as vague and misleading because it deals with the whole brain and neocortex instead of specialized brain areas. First and foremost, the size of the brain and neocortex, whether relative or absolute, varies enormously between clades: a marmoset brain contains 63 million neurons (Herculano-Houzel et al., 2007), while a human brain contains 86 billion (Azevedo et al., 2009). In itself, this variation is worthy of study. Brain evolution is clearly not happening only between specialized areas trading off in volume within unvarying cranial constraints but also in overall brain size and neuron numbers. Second, evidence is mounting in primates (Deaner et al., 2006; Reader and Laland, 2002; Reader et al., 2011), but also in birds (Lefebvre et al., 2004), that the many positive correlations observed between cognitive measures across taxa may in part be subsumed by general intelligence. Third, neuroscientists are increasingly interested in distributed networks of multiple, functionally related brain areas involved in several processes, in contrast to the strict modular view that was dominant a few years ago (Bressler and Menon, 2010). Finally, the trends in Tables 1-4 suggest that both corrected whole brain size and corrected, as well as absolute, neocortex size are robust correlates of several functional variables. In the current state of affairs, the "something about brain enlargement" that was alluded to in the first paragraph of this chapter cannot be pinpointed to only one neuroanatomical level.

The question of absolute versus corrected measures of brain and neocortex size is also difficult to resolve. In Tables 1, 3, and 4, corrected measures seem to be the most successful predictors of functional variables, but good neuroanatomical (Herculano-Houzel et al., 2007) and genetic (Montgomery et al., 2011) arguments have been made in favor of absolute measures, which seem to be better predictors of the cognitive correlates in Table 2. Primates differ from most other clades in that the largest-bodied species, the great apes, are also the largest-brained ones, as well as the ones that show the most complex cognition. In contrast, in groups like birds, cetaceans, and dinosaurs, large brains are found in very large-bodied taxa that show little of the lifestyle or cognitive correlates of encephalization, for example, ostriches, baleen whales, and sauropods. It is only when body-size corrections are made that smaller taxa like corvids, dolphins, and theropods surpass their very large-bodied cousins in relative brain size and reveal the predicted associations with lifestyle and cognition. Whether primates are an exception to a general trend (see Burness et al., 2001; Smith et al., 2010 for the relationship between diet and large size) needs to be determined, taking into account the possibility that the primate equivalent of a baleen whale or a sauropod – a very large-bodied species whose diet would not favor complex cognition-might have recently gone extinct. One candidate here might be Gigantopithecus, a 550kg ape that disappeared 300,000 years ago and whose diet, estimated from dental remains, might have been dominated by bamboo and other highfiber vegetable foods (Kupczik and Dean, 2008; Wang, 2009).

Another key point for the future is the reconciliation of the macro-anatomical perspective and comparative approach used by researchers interested in encephalization and the much finer techniques used in proximal studies of cognitive processes, which work at the level of single cells, neurotransmitters, receptors, and genes. Bridging this gap in methods and perspectives is crucial. One example of a combined approach is the comparative study of the neuropeptides involved in social and reproductive behavior. The peptides are relatively conserved over several classes, taking slightly different chemical forms in birds, mammals, and fish (Donaldson and Young, 2008; Goodson and Thompson, 2010). Techniques are available to map their receptor distribution in different parts of the brain, as well as identify and manipulate the biochemical (Goodson et al., 2009b) and genomic (Ferguson et al., 2000; Young et al., 1999) differences that cause behavioral differences. Finally, good comparative work taking into account common ancestry and independent evolutionary events has been done on several species that vary in their social behavior (Goodson et al., 2006); similar comparative analyses have also been done on midbrain dopamine neuron numbers (Goodson et al., 2009a). This integration of approaches, which is also used in contemporary studies of bird song, brings together molecular genetics, neuroscience, ecology, behavior, and evolution. With the added insights of embryology, it is an example of possible directions in which research on encephalization might go.

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