BEHAVIORAL AND BRAIN SCIENCES (2006) **29**, 1–36
Printed in the United States of America

Précis of *Principles of Brain Evolution*

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Abstract: Brain evolution is a complex weave of species similarities and differences, bound by diverse rules and principles. This book is a detailed examination of these principles, using data from a wide array of vertebrates but minimizing technical details and terminology. It is written for advanced undergraduates, graduate students, and more senior scientists who already know something about "the brain," but want a deeper understanding of how diverse brains evolved. The book's central theme is that evolutionary changes in absolute brain size tend to correlate with many other aspects of brain structure and function, including the proportional size of individual brain regions, their complexity, and their neuronal connections. To explain these correlations, the book delves into rules of brain development and asks how changes in brain structure impact function and behavior. Two chapters focus specifically on how mammal brains diverged from other brains and how *Homo sapiens* evolved a very large and "special" brain.

Keywords: basal ganglia; cladistics; development; hippocampus; homology; lamination; mammal; neocortex; neuromere; parcellation; primate

The target book, *Principles of Brain Evolution* (Striedter 2005), was written partly as an upper-level textbook that summarizes most of the ideas and much of the data of evolutionary neuroscience, but it is also an attempt to synthesize the various ideas about how brains evolve into a larger framework that is new, at least in aggregate. This précis will cover mainly the latter, more synthetic aspects of the book. In particular, it highlights the book's central argument, which is that brains evolve not altogether randomly, but in accordance with a set of interacting laws or principles.

1. Philosophical framework

The main aim of evolutionary neuroscience is not just to document the history of brain evolution, but also to explain it. Reconstructions of what likely happened when and where are interesting and important but, ultimately, they are not enough. As Poincaré (1902) once pointed out, scientists must focus not on specific facts but on the regularities that tie them together. Only the recognition of those regularities, including so-called laws, enables scientists to predict future events. This much is widely accepted, but the same logic applies also to past events, for historical explanations are really predictions in hindsight: given what we know, could we have predicted what actually came to pass? If the answer is affirmative, then we have explained the past. This nomological–deductive approach to history has been discussed extensively in the philosophical literature (Hempel 1942), but its application in biology presents some noteworthy problems. Chief among them is that biological history is governed not by a single law but by a plethora of different laws.

Once upon a time, biologists tended to think that evolution was guided by a single law of progression that caused simple organisms to become complex, and "lower" species to ascend "the scale," but that view is no longer tenable (Hodos & Campbell 1969). Nor is it sensible to argue that all of biology can be explained solely in terms of Darwin's law of natural selection (Rosenberg 2001), for natural selection has to work with raw materials that are subject to a variety of other principles or laws, including what we generally call developmental and/or physical constraints (Alberch 1982; Gould & Lewontin 1979). Although this view of biology as being governed by a multitude of laws bothers some philosophers of science (Beatty 1997), it is not really troublesome, for most complex systems, including those studied by physicists, tend to be governed by a variety of laws, forces, and factors that interact. Therefore, biologists ought not to whittle down their set of laws, but seek a unitary theory that accommodates and unifies a lot of different laws. Because that unifying theory is incomplete, most evolutionary explanations are just partial explanatory "sketches" rather than full-fledged theories. Still, they are a good first step.

Within that general framework, the target book's most general purpose is to specify some likely rules, principles, or laws of brain evolution – and to indicate how they might interact. Regrettably, many of the mentioned principles are vague and the synthesis is incomplete. Hopefully, those imperfections will prompt readers to seek more supporting evidence or to construct alternative hypotheses. Most important, I hope that further work will clarify the mechanistic bases of the various brain evolution principles. Once we know those mechanisms, we will know not only why the principles exist, but also why there sometimes are "exceptions to the rule." Although this goal remains

 0140-525X/06 $12.50

distant, recent advances in developmental, behavioral, and computational neuroscience make it possible to envision such a mechanistic grounding of brain evolution principles. Hopefully, that dream will stimulate more neuroscientists to engage in evolutionary research. As Hans Spemann (1927), a famous embryologist, once wrote: "We still stand in the presence of riddles, but not without hope of solving them. And riddles with the hope of solution – what more can a man [or woman] of science desire?"

2. History of evolutionary neuroscience

Evolutionary neuroscience has pre-evolutionary roots but flourished only after Darwin's (1859) *Origin of Species* forced increasing numbers of intellectuals to contemplate "man's place in nature" (as the title of T. H. Huxley's book puts it; see Huxley 1863). Remarkably, Darwin understood that speciation by natural selection produces family trees, bushes, or corals (see Fig. 1), rather than linear scales. Unfortunately, many of Darwin's contemporaries continued to see evolution as unilinear and unfailingly progressive, with lower species gradually ascending some sort of phylogenetic scale (Bowler 1988). This scalar view of evolution has now become extinct among practicing evolutionary neuroscientists (see Hodos & Campbell 1969), but it lives on in many other minds. Similarly, many "classic" notions about how vertebrate brains evolved (e.g., by adding neocortex to an ancestral "smell-brain") continue to hold sway among many non-specialists (MacLean 1990), even though they have long been disproved (Northcutt 1981). Hopefully, the target book will help disseminate the major advances (such as neurocladistics) that so many evolutionary neuroscientists have labored for (see Nieuwenhuys et al. 1998).

An intriguing aspect of the history of evolutionary neuroscience is that it has been marked by a protracted tug-of-war between those who emphasize species differences in brain organization and those who dwell on similarities. One major reason for that tension is that the human mind, when confronted with input as complex and multifarious as the data on vertebrate brain organization, generally seeks order (similarities) amidst confusion; once order has been detected, it can admit that species differences exist and then embark on a new round of seeking similarities. If the human mind indeed works this way, then it is only natural that some scientists, at some points in time, emphasize species similarities while others home in on differences. According to the quantum physicist David Bohm (1957), it is precisely this tension between similarities and differences, between order and disorder, that leads to the discovery of scientific laws and principles – which is why the target book deals with both species similarities *and* differences. The latter are emphasized mainly because they have thus far received less attention.

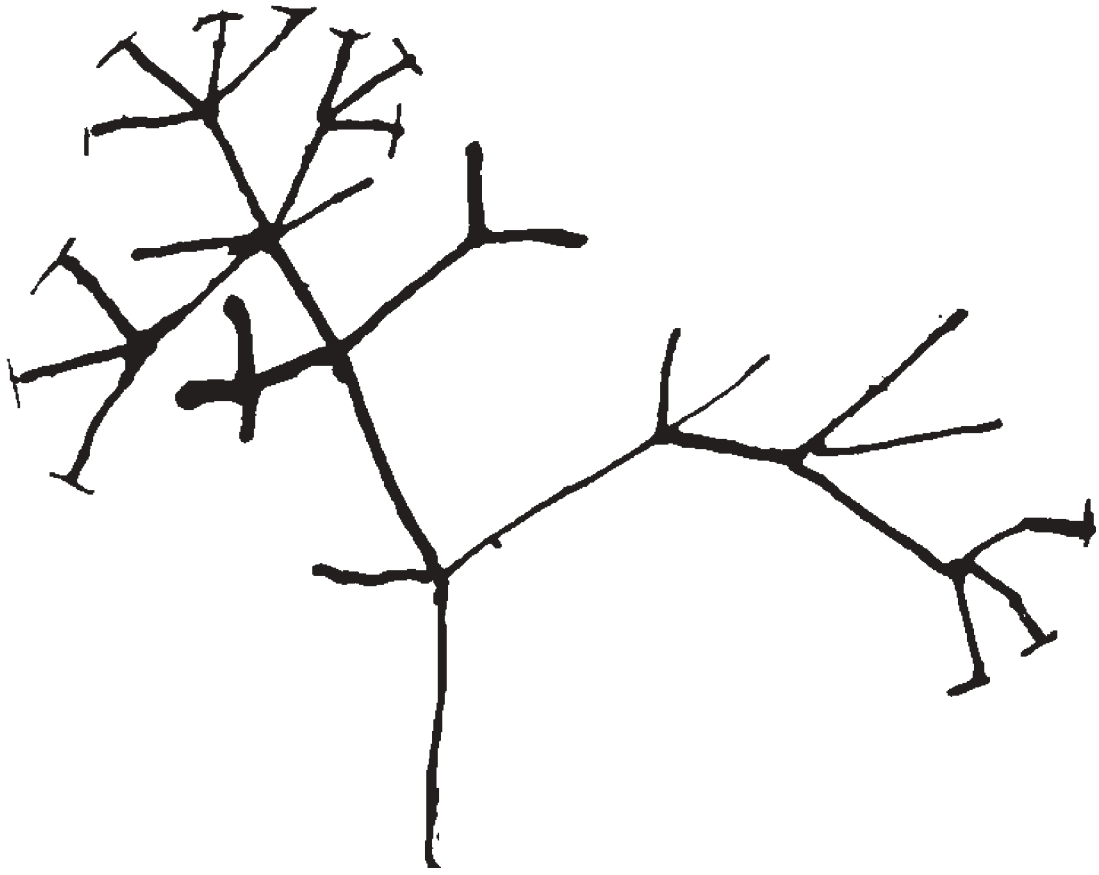

Figure 1. Darwin's "Corals of Life." In one of his early notebooks, Charles Darwin sketched this "coral of life" to illustrate the following thought: "The tree of life should perhaps be called the coral of life, base of branches dead; so that passages cannot be seen – this again offers contradiction to the constant succession of germs in progress" (Notebook B, pp. 25–26, from Barrett 1987).

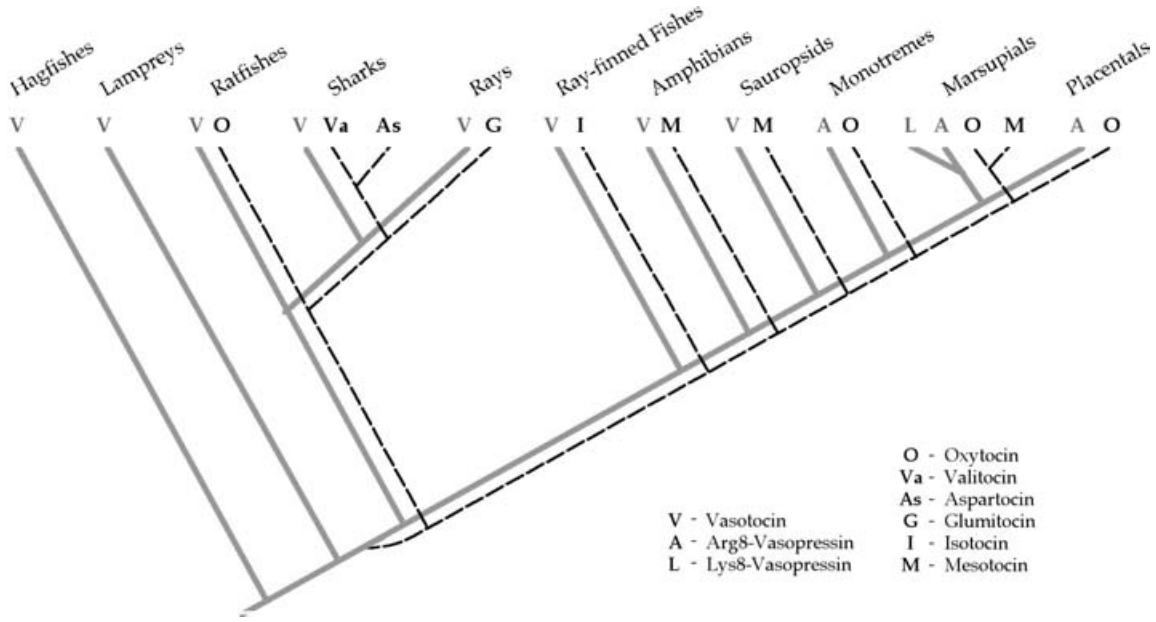

Figure 2. A neuropeptide family. This cladogram depicts a putative phylogeny of peptides in the oxytocin/vasopressin family. The last common ancestor of all vertebrates probably possessed only vasotocin, and extant vertebrates retain this peptide with only minor modifications. Just prior to the origin of jawed vertebrates, the ancestral vasotocin gene probably duplicated and gave rise to a new lineage (dashed lines) that includes a variety of oxytocin-related peptides. Within this more variable lineage, we see both convergent evolution (the evolution of oxytocin in both ratfishes and mammals) and a phylogenetic reversal (the re-evolution of mesotocin in marsupials). Pressed to say which of these peptides is part of the general vertebrate archetype, we can say only that all vertebrates possess at least one member of the oxytocin/vasotocin family. (After Hoyle 1999.)

3. Conservation in vertebrate brains

Adult brains can be studied at several levels of analysis, which form three logically distinct (but interacting) hierarchies of brain structure: regions, cell types, and molecules (Striedter 1999; Striedter & Northcutt 1991). Molecules are generally more conserved than brain regions, but within each hierarchy, conservation generally wanes as one descends levels. For example, individual brain nuclei are less conserved than major brain divisions, and specific neuropeptides are less conserved than the major neuropeptide families (Fig. 2).

Therefore, all adult brain archetypes (common plans of construction) lack detail. In order to obtain more detailed archetypes, we must limit our analysis to smaller taxonomic groups, but even those more limited archetypes remain abstractions, not real brains. Although this is well known, neurobiologists routinely write about "the rodent brain," "the mammalian brain," or even "the vertebrate brain" as if they were talking about a specific brain rather than a highly generalized abstraction. Such linguistic sleight of hand cannot hide species differences for long.

One alternate strategy for obtaining archetypes with more detail is to compare embryonic rather than adult brains, for the embryos of different species generally resemble each other more than the adults do (von Baer 1828). Indeed, molecular studies (Puelles & Rubenstein 1993; 2003) amply confirm that vertebrate embryos go through an early period that is highly, though not perfectly, conserved (Fig. 3).

This high degree of embryonic conservation can help to clarify adult homologies, for experience has shown that homologous adult brain regions tend to be derived from homologous embryonic precursors, even when their

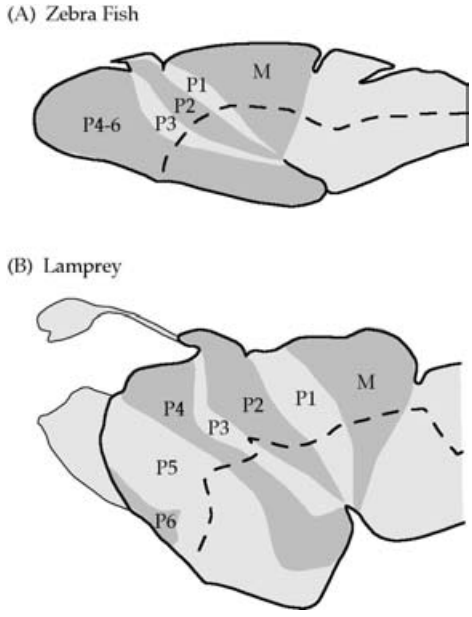

Figure 3. Embryonic brains. At first glance, the brains of embryonic zebra fish and lampreys, here shown from medial perspectives, look very different. However, both are divisible into a distinct series of rostrocaudal rings, or neuromeres, that are highly conserved across the vertebrates. The most rostral neuromeres, called "prosomeres" (P1-P6), give rise to both telencephalon and diencephalon. Behind that lies a single mesomere (M) that gives rise to midbrain tissues. Despite their conservation, neuromeres vary in size and shape, as well as in what adult tissues they produce. (After Pombal & Puelles 1999; Wullimann & Puelles 1999.)

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adult appearance differs dramatically between species (Jiao et al. 2000; Striedter 1997). However, because evolution may occasionally tinker with a structure's embryonic origins, we should not assume that homologous brain regions must *always* develop from homologous precursors, or that the adult derivatives of homologous brain regions must always be homologous to one another (Striedter 1998b; Striedter & Northcutt 1991). Indeed, the more we learn about how brains evolve, the more it seems that evolution can tinker with any neuronal attribute, from embryonic origin to structural complexity and physiological function. A major challenge for the coming years is to reveal the details of that tinkering – to discover how evolution modified neural development to create diverse adult brains, and how those changes altered animal behavior.

4. Evolutionary changes in overall brain size

Relative brain size – that is, brain size relative to what one would expect in an "average" animal of the same type and body size – has increased more often than it has decreased among the vertebrates. Crucially, the increases in relative brain size occurred not in a linear sequence "from fish to man" but independently in a whole slew of different lineages (Northcutt 1984). Although relative brain size is difficult to quantify (Deacon 1990a) and tough to correlate against behavior (van Dongen 1998), it does appear that increases in relative brain size were generally accompanied by increases in social and/or foraging complexity (Byrne & Whiten 1988; Humphrey 1976; Parker & Gibson 1977). In contrast, those lineages that have reduced their relative brain size (e.g., the basking sharks) lead relatively simple lives.

Most evolutionary increases in relative brain size were accompanied by increases in absolute body size. This coincidence is probably related to the fact that proportional brain size generally decreases with increasing body size (von Haller 1762), which means that larger animals tend to have "more room" within their heads for enlarged brains; more formally, I am suggesting that the larger animals were probably more able to expand their brains without changing the shape of either their heads or brains (see Northcutt & Striedter 2002; Striedter & Northcutt, in press). In addition, the coincidence suggests that natural selection for increased social and/or foraging complexity (e.g., among primates or in parrots) brought with it increases in absolute brain size (Byrne 1997). Indeed, absolute brain size correlates with some measures of social complexity better than does relative brain size (Barton & Dunbar 1997). Moreover, within a given taxonomic group, larger species generally seem "smarter" than their smaller relatives, even when relative brain size is held constant (Rensch 1960). All of this suggests that changes in absolute brain size (Fig. 4) may have been far more important than evolutionary neuroscientists traditionally assumed (Jerison 1973).

Perhaps the most fascinating aspect of increasing absolute brain size is that it necessitates changes in the brain's internal connectivity. Specifically, the brain's average connection density (the likelihood that any one neuron connects to any other) must decrease with increasing brain size; otherwise the number of axons would increase

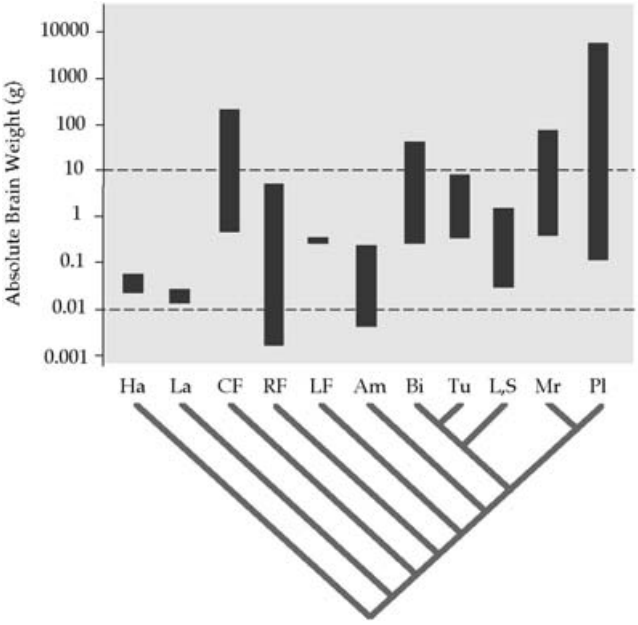

Figure 4. Variation in absolute brain size. Absolute brain size varies across 7 orders of magnitude among the vertebrates. It probably increased independently in several lineages, notably in cartilaginous fishes (CF), birds (Bi), and placental mammals (Pl). Although differences in absolute brain size are rarely discussed in the brain evolution literature, they are likely to be functionally significant (even if they are accompanied by major differences in body size). Other abbreviations: Ha = hagfishes; La = lampreys; RF = ray-finned fishes; LF = lungfishes; Am = amphibians, Tu = turtles; L,S = lizards and snakes; Mr = marsupials.

explosively with neuron number, racking up enormous costs in terms of space and metabolic energy (Deacon 1990b; Ringo 1991). Combined with the general tendency of brains to minimize their axon lengths (Cherniak 1995; Chklovskii et al. 2002; Ramón y Cajal 1909), this decrease in average connection density implies that brains become more modular, both structurally and functionally, as they increase in size (Jacobs & Jordan 1992). Increasing modularity, in turn, allows for increased "division of labor," which generally improves task performance. However, decreasing connection density also reduces a brain's ability to exchange information between distant sites – even if those brains are wired as "small worlds" (Watts & Strogatz 1998). Overall, we can conclude that evolutionary increases in absolute brain size entail both benefits and costs.

5. Evolutionary changes in brain region size

Evolutionary changes in absolute brain size also tend to be associated with fairly predictable changes in brain region proportions (Sacher 1970). Most notably, the proportional size of the neocortex increases steadily with increasing brain size, such that large-brained mammals end up being far more "neocorticalized" than their small-brained relatives (Stephan et al. 1981). The most likely explanation for this scaling rule is that evolution generally enlarges brains by prolonging brain development (letting all precursor cells divide more frequently) but conserves the

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"birth order" of the various brain regions (Finlay & Darlington 1995). This birth order constraint would cause late-born regions, such as the neocortex, to become disproportionately large as absolute brain size goes up (Fig. 5). Although this principle of "late equals large" is logically sound and supported by a considerable amount of evidence, at least within mammals (Clancy et al. 2001; Finlay & Darlington 1995), it remains hotly contested (see commentary in Finlay et al. 2001).

One reason for the controversy surrounding the principle of "late equals large" is that correlations between absolute brain size and proportional brain region size are not always very tight – there are exceptions to the rule! For example, at any given absolute brain size, the neocortex is proportionately larger in simians than in prosimians (Barton & Harvey 2000). Similarly, the superior colliculus is roughly ten times as large in squirrels as in rats, even though both species are similar in absolute brain size (Kaas & Collins 2001). Although such instances of "mosaic evolution" are likely to cause major changes in brain function, they are rare – at least among mammals. Moreover, some of those exceptions to the rule can be explained as the result of real – but rare – changes in the timing of brain region births (Clancy et al. 2001; Finlay et al. 2001). Finally, one should point out that mosaic evolution might occur even if birth order is strictly conserved. The unusually large size of a parrot's telencephalon, for instance, might be due to a parrot-specific increase in the amount of precursor tissue that is initially "specified" to become telencephalon; such an increase would cause adult parrots to have proportionately more telencephalon *even if* birth order is conserved. Unfortunately, we have as yet no published data relevant to that hypothesis.

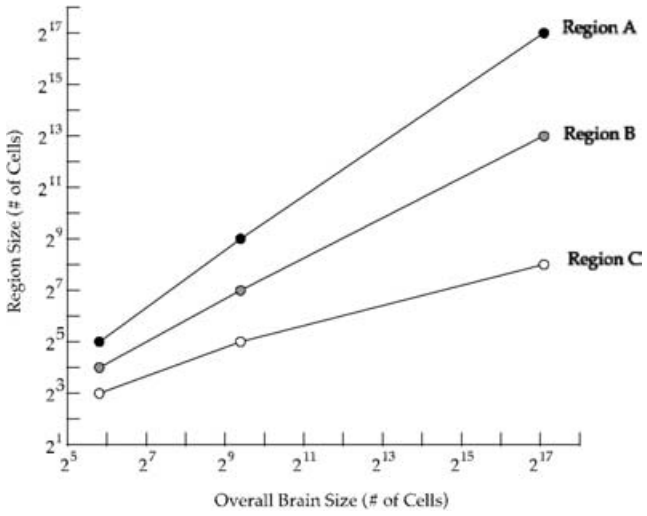

Figure 5. The principle of "late equals large." Imagine 3 neuronal precursor regions (a, b, and c) that each contain 2 precursor cells at the time that they are first formed. Imagine further that the cells in regions a, b, and c undergo 2, 3, and 4 rounds of symmetrical cell division, respectively, before they become post-mitotic and differentiate into the adult regions A, B, and C. According to this scenario, regions A, B, and C are "born" sequentially and, in adulthood, contain 2^3, 2^4, and 2^5 cells, respectively. Now imagine a second scenario – a second species if you will – in which the neurogenetic schedule is doubled in length (conserving birth order!) but the rate of cell division is maintained. In this case, regions a, b, and c would undergo 4, 6, and 8 rounds of cell division, yielding adult regions with 2^5, 2^7, and 2^9 cells, respectively. If we double the neurogenetic schedule again and plot all data in double-logarithmic coordinates, we obtain the illustrated graph. Computing the proportional size of each adult region in each scenario (imaginary species), we see that the later a region was born, the more it increased in proportional size. That, in a nutshell, is Finlay and Darlington's (1995) principle of "late equals large."

Another frequently debated issue is whether changes in proportional brain region size that follow the rule of late equals large can be "selected for." If increasing absolute brain size "automatically" changes the proportional size of the individual brain regions, can any of those individual changes be adaptive? As far as I can tell, the answer must be yes – as long as the benefits of changing any one region outweigh the net cost of changing all the others (see also Finlay et al. 2001). Central to this notion is the realization that changes in a region's proportional size may be of functional significance even if they are in line with allometric expectations. By the same logic, evolutionary changes in absolute brain size may be functionally significant and adaptive even if they are an "automatic" consequence of changing body size. As I see it, any major change in absolute brain size, whether expected or not, is likely to cause changes in an animal's behavior that are visible to natural selection even if selection *also* acts on body size. In other words, I argue that natural selection can operate within constraints imposed by scaling rules.

6. Evolutionary changes in brain region structure

As brain regions increase in absolute and/or proportional size, they frequently change in internal organization. Most dramatic is that enlarged brain regions often become laminar, with cells partitioned into sheets. One likely benefit of lamination is that it allows for corresponding points in sensory or motor maps to be interconnected with a minimum of dendritic or axonal wiring, which saves space and speeds up information processing, particularly as neuron number becomes large. In addition, lamination probably evolved so frequently because it is fairly easy to develop. Specifically, I argue that the development of laminar structures is probably based on the same set of molecular mechanisms that are already used to build topographic maps within the brain. If that is true, then the evolution of laminar brain regions from unlaminated homologs is explicable in terms of both developmental ease and functional significance.

Another crucial correlate of increasing brain region size is the proliferation of regional subdivisions (Fig. 6). The dorsal thalamus, for instance, is both larger and more subdivided in mammals than in other vertebrates (Butler 1995; Jones 1985). Similarly, the posterior tubercular region of the diencephalon is considerably larger and more subdivided in teleost fishes than in other species (Braford & Northcutt 1983). In both instances, most of the new subdivisions probably evolved by the phylogenetic *segregation* of a single ancestral region into several distinct parts. In other instances, however, novel subdivisions most likely evolved by the phylogenetic *addition* of new parts to a conserved ancestral set. The mammalian neocortex, for

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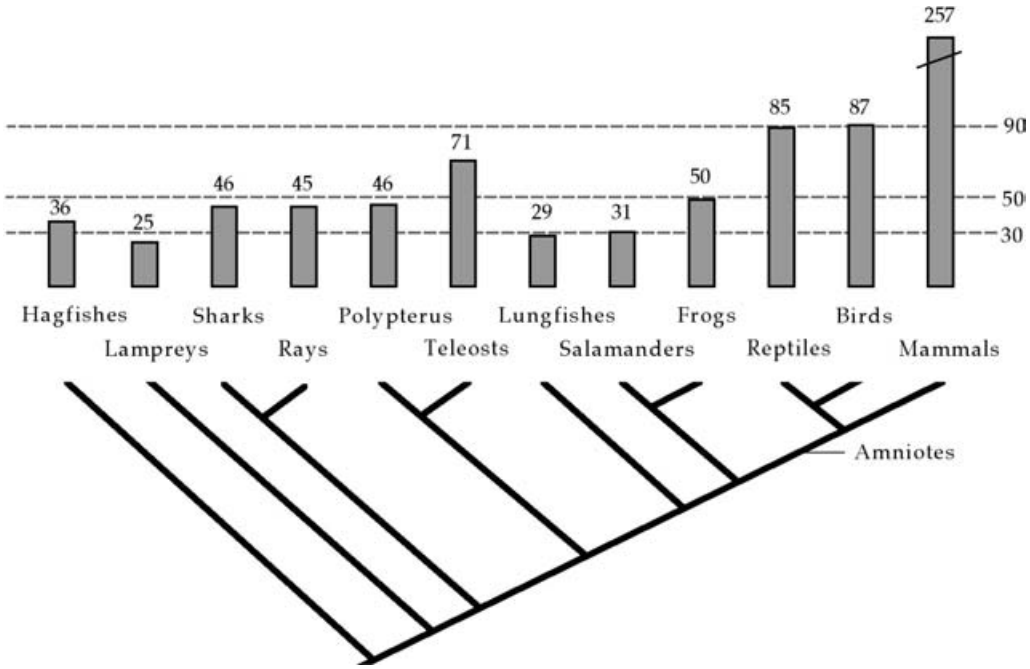

Figure 6. Cladogram of forebrain complexity. Shown across the top are counts of how many different cell groups experts have described in the diencephalon and telencephalon of representative species from each major group of vertebrates. The most parsimonious interpretation of these data is that forebrain complexity increased in amniotes, mammals, and teleosts – and decreased in lungfishes and salamanders. (For references see Wicht & Northcutt 1992.)

example, added several new areas to a highly conserved set of primary sensory and motor cortices as it increased in size. Therefore, it is rash to claim, as some have done (Johnston 1923), that the evolution of more complex brains never involved the creation of new parts.

The mechanisms by which new brain regions are added to existing brains are still unclear. Some even doubt that it is possible for brain regions to evolve "from nothing" (Ebbesson 1984). However, whenever an embryonic precursor increases in size, relative to the ancestral condition, then part of it might be subjected to a hitherto unseen combination of developmentally important molecules (e.g., morphogens), and that could well cause an additional adult brain region to appear (Striedter 1998b). To date, there is little concrete evidence for or against this hypothesis, but future research is likely to yield relevant data. As we learn to explain phylogenetic addition in terms of changes in development, it will become important, once again, to keep in mind that developmental explanations need not be at odds with adaptive scenarios. Although increases in brain or region size may "automatically" lead to the creation of more subdivisions, those increases in structural complexity could be useful to an animal if they allow the various subdivisions to become functionally specialized (Jacobs & Jordan 1992; Nolfi 1997).

7. Evolution of neuronal connectivity

Although neuronal connections are frequently conserved in evolution, they also vary between species. One prominent attempt to explain that variation was Ebbesson's parcellation theory (Ebbesson 1980; 1984). It postulated that, as brains segregate into more and more subdivisions (see above), they selectively lose connections from their daughter aggregates (Fig. 7). The strong version of this theory, which states that neurons only lose connections and *never* evolve new ones, is clearly false. However, the weak version of Ebbesson's theory, which allows for at least some novel projections, remains a possibility. In fact, the weak version is probably correct because, as I mentioned above, a brain's average connection density must decrease with increasing brain size. If connections were not lost as brain regions proliferate, brains would become prohibitively expensive in terms of wiring costs (see Fig. 7).

Another principle that helps explain some variation in neuronal connectivity is Deacon's (1990b) displacement hypothesis, which I call the rule of "large equals well-connected." It holds that, whenever a brain region increases in proportional size, it tends to receive more inputs and project to more targets than it did ancestrally (conversely, decreases in proportional size should reduce connectivity). This rule is grounded in the observation that neural connections generally compete during development for access to target sites. Although Deacon's rule does not yet have broad empirical support, it is consistent with several lines of evidence, such as the discovery that neocortical projections to the medulla and spinal cord become more extensive as the neocortex becomes disproportionately large (e.g., Nakajima et al. 2000). This specific finding is intriguing because it may well explain why mammals with proportionately large neocortices tend to be more dexterous than mammals with smaller neocortices (Heffner & Masterton 1983).

Finally, it is worth noting that evolutionary changes in the size of one brain region may affect the size of other regions by means of axon-mediated trophic (or inductive)

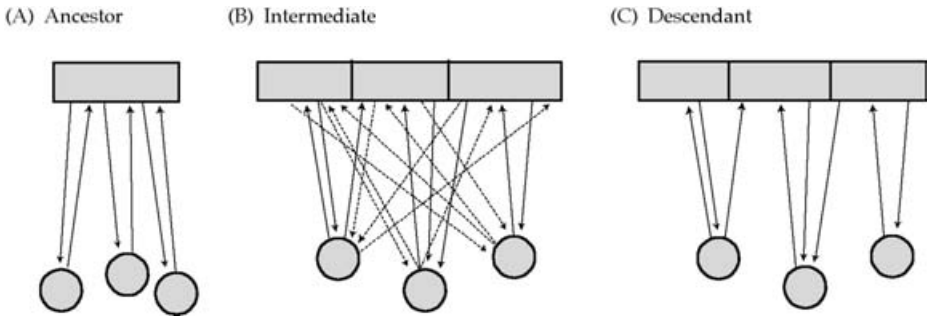

Figure 7. Ebbesson's parcellation theory. In order to grasp Ebbesson's parcellation theory, imagine (A) an ancestral brain region (gray rectangle) that has reciprocal connections with three other regions (gray circles). Now imagine (B) that the rectangular brain region segregates phylogenetically into three distinct brain regions, all of which retain the same set of three reciprocal connections (solid and dashed arrows combined). According to Ebbesson, some of those connections would be lost (dashed arrows), leaving a derived condition (C) in which each of the three rectangular regions retains only a subset of their ancestral connections. Obviously, (C) has a lower connection density than (B) and is, therefore, cheaper in terms of wiring costs. (After Ebbesson 1984.)

interactions (Katz & Lasek 1978). For example, evolutionary decreases in eye size are reliably associated with decreases in the size of retinal targets that depend on the retina for trophic support (Cullen & Kaiserman-Abramof 1976). Decreases in the size of the primary retinal targets, in turn, tend to cause decreases in the size of secondary visual areas, such as striate cortex (Rakic et al. 1991). Apparently, however, such "epigenetic cascades" (Wilczynski 1984) are relatively limited and rare, because (1) not all brain regions require trophic support, (2) many regions may obtain trophic support from multiple sources (Finlay et al. 1987), and (3) decreases in the size of one input frequently lead to compensatory increases in other, previously minor or non-existent projections (Schneider 1973). Overall, we can conclude that epigenetic cascades should occur mainly in unbranched pathways or loops but not in the reticulate circuits that are so typical of brains.

8. Interim conclusion

Evolving brains are subject to a tangled web of rules and principles, containing many constants and some crucial variables, chief among them absolute brain size. Of course, brain size is not everything, but absolute brain size is a more interesting variable than many neuroscientists appreciate. This conclusion may seem odd in our modern age, where most research is aimed at neuroanatomical or physiological details, but it is incorrect to think of size as being independent of details. Evolutionary changes in absolute brain size frequently go hand in hand with major changes in both structural and functional details; indeed, they often demand them!

This insight is not new (Deacon 1990b; Finlay & Darlington 1995; Ringo 1991), but it has never been presented as extensively as in the target book. Therefore, it is fair to ask how well this size-driven view of vertebrate brain evolution "works" in the real world. It is fine to talk of abstract rules and principles, but can they help us understand specific brains? In an effort to answer this question, I devote the next two sections of this précis (Chs. 8 and 9 of the target book) to the brains of mammals and, within mammals, humans. How do the brains of these two taxonomic groups differ from the brains of other vertebrates? What, if anything, makes them special? How well do the aforementioned scaling principles apply?

9. What's special about mammal brains?

Although mammals comprise only about one-tenth of all the vertebrates, they are a reasonably successful class of animals, particularly if we include humans. This success is due to various factors, notably the ability to generate internal body heat and an extended hearing range. Neurobiologically, mammals are distinguished mainly by their neocortex, which has nonmammalian precursors (Aboitiz et al. 2003; Medina & Reiner 2000; Puelles 2001; Striedter 1997) but is highly modified and genuinely new. Although several alternative hypotheses of neocortex origins have been proposed (Butler 1994; Karten 1969; Reiner 2000; Striedter 1997), most data indicate that the six-layered mammalian neocortex evolved from a tri-laminar reptilian precursor (called dorsal cortex) by adding several cellular layers (Reiner 1993), adding an auditory processing region (Puelles 2001), and modifying the trajectory of incoming sensory projections (from tangential to radial; Fig. 8).

The above hypothesis – that mammalian neocortex evolved from something like a turtle's dorsal cortex – is supported by a great deal of comparative neuroembryological data (Holmgren 1925; Puelles et al. 2000), but it is not consistent with some of the connectional data. Most incongruous is the finding that, in contrast to the mammalian neocortex, the reptilian dorsal cortex receives no auditory input from the dorsal thalamus. In order to make this observation fit with the hypothesis of a one-to-one homology between neocortex and dorsal cortex, we must assume that connections from the auditory region of the dorsal thalamus "invaded" the lateral neocortex as mammals evolved. This idea is startling at first, but makes more sense when we consider that early mammals also modified dramatically their peripheral auditory system (Kermack & Mussett 1983). Further support for the invasion scenario derives from the discovery that, among mammals, the ascending auditory axons lose their subcortical projections and expand their neocortical target

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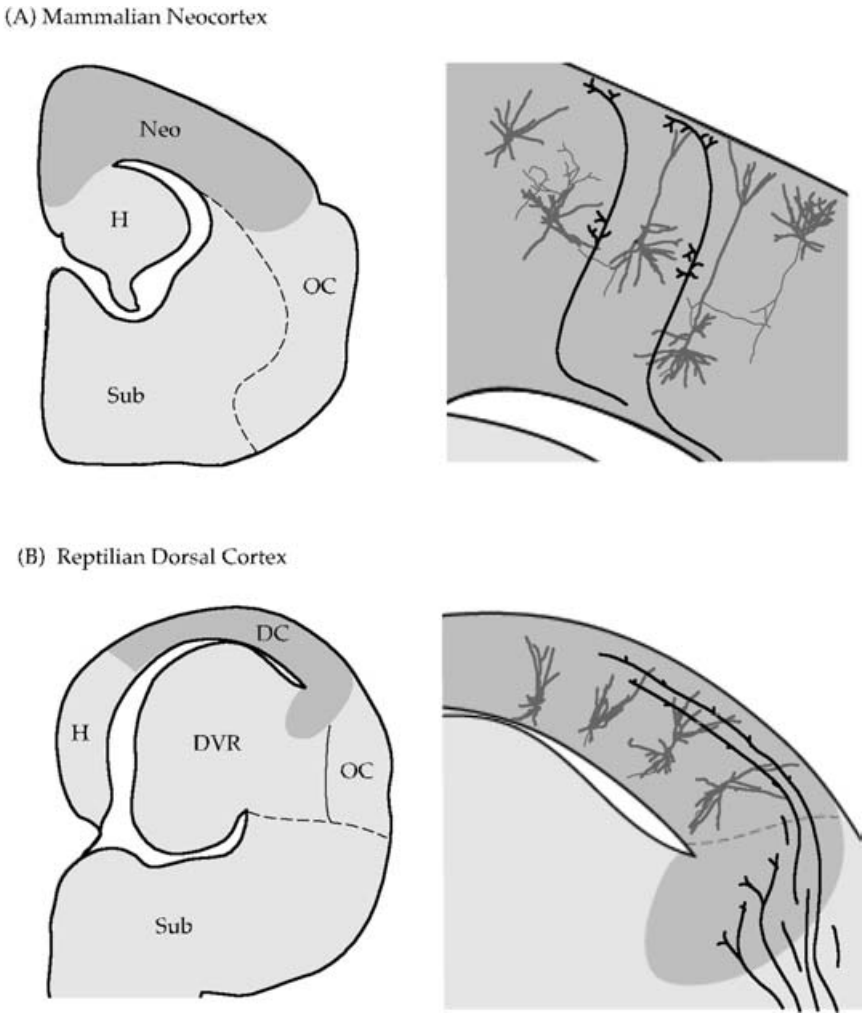

Figure 8. Neocortex and its reptilian homolog. The mammalian neocortex (Neo) and the reptilian dorsal cortex (DC) occupy topologically similar positions within the telencephalon (cross sections shown on left). Both structures also receive sensory inputs from the dorsal thalamus (dark lines in the close-ups on the right). However, in the mammalian neocortex those dorsal thalamic axons course radially through the neocortex, whereas in reptiles they course tangentially. The latter arrangement does not allow for the formation of fine-grained topographic maps. (After Connors & Kriegstein 1986; Heller & Ulinski 1987; Mulligan & Ulinski 1990; Valverde 1986; Valverde et al. 1986.)

as hearing capacity improves (Fig. 9). Most likely, that expansion of the cortical auditory region helped to augment the newly evolved mammalian capacity for high frequency hearing (Manley 2000).

Looking beyond the neocortex, we find that mammal brains are similar, though not identical, to reptile brains. For instance, a hippocampal formation is clearly present in all amniotes (indeed, it seems, in all jawed vertebrates), but the mammalian hippocampus did acquire some specializations. Most notably, the classic "trisynaptic circuit" of the mammalian hippocampal formation appears to be absent from reptile or amphibian brains (Neary 1990; Ulinski 1990). Also unusual is that the mammalian hippocampal formation receives most of its sensory input from the neocortex. In contrast, the neocortical homologs in reptiles and amphibians receive most of their sensory inputs from the dorsal thalamus. In other words, the flow of information into the hippocampal formation was apparently rerouted through the neocortex as mammals evolved. A comparative analysis of basal ganglia circuitry reveals a similar phylogenetic rerouting of information flow. Overall, these data indicate that even when you have a conserved "fundamental scheme" of brain regions and circuitry (Marín et al. 1998; Reiner et al. 1998), many minor changes in brain region size and connectivity may conspire to yield major changes in how information flows through the system and, consequently, in how the whole brain "works."

How well do the principles I have reviewed thus far explain mammalian brain evolution? Clearly, mammal brains are not just reptile brains scaled up or down. The emergence of mammalian neocortex certainly involved several highly specific changes in brain anatomy that are not explicable in terms of any scaling rules. On the other hand, the appearance of "new" auditory projections to the neocortex in early mammals is consistent with the principle of "large equals well-connected" because early mammals probably enlarged their neocortex (compared to the proportional size of its reptilian homolog). More important, much of the neural variation *within mammals* is explicable, at least in part, in terms of changing brain and neocortex size. Indeed, part of what makes mammalian

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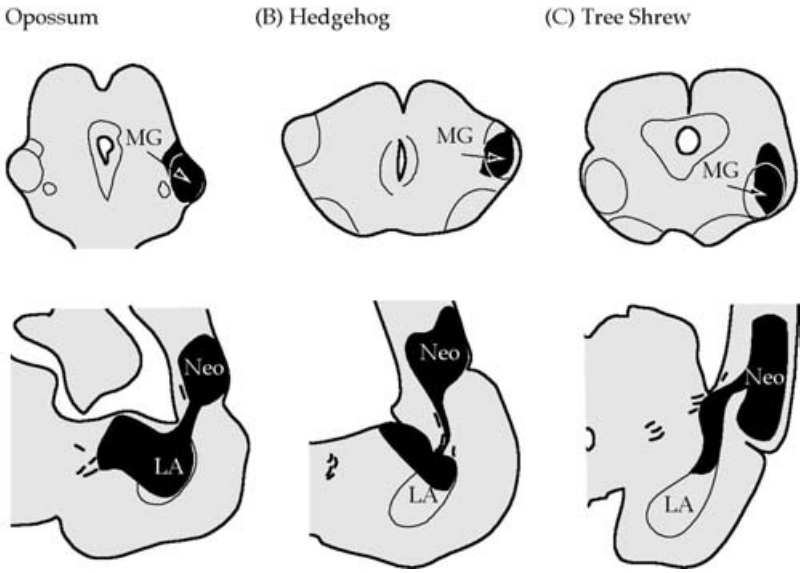

Figure 9. Variation in the ascending auditory pathway. Tracer injections into the main dorsal thalamic auditory nucleus, the medial geniculate (MG, top row), reveal ascending projections (black areas, bottom row) that vary between species. In short-tailed opossums, roughly 50% of MG cells project to the subcortical telencephalon, including the lateral amygdala (LA). In hedgehogs, that percentage is reduced to 25%, and in tree shrews, which are closely related to primates, merely 5% of MG neurons project to subcortical targets. Apparently, MG axons shifted their projections away from the lateral amygdala and into the neocortex as mammals evolved and increased the proportional size of their neocortex. This expansion of the neocortical auditory projection may well be related to the increased capacity for high frequency hearing that occurred early in mammalian phylogeny. (After Frost & Masterton 1992).

brains special is that the neocortex is eminently scalable. Its modular architecture, with afferents coming in radially (Diamond & Ebner 1990; Supèr & Uylings 2001) and long axons coursing through the underlying white matter, is much more efficient, at least in terms of wiring costs, than the architecture of its reptilian precursor (Murre & Sturdy 1995). That probably explains why mammals with large neocortices evolved more frequently than reptiles with large dorsal cortices.

An intriguing sidebar to the story of mammalian brain evolution is that avian forebrains evolved along a very different trajectory. Instead of expanding their dorsal cortex, most birds expanded their dorsal ventricular ridge (DVR; see Fig. 8B), which is most likely homologous to the mammalian claustrum and part of the amygdala (see Striedter 1997). This expanded DVR is the major sensorimotor region of the avian telencephalon (Ulinski 1983), and it is functionally so similar to the mammalian neocortex that some authors have argued the two structures must be homologs (Karten 1969; Karten & Shimizu 1989). However, since DVR and neocortex develop from very different developmental precursors (Puelles et al. 2000), their functional similarities are most parsimoniously interpreted as the result of convergence (i.e., the independent evolution of very similar features from different starting points). This conclusion is astonishing but becomes less remarkable once we consider that birds and mammals have converged also in many other attributes, including endothermy, upright gait, high frequency hearing (Manley 2000), and a trisynaptic circuit in the hippocampal formation (Kahn et al. 2003). Collectively, all these convergences help to explain why many birds are at least as "intelligent" as most mammals (Macphail 1982).

10. What's special about human brains?

Are we "the paragon of animals," as Shakespeare put it, or was Nietzsche right to call us "clever beasts" (Smith 1987)? Neurobiologists tend to approach this question by asking what, if anything, is special about human brains. Unfortunately, that neurobiological question is tricky to resolve decisively (see Cosans 1994). For one thing, most of the techniques that are routinely used to study neuronal connections and activity in nonhumans are, by and large, not feasible in humans. We do have a rapidly expanding set of functional imaging data on human brains, but those are difficult to compare with the more detailed anatomical and physiological data available for nonhumans. The most serious problem is that, if we want to find uniquely human traits, we cannot compare humans to mice, rats, or even macaques. Instead, we must compare our brains to those of our closest living relatives, the chimpanzees. Since neurobiological data on chimps and other apes remain extremely scarce, the literature on human brain evolution is rife with speculation and uncertainty. Nonetheless, we can discern at least a tentative outline of how human brains evolved.

In the roughly six million years since hominins (bipedal apes) diverged from other apes, absolute brain size increased dramatically (roughly fourfold). Particularly intriguing is that this increase in absolute brain size was not "slow and steady," but occurred in several bursts (Hofman 1983). When the genus *Homo* first evolved, absolute brain size increased dramatically, from about 400 cm^3 to about 800 cm^3 (Fig. 10). During the next 1.5 million years, when *Homo erectus* reigned, absolute brain size remained relatively constant. However, when *Homo sapiens* emerged, absolute brain size increased

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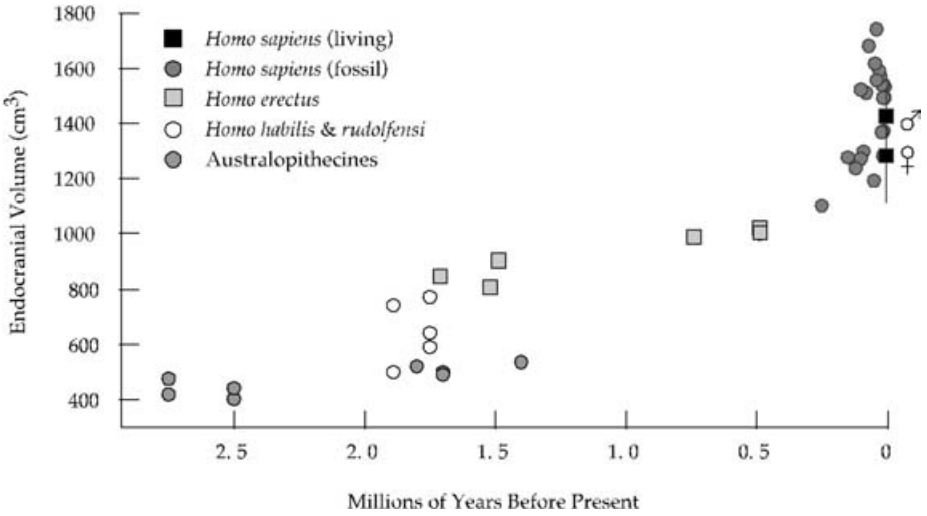

Figure 10. Endocranial volumes of fossil and extant hominins. Plotting endocranial volume (which reflects absolute brain size) against the estimated age of fossil specimens, we find that endocranial volume increased in fits and starts, rather than steadily. One major brain growth spurt occurred with the origin of the genus *Homo*; another one occurred as *Homo sapiens* evolved. The brains of some early *H. sapiens* (particularly Neanderthals) were larger than those of average living humans, but the variability in absolute brain size among both early and recent humans is great. Moreover, recent humans most likely evolved from early humans that had brains about as large as ours (Ruff et al. 1997). Therefore, I conclude that absolute brain size did not, on average, decrease as recent humans evolved. (Data from Kappelman 1996).

again until, roughly 100,000 years ago, our species reached its current level of 1,200 to 1,800 cm^3 (Lee & Wolpoff 2003). Linking this pattern to behavior is difficult but one thing seems fairly clear: the factors driving evolutionary changes in hominin brain size probably changed over time. The first big jump in absolute brain size was probably related mainly to changes in the hominin diet (Aiello & Wheeler 1995; Leonard & Robertson 1994; Leonard et al. 2003). In contrast, the second major brain growth spurt, the one in *H. sapiens*, was likely driven by the need to outwit other humans for access to mates and other resources (Alexander 1990; Byrne 1997; Humphrey 1976; Rose 1980).

Given these increases in absolute brain size, the principle of "late equals large" (see sect. 5) predicts that humans should have disproportionately large neocortices. Indeed, the human neocortex:medulla ratio is roughly double that of chimpanzees (Frahm et al. 1982; Stephan et al. 1981). Furthermore, Deacon's principle of large equals well-connected (see sect. 7) predicts that the enlarged human neocortex should have expanded its projections to other, proportionately smaller, brain regions. It is not surprising, therefore, that humans seem to have unusually extensive projections from the neocortex to the motor neurons of the medulla and spinal cord (Iwatsubo et al. 1990; Liscic et al. 1998; Pearce et al. 2003; Rödel et al. 2003). These expanded neocortical projections probably allowed modern humans to produce more finely controlled movements of the hands, respiratory muscles, eyes, jaws, lips, tongue, and vocal folds. Those increases in manual, ocular, oral, and vocal dexterity were probably prerequisite for the emergence of human language, some 50,000 to 100,000 years ago.

Within the neocortex, the lateral prefrontal cortex became disproportionately large in *Homo sapiens* (Fig. 11). That increase in proportional size probably increased the amount of influence that the lateral prefrontal cortex has within the brain. Although it is difficult to specify the nature of that influence (Passingham 1993), I propose that the expansion of the human lateral prefrontal cortex (and its associates, such as the dorsal pulvinar) increased the ability of humans to perform what I call "unconventional" behaviors, such as looking or pointing away from salient stimuli (Connolly et al. 2000; Everling & Fischer 1998) and reaching around barriers to obtain food (Santos et al. 1999). That increased capacity for unconventional behavior may well have combined with the improved dexterity mentioned above to give us *symbolic* language (Deacon 1997). If that is true, then human language probably evolved, at least in part, as an automatic but adaptive consequence of increased absolute brain size. Once language had evolved, human behavior changed dramatically without further changes in average absolute brain size.

An interesting question is whether humans have come to the "end of the road" (Hofman 2001) in terms of increasing their absolute brain size. Although there are no definitive answers to that question, it is worth pointing out that there are serious costs to increasing brain size. As I mentioned above, brains are metabolically expensive organs (Aiello & Wheeler 1995), and those costs must be paid for by improving diet quality or reducing other energy expenditures. In addition, human neonates cannot evolve much larger brains if they are to pass through their mothers' birth canals (Rosenberg 1992). Finally, the decrease in connection density that comes with increasing brain size (see sect. 7) imposes serious "computational costs," one of which is that the two cerebral hemispheres tend to become functionally more independent of one another (Fig. 12). That increased independence probably encouraged the two hemispheres to become more asymmetrical (Gannon et al. 1998;

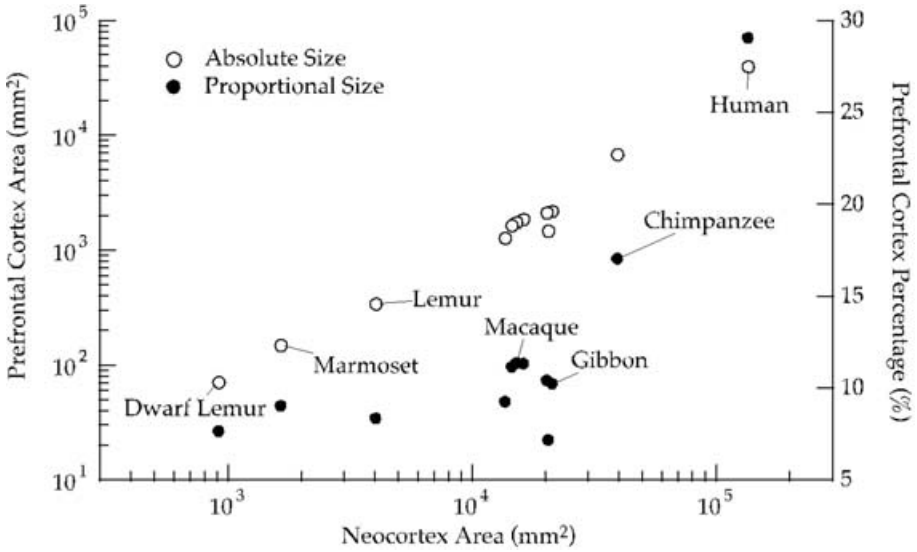

Figure 11. Lateral prefrontal cortex scaling in primates. Few topics in evolutionary neuroscience are as heavily debated as whether or not humans have enlarged frontal and/or prefrontal cortices. Illustrated here are Brodmann's (1912) data on lateral prefrontal scaling in primates. They illustrate rather convincingly that, in terms of both absolute and proportional size, the lateral prefrontal cortex is larger in humans than in other primates. Whether it is also larger than we would expect, given our absolute neocortex size, remains debatable (see Passingham 2002).

Gilissen 2001; Rilling & Insel 1999). Increased asymmetry is "good" because it decreases redundancy, but it also makes human brains unusually vulnerable to brain damage, since the contralateral "back-up systems" are removed. Collectively, these diverse costs of increasing brain size help to explain why hominin brain size essentially plateaued 100,000 years ago[1] (see Fig. 10).

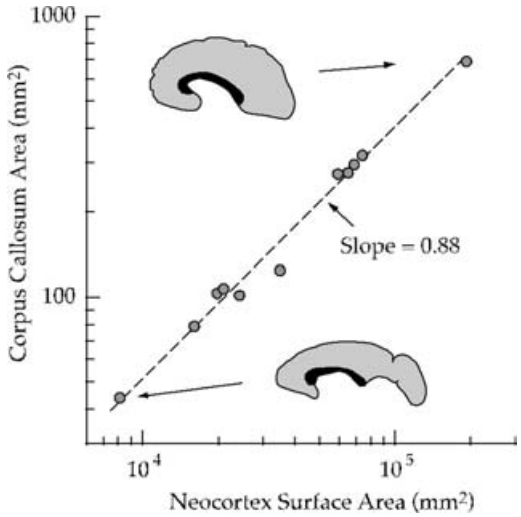

Figure 12. Corpus callosum scaling in primates. As the neocortex increases in surface area, so does the corpus callosum. However, the increases in corpus callosum size fail to keep up with the increases in neocortex size, which means that, as the neocortex increases in size, it becomes less densely interconnected by callosal axons. That, in turn, is likely to make it more difficult for the two hemispheres to work together and, therefore, promotes an increase in hemisphere asymmetry (based on Rilling & Insel 1999).

11. Overall conclusions and prospects

Absolute brain size increased repeatedly in several different lineages, and those increases were associated with several law-like changes in brain organization. Specifically, the increases in absolute brain size were linked to increases in brain complexity, as measured by the number of distinct brain regions, and to changes in brain region proportions, with late-born regions generally becoming disproportionately large. The size-related changes in regional proportions probably caused major changes in neuronal connectivity, with proportionately enlarged regions becoming "better connected." This, in turn, most likely led to major changes in brain function, with the more widely connected regions becoming disproportionately influential. In addition, evolutionary increases in absolute brain size were generally accompanied by decreases in average connection density, causing larger brains to become structurally and functionally more modular. Thus, the single variable of absolute brain size ties together many different attributes of brains. As those links are causal in nature, they have explanatory force.

It is important to stress, however, that the variable of absolute brain size does not capture or explain *all* of the variation that we see in brains. The evolutionary origin of the mammalian neocortex, for example, is not explicable in terms of any change in absolute brain size. Clearly, some evolutionary changes in brain structure were causally independent of changes in absolute brain size. Indeed, I suspect that the origin of most major vertebrate lineages involved some key neuronal innovations (such as the neocortex) that were causally unrelated to changes in absolute brain size but crucial to that lineage's overall success. Nonetheless, those key innovations were relatively rare. Occasionally they pushed brain evolution onto novel "tracks," but within those tracks, brains varied mainly in absolute brain size (and its diverse correlates). Using this

https://doi.org/10.1017/S0140525X06229017 Published online by Cambridge University Press

metaphor of "tracks," we can say, for instance, that human brains are firmly on the primate track but became so large that they evolved a plethora of size-related specializations.

Because distantly related lineages evolve along divergent tracks, it makes little sense to compare the brains of distant relatives in terms of absolute brain size. For example, it is not very informative to know that the brains of some large whales weigh five times as much as human brains, because whale brains also differ from human brains in many other attributes (e.g., whales have an unusually thin, poorly laminated neocortex). Whenever we compare taxonomic groups that are as distantly related as humans and whales, relative brain size tends to be a more useful variable than absolute brain size. Indeed, a relative brain size analysis immediately reveals that humans and toothed whales both have considerably larger brains than "average" mammals of their body size. However, in comparisons between closely related species (such as dolphins versus whales, or humans versus chimpanzees), differences in absolute brain size are very informative.

Overall, my take-home message is that comparisons of absolute brain size are far more interesting and meaningful than comparative neurobiologists historically assumed. Specifically, I argue that major changes in absolute brain size are likely to have important consequences for brain structure and, ultimately, animal behavior even if they are not accompanied by major changes in relative brain size. Because prior books on vertebrate brain evolution have generally not emphasized these causal correlates of changing absolute brain size, the target book will likely spawn a few debates and, hopefully, some new research.

ACKNOWLEDGMENTS

I thank the Institute for Advanced Studies in Berlin for a generous fellowship that got my writing project off the ground. I also thank my home department at the University of California at Irvine for sabbatical support, and the National Science Foundation for funding my research. Finally, I thank my wife, Anna, and son, Ian, for coming to Berlin with me.

NOTES

1. Shortly after publication of the target book, fossils of *Homo floresiensis* were discovered on an Indonesian island (Brown et al. 2004). Members of this species lived as recently as 18,000 years ago, were about 1 m tall, and had brains weighing approximately 430 g. Evolution of this species almost certainly involved a phylogenetic reduction in both absolute brain and body size, but how this affected their behavior remains largely unknown (Brown et al. 2004). Moreover, this discovery does not negate my assertion that the evolutionary history of modern *Homo sapiens* did not involve a phylogenetic reduction in average absolute brain size.

Open Peer Commentary

Brain evolution: Part I

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Abstract: Striedter's accessible concept-based book is strong on the macroevolution of brains and the developmental principles that underlie how brains evolve on that scale. In the absence of greater attention to microevolution, natural selection, and sexual selection, however, it is incomplete and not fully modern on the evolution side. Greater biological integration is needed.

Brain evolution is an odd field, at least as presented in Striedter's (2005) book. On the one hand, the subject is fascinating because the vertebrate brain is a very complicated organ producing a juicy array of behavioral routes to fitness. Explaining its evolution through broad principles is exactly what any good science should do. The subject has a rich intellectual history, perhaps too rich for the data, as Striedter points out. The field has experienced a near revolution as a result of better information, more modern understanding of phylogeny, and an infusion of evolutionary developmental biology ("evo-devo"). This good news deserves wider dissemination, making this highly accessible and well-produced account timely and welcome.

On the other hand, even with Striedter's fresh and engaging treatment, the content still has an old-fashioned feel to it. The book does an excellent job with macroevolution, that is, the evolutionary changes that have occurred over relatively long periods of time, studied by comparing clades on a relatively coarse scale. It does equally well with the recent discoveries of molecular developmental tweaks that underlie major clade differences. But it does not have much to say about microevolution – evolutionary change over shorter time periods. The nuts and bolts of natural selection are missing. In a book about the evolution of other features of vertebrates, this would include studies of wild populations to track changes in gene frequencies and their measurable phenotypic consequences in response to changing selection pressures. There is relatively little discussion of tests of hypotheses about the selective pressures responsible for the origin and maintenance of traits. The book is very quiet on the subject of sexual selection and its role in producing bizarre behavior and its neural underpinnings. The author would seem to be experiencing symptoms of discomfort with the concept of adaptation. The book pays a hefty chunk of attention to evolutionary increases in brain size, on the grounds that *Homo sapiens* has a large brain. The result is a balance of perspectives teetering on the edge of falling back into the mid-twentieth century, with evo-devo and a contemporary allometric approach to analyzing brain size coming to the rescue (fortunately) to pull it back from the brink and get it moving toward the twenty-first.

Striedter is up-front about this hole in the content. His explicit aim is to focus on how, not why, brains have changed. He is aware that without much "why," there is a nearly unbridgeable chasm between what brain evolutionists and behavioral evolutionists are interested in. He explains that neuroscience does not yet know enough about what to look for in brains that would correspond to microevolutionary changes in behavior. Neurons are numerous, complex, and plastic. They do not seem like very good traits for studying evolution. The relevant level of brain organization might lie in the functioning of highly distributed networks of neurons, and not in any measurable aspect of neuroanatomy. Such functionality might not even be heritable. In an effort to try to include the "why" of brain evolution, Striedter does refer at a few points to work, for example, relating hippocampus size to food-storing within two families of birds (Krebs et al. 1989) and relating the size of the song control region HVC to song repertoire size in a family of European warblers (Székely et al. 1996). Nonetheless, on the whole he does not seem to think much progress has been made or expect the hole to start getting filled any time soon. He is surely right that there has been much more progress to date on the macro than on the micro side.

But is such pessimism about the near future justified? Why should tackling the microevolution of brains be intrinsically any more difficult than tackling the microevolution of any complex

and plastic aspect of a phenotype? Evolutionary biologists also struggle with complexity and plasticity (see, e.g., West-Eberhard 2003). If crest size, beak size, tail size, and adrenal size can be studied on a microevolutionary scale, why cannot brain size? Why cannot the kind of research on evolution in real time that the Grants have done with Darwin's finches (Grant & Grant 1989), that Endler and others have done with guppies (Endler 1986), and that is being pursued with rapidly evolving stickleback and cichlid fish (Bell 1995; Kocher 2004; Meyer 1993; Peichel et al. 2001) be applied to brains? Striedter's last chapter refers to Endler's work but with a discouraging spin that glosses over all its positive contributions. Sexual selection can produce quite rapid evolutionary change, and so ignoring it will miss some of the best opportunities to study brain evolution in real time. Yes, there has to be heritable variation, but seldom has anyone looked carefully without finding some.

Furthermore, there are already some notable successes in brain evolution science on a more micro scale. One obvious example is the congeneric voles story (Carter et al. 1995; Young et al. 1998). It suggests that a possible brain basis for a relatively rapid evolutionary change in mating system from promiscuity to monogamy in *Microtus* voles was a gene duplication that altered the distribution of the vasopressin 1A receptor subtype. Viral vector transfer of the gene for the receptor into the ventral forebrain of promiscuous male meadow voles caused them to take on the receptor distribution of monogamous prairie voles and to huddle more with familiar than with unfamiliar females, a measure of monogamous pairing tendency (Lim et al. 2004). One species was, in effect, turned into the other with respect to the traits of interest. This thrillingly direct attack on a brain evolution hypothesis gives one hope that the field can escape the limitations of fundamentally correlational comparative approaches. In all likelihood this particular experiment appeared too late to be included in Striedter's book, but why did the rest of the vole story rate only one sentence? This raises the possibility that Striedter (or perhaps the brain evolution community more generally, whose work he is synthesizing) is shying away from the micro scale not because it is intractable, but because its secrets lie in brain chemistry and gene expression, not anatomy.

The ideal book about brain evolution would be more broadly integrative. Student audiences should get to see the whole landscape. Even high-quality renderings of brain evolution as comparative neuroanatomy, like this one, are destined to seem incomplete and behind the times with respect to where both neuroscience and evolutionary biology are. There needs to be more intellectual cross-fertilization with people studying the evolution of other parts of animal phenotypes, including behavior, the point of the brain.

ACKNOWLEDGMENT

Dennis T. Regan's editorial revisions greatly improved this commentary.

Neuroscientists need to be evolutionarily challenged

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Abstract: Evolutionary theory and methods are central to understanding the design of organisms, including their brains. This book does much to demonstrate the value of evolutionary neuroscience. Further work is needed to clarify the ways that neural systems evolved in general (specifically, the interaction between mosaic and coordinated evolution of brain components), and phylogenetic methods should be given a more prominent role in the analysis of comparative data.

The major goal of neuroscience is to understand how brains work. Given that brain mechanisms are products of natural selection, a central strategy in neuroscience should be to use the methods of evolutionary biology, which have been so successful in helping us to understand the mechanisms and design of organisms generally. Yet, with a few honorable exceptions (mainly within the neuro-ethology tradition), neuroscience has signally failed to apply evolutionary reasoning and methods. A typical "comparative" study in neuroscience, for example, involves comparing two or three species (commonly rats or mice, monkeys, and people), which is obviously hopeless as a strategy for interpreting the differences observed in terms of general principles of scaling and natural selection. Yet proper comparative studies are capable of both generating and resolving major questions in systems neuroscience (e.g., Barton et al. 2003; Krebs 1990; Stevens 2001). It is therefore greatly heartening to be able to greet the publication of a major new work in evolutionary neuroscience. I use the term "evolutionary neuroscience" because it is important to avoid the ghettoization of evolutionary approaches that tends to happen when such studies are characterized as being "merely" about brain evolution, as though the latter is a quaint byway. Striedter's (2005) book does an excellent job of demonstrating that evolutionary approaches are not optional extras, but should have a central role to play in organizing and interpreting the mass of complex information about brains currently being generated.

Among the features I particularly liked in the book was the beautiful description, and explanation in developmental and functional terms, of the evolution of lamination in diverse structures and species. Another is the survey of early mammalian brain evolution, an extremely useful update, both empirically and theoretically, on Jerison's (1973) landmark book. One could go on, but an enumeration of the things I agree with would be somewhat dull and pointless. Instead, I wish to raise two areas where I believe further thought is needed.

The first area concerns the discussion of "concerted versus mosaic evolution" (Ch. 5). This debate, in which I have some interest (Barton & Harvey 2000), concerns whether inter-specific variation in brain structure is best explained by scaling laws that tightly constrain the size of various brain components in relation to each other, or by natural selection causing individual neural systems to evolve independently. In general, Striedter presents a balanced viewpoint, correctly emphasizing that both have a role. The real issues, however, are (1) whether the patterns of coordinated evolution of brain components are products of constraints imposed by conserved ontogenetic schedules (Finlay & Darlington 1995), or of functional/connectional constraints (that could be mediated by a variety of developmental mechanisms), and (2) the relative importance of "concerted" versus "mosaic" evolution. Although the idea of constraints through an evolutionarily conserved developmental program is alluring, because of its potentially elegant simplicity in explaining variation, there is currently little evidence for it. The schedule of neurogenesis varies substantially across species, appearing to be only moderately conserved (Barton 2001). Even if it were strongly conserved, there is still great scope for the ontogeny of single components or systems to vary through alterations to the sizes of precursor regions and/or to rates of neurogenesis (as indeed pointed out by Striedter). Striedter nevertheless concludes that "severely mosaic evolution (deviations that are greater than 2- to 3-fold) is less frequent than concerted evolution" (p. 175). The distinction between "mildly" and "severely" mosaic evolution, seems of questionable value (either systems evolved independently of one another, or they did not), as does any attempt to quantify the relative frequency of mosaic and concerted evolution. Striedter's reasoning is based on the observation that comparisons of relatively closely related species show differences in relative brain components size that are generally within a 2- to 3-fold range, whereas comparisons of more distantly related species reveal larger differences. This is,

however, simply the pattern that one would expect to see under a gradualist model of evolution, and does not imply that mosaic evolution is a rare event. The hypothesis that *all* brain evolution is simultaneously mosaic (individual systems evolve independently) and concerted (the components of such systems tend to evolve together because of functional constraints) is still out there.

The second area of concern is the lack of phylogenetic comparative analysis. Striedter goes half way, by drawing attention to the importance of cladistics in reconstructing morphological evolution, and by noting the problem of phylogenetic non-independence between species, and the associated statistical problem. It is something of a missed opportunity therefore, that he does not go on to explain how phylogenetic methods can solve this problem. For example, scaling exponents calculated across species values without reference to phylogenetic effects, and consequently the biological interpretations of them, are not valid (see Harvey & Pagel 1991). One specific case is the analysis of brain structure scaling (Figs. 5.4 and 5.6 in the book), which feeds into the analysis of developmental constraints mentioned above. Another is the scaling exponent for brain size against body size: the perplexing variability according to which taxonomic groups are included (Ch. 4) is at least partly a function of phylogenetic differences in the intercepts (sometimes referred to as "grade shifts"). Phylogenetic methods designed to treat evolutionary events, not species, as independent data points, have provided new estimates of brain size scaling (e.g., Pagel 1999) that may be more consistent and more amenable to biological interpretation. Finally, phylogenetic methods provide a means for testing hypotheses about correlated evolution (i.e., for determining which neural traits evolved with which other neural or behavioural traits). Greater awareness of these methods would promote the development of evolutionary neuroscience.

Practical use of evolutionary neuroscience principles

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Abstract: As Striedter explores the concerted principles that drive brain evolution and the departures that create uniqueness, he scrutinizes and ultimately supports the (unnecessarily controversial) Finlay/Darlington model in which mathematical relationships across mammalian neural development are identified (Finlay & Darlington 1995). Pragmatic impact includes the ability to make novel comparisons across developing species, including humans.

Brain evolution is studied for a variety of reasons, and my interest in Striedter's (2005) book stems from very practical questions. These include how to best relate neurodevelopment across the variety of species in which it is studied, as well as how to make extrapolations to human brain development. These questions can be addressed using evolution theories because, to paraphrase Striedter's translation from Poincaré, scientific facts can and should be used to make predictions. But a predictive approach is supported only after carefully isolating and explaining unifying principles of brain evolution, what Striedter calls "predictions in hindsight." He has expertly accomplished these analyses in his well-organized, well-referenced, and remarkably readable book. Striedter meticulously clarifies the problems we face identifying similarities across species while giving full weight to the complexities, and avoiding "overeager homologizing."

Striedter discusses a central debate in evolution theories, summarized here simply as the "mosaic versus constraint" dispute. Although he praises a resurgent interest in species differences (mosaic), he does not let the pendulum swing too far away from the obvious – there are clear similarities (constraints) in brain evolution. After scrutinizing and critiquing one method of identifying mammalian constraints, the Finlay/Darlington model (Finlay & Darlington 1995), Striedter ultimately supports it. Although this model stirred some controversy, its premise is unarguable; using statistics, correspondences across mammalian brain development are clearly identified. Striedter analyzes the model over its own period of evolution (Clancy et al. 2000; 2001; Darlington et al. 1999; Finlay & Darlington 1995), something previous critics may have failed to do. Striedter realizes that there is not much to argue about in the "mosaic versus constraint" dispute, at least not as far as the Finlay/Darling model is concerned, because the model does not preclude variability, it adjusts to it. Moreover, in the decade since it was introduced, the model has done precisely what a well-designed model can do – incorporate new neural data and additional species, identify and adjust for variability (read "mosaic" if you like), and, for my part I add that, above all, it permits predictions (Clancy et al. 2000; 2001).

The impact of Striedter's support is significant. He carefully investigates the model, and apparently recognizes that conventional allometrical analyses have great importance in understanding evolutionary principles, but novel mathematical techniques have much potential. If we have learned anything from the past, from the history of stereology for example, we accept that there is enormous value in addressing complex questions using math, statistics, and computer science, which is precisely what the Finlay/Darlington model does. In fact, although modern science has always used state-of-the-art methods to answer biological questions, a new term has been coined for these cross-discipline analyses: bioinformatics. There are enormous advantages to this approach, including identification of relationships that are brought to light when a model is truly dynamic. The Finlay/Darlington model is able to take into account the relationships between every single available data point in every single species in the model, identify the similarities, and adjust to account for the differences. Thus, we can move beyond size comparisons, log graphing, and examination of y intercepts – and incorporate cross-species data on a large variety of data including neurogenesis, outgrowth of fiber tracts, and even behaviors such as eye opening.

Because this "bioinformatics" approach uses statistical principles to analyze a large database, the model has predictive (Darlington 1990) and therefore pragmatic power, including new ways to study evolutionary comparisons. For example, Striedter discusses the concept that delays in the timing of neurogenesis likely produce increases in the size of neural regions simply because increased time in the mitotic ventricular zone results in increased numbers of neurons (Gould 1977). Stated more simply "late equals large" (Finlay & Darlington 1995). He also discusses differences in numbers of rods and cones when comparing nocturnal and diurnal species. It might be useful then to compare the timing of rod and cone genesis between two such species for which neurogenesis data are available, cats and macaques. However, it might be of even more interest to "translate" the timing in the faster developing cat into macaque developmental time so that more precise relative comparisons could be made. Using the Finlay/Darlington model, we can do exactly that. Onset and offset of retinogenesis for these two species are depicted in Figure 1 below; differences in rod numbers between the two species do indeed appear to be related to neurogenesis timing. Late equals large, or rather late equals more rods.

As Striedter points out, neurogenesis data are lacking in humans, so comparative analysis of human and nonhuman data have not been possible; however, the Finlay/Darlington model permits similar predictions and comparisons (Clancy et al. 2000). In addition to applications for evolution theories, the model has value for any researcher who has ever struggled to relate the timing of a neural event in a species reported in the

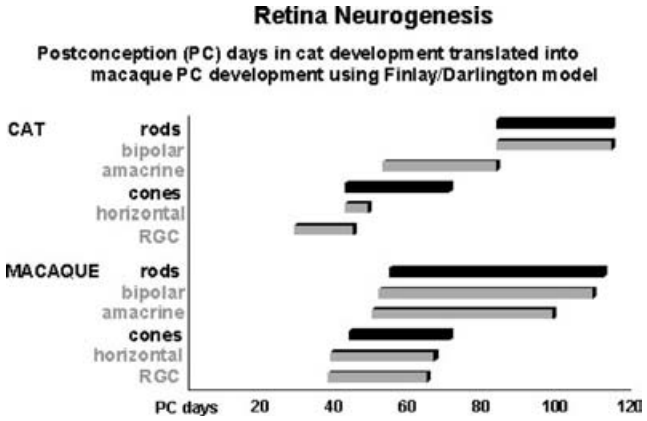

Figure 1 (Clancy). Differences in cat and macaque retinogenesis are obvious when onset and offset are graphed following conversion of cat development timing into macaque time using the Finlay/Darlington model. Onset of rod development is relatively delayed in the rod-dominated cat retina, supporting hypotheses that additional time in the miototic zone equates to increases in cell numbers. Note that cone genesis is surprisingly similar, but there are also clear differences when comparing bipolar, horizontal and retinal ganglion cells (RGC). (From: Finlay et al. 2001; La Vail et al. 1991; Robinson & Dreher 1990.)

literature to the species under study in the lab. Long-range possibilities are considerable and toward that end, we have begun construction of a web-based interactive database that will incorporate additional data and species as they become available (Kersh et al. 2005).

I applaud Striedter for his comprehensive, and comprehensible, summary of the mosaic versus constraint "debate," and for the pragmatic potential advanced by his careful analyses. But while neurobiologists debate evolutionary principles, politicians debate whether or not they can be taught in our schools. Therefore, I also applaud Striedter's book for a more personal reason – the emphasis he places on what is special about human brains. He calls attention to the likelihood that more than one half of university students enter class believing that humans were created, fully formed and quite recently, by a supernatural being. Striedter's chapter on the differences in human brains helps fill a gap for those of us who teach neuroscience in universities where the evolution laws are juxtaposed with state laws. The state legislature is not where the real battles are fought; those battles occur when our reluctant students of evolution go home for the weekend. Survival of the fittest and descent with modification and/or commonality are not antithetical to these students, but evolution of the human species is more troubling, and I cannot teach neuroevolutionary principles without clearly addressing human differences. I welcome the logic, direction, and inspiration that Striedter provides.

ACKNOWLEDGMENT

This work was supported by NIH Grant #P20 RR-16460 from the BRIN Program of the National Center for Research Resources.

Putting humans in their proper place

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Abstract: Striedter's account of human brain evolution fails on two key counts. First, he confuses developmental constraints with selection explanations in the initial jump in hominid brain size around two MYA. Second, he misunderstands the Machiavellian Intelligence explanation.

Georg Striedter is to be congratulated on providing us with a genuinely useful account of brain neurobiology in an evolutionary context (Striedter 2005). We have been sorely in need of such a guide for some considerable time, particularly given the growing convergence between those interested in evolutionary questions and those interested in the hardware for its own sake. Evolutionary neurobiology is surely at long last set for a period of major development. However, Striedter's account of the hominid side of the story requires some reconsideration in two particular respects.

There is an important distinction between developmental constraints and functional selection processes. It is important to remember that the point of Aiello and Wheeler's (1995) expensive tissue hypothesis is that the enormous cost of brain tissue means that some very strong selective advantage is needed to push brain size up a very steep selection gradient. Striedter correctly notes that the initial jump in brain volume with the appearance of *Homo erectus* coincides with an increased meat-based diet. However, changes of diet cannot of themselves cause brain evolution to occur. The energetic costs impose a developmental constraint that has to be solved, but they do not provide a reason why brains should increase in size. That requires a functional explanation in terms of the advantages of having a large brain. Developmental constraints and functional explanations are two very different kinds of explanations (the complete set is commonly referred to as Tinbergen's "Four Why's"; see Barrett et al. 2002), which are equally necessary, but not individually sufficient, to explain any given biological phenomenon.

Conversely, although Striedter offers a genuinely functional explanation for the second phase of exponential brain growth (beginning with the appearance of archaic humans around 0.5 MYA) – namely, the capacity to "outwit other humans for access to mates and other resources (page 10 of the précis) " – this is really less than convincing. The need to outwit others is no greater in human groups than in the groups of other primate species. What is different is the scale of the problem that those exposed to being outwitted have to cope with: the larger the group, the more scope for free riders (those who take the benefits of sociality without paying the costs) to exploit the members of the group, hence the greater the vigilance that group members have to exert. Failure to control free riders leads inexorably to the collapse of the implicit social contract that underpins any such group's existence: the group will continue to exist (and so provide the benefits for its members for which it was created) only so long as members can detect and counteract the behaviour of free riders. The intrusiveness of this problem, however, is a function of group size. The issue of free riding depends on the prior existence of large groups, and it is the evolution of large groups that we really have to explain in cognitive terms. That is what the social brain hypothesis (Dunbar 1998) does.

The second point on which I have to take issue with Striedter's account is his claim that "human language probably evolved . . . as an automatic but adaptive consequence of increased absolute brain size (page 10 of the précis)." I concur entirely with Striedter's argument that it is absolute brain size (or brain part size) that is critical, not relative brain size: we have been dragooned into worrying about relativizing brain size by a very peculiar view that body size must be the default determinant of brain volume (a view for which Jerison's (1973) classic work is invariably cited as justification, even though I would argue that Jerison himself did not necessarily intend such a demand).

The substantive question is whether language could emerge as a simple byproduct of a large brain. I do not believe that it could, not least because it has not done so in other large-brained mammals (e.g., cetaceans and elephants). Language is dependent

on a cognitive capacity that itself requires significant computing power (and hence neural volume), but those cognitive capacities would not evolve *ipso facto* as a consequence of simply having a large brain. Humans have language because they represent the current endpoint of an evolutionary development that focused on specific cognitive skills (principally, those involved in social cognition such as theory of mind; Barrett et al. 2003) that underpin the capacity to bond groups in a particularly intense way that seems to be uniquely characteristic of primates. Language provides the possibility for breaking through what amounts to a glass ceiling in the conventional primate mechanisms of social bonding (Dunbar 1993). A big brain is not required to support language per se, but rather, to support the social cognitive processes that underpin language. And that is a very different issue.

Scaling patterns of interhemispheric connectivity in eutherian mammals

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Abstract: Because network scaling costs tend to limit absolute brain size, Striedter suggests that large cetacean brains must have evolved some novel ways to cope with these costs. A new analysis of available data shows that the scaling pattern of interhemispheric connectivity in cetaceans is isometric and differs from that observed in terrestrial mammals.

Among the various mechanisms driving brain diversity and evolution, Striedter (2005) makes a fundamental point when considering that size-related increased brain fractionation into functionally and anatomically distinct modules is accompanied by a decrease in the density of connection between the two cerebral hemispheres. As a consequence, the two hemispheres become functionally more independent as brain size increases. This is probably the essence of cerebral asymmetry (Ringo et al. 1994), as hemispheres that cannot cooperate might specialize for different tasks. Within primates, this decrease in interhemispheric connectivity is expressed by the fact that the corpus callosum, which interconnects the two cerebral hemispheres, becomes proportionately smaller as neocortex size increases.

As such, the corpus callosum is part of the neocortical white matter. It is therefore relevant to control whether the relationship between corpus callosum surface area and brain size shows a scaling pattern similar to the one that characterizes the relationship between white matter volume and brain size. There is a broad agreement about the fact that neocortical white matter increases disproportionately with brain size (hyperscaling or positive allometry) (Bush & Allman 2003; Frahm et al. 1982; Rilling & Insel 1999; Zhang & Sejnowski 2000). In anthropoid primates, Rilling and Insel (1999) showed a similar scaling pattern when plotting cross-sectional area of the corpus callosum on brain volume. Indeed, the slope of the regression line is 0.71, which is more than two-thirds, namely the value of the slope that would express isometry between an area and a volume. The corpus callosum therefore becomes proportionately larger as brain size increases.

We show that this scaling pattern characterizes the whole primate order. As x variable, we use the brain volume exponent 2/3. The slope of the regression line expressing isometry between an area and a volume exponent 2/3 is 1. The empirical allometric equation of a regression of callosal area on brain volume exponent 2/3 is $\log y = 1.16 \log x - 2.13$ for primates.

If we now consider a group of small-sized mammals such as Insectivora, the scaling pattern of corpus callosum surface area on brain volume exponent 2/3 shows an even more accentuated positive allometry. The equation here is $\log y = 1.27 \log x - 2.55$. A positive allometry appears to characterize eutherian mammals in general, as it is suggested by the study of Olivares et al. (2000) for various terrestrial mammals. Again, these authors showed a slope of 0.76 (more than 2/3) for total callosal area on brain weight. Little variance is expected around this value because $r = 0.99$.

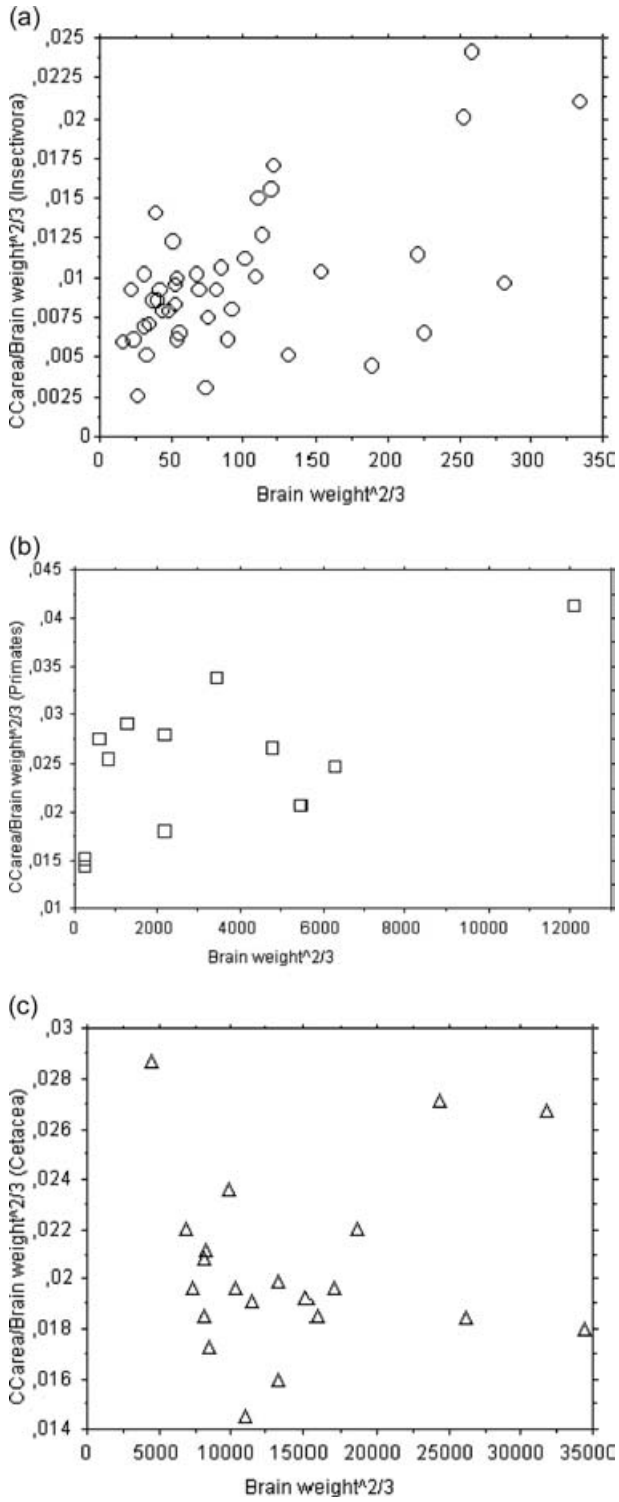

Figure 1 (Gilissen). Ratio between corpus callosum surface area and brain weight exponent 2/3 (^2/3) against brain weight exponent 2/3 (^2/3) for Insectivora (Fig. 1a), primates (Fig. 1b), and Cetacea (Fig. 1c). Data from Gilissen (unpublished), Saban et al. (1990), Stephan et al. (1991), Tarpley & Ridgway (1994).

https://doi.org/10.1017/S0140525X06229017 Published online by Cambridge University Press

When looking at this relationship within large mammals such as Cetacea, the scaling pattern is different from other mammals. Indeed, the empirical equation shows isometry ($log\ y = 0.99\ log\ x - 1.65$ for callosal area on brain volume exponent 2/3). The cetaceans have no tendency toward corpus callosum hyperscaling. The 0.99 slope (95% confidence interval, 0.84–1.10) is not significantly different from isometric scaling.

By contrast, the 95 percent confidence interval for the slope is 1.10–1.44 for Insectivora and 1.03–1.29 for primates. The lower bound of the primate and insectivore 95 percent confidence interval is >1. The scaling is therefore significantly greater than isometric in both groups. Overall, it appears that the value of the slope of the regression between corpus callosum and brain size decreases with increasing brain size and reaches isometry in cetaceans.

Another way to present these data is by plotting the ratio between corpus callosum surface area and brain weight exponent 2/3 against its own denominator (Figs. 1a–c). For Insectivora (Fig. 1a), the correlation between y and x is 0.570 ($p < 0.01$), for primates (Fig. 1b) the correlation is 0.637 ($p = 0.02$), but for Cetacea (Fig. 1c), there is no correlation.

These empirically observed scaling patterns of interhemispheric connectivity in Insectivora, primates, and Cetacea indeed suggest that proportional connectivity decreases with increasing brain size. It also echoes the suggestion made by Striedter that, if network scaling costs tend to limit absolute brain size (Hofman 2001), then large cetacean brains, weighing in at more than twice the size of a human brain, must have evolved some other novel ways to cope with network scaling costs. Specifically, the cetacean callosal scaling pattern appears to largely differ from that of terrestrial mammals.

The evolution of computation in brain circuitry

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Abstract: The attempt to derive mental function from brain structure is highly constrained by study of the allometric changes among brain components with evolution. In particular, even if homologous structures in different species produce similar computations, they may be constituents of larger systems (e.g., cortical-subcortical loops) that exhibit different composite operations as a function of relative size and connectivity in different-sized brains. The resulting evolutionary constraints set useful and specific conditions on candidate hypotheses of brain circuit computation.

As reptilian precursors of avian wings presumably had utility in their own right (e.g., speed, swimming, jumping) before they made the evolutionary transition to full-fledged instruments of flight (e.g., Dial 2003; Zhao 2004), so phylogenetic changes among brain areas are studied for their differing contributions to mental function as homologous structures become successively engaged in evolving circuit designs. Human brain has by far the largest brain-body ratio, and by far the largest ratio of telencephalon to remaining brain components; it is also the structure that uniquely yields complex language, extensive manufacture of artifacts, scientific investigation, and elaborate economic, social, and political constructs. Whether this fount of unique species-specific behaviors arises from correspondingly unique circuitry, absent from nonhuman brains, or strictly from humans' unprecedented telencephalic enlargement of that circuitry, is a hotly debated question (see, e.g., Preuss 2000; Striedter 2005, pp. 308–309). Via compelling reviews of a broad range of principles from the literature (cladistics, "late equals large," "large equals well-connected"; Finlay & Darlington 1995; Kirsch et al. 1997; Murphy et al. 2001), Striedter organizes *Principles of Brain Evolution* to build from earliest vertebrate brains through humans, asking at each new level what it is that makes a particular organization (reptiles, mammals, primates, humans) unique, while not falling into the monotonic evolution fallacy. The book builds through phylogenetically shared and distinct components, and allometric growth and reorganization of those components, to arrive at discussions of advanced human cognitive abilities from planning and social interaction to language.

(A note of regard is in order for Striedter's extensive illustrations: any discussion of comparative neuroanatomy is enhanced by informative figures, and, despite the use of only two colors throughout, Striedter's attractive graphs and schematics provide a richly designed and cumulatively well-organized set of features that cleanly and elegantly illustrate both broad points and details.)

Mammalian cortico-striatal loops, in particular, enlarge allometrically and alter in configuration as brain size grows, becoming the system architecture that accounts for the vast majority of territory in human brain (schematically illustrated in Fig. 1 here). The figure highlights three of the primary changes that occur with allometric growth: (1) the growth of longitudinal fasciculi connecting anterior and posterior cortical regions (AC, PC); (2) a shift in targets of the pallidal (P) output stage, from descending motor nuclei (dashed box) to the thalamocortical circuitry that provided its striatal (S) inputs, "closing" the loop (gray "card"); and (3) anterior cortex "invasion" of motor targets formerly innervated by pallidum (see Nudo & Masterson 1990; Striedter 2005, Fig. 8.13 and pp. 324–27). To the extent that cortical, thalamic, striatal, and pallidal circuitry compute similarly in small and large brains, they must be able to contribute to the range of different configurations in which they find themselves embedded; for instance, the basal ganglia's outputs must presumably be intelligible both to motor nuclei and to thalamocortical circuitry (Granger 2004; in press).

As components of telencephalon grow allometrically with brain size, how do the resulting interactions confer new computational capabilities to larger assemblies? This property is far from universal; many algorithms scale poorly with size, and even those that scale linearly or better, do not typically acquire the power to solve new kinds of problems as they grow larger. Striedter (2005, Ch. 8) notes that laminar organization of neocortex (and, possibly, of avian Wulst) may have enabled it to scale to large size without incurring prohibitive space complexity (wiring) costs, but the larger question of added function remains open. It is intriguing to note that grammars are structures that can carry out abstract string processing operations, and grammatical engines exhibit capabilities that enlarge with the size of the grammatical database on which they operate; that is, as the grammatical rule database grows, grammar systems acquire new capabilities, despite performing the same set of functions on this larger database. This is forwarded as a framework within which to think about the quandary: if thalamocortico-striatal circuitry constructs, by its nature, nested sequences of clusters of sequences of clusters, and so on, as has been proposed (Granger 2004; in press; Rodriguez et al. 2004), then these data structures, comprising a specified form of "sensorimotor grammar," provide a candidate explanation for the challenging phenomenon of new behavioral abilities emerging as telencephalic brain structures phylogenetically proliferate.

A long-simmering question of brain evolution has recently been brought to a boil: birds and mammals both have species with very large brain–body ratios, each based on quite different telencephalic expansions (cortex in mammals; dorsal ventricular ridge in birds), but the homological relations have been unclear. Recent relevant findings have focused on the avian song system. In particular, lesions to the lateral magnocellular nucleus of the anterior nidopallium (LMAN) eliminate generative variability, whereas stimulating LMAN increases variability (Olveczky

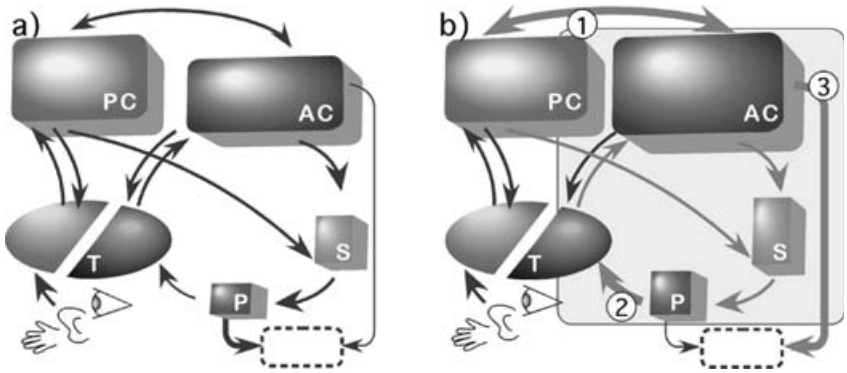

Figure 1 (Granger). Allometric changes in primary components of telencephalon. The anatomical connection pathways among posterior and anterior neocortex (PC, AC), striatum (S), and pallidum (P) are shown for small-brained (a) and large-brained (b) mammals. Sensory inputs (vision, audition, touch) arrive at thalamus (T); projection loops connect thalamus with cortex and cortex to striatum to pallidum and back to thalamus; both pallidal and motor cortex efferents target brainstem motor nuclei (dashed box). (a) In small-brained mammals, primary output from pallidum is to motor systems; primary output of anterior cortex is to striatum. (b) Prominent allometric connection changes in large-brained mammals: (1) Substantial growth occurs in projections between anterior and posterior cortical regions (fasciculi). (2) Pallidal outputs increasingly target thalamus, completing the large cortico-striatal-thalamo-cortical loops. (3) Anterior cortical projections to motor targets grow large (pyramidal tract).

et al. 2005) as a young zebra finch learns to produce the song "taught" by his father. The question of possible mammalian homologues naturally arises, but none has yet been proposed. On predominantly computational grounds, an otherwise unlikely candidate emerges: tonically active neurons (TANs) comprise only about five percent of mammalian basal ganglia but project broadly and diffusely to striatal matrisomes and receive inhibitory input from striosomes (Aosaki et al. 1995; Shimo & Hikosaka 2001; Yamada et al. 2004). In modeling studies (see Granger 2004; in press), nonspecific excitatory TAN activity disrupts matrix responses to a given cortical input, causing small, near-random variations. If cortico-striatal LTP in striosomes corresponds to accretion of statistical "predictions" of dopaminergic rewards that have followed a particular learned matrix response to a cortical input (see, e.g., Granger 2004; Schultz 1998; 2002), then striosomal inhibition of TANs will increase as reward efficacy of a particular response is learned. The resulting model behavior resembles exploratory variability of action during learning, which diminishes as learning succeeds over trials. If so, then TAN damage should selectively impede (and stimulation should increase) early behavioral variability in exploration-based learning; and blocking striosomal inhibition of TANs should prevent the reduction in variability, impeding such learning.

The literature suggests that TANs and LMAN are unlikely to be homologically related, as the former is presumed to be cholinergic (Aosaki et al. 1995; Bennett & Wilson 1999; Koos & Tepper 2002), as well as acting via substance P and neurokinins A and B, whereas LMAN is reported to be glutamatergic (Livingston & Mooney 1997; Stark & Perkel 1999); though it is worth noting that LMAN has repeatedly been reported to exhibit at least sparse cholinergic labeling (Ball et al. 1990; Ryan & Arnold 1981; Sadananda 2004; Sakaguchi & Saito 1991; Watson et al. 1988; Zuschratter & Scheich 1990) and tyrosine hydroxylase immunoreactivity (Bottjer 1993). The literature also suggests that NMDA (such as the receptor targets of LMAN) evokes ACh release at least in mammalian striatum (Kemel et al. 2002); and that LMAN's target nucleus RA is differentially responsive to ACh during the sensitive period of song learning (Sakaguchi 1995), raising the possibility that future findings may identify further points of comparison between these avian and mammalian mechanisms.

Striedter (e.g., 1997; 2005) has been a key player in the momentous consortium that, based on knowledge accreted over decades from fields ranging from behavior to genetics, has very recently (Jarvis et al. 2005; Reiner et al. 2004) transformed the nomenclature of avian telencephalon, not just renaming but entirely recasting the relations among avian brain structures and their homologues among mammals (and other amniotes). It is often asked whether those living in a time of revolutionary change can see or sense the importance of the events as they are occurring. The 1990s may have been termed the decade of the brain, but the present decade seems to be one in which the profusion of data from fields of biology, behavior, and computation is beginning to cohere into theory. The timely appearance of *Principles of Brain Evolution* is a harbinger of such theory, providing an insightful, integrative summary of a vast array of data and opinion on the construction of brains: ours, our near and distant relatives', and glimpses of those yet to be.

ACKNOWLEDGMENTS

The work described here was supported in part by grants from the Office of Naval Research and from the Defense Advanced Research Projects Agency.

Principles of brain connectivity organization

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Abstract: Increases of absolute brain size during evolution reinforced stronger structuring of brain connectivity. One consequence is the hierarchical cluster structure of neural systems that combines predominantly short, but not strictly minimal, wiring with short processing pathways. Principles of "large equals well-connected" and "minimal wiring" do not completely account for observed patterns of brain connectivity. A structural model promises better predictions.

One of Striedter's central and most stimulating ideas is that increases in absolute brain size matter, and have brought with them changes in connectivity as well as greater brain complexity and modularization (Striedter 2005). Interestingly, this modularized architecture may be even more intricate than implied by the general description as a small-world network. Neural networks might be best characterized as clusters of clusters, that is, as a hierarchical cluster architecture. For example, neurons within a cortical column are more densely connected with

each other than with other neurons in the same area. At the same time, neurons within an area receive more area-intrinsic signals than input from other areas, and cortical areas themselves are also arranged in modality-specific clusters (Hilgetag et al. 2000).

This hierarchical organization appears to become more pronounced as the absolute size of the brain increases, together with the size and number of modules. For example, large mammalian cortical areas have tightly integrated subcomponents, such as blobs or stripes, which seem to be missing in smaller areas. At a more global level, the increased number of visual cortical areas in the primate brain results in a segregation into area clusters or "streams," which has not been found for the fewer cat or rat visual cortical areas (Hilgetag et al. 2000). Computational simulations may be able to demonstrate at what group size neural components should be linked into clusters, in order to best support critical network behavior.

One consequence of the efficient multilevel cluster arrangement is that average shortest paths (reflecting the minimal separation of neural components by intermediate projections) between individual neurons in the mammalian CNS may not be much longer than those in the nematode *C. elegans*. Whereas average shortest paths between individual neurons in *C. elegans* ganglia are 3.65 steps long (M. Kaiser, personal communication), they measure 1.79 and 2.16 steps between cortical areas in the cat and rhesus monkey, respectively (Hilgetag et al. 2000). To this one would have to add further synaptic steps within the areas, but due to the hierarchical cluster architecture, that number may be on the same order as for inter-area paths. The formidable capability to maintain very short links between any two neurons in the network (even as brain size increases) comes at a price: the network needs to contain a significant proportion of long-distance projections. This requirement, of course, is opposed to the drive toward strict wiring minimization. Thus, as Striedter also points out, brains evolved not just under one structural or functional constraint, but had to accommodate multiple, partly opposing requirements.

How well do general evolutionary principles allow us to forecast the specific layout of connections in a species? Two principles for predicting the organization of connections arise from Striedter's arguments.

First, according to the principle of "large equals well-connected," or Deacon's rule (Deacon 1990b), larger structures should send and receive more and denser projections. However, this does not necessarily mean that all projection targets of a structure receive axonal input in proportion with the structure's size; individual input patterns, at least in the cat visual cortex, can be more specific (Hilgetag & Grant 2000).

Extensive compilations of cortical and thalamocortical connections in the cat (Scannell et al. 1999) and cortical connections in the rhesus monkey (Young 1993) permit a more general test of the principle. The straightforward test by correlation analysis, however, fails to show any correlation between the size of 16 visual cortical areas in the cat (Hilgetag & Grant 2000) or 30 visual cortical areas in the monkey (Felleman & Van Essen 1991) and the number or summed density of projections that these areas issue. The same is true for the number and summed density of projections that the areas receive (Table 1). Unless current compilations of cortical connectivity are missing most of the existing connections (perhaps with subcortical stations), the "large equals well-connected" principle does not appear to be generally applicable.

Second, the principle of "minimal wiring" suggests that the layout of connections should strongly depend on the distance of interconnected regions, by favoring short-range projections. It does to an extent, but as Striedter notes, there exist a significant number of connections which project over considerable distances and provide "shortcuts" between spatially separate regions. So, while distance correctly predicts that neighboring regions should be connected (Young 1992), it also wrongly forecasts that remote regions should remain unconnected. So far, no model seems to have been published that can specifically predict these remote projections.

Table 1 (Hilgetag). *Correlations (expressed by Pearson's correlation coefficient r) between sizes of visual cortical areas and number or summed density (calculated by adding individual projection strengths) of their total projection outputs and inputs*

Data sets	Outputs	Inputs
Cat thalamocortical (projection number)	−0.12	−0.18
Cat thalamocortical (summed projection density)	0.11	0.03
Rhesus cortical (projection number)	−0.12	−0.04

Thus, the two general principles presented by Striedter only imperfectly explain the specific organization of cortical connections in cat and rhesus monkey. Another principle, not mentioned in the text, offers a promising alternative. Helen Barbas's structural model (Barbas 1986; Barbas & Rempel-Clower 1997) has been very successful in predicting features of cortical projections, in particular their laminar origin and termination patterns. The model proposes that projection origins and terminations depend on the structural similarity of connected areas. For example, more-clearly laminated areas project mainly from their superficial layers into the deep layers of areas with less-pronounced lamination, and vice versa. The model has been verified for ipsi- and contralateral projections of the rhesus prefrontal cortex (Barbas 1986; Barbas et al. 2005), as well as recently for projections among cat visual cortices (Grant & Hilgetag 2004). It will be interesting to see which developmental mechanisms underlie these observations, and if this structural model can be extended to other features of brain connectivity as well (Hilgetag & Barbas 2003).

Despite limitations on the generality of his principles, Striedter has opened a treasure trove of facts and concepts. One looks forward to the evolution of his ideas as further experimental data emerge.

ACKNOWLEDGMENT

Marcus Kaiser provided helpful comments on this manuscript.

Mental attention, not language, may explain evolutionary growth of human intelligence and brain size

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Abstract: Using neoPiagetian theory of mental attention (or working memory), I task-analyze two complex performances of great apes and one symbolic performance (funeral burials) of early *Homo sapiens*. Relating results to brain size growth data, I derive estimates of mental attention for great apes, *Homo erectus*, Neanderthals, and modern *Homo sapiens*, and use children's cognitive development as reference. This heuristic model seems consistent with research.

Striedter's excellent study of brain evolution offers many insights. He interprets human brain growth as serving to master social relations with conspecifics (Striedter 2005, p. 318) and language – his explanation of intelligence (p. 321). This leaves unanswered the question of where symbolic language comes from. Growth

of mental attention (working memory) is a causal interpretation of these skills, consistent with Striedter's findings. This model interprets mammals' dynamic syntheses of *truly novel* (Striedter's "unconventional," p. 333) performances as expressing *developmental* (including phylogenetic) *intelligence* caused by mental attention and learning (Pascual-Leone & Johnson 2005; in press).

Schemes are collections of neurons cofunctional and often coactivated. Truly novel performances are attained via *overdetermination*, that is, conjoint coordination of many sorts of compatible schemes. High cognition begins when the animals' *executive schemes* (located in the prefrontal lobes) can use endogenous attention to boost activation of task-relevant schemes seldom activated by the situation, and also can inhibit schemes irrelevant or misleading for the task. In *misleading* situations (e.g., antisaccade task; Striedter 2005, p. 335), schemes strongly activated by the situation go counter to the intended performance, which can be achieved only when *power of attention* (i.e., *M*-capacity, working memory) is large enough to boost all necessary schemes into high activation. The animal's *M*-capacity limits problem solving in misleading situations. Such situations, therefore, could be used to rank and compare animals' mental-attentional power, even when (as Striedter prescribes, p. 121) each species is studied within a misleading situation suitable for it. NeoPiagetian methods of theory-guided mental-processing task analysis (e.g., Case 1998; Pascual-Leone & Johnson 2005) can aide in this endeavor.

Consider, for example, chimpanzees' use of multiple tools (hammer-stone, anvil-stone, wedge-stone to stabilize the anvil) for cracking nuts (Matsuzawa & Yamakoshi 1996; Russon 2004;Yamakoshi 2004). The infrastructure of this behavior includes two nested functional components. One is an operative scheme (PLACE) for positioning the nut (*nut-food) on the anvil-stone (*anvil) during nut cracking. The other is an operative (CRACK) that uses the hammer-stone (*hammer) as a tool and nut as target (*nut-target). Notice that although the schemes *nut-food and *nut-target both refer to the same actual object, they are distinct schemes, because they belong to different functional activities or practical skills. To crack a nut, chimpanzees must coordinate at least these schemes: CRACK(*hammer, PLACE(*anvil, *nut-food), *nut-target) (f#1).

Because the situation is misleading (e.g., prior schemes would lead chimpanzees to take the nut to their mouth or ignore the stones), this performance requires the simultaneously boosting of all schemes with endogenous attention. Specifically, six distinct schemes must be *M*-boosted (seven if a wedge-stone were used). Thus, six or seven schemes is the highest *M*-complexity level exhibited by great apes, which places them on par with the *M*-capacity of 2- or 3-year-old children (Blake 2004; Pascual-Leone & Johnson 1999; 2005; Russon 2004).

Striedter (pp. 122–25) emphasizes the "grooming clique size" phenomenon – a very different social-intelligence performance of similar complexity. The two nested components (see f#2) now are: (a) The chimp's procedure for social coordination (COOR) with a particular partner and other conspecifics, which subserves (b) a grooming procedure (GROOM) that uses *chimp-self as tool and *chimp-partner as target: GROOM(*chimp-self, COOR(*chimp-partner, *chimp-others), *chimp-partner) (f#2).

Chimp-partner has two schemes to represent two different functional activities. Notice that whether purely cognitive or social cognitive, the performances represented in f#1 and f#2 are sensorimotor: they express external (not internally mediated or mental) intercourse with the environment. Consider, in contrast, human burials (Riel-Salvatore & Clark 2001; Smirnov 1989) – an example of symbolic (i.e., mentally mediated) performance that Striedter examines (p. 312). Two complementary emotional-and-cognitive components may have prompted early humans to make burials. One is the experience that dead bodies (*corpse) enter into putrefaction (*putrefaction) – a shocking experience if one was attached to the living person (*corpse-loved), and this might prompt the idea of burying them (BURY). The other is the feeling of bereavement and mourning for that person, which may elicit need for his symbolic presence, that is, for having a burial ceremony (MEMORIALIZE-Dead) with an added commemorative or symbolic marker (*sym-marker). Mental combination of these two components might have led to the invention of burials. The infrastructure for this symbolic performance is: MEMORIALIZE-Dead(*sym-marker, BURY(*putrefaction, *corpse-loved)) (f#3).

Note that most of the schemes in f#3 are not sensorimotor but symbolic (i.e., mental; Pascual-Leone & Johnson 2005), because they refer not just to the Present (as does *corpse-loved) but to the Future (FUNERAL, BURY, *sym-marker, *putrefaction). The minimum number of schemes to be coordinated is four, if we assume that BURY and *putrefaction (i.e., its motive) should be chunked. In our neoPiagetian cognitive-developmental research, we have found (Case 1998; Pascual-Leone 1970; Pascual-Leone & Johnson 2005) that symbolic/mental schemes carry an attentional (*M*-) demand greater than that of sensorimotor schemes, and the sensorimotor demand (symbolized as "*e*") is still needed to activate executive schemes. There are, thus, two different complexity-counting scales: one for sensorimotor tasks, which we call *Me* (i.e., $M = e$) scale, and another for symbolic tasks, which we denote as *Mk* (i.e., $M = e + k$) scale (see Pascual-Leone & Johnson 2005, for detail). These two sorts of attentional resource appear to have different and separate brain infrastructure (Pascual-Leone & Johnson 2005; in press; Thatcher 1997).

According to our task analysis, formulas f#1 and f#2 illustrate the mental-attentional (*Me*-) capacity of most great apes (equivalent to that of 26-month-old humans), and formula f#3 (equivalent to the mental capacity of 9–10-year-olds) illustrates the (*Mk*) capacity of early *Homo sapiens* (possibly Neanderthals – Riel-Salvatore & Clark 2001; Striedter 2005, p. 312).

These two attentional resources might serve to explain the two spurts of brain growth in Homo genus that Striedter reports (Striedter 2005, Fig. 9.7, p. 314). If we take human development (Pascual-Leone & Johnson 2005) as an approximate model, tentative estimates of mental-attentional capacity in great apes and humans might be: *Hominids.* [*Me.*=6 or 7]; *Homo erectus.* [*Mk.* = e + 1, e + 2]; *Neanderthals.* [*Mk.* = e + 3, e + 4]; *Homo sapiens.* [*Mk.* = e + 5, e + 6, e + 7]. This model would explain both the long-lasting spurt of brain volume in *Homo erectus* and the curvilinear (exponential) growth of brain volume in *Homo sapiens*. Further, it agrees with the task analyses above and some others. For instance, Riel-Salvatore and Clark (2001) report that grave goods (ornaments or objects accompanying the dead) are much more common in burials of the Upper Paleolithic, when *Homo sapiens sapiens* dominates, than in the Middle Paleolithic. If we insert a grave-goods scheme under the parentheses of BURY in f#3, the total mental demand of the burial formula becomes $M = e + 5$, which suggests that *Neanderthals* may not have attained this mental capacity. Using Developmental Stages of Piaget (suitably reinterpreted), this sequence suggests that *Neanderthals.* attained the processing capacity called (mental) Concrete Operations, whereas *Homo sapiens sapiens* attains in addition that of Formal Operations (suitably redefined).

From our perspective (Pascual-Leone & Johnson 1999; 2005; in press), the mental power of symbol-expressing schemes (i.e., symbolic function) is attained progressively, starting with comprehension of simple symbols when the baby is about 12 months old ($Me = 4$). The more mental (*M*-) capacity a human has available, the greater his/her symbolic function will be. Neanderthals must have enjoyed a good symbolic function and may have invented language. In contrast with Striedter, we believe that growth of mental attentional capacity, and not the emergence of language, is at the origin of human developmental intelligence. Nonetheless, the symbolic mind invents language as a tool for thought, and language makes thinking fly.

ACKNOWLEDGMENTS

Cognitive developmental research informing this commentary was funded by the Social Science and Humanities Research Council of Canada. Dr. Janice Johnson, my co-investigator in this research, and Dr. Sergio Morra have contributed much to the commentary with their editing.

Velocity and direction in neurobehavioral evolution: The centripetal prospective

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Abstract: Selection for or against muscle initiates a cascade of centripetal (outside-in), trophically mediated, neurological events through which the environment programs heritable neuromuscular and neuronal connections in a rapid and specific fashion. The velocity, direction, and efficiency of this process are a consequence of the environment acting directly on muscle, the organ of action, and behavioral interface between organism and environment.

A challenge to hypotheses of neurobehavioral evolution is the efficiency of the selective process. For example, is there sufficient time during the biological history of a species for selection acting at the level of individual synapses to have significant effect? I argue that a *centripetal* (outside-in), trophic process acting on muscle, the organ of behavior, shapes the course of neurobehavioral evolution in a much more direct and rapid way than is usually appreciated, and I describe a research program that explores this mechanism. Trophic influences are considered by Striedter (2005) in Chapter 6, "Evolution of neuronal connectivity," but their implications are not fully developed.

A means of exploring the relation between ontogeny and phylogeny became clear to me when, late in his career, my mentor Viktor Hamburger (1975) published his seminal research about naturally occurring cell death of lateral motoneurons in the embryonic chick spinal cord. About half of already produced motoneurons die because they compete unsuccessfully for limited trophic substance provided by the adjacent limb-bud. Hamburger's evidence for a trophically mediated process of motoneuron survival and death was that neuron death is increased by limb-bud ablation and decreased by the addition (transplantation) of a second, "supernumerary" limb-bud. Neuron number is controlled through cell death, not proliferation.[1]

My approach to the phylogenetic consequences of motoneuron death involved the effects of millennia of flightlessness on ratites, large ostrich-like birds presumed to have evolved from flighted ancestors (Provine 1984; 1994). Birds often become flightless in hospitable environments because an environment that does not select for flight selects against the heavy and energy-consuming pectoral flight apparatus. Consistent with this evolutionary scenario is the meager pectoral musculature of the non-keeled, ratite sternum, small brachial spinal motor column, and secondary effects on the neuronal circuitry producing wing-flapping. The tested ratites (emus, two species of rhea, and cassowaries) could move their wings, but they never produced the rhythmic, bilaterally symmetrical, flapping movements of flighted birds, either spontaneously or when dropped or chased. (The game but badly outclassed experimenter chased both young and mature birds. In the interest of subject safety and self-preservation, only young birds were drop-tested while cradled in the experimenter's hands.) If the ratite's ancestors could flap their wings, this circuitry has been lost or could not be activated by extant birds, the probable neurological consequence of selection against pectoral muscles and their neurons. (Conversely, if ratites did not evolve from flighted ancestors, their lack of wing-flapping provides novel behavioral evidence of this flightless heritage.)

Other flightless birds offer informative contrasts to the ratites (Provine 1984; 1994). Four species of penguins, descendents of gull-like birds of the Southern hemisphere, were flightless in air, but were gifted submarine "flyers," propelling themselves through the water with wing-strokes driven by massive pectoral muscles. Although the penguins' neurological flap-producing apparatus obviously is intact, their history of aerial flightlessness did have a neurological consequence – penguins lost the reflex of flapping their wings when dropped, a response of their ancestors and other aerial flyers. Some large birds are rendered flightless because of their weight. Flightless "steamer ducks" from the Falklands that paddle through the water with their wings (like a side-wheel steamer ship), and massive, domestic meat chickens (Cornish x Rock), both retained normal spontaneous and drop-evoked flapping. Unlike the ratites, both retain massive pectoral musculature and associated neurological flap circuitry, and have a relatively short history of flightlessness, only decades in the case of the meat chicken. Other candidates for study are the ostrich, kiwi, flightless owl parrot, the Galapagos flightless cormorant, and a variety of flightless rails and other birds that, if not completely flightless, are nearly so. I predict conservation of the neurological motor pattern generator unless there is marked selection against muscle. Heritable motor disorders such as chicken dystrophy, a non-lethal disorder almost exclusive to the muscles of flight, may be a place to look for dramatic but unsuccessful natural experiments in motor evolution (Provine 1983).

The turtle is an interesting case of the neurological consequence of selection against muscle as noted by Striedter (pp. 221–23). The turtle spinal cord lacks a thoracic lateral motor column, the probable, secondary result of selection against thoracic muscles made redundant because of the rigid shell. However, a developmental study did not discover the predicted ontogenetic recapitulation of phylogeny, with massive attrition of over-produced motoneurons triggered by the absence of thoracic muscles (McKay et al. 1987). The turtle thoracic spinal cord did not over-produce lateral motoneurons that died. The antiquity of the turtle was probably so great that the initial phylogenetic process did not leave its footprints. A better place to look for phylogenetic footprints would be in species that had more recently lost some behavioral capacity and associated musculature.

Centripetal processes have interesting implications beyond those considered above, including a mechanism through which *quantitative* changes in muscles or motoneurons can have *qualitative* consequences. For example, motoneurons that have lost their traditional site of innervation may seek novel muscles and produce novel motor patterns. And interneuronal motor pattern generators that have lost their traditional motoneuron connections may seek other motoneuron targets, producing yet other options for motor novelty. Interneuronal spinal motor pattern generators are the conservative element in this process, escaping the cascade of neuron death triggered by muscle loss (McKay & Oppenheim 1991), lingering in the neurological parts bin, awaiting new motor initiatives. We may be the repositories of obsolete motor programs. The human grasp reflex and the curious Babinski reflex – extending the big toe and fanning the others in response to being stroked on the sole of the foot – are probably the behavioral manifestation of such programs. At present, these reflexes can be elicited only in neonates and in victims of brain damage, being suppressed at other times. What other archaic behavior is programmed within our central nervous system? The most promising place to begin this excavation for the artifacts of our behavioral past is in embryos and adults of animals that have experienced major structural and behavioral change in fairly recent evolutionary history.

NOTE

1. A related and less known fact is that *behavior* also regulates motoneuron number, a conclusion supported by the synchronization between

the onset of limb movement and motoneuron death and the almost total preservation of motoneurons by curare-produced paralysis (Oppenheim & Nunez 1982; Pittman & Oppenheim 1978).

The key role of prefrontal cortex structure and function

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Abstract: The tension between focusing on species similarities versus species differences (phylogenetic versus adaptationist approaches) recurs in discussions about the nature of neural connectivity and organization following brain expansion. Whereas Striedter suggests a primary role for response inhibition, other possibilities include dense recurrent connectivity loops. Computer simulations and brain imaging technologies are crucial in better understanding actual neuronal connectivity patterns.

Striedter's (2005) book represents an important synthesis of ideas and approaches to brain evolution across different levels, with general constraints and evolutionary principles clearly related to neural structures and functions throughout the chapters. It also includes an excellent review of the history of comparative neurobiology (Ch. 2) that concludes by noting the existence of a "tug-of-war between those who emphasize species differences in brain organization and those who dwell on similarities" (p. 50). It is useful to realize that this tug-of-war is being waged today on fields beyond just neuroscience. More generally, these contrasting views are often referred to as the *phylogenetic approach* (emphasizing the continuity and similarities across species) and the *adaptationist approach* (emphasizing the adaptive specializations within each species).

These contrasting views play out at several levels, on many topics, across the behavioral sciences. Most recognize and accept the phylogenetic view of species (ironically, the most controversial aspect of evolutionary theory in the nineteenth century); research with model animals such as pigeons, rats, and apes demands at least an implicit phylogenetic view. On the other hand, the adaptationist view, sometimes labeled as "evolutionary psychology," is currently quite controversial and prone to both adamant support and vigorous opposition (e.g., Rose & Rose 2000; Tooby & Cosmides 1992).

Tension between phylogenetic and adaptationist approaches recurs within Striedter's argument for the importance of absolute brain size. It is an important insight that increases in absolute brain size have implication for patterns of connectivity and organization in general (i.e., more widely connected regions, decreases in average connection density, and more structural and functional modularity as a consequence). Yet there are unaddressed issues within these general implications. How was the brain parsed into modular aspects in the course of evolution? Was it cut like a cake with a chainsaw: random, messy, and in random bits? Or was it like the fissioning of cells, families, and academic departments: the parts already functionally relevant to each other were maintained (relatively higher connection densities), whereas the connections across these parts were reduced? This is precisely the sort of adaptationist question that has been relatively neglected, and is a key to stronger linkages between neuroscience and psychology.

Striedter addresses the core issue of what is special about human brains (Ch. 9), and emphasizes the importance of the enlargement of the lateral prefrontal cortex and its various associated regions, which seems to be well motivated due to the involvement of the prefrontal cortex in high-level cognitive control, selective attention, working memory, and planning, as well as standard intelligence tests, as shown by neuroimaging studies (see Duncan 2001; Miller & Cohen 2001). This chapter also suggests that "response inhibition" plays a major role in enabling the human prefrontal cortex to mediate the production of novel solutions to behavioral problems, but we submit that the nature of the neural mechanisms and architectures supporting flexibility of human behavior and cognition is not yet so clearly specified.

It is also possible that (alternatively or additionally) dense recurrent connectivity loops in the lateral prefrontal cortex enable the formation of stable reverberatory states in working memory, planning, goal representation, and effect anticipation. These active neural representations would "go beyond the stimulus given," and mediate context-sensitive input-output associations, based on a representation of the task context (Duncan 2001; Miller & Cohen 2001). Response inhibition and top-down control of input-output associations would therefore be achieved by means of these stable states in competition (via mutual inhibition) with bottom-up context-independent associations (e.g., impulsive responses). Feedback connections from dorsolateral prefrontal cortex to posterior cortical areas would mediate control of unimodal and multimodal representational states in perception and memory retrieval. In this view, more stable states emerging in the lateral prefrontal cortex via extensive recurrent loops would dominate more transient representations in the brain encoding for current stimuli, responses, and their closer associates. For example, sustained working memory (delay) activity in the lateral prefrontal cortex is immune to interference, whereas delay activity in the inferotemporal cortex is vulnerable to task-irrelevant interfering distracters (Miller et al. 1996). In other words, we share Striedter's view about the crucial role of the enlargement of dorsolateral prefrontal cortex in increasing the flexibility of human behavior and cognition, but we propose that the emergence of convergent recurrent loops within the dorsolateral prefrontal cortex and between the dorsolateral prefrontal cortex and posterior cortical areas (as well as premotor areas) was to mediate this increase in functional flexibility. Another possibility is that the human dorsolateral cortex has evolved to support massive adaptive coding of its neuronal populations (Duncan 2001), combining inputs and outputs in novel context-dependent bindings, in an ongoing dialog with relatively-specialized modules in posterior cortical areas and premotor cortex.

Computer simulations are likely to play a crucial role in shedding light on how different kinds of neuronal connectivity patterns can lead to optimal function-related neuronal coherence within and between brain regions, with special reference to the orchestrating role of the dorsolateral prefrontal cortex. Striedter (Ch. 7) clearly considers the importance of small-world networks, of which the visual cortex can be regarded as an example, and related theoretical studies. Other large-scale simulations (Tononi & Edelman 1998; Tononi et al. 1996) have emphasized the importance of recurrent or re-entrant connectivity systems in binding of neural representations and the emergence of consciousness. The dorsolateral prefrontal area may play a crucial role in coordinating neural synchrony and multiregional cooperative signaling in the brain (see Ch. 9) in a task-dependent fashion, and in encoding action contexts, because of the high number of convergent re-entrant circuits coding multiple modalities and synapses mediating maintenance of stable activation patterns, such as NMDA-synapses (Wang 1999).

Relating structure to function by means of EEG/MEG (combined with high spatial resolution fMRI) and single-cell recording studies, as well as large-scale computer simulation and neuropsychological evidence, may provide a crucial contribution to clarify the role of the dorsolateral prefrontal cortex in making humans superior to other animals in cognition and flexible behavior. Chapter 9 of Striedter's book can be regarded as a good starting point for this challenge.

An evolutionary niche for quantitative theoretical analyses?

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Abstract: Striedter's book offers precious insight into the comparative neuroanatomy of vertebrate brains, but it stops short of addressing what their evolution is all about: how effectively neural networks process information important for survival. To understand the principles of brain evolution, neuroanatomy needs to be combined not only with genetics, neurophysiology, and ethology, but also with quantitative network analyses.

Principles of Brain Evolution (Striedter 2005) is a joy to read. Similar in its orderly complexity, the circuitry of cerebellar cortex is a joy to behold. Nevertheless, the need to adequately process information relevant to the survival of vertebrates has led to the evolution of a diversity of neural tissues, besides cerebellar cortex. Likewise, the ambition to make sense of the structure of modern brains, including our own, has led to a diversity of intellectual approaches, besides the one so valiantly championed by Georg Striedter. *Principles of Brain Evolution* is a major undertaking, though also manageable in size relative to the tome by Nieuwenhuys, Ten Donkelaar, and Nicholson (Nieuwenhuys et al. 1998). We are deeply grateful to Striedter for having written such a trustworthy and insightful resource for all those interested in understanding vertebrate brains; and we immediately proceed, in our commentary, to unceremoniously underscore what the book cannot offer.

It is unorthodox but helpful to contrast *Principles of Brain Evolution* with *On Intelligence* (Hawkins & Blakeslee 2004), the recent proposal on cortical function put forward by Jeff Hawkins, the inventor of the PalmPilot. The two books could hardly be more different: the former being scholarly, comprehensive, and methodical and accurate in its statements carefully polished for a critical audience of peer neuroanatomist readers; the latter being colloquial, fragmentary, and cursory, with its argumentation replete with half-appropriate examples from everyday life. Yet *On Intelligence* offers a theory of how the cortex may work that we can think about, argue with, possibly reject out of hand, and seek to falsify. It is a theoretical perspective with several evident flaws, but it is a useful tool for shaping our insight and eventually our understanding, one that *Principles of Brain Evolution* cannot offer, not *despite* but *because* of its erudition. Both books are a joy to read – in a different number of hours – and both are stimulating. Neither book, though, relies on the quantitative analyses afforded by mathematical models of cortical networks.

In Chapter 8, Striedter considers what is "special about mammal brains," but his thorough discussion of the comparative anatomy of the neocortex and of the hippocampus does not really include an analysis of how these two structures may process information. Hawkins instead sketches an analysis of information processing in neocortical layers, but without elaborating on the comparison with alternative architectures, for example, the avian brain nuclei crucial for generating birdsong (Laje & Mindlin 2002) – thus forfeiting the strength of Striedter's comparative approach. Neither Striedter nor Hawkins attempt a quantitative information theoretical analysis, which should be the ultimate benchmark to assess the efficiency of information processing networks. We have introduced an approach to quantitatively compare the informational efficiency of a laminated versus non-laminated sensory neocortical patch (Treves 2003) as well as a differentiated versus non-differentiated hippocampal circuit (Treves 2004), in order to assess the functional advantages that neocortical lamination and hippocampal differentiation may have brought to mammals. This general comparative approach needs to include specific functional hypotheses which may require revision. (In particular, new experimental data [Lee et al. 2004; Leutgeb et al. 2004] suggest that the CA3-CA1 differentiation may be tightly related to the pattern separation capability afforded by the dedicated DG-CA3 circuitry [Treves & Rolls 1992].) Although such hypotheses may have to be revised or even replaced, the method for testing them on a quantitative comparative basis appears essential in order to understand the structural "phase transition" that, in the simplified language of theoretical physics, seems to have occurred at the early stages of the mammalian radiation.

In Chapter 9, discussing what may be "special about human brains," Striedter briefly mentions, but rather to dismiss them, "Rubicon models," that is, the notion that the faculty of *language* could only be acquired by brains in which the *number* of neurons surpasses a certain threshold (p. 322). He then proceeds to advocate more sensible approaches based on structurally and functionally specific changes in human brain anatomy and physiology. We wonder where such structurally specific changes may be hiding, if they have successfully escaped detection by a century of systematic neuroanatomical investigation; and we suspect that Striedter may be too precipitous in dismissing the possibility of a phase transition allowing the emergence of *qualitatively* novel functionalities – including language – when some anatomical parameter crosses a *quantitative* threshold. Phase transitions play a crucial role in understanding the behavior of systems far simpler than the brain (Amit 1989), and we have shown how a percolation phase transition, triggered by a quantitative increase in cortical connectivity, can endow a simple semantic associative network with a capability for latching between attractor states, offering dynamical support to infinite recursion and syntactic processes (Treves 2005). In this scenario, it is not brain weight or the number of neurons that is important, but the number of neocortical modules (related to the process of arealization; Krubitzer & Huffman 2000) and their connectivity (related to increased spine numbers; Elston 2000; Elston et al. 2001). Although the Potts model we have analyzed is certainly oversimplified, it motivates more detailed network models to assess the implication of the neocortical organization of connectivity, beyond the qualitative "small world" characterization. In fact, we have shown analytically that such connectivity results in localized auto-associative retrieval of activity patterns, thus allowing for combinatorial memory storage of composite memory patterns without the need for in-built modularity (Roudi & Treves 2004; 2005). The responsibility of carrying forward these analyses rests of course with network theorists, as does the need to make them transparent and relevant to evolutionary neurobiologists; we believe that the latter community is now ready to appreciate the import of quantitative information-theoretical analyses (Treves & Roudi 2005), and to include them into their "principles of brain evolution."

ACKNOWLEDGMENTS

We had insightful discussions with Daniele Amati and Mathew Diamond in preparation of this commentary, and partial support from Human Frontier Science Program RG P0047/2004-C.

Brain evolution by natural selection

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Abstract: *Principles of Brain Evolution* (Striedter 2005) places little emphasis on natural selection. However, one cannot fully appreciate the diversity of brains across species, nor the evolutionary processes driving such diversity, without an understanding of the effects of

natural selection. Had Striedter included more extensive discussions about natural selection, his text would have been more balanced and comprehensive.

Striedter's *Principles of Brain Evolution* (2005) is an excellent textbook on evolutionary neuroscience. In particular, I praise Striedter's effort to explicate multiple complicated principles in a well-organized fashion based on a perspective that brain evolution is governed by, not a single grand theory, but "a spidery web of interacting principles" (p. 355). The book is clearly written, avoids annoying technical jargon, and deals with many controversial issues candidly and fairly. Because of the complexity of the subject, these features are important criteria for a textbook intended for students, as well as experienced researchers, who are interested in a more in-depth discussion of brain evolution. However, it is disappointing that little emphasis has been given to the effects of natural selection on brain structures. My commentary focuses on three points specifically associated with this issue.

First, Striedter declares in Chapter 1 that the issue of natural selection is not dealt with very much in this book on account of a serious lack of empirical data and that to do so would be beyond the scope of a book focusing on neuroanatomy. Both of these arguments are reasonable and understandable. However, one cannot fully appreciate the diversity of brains across species, nor the evolutionary processes driving such diversity, without an understanding of the effects of natural selection. Striedter proposes that conservation and size variation are the core principles of brain evolution. No doubt, these are important principles; however, they are neither sufficient nor useful in order to understand some important events in vertebrate brain evolution. For example, the emergence of the mammalian-type brain, which is markedly different from the reptilian- (and avian-) type brain, was a major event in the history of vertebrate evolution (Ch. 8). Specifically, although sauropsids (reptiles and birds) and mammals evolved from a common ancestor, only mammals have a neocortex. In contrast, sauropsids have developed a large brain region called the dorsal ventricular ridge (DVR), which is not present in other vertebrates – including mammals. Why do only reptiles and birds have a DVR whereas mammals have a neocortex? The principles of conservation and size variation can offer little to explain the differences between reptilian- (avian-) and mammalian-type brains. Thus, as Striedter points out, "brain size is not everything! If it were, then same-sized bird and mammal brains should be identical, which they are definitely not" (p. 11). Extended discussion about the behavioral benefits of reptilian- and mammalian-type brains, even if only speculative based on limited empirical data, would be definitely thought-provoking and could perhaps provide new research directions for futures studies.

Second, despite his declaration at the beginning, Striedter offers short, informative discussions about species differences and adaptation on several occasions throughout the book. I would like to comment on two of the discussions – first, on the significance of the avian DVR. In Chapter 8, Striedter points out examples of "intelligent" behaviors in birds, whose DVR is larger than the DVR of reptiles. He writes that "birds apparently attained that high intelligence mainly by elaborating not their neocortex homologue (i.e., the Wulst) but the DVR" (p. 296), which suggests a correlation between the enlarged DVR and high cognitive abilities in birds. However, we should be careful before concluding a direct relationship between the elaboration of DVR and increased "intelligence" for three reasons. (As did Striedter, I will also "avoid the mire of comparative intelligence analyses" (p. 258) by trying to define "intelligence.") First, much previous research on the functions of DVR has revealed its roles in sensory processing and motor control, rather than intelligent behaviors. Evidence demonstrates that DVR is involved in sophisticated sensory processing (e.g., various types of visual discrimination in pigeons) and fine motor control (e.g., song production in songbirds). In contrast, there have been only a limited number of studies on how exactly DVR plays a role in various types of intelligent behaviors (e.g., Güntürkün 1997). Second, the avian Wulst is not necessarily a diminutive entity in many birds other than owls. The Wulst has extensive connections with other brain structures including sensory and limbic thalamic nuclei, the DVR, and the optic tectum (e.g., Shimizu et al. 1995). In addition to the DVR itself, it is likely that the Wulst may be closely involved in "intelligent" behaviors via extensive circuits with these subcortical structures. Third, Striedter supports the claustroamygdalar DVR hypothesis that the DVR is homologous to the claustroamygdalar complex of mammals. In some mammals, the thalamo-amygdala connection is associated with the information processing system for emotional responses (e.g., fear conditioning). If the claustroamygdalar DVR hypothesis is correct, we should consider the possibility that the DVR was originally, and may still be, associated with "affective" aspects of behavior.

Finally, I would like to comment on the significance of the avian hippocampus – in particular, the size differences between food-storing and non-food-storing birds. Striedter cites studies about the avian hippocampus and food storing behavior as examples of "a careful analysis of how individual brain regions have changed in absolute, proportional, and relative size, combined with a detailed functional analysis of what the region does" (p. 175). However, some researchers may not completely agree with this statement. The relationship between avian hippocampus size and spatial memory is still controversial (Bolhuis & Macphail 2001; Macphail & Bolhuis 2001). These authors reviewed related literature and pointed out that "although there have been reports (in corvids and parids) of significant positive correlations in storing species between intensity of storing and relative hippocampus size, there are some notable exceptions to the principle" (Bolhuis & Macphail 2001, p. 428). They concluded that "dependence on stored food does not, then, give a reliable guide to the relative size of a bird's hippocampus" (p. 428). Perhaps, as Striedter suggests, appropriate cladistic reconstruction of storing behavior may be necessary before further discussion is possible. Nevertheless, if Striedter had included the views of these critics, as well as other studies such as lesion data, readers of his book could have a more balanced and unbiased perspective on these issues.

Despite the points discussed above, Striedter's book is highly recommended to students and researchers alike. It provides remarkably clear viewpoints on two sides of brain evolution: similarities and differences in brain organization across species. His book will surely encourage new research that should elucidate both sides of brain evolution.

ACKNOWLEDGMENTS

Preparation of this commentary was supported in part by a research grant from National Science Foundation (IBN-0091869) and an Established Researcher Grant from the University of South Florida (R0-13666).

Brain design: The evolution of brains

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Abstract: After reviewing historical aspects of brain evolution, this accessible book provides an enjoyable overview of several general principles of brain evolution, culminating in discussions of mammalian and human brains and a framework for future research.

From the birth of comparative neuroanatomy in ancient Greece, to efforts to describe brain evolution in the first half of the

twentieth century, investigators have worked with limited data. With modern techniques, investigators have amassed detailed information on the macroscopic as well as microscopic brain organization, the electrophysiological and biochemical *in vivo* functioning of a broad range of species of extant vertebrate brains. This has led to systematic approaches for reconstructing evolutionary history species, and theories of brain evolution have been enriched by historical data on brain sizes and shapes from the an accumulating fossil record. Georg Striedter has produced a fascinating book (Striedter 2005) that discusses current understandings of brain evolution, focusing on certain recent advances.

Striedter provides a survey of brain evolution through general principles that organize the data now available and provide a guide to the interpretation of new observations. For example, certain brain features have been conserved from distant ancestors across members of a taxonomic group. Of course, the degree of similarity across members of a group is dependant upon how much evolutionary time has passed. Thus, the thalamus may be comprised of different nuclei depending on evolutionary stage: one for fish and amphibians, and many for mammals. This raises the question of how certain structures form by possibly having new structures added onto old ones. Alternatively, new structures may emerge in development as duplications of old structures which have provided some survival value, as a result of changes in gene expression and gene duplication. Another example of the means by which evolution may act on brain evolution is found in the lateral geniculate nucleus of the visual thalamus that has been conserved in all mammals. It varies in laminar pattern and may confer binocularity of vision in more advanced mammals – so particular ways of organizing structures may be another end of evolution, by conferring survival advantages. Besides the organization of discrete structures, Striedter discusses how connectivity also varies across animals of different evolutionary stages – such as with the hippocampus, which may be increasingly complex across evolution. Many of these hypotheses, however, are also fraught with exceptions such as with the hippocampus, which appears to be as highly organized and interconnected in avians as in mammals.

An interesting apparent general rule is that as bigger brains evolve, "late makes large," that is, structures that develop late in embryonic development become disproportionately large in bigger brains. Thus, humans have much bigger forebrains relative to their brainstem than do rats. This rule is frequently modulated by adaptive enlargement of useful brain parts: so-called mosaic evolution. For example, because of mosaic evolution, the superior colliculus might be ten times larger in a highly visual rodent than in a weakly visual rodent, even though their brains are the same size. However, most deviations from the rule are much smaller.

Brain size influences brain organization in another way as well. As neurons do not change very much in cell body size, large brains have many more neurons than small brains. This usually means that neurons in bigger brains do not connect with the same proportion of other neurons. In addition, as brains get bigger, connection lengths between neurons, as well as transmission times, get longer, and more of the brain must be devoted to connections unless brain organization is modified. Taken together, this means that large brains require a design different from small brains. Such design problems were addressed in evolution by increasing the number of brain parts and emphasizing local over distributed processing and efficient "highways" of white matter.

Although Striedter highlights the many conserved features between our presumed primate ancestors and humans, he also reviews the significant reorganization that our large human brains have recently undergone. There is, of course, much evidence for this from comparative studies of monkey and human brains. Most notably, human brains are structurally and functionally much less symmetrical, with the right and left cerebral hemispheres differently specialized. This reduces the need for ancestral proportions of the long connections between the hemispheres. Perhaps a future edition could include sections on how neuro- and psychopathology might be understood in an evolutionary light. For example, why humans are more likely to suffer certain illnesses or be protected from others as a result of evolutionary forces; and how evolution may have also solved certain predispositions to illness. It might be particularly interesting to add discussions of the parallels and impact of cultural evolution on brain function of recent millennia.

A range of readers would enjoy this book, from undergraduate to advanced students. Only a basic understanding of neuroanatomy is needed to understand the text. Experts will also find the book well written, interesting, and informative; even if they might disagree with some of the statements and interpretations which, Striedter admits, are often controversial. He concludes with encouragements to colleagues and future researchers to provide more data and affirm or strike down current ideas.

Author's Response

Evolutionary neuroscience: Limitations and prospects

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Abstract: Overall, most of the reviewers agree that *Principles of Brain Evolution* was a welcome addition to the field, and kindly describe it as carefully researched and lucidly written. Thereafter, they note some gaps – principally, adaptive scenarios, microevolutionary studies, and computational models. I here admit to those deficiencies but explain why they exist and how they might be filled. In addition, one commentator criticizes my analysis of hominin brain evolution, and another finds my principle of "large equals well-connected" to be inconsistent with the data. I rebut those two critiques. Hopefully, this process of critique and counterpoint will stimulate some readers to pursue the mentioned thoughts and to engage in new research.

R1. Introduction

Nerve wracking as it is to have one's writing scrutinized by experts who are instructed by the journal to provide "substantive criticism," I am grateful that my *Principles of Brain Evolution* (Striedter 2005) has received such careful attention. Back when I wrote the book, I often wondered how readers would respond to my opinions, strategic decisions, and omissions. Now I know, at least to some extent, and so do you. Moreover, I can now share my response.

Thankfully, most reviewers agreed that the book is valuable, not only as a text for specialists, but also as a vehicle for reducing what **Barton** calls the "ghettoization of evolutionary approaches" in modern neuroscience. This was indeed one of my major aims. I am also relieved that none of the commentators took me to task for placing so

much emphasis on absolute, rather than relative, brain size. I was nervous about this emphasis because, as **Dunbar** notes, comparative neurobiologists have traditionally been "dragooned into worrying about relativizing brain size by a very peculiar view that body size must be the default determinant of brain volume." Given that historical context, it was nice to read **Hilgetag**'s assent that "increases in absolute brain size matter, and have brought with them changes in connectivity as well as greater brain complexity and modularization." Similarly satisfying was **Gilissen**'s analysis of corpus callosum scaling, which supports my suggestion that large-brained cetaceans evolved some novel ways to cope with network scaling costs. Thus, two of my main concerns – addressing the right audience and rehabilitating absolute brain size as an important variable – are allayed. Of course, several other concerns remain, and they are highlighted in the commentaries. Below, I address those concerns, beginning with the most severe.

R2. Natural selection

Adkins-Regan and **Shimizu** both lament that my book included little on the role that natural selection played in shaping brains. They wonder whether I am generally uncomfortable with the concept of adaptation and regret that I discussed far more extensively the "how" of vertebrate brain evolution than its "why." Adkins-Regan, in particular, points out that other areas of evolutionary biology abound with "studies of wild populations to track changes in gene frequencies and their measurable phenotypic consequences in response to changing selection pressures." Why, she asks, are such studies absent from evolutionary neuroscience or, at least, from my account? She admits that studying natural selection in the wild for brains is difficult, but argues that it should be no more difficult than studying any other "complex and plastic aspect of a phenotype." Moreover, Adkins-Regan complains that I seem rather pessimistic about ever learning much about the role that natural selection played (or currently plays) in the evolution of brains. I have three responses to this challenging critique.

First, I feel compelled to state that my reticence on natural selection was deliberate. Although I personally believe that natural selection played a crucial role in shaping most neuronal attributes, proving this is difficult, mainly because neuronal attributes are hard to see in living animals, tough to manipulate experimentally, and difficult to link to specific behaviors. Therefore, most previously published (or uttered in safer venues) statements about natural selection and the brain are quite speculative and suspect. In my opinion, such excessively adaptationist just-so-stories (Gould & Lewontin 1979) do not advance the cause of evolutionary neuroscience, because they reinforce the already too common view that comparative neurobiology is not a very scientific field. In order to combat that perception, I intentionally minimized my speculations about natural selection and, instead, emphasized other principles of brain evolution. However, as **Shimizu** notes, I did let several ideas on neural adaptation creep into the book, especially into the chapters on human and mammalian brains. Again, this was deliberate. It is worth noting, though, that those adaptational hypotheses elicited some strong objections from at least some commentators (notably **Dunbar**). Such disagreements can be productive, but I did not want them to obscure my basic point, which is that evolutionary neuroscience need not be a speculative enterprise full of interminable debates (see also Striedter 1998a).

Second, I did in the book discuss some relatively solid work on natural selection and the brain. For example, I discussed extensively the data showing that hippocampus size in birds correlates with food storing behavior, suggesting that hippocampal enlargement in those species is an adaptation for food storing. Those correlative data have been criticized (Brodin & Lundborg 2003), but a recent analysis supports the adaptational hypothesis (Lucas et al. 2004). Nonetheless, **Shimizu** notes that some authors have serious objections to this kind of "neuroecological" research. Specifically, Bolhuis and Macphail (2001) have argued that knowing how selection shaped a brain region tells you nothing about how that structure works and that, therefore, evolutionary considerations "cannot explain the neural mechanisms of behaviour" (p. 432). To the extent that natural selection can find multiple neuronal solutions to particular behavioral problems (a still open, empirical question), that critique has some merit. However, I find it unproductive to erect such rigid barriers between "how" and "why" questions. Purely mechanistic studies can tell us how a feature works, but they cannot tell us why it works the way it does. For that we need to know the feature's developmental and evolutionary history. Conversely, correlations between neural features and behavior (or ecology) are difficult to interpret unless we have some mechanistic understanding of what the neural features do. Indeed, the latter insight may explain why Bolhuis and Macphail (2001) focused their critique on cognitively complex traits, specifically learning and memory, whose neural substrates are tough to nail down.

Third, my "pessimism" about the study of neuronal adaptation is not as severe as **Adkins-Regan** intimates. In fact, my collaborators and I have performed an experiment that qualifies, I think, as neuroecological. Specifically, we have examined how lesions of a small brain area necessary for vocal imitation in male parrots affect the ability of the lesioned males to find female mates (Hile et al. 2005). We found that the imitation-impaired males had no trouble finding mates initially, but later were more likely to be cuckolded. This study was labor intensive, but had we wanted to make strong statements about natural selection acting on the lesioned vocal control region, we would have had to show also that variation in the brain region's size or structure occurs naturally, is heritable, and has measurable effects on a male's reproductive success. That kind of work is doable, given sufficient resources, but it is extremely arduous. Therefore, I feel that, on the topic of natural selection and the brain, I am not a pessimist but, as the saying goes, an optimist with experience.

R3. Micro- versus macroevolution

Adkins-Regan also bemoans that my book dealt at length with macroevolution but said little about microevolution, loosely defined as evolutionary changes that occurred over relatively short periods of time and, therefore, between

close relatives. Although the other commentators in this issue do not echo this concern, Airey and Collins (2005) did bring it up in their review, published in *Genes, Brain and Behavior*. Therefore, I take this concern quite seriously.

Generally speaking, microevolutionary studies are useful because closely related species (or populations within a species) tend to differ in fewer respects than distant relatives. This high similarity facilitates discovery of correlations between neuronal and behavioral features. It also simplifies the task of "re-creating" in experimental animals the hypothesized changes in brain organization and testing whether these laboratory "monsters" exhibit the expected behavioral phenotype. All those advantages are beautifully illustrated in the recent work on voles, which **Adkins-Regan** properly highlights. The most recent of these studies, which appeared after my book had been composed, showed that increasing the expression level of vasopressin receptors in the ventral forebrain of voles can transform one species into another, at least with regard to some aspects of social behavior (Lim et al. 2004). Such experimental re-creations of real evolutionary change are powerful because they are, in essence, strong experimental tests of evolutionary hypotheses. If I ever write a second edition, I will certainly review such microevolutionary studies more extensively.

Currently, however, microevolutionary "model systems" are quite rare in neuroscience, and most of the existing work does not include experimental re-creations of real species or individual differences (but see Balaban et al. 1988). For example, the research on the relationship between avian hippocampus size and food storing is fairly microevolutionary, but no one has tested experimentally whether having a larger hippocampus makes a bird more capable of storing and retrieving food. Similarly, there are numerous comparative studies on the avian song system, but none convert a species with innate vocalizations into one that learns its songs (though several studies have shown that selective brain lesions impair song learning). Such experiments are currently not feasible, mainly because the evolution of song learning surely involved complex changes in developmental gene expression (i.e., changes more complex than those that altered the social behavior of voles). Some day, however, we may know enough about the differences in brain development between songbirds and non-songbirds to "transform" one into the other. Further advances in evo-devo neuroscience will surely hasten the arrival of that day.

Yet another reason why my book includes scant coverage of microevolutionary brain research is that I thought my target audience would have only limited patience for disquisitions on diverse and often obscure birds, fishes, or, for that matter, voles. Because the majority of neuroscientists, psychologists, and anthropologists tend, in my experience, to be most keen to learn about the human brain, I chose primate brains as the microevolutionary "model system" in my book. This choice was less than ideal because hypotheses about primate brain evolution will always be relatively difficult to test, if only because many crucial primate species are already endangered or extinct. Again, however, I am not entirely pessimistic. Many excellent comparative studies on primate visual systems have already been published (e.g., Kaskan et al. 2005; Rosa & Tweedale 2005) and, as **Clancy** points out, good work is underway to reveal how evolution tweaked development to adapt primate retinas to nocturnal or diurnal niches. Finally, we have good data on brain variability in at least one primate, namely *H. sapiens* (e.g., Andrews et al. 1997), which should make it easier to decipher the rules that underpin brain variability in primates generally.

This last point deserves elaboration. Considerable amounts of data on brain region variability in humans and in laboratory mice have been published (e.g., Seecharan et al. 2003) but, beyond those two species, data are scarce. Furthermore, the available data are solely volumetric. Because the methods currently used to trace neuronal connections require the sampling of many individuals to obtain a species-typical result, we know essentially nothing about intraspecific variation in neuronal connections. In order to overcome this limitation, we would need a novel tracing method that consistently (from case to case) stains the axons of all neurons in an identified population of cells. As far as I know, such a method has not yet been described, but I think that its essential elements already exist (Mombaerts et al. 1996; Soriano 1999; Yu et al. 2005). Hopefully, some clever person will combine those elements, for then we could begin to study how neuronal connections vary within a species, how heritable that variation is, and how it correlates with differences in brain physiology and organismal behavior. Furthermore, we could compare intraspecies variation with interspecies differences and debate whether macroevolutionary changes are merely extensions of microevolutionary trends. In the absence of data, however, such discussions would be premature.

R4. Computational neuroscience

Roudi & Treves, as well as **Granger** and **Raffone & Brase**, point out that I wrote little on the role of computational models in helping to explain the functional significance of evolutionary changes in brain organization. Indeed, this is an important role. For years, I have been intrigued by the unusual interest that computational neuroscientists tend to express in comparative neuroanatomy (perhaps because their daily work deals with network structure/function relationships). I, in turn, have long been fascinated by the potential of neural network models to provide a level of analysis between circuit structure and behavior. The problem is that correlations between brain structure and behavior (e.g., hippocampus size and food storing behavior) tempt us to conclude that the structure causes the behavior. This view is simplistic, of course, because many structures may collaborate to generate a specific behavior, while any one structure is probably involved in numerous behaviors (which, by the way, is another serious obstacle to demonstrating adaptation in the nervous system). Therefore, what we really need to know is this: what computations does the structure in question perform and what do those computations contribute to the behavior(s) of interest? Furthermore, we want to know how variations in neuronal structure change those computations, and how variations in the computations affect behavioral performance or capacity. Network models can, at least potentially, help to answer those questions.

For example, **Shimizu** asks in his commentary what behavioral benefits early mammals derived from evolving a six-layered neocortex. I offered no detailed answer in my book, except to say that the columnar organization of the neocortex allows for finer, more expandable topographic maps than you can have in a reptilian cortex. **Roudi & Treves**, in their commentary, point out that computational studies suggest a deeper, more detailed answer. Specifically, Treves (2003) has compared the computational abilities of a simple non-laminated thalamocortical network with one that contains a two-layered cortex and found that the laminated cortex was better at encoding where (on the receptor array) a stimulus was presented, while simultaneously computing (based on learned information) the identity of the presented stimulus. This makes sense to me. Moreover, if the hypothesis is true, then we would expect the evolution of mammalian neocortex to correlate not just with a single behavior, but with a whole slew of different behaviors (especially if it applies not only to sensory cortex, but also to "association" cortices), as it probably does. Thus, the computational level can bridge the structural and behavioral levels of analysis. Few such bridges now exist, but they are well worth constructing.

Shimizu also asks about the benefits of birds evolving a large and complex dorsal ventricular ridge (DVR) (the likely homolog of the mammalian claustroamygdalar complex). He recommends we be cautious about accepting my proposal that birds became "more intelligent" as they built out their DVR because the DVR is not the only enlarged forebrain region in most birds, and because the DVR supports a number of sensory and motor functions that are not obviously intelligent. Both concerns are reasonable. However, I think that the learning and use of complex songs is an intelligent behavior that clearly is (as Shimizu acknowledges) dependent on the DVR. Furthermore, Timmermans et al. (2000) have shown that the size of one major DVR component correlates quite well with "feeding innovation rate," which I consider to be a good measure of intelligence. Of course, such correlations cannot tell us *how* the elaboration of the DVR facilitates intelligent behavior; they only suggest the presence of a causal link. Personally, I think that the structural elaboration of the DVR probably provided birds with computational powers that are analogous, at least in part, to the computational advantages mammals derived from evolving their neocortex. In order to test this hypothesis, one would need to have some computational models of the DVR in both its simple, reptilian form and in its complex, avian design. To my knowledge, such models are not yet available.

As the preceding paragraph suggests, the main difficulty one encounters in attempting to explain brain evolution at the computational level of analysis is that computational models of real differences in neural circuitry are exceedingly rare. Moreover, the published models are themselves "evolving" (see Treves 2004) and subject to a number of untested assumptions. Given those uncertainties, I decided in my book to shy away from discussing any model specifics (which are generally beyond my grasp in any case) and instead to emphasize some general computational principles. In particular, I focused on some principles of network scaling that have recently attracted a great deal of general interest. For example, we have learned that many real networks, including the internet, grow by preferentially connecting new nodes to old nodes that already have a lot of connections (Barabâsi 2003). Brains, in contrast, do not scale like that. Instead, they adopt a small-world topology that keeps most connections very short but interconnects disparate clusters by long-range "short-cuts" (Watts & Strogatz 1998). **Hilgetag** rightly points out that the small-world designation fails to capture all of the complexity of brains, which he describes "clusters of clusters." However, even his more complex characterization leaves open the possibility that brains are small-world networks whose main nodes are even smaller worlds. Be that as it may, contemplation of these issues serves at least to focus our attention on the network design principles that govern how brains function and evolve. That attention is quite new in evolutionary neuroscience, and I welcome it.

R5. Hominin brain evolution

The commentaries reveal little dissent on my description of hominin brain evolution as having occurred in fits and starts. Nor do they question my central proposition, which is that increases in hominin brain size entailed some forced changes in the brain's internal organization. Instead, the commentaries focus on the question of why hominin brains evolved in the manner they did. What were the functional correlates of changing hominin brain size and organization? What selective pressures drove hominin brains to change?

Dunbar states that I was wrong to claim that early *H. erectus* "probably experienced strong coordinated selection for both larger brains and better dietary quality" (p. 318). In Dunbar's view, "energetic costs impose a developmental constraint that has to be solved, but they do not provide a reason why brains should increase in size." I respectfully disagree. If an evolutionary increase in brain size facilitates the invention of food procurement and/or preparation strategies that *more than* pay for the metabolic costs of having that larger brain, then large-brained individuals would be selected for when food is scarce. Moreover, if the mental capacities that allowed hominins to have a more nutritious diet (e.g., an improved ability to innovate) also enhanced their social skills, then the social intelligence hypothesis, which Dunbar favors, becomes entangled with hypotheses related to changing diet (Parker & Gibson 1977). In fact, one game theoretical analysis has shown that a combination of ecological *and* social selective pressures would give you precisely the sort of nonlinear trajectory in absolute brain size that hominins apparently exhibited (Rose 1980). This idea requires elaboration, but it should caution us against erecting strict dichotomies between the dietary and social intelligence hypotheses or, more generally, between constraint and selection.

Dunbar's second criticism is that human language could not have evolved "as a simple byproduct of a large brain," because whales and elephants also have large brains but lack language. True, but my argument was more nuanced. Specifically, I argued that "there might have been no way to make a *primate* brain as large as ours without also endowing it with most of our brain's organizational features" (emphasis added) (p. 343, Striedter 2005). As I discussed extensively, all primate brains differ from other mammalian

brains in many ways other than size. Therefore, it would be silly to claim that whale or elephant brains mimic all, or even most, of the structural and functional details of human brains. Furthermore, I explicitly dismissed the so-called "Rubicon models" of human language evolution, which claim that language appeared after some brain size threshold was reached. **Roudi & Treves** accurately point out that Rubicon models are not entirely implausible, for some networks exhibit interesting emergent properties when they exceed a certain size, but I continue to be skeptical of Rubicon models as long as they view language evolution as a *simple* byproduct of humans having a large brain. As I stated in the book, the phylogenetic increase in hominin brain size probably led to a slew of changes in the brain's internal organization, some of which then allowed language to evolve. Furthermore, I never claimed that symbolic language sprang fully formed from any one hominin's brow. It almost certainly evolved more gradually.

For me, the crucial question is: which size-related changes in hominin brain organization are most directly linked to language emergence? In the book I pointed out that the evolution of more direct connections from the neocortex to the medulla and spinal cord probably enabled the fine motor control that is essential for human speech. I also stressed that the expansion of the human lateral prefrontal cortex probably increased our capacity for "response inhibition," which I tentatively linked to the transition from signal-based communication to symbolic language. I did mention, however, that there are other views of lateral prefrontal cortical function, some of which are highlighted in the commentaries. **Pascual-Leone**, for example, stresses the prefrontal cortex's role in working memory and argues that its expansion in the hominins increased their attentional bandwidth. **Raffone & Brase** also underline the role of the lateral prefrontal cortex and its associates in working memory. They propose that its expansion in hominins enabled the formation of more stable "reverberatory states in working memory, planning, goal representation, and effect anticipation." I do not disagree with these accounts and they may well, as Raffone & Brase point out, be complementary to mine. *A priori*, it is likely that a structure as complex as the lateral prefrontal cortex performs a variety of different computational functions, which in turn play a role in many different behaviors.

Overall, the commentaries show that human brain evolution remains a highly charged subject. The reasons for that are multiple. First, most of our closest relatives are either on the brink of extinction or already gone. Second, the behaviors that set humans apart, notably symbolic language and inventiveness, are extremely difficult to explain in neural terms. Third, the brain regions that are most likely to be involved in the control of those behaviors (e.g., the prefrontal cortex) are among the most complex. Future modeling studies, both of the computational and the ecological variety, should help to resolve some of those debates. In the meantime, however, partial and vague accounts will have to do. Even so, I hope that my account will stimulate some new research and be a valuable teaching tool. I was happy to read, therefore, that **Clancy** expects it to be useful for teaching students who were raised to think that humans appeared suddenly and fully formed roughly 10,000 years ago. However controversial my take on human brain evolution may be, it surely is less fanciful than the creationist alternatives.

R6. Evo-devo neuroscience

None of the commentators object to my approaching the problem of brain evolution from a pluralistic perspective, which stresses that, as **Hilgetag** puts it, "brains evolved not just under one structural or functional constraint, but had to accommodate multiple, partly opposing requirements." I did not expect such broad agreement on this point (or so little disagreement), because the history of evolutionary neuroscience is replete with arguments for and against specific principles that were once hailed as being the one, true law of brain evolution (e.g., Ebbesson 1984). Of course, the field continues to harbor debates, but those now tend to focus on the relative importance of the various brain evolution principles, on their generality, and on their underlying mechanisms. For example, **Clancy** and **Barton** come down on opposite sides of the dispute about concerted versus mosaic evolution, but both accept at least the possibility that these two modes of evolution coexist.

Barton does, however, criticize my contention that mosaic evolution is relatively rare. He agrees that "comparisons of relatively closely related species show differences in relative brain components size that are generally within a 2- to 3-fold range, whereas comparisons of more distantly related species reveal larger differences" and adds that this is "the pattern that one would expect to see under a gradualist model of evolution." I agree. However, Barton goes on to claim that, therefore, I was wrong to claim that mosaic evolution (larger than 2- to 3-fold deviations) is less frequent than concerted evolution. This conclusion seems illogical or incomplete. To my mind, the observation that large deviations from a brain region's expected size are generally found only in comparisons of distant relatives implies that those "severely mosaic" deviations require long periods of evolution to emerge. This interpretation is supported by my observation that, in at least some instances, phylogenetically intermediate species exhibit brain region proportions that bridge the gap between the distant relatives. In contrast, the available data suggest that large changes in absolute brain size, involving numerous concerted changes in brain region size, can occur more rapidly. Therefore, I conclude that, in any given lineage, severely mosaic evolution is less frequent than concerted change. Of course, as I discussed, both modes of evolutionary change play important, if different, roles in the phylogeny of brains.

Barton also touches on two other issues that I think are crucial for resolving the mosaic/concerted evolution debate. The first is that the available comparative data on neuronal birthdates do not perfectly obey late equals large (Barton 2001). **Clancy** responds that the rule is indeed not hard and fast, yet allows some useful predictions. An interesting alternative is that the deviations might be due to problems with the birthdating data, which were gathered by many different investigators, using diverse methodologies. Perhaps the late equals large rule is actually tighter than the currently available data suggest! The second issue Barton mentions is that we have virtually no comparative data on developmental parameters other than the timing of neurogenesis. The size of early embryonic precursor regions may differ across species, or they might be conserved; we simply do not know. Similarly, a region's rate(s) of cell division

might differ across species, but we have virtually no data relevant to that hypothesis. In other words, we still know very little about the developmental constraints or variables that are important in controlling the size of brain regions as they evolve. In my laboratory, we are beginning to study these factors in embryonic birds (Striedter et al. 2005).

Yet another issue **Barton** mentions is that brain regions might evolve concertedly because of "functional constraints," such as the one proposed by Stevens (2001) to explain the allometric scaling of visual cortex and thalamus. Unfortunately, such functional constraints are difficult to discover, comprehend, and test, mainly because we know so little about the computations being performed by the brain regions of interest. Another kind of functional constraint is that imposed by trophic dependencies between interconnected brain regions. For example, a phylogenetic decrease in retina size might cause a whole "epigenetic cascade" of changes in the size of other visual system structures. As I pointed out, however, the divergent/convergent nature of most neuronal circuits, as well as regional variability in the degree of trophic dependence, tends to dampen or buffer most of those cascades (see also Finlay et al. 1987). **Provine**'s commentary underlines this point, since he reports that peripherally induced spinal motor neuron loss does not cause the upstream spinal interneurons to be lost. What do the rescued interneurons do? Provine suggests they may subserve "obsolete motor patterns." Similar ideas have been discussed before (Kavanau 1987) but they have never, to my knowledge, been examined with rigor. Provine's alternative suggestion, that the rescued interneurons change targets, also has only limited empirical support (Wainwright 2002). Therefore, it seems fair to state that the role of functional constraints in brain evolution remains plausible but scarcely examined.

One of my favorite evo-devo principles is Deacon's displacement hypothesis (Deacon 1990b), which I have dubbed "large equals well-connected." **Hilgetag** reports that his attempt to test this hypothesis, which he understood to predict that "larger structures should send and receive more and denser projections," produced negative results. I disagree with this conclusion because, in my conception, the principle of large equals well-connected applies to interindividual, not intraindividual, comparisons. Specifically, I wrote that "in evolution, when a brain region becomes disproportionately large, it tends to invade novel targets and receive some novel inputs. Conversely, when a brain region becomes disproportionately small, some of its inputs and outputs may be lost" (p. 237 of my book). According to this definition, the principle of large equals well-connected does not guarantee that, after many years of evolution, proportionately large brain regions will have more connections than proportionately small regions in the same individual, because the various brain regions likely began their evolutionary history from different starting points, with different sets of connections. Therefore, if we really want to test the principle of large equals well-connected, we must compare the connections of *homologous* brain regions that have changed their size relative to other brain regions. Such tests are more difficult than the test Hilgetag performed because we need data from a range of different, carefully selected species. However, as I tried to demonstrate, they are hardly impossible.

Hilgetag also suggests that my review of putative brain evolution principles is incomplete. Yes, it probably is. However, it is no easy task to find those other principles. For example, Hilgetag mentions the idea of Barbas (1986) that, as Hilgetag puts it, "more-clearly laminated areas project mainly from their superficial layers into the deep layers of areas with less-pronounced lamination, and vice versa." This is indeed an interesting idea with good empirical support. However, the tests of Barbas's principle that Hilgetag cites all deal with intra-individual comparisons, not with phylogenetic change. Maybe cortical areas do systematically change the laminar origins and targets of their intracortical connections as they become more or less laminar in evolution, but I know of no evidence to support (or contradict) this hypothesis. The necessary work has not been done. Furthermore, it still remains unclear what developmental mechanisms (or functional constraints) might underlie Barbas's principle, either in its original intra-individual version or in the modified, phylogenetic form that Hilgetag implies.

This last point bears emphasis. I focused the book on the twin principles of late equals large and large equals well-connected because we have at least a partial understanding of the mechanisms that could underpin those principles. In the case of late equals large, we know that a phylogenetic stretching of neurogenetic schedules should cause late-born structures to become disproportionately large. In the case of large equals well-connected, we know that activity-dependent competition between developing axons should cause phylogenetically enlarged areas to retain more diverse connections. Having mechanistic explanations for these principles increases my confidence in the principles' existence, but it does not guarantee their generality, because neurogenetic schedules might sometimes be rearranged rather than stretched, and developing axons may not always compete (or compete on tilted playing fields). In the long run, we need both: good comparative data to show which brain evolution principles apply reliably (whenever specific conditions are met) *and* a solid understanding of the mechanisms that cause those principles to manifest.

R7. Clinical relevance

Swain in his commentary hopes that future editions of my book will "include sections on how neuro- and psychopathology might be understood in an evolutionary light." Indeed, clinical relevance is an important issue, because if one could demonstrate to clinically inclined neurologists that evolutionary theory is useful in their work, then evolutionary neuroscience would become a much more vibrant, better-funded field. Unfortunately, clinical neurology and evolutionary neuroscience have thus far remained worlds apart.

Back in the nineteenth century, Hughlings Jackson did attempt to integrate evolutionary and clinical concepts by arguing that the brain is organized from low to high, that this neuronal hierarchy parallels the brain's phylogeny, and that damage to the brain's highest regions causes the whole system to rely more on the lower, phylogenetically older brain regions (see Hughlings Jackson 1958). This synopsis makes it seem as if Hughlings Jackson thought decorticate mammals act like early reptiles or

amphibians, which lacked neocortex, but he realized that neocortical lesions cause a complex mix of what he called negative and positive symptoms. The former are due to damage in the neocortex, whereas the latter are caused by the removal of the normal cortical projections to the lower brain regions. In other words, Hughlings Jackson realized that cortical damage can cause not only paralysis but also the appearance of abnormal new behaviors, such as the Babinski sign, which is the replacement of the normal plantar (toe flexor) reflex by an abnormal toe extensor reflex. These insights were profound and established Hughlings Jackson as a founding father of neurology. However, in my opinion he failed to explain neuropathology and/or psychopathology in evolutionary terms. After all, can the Babinski sign, or the post-lesion emergence of other abnormal reflexes, really be explained as the unveiling of ancestral behaviors? I do not think so and, as far as I know, Hughlings Jackson himself never offered any explicitly phylogenetic explanations for the symptoms he described.

This does not mean that evolutionary insights can never be useful. I am intrigued, for example, by Temple Grandin's finding that the minds of high-performing autistics, such as herself, resemble "animal minds" in being highly visual and detail-oriented, rather than abstract (Grandin & Johnson 2005). Grandin does not claim that autistic people are atavistic throwbacks to some earlier phylogenetic stage. Instead, she states that autism reveals a nonverbal mode of thinking that is common to most animals (notably mammals and birds) but is frequently hidden beneath a swirl of abstract thoughts in non-autistic folk. Grandin also does not claim autistic minds to be exactly like the minds of nonhumans; instead, she says their minds are similar. Grandin's analogy (homology?) remains poorly defined but it has given her profound insights into animal minds, which in turn has improved farm animal welfare (Grandin & Johnson 2005). Conversely, Grandin's insights into animal thinking have modified our understanding of autistic minds, which has improved autistic lives. Grandin herself is living proof, I think, of the analogy's merit.

Finally, it must be said that evolutionary theory can impact medical science by highlighting that species differences exist and complicate extrapolations from model systems to humans. As far as I can tell (but I have not studied this issue thoroughly), drugs or other therapies often work well in nonhumans but are much less effective or harmful in humans. Conversely, some drugs that work well in humans are ineffective or injurious in some nonhuman species. The biomedical community tends to regard these species differences as "noise" that is both unavoidable and inexplicable, whereas animal rights advocates are predisposed to see them as clear proof that animal research wastes time, money, and lives (Greek & Greek 2000). Evolutionary thinking opens up a middle ground between those two extremes, because it implies that species similarities and differences are both bound to exist. More important, evolutionary analyses allow us to develop rational strategies for deciding which model systems are best suited to what purposes. Finally, the evolutionary approach may guide some biomedical research. We may inquire, for example, why species differ in their ability to repair damaged brains, or why humans are exceptionally susceptible to neurodegenerative diseases. Discovering the basis of those species differences may lead to novel therapies. To date, however, comparative neurology is a barely existent field (even though the *Journal of Comparative Neurology* is an extremely venerable, influential publication). Hopefully the field will grow.

R8. Conclusion

For me, the most interesting aspect of evolutionary neuroscience is that it attempts to synthesize many different strands of thought from a variety of disciplines. It has been nice, therefore, to see my book elicit comments from respected representatives of many different fields, including behavioral ecology, computer science, anthropology, neurology, and my own intellectual home, comparative neuroanatomy. Molecular neurobiology is noticeably absent from this list. In part this merely reflects the readership of *BBS*, which seems to prefer cognition to molecules, but it may also reflect the fact that my book was relatively mute on the genes that generate those wonderfully diverse brains. I suspect that any future edition would include much more on that molecular machinery. In any case, my book appears to have accomplished two of my major aims: to make evolutionary neuroscience palatable to a broad audience of scientists, and to stimulate debates about the field's shortcomings and possibilities. Those aims are well reflected in **Granger**'s statement that "the present decade seems to be one in which the profusion of data from fields of biology, behavior, and computation is beginning to cohere into theory." Hopefully my book and this multiple book review will serve to further that emerging theory.

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