The evolution of brain size and organization in vertebrates. A program for research

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In vertebrates, brain size variability relates to two main parameters: body size and ecological factors (in particular diet and foraging strategy). It has been considered by many authors that evolutionary brain growth is a unitary phenomenon whose main effect is to increase processing capacity. Alternatively, in this paper it is considered that brain growth is significantly associated with higher processing capacity only when it occurs associated with ecological circumstances (selection of behavioral or perceptual skills). This process is referred to as "active" growth. When the brain scales on body size, there is little change in processing capacity, and this will be referred to as "passive" growth. I propose that these two modes of phylogenetic brain growth relate to different developmental/evolutionary processes and are distinguishable at the level of adult and developing structure. Shortly, growth due to selection of behavioral capacities is associated with more differentiated brains in terms of number of areas, connectional rearrangements and cell types. Growth due to scaling of body mass produces little brain rearrangements, and many of those that occur relate to the maintenance of functions in a larger brain. In addition, active selection of brain size is triggered by plastic, ontogenic rearrangements of connectivity in the organisms, while passive growth produces the minor rearrangements that take place. Finally, I propose a research program oriented to test this model by separating the effects of body size and ecological variables in brain organization across species.

Key words: *allometry, brain size, ecology, evolution, vertebrates.*

INTRODUCTION

There is a wide diversity of brain size across vertebrates, most of it determined by body weight (explaining more than 90% of the variance in mammals; Harvey and Krebs, 1990). In order to eliminate the effect of the latter, an index of relative brain size referred to as encephalization has been developed (Jerison, 1973; Bauchot, 1978; Hofman, 1989). For example, relative to body mass, brain size is the smallest in agnathans (lampreys and myxinoids), increases in bony fish, amphibians and reptiles, and reaches its highest values in birds and mammals (homeotherms). Humans have the largest relative brain weight, closely followed by dolphins (Jerison, 1973, 1991; Northcutt, 1981). This has led several authors to propose that encephalization closely relates to cognitive capacity across species (Jerison, 1973, 1991; Bauchot, 1978; Hofman, 1982a,b).

The question arises of why body size is such a good determinant of brain weight. According to Jerison (1973, 1991) and others (for example, Hofman, 1982a,b), a larger body needs a larger brain in order to control

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an increased number of cells. Therefore, increase in brain size always results in enhanced processing capacity that may be used either in body control or in cognitive skills. In fact, Jerison (1991) claims that the "amount of information processed per unit volume [is] constant across species" (p. 35), which means that processing capacity increases with volume, regardless of how this brain volume has been acquired.

This view is difficult to reconcile with the fact that structures not related to body control, like visual and higher telencephalic components also scale with body size (Stephan and Frahm, 1984; Fox and Wilczynski, 1986). It is conceivable that for example, spinal motor circuits, the autonomic system and hypothalamic structures producing neurohormones will scale for functional reasons, but the whole brain is quite a different story. A perhaps better interpretation is that if the body increases its number of cells it also increases the cells of the brain as a consequence (Gould, 1975, 1977; Riska and Atchley, 1985). In other words, the body makes the brain to grow because the two are developmentally coupled. This still leaves open the question of whether these additional nerve cells have an effect on the brain's capacity. Gould (1977) argues that in evolution, one way to develop intelligence is to increase body size, that consequently produces a brain size increase. Although there may be something to this argument (see below), it implies that brain size and intelligence are closely related.

My approach will be different. I will argue that in evolution, when brain size increases by following body growth, this does not imply by itself significantly enhanced neural capacity. This is because rearrangements in connectivity and brain organization are very limited during this process, which will be referred to as passive evolutionary brain growth. On the other hand, if there is selection of increased behavioral or perceptual capacity, connectional rearrangements will be quite important both as a plastic ontogenic response and as a result of selection of more specific neural circuits. Brain size in this case works mainly in helping those rearrangements to occur. In this way, neural reorganization is proposed as the main event relating to higher processing skills. This pattern of phylogenetic brain growth is referred to as the active mode.

In this paper I will present evidence supporting the above view, and will offer an evolutionary scenario for increase in brain size in the two different modalities presented above. I will also propose a research line oriented to verify or disprove this perspective.

BRAIN SIZE AND BODY WEIGHT (PASSIVE GROWTH)

When comparing different species, the brain usually grows slower than the rest of the body. In other words, larger species tend to have relatively smaller brains. In mammals, the brain scales with body size with an exponent of 0.75; that is, $B \sim W^{0.75}$, where B = brain size and W = body weight (Pagel and Harvey, 1990). In birds, the exponent is close to 0.56 (Martin, 1981); in reptiles is around 0.6, while in amphibians it is about 0.47. Bony and cartilaginous fish have exponents of 0.65 and 0.94, respectively (Platel, 1979; Deacon, 1990).

What this exponent means has been the matter of some debate. Jerison (1973), believing that the exponent in mammals was around 0.67 instead of 0.75 as has been now confirmed (Martin, 1981; Harvey and Bennett, 1983; Pagel and Harvey, 1990), proposed that it represented the need to innervate the body surface which for geometrical reasons scales with volume with an exponent of 2/3. When the exponent was found to be around 0.75, it was suggested that basal metabolic rate, also scaling with body size with an exponent of 0.75, determined how much the brain could grow during pregnancy (Martin, 1981; Armstrong and Bergeron, 1985). For birds, the situation was supposed to complicate because there were two metabolic constraints of brain growth: production of the egg plus growth within the egg, which lowered the slope to 0.56 (see Martin, 1981). However, it has been found that when the influence of body weight on both brain size and basal metabolic rate is eliminated, the relation between brain weight and metabolism vanishes (Harvey and Bennett, 1983;

McNab and Eisenberg, 1989; Harvey *et al*, 1991), indicating that metabolic rate has no direct effect on brain growth.

Alternatively, the 0.75 exponent of mammals has been interpreted as a direct consequence of the relation between prenatal and postnatal growth in ontogeny (Riska and Atchley, 1985). In the brain of placental mammals, the prenatal phase mainly consists of neuron production, while postnatal growth is due to glial proliferation, increase in cell size and elongation of processes (Jacobson, 1991). Most of adult brain weight is achieved prenatally (during the phase of neuron production), but the rest of the body keeps growing at a fast rate for a long time after birth. This means that brain size is determined mainly by prenatal growth, while body size depends on both the prenatal and postnatal phases. In larger species the postnatal period is increasingly important in determining body size. This limits prenatal growth, resulting in a relatively smaller brain size. The case of marsupials may be similar, the only difference being that birth occurs very early and does not correspond to the point where neuron production ends. Therefore, in marsupials birth does not serve as a marker of the period where the brain slows its growth and consequently decouples from body growth.

Concerning birds, it has been found that post-hatching growth has a significant influence in brain size (Bennett and Harvey, 1985), a situation that makes them not comparable to mammals. This indicates that Martin's (1981) model of both egg and hatchling sizes limiting brain growth is untenable. In this context, it has been found that in songbirds there is continued neurogenesis into adulthood (Nottebohm, 1991). However, the new neurons do not incorporate only in brain nuclei related to song, suggesting that this may involve more general functions than just song learning. If so, post-hatching neurogenesis could be a widespread phenomenon among birds (and perhaps also reptiles). This would explain the influence of post-hatching growth on adult brain size in birds. This phenomenon has not been investigated in other birds, nor in reptiles, which makes it an interesting matter of future research.

As mentioned above, most brain components scale with body growth, although they do not do so at the same rate (Fox and Wilczynski, 1986). In other words, the allometric exponent with body size differs between brain components, indicating that as the brain follows body growth in evolution, it slightly changes its shape. Moreover, larger brains tend to be more differentiated in terms of number of components (Northcutt, 1981; Ebbesson, 1984). It has been proposed that the differentiation of brain areas that occurs in species with larger brains is simply a size-related event that has no effect in processing capacity (Deacon, 1990). Alternatively, here I will show evidence that the modifications that occur as a consequence of size are limited and work largely to compensate for difficulties related to perform the same function in an increased volume. I will argue below that the most important source of brain differentiation is not size but natural selection of behavioral capacity, and this does result in enhanced processing abilities.

One example of rearrangements due to brain size is provided by the structure of the corpus callosum (Aboitiz, 1992a; Aboitiz *et al*, 1992). Across species differing in brain size, the great majority of fibers connecting the two hemispheres tend to be rather uniform in terms of diameter and conduction velocity. However, there is a small population of gigantic fibers that connect primary sensory areas whose diameter increases with brain size. Considering the linear relation between fiber diameter and conduction velocity, we have proposed that those fibers increase their conduction velocity in order to compensate for the imposed transmission delay due to longer distances in larger brains (Aboitiz *et al.*, 1992). Time constraints may be more stringent in primary and secondary sensory areas because they are at the earliest processing phases. Fibers connecting these areas therefore suffer more dramatic changes in size than fibers corresponding to the so-called association areas. This is an example of cellular and histological differentiation according to size that occurs in order to maintain the properties of the respective neural networks.

Other arrangements associated with increased size are decreased neuronal density,

increase in the size of some neurons and increased myelination in some parts (Haug, 1987). As said, some of these changes occur to maintain function, but others are developmental correlates of a larger size. In any case, it is important to note that these modifications occur caused by the larger size of the brain and not vice versa.

Therefore, when the brain grows in evolution by following body size increases, there are some modifications in the relative size of components as well as modest cellular and connectional rearrangements. However, I propose that these changes are not enough to make a notorious increase in processing capacity. This explains why a cow's brain, with about 400 grams, is not capable of performing the cognitive operations that a macaque can do, with only 80 grams of brain. Of course those additional neurons in the cow are not very busy controlling the larger body (Jerison, 1973), because besides the reasons exposed above, this would imply the unlikely possibility that if liberated from its body, the cow's brain would increase its cognitive capacities beyond those of the monkey. Or that the smaller macaque brain is not capable of controlling a cow's body but is able to perform much more complicated cognitive functions than that. The same holds for the case of humans (with brains of 1,300 grams) and whales (with brains of 7,000 grams or more). Below, I will further illustrate this point with some examples.

The above situation is comparable to what occurs in the ontogeny of brain nuclei involved in song in songbirds. These nuclei increase their size triggered by the androgen testosterone, but the individual needs to be exposed to conspecific songs in order to develop its own song (Nottebohm, 1991). In this way, although growth permits the acquisition of a perceptual and motor skill, it needs the influence of the environment and behavior in order to achieve a working structure. Growth per se is undifferentiated, resulting in no specific neural network. I suggest that a similar phenomenon occurs in phylogeny. For evolutionary brain growth to result in a better processing brain, there needs to be selection of increased behavioral skills. This aspect will be discussed in the following sections.

The above digression does not necessarily mean that an additional number of neurons will have no effect whatsoever in brain performance. It is known that polyploid salamanders, with a reduced number of very large neurons with respect to diploid ones, are somewhat impaired in learning abilities. Also, increased brain size produced by hormonal manipulation can enhance learning skills in rats (for a review, see Jacobson, 1991). However, my position is that just increasing the amount of neurons is not sufficient to produce a dramatic effect in brain organization, such as the differentiation of the temporal lobe that occurs in primates (Pandya and Yeterian, 1990). The action of natural selection on behavioral capacity is necessary in order to trigger a reorganization of the brain.

ECOLOGICAL SPECIALIZATIONS AND BRAIN SIZE (ACTIVE GROWTH)

The encephalization quotient

Brain/body graphs show some residual variation. This dispersion has been proposed to correlate with the species' intelligence and has been measured as the encephalization quotient (EQ), defined by Jerison (1973) as the ratio between the real brain size and expected brain size of a "basal" animal of the same body weight. In this way, when compared to "basal" insectivorous primates have an EQ of 2.6, and Homo of 7.6. However, this measure strongly depends on the group chosen as "basal", and since as will be seen below, the allometric exponent widely changes with the taxonomic level at which comparisons are being made (class, order, family, etc.; Platel, 1979; Pagel and Harvey, 1989), this measure is questionable unless using closely related species.

Diet

This dispersion in brain/body graphs is explained in large part by ecological factors (Mace *et al*, 1980). It has been found that diet, which relates to foraging strategy, is a good predictor of relative brain size in mammals. In general, the more predictable

the food source is, the smaller relative brain size the species has. For example, folivorous primates, having shorter home ranges than frugivorous ones, tend to have relatively smaller brains than the latter ones (Mace *et al*, 1980; Harvey *et al*, 1987). In the same line, folivorous rodents have smaller brains than granivorous ones (Mann *et al*, 1988). Among bats, fruit-eating and nectarivorous species have larger brain sizes than echolocating insectivorous ones (Mann, 1963; Pirlot, and Pottier, 1977; Eisenberg and Wilson, 1978; Pirlot, 1987). Similar findings have been obtained for teleost fish (Bauchot *et al*, 1989). It has been proposed that the association between encephalization and diet has to do with the quality of the food (Foley, 1990; Foley and Lee, 1992; Milton, 1993): with an energy-rich diet it would be possible to afford the growth of a larger brain. However, this argument relies on the concept that basal metabolic rate puts a limit on brain growth, assumption that has been disproved. Furthermore, the relationship between diet and metabolic rate in mammals has been also questioned (Harvey *et al*, 1991; see above).

Brain components.

However, it appears that specific brain components are better predictors of ecological specializations than the whole brain. For example, in insectivorous it has been found that distinct brain regions correlate in size with specific ecological niches (Stephan *et al*, 1991). Fossorial species tend to reduce the visual system while their somatosensory system and striatum have a marked tendency to increase in size. Limbic structures are better developed in species that display active antipredator behaviors (fight or flight) as opposed to species with passive antipredator behaviors (i.e., rolling into a ball of spines as in the case of hedgehogs). In semiaquatic species neocortical, striatal, cerebellar, vestibular and trigeminal structures are more developed than in terrestrial insectivorous, but the olfactory and limbic components are reduced. Findings consistent with these have been obtained for bats (Pirlot and Jolicoeur, 1982; Jolicoeur and Baron, 1982; Pirlot, 1987) and primates (Armstrong *et al*, 1987; Sawaguchi, 1988, 1989).

Among birds, foodstoring species, that must keep a memory of the places where they keep their food, tend to have larger hippocampi than other species (Sherry *et al*, 1989; Krebs *et al*, 1989; Healy and Krebs, 1992). Another case is the brood parasitic cowbird, in which females must remember the location of several potential host nests, while males do not assist in the task. In this species, females have a larger hippocampus than males (Sherry *et al*, 1993). It is noteworthy that in these instances, overall brain size is not different from other birds, presumably because the hippocampus is such a small part of the brain. These considerations lead me to propose that in general, ecological factors affect the size of specific brain components involved in particular functions. Sometimes, these components are large enough to make a difference in overall brain size, as in the case of the neocortex, but this does not need to be so. In this way, I conclude that allometric relations *between brain components* will be much steeper when ecological factors induce brain growth than when body size triggers it. Body size produces generalized brain growth, while behavioral specializations relate only to specific brain parts, thus generating more dramatic differences between components.

The taxon-level effect

It has been determined that at least in mammals, the brain/body exponent is not the same if comparing species of different orders, species of different families within an order, different genera within a family, etc. (Riska and Atchley, 1985; Pagel and Harvey, 1989). In mammals, the exponent decreases from 0.75 (when comparing different orders) to about 0.2 or less when comparing individuals within a species (see Fig. 1). Some exceptions are the order primates and the genus Homo (fossil and living), with exponents of 0.94 and 1.73, respectively (Harvey *et al.*, 1987; Pilbeam and Gould, 1974). The decrease in the brain/body exponent with decreasing taxonomic range is referred to as the taxon-level effect. There is more than one interpretation of this phenomenon. Lande (1979) proposed that the effect results from selection on body size

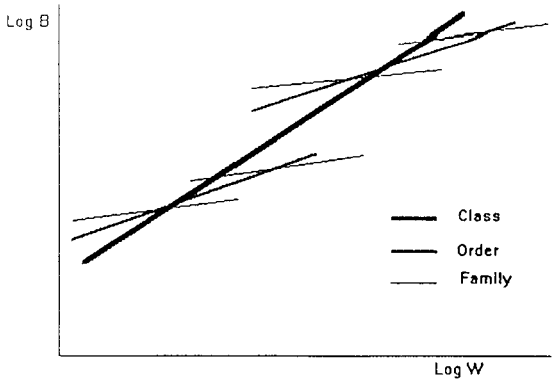

Fig 1. The taxon-level effect. This scheme shows that the brain/body exponent (shown as a slope in this log-log graph) is higher when comparing different orders within a class and progressively decreases when comparing families within an order, genera within a family, etc., indicating that brain size is more conservative at lower taxonomical levels. The thickness of the lines indicates the taxonomic level at which comparisons are being made. For simplicity, the class consists of two orders and the orders have two families each.

alone among close species, while among distant species selection acts on both body and brain size. A second interpretation (Riska and Atchley, 1985) states that, across closely related species, body size differences involve the late, postnatal period of growth, having little incidence on brain size. However, among more distant species, body size differences also involve the prenatal growth phase, in which brain and body growth are genetically and developmentally coupled. This results in a concomitant difference in brain size, therefore increasing the brain/body exponent. The third view (Pagel and Harvey, 1989; Harvey and Pagel, 1992) proposes that relative brain size differences across distant species result from dietary differences. When these ecological variables are statistically controlled for, a large part of the taxon-level effect disappears (Pagel and Harvey, 1989). Although ecological differences (and hence selection on brain size; Lande, 1979) explain large part of the data, there are still cases that cannot be explained in this way, indicating that Riska and Atchley's (1985) developmental/genetic model still has some room in this phenomenon.

Therefore, the steepest brain/body slopes across higher taxa tend to be produced by active growth, and have resulted in the large brains of birds, mammals, primates and of course humans. However, it must be recalled that this occurs only when those brain components are large enough to influence total brain size. There are many (perhaps most) cases where active growth relates to average brain/body slopes simply because the relevant brain components are not large enough to make a difference in brain weight (recall the case of the hippocampus in foodstoring birds). On the other hand, considering Riska and Atchley's (1985) developmental model for the taxon level effect, it is still possible that "passive" brain/body slopes tend to increase with taxonomic distance, thus eventually producing a moderately high slope just by scaling of body mass. As said, more diagnostic of active growth than brain/body slopes may be the relative sizes of different brain components.

Developmental periods involved

The above consideration poses theoretical constraints with respect to the developmental periods that should affect brain development. If the brain follows increases in body mass, it is expected that nerve cell proliferation will be a relatively generalized phenomenon, affecting in greater or lesser extent all brain parts. However, in active growth only those brain components involved in the respective tasks will increase their cell number. Therefore, in passive growth cell proliferation may affect either early periods of brain development, when brain parts are not yet defined (thus guaranteeing size increases in all brain components), or produce generalized increases in the whole brain in later developmental periods. If Riska and Atchley's (1985) developmental model of the taxon-level effect is correct, we should expect that during evolution, progressively earlier developmental periods will be affected as evolutionary divergences increase in time. At least in some cases, nerve cell proliferation may be eventually displaced to earlier and earlier developmental phases as evolutionary time passes. On the other hand, in active growth the situation is different. Here, increases in cell number must affect only late developmental periods, when the cell lineages corresponding to the respective brain nuclei are already specified (Wimer, 1990). In this

way, only the selected brain components will increase in size. This is what has happened in the origin of the mammalian cerebral cortex, where all the additional cells that have emerged are produced during late cortical development (Reiner, 1991).

NEURAL REORGANIZATION

There are also cellular and connectional rearrangements of neural structures associated with ecological conditions. For example, the cellular structure of the retina depends on the habitat in several groups (fish: Collin and Pettigrew, 1988; birds: Inzunza *et al*, 1991; mammals, Fisher and Kirby, 1991); auditory structures are better developed in echolocating than in non-echolocating bats (Suga, 1989; Riquimaroux *et al*, 1991; Habersetzer and Storch, 1992); subterranean animals have somatosensory specializations and regressive visual and auditory structures (Nevo *et al*, 1991; Cooper *et al*, 1993; Heffner and Heffner, 1993); the cortical representation of the hand is better developed in animals who manipulate objects with them (Welker and Seidenstein, 1959; Carlson, 1990; Johnson, 1990); tactile vibrissae have a distinct columnar representation in rodents (Johnson, 1990), and the star-nosed mole has a similar representation of the nose (Catania *et al*, 1993); and of course we must mention the case of ocular-dominance columns in the visual cortex of cats and old-world monkeys (Hubel and Wiesel, 1972). More conspicuous connectional rearrangements are associated with ecological conditions in the origin of snakes (Ulinski, 1971) and the origin of mammals (Aboitiz, 1992b, 1993).

I suggest that these rearrangements occur through selection of increased behavioral capacity. The following is an evolutionary scenario in which animals change their behavior as a response to new ecological conditions, and this behavioral change relates to ontogenic rearrangements in brain connectivity. Evolutionary selection of an increased number of nerve cells is a strategy that aids in these connectional rearrangements.

It has been proposed that in evolution, peripheral modifications precede those in the central nervous system (Van der Loos and Dörfl, 1978; Wilczynski, 1984; Merzenich, 1985; Edelman, 1988). The central nervous system will initially accommodate its connections to the changing periphery by means of plastic, ontogenic rearrangements of the sort observed in the variations of the cortical somatosensory maps that occur according to changes in individual experience (Jenkins *et al*, 1990; Merzenich and Samachima, 1993). A large part of species-specific differences in somatosensory maps have been proposed to result from differences in use of the respective organs rather than to genetic differences, especially among closely related species (Merzenich, 1985). These reorganizations may result in the segregation of nerve terminals, producing a consequent parcellation of different projection systems, and eventually leading to the proliferation of new brain areas (Ebbesson, 1984). I suggest here that this sort of ontogenic rearrangements are the principal event in the adaptation of the vertebrate central nervous system to new functions. These rearrangements have directly to do with increased processing capacity in the respective brain regions. I will discuss now the role of size in this process.

If there is selection of those brains that are better at rearranging their connections, a larger size of the respective components will be a favourable condition. According to this view, those individuals that have a genetically determined increase in cell proliferation in the respective brain regions will have a larger space to perform those connectional modifications, and thus will be better able to separate neural circuits concerning different aspects of sensorimotor coordination. Therefore, increasing the size of the respective components has to do mainly with helping the ontogenic rearrangements to take place. This is probably what happens with the effects of neuron number on learning skills in salamanders and rats (see above). Of course, with an increased number of cells brain capacity will also be better, but I suggest that the main role of cell proliferation is to permit connectional rearrangements leading to the progressive specification of neural circuits. In this way, brain growth is a secondary factor in phylogenetically increased neural processing, while connectional

rearrangements are the most important event (Fig 2).

In this scenario there will also be selection of genetically-determined neural connections and cell differentiation, that may be favoured by increases in brain size as well (Fig 2). These events –ontogenetic rearrangements and genetic increases in cell number and cell differentiation– are supposed to overlap extensively in evolutionary time. I am emphasizing the concept that plastic rearrangements precede the whole process because they are ontogenic, and furthermore they set the path for natural selection to work on (see Aboitiz, 1990). In this way, behaviour and ontogeny act as a frame determining the direction of evolutionary changes in brain size and structure (Fig. 2; see Aboitiz, 1990).

Adaptive (active) brain growth is proposed to differ from growth due to scaling of body mass because in the former, increase in nerve cell proliferation takes place as a secondary response to increasing processing demands. On the other hand, when the brain follows body growth, increased neuroblast proliferation is the primary factor, and the limited onto and phylogenic connectional rearrangements are supposed to occur as a consequence of this enlargement. Another point of caution is that it is likely that in many cases the two modes, passive and active growth, occur together, as in the cases of higher primates and human evolution (Gould, 1975, 1977; Fox and Wilczynski, 1986). Here, connectional rearrangements may make use of the active *and* the passive growth components in order to develop the new connectivities. Thus, those components growing actively show the highest exponents with body size of all brain parts (Fox and Wilczynski, 1986).

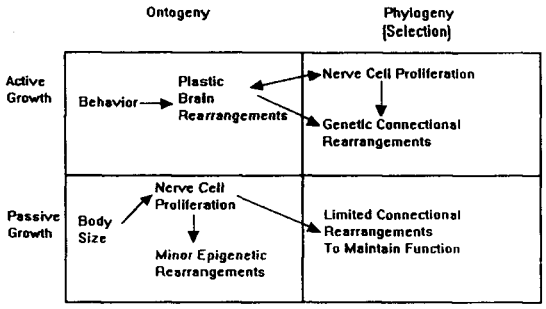

Fig 2: This diagram depicts the proposed evolutionary scenario for the two modes of brain growth. In passive growth (below), a larger body size produces a correlated increase in neuroblast proliferation, which eventually leads to both epigenetic and phylogenetic rearrangements, although limited in their extent. In active growth (above), behavior induces plastic rearrangements that result in phylogenetic selection of increased brain cell proliferation and also genetic changes in cell specificity and differentiation in distinct brain parts. The increased size of these parts permits further onto- and phylogenetic connectional rearrangements.

OF DOLPHINS, BATS AND SALAMANDERS

I will now indicate some instances of adaptive or "active" increase in brain size. In primitive marsupials and insectivorous, cortical areas related to different modalities tend to overlap with each other and with motor areas; but in more derived marsupial and placental orders (and also in the echidna), there is a progressive tendency to separate these areas (Sarnat and Netsky, 1981; Ebbesson, 1984; Johnson, 1990; Rowe, 1990). Although it has been claimed that this reorganization is a simple consequence of brain size increase (Deacon, 1990), the general consensus is that it is related to increased specificity of neural interactions and enhanced processing capacity.

There is still a further degree of brain differentiation in mammals. In most relatively advanced mammalian orders, the primary sensory representations corresponding to the different modalities (visual, auditory and tactile) lie separate but almost adjacent to each other (Johnson, 1990). For example, in orders such as Artiodactyla, brain size does not appear to be related to the differentiation of areas or to changes in cortical organization, indicating that these may be cases of largely passive growth. In carnivores and primates, animals with frontal vision respectively associated with predatory and arboricoral habits, the primary areas tend to separate from each other, and the so-called "higher order" sensory areas disposed between the primary areas have acquired a dramatic expansion. This is especially obvious in the visual cortex (Johnson, 1990; Payne, 1993). It is not clear as yet to what extent this expansion has involved the acquisition of new areas or the expansion of preexisting ones. For example, Montero

[1993] recently found in the rat that the number of extrastriate visual areas receiving projections from area 17 may be comparable to those in carnivores and primates, although he acknowledges that at higher processing levels new cortical areas must have evolved in the latter groups. In any case, this indicates that in these orders a significant reorganization of the brain has taken place, associated with growth and differentiation of at least the visual modules in the case of carnivores, and of the whole brain in primates. Recall that in primates, extensive connections between parietotemporal and prefrontal areas develop, associated with a dramatic expansion of the cerebral cortex (Pandya and Yeterian, 1990; Aboitiz, 1992b).

Another case is the origin of the mammalian cerebral cortex. I have argued (Aboitiz, 1992b, 1993) that in the origin of mammals a significant reorganization of the brain has taken place, associated with an increased neuroblast proliferation in the cerebral cortex. This reorganization resulted from evolutionary adaptation to specific ecological circumstances (see Aboitiz, 1990). In general, every vertebrate class is characterized by its own variant of brain organization (Northcutt, 1981, 1985; Ulinski, 1992). It is tempting to suggest that the most dramatic events of brain reorganization in the history of vertebrates have occurred in the origins of each of the classes that compose the type. Within these classes, brain evolution has been more conservative.

There are also some exceptions to the rule. One of these is the case of dolphins, who have suffered a dramatic cortical expansion without an accompanying differentiation of cortical areas (Glezer *et al*, 1988; Morgane *et al*, 1990). In fact, cetacean cortex is extremely primitive in anatomical and neurochemical design, lacking granular cells of layer IV and receiving its thalamic projections in layer I instead of layer IV (Glezer *et al*, 1988; Hof *et al*, 1992; however, see Revischin and Garey [1990] for a different view on thalamic terminals in cetacean cortex). In this context, it is of interest to note that, although brain volume and cortical surface are enormous in dolphins, in comparison to other mammals *cortical volume* is surprisingly small in relation to other brain cell groups (Glezer *et al*, 1988). There is little question that the expansion of the dolphin cerebrum relates to increased behavioral abilities and is a case of active growth. I suggest that this situation closely resembles the case of the cerebellar cortex. This structure has also dramatically increased its size in the history of vertebrates and especially in human evolution (Leiner *et al*, 1993), although its intrinsic organization has remained quite conservative when compared to the cerebral cortex (Sarnat and Netsky, 1981; Meek, 1992; Ate, 1993). Perhaps this has to do with the tangential organization of the cerebellar cortex, where parallel fibers run in a mediolateral direction in the molecular layer (comparable to layer I of the cerebral cortex). As said, the mode of termination of thalamic terminals in primitive cortices such as the dolphin's is also tangential, ending in layer I. Afferent organization in the cerebellum and in dolphin neocortex might have precluded them from undergoing much regional differentiation, as overlapping of terminals tends to be the rule in a tangential pattern. When afferents are oriented in a radial pattern such as in the neocortex of advanced mammals, overlapping of terminals is less extensive and topographic relations are better defined, possibly facilitating the process of parcellation of connections and eventual differentiation of brain areas. Shortly, the cerebellum and the dolphin's neocortex may have evolved mainly by modifying their extrinsic connections and by drastically increasing their size while maintaining the intrinsic neural architecture of the system, perhaps due to their common tangential organization of afferents.

Brain reorganization may also occur without growth. This has happened in the case of echolocating bats who, despite having the smallest relative brain sizes of their group (Pirlot, 1987; see above), have developed an exquisite differentiation of cortical auditory areas (Suga, 1989). In addition, the paedomorphically-reduced brains of salamanders are able to perform notably complex behavioral tasks, provided adequate microscopic rearrangements of neural architecture (Roth *et al*, 1993).

Summarizing, although in most cases brain reorganization is associated with growth in particular regions, sometimes there is (active) growth with limited rearrangements, and there are also rearrangements without growth. This indicates that reorganization is not a consequence of increased size (as Deacon [1990] implies), even if it is active growth. On the contrary, growth and reorganization are two converging strategies for increasing brain performance. In some instances, one of these possibilities is limited for some reason and there is dissociation of the two phenomena, as happens in dolphins on one hand and bats and salamanders on the other.

CONCLUSION: A PROGRAM FOR RESEARCH

This article has reviewed some of the literature on the evolution of brain size and structure, and proposes a conceptual framework to understand this process. A main concept introduced here is the distinction of two modalities of evolutionary brain growth, one (passive) that follows changes in body size and is characterized by conservative allometric relations between brain components, limited cellular and connectional rearrangements that occur a posteriori of the change in brain size, and where cell proliferation involves either early phases of brain development or is a generalized effect in late phases. The second mode of brain growth is the active one, that results of selection of increased behavioral capacities. Here, allometric relations between components are steeper than in the anterior case, and there are important cellular and connectional rearrangements many of which occur prior to and even determine the selection of larger size in distinct brain components. In active growth, increases in cell proliferation are restricted to late developmental periods, when the respective cell lineages are already committed to distinct brain parts. Finally, the two modes of growth may coexist in many instances, complicating the scene as in the case of primate and human evolution.

Most of the proposed concepts remain speculative. In order to verify or disprove this model, I suggest a specific research program oriented in first instance to discriminate between the effects of body size and ecological variables on both brain size and structure. Of particular interest is the effect of the two factors on the size, number and also quality of specific areas in the cerebral cortex. For this purpose, it may be recommendable to choose a specific taxon of high ecological diversity and relatively easy access, such as the group of South American rodents, that conform a diversified group with a well-known ecology (Redford and Eisenberg, 1992). Of course, larger brained taxa such as carnivores or primates are also of interest. One main thing to be done is, after characterizing the anatomy of the respective brain parts, to determine the effects of size and ecology in the different species. Some ecological parameters of relevance are, of course, diet, habitat and social organization. Other likely factors are for example, life history strategy: does brain organization relate to the position in the K-r axis? In this general context it is of interest to note that preliminary findings indicate that carnivores and primates, respectively predatory and frugivorous taxa, both with strong frontal vision, tend to have a larger size of the posterior (visual) part of the corpus callosum than herbivores such as perissodactyls and artiodactyls with lateral vision (Olivares and Aboitiz, 1993). There are suitable statistical devices for these analyses, that can discriminate between the effects of size and ecology even if they coexist to a large extent (Pagel and Harvey, 1989; Harvey and Krebs, 1990). This work requires extensive sampling and tedious anatomical analyses that may not give results in the short time, for which reason it has probably been avoided in the past. However, it will shed light on important evolutionary and comparative problems on brain evolution, reason for which it is a recommendable line of research.

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