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We dedicate this book to everyone who contributes to the preservation of biodiversity and our fascinating primate relatives in particular.

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Primate Brains and Life Histories:
Renewing the Connection

10 Primate Brains and Life Histories: Renewing the Connection

Robert O. Deaner, Robert A. Barton, and Carel P. van Schaik

Across primate species, brain size correlates with several life history variables, including maximum recorded life span, gestation length, and age at first reproduction (Harvey, Martin, and Clutton-Brock 1987; Austad and Fischer 1992; Allman, McLaughlin, and Hakeem 1993a; Allman 1995; Hakeem et al. 1996; Allman and Hasenstaub 1999; Barton 1999; Ross and Jones 1999; Judge and Carey 2000; Ross, chap. 11, this volume; see also Friedenthal 1910; Sacher 1959, 1975, 1978; Sacher and Staffeldt 1974; Mallouk 1975). These life history variables are themselves correlated, meaning that large-brained primates generally have slow, prolonged growth periods, late sexual maturation, and long lives (Harvey, Martin, and Clutton-Brock 1987; Charnov and Berrigan 1993; for nonprimates see Western and Ssemakula 1982; Stearns 1983; Read and Harvey 1989). To account for these patterns, researchers have offered a number of hypotheses about brain development, function, and degeneration (table 10.1). Thus far, however, these hypotheses have not been examined rigorously, especially with respect to their theoretical bases (but see Allman 1995; Hakeem et al. 1996). One reason for this neglect is uncertainty as to whether the brain size-life history correlations that suggested these hypotheses actually indicate direct evolutionary links between the variables. In particular, it has been proposed repeatedly that these correlations may be artifacts of statistical methodology or unconsidered evolutionary processes (Calder 1976; Economos 1980a; Prothero and Jürgens 1987; Harvey, Martin, and Clutton-Brock 1987; Harvey and Krebs 1990; Barton 1999).

Our goal in this chapter is to evaluate these hypotheses. Given the uncertainty regarding the correlations that gave rise to them, however, we begin by considering the factors that could have spuriously produced those correlations. In the first section, we show that there are tractable statistical

methods for considering the most problematic potential confounding factors; namely, body size, phylogeny, and socioecology. We demonstrate that even when conservative methods are used to deal with these confounds, there remain indications of correlated evolution between brain size and life history. With this evidence in hand, we turn in the following section to the hypotheses proposed to explain the correlations. Although several of the hypotheses are mutually compatible, we evaluate the logic and premises of each separately. Although some of the hypotheses can be refuted a priori, most are at least plausible. For these, we derive additional predictions and test several of those predictions with data from primates and other mammals.

Are Life History and Brain Size Truly Linked?

The hypotheses in table 10.1 assume that brain size and life history variables have direct evolutionary links, such that when there is a change in either brain size or a life history variable, the other trait shows, or becomes more likely to show, some corresponding change, *regardless of other biological processes.* In other words, the challenge is to show that a given brain size-life history correlation is not an artifact of some other process that simultaneously affects both variables. Although there are numerous potential confounds, body size, phylogeny, and socioecology are thought to be most problematic.

Body Size

Body size is arguably the most informative single variable for understanding an organism's physiology, morphology, and life history (Schmidt-Nielsen 1984; Calder 1984). It is highly correlated with brain size and with virtually all aspects of life history; therefore, the influence of body size alone can be expected to produce brain size-life history correlations. Several methods have been employed to examine whether brain size and life history traits are linked independently of body size, but all of them have been plagued by the fact that measures of body size are influenced by numerous factors. Body mass, for instance—the most commonly used body size measure—is highly sensitive to nutritional and reproductive status (e.g., Pagel and Harvey 1988b; Dunbar 1992; Smith and Jungers 1997).

Sacher (1959) made the first attempt to show that life history is related to brain size independently of body size. He found that in a sample of sixty-three mammals, maximum recorded life span (hereafter life span) was more strongly correlated with brain mass than with body mass. Nevertheless, the fact that the brain mass-life span correlation statistically exceeds the body

Table 10.1 Hypotheses generated by comparative studies of primate brains and life history

Hypothesis	Premise and claim	Hypothesis type
Physiological regulator	Larger brains allow greater precision of physiological regulation and thus longer life. Longer life can be achieved only with larger brains, and vice versa.	Strong constraint
Growth regulator	Brain growth governs body growth; larger brains grow for longer periods, and thus larger-brained animals exhibit prolonged somatic growth. Prolonged somatic growth can be achieved only with larger brains, and vice versa.	Strong constraint
Neuronal investment	The functioning population of neurons inevitably diminishes; the only way to maintain a functioning brain (and population of neurons) at advanced age is to grow a large brain initially. The evolution of increased longevity must *often* be accompanied by an increase in brain size.	Weak constraint
Maturational constraints	Brain and behavioral maturation is an inherently slow process that takes longer to complete for large-brained animals. If there is selection for increased brain size, the period of development must *often* be extended.	Weak constraint
Cognitive buffer	The behavioral flexibility permitted by larger brains reduces extrinsic mortality. An evolutionary expansion of the brain increases the likelihood of an evolutionary increase in life span.	Adaptive
Brain malnutrition risk	Developing brains require a constant energy supply to avoid irreversible damage; to keep risks of brain damage low in unpredictable environments, large-brained animals grow their brains slowly; slow brain growth requires slow somatic growth. An evolutionary extension of somatic growth period increases the likelihood of an evolutionary increase in brain size.	Adaptive
Delayed benefits	Brains, and the learning they allow, provide delayed benefits that continue to accrue throughout the animal's life; long-lived animals benefit more from large brains than short-lived animals. An evolutionary extension of life span increases the likelihood of an evolutionary increase in brain size.	Adaptive

mass-life span correlation does not automatically demonstrate that a portion of the life span-brain mass correlation cannot be explained by body size. The reason is that body mass is more subject to sampling error than brain mass is (Pagel and Harvey 1988b); hence, body mass could be worse than brain size as an estimator of true body size (Lindstedt and Calder 1981; Harvey, Martin, and Clutton-Brock 1987; Harvey and Krebs 1990; Barton 1999). Economos (1980a; see also Prothero and Jürgens 1987) argued this point forcefully, showing that two other size-related organs, the liver and the adrenals, are more highly correlated with life span than is body mass.

Other investigators have asked whether brain size and life history variables are related once the influence of body mass is statistically removed from both variables. The procedures used have included the residuals method (or partial correlation), in which the variables of interest are first regressed on body mass to generate residual or "relative" values (e.g., Harvey, Martin, and Clutton-Brock 1987; Allman, McLaughlin, and Hakeem 1993a,b), and multiple regression (Sacher 1975, 1978; Barton 1999). Nevertheless, because these methods are closely related to each other, they are both sensitive to the same potential pitfall. The problem is that if species-specific body mass estimates are highly prone to error, then using them to remove the effects of size from any two variables of interest could lead to spurious correlations between those variables (Harvey and Krebs 1990; Barton 1999). We can show this most easily by considering the results obtained with the residuals method when body mass estimates overestimate or underestimate true body size (fig. 10.1). For any given species (or independent contrast: see below), the error in body mass will create a bias in the same direction in both residuals: if the body mass estimate is erroneously too large, both residuals will be smaller than they should be; if the estimate is too small, both residuals will be larger than they should be. If the error in body mass is substantial, the two residuals may end up showing a significant correlation, even if there is not a biological relationship. Multiple regression is prone to the same problem because multiple regression coefficients represent the relationship between two variables (e.g., brain size and a life history variable) once the effects of another predictor (e.g., body size) have been partialled out.

Without "correct" body size measures, there may be no completely satisfactory way to control for body size. Nevertheless, the problem of correlated errors can be ameliorated with the residuals method if independent estimates of size are used for calculating the residuals for each variable (Harvey and Krebs 1990). In this case, an over- or underestimated body size estimate will produce an over- or underestimated brain size residual; however, because the life history residual is derived from a different body size estimate, it will not (generally) be over- or underestimated in the same manner.

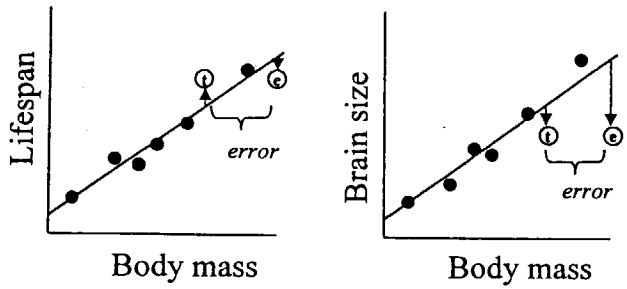

FIG. 10.1. The problem of correlated errors. Consider one species (open circles) in a hypothetical case in which true body size (t) and estimated body mass (e) are both known. If the residuals are generated from true body size, the life span residual will be positive and the brain size residual will be negative. In this instance, the hypothesis that the residuals are correlated will not be supported. On the other hand, if the residuals are generated from (over) estimated body size, both residuals will be negative, falsely supporting the hypothesis of correlation.

To explore the use of independent estimates of size in primates, we first calculated brain mass residuals from the regression of $\log_{10}$ brain mass on $\log_{10}$ body mass, taking brain and body mass data from Stephan, Frahm, and Baron (1981; H. Stephan, H. D. Frahm, and G. Baron, unpub.; see Deaner and Nunn 1999).[1] Next, we calculated life span residuals from the regression of $\log_{10}$ life span on $\log_{10}$ body mass, taking life span as the maximum value in primary and secondary sources (Hakeem et al. 1996; Ross and Jones 1999; unpublished data from the Duke University Primate Center) and taking body mass from Smith and Jungers (1997). For the sake of comparison, we also repeated the analysis using the body mass data from Stephan, Frahm, and Baron to derive both brain mass and life span residuals.[2]

As shown in table 10.2, the correlation between brain mass and life span residuals was positive and significant, both when the residuals were derived from the same body mass values and when they were derived from different values. As expected, however, the relationship was stronger when the residuals were derived from the same values. When we performed these analyses with gestation length and age at first reproduction (age at first reproduction from Ross and Jones 1999; gestation length from Harvey, Martin, and Clutton-Brock 1987), we found highly significant relationships in all cases,

Table 10.2 Relationships between brain size residuals and life history residuals in primates when the residuals are derived from the same or different body mass estimates

	Same estimates			Different estimates		
	r	*P*	*N*	*r*	*P*	*N*
Life span	.46	.0002	56	.43	.0005	56
Age at first reproduction	.52	.0002	47	.47	.001	47
Gestation length	.38	.0087	46	.34	.0153	46

Note: All results are based on species values.

but again, the relationships were somewhat stronger when based on the same body size estimates. Thus, this brief investigation of correlated errors in body mass shows that using the same body mass estimates to calculate brain size and life history residuals may in fact lead to artificially strong associations between brain size and life history. Although much more work is needed on the issue, we provisionally recommend that, whenever possible, workers employ different body mass estimates for calculating each of the residuals that will later be compared.

Phylogeny

Early studies that documented statistical relationships between brain size and life history traits treated species values as independent data points (e.g., Sacher 1959; Sacher and Staffeldt 1974; Austad and Fischer 1991, 1992; Allman, McLaughlin, and Hakeem 1993a,b; Hakeem et al. 1996). Such analyses implicitly assume that the brain size and life history of each species represents an independent evolutionary event. This assumption is clearly incorrect, however: a species' brain size and life history, as well as most other aspects of its biology, are partially predictable from its phylogenetic history (for a discussion of the reasons, see Harvey and Pagel 1991). In primates, for example, relative brain size is largely a function of whether the species in question is a strepsirrhine or a haplorhine (e.g., Martin 1990; Barton 1999). The fact that trait expression is dependent on phylogeny means that statistical tests that treat species (or other taxonomic units) as independent data points greatly overestimate degrees of freedom, a situation that may lead to incorrect conclusions (Harvey and Pagel 1991; Martins and Hansen 1996; Purvis and Webster 1999).

There is a rapidly growing literature on the methods that are most appropriate for treating phylogenetically dependent data (reviewed by Harvey and Pagel 1991; Martins and Hansen 1996; Purvis and Webster 1999). At present, the method of independent contrasts (ICs) is considered best for

calculating independent instances of evolutionary change in continuous variables (Purvis and Webster 1999). In brief, this method calculates evolutionary contrasts (differences between paired taxa) throughout a phylogenetic reconstruction of a character's evolution. The contrasts calculated at each node of the reconstruction should be fully independent of the other nodes and are therefore, in principle, suitable for standard statistical analysis (Felsenstein 1985; Harvey and Pagel 1991; Martins and Hansen 1996). Most importantly, because the ICs are representative of evolutionary change, a hypothesis of correlated evolution (e.g., that evolutionary changes in relative brain size are associated with evolutionary changes in life history variables) can be addressed explicitly.

We explored the associations between relative brain size and life span, age at first reproduction, and gestation length, employing Purvis and Rambaut's (1995) CAIC computer program and Purvis's (1995) phylogeny for calculating ICs. We followed standard practice in obtaining residuals after employing least-squares regressions, forced through the origin, on $\log_{10}$ values (Harvey and Pagel 1991; Garland, Harvey, and Ives 1992). There are theoretical reasons to expect that more recent contrasts could be misleading for variables that are sensitive to measurement or sampling error (Purvis and Rambaut 1995; Purvis and Webster 1999), and this problem could be acute for life history variables. Life span, in particular—a maximum measure—should be highly sensitive to the size and quality of the sample from which it is drawn (i.e., species that are poorly represented in captivity should often have appreciably underestimated life spans: Allman, McLaughlin, and Hakeem 1993a). Hence, we divided the contrasts into two groups of equal size, based on the corresponding nodes in the phylogeny (i.e., "young contrasts" and "old contrasts"). Then we repeated all of the tests with only the old contrasts (Purvis and Harvey 1995). The logic here is that because old contrasts usually incorporate data from several species, any error associated with a particular species estimate will have reduced influence.

Table 10.3 presents the results of our contrasts analyses and, for comparison, the results obtained when we considered species as independent data points (as presented in table 10.2). For all of these tests, we used different body mass estimates for calculating brain size and life history residuals (see above). Although these analyses indicate positive evolutionary relationships between the life history variables and brain size, the relationship reaches significance only for life span (fig. 10.2). For the analyses based only on old contrasts, the results are similar, although the life span-brain size relationship is no longer significant (but the correlation coefficient is slightly greater). These analyses confirm that life span is a genuine correlate of brain

Table 10.3 Relationships between brain size residuals and life history residuals in primates when residuals are derived from species, all independent contrasts, or old independent contrasts

	Species			All ICs			Old ICs		
	r	*P*	*N*	*r*	*P*	*N*	*r*	*P*	*N*
Life span	.43	.0005	56	.31	.03	52	.36	.07	26
Age at first reproduction	.47	.001	47	.20	.18	45	.27	.21	23
Gestation length	.34	.0153	46	.21	.16	44	.18	.43	22

size, but suggest that there might not be evolutionary relationships between age at first reproduction and brain size and gestation length and brain size, despite the apparent relationships when species were considered as independent data points. It is worthwhile to bear in mind that because life history variables may be highly error-prone, the absence of statistical correlations is not conclusive evidence that there are not biological relationships.

Socioecology

Several socioecological variables have been linked to differences in relative brain size (reviewed by Harvey and Krebs 1990; Barton and Dunbar 1997; Deaner, Nunn, and van Schaik 2000). There are also indications that some socioecological variables are associated with life history (reviewed by Ross and Jones 1999). It is possible, therefore, that brain size-life history correlations are by-products of socioecological factors (Harvey, Martin, and Clutton-Brock 1987; Ross and Jones 1999). For instance, a large group size (presumably meaning increased social demands: see Dunbar 1992; Dunbar, chap. 12, this volume) may select for both extended juvenility and larger brains, thereby producing a brain size-juvenility correlation (see Joffe 1997; Ross and Jones 1999).

The most direct way to evaluate whether socioecological factors are responsible for brain size-life history correlations is to test whether the same socioecological variables that correlate with brain size also correlate with

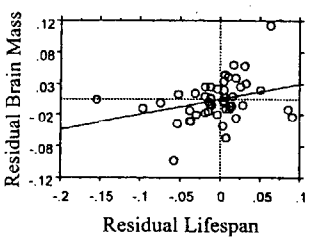

FIG. 10.2. Relationship between life span residuals and brain size residuals in primates, based on all independent contrasts. (See table 10.3 for statistics.)

Table 10.4 Relationships between life history residuals and socioecological variables in primates

	Group size			Home range residual			Frugivory		
	r	*P*	*N*	*r*	*P*	*N*	*r*	*P*	*N*
Life span	.19	.14	64	.24	.08	53	.10	.49	51
Age at first reproduction	.20	.15	54	−.34	.01	50	.13	.39	44
Gestation length	.01	.97	52	−.33	.03	45	.04	.78	40

Note: Life history residuals and home range residuals were calculated from independent body mass estimates. All results are based on all independent contrasts.

life history. Home range size, group size, and percentage of fruit in the diet have been previously shown to correlate with relative whole brain or neocortex size (Harvey and Krebs 1990; Barton and Dunbar 1997; Deaner, Nunn, and van Schaik 2000). Thus, we asked whether any of these factors correlate with life history variables. For these analyses, we took data on home range size and group size from Nunn and van Schaik (2002) and on frugivory from Barton (1999). We followed the procedures described above in calculating ICs, including generating residuals to remove the effects of body size from the life history variables. In addition, we used residual home range size, rather than absolute home range size, because we found this variable to be correlated with body mass. Again, for the reasons discussed above, we used different body mass estimates in generating the home range size residuals than we used when calculating the life history residuals.

We found that the relevant socioecological variables were not significantly correlated with life history variables (table 10.4; see also Harvey, Martin, and Clutton-Brock 1987; Ross and Jones 1999). The only significant findings were negative correlations between home range size and age at first reproduction and gestation length. Clearly, these *negative* correlations cannot explain why home range size should cause age at first reproduction and brain size to be *positively* correlated. Furthermore, Barton (1999; see also Allman 1999) has shown that in multiple regression, ecological and life history variables independently explain variation in brain size. Thus, it is highly unlikely that brain size-life history correlations are products of the common influence of socioecology.

Hypotheses to Explain Brain Size-Life History Correlations

The results presented in the previous section indicate that within primates, there are evolutionary associations between brain size and at least one life history variable—life span—that cannot be attributed to obvious confounds.

Hence, in this section, we evaluate the hypotheses that argue for direct brain size-life history associations (see table 10.1). Although each of these hypotheses deals chiefly with one aspect of life history, the strong correlations among life history variables (Harvey, Martin, and Clutton-Brock 1987; Charnov 1993; Charnov and Berrigan 1993) mean that any single hypothesis may turn out to have fairly general explanatory value. Conversely, these intercorrelations could ultimately make it difficult to distinguish among the hypotheses.

Although most of the hypotheses we review here have already been introduced in the literature, they have been underdeveloped with regard to their crucial assumptions and plausible evolutionary mechanisms. This situation has hampered attempts to understand how compatible these hypotheses are with modern life history theory and how general their predictions are (e.g., whether exceptions are allowed). Thus, besides testing predictions derived from the most plausible hypotheses, we devote considerable attention to exploring these fundamental issues.

Physiological Regulator Hypothesis

The physiological regulator hypothesis holds that brain size determines the overall regulation of physiological function: because larger-brained animals have more precise regulation of function, they are able to live longer (Sacher 1959, 1978; Hofman 1983; see also Friedenthal 1910; Mallouk 1975; Cutler 1976). According to this view, brain size determines longevity directly through physiological mechanisms, so that if there is selection on either brain size or longevity, the other trait necessarily changes.

The physiological regulator hypothesis is best understood as an extension of the now discredited rate-of-living theory (Rubner 1908; Pearl 1928; Stahl 1962; Lindstedt and Calder 1976, 1981; Boddington 1978). This theory holds that molecular damage is the inevitable by-product of energy consumption, and that a lower rate of energy consumption leads to a slower rate of molecular deterioration and therefore enhanced longevity. The basis of the rate-of-living theory was the general correlation between long life span and low metabolism (both of which are correlated with body size). However, in order to explain exceptional cases in which life span exceeds predictions based on metabolism (e.g., humans), brain size was incorporated via the physiological regulator hypothesis.

The rate-of-living theory no longer enjoys support because growing evidence indicates that metabolic rate is not directly related to either accumulated molecular damage or longevity. First, although metabolic by-products

are indeed major sources of molecular damage, the key to predicting accumulated damage is the efficiency of mechanisms that mitigate that damage (Orr and Sohal 1994). For instance, aerobic respiration produces free oxygen radicals, or oxidants, which damage other molecules by dislodging their electrons. However, cells possess mechanisms to remove or repair such damage and specialized molecules (antioxidants) that bind to oxidants before they can do their damage. Species differ greatly in their mitigating mechanisms (e.g., Hart and Setlow 1974; Sohal, Sohal, and Brunk 1990; Sohal, Sohal, and Orr 1995; Ku, Brunk, and Sohal 1993; Austad 1997b), a fact that is inconsistent with the rate-of-living theory (cf. Sacher 1978). A second line of evidence indicating a dissociation between metabolism and life span is experiments in which reduced caloric intake is found to extend longevity; in these cases, metabolic rate is not reduced (reviewed by Masoro 1995). Finally, the rate-of-living theory is weakened by the fact that several long-lived taxa (e.g., bats, birds) have high metabolic rates (Lindstedt and Calder 1981; Austad and Fischer 1991).

Could the brain provide the key link between metabolism and life span, as the physiological regulator hypothesis claims? Probably not. Although progress has been made in identifying mechanisms for reducing or repairing molecular damage (reviewed by Austad 1997b; Finch and Tanzi 1997), there is no evidence that the efficiency of these mechanisms is in any way dependent on the size of the central nervous system. Moreover, it seems unlikely on theoretical grounds that there must automatically exist a tight relationship between brain size and the accumulation of molecular damage. We would expect, for instance, that novel enzymes for eliminating oxidized DNA would occur through specific genetic alterations rather than as correlates of increased overall brain size. In summary, the physiological regulator hypothesis is a mechanistic hypothesis without a plausible mechanism (see also Economos 1980b).

Growth Regulator Hypothesis

The growth regulator hypothesis holds that brain growth governs body growth: specifically, for a given body size, that a large-brained animal will be characterized by slow, prolonged somatic growth. The impetus for this hypothesis was the observation of cross-species correlations between brain size indices and life history variables (Sacher and Staffeldt 1974; Western and Ssemakula 1982; cf. Friedenthal 1910; Ricklefs 1979). In particular, Sacher and Staffeldt (1974) emphasized the relationship between prolonged gestation length and large neonatal brain size.

Like the physiological regulator hypothesis, the growth regulator hypothesis holds that brain size and life history are not distinct traits; rather, large brain size and prolonged somatic growth co-occur because, for physiological reasons, they must. There is no doubt that numerous mechanisms (e.g., the endocrine system) are in place to ensure that growth in each part of the body, including the brain, is complementary to that of other areas (harmony of growth: reviewed by Bogin 1999). Nevertheless, the tremendous diversity of brain and body growth trajectories across species indicates that natural selection can alter these mechanisms (Case 1978; Read and Harvey 1989). Moreover, there is no plausible physiological account of how larger brains are constrained to produce slower body growth. The only such proposal is Sacher and Staffeldt's (1974) "minimax" theory, and it is underpinned by assumptions that the brain is "the slowest growing organ in the mammal" and "the pacemaker for growth of all other somatic tissues, which are constrained to grow at its pace." Both of these claims are incorrect: in mammals, relative to adult body size, brain growth substantially outpaces body growth in the fetal and early postnatal periods (e.g., Count 1947; Deacon 1990; Bogin 1999). Thus, the argument that large brains are physiologically constrained to "drag out" body growth is unsupported (see also Read and Harvey 1989).

Neuronal Investment Hypothesis

The neuronal investment hypothesis holds that brain size limits longevity because the functioning population of neurons diminishes throughout an organism's life, the only way to maintain adequate behavioral performance at an extended age is to invest initially in more neurons, and thus a larger brain. A crucial assumption of this hypothesis is that neurogenesis in mammals occurs only early in development. Allman (1995) proposed this hypothesis in light of evidence that neuronal damage, particularly in the cerebellum, accumulates in older animals and that this damage causes a decline in behavioral performance. To appreciate this idea, imagine the case of an old arboreal animal that is at high risk of falling from trees because it has a reduced population of cerebellar neurons. According to the neuronal investment hypothesis, if the animal's cerebellum initially had more neurons, more would be functioning for the animal in old age, and it would be less likely to fall.

Given the costs of growing and maintaining brain tissue (e.g., Holliday 1971; Aschoff, Gunther, and Kramer 1971; Armstrong 1985a; Aiello and Wheeler 1995), having to generate and maintain "extra neurons" to ensure that some will be available later in life would be highly inefficient; it would be better to invest in mechanisms to minimize or repair neuronal damage. Therefore, the neuronal investment hypothesis must assume that mechanisms to prevent neuronal damage do not exist, or are extremely limited in scope. A strong adaptationist would question this assumption because natural selection has a tremendous capacity to create mechanisms for repairing or avoiding cellular damage. For instance, a major cause of neuronal damage is ingested toxins (Allman 1995); selection for longevity might therefore act through the development of mechanisms to ensure that fewer toxins reach the brain, or, more simply, animals might change their diets. Despite this a priori argument, it is possible that constraints do exist such that in some contexts, neuronal damage is truly unavoidable. Allman (1995) implied that this might be the case for highly active inhibitory neurons, such as Purkinje cells of the cerebellum.

Fortunately, the validity of the unavoidable damage assumption can be tested empirically. If this assumption is correct, then neuron numbers should decrease throughout the life span. On the other hand, if the number of neurons does not begin declining until mid- to late adulthood, this finding would indicate that selection has acted to mitigate early damage. Because the Purkinje cells of the cerebellum are one of the few neuronal populations in which declines are undisputed (see Haug 1985; Allman 1995; Albert and Moss 1996; Scheibel 1996; Peters, Sethares, and Moss 1998), studies of this area are most relevant. Hall, Miller, and Corsellis (1975) and Torvik, Torp, and Lindboe (1986) both found that human Purkinje cells did not decline in number until about sixty years of age (see also Ellis 1920, chart 6). Thus, the assumption of unavoidable neuronal damage appears weak.

The neuronal investment hypothesis holds that the evolution of increased longevity must *often* be accompanied by increases in brain size. Conversely, evolutionary decreases in brain size must *often* be accompanied by evolutionary decreases in longevity. We stress the word "often" because it is conceivable that some animals will have brains that, for some adaptive reason (see the hypotheses below), are larger than the minimum size necessary to achieve their current life span (i.e., in this case, life span would be intrinsically limited by the deterioration of systems other than the brain). In this way, the neuronal investment hypothesis can be viewed as a "weak" or limited constraint hypothesis (as compared with the former two hypotheses, which argue for "strong" constraints). Because it allows exceptions, this hypothesis predicts a positive, but imperfect, evolutionary correlation between brain size and life span.

As already noted, there is some evidence for this brain size-life span linkage. Nevertheless, this prediction is readily derived from other hypotheses that have better-supported assumptions. Hence, we will focus here on the following prediction, which, as far as we know, is unique to the neuronal investment hypothesis: Because the cerebellum is apparently more sensitive to neuronal loss than other brain parts, cerebellum size and life span should be evolutionarily correlated, and the cerebellum's relationship to life span should be stronger than that of the whole brain or that of another large brain structure, such as the neocortex (Allman 1995).

We tested this prediction in three mammalian taxa: primates, bats, and insectivores.[3] Contrary to the prediction of the neuronal investment hypothesis, we found no evolutionary relationship between cerebellum size and life span in bats or insectivores (table 10.5). In primates, we did find evidence of a significant relationship, but, contrary to the prediction, it was not stronger than the relationship between neocortex and life span (cf. Allman, McLaughlin, and Hakeem 1993b; Hakeem et al. 1996). In summary, neither the chief assumption nor the unique prediction of this hypothesis is supported.

Maturational Constraints Hypothesis

In considering the evolution of life history and brain size in hominids, it has commonly been noted that prolonged juvenility may permit greater learning opportunities (e.g., Dobzhansky 1962; Mann 1972; Poirier and Smith 1974; Gould 1977; Lancaster and Lancaster 1983; Bogin 1999; Kaplan et al. 2000). Some researchers have therefore examined the hypothesis that large brains and slow life histories (especially prolonged juvenility) are evolutionarily associated across primates for the straightforward reason that large-brained animals need more time to learn adult skills than smaller-brained animals do (Janson and van Schaik 1993; Ross and Jones 1999). These and other researchers concluded, however, that this "needing-to-learn" hypothesis is weak on both empirical and theoretical grounds (Janson and van Schaik 1993; Ross and Jones 1999; Blurton Jones, Hawkes, and O'Connell 1999; N. G. Blurton Jones and F. W. Marlowe, unpub.; but see Kaplan et al. 2000). Nevertheless, we believe that a modified version of this hypothesis can withstand previously raised criticisms. We call this new version the maturational constraints hypothesis. Instead of focusing on the need for learning opportunities, the maturational constraints hypothesis emphasizes that some types of complex behavior can be supported only by mature nervous systems (cf. Altmann and Alberts 1987; Allman 1999; All-

Table 10.5 Relationships of life span residuals to whole brain residuals, neocortex residuals, and cerebellum residuals in five mammalian taxa

	Whole brain						Neocortex						Cerebellum					
	All ICs			Old ICs			All ICs			Old ICs			All ICs			Old ICs		
	r	*P*	*N*	*r*	*P*	*N*	*r*	*P*	*N*	*r*	*P*	*N*	*r*	*P*	*N*	*r*	*P*	*N*
Primates	.31	.03	52	.36	.07	26	.17	.32	35	.48	.04	18	.16	.36	35	.46	.05	18
Insectivores	.11	.76	9	*	*	*	.22	.54	9	*	*	*	.06	.87	9	*	*	*
Bats	.10	.71	16	.08	.83	8	.27	.40	18	*	*	*	.45	.14	18	*	*	*
Carnivores	.35	.02	41	.31	.16	21	*	*	*	*	*	*	*	*	*	*	*	*
Odontocetes	.40	.12	11	*	*	*	*	*	*	*	*	*	*	*	*	*	*	*

Note: Asterisk denotes insufficient data available for analysis.

man and Hasenstaub 1999). Its claim is that an evolutionary increase in brain size often delays maturation.

The first assumption of this hypothesis is that a lengthy period of brain and behavioral maturation is unavoidable. In other words, no matter what environmental stimuli or nutritional resources are available, (some) complex behaviors, and the neural circuitry underlying them, cannot emerge immediately, but instead require long maturational periods. This proposition is supported by numerous findings that most skills emerge at approximately the same age across individuals of a given species (reviewed by Parker and McKinney 1999). The paradigmatic example is language: virtually all humans, in virtually all cultures, begin to understand and produce words at roughly one year of age (Pinker 1994).

Why maturational constraints exist is not entirely clear, but one reason seems to be that brain and behavioral development occurs in a stepwise fashion: because more complex patterns of behavior and neural connectivity are built on simpler ones, complex behavior cannot emerge immediately (e.g., Hebb 1949; Piaget 1980; Case 1992; Elman et al. 1996; Bjorklund 1997). Some support for this interpretation can be drawn from research on neural networks. In general, powerful networks grow or add nodes slowly, keeping pace with their learning, so that larger networks take longer to establish (reviewed by Quartz and Sejnowski 1997).

The second assumption of the maturational constraints hypothesis is that brain and behavioral maturation often takes so long that it actually affects life history. Most importantly, the duration of brain and behavioral maturation determines the onset of sexual maturity (the hypothesis is silent with regard to juvenile growth rates). If this assumption is correct, then animals should reach neuroanatomical and behavioral markers of maturity just prior to reaching adulthood (Janson and van Schaik 1993). Studies of corticospinal pathways, the fiber tracts that transmit efferent signals from the motor cortex to the extremities, support this claim: white matter density and conduction velocity increase until the age of three years in captive macaques (for which sexual maturity may occur as early as three and a half years: Melnick and Pearl 1987) and until the mid-teens in humans (Nezu et al. 1997; Olivier et al. 1997; Paus et al. 1999). In addition, in corresponding experimental tests of fine manual motor control, there are significant improvements until three years in the macaque and the early teens in humans (reviewed by Olivier et al. 1997).

The evidence regarding development in wild primates is more difficult to evaluate. Janson and van Schaik (1993) reviewed the ontogeny of foraging skill in wild primates and concluded that, although juveniles are usually less successful than adults, this fact is better attributed to differences in body size than to differences in experience or skill (see also N. G. Blurton Jones and F. W. Marlowe, unpub.). Nevertheless, this conclusion was based on only a few types of foraging in a few species (see Janson and van Schaik, 1993, p. 64), and it is possible that there are more difficult techniques that cannot be mastered until adulthood. Indeed, there is some evidence of late-developing skills that are apparently not limited by strength. For instance, mountain gorillas employ a variety of complex manual movements in leaf gathering, and adults (nine years or older) show a larger repertoire of these techniques than subadults (Byrne and Byrne 1993). Similarly, while most types of chimpanzee feeding tool use are established by five or six years, nut cracking with stone tools is not mastered until roughly ten years (Matsuzawa 1994; Boesch and Boesch-Achermann 2000; chimpanzee sexual maturity may occur as early as eleven years: see Goodall 1986). Kaplan et al. (2000) argue that in many hunter-gatherer societies, men and women do not become fully proficient in acquiring many types of crucial resources until their third decade, or even later. Thus, although more evidence is needed, the assumption that the length of brain and behavioral maturation delays adulthood is at least plausible.

The maturational constraints hypothesis is similar to the neuronal investment hypothesis in that it is most plausibly understood as arguing for a "weak" constraint. It predicts a positive, but imperfect, evolutionary correlation between brain size and the duration of brain and behavioral development. This correlation is imperfect because in some cases brain development might not be limiting the length of the juvenile period (i.e., some animals will have periods of development that are "longer than necessary" for their brain sizes).

The life history variable that captures the developmental period best is probably age at first reproduction, but we showed above that there is little evidence of an evolutionary association between age at first reproduction and brain size in primates. To further test this prediction, we examined whether age at first reproduction and brain size are evolutionarily correlated in four nonprimate taxa: insectivores, bats, carnivores, and odontocetes. Contrary to the maturational constraints hypothesis, brain size-age at first reproduction correlations were not significant in any of these nonprimate taxa (table 10.6). Nevertheless, in odontocetes, there was one obvious and significant outlier, and when it was removed, the relationship became significant ($n = 8$, $r = .78$, $p = .008$). The outlying contrast involved the

genus *Platanista*, a riverine dolphin that apparently evolved a slow life history without a corresponding brain enlargement, an occurrence that does not contradict this hypothesis.

Deriving other predictions from the maturational constraints hypothesis is difficult. Allman and Hasenstaub (1999) suggest that, although brain size should be correlated with age at first reproduction, it should be negatively correlated or uncorrelated with gestation length. Their idea is that a shorter gestation will allow the larger-brained organism to spend a greater proportion of its development outside the womb, where, compared with prenatal development, environmental stimulation is greater; this would in turn allow maturation to occur more rapidly. We tested for evolutionary correlations between brain size and gestation length and found no evidence for them (see table 10.6). Nevertheless, we do not believe that these results truly bear on the maturational constraints hypothesis. Our objection is that environmental stimulation, although necessary for brain and behavioral development, probably does not limit it. In fact, in young animals, high levels of stimulation in one modality often interrupt development in another one (overstimulation: reviewed by Bjorklund 1997). Mounting evidence also shows that there is substantial stimulation in the womb (e.g., DeCasper and Spence 1986). Furthermore, aspects of natural history, such as relative predation risk, apparently determine the relative timing of birth (i.e., precociality vs. altriciality) independently of general fast-slow life history trends (Read and Harvey 1989). Hence, predictions regarding gestation length and brain size are probably irrelevant to the maturational constraints hypothesis.

Ross and Jones (1999) attempted to test the needing-to-learn hypothesis by asking whether evolutionary changes in the length of juvenility are negatively correlated with evolutionary changes in percentage of foliage in the diet and positively correlated with evolutionary changes in group size. They suggested that these were reasonable predictions of the needing-to-learn hypothesis because both a larger group size and a nonfolivorous diet might require more complex behavior or learning, which should take longer to master. Ross and Jones (1999) did not find evidence of the predicted relationships. Whatever their implications for the needing-to-learn hypothesis, these tests certainly do not weaken the maturational constraints hypothesis. For one thing, although these relationships are often taken for granted, group size and folivory are variables that have never been demonstrated to correlate with behavioral complexity or learning (cf. Dunbar 1992; Deaner, Nunn, and van Schaik 2000). Even more crucial is the fact that the analyses of Ross and Jones (1999) controlled for differences in brain size. According to the maturational constraints hypothesis, brain size and

Table 10.6 Relationships of brain size residuals to age at first reproduction residuals and gestation length residuals in five mammalian taxa

	Age at first reproduction						Gestation length					
	All ICs			Old ICs			All ICs			Old ICs		
	r	P	N	r	P	N	r	P	N	r	P	N
Primates	.20	.18	45	.27	.21	23	.21	.16	44	.18	.43	22
Insectivores	.15	.57	16	*	*	*	−.23	.27	24	−.42	.16	12
Bats	.23	.22	29	.47	.07	15	.21	.11	58	.09	.64	29
Carnivores	.19	.21	44	.11	.59	22	.10	.41	72	.04	.83	37
Odontocetes	.36	.26	9	*	*	*	−.44	.59	11	*	*	*

Note: All results are based on independent contrasts. Asterisk denotes insufficient data available for analysis.

behavioral demands are two sides of the same coin; thus, once brain size is controlled, juvenility *should not* be correlated with behavioral demands, even if they could be identified.

Cognitive Buffer Hypothesis

The cognitive buffer hypothesis holds that a large brain, and its concomitant behavioral flexibility and improved learning ability, serves to reduce extrinsic mortality (e.g., deaths due to food shortages or predation). Because larger-brained animals are better buffered against ecological dangers, they are likely to experience reduced mortality—a necessary condition for selection of slower life history (Allman, McLaughlin, and Hakeem 1993a; Hakeem et al. 1996; Allman and Hasenstaub 1999; cf. Kaplan et al. 2000).

The crucial underlying assumption of the cognitive buffer hypothesis is that larger brains substantially reduce extrinsic mortality. Although there are countless cases of wild animals employing unusual behavior to solve life-threatening problems, it is extremely difficult to test whether large-brained animals do this more frequently. Nevertheless, researchers have quantified the frequency of foraging innovations reported in the literature per given species and tested whether this measure is correlated with measures of brain size. Lefebvre et al. (1997, 1998; see also Timmermans et al. 2000) found significant positive cross-species correlations in birds, and S. M. Reader (unpub.) did likewise in primates. If feeding innovations do indeed lead to better survival, then these studies support the assumption that larger brains reduce mortality. In addition, cross-species comparisons of learning abilities, although contentious (e.g., Macphail 1987 and commentaries therein), have generally found that brain size (or related measures) predicts performance on learning tasks (Passingham 1975a; Riddell and Corl 1977; Rumbaugh, Savage-Rumbaugh, and Washburn 1996; R. O. Deaner et al., unpub.). Thus, although more work is needed to test this assumption, it is likely to hold.

Like the previous two hypotheses, the cognitive buffer hypothesis predicts positive, but imperfect, correlations between brain size and life history variables. Correlations will be imperfect because organisms might achieve slower life histories in other ways than through enhanced cognition, and, conversely, a larger brain could evolve without a corresponding increase in longevity. In other words, the cognitive buffer claims only that once larger brains have evolved, all else being equal, enhanced longevity becomes *more likely*.

The first prediction of the cognitive buffer hypothesis is that evolutionary changes in brain size and longevity should be positively correlated.

Several previous studies have found this association in primates (e.g., Harvey, Martin, and Clutton-Brock 1987; Austad and Fischer 1992; Allman, McLaughlin, and Hakeem 1993a), and above we showed that it holds even when conservative methods are employed to account for body size and phylogeny.

We can also test for a brain size-longevity relationship in nonprimate mammals. Earlier studies found little or no evidence for this relationship (Mace 1979; Economos 1980b; Gittleman 1986b; Read and Harvey 1989; Austad and Fischer 1991, 1992; cf. Sacher 1959, 1975; Mallouk 1975), but this could be because they did not consider the issue of phylogenetic independence. An evolutionary association between two traits could be obscured in an analysis taking species as independent data points because one (or more) major evolutionary event or grade shift (e.g., Martin 1990; Barton and Harvey 2000) at the base of the phylogenetic tree occurred contrary to the general association (Purvis and Webster 1999). Because the method of ICs calculates all changes within a phylogeny, it should not be unduly weighted by any particular event. Thus, we used ICs to test for an association between brain size and life span in carnivores, bats, insectivores, and odontocetes.

As shown in table 10.5, there was not a significant life span-brain size correlation within insectivores or bats. Within carnivores, there was a positive life span-brain size correlation in the "all contrasts" analysis, but not the "old contrasts" analysis. Within odontocetes, there was an apparent positive life span-brain size correlation, although it did not reach significance. Small sample size ($n = 11$ contrasts), partially due to several unresolved phylogenetic relationships, clearly limited statistical power. We thus decided to reconsider this relationship in an analysis taking species as independent data points. In this case, the relationship reached significance ($n = 14$, $r = .70$, $p = .005$), providing preliminary evidence of a life span-brain size linkage in odontocetes.

A second prediction can also be derived from the cognitive buffer hypothesis: if longevity and cognition are truly associated, then life span should be positively correlated with the size of the neocortex, even if it is not correlated with total brain size. This prediction derives from the fact that most structures implicated in higher-order cognition (e.g., planning, working memory) are located in the neocortex, which can therefore be considered superior to the whole brain as a cognitive assay (e.g., Sawaguchi and Kudo 1990; Dunbar 1992). Although some investigators argue that viewing any part of the brain as more "cognitive" than another is overly simplistic (e.g., Barton 1996; Barton and Dunbar 1997), to the extent that such a dis-

tinction is possible, the neocortex is clearly the best cognition candidate. We therefore tested for a neocortex-life span association within primates, insectivores, and bats. We found no evidence for this relationship in insectivores or bats, but in primates there was a significant relationship in the analysis of "old contrasts," although not in the analysis of "all contrasts" (table 10.5; cf. Allman, McLaughlin, and Hakeem 1993b; Hakeem et al. 1996). Overall, then, both predictions of the cognitive buffer hypothesis have received some support.

Brain Malnutrition Risk Hypothesis

The high energy costs of growing and maintaining the brain are often hypothesized to be linked to the evolution of brain size and life history (Martin 1981; Armstrong 1983, 1985a; Leonard and Robertson 1992; Aiello and Wheeler 1995; Martin 1996; Ross and Jones 1999; Kaplan et al. 2000). Nevertheless, evolutionary biologists have generally overlooked the implications of the growing brain's unique sensitivity to energy shortages (but see Nowicki, Peters, and Podos 1998). Here we present a brain malnutrition risk model showing that a large brain's high energy demands and extreme sensitivity to nutritional perturbations during development are most compatible with conservative brain and body growth trajectories. Hence, an evolutionary extension of the body growth period, and a corresponding decrease in the body growth rate, should increase the probability of an evolutionary increase in brain size.

This model is based on two assumptions about mammalian brain and body growth. First, if the energy needs of the developing brain are not met, even for a brief period, there is a high risk of long-lasting or even permanent brain damage and behavioral abnormalities (Shoemaker and Bloom 1977; Smart 1991; Levitsky and Strupp 1995). The timing of the energy shortage is crucial to predicting its consequences: if it occurs while the brain is growing, it will be far more debilitating than if it occurs after brain growth has been completed (Winick and Noble 1966; Shoemaker and Bloom 1977; Smart 1991; Levitsky and Strupp 1995). Because energy shortages during brain growth often lead to permanent or long-term incompetence, they are likely to severely curtail survival and reproductive success. In contrast, if body growth is stunted due to resource scarcity, the body retains a remarkable capacity to achieve the target adult size, provided resources later become abundant (catch-up growth: Prader, Tanner, and van Harnack 1963; Elias and Samonds 1977; Tanner 1986). Although the mammalian data showing the brain's relatively greater sensitivity to energy shortfalls come from only a few species, the phenomenon may occur generally (Schew and Ricklefs 1998).

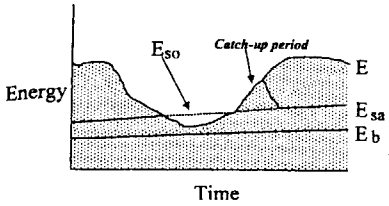

FIG. 10.3. Schematic representation of the brain malnutrition risks model. E represents the animal's potential energy intake; E_b represents the energy needed to maintain and grow the brain; E_{so} represents the energy needed to grow and maintain the soma optimally; E_{sa} represents the actual energy used to grow and maintain the soma. E fluctuates, but there is always enough energy to meet E_b. When E cannot also meet E_{so}, the soma is compromised. Later, when E is high, more energy is devoted to E_{sa}, so that the soma can return to its optimal growth trajectory. Note that E_b and E_{so} increase slowly, reflecting the demands of a growing animal.

The model's second assumption is that body growth trajectories lag behind brain growth trajectories in a similar fashion across mammals. In particular, the brain reaches its peak growth velocity and its adult size considerably before the body does (Count 1947; Holt et al. 1975; Deacon 1990). This relationship between brain and body growth trajectories probably reflects selection for a functional balance between behavioral capabilities and body size. In other words, an animal with a very small brain in an adult-sized body would probably lack the experience and motor control to effectively forage and move (see Mace 2000). Conversely, an animal possessing an adult-sized brain in an extremely small body would have great difficulty obtaining enough energy to maintain its large brain as its body grew.

Given these two assumptions, we can model brain and body growth as follows (fig. 10.3). At any given time during development, an animal has the potential to ingest a certain amount of energy, E, which fluctuates due to temporal variation in food abundance and the animal's improving foraging skills. In the allocation of E, first priority is given to the needs of the growing brain (E_b). The remainder of E is allocated to optimally growing and maintaining the soma (E_{so}). If there is not enough energy to maintain both optimal brain and optimal body growth trajectories, the brain is spared and the body is compromised (Stewart 1918; Shoemaker and Bloom 1977; Smart 1991; Peeling and Smart 1994). In this case, the actual energy devoted to the soma (E_{sa}) falls below the optimal amount (E_{so}). When E exceeds the joint needs of E_b and E_{so} (i.e., periods of superabundance), the animal limits its food consumption to maintain a functionally balanced relationship between brain and body size. If, however, the animal's body growth has been previ-

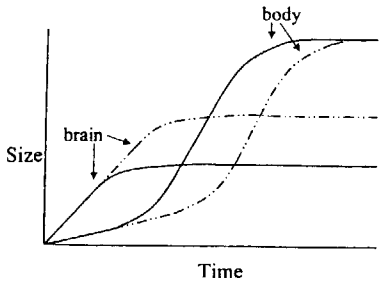

FIG. 10.4. Extension of brain growth and somatic growth could promote the evolution of a larger adult brain. In the ancestor (solid lines), brain growth occurs at a high rate and is finished quickly. Somatic growth first occurs slowly, but when brain growth is complete, it accelerates. In the larger-brained descendant (dashed lines), brain growth follows the ancestral trajectory, but continues at the high rate for a prolonged period, ultimately leading to a larger adult brain size. The descendant's somatic growth curve is also similar to the ancestor's, but the period of slow somatic growth is extended until brain growth terminates. Both the ancestor and its larger-brained descendant end up at the same adult body size, but the descendant reaches it later.

ously stunted, any surplus energy is devoted to the soma (E_{sa}) until body size has returned to its optimal trajectory.

The mean and variability of E are the main factors that select for the level of E_b during brain growth. In particular, natural selection is expected to set E_b only as high as allowed by the minimum reliable E, so that brain damage will be avoided even during periods of resource scarcity. E_{so}, in turn, will be set according to the requirement of maintaining a functional balance between brain and body size. So, although there may be some circumstances in which animals use E fully (e.g., food shortages, during catch-up growth), body growth will be generally limited by brain growth, not by total available energy. Nevertheless, once brain growth is complete, body growth may occur as rapidly as available resources permit, often resulting in growth spurts (Leigh 1996; Bogin 1999).

The evolutionary link between a slower, longer body growth trajectory and a larger adult brain size follows from ontogenetic consideration of how a descendant can achieve a larger adult brain size than its ancestor. Essentially, there are two potential evolutionary pathways by which this can occur. First, the brain may grow faster, allowing the descendant to reach a larger adult brain size without changing the ancestral duration of brain growth. Second, as illustrated in figure 10.4, the duration of brain growth may be prolonged, so that the ancestral brain growth rate eventually produces a larger brain (hypermorphosis: McKinney and McNamara 1991; McKinney 1998; see also Gould 1977). The first pathway—accelerated brain growth—

requires that more energy be allocated to E_b at any given time. Unless the minimum reliable E also increases in the descendant, which is generally unlikely (see below), this change will increase its risk of facing an energy crisis and suffering long-lasting brain and behavioral defects. Hence, if there is no evolutionary change in the somatic growth trajectory, an evolutionary increase in brain size is improbable. The second ontogenetic pathway for gaining a larger brain—extending the ancestral brain growth trajectory—is more likely because it is less risky. The risk of brain damage is low because the energy devoted to E_b at any given time remains the same as in the ancestor. Because of the functional balance requirement, an extended period of brain growth is possible only if there is an extended period of slow body growth. Therefore, on an evolutionary scale, a slower, extended body growth trajectory should increase the likelihood of an evolutionary increase in brain size.

We suggest that this argument applies throughout mammalian development, but that the degree of maternal dependence is an additional, crucial factor. In particular, during gestation and lactation (i.e., when all or most energy comes from the mother), E will tend to be high and subject to relatively less variability. This reflects the fact that adults tend to be large, efficient, and consistent foragers and may also have access to nutritional reserves. Thus, the mother serves as an energetic buffer for her offspring. Just after weaning, however, the offspring's potential energy intake will be low and variable, because it will have no foraging experience and its brain and body will not yet have achieved adult size. As the animal's brain size, body size, and experience all increase, E will increase and become less variable. In sum, the higher and more consistent energy supply during the period of maternal dependence can explain why brain growth is most rapid early in development, and why juvenile somatic growth is reduced relative to preweaning levels in animals with large brains (Janson and van Schaik 1993; Bogin 1999).[4]

The brain malnutrition risk model can be viewed as an extension of Janson and van Schaik's (1993) juvenile risks model, which holds that primate juvenility should be primarily viewed as a period of slow, conservative growth, serving to maximize survivorship to breeding age. However, unlike the juvenile risks model, the brain malnutrition risk model is able to explain why primates do not markedly increase their growth rates when food is abundant.

One objection to the brain malnutrition risk model can be derived from the "expensive tissue" hypothesis, which holds that the energetic costs of maintaining a larger brain can be paid by reducing the size of the gut, another metabolically expensive organ (Aiello and Wheeler 1995; but see

Hladik, Chivers, and Pasquet 1999). Specifically, one could argue that when E_b is raised, E_{so} could be lowered in a corresponding fashion while still achieving the ancestral body size. In other words, growing to and maintaining a certain body size may be cheaper for a larger-brained organism because a smaller gut consistently accompanies a larger brain (Foley 1995). This is an interesting idea, but it does not seriously weaken the model, the central point of which is not simply that if more energy is required to grow the brain, less must be available to grow the body. Instead, the model holds that body growth trajectories are usually determined by conservative brain growth trajectories, not by total E.

A second potential objection is that the evolution of a larger brain size is invariably accompanied by an increase in E or a reduction in E's variance. If so, then a larger-brained animal would be able to allocate more energy to both E_b and E_{so} and thus achieve a larger adult brain size without any change in life history. In this view, the proposed model becomes irrelevant because one of its assumptions—that E is unchanged by an increase in adult brain size—is met so rarely. Martin (1981, 1996) has argued similarly, noting that maternal basal metabolic rate (BMR), a measure that may be linked to E, is tightly linked to neonatal brain size. Although this neonatal brain size-maternal BMR relationship holds in primates (Martin 1996; R. A. Barton, unpub.), it does not generalize to nonprimates (Pagel and Harvey 1988a). Even more importantly, once the effects of body size have been controlled, BMR appears to be unrelated to adult brain size (McNab and Eisenberg 1989; Allman, McLaughlin, and Hakeem 1993a; Martin 1996; Barton 1999). Hence, there is little evidence that changes in adult brain size are achieved by altering E.

Therefore, we conclude that the brain malnutrition risk model is based on reasonable assumptions and is not susceptible to two potentially damaging criticisms. To further test its validity, we should derive and test additional predictions.

The first prediction of the model is that evolutionary changes in brain size should co-occur with evolutionary changes in the duration of brain growth; evolutionary changes in the rate of brain growth, by contrast, should be less common. Unfortunately, this prediction is presently difficult to test, despite the fact that patterns of brain and body growth have now been documented in approximately ten mammalian species (humans, chimpanzees, rhesus monkeys, dogs, cats, rats, mice, guinea pigs, rabbits, pigs: Holt et al. 1975; Dobbing and Sands 1979; see also Pereira and Leigh, chap. 7, this volume). The problem is that the model applies mainly to closely related species that share broadly similar body sizes and foraging patterns; in these cases, E is likely to be similar. The species in which brain and body growth patterns have been documented, however, are highly diverse in these characteristics and thus cannot offer reasonable tests. Nevertheless, one relevant comparison is clearly in agreement with the prediction: chimpanzees versus humans. These closely related species are similar in body size and BMR (Aiello and Wheeler 1995), but have dramatically different adult brain sizes. Crucially, they have highly similar early brain and body growth trajectories; the difference in adult brain size is due to the fact that humans greatly extend the most rapid period of brain growth and reach adult brain and body size much later (Count 1947; Passingham 1975b; Holt et al. 1975; McKinney and McNamara 1991).

A second prediction of the model is that if resources are abundant, a somatic growth spurt will occur shortly after the termination of brain growth. This prediction follows because, according to the model, the end of brain growth permits body growth to begin closely tracking available resources (E) (see also Leigh 1996). To our knowledge, information on both brain and body growth trajectories is available for only four taxa that show somatic growth spurts: macaques, mangabeys (*Cercocebus* spp.), chimpanzees, and humans.[5] Nevertheless, these limited data support the prediction. Rhesus macaques complete brain growth just after their first birthdays (Cheek 1975, fig. 1.7) and show somatic growth spurts as early as eighteen months (Leigh 1996, fig. 5). Mangabeys complete their brain growth between three and four years of age (Pereira and Leigh, chap. 7, this volume) and show growth spurts at the same age (Leigh 1996, fig. 6). Chimpanzees complete their brain growth between four and five years of age (Count 1947, fig. 4 and table 2), and their growth spurts begin at approximately five years (Leigh 1996, fig. 8). In well-fed humans, the adolescent growth spurt begins between ten and twelve years of age (Bogin 1999), and the vast majority of human brain growth is completed by eight or nine years, although there are slight increases until the late teens (Dobbing and Sands 1973; Dekaban and Sadowsky 1978).

Because its underlying model is reasonably well supported, the brain malnutrition risk hypothesis could be correct, and it is therefore worthwhile to consider predictions that follow directly from it. The main prediction is that there should be a modest positive correlation between evolutionary changes in brain size and evolutionary changes in the duration of somatic growth. Although there is relatively little cross-species data on growth durations, age at first reproduction is probably an excellent first approxima-

tion. Thus, the comparative tests performed above bear on the model: the fact that we found no robust correlations in any mammalian taxa fails to support the prediction.

Delayed Benefits Hypothesis

Modern life history theory holds that animals delay reproduction only if the costs of doing so are eventually offset by benefits (Williams 1957; Gadgil and Bossert 1970; Harvey, Read, and Promislow 1989; Stearns 1992; Charnov 1993). Brain investment is an excellent example of this trade-off (the immune system could be another). In particular, even if it is physiologically possible to quickly grow a large brain (see the maturational constraints hypothesis) and the energetic risks of rapid brain growth can be borne (see the brain malnutrition risk hypothesis), investing in a large brain still makes less sense for an animal with a fast life history than for an animal with a slow life history. The reasoning is as follows: A primary benefit of a large brain is that it allows an animal to develop experientially based skills or knowledge that eventually lead to fitness benefits. The development of these abilities, by definition, involves costs of time and risk. Hence, the delayed benefits of such abilities must exceed the costs of developing them. Life history is relevant because once the initial costs of learning are paid, the benefits may accrue indefinitely. Thus, long-lived animals have a greater opportunity than short-lived animals to take advantage of large brains and the learning they allow (cf. Dukas 1998; Kaplan et al. 2000). Therefore, an evolutionary increase in longevity should increase the likelihood of an evolutionary increase in brain size.

The crucial assumption of the delayed benefits hypothesis is that brain size is closely related to an animal's learning ability. As noted under the cognitive buffer hypothesis, this assumption has some support.

The first prediction of the delayed benefits hypothesis is that there should be a modest positive evolutionary correlation between brain size and life span. We have addressed this prediction under the neuronal investment and cognitive buffer hypotheses and found some evidence to support it, in both primates and nonprimates.

The second prediction of the delayed benefits hypothesis is that there should be a positive evolutionary correlation between neocortex size and life span. This prediction is again shared by the cognitive buffer hypothesis, and it likewise follows from the reasoning that the neocortex is especially implicated in learning and other higher-order cognitive processes. Although this prediction was supported in the analysis of old contrasts within primates, it was not supported at all among bats or insectivores. Thus, the main assumption of the delayed benefits hypothesis is reasonable, and the comparative tests provide some support for its predictions.

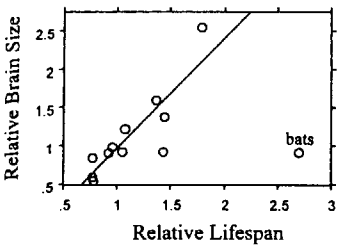

FIG. 10.5. The relationship between relative brain size and relative life span across eutherian mammals. Each point represents an "order average." The regression line is fit to all points save bats, which is a significant outlier.

Discussion

Did Brains and Life History Coevolve in Mammals?

Other than the brain size-life history correlation in primates, there is limited evidence for robust evolutionary relationships between life history variables and the size of the brain or brain structures. One might reasonably ask, then, "Is there truly a brain size-life history phenomenon?" We obviously do not yet know the answer to this question, but, for several reasons, we believe that the linkage is real.

First, although we found relatively few significant relationships, the correlation coefficients were, as predicted, overwhelmingly positive (e.g., a positive increase in brain size and a positive increase in longevity: see tables 10.5 and 10.6). The only exceptions to this pattern involved gestation length, a variable that is only weakly related to general fast-slow life history trends (see discussion of the maturational constraints hypothesis above; Read and Harvey 1989). It might be argued that the predominance of positive correlations reflects the fact that many of our analyses employed the same body mass estimates in calculating brain and life history residuals (i.e., the correlated errors problem). However, this criticism does not apply to the analyses involving primates, bats, and odontocetes.

Second, there appears to be a brain size-life span relationship across eutherian mammalian orders. Read and Harvey (1989) and Austad and Fischer (1992) did not report this relationship, but it is clear from plotting relative life span against relative brain size that the relationship is quite strong once one significant outlier—the bats—is removed (fig. 10.5; with bats: $r = .40$, $n = 12$, $p = .20$; without bats: $r = .86$, $n = 11$, $p = .0007$).[6] It could be argued that outliers should not be omitted, for they are the strongest refutation of a brain size-life history link. Although this argument applies to the "strong constraint" hypotheses, it does not pertain to any of

the hypotheses that we deem plausible. The cognitive buffer hypothesis, for instance, is perfectly compatible with the possibility that some taxa evolve adaptations other than enhanced cognition to reduce their extrinsic mortality. Flying in bats is an excellent example.

Third, because our analyses strictly controlled for body size, they might have obscured or weakened genuine relationships between cognition (or other information-processing capabilities) and life history. The problem is that larger animals could systematically have greater cognitive capabilities than smaller ones (Deacon 1997), meaning that controlling for body size would be tantamount to controlling for cognition, the variable of interest (see Harvey and Pagel 1991). Of course, the reason that we (and others) controlled for body size is that the interrelationships among cognitive capabilities, brain size, and body size are unresolved, and the prudent approach at this stage is to ensure that neuroanatomical and life history relationships do not merely reflect body size. Nevertheless, if future studies (R. O. Deaner et al., unpub.) are able to establish the interrelationships among these variables, such studies will be likely to pave the way for stronger cognition and life history correlations.

Finally, it bears noting that we found at least some support for the brain size-life history linkage in primates, odontocetes, and carnivores, but not in bats or insectivores (tables 10.5 and 10.6). The former taxa are relatively large-brained, whereas the latter are relatively small-brained (Jerison 1973; Austad and Fischer 1992; Marino 1998). Similarly, within primates, the brain size-life history linkage appears to be far stronger in haplorhines than in the relatively small-brained strepsirrhines (Allman, McLaughlin, and Hakeem 1993a; Hakeem et al. 1996; Judge and Carey 2000; see also Kappeler 1996). Thus, it is possible that the forces that produce the brain size-life history linkage operate only in large-brained taxa.

The Competing Hypotheses Are Compatible

Correlations among life history variables are widely known and theoretically expected (Williams 1957; Gadgil and Bossert 1970; Harvey, Read, and Promislow 1989; Stearns 1992; Charnov 1993). Perhaps most basically, an animal with a long life expectancy can afford to delay its reproduction. Thus, despite the fact that each of the candidate hypotheses bears most directly on either maturation or longevity, any one of them could potentially explain virtually all associations between brain size and life history. For instance, if the cognitive buffer hypothesis holds, the following scenario might unfold: an evolutionary increase in brain size is followed by an evolutionary increase in life span, which is followed by an evolutionary increase in age at first reproduction. Therefore, although it is desirable to set up comparative tests so that the hypotheses may compete (i.e., make differing predictions about the importance of particular variables), it should be remembered that such tests are unlikely to be decisive (Harvey, Martin, and Clutton-Brock 1987). We especially urge caution when investigators employ variables that are likely to differ in their degree of error (i.e., maximum longevity vs. age at first reproduction).

Another important point is that, although the hypotheses might be viewed as competing to explain variation in brain size-life history associations, two or more explanations could be correct (see Kaplan et al. 2000). The maturational constraints and cognitive buffer hypotheses, for example, are complementary. According to the maturational constraints hypothesis, a lineage undergoing selection for increased brain size would be forced to postpone sexual maturation. The cognitive buffer hypothesis would then predict that the larger brain would promote the evolution of greater longevity. The brain malnutrition risk and delayed benefits hypotheses are similarly compatible. The former holds that only when the somatic growth period has been extended can animals afford to grow large brains with minimal risk, whereas the latter argues that only when life span is extended do the delayed benefits of large brains favor their evolution. In this way, the evolution of a slower life history could favor increased brain size from the point of view of both costs and benefits.

The fact that the plausible hypotheses are compatible should not deter attempts to falsify them individually. Clearly, it is unlikely that all of them are important, and it is certainly desirable to know which ones hold, and in which taxa. As we have noted, future comparative tests of their predictions, although important for investigating the generality of the brain size-life history phenomenon, are unlikely to reject any of the plausible hypotheses conclusively. We recommend, therefore, that investigators also pay attention to testing the assumptions unique to each hypothesis.

Summary and Conclusions

In the past decade, the link between brain evolution and life history evolution has generally been deemphasized (e.g., Read and Harvey 1989; Harvey, Read, and Promislow 1989; Austad and Fischer 1992; Kappeler 1996; but see Allman 1995; Hakeem et al. 1996). This can be attributed to two factors. First, there existed uncertainty as to whether the brain size-life history correlations would remain once important confounds—body size, phylogeny, socioecology—were removed. Here we have shown that in primates, there is evidence that these correlations are robust, at least in the case of

al about roles the environment plays in shaping survival, re-
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ɔhool of Chicago. He is coeditor of *Juvenile Primates: Life His-
d Behavior,* also published by the University of Chicago Press.

ı mouse lemur carrying infant,
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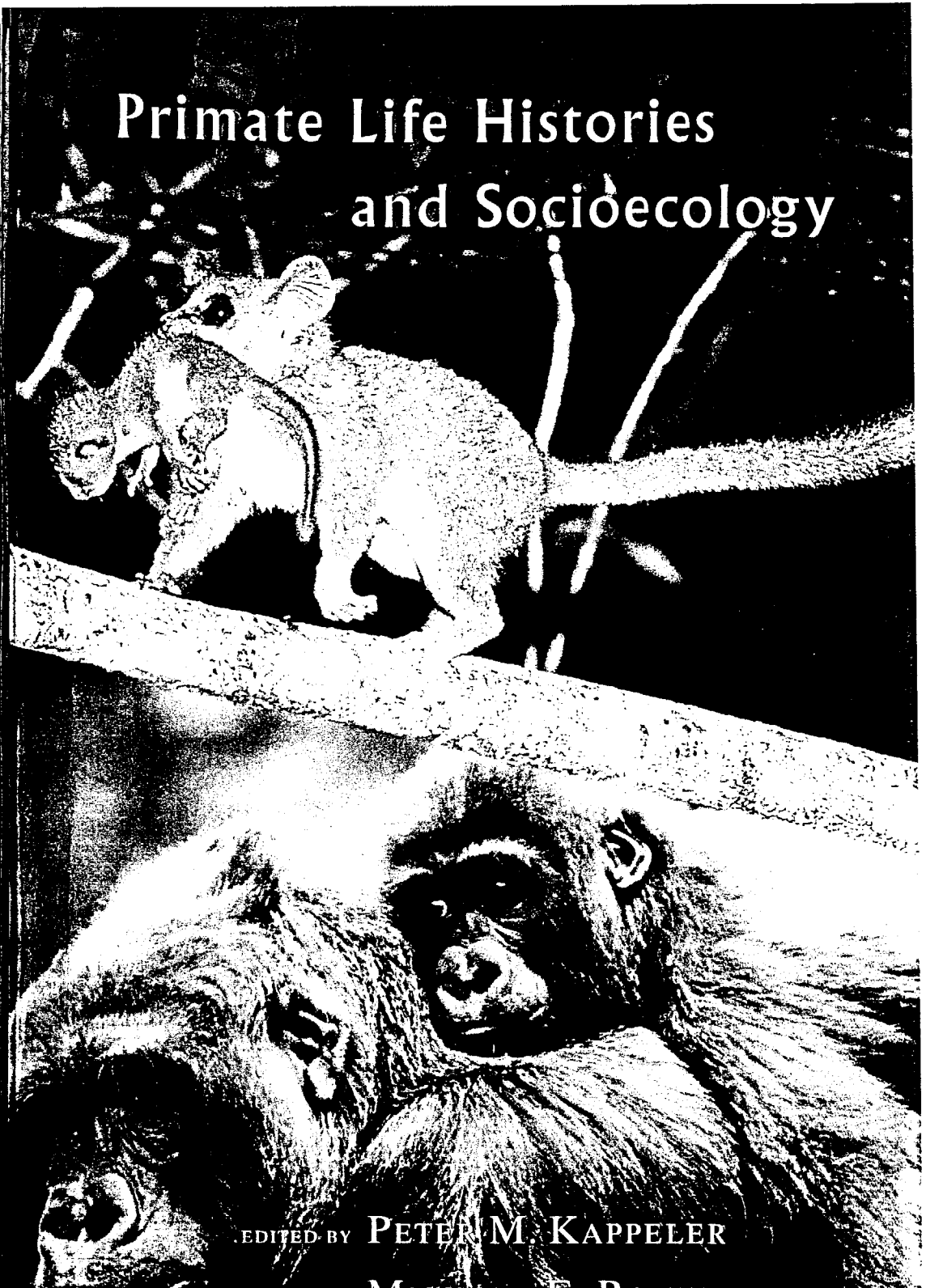

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