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How quickly do brains catch up with bodies? A comparative method for detecting evolutionary lag

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A trait may be at odds with theoretical expectation because it is still in the process of responding to a recent selective force. Such a situation can be termed evolutionary lag. Although many cases of evolutionary lag have been suggested, almost all of the arguments have focused on trait fitness. An alternative approach is to examine the prediction that trait expression is a function of the time over which the trait could evolve. Here we present a phylogenetic comparative method for using this 'time' approach and we apply the method to a long-standing lag hypothesis: evolutionary changes in brain size lag behind evolutionary changes in body size. We tested the prediction in primates that brain mass contrast residuals, calculated from a regression of pairwise brain mass contrasts on positive pairwise body mass contrasts, are correlated with the time since the paired species diverged. Contrary to the brain size lag hypothesis, time since divergence was not significantly correlated with brain mass contrast residuals. We found the same result when we accounted for socioecology, used alternative body mass estimates and used male rather than female values. These tests do not support the brain size lag hypothesis. Therefore, body mass need not be viewed as a suspect variable in comparative neuroanatomical studies and relative brain size should not be used to infer recent evolutionary changes in body size.

Keywords: evolutionary lag; comparative methods; pairwise contrasts; relative brain size

1. INTRODUCTION

Across taxa, adult brain and body mass values can be related by allometric equations of the form $\log(\text{brain mass}) \sim \log(a) + b \log(\text{body mass})$. The extent to which species deviate from such interspecific allometric lines has long been of interest because the deviations are commonly thought to indicate adaptation for neural processing or intelligence (reviewed in Deacon 1990*a*; Harvey & Krebs 1990). Nevertheless, it has been hypothesized that some residual variation around the regression line may represent instances of evolutionary lag, where brain size has not had sufficient time to catch up to change in body size (Jerison 1973; Lande 1979; Martin & Harvey 1985; Willner & Martin 1985; Deacon 1990*b*). In this view, species with relatively large brains have recently experienced a decrease in body size, while species with relatively small brains have recently experienced an increase in body size. All else being equal, it is expected that brain and body size relations will eventually return to the functional line.

Breeding experiments provide good evidence that body size can undergo selection independent of brain size in the short term (Riska & Atchley 1985). However, the claim that brain size lag can persist over extended evolutionary periods is not as well supported. In fact, it is based on one line of evidence: the existence of the taxon-level effect (TLE) (e.g. Gould 1975; Martin & Harvey 1985; Pagel & Harvey 1989). The TLE refers to the phenomenon that interspecific brain:body mass regression coefficients differ appreciably depending on the taxonomic level under consideration. For instance, when regression coefficients are calculated across all species of a genus values are typically 0.3–0.5, but when they are calculated across family averages values range up to 0.75 (Martin & Harvey 1985). Although workers have often cited the existence of the TLE as indicating evolutionary lag (Lande 1979; Martin & Harvey 1985; Willner & Martin 1985; Aboitiz 1996), this evidence is inconclusive. The main problem is that the theoretical basis for the TLE as an indicator of evolutionary lag has never been clarified. Lande (1979) suggested that evolutionary lag might produce a TLE under some sort of species selection. Other solutions are also possible (R. O. Deaner, unpublished data) but, like Lande's (1979), they require additional, as yet unverified assumptions about how evolution has occurred. Furthermore, it is now known that the TLE can be explained without recourse to the lag hypothesis: Pagel & Harvey (1989) showed that when realistic regression models are employed and major ecological differences are accounted for, the TLE disappears in most mammalian taxa (see also Pagel & Harvey 1988; Harvey & Pagel 1991). Thus, although the brain size lag hypothesis is widely accepted (Dunbar 1992, 1998; Aboitiz 1996; Deacon 1997), there is no empirical support for it (Pagel & Harvey 1989; Barton 1998).

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Here we address the brain size lag hypothesis in a novel way. Specifically, we test the prediction that evolutionary changes in brain size should lag behind evolutionary changes in body size as a function of how much time is available for brain size response. To test this prediction, we employ a method for examining time-dependent evolutionary changes based on the phylogenetic comparative method of independent contrasts (Felsenstein 1985). We test this prediction in primates, a taxon for which there is excellent morphological and phylogenetic information. Our general approach, however, can be applied to any system where one trait is hypothesized to lag in relation to another.

2. METHODS

(a) *Rationale*

Our general approach is illustrated in figure 1. To test the brain size lag hypothesis, information is needed on evolutionary changes in brain and body mass and the time over which these changes took place. Evolutionary changes can be represented with pairwise contrasts, which are calculated as the difference in the value of a trait between two living species (Felsenstein 1985). The time over which the changes occurred is estimated as the time since the paired species diverged.

Contrasts can be either positive or negative. To test the lag hypothesis, we force the independent variable (body mass) to be positive; the direction of subtraction is then retained for calculating contrasts in the dependent variable (brain mass), so that these contrasts are either positive or negative. Thus, if brain size lags occur, then, for a given positive contrast in body mass, the magnitude of a brain mass contrast will be a function of the amount of time available for this change to take place. Statistically, brain size lag can be detected by examining residuals calculated from the regression of brain mass contrasts on body mass contrasts. According to the lag hypothesis, smaller residuals (i.e. a relatively small brain mass change for a given positive body mass change) will be associated with shorter divergence times.

(b) *Calculating pairwise contrasts*

To pair species, we used a method of pairwise contrasts which is similar to that of Møller & Birkhead (1992) and Mitani *et al.* (1996). We implemented this method by first identifying all species for which brain and body mass information was available. We then used standard methods (Felsenstein 1985) to pair the most closely related species which had not been previously paired.

Although this pairwise method produces fewer species comparisons than do standard independent contrasts methods (a maximum of $n/2$ versus $n-1$, where n is the the number of available species), it has a crucial advantage in the present context: because comparisons are always between extant species, each contrast requires only a single estimate of the divergence time between the two taxa (e.g. t_1 for contrast 1 in figure 1). In comparison, determining divergence dates for non-terminal independent contrasts (i.e. higher nodes) requires three divergence estimates, one for each of the nodes involved (e.g. in figure 1, estimating the non-terminal contrast, indicated with dashed lines, requires dates for t_1, t_2 and the common ancestor of these nodes). Estimating each of these dates would therefore add additional error to the analysis. Thus, we opted for fewer contrasts but ones which should be more accurate.

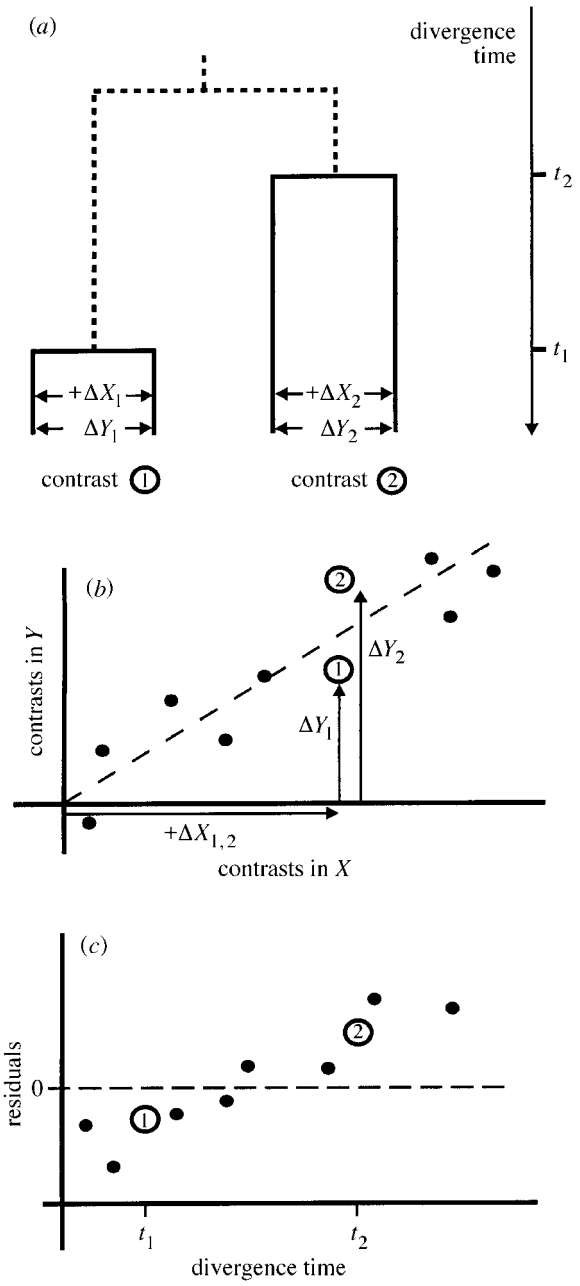

Figure 1. Detecting evolutionary lag using pairwise contrasts. (*a*) Evolutionary change is represented by pairwise contrasts (ΔX_1, ΔX_2, ΔY_1 and ΔY_2). Contrasts are calculated from living species only (i.e. no interior nodes are reconstructed). Contrasts in the independent variable (body mass) are constrained to be positive and divergence time (t) is the time at which the species last shared a common ancestor. For simplicity, only two sets of pairwise contrasts are shown here (dark branches of the phylogeny, labelled as 1 and 2). In this example, which illustrates a case of evolutionary lag, $\Delta X_1 = \Delta X_2$, $\Delta Y_1 < \Delta Y_2$ and $t_1 < t_2$. (*b*) Contrasts are plotted and a least-squares regression line is calculated. Numbered circles correspond to the two sets of contrasts from (*a*), while solid circles indicate other pairwise contrasts not shown in (*a*). If lag exists, then paired contrasts with shorter divergence times (i.e. contrast 1) will have less time available for change in the dependent Y variable (brain mass); Y variable contrasts will therefore tend to be smaller and so will have negative residuals from the empirically fitted regression line. (*c*) Evolutionary lag can therefore be detected by plotting residuals against divergence time: a positive correlation would support the lag hypothesis.

(c) *Data*

Measurements of brain and body mass were taken as means from Bauchot & Stephan (1966, 1969), supplemented with additional unpublished data provided by Dr H. Stephan. This data set contains information on more than 500 individuals of 81 species. Although the data set of Harvey *et al*. (1987) provides information on more species, the Stephan data set is preferable for two reasons. First, the Stephan data set provides actual measurements of brain and body mass, rather than estimates based on cranial capacity and other morphological characteristics (i.e. cranial capacity is an imperfect predictor of brain volume for large-bodied animals; Jerison 1973). Second, many of the specimens in the Stephan data set are of known sex. Although most brain analyses take the mean values from all available specimens, this practice ignores the fact that the sexes may differ considerably in brain and body size relationships (e.g. Willner & Martin 1985; Jacobs 1996). We performed analyses on both sexes separately in order to offer the most sensitive tests. All body mass and brain mass values were log transformed prior to analysis.

In order to account for phylogenetic relationships, we used the composite estimate of primate phylogeny provided by Purvis (1995). We chose this tree because it provides divergence date estimates, which are necessary for the analyses. Not all of the nodes in Purvis' (1995) tree are fully resolved. In cases of polytomies, the species represented with the fewest specimens was excluded from the analysis. When this still left ambiguities over which taxa to include, we calculated pairwise contrasts between all possible remaining contrasts and used only the two taxa that provided the largest body mass contrast.

The majority of divergence dates were taken as the mean values presented in Purvis' (1995) tables. In the case of platyrrhines, we also used information from Barroso *et al.* (1997), as this information clarified ambiguity in this uncertain clade and was unavailable to Purvis (1995). In cases where these sources did not provide divergence dates, we obtained information from other sources (Groves 1993; Delson 1994). If information could not be obtained in this way, we implemented a variant of Losos' (1990) rule by spacing nodes evenly along branches. However, contrary to Losos (1990), we only implemented this rule if the immediately surrounding nodes were dated (including terminal tips, dated at zero million years before present (Myr BP)) and if the time between these surrounding nodes was less than two million years. For instance, in the comparison of the patas monkey *Erythrocebus patas* and the vervet monkey *Cercopithecus aethiops*, the nearest surrounding nodes are dated at 4.0 and 3.0 Myr BP; we therefore used a value of 3.5 Myr BP. Contrasts in body mass and brain mass and the time since the paired species diverged are presented in table 1 in electronic Appendix A on the Royal Society's web site at www.pubs.royalsoc.ac.uk/publish/pro_bs/rpbl420.htm.

(d) *Statistical procedures, confounding variables, assumptions and uncertainty*

We calculated brain mass contrast residuals from the regression of brain mass contrasts on body mass contrasts. In calculating the residuals, we used the least-squares regression technique so that residuals would be uncorrelated with body mass contrasts (Harvey & Pagel 1991). Following standard practice, we forced this regression line through the origin (Harvey & Pagel 1991; Garland *et al*. 1992). We tested for a significant relationship between brain mass contrast residuals and time since divergence using the Pearson correlation coefficient. All probabilities reported are for two-tailed tests.

An important statistical issue is the possibility of an association between divergence time and body mass change. In other words, there might be more change on longer branches (e.g. Brownian motion model; Felsenstein 1985, 1988). Although we are examining residuals, our general approach involves attempting to explain variation in one dependent variable (brain mass change) with two 'independent' variables (body mass change and time since divergence). If the two independent variables are not actually independent, then assessing their separate effects is highly problematic (i.e. collinearity; Kachigan 1991). Fortunately, for the contrasts used in our analyses, there was no correlation between divergence times and body mass contrasts (females $n = 24$ observations, $r = 0.06$ and $p = 0.78$, and males $n = 22$ observations, $r = -0.12$ and $p = 0.58$) so we did not have to deal with this problem.

We did not standardize branch lengths for time since divergence (Felsenstein 1985), as in the computer program CAIC (Purvis & Rambaut 1995), because our goal was to examine residuals relative to divergence dates. However, we did check for heteroscedasticity by examining the association between absolute brain mass contrast residuals and body mass contrasts for our two main data sets. In neither case was there a significant association (females $n = 24$, $r = -0.08$ and $p = 0.36$, and males $n = 22$, $r = 0.11$ and $p = 0.60$), indicating that standardization was unnecessary.

Correlations between ecological factors and brain size are well-documented and may confound our analyses. For example, diet is thought to affect brain size, with frugivorous taxa having larger brains than folivorous ones (Harvey & Krebs 1990). Because ecological transitions may be more likely to occur on longer branches, support for the lag hypothesis might be an artefact of ecological factors (Pagel & Harvey 1989). We dealt with this problem by examining contrasts in 'ecological syndromes', as used by Nunn & Van Schaik (1999; similar to the ecological categories of Clutton-Brock & Harvey (1977)). Although Nunn & Van Schaik (1999) presented information on many ecological variables, we only used information on activity timing (e.g. nocturnal) and diet because these are the variables thought to be most correlated with relative brain size (Harvey & Krebs 1990). In addition, we considered the possible confounding effects of group size because several recent analyses have suggested that this variable may be important in explaining variation in brain structures (Dunbar 1992, 1998; Barton & Dunbar 1997). Data on (population) group size was also taken from Nunn & Van Schaik (1999) and log transformed before analysis.

We incorporated socioecological information in two ways. First, we eliminated contrasts where activity timing or diet differed between the paired species and repeated the analysis with the remaining contrasts (see Appendix A on the Web site). In addition, for these analyses we eliminated the contrast between humans (*Homo sapiens*) and chimpanzees (*Pan troglodytes*) because of the fundamental differences in lifestyle between these species and the extraordinary brain expansion in humans. Second, to examine the possible confounding effects of group size, which is a continuous variable, we used multiple regression through the origin to test whether divergence dates predict brain mass contrast residuals when group size contrasts are incorporated.

Contrasts assume that the overall difference between two species in a trait represents, on average, the total evolutionary change in that trait since the species diverged. However, if there is a trend such that a trait has regularly decreased or increased over evolutionary time in all taxa of a particular clade (Garland

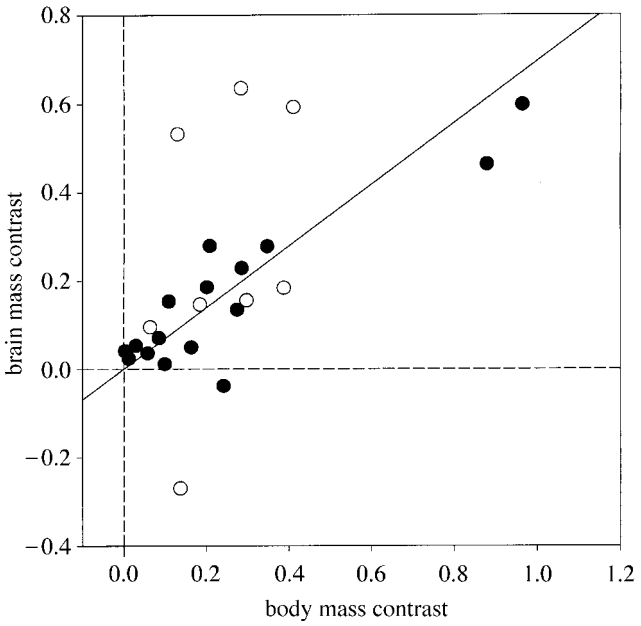

Figure 2. Regression of brain mass contrasts on body mass contrasts for female primates. Solid circles are non-ecological contrasts and open circles are ecological contrasts. The regression line is least-squares regression through the origin and includes all pairwise contrasts.

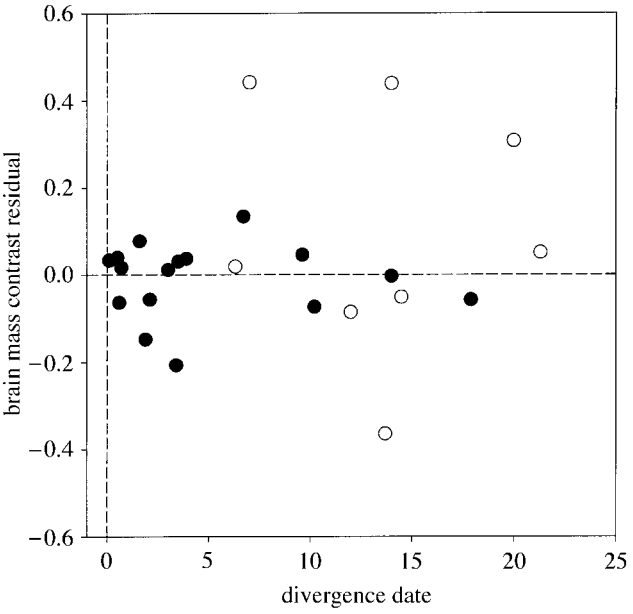

Figure 3. Brain mass contrast residuals plotted against time since divergence for female primates. Brain mass contrast residuals were calculated by regressing brain mass contrasts on body mass contrasts. Solid circles are non-ecological contrasts and open circles are ecological contrasts.

et al. 1993), then contrasts calculated between extant species may consistently underestimate actual evolutionary change. For instance, two extant species might have the same brain size, not because their ancestor had that brain size, but because the ancestor had a smaller brain and both daughter lineages independently evolved larger brains. Pertinent here is that relative brain size is known to have increased during primate evolution (Jerison 1973; Pickford 1988; Martin 1990). However, with the exception of the hominids, it appears that this trend has slowed markedly since the Oligocene period, approximately 25 Myr BP (Pickford 1988). Therefore, we excluded contrasts where species diverged prior to 25 Myr BP (e.g. red colobus *Colobus badius* versus golden lion tamarin *Leontopithecus rosalia* in Appendix A).

Brain mass is thought to be subject to relatively little sampling error (Stephan *et al*. 1981; Pagel & Harvey 1988); thus, we do not deal with variation in brain mass here. Body mass, however, can be influenced by numerous environmental factors (e.g. obesity and pregnancy) and, hence, is more problematic (Pagel & Harvey 1988; Dunbar 1992; Smith & Jungers 1997). To explore this issue, we repeated all tests using alternative body mass estimates. These alternative estimates were compiled from Plavcan & Van Schaik (1997, first value in their Appendix A) and, secondarily, from Smith & Jungers (1997, first value in their table 5). These sources reported data almost exclusively from wild specimens and, in the few cases where data was reported from captive specimens, there was no indication that these specimens were also included in the data sets of Bauchot & Stephan (1966, 1969) or H. Stephan (unpublished data). In the case of the sportive lemur *Lepilemur mustelinus*, we found no alternative estimate and therefore used the original information even in alternative tests.

3. RESULTS

(a) *Females*

We were able to calculate 24 pairwise contrasts dated at less than 25 Myr BP for female primates (see Appendix A on Web site). From these contrasts, we calculated brain mass contrast residuals from the regression of brain mass contrasts on positive body mass contrasts ($b = 0.70$ and $p < 0.0001$; figure 2). Contrary to the lag hypothesis, divergence dates were not significantly correlated with brain mass contrast residuals ($r = 0.15$ and $p = 0.48$; figure 3). Using alternative body mass estimates produced a weaker relationship between brain and body mass contrasts ($b = 0.60$ and $p < 0.0001$) and the resulting brain mass contrast residuals were again uncorrelated with divergence dates ($r = 0.16$ and $p = 0.47$).

Next we explored the possibility that ecology obscured the lag effect. First, we repeated the analysis with the nine discrete ecological transitions removed. The regression of brain mass contrasts on body mass contrasts for the 15 remaining contrasts was similar to that obtained when the ecological contrasts were included ($b = 0.64$ and $p < 0.0001$). The brain mass contrast residuals calculated from this regression were uncorrelated with divergence dates ($r = -0.08$ and $p = 0.77$). Alternative body mass estimates yielded a similar regression of brain mass contrasts on body mass contrasts ($b = 0.70$ and $p < 0.0001$) and the resulting brain mass contrast residuals were again uncorrelated with divergence dates ($r = 0.08$ and $p = 0.78$). Second, we examined whether incorporating the effects of group size would reveal a lag effect. Divergence dates did not explain significant variation in brain mass contrast residuals with group size contrasts entered into a multiple regression model (d.f. $= 18$, $F = 0.45$ and $p = 0.51$ and group size $F = 1.94$ and $p = 0.18$). Repeating this analysis with alternative body mass estimates produced the same result (d.f. $= 18$, $F = 0.061$ and $p = 0.81$ and group size $F = 0.189$ and $p = 0.67$).

(b) *Males*

For males, we were able to date 22 contrasts within the last 25 Myr BP (15 of these were the same pairings as for

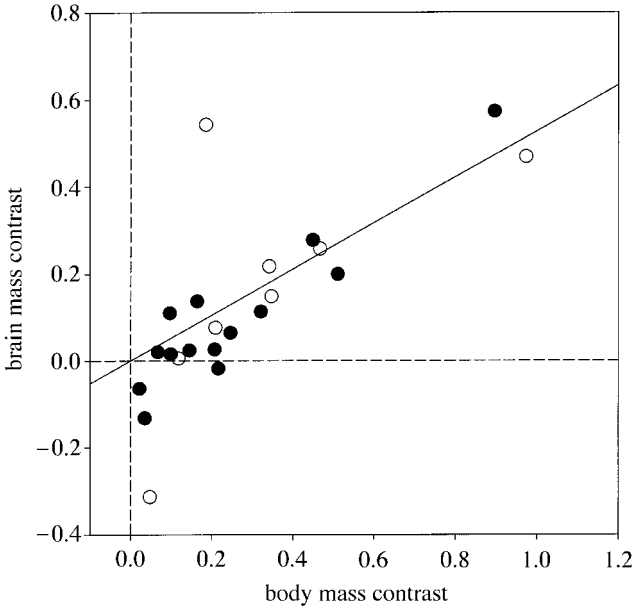

Figure 4. Regression of brain mass contrasts on body mass contrasts for male primates. Solid circles are non-ecological contrasts and open circles are ecological contrasts. The regression line is least-squares regression through the origin and includes all pairwise contrasts.

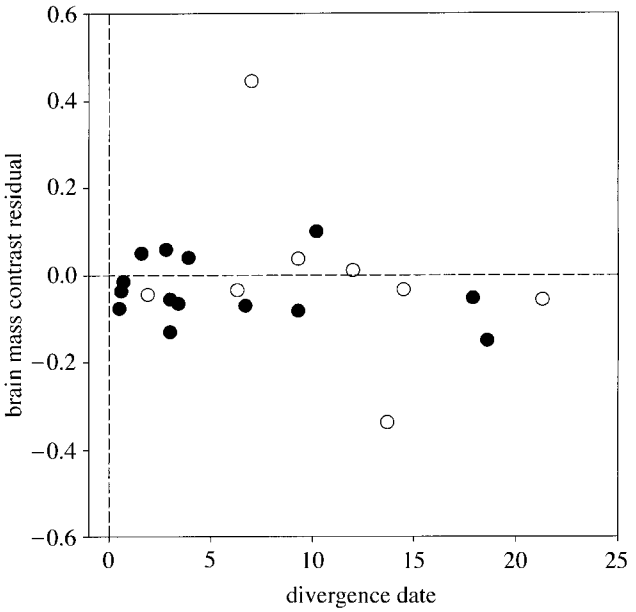

Figure 5. Brain mass contrast residuals plotted against time since divergence for male primates. Brain mass contrast residuals were calculated by regressing brain mass contrasts on body mass contrasts. Solid circles are non-ecological contrasts and open circles are ecological contrasts.

females; see Appendix A). The brain mass contrast residuals, resulting from the regression of brain mass contrasts on body mass contrasts ($b = 0.53$ and $p < 0.0001$; figure 4), were uncorrelated with divergence dates ($r = -0.20$ and $p = 0.38$; figure 5). With alternative body mass estimates, the regression of brain mass contrasts on body mass contrasts had a smaller regression coefficient ($b = 0.46$ and $p < 0.0001$) and the brain mass contrast residuals were again uncorrelated with divergence dates ($r = 0.02$ and $p = 0.92$).

With the nine discrete ecological contrasts removed, the brain mass contrast on body mass contrast regression was similar to the previously calculated regressions ($b = 0.56$ and $p < 0.0001$) and the resulting brain mass contrast residuals were uncorrelated with divergence dates ($r = -0.26$ and $p = 0.37$). With alternative body mass estimates, the regression of brain mass contrasts on body mass contrasts was similar ($b = 0.54$ and $p < 0.0001$) and the resulting brain mass contrast residuals were again uncorrelated with divergence dates ($r = 0.42$ and $p = 0.13$). In comparison to the rest of the tests, divergence dates explained significant variation in brain mass contrast residuals with group size contrasts entered into a multiple regression model (d.f. = 16, $F = 6.96$ and $p = 0.01$ and group size $F = 3.1$ and $p = 0.09$). However, the divergence date partial regression coefficient was negative ($b = -0.0005$), which is contrary to the lag hypothesis. Finally, alternative body mass estimates did not reveal that divergence dates were associated with relative brain mass once group size was included (d.f. = 16, $F = 0.338$ and $p = 0.57$ and group size $F = 0.934$ and $p = 0.35$).

4. DISCUSSION

Here we have addressed the lag hypothesis by testing its prediction that evolutionary changes in brain mass lag behind evolutionary changes in body mass as a function of how much time is available for brain mass response. We used pairwise contrasts of extant species to estimate evolutionary changes in body and brain mass and the time since the species last shared a common ancestor to estimate how much evolutionary time was available for brain mass change to take place. Our tests did not find that time since divergence was significantly associated with brain mass contrast residuals and so do not support the hypothesis that brain size lags are persistent enough to explain interspecific variation in brain size (Jerison 1973; Lande 1979; Martin & Harvey 1985; Willner & Martin 1985; Deacon 1990*b*). We found the same result when we accounted for socioecology, used alternative body mass estimates and used male rather than female values.

(a) *Alternative explanations for a negative result*

There are several potential explanations as to why the lag hypothesis might not be supported by our tests, even if it is correct. First, the data in the analysis may not have been accurate enough to provide sufficiently sensitive tests. This is unlikely for body mass, brain mass and ecological classifications because, using this data, we have found the well-known relationships between ecology and relative brain mass (Clutton-Brock & Harvey 1980; Harvey & Krebs 1990; R. O. Deaner, unpublished data). In addition, analyses using alternative estimates for the most problematic variable, body mass, gave the same results. The fact that group size was not a significant predictor of relative brain mass in multiple regression analyses might seem surprising in view of the social brain hypothesis (Barton & Dunbar 1997; Dunbar 1998). However, previous analyses have documented relationships between group size and the neocortex, not the whole brain.

A second potential source of error is phylogenetic information, including both the topology of pairwise contrasts and their dates of divergence. Topological error is unlikely to have obscured lag effects because alternative

phylogenetic trees (e.g. Fleagle 1988) produce only slightly different pairwise contrasts (R. O. Deaner, unpublished data). Moreover, we performed analyses for males and females separately. Because the availability of sex-specific information differed by species, the pairwise contrasts varied somewhat between the sexes. Nevertheless, the lag hypothesis was not supported within males or females, suggesting that the tests were not sensitive to specific contrast pairings.

However, errors in divergence date estimates cannot be as easily discounted, since we did not repeat the tests with an alternative, independent set of divergence dates. Unfortunately, such an alternative data set does not exist and would be difficult to generate as our main source (Purvis 1995) already incorporates most of the information in the literature. However, statistical tests did not approach significance except in one case where group size was included and this significant relationship went in the direction contrary to the lag hypothesis.

A third reason lag effects may not have been identified is that lags persist only for fairly short periods. In other words, if we had restricted our analysis to the most recent contrasts, lags would have been detected. Contrary to this suggestion, when we repeated our tests with only non-ecological contrasts dated at less than 4 Myr BP, we again found no association between brain mass contrast residuals and divergence dates (females $n=10$, $r=-0.11$ and $p=0.77$, and males $n=9$, $r=0.21$ and $p=0.60$).

Finally, it is possible that we have not identified lag effects because our method is somehow unsuitable for detecting them. The main difficulty we envision is that our method requires either that (i) most body mass change occurs coincidentally with the diversification of paired species, or that (ii) body mass change occurs, on average, further back on longer branches. The only way to test these assumptions is by studying the fossil record. Unfortunately, conclusive results may not emerge since fossil body size estimates typically have wide confidence intervals (Fleagle & Kay 1985; Smith 1996). Besides testing these assumptions, however, it is also possible to investigate the lag hypothesis with additional comparative tests. For instance, it can be predicted that, in lineages characterized by high speciation rates (e.g. Cercopithecidae; Purvis *et al*. 1995; Paradis 1998), brain and body size changes should be particularly decoupled, since extant species in these lineages are more likely to have recently changed in body size. Similarly, if one sex is especially prone to intrasexual selection and rapid changes in body size, then that sex can be predicted to have relatively uncorrelated brain and body mass changes (see Willner & Martin 1985). We have undertaken preliminary assessment of these alternative predictions and have found no support for the lag hypothesis (R. O. Deaner and C. L. Nunn, unpublished data).

In summary, it is likely that our tests did not detect brain size lags because they do not occur. It is important to note that this conclusion applies only to the hypothesis that brain size lags explain interspecific variation in brain size. Our tests cannot address a weaker version of the lag hypothesis, namely that cases of brain size lag occur in nature but are too ephemeral or rare to be detected with interspecific analyses.

(b) *Implications*

Our study has several implications. First, the results presented above indicate that relative brain size should not be used as an indicator of recent body size selection (Bauchot & Stephan 1969; Stephan 1972; Jerison 1973; Gould 1975; Willner & Martin 1985; Martin *et al*. 1994). For example, Willner & Martin (1985; see also Martin *et al*. 1994) attempted to discern whether sexual dimorphism was produced by evolutionary changes in male or female body size, reasoning that a relatively large female brain indicates a recent evolutionary reduction in female body size while a relatively small male brain indicates a recent evolutionary increase in male body size. Given the lack of evidence for brain size lag, Willner & Martin's (1985) conclusion—that sexual dimorphism in primates was produced mainly by reductions in female body size—should be re-examined.

Second, the absence of brain size lag bears upon the question of how brains and brain structures should be scaled in comparative neuroanatomical studies. Most recent studies of brain structure size, particularly in primates, have examined the size of brain structures relative to one another (e.g. the neocortex relative to the brainstem) rather than relative to body mass (e.g. Dunbar 1992; Sawaguchi 1992; Barton & Purvis 1994; Barton & Dunbar 1997; Lefebvre *et al*. 1997). This method has been used mainly because brain size lag was assumed (Dunbar 1992, 1998; Barton & Purvis 1994; Barton & Dunbar 1997) and because body mass estimates were thought to be subject to great sampling error (Dunbar 1992, 1998). We have shown that there is no evidence for the persistence of evolutionary lag. Sampling error may also be less problematic than often supposed. For example, pairwise contrasts of body size calculated from our original and alternative data sets are in excellent agreement (females $n=31$, $r=0.89$ and $p<0.0001$ and males $n=32$, $r=0.92$ and $p<0.0001$). Since these two criticisms of employing body mass are weak, investigators should give the question of how to scale brain structures renewed attention (Deaner *et al*. 2000).

Finally, our study illustrates an alternative approach to examining claims of evolutionary lag in other systems. While many such claims have been made, the arguments supporting them have generally been couched in terms of putative fitness (e.g. Van Schaik & Kappeler 1996; Sterck 1998) and plausible adaptive counter-arguments can usually be formulated. For example, workers have suggested that, in some bird species, the absence of defences for expelling parasitic eggs indicates evolutionary lag (Mayfield 1965; Rothstein 1982; Davies & Brooke 1989), while other workers have argued that the absence of defences may reflect their costs and thus may be adaptive (Rohwer & Spaw 1988; Lotem *et al*. 1992; Takasu 1998). In contrast, the 'time' approach examines an alternative, independent prediction: trait expression is a function of the time over which the trait could have evolved. The advantage of this approach is that if time dependency were demonstrated, it would be difficult to develop an alternative explanation for the pattern. Nevertheless, the time approach has been seldom used, probably because it requires good estimates of the temporal pattern of trait change. We suggest that this problem can be

rectified with phylogenetic comparative methods such as the one presented here.

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