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Lag Effects in Primate Brain Size Evolution:

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A Re-Evaluation

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Abstract

21 The question as to whether there is a lag between brain and body mass evolution was
 22 ostensibly solved two decades ago by Deaner & Nunn (1999) who used phylogenetic
 23 methods to show that there was no evidence to suggest that changes in brain size
 24 lagged behind changes in body size. However, their assumption that body size would
 25 always change ahead of brain size is open to question. In addition, many of their
 26 datapoints are confounded by grade shift effects. A reanalysis of their data controlling
 27 for these confounds shows that there is in fact a strong lag effect, but that the direction
 28 of the lag is the reverse of that originally assumed: brain size typically changes first,
 29 and does so under selection from changes in group size. The data suggest that it takes
 30 about 2.0 million years for body size to converge back onto the conventional
 31 allometric relationship with brain size. In the meantime, species that have increased
 32 brain size are likely to incur a significant energy cost that must be met from
 33 elsewhere. I show that they seem to do so by changing to a more nutrient-rich diet.

34

35 **Key words:** brain mass, body mass, group size, diet, allometry, energy deficit

36

37 **1. Introduction**

38   Ever since Jerison's (1973) seminal analyses, it has been known that, across
39 mammals in general and primates in particular, brain size is correlated with body
40 mass in an allometric power relationship. Nevertheless, there is considerable variation
41 around the common regression line, and it was commonly suggested that this is a
42 consequence of a lag effect in which brain mass takes time to catch up with changes
43 in body mass (Jerison 1973; Lande 1979; Martin & Harvey 1985; Willner & Martin
44 1985; Deacon 1990b, 1997; Aboitiz 1996). This assumption is based mainly on the
45 fact that body size is relatively labile, and can vary considerably within species as a
46 function of local environmental conditions (Dunbar 1990; Bettridge et al. 2010),
47 whereas the complex interconnectivity of brain systems means that it is likely to take
48 longer to bring together the necessary genetic changes without disrupting functional
49 neural systems. Such an effect might explain the well established fact that body size
50 has outstripped brain size in most domesticated species (Hemmer 1990). It is, however,
51 assumed that, given enough time, brain and body size converge on the common
52 regression line under pressure from some form of stabilising selection. That brain and
53 body mass are not yoked in close genetic linkage is confirmed by breeding
54 experiments showing that brain size and body size can undergo independent selection,
55 at least in the short term (Riska & Atchley 1985).

56   Deaner & Nunn (1999) developed a novel method for testing the lag
57 hypothesis that involved plotting the residuals of phylogenetic contrasts in brain mass
58 regressed on contrasts in body mass for a sample of primates against date of
59 divergence. They tested the explicit hypothesis that the lag would be directional: body
60 mass would always change first (hence this was always taken as a positive change),
61 and the lag would thus necessarily be brain mass lagging behind body mass. This

62 being so, a lag, if present, should be evidenced by a positive correlation because
 63 ‘young’ nodes would consist of species pairs with negative residuals when body mass
 64 contrasts are constrained always to be positive. They found that there was no
 65 correlation between the two variables, either for males or for females, or when
 66 controlling for ecology, and concluded that there was no evidence for lag effect.

67 However, there is no principled reason why body size has to change first.
 68 Much will depend on the ecological pressures acting on brain and body mass, and
 69 these can be very different. Brain size is known to be driven by changes in the
 70 cognitive demands imposed by increasing social group size (Dunbar 1998; Perez-
 71 Barberia et al. 2007; Shultz & Dunbar 2007; Dunbar & Shultz 2007, 2010). If brain
 72 size is responding mainly to pressures to evolve larger group sizes but body size
 73 responds mainly to ecological pressures (e.g. nutrient availability), then the two need
 74 not be in close linkage. A further problem that emerges with their analysis is that
 75 many of the nodes they use are not closely related and involve major grade shifts,
 76 potentially resulting in further confounds. In this paper, I reanalyse their data and
 77 show that there is, in fact, clear evidence for a lag effect.

78

79 **2. Methods**

80 In order to ensure that any differences between Deaner & Nunn’s original
 81 results and the new analyses are due to methodology and not to different data
 82 samples, I use the dataset provided by Deaner & Nunn (1999). Their data are CAIC
 83 contrasts without reference to divergence times on the grounds that they wanted to use
 84 divergence time as an independent variable in the analysis. They explicitly used only
 85 nodes that were tip comparisons (i.e. comparisons between living species) and
 86 avoided nodes at higher levels in the phylogeny (which hence have to be estimated).

87 They used the Stephan et al. (1969) brain dataset because this provides data on actual
 88 brain and body masses for the same individual specimens. This dataset yields a set of
 89 24 nodes for which contrasts in brain mass and contrasts in body mass are available.
 90 Although a much larger sample of species is available for cranial volumes (e.g. Isler
 91 et al. 2008), Deaner & Nunn (1999) argued (rightly) that these datasets risked
 92 introducing unnecessary error variance: their main problem is that the body mass and
 93 cranial volume data derive from different animals. I did, however, check out this data
 94 source, but the sample size is no larger once the data are filtered for group size and
 95 divergence dates. I use the divergence times as given by Deaner & Nunn (1999)
 96 (based on Purvis 1995) as well as more recent estimates provided by Perelman et al.
 97 (2011). Group size and dietary data for individual species are from Campbell et al.
 98 (2007), with the exception of baboon group sizes which derive from Bettridge et al.
 99 (2010).

100 Where we are testing a directional hypothesis, one-tailed statistical tests are
 101 appropriate: in such cases, a significant correlation in the opposite direction would be
 102 evidence *against* the hypothesis. In testing all other hypotheses, 2-tailed tests are
 103 used.

104

105 **3. Results**

106 Fig. 1 plots the residuals in the contrasts for brain mass as a function of body
 107 mass against divergence time. Essentially these are the data as presented by Deaner &
 108 Nunn (1999) and are the data they use to justify their conclusion. Deaner & Nunn
 109 (1999) tested for a positive relationship with time, but found a regression slope that
 110 did not differ significantly from $b=0$, and concluded from this that there was no
 111 evidence for any lag effect. However, inspection of Fig. 1 indicates that the datapoints

are evenly distributed either side of zero, with a variance that has a funnel shape, so it is no surprise that there was the correlation did not differ from zero: this is simply a consequence of using residuals from the common regression. More importantly, a funnel-shaped pattern of variance is exactly what would be expected if there is a lag effect but the direction of the lag is unspecified (i.e. sometimes body size changes first, and sometimes brain size changes first).

This being so, absolute residuals should be plotted against divergence date (Fig. 2). The relationship is clearly negative, albeit not significant (Spearman $r_s = -0.074$, $p = 0.362$ 1-tailed). One obvious problem with these data is that they mix contrasts of very different taxonomic status, namely contrasts between closely related species (i.e. those within the same genus) and species that belong to different genera, and in some cases even different families (highlighted as solid and open symbols). Most of the latter are strepsirrhines, but they also include the contrast between *Homo* and *Pan* and that between *Allouatta* and *Lagothrix*, both of which involve very large differences in sociality (especially group size) as well as brain volume. These risk confounding the lag relationship with major grade shifts in brain size (see Aiello & Dunbar 1992; Dunbar 1993). This is especially problematic for the strepsirrhines, where there are grade shifts between nocturnal and diurnal species (Barton 1998), as well as the grade shift between strepsirrhines and haplorhines. They also all entail large differences in sociality involving the transition between semi-solitary species and those that live in multimale/multifemale groups. If we consider only pairwise comparisons for closely related species (i.e. those belonging to the same genus), the correlation between residual brain mass and divergence date is in fact highly significant ($r_s = -0.593$, $N = 13$, $p = 0.017$ 1-tailed). In contrast, the correlation for the

136 between-genus contrasts is not significant (Spearman $r=-0.210$, $N=12$, $p=0.256$ 1-
137 tailed), although the slope is similar to that for within-genus nodes.

138 Deaner & Nunn (1999) tested for a relationship between social group size and
139 residual brain size, but found none. However, using more up-to-date data on species
140 mean group sizes, there is in fact a significant linear regression between contrasts in
141 mean taxon group size and residuals in brain size contrasts (Fig. 3; $F_{1,17}=6.15$,
142 $r^2=0.266$, $p=0.024$ 2-tailed). Two things are immediately apparent, however: first,
143 there is a very striking grade difference between strepsirrhines (prosimians) and
144 haplorhines (anthropoids) and, second, the relationship is clearly non-linear (hence,
145 the likely reason why Deaner & Nunn obtained a non-significant result when using a
146 linear regression). Both effects are in fact well established features of the social brain
147 hypothesis (Dunbar 1993, 1998), and were well known at the time. Partitioning the
148 data by sub-order, and logging group size, yields significant improvements in fit
149 (prosimians: $F_{1,2}=7.96$, $r^2=0.726$, $p=0.067$ 2-tailed; anthropoids: $F_{1,12}=19.955$,
150 $r^2=0.624$, $p=0.001$, with a considerable further improvement in fit in the latter case for
151 a quadratic relationship, $r^2=0.760$). Pooling these results using Fisher's meta-analysis
152 (Sokal & Rolf 1969) yields a highly significant result ($\chi^2=21.99$, $df=4$, $p=0.0002$),
153 indicating that there is a consistent common trend underlying both these datasets.

154 Given that there is an effect of group size, we perhaps need to reconsider the
155 brain lag effect with this in mind. Fig. 4 plots the residuals from a multiple regression
156 of contrasts in brain mass regressed on both the contrast in body mass and the contrast
157 in group size, plotted against divergence date, for within-genus contrasts only. The
158 multiple regression is highly significant, with significant main effects (body mass:
159 $t_{16}=2.97$, $p=0.009$ 2-tailed; group size: $t_{16}=2.48$, $p=0.025$ 2-tailed). Since, in primates,
160 changes in group size over phylogenetic time are almost never negative (Perez-

161 Barberia et al. 2007), the plotted residuals are absolute values. A linear regression for
162 these data is not significant ($F_{1,8}=1.70$, $r^2=0.229$, $p=0.115$ 1-tailed), but a power
163 relationship is significant ($F_{1,8}=4.24$, $r^2=0.358$, $p=0.035$ 1-tailed, since a negative
164 relationship is not possible). Note that the asymptote is at a residual value of ~ 0.035 ,
165 and thus lies just above the common regression line. Mathematically, the point of
166 inflexion is defined by the value on the X axis that is equivalent to $1/e$ back from the
167 asymptotic value on the Y axis. Taking $Y=0.035$ as the asymptotic value and the
168 highest datapoint as the origin ($Y=0.153$), this gives a value of 0.7 million years as the
169 time it typically takes for the brain-body mass relationship to come back into balance.

170 Although the dates given by Deaner & Nunn (1999) correlate significantly
171 ($r=0.659$, $N=19$, $p=0.002$) with the more recent estimates by Perelman et al. (2011),
172 the latter tend to be deeper (the intercept for the regression equation plotting the more
173 recent values against the older values is $+2.395$ Ma). Recalculation of the inflexion
174 point for the same dataset using the Perelman et al. (2011) dates yields an estimate of
175 3.6 Ma. Since the Perelman et al. estimates are based exclusively on molecular data,
176 their divergence dates identify, in effect, a last common ancestor, and thus constitute
177 an upper limit. The date of population divergence (i.e. speciation *sensu stricto*) is
178 likely to be a great deal less. Something in the order of 2 million years is thus
179 probably a reasonable suggestion.

180 Deaner & Nunn (1997) implicitly assumed a causal relationship in which
181 changes in body size drive changes in brain size (presumably as an inevitable
182 consequence of the allometric relationship between the two), with changes in group
183 size presumably being a consequence of changes in brain size (i.e. a default by-
184 product benefit). A path analysis of the relationship between the three variables yields
185 a best fit model in which brain size independently predicts both body mass and group

size (Fig. 5). This pattern is confirmed by a mediation analysis: body size does not significantly influence group size, or *vice versa*, via brain size. Note that by ‘predict’ here is meant ‘constrains’, and not ‘evolutionary cause’ (or driver). In evolutionary (or selection) terms, the causal arrows are reversed: increases in group size select for increases in brain size, but changes in brain size are at the same time dependent on changes in body mass to provide the sufficient energy surplus through the allometric relationship between basal metabolic rate (BMR) and body mass (Schmidt-Nielson 1984; Martin 1990) to fuel brain growth.

Species that have undergone significant change in brain size in response to the need to increase group size will be paying an energetic cost: until body size comes back into line with brain size, they cannot benefit from the spare nutrient capacity made possible by the allometric relationship between BMR and body size (Martin 1990). To meet this demand, species will be obliged to find the additional energy and other nutrients required to fuel brain growth either through a change in diet, or by switching energy demand from other parts of the body (the expensive tissue hypothesis: Aiello & Wheeler 1995) or by reducing the energetic costs of foraging (Dunbar et al. 2009). In fact, it seems that most of the adjustment, in this sample at least, is provided by a shift to a more frugivorous (and hence more nutrient-rich) diet: on average, nodes with large residuals (absolute residual >0.05) in the contrast in brain size in Fig. 4 have a significantly greater contrast in dietary frugivory in favour of the bigger brained species than nodes with smaller residuals, although the sample is small (means of $+12.7 \pm 5.8\%$ vs $-32.5 \pm 34.4\%$, $\eta^2=0.766$; $F_{1,5}=7.09$, $p=0.045$ 2-tailed).

4. Discussion

210 Taken together, these results imply that, as often as not, changes in group size
 211 trigger correlated changes in brain volume, initially without necessarily affecting
 212 body size (leading to high residuals); with time, body size catches up, but Fig. 4
 213 suggests that this probably takes around 1-3 million years. The path analysis indicates
 214 that this change is invariably driven by increases in group size, and that the
 215 relationship between brain size and body size is independent of group size (i.e. is not
 216 directly determined by changes in group size). Although they endeavoured to control
 217 for ecological changes in their analysis, it seems that Deaner & Nunn (1999) failed to
 218 control for a much more important source of confound, namely the grade changes in
 219 relative brain size that occur within the primates. They also failed to control properly
 220 for social group size, mainly because they used a linear regression when the social
 221 brain relationship is explicitly non-linear (Dunbar 1992) and contains very distinct
 222 grades in the group size/brain size relationship (Dunbar 1993). Had they had more
 223 species available to them, the grade shift effects might have been lost in the error
 224 variance, but with a relatively small sample their impact is significant. Unfortunately,
 225 we are in no better position now in terms of available data than they were two decades
 226 ago because the most extensive brain dataset is still the one they used.

227 The initial impact of changes in brain size on the relationship between brain
 228 and body size places a significant strain on nutrient balance in species that make this
 229 change, and to balance their nutrient budget they have to increase nutrient throughput.
 230 For most monkeys, this means a more frugivorous, and less folivorous, diet. This shift
 231 to a richer diet may explain why the asymptotic value lies just above the common
 232 regression line: species that make this transition do not fully return to the common
 233 regression, but exploit their improved diet to maintain a slightly smaller body mass
 234 than would be expected. In cases, such as the *Pan-Homo* transition, where the change

235 in brain size is massive, the increase only seems to have been possible by switching
236 resources away from other expensive anatomical regions (notably the gut) to the brain
237 (the expensive tissue hypothesis: Aiello & Wheeler 1995). This is, however, unlikely
238 to be a general solution since it imposes major restrictions through gut specialisation.

239 This perhaps suggests a reason why several, somewhat misconceived, attempts
240 to test the expensive tissue hypothesis on New World monkeys (Allen & Kay 2012;
241 Hartwig et al. 2011) and mammals more generally (Navarette et al. 2011) have
242 produced negative results: in fact, these species adopted a much simpler strategy for
243 meeting their energy deficits, namely switching to a richer diet. In contrast, the diets
244 of great apes are much more frugivorous than those of any monkeys (on average, 69%
245 fruits vs 51%, N=5 and N=116 species, respectively: Campbell et al. 2007); as a
246 result, there would have been limited room for further movement in the same
247 direction in the transition into *Homo*, and hence a need for a more radical alternative
248 strategy of the kind suggested by Aiello & Wheeler (1995). In fact, of course, the
249 expensive tissue hypothesis was never offered as an explanation for primate brain
250 evolution, but rather for brain size evolution in *Homo*. Indeed, this strategy may only
251 be possible when the larger body mass of great apes allows more gut volume that can
252 be spared.

253 In effect, it seems that Deaner & Nunn (1997) were looking at the problem the
254 wrong way around. The assumption that the lag is based on initial changes in body
255 size because this is physiologically and/or genetically more labile is incorrect, at least
256 for primates. The analyses presented here suggest that quite the reverse is true: in
257 many cases, body size change occurs *because* change has occurred in brain size,
258 which in turn is driven by change in group size. While it is possible that the change in
259 body size is an independent response to the same selection factor that is driving group

260 size change (namely, predation risk: van Schaik 1982; Shultz et al. 2004; Dunbar &
 261 Shultz 2007; Bettridge & Dunbar 2012), it remains a possibility to be tested that
 262 species who opt to increase brain size ultimately need to evolve a larger body size in
 263 order to benefit from the allometric relationship between BMR and body size so as to
 264 pay for some of that increase in brain mass.

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270

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Legends to Figures

349

350

351 Fig. 1. Residuals of contrast in brain mass regressed against contrasts in brain mass
352 plotted against time of divergence for the species pairs. Dashed lines are the
353 upper and lower bounds.

354

355 Fig. 2. Absolute residuals of contrast in brain mass, plotted against time since
356 divergence. Dashed line is the least squares regression for these data points.
357 Data points from Fig. 1.

358

359 Fig. 3. Residual of contrast in brain mass against contrast in body mass, plotted
360 against contrasts in mean group size. Solid symbols: nodes between species
361 pairs from the same genus; open symbols: nodes between species pairs from
362 different genera. Hashed lines are the least squares regression lines through the
363 two sets of data.

364

365 Fig. 4. Absolute residual in contrast in brain mass against the common regression line
366 for contrasts in both body mass and social group size, plotted against time
367 since divergence. Linear and power regression lines are plotted.

368

369 Fig. 5. Path analysis of the functional relationships between the three main variables,
370 contrasts in brain mass, contrasts in body mass and contrasts in mean social
371 groups size. The plotted relationships, and the numbers given on the graph, are
372 the significant ($p < 0.05$) standardised slopes (β).

373

Figure 1

Figure 2

Figure 3

Figure 4

Figure 5