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Large-brained mammals live longer

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Abstract

Many mammals have brains substantially larger than expected for their body size, but the reasons for this remain ambiguous. Enlarged brains are metabolically expensive and require elongated developmental periods, and so natural selection should have favoured their evolution only if they provide counterbalancing advantages. One possible advantage is facilitating the construction of behavioural responses to unusual, novel or complex socio-ecological challenges. This buffer effect should increase survival rates and favour a longer reproductive life, thereby compensating for the costs of delayed reproduction. Here, using a global database of 493 species, we provide evidence showing that mammals with enlarged brains (relative to their body size) live longer and have a longer reproductive lifespan. Our analysis supports and extends previous findings, accounting for the possible confounding effects of other life history traits, ecological and dietary factors, and phylogenetic autocorrelation. Thus, these findings provide support for the hypothesis that mammals counterbalance the costs of affording large brains with a longer reproductive life.

Introduction

Large brains have evolved multiple times and in multiple taxa (Jerison, 1973). This is puzzling because a brain disproportionately large for a given body size is metabolically expensive (Aiello & Wheeler, 1995; Isler & van Schaik, 2006, 2009a,b) and takes a substantial time to reach structural and functional maturity (Casey *et al.*, 2005). Long developmental periods result in significant fitness costs for large-brained species, both in terms of increased offspring mortality risk (Sacher & Staffeldt, 1974; Stearns, 2000; Deaner *et al.*, 2003; Barrickman *et al.*, 2008) and delayed age of first reproduction (Deaner *et al.*, 2003; Barrickman *et al.*, 2008). Consequently, natural selection should have favoured the evolution of large brains only if they provide advantages that counterbalance their production and maintenance costs.

Several hypotheses have been proposed to explain the adaptive advantages of larger brains (see Deaner *et al.*, 2003; van Schaik & Deaner, 2003; Dunbar & Shultz, 2007a; Sol, 2009a), most of which assume that enlarged brains carry cognitive advantages. Amongst others, these include monitoring food sources that vary in space and time (Clutton-Brock & Harvey, 1980; Milton, 1988), using hard-to-eat foods (Parker & Gibson, 1977, 1979), exploiting novel foraging opportunities (Lefebvre *et al.*, 1997) and modifying behaviour in response to conspecifics (Jolly, 1966; Humphrey, 1976; Cheney & Seyfarth, 1986; Byrne & Whiten, 1988; Whiten, 2000; Dunbar & Shultz, 2007b). The above hypotheses focus on selective advantages of enlarged brains but do not provide an explicit explanation for how these benefits balance the developmental costs of affording large brains. However, if these benefits reflect general cognitive capacities for constructing behavioural responses to novel socio-ecological challenges, then this should reduce extrinsic mortality and partially compensate the developmental costs with a longer reproductive life (Allman *et al.*, 1993; Allman, 2000; Deaner *et al.*, 2003; Sol *et al.*, 2007; Sol, 2009a,b). This interpretation, the so-called 'cognitive buffer hypothesis', thus integrates previous hypotheses, acknowledges that brains carry out multiple functions and provides an explicit explanation of the benefits of

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brain enlargement (Allman *et al.*, 1993; Allman, 2000; Deaner *et al.*, 2003; Sol, 2009a).

Recently, comparative work on brain evolution has been criticized because diverse findings regarding correlates of brain enlargement have not been integrated (Healy & Rowe, 2007). The lack of consideration of alternative hypotheses for the evolution of enlarged brains is a repeated criticism (Deaner *et al.*, 2000; Reader & Laland, 2002; Dunbar & Shultz, 2007b). The diversity of reported correlates of brain enlargement probably reflects the fact that the brain performs multiple functions: postulating a single cognitive benefit for brain enlargement is unlikely to be successful. There is considerable evidence that species with enlarged brains for their body size show enhanced cognitive capacities, although the mechanisms behind these relationships are obscure and warrant study (reviewed in Healy & Rowe, 2007; Lefebvre & Sol, 2008). For example, multiple studies have demonstrated associations between brain size and components of behavioural flexibility, such as innovation, tool use, tactical deception, social learning, reversal-learning and combined measures of laboratory learning performance, in both birds and primates (Lefebvre *et al.*, 1997, 2004; Reader & Laland, 2002, 2003; Reader, 2003; van Schaik & Deaner, 2003; Byrne & Bates, 2007; Deaner *et al.*, 2007). Evidence is also accumulating that flexibility in behaviour facilitates the production of adaptive responses to a wide array of ecological challenges (reviewed in Sol, 2009a). In birds and mammals, for example, large-brained species are more likely to be successful when introduced by humans in novel environments than are small-brained species (Sol *et al.*, 2005, 2008). Moreover, amongst British birds, species with relatively large brains were less likely to suffer population declines (Shultz *et al.*, 2005). Thus several lines of evidence support the idea that brain volume is associated with diverse measures of behavioural flexibility and with success in novel or changed environments, providing a route to integrate previous findings.

Surprisingly, however, evidence for a critical prediction of the cognitive-buffer hypothesis that brain enlargement translates to increased life expectancy remains mixed. In mammals, the animals with the largest relative brain sizes, some studies have demonstrated a significant relationship between brain size and lifespan (e.g. Hakeem *et al.*, 1996; Deaner *et al.*, 2003; Kaplan *et al.*, 2003; Isler & van Schaik, 2009a,b), but others did not (e.g. Barton, 1999; Ross & Jones, 1999; Judge & Carey, 2000). The disparity of results may arise from differences in the way that previous studies controlled or failed to control for confounding factors and phylogenetic effects. Moreover, previous analyses were generally based on a reduced number of species and were biassed towards primates (reviewed in Barrickman *et al.*, 2008). This focus potentially reduces the interspecific variation observed in brain size and lifespan, which could reduce the possibility of detecting patterns. Understanding the evolution of large brains is only possible if we further validate the brain–lifespan association in many taxa and with approaches that properly deal with phylogenetic and confounding factors (Lefebvre *et al.*, 2004; Sol, 2009a). Here, we ask whether large-brained mammals live longer with a global phylogenetic-based comparative analysis covering 493 mammalian species. We extend on recent similar analyses (Isler & van Schaik, 2009a,b) by taking into account previously unconsidered confounding variables, using datasets covering additional taxa (e.g. marsupials), and directly estimating and accounting for phylogenetic effects (Hansen & Orzack, 2005). We show that the association of larger brains with longer lifespan holds independently of other life history traits, of research effort, and of energetic, environmental, dietary and habitat variables, thus providing unambiguous support for the idea that the costs of delaying reproduction in large-brained species can be partly compensated by a longer reproductive life.

Material and methods

Lifespan

As an estimate of reproductive lifespan, we gathered information on maximum-recorded lifespan (in years) for 493 species of mammals from de Magalhaes & Costa (2009; see references therein). The bulk of these data come from Weigl (2005). There exist alternative, more accurate estimates of reproductive lifespan (Ricklefs & Scheuerlein, 2001), but maximum-recorded lifespan provides a reasonable estimate that is available for many species (Barrickman *et al.*, 2008; Isler & van Schaik, 2009a). Barrickman *et al.* (2008) proposed that the age at first reproduction must be first subtracted from maximum lifespan to provide a measure of the duration of reproductive life (hereafter, 'reproductive lifespan'), and thus to test the idea that enlarged brains are associated with longer periods of reproductive life. We thus used two lifespan measures as dependent variables: 'lifespan' and 'reproductive lifespan'.

The available maximum lifespan data have some potential problems. First, they are derived from both captive and wild records. As lifespan recorded under captive conditions may not represent that in the wild, pooling these captive and wild data could potentially obscure any true relationships (Barrickman *et al.*, 2008). To account for this issue, we included whether lifespan was measured in captivity or the wild as a factor in the statistical model. Second, maximum lifespan estimates increase with research effort (Møller, 2006, 2007; de Magalhaes & Costa, 2009). To account for this possible bias, we estimated research effort from the number of articles listed in ISI Web of Science in July 2009 for each species. Research effort was log transformed and included as covariate in statistical models.

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Brain size

The use of whole-brain vs. brain-part volumes is an important issue in testing the cognitive buffer hypothesis (Deaner *et al.*, 2007). While a focus on one brain component may be advantageous in studying a specialized cognitive function (Healy & Rowe, 2007), the use of whole brain size is likely to be more appropriate in testing the cognitive buffer hypothesis (Barton & Harvey, 2000; Sol & Price, 2008; Sol *et al.*, 2008). First, behavioural flexibility has multiple underlying mechanisms and arises from several processes such as perception, motor ability and cognitive processing (Changizi, 2003; Deaner *et al.*, 2003), unlikely to be localized in a single brain area (Lewis, 2006). Second, several brain component volumes are consistently correlated with whole brain size, particularly larger parts that are involved in higher order and multimodal integration (Timmermans *et al.*, 2001; Iwaniuk *et al.*, 2004). Finally, as already noted, a growing number of studies have found support for an association between brain size and different measures of behavioural flexibility, such as innovation, tool use, tactical deception and learning (reviewed in Lefebvre *et al.*, 2004; Dunbar & Shultz, 2007a; Deaner *et al.*, 2007; Lefebvre & Sol, 2008). Thus, we used data on whole brain size, which has the additional advantage that it is available for many more species than are brain component volumes. Data on brain mass for 493 species were compiled from published information from multiple sources (see Appendix I). We subtracted 0.59 g from each rodent species datum in Mace *et al.* (1981), following the corrective procedure recommended by Isler & van Schaik (2006). Brain masses were either calculated from endocranial volumes or were whole brain masses. Although the use of endocranial volumes to calculate brain masses has been debated (Röhrs & Ebinger, 2001), we utilized these data because recent studies have demonstrated that it provides a reliable proxy of brain mass (Ashwell, 2008; Isler *et al.*, 2008; Finarelli & Flynn, 2009). The reliability of the brain measures utilized was previously evaluated by Sol *et al.* (2008) using a variance component analysis, which showed that variation across species was higher than within species. This validated the treatment of brain mass as a species character.

Larger species have larger brains, so it is necessary to estimate brain mass controlling for the allometric effect of body size. At least three methods have been proposed to do this: (i) to estimate the residuals of a log–log least squares linear regression of brain mass against body mass; (ii) to calculate the fraction of the body mass that corresponds to brain mass; and (iii) to include absolute brain mass and body mass as covariates in a multivariate model (Deaner *et al.*, 2000). We used all methods, and the results are consistent. We present in the text the results obtained with the residual method, as this approach has the advantage of eliminating problems of collinearity while effectively removing body size effects (Sol *et al.*, 2007). Body mass was obtained from the same sources as brain mass when available and complemented with published data as needed (Smith & Jungers, 1997). Following Sol *et al.* (2008), when more than one source per species was available, the mean values of brain mass and body mass (in grams) were utilized, and when only a range of values was available, the midpoint was used. To reduce measurement error, for each species, the coefficient of variation was calculated for both brain and body mass. We removed extreme values where the coefficient of variation was extremely high (> 50%), apart from highly sexually dimorphic species (Weckerly, 1998), as these high variances were probably the consequence of a measuring error. We removed extreme data values for 11 species.

Before estimating residuals of brain mass, it is necessary to control for the 'grade shift' phenomenon (Pagel & Harvey, 1988; Nunn & Barton, 2000; Sol *et al.*, 2008). Grade shifts represent the fact that in mammals the intercept of the regression line between brain mass and body mass differs across taxonomic groups, leading to biases in residuals if left unaccounted for. To deal with this problem, Nunn & Barton (2000) proposed the estimation of the slope (b) of the regression with phylogenetic independent contrasts (Felsenstein, 1985). As only a few independent contrasts will be affected by grade shifts, the effect of grade shifts on the global relationship between contrasts should be weak (Nunn & Barton, 2000; Sol *et al.*, 2008). Following Blomberg *et al.* (2003), we computed the size-corrected values for brain mass in three steps. First, independent contrasts were estimated for brain mass and body mass (both log transformed) with the PDAP module of the MESQUITE program (Garland *et al.*, 1999; Garland & Ives, 2000). The phylogenetic tree was that proposed by Bininda-Emonds *et al.* (2007; corrigendum, 2008), which includes a great number of extant mammals. Second, a least squared linear regression through the origin of these contrasts (brain mass on body mass) was computed to estimate the allometric exponent (b). The relationship between contrast of brain mass and body mass was strong ($R^2 = 0.90$); therefore, the use of alternative line-fitting techniques was not necessary (Barton & Harvey, 2000). Third, size-corrected values of brain mass were computed as log [brain mass/body mass^b] using raw values (not independent contrasts). Hereafter, this variable will be called 'residual brain mass'. The slope (b) was estimated as 0.64, close to that estimated by other studies (Harvey & Krebs, 1990; Sol *et al.*, 2008). Because the residual brain values obtained do not completely remove the effect of body mass (correlation coefficient = 0.51), log body mass was included in all the models testing the relation between residual brain mass and lifespan.

Confounding variables

As the analyses are correlational, any relation between lifespan and brain mass could be obscured or inflated by

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the effect of other variables. We thus accounted for several factors that potentially can affect lifespan variation. First, metabolism could be an important determinant of lifespan (Harvey *et al.*, 1991; Allman *et al.*, 1993; Hofman, 1993; Ricklefs & Wikelski, 2002; Speakman, 2005). To control its possible effect, data for basal metabolic rate (BMR) were obtained from White *et al.* (2009) and included in the statistical model.

Second, life history traits are known to covary systematically across species (Harvey & Clutton-Brock, 1985; Promislow & Harvey, 1990; Stearns, 2000; Bielby *et al.*, 2007). It is thus important to ensure that the apparent association between brain mass and lifespan is not spuriously created by the effect of another life history trait. Information on gestation, weaning, age at first reproduction, litter size and litters per year was taken from published literature (Ernest, 2003; de Magalhaes & Costa, 2009; Bielby *et al.*, 2007). These life history traits are highly correlated with lifespan (e.g. Harvey & Clutton-Brock, 1985) as well with each other (van Schaik & Deaner, 2003; Bielby *et al.*, 2007).

Third, life history strategies vary across regions (Ricklefs, 2000; Forsyth *et al.*, 2004; Martin *et al.*, 2006; McNamara *et al.*, 2008). For example, latitude has been reported to predict lifespan in birds (Møller, 2006, 2007). To account for geographical biases, maximum northern latitude (MNL) and maximum southern latitude (MSL) were gathered from breeding ranges published in the literature (Dorst & Dandelot, 1973; Schilling *et al.*, 1987; Strahan, 1995; Kingdon, 1997; Mitchell-Jones *et al.*, 1999; Folkens *et al.*, 2002; Long, 2003; Patterson *et al.*, 2003; Jackson, 2007; IUCN, 2008). We calculated three proxy variables for geographical factors from these breeding ranges: 'geographical range' (the total latitude degree of breeding range), 'mid-latitude point of breeding range' (calculated as (MNL+MSL)/2, following Newton, 1995), 'discontinuous distribution' (coded as 'discontinuous' or 'continuous', accounting for discontinuous or continuous occupancy along the latitudinal breeding distribution).

Fourth, species diet and habitat thought to be linked to lifespan (Bennett & Harvey, 1985; Harvey & Clutton-Brock, 1985; Allman *et al.*, 1993; van Schaik & Deaner, 2003). Thus, these variables were considered in the analyses and coded as follows: primary dietary type (herbivorous, carnivorous, omnivorous and insectivorous), feeding generalism (number of these diet categories, range 1–4), primary habitat type (coastal habitat, inland waters, wetland, desert, forest, mountain, tropical rainforest, savanna, grassland, woodland, scrub-tundra, rural and urban areas) and habitat breadth (number of these habitat types used, range 1–13). Data were compiled from multiple sources (Kingdon, 1997; Long, 2003; Patterson *et al.*, 2003; Wilson & Reeder, 2005; Jackson, 2007; IUCN, 2008).

Finally, both lifespan and reproductive lifespan scale allometrically with body size (Harvey & Clutton-Brock, 1985; Blumstein & Møller, 2008; this study), so it is relevant to examine whether brain mass correlates with lifespan when the body size effect is controlled for. To account for body size effects on lifespan, we estimated the residuals of a log–log regression of lifespan (or reproductive lifespan) against body size (termed 'residual lifespan' or 'residual reproductive lifespan', respectively). Because body mass has a high phenotypic variability (Economos, 1980; Smith & Jungers, 1997), the average body mass calculated can be an under-estimate or over-estimate of the true value. This is problematic, as it causes the residuals of the response and predictor variables to be biassed in the same direction, increasing the chance of type I errors (Harvey & Krebs, 1990; Barton, 1999). To avoid this problem, we separately obtained residuals of dependent and independent variables by using a different set of body masses (Harvey & Krebs, 1990; Barton, 1999; Deaner *et al.*, 2003; Barrickman *et al.*, 2008). This second set of body masses was obtained from Ernest (2003), complemented by other sources (Jackson, 2007; de Magalhaes & Costa, 2009).

Analyses

Closely related taxa share many traits from common ancestors rather than from independent evolution, thus species' traits cannot generally be treated as statistically independent points (Felsenstein, 1985). To deal with this problem, we modelled lifespan values for species with a phylogenetic generalized least squares approach (PGLM) (Freckleton *et al.*, 2002; Phillimore *et al.*, 2006; Shultz & Dunbar, 2007). This method takes the phylogenetic variance/covariance matrix derived directly from the phylogenetic supertree of the species, and hence evaluates the association between variables taking into account the correlated error structure. This is performed by estimating a parameter lambda (λ), which measures the degree to which the matrix follows a Brownian model (λ values near 0 implying no phylogenetic autocorrelation and values near 1 maximum phylogenetic autocorrelation). The fitted generalized least squares model (GLM) and λ were simultaneously estimated to test the effect of brain mass on maximum lifespan across species. We included interactions between predictor variables in the analyses, but none were statistically significant and thus are not reported below. PGLM analyses were conducted with R 2.7.0 (R Development Core Team, 2005), the R code kindly provided by R. P. Freckleton, and the phylogenetic hypothesis proposed by Bininda-Emonds *et al.* (2007; corrigendum, 2008).

Following Sol *et al.* (2008), a minimum adequate model (MAM) was constructed by means of a backward selection approach. The initial PGLM model was composed by residual brain mass and the rest of confounding variables. Then, we sequentially dropped the variables resulting in the lowest improvement to model fit. We investigated the significance of alternative models by

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adding the previous variable removed from the model. Variables with $P < 0.2$ were retained in the MAM, to avoid the removal of confounding variables of weak influence. However, the standard criterion for statistical significance ($P < 0.05$) was applied throughout. Diagnostic plots were examined to check for outliers, heteroscedasticity and non-normal errors.

Results

We found extensive variation both in brain mass and maximum lifespan across species (Fig. 1). To test whether the lifespan of mammalian species can be explained to some degree by residual brain mass, we first used a conventional linear model (LR: linear regression), so that our results could be compared with previous studies. This model revealed a very strong relationship between residual brain mass and lifespan (coefficient ± S.E., $b = 0.49 \pm 0.04$, $t_{486} = 10.98$, $P < 0.0001$, Fig 2a), even when the effect of body mass on lifespan was removed (residual brain mass vs. residual lifespan: $b = 0.48 \pm 0.04$, $t_{486} = 10.86$, $P < 0.0001$, Fig. 2b).

The LR analyses above did not include phylogenetic corrections, but it is well known that disregarding phylogenetic effects can cause misleading results when the studied traits show high phylogenetic autocorrelation. Indeed, lifespan showed significant phylogenetic autocorrelation, with a lambda estimate close to 1 ($\lambda = 0.97$; $X^2 = 437.10$, $P < 0.0001$ that λ is 0; $X^2 = 10.89$, $P = 0.0009$ that λ is 1). We thus used a PGLM approach. The relationship between residual brain mass and lifespan was positive and highly significant (partial regression coefficient ± S.E., $b = 0.26 \pm 0.04$, $t_{486} = 5.37$, $P < 0.0001$). When the allometric effect of body mass on lifespan was incorporated in the analysis, the residuals of brain mass remained strongly associated with residuals of lifespan (PGLM: $b = 0.20 \pm 0.04$, $t_{486} = 4.26$, $P < 0.0001$).

Because of the correlative nature of the analyses, the relationship between residual brain mass and lifespan could be spuriously caused by their common association with a third variable. None of the ecological (i.e. basal metabolic rate, primary habitat, primary diet, feeding generalism or habitat breadth) and geographical variables (i.e. geographical range, mid-latitude point or discontinuous distribution) evaluated were found to be significantly associated with lifespan in the MAM (PGLM: $P > 0.05$ in all variables), and did not alter the relationship between brain mass and lifespan. From the life history traits we considered, only the age at first reproduction was significantly associated with lifespan (PGLM: $P < 0.001$, $N = 417$; all other variables $P > 0.2$). However, residual brain mass remained significantly associated with lifespan when age at first reproduction was taken into account (Table 1). The MAM included age at first reproduction along with residual brain mass, lifespan measure (wild or captive), research effort (log transformed) and body mass (log transformed). Recorded lifespan was longer in captive animals, in better-studied species, in heavier species and in species with an older age at first reproduction (Table 1). The models explained 42% of variance in lifespan and 21% of variance in residuals of

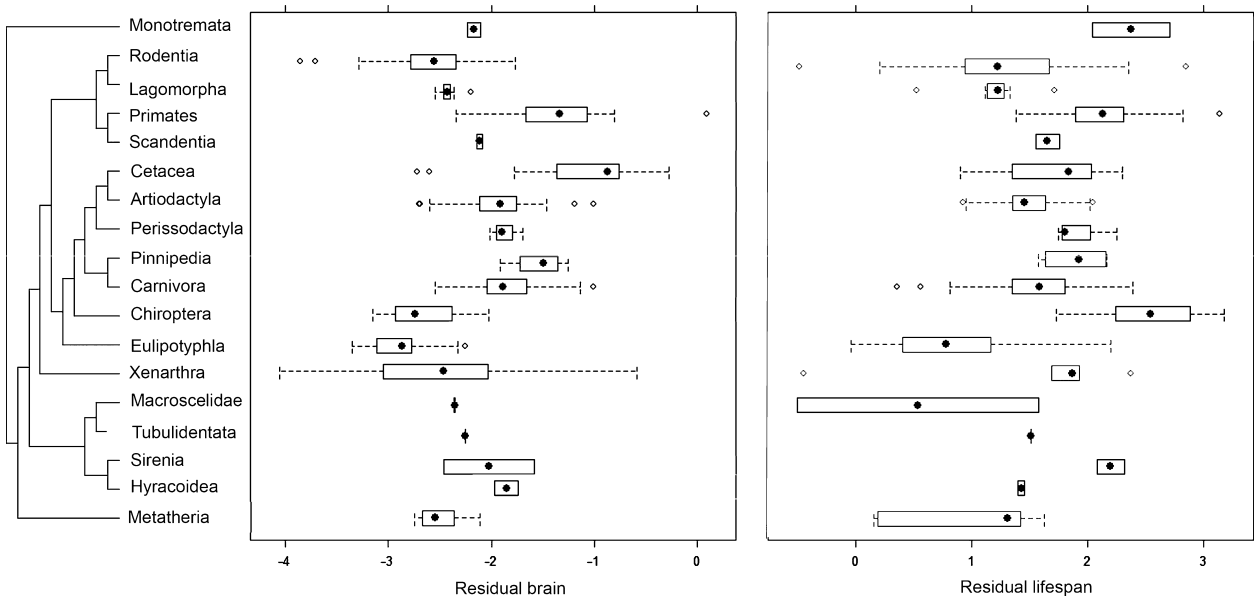

Fig. 1 Box plots (median and 25% and 75% percentiles) of residual brain mass (accounting for body mass) and residual maximum lifespan (accounting for body mass) across mammalian orders, with phylogenetic relationships between taxa indicated on the left (phylogeny: Bininda-Emonds *et al.*, 2007; corrigendum, 2008).

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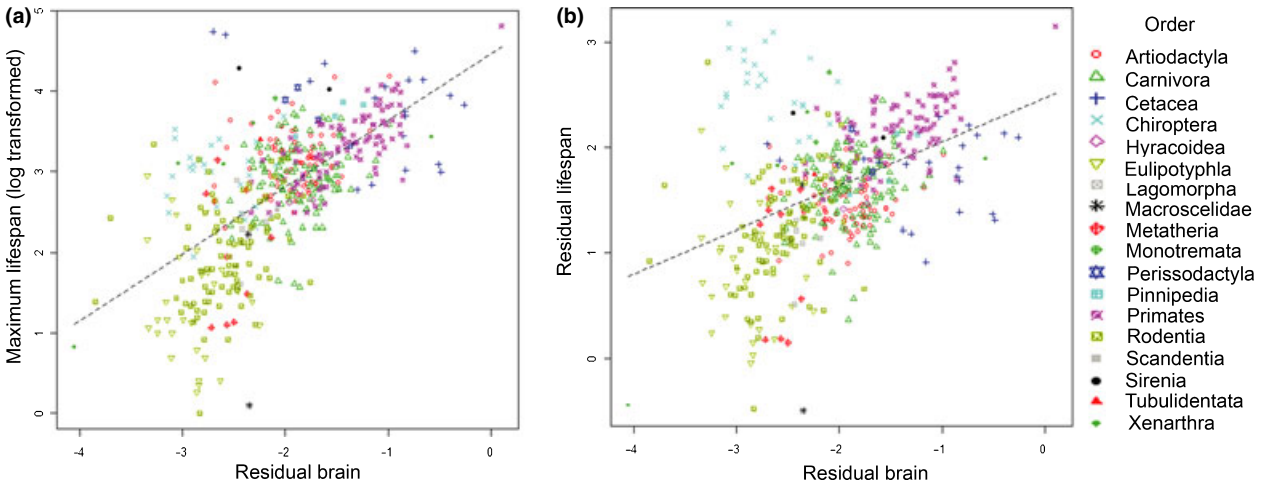

Fig. 2 Relationship between residual brain size and maximum lifespan in 493 species of mammals (a) without (linear regression: $F_{4,486} = 176.9$, $R^2 = 0.59$, $P < 0.0001$) and (b) with control for the allometric effect of body size on lifespan (linear regression: $F_{4,486} = 36.67$, $R^2 = 0.22$, $P < 0.0001$). Equivalent results were obtained using PGLM analysis: a: $F_{5,491} = 57.17$, $R^2 = 0.31$, $P < 0.0001$; b: $F_{5,491} = 19.34$, $R^2 = 0.13$, $P < 0.0001$).

Table 1 Minimum adequate PGLS model of lifespan for 384 mammalian species (adjusted $r^2 = 0.42$, for lifespan, 0.21, for residual lifespan, 0.35 for reproductive lifespan and 0.16 for residual reproductive lifespan).

	Lifespan				Residual lifespan				Reproductive lifespan				Residual reproductive lifespan			
Predictors	*b*	se	*t*	*P*	*b*	se	*t*	*P*	*b*	se	*t*	*P*	*b*	se	*t*	*P*
Residual brain size	0.17	0.05	3.4	0.0006	0.14	0.05	2.7	0.0006	0.19	0.1	3.2	0.001	0.15	0.1	2.6	0.009
Age at first reproduction	0.23	0.03	7.1	< 0.0001	0.23	0.03	7.2	< 0.0001	0.15	0.03	4.2	0.001	0.15	0.03	4.2	< 0.0001
Body mass	0.09	0.01	7.2	< 0.0001	−0.10	0.01	−4.0	< 0.0001	0.1	0.01	7.2	< 0.0001	−0.03	0.01	−2.6	0.0008
Research effort	0.03	0.01	3.6	0.0003	0.03	0.01	3.6	0.0004	0.03	0.01	3.7	0.0002	0.03	0.01	3.6	0.0003
Origin lifespan data	−0.24	0.05	−4.2	< 0.0001	−0.30	0.05	−4.5	< 0.0001	−0.3	0.06	−4.6	< 0.0001	−0.3	0.1	−4.8	< 0.0001

*The parameters (*b*) are the partial regression coefficients relating the predictors (residual brain size, age at first reproduction, research effort, origin of lifespan data (captive/wild) and body mass) with lifespan. Four lifespan measures are used as dependent variables: maximum lifespan, residual maximum lifespan (controlling for the allometric effects of body size), maximum reproductive lifespan (maximum lifespan-age at first reproductive) and residual reproductive lifespan. Analysis was via the phylogenetic generalized least squares method. Three confounding variables were kept in the model (criterion: $P < 0.2$) but were not statistically significant ($0.12 > P > 0.07$): discontinuous distribution ($b = 0.04$ for all four dependent variables), desert habitat ($b = 0.06$–0.07) and herbivorous diet ($b = 0.07$–0.08).

lifespan. Equivalent results were obtained with reproductive lifespan as the dependent variable (Table 1).

Discussion

Species of mammals with larger brains than expected for their body size tended to live longer than those with smaller brains. Although residual brain size explained only a small fraction of the variance in residual lifespan across species (about 13%, Fig. 2b), this relationship was robust and largely independent of ecological, geographical and phylogenetic effects. Thus, our results provide robust evidence that in large-brained animals, the costs of delaying reproduction are in part compensated with a longer reproductive life.

Lifespan is difficult to quantify, and thus estimates are subject to error, which might detract from our ability to resolve the strength of an association between brain size and lifespan. Although some of the highest values of maximum lifespan are reported in captive animals (de Magalhaes & Costa, 2009), the mixture of wild and captivity lifespan records was unlikely to affect the correlation between lifespan and brain size (Table 1; see also Allman *et al.*, 1993; Barrickman *et al.*, 2008). Captive conditions could be argued to not replicate the pressures faced in natural environment, but maximum lifespan may be seen as representing a physiological limit to life duration (Barrickman *et al.*, 2008; de Magalhaes & Costa, 2009). In the same way, research effort may bias lifespan estimates (Møller, 2006, 2007; de Magalhaes & Costa,

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2009), as our results show. However, the brain–lifespan association remained significant when research effort and data source were accounted for in the analyses.

Many previous studies have examined the brain size–lifespan relationship (Sacher, 1959; Sacher & Staffeldt, 1974; Economos, 1980; Hofman, 1993; Allman *et al.*, 1993; Ricklefs & Scheuerlein, 2001; Kaplan *et al.*, 2003; Barton, 1999; Hakeem *et al.*, 1996; Deaner *et al.*, 2003; Barrickman *et al.*, 2008; Isler & van Schaik, 2009a,b). Our findings extend on these studies, expanding the taxonomic range studied. Moreover, several of these previous studies did not take into account the phylogenetic relationships amongst species and, if they did, did not estimate the level of phylogenetic autocorrelation (Hansen & Orzack, 2005). In contrast, we performed the analysis on 493 mammalian species, and the degree of shared evolutionary history was directly included into the analysis, which ensured a better estimation of the model parameters. This proved to be essential as phylogeny accounted for a substantial part of the link between lifespan and brain size ($\lambda = 0.91$). Thus, the high correlations between lifespan and brain size that have been previously reported (e.g. for primates: $r = 0.65$, Allman *et al.*, 1993; and for mammals: $r = 0.83$, Hofman, 1993) could in part be explained by shared evolutionary history amongst related species.

van Schaik & Deaner (2003) argued that the inclusion of some orders (e.g. *Chiroptera, Monotremes*, *Edentates*) may hide the relationship between lifespan and brain size in mammals. These taxa show lower metabolic rates, which tend to be associated with increased longevity despite their small brain size (Allman *et al.*, 1993; Hofman, 1993). In the present work, although these taxa were included, neither inclusion of these taxonomic groups nor metabolic rate in the analysis accounted for the lifespan–brain size correlation we document. Likewise, Harvey *et al.* (1991) did not find evidence for the association between basal metabolic rate and life histories. It is possible that BMR is not the most appropriate metabolic measure (Speakman, 2005). We found that although BMR and lifespan correlated, the relationship was not statistically significant when body mass was included as a covariate and phylogenetic effects were taken into account (LM: $b = -0.38$, $t_{189} = 4.70$, $P < 0.0001$; PGLM: $b = -0.08$, $t_{189} = 1.13$, $P = 0.25$).

In terms of the remaining confounding factors, our results do not indicate any significant association between lifespan and ecological variables (habitat, diet, feeding generalism or habitat breadth), in line with findings by Harvey & Clutton-Brock (1985). Previous work in birds showed a negative association between lifespan and latitude, which can be explained by differential effects of biological and environmental interactions at different latitudes (Møller, 2007; Blumstein & Møller, 2008). Likewise, Duncan *et al.* (1999) found a significant correlation between lifespan and geographical range in birds. In contrast, in our study of mammals, we did not find any significant relationship between lifespan and geographical variables (mid-latitude point, range size and discontinued distribution). Mid-latitude point and range size were significantly associated with lifespan, but these association disappeared when research effort and lifespan measure (captive vs. wild) were included as covariates in the model. Even after controlling for ecological and geographical factors, the predicted brain–lifespan association remained strong.

Age at first reproduction was the only life history trait retained in the MAM as a predictor of lifespan, along with body mass and residual brain size. This finding is consistent with previous studies in birds and mammals (Rushton, 2004; Møller, 2006; Blumstein & Møller, 2008; Isler & van Schaik, 2009a). Barrickman *et al.* (2008) proposed that associations between brain enlargement and duration of the reproductive life must be tested by subtracting the growth period from maximum lifespan. Performing such an analysis, we found that the correlation between lifespan and relative brain size holds. Thus, the observed correlation is not the result of an elongated juvenile period confounding the lifespan measure.

Our results thus add to evidence for the cognitive buffer hypothesis by which a large brain assists in buffering individuals against environmental challenges by facilitating flexible behavioural responses (Allman *et al.*, 1993; Deaner *et al.*, 2003; Sol, 2009a,b). This buffer effect should increase survival rates (Shultz *et al.*, 2005; Sol *et al.*, 2007) and favour a longer reproductive life, thereby partially compensating for the costs of delayed reproduction associated with the need to grow a large brain. Nevertheless, it is possible that an extended reproductive period is insufficient to fully counterbalance the costs of delayed reproduction in large-brained mammals. For example, Isler & van Schaik (2009a) demonstrated a negative correlation between the maximum rate of population increase and mammalian brain size. This raises the issue of additional counterbalancing advantages to brain enlargement (Isler & van Schaik, 2009a,b).

The evidence for the brain size–lifespan association is correlational and does not necessarily reflect a causal relationship. In fact, the cognitive buffer hypothesis is just one of a set of theories that predict the brain size–lifespan correlation (Deaner *et al.*, 2003; Sol, 2009a). For example, while the cognitive buffer hypothesis argues that large brains facilitate a longer lifespan, it is also possible that a longer lifespan selects for larger brains (Deaner *et al.*, 2003; Sol, 2009a,b). Our results do not allow us to distinguish between these possibilities. Moreover, these different theories are not mutually exclusive and may act together to generate positive feedback favouring further increase in brain volume and longevity (Sol, 2009a,b). For instance, longevity can favour a delayed onset of reproduction, which should give parents the opportunity of prolonged investment in and contact with offspring (Covas & Griesser, 2007). This can facilitate an increase in brain size if, as the social

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intelligence hypothesis suggests, individuals living in stable social groups face higher cognitive demands than that individuals living alone (Byrne & Corp, 2004; Dunbar & Shultz, 2007a; Shultz & Dunbar, 2007). Despite its correlative nature, the finding that large-brained mammals live longer is important because it provides a solid basis from which integrate brain size evolution within a life history framework (Deaner *et al.*, 2003; Isler & van Schaik, 2009a,b; Sol, 2009a,b). As Ricklefs (2004) notes, the evolution of large brains and cognition is rarely considered in this manner. A fruitful avenue for future research would be to elucidate the complex causal links that may help integrate brain size into the life history strategy of the species.

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Appendix I

Sources of Brain Data.

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