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Review

Maternal effects and the evolution of brain size in birds: Overlooked developmental constraints

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Abstract

A central dogma for the evolution of brain size posits that the maintenance of large brains incurs developmental costs, because they need prolonged periods to grow during the early ontogeny. Such constraints are supported by the interspecific relationship between ontological differences and relative brain size in birds and mammals. Given that mothers can strongly influence the development of the offspring via maternal effects that potentially involve substances essential for growing brains, we argue that such effects may represent an important but overlooked component of developmental constraints on brain size. To demonstrate the importance of maternal effect on the evolution of brains, we investigated the interspecific relationship between relative brain size and maternal effects, as reflected by yolk testosterone, carotenoids, and vitamins A and E in a phylogenetic study of birds. Females of species with relatively large brains invested more in eggs in terms of testosterone and vitamin E than females of species with small brains. The effects of carotenoid and vitamin A levels on the evolution of relative brain size were weaker and non-significant. The association between relative brain size and yolk testosterone was curvilinear, suggesting that very high testosterone levels can be suppressive. However, at least in moderate physiological ranges, the positive relationship between components of maternal effects and relative brain size may imply one aspect of developmental costs of large brains. The relationship between vitamin E and relative brain size was weakened when we controlled for developmental mode, and thus the effect of this antioxidant may be indirect. Testosterone-enhanced neurogenesis and vitamin E-mediated defence against oxidative stress may have key functions when the brain of the embryo develops, with evolutionary consequences for relative brain size.

Keywords: Androgens; Antioxidants; Brain size evolution; Comparative analyses; Egg carotenoids; Development; Maternal effects; Retinol; Maternal hormones; Vitamins

Contents

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1. Introduction

The costs of elevated cognitive capacities involve the maintenance of large brains and the prolongation of the development of complex neural structures (Ricklefs, 2004). Species that possess elaborate neural machinery in terms of large brain in adulthood may thus need to deploy more resources and invest more time in brain development. Several interspecific studies of birds and mammals have repeatedly supported this hypothesis of developmental constraints by showing an association between mode of development and relative brain size (Bennett and Harvey, 1985a; Iwaniuk and Nelson, 2003; Pagel and Harvey, 1988; Portmann, 1947). In birds, altrical hatchlings have relatively smaller brains than precocial hatchlings, but this difference is reversed in adulthood when altrical birds have larger brains for their body mass than precocial birds (Bennett and Harvey, 1985a, b). However, among mammals, there is no equivalent correlation between variation in adult relative brain size and mode of neonatal development (Bennett and Harvey, 1985a). In this class of animals, gestation length seems to primarily determine neonatal brain size (Pagel and Harvey, 1988). In fact, relative brain size in birds also covaries with other developmental traits, such as the duration of the incubation period, age at fledging and the duration of parental care, and these relationships showed patterns dependant on developmental mode and taxonomical order (Iwaniuk and Nelson, 2003). Despite the difficulty of generalization, the close link between development and brain size demonstrates that cognitive function evolves in conjunction with life-history (Ricklefs, 2004).

It is often overlooked that in birds, maternal effects may also mediate the organization of the brain, and such effects can thus impose developmental constraints for the evolution of relative brain size. In general, the deposition of biologically active molecules, such as androgens and different antioxidants into egg yolk, are chief components of maternal effects that can have short- or long-term effects on offspring either in positive or negative direction (reviews in Blount et al., 2000; Gil, 2003; Groothuis et al., 2005b; Surai, 2002). For example, increased levels of egg testosterone (T) can enhance begging vigour of chicks, their competitive ability, and growth rate (Eising et al., 2001; Schwabl, 1993, 1996), while yolk testosterone can have long-term consequences for survival (Navara et al., 2005; Sockman and Schwabl, 2000), development of secondary sexual characters (Rubolini et al., 2006b; Strasser and Schwabl, 2004), and potentially future reproductive success (Groothuis et al., 2005b). Likewise, carotenoids and vitamins A and E can reduce oxidative damage and result in increased hatching success and improved immune response in young birds, and such effects may ultimately affect their survival and phenotypic quality in adulthood (Blount et al., 2002, 2004; Biard et al., 2005, 2007; Haq et al., 1996; Saino et al., 2003; Surai and Speake, 1998). Below we review the consequences of such maternal effects for brain development and emphasize implications for the evolution of brain size.

1.1. Maternal effects via androgens and brains

Yolk androgens have been suggested to have organizational effects on behaviour (reviews in Dufty et al., 2002; Groothuis et al., 2005b). Since the complexity of behaviour corresponds to the extension of the associated neural substrate (Healy et al., 2005; Lefebvre et al., 2004), maternal T-mediated behavioural effects should be evolutionarily linked to brain space. For example, variation in early exposure to androgens could cause variation in several kinds of reproductive behaviour in both sexes that is likely to result from endocrine activation of the neural system (Rhen and Crews, 2002).

T seems to enhance cognitive function and increase brain volumes. In adult rodents and humans, T can influence spatial cognition, spatial learning, visual and verbal memory, visuo-spatial functioning and visuo-motor scanning (e.g., Janowsky et al., 1994; Moffat and Hampson, 1996; Moffat et al., 2002), and it is positively related to the size of different brain areas, such as hippocampus, amygdala, corpus callosum and overall brain size (e.g., Galea et al., 1999; Malsbury and McKay, 1994; Moffat

et al., 1997; Perrot-Sinal et al., 1998). Similar effects of maternal T levels have also been documented for cognitive and neurological traits. The administration of T derivatives to pregnant rats positively affected brain development in their offspring in terms of brain weight and neocortex thickness (Ryzhavskii, 2002; Ryzhavskii et al., 2004). Studies on second-to-fourth digit ratio, a marker of early sex hormone exposure, suggested that prenatal levels of steroids may influence cognitive function in childhood and adulthood of men (Kempel et al., 2005; Williams et al., 2003). The cognitive and neural enhancement that T causes is likely to be due to its positive impact on proliferation, survival, dendritification, the expression of receptors and activation of neurons (Cooke and Woolley, 2005; Fink et al., 1999; Fowler et al., 2003; Garciasegura et al., 1994; Hutchison et al., 1997; Wang and Devries, 1995; Zhang et al., 1999). In adult birds, T-induced neurogenesis may also exist, as androgens appear to produce organizational effects on different brain regions (Absil et al., 2003; DeVoogd and Nottebohm, 1981; DeVoogd, 1991; Gurney and Konishi, 1980; Louissaint et al., 2002; Panzica et al., 1996; Van Meir et al., 2004). In addition, accelerated cerebral development due to maternal T may also be in effect. Prenatal steroid exposure may have long-term neurological and behavioural consequences (Abdelnabi and Ottinger, 2003; Adkins, 1979; Panzica et al., 1996; Seth et al., 2003). The expression of androgen receptors in the embryo suggests that maternal androgens can act via these receptors in the brainstem and syrinx to influence hatching as well as acoustic and visual components of food-begging behaviour (Godsave et al., 2002).

If maternal T can generally enhance neurogenesis in the avian offspring, we hypothesize that mothers of large-brained species would transfer more T to their eggs than small-brained species to provide the hormonal basis for intense neural growth. Such an interspecific pattern would be the first phylogenetic signature of yolk T being a pivotal component of developmental constraints acting on brain size due to its neurogeneric effects.

1.2. Maternal effects via antioxidants and brains

Other molecules may also be stimulating for the maturing brain in the embryo. Specifically, carotenoids and vitamins A and E have antioxidant and immuno-stimulant roles that are of vital importance for developing embryos and young birds, which are subject to increased oxidative stress as a by-product of metabolism associated with rapid development (Blount, 2004; Blount et al., 2000; Surai, 2002). Because of its high rate of oxygen utilization, high polyunsaturated $n-3$ fatty acid content, and relatively low level of endogenous antioxidants, the brain is very susceptible to oxidative stress (Butterfield, 2002; Connor and Menzies, 1995; Hilscherova et al., 2003; Noseworthy and Bray, 1998; Tyurin et al., 2000). Mismanagement of oxidative stress is considered a key component in several neuro-degenerative diseases including Alzheimer's disease, Parkinson's disease, tardive dyskinesia, and amyotrophic lateral sclerosis (Butterfield, 2002; Jenner et al., 1992; Noseworthy and Bray, 1998). As the events related to neurogenesis pass through a succession of stages such as cell birth, migration, differentiation, maturation and cell death, brain ontogeny is especially associated with high rates of metabolic activity (Dodge et al., 1975), which renders it particularly susceptible to oxidation by free radicals (Ahmad, 1995; Buonocore et al., 2001; Slotkin et al., 2005; Surai, 2002). Studies in birds and mammals have shown that antioxidants involving carotenoids and vitamins A and E suppress lipid peroxidation and avoid malformation of the foetal brain (e.g., Feng et al., 2005; Hwang et al., 2004; Sakaki et al., 2001; reviewed in Ramakrishna, 1999).

If oxidative stress by free radicals can have negative effects on brain development, and antioxidants may help eliminate these harmful consequences, this may set the scene for coevolution between brain size and maternally derived carotenoids and vitamins A and E deposited in the egg. Accordingly, we predict that species that have evolved relatively large brains, maternal investment into egg antioxidants is increased to overcome problems of oxidative stress on brain development.

1.3. A comparative study of birds

To demonstrate that maternal effects can mediate the evolution of brains through development, we used data on yolk T, total carotenoids, and vitamins A and E content in 95 bird species, and tested for their positive phylogenetic relationship with relative brain size, as predicted above. Although intraspecific variation in the concentration of these substances exist (e.g. Schwabl, 1999), this is exceeded by even greater interspecific variation, as indicated by the significant repeatability and biologically relevant associations of these maternal traits (Cassey et al., 2005; Gil et al., 2007; Gorman and Williams, 2005). Therefore, evolutionary constraints that arise from the phylogenetic consequences of maternal effects should have consequences for the evolution of overall brain size. Accordingly, we estimated the pair-wise interspecific relationships between relative brain size and different maternal effects while taking common ancestry into account.

We also assessed the simultaneous associations between relative brain size, yolk T and antioxidants, because these may interact in a complex way. For example, T can increase physical activity and early development of musculature (e.g., Henry and Burke, 1999; Park et al., 1999; Sheffield-Moore, 2000; Xu et al., 2004), which may further increase metabolic rate resulting in the production of free radicals and therefore the need for antioxidants. Alternatively, the immuno-suppressive effects of T (Folstad and Karter, 1992; Roberts et al., 2004) would have implications for maternal allocation of immuno-stimulants to eggs, the other functions of carotenoids and vitamins (Blount, 2004; Blount et al., 2000; Groothuis et al., 2005a;

Møller et al., 2000). Accordingly, concentrations of antioxidants in egg yolks may positively correlate with yolk androgen content (Navara et al., 2006). In addition, we also investigate the second-order derivatives, as extremely high T levels may negatively affect the size of brain volumes (Smulders, 2002), and certain cognitive functions (Garamszegi et al., 2007) which may set up different schemes for the evolution of brains at high and low T concentrations.

Finally, we considered two potentially confounding variables, as the relationship between maternal effects and relative brain size may not necessarily arise due to development, but may be indirect due to a third confounding factor. For example, large brained species may be hypothesized to live longer, because long life span may permit extended learning periods that support experience-based cognitive function (Ricklefs, 2004). However, as yolk T can have long-term consequences for survival (Navara et al., 2005; Sockman and Schwabl, 2000), females may be selected to invest more in the yolk to allow longer life span. Therefore, longevity may be related to brain size across species (Ricklefs, 2004), but one can also predict an interspecific relationship between maternal effects and longevity. Longevity would then mediate a relationship between relative brain size and maternal effects without providing a causal relationship between them. In addition, we controlled for developmental traits, as previous comparative studies have revealed complex associations between development and relative brain size and maternal effects, respectively (Bennett and Harvey, 1985a; Gil et al., 2007; Gorman and Williams, 2005; Iwaniuk and Nelson, 2003). Again, if development is related to brain size and maternal effects, this could result in an apparent relationship between the latter two variables.

2. Materials and methods

2.1. Egg T levels

We used published data for yolk T levels from Gil et al. (2007), who present data for more than one hundred species. These data rely on the collection of freshly laid eggs (mean $\pm$ SE $= 7 \pm 1$ per species) from several parts of the world (Europe, America and Africa). Extraction recoveries, cross-reactivity of antibodies, intra- and inter-assay coefficients of variation, but also within-species and between-laboratory repeatabilities are presented in Gil et al. (2007). Repeatability between sources was found to be significant and high ($R > 0.80$), which indicates that any intraspecific variation due to methodological differences between laboratories is negligible when the interest is to explain interspecific variation, and comparison of species-specific mean values thus makes biological sense. Therefore, we also used information on egg T levels from Gorman and Williams (2005), with the exception of *Sayornis saya*, which was an extreme outlier. The repeatability between the two sources based on five overlapping species was $R = 0.822$ ($F_{4,5} = 10.25$, $P = 0.013$). If we only rely on data from Gil et al. (2007), the results and conclusions are very similar. Here we used information on 75 species for which we were able to locate information on relative brain size. Mean yolk T concentrations (in pg mg^{-1}) for each species are given in Appendices A and B.

2.2. Carotenoids and vitamins in eggs

Frozen eggs, kept at $-70\,^{\circ}$C, for 68 species from the above collection were available for antioxidant extraction and assays. Antioxidants were extracted from eggs using 0.100–0.200 g of yolk. Samples were homogenized with 0.7 ml NaCl (5%) and 1 ml ethanol, after which antioxidants were extracted adding 2 ml hexane and further homogenization, centrifugation and collection of the hexane phase (extraction repeated twice). Hexane extracts were pooled and evaporated at 60–65 °C under nitrogen flow, the residue was then dissolved in 0.1 ml dichloromethane and 0.1 ml methanol. Carotenoid concentration, vitamins A and E concentration were determined following previously published procedures (Hõrak et al., 2002; Surai, 2000). Total carotenoid concentration was determined by HPLC with a Spherisorb type S5NH$_2$ reverse-phase column 25 cm $\times$ 4.6 mm (Phase separation, Clwyd, UK) with a mobile phase of methanol-distilled water (97:3), at a flow rate of 1.5 ml min^{-1}. Lutein was used for the calibration (Sigma, Poole, UK). Concentrations of vitamins A and E were determined by injection of samples onto a Spherisorb type ODS2 3-μ C18 reverse-phase column, 15 cm $\times$ 4.6 mm (Phase Separation, Clwyd, UK) with a mobile phase of methanol/distilled water (97:3), at a flow rate of 1.05 ml min^{-1} using fluorescence detection by excitation and emission wavelength of 295 and 330 nm, respectively, for vitamin E and 330 and 480 nm for vitamin A. Peaks of retinol, δ-, γ- and α-tocopherol were identified by comparison with the retention time of standards of tocopherols (Sigma, Poole, UK). All sampled eggs were analysed for total carotenoids, vitamins A and E concentrations. Vitamin E calculated as the summed concentrations of δ-, γ- α-tocopherol is used in subsequent analyses. However, results obtained considering only the major antioxidant form, α-tocopherol (Surai, 2002) were qualitatively similar. Concentrations and not quantity of antioxidants were used as the variable of interest in statistical analysis, because concentration is the main factor in determining physiological action of antioxidants at the level of tissues (Surai, 2002). We note, however, that when we controlled statistically for egg mass and clutch size, the results and the conclusions remain similar to those we report below (this also applies to yolk T levels).

Based on species for which we assayed more than one egg, we calculated within species repeatability for different antioxidants, which were highly significant for each measures (carotenoids, $R = 0.764$, $F_{50,404} = 19.42$, $P < 0.001$; vitamin A, $R = 0.779$, $F_{50,319} = 22.29$, $P < 0.001$; vitamin E, $R = 0.556$,

$F_{50,318} = 8.10$, $P < 0.001$). This suggests that species-specific values of antioxidant levels also make biological sense in an interspecific context. Mean antioxidant concentrations ($\mu g\, g^{-1}$ fresh tissue) for each species are given in Appendices A and B. Here, we used data on antioxidant levels in eggs of 62 species with available information on relative brain size.

2.3. Brain size

Data for brain size (in grams) and the associated body mass were derived from three different sources (Garamszegi et al., 2002; Iwaniuk and Nelson, 2003; Mlíkovsky, 1990). Iwaniuk and Nelson (2003) provide information on volumetric size, which can be readily converted to brain mass (Iwaniuk and Nelson, 2002). In addition, highly significant repeatabilities among studies indicate that information on relative brain size can be combined across sources (Garamszegi and Eens, 2004; Garamszegi et al., 2005b; Lefebvre et al., 2004). We included information on body mass to control for allometric effects, but also to eliminate potential size-dependant bias in maternal effects (we found strong allometric effects on antioxidant levels, but see Gil et al., 2007 for yolk testosterone levels).

2.4. Confounding variables

Information on developmental mode and developmental periods (duration of incubation and nestling periods in days) were obtained from Cramp and Perrins (1985–1994), Glutz von Blotzheim and Bauer (1985–1997), Poole et al. (1993–2002) and Marchant and Higgins (1990). We used median values when a range of values was reported. Developmental mode was coded along a four-point continuous axis based on Starck and Ricklefs (1998): precocial, semiprecocial, semialtricial and altricial. The finer gradations of developmental mode by Starck and Ricklefs were not adopted, because detailed information on nestling morphology and behaviour was not always available, and the sample size for many of their subcategories would have been too small for multivariate analyses, as detailed below (see also Iwaniuk and Nelson, 2003). For simplicity, developmental mode was treated as a continuous variable in the comparative analyses. The statistical reason behind this choice is that although these variables were scored as discrete, intermediate states are biologically meaningful, and different states are thus arbitrary points along a continuum (Sokal and Rohlf, 1995). In addition, in an evolutionary context, a transition between two states of these variables follows non-discrete gradual evolutionary changes. Therefore, the continuous treatment is generally applied in comparative studies that are constrained to use qualitative data for a larger set of species (Bennett and Owens, 2002; Harvey and Pagel, 1991). Thus as a result, one can show qualitatively that a given trait plays a role in the evolution of another, but cannot assess its quantitative importance.

We also extracted information on maximum longevity of birds from standard ornithological handbooks (Cramp and Perrins, 1985–1994; Glutz von Blotzheim and Bauer, 1985–1997; Marchant and Higgins, 1990; Poole et al., 1993–2002), and from online databases provided by USGS Patuxent Wildlife Research Centre (http://www.pwrc.usgs.gov/bbl/homepage/longvlst.htm) and the Max Planck Institute for Demography (http://www.demogr.mpg.de/longevityrecords/ by Carey and Judge, 2002). The information reported in these sources is based on extensive literature search and data provided by major bird ringing schemes. There was a high repeatability between these sources ($R = 0.800$, $F_{493,563} = 9.55$, $P < 0.001$). A previous study adjusted longevity records for sampling effort, because rare and very old individuals may be more likely caught in intensively studied species than in less studied species (Møller, 2006). However, when we estimated research effort by using the number of studies published since 1972 on each species as cited in the ISI Web of Science (http://www.isiknowledge.com/), we found no significant relationship between longevity and research effort ($F_{1,77} = 2.25$, $P = 0.138$). Therefore, we did not consider research effort further.

The entire data set is reported in Appendices A and B. With the exception of developmental mode, all variables were $\log_{10}$-transformed before analyses.

2.5. Comparative analyses

We applied the general method of comparative analysis for continuous variables based on generalized least squares (GLS) models using the statistical software Continuous (Pagel, 1997, 1999a). The GLS model characterizes evolutionary changes along each branch of a phylogenetic tree through the variance components of traits (Pagel, 1997). Hypotheses are tested with likelihood ratio statistics. This compares the log-likelihood of the model corresponding to a null hypothesis (H_0) over the model for an alternative hypothesis (H_1), where the likelihood ratio $= -2\log_e[H_0/H_1]$. The likelihood ratio statistic is asymptotically distributed as a χ^2 variate with degrees of freedom equal to the difference in the number of parameters between the two models. First, we assessed the contribution of scaling parameters sequentially by estimating the maximum likelihood values of the branch length scaling parameter κ, and the phylogeny scaling factor λ (recent simulations showed that the estimation of δ, the overall path length scaling factor is biased (Freckleton et al., 2002), and thus we avoided estimating this parameter). The κ parameter by differentially stretching long and short branches would yield a punctuational mode of trait evolution at $\kappa = 0$, while $\kappa \geqslant 1$ indicates the importance of long branches in trait evolution (gradualism). Values of $\lambda < 1$ would correspond to traits being less similar amongst species than expected from their phylogenetic relationship, while $\lambda = 1$ suggests the reverse. Any of these potential effects present in the data can be detected by

comparing the log-likelihood of a H_0 model containing default ($=1$) values for the scaling parameters with the log-likelihood of an alternative H_1 model in which one parameter is permitted to take its maximum likelihood value. If a significant effect was found ($P<0.05$), the estimated values were used in the final model, otherwise default settings were used. Second, using the appropriate scaling parameters the correlation between pairs of traits

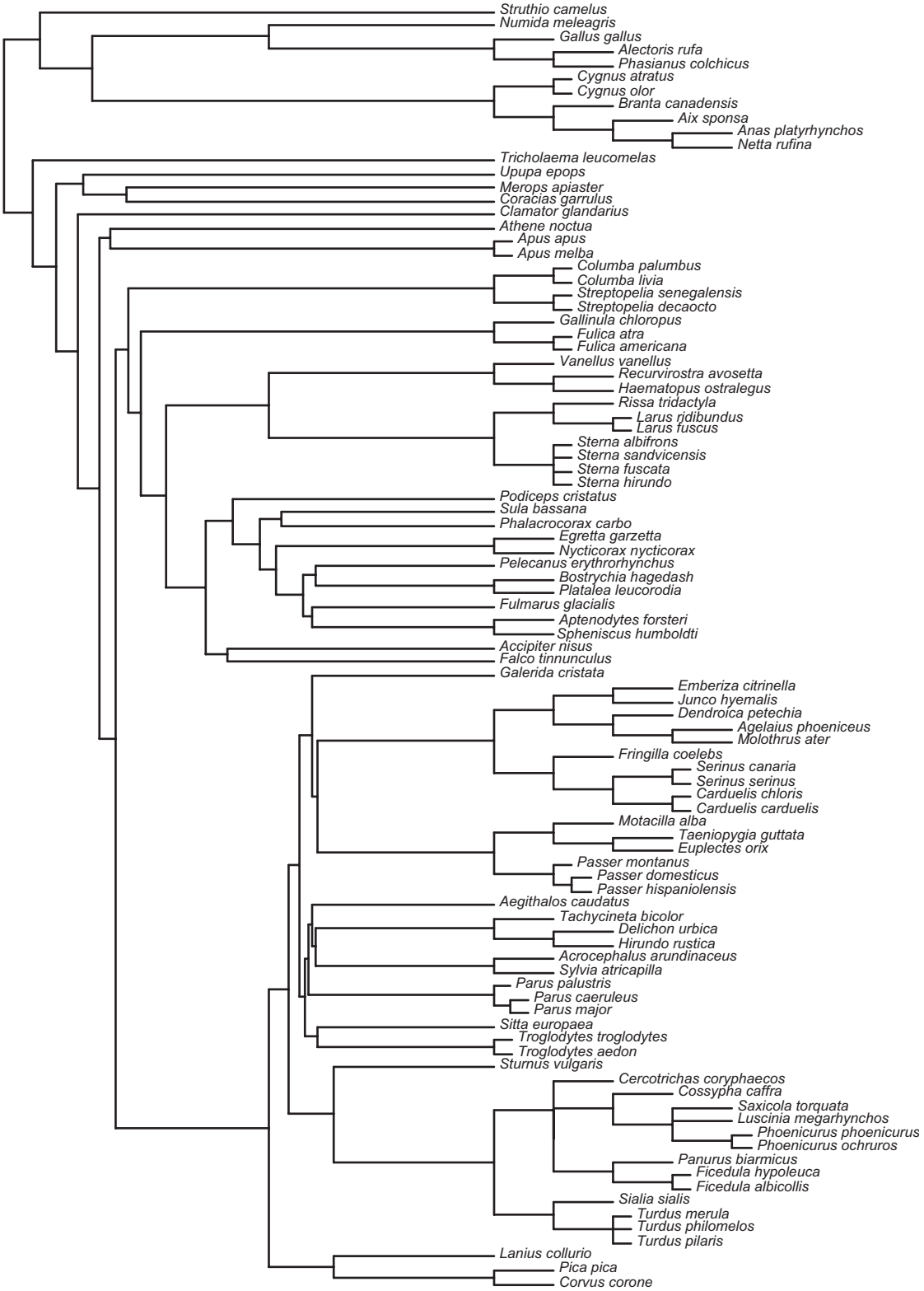

Fig. 1. Phylogenetic hypothesis of passerine birds used for comparative analyses of maternal effects and relative brain size. For sources, see Section 2.

was tested by log-likelihood ratio statistics comparing model H_0 that fits the data forcing the correlation to be zero with the alternative H_1 model, permitting correlated evolution of the two characters. Third, using the best model fitting the data, we estimated the phylogenetic correlation between traits. We assumed that the evolution of traits followed a standard constant-variance random walk evolutionary model, and thus we used the corresponding settings in Continuous (Model A). The appropriate scaling parameters and the log-likelihood ratio statistics testing for correlated trait evolution are presented. When we controlled for potentially confounding factors, such as longevity and developmental mode, we entered these variables one by one together with the variables of interest in the same model, and after estimating the scaling parameters, we calculated the partial phylogenetic correlation for the relationship in question. Each model involved body size to control for its allometric effect on brain size (and its correlation with other traits). The phylogenetic method implemented in the programme Continuous does not allow insight on the phylogenetically transformed data (Pagel, 1999b). For illustrative purposes we present figures based on the raw species data on which we superimpose the phylogenetically corrected regression lines.

Phylogenetic information for our comparative analyses originated mainly from Sibley and Ahlquist (1990) that relied on extensive studies of DNA–DNA hybridization. This phylogeny was supplemented with information from Kimball et al. (1999) for Phasianidae, Suhonen et al. (1994) for Paridae, Møller et al. (2001) for Hirundinidae and Badyaev (1997) for Cardueline finches. We applied branch lengths from the tapestry tree of Sibley and Ahlquist (1990) above the family level. To calculate branch length below the family level, we used the convention that within families the distance between different genera is 3.4 $\Delta T_{50}H$ units, and between species within genera is $1.1\,\Delta T_{50}H$ units (Bennett and Owens, 2002; Sibley and Ahlquist, 1990). The phylogeny is given in Fig. 1. Recent studies indicate that the phylogeny of Sibley and Ahlquist (1990) may be incorrect for some taxa (Barker et al., 2002; Sheldon and Gill, 1996). However, when we reconstructed our composite phylogeny based on Barker et al. (2002) and using equal branch lengths, the conclusions of this paper did not change.

3. Results

3.1. Yolk T and brain size

We found a significant linear relationship between yolk T and brain size corrected for body size in a multiple regression model without taking into account phylogenetic information (full model on brain size including body mass and yolk T as independent variables: $F_{2,73} = 491.8$, $P < 0.001$; body mass: $F_{1,73} = 977.0$, $P < 0.001$; yolk T: $F_{1,73} = 5.339$, $P = 0.024$; Fig. 2a). However, this linear relationship was not significant when similarity among species due to common descent was taken into account (analysis based on Continuous: partial correlation at $\kappa = 0.691$ and $\lambda = 0.959$: $r_{phyl} = 0.142$, $P = 0.223$, $N = 76$).

The graphical inspection of the data revealed that the relationship between relative brain size and T was non-linear. At moderate yolk T levels, increasing hormone levels were accompanied by increasing relative brain size, whereas this effect levelled off at high T levels (Fig. 2b). We modelled this non-linear effect by using a second-order polynomial regression model to the data, and found a significantly improved fit ($F_{1,72} = 8.178$, $P = 0.006$; Fig. 2b). The maximum of the function was found at 21.91 pg mg^{-1} T.

To achieve an analogue modelling in our phylogenetic framework, we added squared yolk T levels to the original Continuous model. From this model, we obtained the phylogenetically corrected correlation matrix of traits, and calculated partial correlations controlling for body size and for first- or second-order derivate of effects of T. Controlling for the second-order derivate, we found that the relationship between yolk T levels and relative brain size was significantly positive (partial correlation at $\kappa = 0.506$ and $\lambda = 0.895$: $r_{phyl} = 0.279$, $P = 0.016$). On the other hand, when first-order T levels were held constant, the model showed significant and negative correlation between brain size and squared yolk T levels (partial correlation: $r_{phyl} = -0.257$, $P = 0.027$). Therefore, the phylogenetic modelling confirmed the curvilinear relationship between relative brain size and yolk T levels. In addition, when we focused on moderate hormone levels (<21.91 pg mg^{-1}) only, which corresponds to the majority of species, by using linear approaches, the phylogenetic correlation between yolk T and relative brain size was positive and significant (partial correlation at $\kappa = 0.679$ and $\lambda = 0.942$: $r_{phyl} = 0.252$, $P = 0.043$, $N = 66$). When we added the second order effect to the model, it was not significantly related to relative brain size (partial correlation at $\kappa = 0.447$ and $\lambda = 0.830$: $r_{phyl} = -0.186$, $P = 0.360$, $N = 66$). This indicates that the relationship between yolk T levels and relative brain size is linear at moderate hormone levels. When we focused on species with high yolk hormone levels ($\geqslant 21.91$ pg mg^{-1}), the partial correlation showed strongly negative effects, but it was non-significant due to the small sample size (partial correlation at $\kappa = 0.000$ and $\lambda = 0.000$: $r_{phyl} = -0.642$, $P = 0.062$, $N = 10$). Hence, the slope for the relationship between relative brain size and yolk T levels is more likely to level off after reaching a maximum than to converge to an asymptote.

To assess the effect of potentially confounding variables, we added developmental mode, the duration of incubation and nestling periods and relative longevity, one by one to the Continuous model which included T levels and its second-order derivative. However, the focal relationship between yolk T and relative brain size remained unaffected (partial correlations at $\kappa = 0.333$–0.653 and $\lambda = 0.731$–0.950,

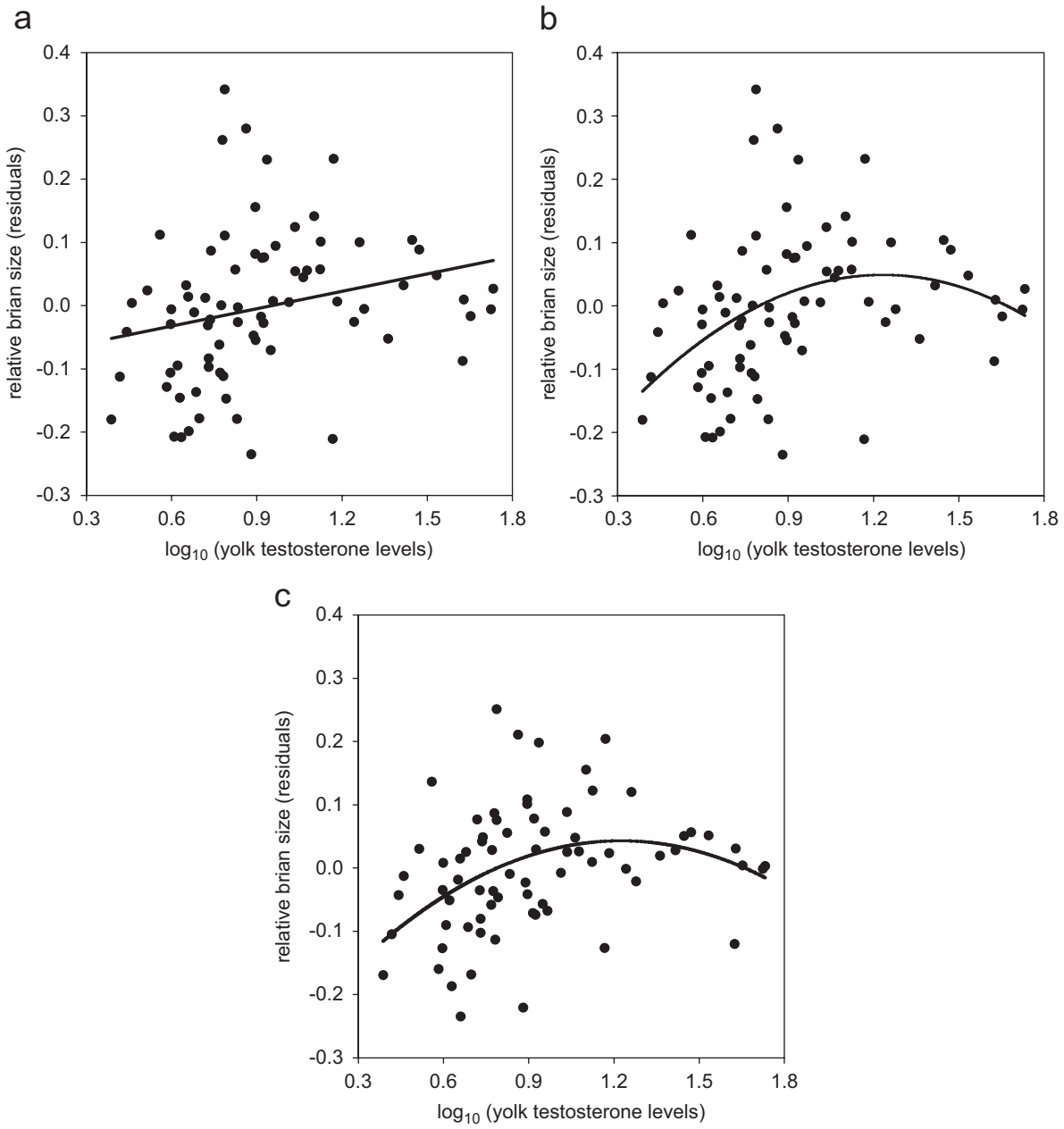

Fig. 2. The interspecific relationship between yolk testosterone levels ($pg\,mg^{-1}$, log-transformed) and brain size when controlling for allometric effects. For illustrative purposes, relative brain size is estimated as residuals from the regression of brain size on body size: (a) The association between traits when assuming a linear relationship. The equation for the regression line is $y = 0.09x - 0.09$. (b) The same data, but when using a second-order polynomial fit. The regression line has the following equation: $y = -0.26x^2 + 0.16x - 0.12$. (c) A curvilinear regression, with the equation: $y = -0.23x^2 + 0.14x - 0.10$, which controlled for the effect of developmental traits on brain size in a form of residuals from the relevant regression. Species-specific values are presented. For phylogenetically corrected statistics, see text.

T: $r_{phyl} = 0.259$–0.334, $P = 0.007$–0.026; T-squared: $r_{phyl} = -0.307$ to -0.241, $P = 0.015$–0.036, $N = 64$–76).

3.2. Antioxidants in eggs and brain size

Following the approaches we employed for yolk T levels, we found significant and positive linear associations between relative brain size and vitamin E levels when controlling for phylogenetic and allometric effects (partial correlation at $\kappa = 0.534$ and $\lambda = 0.908$, $r_{phyl} = 0.275$, $P = 0.035$, $N = 60$, Fig. 3). We found no significant effects for carotenoid levels or vitamin A (partial correlations: carotenoids, $\kappa = 1.000$ and $\lambda = 1.000$, $r_{phyl} = 0.235$, $P = 0.068$, $N = 62$; vitamin A, $\kappa = 1.000$ and $\lambda = 1.000$, $r_{phyl} = 0.073$, $P = 0.589$, $N = 58$). These results were similar when analyses were based on species-specific data,

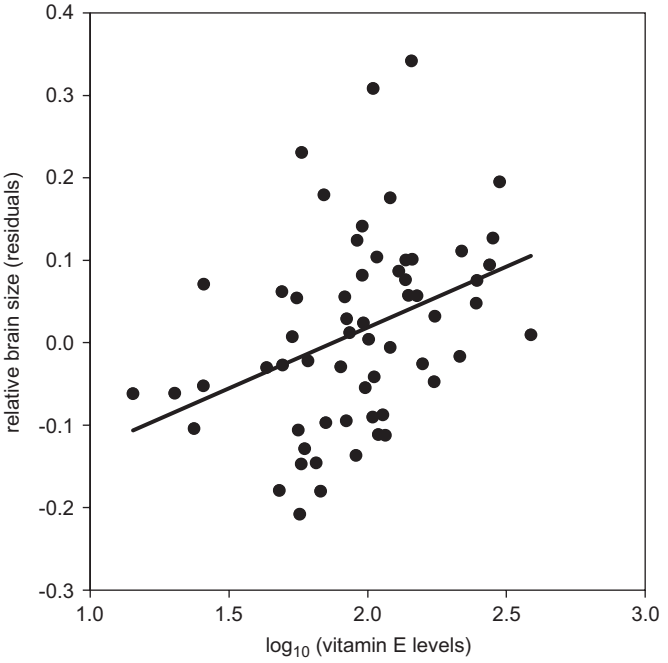

Fig. 3. The association between relative brain size and vitamin E content of the egg ($\mu g\,g^{-1}$, log-transformed). Relative brain size is estimated as residuals from the allometric regression as in Fig. 2. For the corresponding phylogenetic models, see text. The equation for the regression line based on the raw species data: $y = 0.15x - 0.28$.

and polynomial fits to the data or the consideration of two-way interactions did not explain more variance in relative brain size in either case (data not shown).

When we controlled for the potentially confounding variables listed above, the effect size for the relationship between relative brain size and vitamin E remained similar, but its significance was affected (partial correlations at $\kappa = 0.346$–1.000 and $\lambda = 0.846$–0.946: $r_{phyl} = 0.223$–0.301, $P = 0.034$–0.093, $N = 47$–59). The only non-significant effect ($P = 0.093$) emerged, when we controlled for developmental mode, which was significantly related to vitamin E (partial correlation at $\kappa = 1.000$ and $\lambda = 0.949$, when controlling for body size: $r_{phyl} = 0.290$, $P = 0.026$, $N = 60$).

3.3. Associations between maternal effects and brain size

Using the raw species data, we found a positive and significant relationship between levels of yolk T and vitamin E ($r = 0.383$, $P = 0.009$, $N = 46$), but this correlation was not significant when we controlled for phylogenetic effects ($\kappa = 0.111$ and $\lambda = 0.662$, $r_{phyl} = 0.127$, $P = 0.388$, $N = 46$). However, in theory, it remains possible that an apparent relationship between relative brain size and a component of maternal effects is mediated through the other component of maternal effects (see Section 1 for possible mechanisms). To control for this possibility, we simultaneously estimated the effects of yolk T levels and vitamin E levels on relative brain size. Partial phylogenetic correlations showed that yolk T and E vitamins are independently related to brain size when controlling for body size (partial correlations at $\kappa = 0.276$ and $\lambda = 0.737$: T, $r_{phyl} = 0.358$, $P = 0.017$; T^2, $r_{phyl} = -0.324$, $P = 0.032$; vitamin E, $r_{phyl} = 0.374$, $P = 0.012$, $N = 46$).

Table 1
Results of a multiple regression model on brain size based on raw species data, which included all effects as independent variables. The associated AIC values are derived from the model, from which the corresponding effect was excluded

Dependent variable: brain size	F	d.f.	P	AIC
Initial model ($R^2 = 0.955$)	60.77	10.26	<0.001	−176.18
Body size	174.65	1.26	<0.001	−102.57
Yolk testosterone	8.97	1.26	0.006	−167.21
Yolk T × yolk T	8.01	1.26	0.009	−168.24
Carotenoids	0.09	1.26	0.772	−178.05
Vitamin A	0.06	1.26	0.809	−178.09
Vitamin E	0.88	1.26	0.356	−176.94
Incubation period	6.75	1.26	0.015	−169.63
Nestling period	3.25	1.26	0.083	−173.82
Developmental mode	3.85	1.26	0.061	−173.07
Longevity	0.03	1.26	0.871	−178.14

We previously found similar results for analyses conducted at the species level and in a phylogenetic context (note that in the majority of cases λ was significantly smaller than 1, which indicates a weak role for phylogenetic inertia in the data). Hence, we constructed more complex models relying on the raw species data, because modelling the evolution of multiple characters is difficult in comparative programs. We created a full multivariate model, which included brain size as the dependant variable, and maternal effects and all potentially confounding factors as independent predictor variables. This model revealed significant effects for body size, yolk T levels, squared yolk T levels and incubation period, and marginally significant effects for developmental mode and nestling period (Table 1). The effect of vitamin E was not significant due to the effect of developmental mode (if the latter was removed from the model the effect of vitamin E: $F_{1,27} = 5.39$, $P = 0.028$).

4. Discussion

We hypothesized that maternal effects should have consequences for the evolution of relative brain size in birds. Corroborating the predictions of this hypothesis, we found that interspecific variation in yolk T levels was associated with interspecific variation in relative brain size. Interestingly, this relationship appeared to be curvilinear suggesting a role for stabilizing selection. The detected pattern should be robust, as they were independent of other developmental traits, longevity and maternal effects due to other antioxidants. Analyses of antioxidant levels in eggs

revealed that vitamin E concentration was significantly and positively related to relative brain size among species. However, the effects for vitamin E levels were weaker than those for T, as the results were sensitive to developmental mode. When this variable was factored out, the relationship between relative brain size and vitamin E became non-significant. A multiple regression model of brain size showed significant a effect for incubation period and marginally significant effects for developmental mode and nestling period (Table 1) in accordance with earlier findings (e.g. Iwaniuk and Nelson, 2003). We discuss the implications of our findings in association with maternal effects for the evolution of brain size. The evolutionary role of developmental traits is discussed in detail elsewhere (Bennett and Harvey, 1985a; Iwaniuk and Nelson, 2003; Pagel and Harvey, 1988; Portmann, 1947).

4.1. The correlated evolution of brain size and yolk T

Considering the considerable amount of evidence for the neurogeneric effect of T, we predicted that mothers of large brained bird species should invest more T in their eggs than mothers of species with smaller brains. Consistent with this prediction, we found a positive interspecific relationship between yolk T levels and relative brain size. However, this relationship may apply only to moderate levels of T, because at high levels, the relationship was negative. We captured this pattern by applying a curvilinear model, which implied that yolk T levels may have dual effects. At low and intermediate concentrations it may enhance brain development, while at high levels T seems to be suppressive. Therefore, we suggest that the benefits of high yolk T levels in terms of enhanced nestling competition or future reproductive success may be larger than the costs of suppressed brain growth.

Different physiological mechanisms may mediate the negative effects of high T. In his meta-analysis, Smulders (2002) found that different brain volumes were enlarged by seasonally increasing T levels, while the size of the same structures appeared to be reduced after artificial T titre increase by chronic implants. To explain this dual pattern, Smulders (2002) suggested that the administration of T implants does not recreate the complex endocrinological environment, which may lead to unbalanced T metabolism and accumulation in certain brain regions with its associated negative consequences (e.g., Brännvall et al., 2005; Freking et al., 1998). Furthermore, chronic T treatment typically targets high hormone concentrations, which may have negative effects on brain volumes (Smulders, 2002). Finally, T implantation may produce an accompanying rise in glucocorticoids, such as corticosterone, which suppresses neurogenesis (Smulders, 2002, see also Devenport et al., 1992; McEwen, 1999; Sapolsky, 1985). We suggest that these mechanisms may likewise apply to yolk T levels. Via maternal effects, females may be unable to provide all hormone-processing enzymes (aromatase, 5α-reductase, 5β-reductase) that are necessary for T metabolism in the growing brain, which may not produce these enzymes in the corresponding concentration during very early embryonic stages. Additionally, yolk T may also interact with corticosterone, which is produced by the developing embryo (Porter, 2005), or deposited in the egg by the mother (Eriksen et al., 2003; Hayward and Wingfield, 2004; Rettenbacher et al., 2005; Rubolini et al., 2005). Therefore, the detrimental effects of yolk T levels on brain development may arise as a result of a complex interplay with the entire neuro-endocrine milieu that may not be controlled at high hormone levels. Obviously, this suggestion needs further experimental investigation.

The curvilinear relationship between yolk T and relative brain size may also arise due to maternal effects having consequences that differ for sons and daughters. Females may strategically increase yolk T levels when mated to males with a certain quality to produce the most attractive or viable sons (Gil et al., 1999, 2004, 2006; Michl et al., 2005; Strasser and Schwabl, 2004; Uller et al., 2005). Such differential allocation may benefit sons, but have unfavourable consequences for daughters if mothers are unable to differentially allocate maternal factors to eggs of different sex as observed in two different species (Rubolini et al., 2006a; Saino et al., 2006). On the other hand, sex-specific effects of yolk T may sometimes favor females, as it was shown in the zebra finch *Taeniopigya guttata*, in which increased hormone levels caused developmental and behavioural effects in females only (von Engelhardt et al., 2006). Currently, we do not know how maternal androgens specifically mediate sex-specific brain development. However, if such mechanisms exist, selection should act to balance the cost paid by one sex and the benefits of the other sex (Ketterson et al., 2005; Møller et al., 2005). Moreover, if this trade-off differentially constrains investments in female and male offspring at different T levels, such dose- and sex-dependant roles may shape the evolution of brain size differently in males and females. Hence, it is plausible that stabilizing selection on relative brain size in association with yolk T levels arises from several complex sex-dependant mechanisms. We made an effort to study the potential role of such sex-dependant patterns by using information on sex-specific brain size (see Garamszegi et al., 2005a, b), but we were unable to draw convincing conclusions from these analyses due to small sample sizes.

4.2. The correlated evolution of brain size and antioxidants

Vitamin E is one of the best candidates to function as a biological antioxidant, as it truly ameliorates the effect of oxidative stress (Hartley and Kennedy, 2004). In contrast, carotenoids are less effective in this defence, because they can be oxidized at many different sites in their long conjugated aliphatic chains (Hartley and Kennedy, 2004). However, vitamin A is involved in vertebrate embryonic development (Zile, 2001) and certain carotenoids are precursors of vitamin A (Halliwell and Gutteridge, 1999),

and thus they may indirectly affect brain development. We found a significant and positive relationship between vitamin E levels and relative brain size, while vitamin A and carotenoid content varied independently of the rate of encephalization (we note, however, that these latter effects were in the expected directions, and the relationship for egg carotenoids nearly reached statistical significance). The results for vitamin E appear in line with our working hypothesis that maternal effects favor the evolution of large brains. Hence, the antioxidant role played by vitamin E may be important due to high levels of metabolism for brain development that is particularly susceptible to oxidative stress.

However, the positive relationship between relative brain size and vitamin E should be interpreted with caution, as the significance of the relationship was affected when we considered developmental mode as a confounding factor, which warrants discussion. First, the lower power of the corresponding tests may have contributed to this phenomenon. These analyses relied on smaller sample sizes, and the phylogenetic model that involved developmental mode still revealed effect sizes for vitamin E larger than 0.2. Second, the relationship between vitamin E and relative brain size may be indirect and mediated through a third factor. For example, yolk T enhances the activity and development of several embryonic tissues potentially elevating levels of free radicals, whereas it may also suppress the maturing immune system (see Section 1). Similarly, in altrical nestlings, intense growth occurs post hatching (Starck and Ricklefs, 1998), which may also have consequences for the production of free radicals. Therefore, when females put large amounts of T in their eggs or have nestlings with fast development, to compensate for the associated side effects due to oxidative stress, they may also increase the amount of antioxidants and immuno-stimulants transferred. Under this scenario, females vary the levels of vitamin E irrespective of its effect on brain development, which would in fact lead to the uncoupled evolution of brain size and maternal effects due to vitamin E. However, given the correlative nature of our study, we cannot disentangle such indirect and direct effects of vitamin E on brain development.

4.3. Constraints on brain development and the evolution of large brains

Several comparative studies suggested that that the possession of elaborate cognitive capacities and the associated neural space involve costs, as the development of large brains requires substantial investment (Bennett and Harvey, 1985a; Iwaniuk and Nelson, 2003; Pagel and Harvey, 1988; Portmann, 1947; Ricklefs, 2004). Here, by exploring the interspecific association between maternal effects and relative brain size, we demonstrated that conditions during early development may have consequences for the evolution of relative brain size. Accordingly, selection on brain size can only act in species that already have the molecular basis for brain development due to maternal effects, or intense selection on brain size may also favor females that increase the amount of maternal effects allocated to eggs. These evolutionary scenarios suggest that mothers through their maternal effects are able to influence the maturation and the growth of the brain of their offspring, which has effects on the relative size of the brain in adulthood. Given the taxon specific patterns (Bennett and Harvey, 1985a; Iwaniuk and Nelson, 2003; see also Pappas et al., 2006), covariation between different developmental traits and maternal effects (Gorman and Williams, 2005; Iwaniuk and Nelson, 2003; Navara et al., 2006, this study) and the potential for non-linear associations between traits (this study), the evolutionary relationship between development and relative brain size may turn out to be very complex. Here, we provided the first indication that maternal effects should be considered as important developmental constraints on the evolution of the central nervous system.

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Appendix A

Yolk testosterone levels (T), antioxidant content (carotenoids, vitamins A and E) and brain size in birds. Data used in the phylogenetic analyses, sorted alphabetically by species (Table A.1). For sources, see Section 2.

Appendix B

Phenotypic traits of birds that are used as confounding variables in the phylogenetic analyses (Table B.1). See Section 2 for sources.

Table A1
Yolk testosterone levels (T), antioxidant content (carotenoids, vitamins A and E) and brain size in birds

Species	T (pg mg^{-1})	Carotenoid (μg g^{-1})	Vitamin A (μg g^{-1})	Vitamin E (μg g^{-1})	Brain size (g)
Accipiter nisus		7.40	0.745	25.69	3.03
Acrocephalus arundinaceus	5.48	36.06	1.023	129.59	0.94
Aegithalos caudatus	3.27	52.13	7.066	96.43	0.47
Agelaius phoeniceus	27.99	94.77	1.830	107.77	1.76
Aix sponsa	6.81				4.30
Alectoris rufa	4.06				2.00
Anas platyrhynchos	5.44	15.60	1.099	60.88	5.88
Aptenodytes forsteri		8.59		218.27	46.08
Apus apus	4.98				0.69
Apus melba	2.44	2.87	0.296	67.64	1.13
Athene noctua	7.28				3.92
Bostrychia hagedash	8.62	6.90	2.099	57.84	10.55
Branta canadensis		22.08		83.95	10.78
Carduelis carduelis	42.53	101.93	1.941	387.57	0.64
Carduelis chloris	26.08	103.71	1.194	174.67	0.91
Cercotrichas coryphaecos		22.81	0.380	49.35	0.75
Clamator glandarius	2.77	87.85	1.286	105.49	1.88
Columba livia	3.94	36.70	0.759	56.27	2.33
Columba palumbus	4.58				2.46
Coracias garrulus	4.48				2.30
Corvus corone		72.11	2.630	104.47	8.28
Cossypha caffra	8.42	36.97	1.010	136.89	1.06
Cygnus atratus	23.02	14.26	4.406	25.59	12.19
Cygnus olor		12.59	3.581	23.66	15.40
Delichon urbica	4.86	14.71	3.194	90.73	0.46
Dendroica petechia	54.00				0.54
Egretta garzetta	5.34				3.70
Emberiza citrinella	42.19	70.94	1.473	113.22	0.72
Euplectes orix	8.30	162.09	4.753	247.90	0.79
Falco tinnunculus		58.68	2.848	69.61	3.79
Ficedula albicollis	7.73	45.76	0.782	173.30	0.47
Ficedula hypoleuca	6.05	27.25	1.386	109.11	0.45
Fringilla coelebs	6.81				0.77
Fulica americana		130.92			2.97
Fulica atra	5.90				3.20
Fulmarus glacialis		7.28	5.546	120.50	6.68
Galerida cristata	6.67	73.18	1.018	150.40	1.15
Gallinula chloropus	6.19	27.66	2.363	57.61	2.18
Gallus gallus	4.31	10.65	3.182	56.99	2.94
Haematopus ostralegus		34.17	2.185	43.23	3.90
Hirundo rustica	7.87	46.58	2.326	97.97	0.63
Junco hyemalis	29.67				0.86
Lanius collurio	11.93	22.20	0.699	82.80	1.02
Larus fuscus		71.61			5.70
Larus ridibundus	15.25				2.93
Luscinia megarhynchos	5.95				0.73
Merops apiaster	4.25	67.25	3.302	65.30	0.90
Molothrus ater	14.81				2.19
Motacilla alba	5.38	42.63	1.491	70.74	0.62
Netta rufina	5.86	12.59	2.588	14.26	5.23
Numida meleagris	7.59				3.98
Nycticorax nycticorax	6.12				6.40
Panurus biarmicus	3.95	27.04	0.123	79.87	0.57
Parus caeruleus	13.32	16.59	3.010	144.74	0.66
Parus major	18.27	25.18	2.876	137.27	0.86
Parus palustris	7.85	16.86	1.828	95.69	0.68
Passer domesticus	34.16	7.88	1.264	246.28	0.97
Passer hispaniolensis		13.19	2.303	49.12	0.97
Passer montanus	17.48	17.74	2.823	157.94	0.75
Pelecanus erythrorhynchus		151.01	5.105	299.23	24.81
Phalacrocorax carbo	9.26	22.69	1.657	275.39	10.73
Phasianus colchicus					3.83
Phoenicurus ochruros		42.81	0.814	104.36	0.70

Table A1 (*continued*)

Species	T ($pg\,mg^{-1}$)	Carotenoid ($\mu g\,g^{-1}$)	Vitamin A ($\mu g\,g^{-1}$)	Vitamin E ($\mu g\,g^{-1}$)	Brain size (g)
Phoenicurus phoenicurus	5.38				0.51
Pica pica	6.12	86.58	1.621	143.54	5.49
Platalea leucorodia	7.85				10.80
Podiceps cristatus					3.52
Recurvirostra avosetta	14.69				2.00
Rissa tridactyla	11.59				4.13
Saxicola torquata	18.95				0.63
Serinus canaria	53.07				0.64
Serinus serinus	45.00	115.86	0.968	214.85	0.48
Sialia sialis	10.83	71.88	0.607	91.53	1.23
Sitta europaea	12.64	16.75	2.163	95.57	1.09
Spheniscus humboldti		1.50	3.327	283.14	17.03
Sterna albifrons	8.89				0.90
Sterna fuscata	9.05	21.50	0.300	53.54	2.40
Sterna hirundo	8.39				1.82
Sterna sandvicensis	10.31				2.80
Streptopelia decaocto					1.58
Streptopelia senegalensis	3.82	28.58	0.316	59.20	1.29
Struthio camelus	6.76	28.90	3.112	48.07	41.69
Sturnus vulgaris	3.62				1.99
Sula bassana	6.01				19.20
Sylvia atricapilla	4.55				0.71
Tachycineta bicolor	2.62	17.19	0.998	115.66	0.57
Taeniopygia guttata	4.18	60.52	1.123	83.71	0.44
Tricholaema leucomelas	5.22	44.02	0.552	86.05	1.02
Troglodytes aedon	4.78				0.54
Troglodytes troglodytes	2.89	26.09	2.030	100.68	0.50
Turdus merula	8.22				1.63
Turdus philomelos	13.28	36.03	1.680	140.29	1.59
Turdus pilaris	10.85	11.50	1.529	55.60	1.92
Upupa epops	3.97	17.27	0.426	120.44	1.22
Vanellus vanellus		6.18	1.658	20.19	2.18

Data used in the phylogenetic analyses, sorted alphabetically by species. For sources, see Section 2.

Table B1
Phenotypic traits of birds that are used as confounding variables in the phylogenetic analyses

Species	Body mass (g)	Incubation period (day)	Nestling period (day)	Developmental mode	Longevity (years)	Research effort
Accipiter nisus	218.63	33.5	32.0	3	13.3	199
Acrocephalus arundinaceus	21.90	14.0	12.8	4	7.9	141
Aegithalos caudatus	7.80	14.3	16.0	4	8.1	41
Agelaius phoeniceus	67.17	12.0	10.3	4	15.8	446
Aix sponsa	651.50	30.0		1	21.3	85
Alectoris rufa	335.00	23.5	50.0	1	6.1	104
Anas platyrhynchos	1165.66	27.6	55.0	1	26.6	1375
Aptenodytes forsteri	33000.0	64.5	149.8	3		106
Apus apus	38.90	20.2	42.5	4	21.0	60
Apus melba	100.00	20.0	50.0	4	26.0	14
Athene noctua	142.00	27.5	31.5	3	9.5	46
Bostrychia hagedash	1168.00	27.7	39.7	2		4
Branta canadensis	2958.00	26.5	56.0	1	25.9	293
Carduelis carduelis	14.87	12.2	15.0	4	8.4	18
Carduelis chloris	25.75	13.0	15.2	4	12.6	88
Cercotrichas coryphaecos	23.10	14.5	16.5	4		0
Clamator glandarius	144.25	12.9	20.9	4		54
Columba livia	288.17	17.5	36.0	4	6.2	1869
Columba palumbus	478.94	17.0	33.0	4	16.3	54
Coracias garrulous	153.00	18.0	26.5	4	9.1	8
Corvus corone	522.61	18.5	33.0	4	19.0	134

Table B1 (*continued*)

Species	Body mass (g)	Incubation period (day)	Nestling period (day)	Developmental mode	Longevity (years)	Research effort
Cossypha caffra	28.50	16.1	16.3	4		4
Cygnus atratus	5342.50	40.5	160.0	1		30
Cygnus olor	10500.0	36.0	135.0	1	20.8	121
Delichon urbica	14.83	14.7	25.7	4	14.5	107
Dendroica petechia	9.80	11.3	8.3	4	9.9	115
Egretta garzetta	500.00	21.5	42.5	3		96
Emberiza citrinella	27.93	13.0	12.2	4	9.5	94
Euplectes orix	16.30	13.2	15.2	4	11.8	16
Falco tinnunculus	207.74	28.0	31.3	3	16.1	308
Ficedula albicollis	10.30	12.9	16.1	4	7.9	176
Ficedula hypoleuca	12.76	13.7	15.1	4	15.0	744
Fringilla coelebs	22.07	12.6	13.6	4	14.0	181
Fulica americana	540.50	23.5	75.0	1	21.6	101
Fulica atra	526.21	22.5	58.0	1	18.3	90
Fulmarus glacialis	622.00	50.5	49.5	2	27.2	111
Galerida cristata	36.22	11.7	13.5	4	6.0	14
Gallinula chloropus	303.01	21.5	36.3	1	10.7	65
Gallus gallus	700.00	21.0		1		1258
Haematopus ostralegus	550.00	15.0	36.0	1	36.0	360
Hirundo rustica	18.78	14.4	20.1	4	11.0	592
Junco hyemalis	18.23	12.5	10.5	4	9.7	220
Lanius collurio	29.02	14.0	14.5	4	7.4	70
Larus fuscus	870.00	25.5	35.0	2	26.1	99
Larus ridibundus	270.77	24.0	35.0	2	25.1	172
Luscinia megarhynchos	19.40	13.0	11.0	4	7.9	82
Merops apiaster	55.80	20.0	29.2	4		41
Molothrus ater	57.90	11.0	10.5	4	16.6	428
Motacilla alba	21.84	12.7	13.9	4	9.9	43
Netta rufina	1105.50	27.0	31.5	1	7.2	18
Numida meleagris	1406.33	24.5	30.0	1		179
Nycticorax nycticorax	763.05	22.5	38.8	3	21.1	119
Panurus biarmicus	13.94	12.0	11.5	4	6.0	23
Parus caeruleus	10.40	14.1	18.5	4	12.3	338
Parus major	17.24	13.7	19.0	4	15.0	1277
Parus palustris	12.00	14.0	18.5	4	11.5	94
Passer domesticus	27.40	12.2	14.5	4	13.2	712
Passer hispaniolensis	25.67	11.3	13.1	4	11.2	15
Passer montanus	23.19	12.5	17.5	4	8.4	72
Pelecanus erythrorhynchus	7000.00	30.0	40.3	4		2
Phalacrocorax carbo	2196.11	29.2	42.7	4	16.7	247
Phasianus colchicus	1168.67	23.0		1	7.6	288
Phoenicurus ochruros	27.00	14.0	15.5	4	8.4	27
Phoenicurus phoenicurus	14.29	13.0	14.5	4	9.5	38
Pica pica	206.06	21.1	27.1	4	9.9	250
Platalea leucorodia	1700.00	24.5	47.5	3	28.1	16
Podiceps cristatus	913.64	26.0		1	9.7	51
Recurvirostra avosetta	340.00	23.4	38.0	1	24.5	35
Rissa tridactyla	442.70	26.8	42.1	2	17.5	312
Saxicola torquata	15.30	13.5	13.5	4	6.0	64
Serinus canaria	15.60	13.5	16.0	4		120
Serinus serinus	9.50	12.8	15.1	4	8.6	33
Sialia sialis	31.07	15.7	17.3	4	9.2	100
Sitta europaea	22.52	15.4	23.8	4	9.0	92
Spheniscus humboldti	4600.00			3		64
Sterna albifrons	40.00	21.5	19.5	2	22.1	22
Sterna fuscata	185.50	28.3	59.0	2	33.9	42
Sterna hirundo	127.50	21.3	14.3	2	25.0	286
Sterna sandvicensis	250.00	25.0	29.0	2	20.6	25
Streptopelia decaocto	225.27	15.0	17.0	4	13.7	54
Streptopelia senegalensis	103.00	13.3	16.3	4	5.7	23
Struthio camelus	97666.7	45.5		1		452
Sturnus vulgaris	81.51	12.2	21.0	4	18.9	1253
Sula bassana	3200.00	44.0	90.0	4	21.0	36

Table B1 (*continued*)

Species	Body mass (g)	Incubation period (day)	Nestling period (day)	Developmental mode	Longevity (years)	Research effort
Sylvia atricapilla	17.59	11.3	11.3	4	8.0	194
Tachycineta bicolor	20.10	14.8	20.5	4	11.0	234
Taeniopygia guttata	11.50	13.5	21.0	4		608
Tricholaema leucomelas	35.00	14.5	35.0	4		0
Troglodytes aedon	11.58	12.5	17.0	4	8.0	160
Troglodytes troglodytes	9.42	16.0	16.9	4	4.9	138
Turdus merula	98.79	13.4	13.6	4	20.3	281
Turdus philomelos	67.48	13.3	13.1	4	13.8	64
Turdus pilaris	98.60	12.0	13.5	4	18.0	53
Upupa epops	53.81	15.5	28.2	4	11.1	19
Vanellus vanellus	208.13	26.5	33.0	1	19.9	180

See Section 2 for sources.

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