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Within-Clutch Variation in Yolk Testosterone as an Adaptive Maternal Effect to Modulate Avian Sibling Competition: Evidence from a Comparative Study

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abstract: In many species, embryos are exposed to maternal hormones in utero, in the egg, or in the seed. In birds, mothers deposit substantial testosterone into their eggs, which enhances competitive ability of offspring. These maternal testosterone concentrations vary systematically within clutches in different patterns and may enable mothers to adaptively fine-tune competitive hierarchies within broods. We performed a comparative analysis to investigate this hypothesis using a broad set of avian species. We expected species with small size differences among siblings (arising from small hatching asynchrony or slow growth rates) to aim for survival of the whole brood in good years and therefore compensate last-hatching eggs with relatively more testosterone. We expected species with large size differences among siblings (large hatching asynchrony or fast growth rates) to produce surplus young as insurance against failed offspring and to facilitate elimination of redundant surplus young by bestowing last-hatching eggs with relatively less testosterone. As predicted, we found that maternal testosterone compensation to last-hatching eggs is stronger when size differences among siblings become smaller. Maternal testosterone compensation to last-hatching eggs also correlated negatively with hatching asynchrony and growth rates. These findings provide evidence for correlated evolution of several maternal effects that together support different maternal reproductive strategies.

Keywords: maternal effects, testosterone, hatching asynchrony, sibling competition, siblicide, brood reduction.

Introduction

Maternal effects, which occur when the phenotype of the mother influences the phenotype of her offspring, were once considered to be little more than nuisance variation in heritability estimates (Wolf and Wade 2009). But since the late 1990s, evolutionary ecologists have pushed maternal effects into the spotlight as one of the most important influences on offspring phenotype (Mousseau and Fox 1998; Wolf and Wade 2009). This new interest in the potential adaptive nature of many maternal effects (Marshall and Uller 2007) stimulated substantial theoretical and empirical work that addressed whether mothers enhance the reproductive value of individual offspring by matching offspring phenotype to the local environment (Marshall and Uller 2007). Less attention has been paid to the fact that maternal effects can enhance reproductive value at the brood level by providing an efficient mechanism for mothers to adjust brood size to current food conditions, because food availability is often unpredictable. Throughout the animal and plant kingdom, parents chronically overproduce young, engendering sibling rivalry when young share limited space and resources (e.g., parasitoid wasps [Pexton and Mayhew 2002], damselfly larvae [Anholt 1994]; cleistogamous grass [Cheplick 1992]; piglets [Fraser and Thompson 1991]; see also Mock and Parker 1997). Surplus young can provide extra reproductive value in years when resources are plentiful or can serve as replacement units when other progeny fail (Mock and Parker 1997). However, they become a liability when the number of young exceeds what parents can afford to rear by reducing the per capita share in parental care, which undermines average offspring quality and recruitment prospects.

Parental favoritism (Mock and Parker 1997) constitutes a specific class of maternal effects that create competitive hierarchies among offspring of the same reproductive attempt (such as seeds of the same fruit, eggs of the same clutch, and pups of the same litter). These hierarchies channel the bulk of the resources to the more competitive elements of the sibship when resources become scarce, efficiently culling redundant offspring via targeted starvation. Mothers can create such competitive asymmetries by varying propagule size, hormones, cytoplasmic factors, the time at which propagules are released, and when they

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initiate development and also how fast they develop (Mousseau and Fox 1998; Groothuis et al. 2005*b*). Such mechanisms have been reported in a taxonomically wide range of species (e.g., O'Gara 1969; Ganeshaiah and Uma Shaanker 1988; Fox and Czesak 2000), although whether they represent adaptive maternal strategies is rarely tested. Furthermore, evolutionary studies rarely address more than one maternal effect despite the diverse nongenetic mechanisms by which mothers can influence offspring phenotypes that may act simultaneously. Theoretically, multiple maternal effects should function in concert to support a reproductive strategy that will maximize the mother's fitness (Marshall and Uller 2007). Here, we investigate interspecific correlations among several maternal effects that produce varying degrees of competitive asymmetries within sibships and may adaptively support a maternal reproductive strategy of either culling offspring or promoting survival of all offspring.

One primary avenue by which mothers can produce competitive hierarchies among siblings is by varying propagule size (e.g., echinoderms [Turner and Lawrence 1977]; bryozoa [Marshall et al. 2003]; iguanas [Castro-Franco et al. 2011]; wild radish [Stanton 1984*a*]). Variation in propagule size creates size differences among siblings. Larger propagules produce larger offspring that are more likely to survive (e.g., plants [Black 1958]; arthropods [Fox and Csesak 2000]; fish [Einum and Fleming 2004]; birds [Krist 2011]). In many bird species, egg size increases or decreases over the laying sequence within the clutch in relation to their ecology or reproductive strategy (Slagsvold et al. 1984).

Mothers can create size advantages in certain offspring also by accelerating their time of emergence from the egg or seed. For example, in plants, mothers can vary seed coat thickness, which determines permeability and light exposure and therefore timing of germination (Donohue 2009). Earlier germination confers a size advantage (Stanton 1984*b*) in sibling competition (Cheplick 1992). In passalid beetles, intermittent oviposition creates hatching asynchrony, which gives early hatching beetles a developmental advantage in lethal sibling combat (Ento et al. 2010). In birds, mothers commence incubation before clutch completion so that early-laid eggs (eggs laid before the start of incubation) hatch together (core chicks) followed by the later-hatching eggs (eggs laid during the days that follow the onset of incubation), which produce the asynchronous marginal chicks (sensu Mock and Forbes 1995; Mock and Parker 1997). Core chicks grow substantially before the last eggs hatch, so marginal young experience a significant size and developmental disadvantage in the competitive arena and consequently are more likely than core young to die before maturity (Mock 1984; Magrath 1990).

In addition to varying size of offspring, mothers provision them with variable concentrations of maternal hormones. Maternal hormones influence gene expression and metabolism in seeds (Donohue 2009), can cause diapause in insect eggs (Mousseau and Dingle 1991), determine offspring sex in turtles (Bowden et al. 2000), behaviorally lateralize fish (Schaafsma and Groothuis 2012), and affect later stress sensitivity in mammals (Cottrel and Seckl 2009; Weinstock 2008). Their effect on sibling competition has been studied most extensively in birds. The seminal finding of Schwabl (1993) that avian egg yolks contain high concentrations of maternal androgens and that these concentrations increase or decrease over the laying sequence (reviewed in von Engelhardt and Groothuis 2011) opened up the possibility that mothers might adaptively modulate the asymmetries in competitive ability among brood mates via hormonal means (Schwabl 1993, 1996; Groothuis et al. 2005*b*). More often than not, maternal androgens in avian eggs accelerate pre- and postnatal development rate and result in more competitive behavior in young birds, although they also carry some costs, such as compromised immune function and elevated energy expenditure (reviewed in von Engelhardt and Groothuis 2011). The balancing of these benefits and costs to the offspring can therefore yield unique optima for different positions in the laying sequence and may explain the substantial variation in yolk androgen concentrations within clutches. Several studies indicate that genes determine a significant portion of the variation in maternal testosterone transfer to clutches (Gil and Faure 2007; Bertin et al. 2008; Okuliarova et al. 2011), including the pattern in which mothers deposit testosterone in subsequent eggs over the laying sequence (Groothuis et al. 2008). This establishes clear potential for natural selection to shape maternal strategies of differential yolk androgen allocation according to positions of offspring in the brood competitive hierarchy and their corresponding reproductive value.

Birds have proven to be a useful model for studying maternal hormone allocation as well as other maternal effects associated with parental favoritism for several reasons. First, the resources and other maternal substances that mothers transfer to eggs are easily measured and manipulated, because the egg is relatively large and the embryo develops outside the mother's body. Second, competitive hierarchies within broods, individual offspring quality, and mortality are easily assessed, both in the laboratory and in the field. We therefore capitalize on the wealth of data available for avian taxa to investigate whether suites of correlated maternal effects that influence sibling competition might have arisen from long-term evolutionary change driven by the emergence of alternative maternal reproductive strategies that promote survival of the whole brood versus partial reduction of the brood.

The potentially adaptive functions of systematic variation in avian egg size over the laying sequence and size asymmetries within avian broods that arise from hatching asynchrony have already been studied using a comparative approach (Slagsvold et al. 1984; Magrath 1990; Amundsen and Slagsvold 1991; Stoleson and Beissinger 1995). To date, however, such a study is lacking for patterns of yolk androgens over the laying sequence. The main adaptive explanation (Schwabl 1993; Lipar et al. 1999) for within-clutch variation in yolk testosterone (T), the most well-studied yolk androgen, proposes that species compensate marginal young with relatively higher maternal T exposure to boost their competitive ability and mitigate the disadvantage hatching asynchrony imposes on marginal young. Therefore, when breeding conditions are favorable, yolk T exposure should enable all marginal young to survive alongside the core and contribute extra reproductive value to the brood. However, such species should produce broods that contain only small size asymmetries among siblings, so that, via maternal T compensation, marginal young are able to overcome their initial competitive disadvantage when parents can afford to rear all young. This prediction arises from the fact that past experimental work shows that the increase in competitive ability attributable to exposure to elevated yolk T is relatively small compared with the large handicap imposed on marginal chicks by a big delay in hatching (Schwabl 1993, 1996; Eising et al. 2001). Because small size asymmetries among siblings, coupled with an expectation of high maternal T compensation in marginal eggs, should help marginal offspring to survive, we will refer to these species as brood survival species.

Species should not compensate marginal eggs with elevated T if sibling size asymmetries are large, because the extra maternal T exposure could not significantly counteract a large size disadvantage. Moreover, marginal young would still be burdened with the costs of elevated maternal T exposure. Species produce large size asymmetries to facilitate adaptive brood reduction of redundant marginal young (Forbes and Mock 2000). These asymmetries might actually be enhanced by giving marginal offspring a lower dose of yolk T compared with that given to the core offspring (Schwabl et al. 1997). Species with large sibling size asymmetries have been suggested to produce marginal young as "insurance" in case a core offspring fails because of stochastic processes, such as developmental problems or predation (Forbes 1990; Forbes and Lamey 1996). For example, in obligately siblicidal species, if the core offspring survives, it almost always eliminates the marginal offspring in a fatal fight to the death (Anderson 1990; Mock et al. 1990). The inevitable victory of the core chick is fixed in advance by the mother via an insurmountable size advantage caused by extreme hatching asynchrony (Forbes and Mock 2000). Insurance value for marginal young has also been documented for a wide range of facultative brood reducers, including raptors, parrots, and passerines (Wiebe 1996; Forbes et al. 1997). Evolutionary models demonstrate that producing insurance offspring benefits species with large clutches (Forbes 1990), which have a higher cumulative probability of failure of a core offspring. Broods from these species are expected to contain substantial size disparities among siblings despite relatively narrow hatching spreads, because growth rates tend to be fast (Ricklefs 1968), which would cause core chicks to attain considerable mass by the time marginal chicks hatch. In species that produce marginal young for insurance, brood reduction occurs often, whether it is stochastic mortality of a random nestling or nonrandom elimination of a costly redundant marginal offspring (Dorward 1962; Forbes 1990), so we will refer to these species as brood reducers. If large size asymmetries and low maternal T compensation are adaptations to promote brood reduction in species producing insurance offspring and small size asymmetries, and if high maternal T compensation are adaptations to promote survival of marginal young in good years, then species with small size asymmetries between core and marginal offspring are expected to provide more compensatory maternal T to marginal eggs than are species with large size asymmetries.

In this study, we perform an interspecific comparative study of within-clutch patterns of yolk T concentrations to investigate whether mothers adaptively allocate yolk T to modulate differences in competitive ability among brood mates. The comparative approach has been successfully applied for testing hypotheses about average clutch levels of yolk androgens (Gorman and Williams 2005; Gil et al. 2007; Schwabl et al. 2007; Martin and Schwabl 2008). To the best of our knowledge, this is the first comparative study of within-clutch variation of these hormones.

First, we tested our prediction that compensatory maternal T for marginal offspring correlates negatively with size disparities within broods. We then investigated how compensatory maternal T correlates with three different factors that cause size disparities within broods. As mentioned above, size asymmetries among siblings develop in species with large hatching asynchrony and in species that have relatively small hatching asynchrony but fast growth rates. In addition, egg size changes linearly over the laying sequence in many species, contributing to some extent to additional size variation among siblings, although much less than hatching asynchrony (Slagsvold et al. 1984). We therefore expect compensatory maternal yolk T to correlate with any size disadvantage experienced by marginal young, whether it arises as a result of a severe delay in hatching, fast growth in core young, or small eggs. We

tested our prediction that compensatory maternal T correlates negatively with hatching spread and growth rates and correlates positively with changes in egg size over the laying sequence.

Material and Methods

Life-History Variables

We collected data on average hatching spread, clutch size, laying intervals between subsequent eggs within a clutch, logistic growth rate constants, and average egg mass from databases and handbooks (Cramp 1998; Poole 2005; de Magalhaes and Costa 2009), the primary literature, or our own laboratory data (available in Dryad: http://dx.doi.org/10.5061/dryad.b87j7). If estimates for certain parameters were available from several sources, we averaged all values.

Siblings can be divided into two distinct classes of competitive ability: the synchronously hatching core part of the brood, and the asynchronously hatching marginal part of the brood (Mock and Forbes 1995; Mock and Parker 1997). We calculated the number of core and the number of marginal offspring for each species using information about average clutch size, hatching spread, and laying interval between subsequent eggs within a clutch. First, we calculated the average number of days that mothers require to lay the entire clutch (laying days). Hatching asynchrony occurs when mothers initiate incubation before completing the clutch; therefore, hatching spread reflects the number of laying days during which mothers have already started incubating. The number of asynchronously hatching, or marginal, eggs can therefore be identified by dividing the hatching spread by the laying interval between subsequent eggs. If these calculations produced nonintegers, we rounded the values up. The number of core eggs was calculated by subtracting the number of marginal eggs from the average clutch size.

We collected growth curves from the primary literature and estimated the mass of the core nestlings at the time that the last marginal chick hatched on the basis of the average hatching spread for that species. We divided this value by the estimated average mass of a newly hatched chick. This represents the initial proportional mass advantage of core chicks over marginal chicks and is later referred to as percentage initial size difference between core and marginal chicks. Growth curves of core offspring are likely to be slightly higher than the mean in species with large asymmetries, which would make our estimation of initial size hierarchies via the mean growth curve conservative.

Within-Clutch Variation in Yolk Testosterone and Egg Size

Information about patterns of yolk testosterone concentrations and changes in egg size over the laying sequence were collected from several sources. We searched the ISI Web of Science database and collected what was available in the primary literature for species with clutches containing more than one egg until August 2011. We drew from our own unpublished laboratory data and contacted colleagues for additional unpublished data.

From these different sources, we extracted mean T concentration values for each egg in the laying sequence, excluding sources that reported only combined measures of several androgens. The most straightforward approach to capture the relevant variation in yolk T was to calculate the difference in average yolk T concentrations between core and marginal eggs (mean marginal T minus mean core T). Yolk T concentrations often do not change over the laying sequence in a linear way, so a comparison of linear slopes of yolk T over the laying sequence, for example, would have introduced substantial noise into the correlation. In our calculations, we included only T concentrations from eggs within the confines of average clutch size and excluded data from additional eggs from clutches of larger size because those values usually came from very small sample sizes and easily introduce biases. If yolk T data came from experimental studies, we used only values from the control group. If studies reported yolk T patterns for both initial clutches and replacement clutches, we included only yolk T from initial clutches in our analyses. For species in which we were able to calculate mean yolk T compensation in marginal eggs using data from several studies, we averaged the means from all studies into a single species value. We also compiled mean egg size for consecutive positions in the laying sequence and calculated the proportional mean difference in size between core and marginal eggs by subtracting mean core egg size from mean marginal egg size and dividing it by mean core egg size.

Our literature search yielded calculations of yolk T compensation for 29 species, including the following representation of avian orders: one Anseriformes, five Charadriiformes, one Ciconiformes, two Columbiformes, two Falconiformes, one Gruiformes, 13 Passeriformes, three Pelecaniformes, and one Psittaciformes. In our statistical analyses, sample sizes differed between models, because life-history data were not always available for all parameters for all species. Analyses that included hatching spread and growth rate as predictors were performed using a data set that contained 25 species. Analyses that contained initial within-brood size hierarchy as a predictor were performed using a data set containing 28 species.

Phylogenetic Analyses

We performed all of our ordinary least squares regressions (OLS) and phylogenetic least squares regressions (PGLS) in the R statistical computing environment (R Development Core Team 2012) using the Ape package (Paradis et al. 2004). The PGLS approach corrects for phylogenetic nonindependence of data coming from different species by incorporating a covariance matrix that reflects the degree of their evolutionary relatedness. For this purpose, we constructed a composite phylogeny (fig. A1) from several recent molecular bird phylogenies (Crochet et al. 2000; Ericson et al. 2006; Johnsson and Fjeldsa 2006; Hackett et al. 2008). Because we composed a phylogeny from several different published trees, we set all distances between nodes to equal length, which has been demonstrated to be the most conservative approach (Garland et al. 1993; Pagel 1994; Purvis et al. 1994), and branch lengths have been shown not to affect results qualitatively (Poiani and Pagel 1997; Møller et al. 1998; Nunn 1999; Poulin 1999).

We used a PGLS regression that accommodates the degree to which trait evolution depends on phylogeny by estimating the measure of phylogenetic correlation (λ) via likelihood ratio statistics (Freckleton et al. 2002; Hansen and Orzack 2005). A λ of 0 indicates complete phylogenetic independence and produces the same results as a correlation that does not correct for phylogenetic relatedness. A λ of 1 indicates evolution of traits according to the assumption of Brownian motion, which expects consistent phenotypic divergence over time because of small random changes that accumulate at a constant rate, which renders more closely related species more similar. A λ value between 0 and 1 suggests smaller similarity because of common descent than what would be expected under the Brownian model of evolution. In addition to our phylogentically controlled models, in which the PGLS regression estimated λ, we present results from analyses with raw data, as suggested by Freckleton et al. (2002), using OLS regressions (in which $\lambda = 0$). We checked each model for nonnormality of errors, heteroscedasticity, and outliers.

Statistical Models

We quantified yolk T compensation as mean T concentrations in core eggs subtracted from mean T concentrations in marginal eggs. Because clutch averages in yolk T concentrations varied widely between species, and because the biological importance of a certain difference in yolk T compensation between core and marginal eggs is likely to depend on the average absolute T levels within clutches, all models included average core T concentrations as a covariate to scale the level of T compensation to the species average. Including average core T concentrations in our models also corrects for assay differences between studies.

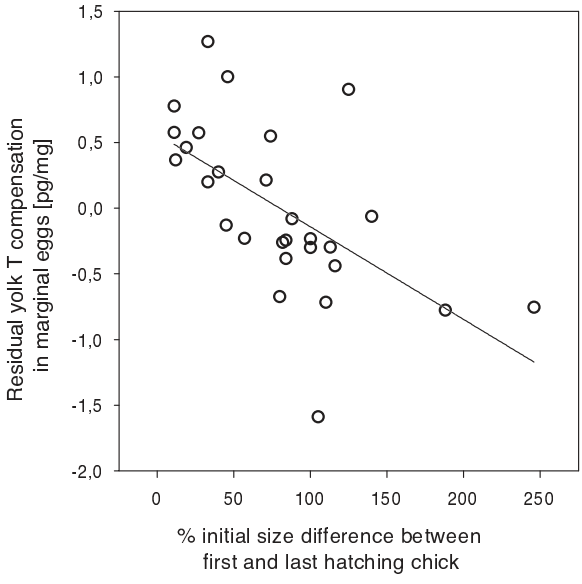

Figure 1: Maternal compensatory yolk testosterone (T) in marginal eggs, controlled for mean core yolk T concentrations and proportional size difference among core and marginal eggs, in relation to percentage initial size advantage of core chick over marginal chick ($n = 28$). Yolk T compensation calculated as mean T concentrations in core eggs subtracted from mean T concentrations in marginal eggs. Percentage initial size difference between first and last hatching chick calculated from species growth curves as mass of the core nestlings by the time the last marginal chick hatched (timing estimated from hatching spread), divided by the mass of the marginal chick.

First, we performed an OLS regression with yolk T compensation as a response variable that contained the following predictors: (1) the percentage initial size advantage of core chicks over marginal chicks, (2) the proportional difference in size between core and marginal eggs (calculated by dividing the mean difference in size between core and marginal eggs by mean core egg size), and (3) average core T concentrations. The first predictor combined the contributions of hatching spread and growth rates to initial size asymmetries. The second predictor adds the contribution of within-clutch egg size variation to initial size asymmetries. We included the third predictor to correct for the species' average clutch T concentrations. We repeated this model using a PGLS regression that corrected for phylogenetic relatedness.

Second, we performed an OLS regression with yolk T compensation as a response variable with predictors that represent the three main contributing factors to initial size

asymmetries: hatching asynchrony (predictor 1), growth rate (predictor 3), and the differences in size between core and marginal eggs (predictor 5), plus several additional covariates (predictors 2, 4, and 6) that we included to correct predictors 1, 3, and 5. In these models, the predictors therefore included (1) average hatching spread, (2) the number of marginal eggs (to correct hatching spread for the number of marginal eggs hatching during the time interval), (3) logistic growth rate constant, (4) mean core egg size (to correct for the fact that growth rate depends to some extent on body size), (5) difference in size between core and marginal eggs (marginal egg size minus core egg size), and (6) mean core T concentrations. We repeated this model in a PGLS regression that corrected for phylogenetic relatedness.

Each model represented a specific hypothesis about the contributions of groups of factors to initial size asymmetries that might have driven the evolution of different patterns of yolk testosterone over the laying sequence. Some predictors were included as covariates to correct other data (e.g., in the second model, we included the number of marginal eggs, mean core egg size, and mean core T concentrations). Because all predictors were important for properly testing our hypotheses, we perform no model simplification. The PGLS package we used in R did not produce residuals corrected for phylogeny, so we present phylogentically uncorrected residuals in figures. All of our analyses were two-tailed with a 95% confidence level.

Results

Initial Size Asymmetries within Broods

As predicted, in our raw species data analysis using an OLS regression ($n = 28$), in species in which core young have a relatively small initial size advantage over their marginal siblings, mothers provide relatively more compensatory yolk T to marginal eggs compared with species in which core young have a relatively large size advantage over their marginal siblings ($b = -0.76$, SE = 0.19, $P = .001$; fig. 1). Maternal yolk T compensation did not vary with the degree to which egg size increased or decreased over the laying sequence ($b = 1.20$, SE = 2.73, $P = .665$) or with average yolk T concentrations in core eggs ($b = 0.16$, SE = 0.09, $P = .101$).

We then performed the same regression on the same species but controlled for phylogenetic relatedness (PGLS regression), and we found qualitatively the same results: in species in which core young have a relatively small initial size advantage over their marginal siblings, mothers provide relatively more compensatory yolk T to marginal eggs compared with species in which core young have a relatively large size advantage over their marginal siblings ($b = -0.70$, SE = 0.20, $P = .002$). Again, maternal yolk T compensation did not vary with the degree to which egg size increased or decreased over the laying sequence ($b = -0.93$, SE = 2.93, $P = .754$) or with average yolk T concentrations in core eggs ($b = 0.13$, SE = 0.09, $P = .168$). This model estimated λ at 0.31, indicating some dependence on phylogeny.

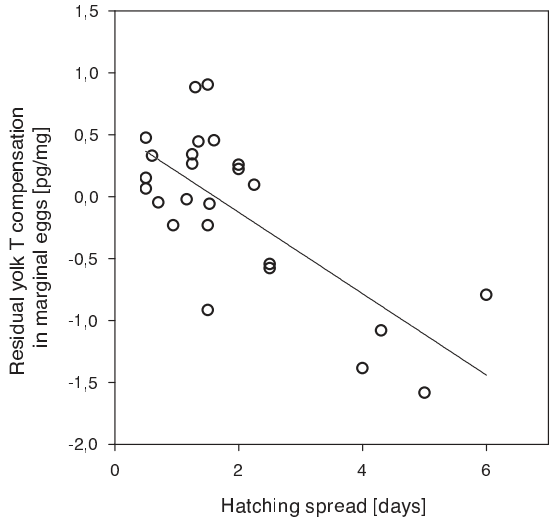

Figure 2: Maternal compensatory yolk testosterone (T) in marginal eggs, controlled for number of marginal eggs, growth rate, mean core T concentrations, mean egg size, and difference in size between core and marginal eggs, in relation to hatching spread ($n = 25$). Yolk T compensation calculated as mean T concentrations in core eggs subtracted from mean T concentrations in marginal eggs.

Hatching Spread, Growth Rate, and Change in Egg Size

As predicted, when using raw species data in an OLS regression ($n = 25$), in species with small hatching asynchrony, mothers provide relatively more compensatory yolk T to marginal eggs compared with species with large hatching asynchrony ($b = -0.43$, SE = 0.09, $P < .001$; fig. 2). In the same model, we found that, in species with slow growth rates, mothers also provide relatively more compensatory yolk T to marginal eggs compared to species with fast growth rates ($b = -3.57$, SE = 0.99, $P = .002$; fig. 3). In the same model, maternal yolk T compensation did not vary with the degree to which egg size increased or decreased over the laying sequence ($b = 0.09$, SE = 0.12, $P = .476$), average core egg size ($b = -0.01$, SE = 0.005, $P = .076$), or with the number of marginal eggs ($b = 0.11$, SE = 0.10, $P = .249$). In the same model, we found that species that produced eggs

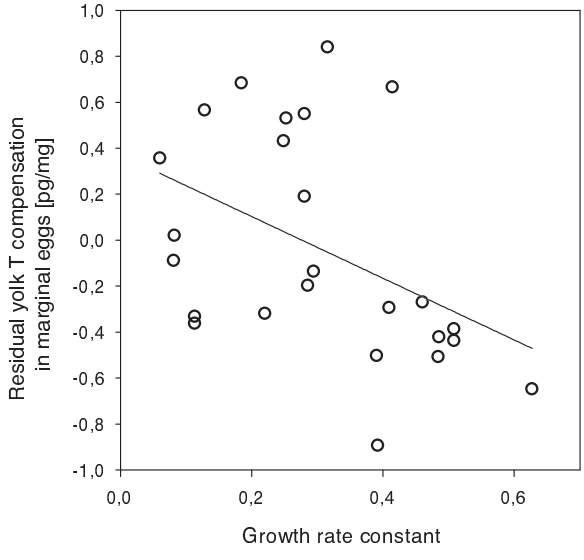

Figure 3: Maternal compensatory yolk testosterone (T) in marginal eggs, controlled for number of marginal eggs, hatching spread, mean core T concentrations, mean egg size, and difference in size between core and marginal eggs, in relation to logistic growth rate constant ($n = 25$). Yolk T compensation calculated as mean T concentrations in core eggs subtracted from mean T concentrations in marginal eggs.

with higher average core yolk T concentrations also provided marginal eggs with relatively more compensatory yolk T ($b = 0.17$, SE $= 0.08$, $P = .046$) compared with species with lower average core yolk T concentrations.

When we performed the same regression on the same species but controlled for phylogenetic relatedness by allowing the regression to estimate λ, we again found that mothers provide relatively more compensatory yolk T to marginal eggs compared with species with large hatching asynchrony ($b = -0.35$, SE $= 0.10$, $P = .002$). We also found that, in species with slow growth rates, mothers provide relatively more compensatory yolk T to marginal eggs compared with species with fast growth rates ($b = -3.82$, SE $= 1.13$, $P = .003$). In this model, mean core egg size ($b = -0.01$, SE $= 0.005$, $P = .087$), the degree to which egg size increased or decreased over the laying sequence ($b = 0.11$, SE $= 0.10$, $P = .291$), the number of marginal eggs ($b = 0.06$, SE $= 0.10$, $P = .548$), and average core yolk T concentrations ($b = 0.10$, SE $= 0.076$, $P = .196$) did not vary significantly with the degree to which mothers provided compensatory yolk T to marginal eggs. In this latter model, $\lambda = 0.69$, indicating moderate phylogenetic dependence in the correlations.

Discussion

In a wide variety of plant and animal species, maternal effects can have important consequences for individual offspring, but their combined effect on a set of propagules that comprise the same reproductive attempt has been little studied (see "Introduction"). In addition, maternal effects encompass a wide array of pathways, potentially providing the mother with a diverse tool kit for optimizing her reproductive performance in different environmental conditions, and are expected to have evolved to work in concert. Understanding their interplay might therefore be crucial for unlocking the importance and evolution of maternal effects. We performed, to our knowledge, the first phylogenetic comparative study to explore correlations among several maternal effects across a broad array of avian species that encompass long periods of evolutionary change in diverse environments. As in many other taxa, avian mothers expose their embryos to substantial amounts of maternal androgens by depositing testosterone in their eggs, affecting behavior and physiology of the chicks (von Engelhardt and Groothuis 2011). Because avian mothers differentially deposit testosterone according to the position of the egg in the laying order of the clutch, these maternal hormones affect the chicks' competitive positions in the sibling hierarchy, potentially affecting the reproductive value of the brood as a whole (Schwabl 1993; Groothuis et al. 2005*b*). Our aim was to test the prediction that within-clutch patterns of maternal androgens evolved together with maternal effects that cause size asymmetries within broods, (egg size, hatching asynchrony, and growth rate) to support maternal reproductive strategies of brood survival or brood reduction. Our study focused on the parental favoritism hypothesis, which postulates that mothers confer competitive advantages and handicaps on different individuals of their broods to create competitive hierarchies. These hierarchies facilitate a secondary adjustment of brood size if the number of young exceeds what parents can afford to rear (Mock and Parker 1997).

We found strong evidence that last-hatching eggs (marginal eggs) from species in which siblings are relatively similar in age and size (brood survival species) receive more maternal T relative to first-hatching eggs (core eggs) compared with species in which age and size differ substantially between siblings (brood reducing species; fig. 1). This suggests that mothers in species that produce marginal young that are primarily intended to provide a reproductive bonus in good years, compared with species that produce marginal young mostly for replacement value, compensate the disadvantage of marginal young with respect to hatching order and sibling rivalry by providing relatively more yolk T. In the latter case, marginal

chicks should only survive in cases in which the core chicks did not hatch or died early in life.

To further explore how maternal androgens relate to the specific processes that give rise to size asymmetries among siblings, we tested the correlation of yolk T compensation with three contributing factors to within-brood variation in age and size: hatching spread, growth rate, and difference in size between core and marginal eggs. We found no relationship between yolk T compensation and the egg size differences in any of our models. The lack of a correlation between these factors probably comes from the fact that variation in egg size contributes little to within-brood size asymmetries compared with the combined contributions of hatching spread and growth rate. In our data set, the maximum difference in size between core and marginal eggs was only 7.5% (*Sula leucogaster*; Ceyca and Mellink 2009), whereas in 10 of the 28 species that we analyzed, the first chick grew to be at least 100% larger than the last chick by the time all chicks had hatched. Clearly, most of the variation in age and size among brood mates arises as a result of hatching asynchrony (Parker 1982) and because of the growth of the early hatching core part of the brood during the time lag before the marginal young hatch. In line with our predictions, we found that species with high compensatory maternal T in marginal eggs had a relatively small degree of hatching asynchrony (fig. 2) and slow growth rates (fig. 3) compared with species with low compensatory maternal T in marginal eggs. Species with large hatching spreads are known to employ an insurance strategy (Forbes and Mock 2000). Species with fast growth rates are also expected to employ an insurance strategy because of their tendency to produce large clutches (Ricklefs 1968), which is a trait that favors the production of insurance offspring (Forbes 1990). We show that both of these traits are associated with very low maternal T compensation.

David Lack's (1947, 1954, 1968) resource-tracking hypothesis proposes that many avian species produce optimistic clutch sizes so that they can capitalize in years when food is abundant but use hatching asynchrony to designate marginal chicks as expendable for when food becomes too scarce for the parents to rear the whole brood. Marginal chicks therefore contribute "extra" reproductive value to the brood by surviving alongside the core under favorable conditions (increasing the quantity of young). However, they don't compromise the value of the core under poor conditions (Forbes et al. 2002), because they compete ineffectively when food is very limited and die of starvation (preserving quality of young). We suggest that these species should produce broods with narrower size hierarchies and more yolk T compensation in marginal eggs. This combination of traits would enhance the survival of marginal young in years when resources are plentiful because food returns would more than compensate for the extra begging effort and growth (reviewed in von Engelhardt and Groothuis 2011) and allow them to overcome their initial small size disadvantage.

In poor years, however, yolk T might actually accelerate mortality in marginal offspring, because such offspring receive insufficient food returns to compensate for their higher energy requirements (as suggested in Tobler et al. 2007). Considering that the behavioral and physiological downstream effects of yolk T exposure place a substantial burden on a chick's energy budget (Tobler et al. 2007), and perhaps therefore also on its investment in immunity (Groothuis et al. 2005*a*), such context-dependent effects like in good versus poor food conditions are very likely to occur, although they have not yet been directly tested. The fact that yolk T compensation might benefit marginal young in good conditions and harm them in poor conditions has already been suggested by Royle et al. (2001) and Groothuis et al. (2005*a*). If this is the case, then, as we showed in our correlations, the insurance species, which have large sibling size hierarchies, should not compensate marginal eggs with yolk T. In these species, redundant marginal young inevitably experience "poor conditions," and yolk T compensation would risk accelerating brood reduction when ineffective high begging effort causes them to perish prematurely before the core is out of the high-risk period of failing.

Here, we have suggested that the patterns of yolk T compensation, as correlated with within-brood size asymmetries, support contrasting reproductive strategies in which mothers produce marginal chicks for "extra" reproductive value versus "insurance" value. For the former strategy, mothers produce broods with narrow hatching spreads and substantial yolk T compensation in marginal eggs so that marginal young have a chance to survive in years with abundant food. For the latter strategy, mothers produce broods with large hatching spreads and little yolk T compensation to facilitate the elimination of redundant marginal young when they are not needed to replace failed core offspring. This model rests on the assumption that elevated yolk T exposure benefits marginal young in good breeding conditions but harms them in poor breeding conditions. Inconsistencies in the outcomes of experimental studies that manipulated yolk T suggest that this is likely to be the case, but it needs to be tested systematically within one experiment. However, context dependency of maternal effects has already been demonstrated convincingly as a broad phenomenon that occurs in diverse taxa (e.g., frogs [Kaplan 1992], trout [Einum and Flemming 1999], beetles [Fox 2000], and soil mites [Plaistow et al. 2006]).

In our analyses, we averaged the patterns of yolk T over the laying sequence for each species and assume it to be

the product of evolutionary pressures related to competitive hierarchies among siblings. We find the strong relationships that we expected, which supports this assumption, but variation among species averages may not be the whole story. Substantial variation in yolk T patterns over the laying sequence also exists within species, and several studies have shown that this variation correlates with characteristics of the mother or to aspects of her environment, such as food abundance (Rutstein et al. 2004; Sandell et al. 2007, reviewed in von Engelhardt and Groothuis 2011). If species are flexible to adjust patterns of yolk T over the laying sequence according to breeding conditions, then mothers could provide marginal young with more compensatory yolk T to promote their survival in good breeding conditions (e.g., Vergauwen et al. 2012). With such flexibility, mothers could also provide marginal young with less compensatory yolk T in anticipation of poor conditions and a high likelihood of brood reduction (the hatching asynchrony adjustment hypothesis; Groothuis et al. 2005*b*). It is also known that the hatching spreads can vary substantially within species, and mothers have been shown to produce more asynchronous broods in anticipation of difficult rearing conditions (e.g., Wiebe and Bortolotti 1994). If mothers are able to adjust hatching spreads and yolk T compensation in concert and in response to environmental conditions, as indicated for the black-headed gull (Muller et al. 2004), then we might expect to find the negative correlation between those two factors that we observe in a between-species comparison in a within-species comparison as well.

In this study, we have investigated the correlated evolution of a suite of maternal effects that appear to function together to allow mothers to optimize the number of progeny that they rear in a given breeding attempt. We focused on variation in maternal androgen exposure among siblings, variation in propagule size among siblings, and variation in relative timing of emergence from the egg. Other studies suggest that similar processes may have evolved in other taxa as well. For example, studies on montane lizards show complex interactions between maternal hormones, egg size, and sex on prenatal development rates that cause variation in hatching times and thus confer developmental advantages on select offspring (Radder and Shine 2007). Subsocial bugs covary egg size and spatial position within the clutch so that peripheral (insurance) eggs that are exposed to higher predation risk are smaller (Kudo 2006). In plants, the potential for maternal effects in enhancing growth rates, accelerating emergence, or increasing seed weight as an avenue toward conferring an early size advantage to seedlings has been implied (e.g., Stanton 1984*a*) but not directly studied in the context of sibling rivalry. We suggest that the hypothesis that we tested in this study extends to all taxa with sibling rivalry: mothers that anticipate a high likelihood of brood reduction should create competitive asymmetries among young that support the best-quality offspring. However, mothers that aim for survival of all offspring should try to equalize the competitive abilities of siblings so that no offspring are outcompeted.

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APPENDIX

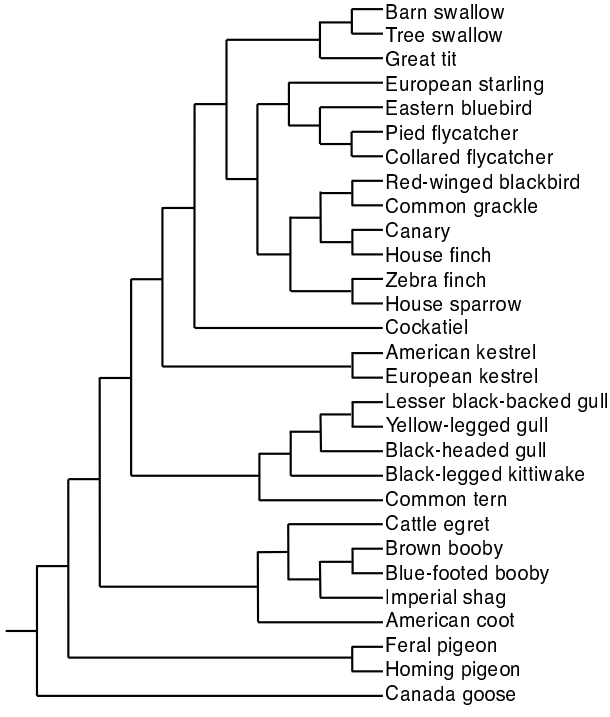

Figure A1: Topology of the composite phylogeny used in the study.

Literature Cited

Amundsen, T., and T. Slagsvold. 1991. Hatching asynchrony: facilitating adaptive or maladaptive brood reduction. Acta XX Congressus Internationalis Ornithologici 1990:1707–1719.

Anderson, D. J. 1990. Evolution of obligate siblicide in boobies. 1. A test of the insurance-egg hypothesis. American Naturalist 135: 334–350.

Anholt, B. R. 1994. Cannibalism and early instar survival in a larval damselfly. Oecologia (Berlin) 99:60–65.

Bertin, A., M. A. Richard-Yris, C. Houdelier, S. Lumineau, E. Mostl, A. Kuchar, K. Hirschenhauser, and K. Kotrschal. 2008. Habituation to humans affects yolk steroid levels and offspring phenotype in quail. Hormones and Behavior 54:396–402.

Black, J. N. 1958. Competition between plants of different initial seed sizes in swards of subterranean clover (*Trifolium subterraneum*) with particular reference to leaf area and the light microclimate. Australian Journal of Agricultural Research 9:299–318.

Bowden, R. M., M. A. Ewert, and C. E. Nelson. 2000. Environmental sex determination in a reptile varies seasonally and with yolk hormones. Proceedings of the Royal Society B: Biological Sciences 267:1745–1749.

Castro-Franco, R., M. G. Bustos-Zagal, and F. R. Mendez-De la Cruz. 2011. Variation in parental investment and relative clutch mass of the spiny-tail iguana, *Ctenosaura pectinata* (Squamata: Iguanidae) in central Mexico. Revista Mexicana de Biodiversidad 82:199–204.

Ceyca, J. P., and E. Mellink. 2009. Reproductive ecology of brown boobies (*Sula leucogaster*) at Morros El Potosí, Guerrero, México. Revista Mexicana de Biodiversidad 80:793–800.

Cheplick, G. P. 1992. Sibling competition in plants. Journal of Ecology 80:567–575.

Cottrell, E. C., and J. R. Seckl. 2009. Prenatal stress, glucocorticoids and the programming of adult disease. Frontiers in Behavioural Neuroscience 3:19.

Cramp, S. 1998. The complete birds of the western Palearctic. CD-ROM. Oxford University Press, Oxford.

Crochet, P. A., F. Bonhomme, and J. D. Lebreton. 2000. Molecular phylogeny and plumage evolution in gulls (*Larini*). Journal of Evolutionary Biology 13:47–57.

Cucco, M., B. Guasco, G. Malacarne, R. Ottonelli, and A. Tanvez. 2008. Yolk testosterone levels and dietary carotenoids influence growth and immunity of grey partridge chicks. General and Comparative Endocrinology 156:418–425.

de Magalhaes, J. P., and J. Costa. 2009. A database of vertebrate longevity records and their relation to other life-history traits. Journal of Evolutionary Biology 22:1770–1774.

Donohue, K. 2009. Completing the cycle: maternal effects as the missing link in plant life histories. Philosophical Transactions of the Royal Society B: Biological Sciences 364:1059–1074.

Dorward, E. F. 1962. Comparative biology of the white booby and brown booby *Sula* spp. at Ascension. Ibis 103:74–220.

Einum, S., and I. A. Fleming. 1999. Maternal effects of egg size in brown trout (*Salmo trutta*): norms of reaction to environmental quality. Proceedings of the Royal Society B: Biological Sciences 266:2095–2100.

———. 2004. Does within-population variation in egg size reduce intraspecific competition in Atlantic salmon, *Salmo salar*? Functional Ecology 18:110–115.

Eising, C. M., C. Eikenaar, H. Schwabl, and T. G. G. Groothuis. 2001. Maternal androgens in black-headed gull (*Larus ridibundus*) eggs: consequences for chick development. Proceedings of the Royal Society B: Biological Sciences 268:839–846.

Ento, K., K. Araya, and S. Kudo. 2010. Laboratory observation of siblicide with hatching asynchrony in an insect with parental provisioning. Journal of Ethology 28:405–407.

Ericson, P. G. P., C. L. Anderson, T. Britton, A. Elzanowki, U. S. Johansson, M. Kallersjo, J. I. Ohlson, T. J. Parsons, D. Zuccon, and G. Mayr. 2006. Diversification of Neoaves: integration of molecular sequence data and fossils. Biology Letters 2:543–547.

Forbes, L. S. 1990. Insurance offspring and the evolution of avian clutch size. Journal of Theoretical Biology 147:345–359.

Forbes, L. S., and T. C. Lamey. 1996. Insurance, developmental accidents, and the risks of putting all your eggs in one basket. Journal of Theoretical Biology 180:247–256.

Forbes, S., Grosshans, R., and B. Glassey. 2002. Multiple incentives for parental optimism and brood reduction in blackbirds. Ecology 83:2529–2541.

Forbes, S., and D. W. Mock. 2000. A tale of two strategies: life-history aspects of family strife. Condor 102:23–34.

Forbes, S., S. Thornton, B. Glassey, M. Forbes, and N. Buckley. 1997. Why parent birds play favourites. Nature 390:351–352.

Fox, C. W. 2000. Natural selection on seed-beetle egg size in nature and the laboratory: variation among environments. Ecology 81: 3029–3035.

Fox, C. W., and M. E. Czesak. 2000. Evolutionary ecology of progeny size in arthropods. Annual Review of Entomology 45:341–369.

Fraser, D., and B. K. Thompson. 1991. Armed sibling rivalry among suckling piglets. Behavioral Ecology and Sociobiology 29:9–15.

Freckleton, R. P., P. H. Harvey, and M. Pagel. 2002. Phylogenetic analysis and comparative data: a test and review of the evidence. American Naturalist 160:712–726

Ganeshaiah, K. N., and R. Uma Shaanker. 1988. Seed abortion in wind-dispersed pods of *Dalbergia sissoo*, maternal regulation or sibling rivalry? Oecologia (Berlin) 75:135–139.

Garland, T., Jr., A. W. Dickerman Jr., C. M. Janis, and J. A. Jones. 1993. Phylogenetic analysis of covariance by computer simulation. Systematic Biology 42:265–292.

Gil, D., C. Biard, A. Lacroix, C. N. Spottiswoode, N. Saino, M. Puerta, and A. P. Møller. 2007. Evolution of yolk androgens in birds: development, coloniality, and sexual dichromatism. American Naturalist 169:802–819.

Gil, D., and J. M. Faure. 2007. Correlated response in yolk testosterone levels following divergent genetic selection for social behaviour in Japanese quail. Journal of Experimental Zoology A 307: 91–94.

Gorman, K. B., and T. D. Williams. 2005. Correlated evolution of maternally derived yolk testosterone and early developmental traits in passerine birds. Biology Letters 1:461–464.

Groothuis, T. G. G., C. Carere, J. Lipar, P. J. Drent, and H. Schwabl. 2008. Selection on personality in a songbird affects maternal hormone levels tuned to its effect on timing of reproduction. Biology Letters 4:465–467.

Groothuis, T. G. G., C. M. Eising, C. Dijkstra, and W. Muller. 2005*a*. Balancing between costs and benefits of maternal hormone deposition in avian eggs. Biology Letters 1:78–81.

Groothuis, T. G. G., W. Muller, N. von Engelhardt, C. Carere, and C. M. Eising. 2005*b*. Maternal hormones as a tool to adjust offspring phenotype in avian species. Neuroscience and Biobehavioral Reviews 29:329–352.

Hackett, S. J., R. T. Kimball, S. Reddy, R. C. K. Bowie, E. L. Braun,

M. J. Braun, T. Yuri, et al. 2008. A phylogenomic study of birds reveals their evolutionary history. Science 320:1763–1768.
Hansen, T. F., and S. H. Orzack. 2005. Assessing current adaptation and phylogenetic inertia as explanations of trait evolution: the need for controlled comparisons. Evolution 59:2063–2072.
Jonsson, K. A., and J. Fjeldsa. 2006. A phylogenetic supertree of oscine passerine birds (Aves: Passeri). Zoologica Scripta 35:149–186.
Kaplan, R. H. 1992. Greater maternal investment can decrease offspring survival in the frog *Bombina orientalis*. Ecology 73:280–288.
Krist, M. 2011. Egg size and offspring quality: a meta-analysis in birds. Biological Reviews 86:692–716.
Kudo, S. 2006. Within-clutch egg-size variation in a subsocial bug: the positional effect hypothesis. Canadian Journal of Zoology 84: 1540–1544.
Lack, D. 1947. The significance of clutch size. Ibis 89:302–352.
———. 1954. The natural regulation of animal numbers. Clarendon, Oxford.
———. 1968. Ecological adaptations for breeding in birds. Methuen, London.
Lipar, J. L., E. D. Ketterson, and V. Nolan Jr. 1999. Intraclutch variation in testosterone content of red-winged blackbird eggs. Auk 116:231–235.
Magrath, R. D. 1990. Hatching asynchrony in altricial birds. Biological Reviews 65:587–622.
Marshall, D. J., T. F. Bolton, and M. J. Keough. 2003. Offspring size affects the post-metamorphic performance of a colonial marine invertebrate. Ecology 84:3131–3137.
Marshall, D. J., and T. Uller. 2007. When is a maternal effect adaptive? Oikos 116:1957–1963.
Martin, T. E., and H. Schwabl. 2008. Variation in maternal effects and embryonic development rates among passerine species. Philosophical Transactions of the Royal Society B: Biological Sciences 363:1663–1674.
Mock, D. W. 1984. Infanticide, siblicide, and avian nestling mortality. Pages 3–30 *in* G. Hausfater and S. B. Hrdy, eds. Infanticide: comparative and evolutionary perspectives. Aldine, New York.
Mock, D. W., H. Drummond, and C. H. Stinson. 1990. Avian siblicide. American Scientist 78:438–449.
Mock, D. W., and L. S. Forbes. 1995. The evolution of parental optimism. Trends in Ecology & Evolution 10:130–134.
Mock, D. W., and G. A. Parker. 1997. The evolution of sibling rivalry. Oxford University Press, Oxford.
Mousseau, T. A., and H. Dingle. 1991. Maternal effects in insect life histories. Annual Review of Entomology 36:511–534.
Mousseau, T. A. and C. W. Fox. 1998. The adaptive significance of maternal effects. Trends in Ecology & Evolution 13:403–407.
Müller, W., C. M. Eising, C. Dijkstra, and T. G. G. Groothuis. 2004. Within-clutch patterns of yolk testosterone vary with the onset of incubation in black-headed gulls. Behavioral Ecology 15:893–897.
Nunn, C. L. 1999. The number of males in primate social groups: a comparative test of the socioecological model. Behavioral Ecology and Sociobiology 46:1–13.
Møller, A. P., G. Sorci, and J. Erritze. 1998. Sexual dimorphism in immune defense. American Naturalist 152:605–619.
O'Gara, B. 1969. Unique aspects of reproduction in the female pronghorn (*Antilocapra americana*). American Journal of Anatomy 125: 217–232.
Okuliarova, M., T. G. G. Groothuis, P. Skrobanek, and M. Zeman. 2011. Experimental evidence for genetic heritability of maternal hormone transfer to offspring. American Naturalist 177:824–834.
Pagel, M. D. 1994. Detecting correlated evolution on phylogeneties: a general method for the comparative analysis of discrete characters. Proceedings of the Royal Society B: Biological Sciences 255: 37–45.
Paradis E., J. Claude, and K. Strimme. 2004. APE: analyses of phylogenetics and evolution in R language. Bioinformatics 20:289–290.
Parker, G. A. 1982. Phenotype-limited evolutionary stable strategies. Pages 173–201 *in* B. R. Bertram, T. H. Clutton-Brock, R. I. M. Dunbar, D. I. Rubenstein, and R. Wrangham, eds. Current problems in sociobiology. Cambridge University Press, Cambridge.
Pexton, J. J., and P. J. Mayhew. 2002. Siblicide and life-history evolution in parasitoids. Behavioral Ecology 13:690–695.
Plaistow, S. J., C. T. Lapsley, and T. G. Benton. 2006. Context-dependent intergenerational effects: the interaction between past and present environments and its effect on population dynamics. American Naturalist 167:206–215.
Poiani, A., and M. Pagel. 1997. Evolution of avian cooperative breeding: comparative tests of the nest predation hypothesis. Evolution 51:226–240.
Poole, A. 2005. The birds of North America online. http://bna.birds.cornell.edu/BNA/. Cornell Laboratory of Ornithology, Ithaca, NY.
Poulin, R. 1999. The intra- and interspecific relationships between abundance and distribution in helminth parasites of birds. Journal of Animal Ecology 68:719–725.
Purvis, A., J. L. Gittleman, and H. K. Luh. 1994. Truth or consequences: effects of phylogenetic accuracy on two comparative methods. Journal of Theoretical Biology 167:293–300.
R Development Core Team. 2012. R: a language and environment for statistical computing. http://www.R-project.org. R Foundation for Statistical Computing, Vienna.
Radder, R. S., and R. Shine. 2007. Sex-based hatching asynchrony in an oviparous lizard. Austral Ecology 32:502–508.
Ricklefs, R. E. 1968. Patterns of growth in birds. Ibis 110:419–451.
Royle, N. J., P. F. Surai, and I. R. Hartley. 2001. Maternally derived androgens and antioxidants in bird eggs: complementary but opposing effects? Behavioral Ecology 12:381–385.
Rutstein, A. N., L. Gilbert, P. J. B. Slater, and J. A. Graves. 2004. Sex-specific patterns of yolk androgen allocation depend on maternal diet in the zebra finch. Behavioral Ecology 16:62–69.
Sandell, M. I., E. Adkins-Regan, and E. D. Ketterson. 2007. Prebreeding diet affects the allocation of yolk hormones in zebra finches *Taeniopygia guttata*. Journal of Avian Biology 38:284–290.
Schaafsma, S. M., and T. G. G. Groothuis. 2012. Sex-specific effects of maternal testosterone on lateralization in a cichlid fish. Animal Behaviour 83:437–443.
Schwabl, H. 1993. Yolk is a source of maternal testosterone for developing birds. Proceedings of the National Academy of Sciences of the USA 90:11446–11450.
———. 1996. Maternal testosterone in the avian egg enhances postnatal growth. Comparative Biochemistry and Physiology A 114: 271–276.
Schwabl, H., D. W. Mock, and J. A. Gieg. 1997. A hormonal mechanism for parental favourtism. Nature 386:231–231.
Schwabl, H., M. G. Palacios, and T. E. Martin. 2007. Selection for rapid embryo development correlates with embryo exposure to maternal androgens among passerine birds. American Naturalist 170:196–206.
Slagsvold, T., J. Sandvik, G. Rofstad, O. Lorentsen, and M. Husby.

1984. On the adaptive value of intra-clutch egg-size variation in birds. Auk 101:685–697.

Stanton, M. L. 1984*a*. Developmental and genetic sources of seed weight variation in *Raphanus raphnistrum L.* (Brassicaceae). American Journal of Botany 71:1090–1098.

Stanton, M. L. 1984*b*. Seed variation in wild radish: effect of seed size on components of seed size on components of seedlings and adult fitness. Ecology 65:1105–1112.

Stoleson, S. H., and S. R. Beissinger. 1995. Hatching asynchrony and the onset of incubation in birds, revisited: when is the critical period? Pages 191–270 *in* D. M. Power, ed. Current ornithology. Vol. 12. Plenum, New York.

Tobler, M., J. A. Nilsson, and J. F. Nilsson. 2007. Costly steroids: egg testosterone modulates nestling metabolic rate in the zebra finch. Biology Letters 3:408–410.

Turner, R., and J. M. Lawrence. 1977. Volume and composition of echinoderm eggs: implications for the use of egg size in life-history models. Pages 25–40 *in* S. E. Stancyk, ed. Reproductive ecology of marine invertebrates. University of South Carolina Press, Columbia.

Vergauwen, J., V. C. Goerlich, T. G. G. Groothuis, M. Eens, and W. Müller. 2012. Food conditions affect yolk testosterone deposition but not incubation attendance. General and Comparative Endocrinology 176:112–119.

von Engelhardt, N., and T. G. G. Groothuis. 2011. Maternal hormones in avian eggs. Pages 91–127 *in* D. O. Norris and K. H. Lopez, eds. Hormones and reproduction of vertebrates. Vol. 4. Birds. Academic Press, New York.

Weinstock, M. 2008. The long-term behavioural consequences of prenatal stress. Neuroscience & Biobehavioral Reviews 32:1073–1086.

Wiebe, K. L. 1996. The insurance-egg hypothesis and extra reproductive value of last-laid eggs in clutches of American kestrels. Auk 113:258–261.

Wiebe, K. L., and G. R. Bortolotti. 1994. Food supply and hatching spans of birds: energy constraints or facultative manipulation? Ecology 75:813–823.

Wolf, J. B., and M. J. Wade. 2009. What are maternal effects (and what are they not)? Philosophical Transactions of the Royal Society B: Biological Sciences 364:1107–1115.

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