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E-Article

The Adaptive Value of Stress-Induced Phenotypes: Effects of Maternally Derived Corticosterone on Sex-Biased Investment, Cost of Reproduction, and Maternal Fitness

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abstract: The question of why maternal stress influences offspring phenotype is of significant interest to evolutionary physiologists. Although embryonic exposure to maternally derived glucocorticoids (i.e., corticosterone) generally reduces offspring quality, effects may adaptively match maternal quality with offspring demand. We present results from an interannual field experiment in European starlings (*Sturnus vulgaris*) designed explicitly to examine the fitness consequences of exposing offspring to maternally derived stress hormones. We combined a manipulation of yolk corticosterone (yolk injections) with a manipulation of maternal chick-rearing ability (feather clipping of mothers) to quantify the adaptive value of corticosterone-induced offspring phenotypes in relation to maternal quality. We then examined how corticosterone-induced "matching" within this current reproductive attempt affected future fecundity and maternal survival. First, our results provide support that low-quality mothers transferring elevated corticosterone to eggs invest in daughters as predicted by sex allocation theory. Second, corticosterone-mediated sex-biased investment resulted in rapid male-biased mortality resulting in brood reduction, which provided a better match between maternal quality and brood demand. Third, corticosterone-mediated matching reduced investment in current reproduction for low-quality mothers, resulting in fitness gains through increased survival and future fecundity. Results indicate that the transfer of stress hormones to eggs by low-quality mothers can be adaptive since corticosterone-mediated sex-biased investment matches the quality of a mother to offspring demand, ultimately increasing maternal fitness. Our results also indicate that the branding of the proximate effects of maternal glucocorticoids on offspring as negative ignores the possibility that short-term phenotypic changes may actually increase maternal fitness.

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Although the proximate effects of maternally derived steroid hormones on offspring phenotype have garnered significant attention from ecological physiologists for the last 15 years (e.g., Schwabl 1993; Groothuis et al. 2005*a*, 2005*b*), virtually nothing is known about how these maternal effects influence fitness (Groothuis et al. 2005*b*; Love et al. 2005, 2008). Moreover, almost all of these studies have focused on the effects of yolk androgens (reviewed in Groothuis et al. 2005*b*), despite there being numerous steroids that are excellent candidates for causing numerous life-history trade-offs (Love et al. 2005). A particularly good example are glucocorticoids, such as corticosterone in birds, reptiles, and amphibians, which mediate adaptive physiological and behavioral responses to "stressful" events (Wingfield et al. 1998; Sapolsky et al. 2000; Wingfield 2005) within the larger context of maintaining daily homeostatic energetic balance (Harvey et al. 1984; Dallman et al. 1993; Remage-Healey and Romero 2001). As such, corticosterone can be linked to an individual's energetic state (Holberton et al. 1996; Love et al. 2005, 2008; Kitaysky et al. 2006; Blas et al. 2007; Love and Williams 2008). Importantly, embryos in a wide range of taxa are sensitive to maternally derived glucocorticoids during development and express many phenotypic adjustments (fish: McCormick 1998, 1999; reptiles: Sinervo and DeNardo 1996; Meylan and Clobert 2005; Lovern and Adams 2008; birds: Hayward and Wingfield 2004; Love et al. 2005; Saino et al. 2005; Hayward et al. 2006; Love and Williams 2008; mammals: Seckl 2001, 2004). Again, however, the result of many of these studies has been the branding of effects as simply positive or negative to the offspring, largely ignoring the possibility that proximate adjustments in offspring may provide fitness costs/benefits to mothers in both the present and the future (Love et al. 2005; Love and Williams 2008). Moreover, while many of the proximate effects of maternally derived glucocorticoids have

eggs; incubation length: 10.3 ± 0.1 days; postnatal period: 21 ± 0.6 days; Love et al. 2008). Nest boxes were checked daily to determine clutch initiation, laying sequence, and clutch completion dates. On the morning of the laying of the first egg for a given female, individuals were randomly (within the colony) assigned to either an oil injection (hereafter referred to as "sham"; $n = 34$ females) or corticosterone injection ($n = 34$ females) treatment group (treatments paired each day to control for laying date) within the synchronous 7–8-day peak of laying of first clutches in our colony. Within 3 h of a new egg being laid, it was removed from the nest, the injection site was cleaned with ethyl alcohol, and the injection was made into the yolk. The hole was sealed using cyanoacrylate glue (Loctite Superglue Control Gel, Henkel), the egg's laying order was marked, and the egg was measured and returned to the

Smits et al. (1999). Repeatability of both initial (r p 0.91, P ! .0001) and final (r p 0.87, P ! .0001) measurements was high, and we used mean values of the three measurements.

Molecular Sexing

The blood sample at hatching was collected on a piece of filter paper and stored frozen at 20°C. On the basis of techniques reported by Love et al. (2005), nestling sex was determined using polymerase chain reaction amplification. DNA was isolated from the blood samples using InstaGene matrix (Bio-Rad Laboratories, cat. no. 732–6030) and from tissue samples collected from unhatched eggs using DNeasy kits (Qiagen) following manufacturer's protocols. Polymerase chain reaction amplification was carried out in a total volume of 10 L and run using the P2 (5′-TCTGCATCGCTAAATCCTTT) and CW (5′-AGAAATC-ATTCCAGAAGTTCA) primers followed by digestion with HaeIII enzyme.

Statistical Analysis

Since the goal of this study was to determine whether exposing offspring to maternally derived corticosterone was adaptive for a low-quality mother, we pay particular attention in the "Results" and "Discussion" to differences between the CORT-clipped and sham-clipped groups. The remaining control groups (CORT-nonclipped and sham-nonclipped) allow us to (1) examine the fitness of a low-quality mother if her quality can increase from laying to chick rearing (CORT-nonclipped) and (2) compare all of the manipulated treatments with an unmanipulated baseline control mother that raises normal young (sham-nonclipped). To examine hatching traits, we statistically analyzed data in relation to the hormone treatment only, since hatchlings had not yet been exposed to the effects of their mother's treatment.

We used ANOVA to analyze potential differences between females assigned to the various treatments (body mass, clutch size) as well as differences in maternal/paternal provisioning rates. We used repeated-measures ANOVA to examine changes in maternal body mass and reproductive parameters between reproductive attempts (repeated statistic is referred to as "time" in "Results"). We used general linear mixed models (GLMMs) to analyze sex-specific effects of the hormonal and overall treatments on nestling traits (body mass/size and CMI) by including nestling sex and the relevant treatment as fixed factors; maternal identity was included as a random factor to control for nonindependence due to the inclusion of siblings in the analysis, and both laying sequence and clutch size were included as covariates since clutch-specific laying sequence patterns of yolk corticosterone have been found (Love et al. 2008). Post hoc comparisons for significant treatment effects were carried out using the sequential Bonferroni post hoc procedure, with the P value corrected for the number of pairwise comparisons made depending on the type of analysis (Rice 1989); for the sex × treatment interaction, post hoc comparisons were made within sexes. Sex ratio of the brood (a measure of sex-specific mortality), as a function of maternal treatment, was analyzed using GLMM with a binomial error structure; maternal identity was included as a random factor, and laying sequence was included as a covariate (Love et al. 2005). Sex-specific mortality as a function of maternal treatment and in relation to laying sequence was analyzed within sexes using a GLMM with laying sequence included as a covariate. For all GLMM models involving sex ratio/mortality, the significance of the explanatory variables was determined by their Wald statistic using the χ^2 distribution with set

Figure 1: Treatment effects on (*A*) maternal and parental provisioning rates in first broods and (*B*) maternal body mass changes during chick rearing in first and second broods of European starling mothers (mean

Table 1: Effects of maternal and hormonal treatments on fledgling phenotypic traits in male and female starling nestlings in first broods in 2005

Traits	S_C	B_C	S_{NC}	B_{NC}
Males:				
Body mass (g)	$76.42 \pm .91^A$	77.53 ± 1.12^A	$74.37 \pm .98^A$	$76.64 \pm .91^A$
Tarsus (mm)	$34.40 \pm .30^A$	$34.48 \pm .37^A$	$35.16 \pm .32^B$	$35.26 \pm .30^B$

a reduction in daughter body mass and structural size at fledging. Exposure to elevated prenatal corticosterone reduced the hatching mass and begging of sons, and CORT-clipped mothers experienced rapid, male-biased brood reduction. We argue that these smaller broods provided feather-clipped mothers with a better "match" between their offspring-rearing capability and their brood's demand. As a result, these mothers produced both high-quality sons and daughters at fledging. This assertion is supported by data indicating that the "mismatched" mothers began and ended second broods with lower body masses than "matched" mothers. As a result, raising corticosterone-exposed offspring in effect "rescued" feather-clipped mothers during the current breeding attempt and resulted in an increase in future fecundity and survival prospects compared with mismatched mothers.

Figure 2: Treatment effects on (*A*) changes in brood size, (*B*) sex ratio (proportion of males), and (*C*) male survival in relation to laying order during postnatal development in first broods of European starling mothers (treatments: sham-clipped [S_C], CORT-clipped [B_C], sham-nonclipped [S_{NC}], and CORT-nonclipped [B_{NC}]; asterisks represent significant differences between B_C and all other treatment groups).

Discussion

Sham-clipped mothers showed reduced maternal provisioning ability and were therefore "mismatched" to the relative demand of their unmanipulated brood, leading to

Table 2: Effects of first brood maternal treatment on fledgling phenotypic traits in male and female starling nestlings in second broods in 2005

Traits	S_C	B_C	S_{NC}	B_{NC}
Males:				
Body mass (g)	66.55 ± 3.23^A	73.53 ± 2.70^B	75.49 ± 1.94^B	73.17 ± 2.38^B
Tarsus (mm)	$34.16 \pm .47^A$	$35.11 \pm .63^B$	$35.16 \pm .28^B$	$35.24 \pm .35^B$
Wing cord (mm)	84.33 ± 2.52^A	88.45 ± 2.28^B	90.10 ± 1.69^B	89.94 ± 2.09^B
CMI (mm × 10)	13.23 ± 2.68^A	36.42 ± 5.08^B	14.62 ± 1.89^A	12.02 ± 1.52^A
Females:				
Body mass (g)	72.09 ± 3.09^A	70.48 ± 2.68^A	67.53 ± 1.79^A	70.39 ± 2.59^A
Tarsus (mm)	$34.58 \pm .45^A$	$33.95 \pm .52^A$	$34.65 \pm .26^A$	$34.16 \pm .38^A$
Wing cord (mm)	88.61 ± 2.50^A	82.09 ± 3.06^B	90.52 ± 1.56^A	90.09 ± 2.26^A
CMI (mm × 10)	14.91 ± 2.61^A	41.29 ± 3.10^B	21.04 ± 1.68^A	17.57 ± 2.28^A

Note: Different letters represent significant differences within sexes across treatments. CMI, cell-mediated immunity. Treatments: sham-clipped (S_C), CORT-clipped (B_C), sham-nonclipped (S_{NC}), and CORT-nonclipped (B_{NC}).

In our previous work (Love et al. 2005), starling mothers implanted with corticosterone before laying deposited the hormone into yolks, resulting in an investment in less expensive daughters, whereby sons showed increased embryonic mortality, reduced hatching masses, and reduced postnatal growth. These results suggested that the deposition of yolk corticosterone would benefit mothers in poor condition by providing an adaptive mechanistic link between maternal quality and sex-specific allocation. Deposition of maternally derived corticosterone to eggs in relation to maternal condition would be a beneficial bet-hedging strategy in stochastic environments where the correlation between environmental cues at laying (and therefore potentially maternal condition) and conditions during chick rearing might be low and unpredictable (Love et al. 2005). The direct yolk corticosterone manipulation used in this study specifically tests whether it is yolk corticosterone per se that is responsible for the documented changes in sex allocation rather than potential behavioral/physiological side effects associated with elevating the hormone in the mother. Results of this study are supported by recent work in captive avian species indicating that males show reduced hatching masses and slower postnatal growth in response to exposure to elevated yolk corticosterone (Hayward et al. 2006; Satterlee et al. 2007). We would predict that if low maternal quality at laying continues into chick rearing, then lighter-hatched sons should experience increased mortality, since low mass at hatching has a significant negative effect on survival during early postnatal development in altricial birds when postnatal conditions are harsh (reviewed in Williams 1994); the outcome would be a further relative investment in daughters via a reduction in competition by sons. In support of these predictions, we found that brood sex ratios of CORT-clipped mothers were significantly female biased by fledging (0.36 male/female ratio) compared with sham-clipped mothers (0.52 male/female ratio), indicating an indirect investment in daughters in the hormone-exposed group. Moreover, CORT-clipped mothers directly invested in daughters through increases in daughter quality (likely mediated via a decrease in the number of competing brothers), where these daughters were significantly heavier and structurally larger than daughters of sham-clipped mothers and the least physiologically stressed (lower baseline plasma corticosterone; Love and Williams 2008) of all groups. We also predicted that if maternal quality improved following laying, mothers adopting the flexible strategy of exposing young to yolk corticosterone could fledge both good-quality sons and daughters, given that small males are expected to catch up during postnatal development (Love et al. 2005). This prediction was also

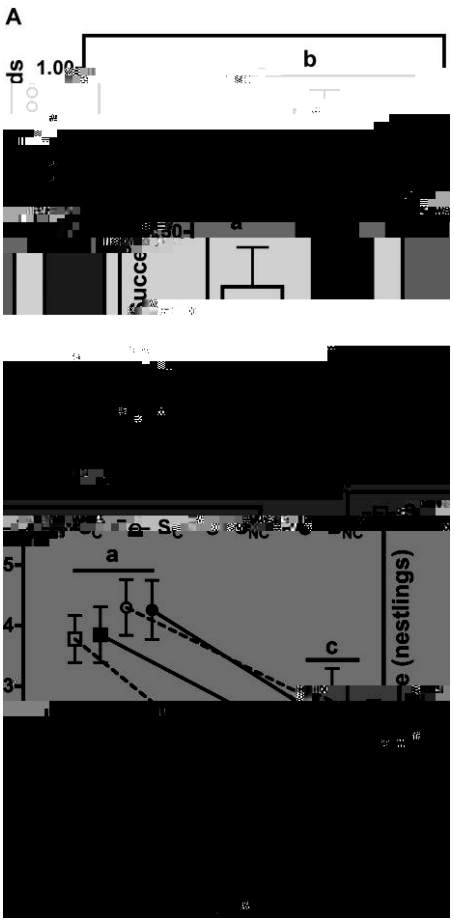

Figure 3: Treatment effects on (*A*) the proportion of successful second broods (at least one chick surviving to fledging age) and (*B*) brood size during postnatal development in second broods of European starling mothers (mean ± SEM; different letters represent significant differences between groups; treatments: sham-clipped [S_C], CORT-clipped [B_C], sham-nonclipped [S_{NC}], and CORT-nonclipped [B_{NC}]).

growth effects of the hormonal treatment while at the same time exhibiting positive effects on their survival. Hayward et al. (2006) reported that male Japanese quail (*Coturnix coturnix japonica*) were more developmentally sensitive (slower postnatal growth) than females following exposure to experimentally elevated yolk corticosterone. Finally, Satterlee et al. (2007) recently reported that prenatal corticosterone exposure negatively affected reproductive function in male Japanese quail. In this study, male offspring exposed to corticosterone hatched at lower body masses and begged at lower rates at hatching than did sham-exposed males, traits that likely impacted survival probability. However, sons of CORT-clipped mothers that survived to fledging showed no impacts on fledging body mass, structural size, or baseline plasma corticosterone levels (Love and Williams 2008). Nonetheless, we found that sons of CORT-clipped mothers had lower CMI compared with sham-clipped counterparts, suggesting an obligate cost to the male immune system following exposure to maternal/environmental stress (Chin et al. 2005; Love et al. 2005; Rubolini et al. 2005). However, this effect was not present in sons of CORT-nonclipped mothers, strongly indicating that costs of corticosterone exposure on the immune system are indirect and context dependent rather than a direct result of corticosterone-induced suppression of the immune system. Instead, it may be that CORT-exposed sons must "catch up" during development (Metcalfe and Monaghan 2001), resulting in a redirection of resources away from the immune system (Love et al. 2005). To make things even more complex, we found that even offspring of sham-clipped mothers had higher CMI than did sham-nonclipped offspring, further suggesting that passerine nestlings may have some capacity to increase CMI in response to stressful rearing conditions. Regardless, together with our previous work (Love et al. 2005), results from this study indicate that maternal corticosterone can act as an hormonal mechanism adaptively modifying sex allocation decisions in relation to maternal quality.

Carryover Effects of Yolk Corticosterone on Maternal Fitness

It has been proposed that maternal corticosterone modulates the cost of reproduction (Sinervo and DeNardo 1996; Love et al. 2005), given that glucocorticoids play a central role in the regulation of allostatic load during reproduction (Wilson and Wingfield 1992; Sinervo and

Figure 4: Effects of the 2005 treatment on local survival of European starling mothers to 2006 and 2007 (different letters represent significant differences between groups within years; treatments: sham-clipped [S_C], CORT-clipped [B_C], sham-nonclipped [S_{NC}], and CORT-nonclipped [B_{NC}]).

smaller brood benefits CORT-clipped mothers in three ways: (1) increased body condition at both the beginning and the end of the second (within-year) reproductive attempt, (2) increased future fecundity (e.g., more young, better-quality young) in both second (within-year) and future (across-year) broods, and (3) increased survival to future breeding attempts. First, we found that CORT-clipped mothers lost less body mass raising nestlings in their first (focal) broods compared with sham-clipped mothers. Moreover, CORT-clipped mothers began and

Figure 5: Effects of the 2005 treatment on (*A*) egg mass in 2006 and (*B*) brood size changes in 2006 of European starling mothers (mean ± SEM; different letters represent significant differences between groups; treatments: sham-clipped [S_C], CORT-clipped [B_C], sham-nonclipped [S_{NC}], and CORT-nonclipped [B_{NC}]).

DeNardo 1996; Love et al. 2004; Boonstra 2005; Wingfield 2005). The cost of reproduction is a central concept in evolutionary biology, where increased investment in current reproduction is predicted to lead to a decrease in maternal condition, future fecundity, and even survival (Williams 1966; Stearns 1992; Williams 2005). According to theories on costs of reproduction (Williams 1966; Reznick 1985) and data from empirical studies (Nur 1984, 1988; Orell and Koivula 1988; Dijkstra et al. 1990; Daan et al. 1996; Young 1996; Golet et al. 1998, 2004), the corticosterone-mediated decrease in brood size for CORT-clipped (matched) mothers should represent a reduction in maternal energetic investment in the current reproductive attempt compared with sham-clipped (mismatched) mothers. This energetic saving of raising a

mones such as thyroid hormones (de Jesus et al. 1990; Redding et al. 1991). Glucocorticoids also act as transcription factors since many genes have glucocorticoid response elements on DNA, and thus any potential change in glucocorticoid levels can have profound effects on development (for review, see Byrne 2001). In a sexually size-dimorphic species, these developmental effects could be sex specific, given that the sexes may require different levels of growth hormones, insulin-like growth factors, or even receptors for these hormones during prenatal or postnatal development. Since larger adult male size ultimately provides fitness benefits to not only sons but also their mothers, the need for an individual son to grow larger and faster during postnatal development could be co-opted by mothers taking evolutionary advantage of male sensitivity to prenatal glucocorticoid-dependent developmental pathways (Love et al. 2005). If this were the case, one may expect there to be parent-offspring conflict between males and mothers as sons attempt to break from their mother's developmental control (Crespi and Semeniuk 2004; Wendt Müller et al. 2007). However, if glucocorticoid-mediated developmental pathways are fixed and likely evolved before starlings became sexually size dimorphic, then males may not be able to developmentally ignore the presence of elevated yolk corticosterone. Complex molecular studies of differences in growth factors, growth hormones, and receptor density will begin to disentangle the two mechanistic hypotheses presented here and will help to address further technical differences between adaptation, exaptation, and constraint (Ketterson and Nolan 1999).

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