

# Multiple Environmental Stressors Induce an Adaptive Maternal Effect

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**ABSTRACT:** Evolution of adaptation requires predictability and recurrence of functional contexts. Yet organisms live in multifaceted environments that are dynamic and ever changing, making it difficult to understand how complex adaptations evolve. This problem is particularly apparent in the evolution of adaptive maternal effects, which are often assumed to require reliable and discrete cues that predict conditions in the offspring environment. One resolution to this problem is if adaptive maternal effects evolve through preexisting, generalized maternal pathways that respond to many cues and also influence offspring development. Here, we assess whether an adaptive maternal effect in western bluebirds is influenced by maternal stress pathways across multiple challenging environments. Combining 18 years of hormone sampling across diverse environmental contexts with an experimental manipulation of the competitive environment, we show that multiple environmental factors influenced maternal corticosterone levels, which, in turn, influenced a maternal effect on aggression of sons in adulthood. Together, these results support the idea that multiple stressors can induce a known maternal effect in this system. More generally, they suggest that activation of general pathways, such as the hypothalamic-pituitary-adrenal axis, may simplify and facilitate the evolution of adaptive maternal effects by integrating variable environmental conditions into preexisting maternal physiological systems.

**Keywords:** stress phenotypes, aggressive behavior, personality, corticosterone, dispersal.

## Introduction

Organisms inhabit a wide variety of complex and multifaceted environments, persistence in which requires accommodation of or buffering against environmental variation. Which outcomes prevail depends on preexisting adaptations and the diversity and predictability of the en-

vironments that an organism encounters (Schmalhausen 1949; West-Eberhard 2003). Yet the properties that allow organisms to maintain existing adaptations while accommodating novel and variable environmental inputs are not well known.

Adaptive maternal effects—where an environmental cue in the maternal generation influences maternal physiology in a way that can induce adaptive offspring trait variation—are a particularly clear example of this problem. The evolution of adaptive maternal effects is thought to require reliable environmental cues that predict future offspring environments (Nettle et al. 2013; Burgess and Marshall 2014; Auge et al. 2017), and an implicit assumption of most studies is that they evolve in response to a single specific cue. Yet such specificity necessarily limits the breadth of environments in which a particular adaptive maternal effect can function. Thus, in systems with extensive environmental variation over space and time, it is unclear how such precise environment-specific responses can evolve. Normal development always requires maternal input of resources and protection from external threats (Badyaev 2005a; Marshall and Uller 2007), but how these maternal pathways can channel offspring development to match future environments is a key problem (Mousseau and Fox 1998; Mateo 2014; Sheriff et al. 2018). One possible resolution to this problem is that adaptive maternal effects are not evolving to track specific environmental cues but instead are induced by a generalized preexisting pathway that integrates maternal experience of multiple environmental factors. Consequently, rather than evolving discrete pathways of information transfer from mothers to offspring, a diversity of environmental factors can influence the same inducible offspring traits, solely because both the cues and the offspring traits interact with the same maternal pathway.

Maternal stress responses mediated through the hypothalamic-pituitary-adrenal axis (HPA axis) are a likely pathway through which integration of multiple environmental cues may occur. The HPA axis is activated in response to environmental challenges (Denver 2009), leading to increases in circulating glucocorticoid hormones that

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can produce a suite of changes in organismal behavior and physiology (MacDougall-Shackleton et al. 2019). These changes are achieved through a multitude of effects that glucocorticoid hormones have on organismal metabolism, reproductive physiology, neuroendocrine systems, and immune response (Sapolsky et al. 2000). Not only do a wide variety of environmental factors raise baseline levels of glucocorticoid hormones (Ottenweller et al. 1992; Narayan and Hero 2014), such as poor nutrition, competition, predation, parasite load, and abiotic conditions (Harvey et al. 1984; Bowen et al. 2014), but maternal responses to stressors have also been shown to influence offspring development across taxa (table S1 [tables S1–S3 are available online]; for reviews, see Badyaev 2005*b*; Chaby 2016). Thus, offspring sensitivity during development to maternal stress responses may simplify the evolution of adaptive maternal effects, as the information content of variable, changing, and complex environments experienced in the maternal generation would be collapsed into a single nonspecific cue: whether current environmental conditions are benign or challenging.

Although the cues that induce these effects are either often unknown or are studied in isolation, a review of the literature (table S1) makes it apparent that similar patterns of offspring trait variation can be interchangeably produced by a variety of environmental stressors across taxa. For example, across both mammals and reptiles, variation in predation risk, abiotic conditions, and resource availability all activated maternal stress responses and led to the production of offspring that showed higher anxiety (table S1). This across-species survey supports the idea that maternal stress can link a diversity of environmental contexts to influence similar offspring traits. Yet a definitive test of this idea requires assessing whether the same pattern holds within a species.

Here, we investigate this in a system with a well-characterized adaptive maternal effect where multiple relevant aspects of environmental variation can be studied simultaneously to determine their effects on maternal stress response and, ultimately, on maternal effect expression. We combine long-term sampling of maternal corticosterone across diverse environmental contexts with a large-scale field experiment in western bluebirds (*Sialia mexicana*), a species with an egg-based maternal effect that adaptively influences offspring aggression and dispersal (Duckworth et al. 2015; Potticary and Duckworth 2018). This maternal effect is known to be induced by competition over nest cavities—the main limiting breeding resource in this species (Brawn and Balda 1988). Females experiencing heightened interference competition from other cavity-nesting species over their primary nest cavity produce aggressive sons that are more likely to disperse, whereas females experiencing less competition produce less aggressive sons that remain in their natal population

and acquire territories next to relatives (Duckworth 2008; Duckworth 2009; Aguillon and Duckworth 2015). This maternal effect is mediated by androgen allocation to the clutch and is correlated with sex-biased lay order, such that clutches where more males are produced early in the laying sequence have higher androgen levels than clutches where more males are produced later (Duckworth et al. 2015). Previous work has shown that this maternal effect is adaptive, as nonaggressive males generally have high fitness when they can breed adjacent to family members, whereas aggressive, dispersive males perform better in lower-density populations, where they acquire large resource-rich territories (Duckworth 2006*b*, 2008). Lastly, western bluebirds evolved in postfire habitat that shows predictable changes in intra- and interspecific competition, the presence of kin, and territory quality as a result of ecological succession (Hutto 1995; Kotliar et al. 2007; Duckworth 2008; Hutto et al. 2015), making it a particularly good system to study how an adaptive maternal effect can evolve given that a diverse array of environmental factors vary over space and time.

If elevation of maternal glucocorticoids functions as a general cue of environmental conditions, we predict that variation in multiple environmental contexts should be correlated with variation in maternal corticosterone levels and induce the maternal effect on offspring aggression. Alternatively, if this maternal effect is induced by pathways that are specific to competitive environment, then maternal experience of competition over nest cavities will be the only cue that induces it, and expression will not be correlated with maternal stress physiology. We tested these predictions using a 17-year data set of maternal effect expression and baseline corticosterone sampling to determine whether maternal glucocorticoid elevation is related to natural variation in multiple environmental contexts and, in turn, whether both are correlated with sex-biased lay order and offspring aggression. We combine this long-term data with experimental manipulations of female competitive environment to determine how competition influences baseline maternal corticosterone levels and variation in the maternal effect. Finally, we also determine whether maternal condition might serve as a proximate link between female glucocorticoid response and offspring phenotype by assessing the influence of residual body mass on glucocorticoid levels and sex-biased lay order.

## Methods

### *Study Populations*

Data were collected from two nest box populations that vary in time since population establishment during the 2001–2018 breeding seasons (mid-April to mid-August) in western Montana (table S2). Bluebirds typically forage

and defend a territory that spans 150–300 m around their primary nest box (Duckworth 2014). Distances between nest boxes are variable across sites, ranging from approximately 20 to 450 m apart; thus, the number of nest boxes on individual territories is highly variable. Most adults at these sites were captured to collect blood samples for hormone analyses and were color-banded for individual recognition and to take standard morphological measurements, including tarsus length and body mass. We measured tarsus length to the nearest 0.01 mm using calipers and body mass to the nearest 0.1 g on an electronic scale. Individuals captured as adults were aged using validated feather wear indices (Shizuka and Dickinson 2005).

#### *Assessing Environmental Contexts*

To determine whether multiple environmental contexts influence baseline maternal corticosterone levels and sex-biased lay order, we assessed variation in three social and ecological contexts that previous research has indicated to influence measures of fitness in bluebirds (Duckworth 2008; Duckworth et al. 2017; Potticary and Duckworth 2018), including nest cavity abundance, susceptibility to cold snaps, and distance to kin. We then determined whether variation in these factors correlated with sex-biased lay order, our measure of the maternal effect.

*Nest Cavity Abundance.* Nest cavities are the limiting breeding resource for bluebirds (Brawn and Balda 1988; Guinan et al. 2000), and bluebirds prefer territories with multiple nest cavities (Duckworth 2006a). Western bluebirds experience less direct interference competition at their primary nest box from other cavity nesting species when they have multiple nest cavities on their territory (see “Results” and Duckworth et al. 2015). Therefore, we assessed nest cavity availability by measuring the distance (in meters) to the nearest nest box that was not occupied by a conspecific.

*Susceptibility to Cold Snaps.* In insectivorous birds, cold snaps are an important source of nest mortality because they can disrupt the food supply during nest provisioning by making ectothermic insects immobile during cold weather (Winkler et al. 2013; Coe et al. 2015). Western bluebirds are insectivorous during the breeding season (Pinkowski 1979; Guinan et al. 2000), and cold storms accompanied by heavy rain or snow makes foraging for ectothermic insects difficult, such that many females will begin abandoning nests after several days of cold precipitation (Duckworth et al. 2017). In our study populations, cold snaps occur every year and generally overlap with the peak of bluebird breeding, during late May and early June (Duckworth 2006b; Duckworth et al. 2017). While

all females experience cold snaps, there is extensive variation in the likelihood that any given nest will fail. Females are more likely to abandon a nest when (a) the breeding male is not feeding her adequately at the nest during cold snaps (Duckworth 2006b) and (b) when a cold snap occurs during the nestling stage, especially when nestlings are older and are more demanding of food (Duckworth et al. 2017). Thus, nests that are most susceptible to cold snap mortality reflect a more challenging foraging environment, and we predict that susceptible females should have higher baseline corticosterone levels.

We defined susceptibility to cold snaps as whether females lost at least one offspring during a cold snap. We defined cold snaps as a period of two or more days in which the daily maximum temperature was less than 15°C and there was at least 10 mm of precipitation. These criteria were selected because prior research has shown them to be meaningful thresholds at which nests begin to fail as a result of inclement weather (Duckworth 2006b; Duckworth et al. 2017).

*Kin Adjacency.* Western bluebirds are known to have neutral territory boundaries with relatives, such that bluebirds living adjacent to kin experience fewer border disputes (Aguillon and Duckworth 2015) and often tolerate each other on their territories (Potticary and Duckworth 2018). Thus, having kin as neighbors effectively increases territory size and also increases the time budget for other activities (such as self-maintenance and foraging). For this reason, we assume that bluebirds breeding next to kin experience a reduction in conspecific competition. We assigned a categorical variable to each nest designating whether kin of the breeding male or female were on an adjacent territory. Kin that did not live immediately adjacent to the focal pair were unlikely to interact with them regularly and were not included in analyses. Individuals were considered relatives if they were (a) a social parent, (b) a sibling born in the same breeding season, or (c) a social offspring of the breeding male or female. While there is extrapair paternity in this system, western bluebirds identify relatives on the basis of social interactions (i.e., whom they were raised by or with, or nestlings hatched in their nest; Akçay et al. 2013), and for this reason only social kin were used for these analyses.

#### *Experimental Manipulation of Competition and Resource Availability*

To test whether competitive interactions over nest cavities influence female corticosterone levels and induce the maternal effect, we manipulated local nest cavity availability on 92 bluebird territories (for details, see table S2). Once a western bluebird initiated nest building, either a second nest box was placed to simulate high resource availability/

low competition (hereafter, “double-box” territories) or the territory was visited but no additional nest boxes were added to simulate low resource availability/high competition (hereafter, “single-box” territories). For double-box territories, the second nest box was placed in the same direction as and within 10–30 m of the primary nest box, based on availability of mounting substrate. Control and experimental territories were chosen so that they were spatially intermixed across the study area and so that the two groups did not differ in initiation date (for first nesting attempts, see below;  $t$ -test;  $t_{55} = 0.54$ ,  $P = .59$ ,  $n = 56$ ).

To confirm that our treatment affected heterospecific competition at the primary nest, we recorded behavioral interactions at each single- or double-box territory for 2 h between 06:00 and 12:00 once during late-building/early-incubation stages using Sony Handycam DCR-SX85 camcorders. This stage was selected because it is representative of the competition females experience during oogenesis and egg laying, which is when females are yolking eggs and androgen allocation is determined (Duckworth et al. 2015). All videos were transcribed by a single observer to minimize interobserver error.

For each video, we recorded (a) the percentage of time each breeding individual spent guarding (defined as time spent >1 min within 2 m of the nest box) and (b) the number of intrusions made by nest competitors onto the bluebirds’ territory (i.e., intrusions within 10 m of the nest box). These measures were taken for the pair’s primary nest box for all territories and for both nest boxes in territories with the double-box setup. The total amount of time the breeding male and female spent guarding the primary and/or secondary nest box (individually and cumulatively scored across both individuals) was calculated as a percentage of the total observation time. For nests in the incubation stage, the time the female spent incubating was subtracted from the total observation time. The number of intrusions onto the territory was assessed as a rate (number per unit of trial time).

Extended cold snaps occurred in both experimental years, and thus sex-biased lay order data from many first nesting attempts were unavailable. In this case, second nesting attempts were included if the first nest failed and no data were available on lay/hatch order. Sometimes a single nest box was used by multiple pairs across the breeding season, and first attempts for all females were included in analyses, even if a female was not the first to breed in the nest box that season.

#### *Hormone Sampling and Measurement*

To determine whether variable environmental conditions influenced female stress levels, we assessed natural and experimental variation in female baseline corticosterone

(for sampling details, see table S2). For the naturally varying hormone data, females were captured during the building, prelay, laying, and incubation stages using traps baited with mealworms. Only females bled in under 5 min were included ( $n = 62$  females). We selected this threshold because there was no relationship between bleed time and corticosterone levels for females bled within 5 min (linear regression;  $F = 1.49$ ,  $r^2 = 0.03$ ,  $P = .23$ ). In the experimental manipulation of resource availability, females were captured during late building, prelay, or the first week of incubation and were all bled in less than 2 min ( $n = 46$  females). Blood samples were kept on ice in the field until they could be centrifuged to separate red blood cells and plasma, which were stored at  $-20^{\circ}\text{C}$  prior to analysis.

After thawing, 10–20  $\mu\text{L}$  of plasma were extracted two times with 2 mL of diethyl ether. The ether fractions were decanted, and the combined fractions for each sample were vacuum dried for 50 min ( $55^{\circ}\text{C}$ ) using a CentriVap Benchtop Vacuum Concentrator. We measured the plasma corticosterone concentrations using a commercial enzyme-linked immunoassay kit (Corticosterone ELISA Kit, item 501320; Cayman Chemical Company, Ann Arbor, MI) according to the manufacturer’s instructions. The assay has a range of 8.2–5,000 pg/mL and a detection limit of 30 pg/mL. We first carried out a validation of the kit using an aliquot of pooled plasma that was stripped of endogenous steroid using dextran-coated charcoal and then spiked with 5,000 pg/mL corticosterone standard. Serial dilutions of pooled plasma from breeding western bluebirds demonstrated strong binding to the antiserum coated plates—the slope of the curve for western bluebird plasma ( $F = 2,701.59$ ,  $P < .01$ ,  $b_{\text{ST}} = -0.99$ ) did not differ from the slope of the standard used in the kit ( $F = 1,858.61$ ,  $P < .01$ ,  $b_{\text{ST}} = -0.99$ ; test for slope differences:  $F = 0.45$ ,  $P = .52$ ). Plasma samples collected from different populations, different years, and different treatments were randomly distributed across assays to reduce the potential for conflating assay-specific variation with year-, population-, or treatment-specific variation. The average intra- and interassay coefficients of variation were 6.61% and 15.18%, respectively.

This study was carried out in accordance with the recommendations and guidelines approved by university institutional and animal care and use committees and complied with all state and federal permitting guidelines.

#### *Assessment of the Maternal Effect*

*Sex-Biased Lay Order.* Previous work has established that sex-biased lay order (the number of males produced early vs. late in a clutch) is a reliable proxy for this maternal effect, as clutches where males are produced earlier in the lay order result in more dispersive/aggressive sons, while

those where males are produced later lead to more philopatric/nonaggressive sons (Duckworth 2009; Duckworth et al. 2015). Using this proxy allowed us to assess the maternal effect on offspring aggression in a consistent way across multiple years and sites. In our populations, the clutch sizes of first nesting attempts vary from four to seven eggs, with a modal clutch size of six. We measured sex-biased lay order as the number of males produced early in the lay order (position 3 or earlier) minus the number of males produced late (positions 4–7). We assessed sex-biased lay order across environmental contexts and in our field experiment by marking eggs as they were laid and marking nestlings on hatch by unique combinations of nail clipping until they could be banded with a USGS metal band. When exact lay order was not known, we used measurements of nestlings at 7–8 days of age to determine order, as previous work has shown that it is strongly positively correlated with both lay order and hatch order (Duckworth 2009; Duckworth et al. 2015). Nest boxes were visited at least once a week to monitor nest progress, to mark eggs, and to band, bleed, and measure nestlings. Nestlings were sexed using plumage differences that appear at day 14 of the nestling period or molecularly for those did not survive to that age. We extracted DNA from tissue of nestlings and eggs that died using the Blood and Cell Culture DNA Midi Kit (cat. no. 13343; Qiagen, Venlo, Netherlands). For molecular sexing, we used polymerase chain reaction (PCR) primers P2 and P8 (Griffiths et al. 1998), which anneal to conserved exonic regions and amplify across an intron in both *CHD1-W* and *CHD1-Z* genes. PCR on extracted DNA was carried out according to the protocol in Badyaev et al. (2005).

*Offspring Aggression in Adulthood.* We assessed the aggression of sons for a subset of nests for which we had measures of maternal corticosterone. Aggression is routinely measured for all adults using standardized protocols that simulate a territorial intrusion by a heterospecific competitor for nest boxes, the tree swallow (*Tachycineta bicolor*; for full details on the aggression trial protocol, see Duckworth 2006b). Tree swallows were used because they are the most frequent nest site competitor of bluebirds, and western bluebird aggressive response toward a heterospecific competitor is strongly and positively correlated with aggressive responses toward conspecific competitors (Duckworth 2006b). Moreover, aggression level is highly repeatable both within and across breeding stages as well as across a male's life (Duckworth 2006a, 2006b; Duckworth and Sockman 2012).

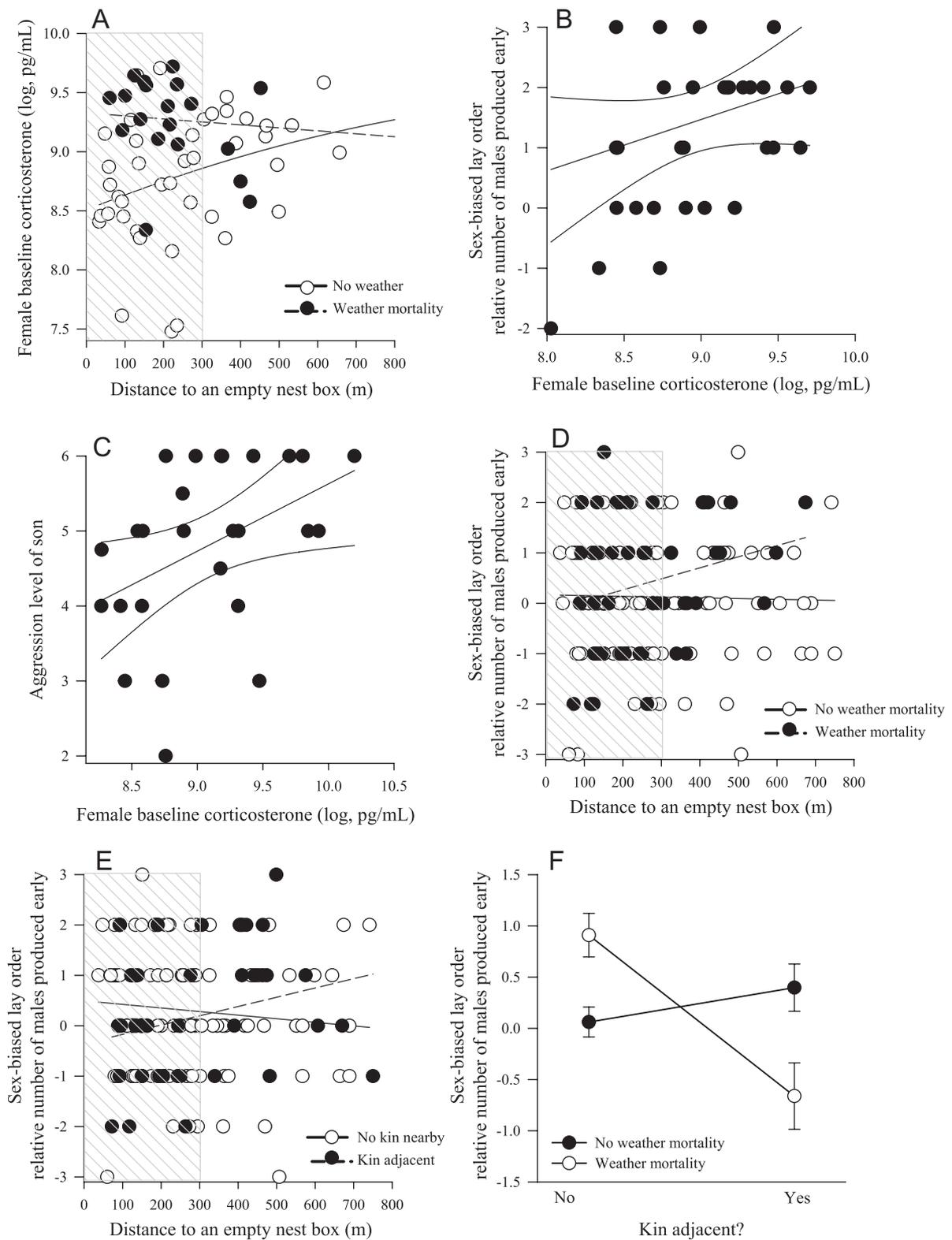
In brief, to conduct aggression trials, we placed a live tree swallow in a wire cage at a focal male's nest box and responses were recorded concurrently by an observer and a Panasonic HC-X920K HD camcorder. Observers

noted the number of times an individual flew by, attacked, and hovered, and aggression scores were assigned according to the following scale: 1, no aggressive behaviors; 2, hovering or flying by 1 to five times and zero attacks; 3, hovering or flying by more than five times and zero attacks; 4, one to five attacks; 5, six to nine attacks; and 6, ten or more attacks. Counts of these behaviors by the real-time observer were independently verified by a separate observer from the video. The measurement error due to variation between these two observers was assessed using a subsample of 10 individuals that had been measured in the field in real time and by an observer in the laboratory from video using one-way ANOVA. The effect of interobserver measurement error was less than 2% of the individual identity effect (mean squares: 0.11 vs. 8.1) and was therefore negligible.

#### Statistical Analyses

All statistical analyses were performed using SAS (ver. 9.4). Results are means  $\pm$  SE. All measures of plasma corticosterone were log transformed prior to analyses. For all corticosterone analyses, only a single sample from each female was used from her first breeding attempt of the season to ensure independence of data points. For all measures of sex-biased lay order, only a single clutch was included from each female. For unbanded females, we assumed that second nesting attempts in the same nest box were by the same female and excluded these attempts unless other information was available.

From the long-term naturally varying data, we used linear mixed models (PROC MIXED) utilizing a maximum likelihood estimation method. All continuous variables were centered at the mean. We included sex-biased lay order or female baseline corticosterone levels as the dependent variable and nest cavity availability, cold susceptibility, and distance to kin and their interactions as fixed effects. The effect of ecological conditions on female corticosterone was originally analyzed including female age, nesting stage, and capture date as covariates, but these were excluded from the final model, as none were related to baseline corticosterone levels ( $P > .15$  for all; table S3). For corticosterone analysis, we also included a second-order effect of nest cavity availability (measured as distance to the nearest nest box), based on a nonlinear pattern observed in plots of the raw data. Year and nest box identity were initially included as random effects in both models, but only year was retained in the final models ( $P > .15$  for all other factors; table S3). Clutch size was correlated with sex-biased lay order ( $r = -0.35$ ,  $P < .01$ ), so we included it as a covariate in the models. We also tested whether maternal corticosterone influenced clutch size using females for whom clutch size was known using



**Figure 1:** Natural variation in maternal baseline corticosterone levels, environmental contexts, and the maternal effect on sex-biased lay order. A, Females that were susceptible to cold weather (black circles, dashed line) had higher baseline corticosterone levels irrespective of the distribution of nest boxes on their territories, while females that were not susceptible (white circles, solid line) had higher corticosterone levels only

ANOVA ( $n = 70$ ). Only significant interaction terms were included in the final models. We performed either  $\chi^2$  or  $t$ -tests to determine whether any of the environmental factors covaried. The influence of female baseline corticosterone levels on sex-biased lay order was analyzed using linear regression for a subset of females that were captured prior to egg laying, during laying, or within 4 days of clutch completion ( $n = 27$ ). Last, we measured the influence of maternal corticosterone on the aggression phenotypes of her male offspring when they reached adulthood using linear regression. Only sons whose mothers were captured and bled in the nesting attempt that the son was born in were included ( $n = 26$ ).

Female condition was quantified as the residuals of a linear regression of body mass on tarsus length (Brown 1996). Body mass and tarsus showed a significant positive relationship ( $F = 4.14$ ,  $P = .04$ ,  $b_{ST} = 0.16$ ) where larger females were heavier in general; however, there was substantial scatter, justifying the use of residuals as a proxy for condition. The influence of female condition on sex-biased lay order was assessed using linear regression. For the influence of condition on baseline corticosterone levels, we used a mixed model with year as a random effect. Given that females carrying eggs are substantially heavier than other females irrespective of their condition, we excluded females that were captured 3 days before laying (during rapid yolk deposition) through the end of laying.

Data from the experimental resource manipulation were initially analyzed using linear mixed models with either female baseline corticosterone or sex-biased lay order as the dependent variable, status as a double- or single-box territory holder as a fixed effect, and population, year, and nesting attempt as random effects. However, given that these random effects did not explain a significant proportion of the variance, they were removed from subsequent analyses ( $P > .15$  for all). Therefore, we tested the influence of single- and double-box treatments on female corticosterone using  $t$ -tests with pooled variances because variances were equal (folded  $F$ ;  $F = 1.0$ ,  $P = .99$ ,  $n = 46$ ). We assessed the influence of experimental resource manipulation on sex-biased lay order using a  $t$ -test with the Satterthwaite approximation after determining that var-

iances were unequal (folded  $F$ ;  $F = 1.82$ ,  $P = .05$ ,  $n = 92$ ). All proportions from behavioral observations were arcsine-root transformed, assessed for normality using Shapiro-Wilk  $W$ -tests, and analyzed with  $t$ -tests and general linear models (GLMs) if normally distributed; if transformation failed to normalize distributions, we used two-sided Wilcoxon rank sum tests. The relationship between female corticosterone and competitive interactions was assessed using linear regression.

## Results

### *Natural Variation in Multiple Environmental Contexts Influenced Maternal Corticosterone and the Maternal Effect*

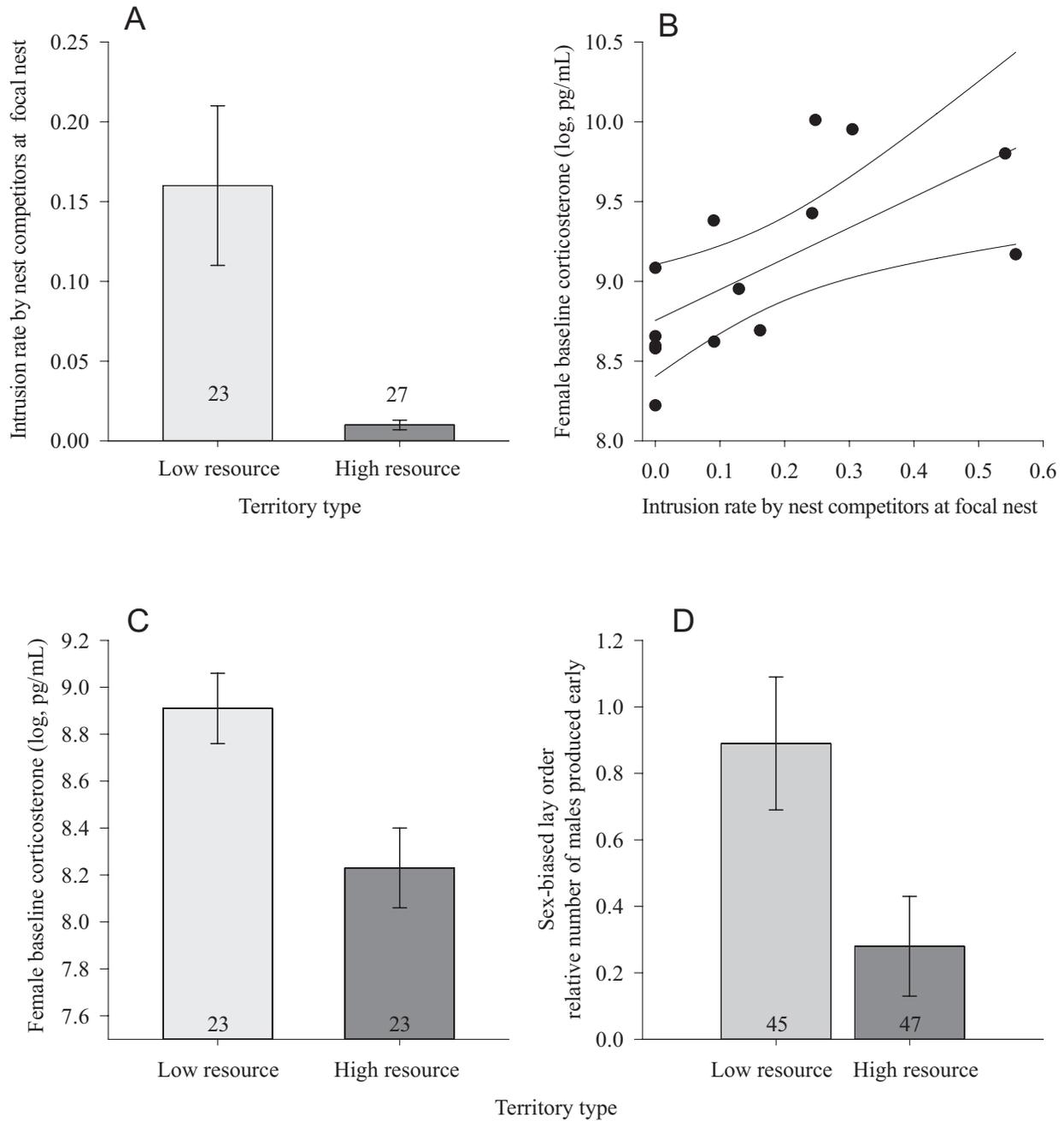
Both susceptibility to cold weather and nest box availability influenced female corticosterone, as females had higher baseline corticosterone levels if they did not have an extra nest box on their territory (as indicated by the significant second-order [ $F = 3.99$ , 95% confidence interval (CI) = 0.00 to 0.33,  $P = .05$ ,  $n = 62$ ] but not first-order [ $F = 0.01$ , 95% CI =  $-0.73$  to 0.16,  $P = .94$ ] relationship; fig. 1A) and if they were more susceptible to cold weather (least square means: susceptible vs. not susceptible =  $9.24 \pm 0.15$  vs.  $8.72 \pm 0.13$  log pg/mL;  $F = 17.38$ , 95% CI =  $-1.46$  to  $-0.51$ ,  $P < .01$ ). There was also a significant interaction between these effects ( $F = 4.80$ , 95% CI = 0.05 to 1.04,  $P = .03$ ; fig. 1A), such that females that were susceptible to cold weather showed higher corticosterone levels irrespective of the nest cavity availability on their territory, while females that were not susceptible to cold weather showed a positive relationship between corticosterone levels and distance to the nearest empty nest box. There was no relationship between susceptibility to cold weather and the distance to the nearest available nest box ( $t$ -test:  $t = -0.15$ ,  $P = .88$ ), suggesting that the effects of each on maternal corticosterone are independent. Females with higher baseline corticosterone during oogenesis produced sons earlier in the lay order than did females with lower baseline corticosterone (linear regression;  $t = 3.59$ , 95% CI = 0.25 to 0.92,  $P < .01$ ,

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when nest box availability was low (maximum territory size for western bluebirds is indicated here and in *D* and *E* by hatched rectangles). *B*, Females with higher baseline corticosterone levels were more likely to produce sons early in the lay order than were females with lower levels. Positive sex-biased lay order indicates that more dispersive sons were produced, while a negative order indicates that more philopatric sons were produced. *C*, Females with higher baseline corticosterone levels had sons that showed higher aggression as adults than females with lower baseline corticosterone levels. *D*, Females that were more susceptible to cold snaps and that experienced higher competition for nest cavities produced more sons early in the lay order, whereas for females that were not susceptible there was no relationship between nest cavity distribution and sex-biased lay order. *E*, Females living next to kin are more likely to produce sons late in the lay order if they had extra nest cavities on their territories. However, there was no relationship between sex-biased lay order and nest cavity distribution for females that did not have kin nearby. *F*, Interaction between proximity to kin and susceptibility to cold snaps. "Kin" indicates that kin of the breeding pair were on adjacent territory, while "weather" indicates that females experienced weather-related offspring mortality. Bars indicate means  $\pm$  SE.

$r^2 = 0.34$ ,  $n = 27$ ; fig. 1B), and adding clutch size did not change these results (sex-bias lay order:  $t = 4.26$ , 95% CI = 0.24 to 0.92,  $P < .01$ ,  $r^2 = 0.35$ ; clutch size:  $t = -0.86$ , 95% CI =  $-0.86$  to 0.43,  $P = .40$ ). More-

over, maternal corticosterone levels were not related to clutch size ( $F = 0.20$ ,  $P = .90$ ,  $n = 70$ ). Sons whose mothers had higher baseline corticosterone during oogenesis were more aggressive when they reached adulthood



**Figure 2:** Experimental manipulation of competitive environment alters female baseline corticosterone levels and the maternal effect on sex-biased lay order. *A*, Females on single-box territories experience more intrusions by nest competitors than do females on double-box territories. *B*, Females that experienced more intrusions had higher baseline corticosterone levels. *C*, Females on single-box territories had higher baseline corticosterone levels than females on double-box territories. *D*, Females on single-box territories produced more sons early in the lay order than did females on double-box territories, indicating that females on single-box territories produced more dispersive sons. Bars indicate means ± SE, and numbers on bars indicate sample sizes.

( $F = 5.29$ , 95% CI = 0.09 to 1.71,  $P = 0.03$ ,  $r^2 = 0.18$ ,  $n = 26$ ; fig. 1C).

Living adjacent to kin did not influence maternal corticosterone ( $F = 0.66$ , 95% CI =  $-0.34$  to  $0.81$ ,  $P = .42$ ,  $n = 62$ ), and there was also not a strong effect of year on corticosterone levels ( $Z = 1.20$ ,  $P = .11$ ). However, there was a small sample size ( $n = 12$ ) for females that were sampled for both corticosterone and had kin living adjacent, lowering the power to detect an effect of kin on female corticosterone. Despite the lack of a clear link between kin adjacency and corticosterone, in the analysis with more power we did find a link between living adjacent to kin and sex-biased lay order ( $F = 5.99$ , 95% CI = 0.47 to 1.61,  $P < .02$ ,  $n = 138$ ). Moreover, in this larger data set, there were significant interactions between all environmental factors and sex-biased lay order. Specifically, females produced more sons early in the lay order when they did not have extra nest boxes on their territory but only when they experienced one of these other contexts at the same time (interaction of nest box availability and susceptibility to cold weather:  $F = 6.15$ , 95% CI =  $-0.74$  to  $-0.08$ ,  $P = .01$ ; fig. 1D; nest box availability with kin adjacency:  $F = 4.78$ , 95% CI =  $-0.65$  to  $-0.03$ ,  $P < .03$ ; fig. 1E). Females that were both susceptible to cold weather and were not breeding near kin produced more sons early in the lay order, whereas females that were more susceptible to cold weather and breeding adjacent to kin produced sons late in the lay order (interaction between susceptibility to cold weather and kin adjacency:  $F = 11.83$ , 95% CI =  $-1.92$  to  $-0.52$ ,  $P < .01$ ; fig. 1F). Neither susceptibility to cold weather ( $F = 0.00$ ,  $P = .96$ ) nor nest box availability ( $F = 3.65$ ,  $P = .06$ ) was significantly related to sex-biased lay order, as main effects and none of the fixed effects covaried (susceptibility to cold weather vs. kin adjacency:  $\chi^2 = 0.51$ ,  $P = .48$ ; nest box availability vs. kin adjacency:  $t = -0.75$ ,  $P = .46$ ; nest box availability vs. susceptibility to cold weather:  $t = 0.37$ ,  $P = .71$ ).

Female condition, as measured by residual body mass, was not related to sex-biased lay order ( $F = 1.71$ , 95% CI =  $-0.49$  to  $0.11$ ,  $P = .21$ ,  $n = 22$ ). However, it was negatively related to female baseline corticosterone levels ( $F = 11.64$ , 95% CI =  $-0.14$  to  $-0.04$ ,  $P < .001$ ,  $n = 149$ ).

#### *Experimental Manipulation of Nest Cavity Availability Influences Competitive Behavior, Maternal Corticosterone, and Sex-Biased Lay Order*

Females on double-box territories guarded their primary nest box less (mean  $\pm$  SE:  $0.07 \pm 0.03$  of observation time) and experienced fewer territorial intrusions from hetero-

specific competitors (mean  $\pm$  SE:  $0.01 \pm 0.004$  intrusion rate) than did females on single-box territories (guarding:  $0.18 \pm 0.04$  of observation time;  $Z = 2.46$ ,  $P < .01$ ,  $n = 50$ ; heterospecific intrusion rate:  $0.16 \pm 0.05$  intrusions;  $Z = 3.45$ ,  $P < .01$ ,  $n = 50$ ; fig. 2A). Moreover, when male and female guarding was scored cumulatively, pairs on double-box territories guarded less overall (mean  $\pm$  SE:  $0.18 \pm 0.04$  of observation time) than did pairs on single-box territories ( $0.32 \pm 0.06$  of observation time;  $Z = 1.78$ ,  $P < .04$ ,  $n = 50$ ). Females that experienced more intrusions from nest competitors at their nest box had higher baseline corticosterone (linear regression:  $F = 7.86$ , 95% CI = 0.37 to 3.11,  $r^2 = 0.42$ ,  $P < .02$ ,  $n = 13$ ; fig. 2B).

Correspondingly, females on double-box territories had lower baseline corticosterone (mean  $\pm$  SE:  $4.52 \pm 0.44$  ng/mL; fig. 2C) than did females on single-box territories ( $8.97 \pm 1.32$  ng/mL;  $t = -3.18$ ,  $P < .01$ ,  $n = 47$ ). Moreover, females on single-box territories produced more sons earlier in the lay order (mean  $\pm$  SE:  $0.89 \pm 0.20$  sons produced early relative to late) than did females on double-box territories ( $0.28 \pm 0.15$  sons produced early relative to late;  $t = 2.43$ ,  $P < .02$ ,  $n = 92$ ; fig. 2D).

## Discussion

A critical question in the evolution of adaptive maternal effects is how complex multigenerational coordination can evolve. Namely, such adaptive maternal effects seem to require the simultaneous evolution of maternal responses to particular environmental conditions, maternal physiological mechanisms that transmit this information to offspring, and the ability of offspring to receive and incorporate this information into trait development. Our findings suggest that a complex adaptive maternal effect can evolve through preexisting maternal stress pathways that link multiple environmental contexts to offspring trait variation. Not only did multiple distinct environmental contexts influence maternal glucocorticoid levels, but we also found that, in both naturally varying populations and experimental manipulations, females with higher baseline corticosterone levels produced more dispersive and aggressive offspring.

Often multiple environmental cues were required to induce the maternal effect, providing further evidence that female production of dispersive offspring is not tied to a particular cue but only to a female's general experience of environmental challenges. For example, females experiencing natural variation in competition over nest cavities and those that were more susceptible to cold weather exhibited higher corticosterone levels than did those solely experiencing competition (fig. 1A). Yet our experimental manipulation of the competitive environment showed

that a single cue, if intense enough, was sufficient to elevate baseline maternal corticosterone levels (fig. 2C). Finally, in the larger data set of ecological factors and sex-biased lay order, no single factor explained variation in this maternal effect (fig. 1D–1F). Instead, variation in sex-biased lay order resulted from interactions between all three factors that we assessed, including presence of kin. This interchangeability and complexity of interactions between environmental cues and their influence on sex-biased lay order is likely explained by the fact that repetition of the same stressor is not required to increase baseline corticosterone levels in vertebrates, only that the exposure to environmental challenge is chronic (Ottenweller et al. 1992; Narayan and Hero 2014).

Perturbation of homeostatic systems, often referred to broadly as “stress” (e.g., Ellis and Del Giudice 2019), during development is expected to increase offspring trait variation because, by definition, contexts that perturb organismal functioning or development are situations that organisms are not buffered against (McEwen and Wingfield 2003; Badyaev and Young 2004; Badyaev 2005b). Indeed, direct transfer of glucocorticoids to developing offspring can have disruptive effects on development (Reynolds 2013; Moisiadis and Matthews 2014), which likely explains why there are both maternal and embryonic mechanisms to buffer embryos from maternal glucocorticoids across taxa (e.g., Painter and Moore 2005; Vassallo et al. 2014; Paitz et al. 2016; Zhu et al. 2019). This begs the question of how maternal glucocorticoid elevation can become a cue to induce adaptive offspring phenotypes when its main effect is to disrupt offspring development. One possibility is that the proximate effects of maternal glucocorticoids are indirect. The multitude of physiological systems affected by glucocorticoid elevation, from immune response to metabolism to neuroendocrine changes, provide a diversity of targets by which maternal response to stressors can impact offspring trait development. Such a diversity of mechanisms suggest that what is evolving with stress-induced maternal effects are not novel maternal pathways to detect and react to predictive cues but instead are the links between the already existing “infrastructure” of the HPA axis and offspring trait variation. In bluebirds, we did not find any correlation between female condition and sex-biased lay order; however, previous work found that yolk androgens were higher in clutches that produced more dispersive offspring (Duckworth et al. 2015), suggesting that elevated glucocorticoids may influence offspring phenotype through stimulation of maternal hormones produced at the egg follicle. In other avian species, maternal stress response has been shown to influence production of local gonadal androgens (Henriksen et al. 2011; de Haas et al. 2017), which are transferred to egg yolk (e.g., Ahmed et al. 2014a). The relationship between circulating

maternal glucocorticoids and yolk androgens is variable (Henriksen et al. 2011; de Haas et al. 2017) and is expected to be influenced by life history (Bentz et al. 2016). Thus, this may be a plausible mechanism in bluebirds, although further research is needed.

While some studies have shown that mothers induce adaptive offspring variation to specific stressors (e.g., Storm and Lima 2010), stress-induced maternal effects may be particularly likely to evolve for offspring traits that are generalized responses to environmental conditions, such as dispersal polymorphisms. Dispersal is costly (Bonte et al. 2012), often requires specific traits to ensure success (Duckworth 2012; Duckworth et al. 2018), and dispersal decisions are usually made early in life (e.g., Roff 1986; O’Riain et al. 1996). Since persistent adverse conditions increase baseline corticosterone over time (Ottenweller et al. 1992; Narayan and Hero 2014), baseline maternal corticosterone may contain more long-term information about environmental suitability than an offspring’s brief experience of the postnatal environment prior to dispersal (Lea et al. 2017). For these reasons, maternal experience of current environmental conditions may be as—or more—useful than offspring’s direct experience, particularly if offspring dispersal depends on traits that develop during a sensitive period early in ontogeny (Harrison 1980; Cremer and Heinze 2003; Duckworth et al. 2018). An important goal for future work in this field is to determine under what circumstances maternal effects evolve in response to specific cues versus more generalized responses to challenging environments.

It is contentious whether stress-induced variation in offspring is adaptive, particularly in humans, where early-life exposure to stress leads to chronic disease phenotypes in adulthood (Hales and Barker 1992; Gluckman et al. 2005; Chaby 2016). While many adaptive hypotheses have been proposed to explain such observations (Bateson et al. 2014; Berghaenel et al. 2016), they have been criticized because the window of information transfer from mother to offspring is often short compared with the life span of long-lived organisms, making maternal experience a poor predictor of the environment offspring will experience (Lea et al. 2015). However, these criticisms ignore the possibility that the induced phenotype is not preparation for the specific environment experienced by the mother but instead prepare offspring to construct their own environment. Our results provide an example of this, as the maternal effect prepares offspring to leave the maternal environment and disperse to a new area. Similarly, many of the behavioral and physiological traits that are frequently observed in stress-induced phenotypes of other organisms (e.g., heightened vigilance to threat, fear responses, and thrifty metabolism) are potentially useful in enabling organisms to seek out and survive in unfamiliar environments.

Why would an adaptive maternal effect depend on a maternal pathway (i.e., HPA axis) that is responsive to many cues instead of tracking a specific cue? Historically, bluebirds depended on postfire habitat that changed continuously during forest succession. Not only does intraspecific competition vary over time, based on the composition of aggressive birds (Duckworth and Badyaev 2007) and presence of kin (Aguillon and Duckworth 2015), other factors, such as interspecific competition, nest cavity availability, predation, and food abundance, also change over time (Saab et al. 2007; Swanson et al. 2011). The dynamics of these changes vary across postfire habitats depending on the particular plant community and burn severity (DellaSala and Hanson 2015), and they can also vary extensively within postfire patches (Agee 2004). For this reason, the specific stressors any given female may experience are multidimensional and highly variable over space and time. Elevated glucocorticoid levels may thus act to summarize the effects of multiple interacting factors to reduce the dimensionality of such complex, variable habitats.

A common theme in the evolution of complex systems is the bow-tie structure, where diverse inputs are accommodated by a conserved “waist” to produce multiple outputs. Bow-tie structures are a frequently observed property of complex systems—from embryonic development (Raff 1996; Irie and Kuratani 2011) to variation in brain structure (Briscoe and Ragsdale 2018) to the infrastructure of the internet (Akhshabi and Dovrolis 2011)—and are thought to confer both robustness and adaptability because they can accommodate perturbations and fluctuations in inputs while maintaining stability of outputs across time and space. In our case, the ability of the maternal HPA axis to respond to a diversity of environmental factors likely increases the robustness of the maternal effect across a broad spectrum of contexts. Moreover, such a structure may increase adaptability on an evolutionary scale because the core physiological pathways (e.g., HPA axis) can be readily recombined with a multitude of output traits without disrupting overall system integration (Akhshabi and Dovrolis 2011; Briscoe and Ragsdale 2018; Telemeco et al. 2019). The appearance of bow ties across scales in biology, business, and technology suggest that this type of organization reflects a fundamental structural principle of complex systems that facilitates integration of heterogeneity in inputs while simultaneously allowing for robust regulation of outputs. As such, this perspective may provide a useful framework for understanding the diversity of maternally induced stress effects that have evolved across species (table S1).

Stressors are highly species and population specific, but the maternal stress response is a general pathway that can respond to many environmental contexts and influence offspring development. Our results show that an array of ecologically relevant factors induce a complex maternal ef-

fect, the ultimate expression of which has been shown to be strongly adaptive in the offspring generation. The ability of maternal stress to integrate multiple cues into a diversity of physiological effects in the mother may facilitate the evolution of adaptive maternal effects, as it provides a preexisting pathway for the environment to influence offspring development.

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### Statement of Authorship

A.L.P. wrote the original draft; reviewed and edited subsequent drafts; contributed to conceptualization, funding acquisition, methods development/experimental design, data collection, data analysis, data validation, and data visualization; and provided resources. R.A.D. contributed to conceptualization, funding acquisition, methods development/experimental design, data collection, data analysis, supervision, and the review and editing of the manuscript and provided resources.

### Data and Code Availability

Data have been deposited in the Dryad Data Depository (<https://doi.org/10.5061/dryad.z08kpr99>; Potticary and Duckworth 2020).

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