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Delayed effect of early-life corticosterone treatment on adult anti-predator behavior in a common passerine

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ABSTRACT

Acute, short-term effects of early-life stress and associated glucocorticoid upregulation on behavior are widely documented across vertebrates. However, the persistence and severity of these effects are largely unknown, especially through the adult stage and in wild species. Here, we investigate long-term effects of experimental post-natal increases in a circulating glucocorticoid on antipredator behavior in wild house sparrows (*Passer domesticus*) tested in captivity. We manipulate circulating corticosterone concentration in wild, free-living nestlings, transfer fledglings to captivity, and test juveniles and adults for two measures of antipredator behavior: evasiveness during a direct human encounter, and propensity to escape from a risky environment. We find no effect of early-life stress hormone manipulation on escape behavior, but a delayed effect on evasive behavior: evasive behavior was depressed in adults but not juveniles, and influenced by current body condition. These results highlight the importance of state-behavior interactions and life stage in assessing long-term effects of early-life stress, and provide rare evidence for delayed effects of early-life stress to adults of a wild avian species.

1. Introduction

Stressors that elicit a physiological stress response early in life can have long-term impacts on an animal's behavior, physiology, and fitness [1–5]. Understanding of such "programming" or "organizational" effects [4,5] has broad implications for human health, captive animal health and productivity, and for managing wildlife populations [6]. However, the persistence of programming effects over the lifespan of animals, especially for wild species, and their relevance for natural systems is not well established [2]. Non-viviparous vertebrates, such as birds, are useful models for study of long-term behavioral effects of early-life stress because they lack a direct physiological postnatal link between mothers and offspring (*i.e.*, maternal lactation), which simplifies the isolation of endogenous effects of stress in offspring [7]. However, the few existing studies of wild birds have provided mixed results regarding long-term programming effects, casting doubt on the persistence of these effects in wild species, although most studies do not follow subjects to adulthood [2,3].

Two general models have been proposed to explain the pattern of long-term effects expected due to early-life stress: inoculation and adaptive tuning [6]. The inoculation model predicts an inverted U-shaped relationship between early-life stress and future resilience. Too little stress and animals are unprepared; too much stress overwhelms the organism leaving it unable to cope, while moderate early-life stress inoculates young animals by preparing them for future stresses [6]. For example, laboratory rats exposed to high neonatal stress exhibit later life cognitive impairment and depression-like syndrome [8],while rats exposed to moderate neonatal stress later display a dampened stress-induced hypothalamic-pituitary-adrenal (HPA) response [9] and decreased anxiety-like behavior [10]. In contrast, the adaptive tuning model predicts that long-term effects of early-life stress are adaptive at all stress levels, if the early-life environment accurately predicts the adult environment [11]. In one well developed form of this model, the "adaptive calibration model" (ACM), degrees of early-life stress result in specific, adaptive long-term responses [6]. Under the ACM, low early life stress signals a low stress adult environment, thus adults should exhibit low anxiety and vigilance, and high engagement and exploration; high early-life stress signals a high stress adult environment, thus individuals should be vigilant with strong anti-predator behavior (*e.g.*, fight or flight responses) [6,12]. For example, rats exposed to high stress early in life display enhanced fear-memory formation as young adults under high stress conditions [13,14], an adaptive response to stressful environments. While both the inoculation and adaptive tuning models have gained some empirical support, long-term studies of trait

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persistence across life stages, especially in wild species, are lacking [3].

Glucocorticoids (including corticosterone, the primary avian glucocorticoid) are a class of steroid hormones [15] that can have activational or organizational/programming effects [16] on the brain [5,17], physiology [1,7,18] and behavior [4]. Programming effects typically occur when steroid hormones act during ontogenetic transitions [16,19], such as the perinatal period [17,19]. Early-life stress produces programming effects in laboratory rodents and birds, including changes in the corticosterone (CORT) stress response of juveniles and young adults [7,20–23] and in behavioral traits including neophobia, anxiety, and aggression [21,24–26], as well as changes in other physiological, behavioral, and life history traits [2,27,28]. However, long-term effects of early-life glucocorticoid exposure are not well established for wild species. Most studies follow individuals only to the juvenile or dispersal stage, and mixed results from the few long-term studies of early life stressors in wild birds cast doubt on the persistence of such effects in wild species [2]. Furthermore, many studies of wild species manipulate environmental stressors, not glucocorticoids directly, making isolation of proximate mechanisms for long-term effects difficult.

This study examines long-term effects of early-life glucocorticoid exposure in a wild bird species, the house sparrow (*Passer domesticus*). Previous work in this species has shown that early-life glucocorticoid treatment decreases nestling immune response [29,30] and body mass [30], with no change in structural size [unpublished data]. These effects are relatively short-term and disappear by the juvenile and adult stages [unpublished data]. Here, we expand upon this work by investigating long-term behavioral effects of repeated exposure to high concentrations of CORT ([CORT]) at the nestling stage on two aspects of juvenile and adult antipredator behavior: a direct "predator" encounter (*i.e.*, "evasiveness"), and a high risk situation with cues of predator presence (*i.e.*, "propensity to escape").

Antipredator behavior is critical to fitness because it directly influences the survival outcome of predation encounters. Appropriate antipredator behavior involves response to both direct encounters with predators and cues of predator presence. Wild house sparrows are ground-foraging cavity-nesters [32] and their direct encounters with predators typically occur in habitat consisting of many narrow openings (*e.g.,* shrubs, grain fields) [33]. Thus, maneuverability on the ground and air are a priority over flight speed and endurance while evading predators in this species. Adult depredation while in the nest is also known to occur in house sparrows [34] and in both males and females in this study population (personal observation). Predators can trap adults in the nest by blocking the entrance, causing significant adult mortality in other cavity-nesting species [35,36]. Escape from the cavity in response to predator cues is critical to survival in these encounters. Hence, both evasive behavior in a direct predator encounter, and escape from a high risk environment given predator cues are essential aspects of house sparrow antipredator behavior. In this study, we use humans in predator encounter/cue testing; a technique used in the laboratory [37,38] and wild [39–41] to evaluate antipredator behavior in this species and others. Wild house sparrows flush in response to human approach [42]and humans have occasionally been observed destroying nests with young at our study site, thus human disturbance is an ecologically relevant threat to this population. Antipredator behavior is also a probable target of behavioral programming due to early-life stress because it can be affected by early-life experience [42] and perinatal glucocorticoid exposure [38,43,44], in other taxa.

Here, we manipulate glucocorticoid concentrations in wild house sparrow nestlings, then measure antipredator behavior at the juvenile and adult stages in captivity. We base our hypotheses off of the adaptive tuning and inoculation models predicting outcomes in response to high early life stress, and evaluate if hormonal signals of stress alone, without environmental cues, are sufficient to induce long-term behavioral changes. If high early-life stress adaptively tunes animals for stressful later life environments, we expect that birds that were exposed to high corticosterone as nestlings will exhibit enhanced antipredator behavior (*i.e.,* increased evasiveness in a direct encounter and propensity to escape given cues of a predator) as juveniles and adults. Alternatively, if high early-life stress overwhelms young birds (the inoculation model), we expect that birds exposed to high corticosterone as nestlings will exhibit poor antipredator behavior (decreased evasiveness and propensity to escape) as juveniles and adults. In either case, we predict these effects to be long-lasting if early-life stress has programming effects on antipredator behavior.

2. Materials and methods

Permission to capture, sample, and hold house sparrows in captivity was issued by the French government (DREAL Poitou-Charentes, permit delivered to FA) and by the Muséum National d'Histoires Naturelles. All experimental procedures were approved by the French Government (R45GRETAF1-10) and Centre National de la Recherche Scientifique, and conform to guidelines set forth by the French Ministry of Higher Education and Research and Ministry of Agriculture and Fisheries.

2.1. Study population and reproductive monitoring

House sparrows (*Passer domesticus)* were part of a free-living population breeding in nest boxes in the vicinity of Prissé la Charrière (46°09′12″N 0°28′59″W), a small rural and agricultural town located in Deux-Sèvres, France. Nest boxes were monitored for clutch initiation and hatching dates, clutch size, hatching success, and chick mortality until eight days post-hatching. Brood size was the number of nestlings in the nest at hatching. Nestling age was calculated from hatch date as day zero, one day post-hatching as day one, and so on. All nests within the study area with more than one nestling were used in the study, and all nestlings were used in each nest (N = 131 nestlings).

2.2. Nestling manipulation

All nestlings were given a plastic color band for individual identification and at eight days post-hatching hormonal manipulation began. The hypothalamic-pituitary-adrenal axis may develop slowly in altricial species such as the house sparrow [19], hence we began treatment in the mid to late nestling period, when eyes are open and a related species, the white-crowned sparrow (*Zonotrichia leucophrys*), is able to mount a strong HPA axis stress response [45]. Half of the nestlings in each nest were assigned to corticosterone-fed ("CORT-fed") and half to control groups. Assignment was alternated between nestlings as they were removed from the nest box and assignment order was alternated between nests (*e.g.,* CORT-fed first, control second; control first, CORT-fed second, *etc.*). CORT was delivered to CORT-fed nestlings non-invasively following the method of Breuner et al. [46] and used successfully in subsequent studies [47–49]. Mealworms (*Tenebrio molitor*) were injected with 20 μL of 0.6 mg/mL CORT for 8 and 9 day-old chicks and 0.9 mg/mL CORT in dimethyl sulfoxide (DMSO) for 11 day-old nestlings. Control nestlings were fed worms injected with 20 μL of DMSO. CORT concentrations were determined based on previous work with house sparrow nestlings [30] and white crowned sparrows [46,47], scaled to the average mass of house sparrow nestlings at days 8, 9 and 11 post-hatching, and verified in this population. At 8 days post-hatching, this dosage increased circulating CORT concentration 8.6-fold within 40 min after worm ingestion (CORT-fed: mean [CORT] ± SE = 76.74 ± 11.44 ng/mL; Control: 8.95 ± 1.45 ng/mL), consistent with a high early-life stress (Fig. 1). This concentration is lower than the average [CORT] obtained by Loiseau et al. [30] for house sparrow nestlings injected with CORT suspended in peanut oil, and previous work in house sparrows has shown that 9-day old nestlings can reach between 65 and 70 ng/mL and in some cases over 100 ng/mL during a standardized capture-restraint test [50]. Thus, the CORT concentrations we obtained were within physiological range for a

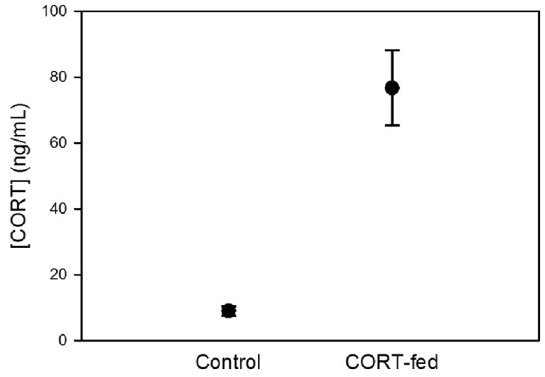

Fig. 1. Circulating [CORT] in house sparrow nestlings 10–40 min. Following ingestion of a control worm or CORT-fed worm (N = 127 individuals). Nestlings were all at 8 days post-hatching, the first day of treatment, and were from the same population and year of this study. Points indicate means, error bars are standard errors. There was no effect of time between worm ingestion and sampling on CORT concentration. Control standard errors are too small to be visible.

strong, acute stressor.

Worms were chilled at − 20 °C prior to injection to limit movement, and were injected ventrally, into the central abdomen, between exoskeletal segments. If any fluid leaked from the mealworm it was discarded. CORT- and vehicle-injected worms were fed to nestlings with blunt-end forceps, at their nest box. Nestlings beg in response to noise and movement, but if nestlings did not beg, the beak was gently opened, and one end of the worm inserted into the mouth, after which an automatic swallow reflux facilitated swallowing. The schedule of feedings was designed to be unpredictable, to discourage habituation by nestlings. To this end, CORT-fed nestlings received a CORT-injected worm on day 8 (0.6 mg/mL), day 9 (twice – morning and evening; 0.6 mg/mL), and day 11 post-hatching (0.9 mg/mL), while control nestlings received a vehicle injected worm on the same schedule. Worms were delivered in the mornings (8:00–12:00), except for on day 9 when a worm was delivered in the morning and evening (18:00–20:30). On day 9 post-hatching, all nestlings were banded with a permanent metal ring with a unique identifying number.

2.3. Captive housing

At and after twelve days post-hatching, nests were checked daily and nestlings that appeared ready to fledge (alert, active, and showing signs of flight) were taken into captivity (median = 13 days post-hatching, maximum = 15, minimum = 12, N = 97). Two to four fledglings were removed from each nest, and a total of 97 fledglings from 23 nests were taken into captivity. Equal numbers of control and CORT-fed fledglings from each nest were brought into captivity (*i.e.,* one control and one CORT-fed, or two control and two CORT-fed fledglings) except in three instances where only three fledglings were in the nest, all were small, and we were concerned about survival. In these cases we brought all fledglings in the nest into captivity. In one of those nest groups a fledgling died within one week of captivity, thus only two nests had unequal numbers of CORT-fed and control birds that were measured at juvenile and adult stages, one nest with an extra CORT-fed bird, and the other with an extra control bird.

Fledglings were hand-fed by caretakers blind to treatment category until they were capable of feeding on their own. Fledglings were housed in wire bird cages (Vision S01, 45.5 × 35.5 × 51 cm) with all siblings (2–4 birds per cage) until birds reached basic plumage, after which pairs consisting of one CORT-fed and one control bird were housed together (*i.e.*, two birds per cage). When possible these pairs were sibling pairs, otherwise pairs were age-matched. Birds were not separated visually or acoustically within the care room. Birds were supplied with *ad libitum* mixed seeds, vitamin and mineral soaked cat food, mineral and salt blocks, water (changed daily), and millet on the stalk. Grit was supplied three times per week and cages were equipped with perches of varying heights. Birds were kept on natural daylight schedules.

2.4. Behavioral testing

At the juvenile (54–75 days post-hatching, basic plumage) and adult (159–171 days post-hatching, alternate plumage) stages we conducted evasiveness (N = 35 control, 34 CORT-fed juveniles; 27 control, 23 CORT-fed adults) and propensity to escape (N = 31 control, 35 CORT-fed juveniles; 30 control, 28 CORT-fed adults) testing. At the time of juvenile testing, birds had spent at least 40 days in captivity, a sufficient period of time for captive habituation and longer than commonly used for behavioral studies [51]. All testing occurred between 10:00 and 17:45, at least 2 h after sunrise and 1 h before sunset. This time frame allowed birds to eat before testing and avoided dawn and dusk, the most active and social periods of the day for house sparrows [32]. When testing was repeated on consecutive days it was conducted at the same time of day for each bird. Each time point of testing initiation was approximately evenly split between control and CORT-fed birds.

2.4.1. Evasiveness

Evasiveness was cumulative time to capture, by hand, in the home cage. This is a variant of a test used to evaluate antipredator response in lizards [37,38] and is very similar to measurements of “docility” (*i.e.*, response to trapping and handling) in other animals [52–54]. We do not use this terminology because all birds responded to attempted capture with evasive maneuvers; we felt that “evasiveness” was a better description of the trait we measured.

The cage was placed on the floor and the experimenter kneeled next to it. Time began when the experimenter placed her hand in the cage and began attempting to capture the birds. Time stopped when a bird was captured and under control of the experimenter. Individual “Time to capture” was cumulative time spent attempting to capture birds in the home cage until that individual was caught. For example, in cage X, bird A was caught after 20 s. Time to capture for bird A is 20 s. Bird B was caught 10 s after bird A. Time to capture for bird B is 30 s (*i.e.*, 20s + 10s). Birds were identified by unique metal band numbers following capture and the experimenter did not attempt to identify birds prior or during capture, to minimize any capture bias. The same experimenter (JKG) captured all birds to avoid a potential bias. Juvenile birds were captured six times: once per day for three consecutive days, followed by a rest period of four days, and then again once per day for three consecutive days (to test for short-term repeatability of evasiveness). Adult birds were captured three times: once per day for three consecutive days.

2.4.2. Propensity to escape

The testing arena (Fig. 2) consisted of a wooden box with a central square (100 cm wide × 100 cm long × 40 cm deep) and four arms (30 cm wide × 60 cm long × 40 cm deep) angled slightly upward radiating out from the center of each side of the square. The arena was elevated approximately 70 cm off of the ground. Three of the arms were closed with wooden covers, the fourth was “open”, but enclosed by a mist net bag supported by wire that extended approximately 30 cm out from the arm, then dropped down approximately 30 cm (Fig. 2, B). The open arm was the same for all trials. The entrance to the arena consisted of a square hole cut into the top of the center of the arena, and closed with a removable wooden cover (Fig. 2, A). The upward angle of each arm of the arena forced birds to fly, not walk to the upward edge, thus preventing inspection of the “open” exit (and detection of the mist net which may have discouraged “escape”).

Juvenile birds were tested twice to examine short-term repeatability of behavior. The second juvenile testing occurred one week after the first juvenile testing. Adult birds were tested only once. Birds were taken from their home cage, placed in a dark cloth weigh bag and

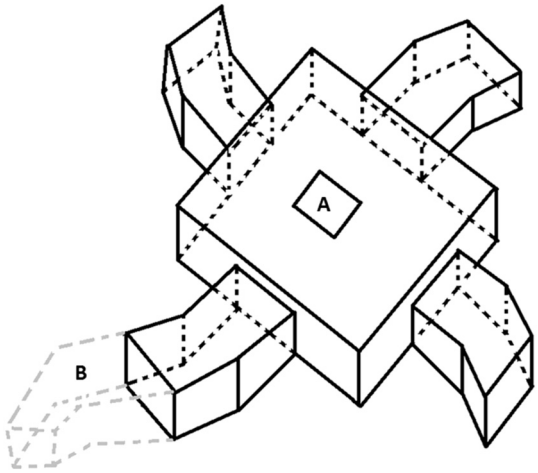

Fig. 2. Diagram of the testing arena for propensity to escape. The arena was elevated approximately 70 cm off of the ground. Black lines indicate wood material; grey lines indicate mist net material. Each arm was tilted slightly upward to force flight (ascent was too slippery to walk) and discourage investigation of the mist net. Birds were placed in the center of the maze (A), after which the entrance was quickly covered to prevent escape back out the entrance. The central square opened into four arms, three of which were closed with wood and one with a mist net bag (B) that extended out and down from the arm.

individually transported to the testing room which was separated visually and acoustically from the home cage room. The experimenter removed the entrance cover (Fig. 2, A) at the center of the maze, placed the bird in the arena, and rapidly replaced the cover. Time began as soon as the bird was placed in the arena. A pilot study indicated that birds that exited the arena did so within 6 min, thus each bird was given 6 min to exit the arena. If the bird escaped the arena it was caught by the mist net at the end of the "open" arm. Because a large number of birds did not escape the maze and chose to remain inside, we were unable to analyze time to escape and instead examine propensity to escape as a binary variable (escape, or not).

2.5. Body size and condition measurements

Birds were given one week to recover from behavioral testing, after which they were weighed (electronic balance: ± 0.1 g) and tarsus (caliper: ± 0.1 mm) was measured. Weight and measurements were taken at least 2 h after sunrise to allow all birds to eat. Tarsus length was chosen to represent body size because it is not prone to temporary damage. Body condition was calculated as the residuals of a regression of mass on tarsus for each life stage, separately, because the relationship between mass and tarsus length changes throughout development. Size and weight measurements were not conducted prior to behavioral testing to limit handling stress and because preliminary results showed that body condition and body size are repeatable in the long-term in this species. Both body condition and tarsus length were repeatable characteristics of the individual after the first pre-basic molt, between the juvenile (approximately 61 days post-hatching) and adult stages (approximately 172 days post-hatching): consistency repeatability (± 95% CI; see Statistics for details on calculation of repeatability estimates) for body condition was estimated at 0.46 ± 0.23 ($P < 0.001$ estimated by a likelihood ratio test, $N = 116$, 58 individuals), and for tarsus length at 0.89 ± 0.07 ($P < 0.001$ estimated by a likelihood ratio test, $N = 116$, 58 individuals). Unpublished data indicates that CORT treatment has no long-term effect on either parameter in house sparrows. Thus, we do not expect results obtained in this study to result from differences in body size or condition between CORT-fed and control sparrows.

2.6. Statistics

All statistical analyses were conducted in R (version 3.0.3) and were evaluated within a multimodel inference framework, which has the benefit of evaluating the relative information explained by multiple models. General linear mixed models (GLMMs) were ranked using Akaike's Information Criterion corrected for small sample size (AICc), where AICc = N*log(RSS/N) + (2 K(K + 1))/(N − K − 1) + 2 K; N is the sample size, RSS is the residual sums of squares for the model, and K is the number of parameters, including error [55]. Thus, AICc balances information explained and complexity of a model [56] (Burnham et al. 2011). We evaluated models first by ΔAICc (the difference in AICc between the candidate model and the model with the lowest AICc), followed by examination of the beta coefficient and associated 95% confidence interval (95% CI). We only consider models whose AICc value was less than that of the null to be important models regarding interpretation of the effects of treatment [57,58]. We present model-averaged beta coefficients using the subset of models within Δ4 of the top model, here. Please see Supplementary information for model sets and AICc comparison parameters.

GLMM analyses were conducted in R using the package 'lme4' [59]. For analyses evaluating the effect of treatment on behavior, models were derived from all subsets of a global model and were compared *via* AICc model selection with the R package MuMIn [60]. We report both marginal r-squared values (*i.e.*, proportion of variance explained by fixed factors alone) and conditional r-squared values (*i.e.*, the proportion of variance explained by both random and fixed factors) obtained using the r.squarredGLMM function in R [61,62] for top models. Any birds that exhibited signs of illness (*e.g.,* rapid weight loss or behavioral changes) prior to testing were excluded from analyses (9 juveniles, 4 adults). We analyzed juvenile and adult stages separately because preliminary analysis indicated that interactions between life stage, sex, treatment, body size, and condition resulted in overspecification of models if life stages were combined.

2.6.1. Evasiveness

Preliminary analyses indicated that residuals were not normally distributed, thus we log-transformed time to capture (s), after which residuals approximated normality. Sex differences, body size (*e.g.,* larger birds provide larger targets for capture), and body condition could all affect speed of human capture, hence these parameters were included in the global model, and allowed to interact with each other and treatment. For juveniles, number of cage mates was also included as an additive fixed factor (binary factor: 1 = one other bird in the cage, 2 = two or three other birds in the cage). Finally, "capture event" was also included as a fixed additive effect (juveniles were captured six times over ten days). Random effects included in all models were nest of origin, bird ID, and cage number. Thus, the global general liner mixed model (Gaussian family, identity link) in lme4 syntax for juvenile evasiveness was: log(Time to catch) ~ Treatment * Body Condition * Tarsus Length * Sex + #Cage mates + Capture event + (1|Nest) + (1|ID) + (1|Cage). Where ~ means "regressed on", "*" indicates an interaction, and parentheses are used to denote random factors. Tarsus length was centered by z-scoring and treatment was coded as 1 for control and 2 for CORT-fed.

At the adult stage, our smaller sample size restricted the number of interactions we could evaluate, thus, sex was also not allowed to interact with other fixed effects. Number of cage mates was not included in adult models because adults were all housed with one cage mate. For adult evasiveness, our global model was: log(Time to catch) ~ Treatment * Body Condition * Tarsus Length + Sex + Capture event + (1|Nest) + (1|ID) + (1|Cage).

2.6.2. Propensity to escape

For escape testing, the greater power required for analyzing a binary variable and our smaller sample size (two measures for juveniles, one

for adults) restricted the number of interactions we could evaluate, thus, sex and capture event were not allowed to interact with other fixed effects. The global model consisted of the fixed effects of treatment, body condition, tarsus length (z-scored), and sex, and the random effects of nest of origin. For juveniles we also included the fixed effect of escape event and random effect of bird ID, because juveniles were tested twice. Thus, the global GLMM (binomial family, logit link) in lme4 syntax for juveniles was: Escape ~ Treatment * Body Condition * Tarsus Length + Sex + Escape event + (1|Nest) + (1|ID). For adults the global GLMM was: Escape ~ Treatment * Body Condition * Tarsus Length + Sex + (1|Nest).

2.6.3. Repeatability of behavior

To evaluate the extent to which evasiveness and escape behavior are consistently performed by individuals within the population [63,64] we calculated repeatability (*R*) estimates. Repeatability is the proportion of the total variation that can be accounted for by differences between individuals (*i.e.*, between-individual variance divided by total phenotypic variance). If within-individual variance is high (denominator) and between-individual variance is low (numerator), repeatability will be low. Conversely, if within-individual variance is low and between-individual variance is high, repeatability will be high [65].

For evasiveness, we square-root transformed time to capture because residuals exhibited heteroscedasticity and the square root transformation is variance stabilizing. Agreement repeatabilities were calculated for evasiveness [65] at the juvenile and adult stages (short-term), and between these stages (long-term), after adjusting for the random effect of cage number. To ensure that the juvenile and adult stages were equally represented within the long-term data set we used only the first week of testing at the juvenile stage, and only individuals who were tested three times at each life stage. For escape testing, we calculated consistency repeatabilities and present link-scale estimates of repeatability [65], at the juvenile stage (short-term) and between the juvenile and adult stages (long-term). GLM-based repeatability estimates for both behavioral tests and associated 95% confidence intervals were calculated in R using the package rptR [65].

3. Results

3.1. Evasiveness

There was no effect of treatment on juvenile evasiveness (Fig. 3A); the top model included the fixed effects of capture event (6 captures, once per day), body condition, and number of cage mates (ΔAICc of the closest model including treatment = 0.73, marginal r^2 of the top model = 0.04, conditional r^2 of the top model = 0.40, N = 407, 69 individuals). There was an effect of treatment on adult evasiveness: the top model included interactions between treatment and body condition, and tarsus and body condition (ΔAICc of the closest model excluding treatment = 7.74, marginal r^2 of the top model = 0.13, conditional r^2 of the top model = 0.16, N = 149, 50 individuals). CORT-fed adults were generally less evasive than control adults (Table 1, Fig. 3B). However, that effect was dependent on body condition, such that CORT-fed adults in very poor body condition were no less evasive than controls and the difference between treatment groups was only apparent for birds in good body condition (Fig. 4). As body condition increased, evasiveness decreased for CORT-fed adults (Fig. 4B), with little effect on controls (Fig. 4A). Irrespective of treatment, for large adults, evasiveness increased with body condition, while that relationship was (more weakly) reversed for small adults (interaction Body Condition * Tarsus, see Table 1).

To evaluate whether the lack of treatment effect at the juvenile stage was due to differential mortality between the juvenile and adult stages (*e.g.,* birds whose behavior was unaffected by treatment had a higher rate of mortality), we re-analyzed juvenile data using only birds that survived to adulthood. Using this survivor data set, treatment still had no effect on juvenile evasiveness: the top model included only the fixed effect of capture event (ΔAICc of the closest model including treatment = 0.69, marginal r^2 of the top model = 0.01, conditional r^2 of the top model = 0.74, N = 288, 48 individuals). Thus, phenotypic plasticity and not differential mortality appears to be responsible for life stage differences in evasiveness.

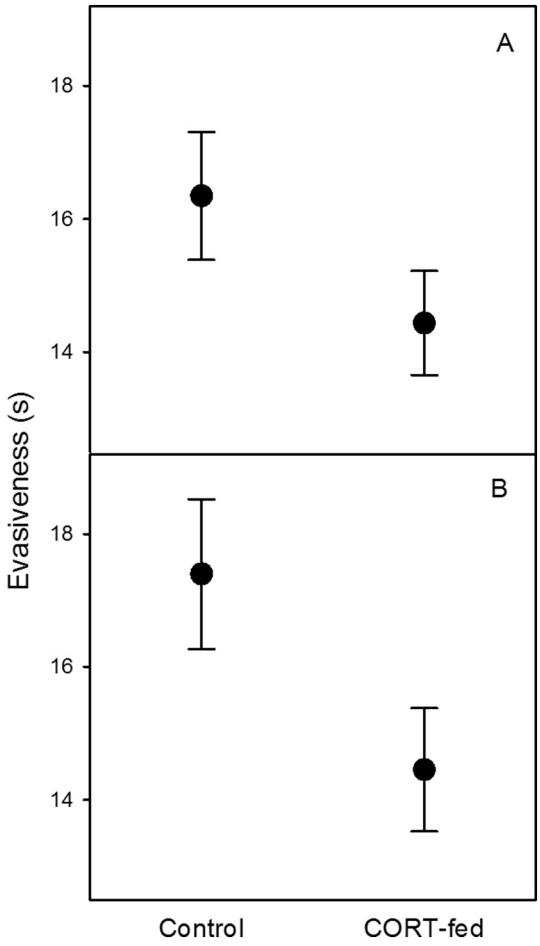

Fig. 3. Effect of treatment on (A) juvenile (N = 407, 69 individuals) and (B) adult evasiveness (N = 149, 50 individuals). The y-axis indicates evasiveness, which was measured in cumulative time in seconds until capture by hand in the home cage. Dots are means, error bars are standard error. There was no important difference between control and CORT-fed juveniles, while birds that were CORT-fed as nestlings were less evasive as adults than control birds.

Evasiveness was repeatable at the juvenile and adult stages over the short-term. At the juvenile stage, agreement repeatability (± 95% CI) was 0.36 ± 0.11 (P < 0.001 by likelihood ratio test) and the GLM model including the random effect of bird ID was the top model (ΔAICc of the model not including bird ID = 56.0, N = 400, 68 individuals). For adults, agreement repeatability was lower, but still significant (0.19 ± 0.18; P = 0.02 by likelihood ratio test) and the top GLM model included the random effect of bird ID (ΔAICc of the model not including bird ID = 0.27, N = 139, 48 individuals). Evasiveness was also moderately repeatable over the long-term, between the juvenile and adult stages (6 captures: 3 at the juvenile stage, 3 at the adult stage). Long-term agreement repeatability (± 95% CI) was estimated at 0.26 ± 0.12 (P < 0.001 by likelihood ratio test), and the top model included the random effect of bird ID (ΔAICc of the model not including bird ID = 21.33, N = 272, 47 individuals).

Table 1
Model averaged beta coefficients and 95% confidence intervals (CIs) for parameters within Δ4 of the top model predicting juvenile and adult evasiveness (time to capture by hand). Parameters were estimated over all models, not just those where a predictor occurs. 95%CIs are calculated from adjusted standard error. All models included the random effect of nest of origin, bird ID, and cage number. "BC" indicates body condition; tarsus length was z-scored. For fixed factors, the non-reference factor is indicated in parenthesis. A * indicates an interaction term. Beta coefficients of predictors for which 95% CI did not overlap zero are highlighted in bold.

Predictor	Beta	± 95% CI
Juveniles		
Capture event	0.065	0.102
BC	**0.049**	**0.036**
#Cage Mates (> 2)	0.097	0.267
Sex (Females)	− 0.081	0.218
Treatment (CORT-fed)	− 0.042	0.157
BC * Sex	− 0.010	0.076
Tarsus	− 0.004	0.104
Treat * Tarsus	0.021	0.134
Sex * Tarsus	0.005	0.067
Adults		
BC	0.028	0.106
Treatment (CORT-fed)	**− 0.317**	**0.192**
Tarsus	0.044	0.101
BC * Treatment	**− 0.259**	**0.177**
BC * Tarsus	**0.146**	**0.093**
Sex (Females)	0.005	0.049
Tarsus * Treatment	0.007	0.078
Capture event	0.007	0.096
BC * Treatment * Tarsus	0.014	0.096

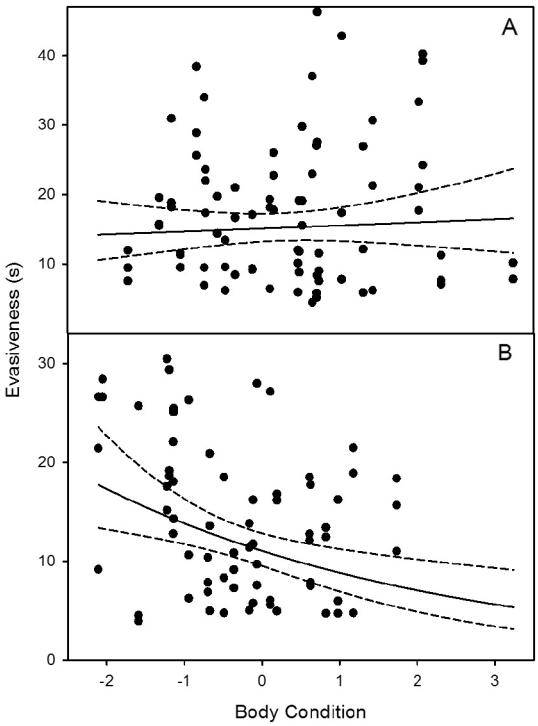

Fig. 4. Effect of body condition on adult evasiveness for (A) control (N = 80, 27 individuals) and (B) CORT-fed (N = 69, 23 individuals) birds. The y-axis indicates evasiveness, which was measured as time in seconds until capture by hand in the home cage. Solid lines are regression lines, dotted lines are 95% confidence intervals for the regression estimate, and dots are raw data points. There was no relationship between evasiveness and body condition for control birds, while evasiveness decreased with body condition for CORT-fed birds.

3.2. Propensity to escape

Treatment had no effect on propensity to escape for juveniles or adults. The top model predicting escape from the testing arena for juveniles included the fixed effects of sex and random effects of nest and bird ID (ΔAICc of the closest model including treatment = 1.44, marginal r^2 of the top model = 0.03, conditional r^2 of the top model = 0.17, N = 132, 68 individuals). For adults, the top model was the null model (ΔAICc of the closest model including treatment = 2.22, N = 58 individuals) (Table 2).

Table 2
Model averaged beta coefficients and 95% confidence intervals (CIs) for parameters within Δ4 of the top model predicting juvenile and adult propensity to escape. Parameters were estimated over all models, not just those where a predictor occurs. 95%CIs are calculated from adjusted standard error. All models included the random effect of nest of origin. "BC" indicates body condition; tarsus length was z-scored. For fixed factors, the non-reference factor is indicated in parenthesis. A * indicates an interaction term.

Predictor	Beta	± 95% CI
Juveniles		
Sex (Female)	− 0.406	0.968
BC	− 0.047	0.227
Treatment (CORT-fed)	0.099	0.590
Escape event	0.016	0.324
Tarsus	− 0.004	0.214
Sex * Treatment	0.020	0.367
BC * Sex	− 0.001	0.106
Sex * Tarsus	0.007	0.157
Adults		
Sex (Females)	− 0.209	0.896
Tarsus	0.039	0.302
BC	0.042	0.342
Treatment (CORT-fed)	0.009	0.486
BC * Treatment	− 0.035	0.413

Over the short-term, propensity to escape was not repeatable at the juvenile stage; consistency repeatability (± 95% CI) on the link-scale was only 0.14 ± 0.18 (P = 0.11 by likelihood ratio test), and the top model did not included the random effect of bird ID (ΔAICc of the model including bird ID = 1.22, N = 128, 64 individuals). Propensity to escape was also not repeatable over the long-term, between the juvenile and adult stages; consistency repeatability (± 95% CI) on the link-scale was 0.00 ± 0.15 (P = 0.5 by likelihood ratio test) and the top model did not include the random effect of bird ID (ΔAICc of the model including bird ID = 2.16, N = 108, 54 individuals).

4. Discussion

Our results indicate that early-life exposure to high glucorticoid concentration affects evasiveness during a direct predator encounter, but not propensity to escape from a high risk environment in the presence of predator cues (*i.e.,* handling by a human). These results provide no support for adaptive tuning in response to early-life glucocorticoid exposure, and instead support the inoculation model of programming effects [6], that development is constrained by stress experienced during critical periods. Sex-specific effects of early-life stress have been reported for some other species (*e.g.* neurogenesis and anxiety-like behavior in laboratory rats, *Rattus norvegicus*, [66,67]; neophobia in zebra finches, *Taeniopygia guttata,* [26]; growth and metabolic rate in song sparrows, *Melospiza melodia*, [68]). However, we found no evidence of sex-specific effects in house sparrows.

Corticosterone (CORT) treatment during the mid to late postnatal nestling period resulted in a strong, but delayed effect on evasive behavior, especially for birds in good body condition: CORT-fed house sparrows in good body condition displayed poor evasive behavior as adults. Poor body condition at adulthood appeared to overwhelm the negative effect of treatment by increasing evasive behavior in CORT-fed birds to control levels. This response may confer an advantage during a direct predator encounter to CORT-fed birds in poor body condition over those in good body condition, and suggests that early-life stress exposure interacts with current physiological state. State-behavior interactions are well known in behavioral ecology, and positive/

negative feedback loops between state and behavior are hypothesized to play a key role in maintaining (and destroying) behavioral syndromes and the prolonged effects of early-life stress [6,69,70]. State-dependence in our study may have developed through permanently altered physiology (*e.g.* energy regulation, [68]) and/or gene by environment interactions [71] associated with perinatal glucocorticoid exposure. Our results support the framework proposed by Luttbeg and Sih [69] and refined by Sih [6] that the long-term effects of early-life stress depend on ecological cost-benefit and state-behavior interactions.

Similar findings that body condition interacts with perinatal stress are documented in other species, notably the threespined stickleback (*Gasterosteus aculeatus*). Perinatal glucocorticoid exposure in this species also interacts with body condition to negatively impact some, but not all [72], antipredator behaviors [43]. Offspring of stickleback mothers exposed to predation stress while gravid are less likely to orient toward a predator and are more easily caught by predators than offspring of control mothers, with a negative impact on survival [43]. Thus, parents are motived to limit exposure of offspring to stressors. Additionally, in perinatal CORT-exposed sticklebacks, as in our sparrows, size/body condition is negatively related to antipredator behavior, with large CORT-exposed offspring being most easily caught by predators [43]. However, other antipredator behaviors in response to predator cues appear to be enhanced by maternal predator-exposure, including freeze responses and shoaling in sticklebacks and other species [42,72,73] and social clinging behavior in rhesus macaques [14]. Generalizing effects of perinatal stress on antipredator behavior across species and behaviors is difficult due to methodological differences in timing, application, and severity of early-life stressors. In spite of these differences, an emerging trend might be that in response to early-life stress defensive behaviors [14] in response to predator cues are enhanced, but ability to respond adaptively to direct encounters with predators is decreased. This may be especially true when animals are large or in good body condition. Our study supports only the latter part of this trend, as we found no support for enhancement of defensive escape behavior in sparrows.

A possible explanation for poor performance of perinatally stressed animals in good body condition may be long-term detrimental effects of catch-up growth following early-life stress. Previous work in this species shows that early-life CORT treatment depresses nestling growth [30]. Poor neonatal nutrition and subsequent catch-up growth is associated with poor endurance and locomotor activity under challenging conditions in fish [74] and birds (zebra finch, [75]). CORT-fed birds in high body condition may have invested more in catch-up growth than those in low body condition, and thus incurred higher costs. Our early-life stress treatment may also have affected muscular or metabolic capacity, due to poor early growth conditions [76], or cognitive/neuromotor abilities [77,78]. Early-life stress exposure is linked to smaller hippocampi and fewer hippocampal neurons in Western scrub-jays (*Aphelocoma californica*) [79], impairment of neural synaptic plasticity [80,81] and deterioration of complex cognitive behavior [81,82] in laboratory rodents, and poor neuromotor skills in primates [77] and humans [78]. Impaired synaptic activity is associated with deficits in spatial learning [83], an effect of early-life stressors (*i.e.,* nutritional deprivation and CORT implantation) that has been documented in other wild avian species in captivity (Western scrub-jay [84]; black-legged kittiwake, *Rissa tridactyla* [28]), suggesting widespread cognitive impairment following early-life stress. Of course, our test for evasive antipredator behavior involved several factors that could affect time to capture (*e.g.,* neuromotor skills, endurance, spatial orientation, fear response, motivation). However, deteriorating neuromotor/cognitive abilities in CORT-fed sparrows, perhaps due to long-term costs of catch-up growth, fit with our observations and those of other species. Assessments of cognitive, metabolic, and locomotor abilities in relation to early-life stress are obvious next steps to evaluate these hypotheses.

There is little literature regarding delayed appearance of programming effects at the adult but not juvenile stage, especially for birds and wild species, because very few studies have followed individuals exposed to early-life stress through both juvenile and adult stages [2]. Some evidence that behavioral effects can be delayed comes from zebra finches, for which postnatal CORT treatment results in a trend toward increased neophobic behavior with age [26] (although that trend was not significant). Stronger support for delayed programming effects is described in laboratory rodents. Adult, but not juvenile laboratory mice (*Mus musculus*) exposed to early-life stress in the form of postnatal maternal separation or fragmented maternal care exhibit later impairment of neural synaptic plasticity [80,81], and deterioration of complex cognitive behavior [81]. Our results support these previous studies by suggesting that programming effects of early-life stress may not appear until relatively late in life.

Our results for propensity to escape reveal no persistent effects of early-life stress and so do not support either the inoculation or adaptive calibration models, both of which require a long-term response to early-life stress [6,12]. Individuals were also highly variable in their responses to escape testing, indicating a potential problem with our testing scheme. This lack of consistency may have been a statistical artifact due to the small number of measurements/individual and binary nature of the outcome variable that resulted in low population variance on which estimates of repeatability depend. Alternatively, escape behavior may be linked to a highly labile state variable which rapidly changes (*e.g.*, immediate energy reserves, blood pressure) [69,85]. Individuals also may have exhibited high variability in escape response to confuse the “predator” (experimenter) waiting outside the arena. Or, our testing arena may not have replicated a natural situation well enough to allow for consistent decision-making between hiding/freezing and escape, both natural responses to dangerous situations [32]. Regardless of the cause of the lack of individual consistency, our results indicate that post-natal increases in CORT have no effect on the propensity of individuals to escape from a risky environment with predator cues, although we caution that our measure of escape behavior may not be a reliable measure of the overall strategy that individuals use to cope with danger.

We report no evidence for adaptive tuning in this study, however, an alternative explanation is that glucocorticoid exposure, alone, may not have provided context in which adaptive phenotypic adjustments could be made, or that captivity was not an adequate setting for measurement of adaptive responses. While captivity is a potentially stressful environment for wild house sparrows due to limited flight range and proximity to humans, the stresses of captivity may differ from those of the wild obscuring our ability to detect adaptive changes. We also provided no early-life exogenous clues of predation threat, only increased circulating [CORT], in an attempt to isolate the proximate signal of behavioral programming. Birds may have adaptively adjusted to other stressors not tested here, such as food stress or temperature stress and evaluation of this possibility is a logical next step for this research. Humans may also have inadequately simulated a predator, especially if birds in our study had habituated (*i.e.,* decreased in response intensity) [86] to human presence. However, we found no empirical or observational evidence of habituation. Captive birds consistently responded to human presence with increased vigilance and activity, and always attempted to avoid capture. Juvenile evasiveness also increased with capture experience in our study, suggesting that birds may have learned about the test but had not habituated to human presence. Thus, in regards to antipredator behavior, our study suggests that glucocorticoid upregulation alone is not sufficient to induce adaptive behavioral changes, and environmental cues in tandem with hormonal signals may be necessary for adaptive tuning.

In summary, we showed that early-life exposure to repeatedly elevated circulating glucocorticoids is sufficient to induce long-term effects on the ability to avoid capture during a direct predator encounter as an adult. These findings provide rare evidence of delayed behavioral effects of early-life stress in a wild species and highlight the importance of state-behavior interactions [6,69] in modulating these

effects. Notably, we observed effects at the adult, but not juvenile stage, suggesting that long-term measurements beyond the juvenile and early-adult stages may be necessary for detection of programming effects in future studies.

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Appendix A. Supplementary data

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