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Experimental effects of early-life corticosterone on the hypothalamic–pituitary–adrenal axis and pre-migratory behaviour in a wild songbird

Jesse J. Pakkala[1], D. Ryan Norris[1], James S. Sedinger[2] and Amy E. M. Newman*[,1]

[1]*Department of Integrative Biology, University of Guelph, Guelph, Ontario, N1G 1E4 Canada; and* [2]*Department of Natural Resources and Environmental Science, University of Nevada, Reno, Nevada 89512, USA*

Summary

1. Although laboratory studies have shown that chronic exposure to elevated glucocorticoids during development can have profound effects on the physiology and behaviour of animals, we still have a poor understanding of the proximate and ultimate consequences of early life stress on individuals in the wild.

2. In an island population of Savannah sparrows (*Passerculus sandwichensis*), we examined multiple hypotheses to explain how elevated glucocorticoid exposure during the nestling period influenced both hypothalamic–pituitary–adrenal (HPA) axis function during the nestling period and the subsequent movement and survival of young after they fledged the nest.

3. We exposed nestlings to exogenous corticosterone from 2 to 6 days of age and then measured both baseline and stress-induced (30-min restraint) plasma corticosterone levels prior to fledging (day 7). We then recaptured young throughout the pre-migratory period and used mark–recapture analysis to estimate temporary emigration from the capture site (movement) and survival.

4. Corticosterone-treated nestlings had higher baseline corticosterone levels and lower stress reactivity than untreated individuals, and were more sensitive to inclement weather. Although there was no evidence that corticosterone treatments influenced survival, treated individuals had higher rates of temporary emigration outside of the study site than sham or controls.

5. Our results provide support for the *ceiling hypothesis*, which suggests that individuals with chronic elevated glucocorticoids can lead to a dampened HPA axis response. We also provide support for the *CORT-activity hypothesis*, which suggests that elevated glucocorticoids can increase activity levels, at least 1–2 months after leaving the nest. Our study highlights the importance of tracking individuals across multiple stages of the annual cycle to understand how early life events carry over to influence both physiology and behaviour.

Key-words: Savannah sparrow, annual cycle, carry-over effects, mark–recapture, *Passerculus sandwichensis*, robust design, stress

Introduction

Early life stress, and exposure to elevated glucocorticoids, can have profound long-term effects on the physiology, behaviour and fitness of vertebrates (Meylan & Clobert 2005; Saino *et al.* 2005; Eriksen *et al.* 2006; Blas *et al.* 2007). However, understanding the mechanisms that link the early life environment with individual fitness requires both the ability to follow individuals from birth to later life stages and knowledge of the proximate consequences of exposure to early life stress. One physiological mechanism that links vertebrates to their environment is the hypothalamic–pituitary–adrenal (HPA) axis. Early life conditions influence developmental programming of the HPA axis (Matthews 2002; Seckl 2004), and exposure to early life stressors can disrupt HPA axis regulation, resulting in elevated basal glucocorticoids (Kitaysky *et al.* 2005; Walker, Wingfield & Boersma 2005; Quillfeldt *et al.* 2007; Rensel, Boughton & Schoech 2010) and increased magnitude of the glucocorticoid response to acute stressors (Pravosudov & Kitaysky 2006; Vieau *et al.* 2007; reviewed in Schoech, Rensel & Heiss 2011). For example, in western scrub jays (*Aphelocoma californica*), post-natal food

*Correspondence author. E-mail: newman01@uoguelph.ca

restriction elevated baseline levels of corticosterone in nestlings and exaggerated subsequent responses to acute stress in adults (Pravosudov & Kitaysky 2006). Such effects are known to persist beyond the early life environment across several taxa (mammals: Meaney *et al.* 1991; Liu *et al.* 1997; Meerlo *et al.* 1999; Pryce *et al.* 2005; Navarrete *et al.* 2007; birds: Spencer & Verhulst 2007, 2008; Vieau *et al.* 2007; amphibians: Hu, Crespi & Denver 2008; Spencer, Evans & Monaghan 2009; Marasco *et al.* 2012; Schmidt, MacDougall-Shackelton & MacDougall-Shackelton 2012; Schmidt *et al.* 2014; Drummond & Ancona 2015). Nonetheless, to our knowledge, no studies have examined the direct effects of chronic early life stress on both HPA axis function and how these effects carry over to influence subsequent components of fitness, such as survival and dispersal, in the wild.

In this study, we examine multiple proximate and ultimate hypotheses related to HPA axis function in a free-living population of Savannah sparrows (*Passerculus sandwichensis*). At the proximate level, we examined two competing hypotheses. Firstly, in terms of endocrine regulation, several studies suggest that exposure to chronic early life stress leads to both elevated baseline (Kitaysky *et al.* 2005; Walker, Wingfield & Boersma 2005; Quillfeldt *et al.* 2007; Rensel, Boughton & Schoech 2010) and acute stress-induced corticosterone levels (Pravosudov & Kitaysky 2006; Vieau *et al.* 2007; Spencer, Evans & Monaghan 2009) due to impaired negative feedback of the HPA axis: the '*hyper-reactivity hypothesis*'. However, a recent study suggests that early-life corticosterone exposure in birds can lead to higher baseline corticosterone levels, but a lower response to an ACTH challenge (Kriengwatana *et al.* 2014), which implies unaltered or even enhanced HPA axis negative feedback, or a maximum capacity of the adrenals to produce glucocorticoids. We have termed this competing explanation the '*ceiling hypothesis*', which predicts that chronic early life stress increases baseline corticosterone but, due to an upper 'ceiling' on glucocorticoid production, does not magnify acute stress-induced levels.

At the ultimate level, two potential, non-mutually exclusive, hypotheses attempt to explain the consequences of chronic early life stress in relation to individual success. The first, known as the '*CORT-fitness hypothesis*' (see Bonier *et al.* 2009a,b), proposes that individuals with elevated baseline and stress-induced corticosterone levels will have lower survival due to the deleterious effects associated with chronic HPA axis activation (Wingfield *et al.* 1998; Sapolsky, Romero & Munck 2000; Zera & Harshman 2001). For example, higher corticosterone levels were negatively correlated with the annual survival of free-living Galapagos marine iguanas (*Amblyrhynchus cristatu*; Romero & Wikelski 2001), European white storks (*Ciconia ciconia*; Blas *et al.* 2007) and song sparrows in some years (*Melospiza melodia*; MacDougall-Shackleton *et al.* 2009, 2013). Another hypothesis is that elevated baseline corticosterone levels could increase movement due to the physiological and behavioural changes associated with HPA axis activation, known as the '*CORT-activity hypothesis*' (Breuner & Hahn 2003; Rivers *et al.* 2012). An increased degree of movement could also serve to enhance survival prospects by increasing anti-predator and locomotory functions (Astheimer, Buttemer & Wingfield 1992; Belthoff & Dufty 1998; Breuner & Hahn 2003). This was recently supported in a wild population of migratory Swainson's thrush (*Catharus ustulatus*), where juveniles with higher baseline corticosterone as nestlings had greater subsequent survivorship throughout the post-fledging period (Rivers *et al.* 2012).

To examine these hypotheses, we experimentally increased corticosterone exposure in nestling Savannah sparrows during development (June/July) and recaptured birds throughout the pre-migratory period (August/September). To address proximate hypotheses, we measured their HPA reactivity just prior to leaving the nest and then, to address ultimate hypotheses, we estimated juvenile survival and movement (temporary emigration from the study site) using a robust-design mark–recapture protocol. Following the '*hyper-reactivity hypothesis*', we predicted that corticosterone-treated individuals would have increased baseline and acute stress-induced corticosterone levels compared to the sham group, but the net increase in corticosterone during stress exposure of both groups would be similar (Fig. 1a). Following the '*ceiling hypothesis*', we predicted that corticosterone-treated nestlings would have increased baseline, but similar acute stress-induced corticosterone levels compared to the sham group, and as such, the stress reactivity of corticosterone-treated individuals would be lower than sham individuals (Fig. 1b). At the ultimate level, following the '*CORT-fitness hypothesis*', we predicted that treated individuals would have lower survival during the pre-migratory period than the sham group. Following the '*CORT-activity hypothesis*', we predicted that treated individuals would have higher rates of movement, inferred from estimates of temporary emigration outside of the capture area. Understanding the proximate and ultimate consequences of post-natal glucocorticoids in a natural environment is paramount to predicting long-term effects of stress on individuals and populations.

Materials and methods

STUDY SITE AND SPECIES

From 27 May to 12 September 2013, we studied a marked population of migratory Savannah sparrows on Kent Island, New Brunswick (44°35′N, 66°45′W), an isolated 100-ha island in the Bay of Fundy. The main study area is comprised of two open fields (i.e. north field: 1·5 ha, south field: 6 ha) in the centre of the island, and each field is delineated by 50 × 50 m quadrats (Fig. 2; Wheelwright & Mauck 1998). We observed all breeding adults (n = 76 pairs) within the main study area, and used mist nets to capture any new individuals that had not previously been marked. All

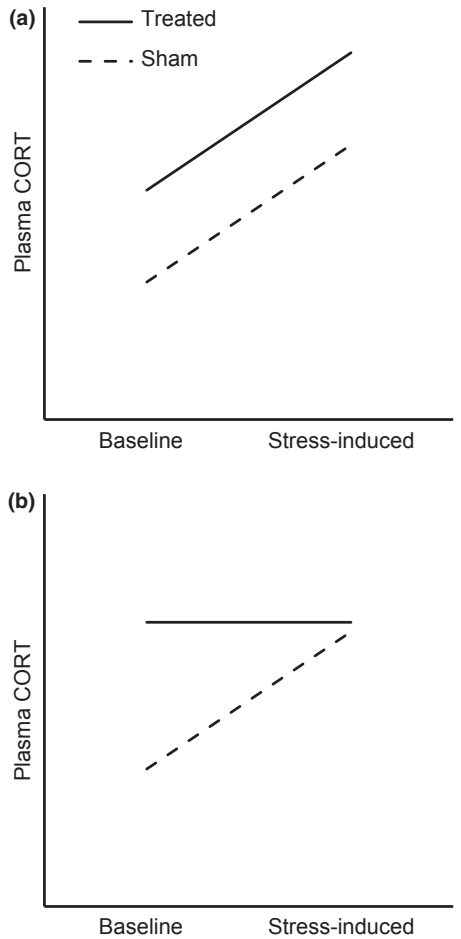

Fig. 1. The predicted effects of early life stress on the HPA reactivity of nestlings. (a) Following the '*hyper-reactivity hypothesis*', individuals treated daily with small doses of corticosterone (CORT) dissolved in peanut oil (treatment) should have higher baseline CORT and stress-induced CORT (acute restraint or sampling 30 min after capture) levels compared to birds that there given peanut oil alone (sham), but both groups should have a similar change in CORT between baseline and stress-induced samples. (b) Following the '*ceiling hypothesis*', treated individuals should have higher baseline CORT values compared to sham birds but similar stress-induced CORT values. As a result, treated individuals would have a much lower change in CORT between baseline and stress-induced samples.

adults (> 1 year of age) were marked using a unique combination of three plastic colour leg bands and a single aluminium leg band (2 bands on each leg). All nests were located on all breeding territories from 27 May to 17 July (n = 109 nests) by observing females during the nest building and incubation stages. Nests were left undisturbed (i.e. not visited from days 2 to 6 post-hatching) throughout development (except for nests within the experimental study site, see below) until nestlings were banded at 7 days of age. All nestlings were marked using a single plastic colour leg band and an aluminium leg band on the opposite leg, and mass (g) and tarsus length (mm) were also recorded. Blood samples were also collected on day 7 to measure plasma corticosterone (see below for details). Nestlings typically fledge the nest at 9 days (range: 9–11 days; Wheelwright & Rising 2008). Fledge dates were recorded by visiting the nest only once after day 9 to reduce the risk of premature fledging, and confirmed by observing parents feeding young out of the nest.

EXPERIMENTAL TREATMENTS

To assess the effect of experimentally elevated corticosterone levels on birds as nestling and later as juveniles, we selected a smaller experimental study site (approx. 250 × 150 m; hereafter 'study site') within the main south field study area (Fig. 2a). Nests on all territories within the study site (n = 42 nests) were located throughout the breeding period (as above). However, only 15 of these nests successfully fledged nestlings due to high rates of nest predation by ravens (*Corvus corax*) and American crows (*Corvus brachyrhynchos*; 31% nest predation within study site in 2013, see Dixon 1972) and also inclement weather conditions (J.J. Pakkala, D.R. Norris, A.E.M. Newman, personal observations). To restrict nest predation, we placed predator exclosures (triangular frames fitted with 2″ chicken wire: 0·75 × 1·0 × 1·0 m) over newly located nests that were deemed to be at a high risk of predation (i.e. most nests > 5 m from the shoreline; n = 12 nests fitted with exclosures). Predator exclosures protected nests until the nestlings successfully fledged, and were monitored at a distance to ensure parental provisioning was not affected by the exclosure.

From 7 June to 18 July, we experimentally manipulated nestling corticosterone levels to establish a causal link between elevated early-life corticosterone and HPA axis function in free-living songbirds. In total, 41 nestlings from 15 broods (Fig. 2b) within the study site were used during the corticosterone experiments. At 2 days of age, nestlings within each brood were weighed and randomly assigned to one of two treatment groups (treated or sham). Due to two severe storms, several broods drowned before day 7; thus, final sample sizes were as follows: treated (n = 14 individuals) or sham (n = 19 individuals). If a brood consisted of four or more nestlings, the individual with the lowest mass was not included in the experiment due to low chances of survival (D.R. Norris, A.E.M. Newman, personal observations). Following Schmidt *et al.* (2014), treated individuals were given an oral dose of exogenous corticosterone dissolved in peanut oil (0·87 μg g^{-1} of body mass) twice daily at the nest from days 2 to 6 post-hatching (Fig. 3a), which included once in the morning (0900–1130 h AST) and once in the afternoon (1500–1730 h AST). At the same time, sham individuals within each brood were fed the equivalent volume of plain peanut oil. Micropipettes (200 μl) were used to administer the peanut oil solutions to each group, and allowed us to accurately increase the volume daily across the treatment period in relation to the daily growth profiles of the nestlings (N.T. Wheelwright, unpublished data). Plasma corticosterone was measured in blood collected from all nestlings within the study site on days 7 post-hatching, and each individual was marked, weighed and tarsus length was measured.

To control for observer presence at experimental treatment nests and to increase statistical power within robust design models (see below), we included a third group of individuals from successful nests in all territories outside, but within 100 m north, of the study site in south field (n = 52 nestlings from 14 broods). These 'control (undisturbed)' nests were located during the breeding season (as above), but nestlings were not visited from days 2 to 6 post-hatching. Control (undisturbed) individuals were marked at 7 days (as above), but plasma corticosterone levels were not measured in this group of nestlings.

BLOOD COLLECTION AND CORTICOSTERONE ASSAY

Following experimental treatments, we collected blood samples (~75 μL) from nestlings on days 7 post-hatch to determine plasma corticosterone concentrations. Firstly, a baseline blood sample (~35–40 μL) was taken from each nestling within 3 min of approaching the nest (see Romero & Reed 2005). Blood was extracted via brachial venipuncture using a 26-gauge needle and quickly drawn into heparinized microhematocrit capillary tubes

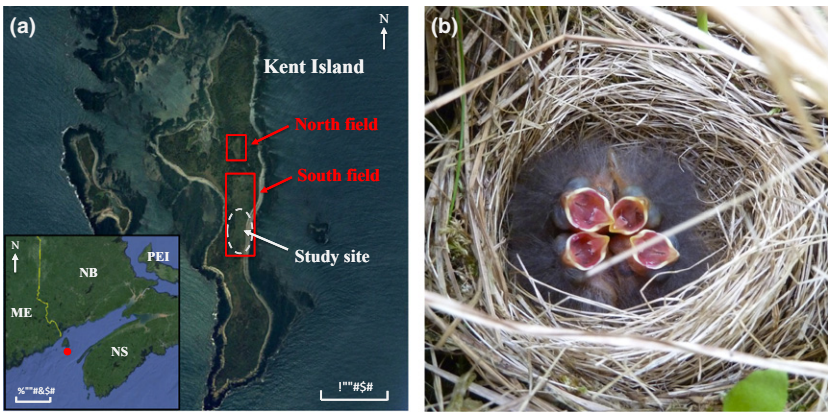

Fig. 2. (a) Map displaying the *Three Islands* archipelago with the study site (white dashed line) and the main study area (red solid lines) located on Kent Island, New Brunswick, Canada (44°35′N, 66°45′W). (Inset) Shows the location of Kent Island (red dot) within the Bay of Fundy in relation to Maine, USA (ME), and New Brunswick (NB), Nova Scotia (NS) and Prince Edward Island (PEI), Canada. (b) Nestling Savannah sparrows at approximately 3–4 days of age on Kent Island, NB (Photo: A.E.M.N.). Maps were created with Google Earth 2014.

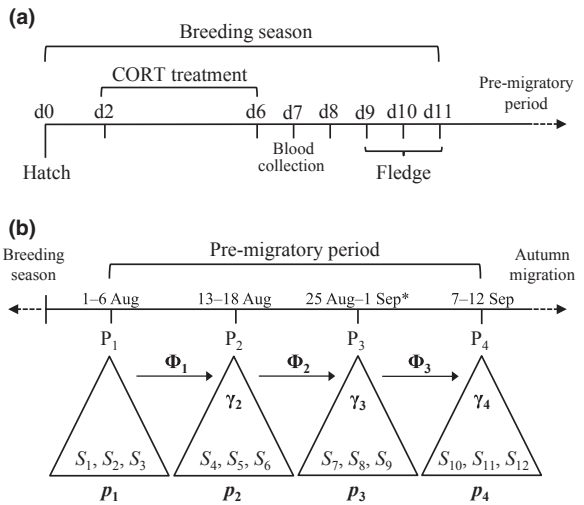

Fig. 3. Sampling (a) and recapture (b) timeline used to estimate the effects of early life stress on the HPA reactivity of nestlings ('blood sampling' in a) and on subsequent juvenile fitness components during the pre-migratory period (b) for first-year Savannah sparrows on Kent Island, NB. (a) For experimental treatments throughout the breeding season, nestlings were fed daily small doses of corticosterone (CORT) dissolved in peanut oil (treated) or peanut oil only (sham) from days 2 to 6 post-hatching. Blood samples were collected on d 7 post-hatching to measure plasma CORT prior to fledging (days 9–11). (b) We used Pollock's robust design to test the effects of treatment (as above) on apparent survival (Φ_i), emigration from the study site (γ_i) and detection (p_i) probabilities of juveniles during the pre-migratory period. γ_i is simplified notation for emigration probability ($\gamma_i'' = \gamma_i'$) within the classical robust design (see Kendall, Pollock & Brownie 1995; Kendall, Nichols & Hines 1997). 'P_i' represents a 6-day primary sampling period with the corresponding date range labelled above, while 'S_j' denotes the 2-day secondary sampling occasions within each primary period.

(Hoysak & Weatherhead 1991), after which, blood flow was staunched by gently applying pressure with a sterile piece of cotton. Nestlings were then immediately placed inside a warm cloth bag and restrained for 30 min to elicit a stress-induced HPA axis response (see Wingfield, Vleck & Moore 1992; Wingfield *et al.* 1994). A second blood sample (~35–40 μL) was collected after 30 min of acute restraint using the same methods described above. On day 7, nestlings weigh 14–16 g; thus, blood volumes were approximately one-half of what is recommended given body size (Sheldon *et al.* 2008). Blood samples were kept on ice in the field and centrifuged within 1–4 h after collection to separate the plasma from red blood cells. Plasma samples were stored at −20 °C until analysis.

Plasma corticosterone concentrations were quantified using a radioimmunoassay (ImmuChem 07-12013; MP Biomedicals, Orangeburg, NY, USA) modified for avian plasma (Washburn *et al.* 2002; Newman, Pradhan & Soma 2008). All samples were measured in duplicate in one assay and the manufacturer's instructions were followed, except that the volumes of all reagents were halved and plasma was diluted to 1:16 (16 μL plasma + 234 μL assay buffer). The minimum and maximum detection limits were 3·1 and 250 pg, respectively, and intra-assay variation was 2·9%.

FIELD PROTOCOL DURING THE PRE-MIGRATORY PERIOD

To determine whether the effects of experimentally elevated post-natal corticosterone carry forward to influence subsequent juvenile survival and dispersal during the pre-migratory period (defined as after fledging and prior to departure for fall migration), a mark–recapture protocol was established using a robust design framework (Pollock 1982; Kendall, Pollock & Brownie 1995; Kendall, Nichols & Hines 1997). Sampling effort during the pre-migratory period was divided into four 6-day primary periods (i.e. P_1–P_4) that were separated by 6 days, and each primary period contained three consecutive 2-day secondary periods (i.e. P_1: S_1-S_3; P_2: S_4–S_6… etc.; Fig. 3b). Recaptures from all three groups (treated, sham, control [undisturbed]) occurred during the primary periods from 1 to 6 August and 13 to 18 August; 25 August to 1 September (28–29 August were omitted due to poor weather); and 7–12 September, respectively (Fig. 3b).

To recapture fully flighted juveniles, we flushed juveniles from the surrounding field into a line of mist nets 162 m long (as in Mitchell *et al.* 2011, 2012). Because these are grassland birds that are moulting and preparing for migration, they tend to stay very low within the grass and are not frequently sitting high on perches or flying at this time of the year. Thus, flushing them is an extremely effective method for capturing both adults and young of the year, and very few birds are seen to evade the nets once they have been flushed. To cover the entire study site, we defined four separate locations across the study site where we set up the single row of mist nets (i.e. net locations 1–4). Net locations were then set up in both the morning (0730–1130 h AST) and the afternoon (1300–1700 h AST) for the 2 days within each secondary period (e.g. S_1: day 1 = net 1 [morning], net 2 [afternoon], days 2 = net 3 [morning], net 4 [afternoon], etc.). The order in which net locations were used within each secondary period (i.e. days 1 or 2, morning or afternoon) was randomized to eliminate potential sampling bias.

STATISTICAL ANALYSIS: EFFECTS OF TREATMENT ON PLASMA CORTICOSTERONE

We used linear mixed models to examine the effects of experimental exposure to post-natal glucocorticoids on circulating corticosterone levels in nestlings. Plasma corticosterone levels measured at 'baseline' (< 3 min) and after acute restraint stress ('30 min') were the response variables. We included nest identity (hereafter 'nest ID') as a random effect within all models to account for the potential effects of the nest or mothers within the experimental design. Since inclement weather can activate the HPA axis (Wingfield, Moore & Farner 1983), we considered the amount of rainfall (in.), collected over a 24-h period on Kent Island when nestlings were 7 days old (hereafter 'precipitation'), as a fixed effect within additive and interactive models to account for environmental perturbations during development. We also included hatch date (hereafter 'hatch day') as a fixed effect within additive and interactive models to account for the potential effects of seasonality on the plasma corticosterone levels of nestlings. Six linear mixed models were constructed for baseline and 30-min response variables to explain variation in plasma corticosterone concentrations, and compared using Akaike's information criterion model selection corrected for small sample sizes (AIC_c; Burnham & Anderson 2002). The null model contained only an intercept and the random effect (1 + nest ID). The remaining five models all contained treatment (treated, sham) as a fixed effect. A stepwise approach was then used to incorporate hatch day (julian date) and precipitation (rainfall amount on d 7) as fixed effects within additive models, and the interactions between treatment and each explanatory variable (i.e. treatment × hatch day, treatment × precipitation) as fixed effects within interactive models. We also analysed stress reactivity of individuals using a repeated measures ANOVA on baseline and 30-min corticosterone concentrations to estimate the overall capacity of individuals to respond to acute stress (see Schmidt *et al.* 2014). However, since this response variable contained the same corticosterone data as the previous models, we used the fixed effects from the top baseline and 30-min models and added the fixed effect of acute restraint stress to examine variation in plasma corticosterone within individuals.

Analyses were performed in JMP PRO (ver. 11·2, SAS, Cary, NC, USA). Baseline and stress-induced corticosterone were log-transformed to reduce heteroscedasticity. To be consistent with AIC model selection, we used 85% confidence intervals (CI) to assess parameter estimates (Arnold 2010); all *P*-values were two-tailed. Means are presented ± standard error.

MODELLING JUVENILE SURVIVAL AND TEMPORARY EMIGRATION

Robust design models were used to estimate apparent survival (Φ_i), temporary emigration ($\gamma_i'' = \gamma_i'$; hereafter γ_i), and conditional capture (hereafter 'detection'; p_i) and recapture (c_{ij}) probabilities of juvenile Savannah sparrows (Pollock 1982; Kendall, Pollock & Brownie 1995; Kendall, Nichols & Hines 1997; Schwarz & Stobo 1997). Following Pollock (1982) and Kendall, Pollock & Brownie (1995), parameter estimates from robust design models were obtained by using data from both primary and secondary sampling periods within the experimental design (see above; Fig. 3b). To remove unnecessary estimates of population size from the likelihood, we used robust design models with the Huggins' estimator (Huggins 1989, 1991). Our candidate models were chosen *a priori*, and are based on Savannah sparrow biology and well-defined hypotheses (Burnham & Anderson 1998). Model notation follows that of Kendall, Pollock & Brownie (1995), Kendall, Nichols & Hines (1997).

We assumed closure for juveniles from the experimental groups during each secondary period, such that all mortality and temporary emigration occurred outside of the secondary samples (Pollock 1982; Kendall, Pollock & Brownie 1995). This assumption was supported by the fact that juvenile Savannah sparrows are known to remain near their natal territories and are confined to the island throughout the pre-migratory period (Mitchell *et al.* 2011, 2012). Recaptures of marked birds were summarized into encounter history format from fledge day until the end of the pre-migratory period with three groups (treated, sham, control [undisturbed]), and models were structured using design matrices.

We began by fitting a base model with constant survival (Φ.), temporary emigration (γ.), detection (P.), and recapture (c.) probabilities to use for comparisons with other models. We then considered variation in survival across treatment groups ($\Phi_{treatment}$) and over time (Φ_t). However, there was no support for temporal variation in survival (Φ_t), so we did not further consider models including temporal variation in survival. We then explored variation in survival between juveniles from nests within the study site ($\Phi_{treated} = \Phi_{sham}$) and juveniles from outside of the study site ($\Phi_{control\ (undisturbed)}$), and found that survival did not vary between these groups. Therefore, we considered each treatment group separately in our survival analysis ($\Phi_{treated}$, Φ_{sham}, $\Phi_{control\ (undisturbed)}$, i.e. $\Phi_{treatment}$). We also explored temporal variation in temporary emigration (γ_t), detection (P_t) and recapture (c_t) probabilities of juveniles and found no support for these models, so we excluded time as a variable within these models as well.

For the temporary emigration probabilities of juveniles (γ), we were interested in the effects of treatment on pre-migratory movements from the study site. Juvenile Savannah sparrows form loose flocks throughout the pre-migratory period and remain confined to the island, typically near their natal territories, until departing for migration (Wheelwright & Mauck 1998; Mitchell *et al.* 2011, 2012). Therefore, we assumed temporary emigration from the study site could take place during any interval between primary periods (Kendall, Pollock & Brownie 1995; Kendall, Nichols & Hines 1997), and examined whether elevated early-life corticosterone was a predictor of juvenile movement, as estimated by temporary emigration. We first parameterized models that considered each treatment group separately ($\gamma_{treated}$, γ_{sham}, $\gamma_{control\ (undisturbed)}$, i.e. $\gamma_{treatment}$), and then further explored the variation between individuals with elevated early-life CORT levels ($\gamma_{treated}$) and those without experimentally elevated corticosterone ($\gamma_{sham} = \gamma_{control\ (undisturbed)}$).

We modelled the detection and recapture probabilities of juvenile Savannah sparrows using the same structure for both P_{ij} and c_{ij} (e.g. $P_{treated} = P_{sham}$) but allowed recapture probabilities (c_{ij}) to differ from initial capture probabilities (P_{ij}). We also investigated potential differences in detection probability between individuals that fledged within the study site ($P_{treated} = P_{sham}$) vs. individuals from outside of the study site ($P_{control\ (undisturbed)}$).

We parameterized robust design models in program MARK (White & Burnham 1999) and then used Akaike's information criterion adjusted for small sample sizes (AIC_c) to compare competing models (Burnham & Anderson 2002). We considered models within ≤ 2 AIC_c units of the top model as competitive (Burnham & Anderson 2002). If there were multiple competitive models, we used model averaging to derive parameter estimates from these models. Parameter estimates are reported using the mean and, to be consistent with AIC model selection analysis (Arnold 2010), 85% confidence intervals.

Results

PLASMA CORTICOSTERONE LEVELS

For baseline plasma corticosterone levels in nestling Savannah sparrows (mean baseline plasma corticosterone for treated birds: 14·87 ± 2·93 ng mL^{-1}; sham: 11·42 ± 1·51 ng mL^{-1}), the top supported model included the

interaction between treatment and precipitation (AIC_c weight: 0·975; Table 1) and the second highest supported model was the null model with a ΔAIC_c of 7·60. In the top model, the significant interaction between treatment and precipitation (Table 2) suggested that corticosterone-treated nestlings had higher baseline plasma corticosterone levels during periods of higher precipitation compared to sham nestlings. There was a trend for higher baseline in treated nestlings, but the difference between treatment and control was not significant for this main effect ($P = 0·09$). Models that included treatment alone, or the additive effects of precipitation and hatch day with treatment and the interaction between treatment and hatch day had low support (AIC_c weight < 0·05; Table 1).

Similar to the baseline model, the top supported model for restraint stress-induced (30 min) corticosterone levels included the interaction between treatment and precipitation (AIC_c weight: 0·674; Table 1) and the second highest supported model was the null model with a ΔAIC_c of 2·65. Similar to the baseline model, there was very weak evidence for an effect of treatment in the top model (mean 30-min plasma corticosterone for treated birds: 14·34 ± 2·81 ng mL^{-1}, for sham birds: 17·26 ± 2·58 ng mL^{-1}), as 85% CI included zero and the *P*-value was 0·13 (Table 2). Similarly, there was only weak support for an effect of precipitation and the interaction between treatment and precipitation: 85% CI did not overlap with zero, but *P*-values were ≥ 0·10 (Table 2).

For the stress reactivity of nestlings, repeated measures ANOVA revealed that there was a significant interaction between treatment and acute restraint stress, ($F_{1,69} = 4·69$, $P = 0·035$; Table 2; Fig. 4), and post-hoc tests indicated an elevation in baseline but not 30-min levels for treated individuals and thus a lower overall response to a 30-min restraint stress than the sham group (Δ CORT (30 min – baseline plasma corticosterone): treated: −3·38 ± 3·02 ng mL^{-1}, sham: 5·54 ± 2·59 ng mL^{-1}). Furthermore, the mixed-model results on baseline and 30-min plasma corticosterone, and analysis of within individual responses, revealed a significant interaction between treatment and precipitation, suggesting that during rain storms, treated individuals had higher circulating corticosterone (Table 2) and this was due to elevated baseline corticosterone in treated individuals. Worth noting, for nestlings on day 7, there was no effect of corticosterone treatment on body mass (corticosterone treated: 14·09 ± 0·65 g; sham: 13·41 ± 0·59 g, $t = -0·80$, $P = 0·43$) or tarsus length (corticosterone treated: 18·36 ± 0·42 mm; sham: 18·22 ± 0·39 mm, $t = -0·24$, $P = 0·81$).

SURVIVAL, TEMPORARY EMIGRATION AND DETECTION PROBABILITY

A model in which initial capture probabilities and recapture probabilities differed between the study site and control area performed substantially better than a model that constrained initial capture and recapture probabilities to be equal between areas ($\Delta AIC_c > 7$, Table 3). Thus, we incorporated this structure for capture probabilities in subsequent models. The two models within ≤ 2 AIC_c units (AIC_c weight > 0·07; Table 3) were model averaged to estimate parameters for juvenile survival across each treatment group (treated, sham, control [undisturbed]). Models with the most support (AIC_c weight > 0·08; Table 3) included constant survival (Φ.) across primary periods, which suggested that juvenile pre-migratory survival was not influenced by treatment (Fig. 5a). Individuals experimentally treated with early-life corticosterone had a higher

Table 1. Results of model selection for competing linear mixed models used to explain the effects of treatment (treated, sham), precipitation (rainfall amount on day 7) and hatch day (julian date) on (A) baseline and (B) 30-min corticosterone concentrations for nestling Savannah sparrows during the 2013 breeding season on Kent Island, NB. Response variables are associated with a distinct set of models (A: 1–6, B: 1–6). 'Nest ID' was included as a random effect in all models, including the null model

Model	d.f.	*AIC_c	ΔAIC_c	W_i	Deviance
(A) Baseline corticosterone					
1. Treatment × precipitation	6	8·9	0·00	0·975	−6·11
2. Null model	3	16·50	7·60	0·022	9·73
3. Treatment + precipitation	5	20·86	11·96	0·002	8·79
4. Treatment	4	22·77	13·87	0·001	13·44
5. Treatment + hatch day	5	33·46	24·56	0·000	21·39
6. Treatment × hatch day	6	44·83	35·93	0·000	29·83
(B) 30-min corticosterone					
1. Treatment × precipitation	6	24·99	0·00	0·674	9·99
2. Null model	3	27·64	2·65	0·179	20·87
3. Treatment + precipitation	5	28·28	3·29	0·130	16·21
4. Treatment	4	32·30	7·31	0·017	22·96
5. Treatment + hatch day	5	43·26	18·27	0·000	31·19
6. Treatment × hatch day	6	54·18	29·19	0·000	39·18

d.f., degrees of freedom; AIC_c, Akaike's information criterion corrected for small sample sizes; ΔAIC_c, model difference relative to the model with the lowest AIC_c score; W_i, Akaike weight, or the probability in favour of the given model relative to all models considered; Deviance, −2log(L) for each model considered.

Table 2. Results from the top linear mixed models (see Table 1) used to explain the effects of treatment (treated, sham) and precipitation (rainfall amount day 7) on (A) baseline corticosterone, and (B) stress-induced corticosterone for nestling Savannah sparrows. (C) Repeated measures model for plasma corticosterone using the same predictor variables as the top models for baseline and 30-min corticosterone and adding the effect of 30-min acute restraint stress (baseline or 30 min) within an individual. 'Nest ID' was included as a random effect in all models

Model	Estimate	85% CI	*t*	*P*
(A) Baseline corticosterone				
Intercept	1·00	0·85, 1·15	10·38	< 0·0001
Treatment	0·05	0·01, 0·10	1·74	0·09
Precipitation	−0·46	−6·62, 5·70	−0·12	0·91
Treatment × precipitation	5·67	3·63, 7·71	4·16	0·0005
(B) Stress-induced corticosterone				
Intercept	0·99	0·86, 1·12	11·58	< 0·0001
Treatment	−0·07	−0·13, 0·00	−1·56	0·13
Precipitation	6·19	0·70, 11·67	1·76	0·11
Treatment × precipitation	3·69	0·53, 6·86	1·75	0·10
(C) Within individual corticosterone				
Intercept	0·99	0·88, 1·12	12·63	0·0001
Treatment	−0·003	−0·05, 0·04	−0·09	0·93
Precipitation	2·81	−2·27, 7·89	0·86	0·41
Acute restraint	−0·03	−0·07, 0·03	−0·84	0·41
Treatment × Precipitation	4·79	2·68, 6·90	3·41	0·003
Treatment × Acute restraint	0·06	0·02, 0·11	2·08	0·035

CI, 85% confidence intervals around the parameter estimates; *t*, *t* test statistic for each parameter estimate; *P*, the *P* value associated with *t*.

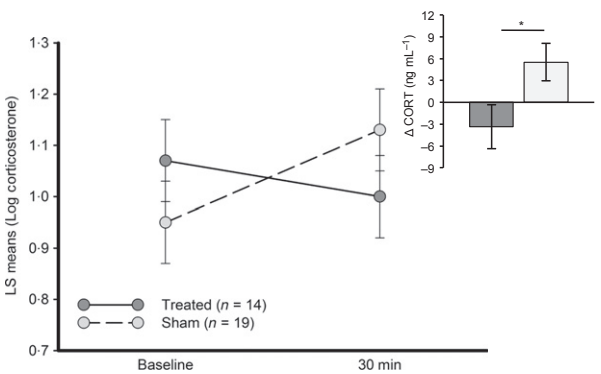

Fig. 4. Least square (LS) means for the effects of early-life corticosterone treatment on the HPA reactivity of nestling Savannah sparrows. Plasma corticosterone concentrations were compared between nestlings that received small daily doses of corticosterone (CORT) dissolved in peanut oil (treated) and nestlings that were treated with peanut oil alone (sham). Treated individuals exhibited a reduced response to 30-min acute restraint (see Table 2). Inset: for illustrative purposes, Δ CORT (30 min – baseline) for treated and sham individuals (*$t = -2{\cdot}59$, $P = 0{\cdot}02$). Sample sizes for each group are indicated in parentheses.

probability of temporary emigration from the capture area between primary sampling intervals compared to the sham or control (undisturbed) birds (Fig. 5b). Juveniles from nests within the study site (treatment and sham) had a higher detection probability within primary sampling periods than individuals from nests outside of the study site (Fig. 5c). Finally, there was no evidence that recapture (c_i) probabilities varied across secondary samples within each primary period (treated: CI = 0·12, 0·30, $c_{1–12} = 0{\cdot}20 \pm 0{\cdot}044$; sham: CI = 0·14, 0·30, $c_{1–12} = 0{\cdot}21 \pm 0{\cdot}042$; control [undisturbed]: CI = 0·04, 0·13, $c_{1–12} = 0{\cdot}071 \pm 0{\cdot}023$).

Table 3. Results of model selection for survival (Φ), temporary emigration (γ), detection (*P*) and recapture (*c*) probabilities with respect to treatment (treated, sham, control [undisturbed]) for juvenile Savannah sparrows during the 2013 pre-migratory period on Kent Island, NB. '.' represents a constant (intercept-only) model. 'β' refers to the beta parameter structure for each treatment group within a model

Model	*K*	ΔAIC_c	W_i	Deviance
1. Φ.	6	0·00	0·583	593·56
$\gamma_{treatment}$: $\beta_{sham} = \beta_{control(undisturbed)}$				
$p_{treatment}$: $\beta_{treated} = \beta_{sham}$				
$c_{treatment}$: $\beta_{treated} = \beta_{sham}$				
2. Φ.	7	1·91	0·224	593·30
$\gamma_{treatment}$: $\beta_{sham} = \beta_{control(undisturbed)}$				
$p_{treatment}$				
$c_{treatment}$				
3. Φ.	8	3·97	0·080	593·14
$\gamma_{treatment}$				
$p_{treatment}$				
$c_{treatment}$				
4. $\Phi_{treatment}$	8	4·17	0·073	593·34
$\gamma_{treatment}$: $\beta_{sham} = \beta_{control(undisturbed)}$				
$p_{treatment}$: $\beta_{treated} = \beta_{sham}$				
$c_{treatment}$				
5. Φ., γ.,	5	5·44	0·038	601·15
$p_{treatment}$: $\beta_{treated} = \beta_{sham}$				
$c_{treatment}$				
6. Φ., γ., *P*., *c*.	4	12·49	0·001	610·33

K, number of β parameters; ΔAIC_c, model difference relative to the model with the lowest AIC_c (Akaike's information criterion corrected for small sample sizes) score; W_i, Akaike weight, or the approximate probability in favour of the given model relative to all models considered; Deviance, −2log(L) of each model considered.

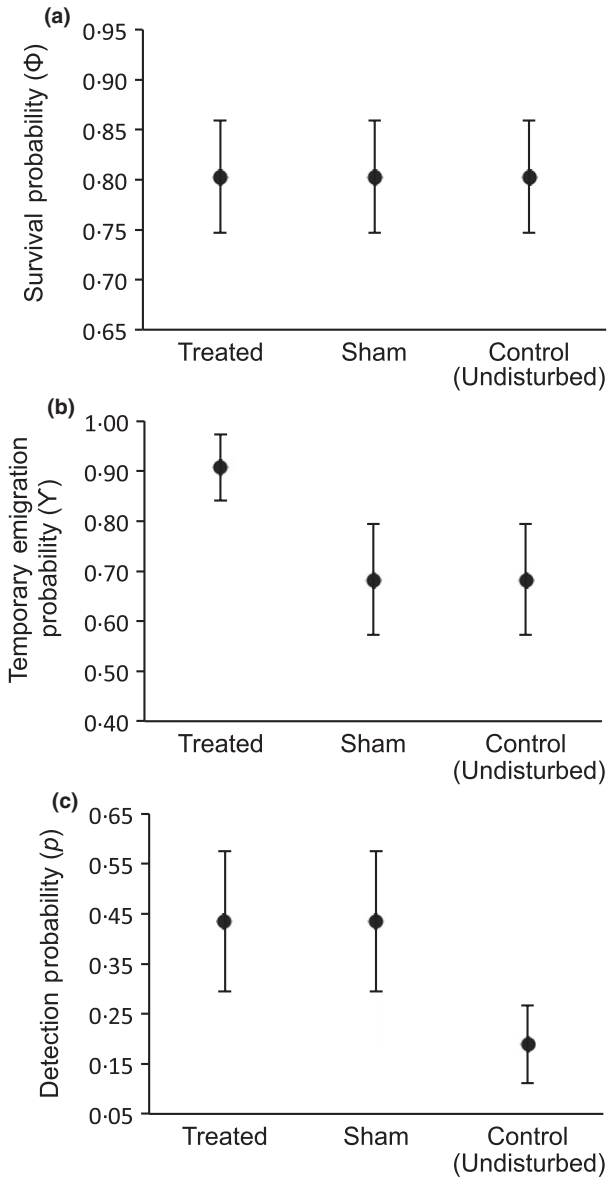

Fig. 5. The effects of early life stress on the (a) apparent survival probability (Φ), (b) probability of detection (*P*) and (c) probability of emigrating from the study site (γ) of juvenile Savannah sparrows during the pre-migratory period. Nestlings within the study site were given either daily small doses of corticosterone (CORT) dissolved in peanut oil (treatment) or peanut oil without CORT (sham). Nestlings raised in broods outside but within 100 m of the study site were not treated and these nests were not visited during the nesting period (control [undisturbed]). We then attempted to recapture juveniles throughout the pre-migratory period (1 August–12 September) using a field protocol based on a robust design framework to examine the variation in survival (Φ), temporary emigration (γ), detection (*P*) and recapture (*c*; not shown in figure) probabilities. Data points are weighted model averaged estimates (± 85% CI) from Pollock's robust design models (see Table 3).

Discussion

To our knowledge, we provide the first experimental evidence for the effects of exogenous glucocorticoids on both early-life HPA axis function and how this effect carries over to influence juvenile pre-migratory movements in a wild population of birds. In doing so, we provide some support for both the *ceiling hypothesis* that attempts to explain the reactivity of the HPA axis at the proximate level and the *CORT-activity hypothesis* that attempts to explain the ultimate consequences of chronically elevated corticosterone levels. We discuss these findings, particularly in the context of examining the effects of early life stress in the wild.

The HPA axis is a fundamental physiological mechanism connecting an organism to its environment and is sensitive to stress and glucocorticoid exposure during development (reviewed in Schoech, Rensel & Heiss 2011; Crespi *et al.* 2013). We found that early corticosterone treatment during days 2–6 post-hatch altered nestling HPA axis function and sensitivity in several important ways. Consistent with the '*ceiling hypothesis*', our results with respect to acute restraint exposure suggest that a standard 30-min acute restraint stress on day 7 increased plasma corticosterone concentrations in sham nestlings but not in corticosterone-treated nestlings (Fig. 4; Table 2). Other studies in captive populations have reported similar findings of a dampened HPA axis response after chronic glucocorticoid treatment (e.g. Eurasian Kestrel nestlings: Müller *et al.* 2009; Zebra finch nestlings: Kriengwatana *et al.* 2014), but our results provide important experimental insight into this effect in a wild population. Chronic corticosterone treatment may down-regulate endogenous adrenal sensitivity due to altered negative feedback, or may increase glucocorticoid metabolism and clearance, as has been suggested for adult song sparrows (*Melospiza melodia*) treated with corticosterone (Newman *et al.* 2010; B. Robertson, A. Newman, S. MacDougall-Shackleton, unpublished data). Future experimental work on glucocorticoid receptor expression in the brain and glucocorticoid metabolite levels in faeces will help to tease apart the mechanism leading to lower plasma corticosterone concentrations after chronic treatment and HPA axis stimulation with acute restraint.

Interestingly, while we found little evidence that a standard 30-min 'acute restraint stress' elevated plasma corticosterone in corticosterone-treated nestlings, our results suggest that corticosterone treatment sensitized nestlings to inclement weather. The amount of rainfall on day 7 post-hatch increased plasma corticosterone levels in treated but not in sham nestlings. Early corticosterone treatment may program HPA axis hypersensitivity to environmental perturbations, which could be an adaptive proximate response to cope with unpredictable conditions. This would contradict the proposed 'silver spoon effect' (Van de Pol *et al.* 2006), which hypothesizes that the optimal early life experience is one of low stress. It remains to be tested if individuals with hypersensitive physiological phenotypes, shaped by early-life corticosterone exposure, have higher lifetime fitness under unpredictable ecological conditions. Nonetheless, our results do add tantalizing support to the '*environmental matching hypothesis*' where individuals pro-

grammed under 'stressful' environments early in life thrive relative to their 'silver spoon' peers under harsh conditions (e.g. unfavourable weather, decreased food availability, increased competition). Further, it has been shown in adult birds that adverse weather activates the HPA axis and alters behaviour to facilitate survival (Wingfield, Moore & Farner 1983; Silverin 1986; Rogers *et al*. 1993; Breuner & Hahn 2003; Boyle, Norris & Guglielmo 2010).

The interaction between early-life glucocorticoid exposure and the subsequent physiological response to rainfall may also depend on the type, timing and duration of the stimulus. In our experimental protocol, repeated handling (2× day during post-hatch days 2–6 for administration of peanut oil) combined with corticosterone treatment could have acclimatized the subsequent 'acute restraint stress' in nestlings on day 7. In contrast, the 'novel' environmental stressor (rainfall) may have revealed an increased sensitivity of corticosterone-treated nestlings. Indeed, avian studies report varying effects on later HPA axis function (attenuated stress response: Rich & Romero 2005; e.g. exaggerated stress response: Pravosudov & Kitaysky 2006; Cyr *et al*. 2007; Lendvai *et al*. 2009; Spencer, Evans & Monaghan 2009; Banerjee *et al*. 2012; Kriengwatana *et al*. 2014; Lattin & Romero 2014; Schmidt *et al*. 2014) and this may depend on how glucocorticoids are experimentally elevated early in life. In addition, the endocrine effect of early life stress may also manifest at different levels of HPA axis function as baseline functions are largely mediated by high-affinity mineralocorticoid receptors, whereas functions related to increased HPA axis activation (i.e. during stress) are mediated by lower affinity glucocorticoid receptors; thus, early-life HPA axis programming may differentially affect expression of these two receptors for corticosterone. Understanding the relationship between endocrine profiles and chronic stress exposure requires further study, as evidenced in an elegant review by Dickens & Romero (2013) and more recently by Drummond & Ancona (2015); both papers highlight the variability around stress exposure and HPA axis dynamics in wild animals.

In addition to providing insight into how the early-life HPA axis responds to chronic and acute stressors, our results provide some support to the notion that early life stress can carry over to subsequent life stages. Corticosterone-treated individuals were more likely to temporarily emigrate from the capture site compared to sham or control birds (Fig. 5b). We interpret higher temporary emigration among treatment juveniles as reflecting an increase in overall movement rate as they move in and out of the smaller capture site (encompasses their natal territories and immediately surrounding territories) embedded within a larger expanse of breeding habitat. Thus, these results are consistent with the '*CORT-activity hypothesis*', suggesting that corticosterone exposure during the nestling period increases locomotory activity when young are independent. Although we did not measure corticosterone concentrations during the pre-migratory period, it is possible that behavioural effects were not directly fuelled by increased plasma corticosterone in juveniles. However, it does not rule out a link between HPA axis programming during early life and 'activity' later in life. Indeed, previous studies on adult birds (i.e. in, at least, their first breeding season) have shown that elevated corticosterone not only promotes locomotory activity (Astheimer, Buttemer & Wingfield 1992; Breuner, Greenberg & Wingfield 1998; Breuner & Hahn 2003), but also foraging rates (Astheimer, Buttemer & Wingfield 1992; Crossin *et al*. 2012), and migratory restlessness (Landys, Ramenofsky & Wingfield 2006). Further, one earlier study on independent juvenile willow tits (Silverin, 1997) suggests that corticosterone implants in late summer increase subsequent disappearance from the study site, which was interpreted as dispersal. Any or all of these specific behavioural responses could have resulted from our experimental treatment. We do have evidence that the date juveniles depart the island for fall migration is partially related to the date that they fledge the nest (Mitchell *et al*. 2012), but early life stress could also play a factor in explaining the timing of migration. Quantifying corticosterone during the pre-migratory phase will yield important results to help explain the long-term effects of early life stress on adult physiology and behaviour.

Although modulation of anti-predator behaviour in the form of increased movement could be important for juvenile songbirds since most mortality is attributed to predation during early life stages (e.g. Dixon 1972; Anders *et al*. 1997; Cohen & Lindell 2004; King *et al*. 2006; Rivers *et al*. 2012), we did not find evidence that corticosterone-treated birds had higher survival rates during the pre-migratory period compared to sham or controls. One explanation may be the lack of power due to the low number of recaptures. However, our parameter estimates showed moderately high precision, suggesting that we likely would have had a high probability of detecting differences had they existed. Another aspect that gave us confidence in the model results was the fact that encounter probabilities were lower in control birds than either sham or corticosterone-treated birds. Unlike the sham birds, our control birds were born just outside of the study site, so we expected these individuals to have lower encounter probabilities. A second explanation for similar survival among groups is that, unlike migration (Sillett & Holmes 2002), the fall pre-migratory period may not be particularly risky for juvenile Savannah sparrows on Kent Island. Although we have occasionally observed small falcons patrolling the fields during the pre-migratory period, there are no predatory small mammals on the island. In this population, there is evidence that nestling mass on day 7 influences first-year survival, as well as fat mass during the pre-migratory period (Mitchell *et al*. 2011), suggesting that juvenile survival may be partly driven by the ability to accumulate sufficient reserves prior to migration. Whether early life stress also influences survival rates during migration or even the stationary non-breeding period remains to be tested. It is extremely challenging to track mortality rates of individual migratory birds across periods of the annual cycle.

Our results also highlight the advantage of using robust-design approach for mark–recapture analysis over the more commonly used Cormak–Jolly–Seber (CJS) approach. The robust design allowed us to separate encounter probabilities into 'availability for encounter' (1-temporary emigration, γ) and 'encounter probability conditioned on availability', whereas encounter probability in a CJS analysis is simply a product of these two probabilities. Models in which we held γ constant (Table 3) or set γ to zero (results not shown) performed poorly compared to models in which we estimated γ, providing strong evidence that there was, indeed, temporary emigration in our sample population. Capturing variation in γ is something we would not have been able to do with a simpler, CJS design.

In conclusion, our work supports the notion that elevated early life stress events can have a significant immediate impact on the HPA axis, as well as carry over to influence movement behaviour once young are independent (Rivers *et al*. 2012). Our results have implications for a wide range of early life stressors experienced in the wild and highlight the importance of tracking individuals across multiple stages of annual cycle to understand how early life events impact individual success (Harrison *et al*. 2011; O'Conner *et al*. 2014).

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Data accessibility

Data are deposited in the Dryad Digital Repository: http://dx.doi.org/10.5061/dryad.8nh60 (Pakkala *et al*. 2015).

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