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Developmental stress increases reproductive success in male zebra finches

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There is increasing evidence that exposure to stress during development can have sustained effects on animal phenotype and performance across life-history stages. For example, developmental stress has been shown to decrease the quality of sexually selected traits (e.g. bird song), and therefore is thought to decrease reproductive success. However, animals exposed to developmental stress may compensate for poor quality sexually selected traits by pursuing alternative reproductive tactics. Here, we examine the effects of developmental stress on adult male reproductive investment and success in the zebra finch (*Taeniopygia guttata*). We tested the hypothesis that males exposed to developmental stress sire fewer offspring through extra-pair copulations (EPCs), but invest more in parental care. To test this hypothesis, we fed nestlings corticosterone (CORT; the dominant avian stress hormone) during the nestling period and measured their adult reproductive success using common garden breeding experiments. We found that nestlings reared by CORT-fed fathers received more parental care compared with nestlings reared by control fathers. Consequently, males fed CORT during development reared nestlings in better condition compared with control males. Contrary to the prediction that developmental stress decreases male reproductive success, we found that CORT-fed males also sired more offspring and were less likely to rear non-genetic offspring compared with control males, and thus had greater overall reproductive success. These data are the first to demonstrate that developmental stress can have a positive effect on fitness via changes in reproductive success and provide support for an adaptive role of developmental stress in shaping animal phenotype.

1. Introduction

The environment animals experience during development can have important effects on phenotype and fitness across life-history stages [1–3]. For example, exposure to stress during development (in the form of elevated glucocorticoid stress hormones or food restriction), has been associated with a range of phenotypic effects across taxonomic groups (reviewed in [4–7]). Developing animals can be exposed to stress indirectly by parental effects (e.g. [8]) or directly when environmental factors such as low food availability, inclement weather or sibling competition stimulate activation of the hypothalamic–pituitary–adrenal axis and the release of glucocorticoid hormones [9–12]. Phenotypic changes induced by developmental stress can be sustained across an animal's life (e.g. [13–15]) and, in this way, could affect fitness at multiple life-history stages.

Although the phenotypic and performance consequences of developmental stress have been well studied (reviewed in [5–7,16]) there are comparatively few studies that have examined the fitness consequences of developmental stress via changes in reproductive success (e.g. [17,18]). Developmental conditions (e.g. enlarged broods, reduced food availability, elevated glucocorticoids) have long-term effects on phenotypic traits such as metabolism, immunocompetence and stress response which could have indirect negative effects on reproductive success (e.g. [13,19,20]). Additionally, developmental stress can reduce the quality of sexually selected traits [21–25] which could decrease reproductive success

via changes in sexual attractiveness. For example, zebra finches (*Taeniopygia guttata*) exposed to either dietary stress or experimentally elevated glucocorticoids during development sing lower quality songs as adults [22,25,26] and are less attractive to females [27]. Cumulatively, these studies suggest that developmental stress can influence adult phenotype in ways that are potentially important for reproductive success. However, no study has directly compared the relative change in reproductive success between control and developmentally stressed males.

A decline in sexually selected trait expression should lead to a decline in paternity, which predicts negative effects on reproductive success (e.g. [28]). However, animals exposed to stress during development may compensate for poor-quality sexually selected traits by pursuing alternative reproductive tactics in order to optimize reproductive success. Alternative reproductive tactics occur when individuals attempt to maximize reproductive success using different behavioural strategies [29]. Individual tactics can be expressed because of the existence of genetic polymorphisms within a population (e.g. [30–32]) or conditionally, based on cues experienced during development (e.g. [33]). In birds with bi-parental care, alternative reproductive tactics could exist if males with poor-quality sexually selected traits maximize their fitness by investing more in parental care (e.g. nestling provisioning). By contrast, males with high-quality sexually selected traits could maximize fitness by pursuing copulations outside of their social mate (i.e. extra-pair copulations; EPCs; [34]) and minimizing costly parental care (e.g. [35,36]). Such tactics have been demonstrated in zebra finches, where unattractive males invest more in parental effort and sire fewer offspring through EPCs compared with attractive males [37]. Likewise, in house finches (*Carpodacus mexicanus*), males with drab plumage provision nestlings more compared with males with elaborate plumage [38]. Adverse developmental conditions (e.g. exposure to stress) could limit the choices available for individual reproductive decisions if certain behaviours lead to unsuccessful reproductive attempts (e.g. pursuing EPCs; [39]). Therefore, by using alternative reproductive tactics, animals exposed to developmental stress could maximize their fitness by pursuing an optimal reproductive tactic for their given phenotype.

We examined the consequences of developmental stress on parental behaviour, bill coloration (a trait important for mate choice [40,41]) and reproductive success in male zebra finches. We elevated endogenous corticosterone (CORT; the dominant avian glucocorticoid) by orally administering CORT dissolved in peanut oil to nestling zebra finches for 16 days during the nestling period (from 12 to 28 days post-hatch). Altricial nestlings respond to environmental perturbations such as anthropogenic disturbances, inclement weather, food restriction and sibling competition with approximately three- to 16-fold elevation in circulating CORT [11,12,42–46]. Oral administration of CORT to nestling zebra finches increases endogenous levels of stress-induced CORT approximately 10-fold, falling within the fold elevation seen in natural stressors across other species of nestlings, while also falling within a biologically relevant range in nestling zebra finches [47]. Developmental stress in the form of elevated CORT and food restriction induces long-term effects on physiology and behaviour [13,47–49], song learning [25,27] and plumage coloration [24], suggesting that our treatment could have organizational effects on adult

phenotype with the potential to influence reproductive behaviour and success.

After male zebra finches reached sexually maturity, we used common garden breeding experiments to determine if CORT exposure during development modulates male reproductive tactics. Specifically, we measured nest attendance, nestling condition and the number of nestlings sired through EPCs. We predicted that males exposed to developmental stress would obtain fewer EPCs, but would instead invest in offspring quality by increasing nest attendance and, thus, rear nestlings in better condition compared with control males. By contrast, we predicted that control males would invest in offspring quantity by siring more nestlings through EPCs compared with CORT-exposed males. Therefore, males exposed to developmental stress would maximize reproductive success by investing in offspring *quality* while control males would invest in offspring *quantity*.

2. Material and methods

(a) Parental birds: housing and breeding

Ten female and 10 male zebra finches were purchased from six pet stores across Montana and Washington, USA. Throughout the course of the experiment, three males and two females were replaced due to mortality. Individuals were given unique colour band combinations and housed communally in a 2.0 × 2.8 m room (14 L : 10 D, 26–27°C with 20–30% humidity). Birds had access to 12 nest-boxes and shredded burlap nesting material. We fed birds commercial finch seed (Silver Song West) and spray millet ad libitum and supplemented their diet daily with hard boiled eggs, spinach and crushed egg shells. Nest-boxes were monitored daily for signs of nest building and egg laying. Over the course of the experiment, 48 clutches of nestlings were produced.

(b) First-generation nestlings: experimental treatment

We marked newly hatched nestlings with an individual combination of leg markings using a black Sharpie marker. We banded nestlings with a numbered plastic leg band between 3 and 4 days post-hatch. Nestlings were then randomly assigned to treatment groups (CORT or control), balancing treatments within broods. Nestlings exposed to the CORT treatment were fed oral boluses (25 µl) of CORT (Sigma Aldrich) dissolved in peanut oil twice daily approximately 5 h ± 1 h apart. From 12 to 15 days post-hatch, nestlings received 0.124 mg ml⁻¹ of CORT in peanut oil for a total daily dose of 6.2 µg of CORT. Starting from 16 days post-hatch, the dose was increased to 0.163 mg ml⁻¹ for a total daily exposure of 8.15 µg of CORT (doses from Spencer *et al.* [13]). Control nestlings were fed 25 µl of peanut oil on an identical feeding schedule. Nestlings were exposed to treatments from 12 to 28 days post-hatch (methods as per Spencer *et al.* [13]). These methods have been shown to elevate baseline and stress-induced corticosterone within the natural physiological range of nestling zebra finches [13].

Nestling zebra finches fledge as early as 17 days post-hatching. Before fledging, we identified the social parents of a nest by observing incubation and provisioning behaviours (observations made in person, or using VehoMuvimicroDV camcorders if necessary). After determining social parents, we moved the nest-box and parents to wire cages (70 × 40 × 44 cm) where they were housed until the nestlings reached nutritional independence at 28 days post-hatch [13]. Following nutritional independence, we returned the parents to the breeding aviary. Nestlings remained in the cages and were fed a diet of commercial finch food, spray millet, boiled eggs and spinach until reaching sexual maturity at 90 days post-hatch. Zebra finches fed CORT during development

were smaller and had higher stress responses than control birds at 28 days post-hatch, but not at 60 or 90 days post-hatch [48]. Another study from this population showed that CORT treatment had long-term effects on the ability of zebra finches to solve a novel foraging task [49]. These results demonstrate that our treatment alters nestling phenotype with some sustained effects.

(c) Breeding experiment: set-up

To measure F_1 reproductive success, we established common garden breeding experiments in aviaries 2.0×2.8 m that consisted of 10 novel females, five adult males from the CORT-fed treatment and five adult males from the control treatment. We obtained females from pet stores from Montana and Washington, USA. All housing conditions (light/dark, temperature, humidity, nest-boxes and nesting material and food) were identical to those described in the parental generation above. We allowed the birds to breed for one reproductive bout. The number of males that successfully fledged one nestling was 8, 8, 6 and 7 for replicates 1–4 (respectively). We replicated the breeding experiment twice to examine the effects of developmental stress on paternal behaviour using unique males and females for each replicate (i.e. no bird was used in more than one replicate). When we examined the effects of developmental stress on paternity, our initial trends were non-significant. To further explore this effect we expanded our sample size to four replicates for the paternity analysis (see below) with unique birds in each replicate.

Prior to starting each replicate, we weighed males to the nearest 0.1 g and measured tarsus length (posterior to anterior tarsus) and wing chord (carpus to longest primary feather) to the 0.1 mm. We used tarsus length and mass to calculate condition using the scaled mass index [50,51]. The scaled mass index accounts for errors associated with residual body mass measurements by using a scaling relationship derived from the population of interest to calculate the expected mass of each individual at a fixed body size. In this way, the scaled mass index standardizes all animals to the same growth phase or body size and is considered to be a more accurate measure of condition [50,51].

(d) Breeding experiment: social paternity and feeding behaviour

To measure parental behaviour, we collected videos of nests using VehoMuvimicroDV camcorders for 3 consecutive days starting 6 days post-hatch. From these videos, we identified social parents and measured the following parental behaviours: occurrences of nest attendance, nest association, and time spent in nest attendance or nest association. We considered birds to be engaging in nest attendance after they entered the nest-box. We defined nest association as behaviours that occurred when the birds were perched outside or on top of nest-boxes.

(e) Second-generation nestlings: measurements and social paternity

We monitored nest-boxes daily for signs of egg laying and hatching. Starting on hatch day, we marked nestlings with an individual combination of leg markings using a black Sharpie marker. Between 3 and 4 days after hatching, we banded nestlings with a numbered plastic leg band. Twelve and 28 days after hatching, we weighed nestlings to the nearest 0.1 g and measured tarsus length (posterior to anterior tarsus) and wing chord (carpus to longest primary feather) to the 0.1 mm. We used the tarsus length and mass to calculate condition using the scaled mass index as described above [50,51].

At 28 days post-hatch we also collected 25 μ l of blood for use in paternity analyses. To collect blood, we punctured the alar vein with a 26-gauge needle and collected blood with heparinized

microcapillary tubes. Immediately after collection, blood was frozen at -20°C before being moved to storage at -80°C .

(f) Paternity analysis

We determined genetic paternity using six microsatellite markers previously developed for the zebra finch [52]. For each locus, we first used a single polymerase chain reaction (PCR) to genotype all parents from the breeding experiment (F_1 males and untreated mothers) to determine the range of allelic diversity for each locus. We then designed two multiplex PCR reactions (multiplex 1: Tgu1, Tgu8, Tgu10; multiplex 2: Tgu5, Tgu9, Tgu12) for subsequent multilocus genotyping of the nestlings (F_2). Three primers were labelled using HEX fluorescent tags (Tgu8, 9 and 10) and three were labelled using FAM fluorescent tags (Tgu1, 5 and 12). We extracted genomic DNA from 5 μ l of the blood from each individual using the DNA Easy Blood & Tissue kit (QIAGEN). We performed multiplex PCR using the Multiplex PCR kit (QIAGEN). Each 10 μ l reaction contained 1–2 ng of DNA, 5 μ l of QIAGEN Multiplex PCR Master Mix, 3 μ l of ddH₂O and 1 μ l of multiplex primer stock containing 1 μ M of each primer. The cycling conditions for both multiplex mixes were: a 2-min initial denaturation step at 94°C , 35 cycles of 30-s denaturation at 94°C , 30-s of annealing at 60°C , and a 20-s extension at 72°C , followed by a final 4-min annealing step at 72°C . Diluted PCR products were genotyped by the University of Arizona Genetics Core using an ABI 3730 Genetic Analyzer under standard conditions. We scored all genotypes using GENE MAPPER 3.7 (Applied Biosystems).

All six microsatellite loci were highly polymorphic with the number of alleles per locus ranging from 11 to 18 (mean = 14.67, s.d. = 2.50). Paternity was initially assigned using a simple exclusion analysis between offspring, the known maternal genotype and all potential fathers. We then verified the results of our exclusion analysis using maximum-likelihood paternity assignment as implemented in Cervus 3.0.3. The combined non-exclusion probabilities calculated by CERVUS 3.0 were $p = 0.0031$ for the first parent and $p = 0.0001$ for the second parent. Two nestlings died before we were able to obtain blood samples for genetic analysis. We were unable to obtain DNA from three samples. Therefore, our sample sizes for social and genetic paternity are 100 and 95, respectively.

(g) Beak colour analysis

We quantified spectral components of beak coloration for a separate cohort of males by taking photographs of each bird and analysing the resulting images in Adobe Photoshop CS (Adobe Systems, San Jose, CA, USA). We photographed the right and left side of each bird using a Cannon Powershot digital camera. We used a white reference to standardize measurements across images (The Tiffen Company, Hauppauge, New York). Using the magic wand tool (tolerance = 30), we selected the beak and used the mean values of red, green, and blue channels displayed in the histogram window to calculate hue, saturation, and brightness using the colour picker function (methods as per [53,54]). Hue values ranges from 359 to 6. We changed hue values of 359 to -1 in order to make all values numerically consecutive for statistical analyses. We subtracted the mode hue, saturation, and brightness measurements for the standard from the corresponding measurements for each sample. We then averaged the value for the right and left picture and used the resulting values in all statistical analyses.

(h) Statistical analyses

We analysed all data using PASW STATISTICS18.0. All measurements of nestling provisioning were non-normally distributed (Shapiro–Wilk, $p < 0.04$) except for the paternal nest attendance and maternal nest association ($p < 0.12$). We log-transformed

non-normal data and used the resulting values in all statistical analyses. We measured parental behaviour for both parents separately and for both parents combined to look at the total amount of parental care nestlings received. We analysed parental data using multivariate general linear models with paternal treatment as a fixed factor and clutch size as a random factor. Clutch size did not affect parental behaviours and was removed from the final model. We used generalized linear models (GLM) to analyse the effect of paternal treatment on nestling morphology and condition with paternal treatment as a fixed factor and nest identity as a random factor to account for multiple measurements per nest [45]. Experimental replicate (1–2) was included as a fixed factor in initial analyses, but was non-significant and removed from the final model. We analysed treatment effects on paternal morphological traits (tarsus, wing, mass, condition) using analysis of variance (ANOVA).

The numbers of nestlings reared by social fathers and the number of offspring sired by males were non-normally distributed (Shapiro–Wilk, $p < 0.001$). We analysed the effects of developmental treatment on social and genetic paternity using GLM using a Poisson distribution with treatment as a fixed factor. We included experimental replicate as a fixed factor (1–4) and male age (days post-hatch) as a covariate in the initial model. These factors were non-significant and removed from final analyses. We further explored the effects of developmental treatment on EPCs using a χ^2 -analysis to evaluate if treatment affected the success of achieving EPCs and the likelihood that a male would raise non-genetic offspring.

Hue, saturation, and brightness measurements were non-normally distributed (Shapiro–Wilk, $p = 0.003, 0.04, 0.06$, respectively, for all). Log transformation failed to normalize data ($p < 0.05$ for all). Age had a non-significant effect on hue values with older birds having less red beaks ($p = 0.055$, Spearman's $\rho = 0.29$, $n = 45$). Age affected brightness measures with birds having brighter beaks ($p = 0.03$, Spearman's $\rho = 0.32$, $n = 45$). To account for age effects, we used a generalized linear mixed model with a Poisson distribution and treatment as a fixed effect, age as a covariate. The sample sizes were $n = 24$ and 21 for control and CORT-fed males, respectively.

3. Results

(a) Treatment effects on males (parental generation): body size and bill colour

There was no difference between CORT-treated and control males in any measure of body size or condition prior to the start of the breeding experiments ($F_{1,39} < 0.42$, $p > 0.52$ for all). These data are congruous with a larger dataset from this population showing that CORT treatment during development decreased body size in zebra finches at 30 days post-hatch, but did not have sustained effects on body size at 60 or 90 days post-hatch [48]. Developmental treatment did not have an effect on hue, saturation or brightness measurements of adult beak coloration ($p = 0.39, 0.18$ and 0.77 for all).

(b) Nestling provisioning

CORT treatment during development increased the total amount of combined time that males and females spent in nest attendance and nest association (figure 1; $F_{1,13} = 5.22$, $p = 0.04$). However, there was no difference between treatment groups in the number of paternal nest association bouts or the time spent in nest association ($F_{1,13} < 0.16$, $p > 0.39$). Likewise, CORT treatment during development did not affect paternal

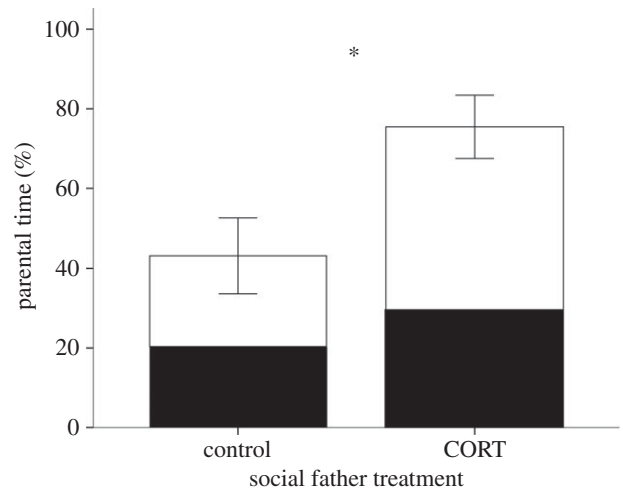


Figure 1. Total percent of time parents invested in parental behaviours for pairs with fathers (F_1) exposed to CORT or control treatments during development. Black portion denotes social father and white the mother.

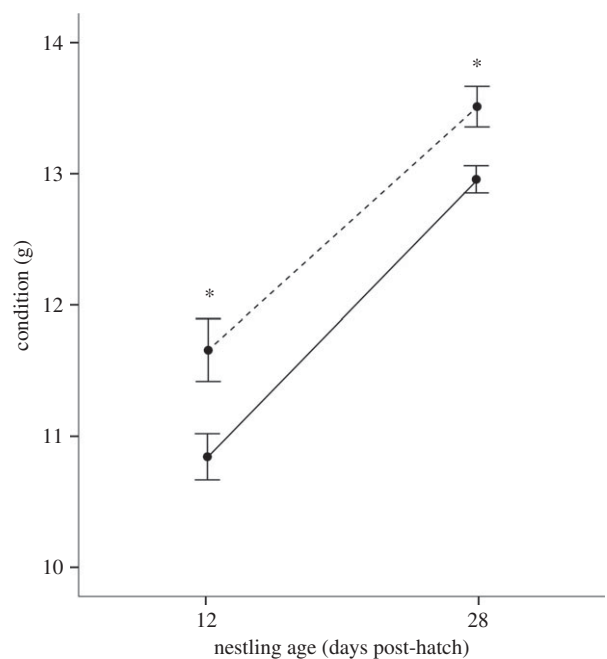


Figure 2. Condition (scaled mass index; g) of nestlings at 12 and 28 days post-hatch reared by fathers fed CORT or control treatments during development. * $p < 0.006$. Dashed lines indicate CORT treatment and solid lines the control.

nest attendance ($F_{1,13} = 1.51$, $p = 0.24$) or the number of nest attendance trips ($F_{1,13} = 1.75$, $p = 0.24$). Paternal treatment group during development did not affect the number of maternal association or attendance bouts or the time spent in nest association or attendance ($F_{1,13} < 1.16$, $p > 0.17$). However, females paired with males fed CORT during development spent more time engaging in parental care (nest attendance and nest association) compared with females paired to control males ($F_{1,13} = 5.81$; $p = 0.03$).

(c) Nestling body size and condition

Fathers exposed to CORT during development reared nestling in higher condition at 12 and 28 days post-hatch compared with control males (figure 2 and table 1, Wald statistic = 7.71, 9.09, d.f. = 1, 1, $p = 0.006, 0.003$, respectively).

Table 1. Body size and condition for nestlings (F_2) reared by fathers (F_1) exposed to CORT or control treatments during development. Significant values are highlighted in bold.

variable	day 12					day 28						
	control ($n = 25$)		CORT ($n = 26$)		difference	control ($n = 25$)		CORT ($n = 26$)		difference		
	mean	s.d.	mean	s.d.		mean	s.d.	mean	s.d.			
tarsus (mm)	13.67	1.12	13.32	0.83	0.190	1.70	14.07	0.69	13.89	0.64	0.330	0.97
wing (mm)	34.98	4.90	32.98	2.28	0.059	3.57	55.18	1.59	54.54	1.84	0.18	1.82
mass (g)	11.16	1.44	11.29	1.33	0.74	0.11	13.08	1.01	13.37	0.77	0.25	1.34
condition (g)	10.84	0.88	11.66	1.22	0.006	7.71	12.96	0.52	13.51	0.79	0.003	9.09

There were no differences between treatment groups in any nestling morphological measurement at 12 or 28 days post-hatch (table 1). However, there was a non-significant trend for nestlings reared by control fathers to have longer wing chords at 12 days post-hatch (Wald statistic 3.57, d.f. = 1, $p = 0.059$).

(d) Offspring sired and extra-pair offspring

Males exposed to CORT during development reared similar numbers of social offspring compared with control males (table 2 and figure 3; Wald $\chi^2 = 1.96$, $p = 0.16$, d.f. = 1,38). Of 95 nestlings produced across four replicates, 29 (30.5%) were sired through extra-pair fertilizations (EPFs). This EPF percentage is similar to what has previously been described in captive zebra finches [55]. Males exposed to CORT during developmental sired more genetic offspring compared with control males (table 2 and figure 3; Wald $\chi^2 = 5.15$, $p = 0.02$, d.f. = 1,38). CORT-fed and control males sired similar numbers of offspring through EPFs ($n = 15$ and 14, respectively, $p = 0.85$, $\chi^2 = 0.03$). However, control males were more likely to rear non-genetic offspring compared with males exposed to CORT during development (table 2; $n = 20$ and 9, respectively, $p = 0.04$, $\chi^2 = 4.17$). CORT-fed and control males were equally likely to be chosen as social mates ($p > 0.9$, Pearson's $\chi^2 = 0.13$, d.f. = 1) and to sire at least one nestling ($p = 0.50$, Pearson's $\chi^2 = 1.03$, d.f. = 1).

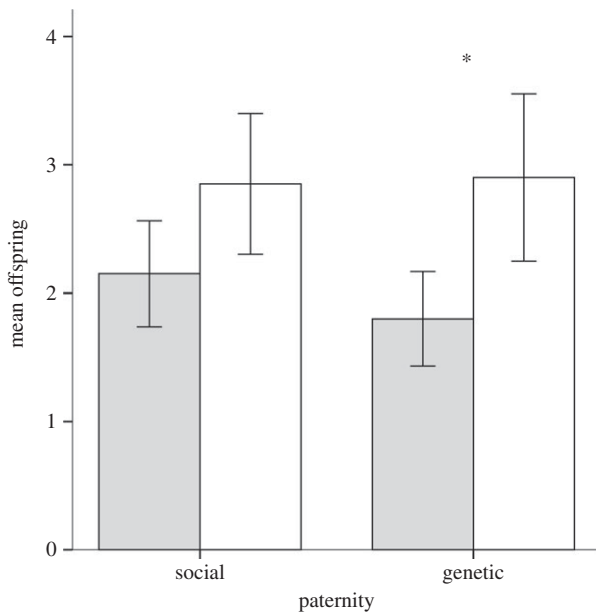
4. Conclusion

We investigated the effects of developmental stress (elevated CORT) on male reproductive success in zebra finches. Male zebra finches exposed to developmental CORT reared higher quality nestlings and sired more nestlings compared with control males. Nestlings reared by social fathers exposed to developmental CORT received more combined parental care from mothers and fathers and were in better condition compared with nestlings reared by control social fathers and their mates. CORT males were equally as likely as control males to sire nestlings through EPCs, but were less likely to rear non-genetic offspring compared with control males. Overall, males exposed to development stress had greater reproductive success than control males in both the *quality* and *quantity* of nestlings produced.

In general, developmental stress has well-known suppressive effects on sexually selected traits and is therefore predicted to decrease reproductive success ([21,22,25], but see: [56]). However, we found no effect of developmental CORT treatment on adult bill coloration and a positive effect of CORT treatment on male reproductive success. Our results demonstrate that exposure to stress during development (via elevated CORT) can have sustained and positive effects on fitness. Exposure to CORT during development has broad effects on physiology and behaviour and it is possible that our surprising results are explained by pleiotropic effects of developmental CORT exposure on aspects of organism phenotype that were not assessed in this experiment. From our experiment, we are unable to determine the phenotypic changes responsible for increased reproductive success or whether these changes arise from direct or indirect effects of CORT treatment. We offer several potential explanations for our data that may serve as fruitful avenues for future research.

Table 2. Number of offspring sired through EPFs and reared by males exposed to CORT or control treatments during development. Significant values are highlighted in bold.

treatment	mean social offspring	mean genetic offspring	offspring sired through EPCs	non-genetic offspring reared
control	2.15	1.80	14	20
CORT	2.85	2.90	15	9
difference	$p = 0.16$	$p = 0.02$	$p = 0.85$	$p = 0.04$

**Figure 3.** The number of nestlings reared (social paternity) and sired (genetic paternity) by CORT-fed and control males. $*p = 0.04$. White bar denotes CORT treatment and grey bar the control.

One possible explanation for increased reproductive success in CORT-exposed males is that developmental CORT decreases longevity and triggers males to increase investment in their current reproductive effort. For animals that reproduce multiple times over their lifespan, life-history theory predicts that reproductive effort should increase with decreased residual reproductive value [57,58]. Current reproduction comes at the costs of future reproduction and animals with limited future reproductive prospects should invest more in current reproductive bouts (e.g. the 'terminal investment' hypothesis; [59,60]). Exposure to developmental stress (elevated CORT) decreases longevity in zebra finches [17,61] potentially through changes in cell function that result in increased oxidative damage [61,62]. It is possible that our developmental CORT treatment increased senescence and triggered males to increase investment in their current reproductive bout. In this scenario, we would expect to see reduced lifespans in CORT-treated males compared with control males, but equivalent reproductive success between treatment groups over the lifespan of the birds [17]. Future experiments could assess the role of developmental stress on alternative reproductive tactics and lifetime breeding success through long-term common garden breeding experiments.

A second explanation of the results lies in possible effects on behavioural phenotype (e.g. personality; [5]). There is increasing evidence that developmental stress can affect adult behaviour in ways that could potentially affect reproductive success (reviewed in [5]). For example, zebra finches fed CORT during the nestling period were less

socially dominant at two months of age compared with control birds [47]. We found that males exposed to developmental CORT sired more offspring and were cuckolded less compared with control males. It is possible that CORT treatment resulted in comparatively reduced social dominance and intrasexual aggression. In this scenario, control males may have engaged in more frequent aggressive social encounters than CORT-fed males reducing their available time to invest in reproductive behaviours such as nestling provisioning. Developmental CORT treatment could have also altered affiliative or mate guarding behaviours. Mate guarding can reduce extra-pair paternity in birds [63,64]. In our experiment, CORT-fed males were less likely than control males to rear non-related nestlings, but were equally as likely as control males to sire nestlings through EPCs. This suggests that males from both treatment groups were equally successful at achieving EPCs or females showed no preference between CORT-fed and control males for EPCs. However, CORT-fed males were more successful at preventing their social mate from engaging in EPCs or females mated to CORT-fed males were less likely to pursue EPCs. These explanations are non-mutually exclusive and provide easily testable hypotheses for future research.

Although many of the phenotypic consequences of developmental stress appear overwhelmingly negative, there is increasing evidence for an adaptive function of developmental stress (reviewed in [65–69]). For example, stress exposure during development may induce phenotypic changes which prepare developing animals to live in harsh environments or match offspring needs to parental capabilities [8,56,66]. In our experiment, males exposed to CORT during development may maximize nestling quality in anticipation of living in a harsh environment. The transition from nestling to fledging is marked by high mortality in free-living birds. Fledglings in better condition are more likely to survive to adulthood [70]. Therefore, by investing in parental care and increasing nestling quality, males living in stressful environments could maximize the survival of their nestlings and, hence, their fitness. In this way, the environment experienced by the male parent during development indirectly affects his offspring's phenotype by shaping his parental behaviour allowing him to maximize fitness in a given environment [71]. This idea of phenotype matching has received some experimental support recently [56]. In our experiment, birds were kept in standard housing conditions with ad libitum food availability (i.e. in seemingly non-stressful conditions). In this scenario, contrary to our results, phenotype-matching models would predict that control males would outperform CORT-fed males [71]. However, it is possible that CORT-fed males only appear to have higher reproductive success than control males because we examined one reproductive attempt. In non-stressful conditions, it is possible that control males would have had higher reproductive success over their

entire reproductive lifetime compared with CORT-fed males; this would support phenotype-matching models. Future studies that alter housing conditions (e.g. restricted food availability) and measure multiple reproductive attempts could begin to examine the role of phenotype matching on alternative reproductive tactics and reproductive success.

The effect of early environment on animal phenotype is fundamentally important to the study of evolution because the variation caused by early environment creates variation upon which natural selection can act [1]. Developmental stress has been associated with a range of seemingly negative phenotypic changes. However, phenotypic changes that occur as a consequence of development could maximize fitness in harsh or stressful environments (e.g. [72,73]). Here, we show that males exposed to stress during development rear offspring in better condition, sire more offspring, and are less likely to rear non-genetic offspring. These data are the first to demonstrate that developmental stress can have a positive effect on

fitness via changes in reproductive success and provide support for an adaptive role of developmental stress in shaping animal phenotype.

Ethics statement. All research and methods described above were approved by the University of Montana Institutional Animal Care and Use Committee (IACUC; AUP 018-11).

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