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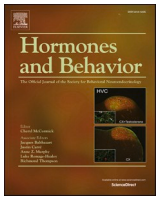

Early life conditions reduce similarity between reproductive partners in HPA axis response to stress

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ABSTRACT

Social environments modulate endocrine function, yet it is unclear whether individuals can become like their social partners in how they physiologically respond to stressors. This social transmission of hypothalamic-pituitary-adrenal (HPA) axis reactivity could have long-term consequences for health and lifespan of individuals if their social partners react to stressors with an exaggerated HPA axis response. We tested whether glucocorticoid levels in response to stress of breeding partners changes after breeding depending on whether partners had similar or dissimilar postnatal conditions. We manipulated postnatal conditions by mimicking early life stress in zebra finch chicks (*Taeniopygia guttata*) via postnatal corticosterone exposure. When they reached adulthood, we created breeding pairs where the female and male had experienced either the same or different early life hormonal treatment (corticosterone or control). Before and after breeding, we obtained blood samples within 3 min and after 10 min or 30 min of restraint stress (baseline, cort10, cort30). We found that corticosterone levels of individuals in response to restraint were affected by their own and their partner's early life conditions, but did not change after breeding. However, across all pairs, partners became more similar in cort30 levels after breeding, although differences between partners in cort10 remained greater in pairs with a corticosterone-treated female. Thus, we show that HPA axis response to stressors in adulthood can be modulated by reproductive partners and that similarity between partners is reduced when females are postnatally exposed to elevated glucocorticoids.

1. Introduction

Early life conditions can permanently alter how the hypothalamic-pituitary-adrenal (HPA) axis responds to stressors throughout life, thereby affecting health across the lifespan (Danese and McEwen, 2012; Harris and Seckl, 2011; Lupien et al., 2009; Matthews, 2002). The HPA regulates glucocorticoids, which become transiently elevated to help vertebrates cope with immediate situations that threaten survival (Sapolsky et al., 2000; Wingfield and Romero, 2011). However, chronic activation of the HPA and chronically high levels of glucocorticoids often cause oxidative stress and cellular damage that ultimately affect ageing rate (Hewitt et al., 2012; Costantini et al., 2011, but see Boonstra, 2013). The HPA axis of individuals that experience early postnatal stress are often more reactive, secreting more glucocorticoids in response to acute stressors (Love and Williams, 2008; Lupien et al., 2009; Sandi and Haller, 2015; Spencer et al., 2009). Consequently, individuals that experienced elevated stress in early postnatal life are more likely to suffer physical and psychological ailments, reduced fitness, and/or premature mortality, even if they live in benign environments without threat of starvation or predation (Danese and McEwen, 2012; Maniam et al., 2014; Marshall et al., 2017; Monaghan et al., 2012).

However, the social environment in later life can also play an important role, with individuals influencing each other (Creel et al., 2013; DeVries et al., 2003; Seeman and McEwen, 1996). Stress contagion – when a previously non-stressed individual increases behavioural and physiological responses to stresses after interacting with a previously stressed individual – appears taxonomically widespread and may coordinate or synchronise activity and states between social group members (Brandl et al., 2022; Buchanan et al., 2012; Carnevali et al., 2020; DeVries et al., 2003; Engert et al., 2019; Nitschke and Bartz, 2023). Via this process of stress contagion, GC levels of individuals that did not themselves directly experience stressful events become elevated after interacting with other conspecifics who have had such experiences.

Stress contagion, however, is not currently hypothesised to

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permanently alter how strongly the HPA axis responds to future stressors. Nonetheless, there is some evidence to imply that HPA axis response to stressors of two individuals could become more similar if they interact frequently over time. In humans, cortisol levels of heterosexually married couples are interdependent and moderated by the amount of a partner's physical presence and marital satisfaction (Liu et al., 2013; Papp et al., 2013; Saxbe and Repetti, 2010). In a passerine bird that provides bi-parental care, baseline GC levels are correlated between breeding partners and become more similar the longer they breed together, and pairs with more similar baseline GCs fledged more offspring (Ouyang et al., 2014). The study by Ouyang et al. (2014) suggests that in species that provide bi-parental care, there might be fitness benefits for individuals to become hormonally similar to their mates. However, as only baseline GCs were measured, it is unclear whether breeding partners also became more similar in how strongly the HPA axis respond to stressors.

Becoming hormonally similar to breeding partners may also be costly for individuals. In cases where one pair member responds with an exaggerated HPA axis response to stressors, its partner may face a trade-off: match physiological states and invest in current reproductive success or resist matching physiological states and invest in future health and longevity. In a study by Monaghan et al. (2012), zebra finches (*Taeniopygia guttata)* that did not themselves experience early life stress had shorter lifespans if they bred with a partner that was early life stressed. There was no sex difference in this effect, with the same lifespan reduction being evident irrespective of whether the male or the female had experienced early life stress. Where both members of the pair had experienced early life stress the lifespan reduction was greatest. Matching of physiological states might explain why control individuals that were paired with an early life stress individual experienced lifespan reduction, but this has not been directly tested thus far.

In this study we examined whether this detrimental effect of survival may result from stress-induced GC levels of individuals becoming more similar over time due to stress contagion. Specifically, our study aimed to understand whether stress contagion is long-lasting: can the magnitude of stress-induced GC levels in adulthood be influenced by social partners, and do the early life conditions of individuals and/or their partners matter? Following the same protocol as Monaghan et al., 2012, we simulated early life stress by administering GCs during early postnatal development, which has been shown to increase HPA axis reactivity to stressors later in life (Spencer et al., 2009). After sexual maturation, individuals were paired with a partner that had experienced the same postnatal GC exposure (experimental bird) or with a partner that had not experienced postnatal GC exposure (control bird). Baseline and stress-induced GCs levels of both pair members were measured before and after breeding to assess HPA axis activity at baseline and in response to a stressor. In our study, we use stress-induced to refer to GC levels after 10 or 30 min of standardised restraint stress. Our prediction was that 1) postnatal GC exposure would lead to higher stress-induced GC levels in adulthood, in line with previous findings; 2) stress contagion would cause control individuals paired with experimental birds to have higher stress-induced GC levels after breeding compared to before breeding; 3) stress-induced GC levels of experimental birds paired with another experimental bird would be the highest; 4) stress contagion would reduce the difference in baseline and stress-induced GC levels between partners in all pairs, supporting what has been reported in great tits (Ouyang et al., 2014). Before breeding, the HPA axis response to stress would be more dissimilar in partners with different postnatal GC but would show the greatest convergence after breeding. We did not predict any sex effects because having an early life stressed partner reduced the lifespan of both males and females (Monaghan et al., 2012).

2. Methods

2.1. Subjects and manipulation

Experimental birds ($N = 131$) were bred from 36 breeding pairs from the University of Glasgow breeding stock in October 2018. Five days after hatching, subjects were assigned to control (CON, $n = 77$ from 36 nests) or experimental (EXP, $n = 54$ from 28 nests) treatment pseudorandomly (to distribute first-hatched birds equally between the two treatments and ensure that all nests had at least one CON and one EXP subject). We replicated the manipulation of corticosterone – the main GC hormone in birds – as described in (Spencer et al., 2009). Briefly, from day 12–28 post-hatch, EXP birds received oral administration of corticosterone (Sigma Aldrich) dissolved in peanut oil twice daily (total 6.2 μg corticosterone per day from day 12–15 and 8.15 μg corticosterone per day from day 16–28) while CON birds received only peanut oil twice daily, approximately 5 h apart. This dosing regimen elevates circulating corticosterone to levels comparable to those observed in the acute stress response of similar aged zebra finches (Spencer et al., 2009). Each nest contained both CON and EXP individuals. Birds were weighed at 12, 16, 21, 28, and 40 days of age to the nearest 0.1 g. When approximately 60 days old, birds were moved to single sex cages (2 m × 1 m × 0.5 m) containing 4–6 birds per cage and remained there until the breeding experiment. Photoperiod was kept at 14:10 light:dark and temperature was kept between 20 and 24 °C. All procedures were in compliance with the UK Home Office project license No. 70/8335 and received institutional approval by the University of Glasgow Animal Welfare and Ethics Review Board. All procedures were performed in accordance with the ARRIVE guidelines for use of animals in scientific research.

2.2. Breeding experiment

Assigning mates for the breeding experiment was pseudo-randomised: the male and female in a pair did not share the same parents or grandparents (i.e. were not siblings or cousins) and individuals from one nest were not always paired with individuals from another nest (i.e. not all birds in nest 1 paired with birds in nest 2). We created four treatment groups (pair types) that included pairwise comparisons of adults that were either CON or EXP. The four pair types were as follows: two CON birds (CON♀ - CON♂), female EXP and male CON (EXP♀ - CON♂), female CON and male EXP (CON♀ - EXP♂) and two EXP birds (EXP♀ - EXP♂). In the experiment, 8 pairs of each pair type were studied ($N = 32$ pairs). Birds were approximately 330 days old at the start of breeding (mean ± SD = 329 ± 18). Pairs were housed in (120 × 50 × 50 cm) cages containing ad libitum food, water, and cuttle bone, and a nest box attached to an opening on one side of the cage. Coconut fibers for nest building were provided until the first egg was laid. Body mass was measured and blood samples were collected from birds between one to three days before pairing in October 2019 and approximately 100 days after a reproductive attempt in January 2020 (difference between pre- and post-paring blood samples mean ± SD = 114 ± 4 days). A summary and timeline of the experiment is shown in Fig. 1.

2.3. Blood samples

Corticosterone was quantified from a baseline blood sample (taken within 3 min of disturbance, mean ± SD = 86.5 s ± 31.5) and after 10 or 30 min of a standardised restraint stressor, which involved placing birds in soft cloth bags. We investigated corticosterone levels at 10 and 30 min after a stressor because they are both affected by postnatal GC manipulation in juvenile zebra finches (Spencer et al., 2009). Blood samples were taken between 10:00 and 13:00 to minimise circadian fluctuations (Breuner et al., 1999). Blood was collected from the brachial vein with a 26-gauge hypodermic needle and birds were weighed to the nearest 0.1 g. Each individual was sampled only twice (i.e. baseline and cort10, or baseline and cort30). A maximum of 10 % total blood volume was

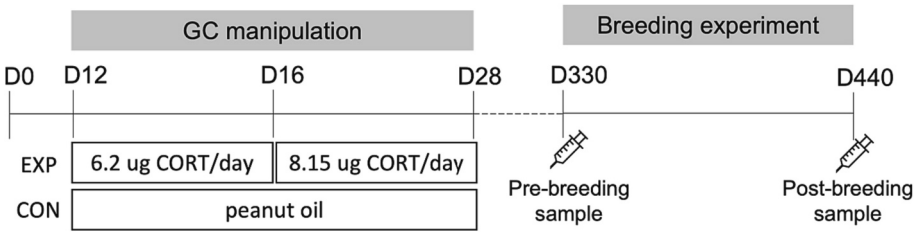

Fig. 1. The timeline and experimental design, with "D" indicating age in days post-hatch. Postnatal GC manipulation occurred during the first month of life, with EXP birds receiving two doses of corticosterone per day for two weeks (dosage increased after the first week). Treatment was within-brood, such that each nest contained both EXP and CON birds. Subjects were allowed to breed almost one year later, with blood samples taken before partners were introduced and after pairs had undergone a reproductive attempt.

collected from each subject. Samples were collected in heparinised capillary tubes and put immediately on ice, then centrifuged at 1500 rpm for 5 min to extract plasma, which was then stored at −80 °C until laboratory analysis.

2.4. Hormone analysis

Corticosterone was assayed in samples using an ELISA kit (ENZO Life Sciences ADI-900-097) that has been previously validated in zebra finches (Wada et al., 2007). Samples were prepared in advance by diluting 14 μL of plasma in a 2.5 % steroid displacement buffer at 1:20 dilution. For samples with insufficient volume, 7 μL was instead diluted into a 1.5 % steroid displacement buffer at 1:40 dilution. All standards and samples were assayed per manufacturer instructions; standards were run in triplicate while samples were run in duplicate. A control standard of unknown concentration was included in duplicate on each plate and was generated by pooling samples from $n = 24$ animals. Plates were read using a Labtech LT-4500 Microplate Reader at 405 nm, corrected at 570 nm. Levels of corticosterone were calculated using a five-point standard curve ranging between 20,000 and 32 pg/mL. The true minimum detection limit was calculated per plate and ranged between 9.53 and 21.7 pg/mL: Intra- and inter-assay variability was 13.93 % and 10.94 % respectively. The range of intra-assay variation was 11.22–17.99 % (SD = 2.58 %). Samples that were below the limit of detection were assigned the value of the lower limit of detection of the respective plate they were measured on ($n = 11$ out of 250 samples). We excluded one value from statistical analyses because there was a very large difference between duplicates (0.06 versus 1.35 ng/mL). Hormone analyses were performed by CM who was blind to individual early life treatment and pair types.

2.5. Statistical analysis

All statistical analyses were performed using the R Statistical language (version 4.3.0; R Core Team, 2023). We used linear mixed models (LMMs) from the *lme4* package (version 1.1.33; Bates et al., 2015) to determine whether a bird's corticosterone levels changed after breeding depending on its own and/or its partner's early life treatment. Each bird was considered as an experimental unit for these analyses. Blood samples collected for each timepoint (i.e. *baseline, cort10,* and *cort30)* were analysed separately to reduce the number of parameters to be estimated and obtain good quality estimates for those parameters, based on the amount of data we had. For baseline corticosterone, we performed analyses on the residuals of a linear regression between corticosterone levels and the time to obtain blood samples because we unintentionally obtained blood from EXP birds significantly earlier than CON birds (mean time in seconds for CON = 97.67; EXP = 79.27). We interpret the residuals used in analyses of baseline corticosterone as corticosterone levels corrected for time to obtain blood.

To test our hypothesis that HPA axis reactivity of individuals would change if they bred with a mate that was exposed to postnatal GCs, we performed model selection using an information theory approach (AIC) with the package *AICcmodavg* (version 2.3.2; Mazerolle, 2023). AIC compares models based on parsimony, i.e. a balance between model fit and model complexity. More parsimonious models are indicated by a smaller Akaike information criterion value, and we use the AIC value corrected for a small sample size (AICc; Symonds and Moussalli, 2011). For each timepoint, we created models with combinations of the predictors *mate treatment* (CON or EXP), *own treatment* (CON or EXP), *breeding stage* (pre-breeding or post-breeding), *sex*, and/or interactions of these terms (Table 1). We created models to specifically test whether 1) corticosterone levels in adult birds are predicted by *own treatment*, *mate treatment, sex*, and *breeding stage*; 2) corticosterone levels are predicted by the interaction of *mate treatment* and *breeding stage*, potentially depending on the *sex* of the subject; 3) corticosterone levels are predicted by the interaction of *own treatment*, *mate treatment, and breeding stage*, potentially depending on the *sex* of the subject. In all models, *body mass* and *clutch size* were scaled and included as fixed covariates with *bird ID* and *birth nest ID* as random effects. Candidate models were compared to a simple model that included only *body mass* and *clutch size* as predictors. We chose to include body mass and clutch size in our models based on knowledge of their effects on moderating circulating corticosterone (e.g. body mass and brood size; Bonier et al., 2011; Lendvai et al., 2007; Hau et al., 2010). The most probable models were ranked according to Akaike weights and delta AICc ≤ 2, and we averaged models with delta AICc ≤ 2 to obtain a final model using the package *MuMIn* (version 1.47.5; Bartoń, 2023; Dormann et al., 2018). If model

Table 1
Fixed predictors of models that were compared to assess whether corticosterone changed after breeding depending on its own or its partner's early life treatment. Model 1 is a simple model without predictors of interest. Model 2 includes predictors that we hypothesised would predict corticosterone, but without interacting (i.e. main effects). Model 3 test the hypothesis that corticosterone levels are predicted by the interaction of *mate treatment* and *breeding stage*, while Model 4 assesses whether this interaction depends on the *sex* of the subject. Model 5 tests whether corticosterone levels are predicted by the interaction of *own treatment*, *mate treatment, and breeding stage*, and Model 6 tests whether the *sex* of the subject moderates these relationships.

	Fixed predictors
1	Mass + Clutch size
2	Mass + Clutch size + Own treatment + Breeding stage + Sex
3	Mass + Clutch size + Mate treatment + Breeding stage + Mate treatment: Breeding stage
4	Mass + Clutch size + Mate treatment + Breeding stage + Sex + Mate treatment: Breeding stage + Mate treatment:Sex + Breeding stage:Sex
5	Mass + Clutch size + Mate treatment + Own treatment + Breeding stage + Mate treatment:Own treatment + Mate treatment:Breeding stage + Own treatment: Breeding stage + Own treatment:Mate treatment:Breeding stage
6	Mass + Clutch size + Mate treatment + Own treatment + Breeding stage + Sex + Mate treatment:Own treatment + Mate treatment:Breeding stage + Mate treatment:Sex + Own treatment:Breeding stage + Own treatment:Sex + Breeding stage:Sex + Mate treatment:Own treatment:Breeding stage + Mate treatment:Own treatment:Sex + Mate treatment:Breeding stage:Sex + Own treatment:Breeding stage:Sex + Mate treatment:Own treatment:Breeding stage: Sex

averaging was performed, coefficients of full average values were reported for final models after averaging. Model assumptions were verified visually by plotting residuals versus fitted values to determine normality of residuals and homogeneity of variance, using the packages *DHARMa* (version 0.4.6; Hartig, 2022) and *performance* (version 0.10.3; Lüdecke et al., 2021).

To determine whether HPA axis reactivity of reproductive partners become more similar, we calculated the absolute difference between partner's corticosterone levels before and after breeding (i.e. difference between baseline CORT of the male and female at pre-breeding, difference between baseline CORT of the male and female at post-breeding). Each breeding pair was considered as an experimental unit for these analyses. Each timepoint (partner difference at *baseline, cort10*, and *cort30*) was analysed separately. For baseline, we analysed the difference between the residuals of a linear regression of corticosterone by sampling time of partners rather than corticosterone levels to correct for effects of sampling time. We used LMMs from the *lme4* package to build models that included the predictors *breeding stage, female treatment, male treatment*, and/or interactions of these terms, with pair ID as a random effect in all models (Table 2). These models tested hypotheses about whether corticosterone levels of partners would become more similar after breeding, and whether female and male treatment explained variation in corticosterone differences between partners. Candidate models were compared to a null model that included only the intercept. Model selection was performed with delta AICc and Akaike weights using the *MuMIn* package, and model assumptions were checked using the *DHARMa* and *performance* packages as described earlier. Results of all model comparison and selection are presented in Table 3.

3. Results

3.1. Does HPA axis reactivity of individuals change if their partners had elevated glucocorticoids in early life?

We found that an individual's stress-induced corticosterone levels, but not baseline corticosterone levels, were predicted by their own and their partner's early life conditions. Table 4 shows final model coefficients for *baseline, cort10*, and *cort30*. Baseline corticosterone was correlated negatively with body mass and clutch size, although time strongly predicted baseline corticosterone levels. Corticosterone levels 10 min after restraint stress (cort10) was affected by *body mass*, *own treatment*, and *breeding stage*. Levels were higher in birds with lower body mass, in EXP birds compared to CON birds, and at pre- compared to post-breeding (Fig. 2). The best fitting model also included *mate treatment, own treatment, breeding stage, clutch size*, and *sex*, although these did not strongly influence corticosterone levels. Corticosterone after 30 min of restraint stress (cort30) was affected by *breeding stage*, *mate treatment,* and *sex*. Levels were higher at pre- compared to post-breeding, in birds with EXP mates compared to birds with CON mates, and in females compared to males (Fig. 3). Other predictors that also contributed to the model but did not strongly influence *cort30* levels included *body mass*, *clutch size*, and *own treatment*.

Table 2
Fixed predictors of models that were compared to test whether HPA axis activity of reproductive partners becomes more similar after breeding. Model 1 is a null model without predictors of interest. Models 2–4 test whether differences in corticosterone between partners are predicted breeding stage, female and male treatment. Models 5–7 test whether differences in corticosterone are predicted by interactions between breeding stage, female or male treatment. Model 8 tests whether differences in corticosterone are predicted by the interactions between breeding stage, and female and male treatment.

	Fixed predictors
1	Intercept only
2	Breeding stage
3	Breeding stage + Female treatment
4	Breeding stage + Female treatment + Male treatment
5	Breeding stage + Female treatment + Male treatment + Breeding stage: Female treatment
6	Breeding stage + Female treatment + Male treatment + Breeding stage: Male treatment
7	Breeding stage + Female treatment + Male treatment + Breeding stage: Female treatment + Breeding stage:Male treatment
8	Breeding stage + Female treatment + Male treatment + Breeding stage:Female treatment + Breeding stage:Male treatment + Breeding stage:Female treatment: Male treatment

Table 3
Model selection tables showing model ID, number of estimated parameters for the model (k), AICc, delta AICc, Akaike weights, log-likelihood (LL), and cumulative Akaike weights. Model ID refers to numbers assigned to models of change in individual corticosterone levels (Table 1) and partner difference in corticosterone levels (Table 2). For example, T1–1 refers to model 1 in Table 1 and T2–1 refers to model 1 in Table 2.

Model ID	K	AICc	Delta AICc	AICc weight	LL	Cum. weight
Baseline corticosterone (individual)						
T1-1	6	482.79	0.00	0.76	−235.03	0.76
T1-3	9	485.83	3.04	0.17	−233.12	0.92
T1-2	10	487.94	5.15	0.06	−232.99	0.98
T1-5	13	490.90	8.11	0.01	−230.78	1.00
T1-4	13	492.87	10.08	0.00	−231.76	1.00
T1-6	21	504.38	21.60	0.00	−226.62	1.00
Cort10 (individual)						
T1-2	10	379.36	0.00	0.72	−177.56	0.72
T1-3	9	381.38	2.02	0.26	−179.99	0.98
T15	13	387.72	8.36	0.01	−177.14	0.99
T1-1	6	390.30	10.95	0.00	−188.40	1.00
T1-4	13	390.68	11.32	0.00	−178.62	1.00
T1-6	21	408.73	29.38	0.00	−172.10	1.00
Cort30 (individual)						
T1-2	10	445.48	0.00	0.77	−210.62	0.77
T1-4	13	449.13	3.65	0.12	−207.85	0.89
T1-3	9	449.52	4.04	0.10	−214.06	0.99
T1-5	13	454.88	9.40	0.01	−210.73	1.00
T1-1	6	460.19	14.71	0.00	−223.35	1.00
T1-6	21	466.28	20.80	0.00	−200.87	1.00
Baseline corticosterone (difference between partners)						
T2-1	3	235.89	0.00	0.57	−114.72	0.57
T2-2	4	237.56	1.67	0.25	−114.40	0.82
T2-3	5	239.92	4.02	0.08	−114.38	0.90
T2-4	6	240.76	4.87	0.05	−113.56	0.95
T2-5	7	241.91	6.02	0.03	−112.84	0.98
T2-6	7	242.97	7.08	0.02	−113.37	1.00
T2-7	9	246.43	10.54	0.00	−112.34	1.00
T2-8	10	248.88	12.98	0.00	−112.10	1.00
Cort10 (difference between partners)						
T2-3	5	181.40	0.00	0.56	−84.45	0.56
T2-1	3	183.62	2.22	0.19	−88.35	0.75
T2-4	6	184.22	2.82	0.14	−84.28	0.89
T2-2	4	186.26	4.85	0.05	−88.33	0.94
T2-6	7	186.87	5.47	0.04	−83.89	0.97
T2-5	7	187.64	6.24	0.02	−84.27	1.00
T2-7	9	193.73	12.33	0.00	−83.37	1.00
T2-8	10	197.64	16.24	0.00	−83.03	1.00
Cort30 (difference between partners)						
T2-2	4	209.17	0.00	0.50	−99.79	0.50
T2-3	5	211.18	2.01	0.18	−99.34	0.68
T2-1	3	211.74	2.57	0.14	−102.41	0.82
T2-4	6	213.21	4.04	0.07	−98.78	0.88
T2-5	7	213.69	4.52	0.05	−97.30	0.93
T2-6	7	213.86	4.69	0.05	−97.39	0.98
T2-7	9	215.84	6.67	0.02	−94.42	1.00
T2-8	10	219.98	10.81	0.00	−94.20	1.00

Table 4
Model details for individual HPA axis activity showing fixed coefficients, standard errors (SE), t-values, degrees of freedom (df), *p*-values, and 95 % confidence intervals. Reference levels for the estimated coefficients for early life treatment was CON, for breeding stage was pre-breeding, and for sex was female. Significant *p*-values are highlighted in bold.

Predictor	Coefficient	SE	df	*t*-Value	*p*-Value	*95 % CI*	
Baseline corticosterone							
(Intercept)	**−0.93**	**0.18**	**27.26**	**−5.17**	**<0.001**	**−1.29**	**−0.58**
Body mass	−0.23	0.16	114.71	−1.46	0.146	−0.53	0.07
Clutch size	−0.05	0.16	106.14	−0.30	0.763	−0.35	0.26
Corticosterone after 10 min of restraint stress (cort10)							
(Intercept)	**9.07**	**1.36**	**39.13**	**6.68**	**<0.001**	**6.55**	**11.61**
Body mass	**−1.49**	**0.64**	**52.71**	**−2.31**	**0.025**	**−2.70**	**−0.29**
Clutch size	0.08	0.63	47.53	0.12	0.903	−1.10	1.26
Own treatment	**2.60**	**1.23**	**46.39**	**2.10**	**0.041**	**0.29**	**4.89**
Mate treatment	1.51	1.27	43.06	1.19	0.239	−0.84	3.88
Breeding stage	**−4.29**	**1.07**	**48.98**	**−4.00**	**<0.001**	**−6.32**	**−2.25**
Sex	−0.57	1.39	29.29	−0.41	0.683	−3.20	2.06
Corticosterone after 30 min of restraint stress (cort30)							
(Intercept)	**17.83**	**2.43**	**32.32**	**7.32**	**<0.001**	**13.32**	**22.33**
Body mass	−1.60	1.10	45.04	−1.46	0.152	−3.66	0.50
Clutch size	−0.44	1.18	26.81	−0.37	0.713	−2.63	1.73
Own treatment	−2.57	2.28	19.88	−1.13	0.274	−6.87	1.61
Mate treatment	**5.08**	**2.37**	**24.64**	**2.14**	**0.043**	**0.39**	**9.47**
Breeding stage	**−6.62**	**1.53**	**32.52**	**−4.34**	**<0.001**	**−9.64**	**−3.62**
Sex	**−5.88**	**2.31**	**27.03**	**−2.54**	**0.017**	**−10.21**	**−1.54**

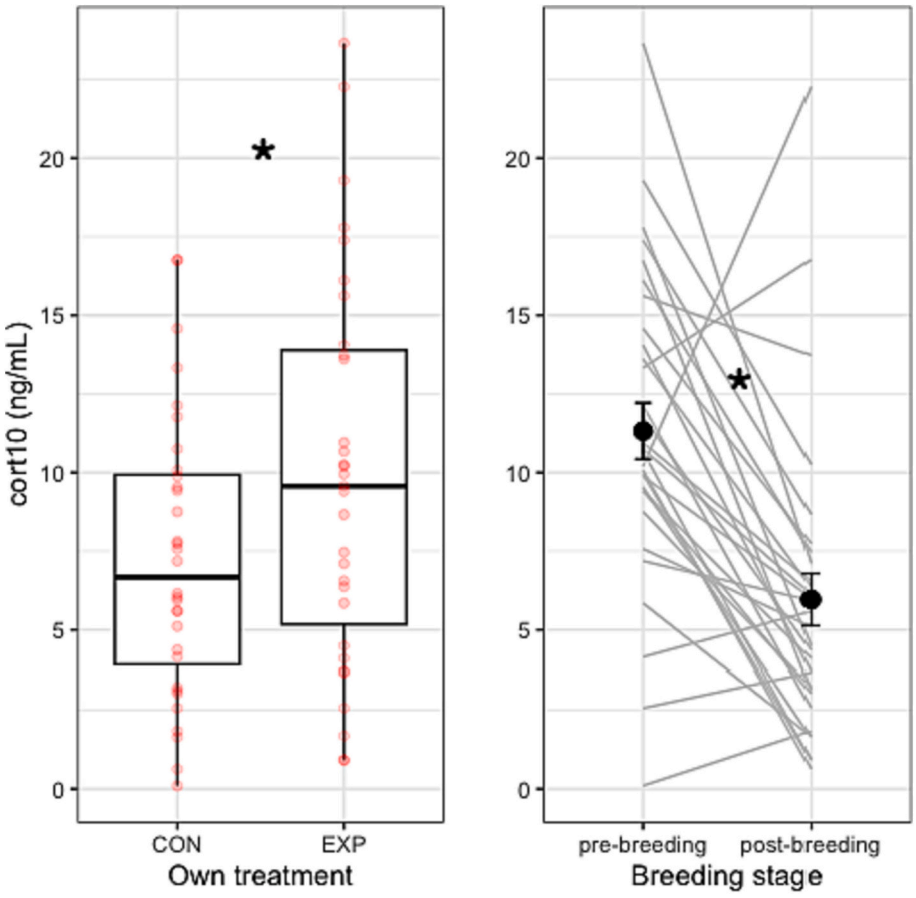

Fig. 2. Stress-induced corticosterone was influenced by early life treatment and breeding stage. EXP birds that received postnatal GCs had higher corticosterone levels after 10 min of restraint stress than CON birds that received only vehicle (A), showing that the effect of postnatal GCs on HPA axis stress reactivity persists until adulthood. Each point in represents a corticosterone sample. Corticosterone levels after 10 min of restraint stress was also lower after breeding (B). Each line represents an individual, with the point representing means for each breeding stage and error bars showing SE.

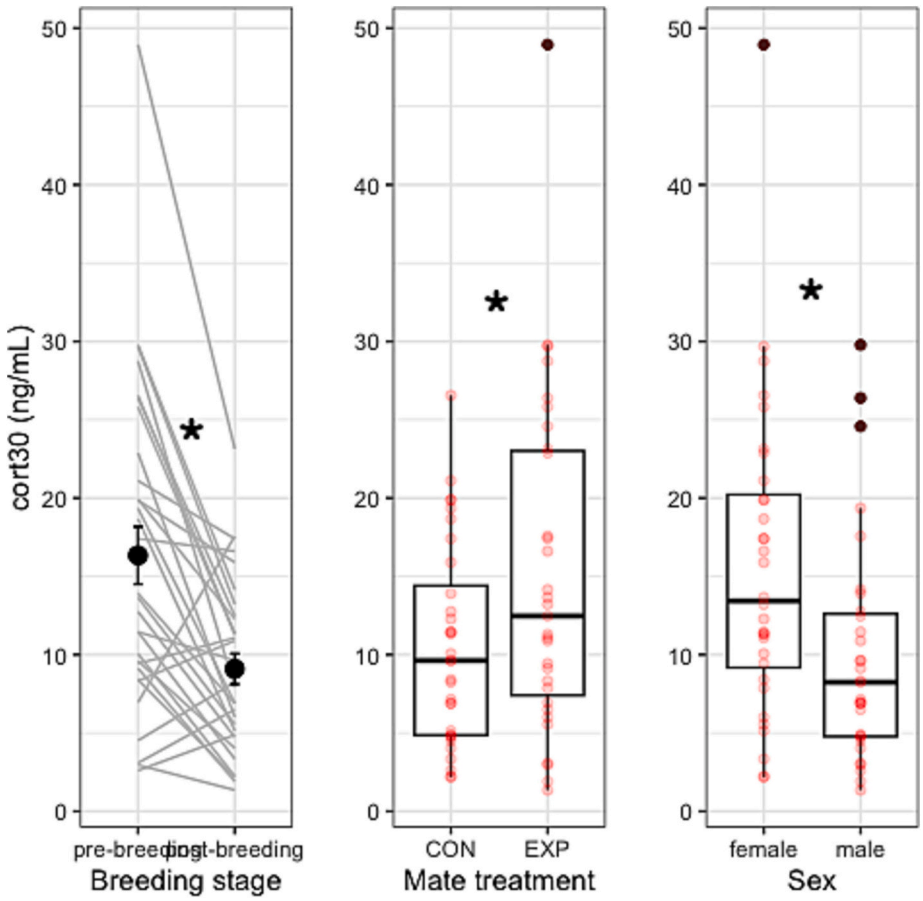

Fig. 3. Corticosterone levels after 30 min of restraint stress was influenced by breeding stage, mate treatment, and sex. Cort30 was reduced after breeding (A). Each line represents an individual, with the point representing means for each breeding stage and error bars showing SE. Corticosterone after 30 min of restraint stress was higher in than birds with an EXP than a CON mate (B) and females compared to males (C). Each point represents a corticosterone sample. Removal of a potential outlier (corticosterone = 49 ng/mL) did not change the results.

3.2. Does HPA axis reactivity of reproductive partners become more similar after breeding?

Early life elevation of GCs affected the degree to which stress-induced HPA axis activity of partners converged after breeding. Table 5 reports the model coefficients of the final models for difference between partners in *baseline*, *cort10*, and *cort30*. The difference between partners in baseline corticosterone was only weakly predicted by *breeding stage*, tending to be smaller after breeding. On the other hand, the difference between partners in corticosterone levels after 10 min of restraint stress was strongly predicted by *female treatment*. Specifically, the difference between partners was greater in pairs with an EXP female compared to pairs with a CON female (Fig. 4A). *Breeding stage* also contributed the model but did not strongly influence difference between partners after 10 min of restraint stress. The difference between partners in corticosterone levels after 30 min of restraint stress was affected by *breeding stage*, as the difference was smaller after breeding compared to before breeding (Fig. 4B). Female treatment also contributed to the

Table 5
Model details for difference between partners in HPA axis activity showing fixed coefficients, standard errors (SE), z-values, degrees of freedom (df, or adjusted SE for averaged models), *p*-values, and 95 % confidence intervals. Reference levels for the estimated coefficients for early life treatment was CON and for breeding stage was pre-breeding. Significant *p*-values are highlighted in bold.

Predictor	Coefficient	SE	df	z-Value	*p*-Value	*95 % CI*	
Difference in baseline corticosterone							
(Intercept)	**1.33**	**0.29**	**0.29**	**4.56**	**<0.001**	**0.76**	**1.91**
Breeding stage	−0.11	0.30	0.30	0.37	0.711	−1.24	0.54
Difference in cort10							
(Intercept)	**3.30**	**1.38**	**27.00**	**2.40**	**0.024**	**0.66**	**5.95**
Breeding stage	−0.35	1.56	27.00	−0.22	0.825	−3.34	2.64
Female treatment	**4.39**	**1.55**	**27.00**	**2.82**	**0.009**	**1.41**	**7.38**
Difference cort30							
(Intercept)	**9.66**	**2.24**	**2.34**	**4.12**	**<0.001**	**5.06**	**14.25**
Breeding stage	**−5.72**	**2.46**	**2.58**	**2.22**	**0.027**	**−10.77**	**−0.66**
Female treatment	1.67	2.49	2.59	0.65	0.519	−3.11	7.88

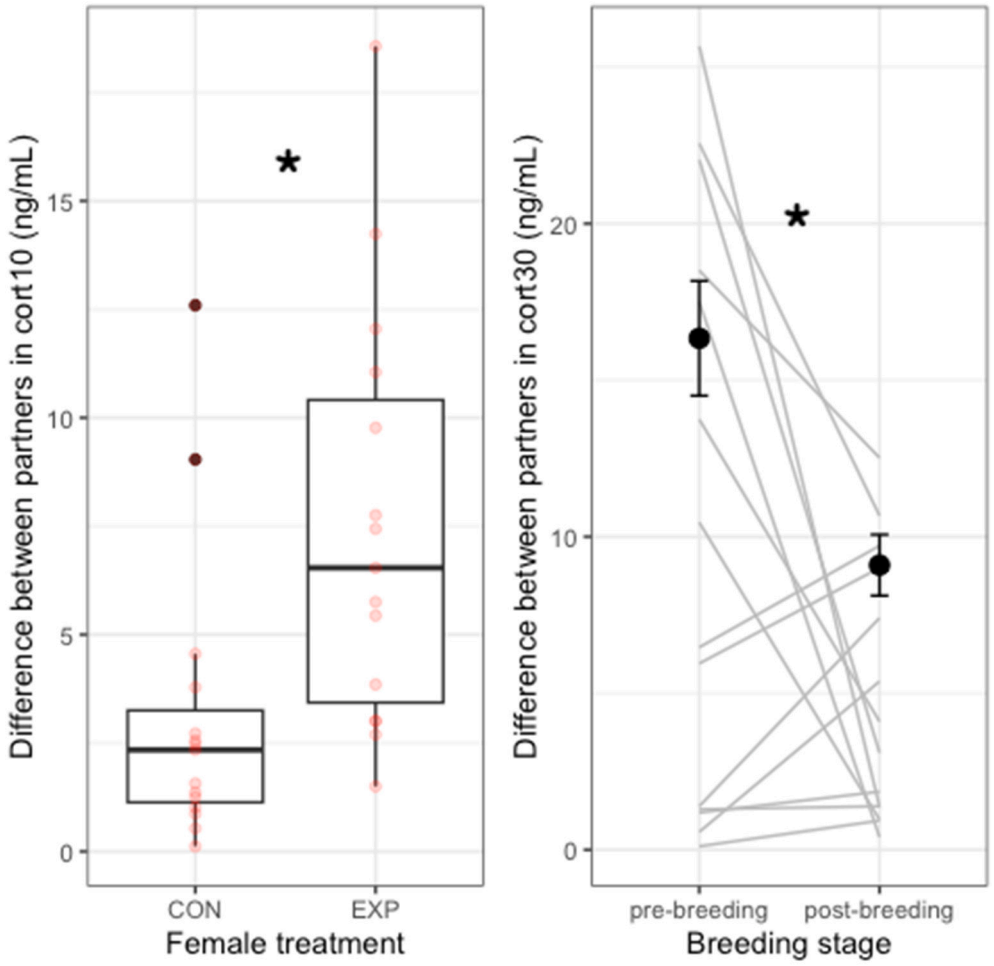

Fig. 4. Difference between reproductive partners in stress-induced corticosterone levels. After 10 min of restraint stress, the difference was greater in pairs which had an EXP female, suggesting that EXP decreases hormonal similarity between partners (A). Each point represents a corticosterone sample. Difference between partners in corticosterone levels after 30 min of restraint stress was reduced after breeding (B), indicating that stress reactivity is moderated by social partners, even if they are absent while the stressor is being experienced. Each line represents an individual, with the point representing means for each breeding stage with error bars showing SE.

model but did not strongly predict difference between partners after 30 min of restraint stress.

We further explored reasons why we found convergence of stress-induced corticosterone levels between partners but did not find that partners affected stress-induced corticosterone levels of individuals. We asked whether partners that showed a larger difference in corticosterone levels before breeding would show a larger change in individual HPA axis reactivity. We calculated 1) the difference in corticosterone levels of partners before breeding and 2) how much the female changed in corticosterone levels compared to the male (defined as the change in corticosterone of a female from pre- to post-breeding minus the change in corticosterone of her mate from pre- to post-breeding). Spearman's rank correlation of these two variables showed that higher dissimilarity between partners in baseline and cort30 before breeding was positively associated with one partner showing a greater change in corticosterone compared to its mate (baseline corticosterone: $r(27) = 0.653$, $p < 0.001$; for cort10: $r(11) = 0.429$, $p = 0.146$; for cort30: $r(12) = 0.833$, $p < 0.001$; Fig. 5). In summary, the greater the initial difference in corticosterone between partners before breeding, the more one pair member would change in baseline or cort30.

4. Discussion

Our study aimed to provide new insights into stress contagion by examining whether adult individuals become more similar to their reproductive partner in how strongly their HPA axis responds to a stressor. We found that after breeding together, zebra finch reproductive partners became more similar to each other in corticosterone responses after 30 min of restraint stress. However, being paired with a partner that had been postnatally exposed to elevated glucocorticoids did not significantly alter an individual's HPA axis reactivity to stress, even though individuals that had been postnatally exposed to elevated glucocorticoids did show a stronger corticosterone response after 10 min of restraint stress. Therefore, these results support two of our hypotheses: 1) that postnatal glucocorticoid exposure would cause individuals to react to stressors with an exaggerated corticosterone response in adulthood, and 2) that partners would become more similar in stress-induced corticosterone levels after being paired. However, they do not support our other two hypotheses that 1) CON individuals will increase stress-induced corticosterone after being paired with an EXP partner, or 2) that EXP individuals paired with another EXP individual would have the greatest levels of stress-induced corticosterone.

4.1. Enduring effects of early life stress on HPA axis reactivity

The elevated corticosterone levels after 10 min of restraint stress in EXP birds support our first hypothesis and provide additional evidence that early postnatal conditions persistently alter HPA axis response to

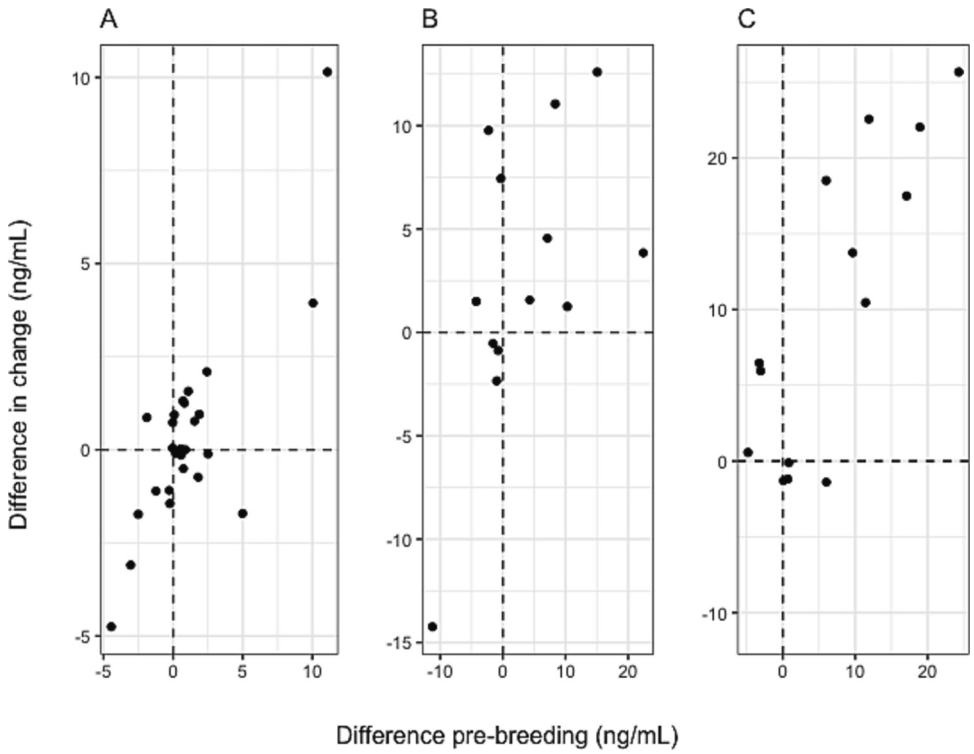

Fig. 5. Relationship between initial difference in corticosterone levels of partners before breeding (x-axis) and the difference in change of corticosterone levels of the female versus the male (y-axis). Partners with corticosterone levels that were more different before breeding changed more in baseline corticosterone (A) and cort30 (C), but not cort10 (B). Values close to 0 on the x-axis indicate very little difference between partners before breeding. Values close to 0 on the y-axis indicate that both partners changed equally. Each data point represents a pair, and units are ng/mL of corticosterone.

stressors in birds (Banerjee et al., 2011; Grace and Anderson, 2018; Pravosudov and Kitaysky, 2006; Schoech et al., 2011; Spencer et al., 2009). Specifically, elevating levels of corticosterone for 2 weeks in the first month of life heighted zebra finches' corticosterone response to stressors almost one year later. This extends the findings by Spencer et al. (2009) who showed that the same manipulation affected birds when they were 60 days old. Unlike Spencer et al., however, we did not find that postnatal GCs elevated corticosterone after 30 min of a stressor. This absence could be due to fine-tuning of GC feedback mechanisms that help to mitigate the deleterious effects of sustained exposure to high levels of GCs (e.g. (Kriengwatana et al., 2014; Rich and Romero, 2005). Nonetheless, some studies manipulating early postnatal stress in birds find that stress-induced corticosterone levels are depressed rather than elevated, or only temporarily altered (Careau et al., 2014; Crino et al., 2014; Grace et al., 2020; Lendvai et al., 2009; Marasco et al., 2012). For zebra finches, genetic differences between subpopulations of laboratory zebra finches could account for some of these discrepancies (Crino et al., 2014; Forstmeier et al., 2007). An additional consideration is variation in housing and husbandry conditions, which are also important sources of variation potentially contributing to programming of the HPA axis (Gerdin et al., 2012; Nevalainen, 2014). Finally, early life stress may have varying effects on birds depending on what type of stressor is used. We acknowledge that for our study, increasing corticosterone to mimic stress exposure does not fully replicate exposure animals to natural stressors (MacDougall-Shackleton et al., 2019). The reason we chose to use this method is because we could standardise exposure across individuals and directly show that stimulation of the HPA axis by corticosterone has long-term effects on its response to acute stressors.

4.2. Does HPA axis activity of individuals change depending on developmental history of their reproductive partner?

We found limited evidence to support our hypothesis that an individual's corticosterone levels at baseline or after a stressor changed depending on the conditions their partner experienced in early life. Instead, we found that stress-induced corticosterone levels were reduced after breeding compared to before breeding regardless of own or partner early life conditions. Such dampening of the response may avoid interference of prolonged high GCs with reproductive behaviour (Kirby et al., 2009; Silverin, 1986). Interestingly, we also found that corticosterone levels after 30 min of restraint stress were higher in birds with an EXP mate, independent of breeding stage.

Individual HPA axis activity may have been unaffected by their partners in our experiment for several reasons. First, the duration of the experiment was relatively short: pre- and post-breeding samples were obtained approximately 3 months apart. In great tits, hormonal similarity between partners increased incrementally over the successive years that a pair remained together (Ouyang et al., 2014). Thus, changes in an individual's HPA axis activity may have been too subtle for us to detect after only 3 months of being with their partner. Sensitivity analyses with G*Power suggests that we would have had a 90 % chance of detecting individual change in cort10 with a moderate effect size ($f^2 = 0.17$), which supports the possibility that only slight changes in HPA axis activity occurred over the short duration of our experiment (Erdfelder et al., 2009; Faul et al., 2007). We further believe that it takes longer than 3 months before statistically significant changes are observed because we found that partners become more similar in HPA axis reactivity (discussed in detail later). This implies that an individual's HPA axis activity is only very gradually affected by their partner.

Alternatively, individual HPA axis activity may not have changed because we did not pair birds that differed sufficiently in corticosterone responses to stressors. Our results provide some support for this, as a larger difference between partners in baseline and cort30 levels before breeding predicted that one partner would change more than the other partner (Fig. 5). Future experiments could pair individuals of known high and low HPA axis reactivity to stressors and measure corticosterone

response to stressors across several breeding attempts. This would clarify the possibility that reproductive partners become more similar in how they physiologically respond to stressors after repeated breeding experience and/or time together.

Finally, an individual's HPA axis response to stressors might be plastic only under certain conditions to reduce any costs of becoming similar to partners. An individual's HPA axis response to stress is shaped by early life experiences (DeWitt et al., 1998; Murren et al., 2015) but it remains unclear whether the magnitude of the HPA axis response to stressors can be futher influenced by experiences in adulthood. Assuming that it can be influenced in adulthood and during reproduction, then changes in HPA axis response to stressors may only occur during periods where tight coordination between partners are necessary (e.g. during incubation and chick rearing). It would be interesting to quantify whether the magnitude of HPA axis response to stress changes during parental care and whether the changes are associated with parental care activities.

4.3. Early life conditions affect similarity of HPA axis activity of reproductive partners

Our hypotheses regarding the effect of early life exposure to glucocorticoids on physiological matching was only partially supported. Corticosterone levels after 30 min of restraint stress of partners became more similar after breeding regardless of an individual's own or their mate's early life experiences. This shows that partners converge in physiological responses to stressors, which could reduce sexual conflict and enable individuals to be more attuned to the state of their partner (Griffith, 2019). However, we did not find that pairs with two EXP individuals had the highest corticosterone response to stress, nor that they converged more or less with their partners compared to pairs with at least one CON individual. However, pairs with an EXP female were more dissimilar in corticosterone levels after 10 min of restraint stress, both before and after breeding. This suggests that postnatal GC exposure in females may impede convergence of HPA axis between reproductive partners. Future work could explore reasons for this impediment by investigating changes in parental care and coordination behaviours.

5. Conclusions

Overall, our results provide evidence of stress contagion by showing that the HPA axis response to stressors of reproductive partners become more similar after breeding. Nonetheless, significant changes in how strongly of the HPA axis of individuals responds to stressors take longer to manifest. Stress contagion may explain how individuals that did not themselves experience early life stress can nevertheless be negatively affected by early life stress (Monaghan et al., 2012). It also shows that social transmission of HPA axis reactivity may be diminished between partners if the female experienced elevated GC levels during early postnatal life. Altogether, our findings highlight a need for a deeper understanding of the causes and fitness implications of convergence of HPA axis response to stressors between reproductive partners, and to determine whether convergence could also occur in non-breeding contexts between social partners in larger groups.

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CRediT authorship contribution statement

Buddhamas P. Kriengwatana: Conceptualization, Data curation, Formal analysis, Funding acquisition, Investigation, Methodology, Writing – original draft, Writing – review & editing, Project administration. **Christopher J. Marshall:** Formal analysis, Investigation, Writing – original draft, Writing – review & editing. **Tyler Stevenson:** Resources, Supervision, Writing – review & editing. **Pat Monaghan:** Conceptualization, Funding acquisition, Methodology, Resources, Supervision, Writing – review & editing.

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Appendix A. Supplementary data

Supplementary data to this article can be found online at https://doi.org/10.1016/j.yhbeh.2024.105508.

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