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Genetic inheritance and environment determine endocrine plasticity to urban living

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As urban areas continue to expand globally, understanding how and why species respond to novel habitats becomes increasingly important. Knowledge of the mechanisms behind observed phenotypic changes in urban animals will enable us to better evaluate the impact of urbanization on current and future generations of wildlife. Physiological changes, such as those involved in the endocrine stress response, may allow individuals to inhabit and thrive in urbanized areas, but it is currently unknown how these changes arise in natural populations. In this study, we performed a four-way cross-foster experiment in free-living house wren chicks, *Troglodytes aedon*, to disentangle whether differences in baseline corticosterone between urban and rural individuals are a result of genetic and/or plastic mechanisms during development. We found that urban chicks already had higher corticosterone levels than their rural counterparts on the day they hatched, which suggests a possible genetic component to the corticosterone phenotype. However, rural offspring that were moved to an urban environment significantly increased their corticosterone levels, mimicking those of urban offspring. Our findings suggest that, although differences in baseline corticosterone concentrations between urban and rural individuals may have a genetic component, plasticity plays a pivotal role and can modify the corticosterone phenotype in response to the environment experienced in the first two weeks of life.

1. Introduction

Rapid human population growth has caused the most dramatic environmental change in the twenty-first century, with 54% of the world's human population living in urban areas and 3% of total land cover considered urban [1]. Urban land cover continues to expand, leading to the destruction of natural habitat and reduced biodiversity [2]. Species responses to urbanization vary greatly, with some unable to occupy these novel habitats, while others thrive [3,4]. A growing list of urban ecology and evolution research has shown that behavioural, morphological and physiological phenotypes of individuals differ between urban and rural areas [5–10], and those that possess certain traits are able to colonize urban areas [11,12]. Although phenotypic differences are well documented among conspecifics that live in urban areas as opposed to their rural counterparts, one outstanding question in the study of urban evolution is how and why these phenotypes arise [13].

There are few experimental studies that were performed in both urban and rural sites during offspring development (although see [14–17]), which limits our understanding of the mechanisms that contribute to the observed phenotypic differences between urban and rural organisms. One potential mechanism that allows individuals to persist in urban environments may be the endocrine system, as hormones can integrate internal condition and external cues. In particular, the hypothalamic–pituitary–adrenal (HPA) axis produces glucocorticoids that allow individuals to rapidly respond to environmental change [18]. For

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example, studies have shown that corticosterone (the main glucocorticoid in birds) levels are different between urban and rural environments, but this difference may differ by year, site, species and sex (see [19,20] for reviews). Moreover, differences in hormone levels in the eggs due to maternal effects and/or poor conditions during rearing can remodel the HPA axis (see [21,22] for reviews), and increases in glucocorticoids during development can lead to phenotypic differences later in life [23,24]. Therefore, glucocorticoids may be a mechanism by which individuals adjust to urban living. However, the labile nature of this hormone in responding to external change makes it difficult to understand how selection would act on this phenotype [25]. Therefore, whether the glucocorticoid phenotype is the result of genetic or plastic response to the environment is currently unknown.

To disentangle the genetic and plastic contribution to glucocorticoid levels, we performed an unprecedented cross-foster experiment between urban and rural populations of house wren chicks, *Troglodytes aedon*. In our populations, urban adults consistently have higher baseline corticosterone levels than rural adults [26]. We predicted that if baseline corticosterone concentrations are a result of purely genetics, then urban chicks fostered to a rural environment would maintain the urban phenotype during development. If baseline corticosterone concentrations are a result of pure phenotypic plasticity, then urban chicks fostered to a rural environment would change their phenotype to a rural one. Lastly, if baseline corticosterone concentrations are a result of both mechanisms, then we would expect intermediate concentrations in between these two extremes (figure 1).

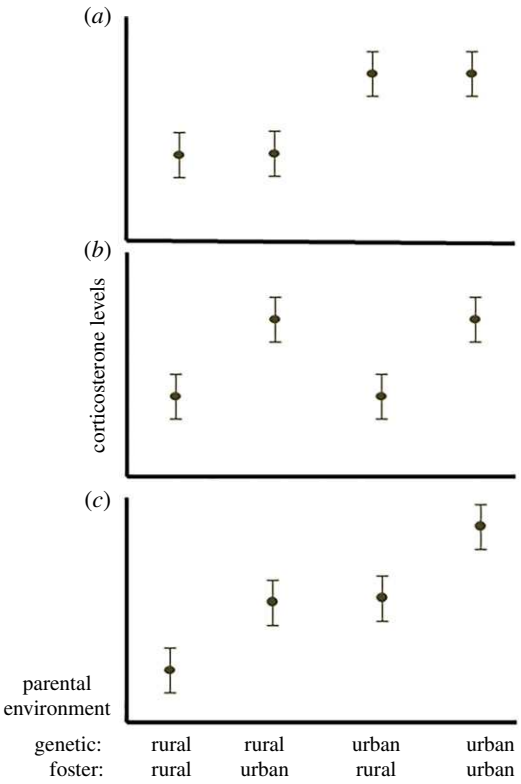

Figure 1. Hypothetical results of house wren chicks after the cross-foster if the glucocorticoid phenotype is a result of (*a*) genetic, (*b*) phenotypic plastic or (*c*) both mechanisms.

2. Methods

We conducted our cross-fostering study from May to July 2017 and 2018 at one urban location in Reno, Nevada, USA, and one rural location in Sparks, Nevada, USA (see [26] for detailed description of the field sites). Briefly, we estimated the land use of each study site, using the validated method described by Seress *et al*. [27]. The urban site has more cells with increased building density and paved surfaced and decreased vegetation density [26]. We monitored house wren nests regularly to determine the hatch date (day 0). In both years, average hatch date did not differ between the two sites (urban: 2 June ± 0.7, rural: 5 June ± 1.1). On day 0 or 1, we obtained a blood sample from all chicks (2.1 ± 0.02 min; mean ± s.e.). Chicks sampled on day 0 or 1 did not differ in their corticosterone levels ($p > 0.2$). Any chick that was blood sampled past 3 min was not included in the analyses. There was over 95% chick survival at both sites, and any chick that did not survive to day 15 was also not included in the repeated measures design (see below). After weighing all chicks (nearest 0.1 g), we individually marked them with toenail polish (reapplied on day 4 if necessary) and toenail clipping for identification. We made quartets of two urban nests and two rural nests to perform a four-way cross-foster by fostering chicks both within and among sites. Briefly, the chicks in each nest were randomly divided into two groups, and one group from each nest was fostered into another nest at the same site (i.e. urban to urban or rural to rural) and the other group from each nest was fostered into a nest at the opposite site (i.e. urban to rural or rural to urban), such that no parent raised their own genetic offspring. We cross-fostered 119 offspring among 29 urban and 26 rural nests both within and among sites (rural to rural $n = 30$, rural to urban $n = 28$, urban to rural = 30, urban to urban = 31). There were four urban and four rural nests in which we could not find a quartet to cross-foster with, so we fostered them within sites. Removing these nests did not alter our results. During the cross-foster process, we transported the chicks in a heated nest-box to either a rural or an urban nest that hatched on the same day (maximum transport time: 20 min, average transport time: 10 min). On day 8, we banded the nestlings with a uniquely numbered tarsal band. On day 15 (house wrens fledge around day 17 and reach asymptotic weight at day 15), we obtained another blood sample from all chicks (1.7 ± 0.02 min) and weighed them to the nearest 0.1 g.

(a) Corticosterone assay

We used enzyme-linked immunosorbent assay kits (Enzo Life Sciences; Farmingdale, NY, USA) following the manufacturer's instructions (Lot 05011701). See [26] for validation of this assay for house wrens. Based on optimization, we diluted plasma 1 : 40 in assay buffer with 0.5% steroid displacement reagent. We randomized samples across plates but an individual's day 0 and day 15 plasma were always next to each other on the same plate. We included a standard curve on each plate that ranged from 32 to 20 000 pg ml^{-1}. The assay sensitivity was 28 pg ml^{-1}. To calculate intra- and inter-plate variation (CV), we included pooled house wren plasma, assayed in triplicate. The intra-plate CV was 9.8%, and the inter-plate CV (5 plates) was 5.6%.

(b) Statistical analyses

We conducted statistical analyses using R (v. 3.4.3). We performed all linear mixed models (LMMs) with the *lmer* function in the *lme4* package [28], and we ran Tukey post hoc tests using the *lsmeans* function implemented in the *lsmeans* package [29]. All final models met assumptions of normality and homoscedasticity of residual errors, and significance was taken at $\alpha = 0.05$. We used an LMM with repeated measures to test if corticosterone levels at day 0 and corticosterone levels at day 15 were different

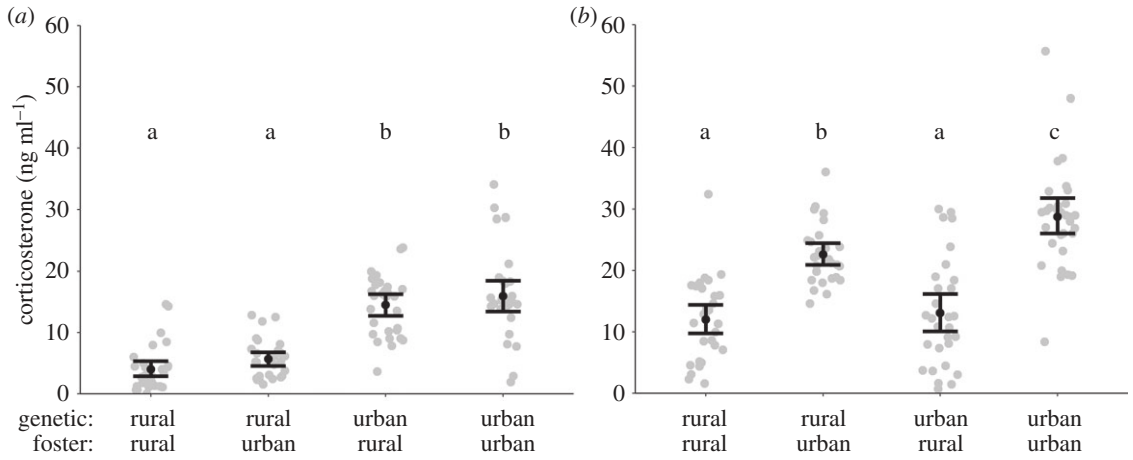

Figure 2. Corticosterone levels at (*a*) day 0 and (*b*) day 15 of house wren chicks that were cross-fostered between an urban and rural site. The genetic parent is the location of the genetic parent, and the foster parent is the location of the foster parent. Grey circles indicate raw data and plotted are mean and 95% confidence intervals. Different letters indicate significant differences among groups in the post hoc tests.

Table 1. Model estimates for the effect of cross-foster treatment on corticosterone levels of nestling house wrens. Individual estimates are given from summary statistics of the LMM. Random effects include individual ID and parental and natal nest identity. Time is either day 0 or day 15 for blood sampling. U, urban; R, rural for cross-foster groups, (e.g. UR is for offspring that started in an urban nest and was cross-foster to a rural nest).

variable	estimate (95% CI)	*t*	*p*-value
LMM for corticosterone levels			
(intercept)	−4.83 (−10.19, 0.50)	−1.730	0.08
cross-foster group RU (*reference group RR*)	4.06 (0.77, 7.39)	2.355	0.02
cross-foster group UR	12.84 (9.52, 16.19)	7.385	<0.001
cross-foster group UU	13.50 (10.39, 16.64)	8.302	<0.001
time (day 15)	7.99 (5.12, 10.87)	5.371	<0.001
date	0.24 (0.11, 0.38)	3.584	0.003
year	−1.69 (−3.64, 0.23)	−1.688	0.10
cross-foster group RU × time (day 15)	8.94 (4.80, 13.08)	4.173	<0.001
cross-foster group UR × time (day 15)	−9.43 (−13.50, −5.36)	−4.482	<0.001
cross-foster group UU × time (day 15)	4.75 (0.72, 8.79)	2.277	0.02
random effects		**variance**	**s.d.**
chick ID		<0.001	<0.001
foster nest ID		<0.001	<0.001
parental nest ID		2.95	1.72

within individuals before and after cross-fostering, with the interaction of treatment (rural to rural, urban to urban, urban to rural and rural to urban) and time (day 0 or day 15) as a fixed effect and year and date of capture as covariates. Individual ID and nest ID of the genetic and foster parents were included as random effects. We calculated the variance explained by random effects using the package sjPlot and model estimates [30]. We ran Tukey post hoc multiple comparison tests for the interaction to test whether each treatment group was different from others. We followed the same analysis for individual body mass changes as with corticosterone changes. We initially included brood size as a covariate, but due to lack of variation (90% of nests had four or five offspring) and statistical significance, we removed it from all models.

We calculated repeatability in baseline corticosterone between days 0 and 15 using the within and between-variance components in an LMM, using the restricted maximum-likelihood method (REML) with bird identity as the grouping random factor [31,32]. We used the package rptR developed and explained in [32] to calculate repeatabilities. Briefly, confidence intervals and standard errors were calculated from parametric bootstraps that created the distributions of likelihood ratios (1000 times).

3. Results

Urban chicks at day 0 had higher corticosterone levels than rural chicks (figure 2*a* and tables 1 and 2). At day 15, the chicks that were fostered to urban environments had higher corticosterone levels than chicks that were fostered to rural environments (figure 2*b* and tables 1 and 2). Specifically, within-individual changes from day 0 to day 15 showed that corticosterone levels increased in chicks moved from rural nests to rural nests (coef = −8.0, s.e. = 1.5, $z = -5.4$, $p < 0.0001$; figure 3*a*), in chicks moved from rural nests to

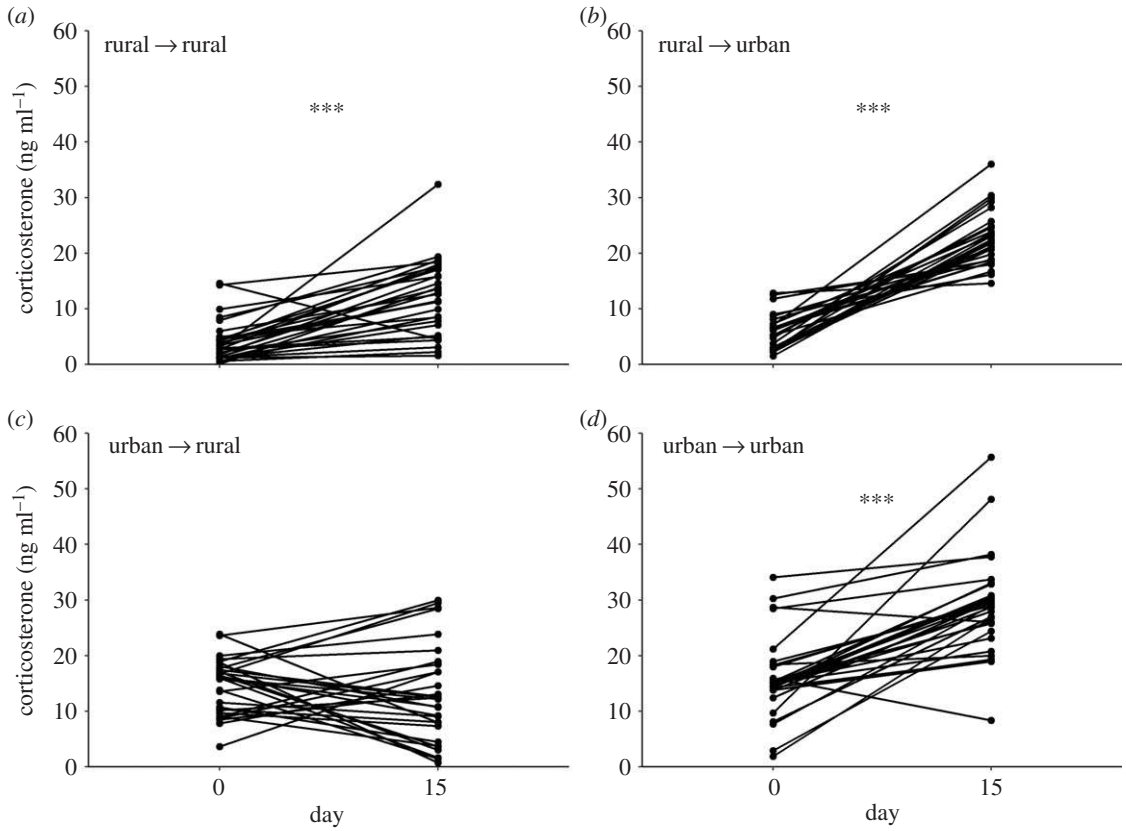

Figure 3. Within-individual changes in corticosterone levels of house wren chicks that were fostered (*a*) within a rural site, (*b*) from a rural site to an urban site, (*c*) from an urban site to a rural site, and (*d*) within an urban site. Asterisks indicate significant ($p < 0.0001$) within-individual changes for each group following post hoc tests.

Table 2. Estimates from Tukey post hoc multiple comparisons to test differences among cross-foster groups. Individual estimates are given from post hoc test on the interaction between time (day 0 or day 15) and cross-foster group. U, urban; R, rural for cross-foster groups (e.g. UR is for offspring that started in an urban nest and was cross-foster to a rural nest).

	estimate	s.e.	d.f.	*t*	*p*-value
post hoc comparisons of corticosterone levels at day 0 (hatch date)					
cross-foster group					
RR–RU	−4.06	1.76	144	−2.30	0.299
RR–UR	−12.84	1.77	158	−7.25	<0.001
RR–UU	−13.50	1.64	167	−8.21	<0.001
RU–UR	−8.78	1.68	121	−5.24	<0.001
RU–UU	−9.44	1.68	157	−5.62	<0.001
UR–UU	−0.66	1.57	110	−0.42	0.999
post hoc comparisons of corticosterone levels at day 15 (~2 days before fledge)					
cross-foster group					
RR–RU	−13.00	1.76	144	−7.38	<0.001
RR–UR	−3.41	1.77	158	−1.92	0.537
RR–UU	−18.25	1.64	167	−11.10	<0.001
RU–UR	9.59	1.68	121	5.72	<0.001
RU–UU	−5.25	1.68	157	−3.13	0.042
UR–UU	−14.85	1.57	110	−9.439	<0.001

urban nests (coef = −16.9, s.e. = 1.5, $z = -11.0$, $p < 0.0001$; figure 3*b*) and in chicks moved from an urban nest to an urban nest (coef = −12.7, s.e. = 1.5, $p < 0.0001$; figure 3*d*). By contrast, chicks moved from urban nests to rural nests did not change their corticosterone levels (coef = 1.4, s.e. = 1.5, $z = 1.0$, $p = 0.98$; figure 3*c*). Although three of the four groups increased corticosterone levels, levels of this hormone were highest in chicks fostered to urban nests (figure 2*b* and tables 1 and 2). Body mass of the chicks did not differ between the four treatment groups on day 0 ($p > 0.05$), but

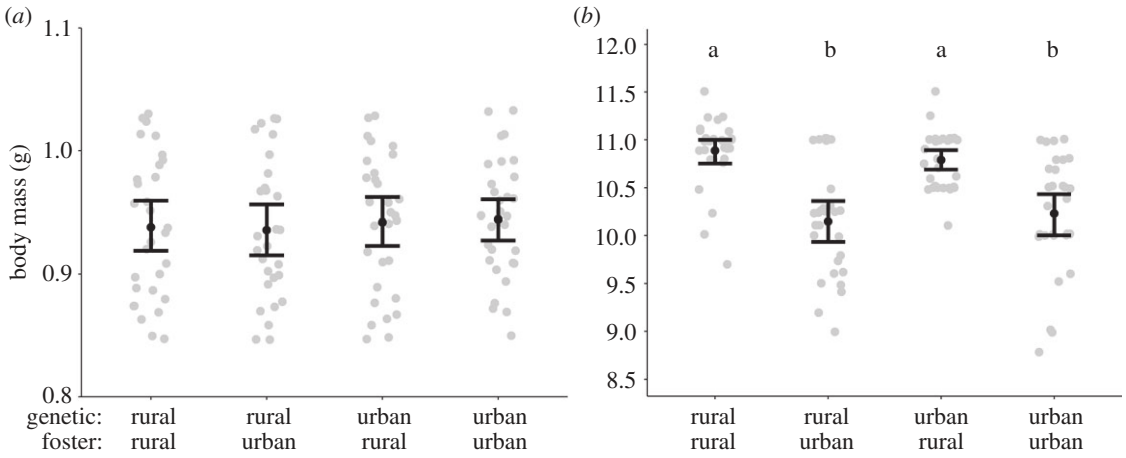

Figure 4. Body mass at (*a*) day 0 and (*b*) day 15 of house wren chicks that were cross-fostered between an urban and rural site. Note differences in scale. The genetic parent is the location of the genetic parent, and the foster parent is the location of the foster parent. Grey circles indicate raw data and plotted are mean and 95% confidence intervals. Different letters indicate significant differences among groups in the post hoc tests.

chicks fostered or remained in the urban site had lower mass than chicks fostered or remained in the rural site (figure 4; post hoc statistics: RU–RR, UU–RR, UR–RU, UU–UR: all $p < 0.0001$). Later nests had chicks with higher corticosterone levels than earlier nests (table 1). The number of fledglings did not differ between the urban and rural sites in either year ($p > 0.05$).

Baseline corticosterone concentrations were not repeatable from day 0 to day 15 ($R = 0.02$ (0, 0.2), s.e. = 0.06, $p = 0.45$). Individual ID and foster nest ID did not explain any variation in corticosterone levels, but the genetic nest ID explained 3% of the variance in corticosterone levels (table 1).

4. Discussion

We conducted a four-way cross-foster experiment to test the contribution of genetic and plastic mechanisms during development of a hormonal phenotype. We provide evidence that baseline corticosterone levels are plastic during development and increase in response to living in the urban environment. Combined with changes in body mass showing heavier rural chicks than urban chicks, we suggest there are correlated phenotypic adjustments to urban life [33].

During two weeks of development within a nest, house wren chicks that were moved from the rural site to the urban site and those that were moved within each site increased their baseline corticosterone levels. If glucocorticoid concentrations were determined purely by genetics, then we would expect that all fostered chicks would match the phenotype of their siblings, (i.e. rural chicks in urban nests would have low baseline corticosterone levels). However, we found that the environment played a much larger role with experimental evidence that the foster environment shapes offspring phenotype. This finding is similar to a finding in telomeres in which rural great tit, *Parus major*, chicks had shortened telomeres once transported to an urban environment [14]. House wren chicks could be responding to a number of cues that differ between urban and rural environments [13,34], including food availability, food quality [35], light and noise pollution [36], and parasite load [37]. It is reasonable to expect that any or a combination of these cues could shape the development of the HPA axis. For example, if parents from urban areas provision their offspring with lower-quality food, then chicks in urban areas will weigh less at fledging and may have higher baseline corticosterone levels. Indeed, we found that chicks cross-fostered to the urban site had lower weights than chicks in the rural site. As hatching date did not differ between the sites, high corticosterone levels in offspring from later nests may be a result of decreasing food availability at both sites [38,39]. Although fledgling number does not differ between urban and rural sites, higher fledgling weight strongly correlates with increased survival and therefore future reproductive attempts [40]. We note that although we cross-fostered at day 0, maternal effects could have contributed to differences in yolk volume and hormone levels in the eggs and/or incubation differences [41], thus affecting the corticosterone phenotype at hatching. A recent study in house wrens found that increasing maternal corticosterone increased offspring survival, but only when these offspring were raised by control mothers [42]. Urban environments are often heat islands [43], so there may also be behavioural differences during incubation between urban and rural mothers due to thermal adaptation.

Why is it that urban chicks did not decrease their corticosterone levels during development if they were moved to rural areas? One possible explanation is that a general development of the HPA axis during the first weeks [44] may constrain and counteract any environmental pressures to decrease corticosterone levels in the urban to rural group. This is supported by the fact that for the remaining three of the four treatment groups (urban to urban, rural to rural, rural to urban), corticosterone levels increased from day 0 to day 15. We found in a previous study that rural adult house wrens respond to experimental increases in noise by increasing corticosterone levels [26], which is consistent with this study showing that rural offspring responded when moved to an urban site but urban ones did not when moved to a rural site. Sensitivity and perception to environmental stressors is an understudied aspect of endocrine evolution [45].

Baseline corticosterone levels of our population of house wren chicks at day 0 already differed between urban and rural sites. This difference, and the fact that the random effect of natal nest explained some of the variance in corticosterone levels, suggests that there is a component of genetics in determining the corticosterone phenotype, as adult urban

house wrens consistently have higher baseline corticosterone levels than rural adults [26]. Offspring from certain nests, regardless of origin, start out with higher corticosterone concentrations than in other nests. However, baseline corticosterone levels were not repeatable similar to other studies (reviewed in [46]), suggesting that glucocorticoids are highly plastic. This plasticity may be due to the development of the HPA axis and/or due to a change in the rearing environment regardless of the identity of the foster nest. Specifically, the identity of the foster nest was not statistically significant, whereas the identity of the genetic nest was. These natal nest effects may be due to maternal effects rather than pure genetics. It would also be interesting to be able to measure the stress response as well as negative feedback for the entire corticosterone phenotype during development. Heritability of the corticosterone phenotype has been shown in laboratory and field settings [47,48]. For 2 years, urban house wren chicks consistently had higher baseline corticosterone levels at day 0 than their rural counterparts. However, moving chicks to a different environment and growing up in this new environment, especially to an urban location, may be more challenging and select for an extreme phenotype at fledging. A system with power to detect recruitment or a long-term study to increase power (i.e. whether cross-fostered chicks have differential dispersal and recruitment rates, and measuring their adult phenotypes during breeding) would allow us to determine whether these phenotypes are adaptive and matched to the environment.

Understanding how species respond to novel habitats is becoming increasingly important as urban areas continue to grow [49]. We show that a proximate mechanism, baseline corticosterone concentrations, potentially has a genetic component at birth but responds during development to the environment in which the offspring was raised. These findings shed light on why individual phenotypes differ between urban and rural environments and how these differences may arise. Figuring out the adaptive advantages will be the next challenge in understanding urban evolution and species persistence.

Ethics. All of the data were collected under the appropriate state and federal permits and approved University of Nevada, Reno IACUC protocols.

Data accessibility. Data available from the Dryad Digital Repository: https://doi.org/10.5061/dryad.5p3q298 [50].

Authors' contributions. J.Q.O. secured funding for the study. J.Q.O. and S.D. conceived the study. J.Q.O., C.M. and S.D. collected the data. J.Q.O. and D.B. analysed the data and wrote the paper. All authors revised, edited and approved the manuscript before submission.

Competing interests. We declare we have no competing interests.

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