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Traffic noise exposure alters nestling physiology and telomere attrition through direct, but not maternal, effects in a free-living bird

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ABSTRACT

Anthropogenic impacts, such as noise pollution from transportation networks, can serve as stressors to some wildlife species. For example, increased exposure to traffic noise has been found to alter baseline and stress-induced corticosterone levels, reduce body condition and reproductive success, and increase telomere attrition in free-living birds. However, it remains unknown if alterations in nestling phenotype are due to direct or indirect effects of noise exposure. For example, indirect (maternal) effects of noise may occur if altered baseline and stress-induced corticosterone in mothers results in differential deposition of yolk steroids or other components in eggs. Noise exposure may also alter nestling corticosterone levels directly, given that nestlings cannot escape the nest during development. Here, we examined maternal versus direct effects of traffic noise exposure on baseline and stress-induced corticosterone levels, and body condition (as measured by size-corrected mass) in nestling tree swallows (*Tachycineta bicolor*). We used a two-way factorial design and partially cross-fostered eggs between nests exposed to differing levels (i.e. amplitudes) of traffic noise. For nestlings that were not cross-fostered, we also investigated the effects of traffic noise on telomere dynamics. Our results show a positive relationship between nestling baseline and stress-induced corticosterone and nestling noise exposure, but not maternal noise exposure. While we did not find a relationship between noise and body condition in nestlings, nestling baseline corticosterone was negatively associated with body condition. We also found greater telomere attrition for nestlings from nests with greater traffic noise amplitudes. These results suggest that direct, rather than maternal, effects result in potentially long-lasting consequences of noise exposure. Reduced nestling body condition and increased telomere attrition have been shown to reduce post-fledging survival in this species. Given that human transportation networks continue to expand, strategies to mitigate noise exposure on wildlife during critical periods (i.e. breeding) may be needed to maintain local population health in free-living passerines, such as tree swallows.

1. Introduction

Traffic noise in terrestrial environments is widespread and continues to increase (Barber et al., 2010). This anthropogenic impact can have negative consequences at both small and large scales. For example, noise exposure has been found to alter molecular (e.g. telomere dynamics) and physiological responses (e.g. glucocorticoids, nestling growth) in birds, which can result in decreased reproductive success and population health (Shannon et al., 2016). Identifying the pathways by which traffic noise affects birds at the molecular and physiological levels is important, especially during critical periods such as nestling development. Indirect (i.e. maternal exposure to noise) and/or direct (i.e. nestling exposure to noise) pathways may contribute to altered nestling phenotypes if traffic noise is perceived as a stressor by free-living birds (Kight and Swaddle, 2011).

The hypothalamic-pituitary-adrenal (HPA) axis becomes activated in response to stressors, resulting in the secretion of glucocorticoids (corticosterone in birds, hereafter "cort"; Wingfield et al., 1998).

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Increased cort secretion results in physiological and behavioral changes that aim to regain homeostasis by reallocating energy only towards processes that aid survival (Wingfield et al., 1998). However, if a stressor is chronic, activation of the HPA-axis may not persist (Rich and Romero, 2005). In western bluebirds (*Siala mexicana*), chronic exposure to anthropogenic noise has been found to be associated with reduced baseline cort (a measure of an individual's standard state), with noise-exposed females also suffering reduced hatching success of their clutches, compared to controls (Kleist et al., 2018). Also, reduced stress-induced cort (how strongly an individual responds to an acute stressor such as handling; Sapolsky et al., 2000) has been found in urbanized areas in adult European blackbirds (*Turdus merula*; Partecke et al., 2006), and northern cardinals (*Cardinalis cardinalis*; Wright and Fokidis, 2016). Reduced baseline and stress-induced cort may suggest that individuals are not successfully coping with chronic anthropogenic stressors (Norris et al., 1999), and can be associated with reduced synthesis of hormones from the HPA-axis and/or enhanced sensitivity to negative feedback of cort (Fries et al., 2005). However, in contrast, elevated cort concentrations (especially baseline) are also often interpreted as an indication of stress and thought to lead to negative fitness impacts (Bonier et al., 2009a). Additionally, some studies of birds have found no impact of chronic anthropogenic noise exposure on baseline cort levels (Angelier et al., 2016; Davies et al., 2017; Meillère et al., 2015; Morgan et al., 2012; Potvin and MacDougall-Shackleton, 2015a,b). These varied results may be due to species (e.g. migratory v. non-migratory) and context-dependent (e.g., geographic location, life history stages) effects of disturbance on cort.

If traffic noise serves as a stressor, nestling physiology may be altered in noisy habitats through a combination of maternal and/or direct effects. For example, female Japanese quail (*Coturnix coturnix japonica*) that were given cort implants during egg-laying laid eggs with increased cort in the yolk, and had nestlings with reduced growth and higher stress-induced cort in response to restraint from handling as adults (Hayward and Wingfield, 2004). Similarly, female barn swallows (*Hirundo rustica*) exposed to greater predation risk laid eggs with greater cort concentrations, which in turn produced fledglings of smaller body size (Saino et al., 2005). Alternatively, maternal effects may prime nestlings in a way that allows them to better cope with stressful environments: nestling great tits (*Parus major*) with greater parasite loads had lower oxidative stress if their mothers also experienced high parasite loads pre-egg laying (De Coster et al., 2012). The potential for noise exposure to result in multi-generational effects (negative or positive) warrants more research.

Traffic noise exposure may have direct effects on nestling phenotype, given that nestlings cannot escape from noise-exposed nests during development (Shors et al., 1989). For example, previous studies found that nestling noise exposure during rearing, rather than maternal noise exposure during egg-laying, was related to reduced fledging and recruitment rates in house sparrows (*Passer domesticus*; Schroeder et al., 2012). Anthropogenic disturbance has also been shown to directly alter telomere dynamics in birds. Telomeres are repetitive segments of non-coding DNA on the ends of chromosomes, which prevent degradation of necessary genetic information (Blackburn, 2001). Telomere attrition has been found to increase with baseline cort in chickens (*Gallus domesticus;* Haussmann et al., 2012) and early-life stressors in European shags (*Phalacrocorax aristotelis*; Herborn et al., 2014). Given that telomere length is positively related to survival in free-living birds (Haussmann et al., 2005), it is important to understand the effects of traffic noise on nestling cort and telomere dynamics. To date, studies suggest that direct, rather than maternal effects may alter nestling phenotype: noise exposure was associated with greater telomere attrition for lab-reared zebra finch nestlings when nestlings themselves were exposed to traffic noise, but not when parents were exposed to noise (*Taenopygia guttata*; Dorado-Correa et al., 2018) and cross-fostered great tit nestlings reared in urban habitats had increased telomere attrition, despite their natal habitats (Salmón et al., 2016). Additional experimental studies in free-living birds would be highly useful to better understand direct vs. maternal effects of noise exposure on nestling phenotype. Such studies would have the ability to isolate the effects of noise from other factors associated with urbanization, while taking into account the suite of additional stressors experienced by free-living birds.

Here, we used a two-way factorial design to partially cross-foster eggs between tree swallow (*Tachycineta bicolor*) nests that were exposed to different levels (i.e. amplitudes) of traffic noise. This experimental design allowed us to assess maternal versus direct effects of traffic noise on nestling baseline and stress-induced cort, as well as nestling body condition (as measured by size-corrected mass) in free-living birds. We also investigated noise impacts on telomere attrition in non-cross-fostered nestlings. Tree swallows offer an excellent system in which to study anthropogenic stressors because they readily occupy nest boxes, allowing for better control of potentially confounding factors (i.e. predation risk based on nest placement, exposure to light pollution, visual disturbance of cars passing, etc.). Previous work in this population found that noise exposure during development is positively related to nestling baseline cort and negatively related to nestling body condition (Injaian et al., 2018c). However, without cross-fostering, Injaian et al. (2018c) could not determine whether these impacts were due to direct nestling noise exposure or maternal noise exposure. This experiment uses cross-fostering to explicitly isolate maternal versus direct effects of noise on nestling phenotype to better understand the mechanism(s) by which noise may impact tree swallows.

We hypothesized that traffic noise exposure during the breeding period affects nestling phenotype through both maternal and direct effects (Fig. 1). We predicted that maternal (Fig. 1, Box A) and nestling (Fig. 1, Box B) noise exposure would be positively related to nestling

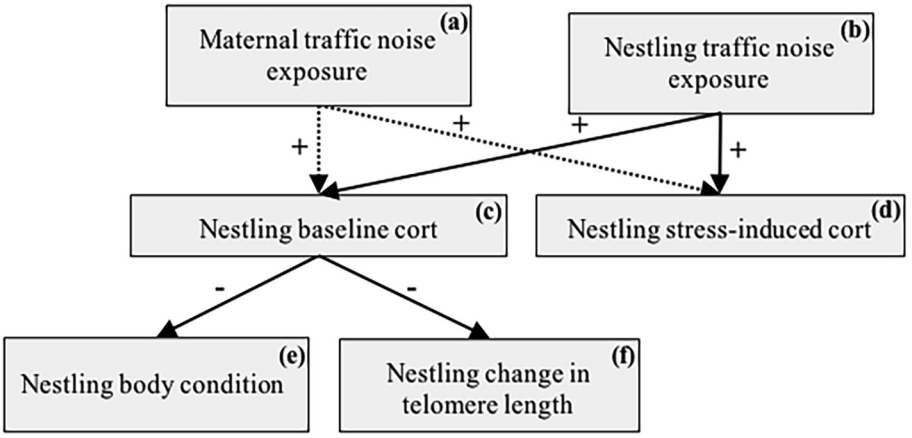

Fig. 1. Hypothesized anthropogenic noise impacts on tree swallows investigated in this study. Arrow direction represents the predicted direction of the impact and +/− symbols indicate predicted increases or decreases in each factor. Solid lines represent the predicted direct effects of traffic noise, while dashed lines represent the predicted maternal (indirect) effects of traffic noise.

baseline (Fig. 1, Box C) and stress-induced cort (Fig. 1, Box D). We also predicted that both maternal and nestling noise exposure would negatively affect nestling body condition through impacts on nestling baseline cort (Fig. 1, Box E). Similarly, we predicted that maternal and nestling noise exposure would negatively affect the change in telomere length (i.e. greater telomere attrition) during development through nestling baseline cort (Fig. 1, Box F). Increased baseline cort, reduced nestling body condition, and increased telomere attrition have potentially long-lasting negative impacts on fitness in this species (Bonier et al., 2009b; Haussmann et al., 2005; McCarty, 2001). Therefore, support for our predictions would suggest that traffic noise exposure needs to be mitigated in avian habitats during critical periods (i.e. breeding) in order to maintain local population health in tree swallows.

2. Methods

2.1. General field methods

This study took place at two sites near Davis, CA: the Putah Creek Riparian Reserve (38°32′18E, 121°51′01S, n = 11), and South Fork Preserve (38°31′04E, 121°41′39S, n = 13). A total of 24 nest boxes, with predator guards, were mounted to metal poles approximately 1.5 m above ground and separated by at least 20 m to prevent aggression associated with territory overlap (Winkler, 1994).

2.2. Noise playbacks

We measured ambient noise levels before traffic noise playbacks began using a sound pressure level (SPL) meter (Model 824, Larson-Davis, Inc., Depew, NY, U.S.A.). All measurements were taken in dBA SPL (re 20 μPa), using previously established methods (Injaian et al., 2018b). The ambient noise levels at each site were typical of U.S. rural areas, as neither field site is near a major roadway (EPA, 1971; Putah Creek Riparian Reserve: Leq of 43.35 ± 0.97 dBA, South Fork Preserve: Leq of 41.1 ± 0.28 dBA). Noise playbacks began 31 March 2016, before egg-laying, but after settlement to avoid non-random settlement of adults with regards to noise exposure (i.e. lower quality individuals settling in noise; Injaian et al., 2018a). We used previously established methods to run noise playbacks, which created a continuous distribution of amplitude exposures across nest boxes (Injaian et al., 2018a). Briefly, we created four unique traffic noise files from recordings of cars, trucks and motorcycles passing along a highway at medium to low traffic intervals (medium traffic: 18.7 ± 4.1 vehicles passing per minute, low traffic: 12.0 ± 1.0 vehicles per minute). Amplitude at the nest boxes ranged from an Leq of 35.0 to 61.4 dBA, with a mean Leq of 46.4 ± 7.5 dBA and a mean peak amplitude of 74.3 ± 4.1 dBA. To control for the visual disturbance of noise playbacks systems, plastic bins of similar size, shape and color were also distributed throughout the field site (each nest box was within 20 m of either a noise playback system or plastic bin). Further, to control for researcher disturbance, we visited plastic bins in the same pattern and for the same amount of time as noise playbacks systems. Noise playbacks ceased on 11 June 2016, the day that the final nestlings included in this experiment fledged.

2.3. Cross-fostering

Nest boxes were monitored every other day to track egg-laying, causing all dates to have an associated error of one day. For cross-fostering, nests were date-matched on day one of egg-laying and eggs were switched between nests once a pair of matched nests each had 3 eggs. This method allowed each brood to be partially cross-fostered prior to incubation, as tree swallows typically lay between 5 and 7 eggs in our population and begin incubating after laying their penultimate egg. This resulted in 3 crossed eggs and 2–4 natal eggs per clutch. We did not alter brood size at any nests, and eggs were not cross-fostered between field sites. The necessity to date-match nests caused the amplitude differences between natal and cross-fostered nests to range from an Leq of 6.7 to 21.4 dBA, with a mean difference of an Leq of 13.5 ± 3.9 dBA. Given that a decrease in 10 dBA is perceived as a halving of loudness for humans, a difference of even 6.7 dBA is likely a substantial change in the acoustic environment for birds (Blickley and Patricelli 2010).

2.4. Blood sampling and morphological measures

We caught adult females (n = 24) by hand in the nest box during incubation, at which time approximately 30 μL of whole blood was taken via brachial venipuncture and put into Queen's Lysis buffer in the field, using previously established methods (Seutin et al., 1991). We later used these blood samples to match natal mothers and nestlings (see Section 2.5 below). At this time, we also measured adult female mass (to the nearest 0.01 g) and tarsus length (to the nearest 0.1 mm) using previously established methods for tree swallows (Wiggins, 1989). We took the residuals of mass regressed on tarsus length to calculate a body condition index (BCI) for each individual (Whittingham and Dunn, 2000). We used these similar methods for nestling measurements, taken on day 10 post-hatch. However, we used mass regressed on wingchord length, instead of tarsus, to calculate nestling BCI, given difficulties associated with measuring nestling tarsus length on day 10. Nestling measurements were taken on day 10 post-hatch because tree swallow nestlings begin to lose mass after day 12 post-hatch to prepare for fledging, thus interpretation of mass later in development is difficult (Zach and Mayoh, 1982).

To analyze noise impacts on telomere length, we took 20 μL blood-samples via brachial venipuncture from nestlings on days 4 and 14 post-hatch (i.e. during the early and late developmental stage). This 10-day span between early and late samples is similar to the timeframe used for telomere analyses in barn swallows (*Hirundo rustica;* Parolini et al., 2015). Upon collection, we immediately put blood samples in 0.75 mL of cryoprotectant buffer (90% newborn calf serum, 10% dimethyl sulfoxide, DMSO). Blood samples in buffer were kept on ice for up to 5 h before we transferred them to a −80 °C freezer. We stored samples for approximately 1 year until DNA extraction. Due to logistical constraints, we only ran telomere analyses for a subset of nestlings that were not cross-fostered (n = 35 nestlings from 21 nests).

Also on day 14 post-hatch, we briefly removed nestlings from nest boxes and conducted a standard restraint test (Romero and Reed, 2005). Within 3 min of capture, we took approximately 30 μL of blood via brachial venipuncture using a heparinized capillary tube. This blood was used for baseline measurements of nestling cort (n = 106 nestlings from 24 nests). We then placed birds in mesh bags for 30 min, at which point we took a second 30 μL blood sample to assay stress-induced cort concentrations (n = 109 nestlings from 24 nests). Day 14 post-hatch was chosen for the standard restraint test because studies have shown that passerines have reduced stress responses earlier in development (Romero, 2004). Samples were placed on ice for up to 5 h before they were centrifuged to separate plasma from red blood cells. Plasma was stored at −80 °C for 3 months until radioimmunoassays were run.

2.5. Radioimmunoassay (RIA)

We measured plasma levels of cort by RIA as described by Wingfield et al. (1992) and González-Gómez et al. (2013). Briefly, we diluted approximately 20 μL of plasma to 200 μL in distilled water and added 2000 pm of tritiated cort for later calculation of recovery (i.e. losses following extraction and re-suspension). After equilibration for at least 2 h, we extracted all plasma samples in 4 mL of freshly re-distilled dicholoromethane. We then aspirated and dried the organic phase under a stream of nitrogen at 35 °C. We re-suspended dried extracts in 0.55 mL of phosphate-buffered saline with gelatin (PBSG). Next, we placed 200 μL aliquots into test tubes in duplicate and combined with 100 μL of 10^4 pm of tritiated cort (NET399250UC, PerkinElmer, Waltham, MA) and 100 μL of cort antiserum (MP Biomedical antibodies: 07-120016,

Lot 3R3-PB-20E). To assess recoveries, we placed a 100 μL aliquot into individual scintillation vials. We stripped unbound steroid from solution by adding 500 μL of dextran coated charcoal followed by centrifugation at 4122 × *g* for 10 min. Each scintillation vial received 3.5 mL of scintillation fluid (Ultima Gold 6013329, PerkinElmer, Waltham, MA) and was counted for 5 min using a Beckman 6500 liquid scintillation counter. We corrected final hormone values using the individual recovery for each sample. Intra-assay variation ranged from 1.6% to 2.9%, and inter-assay variation was 2.61%. The mean recovery was 84.45% ± 0.91% SD.

2.6. Assigning maternal identity for each nestling

Upon hatching, it was not possible to discern between crossed and natal nestlings, therefore we assigned maternity via genotyping. For nestlings, we acquired red blood cells via centrifugation of blood samples taken on day 14 post-hatch (see details in Section 2.3 above), which we then put into 1 mL of Queen's Lysis Buffer (Seutin et al., 1991). Blood samples from both nestlings and adult females were stored in −80 °C for 3 months until DNA was extracted using DNeasy Blood and Tissue Kits (Qiagen, Valencia, CA), according to the manufacturer's instructions.

After DNA extraction, we genotyped individuals via polymerase chain reaction (PCR) at nine microsattelite loci, which were previously developed for tree swallows (Tie16, 19, TaBi1, 4, 8, 25, 34 and Tal6, Makarewich et al., 2009; Tbi104, Stenzler, 2001). We modified the 5′ end of the forward primers by adding a fluorescent label (PET, 6-FAM, VIC, or NED; Applied Biosystems, Foster City, CA) and used a multiplexing PCR protocol (Hailer et al., 2005) to genotype all nine loci using previously established protocols (Stenzler et al., 2009). After PCR, we estimated all allele sizes with *Geneious* (Version 10.1.3, Biomatters, Ltd, Auckland, New Zealand). We then assigned maternity for each nestling among the two possible mothers through manual comparisons of allele sizes. We required allele sizes to match for seven out of nine microsattelites before assigning a nestling to a prospective mother. For five nestlings, amplification failed at up to two loci, however maternity was still able to be assigned using the remaining seven microsattelites.

2.7. Telomere restriction fragment analysis

The mean telomere length in whole blood samples from nestling day 4 and day 14 was calculated for each individual through previously established methods of telomere restriction fragment (TRF) assays (Haussmann and Mauck, 2008). Briefly, genomic DNA was restriction digested followed by Southern hybridization to a radioactive probe containing a terminal repeat to measure mean telomere length from a distribution of TRFs. Gels were imaged on a phosphor screen with a Typhoon Variable Mode Imager (Amersham Biosciences, Buckinghamshire, England) to visualize telomeres. The amount of radioactive signal (optical density, OD) in each lane corresponds with the amount of telomere at that position on the gel (i), and was quantified by densitometry in ImageJ. Background signal from nonspecific binding of the radioactive probe was subtracted from all OD measures. We used a 1 kb DNA extension ladder (1–40 kb), Invitrogen; and the distance each band of the molecular marker migrated (i) was plotted against the molecular weight in kilobases and converted into molecular weights (L) using a three-parameter log-linear function. The mean telomere restriction fragment length (called mean telomere length hereafter for simplicity) for each individual was calculated using: Mean TRF = Σ(ODi * LI)/Σ(ODi), where ODi is the densitometry output at position i, and Li is the length of the DNA (kB) at position I. The difference in mean telomere length between day 4 and day 14 (Δ mean telomere length) was used as a measure of telomere attrition.

2.8. Statistics

We analyzed our data using linear mixed models (LMM) in R (Version 3.2.2, *lme4* package; Bates et al., 2014). In models of nestling baseline and stress-induced cort, and nestling BCI, we included the following fixed effects: natal amplitude, rearing amplitude, natal female BCI, rearing female BCI, and all possible two-way interaction terms. We included 'natal amplitude' and 'rearing amplitude' to test for the impacts of direct versus maternal traffic noise exposure, respectively. We also included 'natal female BCI' and 'rearing female BCI' to account for maternal effects on nestling phenotype, as female BCI may alter nestling cort and BCI through pre and post-natal nutrients provided to nestlings (Blount et al., 2002; Nooker et al., 2005; Winkler and Allen, 1995). We only analyzed telomere attrition for nestlings that were not cross-fostered, therefore we only included 'natal amplitude' and 'natal female BCI' in these models ('rearing amplitude' and 'rearing female BCI' would have been redundant with natal measures). We also included 'nestling baseline cort' as a fixed effect in models of 'nestling BCI', 'mean telomere length on day 4' and 'Δ mean telomere length', because elevated baseline cort has been shown to suppress nestling growth (Hayward and Wingfield, 2004; Wada and Breuner, 2008) and covary with telomere dynamics (Young et al., 2017) in other bird species. We included 'site' as a fixed effect in all models because there were less than 5 levels in the data on which to base an estimate of the variance (Crawley, 2002). We included 'date', 'nest box location', and 'brood size' as random effects in all models. The 'nest box location' parameter provided information on the specific traffic noise file/speaker system or control bin associated with each nest box within a field site. All continuous parameters were centered and standardized using the *scale* function in R.

We used the *model.sel* function in the *MuMIn* package in R to compare all candidate models. For each model set, we used values of Akaike Information Criterion corrected for small sample sizes (AICc) and model weights for model comparisons (Burnham and Anderson, 2004; Hurvich and Tsai, 1989). We considered all models with ΔAICc < 4 to be highly supported (Burnham and Anderson, 2004). Model comparisons allowed us to evaluate the hypothesis that noise exposure during both egg-laying and development affected our dependent variable: if both 'natal amplitude' and 'rearing amplitude' were included in the top-ranked models, our results suggest that both maternal and direct effects explain the variation in our data. Further, for each dependent variable, we estimated the effect size (β parameter estimates) and 95% confidence intervals (CI) of parameters in the top model through the *summary* function in R. We assessed the importance of each parameter based on whether the 95% CI overlapped zero.

3. Results

3.1. Nestling baseline and stress-induced cort

The parameter of 'rearing amplitude', but not 'natal amplitude', was included in the top-ranked model of both nestling baseline and stress-induced cort (Table 1; which shows ΔAICc < 2 only, model comparisons for the full list of candidate models can be found in Appendix S1, Table S1). For every 1 dBA increase in rearing amplitude, nestling stress-induced cort increased by 0.218 ng/mL (Table 2, Fig. 2b). The effect of rearing amplitude on baseline cort was smaller in magnitude, as compared to stress-induced cort. Specifically, for every 1 dBA increase in rearing amplitude nestling baseline cort increased by 0.056 ng/mL (Table 2, Fig. 2a). However, the 95% CI for 'rearing amplitude' overlapped zero in the top model of nestling baseline cort and the null model (site only) was also highly ranked (ΔAICc = 0.223, Table 2).

Table 1
Candidate models for traffic noise impacts on nestling physiology, with the number of parameters (K), AICc values (AICc), the difference between each model and the best fit model (ΔAICc), and AIC model weights (w_i). Only models with ΔAICc < 2 are shown here (the full set of candidate models is provided in Appendix S1, Table S1).

Dependent variable (n)	Model*	K	AICc	ΔAICc	w_i
Nestling baseline cort (n = 109)	rearing amplitude + site	7	478.09	0	0.176
	null model (site only)	6	478.31	0.223	0.158
Nestling stress-induced cort (n = 106)	rearing amplitude + natal female BCI + site	8	775.91	0	0.202
	natal female BCI + site	7	777.60	1.683	0.087
	rearing amplitude + natal female BCI + rearing female BCI + site	9	777.71	1.793	0.082
Nestling BCI (n = 113)	baseline cort + rearing female BCI + natal female BCI + rearing female BCI*natal female BCI + site	10	363.19	0	0.545
Nestling mean telomere length on day 4 (n = 35)	null model (baseline cort + site only)	7	134.58	0	0.519
	natal female BCI	8	136.36	1.779	0.213
Nestling Δ mean telomere length (n = 35)	amplitude + baseline cort + site	8	97.78	0	0.559
	null model (baseline cort + site only)	7	99.33	1.556	0.257

* All models included 'date', 'brood size' and 'nest box location' as random effects.

3.2. Nestling body condition

The top-ranked model of nestling BCI did not include 'natal amplitude' or 'rearing amplitude' (Table 1). In the top model of nestling BCI, 'nestling baseline cort' (which was affected by 'amplitude', see Section 3.1 above) negatively affected nestling BCI, such that nestling BCI decreased by 0.169 units for every 1 ng/mL increase in nestling baseline cort (Table 2; Fig. 3). To ensure that these results were not driven by extreme values of nestling baseline cort, we re-ran the analysis with the two most extreme values removed from the data set (14.47 ng/mL and 9.86 ng/mL; Fig. 3). The results of this new analysis were qualitatively similar to the original analysis, with the new top model of nestling BCI including 'nestling baseline cort' and receiving approximately 60% of the model weight.

3.3. Nestling telomere dynamics

For nestlings that were not cross-fostered, we found no effect of 'amplitude' on telomere length in early development: the null model of mean telomere length on day 4 was ranked best (Table 1). However, the top-ranked model of telomere attrition included 'amplitude' and received 56% of the total model weight (Table 1). The negative relationship between amplitude and the change in telomere length indicates that nestlings from nests with greater noise exposure had greater telomere attrition during development (telomeres length decreased by 0.485 kb for every 1 dBA increase; Table 2, Fig. 4). Again, to ensure that these results were not driven by extreme values of change in telomere length (positive or negative), we re-ran the analysis with the two most extreme values removed from the data set (2.46 kb and −2.18 kb; Fig. 4). The new results were qualitatively similar to the original results, with the new top model of telomere attrition including 'amplitude' and receiving approximately 62% of the model weight.

4. Discussion

Our hypothesis that noise exposure would result in both direct and maternal effects on nestling cort was only partially supported, as top models of nestling baseline and stress-induced cort included rearing amplitude (direct effects), but not natal amplitude (maternal effects, Table 1). Additionally, we did not find support for our hypothesis that noise exposure affects nestling BCI through either direct or maternal effects. Rearing amplitude was positively related to baseline cort and baseline cort was negatively related to nestling BCI, but there was no relationship between rearing amplitude and BCI. This was surprising, given that two past studies in this population found a negative relationship between nestling growth and exposure to traffic noise during development (Injaian et al., 2018b,c). Finally, our hypothesis regarding noise impacts on telomere attrition was supported: nestlings with

Table 2
Observed relationships (β estimates ± 95% CIs, calculated using ± 1.96 SE) between response variables and parameters for top-ranked models.

Dependent variable (n, model weight)	Parameter*	β estimate	95% CI
Nestling baseline cort (n = 109, w_i = 0.176)	(intercept)	−0.966	−3.880, 1.942
	rearing amplitude	0.056	−0.003, 0.114
	site	0.812	−0.220, 1.778
Nestling stress-induced cort (n = 106, w_i = 0.202)	(intercept)	3.044	−7.367, 13.623
	rearing amplitude	0.218	0.005, 0.428
	natal female BCI	−1.499	−2.861, −0.132
	site	−1.715	−5.174, 1.780
Nestling BCI (n = 113, w_i = 0.545)	(intercept)	0.851	−0.126, 2.010
	nestling baseline cort	−0.169	−0.272, −0.066
	rearing female BCI	−0.423	−0.794, −0.028
	natal female BCI	0.297	−0.052, 0.610
	*rearing female BCI*natal female BCI*	0.299	0.119, 0.478
	site	−1.464	−2.556, −0.372
Nestling Δ telomere length (n = 35, w_i = 0.559)	*(intercept)*	2.109	0.282, 3.873
	amplitude	−0.048	−0.082, −0.025
	baseline cort	−0.148	−0.388, 0.098
	site	0.149	−0.621, 0.634

* Italicized text indicates that 95% CI did not overlap zero.

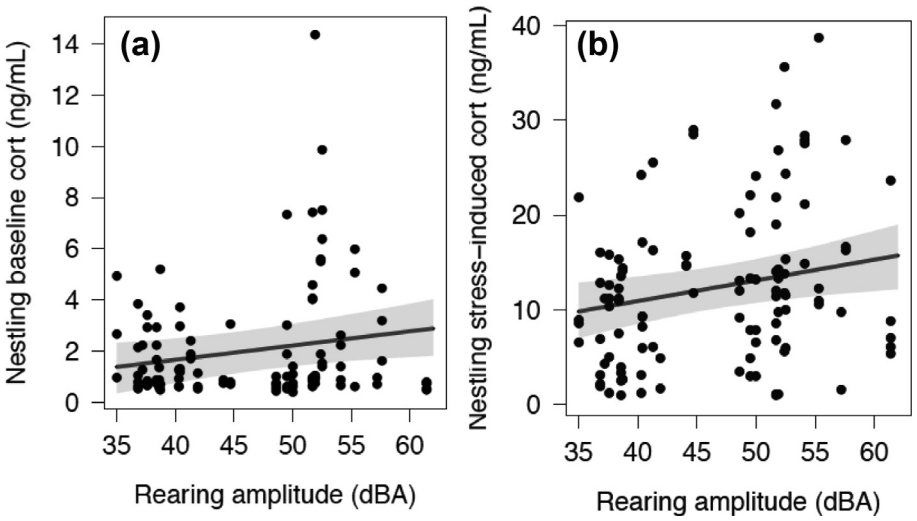

Fig. 2. Model output (with 95% CI) and raw data points for the top-ranked models of (a) nestling baseline (n = 106) and (b) nestling stress-induced (n = 109) cort levels (ng/mL) taken on day 14 post-hatch. Cort data plotted against the rearing amplitude at a given nest box. Best fit lines and CIs created by allowing rearing amplitude to vary and holding all other factors constant. Note that scales for y-axes differ between (a) and (b) to maximize ability to view patterns for each.

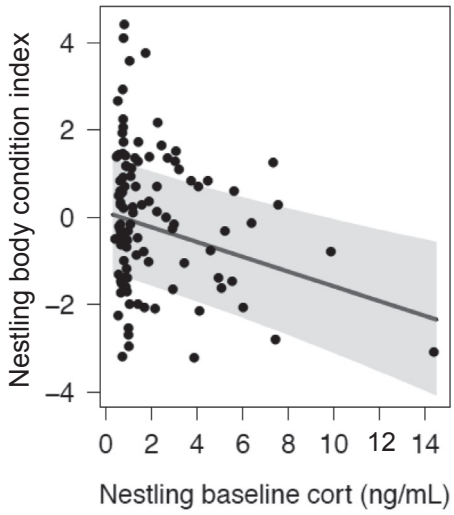

Fig. 3. Model output (with 95% CI) and raw data points for the top-ranked model of nestling body condition index (BCI). Measurements taken on day 10 post-hatch. Graph created by allowing nestling baseline cort to vary and holding all other factors constant.

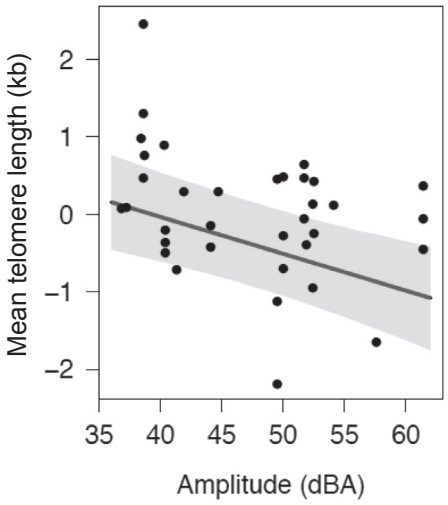

Fig. 4. Model output (with 95% CI) and raw data points for the top-ranked model of Δ mean telomere length (kb). The change in mean telomere length was assessed for each nestling through blood samples taken on days 4 and 14 post-hatch. Only nestlings that were not cross-fostered were included in telomere analyses. Graph created by allowing amplitude to vary and holding all other factors constant.

greater noise exposure during development had increased telomere attrition. Overall, these results suggest that direct exposure to traffic noise during development may result in negative consequences for tree swallows both in the short and long-term.

The positive relationship between direct nestling noise exposure (rearing amplitude) and nestling baseline and stress-induced cort is supported by past work in this system (baseline; Injaian et al., 2018c), as well as other species (baseline and stress-induced; Crino et al., 2011). Again, it is often difficult to relate cort to fitness (Bonier et al., 2009a; Sorenson et al., 2017) and thus potential impacts on populations (Busch and Hayward, 2009). However, when taken in combination with the decreased nestling body condition and increased telomere attrition, our results suggest that increased nestling baseline and stress-induced cort may result in negative consequences for free-living tree swallows. Again, it is important to note that the effect size of 'rearing amplitude' on nestling baseline cort was relatively small (β estimate = 0.056), the 95% CI for the 'rearing amplitude' parameter overlapped zero in the top-ranked model (Table 2), and there was high inter-individual variation in baseline cort levels for individual with similar noise exposures. Additionally, our results differ from recent studies of noise impacts, which found no effect on baseline or stress-induced cort in house sparrow nestlings (Angelier et al., 2016; Meillère et al., 2015), reduced nestling stress-induced cort in noise-exposed white-crowned sparrows (*Zonotrichia leucophrys;* Crino et al., 2013), and reduced nestling baseline cort in several passerine species exposed to compressor noise (Kleist et al., 2018). Additional measures, such as corticosteroid binding globulin (CBG), or more integrative measures, such as fecal glucocorticoid metabolites (FGM), would help gain a more complete understanding of the impacts of noise exposure on regulation of the HPA-axis in developing passerines (Breuner et al., 2013; Dantzer et al., 2014).

Past studies in this system found a negative relationship between traffic noise exposure and adult female stress-induced, but not baseline, cort (Injaian et al., 2018c). These past results may explain the fact that we found no effect of maternal noise exposure on nestling baseline or stress-induced cort, given that maternal baseline, but not stress-induced, cort levels have been shown to be related to yolk corticosteroid levels in other passerines (Hayward and Wingfield, 2004). Unfortunately, we were not able to include a formal analysis of maternal cort in this study due to small sample size. However, preliminary data from 14 females included in this study showed either no relationship (r = 0.016) or a weak relationship (r = 0.196) between natal female

baseline cort and nestling baseline and stress-induced cort, respectively. These data support our results that traffic noise exposure does not impact nestling baseline or stress-induced cort through maternal effects. We do not expect food deprivation to be the explanatory mechanism of increased nestling baseline and stress-induced cort in noise given that past studies in this population found that noise does not decrease parental feeding behavior (Injaian et al., 2018b,c).

Our results, which found no relationship between rearing or natal amplitude and nestling BCI, are supported by some (Leonard and Horn, 2008), but not all (Injaian et al., 2018b,c) previous work in this species. Here, impacts of traffic noise exposure on nestling BCI may be mediated through nestling baseline cort (Table 2). This negative relationship between baseline cort and body condition in nestlings matches the cort-fitness hypothesis (i.e. higher baseline cort levels indicates lower fitness; Bonier et al., 2009a). However, evidence in support of this hypothesized negative relationship varies within and between populations, and even within individuals at different stages of their life history (Bonier et al., 2009a). Also, we took nestling BCI measures on day 10 post-hatch, while cort samples were taken on day 14 post-hatch. It is possible that BCI and/or cort samples taken at different times in development would produce different results.

Given logistical constraints, we were not able to separate out the potential role of direct versus maternal effects of noise exposure on telomere attrition (we only assayed telomere attrition for nestlings that were not cross-fostered). However, the fact that we did not find an effect of increased noise exposure until later in development may suggest that direct, rather than maternal, effects drive telomere attrition in this context: we found a difference in telomere length change (when comparing measurements from days 4 and 14 post-hatch), but we did not find an effect of traffic noise exposure on day 4 measures of mean telomere length alone (Table 2). Past studies in great tits (Salmón et al., 2016) and zebra finches (Dorado-Correa et al., 2018) also suggest direct, rather than maternal, effects of disturbance on telomere attrition. Human disturbance may increase telomere attrition in nestlings by increasing stress-induced cort (Herborn et al., 2014). Although other experimental work in house sparrows found no relationship between cort and telomere attrition, given noise exposure (Meillère et al., 2015). Here, the slight increase in mean telomere length between days 4 and 14 post-hatch for nestlings with low-amplitude traffic noise exposure is interesting, and similar results have been found in other studies of the effects of early life stressors on telomere dynamics (Fig. 4; Young et al., 2017). Given that telomere length is positively related to lifespan in free-living birds (Bize et al., 2009; Haussmann et al., 2005; Heidinger et al., 2012), greater telomere attrition for birds reared in noisier nests suggests that short-term exposure to traffic noise during development may have long-term consequences on both individual survival and population health in noise-exposed areas. We would like to highlight that the impact on telomere attrition found here is likely an underestimate of the total impact of noise: we assayed telomere attrition between days 4 and 14 post-hatch, but nestlings reared in noisy environments would be exposed to noise from hatching until fledging (e.g. approximately 22 days in tree swallows).

5. Conclusion

Overall, our results suggest multiple pathways through which nestling exposure to traffic noise during development may result in long-term consequences in tree swallows. The decreased nestling body condition and increased telomere attrition in nests with greater noise-exposure may be explained by increased nestling baseline and stress-induced cort, however further research is needed to confirm this pathway. We did not find any evidence for maternal effects of noise exposure on nestlings. Given the continued growth of transportation networks and the associated acoustic alterations of breeding habitats for free-living birds, our results suggest that conservation efforts should be allocated towards mitigating noise exposure during critical periods (i.e. nestling development).

Author contributions

ASI, CCT and GLP conceived ideas and designed methodology. ASI, PGG, and AKB collected data. ASI, PPG, AZ, MFH and JCW analyzed and interpreted the data. ASI led the writing of the manuscript. All authors provided feedback on the manuscript.

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Competing interests

Declarations of interest: none.

Appendix A. Supplementary data

Supplementary data to this article can be found online at https://doi.org/10.1016/j.ygcen.2019.02.017.

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