

# Chronic anthropogenic noise disrupts glucocorticoid signaling and has multiple effects on fitness in an avian community

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**Anthropogenic noise is a pervasive pollutant that decreases environmental quality by disrupting a suite of behaviors vital to perception and communication. However, even within populations of noise-sensitive species, individuals still select breeding sites located within areas exposed to high noise levels, with largely unknown physiological and fitness consequences. We use a study system in the natural gas fields of northern New Mexico to test the prediction that exposure to noise causes glucocorticoid-signaling dysfunction and decreases fitness in a community of secondary cavity-nesting birds. In accordance with these predictions, and across all species, we find strong support for noise exposure decreasing baseline corticosterone in adults and nestlings and, conversely, increasing acute stressor-induced corticosterone in nestlings. We also document fitness consequences with increased noise in the form of reduced hatching success in the western bluebird (*Sialia mexicana*), the species most likely to nest in noisiest environments. Nestlings of all three species exhibited accelerated growth of both feathers and body size at intermediate noise amplitudes compared with lower or higher amplitudes. Our results are consistent with recent experimental laboratory studies and show that noise functions as a chronic, inescapable stressor. Anthropogenic noise likely impairs environmental risk perception by species relying on acoustic cues and ultimately leads to impacts on fitness. Our work, when taken together with recent efforts to document noise across the landscape, implies potential widespread, noise-induced chronic stress coupled with reduced fitness for many species reliant on acoustic cues.**

acoustic environment | anthropogenic noise | birds | stress response | perceived predation risk

Human-made noise is a widespread and complex stimulus that alters habitats, degrades natural acoustic conditions, and partially or fully excludes species that are sensitive to noise exposure from affected areas (1–4). The exact mechanisms by which noise disrupts animals and their environments are still debated, but recent work links noise exposure with alterations in vocalization, vigilance, foraging, and parental behavior (5–12). Despite this recent progress, key questions remain unanswered, especially whether anthropogenic noise represents a chronic stressor for free-living animals and the severity of any resulting fitness effects from noise or noise-induced stress. Although not unexplored (13–20), the effect of noise on stress and fitness is complex (21). The identification of mechanisms is especially difficult given the possibility that noise could elicit a stress response directly through extreme exposure (22, 23) or indirectly by altering the interaction between animals and their environment (13). Noise levels in wild systems reach the extreme amplitudes that are observed to directly elicit stress responses in laboratory studies [i.e., 90–105 dB (23); 130 dB (24)] only at very close proximity to noise sources. At lower exposure levels, anthropogenic noise is more likely to elicit stress responses indirectly by increasing the difficulty of coping with

external challenges (e.g., territory defense) or by creating anxiety through reduced detectability and predictability of threats (e.g., acoustic masking of predator alarm sounds) (8, 24–26), or both. Clarifying the potential impacts of chronic noise exposure in wildlife is timely and needed, given the nearly ubiquitous presence of anthropogenic noise worldwide and the forecasted global rise in noise-producing infrastructure (1).

Conservation physiologists often use measurements of an organism's baseline circulating stress hormones, or glucocorticoids (GCs), as an indirect measure of habitat quality and a proxy measure for potential fitness (27). GCs are secreted from the adrenal gland in a coping response to challenge and are the result of activation of the hypothalamic–pituitary–adrenal (HPA) axis, a chemical cascade triggered by neuronal signals sent from hierarchical neural systems to the hypothalamus in response to the brain's perception of a stressor (28, 29). The HPA axis is a highly conserved vertebrate stress system activated during allostasis, the process by which animals maintain stability, or homeostasis, through change (30). While GCs are modulated in ultradian, circadian, and seasonal rhythms to meet challenges related to predictable energy deficits, frequent disturbance from chronic stressors encountered in low-quality habitats can push an

## Significance

Studies examining relationships among habitat disturbance, physiology, and fitness in wild animals often produce contradictory or inconclusive results, casting doubt on current conservation physiology predictive frameworks linking stress and fitness. We apply a new framework drawn from experimental systems utilizing chronic inescapable stressors to explore how noise, an environmental stimulus common to wildlife habitats worldwide, disrupts stress hormone signaling and impacts fitness. We utilize a natural experiment to show that chronic, anthropogenic noise reduced baseline corticosterone levels, increased acute corticosterone response, and, at highest amplitudes, negatively impacted multiple measures of fitness across three species of birds. Our work brings conservation physiology theory involving wild animals into needed alignment with recent theories based on chronic stress in laboratory studies.

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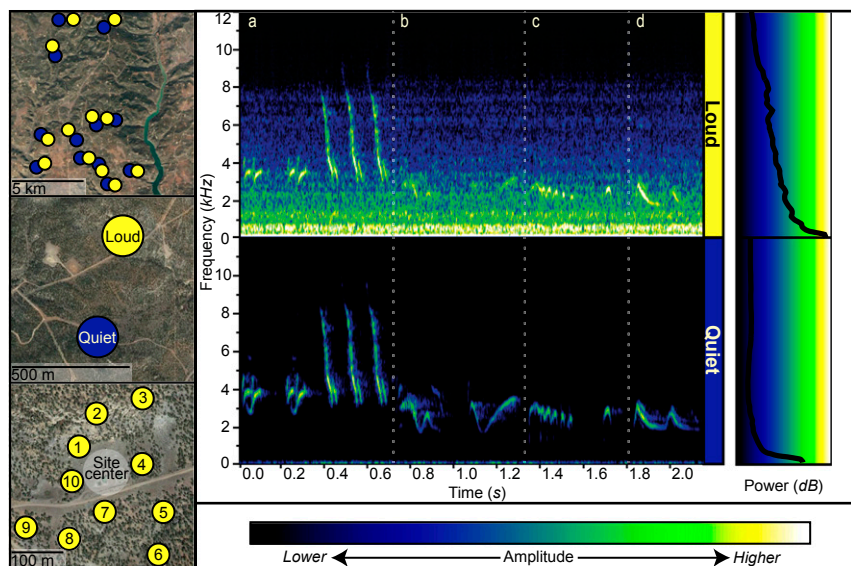
organism's physiological state out of the range of a normal allostasis and into a state of allostatic overload. Overload states are often pathological and have been shown to have fitness consequences (30, 31). Although increased allostatic load may suggest higher baseline GCs, the empirical data have been equivocal at best; different studies find decreased as well as increased GCs in response to chronic stressors (17, 25, 32–36).

Baseline corticosterone (hereafter, “cort”) levels are generally predicted to positively associate with the intensity of habitat disturbance in what has been dubbed the “cort-fitness hypothesis” (21, 27). In contrast to the predictions of this hypothesis, results from laboratory and human systems suggest that exposure to chronic stressors is often associated with decreased basal GC levels, hypocorticism, and GC insensitivity (37, 38). The development of hypocorticism could be a coping mechanism that saves an organism from experiencing the severe effects of allostatic overload (39), but there is evidence that it can lead to reduced fitness. For example, a laboratory-based system using mice shows how chronic subordinate social rank triggers hypocorticism, reflected by reduced basal GC levels and GC insensitivity that can be accompanied by an inflammatory response (25, 40) and reduced weight gain (41). Unlike situations in which animals fail to react to repeated chronic stressors, physiological results in this laboratory system are paired with negative fitness outcomes, suggesting that animals do not habituate (42). Additionally, these studies show that individuals experiencing hypocorticism can develop HPA-axis sensitivity to heterotypic stressors, leading to increased GC responsiveness following experimental acute stress events (25, 43). In natural systems, such heterotypic stressors could include predation attempts, competitor interactions, or confrontations with humans and other anthropogenic disturbances. While much work has focused on human and rodent laboratory models, the sensory blanketing of the acoustic environment by chronic anthropogenic noise provides a unique opportunity to test if a chronic, inescapable stressor can cause similar stress and fitness responses in wild animals.

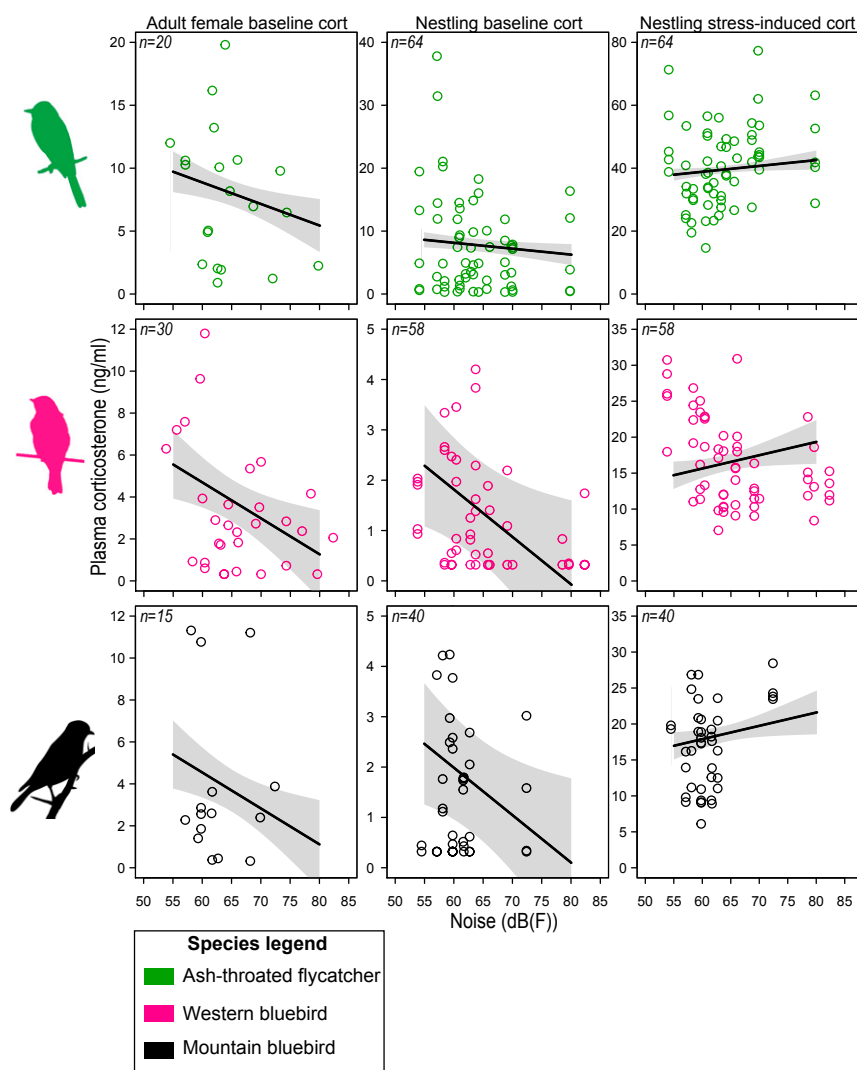
The impact of noise is thought to be most severe when frequencies overlap auditory environmental cues, a phenomenon

known as “acoustic masking,” which leads to reduced detection and discrimination of key acoustic signals and cues. Acoustic signals and cues from both conspecifics and heterospecifics provide important information about the immediate environment, including the quality and location of competitors (44) and the presence or absence of predators through alarm or nonalarm vocalizations and adventitious sounds (45–49). Given the capacity for chronic noise to consistently mask biologically relevant cues (8, 26, 47, 50), animals living in areas with high levels of noise may fail to receive information about their local habitats, leading to a continual state of perceived unpredictability and reduced security (44, 47, 51). The experience of constant environmental uncertainty among both parents and nestlings, whether justified or not (24), could cause chronic activation of the HPA axis and affect both GCs and fitness through reduced hatching success of eggs and provisioning of young (52).

To date, no studies have simultaneously examined relationships among noise, GCs, and fitness in animals that settle and breed in natural areas exposed to chronic anthropogenic noise. To address this significant gap and provide a needed conceptual alignment of recent work in conservation physiology with related work on chronic stress, we performed a natural experiment using wildlands in northern New Mexico's San Juan Basin, where a community of birds breeds along a gradient of noise produced by natural resources extraction (Fig. 1). We measured noise levels, cort, and fitness-relevant variables, e.g., hatching success and nestling body condition, in populations of three species of cavity-nesting birds with different tolerances to noise: western and mountain bluebirds (*Sialia mexicana* and *Sialia currucoides*, respectively), and ash-throated flycatchers (*Myiarchus cinerascens*) (53). We predict that long-term exposure to continuous anthropogenic noise will act as a chronic stressor, disrupting GC signaling in adults and nestlings, with the greatest impact at sites with highest amplitude noise. We also predict that long-term exposure to continuous anthropogenic noise will be linked to reduced fitness. Incorporating multiple species and disturbance (53–57) into a multiyear study defined by a chronic, homotypic stressor represents the most highly integrated study of the effects of anthropogenic noise on wild birds to date.



**Fig. 1.** This conceptual figure shows the study system design and the acoustic properties of sites with and without compressor noise. (Left) The three nested spatial scales included in the study: the full system (Top), loud and quiet pairs (Middle), and individual sites (Bottom). The Bottom shows nest boxes (labeled 1–10) arrayed around the site center, which was the centroid location of a well pad. (Center) A spectrogram of recorded background noise at a louder area (Upper) and quieter area (Lower). The songs of four common species that are found in RCHMA, including representatives from this study are included to depict the masking potential of noise: (a) spotted towhee; (b) plumbeous vireo; (c) ash-throated flycatcher; and (d) western bluebird. (Right) Power spectra that show the amplitude or “power” of compressor noise at frequencies between 0 and 12 kHz.



**Fig. 2.** The graph shows the relationship between cort and noise in all three species and two life stages. Baseline cort levels for both adults and nestlings were negatively affected by noise (Left and Center Columns, respectively), while acute stress-induced cort levels in nestlings were positively affected by noise (Right Column). For each model, the effect of noise was consistent across species, irrespective of species-specific cort levels. The gray-shaded areas indicate the 85% CIs for each estimated effect.

## Results and Discussion

**Effect of Noise on Cort.** The amplitude of compressor noise, measured as equivalent continuous sound level ( $L_{eq}$ , dB unweighted [dB(F)]), had a negative effect on baseline cort levels in adult females [linear mixed model (LMM), Gaussian distribution,  $\beta_{noise} = -1.120$ , 85% CI:  $-1.966$  to  $-0.279$ ] (Fig. 2 and Table 1). This result, which was consistent across species, supports our prediction that chronic anthropogenic noise exposure is an explanatory factor affecting GC signaling. The finding of a negative linear relationship between noise disturbance and baseline cort strongly suggests that suppression of baseline cort occurs in response to the acoustic disturbance gradient. While it remains possible that individual GC responsiveness affects nest site selection, we do not find any evidence of self-sorting based on other quality factors, e.g., body size (Table S1). This relationship is best explained by the development of hypocorticism, a physiological condition consistent with exposure to chronic stress (25, 39, 40). A previous study found that an experimental chronic stress protocol reduced baseline cort levels among wild-breeding female European starlings (*Sturnus vulgaris*) relative to individuals in a control group (32).

Nestlings in our study also showed reduced levels of baseline cort with increased noise levels, and, as in adults, there was support for a common effect across species (LMM, Gaussian distribution,  $\beta_{noise} = -0.660$ , 85% CI:  $-1.243$  to  $-0.080$ ) (Fig. 2 and Table 1). A comparable result was found in a small-scale experimental study that reported reduced baseline cort in white-crowned sparrow (*Zonotrichia leucophrys*) chicks exposed to noise during their first 5 d of life (17). That we find a clear negative effect on cort across a gradient of noise in females and nestlings from three different species strongly suggests that chronic anthropogenic noise induces stress and hypocorticism in birds.

Research from the field of sensory ecology demonstrates how masking of critical acoustic communications by chronic noise could lead to stress. The distance over which birdsong and other sounds are effectively transmitted, their “active space” (58), is significantly reduced by increases in ambient background noise (1, 8, 59–61). Anthropogenic noise, acting as an acoustic blanket, can reduce or inhibit detection of hetero- (46–48) and conspecific (62) vocalizations that birds and other animals (45) use to gain information about predation threats. For example, the presence of birdsong and chatter is thought to signal the absence of nearby predators (49, 63). Thus,



Table 1. Effects of noise and other variables on cort and fitness

Dependent variable	N	Fixed effects	Random effects	k	R <sup>2</sup>	AIC <sub>c</sub>	Null AIC <sub>c</sub>	χ <sup>2</sup>
Cort predicted by noise								
Adult baseline cort	65	Noise*– Species* Life stage*	Site Nest ID	8	0.592	369.801	381.857	<0.001
Nestling baseline cort	162	Noise*– Species*	Year	6	0.319	978.299	1,030.426	<0.001
Nestling acute stressor-induced cort	161	Noise*+ Species* Chicks*+ Time*– Tree cover*+	Nest ID	9	0.766	1,134.000	1,202.198	<0.001
Fitness predicted by noise								
All species hatch success	364	Intercept	None	1	0	218.722	Na	Na
Western bluebird hatch success	133	Noise*– Tree cover*+	None	3	0.039	89.489	91.538	0.045
Ash-throated flycatcher hatch success	138	Noise*+	Year	3	0.024	75.027	75.280	0.126
Mountain bluebird hatch success	93	Intercept	None	1	0	56.586		Na
Nestling feather growth (PC1)	272	Noise <sub>quad</sub> * Species*	Year Nest box Nest ID	9	0.907	295.644	353.080	<0.001
Nestling body size (PC2)	272	Noise <sub>quad</sub> * Species* Chicks*– Distance*– Lay date*–	Nest ID	10	0.739	309.872	364.547	<0.001

All models presented are best-performing models. The term for life stage is included as the effect of the provisioning stage on the dependent variable. The term "distance" refers to the distance in meters from the nest box to the well pad center or compressor station. Models that do not include random effects were run as GLMMs following our described methods. k, parameters estimated; Na, not applicable.

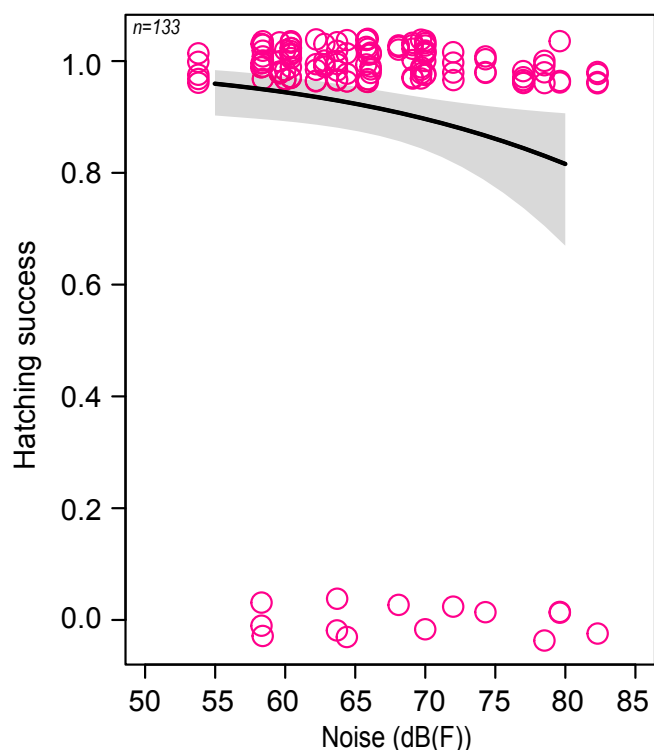
\*Predictors that do not include zero in their 85% confidence limits; all continuous predictors are marked with the direction of their effect on the dependent variable.

hearing birdsong could be used as a safety signal in birds, and continual masking could chronically impair risk perception, leading to environmental uncertainty and activation of the stress response. While birds are away from the nest on foraging trips, noise throughout the territory (53) could decrease hunting efficiency (7, 50), reducing energy intake and leading to increased allostatic load relative to individuals in quieter territories. Unlike adults, nestlings are incapable of escaping nest boxes and are constantly exposed to noise. This chronic, inescapable disturbance could stimulate stress and hypocorticism in nestlings through reduced reception of heterospecific (48) or parental (64) cues. Alternatively, the effect of compressor noise on parental vocal behavior (65) could interact with the effects of masking and lead to reduced detectability and increased uncertainty about environmental risk. Provisioning behavior could also be negatively affected by noise, leading to increased allostatic load in nestlings, discussed more below.

We also tested if, as in other systems, responses to acute stressors in animals under chronic stress were higher when exposed to heterotypic stressors. We measured cort levels in nestlings 10 min after capture and fit models similar to those for baseline measurements. The model with a common effect for noise but with species-specific differences best explained acute stressor-induced cort levels in nestlings. In contrast to baseline cort results, the effect of noise on acute stressor-induced cort was positive (LMM, Gaussian distribution,  $\beta_{\text{noise}} = 1.297$ , 85% CI: 0.067–2.538) (Fig. 2 and Table 1). The finding of suppressed baseline cort and increased acute stress-induced cort is consistent with many laboratory studies on model species, which suggest that hypocorticism results from chronic psychological stress (reviewed in ref. 26). Sensitization of cort response to acute stressors following exposure to a chronic stressor has also been found in nestling European starlings exposed to a chronic psychological stress protocol (32) but not in wild-caught adult birds exposed to a similar treatment in a laboratory setting (33). This

response could be adaptive in nestlings experiencing increased perceived predation risk, as a heightened reactivity during this stage could prime them for more efficient escape effort (66). However, early fledging might trade off with the accumulation of body mass, which can reduce the probability of postfledgling survival (67–69). Although hypocorticism has been proposed as a protective response to increased allostatic load that might allow affected individuals to avoid the most serious effects of overload states (39), the attenuation of baseline cort is also potentially harmful and has been associated with inflammation, disease susceptibility (25), anxiety (43), and reduced exploration of novel objects (70). Thus, studies that explore hormone signaling and survivorship of individuals are necessary to fully quantify the costs of these responses to noise.

**Effect of Noise on Fitness: Hatching Success.** We found that a summary model including all species yielded uninformative results, and we therefore present species-specific models for hatching success (Table 1). Results from these models suggest that hatching success was significantly affected by noise in one species, the noise-tolerant western bluebird (53). In support of our prediction that increased noise would lead to decreased fitness, hatching success in the western bluebird was negatively associated with noise levels at the nest box [generalized linear mixed-effect model (GLMM), binomial distribution,  $\beta_{\text{noise}} = -0.483$ , 85% CI:  $-0.892$  to  $-0.075$ ] (Fig. 3 and Table 1). Egg-hatching rates in ash-throated flycatchers showed a weak, positive relationship with noise (GLMM, binomial distribution,  $\beta_{\text{noise}} = 0.658$ , 85% CI:  $0.036$ – $1.376$ ) (Table 1), but this model was indistinguishable from the null hypothesis [change in corrected Akaike information criterion ( $\Delta\text{AIC}_c$ ) = 0.25]. Similarly, there was no clear effect of noise on hatching rates in mountain bluebirds. Overall, results from all three species may be best explained by previously described nesting patterns.



**Fig. 3.** Hatching success in the western bluebird was negatively affected by noise. The gray-shaded area indicates the 85% CI.

The negative relationship between anthropogenic noise and hatching success in western bluebirds represents the strongest effect of noise on a direct measure of reproductive success in this study and is somewhat surprising, given previous results from this system that showed an even distribution of western bluebird nests across the noise gradient (53). The most parsimonious explanation is that the western bluebird may be caught in an equal-preference ecological trap driven by anthropogenic noise. An equal-preference trap occurs when a species shows equal preference for low- and high-quality habitats but incurs a reproductive cost in low-quality habitats (71). Both the ash-throated flycatcher and the mountain bluebird have been shown to avoid noise (53) and appear to escape the negative effects on hatching success related to high-amplitude noise exposure. That these population-level consequences were not indicated by occupancy data illustrates the importance of studies that combine occupancy, physiology, and fitness data.

One possible mechanism leading to reductions in hatch success is increased distraction and vigilance behavior in areas exposed to higher levels of noise, an effect well documented in the literature among birds, mammals, and arthropods (7, 50, 72–74). During the incubation period, increased vigilance by females could contribute to reduced hatching by a trade-off with incubation time leading to fluctuations of nest temperature and reduced time at optimum temperature (75, 76). Increased perceived predation risk has been shown to elevate vigilance via increased nest attentiveness among several bird species; however, this appears to come at the cost of reduced hatching success (52). Other possibilities include noise leading to a breakdown in courtship and copulation, or males, in contrast to females, might sort themselves by quality with noise, potentially leading to low-virility males in loud areas. Studies that investigate incubation rates and rhythms, courtship, nest attentiveness, and male quality in the context of noise are still needed to more conclusively determine causes of reduced egg hatching in loud areas. Nevertheless, regardless of the mechanisms involved, our results suggest that noise decreases a direct measure of fitness,

hatching success, in the western bluebird, a species previously thought to be noise tolerant.

**Effect of Noise on Fitness: Feather Development and Body Size.** The effect of noise on fitness may manifest not only in hatching failures but also in reduced body condition of hatched chicks. Development and growth during the nestling stage is an important predictor of juvenile survival and thus acts as a useful surrogate to infer fitness potential in individuals exposed to noise (77, 78). We evaluated the effect of noise on nestling feather growth [principal component feather ( $PC_{\text{feather}}$ )] and nestling body size [principal component size ( $PC_{\text{size}}$ )] (see Table S2 for loadings) in a model including all species. Noise exposure was easily the strongest predictor of feather growth, showing a strong quadratic relationship with  $PC_{\text{feather}}$  (LMM, Gaussian distribution, 85% CI:  $-5.410$  to  $-2.184$ ) (Fig. 4 and Table 1). Noise had a positive impact on feather growth until  $\sim 70$  dB(F), after which feather development was strongly reduced. Similarly, body size showed a quadratic relationship with noise, with decreases above  $\sim 70$  dB(F) (LMM, Gaussian distribution, 85% CI:  $-2.589$  to  $-0.499$ ) (Fig. 4 and Table 1). These results also support the hypothesis that noise decreases fitness. However, the nonlinear effect of noise on both feather growth and body size was not predicted. This demonstrates that, either directly or indirectly, acoustic disturbance gradients can elicit complex responses.

High levels of noise exposure could distract parents and lead to increased vigilance and reduced provisioning efforts that result in body size reductions (7), a situation that might also partly explain the development of hypocorticism in nestlings. Additionally, the ability of the birds in our study, which are all insectivorous, to find prey could also be diminished at sites with elevated levels of noise (50, 79). Several consequences are linked to smaller nestlings, such as failure to return from migration (80), occupancy of low-quality breeding habitat (81), and reduced reproductive success (82). Reduced feather development also likely decreases the ability to escape predators during fledgling stage (83).

At intermediate amplitudes nestlings exhibited accelerated growth of both feathers and body size. These moderate levels may increase pressure to develop faster and fledge earlier, while not incurring the costs of reduced provisioning in high-noise exposure. As well, previous results in this system show that avian nest predators are sensitive to and avoid loud areas (2). It could be that, at intermediate amplitudes, breeding birds experience a noise-mediated predator shield (84), in which predators are excluded but the consequences of noise on communication, cue detection, and distraction are minor or not perceived by individuals. This scenario would further facilitate provisioning and should be tested with experimental introductions of multimodal predator stimuli placed within medium-amplitude zones. If the absence of predators in areas where noise has not yet degraded perception creates conditions in which parental care is enabled, we would expect to see negative effects of the proposed predator treatment on nestling development.

At lower amplitudes, nestlings had feather growth and body size more comparable to those in individuals from the loudest locations, perhaps reflecting perceived (and real) presence of predators or increased detectability of con- and heterospecific acoustic signals about predators. It is worth noting that adult females and nestlings experience higher baseline cort in quiet areas, although it is unlikely that habitats with more natural acoustic conditions are poorer-quality environments for breeding. Thus, given these documented physiological changes (e.g., hypocorticism and body condition), further experiments addressing longer-term impacts of noise on survivorship of fledglings are needed.

We argue that the nonlinear effects of noise on development are best explained using the same mechanism that may cause chronic stress and hypocorticism: masking of acoustic cues that impairs risk perception and can lead to decreased provisioning effort. In other words, noise exposure creates sensory conditions

The critical discovery is that there is a strong and consistent multispecies pattern of hypocorticism in response to increased noise amplitude that is linked to negative fitness consequences. This suggests an updated framework for how noise affects wildlife and strengthens the case for reassessment of conservation physiology theoretical models. Although there is an historical bias to expect elevated basal GCs in response to exposure to chronic

**Table 2. Effects of cort on fitness**

Dependent variable	N	Fixed effects	Random effects	k	R <sup>2</sup>	AIC <sub>c</sub>	Null AIC <sub>c</sub>	χ <sup>2</sup>
Fitness predicted by maternal baseline cort								
Hatching success	120	Baseline cort*+ Species*	None	4	0.190	90.649	92.459	0.044
Nestling feather growth (PC1)	96	Baseline cort*– Species* Cort: species* Chicks*+ Distance*+ Tree cover*+ Baseline cort+ Species* Cort: species* Distance*– Lay date*–	Pair Year	12	0.969	77.126	200.930	<0.001
Nestling body size (PC2)	96	Baseline cort+ Species* Cort: species* Distance*– Lay date*–	Site	10	0.802	99.297	184.633	<0.001
Fitness predicted by nestling baseline cort								
Nestling feather growth (PC1)	162	Baseline cort+ Species*	Site Nest ID	6	0.883	189.782	224.463	<0.001
Nestling body size (PC2)	162	Species* Lay date*–	Nest ID	6	0.720	202.290	228.856	<0.001
Fitness predicted by nestling acute-stressor induced cort								
Nestling feather growth (PC1)	161	Acute cort+ Species*	Nest ID	6	0.885	182.780	217.760	<0.001
Nestling body size (PC2)	161	Acute cort+ Species*	Nest ID	6	0.705	183.643	212.253	<0.001

Models are top models or are the highest performing models within two AIC<sub>c</sub> that included a variable for cort. This table follows the formatting described in the legend of Table 1. Acute cort, acute stressor-induced cort; k, parameters estimated.

\*Predictors that do not include zero in their 85% confidence limits; all continuous predictors are marked with the direction of their effect on the dependent variable.

stressors, a careful analysis of field and laboratory studies of diverse nonmammalian and mammalian species revealed that there is no consensus endocrine response to chronic stress (87). Our work raises a subtle but important point. There may be consistent responses, such as hypocorticism, within the context of chronic, inescapable noise exposure for avian breeding communities. However, the body of evidence suggests GC regulation is also context dependent, and rather than expecting simple generalities about GC response to hold broadly, it is more critical to document dysregulation, along with associated negative health consequences (88, 89). The next frontier in conservation physiology research may be understanding the physiological and behavioral consequences of chronic adverse experience and GC dysregulation in the context of the collective adaptive or maladaptive behavioral, immune (e.g., inflammatory), autonomic, and neuroendocrine mediator responses (i.e., those involved in growth, reproduction, and metabolism) (90, 91).

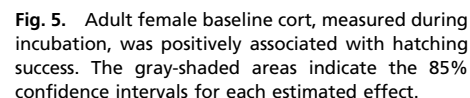
We conclude by noting that conditions at our sites are not unusually loud compared with anthropogenic noise found in many areas across the United States, or globally. In the contiguous United States, estimates of the land area exposed to even moderate amplitude increases of 10 A-weighted decibels [dB(A)] above natural levels is ~485,268.16 km<sup>2</sup> (92), and highly restricted protected areas and critical habitat for species of concern are not immune (3). A 10 dB(A) increase above natural levels translates into a 90% decrease in listening area (1, 3) (i.e., the spatial extent of detection of acoustic signals or cues)—clearly a drastically reduced perceptual world. In this era of unprecedented, large-scale human-driven environmental change, preservation or recovery of natural acoustic conditions should be a key aspect of conservation planning and is a critical step toward successful conservation of protected species.

## Materials and Methods

**Study System.** We conducted fieldwork, following University of Colorado Boulder Institutional Animal Care and Use Committee guidelines (Protocol 1404.03), at a long-term study site in the Bureau of Land Management's Rattlesnake Canyon Habitat Management Area (RCHMA) in NW New Mexico during late spring and early summers from 2011 to 2014. The piñon (*Pinus edulis*)–juniper (*Juniperus osteosperma*) woodland and shrub grassland ecosystem of RCHMA is heavily affected by large-scale development of oil and gas infrastructure. Although developed by industry, RCHMA is remote, and animals in this region have very little contact with humans outside of minimally trafficked dirt roads and exposure to industry workers on well pads. Throughout this system, we installed a network of 240 cedar latched-roof nest boxes (53). Although nest boxes were designed to exclude common opportunistic nest predators such as Woodhouse's scrub-jay (*Aphelocoma woodhouseii*) and the least chipmunk (*Tamias minimus*) that appear largely responsible for nest predation in this system (2, 6), instances of predation by generalists such as deer mice (*Peromyscus* spp.) and bull snakes (*Pituophis catenifer sayi*) were occasionally observed at nest boxes across all noise levels.

We utilized the gradient of noise at RCHMA by placing nest boxes throughout the study area in 12 pairs of control and treatment sites, with 10 boxes per site. All sites were fully operational with nearly identical industrial equipment and similar levels of human activity on treatment and control sites (*SI Materials and Methods*). The key distinction between treatment and control sites was the presence of large compressor engines, which constantly produced high-amplitude, low-frequency noise on treatment sites (Fig. 1). Each control site was located at least 500 but no more than 1,000 m from its treatment pair, assuring that it was close enough to the treatment site to control for localized factors related to land cover composition but was far enough away to not be significantly affected by the noise from the compressor. Each control site was at least 500 m from any other noise-producing wells that were not involved in this study. Noise levels across all pairs of treatment and control sites differed significantly, while forest cover and vegetation did not (2, 53). We placed nest boxes in a circular pattern at ~75 m, 125 m, and 175 m from the center of the well pad on control sites and from the compressor on treatment sites (Fig. 1). By placing the boxes at uniform distances from site centers we control for the effect of disturbance created by industry workers servicing equipment.





The second model, which is the first of two models focused on uncovering the effect of noise on fitness, explored the relationship between noise and egg-hatching success. We defined hatching success as a binomial response in which each egg that was laid in a nest was included; those that hatched were marked as a 1, and those that did not hatch were marked as 0 (107). The



binomial response term for hatching was included in models with noise, species, lay date, distance to well pad, tree cover, and clutch size.

In addition to measuring the effect of noise and other factors on hatching success, a direct measure of reproductive success, we created a third modeling set that included nestling condition as a response term. Nestling developmental changes can be a subtler but equally important indicator of fitness (52, 77). We used principal components analysis (PCA) with varimax rotation (principal function in the psych library for R) to load measurements of nestling wing chord (in millimeters), rectrices (in millimeters), tarsus length (in millimeters), and mass (in grams) onto appropriate axes. These measurements were all taken on the 12th day after hatching, because that is the final day where nest visitation is unlikely to instigate disturbance-induced pre fledgling (North American Bluebird Society, [www.nabluebirdsociety.org](http://www.nabluebirdsociety.org)). These morphological measures loaded on two axes with PC1 explaining 47% and PC2 37% of the variation in the data (Table S2). Wing chord length and length of rectrices loaded strongly onto PC1, while mass and tarsus length loaded strongly onto PC2; because of these loadings, we named these axes "PC<sub>feather</sub>" and "PC<sub>size</sub>," respectively. We placed these axes as response terms in models that include fixed effects for noise, species, tree cover, distance to well pad, lay date, and brood size. Because nestling mass is known to fluctuate throughout the day, we also included a term for time of day in model selection for PC<sub>size</sub>. After initial exploration of data distributions, we included both PC<sub>feather</sub> and PC<sub>size</sub> as second-order polynomials, suggesting that developmental effects of noise might vary nonlinearly with amplitude. In experimental research with humans, vigilance is known to peak at medium amplitudes of noise (108). This has important implications, via noise-mediated foraging-vigilance trade-offs (7), and could translate to reduced provisioning rates and altered development in nestlings, in a similar nonlinear fashion.

Finally, we explored the relationship between cort and fitness. In three separate models, we incorporated nestling body condition as a response to nestling baseline cort, nestling acute stressor-induced cort, and maternal baseline cort measured during provisioning. We also analyzed the effect of maternal baseline cort measured during incubation on hatching success. For all cort-fitness models we included an interaction between cort and species, as cort levels are known to vary systematically across species. Although the focus of this paper is to explore the effect of acoustic habitat disturbance on

physiology and fitness, and not the effect of maternal cort on nestling growth, we included these models to more fully examine if changes to baseline cort resulting from noise exposure are linked to negative fitness consequences. We did not use noise as a predictor in these models to better isolate effects of maternal cort on fitness-relevant measures.

For all models, we included random effects terms to control for variation in data attributable to environmental factors associated with pair, site, and nest box (Fig. 1) and also included a random effect for year. Nestling cort and fitness models included a random effect for Nest ID to control for brood identity in nestlings and for multiple breeding attempts in adults.

Model selection was conducted using an information criterion approach to compare AIC<sub>c</sub> scores between models. All continuous fixed effects were transformed to z-scores using the scale function in R, which allows direct comparison of effects and improves model fit. After initially fitting the full model, random effects that explained near-zero variance (variance  $\leq 0.0001$ ) were removed to improve the fit of the model (109). Additionally, we refit models as GLMM if all random effects were removed during model selection. We considered models with all combinations of hypothesized predictor variables and designated all models within two  $\Delta AIC_c$  and that differed from the null model with identical random effects as determined by a likelihood ratio test as strongly supported. We calculated effect sizes and 85% CIs for individual predictors from supported models (7, 110). For full results, including effects of other predictors and model selection, see Tables S3–S7. All data and the R code used to produce this work will be made available online in a Dryad digital data repository.

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