NOISE POLLUTION

Pre- and postnatal noise directly impairs avian development, with fitness consequences

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Noise pollution is expanding at an unprecedented rate and is increasingly associated with impaired reproduction and development across taxa. However, whether noise sound waves are intrinsically harmful for developing young—or merely disturb parents—and the fitness consequences of early exposure remain unknown. Here, by only manipulating the offspring, we show that sole exposure to noise in early life in zebra finches has fitness consequences and causes embryonic death during exposure. Exposure to pre- and postnatal traffic noise cumulatively impaired nestling growth and physiology and aggravated telomere shortening across life stages until adulthood. Consistent with a long-term somatic impact, early life noise exposure, especially prenatally, decreased individual offspring production throughout adulthood. Our findings suggest that the effects of noise pollution are more pervasive than previously realized.

Noise pollution has become ubiquitous, even reaching Earth's most remote areas (*1*). Widespread impacts of noise pollution on animals have long been documented, primarily on acoustic communication and behavior (*2*, *3*), and more recently on physiology and reproduction (*4*–*8*). Evidence for the effects of noise during development is also starting to accumulate across animal species (*7*–*13*), as well as in humans (*14*, *15*). Notably, noise exposure in early life is associated with changes in stress physiology, telomere length, and even embryonic survival (*7*, *9*–*12*, *15*). Beyond immediate impacts, these effects are particularly concerning given the established consequences of early-life conditions for animal fitness and human health (*16*, *17*). Yet, we know surprisingly little about how noise impairs development and fitness.

The vast majority of developmental effects of noise documented to date in observational and experimental studies [e.g., (*7*–*15*)] cannot be attributed to a direct impact of noise itself on development, because relationships are confounded by the concurrent exposure of parents to noise and its subsequent indirect effects on offspring. Studies exposing only offspring are extremely rare and typically restricted to exposure to very high noise levels (>100 dB) capable of causing physical damage (*18*, *19*), lack comparison to another sound of comparable intensity, or focused on more mature juveniles that were past most major developmental transformations (*20*–*22*). Therefore, it remains unclear whether the mere physical properties of noise sound waves, at

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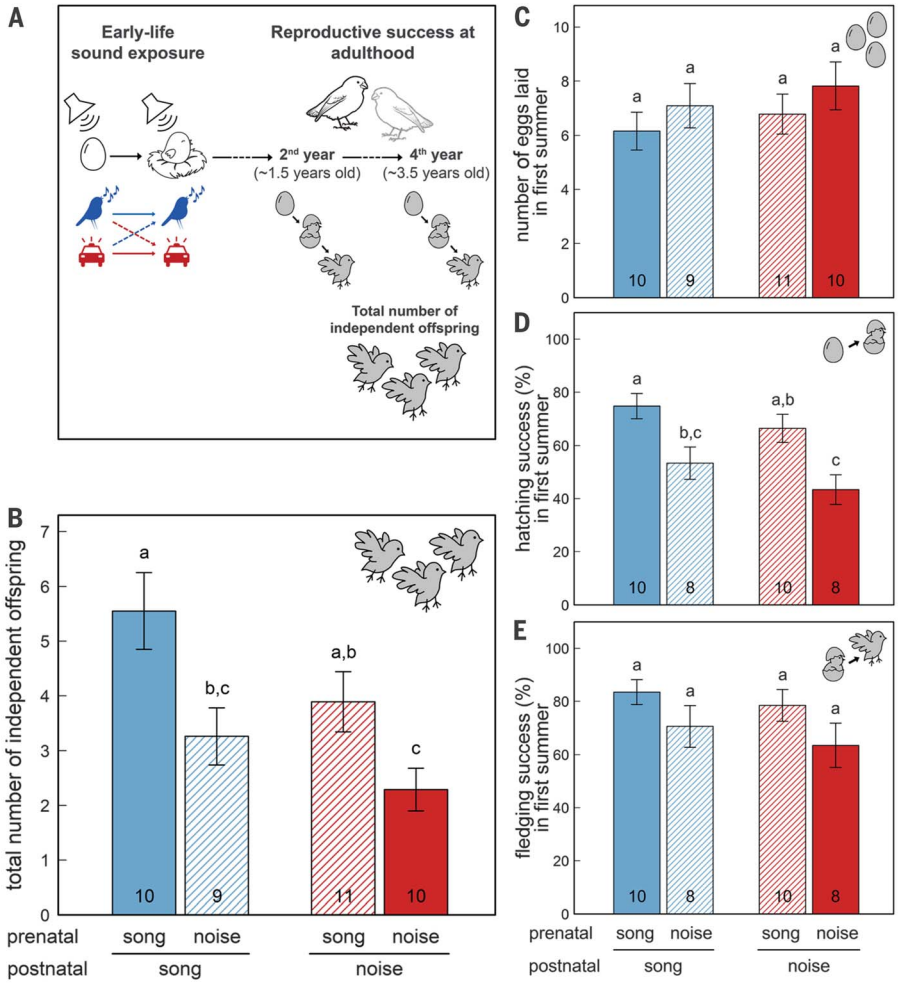

Fig. 1. Fitness consequences of early-life sound exposure. (**A**) During development, experimental birds were exposed to song or noise playbacks pre- or postnatally, or in both life stages. Their subsequent reproductive output and the survival of their offspring were measured at adulthood. (**B**) Total number of independent offspring produced across all breeding opportunities (first summer and fourth year; "realized fitness"); and, for the first summer: (**C**) number of eggs laid, (**D**) proportion of eggs laid that hatched, (**E**) proportion of hatchlings that fledged. (C to E) Effects were similar in late adulthood (i.e., fourth year; see fig. S3 and table S3). Data show estimated marginal mean (EMM) ± SE (also accounting for mass at hatching of experimental birds; tables S1 and S2). Different letters indicate significant differences between groups (Tukey's honest significant difference test adjusted for multiple comparisons, $P_{adj} < 0.05$). Numbers in bars indicate sample sizes.

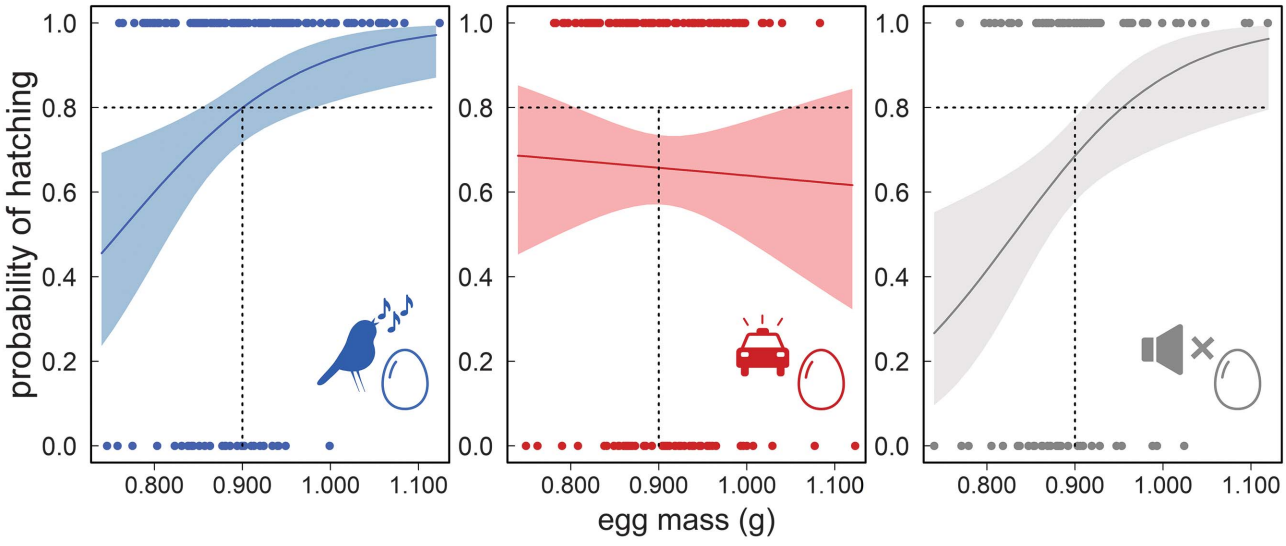

Fig. 2. Direct effects of prenatal sound exposure on embryonic survival of experimental individuals. Probability of hatching in relation to egg mass in eggs exposed to song (left), noise (middle), or silence (right). Regression lines from the GLMM (table S4) and 95% confidence intervals (CIs) (shaded areas) are shown, with raw data points for individual eggs. Horizontal and vertical dotted black lines show the hatching success (80%) in the song group at mean egg mass (0.900 g) on all three panels for comparisons among treatment groups. Hatching success was significantly lower in eggs exposed to noise, compared to song at mean egg mass and to both song and silence for heavier eggs (table S6). Hatchling mass was used as a covariate in all subsequent analyses, to control for differential hatching success relative to egg size.

ecologically relevant levels, can disrupt vertebrate developmental processes. Yet, whereas behavioral habituation to noise (by the parents) may allow adaptation to noise pollution, direct developmental interference by noise would be much harder to counter. Therefore, if we are to predict and mitigate the risks posed by noise pollution, it is essential to establish how sound waves interfere with, or enable, fundamental biological processes.

Indeed, recent evidence suggests that sole exposure to prenatal natural sounds or vibrations can alter postnatal developmental trajectories and adaptively program individual phenotypes (*23*, *24*). This is thought to occur as prenatal natural vibro-acoustic stimuli provide anticipatory cues about postnatal environmental conditions (*23*–*25*). Noise exposure during prenatal life may thus similarly either improve offspring resilience to postnatal exposure, or instead have a cumulative negative effect across life stages. However, the interactive effect of pre- and postnatal noise exposure on phenotype has not been considered in any species, and the fitness consequences are unknown.

Here, in an altricial vertebrate species, the Australian zebra finch (*Taeniopygia guttata castanotis*), we quantified the fitness impact of developing in a moderately noisy environment, when nothing but the acoustic experience differs. Without altering the parental environment, we experimentally manipulated offspring pre- and postnatal exposure to anthropogenic traffic noise, in wild-derived birds (*26*). To test for preparatory versus cumulative effects of prenatal exposure, we used a full factorial match-mismatch design, whereby individuals were exposed to playbacks of traffic noise or species-specific sounds (i.e., zebra finch songs) pre- or postnatally, or in both life stages (*26*) (fig. S1). At the embryonic stage, eggs were artificially incubated and exposed to overnight playbacks during the last 5 days of incubation; while at the nestling stage, individuals were naturally raised by undisturbed parents but taken away overnight to be exposed to playbacks from 4 to 13 days after hatching (*26*). Mimicking peak traffic hours or song chorus times, playbacks in both life stages occurred in evenings and early mornings, for a total of 4.5 hours (*26*). Sound levels at 65 dB for both playbacks were comparable to song and call levels experienced within the nest (*27*), or noise levels birds may encounter in a moderate urban or roadside environment (*28*), and were well below the threshold causing injuries to hearing or other organs (*18*, *19*).

Early-life noise exposure reduced reproductive success throughout adulthood

After pre- and postnatal sound exposure, we measured the fitness impact of such early-life acoustic experiences, by quantifying reproductive output throughout adulthood (*26*) (Fig. 1A). At the beginning and toward the end of their reproductive life (i.e., second year of life (first summer) and fourth year of life), experimental birds were allowed to pair and breed freely with unmanipulated partners, for 3.5 months each year (i.e., long enough to produce two successful broods per season) (*26*). We found that both prenatal and postnatal exposure to traffic noise significantly reduced individual total reproductive output across all breeding opportunities [i.e., "realized fitness"; generalized linear model (GLM), prenatal treatment: $X^2_1 = 9.52$, $P = 0.002$; postnatal treatment: $X^2_1 = 4.65$, $P = 0.031$; $n = 40$ adults] (Fig. 1B and table S1). As a result, individuals that were subjected to noise both pre- and postnatally produced 59% fewer independent offspring overall than those that had never been exposed to noise in early life (i.e., "song-only") (Fig. 1B). This deleterious noise effect on reproduction was strongest in the first breeding season (with twice the offspring sample size) and for prenatal rather than postnatal noise exposure [GLMs, first season (first summer), prenatal treatment: $X^2_1 = 6.74$, $P = 0.009$; postnatal treatment: $X^2_1 = 2.23$, $P = 0.135$; second season (fourth year), prenatal treatment: $X^2_1 = 3.74$, $P = 0.053$; postnatal treatment: $X^2_1 = 2.94$, $P = 0.086$; $n = 40$ and 25 adults, respectively; tables S2 and S3] (fig. S2). In both breeding seasons, the lower offspring production was not due to a reduction in reproductive investment (GLMs, number of eggs laid, prenatal and postnatal treatments: all $P > 0.260$) (Fig. 1C, fig. S3, and tables S2 and S3). Rather, it was largely attributable to a lower hatching success for eggs produced by individuals exposed to noise prenatally (GLMs, first summer: $X^2_1 = 12.47$, $P < 0.001$; fourth year: $X^2_1 = 6.39$, $P = 0.011$) (Fig. 1D, fig. S3, and tables S2 and S3), with fledging success also

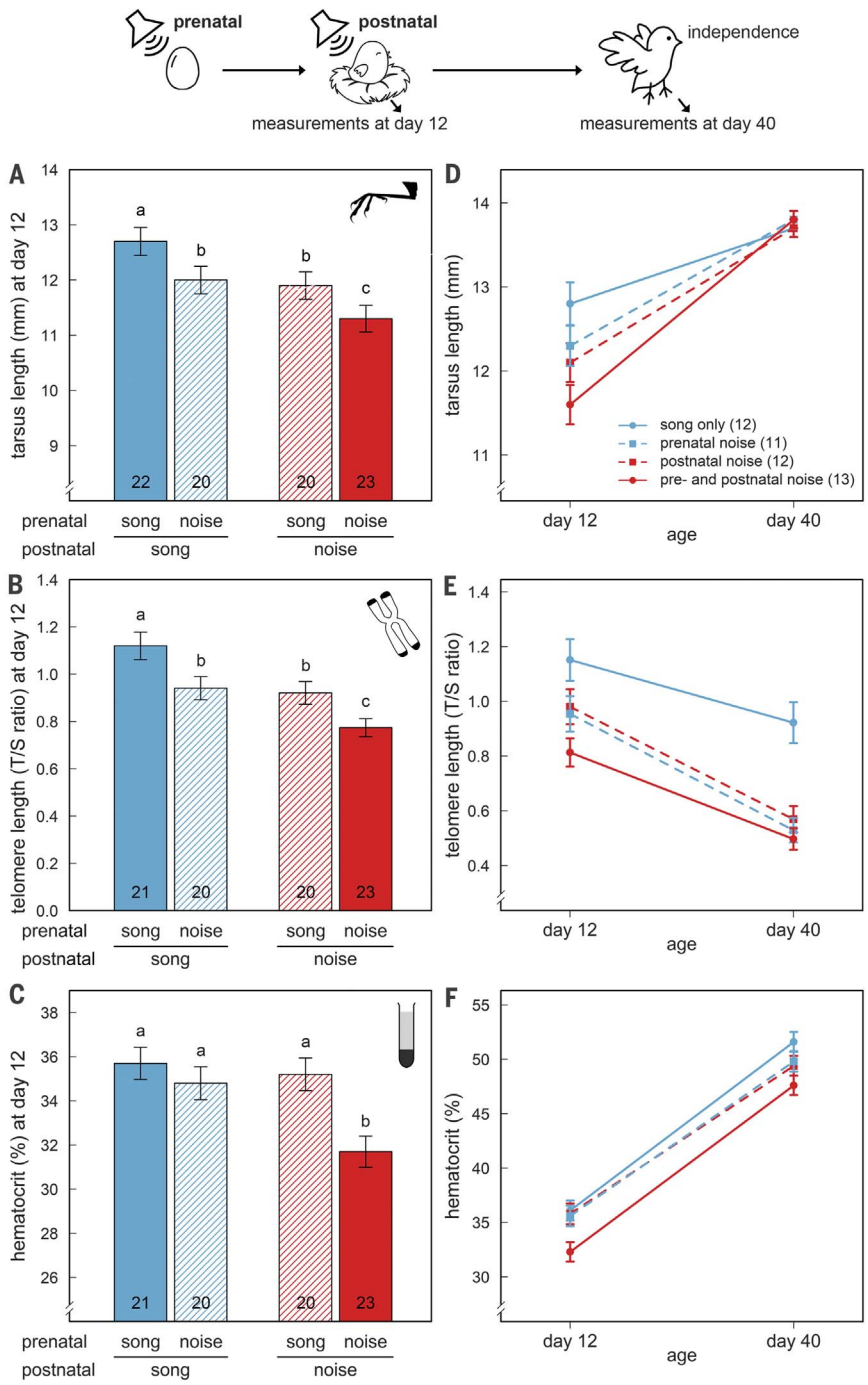

Fig. 3. Effects of prenatal and postnatal sound exposure (song or noise) on experimental individual growth and physiology. Tarsus length, telomere length, and hematocrit level (**A** to **C**) at day 12 (i.e., during postnatal acoustic treatment) and (**D** to **F**) from days 12 to 40 (i.e., up to ~1 month after acoustic treatment). Data show EMM ± SE (also accounting for hatchling mass; tables S8, S10, and S17). [(A) to (C)] Different letters indicate significant differences between groups (P_{adj} < 0.05). Numbers in bars [(A) to (C)] or brackets [(D) to (F)] indicate sample sizes [which were reduced at day 40 owing to mortality of two cohorts during unexpected disease outbreaks (*26*)].

contributing, but to a lesser extent (GLMs, first summer: X^2_1 = 3.34, P = 0.068; fourth year: X^2_1 = 0.21, P = 0.644) (Fig. 1E, fig. S3, and tables S2 and S3). Higher offspring mortality among noise-exposed birds may indicate lower offspring viability and/or follow from reduced parental care from experimental birds or their partners [possibly of lower body condition; but see (*26*)]. Nonetheless, it is noteworthy that noise impacts on hatching success remained consistent in both breeding seasons, in spite of breeding experience and new partners (tables S2 and S3). Overall, regardless of the contributing factors, our results show that noise exposure in early life, especially prenatally, affects individuals' offspring production throughout adulthood, and thereby, their realized fitness.

To elucidate how such fitness reduction came about, we assessed the direct impact of noise on experimental individuals' development, during acoustic exposure pre- and postnatally, and until adulthood.

Prenatal noise directly reduced embryonic survival

To test whether, and how, prenatal noise may impair embryonic development during exposure, and specifically whether this occurs through the disruption of essential auditory input, we included a third concurrent treatment group that was incubated in silence [used for prenatal effects only; see below and (*26*)] (fig. S1). These three prenatal treatment groups allowed for the quantification of the effects of both noise exposure and natural sound deprivation independently. We found that prenatal playback had a direct impact on embryonic survival [generalized linear mixed model (GLMM), X^2_1 = 7.45, P = 0.024, n = 393 eggs] (table S4), but not on hatchling mass (LMM, $F_{2,216.50}$ = 0.83, P = 0.437, n = 275 hatchlings) (table S5). On average, hatching success was significantly lower for embryos exposed to noise, compared to those experiencing song playback (corresponding to an 18.5% decrease at average egg mass), whereas hatching success in the silence group was intermediate (Fig. 2 and table S6). The effect of sound varied with egg mass (GLMM, prenatal treatment × egg mass: X^2_2 = 9.17, P = 0.010) (table S4). As commonly observed in birds (*29*), hatching success increased with egg mass, for embryos exposed to either song or silence (P = 0.003 and P = 0.005, respectively) (Fig. 2). However, this typical pattern was disrupted by noise exposure (P = 0.769), with heavier eggs disproportionally affected by noise (Fig. 2 and table S6). Therefore, prenatal noise exposure itself affected embryonic developmental patterns, independently of any natural sound deprivation effects. Such stimulus-specific effects of noise sound waves on a songbird are particularly compelling, given that hearing is considered to only develop postnatally in altricial avian species (*30*). Effects of prenatal noise in this species, on individual embryonic survival, and on that of their offspring suggest that the impact of anthropogenic noise across taxa may be far more damaging than previously realized.

Pre- and postnatal noise impaired nestling growth and physiology

We quantified the impact of noise or song exposure pre- and postnatally on individual postnatal traits on a subset of broods [the silence

treatment was excluded postnatally, because all nestlings were exposed to natural sounds during daytime and therefore not auditorily deprived (*26*)] (fig. S1). We found that both pre- and postnatal noise exposure impaired nestling growth compared to song playbacks, with additive negative effects on growth trajectory from 5 to 13 days after hatching (LMM, age × prenatal treatment: $F_{1,676.00} = 35.71$, $P < 0.0001$; age × postnatal treatment: $F_{1,676.00} = 13.99$, $P < 0.001$, $n = 85$ nestlings) (fig. S4 and table S7). Individuals that were exposed to traffic noise both as embryos and nestlings were 11% smaller (tarsus length) and 14.5% lighter (mass) at day 12 than those that had never been exposed to noise (Fig. 3A, fig. S5, and table S8). Previous exposure to prenatal noise did not prevent further effects of noise postnatally and was as detrimental as the postnatal exposure occurring concurrently with growth measurement (Fig. 3A: Both mismatched treatments are significantly different compared to noise- or song-only treatments and do not differ from each other). Notably, these morphological differences were not just a consequence of differential prenatal survival relative to egg size, because there was no difference in weight at hatching among treatment groups (table S5), and hatchling mass was accounted for in all models [from nestlings to adulthood (*26*)]. Because growth is an integrative measure of individual development, multiple cellular, physiological, and behavioral factors likely contribute to this trait. For example, direct alteration by noise of nestling phenotype could have in turn affected their interactions with the foster parents, leading to reduced food provisioning, compared with their song-exposed nestmates. However, we found no indication of this, using an indirect measure of food intake [GLMMs, seed count in crop in the evening (*26*), prenatal and postnatal treatments, on day 7: all $P > 0.486$; on day 12: all $P > 0.256$] (table S9). Although future studies are required to identify the underlying mechanistic pathways through which sound waves altered growth, our results show that noise exposure was the direct initial trigger of these changes, because individual experience was otherwise exactly the same, and parents were not exposed.

Likewise, nestling physiology was also broadly affected, with physiological traits at day 12 exactly following patterns observed for growth. Nestling telomere length [the protective ends of chromosomes (*31*)] was significantly reduced by noise exposure at both life stages, with prenatal and postnatal effects being cumulative and equally strong (LMM, prenatal treatment: $F_{1,52.02} = 10.35$, $P = 0.002$; postnatal treatment: $F_{1,76.90} = 11.81$, $P < 0.001$) (Fig. 3B and table S10). These results are consistent with previous findings on the effect of noise on telomere dynamics in wild passerines (*10*, *11*) but reveal that noise itself—without any parental disturbance—has a direct impact. Similarly, nestling hematocrit levels [percentage of red blood cells in total blood volume (*32*)]) also indicated a detrimental impact of noise on nestling condition, with individuals exposed to noise during both pre- and postnatal life having significantly lower levels than all others (LMM, prenatal × postnatal treatments: $F_{1,50.24} = 4.16$, $P = 0.047$) (Fig. 3C and table S10). It is worth noting that, overnight displacement, and playback per se, did not have any detrimental effect on individuals' morphological and physiological traits (fig. S6 and tables S11 to S15), as shown through comparison to control nestlings remaining in (silent) parental nests overnight (*26*). In addition, because all nestlings were likely exposed to higher sound levels from the colony background sound during the day [>80 dB (*27*)] than during overnight noise or song exposure (both at 65 dB), our study demonstrates the harmful impact of the acoustic properties of noise specifically, rather than a broad negative effect of loud sounds.

Overall, our findings show that sole exposure to noise in early life can alter individual development and phenotype. In addition, our cross-factorial design uncovered that these alterations occur even when noise is only experienced prenatally, and that prenatal exposure has no preparatory effect that would lessen the impact of later exposure.

Effects on telomeres persisted after exposure and predicted reproductive prospects

Telomere length reflects the level of cellular damage that the individual has experienced, with attrition in early life expected to be more pronounced than later in life (*33*). As a bioindicator of cellular aging, predictive of longevity in this and other taxa (*34*, *35*) or lifetime reproduction (*36*, *37*), reduction in telomere length may persist after exposure and forecast later-life fitness.

In agreement, effects on telomeres persisted well beyond the end of noise exposure (at day 13) (*26*). Telomere attrition rate between 12 and 40 days after hatching (nutritional independence) was greater in individuals exposed to noise at one or both life stages, compared with individuals that had never been exposed to noise (LMM, prenatal × postnatal treatments: $F_{1,35.36} = 12.18$, $P = 0.001$, $n = 48$ individuals) (Fig. 3E and table S16). Effects on morphology nonetheless had eroded by day 40 (with no difference in tarsus length or mass; fig. S7 and table S17), given that compromised individuals compensated by accelerating their growth after exposure (Fig. 3D), as typically observed in birds after developmental stress (*38*). However, as expected (*38*), such "catch-up growth" was accompanied by telomere erosion, because higher growth rate from 12 to 40 days was associated with more severe telomere shortening during that period (LMM, mass gain: $F_{1,39.47} = 7.10$, $P = 0.011$) (fig. S8 and table S16). As a result, by day 40, all individuals that had experienced noise at some stage during their development

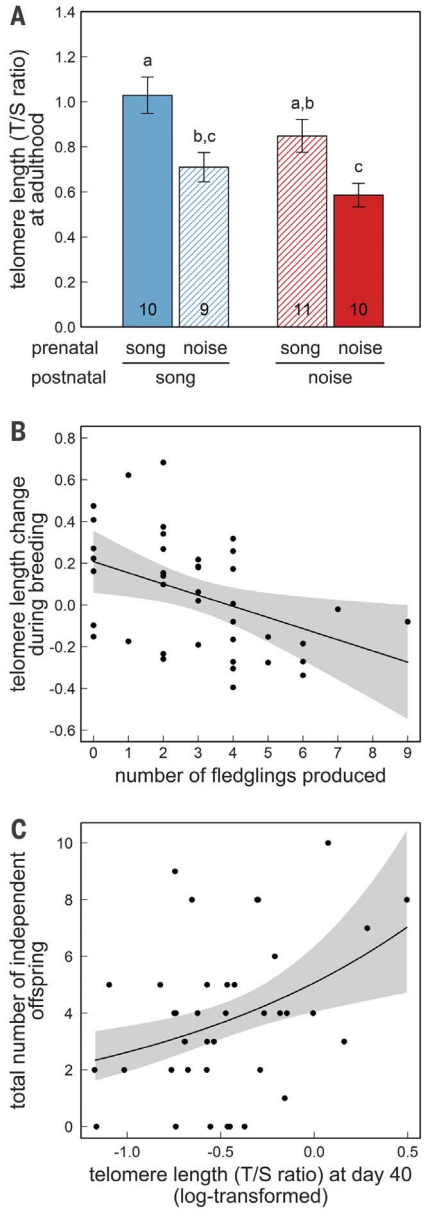

Fig. 4. Long-term effects of early-life sound exposure on telomeres and reproductive prospects. (**A**) Effects of prenatal and postnatal noise exposure on telomere length at adulthood (before first breeding season). (**B**) Relationship between telomere length change and number of fledglings produced during the first breeding season. (**C**) Relationship between telomere length at day 40 (log-transformed) and total offspring production throughout adulthood. (A) Data show EMM ± SE (also accounting for mass at hatching; table S18). Different letters indicate significant differences between groups ($P_{adj} < 0.05$). Numbers in bars indicate sample sizes. [(B) and (C)] Regression lines and 95% CIs (shaded areas) from models in tables S18 and S19 are shown.

had 38 to 46% shorter telomeres than those that had never been exposed to noise (Fig. 3E, fig. S7, and table S17). In addition to shortened telomeres, individuals that were exposed to pre- and postnatal noise had lower hematocrit levels at day 40 than song-only individuals (LMM, prenatal treatment: $F_{1,34.69} = 3.13$, $P = 0.085$; postnatal treatment: $F_{1,42.58} = 4.49$, $P = 0.040$) (Fig. 3F, fig. S7, and table S17). Overall, both pre- and postnatal noise impaired all nestling morphological and physiological traits that we measured, with especially strong and lasting effects on telomere length, which got more pronounced by the time individuals reached nutritional independence.

From day 40 to first reproduction, experimental birds remained undisturbed in communal outdoor aviaries for ~1.5 years (*26*). Even after that time, the impact of early-life noise exposure on telomere length persisted: The prenatal noise effect remained as strong at adulthood (LM, $F_{1,35} = 13.71$, $P < 0.001$), whereas that of postnatal exposure had weakened ($F_{1,35} = 3.67$, $P = 0.063$, $n = 40$ adults) (Fig. 4A and table S18). The impact of prenatal noise exposure on telomere length was present both before and after breeding (table S18). This was despite noise-exposed individuals only producing half as many offspring, and individuals' telomere shortening during the breeding season being negatively related to their offspring production (LM, $F_{1,33} = 6.33$, $P = 0.017$) (Fig. 4B and table S18). This suggests that song-exposed individuals were able to produce more offspring without paying a higher physiological cost (reflected in telomere length). Telomere shortening—or, more likely, the associated somatic state that telomere length reflects—could thus potentially continue to affect individual fitness, without noticeable compensation in later life for noise-exposed individuals. In agreement, individual telomere length at day 40 (and, to a lesser extent, at day 12) predicted their reproductive prospects, with individuals with longer telomeres at day 40 producing more offspring throughout adulthood (GLMs, day 40: $X_1^2 = 11.11$, $P < 0.001$; day 12: $X_1^2 = 4.20$, $P = 0.040$) (Fig. 4C and table S19). Overall, our results demonstrate that directly experiencing noise during development, while receiving normal unaltered parental care, triggers changes in individual developmental trajectory and its associated costs. This acoustic experience, reflected in telomere length as a biomarker until adulthood, has carry-over effects on realized fitness.

Conclusions

Our study echoes recent studies on the pervasive effects of anthropogenic noise on reproduction, species communities, ecosystems, and human health (*6*–*9*, *15*, *39*, *40*) but reveals the disruptive properties of moderate noise sound waves themselves on physiology, development, and ultimately reproduction, leading to life-long fitness reduction by traffic noise exposure. How traffic noise could have such an intrinsic impact on embryos and nestlings that lacked the opportunity to learn to associate traffic noise with a threat is puzzling. Further investigations into the physical properties, biological underpinnings, and evolutionary origins of such interference are therefore needed. We also showed that no habituation to noise occurred from pre- to postnatal life; and, even in an altricial bird with uncertain auditory perception, prenatal development was particularly sensitive to noise, with large effects on phenotype and fitness across life stages until late adulthood. An impact of such magnitude from ecologically relevant noise levels is concerning, for wildlife and humans alike. Our findings therefore call for a reassessment of the threat posed by anthropogenic noise, and the need for mitigation measures to reduce noise production and exposure, not least during early development. Action is particularly urgent, given the ongoing and projected increase in road networks (*41*), but also in mining, industrial, and domestic activities generating noise pollution, from moderate to high levels.

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SUPPLEMENTARY MATERIALS

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Materials and Methods
Figs. S1 to S8
Tables S1 to S19
References (*43*–*58*)
MDAR Reproducibility Checklist

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