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Adult telomere length is positively correlated with survival and lifetime reproductive success in a wild passerine

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Abstract

Explaining variation in individual fitness is a key goal in evolutionary biology. Recently, telomeres, repeating DNA sequences capping chromosome ends, have gained attention as a biomarker for body state, physiological costs, and senescence. Existing research has provided mixed evidence for whether telomere length correlates with fitness, including survival and reproductive output. Moreover, few studies have examined how the rate of change in telomere length correlates with fitness in wild populations. Here, we intensively monitored an insular population of house sparrows, and collected longitudinal telomere and life history data (16 years, 1225 individuals). We tested whether telomere length and its rate of change predict fitness measures, namely survival, lifespan and annual and lifetime reproductive effort and success. Telomere length positively predicted short-term survival, independent of age, but did not predict lifespan, suggesting either a diminishing telomere length—survival correlation with age or other extrinsic factors of mortality. The positive association of telomere length with survival translated into reproductive benefits, as birds with longer telomeres produced more genetic recruits, hatchlings and reared more fledglings over their lifetime. In contrast, there was no association between telomere dynamics and annual reproductive output, suggesting telomere dynamics might not reflect the costs of reproduction in this population, potentially masked by variation in individual quality. The rate of change of telomere length did not correlate with neither lifespan nor lifetime reproductive success. Our results provide further evidence that telomere length correlates with fitness, and contribute to our understanding of the selection on, and evolution of, telomere dynamics.

KEYWORDS
individual fitness, reproductive success, senescence, survival, telomere dynamics

Hannah L. Dugdale and Julia Schroeder share last authorship.

1 | INTRODUCTION

Understanding how an organism's fitness is influenced by its traits is a central tenet in evolutionary biology. While most measurable traits are manifested at the organismal level, for example in reproduction, survival, and behaviour, it is important to examine traits at deeper levels of biological organization, including cell and body physiology, as they underlie organismal performance. One such trait is telomere dynamics, which could reflect the cellular and body state of the organism, bridging together physiology and fitness.

Telomeres are nucleoprotein complexes at the ends of chromosomes consisting of repeating DNA sequences ($TTAGGG_n$ in vertebrates; Blackburn, 1991). Telomeres are vulnerable to erosion due to the end-replication problem, where linear DNA is not fully replicated during cell proliferation (Levy et al., 1992; Olovnikov, 1973), and chemical damage from oxidative stress (Blackburn et al., 2015; von Zglinicki, 2002). They therefore shorten over time. Shortened telomeres can be restored, for example by telomerase, but telomerase activity varies across life stages and species (Haussmann et al., 2007), and is generally thought to be suppressed in adult somatic cells in humans and other mammals (Blackburn et al., 2015; Young, 2018). This creates a decline of telomere length throughout lifespan, typically rapidly during early life due to prominent cell proliferation, and more slowly in adulthood (Heidinger et al., 2012; Spurgin et al., 2018; Stier et al., 2020), though patterns vary across taxa (Remot et al., 2022). When telomeres are critically short, cells enter a senescent state, and can undergo apoptosis, leading to a decline in tissue function (Blackburn et al., 2015; Campisi, 2005). This decline in function could lead to declined body performance and health, linking telomere dynamics with mortality risks. In addition, the link between oxidative stress and telomere damage also means that telomere length could reflect the cumulative energetic costs of stress-inducing activities, for example reproduction (reviewed in Monaghan, 2010). Because of this, telomere length, and the rate of telomere shortening, have gained attention in evolutionary biology and epidemiology, as biomarkers of body state, individual quality (e.g. Angelier et al., 2019; Bauch et al., 2013; Monaghan, 2010), a measurement of physiological costs in life-history trade-offs (e.g. Bauch et al., 2013) and a hallmark of senescence (e.g. López-Otín et al., 2013).

Because telomeres link to cellular senescence, thereby tissue function (Blackburn et al., 2015; Campisi, 2005), and vital rates such as mortality (Wilbourn et al., 2018) and fecundity (e.g. Bauch et al., 2013), and perhaps therefore actuarial senescence, one would expect telomere dynamics to be under selection, and associated with fitness measures. However, studies examining the relationship between telomere dynamics, survival and lifespan have provided mixed results. On average, shorter telomeres are associated with higher mortality, but variation exists for example among studies using different telomere length measurement techniques (Wilbourn et al., 2018). Some studies found positive relationships between early-life telomere length and survival or lifespan (e.g. Eastwood et al., 2019; Fairlie et al., 2016; Heidinger et al., 2012; Sheldon et al., 2022; van Lieshout et al., 2019); while others found such a relationship also in adults (e.g. Bakaysa et al., 2007; Bichet et al., 2020; Froy et al., 2021; Vedder et al., 2022), even at a genetic level (Vedder et al., 2022). There has also been evidence that telomere shortening predicts survival and/or lifespan, both intraspecifically (Boonekamp et al., 2014; Brown et al., 2022; Whittemore et al., 2019; Wood & Young, 2019) and interspecifically (Tricola et al., 2018). To date, however, it remains unclear whether, and how, telomere biology causally contribute to organismal senescence (Simons, 2015; Young, 2018) and fitness variation.

The link between telomere dynamics and reproductive output, another essential component of fitness, also demands attention (Sudyka, 2019). Two main hypotheses link telomere dynamics with variation in reproductive output: (1) the 'individual quality hypothesis' suggests that individuals with longer telomeres and/or slower telomere shortening are of higher quality either due to genetic differences (e.g. Pepke et al., 2023) or environmental variation, e.g. better habitats offering more resources and thus less stress, such that these individuals both live longer and have higher lifetime and annual reproductive output, generating a positive relationship between telomere dynamics and reproduction (e.g. Angelier et al., 2019; Heidinger et al., 2021). (2) The 'pace-of-life hypothesis' suggests that individuals differ in their relative energetic investment in self-maintenance versus reproductive effort, such that individuals with a slower pace-of-life (i.e. investing more into self-maintenance) would exhibit longer telomeres and slower shortening, in exchange for decreased annual reproductive output, resulting in a negative relationship between telomere dynamics and reproduction (Bauch et al., 2020; Bichet et al., 2020; Eastwood et al., 2019; Heidinger et al., 2021; Ravindran et al., 2022). So far, research has largely provided mixed results: Evidence for the 'individual quality hypothesis' was found by for example Angelier et al. (2019); Eastwood et al. (2019) and Heidinger et al. (2021), whereas evidence for the 'pace-of-life hypothesis' was found by for example Bauch et al. (2013) and Pepke, Kvalnes, Lundregan, et al. (2022); Pepke, Kvalnes, Ranke, et al. (2022). Additionally, it is still unclear how telomere shortening relates to reproductive output. For example, Heidinger et al. (2021) using a population of house sparrows (*Passer domesticus*) did not find an association between telomere shortening and reproductive success, while Sudyka et al. (2019) using a blue tit (*Cyanistes caeruleus*) population found a negative association: increased telomere loss was found in parents of enlarged broods compared to controls. Furthermore, under the context of the pace-of-life hypotheses, telomere dynamics are representative of the costs of reproduction, that is energetic investments in egg production, parental care, and so forth, yet this does not predict ultimate fitness consequences, such as the number of recruits (i.e. who also reproduce offspring). Further testing for these fitness associations with telomere length and shortening, especially in longitudinal, natural systems, can thus enable us to better understand the evolutionary mechanism that drives variation in telomere dynamics.

Here, we examined the links between telomere dynamics and fitness in a free-living, insular population of house sparrows (*Passer domesticus*), using longitudinal telomere measurements that span

16 years, and for which we have survival and lifetime reproductive data unconfounded by emigration. Such longitudinal monitoring means that our data consisted of largely adult (post-fledging) telomere dynamics, which we used here to test: (1) whether adult telomere length predicts immediate survival up to 1 year post-measurement; (2) whether average individual telomere length and its rate of change across adulthood are associated with lifespan; (3) whether adult telomere length is associated with annual reproductive output; and (4) whether average telomere length and its rate of change are associated with lifetime reproductive output. Uniquely, by focusing on reproductively mature individuals, this study examines the links between telomere dynamics and fitness independent of early-life mortality.

2 | MATERIALS AND METHODS

2.1 | Study population and life-history data collection

We systematically monitored and collected life-history data from a free-living, nest-box population of house sparrows (*Passer domesticus*) on Lundy Island (51°10′ N, 4°40′ W), 19 km off the coast of Devon, UK, starting in the year 2000, though non-systematically collected observational and genetic data from as early as 1990 were available, and used for pedigree construction (see below). Adult population sizes fluctuate yearly, with a mean of 421 birds (range = 66–787). Annually, we conducted fieldwork during two periods: the breeding season (April–August) and a shorter winter survival monitoring period (November or December). The Lundy sparrows produce a mean of 2.3 clutches of 4.2 eggs each breeding season (Westneat et al., 2014). During the breeding season, we monitored all nest boxes on the island, and tagged >99% of the population with uniquely numbered metal rings from the British Trust for Ornithology and a unique combination of three colour rings, either as ≥12-day-old nestlings for birds in nest boxes, or during their first winter for birds fledged from wild nests. We were thus able to obtain the hatch year and age of virtually all individuals to the precision of 1 year, and the exact hatch dates for birds hatched in nest boxes. To separate biological parental effects, and environmental effects pre- versus post-hatching, routine fieldwork also includes cross-fostering, where 2- to 3-day-old hatchlings are swapped between broods with matching size and hatching dates. On average 39% of broods were cross-fostered every breeding season (Winney et al., 2015). Due to the geographical isolation of Lundy Island, immigration and emigration is almost absent (three confirmed immigrants up to 2015; Schroeder et al., 2015). We collected survival data through the biannual surveys where we recorded the presence/absence of each bird, and the annual re-sighting probability was between 91%–96% (Schroeder et al., 2015). This is done during both winter and summer fieldwork, where we recapture and sample all present sparrows. Except for those with an explicit death record (134 birds, or 10.9% of our telomere dataset), birds that were not sighted for 2 years consecutively since their last sighting were assumed dead, and the year when they were last seen was assumed as the death year, allowing us to calculate minimum lifespan for each individual.

We repeatedly collected blood samples from individuals, typically at 2 days of age, 12 days of age, and at most subsequent captures after fledging. Blood samples were stored in 96% ethanol at room temperature until DNA extraction using the ammonium acetate method following Richardson et al. (2001) and stored at −20°C until analysis. We then assigned genetic parentage using up to 23 microsatellite markers (Dawson et al., 2012). Using the software CERVUS 3.0 we assigned the genetic parents to >99% recruits with 95% confidence (Kalinowski et al., 2007; Schroeder et al., 2015), totalling 10731 birds in the pedigree used in this work. From this pedigree, we calculated annual reproductive success (ARS) for each bird as the annual number of genetic recruits, that is offspring that reproduced and appeared in the genetic pedigree as dams or sires themselves. We also calculated lifetime reproductive success (LRS) as the sum of ARS across the lifespan of each individual.

2.2 | Telomere extraction and assay

We measured relative telomere length (RTL) from blood samples of sparrows after they fledged, collected from 2000 to 2015. The number of samples taken from a bird within a year ranged from one to five. While we obtained blood samples from juveniles, these were chiefly used for pedigree construction (only 77 juvenile RTL measurements were available), and therefore were not included in our analyses. DNA sample concentration was measured using a Nanodrop 8000 Spectrophotometer (Thermo Fisher) and normalized to 20–30 ng/μL. During normalization we checked for DNA purity by ensuring that the 260/280 and 260/230 absorbance ratios ≥1.8 (Morinha et al., 2020). Next, we used a monochrome multiplex quantitative polymerase chain reaction (MMqPCR) method to quantify RTL (Cawthon, 2009) described in detail in (Chik et al., 2023; Sibma, 2021). In brief, MMqPCR quantifies RTL as a ratio of telomeric signals (T) to that of a single-copy reference gene (S; the gene GAPDH was used in our study), where amplification of both target sequences occur within a single well to eliminate error from sample loading. Samples were allocated to plates using a slicing approach (van Lieshout et al., 2020), where we divided samples from each year into thirds (termed 'slices'), and allocated them such that plates contained three slices, with one overlapping slice. As a result, recently and formerly collected samples were analysed together on the same plate, and plate effects could then be separated from sampling year and storage time effects. We measured samples in duplicates in adjacent wells. On each plate, we also included a reference sample, serially diluted to 80, 20, 5, 1.25 and 0.3125 ng/μL, to generate a standard curve used for calibration of Ct values of the T and S signals and subsequent calculation of RTL independent of plate effects. Standard curves were visually assessed for outliers and up to two outliers could be

removed to improve downstream calculations. Using this standard curve we calculated the quantity of T and S amplification products of the experimental sample in each well, and derived T/S ratios as RTL. We also calculated amplification efficiencies using the standard curves. After MMqPCR, we removed samples with Ct values >25, and between-duplicate relative difference > 0.2. We averaged the RTL of the remaining duplicates as the final measure for each sample. Plates were run using two machines, a QuantStudio 12 K Flex Real-Time PCR System (Thermo Fisher Scientific, five plates) and a StepOnePlus (Applied Biosystems, 77 plates), and by two technicians (MEM ran 52 plates and NdR ran 30 plates), but machine identity did not have an effect on RTL (Sibma, 2021). The mean qPCR amplification efficiencies for telomeres and the reference gene were 89.2% (s.d. = 8.1%, range = 70.7%–110%) and 88.0% (s.d. = 6.8%, range = 70.7%–105%) respectively, across all 82 plates. The mean R^2 of the calibration curves for telomeres and the reference gene were 0.987 (s.d. = 0.013, range = 0.926–0.998) and 0.994 (s.d. = 0.005, range = 0.963–0.999) respectively. We calculated qPCR repeatabilities with the R package rptR 0.9.22 (Stoffel et al., 2017), with 1000 bootstraps. Using the experimental telomere samples, and fitting storage time, technician identity, and sample ID, we calculated the intra-plate, between-duplicate repeatability of RTL to be 95.7% (s.e. = 0.2%, N = 4536 observations from 2162 samples). To assess inter-plate repeatability, the repeatability of the same sample ran across different plates, we took two approaches. First, using standard samples across 25 plates run by the same technician (NdR), we calculated the inter-plate repeatability of Ct for telomeres to be 98.3% (s.e. = 0.4%, N = 126 from 63 samples, which include serial dilutions), and that of Ct for the reference gene to be 99.0% (s.e. = 0.2%). Second, when we first verified our MMqPCR protocol, we assayed a set of captive house sparrow samples on two consecutive plates, ran on two consecutive calendar days. Inter-plate repeatability of RTL calculated from this dataset was high at 97.7% (s.e. = 0.9%, N = 57 observations from 34 samples). Furthermore, to assess variability induced by re-analysis and/or storage effects, we re-extracted and re-assayed a subset of samples. Note that storage and extraction variation is rarely assessed, especially in long term studies. We expected repeatability to therefore potentially be lower than that generally reported for inter-plate repeatability, as both potential storage, handling, re-extraction and other unknown effects contribute. After quality control, we fitted group (old versus new extraction) to correct for the effects of groups on the mean, and the sample identity, and calculated repeatability of RTL to be 52.3% (s.e. = 6.9%, N = 232 observations from 48 samples). The final telomere dataset consisted of 2083 telomere length measurements from 1225 birds, 476 of which have telomere length measurements at multiple ages. Of these 2083 measurements, 1142 (54.8%) were taken outside of the breeding season. Using our dataset, we previously found that RTL declines linearly with age within an individual but not across individuals, and has individual repeatability of 14%, and that there was no difference in RTL between the sexes (Chik et al., 2023).

2.3 | Statistical analysis

We conducted all analyses in R 4.1.2 (R Core Team, 2021). To allow the use of RTL as a predictor, we first corrected RTL measurements for technical effects, including storage time, technician identity, and qPCR plate effects. We ran a linear mixed model using the package *lme4* 1.1–28 (Bates et al., 2015), with RTL as the response variable, z-transformed such that effect sizes are comparable between studies (Verhulst, 2020). We fitted the following predictors: duration from when the sample was stored as a blood sample until DNA extraction ('BloodAge', in years), duration from when the DNA sample was stored until RTL measurement ('DNAAge', in years), the squared terms for both storage durations to account for non-linear effects, technician identity as a two-level fixed factor (A/B), and plate identity as a random effect. These effects were previously found to affect RTL (e.g. Precioso et al., 2022); in particular, we found RTL to decrease non-linearly with storage time, with the fastest rate of decline seen in the first 5 years of blood storage (Chik et al., 2023; Sibma, 2021). We fitted this model assuming a Gaussian error distribution. The residual RTL values ('corrected z-RTL') were then extracted for the survival models.

We tested for the correlation between post-fledging telomere length and short-term survival in two ways. First, we ran a generalized linear mixed model (GLMM) (n = 1558 from 900 birds) using *lme4*. We fitted annual survival (whether an adult individual survived one more year after sampling as a binomial response, 0 = did not survive/1 = survived) with a logit link function, and corrected z-RTL as a continuous fixed predictor. We tested for a quadratic relationship between survival and z-RTL, but the squared term was not statistically significant and was thus removed from the final model to facilitate interpretation of the first-order effect. As survival changes with age non-linearly (Simons et al., 2019), we also fitted age at sampling (in years) and its squared term as continuous fixed predictors, along with sex. The interaction between sex and corrected z-RTL, found in (Heidinger et al., 2021), was not statistically significant in our dataset, and was thus also excluded in the final model to aid model interpretation. Finally, we added the following random effects: bird ID to correct for non-independence in observations from the same bird; year of capture to account for yearly stochasticity, and rearing brood identity to account for early-life environmental effects on survival. We checked the variance inflation factor (VIF) of fixed predictors, and concluded that there was minimal collinearity as all VIFs were < 3 (Zuur et al., 2010). Second, to confirm the qualitative results from the binomial model, we fitted a time-dependent Cox proportional hazard model (1211 observations from 670 birds). In brief, at the time of each death event, the model compares covariate values of individuals who died, to the individuals who were still alive and therefore at risk of dying, to estimate the risk score associated with the covariate value. To run this model, we coded time-to-event (death) for each individual in days, in a step-wise manner, with the first step being the time elapsed between the hatch date and first RTL measurement, and subsequent steps being the time elapsed between two consecutive RTL measurements, and the last step being the time elapsed between the

last RTL measurement and the date the bird was last seen, on which it was assumed dead. We excluded 322 birds without an exact hatch date. We then ran the Cox model using the package *coxme* 2.2–18.1 (Therneau, 2022), right-censoring 11 birds that were still alive at the time of the analysis, and using the same fixed effect and random effect structures as retained in the binomial GLMM.

We then tested whether adult telomere dynamics were associated with lifespan, using a bivariate model, which allows estimation of the covariance among the two response variables, and fixed effects to be fitted to only one of the two responses where required. We did so in a Bayesian framework, using *MCMCglmm* 2.32 (Hadfield, 2010), following a similar approach by Heidinger et al. (2021). In this model, we only included the 1214 birds that were dead at the time of analysis. We fitted z-RTL, and individual lifespan as response variables, assuming respectively a Gaussian and a quasi-Poisson distribution accounted for over-dispersion. For z-RTL only, we fitted age at sampling in years, centred around the population mean, so that the random individual intercept in z-RTL can be interpreted relative to the population mean. We also fitted BloodAge, DNAAge, their squared terms, and technician identity as fixed variables, to correct for the age and technician effects on RTL. For the random effect structure, we fitted a random slope function of RTL by age at the individual level, along with the year of capture and plate ID as random variables to RTL. We did not fit social parent identities nor brood identity as they explained minimal variation in RTL (Chik et al., 2023). Because our aim is to test for the phenotypic (apparent) covariation between telomere dynamics and lifespan, regardless of common factors contributing to such covariation, we did not fit additional effects to account for potential variation in lifespan. As each individual had one lifespan value corresponding to multiple RTL values, individual variation in lifespan could be effectively coded as part of the residual variation in the bivariate model in MCMCglmm model specification (Arnold et al., 2019; Thomson et al., 2017). We specified inverse-Wishart priors (V = 1, nu = 1) for capture year and plate ID, and a 3 × 3 variance–covariance matrix (V = diag(3), nu = 3, covu = TRUE) for bird ID, which links the random and residual effects structure, and allows the lifespan values in the residuals to covary with RTL and the rate of RTL change (Thomson et al., 2017). Such a bivariate model is preferred as the random-residual effects structure allows the estimation of the among-individual variance and covariance among RTL, rate of RTL change and lifespan simultaneously, without needing a stats-on-stats approach:

$$\begin{bmatrix} \sigma^2_{\text{RTL}} & \sigma_{\text{RTL,RTL:Age}} & \sigma_{\text{RTL,Lifespan}} \\ \sigma_{\text{RTL,RTL:Age}} & \sigma^2_{\text{RTL:Age}} & \sigma_{\text{RTL:Age,Lifespan}} \\ \sigma_{\text{RTL,Lifespan}} & \sigma_{\text{RTL:Age,Lifespan}} & \sigma^2_{\text{Lifespan}} \end{bmatrix}_{\text{ID}}.$$

To examine the correlations between telomere dynamics and reproduction, we built models with the annual and lifetime number of offspring at three stages: hatchlings, defined as chicks that reached 2 days (hereafter AROHatchlings and LROHatchlings for annual and lifetime reproductive output in hatchlings respectively); fledglings, defined as chicks that reached 12 days (hereafter AROFledglings and LROFledglings); and recruits, derived from the genetic pedigree (ARS and LRS, as mentioned above). For ARO and LRO measures, we used the number of social offspring, i.e. chicks that were reared by the focal parent. Because any cross-fostering is conducted after counting hatchlings and without changing brood size, and extra-pair paternity in the house sparrows are generally low (~18% in our population; [Dunning et al., 2024; Hsu et al., 2014]), hatchling counts thus represent the energetic costs in egg production, incubation, as well as short-term fitness of the biological parent, whereas fledgling data represent reproductive costs, including the rearing process post-hatching, for example parental care. In addition, we used ARS and LRS, i.e. the number of genetic recruits, to measure longer-term fitness, as they are the best proxy for the pedigree-derived genetic reproductive value (Alif et al., 2022). Using these variables, we built six bivariate models, detailed below.

For the lifetime reproductive output models (LRS, LROHatchlings, LROFledglings), we used the same bivariate framework as for the lifespan model, and included the 1214 individuals that were dead at the time of analysis to avoid issues of censoring. The three-level variance–covariance matrices obtained from these lifetime models allow us to quantify the among-individual (co)variation between RTL, rate of RTL change and lifetime reproductive measures.

For the annual reproduction models (ARS, AROHatchlings, AROFledglings), we used the whole dataset (2078 observations from 1220 birds; five birds out of 1225 were excluded due to missing sex). We paired annual reproductive traits with the z-RTL measurement taken in the same year for each bird. For the fixed effect structure, we added age and sex to both response variables, and storage time and technician ID to RTL only to correct for technical effects. We also fitted seasonality (breeding/non-breeding) as a two-level factor to RTL, as measurements taken inside versus outside of the breeding season could show differences in their association with annual reproductive traits. For the random effects structure, we fitted unrestricted 2 × 2 variance–covariance matrices for bird ID and capture year to estimate among-individual and among-year (co)variation in RTL and annual reproductive measures. We fitted plate ID as a random effect without estimating covariance, as the variable is only relevant to RTL, but MCMCglmm does not allow random effects to be fitted to one response variable only. However, because of the MCMCglmm approach, this plate ID variance is not expected to affect the estimation of the other random variables. We fitted an unrestricted variance–covariance structure to the residuals to quantify the residual covariation between RTL and annual reproductive traits. We specified inverse-Wishart priors (V = diag(2), nu = 2) for all random and residual effects.

For all (MCMCglmm) bivariate models, we calculated the among-individual correlation from the bird ID variance–covariance matrices using the posterior.cor() function in MCMCglmm. We adjusted the number of iterations, burn-in and thinning intervals (Table S1), such that convergence was reached based on the following criteria: visual inspection of posterior trace plots showed no distinguishable trend, autocorrelation <0.1, and the effective sample size >1000. In the MCMCglmm framework, we considered effects statistically significant if the 95% credible intervals (CI) did not include zero, or, where

effects were zero-bound (e.g. variances), where the 95% CI was not bound to zero.

To check if the inclusion of the among-individual random slope effect altered any conclusions drawn between RTL and lifetime traits, we further built four univariate, generalized linear models, with lifespan, LROHatchlings, LROFledglings, and LRS as responses respectively, assuming a Poisson distribution for each by fitting a log link function. In each of these models, we fitted corrected z-RTL as the sole predictor.

3 | RESULTS

3.1 | Descriptive statistics

In our dataset, the mean RTL was 1.29 (s.d. = 0.64, range = 0.14–6.61). One thousand two hundred and twenty-five individuals were sampled between the age of 0–7, with a mean of 1.7 samples per bird (range = 1–9). Further summaries are in Tables 1 and 2. At the time of analysis, 11 individuals were still alive. Excluding these individuals, the mean lifespan was 1.7 years (median = 1, s.d. = 1.7, range = 0–9, N = 1214; 572 females and 637 males). The mean LRS was 1.5 recruits (median = 0, s.d. = 2.7, range = 0–16), the mean LROHatchlings was 9.7 (median = 3, s.d. = 13.3, range = 0–86), and the mean LROFledglings was 3.7 (median = 0, s.d. = 6.1, range = 0–40). Mean ARS of all birds in the dataset was 0.6 (median = 0, s.d. = 1.1, range = 0–8, N = 1225; 579 mothers and 641 fathers), mean AROHatchlings was 3.7 (median = 0, s.d. = 4.8, range = 0–30), and mean AROFledglings was 1.5 (median = 0, s.d. = 2.2, range = 0–11).

TABLE 1 Summary of the number of repeated RTL measurements and associated number of individuals in the Lundy house sparrow dataset, for blood samples collected in 2000–2015.

Number of samples	Number of individuals
1	749
2	256
3	126
4	53
5	22
6	14
7	3
8	1
9	1
Total number of birds	1225

TABLE 2 Summary of the number of birds and samples across age classes in the Lundy house sparrow telomere dataset (2083 samples from 1225 birds in total), for blood samples collected in 2000–2015.

Age in years	Number of birds	Number of samples
0	703	800
1	535	669
2	248	298
3	144	175
4	64	78
5	35	40
6	15	16
7	5	7
Total number of samples		2083

3.2 | Telomere length and survival

Both the binomial regression model and the Cox time-dependent proportional hazard model indicated that adult RTL was positively correlated with survival. In the binomial model, corrected z-RTL was statistically significantly related to survival to the next year, with a slope of 0.44 (s.e. = 0.14, p = 0.002, Table 3, Figure 1). Age also had a statistically significant quadratic relationship with survival, with early-life and late-life survival being lower than mid-life (Table 3, Figure 2). There was no difference in survival between the sexes (Table 3).

Similarly, the Cox model showed a statistically significant and negative relationship between corrected z-RTL and mortality. Corrected z-RTL had a negative coefficient of −0.17 (s.e. = 0.06, p = 0.008) on survival, meaning that for every unit increase in corrected z-RTL the hazard ratio is multiplied by a factor of 0.84, that is a 16% decrease in mortality (Table 4). Age also showed a quadratic effect, with high mortality early and late in life (Table 4). There was no significant effect of sex (Table 4).

TABLE 3 Summary of the generalized linear mixed model (GLMM) testing for the effects of corrected, z-transformed relative telomere length (corrected z-RTL), age, and sex on survival as a post-fledgling to 1 year after sampling in the Lundy house sparrows.

	Estimate	s.e.	z-Value	p-Value
Fixed effects				
(Intercept)	−1.491	0.563	−2.649	0.008
Corrected z-RTL	**0.436**	**0.140**	**3.126**	**0.002**
Sex	0.127	0.222	0.575	0.566
Age	**1.084**	**0.201**	**5.395**	**<0.001**
Age^2	**−0.285**	**0.052**	**−5.498**	**<0.001**
Random effects				
	Variance		Number of levels	
Bird ID	3.170		900	
Rearing brood ID	1.115		617	
Capture year	3.063		12	

Note: Statistically significant effects (excluding intercept) are highlighted in bold. N = 1558.

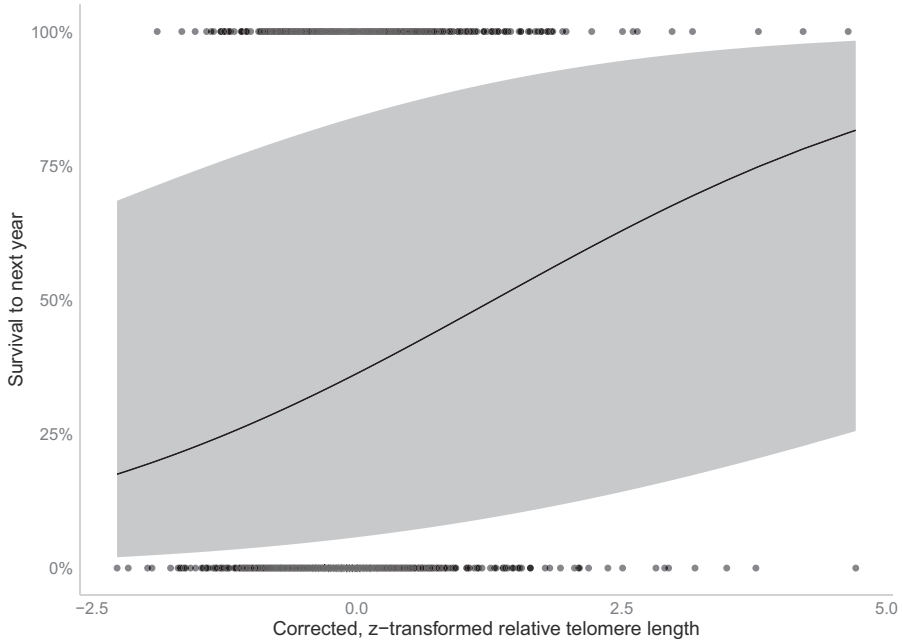

FIGURE 1 The positive relationship between relative telomere length (corrected for technical effects) and survival to 1 year after sampling (0/1) in the Lundy house sparrows, predicted from a binomial model (Table 3), accounting for random effects of bird ID, capture year and rearing brood identity. Solid black line indicates predicted relationship, shaded area indicates 95% confidence interval, and black dots indicate raw data points.

3.3 | Telomere dynamics and lifespan

From the bivariate model, we found significant individual variation in RTL (σ^2=0.12, 95% CrI=0.09–0.15), in the rate of RTL change (σ^2=0.07, 95% CrI=0.05–0.10), and in lifespan (σ^2=0.42, 95% CrI=0.34–0.51; Table 5). While the covariation of RTL and the rate of RTL with lifespan was positive (σ=0.04 and 0.02; r=0.21 and 0.11 respectively), the estimate did not reach statistical significance (Table 5).

The univariate model fitting lifespan and corrected z-RTL returned similar results, where there was a positive but not significant relationship between RTL and lifespan (β=0.026, s.e.=0.019, p=0.169).

3.4 | Telomere dynamics and lifetime reproductive measures

From the bivariate model, there was a statistically significant positive among-individual covariance (σ=0.117, 95% CI=0.035–0.224, Table 6) and correlation (r=0.186, 95% CI=0.058–0.346, Table 6) between RTL and LRS, indicating that individuals with longer mean telomere lengths produced more genetic recruits over their lifetime. There was no among-individual covariation between the rate of RTL change and LRS (Table 6). The univariate model indicated similarly that RTL is positively associated with LRS (β=0.063, s.e.=0.018, p<0.001).

We found statistically significant covariance (σ=0.139, 95% CI=0.010–0.285, Table S2) and correlation (r=0.175, 95% CI=0.112–0.320, Table S2) between LROHatchlings and RTL, but not with the rate of RTL change (Table S2). Its univariate model equivalent found a marginal RTL effect on LROHatchlings (β=0.015, s.e.=0.008, p=0.06). Similarly, we found a significant covariance (σ=0.012, 95% CI=0.001–0.230, Table S3) and correlation (r=0.151, 95% CI=0.111–0.322, Table S2) between the lifetime number of social fledglings (LROFledglings) and RTL, but not with rate of RTL change (Table S3). However, LROFledglings was not associated with RTL in the univariate model (β=0.018, s.e.=0.012, p=0.15). Together, these results indicate that individuals with longer telomeres have higher lifetime fitness and successfully raised more chicks over their lifetime.

3.5 | Telomere length and annual reproductive measures

From the bivariate model with RTL and ARS, we did not find any association between RTL and ARS among individuals (σ=0.000, 95% CI=−0.062 to 0.055; r=0.000, 95% CI=−0.218 to 0.190; Table 7), meaning that individuals with longer telomeres on average did not produce more genetic recruits from year to year. There was also no statistically significant covariance between RTL and ARS within an individual (residual σ=0.038, 95% CI=−0.031 to 0.092; Table 7), meaning annual reproductive output did not change as telomere length varied within a bird.

We did not find any among-individual covariance (σ=0.009, 95% CI=−0.063 to 0.074, Table S4) or correlation (r=0.022, 95% CI=−0.164 to 0.182, Table S4) between RTL and the annual number of hatchlings (AROHatchlings) produced. There was also no among-individual covariance (σ=−0.023, 95% CI=−0.079 to 0.039, Table S5) or correlation (r=−0.095, 95% CI=−0.254 to 0.132, Table S5) between RTL and AROFledglings, suggesting that in our population the link between RTL and immediate productive costs was, if at all, weak.

We summarized the among-individual correlations between RTL and lifetime and annual fitness traits from the bivariate models in Figure 3.

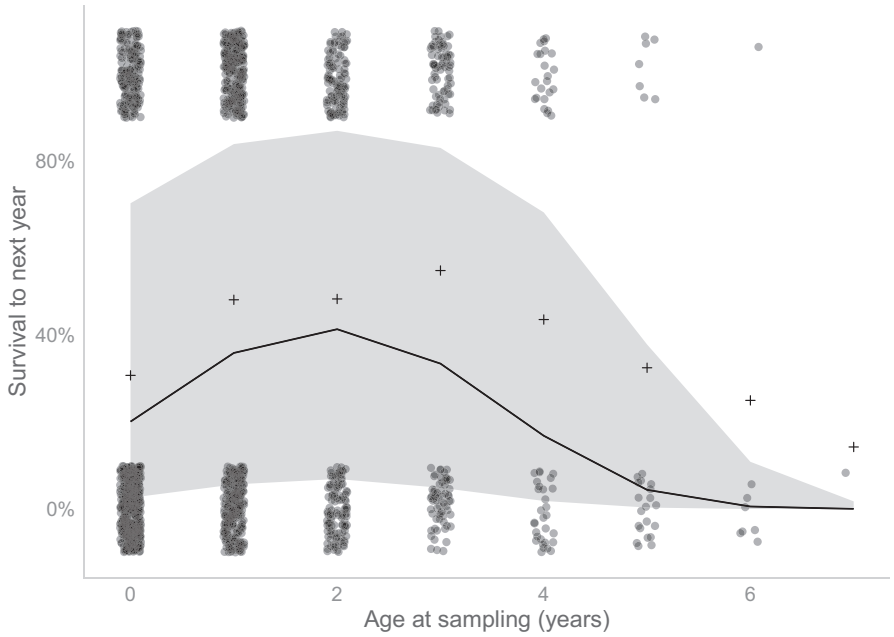

FIGURE 2 The quadratic relationship between age at sampling (in years) and survival to 1 year after sampling (0/1) in the Lundy house sparrows, predicted from a binomial model (Table 3), accounting for random effects of bird ID, capture year and rearing brood identity. Solid black line indicates predicted relationship, shaded area indicates 95% confidence interval, and black dots (jittered) indicate raw data points. Crosses indicate raw age-specific survival probability, i.e. the proportion of birds that survived in each age class.

TABLE 4 Summary of the time-dependent Cox proportional hazards model testing for the relationship between corrected, z-transformed relative telomere length (RTL), age, sex, and mortality risk.

Fixed effects					
	Coefficient	**s.e.**	**Hazard ratio**	**z-Value**	***p*-Value**
Corrected z-RTL	**−0.167**	**0.063**	**0.846**	**−2.66**	**0.008**
Age	**−0.361**	**0.114**	**0.697**	**−3.17**	**0.002**
Age^2	**0.056**	**0.025**	**1.058**	**2.23**	**0.025**
Sex	−0.002	0.087	0.998	−0.03	0.980
Random effects					
	Variance				
Rearing brood ID	0.165				
Capture year	0.319				

Note: Statistically significant effects are highlighted in bold. $N = 1211$.

4 | DISCUSSION

Using longitudinal telomere measurements from the Lundy Island house sparrow population, where ages, death status and reproductive outputs were known, we found that in post-fledging birds, independent of age, longer telomeres were associated with higher chances to survive to the next year, and higher lifetime reproductive outputs but not with annual reproductive outputs.

Our finding that birds with longer telomeres are more likely to survive to the next year is consistent with existing literature on adult telomere length (e.g. Angelier et al., 2013; Barrett et al., 2013) and meta-analytic results (Wilbourn et al., 2018). It also agrees with the speculation of the selective disappearance of older birds with short telomeres in the Lundy sparrows, suggested by a lack of among-individual relationship between RTL and age reported in Chik et al. (2023). The link between telomere length and survival/mortality could be explained by two mechanisms: Telomeres could play a causal and active role, by inducing cell senescence and cell death at a critically short length. The accumulation of senescent cells could hinder tissue functions, lead to organ failure and eventual death (Barrett et al., 2013; Monaghan, 2010; Sahin et al., 2011). Alternatively, telomere length could also not participate directly in causing death, but serve as an indicator of the accumulative damage received by the body, or as a measure of 'frailty', the capacity of the body to withstand and/or recover from damage (Monaghan, 2010). While this study is correlational and cannot prove causality, our finding supports that telomere length could serve as a biomarker of short-term survival.

Nevertheless, the demonstrated association between adult telomere length and short-term survival in our study contradicts

TABLE 5 Summary of the bivariate mixed model, with a random intercept and slope function of relative telomere length (RTL) with age, and a random intercept of lifespan, at the individual level.

Fixed effects				
	Post. Mode	95% CI	Effective sample size	*p*-MCMC
Intercept (Lifespan)	0.324	0.256 to 0.394	13,051	<0.001
Intercept (RTL)	0.373	−0.039 to 0.848	13,521	0.067
Mean-centred age*	−0.070	−0.129 to 0.007	13,067	0.083
BloodAge*	**−0.155**	**−0.232 to −0.087**	**13,500**	**<0.001**
BloodAge^2*	**0.005**	**0.001 to 0.009**	**13,500**	**0.010**
DNAAge*	0.034	−0.056 to 0.110	13,500	0.494
DNAAge^2*	**−0.010**	**−0.018 to −0.004**	**13,500**	**0.002**
Technician (B)*	0.025	−0.213 to 0.222	13,500	0.976
Random effects				
Capture year*	0.142	0.067 to 0.349	13,500	
Plate*	0.101	0.073 to 0.155	13,500	
Bird ID				
Var(RTL)	0.120	0.092 to 0.151	12,510	
Var(RTL:Age)	0.071	0.052 to 0.097	13,500	
Var(Lifespan)	0.416	0.337 to 0.512	13,134	
Cov(RTL, RTL:Age)	−0.007	−0.027 to 0.012	12,714	
Cov(RTL, Lifespan)	0.040	−0.009 to 0.085	12,953	
Cov(RTL:Age, Lifespan)	0.021	−0.016 to 0.059	13,500	
Cor(RTL, RTL:Age)	−0.087	−0.299 to 0.103		
Cor(RTL, Lifespan)	0.207	−0.033 to 0.370		
Cor(RTL:Age, Lifespan)	0.110	−0.087 to 0.326		
Residuals	0.394	0.361 to 0.431	13,143	

Note: This model estimates the variance, covariance and correlation in RTL, rate of RTL change with age, and lifespan among individuals in the Lundy house sparrows. Significant effects are highlighted in bold, excluding fixed intercepts. Shaded rows indicate covariances.

*Effects fitted on RTL only.

others. In another insular house sparrow study in Norway, Pepke, Kvalnes, Ranke, et al., 2022 found no correlation between early-life telomere length and adult survival. This could simply be due to the difference between sampling stage, i.e. early-life versus adult, in that RTL during the nestling stage is less influential on adult survival compared to other (environmental) factors. Alternatively, this difference could also be a result of habitat differences—in the Norwegian population, some sparrows resided on islands with limited food and shelter, leading to higher competition and increased juvenile mortality, ultimately the decoupling of early-life telomere length and adult survival (Pepke, Kvalnes, Ranke, et al. (2022)); whereas in the Lundy population, food and shelter are available to sparrows year round, and mortality was less dependent on resources availability and population density (Simons et al., 2019), thus revealing a stronger effect of telomere length. As telomere dynamics are influenced by environmentally induced oxidative stress (Monaghan & Ozanne, 2018), it is perhaps not surprising that the telomere-mortality link would be context-dependant, necessitating further studies using different ages, populations and taxa (Wilbourn et al., 2018).

Compared with short-term survival, the link between telomere dynamics and lifespan was weaker. This weaker link could be the result of extrinsic factors. Independent of telomere length, age was linked with mortality: the youngest and oldest birds had a higher probability of dying. This could mean that other age-specific factors, such as predation, became the main cause of death in the shortest and longest living birds. This would weaken the link between lifespan and telomere length at the extreme ages, and drive down sample sizes, especially of long-lived birds, or drive down variation in lifespan, such that we could no longer detect an effect of telomere length on lifespan. In the Lundy sparrows, predation pressure predicted mortality in adults than in juveniles (Simons et al., 2019), but we do not know the main cause of death in each age class, nor have we tested for age dependency in TL-mortality association. Further studies should address these topics. Nevertheless, the effect found here agreed with the positive link we found between telomere length and immediate survival (Figure 3).

If telomere length acts as an indicator of somatic redundancy/frailty, then the TL-mortality link would be weaker at older ages, and the rate of telomere shortening could emerge as a better predictor

TABLE 6 Summary of the bivariate mixed model, with a random intercept and slope function of relative telomere length (RTL) with age, and a random intercept of lifetime reproductive success (LRS), at the individual level.

Fixed effects				
	Post. Mode	95% CI	Effective sample size	*p*-MCMC
Intercept (LRS)	−0.890	−0.078 to −0.731	9000	<0.001
Intercept (RTL)	0.438	−0.052 to 0.838	9000	0.082
Mean-centred age*	**−0.061**	**−0.130 to −0.008**	**9000**	**0.026**
BloodAge*	**−0.164**	**−0.226 to −0.083**	**8605**	**<0.001**
BloodAge^2*	**0.005**	**0.001 to 0.009**	**9000**	**0.018**
DNAAge*	0.042	−0.046 to 0.123	9000	0.412
DNAAge^2*	**−0.012**	**−0.018 to −0.005**	**9298**	**0.001**
Technician (B)*	−0.013	−0.216 to 0.211	9229	0.990
Random effects				
Capture year*	0.142	0.070 to 0.355	9000	
Plate*	0.101	0.074 to 0.153	9000	
Bird ID				
Var(RTL)	0.120	0.094 to 0.153	9000	
Var(RTL:Age)	0.070	0.052 to 0.096	9000	
Var(LRS)	3.110	2.666 to 3.744	9000	
Cov(RTL, RTL:Age)	−0.010	−0.026 to 0.012	9000	
Cov(RTL, LRS)	**0.117**	**0.035 to 0.224**	**9000**	
Cov(RTL:Age, LRS)	−0.010	−0.034 to 0.150	8093	
Cor(RTL, RTL:Age)	−0.078	−0.294 to 0.110		
Cor(RTL, LRS)	**0.186**	**0.058 to 0.346**		
Cor(RTL:Age, LRS)	0.120	−0.056 to 0.313		
Residuals	0.393	0.363 to 0.431	9430	

Note: This model estimates the variance, covariance and correlation in RTL, rate of RTL change with age and LRS among individuals in the Lundy house sparrows. Significant effects are highlighted in bold, excluding fixed intercepts. Shaded rows indicate covariances.

*Effects fitted on RTL only.

of lifespan (Boonekamp et al., 2013; Monaghan, 2010). However, we did not find such association here, as covariance between the rate of RTL change and lifespan was not statistically significant, despite finding individual variation in the rate of telomere shortening (Chik et al., 2023). This could be a result of not having enough statistical power: In our dataset, only 270 birds were sampled three times or more, and few individuals lived to old ages (six birds lived to eight years old, three birds to nine). The individual variation in the rate of RTL change was also low, and as a result might not allow the detection of its covariation with lifespan.

In addition to survival, we also found a link between telomere length and reproductive success, such that individuals with longer telomeres on average, produce more genetic recruits and hatchlings, and raised more fledglings over their lifetime, which could indicate that telomere length is under positive selection in our population. In contrast, there was no evidence of any relationship between annual telomere length and reproductive output. Our results suggested that the link between telomere length and fitness is primarily through higher survival (Figure 3), where individuals with longer telomeres survive longer and as a result reproduced more over their lifetime, similar to the finding by Heidinger et al. (2021). One important contributor to such a link is parental age at conception—previously we detected such a Lansing effect in the Lundy sparrows, where birds whose biological parents were older when they hatched, produced fewer recruits annually and over a lifetime, suggesting epigenetic detrimental effects that were carried down generations (Schroeder et al., 2015). However, similar effects on telomere length is less well-studied in house sparrows, and so far results have been mixed: Bennett et al. (2022) found paternal effects on early-life telomere length, while Pepke, Kvalnes, Lundregan, et al. (2022) did not find any parental effects, for example. Further studies are needed to test for a similar Lansing effect in telomere dynamics to better elucidate the intrinsic and extrinsic contributors to variation in individual quality (e.g. Drake & Simons, 2023), and how telomere dynamics is mechanistically linked to quality and reproduction.

We found no relationship between annual telomere length and reproductive output among individuals, regardless of whether the annual reproductive trait better measured reproductive effort (social fledglings' production) or fitness (hatchlings and genetic recruits).

TABLE 7 Summary of the bivariate mixed model estimating the variance, covariance and correlation among relative telomere length (RTL) and annual reproductive success (ARS) among individuals in the Lundy house sparrows. Reference level for sex was female.

Fixed effects				
	Post. Mode	95% CI	Effective sample size	*p*-MCMC
Intercept (ARS)	−1.336	−1.902 to −0.844	17,380	<0.001
Intercept (RTL)	0.410	−0.112 to 0.931	19,882	0.112
Mean-centred age (RTL)	−0.012	−0.050 to 0.022	19,160	0.447
Mean-centred age (ARS)	**0.771**	**0.677 to 0.859**	**9775**	**<0.001**
Sex (RTL)	0.016	−0.061 to 0.082	19,800	0.787
Sex (ARS)	**−0.369**	**−0.560 to −0.141**	**15,527**	**0.001**
Seasonality (Non-breeding)*	−0.005	−0.110 to 0.080	19,800	0.698
BloodAge*	**−0.144**	**−0.231 to −0.072**	**19,800**	**<0.001**
BloodAge^2*	**0.005**	**0.001 to 0.009**	**19,800**	**0.029**
DNAAge*	0.027	−0.060 to 0.121	19,800	0.493
DNAAge^2*	**−0.011**	**−0.018 to −0.004**	**19,800**	**0.002**
Technician (B)*	0.019	−0.202 to 0.255	19,800	0.813
Random effects				
Capture year				
Var(RTL)	0.212	0.101 to 0.519	19,800	
Var(ARS)	0.658	0.339 to 1.708	18,683	
Cov(RTL, ARS)	0.049	−0.251 to 0.364	19,800	
Plate				
Var(RTL)	0.117	0.084 to 0.173	19,149	
Var(ARS)	0.123	0.074 to 0.207	17,655	
Bird ID				
Var(RTL)	0.104	0.076 to 0.134	19,800	
Var(ARS)	0.752	0.558 to 1.208	6496	
Cov(RTL, ARS)	0.000	−0.062 to 0.055	15,422	
Cor(RTL, ARS)	0.000	−0.218 to 0.190		
Residuals				
Var(RTL)	0.435	0.402 to 0.472	19,800	
Var(ARS)	0.232	0.128 to 0.372	7051	
Cov(RTL, ARS)	0.038	−0.031 to 0.092	16,067	

Note: Significant effects are highlighted in bold, excluding fixed intercepts. Shaded rows indicate covariances.

*Effects fitted on RTL only.

Our findings here with annual reproduction did not lend support to the 'pace-of-life hypothesis', under which individuals with a faster pace-of-life are expected to sacrifice somatic maintenance for reproduction, trading higher annual reproductive output for shorter telomeres, which would manifest as a negative relationship. There could be several potential speculations for a lack of relationship between RTL and reproduction at the annual level, but not at the lifetime level. First, there could be little variation in the pace-of-life in the Lundy population, which is in line with another finding by Heidinger et al. (2021). Second, annual reproductive traits could in fact be linked to RTL, but the effects were small, such that this link only manifested at the lifetime level, having accumulated over time. Third, our result could indicate that the physiological costs of reproduction was little reflected in telomere dynamics, regardless of whether telomere was measured inside or outside of the breeding season, as previous research suggested that the trade-off between reproduction and ageing could be not as strong within-species as previously considered, and among-individual trends could be masked by individual quality effects (Winder et al., 2022). Indeed, this possibility could be likely, as the lifetime production of social fledglings, which could represent cumulative post-hatching reproductive costs, was positively associated with telomere length, suggesting that individuals with longer telomere lengths could be of better quality, and were therefore able to rear more chicks to fledging without impacting their own survival.

In contrast to telomere length, we did not find an association between the rate of change of RTL and lifetime reproductive output.

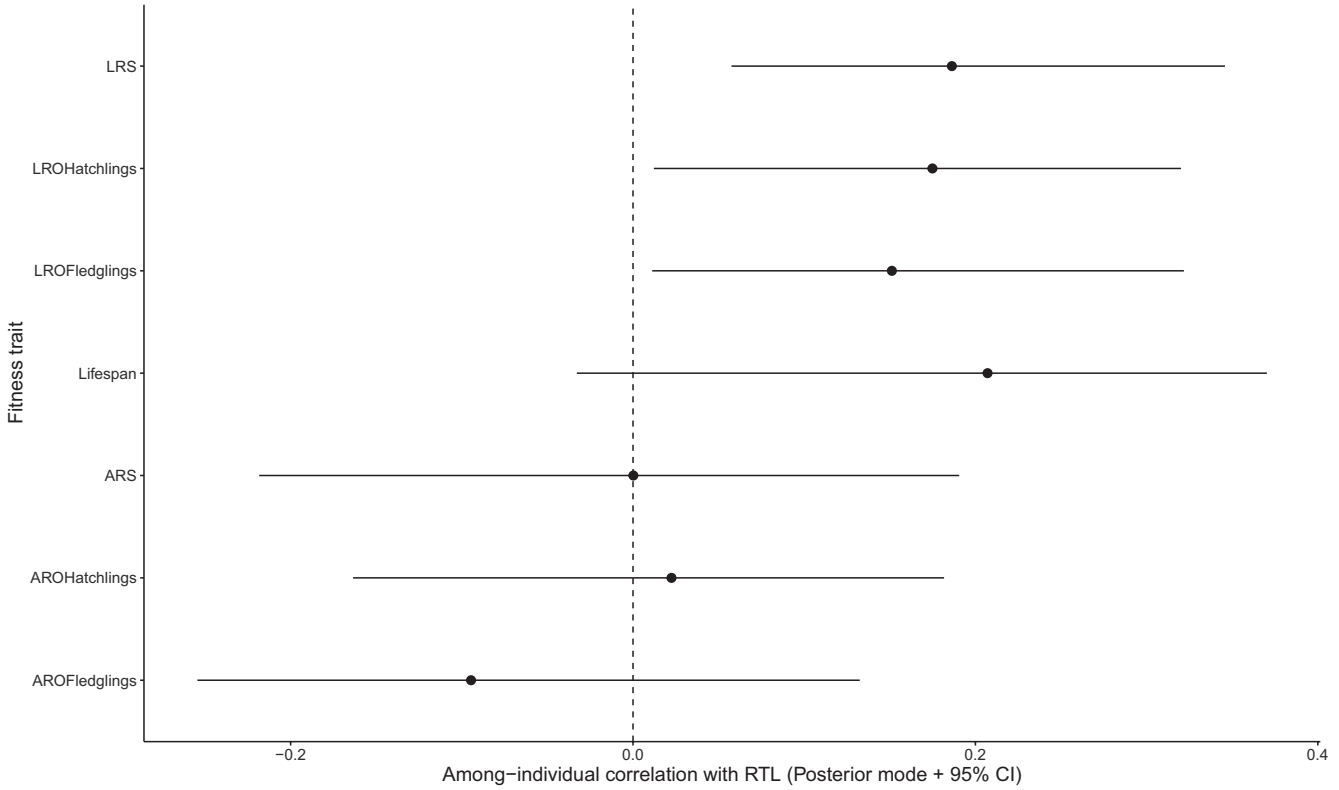

FIGURE 3 Forest plot for the among-individual correlations between fitness traits and relative telomere length (RTL) estimated from Bayesian bivariate models. The dots represent posterior modes, and the bars represent the 95% credible intervals. Dotted line indicates zero correlation. LRS = lifetime reproductive success in terms of the number of genetic recruits; LROHatchlings = lifetime reproductive output in terms of number of social hatchlings; LROFledglings = lifetime reproductive output in terms of number of social fledglings; ARS, AROHatchlings and AROFledglings are their annual equivalents.

We also did not find an association between telomere length and reproductive output within an individual, suggesting that the lack of association could not be attributed solely to differences in individual quality, for example in resource acquisition or stress resistance. Note, however, that the reproductive output measures used here might not fully equate to reproductive effort. For example, contrary to our observational results here, other studies found parents of enlarged broods had shorter telomeres and faster shortening than those with unmanipulated or reduced broods, demonstrating a direct effect of increased reproductive effort on telomere dynamics (Reichert et al., 2014; Sudyka et al., 2014). Further studies should therefore examine the direct effects of for example parental care, to determine whether the telomere dynamics could be used as an indicator of the costs of reproduction in the Lundy sparrows.

In conclusion, this study examined the fitness consequences of telomere dynamics in a longitudinal, closed house sparrow population and found evidence that telomere length was correlated with fitness. Our results provide additional support that telomere length is linked with survival and, possibly through increased survival, with increased lifetime reproductive success. We also add to the debate of the role of telomere shortening as an indicator of senescence, somatic resilience, and fitness. It is important as a next step to determine whether the associations we found are only at the phenotypic level, or occur also at the genetic level, which coupled with heritable variation in telomere dynamics (Chik et al., 2023), would inform how telomere dynamics evolve in the wild.

AUTHOR CONTRIBUTIONS

HYJC, HLD and JS conceptualized the study. NdR and MEM conducted the telomere measurements, and JS and TB curated the telomere and life history datasets. HYJC compiled the datasets used for this study, conducted the statistical analysis and wrote the initial draft of the manuscript, with input from HLD and JS. All authors contributed to the revision of the manuscript and agreed on the final version of the manuscript to be published.

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CONFLICT OF INTEREST STATEMENT

The authors declare no conflicts of interest.

DATA AVAILABILITY STATEMENT

The telomere and life history datasets used in this study, along with the R script used for analysis, are publicly available at http://doi.org/10.6084/m9.figshare.25586205.

ETHICS APPROVAL

All animal procedures are licensed and approved by the British Trust for Ornithology and the UK Home Office (Project Licence PP5873078).

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