Original article

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

Running title: *Telomere length is correlated with fitness*

Heung Ying Janet Chik[1,2], Maria-Elena Mannarelli[3,4], Natalie dos Remedios[3,5], Mirre J. P. Simons[3], Terry Burke[3], Julia Schroeder[6]*, Hannah L. Dugdale[1]*

*H. L. Dugdale and J. Schroeder share last authorship

[1]Groningen Institute for Evolutionary Life Sciences, University of Groningen, Groningen 9747AG, the Netherlands.

[2]School of Natural Sciences, Macquarie University, NSW 2109, Australia.

[3]Ecology and Evolutionary Biology, School of Biosciences, University of Sheffield, Sheffield S10 2TN, United Kingdom.

[4]School of Biological Sciences, University of East Anglia, Norfolk NR4 7TJ, United Kingdom.

[5]School of Social Sciences, University of Auckland, Auckland 1010, New Zealand.

[6]Department of Life Sciences, Imperial College London Silwood Park, Ascot SL5 7PY, United Kingdom.

Corresponding authors: Heung Ying Janet Chik (chikhyjanet@gmail.com), Hannah L. Dugdale (h.l.dugdale@rug.nl)

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Abstract

Explaining variation in individual fitness is a key goal in evolutionary biology. Recently, telomeres, repeating DNA sequences capping the ends of chromosomes, have gained attention as a biomarker for body state, individual quality, and ageing. However, existing research has provided mixed evidence for whether telomere length correlates with fitness components, including survival and reproductive output. Moreover, few studies have examined how telomere shortening correlates with fitness in wild populations. Here, we intensively monitored an insular population of house sparrows on Lundy Island, UK, and collected longitudinal telomere and life history data spanning 16 years from 1,225 individuals. We tested whether telomere length and/or shortening predict fitness measures, namely survival, lifespan, as well as annual and lifetime reproductive success. Telomere length positively predicted immediate survival up to one year after measurement, 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 effect of telomere length on survival translated to reproductive benefits, as birds with longer telomeres produced more genetic recruits over their lifetime, but not annually, suggesting variation in individual quality. The rate of telomere shortening, however, correlated with neither lifespan nor lifetime reproductive success. Our results provided further evidence that telomere length correlates with fitness, and they contributed to our understanding of how telomere dynamics link with individual quality.

Keywords:

Telomere dynamics, survival, reproductive success, senescence, individual quality

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 equally important to examine traits at deeper levels of biological organization, including cell and body physiology, as they underlie organismal performance. One of such traits 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 chromosome consisting of repeating DNA sequences ($TTAGGG_n$ in vertebrates; Blackburn, 1991). Telomeres are vulnerable to erosion due to 1) the end-replication problem, where linear DNA is not fully replicated during cell proliferation (Levy et al., 1992; Olovnikov, 1973); and 2) chemical damage from oxidative stress (Blackburn et al., 2015; von Zglinicki, 2002). They therefore shorten over time. Shortened telomeres can be restored, e.g. 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 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). Because of this, telomere length, and the rate of telomere shortening, have gained attention in evolutionary biology and epidemiology, as a biomarker of body state or 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 ageing (e.g. López-Otín et al., 2013).

Because telomeres link to cellular senescence, thereby tissue function, and thus perhaps ultimately ageing, one would expect telomere dynamics to be under selection, and therefore to be correlated with fitness. However, studies examining the relationship between telomere dynamics and survival and/or lifespan have provided mixed results. On average, shorter telomeres are associated with higher mortality, but variation exists (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 some evidence that telomere shortening predicts survival and/or lifespan (e.g. Boonekamp et al., 2014; Brown et al., 2022; Tricola et al., 2018; Whittemore et al., 2019; Wood & Young, 2019). To date, it remains unclear whether, and how, telomere biology causally contribute to organismal senescence (Simons, 2015; Young, 2018) and fitness variation. This is particularly true for adult telomere dynamics, as most studies focused on early-life telomere lengths.

The link between telomere dynamics and reproductive success, 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 habitat that offers more resources and 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 would exhibit a longer lifespan, have longer telomeres and slower shortening, but decreased annual reproductive success, 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 focused on early-life telomere length and its association with reproductive output, and has provided mixed results: Support for the 'individual quality hypothesis' was found by e.g. Angelier et al., (2019); Eastwood et al., (2019) and Heidinger et al., (2021), whereas support for the 'pace-of-life hypothesis' was found by e.g. Bauch et al., (2013) and Pepke et al., (2022). Additionally, it is still unclear how telomere shortening relates to reproductive output. For example, Heidinger et al. (2021) did not find an association between telomere shortening and reproductive success, while Sudyka et al. (2019) found a negative association. Further testing for 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 precise survival and lifetime reproductive data. As there has been a relative lack of focus on telomere dynamics beyond early life, we selected samples and quantified telomere lengths from birds after they have fledged, and tested: 1) whether adult telomere length predicts immediate survival up to 1 year post-measurement; 2) whether average individual telomere length and rate of telomere shortening across adulthood are associated with lifespan; 3) whether adult telomere length is associated with annual reproductive output; and 4) whether average telomere length and telomere shortening are associated with lifetime reproductive output.

Materials and Methods

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, United Kingdom, starting in the year 2000 though genetic data from as early as 1990 were also available, and used for pedigree construction (see below). Annually, 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 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 one year, and the exact hatch dates for birds hatched in nest boxes. Due to the geographical isolation of Lundy Island, immigration and emigration is almost absent (four suspected immigrants in 2000 - 2011, and three confirmed emigrants up to 2015; Schroeder et al., 2015). We collected survival data through biannual surveys where we recorded the presence/absence of each bird, with annual re-sighting probability between 91-96% (Schroeder et al., 2015). Except for those with an explicit death record, birds that were not sighted for two 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 lifespan for each individual.

We repeatedly collected blood samples from individuals, typically at two 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 subsequently stored at -20°C until analysis. We then

assigned genetic parentage using up to 23 house sparrow microsatellite markers from Dawson et al. (2012) for sparrows hatched in 1990 – 2019. 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 10,731 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, i.e. offspring that reproduced and appeared in the pedigree as dams or sires themselves. We also calculated lifetime reproductive success (LRS) as the sum of ARS across the lifespan of each individual.

Telomere extraction and assay

We measured relative telomere length (RTL) from blood samples of sparrows after they fledged, collected from 2000 to 2015. DNA sample concentration was first measured using a Nanodrop 8000 Spectrophotometer (Thermo Fisher) and normalized to 20–30 ng/µl. Next we used a monochrome multiplex quantitative polymerase chain reaction (MMqPCR) method to quantify RTL (Cawthon, 2009) as described in Chik et al. (2023). In brief, MMqPCR quantifies RTL as a ratio of telomeric signals to that of a single-copy reference gene (GAPDH 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 each 'slice' contained samples obtained from the same year, and each plate contained samples from three consecutive slices, to avoid confounding plate and sample year effects. We measured samples in duplicates in adjacent wells. Plates were run using two machines, a QuantStudio 12K 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). After MMqPCR, we removed samples with Ct values > 25, and a between-duplicate relative difference > 0.2. We averaged the RTL of the remaining duplicates as the final measure for each sample. Mean qPCR amplification efficiencies for telomeres and the reference gene were 90.7% (range: 71 – 109%) and 94.5% (range 76 – 119%) respectively (Sibma, 2021). The inter-plate repeatability was 0.49 (s.e. = 0.07), while the intra-plate repeatability was 0.98 (Sibma, 2021). The final telomere dataset consisted of 2,083 telomere length measurements from 1,225 birds, 476 of which have telomere length measurements at multiple ages.

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 (Chik et al., 2023; Sibma, 2021). 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 variable. 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) using *lme4*. We fitted annual survival (whether an individual survived one more year after sampling as an adult as a binomial response, 0/1) with a logit link function, and corrected z-RTL as a continuous fixed predictor. As survival changes with age non-linearly, we also fitted age at sampling (in years) and its squared term as continuous fixed predictors, along with sex. Finally, we added bird ID and year of capture as random variables to correct for non-independence in observations from the same bird, and from yearly stochasticity. We checked the variance inflation factor (VIF) of fixed predictors, and concluded that there was minimal collinearity as all VIFs were < 5 (Montgomery et al., 2013). Second, we fitted a time-dependent Cox proportional hazard model (n = 1,211). In brief, at the time of each death event, the model compares covariate values of individuals who died, to all other 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 birds without an exact hatch date. We then ran the Cox model using the package *coxme* 2.2-18.1 (Therneau, 2022), right-censoring birds that were still alive at the time of the analysis, and using the same fixed effect and random effect structures as the binomial GLMM.

We then tested whether adult telomere dynamics are associated with lifespan, using a bivariate model, which allows estimation of the covariance among the two response variables, and allows fixed effects to be fitted to only one of the two responses. We did so in a Bayesian framework, fitted with *MCMCglmm* 2.32 (Hadfield, 2010), following a similar approach by Heidinger et al. (2021). In this model, we only included the 1,214 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 Poisson distribution. 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 as they were found to explain minimal variation in RTL (Chik et al., 2023). Because each individual had one lifespan value, a random effect of individual on lifespan could be estimated as a part of the residual variation in the model. The random-residual effects structure therefore allows the estimation of the among-individual variance and covariance among RTL, rate of RTL change, and lifespan:

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

To examine the correlations between telomere dynamics and reproductive success, we fitted two bivariate mixed models, with LRS and ARS respectively. The LRS model had the same framework as the lifespan model, and included only the 1,214 individuals that were dead at the time of analysis. For the ARS model, we used the whole dataset. We paired ARS with the z-RTL measurement taken in the same year for each bird. We retained the same fixed effects structure on RTL, and modelled variance and covariance between z-RTL and ARS explained by bird ID and capture year. We also estimated the residual covariance between z-RTL and ARS in this model to examine the within-individual covariation between the two variables.

For all three bivariate models, we used default priors for fixed effects, inverse-Wishart priors for random effects, and adjusted the number of iterations, burn-in, and thinning intervals for each model, such that convergence is 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.

Results

Descriptive statistics

In our dataset, the mean RTL was 1.29 (s.d. = 0.64, range = 0.14 – 6.61). 1,225 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 (s.d. = 1.7, range = 0 – 9, N = 1,214; 572 females and 637 males), and the mean LRS was 1.5 (s.d. = 2.7, range = 0 – 16). ARS of all birds in the dataset had a mean of 0.6 (s.d. = 1.1, range = 0 – 8, N = 1,225; 579 mothers and 641 fathers).

Telomere length and survival

Both the binomial regression model and the Cox time-dependent proportional hazard model indicated that adult RTL is 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 0.39 (Table 3, Fig. 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, Fig. 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 -0.15 on survival, meaning that for every unit increase in corrected z-RTL the hazard ratio is multiplied by a factor of 0.86, i.e. a 14% decrease in mortality (Table 4). Age showed also a quadratic, U-shaped effect on mortality (Table 4). There was no significant effect of sex (Table 4).

Telomere dynamics and lifespan

From the bivariate model, we found individual variation in RTL, the rate of RTL change, and lifespan. We also found tendencies for RTL and the rate of RTL change to positively covary with lifespan (σ = 0.04 and 0.02 respectively), but the estimates were small and did not reach statistical significance, as their 95% credible intervals overlapped zero (Table 5).

Telomere dynamics and reproductive success

From the bivariate model with RTL and LRS, we found a statistically significant and positive among-individual covariance between RTL and LRS, indicating that individuals with longer mean telomere lengths produce more genetic recruits over their lifetime (σ = 0.12, 95% CI = 0.04 – 0.22, Table 6). There was no among-individual covariation between the rate of RTL change and LRS (Table 6). In contrast, from the bivariate model with RTL and ARS, we did not find any association between RTL and ARS among individuals (σ = 0.002, 95% CI = -0.065 – 0.054, Table 7), meaning that individuals with longer telomeres on average did not produce more recruits from year to year. There was also no statistically significant covariance between RTL and ARS within an individual (residual σ = 0.04, 95% CI = -0.032 – 0.091, Table 7), meaning annual reproductive output did not change as telomere shortens within a bird.

Discussion

Using longitudinal telomere measurements from the Lundy Island house sparrow population, where precise ages, death status and reproductive success are known, we estimated the relationships between telomere dynamics and fitness measures, including survival and reproductive success. We found that in post-fledging birds, independent of age, longer telomeres were associated with higher chance to survive to the next year. This finding was 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 (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). Regardless of causality, our finding supports that telomere length could serve as a biomarker of immediate survival.

Nevertheless, the demonstrated association between adult telomere length and survival in our study contradicts others. In another insular house sparrow study in Norway, authors found no

correlation between early-life telomere length and adult survival (Pepke et al., 2022). This could 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 et al., 2022); whereas in the Lundy population, food and shelter is 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 survival, the link between telomere dynamics and lifespan was much weaker, though still in the expected direction. This weaker link could be the result of the more removed nature of lifespan as an indicator of survival, or 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, such that we could no longer detect an effect of telomere length on lifespan. In the Lundy sparrows, predation pressure was stronger 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.

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 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 of 9 and above.

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 over

their lifetime, which in our population, predicts expected genetic contribution and fitness (Alif et al., 2022). In contrast, there was no evidence of any relationship between annual telomere length and reproductive output. Our results indicated that the link between telomere length and fitness is primarily through higher survival, where individuals with longer telomeres survive longer and as a result reproduced more, similar to the finding by Heidinger et al. (2021), and consistent with the 'individual quality hypothesis', i.e. individuals with a higher quality will have better body conditions, and hence survival and/or reproductive prospects, than poorer quality individuals, a trend found also in classical brood size manipulation studies testing for survival-reproduction trade-offs (Winder et al., 2022). One important contributor to variation in individual quality is parental age at conception – previously we detected such 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). Further studies should 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.

In contrast, there was no relationship between annual telomere length and reproductive output among individuals. This did not align with the 'pace-of-life hypothesis', under which individuals with a faster pace-of-life are expected to sacrifice somatic maintenance for reproduction, trading higher output for shorter telomeres. This could mean that there is little variation in the pace-of-life in the Lundy population, which is in line with another finding by Heidinger et al. (2021). Alternatively, our result could also indicate that the physiological costs of reproduction was not reflected on telomere dynamics, or that the trade-off between reproduction and ageing is not as strong within-species as previously considered, and masked by quality effects (Winder et al., 2022). Indeed, we did not find an association between the rate of telomere shortening and lifetime reproductive output, nor a trade-off 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, e.g. in resource acquisition or stress resistance. Note, however, that reproductive success need not equate to reproductive effort. For example, previous experiments have shown parents of enlarged broods had shorter telomeres and faster shortening than those with unmanipulated or reduced broods (Reichert et al., 2014; Sudyka et al., 2014). Further studies should therefore examine the effect of e.g.

parental care on telomere dynamics, to determine whether the latter is an indicator of the costs of reproduction in the Lundy sparrows.

In conclusion, in this study we examined the fitness consequences of telomere dynamics in a longitudinal, closed house sparrow population, and found evidence that indeed telomere length was correlated with fitness. Our results provide additional support that telomere length is linked with survival and therefore in turn with lifetime reproductive success, but 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.

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Data Accessibility and Benefit-Sharing

Data Accessibility Statement

The telomere and life history datasets used in this study, along with the R script used for analysis, will become publicly available on a data repository e.g. Figshare, upon acceptance for publication.

Authors contribution

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.

Tables and Figures

626 Table 1. Summary of the number of repeated RTL measurements and associated number of
627 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

628

629

Table 2. Summary of the number of birds and samples across age classes in the Lundy house sparrow telomere dataset, 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

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 one year after sampling in the Lundy house sparrows. Statistically significant effects (excluding intercept) are highlighted in bold. N = 2,078.

	Estimate	*Std. err.*	*z-value*	*p-value*
Fixed effects				
(Intercept)	-1.220	0.496	-2.458	0.013
Corrected z-RTL	**0.393**	**0.118**	**3.319**	**<0.001**
Sex	-0.024	0.200	-0.118	0.906
Age	**0.909**	**0.164**	**5.558**	**<0.001**
Age^2	**-0.226**	**0.037**	**-6.053**	**<0.001**
Random effects				
		Variance	*Number of levels*	
Bird ID		4.737	1220	
Capture year		2.975	15	

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, genetic maternal age at conception (MAC), genetic paternal age at conception (PAC) and mortality risk. Statistically significant effects are highlighted in bold. N = 1,211.

Fixed effects					
	Coefficient	*s.e.*	*Hazard ratio*	*z-value*	*p-value*
Corrected z-RTL	**-0.154**	**0.056**	**0.858**	**-2.67**	**0.008**
Age	**-0.284**	**0.106**	**0.752**	**-2.68**	**0.007**
Age^2	**0.051**	**0.024**	**1.052**	**2.17**	**0.030**
Sex	-0.001	0.078	0.999	-0.01	0.990
Random effects					
	Variance			*Number of levels*	
Capture year	0.269			15	

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. This model estimates the variance and covariance 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.

Fixed effects				
	Post. mode	*95% CI*	*Effective sample size*	*p-MCMC*
Intercept (Lifespan)	0.324	0.256 – 0.394	13051	<0.001
Intercept (RTL)	0.373	-0.039 – 0.848	13521	0.067
Mean-centred age*	-0.070	-0.129 – 0.007	13067	0.083
BloodAge*	**-0.155**	**-0.232 – -0.087**	**13500**	**<0.001**
BloodAge^2*	**0.005**	**0.001 – 0.009**	**13500**	**0.010**
DNAAge*	0.034	-0.056 – 0.110	13500	0.494
DNAAge^2*	**-0.010**	**-0.018 – -0.004**	**13500**	**0.002**
Technician (B)*	0.025	-0.213 – 0.222	13500	0.976
Random effects				
Capture year*	0.142	0.067 – 0.349	13500	
Plate*	0.101	0.073 – 0.155	13500	
Bird ID				
Var(RTL)	0.120	0.092 – 0.151	12510	
Var(RTL:Age)	0.071	0.052 – 0.097	13500	
Var(Lifespan)	0.416	0.337 – 0.512	13134	
Cov(RTL, RTL:Age)	-0.007	-0.027 – 0.012	12714	
Cov(RTL, Lifespan)	0.040	-0.016 – 0.085	12953	
Cov(RTL:Age, Lifespan)	0.021	-0.016 – 0.059	13500	
Residuals	0.394	0.361 – 0.431	13143	

*Effects fitted on RTL only

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. This model estimates the variance and covariance 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.

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

*Effects fitted on RTL only

Table 7. Summary of the bivariate mixed model estimating the variance and covariance among relative telomere length (RTL) and annual reproductive success (ARS) among individuals in the Lundy house sparrows. Significant effects are highlighted in bold, excluding fixed intercepts.

Fixed effects				
	Post. mode	*95% CI*	*Effective sample size*	*p-MCMC*
Intercept (ARS)	-1.518	-2.070 – -1.033	15525	<0.001
Intercept (RTL)	0.434	-0.093 – 0.946	19800	0.114
RTL: Mean-centred age	-0.010	-0.049 – 0.022	19800	0.482
ARS: Mean-centred age	**0.746**	**0.665 – 0.846**	**10768**	**<0.001**
BloodAge*	**-0.146**	**-0.229 – -0.074**	**19800**	**<0.001**
BloodAge^2*	**0.004**	**0.001 – 0.009**	**19800**	**0.015**
DNAAge*	0.042	-0.058 – 0.119	19800	0.472
DNAAge^2*	**-0.011**	**-0.018 – -0.004**	**19800**	**0.003**
Technician (B)*	0.041	-0.207 – 0.248	19800	0.802
Random effects				
Capture year				
Var(RTL)	0.214	0.074 – 0.403	19800	
Var(ARS)	0.678	0.324 – 1.718	18742	
Cov(RTL, ARS)	0.008	-0.267 – 0.359	20766	
Plate				
Var(RTL)	0.116	0.084 – 0.174	19800	
Var(ARS)	0.132	0.075 – 0.207	16787	
Bird ID				
Var(RTL)	0.104	0.076 – 0.134	19800	
Var(ARS)	0.798	0.572 – 1.051	6655	
Cov(RTL, ARS)	0.002	-0.065 – 0.054	15619	
Residuals				
Var(RTL)	0.431	0.402 – 0.472	19800	
Var(ARS)	0.222	0.125 – 0.369	7386	
Cov(RTL, ARS)	0.039	-0.032 – 0.091	16503	

*Effects fitted on RTL only

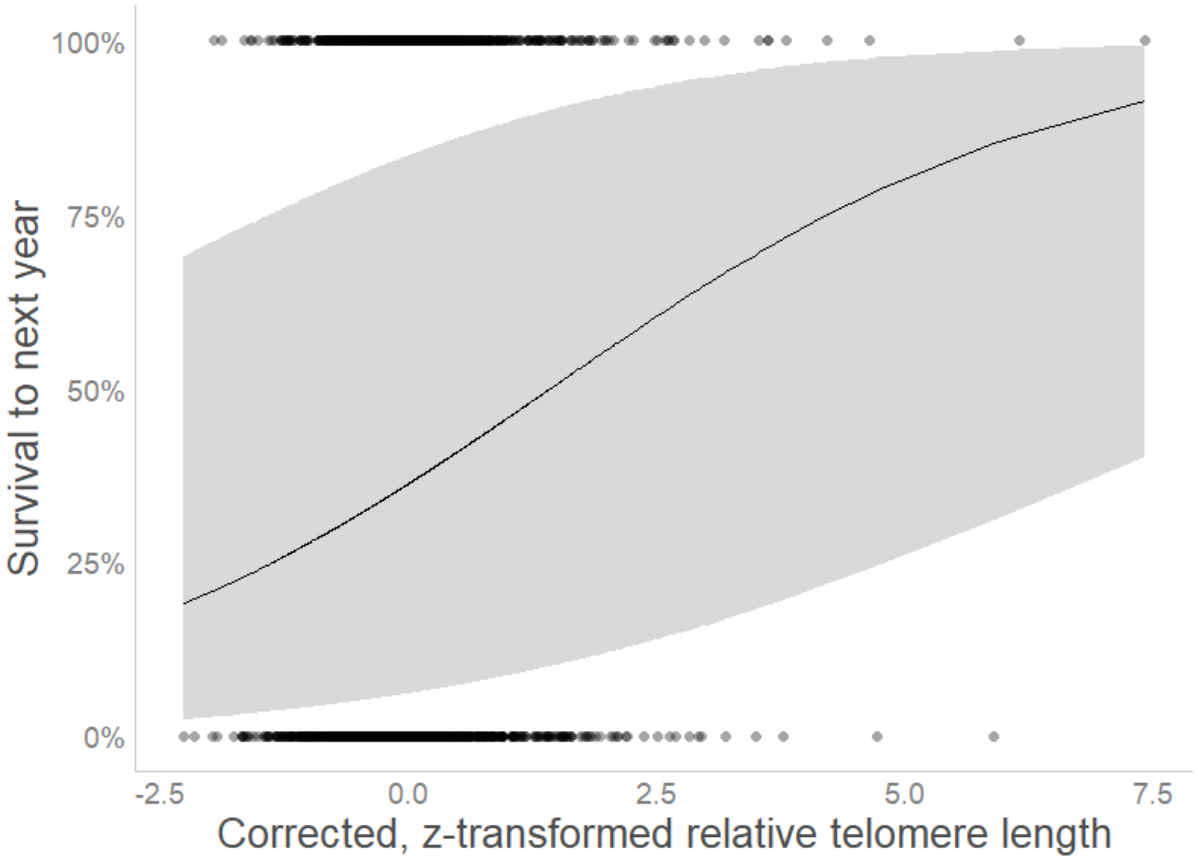

Fig. 1. The positive relationship between relative telomere length (corrected for technical effects) and survival to one year after sampling (0/1) in the Lundy house sparrows. Solid black line indicates predicted relationship, shaded area indicates 95% confidence interval, and black dots indicates raw data points.

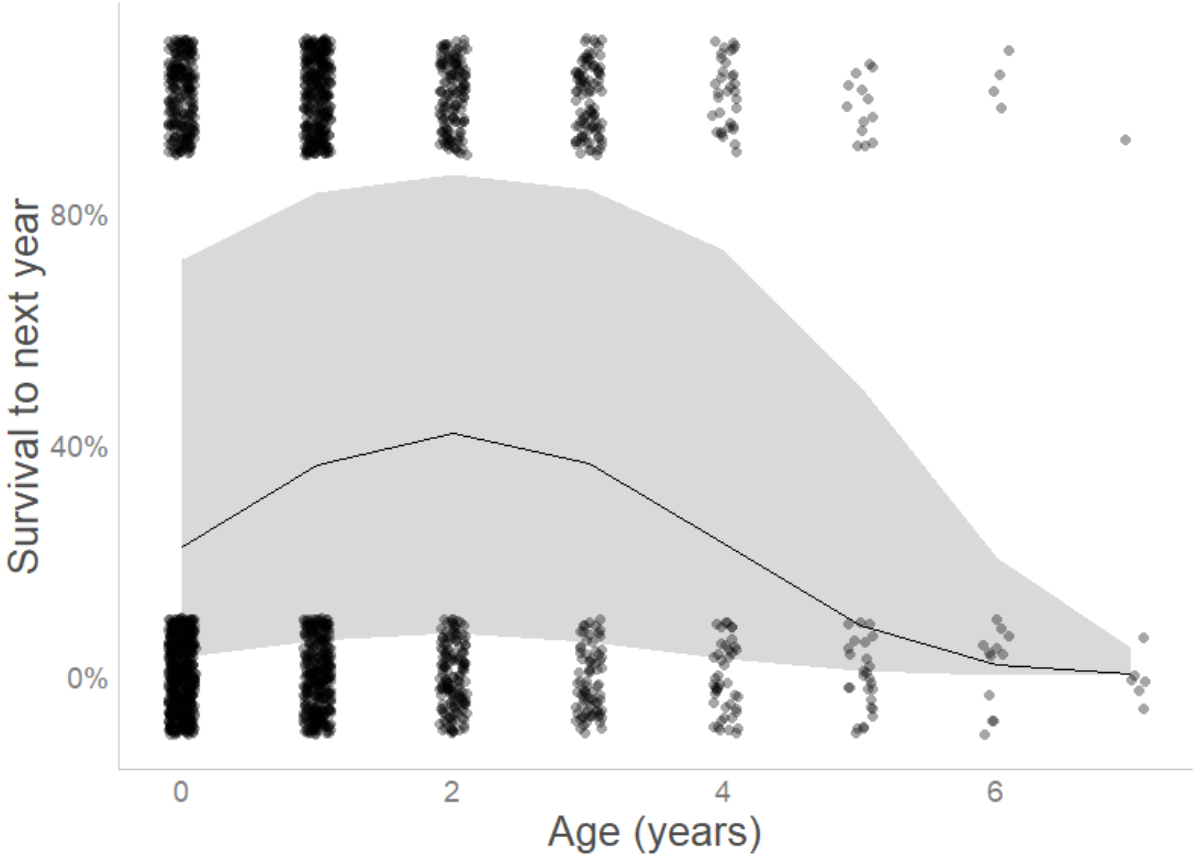

Fig. 2. The quadratic relationship between age at sampling (in years) and survival to one year after sampling (0/1) in the Lundy house sparrows. Solid black line indicates predicted relationship, shaded area indicates 95% confidence interval, and black dots (jittered) indicate raw data points.