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# Telomere dynamics, not absolute telomere length, predicts lifespan in adult zebra finches

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## Abstract

**Telomeres shorten with age, and telomere length (TL) can predict lifespan. *In vitro* studies have established that the shortest telomeres in the genome drive cellular senescence, but whether they also drive *in vivo* lifespan variation is undecided. Moreover, it is not well known to what extent the TL-lifespan association can be attributed to variation in TL per se vs. variation in telomere dynamics prior to sampling. We investigated whether absolute TL, telomere dynamics, or both, serve as predictors of lifespan using longitudinal blood samples from adult captive zebra finches of both sexes that were raised in either small or large broods. We measured TL through telomere restriction fragment analysis, which provides information on the TL distribution within samples in addition to estimates of mean sample TL. Birds with shorter lifespans displayed accelerated telomere shortening and the association between telomere shortening and lifespan was steeper at higher percentiles. Absolute mean TL at any point did not predict lifespan or remaining lifespan and neither brood size nor sex were found to affect TL or telomere dynamics. Collectively, our findings support telomere dynamics—rather than average TL—better predict lifespan, likely more accurately reflecting cumulative physiological stress than TL. This relationship was more pronounced at the longer telomeres in the genome.**

**Keywords** telomere length, telomere dynamics, lifespan, zebra finch

## Introduction

Telomeres are noncoding nucleotide repeats at the ends of eukaryotic chromosomes that contain the repeating 5′-TTAGGG-3′ sequence (Blackburn, 1991) and contribute to maintaining genomic stability and integrity (de Lange, 2004). Telomere sequences are evolutionarily well conserved among vertebrates going back more than 400 million years (Meyne et al., 1989), but average telomere length (TL) at birth is species-specific (e.g., humans, *Homo sapiens* ~6 kbp, (Lai et al., 2023); mice, *Mus musculus* ~50 kbp, (Vera et al., 2012). At the same time, TL typically varies strongly between individuals and a large part of this variation is heritable when measured with high precision (Bauch et al., 2022; Chik et al., 2022). Telomeres shorten with every cell division as telomerase, the enzyme responsible for maintaining TL, stops being active early in life (Blackburn, 1991; Harley et al., 1992), and telomere shortening is known to be accelerated by oxidative stress (Armstrong & Boonekamp, 2023; Tchirkov & Lansdorp, 2003). Critically short telomeres activate a DNA damage response pathway, disrupting telomere protection, causing the cell to lose its ability to divide and triggering cellular senescence and/or cell death (Bendix et al., 2010; Hemann et al., 2001; Zou et al., 2004).

The accumulation of senescent cells is thought to contribute to the aging process by impairing tissue function and promoting age-related decline (Campisi, 2005; van Deursen, 2014), and telomere attrition is one of the hallmarks of aging (López-Otín et al., 2013, 2023). Indeed, TL correlates with fitness proxies including reproductive success (Heidinger et al., 2021) and offspring quality (Young et al., 2021), and longer telomeres have been found to predict survival (Boonekamp et al., 2013; Eastwood et al., 2023; Wilbourn et al., 2018), lifespan (Eastwood et al., 2019; Heidinger et al., 2012; Vedder et al., 2022), and lifetime reproductive success (Chik et al., 2024; Eastwood et al., 2019). Furthermore, early-life stress has previously been shown to accelerate telomere shortening in multiple bird species (Badás et al., 2023; Herborn et al., 2014; Nettle et al., 2013). Parental age has been found to modulate TL across vertebrates, suggesting that telomere dynamics may also mediate intergenerational effects on longevity (Bauch et al., 2019; Eisenberg & Kuzawa, 2018; Marasco et al., 2019; Vlasova et al., 2025). These findings together highlight the potential for telomere biology to play a role in individual aging trajectories and shape lifespan across generations.

Telomere length at any point after conception is the outcome of TL at conception and subsequent telomere dynamics, the change

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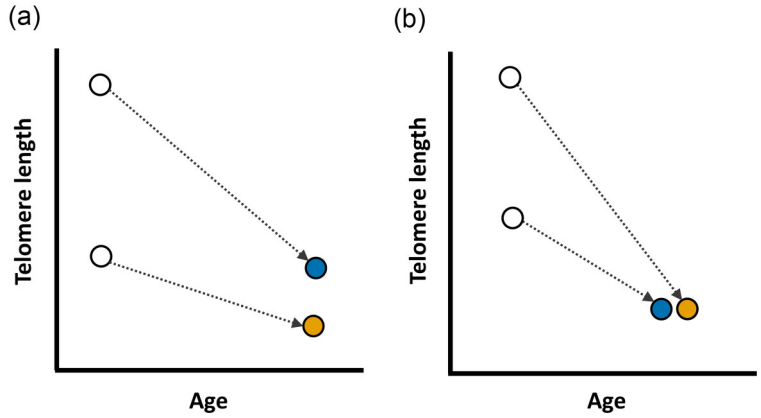


**Figure 1.** Conceptual diagram illustrating different outcomes depending on whether mortality is best predicted by (A) telomere length or (B) telomere dynamics. Circles connected by arrows each represent individuals, with the dashed arrows indicating the change in TL with age prior to sampling. The orange marker indicates the individual with the lower survival probability. In panel (A), individuals have different telomere lengths at sampling and regardless of telomere dynamics prior to sampling the individual with the shorter telomeres has a lower survival probability. In panel (B), individuals have the same telomere length at the later sampling point, but differ in telomere dynamics prior to that point, and the individual that lost more base pairs has a lower survival probability. Note that a and b represent extremes of a continuum, as length and shortening can also both contribute independently to associations between telomere length and lifespan.

in telomere length within individuals over time. Consequently, associations between TL and survival or lifespan can be due to effects of TL at the time of sampling, the telomere dynamics prior to sampling, or both. However, whether absolute telomere length or telomere dynamics is a more accurate predictor of lifespan variation is not yet resolved. This question is of particular interest, because its answer will shed light on the more general question of whether telomeres are *causally* involved in the association between telomere length and lifespan (Boonekamp et al., 2014; Simons, 2015). Specifically, finding that TL dynamics predict survival and/or lifespan, while TL does not, would argue against causal involvement of TL in lifespan variation, indicating it serves as a biomarker of factors that are causally related to survival instead. One such factor could be oxidative stress, which is generally considered to accelerate TL dynamics (Armstrong & Boonekamp, 2023; von Zglinicki, 2002). Finding that absolute TL best predicts survival independent of telomere shortening would be in line with a direct effect of TL on survival, while finding that TL and TL dynamics independently predict survival does not imply a direct effect of TL on lifespan since this association may be due to telomere dynamics prior to the time point the first sample was taken.

Studies using longitudinal telomere measurements are thus instrumental in identifying the contributions of telomere length and telomere dynamics to lifespan variation (Fig. 1). Panel 1a illustrates a scenario where absolute TL better predicts lifespan: regardless of telomere dynamics prior to sampling, the individual with the shorter telomeres has a lower survival probability. Panel 1b illustrates the alternative scenario where telomere dynamics best predict lifespan: despite identical absolute TL at the second sampling point, the individual with higher telomere shortening rate faces higher mortality risk. Studies that have attempted to tease those two scenarios apart have so far have yielded mixed results (Barrett et al., 2013; Chik et al., 2024; Sheldon et al., 2022; Wood & Young, 2019). This is likely to be at least in part due to the fact that individual variation in TL is usually large relative to telomere dynamics. For example, in humans, TL range at any age is approximately 3,000 bp, while telomere shortening in adults is 20–30 bp/year (Lai et al., 2023). Thus, measurement precision that is sufficient to quantify individual variation in TL is considerable lower than the measurement precision that is required to accurately quantify telomere dynamics (Lindrose et al., 2021).

TL is not uniform across tissues within individuals, with proliferative tissues like blood typically exhibiting shorter telomeres than post-mitotic tissues (Demanelis et al., 2020; Sabharwal et al., 2018). This raises the possibility that telomere dynamics in some tissues may contribute disproportionately to aging and longevity. However, in birds, TL in the blood is known to act as a proxy for TL in the whole avian organism (Reichert et al., 2013). TL also varies in chromosomes within and between cells (Karimian et al., 2024; Sanchez et al., 2024), and hence TL measured in a sample is the average emerging from a large number of individual telomeres. Telomeres with different lengths do not contribute equally to cellular senescence: cell culture studies have identified the shortest telomeres in the genome to drive cellular senescence (Hemann et al., 2001; Zou et al., 2004). However, to what extent the shortest telomeres in the genome also drive associations between TL and lifespan or survival at the organismal level is not well known. Some studies have therefore focused on the proportion of short telomeres in the genome, using different approaches to quantify short telomeres. Moreover, multiple studies on free-living birds found the longer telomeres in the genome (i) to be stronger affected by environmental/experimental effects (Andrews et al., 2021; Pineda-Pampliega et al., 2020) and (ii) to show stronger relationships to fitness components (Atema et al., 2022; Bauch et al., 2014) compared to shorter telomeres. However, how long and short telomeres within the genome relate to lifespan variation remains to be identified.

In this study, we tested whether absolute erythrocyte TL or within-individual telomere shortening rate better predict lifespan in captive zebra finches (*Taeniopygia castanotis*) followed from hatching until their natural death. To this end, and to extend on a previous zebra finch study that revealed TL in early life to predict lifespan (Heidinger et al., 2012), we used longitudinal telomere measurements on samples taken throughout adulthood, and

applied a statistical model that allows the simultaneous testing for associations of lifespan with TL and ΔTL (Barrett et al., 2013; Sheldon et al., 2022). Moreover, we explored how this relationship varies across the TL distribution (i.e., long vs. short telomeres within the genome).

We specifically predicted that faster within-individual telomere shortening would be associated with a shorter lifespan, regardless of baseline TL (Fig. 1B). Blood samples were taken from zebra finches that were part of a long-term experiment with a focus on aging in relation to developmental conditions, manipulated by rearing birds in either small or large broods (with two or six chicks, respectively). This revealed that being reared in a large brood, or being female, both (independently) resulted in a shorter lifespan (Briga et al., 2017). We therefore predicted that females and birds reared in large broods display shorter telomeres and/or accelerated telomere dynamics.

# Materials and methods

## Rearing and housing

Rearing conditions and brood size manipulations have been described in detail elsewhere (Briga et al., 2017). In brief, zebra finch pairs were randomly matched and placed in indoor breeding cages with nestboxes, nesting material (hay), food and water *ad libitum*. Nestboxes were checked daily for the presence of eggs or chicks. Chicks were cross-fostered into broods of either two or six chicks, brood sizes within the range observe in the wild (Zann, 1996), until they were a maximum of 5 days old. On the day of cross-fostering, all chicks were weighed while no siblings were placed in the same nests and no parents were taking care of their own offspring. Chicks were ringed when around 12 days old, and at age 35 days, after reaching nutritional independence (Zann, 1996), they were moved to indoor aviaries with two pairs of foster parents for sexual imprinting. When approximately 3 months old, birds were housed in outdoor aviaries (320 × 150 × 210 cm) each containing single sex groups of 18–24 adults (Briga et al., 2017). All birds used in the present study were housed in aviaries where food was supplied in boxes suspended from the ceiling, with holes in the sides (Koetsier & Verhulst, 2011). As there were no perches under the holes, birds had to hover in front of the holes to obtain food.

## Blood samples

Blood samples, taken from the brachial vein of adult zebra finches, were snap frozen and stored in glycerol buffer at −80 °C within days from being collected (Atema et al., 2015). Telomere length was measured in 100 samples which were collected between 2007 and 2015 from 50 known-age individuals (26 males and 24 females). The average interval between the two sampling points was 993 days (range: 135–2,490 days). Individuals were selected based on their lifespan, being either relatively short or long within the experimental population, to maximize statistical power with a given sample size.

## Telomere Restriction Fragment analysis

Telomere Restriction Fragment (TRF) analysis without DNA denaturation was used to quantify terminal telomere sequence length as described in Salomons et al., (2009). In brief, DNA was extracted from the nuclei of 5 $\mu$l isolated erythrocyte cells and digested overnight with Proteinase K at 50 °C following protocol of Bio-rad CHEF Genomic DNA Agaroseplug Kit (Bio-Rad Laboratories, Inc). A quarter of the agarose plug (approximately 1 μg) from each sample was separated by pulsed-field gel electrophoresis (4.8 V/cm, 22h, switch time 1–25 s at 14 °C) with size ladders (Molecular Weight Marker XV, Roche Diagnostics and NEB MidRange PFG Marker I, New England Biolabs) that expanded the measurement range to 2.4–245 kbp. The samples were spread over four gels (Fig. S1) with samples from the same individual next to each other, with the first and second samples first interchangeably. Samples were analyzed blind with respect to all individual characteristics. For each sample, we calculated average TL over the smear, and the TL at percentiles 10–90 (in steps of 10%).

## Statistics

The selected samples were balanced with respect to rearing brood size and sex. Age at sampling and lifespan per brood size category and sex is summarized in Table 1. For graphical purposes only, lifespan was used as a factor with levels "short" and "long." This approach accommodated our selection of individuals with either relatively short or long lifespans within the experimental population (Fig. S2). Individuals with "short" lifespans had an average lifespan of 2.44 ± 0.57 years and individuals with "long" lifespans had an average lifespan of 7.04 ± 0.65 years (Table 1, Fig. S2).

In order to calculate within-individual telomere length repeatability, also known as intra-class correlation coefficient, we used the package *rptR* v.0.9.22 (Stoffel et al., 2017) with average TL as the response variable, delta age (explained below) as fixed effect and individual id and gel as random effects with 1,000 bootstrapping iterations.

We used linear mixed effects models using package *lme4* v. 1.1.35.1 (Bates et al., 2015) in R v. 4.1.2 (R Core Team, 2023) and the significance of interactions between variables were tested using package *car* v. 3.1.2 (Fox & Weisberg, 2019). Gel number and individual (when using both observations per individual) were included as random effects. We further partitioned an individual's age into average age and delta age to distinguish between- and within-individual age effects (Snijders & Bosker, 1999; van de Pol & Verhulst, 2006). Average age was calculated as the average of the ages at collection per individual, while delta age was calculated as the difference between an individual's chronological age at sampling and its average age (delta age = age at sampling-average age). For example, if an individual was sampled at ages 2 and 6 years its average age would be 4 years while the delta age would be −2 years for the sample collected at age 2 years (2 − 4= −2) and +2 years for the sample collected at age 6 years (6 − 4 = 2).

We used TL as the dependent variable, firstly, because our dataset included two TL measurements per individual but only one lifespan per individual, making TL the more appropriate response variable. Second, this approach allowed us to simultaneously explore experimental effects on TL and telomere shortening and further account for gel effects. Lastly, using TL as the response variable ensured that our results would be comparable to the ones from previous studies which have used this approach (Barrett et al., 2013; Sheldon et al., 2022).

To examine whether telomere dynamics across the telomere length distribution varies with lifespan, we fitted linear mixed-

**Table 1.** Sample size and average age at sampling (years) ± s.d. of each sex in each brood size and lifespan category.

| Sex | Brood size | Short lifespan (2.44 ± 0.57 years) | | Long lifespan (7.04 ± 0.65 years) | |
|---|---|---|---|---|---|
| | | Timepoint 1 | Timepoint 2 | Timepoint 1 | Timepoint 2 |
| Female | Small | 4 (1.37 ± 0.53) | 4 (2.63 ± 0.35) | 7(1.81 ± 1.33) | 7(5.81 ± 1.17) |
| | Large | 8 (0.87 ± 0.50) | 8 (2.06 ± 057) | 5 (2.32 ± 0.99) | 5 (6.40 ± 0.68) |
| Male | Small | 7 (1.46 ± 0.50) | 7 (2.40 ± 0.62) | 7 (2.12 ± 1.74) | 7 (6.75 ± 0.83) |
| | Large | 6 (1.09 ± 0.62) | 6 (2.06 ± 0.76) | 6 (2.33 ± 1.65) | 6 (7.05 ± 0.70) |

effects models. Models were fit separately for average TL and TL at percentiles 10–90 (in steps of 10%) with the following formula:

$$TL \sim lifespan + sex + natal\ brood\ size + delta\ age + average\ age + (lifespan : delta\ age) + (1|Bird\ ID) + (1|Gel\ ID).$$

Sample ID was included only when TL at the range of percentiles was the dependent variable. Fixed effect estimates were extracted using the *broom.mixed* v. 0.2.9.4 package (Bolker & Robinson, 2025) and we calculated the predicted slope of ΔTL per year for lifespans of 1, 4, and 8 years. All fixed effects were mean centred.

In addition, we ran a Cox proportional hazards model to further investigate the effects of TL and telomere shortening on lifespan using the function "coxph" of the R package *survival* v. 3.5–7. Lifespan was used as the time variable while average TL per individual (average of two measurements per individual) and telomere shortening were simultaneously included as fixed predictors.

## Results

Mean TL over all samples was 20.04 kbp (S.D. = 2.66, $n = 100$) and average TL varied almost two-fold between individuals, from 14 to 26.7 kbp (Fig. 2A, B). Average TL at first and second measurement was strongly correlated ($r = 0.9$, $t = 13.94$, $df = 48$, $p < 0.0001$, Fig. 2A), as also reflected in the within-individual telomere length repeatability of $R = 0.84$ (95% CI:0.65–0.92, $p < 0.0001$). This level of individual repeatability is in the range previous reported from studies using TRF (Kärkkäinen et al., 2022).

Individuals experienced greater telomere shortening over longer sampling intervals (Fig. 2C) while telomere length decreased with age at a rate of −0.26 ± 0.05 kbp/year (Table 2). Neither TL, nor telomere dynamics, differed significantly between males and females (Table 2) and effect sizes were not in the expected direction based on the observation that females have a shorter lifespan than males (Briga et al., 2017). Similarly, neither TL nor telomere dynamics differed significantly between birds reared in small or large broods (Table 2). The non-significant brood size effect on TL was in the direction expected on the basis of the shorter lifespan of birds reared in large broods, but the non-significant effect on telomere dynamics suggested faster telomere loss in birds reared in small broods, which is opposite to what we predicted. Excluding telomere dynamics outliers (Fig. 3) preserved significance across all models. Moreover, for all models, we tried fitting random slopes, but because there are only two observations per individual, the error term was unidentifiable.

We investigated associations of lifespan with TL and telomere dynamics by adding lifespan (mean-centred) and its interactions with natal brood size, sex and delta age to the model (Table 3, Table S1). This revealed a strong interaction between lifespan and delta age, indicating that individuals who lost telomeres faster lived significantly shorter lives (Fig. 3, Fig. S3). At the same time, lifespan was not significantly correlated with absolute TL (Table 3). A more detailed analysis further showed that telomere length at neither the first nor the second sampling time point predicted lifespan or remaining lifespan (Fig. S4, Table S2-S3).

The Cox model revealed a significant effect of within-individual telomere shortening rate on mortality hazard (HR = 0.66, 95% CI [0.45,0.95], $z = -2.21$, $p = 0.027$), indicating that faster telomere shortening predicts higher mortality rate. Average lifetime TL, simultaneously included in the model, showed no significant association with survival (HR = 1.06, 95% CI [0.95, 1.19], $z = 1.06$, $p = 0.29$). Thus, these results confirm the findings from the general linear modelling approach summarized in Table 3.

We repeated the mixed-effect model in Table 2 for each percentile separately, replacing sample average TL with the TL at percentiles 10%–90% in steps of 10. This revealed firstly that telomere dynamics depended on TL percentile, ranging from 0.007 kbp/year at the 10th percentile to −0.68 kbp/year at the 90th percentile (Fig. 4). Moreover, birds with longer lifespans exhibited less TL attrition across all percentiles, and the three-way interaction between delta age, lifespan, and percentile was significant (Table S4; $t = 4.09$, $p < 0.001$), indicating a stronger lifespan effect on telomere shortening at the higher percentiles; (Fig. 4). Note, however, that the interaction between lifespan and delta age was not significant at any individual percentile when testing the percentiles separately (all $p > 0.05$).

## Discussion

Examining whether telomere dynamics or TL best predicts mortality is of interest, because finding that telomere dynamics predicts mortality, while TL does not, argues against a causal effect of TL on mortality (Boonekamp et al., 2014; Simons, 2015). We here aimed to determine whether absolute TL and/or telomere dynamics best predict lifespan in zebra finches using telomere restriction fragment analysis applied to longitudinal blood samples taken in adulthood.

Previous investigations that aimed to tease apart associations of TL and telomere dynamics with lifespan or survival in birds have yielded mixed results. A longitudinal study on the Seychelles warbler (*Acrocephalus sechellensis*) showed that both adult telomere length and telomere dynamics predicted mortality (Barrett et

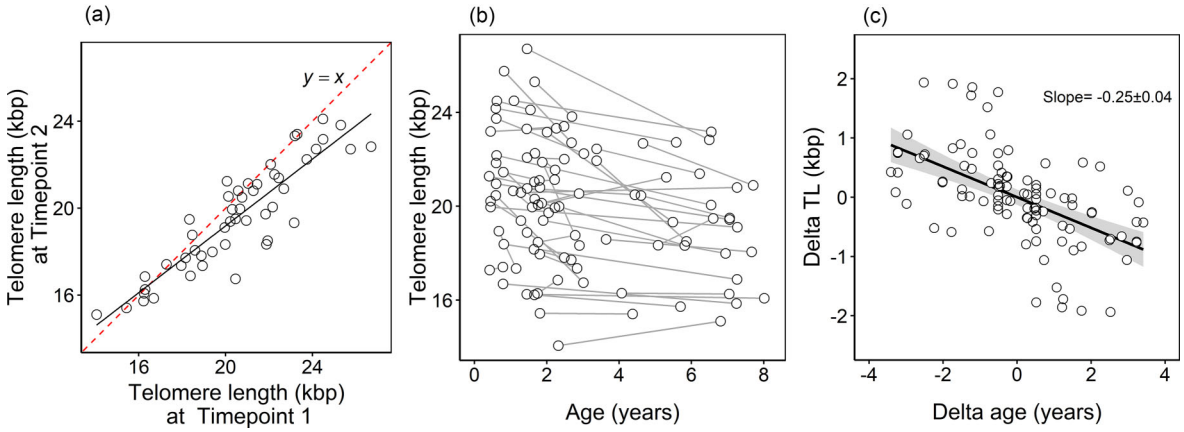


**Figure 2.** Telomere length in relation to age. (A) Telomere length at the two sampling ages plotted against each other. The dashed line indicates equal values ($y = x$) so data points below the dashed line reflect telomere shortening. (B) Within-individual change in telomere length. Longitudinal TL measurements of the same individual are connected with lines. (C) Delta TL and its relationship to delta age (delta values are differences of the first and second sample with the within individual average TL and age). The slope in the plot shows the estimate of linear mixed-effects model with individual as a random effect. Solid lines in panel C represent the best-fit regression through the data and the shaded area is the 95% CI.

**Table 2.** Within- and between-individual changes in telomere length.

| | **Telomere length (kbp)** | | | |
|---|---|---|---|---|
| ***Predictors*** | ***Estimates (±s.e.)*** | ***t-statistic*** | ***df*** | ***Pr(>\|t\|)*** |
| Intercept | 21.33 (±0.85) | 25 | 22.07 | **<0.001*** |
| average age (years) | −0.43 (±0.24) | −1.78 | 45.18 | 0.082 |
| delta age (years) | −0.26 (±0.05) | −4.81 | 47 | **<0.001*** |
| sex | −0.57 (±0.72) | −0.79 | 45.41 | 0.433 |
| natal brood size | −0.47 (±0.73) | −0.64 | 45.44 | 0.524 |
| delta age * sex | 0.16 (±0.11) | 1.46 | 47 | 0.153 |
| delta age * natal brood size | 0.09 (±0.11) | 0.782 | 47 | 0.438 |
| ***Random Effects*** | | | | |
| Bird ID | 5.73 | | | |
| Gel ID | 0.34 | | | |
| Residual | 0.85 | | | |

*Note:* Average age and delta age are expressed in years and telomere size is expressed in kbp. Sex and natal brood size (small broods = 2 chicks, large broods = 6 chicks) were mean-centred and coded −0.5 and +0.5, with males and large natal brood size coded as +0.5.

al., 2013). In contrast, in nestling of jackdaws (*Corvus monedula*) and white-browed sparrow-weavers (*Plocepasser mahali*), post-fledging survival was predicted by telomere dynamics with no additional effect of TL (Boonekamp et al., 2014; Wood & Young, 2019). In purple-crowned fairy wrens (*Malurus coronatus*) , telomere dynamics in the first year of life predicted lifespan, with no additional variation explained by TL (Sheldon et al., 2022). TL predicted survival in house sparrows (*Passer domesticus*), while the association with telomere dynamics did not reach statistical significance (Chik et al., 2024), but the power was low in the latter study due to low individual repeatability. Our main finding is that telomere dynamics was a significant predictor of lifespan, while there was no evidence for an association between TL at any point in adulthood and lifespan. We conclude that telomere dynamics is the primary predictor of lifespan, while TL by itself has little additional effect. This suggests the observed associations between TL and survival or lifespan can be attributed to factors other than TL. Although our dataset included the extreme ends of the lifespan distribution, primary effect estimates from continuous models remained unbiased and their significance was not altered by this selection.

We are aware of one other study that examined the association between TL, telomere dynamics and lifespan in zebra finches. Heidinger et al., (2012) reported a positive correlation of lifespan with TL at the age of 25 days, a significant but weaker correlation of lifespan with TL at the age of 1 year, and no significant association between TL and lifespan at older ages. Most samples in our study were taken from birds older than 1 year (Table 1), and hence the absence of a correlation between TL and lifespan is consistent with the results of Heidinger et al., (2012). With respect to the associations between telomere dynamics and lifespan, Heidinger et al. found no association when considering telomere dynamics in the first year of life; however, they did not examine telomere dynamics later in life, which precludes a direct comparison with our findings. Thus, while our findings differ in important ways from the findings of Heidinger et al., (2012), they are not inconsistent with each other. This suggests that associations of lifespan with TL and

**Table 3.** Telomere length in relation to age and lifespan. Average age, delta age, and lifespan are numeric variables expressed in years and average telomere size is expressed in kbp. Sex and natal brood size (small broods = 2 chicks, large broods = 6 chicks) were mean-centred and coded -0.5 and + 0.5, with males and large natal brood size coded as + 0.5.

| | **Telomere length (kbp)** | | | |
|---|---|---|---|---|
| | | | | *Pr(>\|t\|)* |
| ***Predictors*** | ***Estimates (±s.e.)*** | ***t-statistic*** | ***df*** | |
| Intercept | 21.5 (±0.91) | 23.60 | 23.10 | **< 0.001*** |
| lifespan (years) | 0.06 (±0.39) | 0.15 | 42.53 | 0.88 |
| delta age (years) | −1.28 (±0.29) | −4.78 | 48.01 | **< 0.001*** |
| average age (years) | −0.56 (±0.63) | −0.89 | 42.64 | 0.38 |
| sex | −1.1 (±1.67) | −0.66 | 41.23 | 0.52 |
| natal brood size | −1.69 (±1.74) | −0.97 | 42.78 | 0.34 |
| delta age * lifespan (years) | 0.15 (±0.04) | 3.88 | 48.01 | **< 0.001*** |
| delta age * sex | 0.10 (±0.32) | 0.33 | 40.97 | 0.75 |
| delta age* natal brood size | 0.25 (±0.33) | 0.77 | 40.72 | 0.45 |
| ***Random Effects*** | | | | |
| Bird ID | 6.18 | | | |
| Gel ID | 0.32 | | | |
| Residual | 0.66 | | | |

Note: Average age, delta age, and lifespan are numeric variables expressed in years and average telomere size is expressed in kbp. Sex and natal brood size (small broods = 2 chicks, large broods = 6 chicks) were mean-centred and coded −0.5 and +0.5, with males and large natal brood size coded as +0.5.

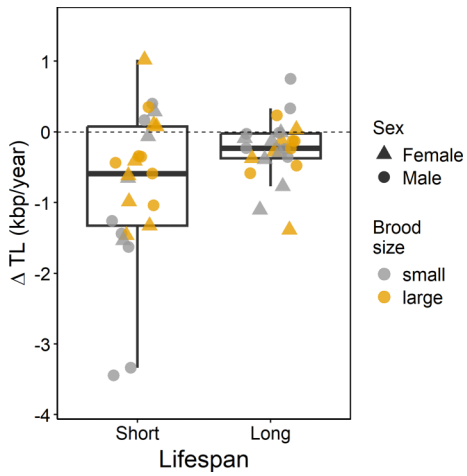


**Figure 3.** Telomere dynamics of average telomere length (kbp/year) in short and long-lived males and females (shapes) that were raised in either small (2 chicks) or large (6 chicks) broods (colours; $n = 50$).

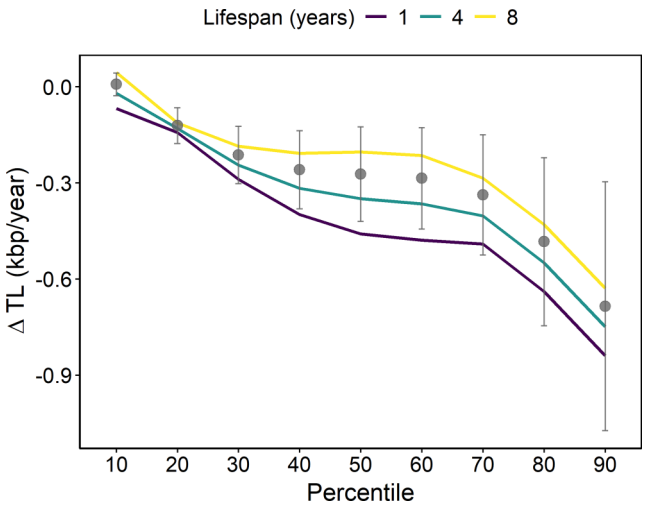


**Figure 4.** Telomere dynamics at the different percentiles (grey points with S.E.) estimated in models as in Table 2 separately for each percentile. Error bars indicate 95% confidence intervals around these estimates. Coloured lines represent estimated rates of ΔTL per year at each percentile for birds with lifespans of 1 (purple), 4 (green), and 8 (yellow) years from linear mixed-effects models fitted separately for each TL percentile.

telomere dynamics are age dependent in zebra finches, with TL potentially being important early in life, and telomere dynamics later in life.

In some species, including humans (Benetos et al., 2019; Verhulst et al., 2016) and barn swallows (Kauzálová et al., 2022), individual variation in telomere dynamics in adulthood is negligible, despite the use of high-precision measurements. Hence, in these species, telomere dynamics in adulthood is unlikely to predict mortality. Early-life telomere dynamics may, however, be modulated by developmental conditions, and early-life induced variation may predict disease and mortality, as demonstrated in humans (Benetos et al., 2018). In jackdaws, variation in telomere dynamics over most of adulthood is negligible (S.V. pers. obs.), while high shortening rate early in life and at the end of life are associated with higher mortality (Boonekamp et al., 2014; Salomons et al., 2009). Taken together, the above findings indicate that the rate of telomere dynamics better reflects phenotypic state than absolute length, at least with respect to survival. At the same time, TL at any time point after conception reflects telomere dynamics up to that point and may, for that reason, be informative with respect to individual phenotypic state.

We also investigated whether TL and telomere dynamics varied depending on developmental conditions (manipulated natal brood size, small broods = 2 chicks, large broods = 6 chicks) or sex, which are factors previously shown to influence lifespan in this population (Briga et al., 2017). Neither sex, nor natal brood size was associated with TL in our study. However, given our finding that telomere dynamics predicts lifespan while TL does not (or at least not significantly), effects of sex and natal brood size on telomere dynamics are more relevant with regard to lifespan than effects on TL. However, neither sex, nor natal brood size was associated with telomere dynamics. Moreover, contrary to our expectation, both non-significant effects (of sex and natal brood size) were such that longer-lived birds (males and birds reared in small broods) lost more telomere base pairs per year. Non-significance of observed differences may furthermore be due to insufficient statistical power, as also illustrated by the finding that lifespan was not significantly associated with natal brood size or sex in the present data set (Fig. S5), which for logistic reasons is a selection of the larger set in which these lifespan effects were documented (Briga et al., 2017).

Telomere dynamics depend on telomere length and long telomeres within the genome are known to shorten at a higher rate than short telomeres, possibly because they form a larger "target" for oxidative stress (Grasman et al., 2011; op den Buijs et al., 2004). Differential telomere dynamics can be inferred indirectly from the shape of the population level TL distribution, which typically becomes more "compressed" over time, both in cell cultures (Martens et al., 2000) and in human populations (Lai et al., 2023). We evaluated length-dependent telomere dynamics by examining the shortening rate at different percentiles of the within-sample TL distribution, and find that telomere shortening was faster at higher percentiles. This pattern has also been previously observed in humans (Sanchez et al., 2024; Zheng et al., 2024) and multiple avian species (Atema et al., 2019; Bauch et al., 2014; Salomons et al., 2009). The "compression" is due to little change in the shorter telomeres combined with a shift of the longer telomeres to lower values; the more constant lower boundary likely reflects senescence of hematopoietic stem cells with short telomeres.

Length-dependent telomere dynamics is of interest, since *in vitro* studies have shown that the *shortest* telomeres in the genome trigger replicative senescence (Bendix et al., 2010; Hemann et al., 2001; Zou et al., 2004). In contrast to the pattern observed *in vitro*, *in vivo* studies generally find the longer telomeres in the genome to be more affected by environmental effects (Pineda-Pampliega et al., 2020) and to show stronger relationships to fitness components (Atema et al., 2019; Bauch et al., 2014). Building on our finding that $\Delta$TL is faster for short-lived individuals, we further explored how this relationship varies across different TL percentiles. In the present data set, the association between $\Delta$TL and lifespan was steeper at higher percentiles, extending earlier observations on fitness components to associations with lifespan. However, these findings at higher percentiles coincide with higher measurement variance, which at least in part is due to the logarithmic nature of molecular ladders used in gel electrophoresis TL measurements. We consider this increased variance to be a likely explanation for the lack of statistical significance of the association between lifespan and age when examined for individual percentiles. In effect, when looking for a marker that best predicts lifespan, shortening of average TL, which integrates information across the whole telomere length distribution, was a better predictor of lifespan when compared to TL measured at individual percentiles. Our findings nevertheless argue against the idea that the finding that the shortest telomeres in the genome induce cellular senescence can be extrapolated to the organismal level.

Our results indicate that TL shortening can potentially better capture the overall physiological state and cumulative cellular aging of an organism while also reflecting the cumulative effects of genetic, environmental, and stochastic factors on the phenotype. However, despite the observed associations, telomere shortening—average or at specific percentiles—does not constitute a very *precise* predictor of lifespan. Since our results suggest that in captive zebra finches, variation in lifespan is associated with physiological differences other than telomere length and shortening, there is scope for additional characterizations of biomarkers that predict lifespan. Epigenetic modifications such as DNA methylation is a potential candidate to mediate the effects of early-life experiences on adult fitness (Anderson et al., 2024; Laubach et al., 2021; Tangili et al., 2025) but also drive (sex-specific) longevity (Tangili et al., 2023).

## Supplementary material

Supplementary material is available at *Journal of Evolutionary Biology* online.

## Data availability

Data and code used in this paper are available at https://doi.org/10.34894/L9EFMD.

## Author contributions

Marianthi Tangili (Data curation [equal], Formal Analysis [lead], Visualization [lead], Writing – original draft [lead]), Ellis Mulder (Data curation [equal], Formal Analysis [equal], Methodology [equal], Project administration [equal], Writing – review & editing [equal]), Blanca Jimeno (Methodology [equal], Project administration [equal], Writing – review & editing [equal]), Michael Briga (Methodology [equal], Project administration [equal], Writing – review & editing [equal]), Simon Verhulst (Conceptualization [equal], Data curation [equal], Formal Analysis [equal], Funding acquisition [equal], Methodology [equal], Project administration [equal], Supervision [equal], Writing – original draft [equal], Writing – review & editing [equal])

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## Conflicts of interest

None declared.

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