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Early-life telomere length predicts lifespan and lifetime reproductive success in a wild bird

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Abstract

Poor conditions during early development can initiate trade-offs that favour current survival at the expense of somatic maintenance and subsequently, future reproduction. However, the mechanisms that link early and late life-history are largely unknown. Recently it has been suggested that telomeres, the nucleoprotein structures at the terminal end of chromosomes, could link early-life conditions to lifespan and fitness. In wild purple-crowned fairy-wrens, we combined measurements of nestling telomere length (TL) with detailed life-history data to investigate whether early-life TL predicts fitness prospects. Our study differs from previous studies in the completeness of our fitness estimates in a highly philopatric population. The association between TL and survival was age-dependent with early-life TL having a positive effect on lifespan only among individuals that survived their first year. Early-life TL was not associated with the probability or age of gaining a breeding position. Interestingly, early-life TL was positively related to breeding duration, contribution to population growth and lifetime reproductive success because of their association with lifespan. Thus, early-life TL, which reflects growth, accumulated early-life stress and inherited TL, predicted fitness in birds that reached adulthood but not noticeably among fledglings. These findings suggest that a lack of investment in somatic maintenance during development particularly affects late life performance. This study demonstrates that factors in early-life are related to fitness prospects through lifespan, and suggests that the study of telomeres may provide insight into the underlying physiological mechanisms linking early- and late-life performance and trade-offs across a lifetime.

KEYWORDS
ageing, development, fitness, late-life, life-history, telomere, trade-offs

1 | INTRODUCTION

As resources are finite, life-history theory predicts that individuals may maximize fitness by investing resources into traits important for current fitness gains, at the expense of somatic maintenance for the benefit of future fitness gains (Ricklefs & Wikelski, 2002; Stearns, 1992). Such trade-offs may account for the plethora of findings that suggest adverse early-life conditions negatively affect lifespan and lifetime reproductive success (LRS; Kruuk, Clutton-Brock, Rose, & Guinness, 1999; Lindström, 1999; Nussey, Kruuk, Morris, & Clutton-Brock, 2007; Tung, Archie, Altmann, & Alberts, 2016), which can occur when animals invest in growth or competitiveness during development at the expense of their later-life fitness (Vedder, Verhulst, Bauch, & Bouwhuis, 2017). The mechanisms behind such patterns

are unclear, but recently it has been suggested that telomeres, the nucleoprotein structures at the terminal end of chromosomes, could link early-life conditions and developmental growth to lifespan and ultimately LRS (Monaghan & Haussmann, 2006; Monaghan & Ozanne, 2018).

Telomeres consist of noncoding TTAGGG sequence repeats that play a role in many important cellular processes (Blackburn, 1991; von Zglinicki, Bürkle, & Kirkwood, 2001). Most importantly, telomeres function to maintain chromosomal integrity and prevent damage to coding DNA by acting as a buffer against the loss of DNA that occurs with each cellular division (Blackburn, 1991; von Zglinicki et al., 2001). Once telomeres shorten to a critical length, the cell enters a senescent state or initiates cell apoptosis (von Zglinicki et al., 2001). In addition, some evidence suggests that TL may also have a role in age-related diseases such as cardiovascular disease and cancer (Aviv & Shay, 2018). The accumulation of cells in these states impairs tissue renewal capacity and function, causing the symptoms associated with organismal senescence (Aubert & Lansdorp, 2008). Telomeres are thought to connect early-life conditions to future life-history because they shorten with each cellular replication and in response to physiological stress such as oxidative (Boonekamp, Bauch, Mulder, & Verhulst, 2017; Reichert & Stier, 2017), nutritional and glucocorticoid stress (Bauch, Riechert, Verhulst, & Becker, 2016; Haussmann & Heidinger, 2015; Paul, 2011). Most often TL attrition is fastest during the initial growth phase, probably due to higher levels of cellular replication (Salomons et al., 2009; Spurgin et al., 2017; Vedder et al., 2017). Therefore, differences in the initial growth phase (prenatal and natal), greater cellular renewal, and physiological stress can have a direct effect on the ageing process and lifespan via telomeres (Meillère, Brischoux, Ribout, & Angelier, 2015; Monaghan & Ozanne, 2018; Nettle et al., 2015). Alternatively, other genetic (DNA damage or methylation) or physiological processes (hormonal, immune or inflammatory responses) which are affected by developmental conditions, can also have a proximate causal role in future life-histories but also affect telomere dynamics (Fagundes, Glaser, & Kiecolt-Glaser, 2013; Monaghan, 2014; Monaghan, Metcalfe, & Torres, 2008; Murgatroyd et al., 2009; Taylor, 2010). Thus, whether TL represents an underlying causal factor or a biomarker that reflects damage to other physiological structures is unresolved, but either way understanding the relationship between early-life TL and remaining lifespan is informative for both scenarios (Monaghan & Haussmann, 2006; Young, 2018).

Early-life TL has been found to predict lifespan in birds. In captive zebra finches (*Taeniopygia guttata*) early-life TL was positively related to lifespan (Heidinger et al., 2012), which suggests that intrinsic factors related to early-life TL contribute to senescence. However, whether this pattern is upheld in the wild, where extrinsic mortality, with a large stochastic component, predominates, and more broadly, whether there is indeed an association between early-life telomere length and lifetime fitness, is unknown, mostly due to challenges associated with obtaining this information for complete cohorts. Several studies have investigated survival probabilities over shorter time periods. A meta-analysis of 27 studies which measured TL at variable stages suggests that longer telomeres are associated with better survival (Wilbourn et al., 2018). Similarly, studies which investigate early-life TL and survival have also demonstrated positive TL effects on short-term survival (post growth period or fledging in birds (Geiger et al., 2012; Stier et al., 2014; Watson, Bolton, & Monaghan, 2015)) or first year survival (Cram, Monaghan, Gillespie, & Clutton-Brock, 2017; Salmón, Nilsson, Watson, Bensch, & Isaksson, 2017) but not always (Boonekamp, Mulder, Salomons, Dijkstra, & Verhulst, 2014; Cerchiara et al., 2017; McLennan et al., 2017; Stier et al., 2014; Ujvari & Madsen, 2009; Vedder et al., 2017). However, because extrinsic factors are always likely to be a major cause of mortality and TL-associated mortality is likely to diminish with age (Boonekamp, Simons, Hemerik, & Verhulst, 2013), the predictive effect of early-life TL on survival may be age-dependent under natural conditions, a plausible yet untested hypothesis in the wild (Supporting Information Table S1).

Positive associations between early-life TL and early-life survival are likely to extend to fitness (e.g., Cram et al., 2017; Watson et al., 2015), while this is likely to be true in many cases, life-history trade-offs, behaviour, and environmental conditions may obscure this association (Ricklefs & Wikelski, 2002). If individuals pursue different pace-of-life strategies (Ricklefs & Wikelski, 2002), those with a slower pace-of-life may favour self-maintenance (associated with longer early-life TL and longer lifespan) over investment in reproductive bouts, resulting in no, or even a negative, association between early-life TL and fitness. For example, common terns with long telomeres have high survival but low reproductive success (Bauch, Becker, & Verhulst, 2013, 2014). Hence, it is not only important to determine whether the capacity of TL to predict survival is age-dependent but also if longer TL translates into an increased LRS or if TL predicts LRS independently of lifespan.

Here we examined if and how early-life TL predicts several key components of fitness in wild purple-crowned fairy-wrens (*Malurus coronatus coronatus*), by utilising a high-quality long-term dataset (>10 years) including complete life-history information for several cohorts. Fairy-wrens are highly philopatric, cooperatively breeding birds with a maximum recorded lifespan of 13 years (Hidalgo Aranzamendi, Hall, Kingma, Sunnucks, & Peters, 2016; Kingma, Hall, Arriero, & Peters, 2010). We examined if TL at the time of near-complete skeletal growth (age 7 days) was related to survival at all life stages, and ultimately lifespan. In addition, we investigated if early-life TL predicted breeding tenure, i.e., if TL was related to the probability of gaining a breeder position, the age it was attained and the duration spent as a breeder. Finally, we examined potential associations with fitness by testing if early-life TL predicts an individual's LRS or genetic contribution to population growth. Because our study, for the first time, combines survival prior to and after recruitment as well as LRS and lifespan, we were able to disentangle in what manner early-life TL is related to future performance under natural conditions.

2 | MATERIALS AND METHODS

2.1 | Study species and data collection

Since July 2005 we studied a population of individually colour-marked *M. c. coronatus* at the Australian Wildlife Conservancy's Mornington Wildlife Sanctuary (S17°31' E126°6'). The core area of the study site is located along a 15 km stretch of Annie Creek and the Adcock River dominated by *Pandanus aquaticus*. At this site, wrens do not occupy habitat without *Pandanus*, which is used for nesting. Social groups, consisting of a dominant breeding pair and 0–8 subordinates, defend year-round stable territories which are distributed linearly along water courses (Kingma, Hall, & Peters, 2011). Breeding pairs, identified by duet song (Hall & Peters, 2008, 2009), are monogamous (with <5% extra-pair paternity [Kingma, Hall, & Peters, 2013]) and can breed all year round with a peak in breeding activity during the wet season (December–March; Hidalgo Aranzamendi et al., 2016). Subordinates, often retained previous offspring, do not gain parentage until they acquire an independent breeding position, on average around 1.5 years of age. They usually acquire a breeding position on their natal territory, or by taking up a vacancy elsewhere (Hidalgo Aranzamendi et al., 2016). We estimate that immigration is ~11% (78/691 birds >1 y.o. are immigrants; dispersal before 1 y.o. is very rare) indicating that philopatry is high.

We sampled 288 nestlings from four complete cohorts (2007–2010), and determined their survival, gaining of a breeder position, and reproductive success. We include all nestlings from nests that successfully fledged, as we do not predict that stochastic extrinsic mortality events during the nestling stage (predation, floods) would be predicted by TL. From 2006–2010, we monitored territories year-round by weekly visits to record the presence and social status of all individuals, and document nesting activity. Nests were checked every 2–3 days to accurately determine egg laying dates. Nestlings were banded on day seven from hatching (nestlings fledge at ~14 days) and a blood sample from the brachial vein was collected in a heparinised capillary tube which was then centrifuged. The plasma was removed and red blood cells stored in Longmire's lysis buffer (4°C). Sex was determined using an established PCR protocol (Griffiths, Double, Orr, & Dawson, 1998), validated in adults by sexually dichromatic plumage (Fan et al., 2017). From 2011, complete censuses of the core study area were conducted before and after the wet season to record the presence and social status of all individuals and to band all new unbanded birds (fledglings from unknown nests or dispersers) before they reach four months, the minimum age when individuals might disperse (Hidalgo Aranzamendi et al., 2016; Kingma et al., 2013; Kingma, Hall, Segelbacher, & Peters, 2009); parentage of unbanded birds was confirmed genetically (for details see, Hidalgo Aranzamendi et al., 2016). To minimise errors in survival estimates due to emigration, we conducted intensive yearly censuses from 2007 onwards to find birds that had dispersed outside the core area. We used playback of conspecific vocalisations to search rivers in the wider catchment area connected to the study site, including all suitable habitat (containing *Pandanus*) within 20 km of the core area, and some up to 60 km away, covering a total of 95 km of river length. The accuracy of this census technique is extremely high (>90% detection) due to the targeted, playback-based search technique and the species' habitat specificity (Hidalgo Aranzamendi et al., 2016). Fewer than 1% of birds that were assumed to have died were resighted inside or outside the core study area over the course of the study (for details up to 2014 see Hidalgo Aranzamendi et al., 2016; no additional resighting of birds assumed to have died have occurred since then). This additional long-distance monitoring provided reliable information on survival and whether or not all our focal nestlings acquired a breeding position, as well as more complete estimates of the LRS (number of offspring, confirmed using genetic parentage assignment, surviving to maturity, i.e., ≥6-months of age and hatched before June 2017, six months prior to the end of the study) of individuals that stayed to breed in the core area. However, since surveys were only conducted annually, accurate estimates of lifespan, age when breeder position was gained, breeder duration and LRS were not available for focal nestlings that emigrated from the core study area (n = 8). Our estimates of the number of immigrants (~11%, see above) and emigrants (~7%, 49/691 birds known to survive >1 year) are similar, which also suggests that our surveys outside the core study area do identify the majority of emigrants, and our estimates of survival and LRS are highly accurate. No bird from our population has ever been resighted beyond the range of our census and dispersal outside the river vegetation has never been recorded for this species (Skroblin, Cockburn, & Legge, 2014; Skroblin & Legge, 2010).

2.2 | Telomere measurement

Following Eastwood, Mulder, Verhulst, and Peters (2018), DNA was extracted using a modified QIAamp DNA kit (Qiagen) and the automated QIAcube HT instrument. DNA purity and concentration was assessed using a NanoDrop (ND-1000) whilst DNA integrity was assessed by running all samples on a 1% Agarose gel (100 V for 30 min). To measure TL we used a qPCR method based on Criscuolo et al., (2009) and validated for use in *M. coronatus* by Eastwood et al., (2018) which provided a relative measure of telomere length compared to a control gene. In brief, qPCR reactions were automated in an EpMotion 5075 on 96-well plates with a total reaction volume of 25 µl. The reaction included 12.5 µl of SYBR Green I (Roche), 300 nM of both the normalising control gene (glyceraldehyde-3-phoshate dehydrogenase; GAPDH) primers or 400 nM of both telomere primers (Integrated DNA Technologies) and 10 ng of sample DNA (except the two-fold serial dilution containing 40, 20, 10, 5 and 2.5 ng of DNA). All samples and controls were run in duplicate. The telomere primers were Tel1b 5' - CGG TTT GTT TGG GTT TGG GTT TGG GTT TGG GTT TGG GTT - 3' and Tel2b 5' - GGC TTG CCT TAC CCT TAC CCT TAC CCT TAC CCT TAC CCT - 3' (8). The GAPDH primers were GT2-GAPDH-forward 5' - CCA TCA CAG CCA CAC AGA AG - 3' and GT2-GAPDH-reverse 5' - TTT TCC CAC AGC CTT

AGC AG - 3' (Atema, Oers, & Verhulst, 2013). Reactions were run on a LightCycler 480 (Roche) machine on separate plates as follows: telomere (95°C for 15 min, followed by 35 cycles of 15 s at 95°C, 30 s at 56°C, 30 s at 72°C) and GAPDH (95°C for 15 min, followed by 40 cycles of 15 s at 95°C, 30 s at 60°C, 30 s at 72°C). A melt-curve analysis followed both reactions to ensure the correct product was amplified. Intra-assay Cq repeatabilities were 0.91 and 0.97 for telomere and GAPDH respectively. qPCR quality control and the calculation of relative telomere length was as per Eastwood et al., (2018). Relative telomere length was highly repeatable at the interassay repeatability = 0.85 and interextraction repeatability = 0.88 levels.

2.3 | Statistical analysis

To investigate if and in what manner early-life TL is associated with future fitness components we divided our analysis into three parts: survival/lifespan, breeding tenure, and LRS. In each analysis, TL and sex were modelled as fixed factors whilst hatch year was included either as a random intercept or in Cox regression survival analyses as a fixed factor to control for cohort effects. Because nest ID or maternal ID are possibly important factors predicting nestling fitness components they were included separately as random effects. However, nest ID and maternal ID explained less than 1% of the variance in all cases and were therefore removed from the final models. TL did not differ between males and females (mean ± *SD* males = 1.22 ± 0.16, females = 1.21 ± 0.15, $t = 0.6$, $df = 286$, $p = 0.56$). There was no need to control for age sampled because telomere length did not vary according to nestling age (regression between exact age sampled (for individuals observed hatching) vs. TL: $n = 19$, $F = 0.17$, $df = 1$, $p = 0.68$), presumably due to low variation in age (mean ± *SD* = 7 ± 1 days). All analyses were conducted in SPSS version 23 (IBM). In all cases, models were assessed for goodness-of-fit and we confirmed that the relevant model assumptions had been satisfied.

To test whether TL predicted survival, we used a survival analysis (Cox proportional hazards model). All individuals except long distance dispersers were included ($n = 280$), and individuals that were still alive at the end of the study were defined as censored ($n = 24$, includes individuals from all cohorts except 2008). To test for age-dependent effects we tested whether TL predicted the probability of surviving early-life period (between 0 and 12 months), and separately for those that survived to 12 months, the probability of surviving up to two years (between 12 and 24 months, when most individuals acquire a breeder position), using binomial logistic regression with a logit link function. The survival intervals of one and two years were based on population survival patterns and differences in life-history, with the first year encapsulating early-life survival during a life-stage when mortality is highest (70%), individuals are most dependent and remain in their natal territory, whilst those that then survive over two years (35% mortality during the second year) enter a life-stage with a much lower mortality rate (18% from the third year) and individuals have most likely gained a breeding position. Separating different biological stages of development in this way allows us to test for differences between an early-life stage with high mortality rates and dependency, and later-life adult stage with low mortality rates and reproduction. Among those that survived more than one year ($n = 56$), we tested whether early-life TL was associated with completed lifespan ($Log_{10}(+1)$ transformed to improve model residuals; excluding $n = 24$ that were still alive and $n = 8$ that emigrated from the core study area) using a linear mixed effects model (LMM).

To determine if early-life TL predicts whether ($n = 213$) or not ($n = 75$) an individual obtains a breeding position during its lifetime, we used a binomial logistic regression with a logit link function. In addition, we ran a Cox regression to test whether TL predicted the age of gaining a breeding position (in months) but removed individuals that had dispersed outside the core study area ($n = 8$). Individuals that never held a breeding position were excluded; breeders do not revert to being subordinate. To investigate whether early-life TL was associated with the number of months in a breeding position ($Log_{10}(+1)$ transformed to improve model residuals) we ran a LMM. Individuals that were still alive ($n = 24$; breeding duration was not complete) or had less accurate breeding position ages were removed, leaving $n = 43$ individuals.

Early-life TL could be a biomarker of adverse conditions that play a lasting physiological role on future reproductive performance. However, TL may also be linked to future performance through links with lifespan and duration spent in a breeding position. We therefore used two approaches to determine if TL predicted LRS. In both analyses we excluded individuals if they were still alive ($n = 24$), dispersed outside the core study area ($n = 8$) or their tenure as breeder lasted less than one month as there was no biological opportunity to reproduce, leaving $n = 43$. First, we ran a LMM with individual contribution to population growth ($p_{t(i)}$; $Log_{10}(+1)$ transformed to improve model residuals) as a dependent variable. We calculated $p_{t(i)}$ for each individual using equation 3.4 in (Coulson et al., 2006). To summarise, $p_{t(i)}$ was calculated based on an individual's survival and fitness for a given time interval (calendar year) which was adjusted for mean population survival, mean reproduction and population size (Coulson et al., 2006). The reproduction component was defined as the number of offspring produced in a year that survive longer than 6 months (multiplied by ½ as we consider both males and females). As $p_{t(i)}$ is annual realized fitness and it is weighted for population size we then summed $p_{t(i)}$ across all years that an individual was in a breeding position. Therefore, $p_{t(i)}$ reported here represents a contribution to population growth for the period in which each individual was in a breeding position. $p_{t(i)}$ was highly correlated with LRS (Pearson correlation $r = 0.89$, $n = 43$, $p < 0.001$) as expected. Second, we used a negative binomial model with LRS as the dependent variable and included breeding duration (number of months in a breeding position until June 2017), average breeding conditions (mean annual population reproductive success averaged over the years an individual spent as a breeder), sex and hatch year as fixed effects. Fixed covariates were examined for possible covariation (Supporting Information Figure S3).

TABLE 1 Relationship between early-life telomere length (TL) and survival and lifespan. In each analysis we controlled for sex and potential cohort effects (hatch year modelled as a random effect and removed from the model if it explained zero variance). Significant fixed effects are presented in bold

Dependent variable	Model	Independent variable	Estimate (*SE*)	Hazard ratio	Test statistic	*df*	*p* value
(a) Probability of survival (months) (*n* = 280, 24 censored)	Survival analysis (Cox proportional hazards)	TL	0.03 (0.42)	1.03	Wald = 0.01	1	0.94
		Hatch year	**—**	**—**	**Wald = 13.98**	**3**	**0.003**
		Sex (female)	0.16 (0.13)	1.18	Wald = 1.69	1	0.19
(b) Early-life survival (y/n) 0–12 months (*n* = 288)	Logistic regression	Intercept	−0.32 (1.05)	—	*t* = −0.31	285	0.76
		TL	−0.32 (0.84)	—	*t* = −0.38	285	0.70
		Sex (female)	−0.26 (0.26)	—	*t* = 1.01	285	0.31
		Hatch year (random)	0.12 (0.15)	—	Wald = 0.79	285	0.43
(c) Late-life survival (y/n) 12–24 months (*n* = 87)	Logistic regression	Intercept	−3.66 (2.10)	—	*t* = −1.76	84	0.08
		TL	**3.71 (1.73)**	**—**	***t* = 2.15**	**84**	**0.035**
		Sex (female)	−0.40 (0.49)	—	*t* = 0.81	84	0.42
		Hatch year (random)	0.39 (0.49)	—	Wald = 0.79	84	0.43
(d) Lifespan (months) >12 months survival (*n* = 56)	Linear	Intercept	0.57 (0.25)	—	*t* = 2.26	53	0.03
		TL	**0.74 (0.20)**	**—**	***t* = 3.69**	**53**	**0.001**
		Sex (female)	0.01 (0.06)	—	*t* = 0.17	53	0.86

To visualize how TL-fitness associations change with age, we calculated Spearman's rank correlations between early-life TL, lifespan and contribution to population growth for successive age categories (all individuals, and those that survived at least 6, 12 and 18 months respectively; Figure 3).

3 | RESULTS

3.1 | Survival and lifespan

The probability of survival from fledging onwards was not predicted by early-life TL or sex, but did depend on hatch year (Survival analysis; Table 1a), with individuals that hatched in 2010 having a longer lifespan than the birds hatched in other years (Supporting Information Figure S1). Effects of developmental conditions may be age-dependent (Briga, Koetsier, Boonekamp, Jimeno, & Verhulst, 2017), and we therefore analysed the association between TL and survival in more detail. The probability of surviving the first 12 months (early-life survival) was not predicted by TL (Table 1b and Figure 1), whilst controlling for sex and hatch year (see Supporting Information Figure S2a showing no association between TL and lifespan for individuals that died within 12 months). However, among birds that survived their first year, individuals with a longer early-life TL were more likely to survive the next 12 months (Table 1c and Figure 1). Moreover, among individuals that survived more than 12 months, lifespan was positively associated with TL (Table 1d and Supporting Information Figure S2a; note that total lifespan is unknown for the subset of birds still alive at the end of the study; see Supporting Information Table S2 and Figure S2b which show the confounding cohort effect of the 24 alive individuals). This effect was considerable, with individuals with the 90th percentile TL estimated to live 2.8 times longer than those with the 10th percentile TL (10th percentile mean =16.5 months, 90th percentile mean = 45.8 months).

3.2 | Breeding tenure

Early-life TL did not predict whether an individual would gain a breeding position during its lifetime (Table 2a; n = 288, all sampled individuals either acquired a breeding position or had died), whilst controlling for sex and hatch year. The age at which individuals gained a breeder position was not associated with TL but there were significant differences between sexes and hatch years (Table 2b). Females obtained a breeding position on average earlier than males, while the long-lived cohort that hatched in 2010 tended to be older than other cohorts when they gained a breeder position. Among birds that acquired a breeding position, TL was positively related with breeding duration (Table 2c and Figure 2b). However, breeding duration is highly correlated with lifespan (Pearson's r = 0.90, p < 0.001), suggesting that TL is related to breeding duration through its association with lifespan. In this study, no individuals were found to lose their breeding position before death.

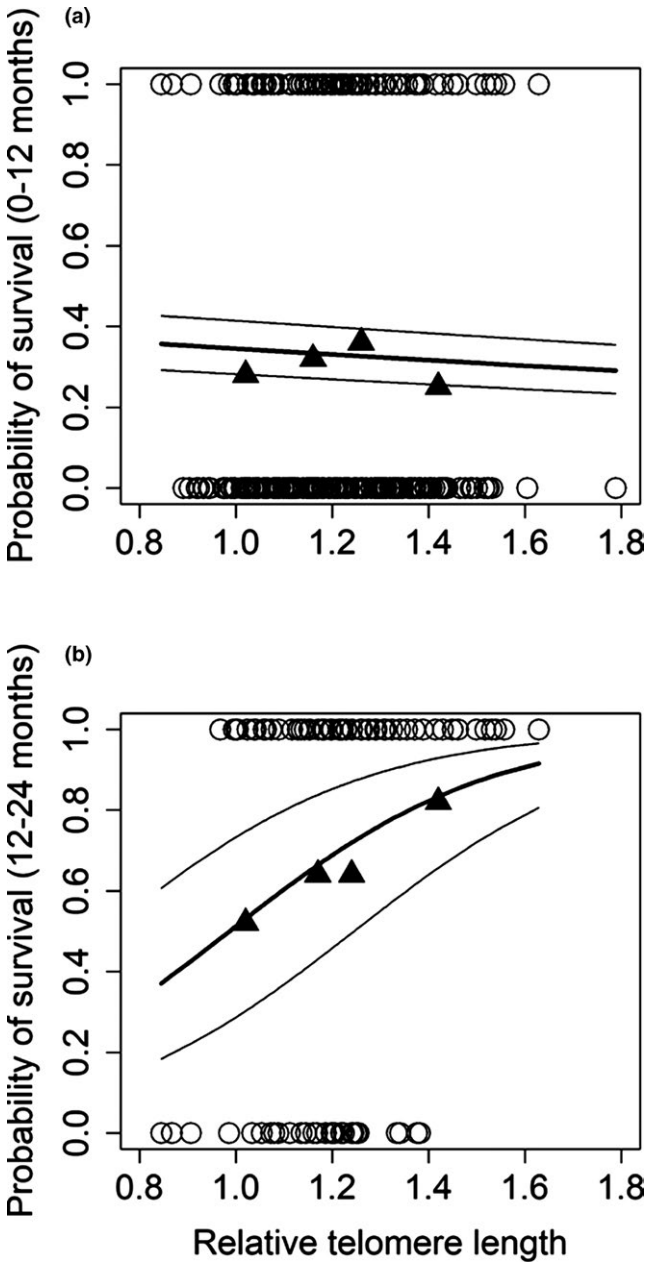

FIGURE 1 Early-life relative telomere length (TL) was not associated with (a) early-life survival (0–12 months) but was positively related to (b) survival in the following year (12–24 months). Lines represent model predicted probabilities and 95% confidence intervals. n = 288 nestlings were included 0 and 12 months of age and n = 87 between 12 and 24 months of age (for those that survived until at least 12 months). Triangles represent mean rTL and survival probabilities for each quartile and open circles represent the raw data

3.3 | Lifetime reproductive success

Among individuals that acquired a breeding position, individuals with a longer early-life TL had higher LRS (number of offspring surviving to maturity, i.e., ≥6 months; p = 0.028) when controlling for sex differences (Supporting Information Table S3). When controlling for breeding duration and average breeding conditions in each year in

TABLE 2 Relationship between early-life telomere length (TL) and breeding tenure. In each analysis we controlled for sex and potential cohort effects (hatch year modelled as a random effect and removed from the model if it explained zero variance). Significant fixed effects are presented in bold

Dependent variable	Model	Independent variable	Estimate (*SE*)	Hazard ratio	Test statistic	*df*	*p* value
(a) Acquired a breeding position (y/n, *n* = 288)	Logistic regression	Intercept	1.47 (1.11)	—	*t* = 1.34	285	0.18
		TL	−0.29 (0.88)	—	*t* = −0.33	285	0.74
		Sex (female)	−0.07 (0.27)	—	*t* = −0.25	285	0.80
		Hatch year (random)	0.14 (0.18)	—	Wald = 0.79	285	0.43
(b) Age obtained breeder position (months; *n* = 67, 213 truncated)	Survival analysis (Cox proportional hazards)	TL	−0.48 (0.81)	0.62	Wald = 0.35	1	0.55
		Hatch year	**—**	**—**	**Wald = 11.55**	**3**	**0.01**
		Sex (female)	**−0.95 (0.28)**	**0.39**	**Wald = 11.50**	**1**	**0.001**
(c) Breeding duration (*n* = 43)	Linear	Intercept	−0.41 (0.71)	—	*t* = −0.58	40	0.57
		TL	**1.26 (0.55)**	**—**	***t* = 2.29**	**40**	**0.03**
		Sex (female)	−0.10 (0.17)	—	*t* = −0.59	40	0.56
		Hatch year (random)	>0.001 (0.02)	—	Wald = 0.003	40	0.99

the same model, the association between TL and LRS remained positive, although the effect was smaller and not statistically significant ($p = 0.096$; Table 3a), which indicates that the positive association between TL and LRS is mostly but perhaps not exclusively derived from an association of TL with lifespan. To account for the possibility that LRS between partners may not be statistically independent we ran the LRS analysis for each sex separately. The conclusions were the same as the overall model and the TL effect sizes for males (effect size = 2.01, *SE* = 2.31, $t = 0.87$, $df = 11$, $p = 0.40$) and females (effect size = 1.89, *SE* = 3.06, $t = 0.62$, $df = 18$, $p = 0.54$) were both positive, of similar magnitude and nonsignificant. Accordingly, early-life TL was positively associated with individual contribution to population growth whilst controlling for sex ($p = 0.019$; Table 3b and Figure 2c; $n = 43$; excludes individuals that lacked accurate reproductive success data because they were still alive or had emigrated from the core study area). Using the back transformed model predicted values, individuals in the TL 90th percentile performed better than those within the 10th percentile, the latter on average performing worse than the population average (10th percentile mean $p_{t(i)} = -0.012$, 90th percentile mean $p_{t(i)} = 0.001$). Accordingly, Spearman's rank correlations between early-life TL and lifespan or contribution to population growth increased strongly when calculated for successive age categories, being negligible when calculated for all individuals, but increasing steeply to those that survived over 6 months and to those that survived at least 12 or 18 months, respectively (Figure 3).

4 | DISCUSSION

For the first time, we find evidence that early-life telomere length is related to fitness prospects in a wild songbird, specifically we show that the association between early-life TL and survival was age-dependent and only apparent after the first year. Early-life TL did not predict the probability of acquiring a breeding position or the age a breeding position was gained. However, TL was positively related to breeding duration and contribution to population growth because of their association with lifespan. Taken together, our findings indicate that variation in early-life TL is associated with fitness among breeders, by linking an individual's genetic contribution to the next generation via lifespan.

4.1 | Early-life as an indicator of fitness

Early-life TL is associated with lifespan in captive zebra finches under benign conditions, where intrinsic factors determine lifespan (Heidinger et al., 2012). Here we show the same pattern in wild birds exposed to the natural environment and its many causes of extrinsic mortality, but only among birds that survived their first year which had the highest mortality risk. This suggests that intrinsic factors associated with TL may also have a role in determining lifespan in wild animals (Heidinger et al., 2012). Exactly how early-life TL links to lifespan is currently unknown but several hypotheses exist (Young, 2018). Longer telomeres in early-life may causally confer a longer lifespan by reflecting younger biological age, whereby the minimum TL threshold takes longer to reach and cellular senescence is delayed (von Zglinicki et al., 2001). Alternatively, when factors that influence TL also damage other cellular processes, TL might be a biomarker of somatic redundancy and not necessarily directly involved in senescence (i.e., a non-causal biomarker; Boonekamp et al., 2013; Young, 2018). Given that the correlation between TL and survival was stronger later in life, when senescence is expected to occur, (Figure 3) our findings are compatible with a TL induced senescence hypothesis. However, we cannot rule out that early-life TL is a biomarker, correlated with other physiological factors related to senescence or

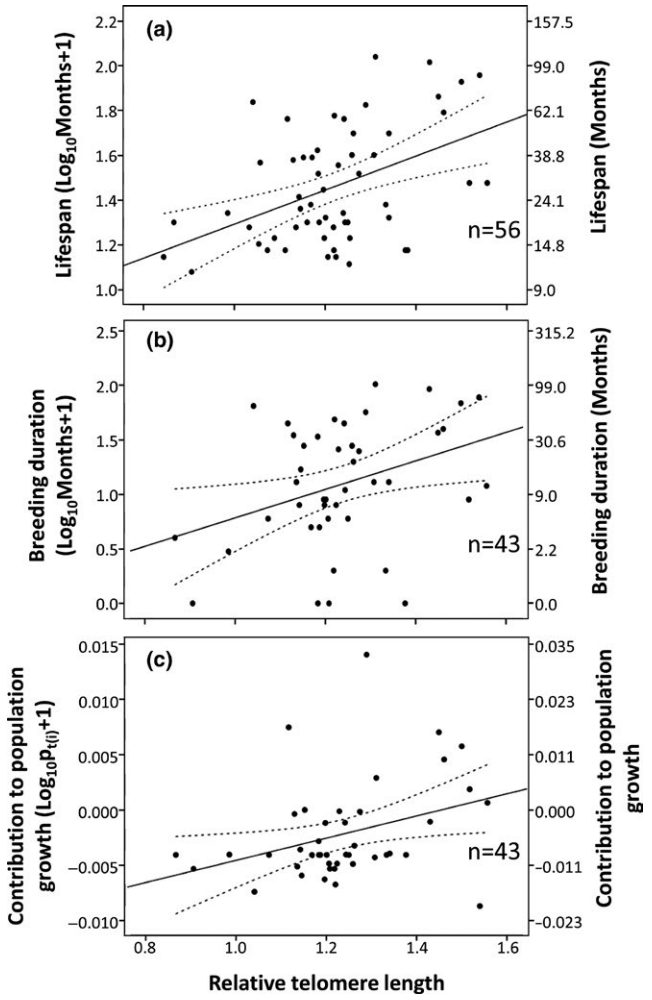

FIGURE 2 Early-life relative telomere length (TL) was positively related to (a) lifespan, (b) breeding duration and (c) contribution to population growth. Data presented do not include birds that were still alive at the end of the study nor those that dispersed outside the core study area. Lifespan does not include those that died before 12 months of age. Contribution to population growth (pt(i)) was calculated as per Coulson et al. (2006). Each regression line and associated 95% confidence intervals were calculated using least squares regression. Note that TL's association with both breeding duration and contribution to population growth is likely driven by lifespan (highly correlated, for details see Discussion and Table 3a)

mortality. Regardless of the mechanisms, because of the association with survival/lifespan we observed, early-life TL reflects individual fitness in the wild among birds that survived their first year.

Lifespan and the period spent as a breeder are key components of fitness. Therefore, studies that find an association between early-life TL and early-life survival or lifespan extrapolate that this translates into LRS (Asghar et al., 2015; Watson et al., 2015). Here we confirm that early-life TL is positively related to LRS and contribution to population growth in the wild: individuals with longer telomeres in early-life had higher fitness relative to others (Figure 2c). Further analysis revealed that TL was primarily associated with LRS through its association with lifespan, with a nonsignificant positive trend between TL and LRS independent of lifespan. Our findings indicate that the association between early-life TL and lifespan is not a result of intrinsic pace-of-life variation, which predicts that individuals with long early-life TL would have longer lifespans, but lower lifespan-adjusted-LRS. Instead, our data suggests that the early-life TL association with lifespan and LRS holds irrespective of adult pace-of-life and TL dynamics. This is in contrast to studies which suggest that telomere dynamics are altered by reproductive effort, and increased parental care can increase the rate of telomere loss (Bauch et al., 2016; Heidinger et al., 2012; Reichert et al., 2014; Sudyka et al., 2014) and reduce survival (Bauch, Becker, & Verhulst, 2013). Telomere loss over the reproductive period is currently unknown in *M. c. coronatus* and further work is necessary to elucidate whether early-life TL is likely to be associated with fitness in other species with different life-histories and pace-of-life strategies.

Our study may add to our understanding of evolutionary implications for telomere dynamics. Because TL is related to fitness via mortality in adulthood, natural selection (direct or indirect) should favour the evolution of longer telomeres. However, the strength of such selection would be weakened by stochastic ecological processes, as individuals must first survive higher mortality rates in their first year to breed (26% of fledglings). Nonetheless, given the cumulative effect over generations of small selection differentials, our data do suggest that there is selection for longer average TL in early-life. Given that TL heritability can be substantial (Dugdale & Richardson, 2018), this raises the question why TL are not longer, suggesting that there may be balancing selection, related to fitness costs associated with a longer TL than currently observed. This suggests that also in wild animals, there may be a balance between benefits of long telomeres and benefits of short telomeres, as has been suggested in humans for TL versus cardiovascular disease and cancer risk (Aviv & Shay, 2018). Alternatively, the TL variance component that associates with fitness is independent of the heritable variance component, in which case there is no selection for longer telomeres to evolve; in this scenario, the TL fitness association can possibly be attributed to early-life conditions that impact early-life telomere dynamics. Elucidation of the causes of early TL variation will be necessary to distinguish between these scenarios.

4.2 | When does early-life TL predict fitness?

Here, we investigated whether early-life TL was associated with survival separately for the first and second year of life because TL-associated mortality likely varies between those two life stages (Supporting Information Figure S1). The finding that the extent to which survival depends on an individual's state varies between life history stages may well be a general phenomenon, and we consider the most likely explanation that early-life mortality is largely due to extrinsic factors that exert their effect independent of individual state. In *M. c. coronatus* this may be due to fledglings having a long period of dependence on parental and subordinate care (3.5 months; Kingma et al., 2010), masking effects of variation in individual state on early-life survival, while individual state becomes important for survival after reaching independence.

TABLE 3 Relationship between early-life telomere length (TL) and lifetime reproductive success (LRS). In each analysis we controlled for sex and potential cohort effects (hatch year modelled as a random effect and removed from the model if it explained zero variance). Significant fixed effects are presented in bold

Dependent variable	Model	Independent variable	Estimate (*SE*)	Test statistic	*df*	*p* value
(a) Lifetime reproductive success (*n* = 43)	Negative binomial	Intercept	−4.68 (2.84)	*t* = −1.65	35	0.11
		TL	2.46 (1.45)	*t* = 1.71	35	0.096
		Breeding duration	**0.039 (0.01)**	***t* = 4.25**	**35**	**<0.001**
		Breeding conditions	0.98 (1.80)	*t* = 0.54	35	0.60
		Sex (female)	0.29 (0.42)	*t* = 0.69	35	0.49
		Hatch year				
		2007	−0.05 (0.79)	*t* = −0.07	35	0.95
		2008	−0.23 (0.46)	*t* = −0.49	35	0.63
		2009	−0.51 (0.52)	*t* = −0.99	35	0.33
		2010	0	0	0	0
(b) Contribution to population growth (*n* = 43)	Linear	Intercept	−0.02 (0.006)	*t* = −2.87	40	0.007
		TL	**0.01 (0.004)**	*t* = 2.44	**40**	**0.019**
		Sex (female)	0.001 (0.001)	*t* = 0.98	40	0.33

A not mutually exclusive alternative scenario is that TL-associated mortality develops late in life, with individuals with long early-life TL reaching a critical TL at later ages than individuals with a shorter early-life TL. In this scenario, as also suggested for humans, TL could represent a measure of somatic redundancy with mortality increasing when the redundancy nears depletion (Boonekamp et al., 2013). Which scenario dominates is likely to vary between species and perhaps even populations or cohorts, and as such may potentially explain variation between studies in TL – mortality associations. Regardless of the mechanisms causing the observed patterns, our findings highlight that the association between TL and survival depends on the life stage for which survival is measured (Figure 3), which has not previously been explicitly considered.

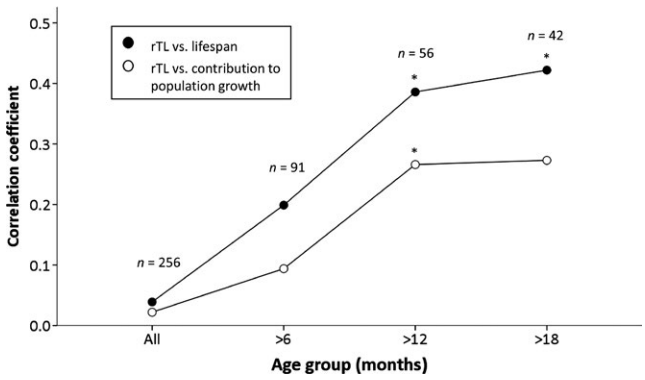

FIGURE 3 The strength of the association between early-life relative TL and fitness increases with age. The correlation (Spearman *r*) between TL and lifespan (closed circles) and contribution to population growth (open circles) is much stronger in individuals that survive their first year of life. Contribution to population growth was calculated using all individuals that fledged over the entire study period including those that never reached a breeding position. *n*, number of individuals remaining in each age category; *, significance $p < 0.05$

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DATA ACCESSIBILITY

All data presented in this manuscript are available from the DRYAD database (https://doi.org/10.5061/dryad.dt96f84/1).

AUTHOR CONTRIBUTIONS

J.E., S.V. and A.P. were involved in the study design. M.H., N.T., S.K., N.A., M.F., M.R. and A.P. conducted the fieldwork and collected samples. J.E. performed the lab work and statistical analyses. The manuscript was written by J.E., S.V. and A.P. with contributions from all authors.

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REFERENCES

Asghar, M., Hasselquist, D., Hansson, B., Zehtindjiev, P., Westerdahl, H., & Bensch, S. (2015). Hidden costs of infection: Chronic malaria accelerates telomere degradation and senescence in wild birds. *Science*, *347*(6220), 436–438. https://doi.org/10.1126/science.1261121

Atema, E., van Oers, K., & Verhulst, S. (2013). GAPDH as a control gene to estimate genome copy number in great tits, with cross-amplification in blue tits. *Ardea*, *101*(1), 49–54. https://doi.org/10.5253/078.101.0107

Aubert, G., & Lansdorp, P. M. (2008). Telomeres and aging. *Physiological Reviews*, *88*(2), 557–579. https://doi.org/10.1152/physrev.00026.2007

Aviv, A., & Shay, J. W. (2018). Reflections on telomere dynamics and ageing-related diseases in humans. *Philosophical Transactions of the Royal Society B: Biological Sciences*, *373*(1741), 20160436. https://doi.org/10.1098/rstb.2016.0436

Bauch, C., Becker, P. H., & Verhulst, S. (2013). Telomere length reflects phenotypic quality and costs of reproduction in a long-lived seabird. *Proceedings of the Royal Society of London. Series B, Biological Sciences*, *280*(1752), 20122540. https://doi.org/10.1098/rspb.2012.2540

Bauch, C., Becker, P. H., & Verhulst, S. (2014). Within the genome, long telomeres are more informative than short telomeres with respect to fitness components in a long-lived seabird. *Molecular Ecology*, *23*(2), 300–310. https://doi.org/10.1111/mec.12602

Bauch, C., Riechert, J., Verhulst, S., & Becker, P. H. (2016). Telomere length reflects reproductive effort indicated by corticosterone levels in a long-lived seabird. *Molecular Ecology*, *25*(22), 5785–5794. https://doi.org/10.1111/mec.13874

Blackburn, E. H. (1991). Structure and function of telomeres. *Nature*, *350*(6319), 569–573. https://doi.org/10.1038/350569a0

Boonekamp, J. J., Bauch, C., Mulder, E., & Verhulst, S. (2017). Does oxidative stress shorten telomeres? *Biology Letters*, *13*(5), 20170164. https://doi.org/10.1098/rsbl.2017.0164

Boonekamp, J. J., Mulder, G. A., Salomons, H. M., Dijkstra, C., & Verhulst, S. (2014). Nestling telomere shortening, but not telomere length, reflects developmental stress and predicts survival in wild birds. *Proceedings of the Royal Society of London. Series B, Biological Sciences*, *281*(1785), 20133287. https://doi.org/10.1098/rspb.2013.3287

Boonekamp, J. J., Simons, M. J. P., Hemerik, L., & Verhulst, S. (2013). Telomere length behaves as biomarker of somatic redundancy rather than biological age. *Aging Cell*, *12*(2), 330–332. https://doi.org/10.1111/acel.12050

Briga, M., Koetsier, E., Boonekamp, J. J., Jimeno, B., & Verhulst, S. (2017). Food availability affects adult survival trajectories depending on early developmental conditions. *Proceedings of the Royal Society of London. Series B, Biological Sciences*, *284*(1846), 20162287. https://doi.org/10.1098/rspb.2016.2287

Cerchiara, J. A., Risques, R. A., Prunkard, D., Smith, J. R., Kane, O. J., & Dee Boersma, P. (2017). Telomeres shorten and then lengthen before fledging in Magellanic penguins (*Spheniscus magellanicus*). *Aging*, *9*(2), 487–493. https://doi.org/10.18632/aging.101172

Coulson, T., Benton, T. G., Lundberg, P., Dall, S. R. X., Kendall, B. E., & Gaillard, J.-M. (2006). Estimating individual contributions to population growth: evolutionary fitness in ecological time. *Proceedings of the Royal Society of London B: Biological Sciences*, *273*(1586), 547–555. https://doi.org/10.1098/rspb.2005.3357

Cram, D. L., Monaghan, P., Gillespie, R., & Clutton-Brock, T. (2017). Effects of early-life competition and maternal nutrition on telomere lengths in wild meerkats. *Proceedings of the Royal Society of London. Series B, Biological Sciences*, *284*(1861), 20171383. https://doi.org/10.1098/rspb.2017.1383

Criscuolo, F., Bize, P., Nasir, L., Metcalfe, N. B., Foote, C. G., Griffiths, K., … Monaghan, P. (2009). Real-time quantitative PCR assay for measurement of avian telomeres. *Journal of Avian Biology*, *40*(3), 342–347. https://doi.org/10.1111/j.1600-048X.2008.04623.x

Dugdale, H. L., & Richardson, D. S. (2018). Heritability of telomere variation: It is all about the environment!. *Philosophical Transactions of the Royal Society B: Biological Sciences*, *373*(1741), 20160450. https://doi.org/10.1098/rstb.2016.0450

Eastwood, J. R., Mulder, E., Verhulst, S., & Peters, A. (2018). Increasing the accuracy and precision of relative telomere length estimates by RT qPCR. *Molecular Ecology Resources*, *18*(1), 68–78. https://doi.org/10.1111/1755-0998.12711

Fagundes, C. P., Glaser, R., & Kiecolt-Glaser, J. K. (2013). Stressful early life experiences and immune dysregulation across the lifespan. *Brain, Behavior, and Immunity*, *27*, 8–12. https://doi.org/10.1016/j.bbi.2012.06.014

Fan, M., Hall, M. L., Kingma, S. A., Mandeltort, L. M., Hidalgo Aranzamendi, N., Delhey, K., & Peters, A. (2017). No fitness benefits of early molt in a fairy-wren: Relaxed sexual selection under genetic monogamy? *Behavioral Ecology*, *28*(4), 1055–1067. https://doi.org/10.1093/beheco/arx065

Geiger, S., Le Vaillant, M., Lebard, T., Reichert, S., Stier, A., Le Maho, Y., & Criscuolo, F. (2012). Catching-up but telomere loss: Half-opening the black box of growth and ageing trade-off in wild king penguin chicks. *Molecular Ecology*, *21*(6), 1500–1510. https://doi.org/10.1111/j.1365-294X.2011.05331.x

Griffiths, R., Double, M. C., Orr, K., & Dawson, R. J. G. (1998). A DNA test to sex most birds. *Molecular Ecology*, *7*(8), 1071–1075. https://doi.org/10.1046/j.1365-294x.1998.00389.x

Hall, M. L., & Peters, A. (2008). Coordination between the sexes for territorial defence in a duetting fairy-wren. *Animal Behaviour*, *76*(1), 65–73. https://doi.org/10.1016/j.anbehav.2008.01.010

Hall, M. L., & Peters, A. (2009). Do male paternity guards ensure female fidelity in a duetting fairy-wren? *Behavioral Ecology*, *20*(1), 222–228. https://doi.org/10.1093/beheco/arn139

Haussmann, M. F., & Heidinger, B. J. (2015). Telomere dynamics may link stress exposure and ageing across generations. *Biology Letters*, *11*(11), 20150396. https://doi.org/10.1098/rsbl.2015.0396

Heidinger, B. J., Blount, J. D., Boner, W., Griffiths, K., Metcalfe, N. B., & Monaghan, P. (2012). Telomere length in early life predicts lifespan. *Proceedings of the National Academy of Sciences of the United States of America*, *109*(5), 1743–1748. https://doi.org/10.1073/pnas.1113306109

Hidalgo Aranzamendi, N., Hall, M. L., Kingma, S. A., Sunnucks, P., & Peters, A. (2016). Incest avoidance, extrapair paternity, and territory quality drive divorce in a year-round territorial bird. *Behavioral Ecology*, *27*(6), 1808–1819. https://doi.org/10.1093/beheco/arw101

Kingma, S. A., Hall, M. L., Arriero, E., & Peters, A. (2010). Multiple benefits of cooperative breeding in purple-crowned fairy-wrens: A consequence of fidelity? *Journal of Animal Ecology*, *79*(4), 757–768. https://doi.org/10.1111/j.1365-2656.2010.01697.x

Kingma, S. A., Hall, M. L., & Peters, A. (2011). Multiple benefits drive helping behavior in a cooperatively breeding bird: An integrated analysis. *The American Naturalist*, *177*(4), 486–495. https://doi.org/10.1086/658989

Kingma, S. A., Hall, M. L., & Peters, A. (2013). Breeding synchronization facilitates extrapair mating for inbreeding avoidance. *Behavioral Ecology*, *24*(6), 1390–1397. https://doi.org/10.1093/beheco/art078

Kingma, S. A., Hall, M. L., Segelbacher, G., & Peters, A. (2009). Radical loss of an extreme extra-pair mating system. *BMC Ecology*, *9*, 15. https://doi.org/10.1186/1472-6785-9-15

Kruuk, L. E. B., Clutton-Brock, T. H., Rose, K. E., & Guinness, F. E. (1999). Early determinants of lifetime reproductive success differ between

the sexes in red deer. *Proceedings of the Royal Society of London B: Biological Sciences*, *266*(1429), 1655–1661. https://doi.org/10.1098/rspb.1999.0828

Lindström, J. (1999). Early development and fitness in birds and mammals. *Trends in Ecology & Evolution*, *14*(9), 343–348. https://doi.org/10.1016/S0169-5347(99)01639-0

McLennan, D., Armstrong, J. D., Stewart, D. C., Mckelvey, S., Boner, W., Monaghan, P., … Williams, T. (2017). Shorter juvenile telomere length is associated with higher survival to spawning in migratory Atlantic salmon. *Functional Ecology*, *31*(11), 2070–2079. https://doi.org/10.1111/1365-2435.12939

Meillère, A., Brischoux, F., Ribout, C., & Angelier, F. (2015). Traffic noise exposure affects telomere length in nestling house sparrows. *Biology Letters*, *11*(9), 20150559. https://doi.org/10.1098/rsbl.2015.0559

Monaghan, P. (2014). Organismal stress, telomeres and life histories. *Journal of Experimental Biology*, *217*(1), 57–66. https://doi.org/10.1242/jeb.090043

Monaghan, P., & Haussmann, M. F. (2006). Do telomere dynamics link lifestyle and lifespan? *Trends in Ecology & Evolution*, *21*(1), 47–53. https://doi.org/10.1016/j.tree.2005.11.007

Monaghan, P., Metcalfe, N. B., & Torres, R. (2008). Oxidative stress as a mediator of life history trade-offs: Mechanisms, measurements and interpretation. *Ecology Letters*, *12*(1), 75–92. https://doi.org/10.1111/j.1461-0248.2008.01258.x

Monaghan, P., & Ozanne, S. E. (2018). Somatic growth and telomere dynamics in vertebrates: Relationships, mechanisms and consequences. *Philosophical Transactions of the Royal Society B: Biological Sciences*, *373*(1741), 20160446. https://doi.org/10.1098/rstb.2016.0446

Murgatroyd, C., Patchev, A. V., Wu, Y., Micale, V., Bockmühl, Y., Fischer, D., … Spengler, D. (2009). Dynamic DNA methylation programs persistent adverse effects of early-life stress. *Nature Neuroscience*, *12*(12), 1559–1566. https://doi.org/10.1038/nn.2436

Nettle, D., Monaghan, P., Gillespie, R., Brilot, B., Bedford, T., & Bateson, M. (2015). An experimental demonstration that early-life competitive disadvantage accelerates telomere loss. *Proceedings of the Royal Society of London. Series B, Biological Sciences*, *282*(1798), 20141610. https://doi.org/10.1098/rspb.2014.1610

Nussey, D. H., Kruuk, L. E. B., Morris, A., & Clutton-Brock, T. H. (2007). Environmental conditions in early life influence ageing rates in a wild population of red deer. *Current Biology*, *17*(23), R1000–R1001. https://doi.org/10.1016/j.cub.2007.10.005

Paul, L. (2011). Diet, nutrition and telomere length. *The Journal of Nutritional Biochemistry*, *22*(10), 895–901. https://doi.org/10.1016/j.jnutbio.2010.12.001

Reichert, S., & Stier, A. (2017). Does oxidative stress shorten telomeres in vivo? A review. *Biology Letters*, *13*(12), 20170463. https://doi.org/10.1098/rsbl.2017.0463

Reichert, S., Stier, A., Zahn, S., Arrivé, M., Bize, P., Massemin, S., & Criscuolo, F. (2014). Increased brood size leads to persistent eroded telomeres. *Behavioral and Evolutionary Ecology*, *2*, 9. https://doi.org/10.3389/fevo.2014.00009

Ricklefs, R. E., & Wikelski, M. (2002). The physiology/life-history nexus. *Trends in Ecology & Evolution*, *17*(10), 462–468. https://doi.org/10.1016/S0169-5347(02)02578-8

Salmón, P., Nilsson, J. F., Watson, H., Bensch, S., & Isaksson, C. (2017). Selective disappearance of great tits with short telomeres in urban areas. *Proceedings of the Royal Society of London. Series B, Biological Sciences*, *284*(1862), 20171349. https://doi.org/10.1098/rspb.2017.1349

Salomons, H. M., Mulder, G. A., van de Zande, L., Haussmann, M. F., Linskens, M. H. K., & Verhulst, S. (2009). Telomere shortening and survival in free-living corvids. *Proceedings of the Royal Society of London. Series B, Biological Sciences*, *276*, 3157–3165. https://doi.org/10.1098/rspb.2009.0517

Skroblin, A., Cockburn, A., & Legge, S. (2014). The population genetics of the western purple-crowned fairy-wren (*Malurus coronatus coronatus*), a declining riparian passerine. *Australian Journal of Zoology*, *62*(3), 251–259. https://doi.org/10.1071/ZO13087

Skroblin, A., & Legge, S. (2010). The distribution and status of the western subspecies of the Purple-crowned Fairy-wren (*Malurus coronatus coronatus*). *Emu*, *110*(4), 339–347.

Spurgin, L. G., Kat, B., Fairfield, E. A., Martijn, H., Jan, K., Terry, B., … Sandra, B. (2017). Spatio-temporal variation in lifelong telomere dynamics in a long-term ecological study. *Journal of Animal Ecology*, *87*(1), 187–198. https://doi.org/10.1111/1365-2656.12741

Stearns, S. C. (1992). *The evolution of life histories*. Oxford, UK: Oxford University Press.

Stier, A., Viblanc, V. A., Massemin-Challet, S., Handrich, Y., Zahn, S., Rojas, E. R., … Criscuolo, F. (2014). Starting with a handicap: Phenotypic differences between early- and late-born king penguin chicks and their survival correlates. *Functional Ecology*, *28*(3), 601–611. https://doi.org/10.1111/1365-2435.12204

Sudyka, J., Arct, A., Drobniak, S., Dubiec, A., Gustafsson, L., & Cichoń, M. (2014). Experimentally increased reproductive effort alters telomere length in the blue tit (*Cyanistes caeruleus*). *Journal of Evolutionary Biology*, *27*(10), 2258–2264. https://doi.org/10.1111/jeb.12479

Taylor, S. E. (2010). Mechanisms linking early life stress to adult health outcomes. *Proceedings of the National Academy of Sciences of the United States of America*, *107*(19), 8507–8512. https://doi.org/10.1073/pnas.1003890107

Tung, J., Archie, E. A., Altmann, J., & Alberts, S. C. (2016). Cumulative early life adversity predicts longevity in wild baboons. *Nature Communications*, *7*, 11181. https://doi.org/10.1038/ncomms11181

Ujvari, B., & Madsen, T. (2009). Short telomeres in hatchling snakes: Erythrocyte telomere dynamics and longevity in tropical pythons. *PLoS ONE*, *4*(10), e7493. https://doi.org/10.1371/journal.pone.0007493

Vedder, O., Verhulst, S., Bauch, C., & Bouwhuis, S. (2017). Telomere attrition and growth: A life-history framework and case study in common terns. *Journal of Evolutionary Biology*, *30*(7), 1409–1419. https://doi.org/10.1111/jeb.13119

von Zglinicki, T., Bürkle, A., & Kirkwood, T. B. L. (2001). Stress, DNA damage and ageing — An integrative approach. *Experimental Gerontology*, *36*(7), 1049–1062. https://doi.org/10.1016/S0531-5565(01)00111-5

Watson, H., Bolton, M., & Monaghan, P. (2015). Variation in early-life telomere dynamics in a long-lived bird: Links to environmental conditions and survival. *Journal of Experimental Biology*, *218*(5), 668–674. https://doi.org/10.1242/jeb.104265

Wilbourn, R. V., Moatt, J. P., Froy, H., Walling, C. A., Nussey, D. H., & Boonekamp, J. J. (2018). The relationship between telomere length and mortality risk in non-model vertebrate systems: A meta-analysis. *Philosophical Transactions of the Royal Society B: Biological Sciences*, *373*(1741), 20160447. https://doi.org/10.1098/rstb.2016.0447

Young, A. (2018). The role of telomeres in the mechanisms and evolution of life-history trade-offs and ageing. *Philosophical Transactions of the Royal Society B: Biological Sciences*, *373*(1741), 20160452. https://doi.org/10.1098/rstb.2016.0452

SUPPORTING INFORMATION

Additional supporting information may be found online in the Supporting Information section at the end of the article.

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