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A multi-tissue view on telomere dynamics and postnatal growth

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Abstract

Trade-offs between growth and self-maintenance are common in nature, such that early-life effects on growth can generate lasting consequences on survival and longevity. Telomeres—putative biomarkers of self-maintenance—may link early growth with these later phenotypic effects, but evidence for growth-telomere trade-offs is mixed. Null or even positive relationships between growth and telomeres may be driven by heterogeneity in resource availability or invariable allocation towards telomere maintenance within a population. We used nestling tree swallows (*Tachycineta bicolor*) to assess the directionality and timing of relationships between growth and telomere length in several tissues. We focused on two important phases of growth: first, the peak of postnatal growth occurring around 6 days old when nestlings grow by ~33% in a single day, and subsequently, the later phase of growth occurring as body mass plateaus near adult size at 12 days old. We quantified telomere attrition in blood during postnatal growth, as well as telomere length in the blood, brain, adrenals, and liver at 12 days old. Growth was unrelated to telomere length in the liver and telomere dynamics in blood. However, brain telomere length was positively correlated with peak growth, and adrenal telomere length was positively related to later growth, particularly for chicks that had experienced a temporary stressor. These observations suggest that variation in resource availability may mask trade-offs, generating positive correlations between growth and telomere length at the population level. They also provide insights into complex relationships between growth and self-maintenance that can be revealed by looking in multiple tissues.

KEYWORDS

ageing, bird, life history, lipopolysaccharide, postnatal growth, tissue

1 | INTRODUCTION

Life-history theory predicts that accelerated early life growth may confer reproductive and survival advantages (Dmitriew, 2011); however, trade-offs among growth, self-maintenance, and reproduction may also generate fitness costs to rapidly attaining a large adult size (Blanckenhorn, 2000; Stearns, 1992; Williams, 1966). For instance, experimental increases in growth rates may trade off with lifespan (Lee et al., 2013; Metcalfe & Monaghan, 2003; Rollo, 2002). These apparent shifts in resource allocation may be driven by increases in metabolic rate (Criscuolo et al., 2008; Stier et al., 2014) and oxidative stress (Smith et al., 2016). However, life-history trade-offs can become masked at the phenotypic level when individuals vary in resource availability (Van Noordwijk & de Jong, 1986). This can generate positive relationships between growth and lifespan, in that individuals with greater access to resources are better able to excel at

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both traits simultaneously (Hamel et al., 2009). Alternatively, invariable resource allocation to one or more traits may effectively decouple growth and self-maintenance, masking any connection between the two (i.e., canalization; Waddington, 1942). In essence, life-history trade-offs are not simply the product of resource allocation rules, but must be viewed in the context of resource availability; therefore, it comes as no surprise that observed patterns between growth and lifespan are mixed.

The telomere has started to emerge as a putative biomarker of self-maintenance used to explore these trade-offs, because notably, telomeres often predict lifespan (Tricola et al., 2018; Wilbourn et al., 2018). Telomeres are ribonucleoprotein structures that buffer the ends of chromosomes from erosion during cellular replication (Allsopp et al., 1995; Zakian, 2012). Stress exposure can promote telomere loss (Chatelain et al., 2020), likely via increases in glucocorticoids (Angelier et al., 2018) and oxidative damage (von Zglinicki, 2002; see also Boonekamp et al., 2017, Reichert & Stier, 2017). Theoretically, telomere loss should therefore become accelerated during metabolically-taxing periods, such as rapid growth (Geiger et al., 2012; Monaghan & Ozanne, 2018), generating trade-offs between growth and longevity. Faster growth is often associated with shorter telomeres (e.g., Boonekamp et al., 2014; McLennan et al., 2016), but many other observational studies and experimental manipulations of resource availability show a positive relationship or no relationship between growth and telomere length (reviewed in Monaghan & Ozanne, 2018; Vedder et al., 2017).

Adding further complication, work on the growth-telomere link is usually limited to a single-tissue focus (e.g., blood in birds or fin in fish), which may not represent processes occurring in other survival-oriented tissues for several reasons. First, telomere length covaries among *some* tissues, but this is not always the case (Kesäniemi et al., 2019; Power et al., 2021; Reichert et al., 2013; Rollings et al., 2019; Wolf et al., 2021b), and covariation may differ among life-history stages (Schmidt et al., 2016). For example, tissues may vary in age-related telomere dynamics (Cherif et al., 2003; Prowse & Greider, 1995; Tarry-Adkins et al., 2021) and responses to stress (Cattan et al., 2008; Ludlow et al., 2012), likely driven by tissue-specific regulation of telomeres (Haussmann et al., 2003; Haussmann et al., 2007; Ulaner et al., 2001). Second, tissues may vary in postnatal timing of development (Ricklefs, 1979, 1983). Therefore, each tissue may have critical periods during which it relates most to structural growth, leading to tissue-specific variation in growth-related telomere dynamics. Third, resource allocation to self-maintenance may also differ under normal versus resource-limited conditions (sensu Barker, 2007; Nowicki et al., 2002), setting the stage for telomere trajectories that diverge among tissues throughout the body. Although past work on telomere dynamics in single tissues has been critical to understanding formative concepts of ageing in the wild (Monaghan, 2014; Olsson et al., 2018), we must also look across the body to understand functional connections between telomeres, growth, and lifespan.

Here, we studied the relationship between growth and telomere length in multiple tissues of nestling tree swallows (*Tachycineta bicolor*). We focused on two important phases of growth: first, the peak of postnatal growth that occurs around 6 days old when nestlings grow by ~33% in a single day, and second, the subsequent phase of growth occurring as body mass plateaus near adult size at 12 days old. We quantified telomere attrition in blood from 5 to 12 days old, as well as telomere length in 12-day-old blood, brain, adrenals, and liver. Importantly, past work suggests that telomere length can change within these several days (Nettle et al., 2015; Stier et al., 2016; Wolf et al., 2021a), especially during this period of incredible somatic growth. First, we assessed the *directionality* of growth-telomere relationships. If increased investment in growth limits resource availability for telomere maintenance, we may expect a negative relationship between growth and telomere length. Alternatively, heterogeneity in resource availability among individuals may mask such trade-offs or generate a positive relationship between growth and telomere length. We next used model comparisons to ask about the *timing* of relationships between growth and telomere length, or specifically, whether peak growth or later growth better predicts telomere length in each tissue. Although these two growth phases are not independent, they do capture biologically unique periods that may shed light on previous mixed results linking growth and telomere dynamics. Specifically, postnatal growth and a tissue's telomere length may more likely covary during a period when maximal development and resource demands are occurring in the focal tissue. Due to limited sample sizes for these terminally collected tissues, we used separate analyses of each tissue to shed light on growth-telomere dynamics. As we elaborate below, a single-tissue view of the putative links between growth and telomere dynamics may mask complex relationships occurring in other survival-oriented tissues.

2 | METHODS

2.1 | Study species and sample collection

We conducted this experiment during spring 2018 in Monroe County, Indiana, USA (39.1851° N, 86.4997° W). We used a nest box population of tree swallows that we regularly monitored to determine lay and hatch dates. Our primary goal was to assess the relationship between postnatal growth and telomere length in several tissues. The peak of postnatal growth occurs at 6 ± 1 days old (Wolf et al., 2021b). Growth then begins to slow after 6 days old and plateaus near adult size by 12 days of age, concurrent with the timing of accelerated feather development. Faster growth rates are commonly implicated in greater chances of short-term survival (Arendt, 1997). Likewise, large adult body size is linked to future survival and fecundity (Haywood & Perrins, 1992; Magrath, 1991; McCarty, 2001).

We initially attempted to manipulate chick growth by injecting mothers with lipopolysaccharide (LPS) because it tends to decrease provisioning; however, the treatment had limited effects on nestling growth (see below). We began our experiment on day 5.1 ± 0.1 of the nestling period (hatch day = day 1, range = 5–6 days). We measured

nestling mass to the nearest 0.1 g, collected blood from the metatarsal vein (30–50 µl), and gave each chick a unique nail trimming for later identification ($n = 119$ chicks from 27 nests). The following day, we weighed chicks again to quantify peak growth. We weighed chicks a final time when they were 12 days old, a time when chicks do not prematurely fledge in response to research activities at their nest. At Day 12, we also banded chicks with one numbered USGS band and collected ≤50 µl blood from the alar vein, to quantify telomere attrition in blood over the study period. Peak growth (5–6 days old) and later growth (6–12 days old) was quantified relative to initial mass as $(mass_{t+1} - mass_t)/mass_t$. We acknowledge that peak and later growth phases are therefore not statistically independent, and additionally, are not biologically independent, as each growth phase can be influenced by prior embryonic and postnatal growth.

To assess the relationship between postnatal growth and telomere length in nonblood tissues, we euthanized one chick from each nest at the end of the study, i.e., 12 days old ($n = 12$ saline, $n = 12$ LPS), except for 3 nests due to permit limitations. We chose the chick with the median mass (relative to siblings) at 5 days old, which presumably experienced a typical growth trajectory, although we cannot completely control for remaining differences in pre- and postnatal environments among nests. Chicks were euthanized with an overdose of isoflurane, followed by decapitation and collection of blood, brain, liver, and adrenals. Samples were snap frozen on dry ice in the field and stored at −80°C. All protocols were approved by Indiana University's IACUC #18-004.

2.2 | Quantifying variation in maternal care

We attempted to enhance variation in peak nestling growth via manipulations to maternal care using LPS, a nonreplicating piece of bacterial cell wall that temporarily triggers an immune response and induces "sickness" behaviors, e.g., lethargy, weight loss, and reduced parental care (Dantzer et al., 2008; Palacios et al., 2011; Wolf et al., 2021a). We administered a subcutaneous injection of either saline or LPS saline-oil emulsion (0.4 mg/kg body weight) in the right dorsal apterium (saline: $n = 14$, LPS: $n = 13$). The LPS saline-oil emulsion consisted of LPS from *Escherichia coli* (serotype 055:B5, lot #086M4146V; Sigma-Aldrich) dissolved in 0.9% sterile saline. We then emulsified the solution at a 1:1 ratio with Freund's incomplete adjuvant (Sigma-Aldrich), which prolongs the expression of sickness symptoms up to 48 h (Owen-Ashley & Wingfield, 2006). We injected all control birds with a saline-oil emulsion. We recaptured 23 of the 27 females 24–48 h later ($n = 21$ within ~24 h, saline: $n = 10$, LPS: $n = 11$) and reweighed them to quantify fluctuations in body mass.

We also quantified changes in maternal visitation rate, which is a reliable indicator of feeding rates (McCarty, 2002). All females were banded with a colored PIT tag, with which we used radiofrequency identification (RFID) boards to measure visitation rate. Nest boxes were equipped with RFID readers, which recorded a female's ID and time stamp every time her PIT tag passed through the antennae at the box entrance (Bonter & Bridge, 2011; Lendvai et al., 2015). We determined the number of visits by filtering out continuous reads occurring within 3 s of another read of the same individual, as seen when a bird is perched at the nest entrance. We halved the number of remaining reads to account for entrances and exits. Because LPS-induced effects peak around 3–6 h postinjection (Dantzer et al., 2008), we focused our analyses on visitation rate occurring 3–6 h post-injection (average hourly number of visits). Baseline visitation rates were taken the day prior during the same window of time ($n = 15$; $n = 9$ excluded due to equipment malfunction).

2.3 | Telomere measurement

We quantified relative telomere length using quantitative PCR (adapted from Cawthon, 2009; Criscuolo et al., 2009). We extracted DNA from blood, brain, liver, and adrenals. We used the automated Maxwell® RSC Instrument (Promega) and Whole Blood DNA Kit (No. AS1520; Promega) to extract DNA from ≤25 µl whole blood. Nonblood tissues were manually homogenized on a sterile chilled block and an aliquot of the homogenate was used for DNA extraction with the Tissue DNA kit (No. AS1610; Promega). We quantified DNA concentration using the Epoch microplate spectrophotometer (BioTek).

Relative telomere length was measured as the ratio (T/S) of telomere repeat copy number (T) to a single gene copy number (S), relative to a pooled reference sample present on all plates. We amplified our single copy gene, glyceraldehyde-3-phosphate dehydrogenase (GAPDH) using primers GAPDH-F (5′-AACCAGCA AAGTACGATGACAT-3′) and GAPDH-R (5′-CCATCAGCAGCAGCCT TCA-3′). We amplified telomeres using primers telg (5′-ACACTA AGGTTTGGGTTTGGGTTTGGGTTTGGGTTAGTGT-3′) 4 and telc (5′-TGTTAGGTATCCCTATCCCTATCCCTATCCCTATCCCTAACA-3′). We conducted qPCR on 384-well plates (ABI Quantstudio 5). For each sample, we ran GAPDH and telomere reactions on the same plate. Before plating, we diluted DNA samples to 3.33 ng/µl using ultra-pure water. Each reaction had a total volume of 10 µl containing 5 µl PerfeCTA SYBR Green SuperMix Low ROX (Quanta Biosciences), 200 nM each GAPDH-F/GAPDH-R or 200 nM each telc/telg, and 3 µl DNA extract (10 ng total). qPCR reaction conditions were: 10 min at 95°C, followed by 30 cycles of 10 s at 95°C, 1 min at 62°C, and 30 s at 72°C, followed by 1 min at 95°C, 30 s at 55°C, and 30 s at 95°C. All samples fell within the bounds of the standard curve and reaction efficiencies were 98.72 ± 2.77 (GAPDH) and 107.55 ± 8.22 (telomere). Samples were run in triplicate, and mean values were used to calculate T/S ratios for each sample using the formula: $2^{-\Delta\Delta Ct}$, where $\Delta\Delta C_t = (C_t^{telomere} - C_t^{GAPDH})_{reference} - (C_t^{telomere} - C_t^{GAPDH})_{reference}$. Telomere attrition was corrected for regression to the mean (Verhulst et al., 2013), where more negative values indicate greater telomere loss. During initial assay optimization, the repeatability of the T/S ratio was calculated using 28 samples run across two plates (Wolf et al., 2021b). The intraclass correlation coefficient for the T/S ratio was 0.86 (95% confidence interval: 0.73–0.93). For analysis of the change in blood telomere length over time, both samples from an

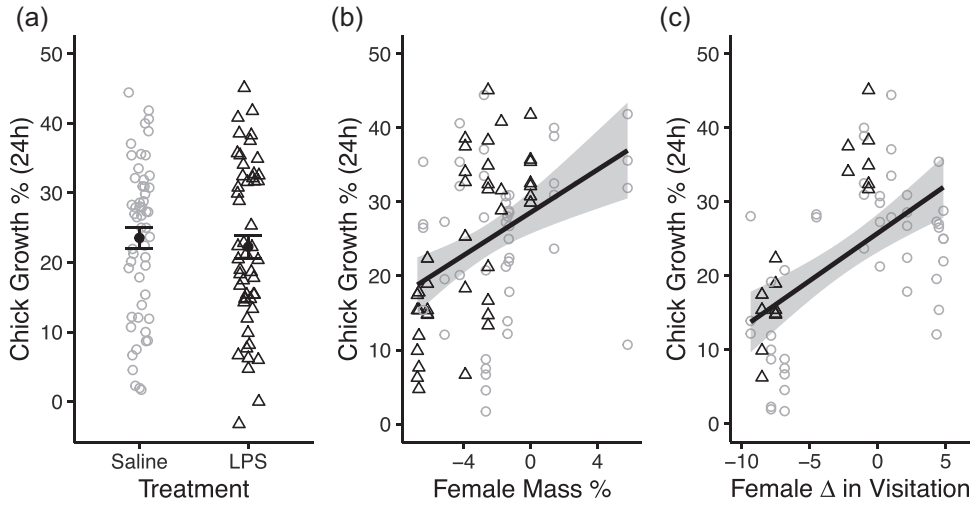

FIGURE 1 Peak postnatal growth of nestlings within the 24 h following maternal injections of either saline (gray, circle) or lipopolysaccharide (LPS; black, triangle), in relation to (a) maternal treatment and both change in maternal (b) mass and (c) visitation rate following maternal injections. Each point represents one nestling, and analyses control for the random effect of nest ID

individual were included on the same plate, and plates ($n = 7$) were balanced by treatment, hatch date, and brood size. Each non-blood tissue was run on a separate plate, which along with limited sample sizes, prevents us from directly comparing growth-telomere relationships across tissues.

2.4 | Statistical analyses

LPS injections did not significantly affect maternal change in body mass ($F_{1,19} = 2.64$, $p = 0.12$) or visitation rate ($F_{1,13} = 0.10$, $p = 0.76$). LPS treatment of mothers also did not affect peak nestling growth in the 24 h following injections ($F_{1,23} = 0.10$, $p = 0.76$, controlling for # siblings and nest ID, Figure 1a). However, we also found an interaction between treatment and chick mass at the start of the experiment in predicting the magnitude of peak growth ($F_{1,77} = 12.77$, $p = 0.006$, see Figure S1). As expected, the initially smallest 5 day old control chicks grew faster during estimated peak growth compared to initially larger age-matched individuals. Chicks of LPS-injected mothers, however, did not show this pattern: initially smaller and larger chicks grew similarly during peak growth. In addition, peak growth was also positively associated with changes in maternal body mass ($F_{1,20} = 5.83$, $p = 0.03$, Figure 1b) and marginally so for changes in visitation rate ($F_{1,14} = 4.24$, $p = 0.06$, Figure 1c). Therefore, maternal treatment showed no main effect on nestling growth, although indirect effects of treatment are possible via changes in maternal phenotypes, and so we did not ignore treatment in our main analyses.

We used an information-theoretic approach to evaluate support for competing hypotheses on the relationship between growth and telomere dynamics. For global models (below), we assessed multicollinearity and removed redundant variables with variable inflation factors ≥5 (Fox & Weisberg, 2011). We used Akaike Information Criterion (AIC_c—to correct for sample size) for model comparisons (Burnham & Anderson, 2002), and we present ΔAIC_c ($AIC_i - AIC_{best\ model}$) and AIC weights (weight of evidence for model) for models within 6 AIC_c of the top model (Burnham et al., 2011; Harrison et al., 2018). We then interpreted our top model sets using model averaging (Barton, 2019) and report full and conditional-averaged coefficients for each predictor variable included within the top model set. A full average assumes that each predictor is included in every model and detects robust effects on response variables, whereas conditional averages are calculated using only models containing the predictor and they, therefore, detect weaker effects (Grueber et al., 2011). All statistical analyses were performed in R (version 3.5.3, RStudio Team, 2019).

To ask whether specific growth periods had more overall support in predicting telomere dynamics within each tissue, we directly compared models including peak growth (5–6 days old) or later growth (6–12 days old). The global model for each tissue assumed a gaussian distribution and included treatment, growth (peak or later), and their interaction; as well as, hatch date, # siblings, and changes to maternal mass. All competing models predicting blood telomere dynamics included random effects of nest ID and qPCR plate. While we saw no major effects of treatment, we included treatment in the global model to account for potentially unseen effects of LPS on parental behavior (e.g., brooding, begging, food quality) and consequently, chick growth and telomere dynamics. Similarly, we included maternal mass fluctuations during peak nestling growth because maternal care at this time likely increases variation in growth and may slow or advance the pace at which nestlings approach asymptotic mass. We used *dredge* (Barton, 2019) to create model sets from the global model, in which all models for a given tissue included the same subset of data, had a maximum of three parameters (to avoid overparameterization), and only included one growth period per model to avoid multicollinearity of statistically-related variables. Blood telomere metrics were log-transformed to achieve normality. From the

$n = 119$ nestlings in the study, $n = 82$ survived to 12 days old (saline: $n = 45$; LPS: $n = 37$), had 5- and 12-day-old blood samples, and were not missing data on any variables included in the global model. After removal of euthanized nestlings with missing data, the final sample sizes were $n = 18$ brain, $n = 19$ adrenals, and $n = 19$ liver.

3 | RESULTS

Coefficients estimated using full and conditional averaging of top model sets are shown in Table 1 and Table S1, respectively. All models within $\Delta AIC_c \leq 6$ are reported in Supporting Information Material.

Relative telomere length was not significantly correlated between tissues (Pearson $r < |0.58|$, $p > 0.15$; Figure S2). However, we observed significant relationships between growth and telomeres in some tissues.

Specifically, in blood, the full average models indicate no relationship between Day 12 telomere length and any component of growth (Figure 2a,b, see Table S2 for AIC_c). While blood telomere length significantly shortened from 5 to 12 days old ($\beta = -0.23$, $t = -4.54$, $df = 81$, $p < 0.0001$), this change in telomere length in the blood was similarly unrelated to either peak growth or later postnatal growth (Figure 2c,d, see Table S3 for AIC_c). However, treatment was the top-ranking model for both blood telomere variables, followed by the intercept-only model, suggesting that LPS treatment of mothers may have had some effect on blood telomere length. Consistent with this view, conditional-averaged estimates support that chicks of LPS-injected mothers exhibited shorter blood telomere lengths at 12 days old ($z = 2.41$, $p = 0.02$, Table S1A) and experienced higher rates of telomere attrition from 5 to 12 days old in blood ($z = 2.30$, $p = 0.02$, Table S1B).

Brain telomere length at the end of the growth period was related to the pace of peak growth. Full-averaged coefficients show a significant positive relationship ($z = 3.23$, $p = 0.001$), where chicks who grew the most during the peak of growth exhibited *longer* brain telomere length at the end of the study (Figure 2e, see Table S4 for AIC_c). Brain telomere lengths were also shortest for chicks whose mothers lost the least mass during the peak of nestling growth ($z = 3.11$, $p = 0.002$). Brain telomeres were unrelated to later nestling growth or maternal treatment (Figure 2f).

For adrenal telomere length, a single model containing later nestling growth, treatment, and their interaction had a $\Delta AIC_c \leq 6$. Here, we see a significant interaction between later growth and treatment ($z = 2.87$, $p = 0.004$, Figure 2g,h, see Table S5 for AIC_c), where faster-growing chicks of LPS-injected mothers had significantly *longer* adrenal telomeres.

For liver telomere length, the top model set included both peak and later growth (see Table S6 for AIC_c). However, model averaging showed no relationship between liver telomeres and either peak nestling growth ($z = 0.26$, $p = 0.79$, Figure 2i) or later nestling growth ($z = 0.47$, $p = 0.64$, Figure 2j).

TABLE 1 Full model-averaged coefficients for the top model sets (≤ 6 ΔAIC_c) investigating the role of postnatal growth on chick telomere length

	Estimate	*SE*	Adj *SE*	*z*	*p*
(A) Blood relative telomere length ($n = 82$ chicks)					
Intercept	0.01	0.14	0.15	0.10	0.92
Treatment (LPS)	−0.13	0.14	0.14	0.93	0.35
# Siblings	0.008	0.03	0.03	0.28	0.78
(B) Change in blood relative telomere length ($n = 82$ chicks)					
Intercept	−0.40	0.22	0.22	1.83	0.07
Treatment (LPS)	−0.18	0.19	0.19	0.95	0.34
# Siblings	0.02	0.05	0.05	0.35	0.73
(C) Brain relative telomere length ($n = 18$ chicks)					
Intercept	−0.51	0.83	0.89	0.57	0.57
Peak growth	0.04	0.01	0.01	3.23	**0.001**
Treatment (LPS)	−0.01	0.06	0.07	0.19	0.85
Mom mass	−0.010	0.03	0.03	3.11	**0.002**
Hatch	0.00	0.00	0.00	0.13	0.90
# Siblings	0.10	0.10	0.10	1.03	0.30
(D) Adrenal relative telomere length ($n = 19$ chicks)					
Intercept	−1.03	0.556	0.59	1.76	0.08
Later growth	0.01	0.004	0.005	2.91	**0.004**
Treatment (LPS)	−1.89	0.63	0.66	2.85	**0.004**
Later growth × treatment	−0.02	0.005	0.006	2.87	**0.004**
(E) Liver relative telomere length ($n = 19$ chicks)					
Intercept	1.17	0.96	1.02	1.14	0.25
Peak growth	0.002	0.006	0.006	0.26	0.79
Later growth	0.001	0.003	0.003	0.47	0.64
Treatment (LPS)	−0.009	0.08	0.09	0.10	0.92
Mom mass	−0.0008	0.009	0.01	0.08	0.94
Hatch	0.00	0.00	0.0001	0.12	0.91
# Siblings	0.02	0.05	0.05	0.31	0.76

Abbreviation: LPS, lipopolysaccharide.

4 | DISCUSSION

Because telomere length has been connected with longevity (Tricola et al., 2018; Wilbourn et al., 2018), the telomere is a potentially useful biomarker for testing the hypothesis that early-life growth generates lasting consequences on lifespan. Past work tests this idea in limited tissue types—both within and among taxon—which may contribute to the inconsistent relationships reported between growth and telomeres (reviewed in Monaghan & Ozanne, 2018). In this study, we assessed this relationship in multiple tissues in a wild system. We

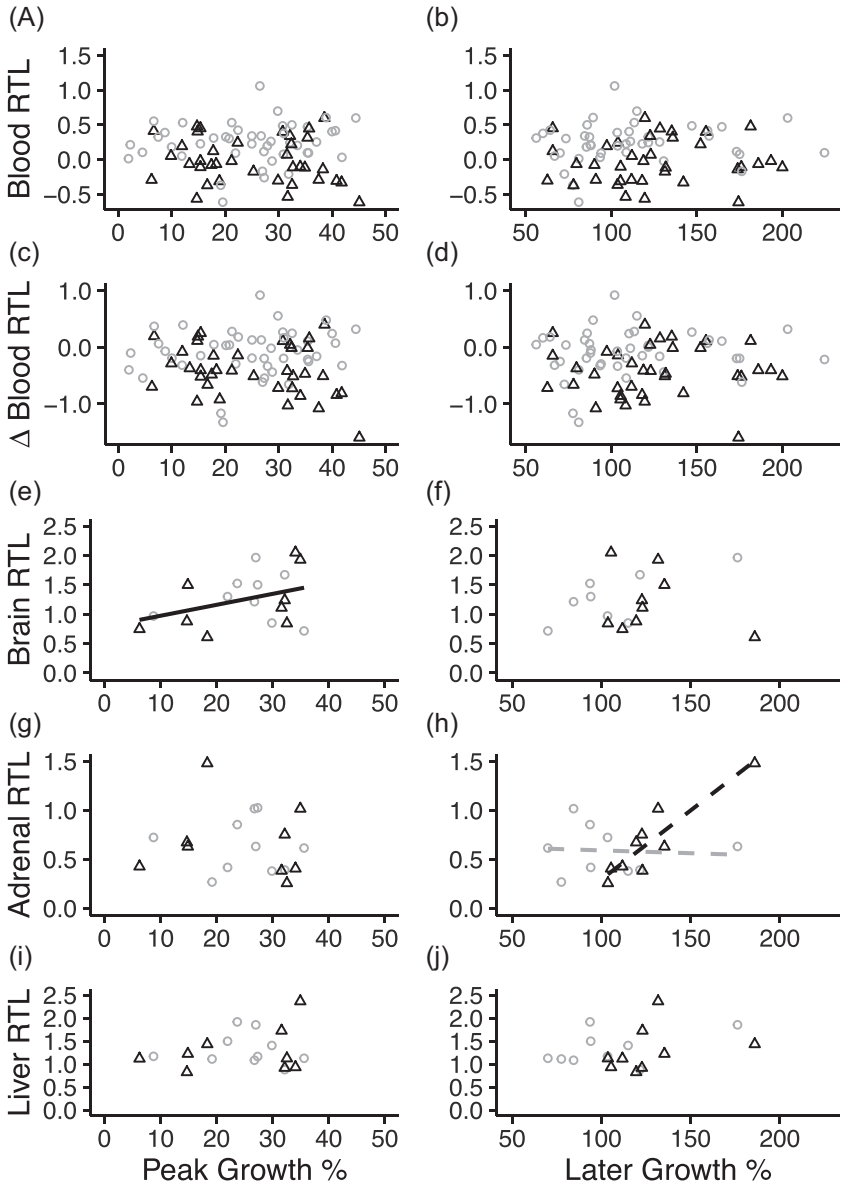

FIGURE 2 Postnatal growth and telomere dynamics in 12-day-old nestlings of females injected with either saline (gray, circle) or lipopolysaccharide (LPS; black, triangle) when nestlings were 5 days old. Relationships between peak growth (i.e., 5–6 days old) or later postnatal growth (i.e., 6–12 days old) and relative telomere length in several tissues: blood (a,b); change in blood from 5 to 12 days old (cd); brain (e,f); adrenals (g,h); and liver (i,j). Blood telomere data is log-transformed. Each point represents one nestling, and models control for the random effect of nest. Predicted lines are shown for significant main effects of growth (solid line) and growth by treatment interactions (dashed line) on relative telomere length

found that postnatal growth and telomeres were not related in the blood or liver. However, we did find that brain telomere length was positively correlated with *peak* growth, and adrenal telomere length was positively related to *later* growth, particularly so for chicks whose mothers had experienced an immune challenge. These observations collectively suggest that variation in resource availability can mask trade-offs in some tissues and generate positive correlations between growth and telomere length at the population level. That this latter pattern was only present in the adrenals of chicks at manipulated nests, but not controls, suggests that these relationships can remain hidden until a challenge. Moreover, a single-tissue view of telomere dynamics can miss important information gained from multiple tissues regarding links with early-life growth.

If increased investment in growth limits resource allocation to self-maintenance, we might expect a negative relationship between growth and telomere length (e.g., Burraco et al., 2017; Herborn et al., 2014; Jennings et al., 1999). However, in our study, early life growth was *positively* related to telomere length in two tissues: the brain and adrenals, which may suggest a "scaling" of resource allocation as resource availability increases (Van Noordwijk & de

Jong, 1986). Under this scenario, a nestling with more resources would be able to allocate additional energy towards both growth *and* telomere maintenance, relative to a resource-limited chick. However, these dynamics are difficult to assess directly given the necessity of terminal sampling in some tissues. Alternatively, the absence of growth-telomere relationships, as shown in liver and blood, could occur under several scenarios: first, with canalization of investment towards one trait (Waddington, 1942), which lowers its variation and decreases the possibility of correlations with other life history traits (as in Vedder et al., 2017); second, due to delayed responses that take longer to accumulate following a change in resource availability (Salmón et al., 2021); and third, with a lack of power to detect patterns. That blood and liver telomeres were not related to growth in either treatment suggests that telomeres in these tissues are less sensitive to early life stress; however, stronger manipulations are needed to separate this outcome from alternatives, e.g., measurement error or low power. Together, our results do not show trade-offs between growth and telomeres, but rather, they reveal some degree of coordinated investment in growth and telomere maintenance, at least in the brain and adrenals.

We also observed variation in the specific timing over which growth related to telomeres within each tissue. We predicted that telomere length would most likely covary with postnatal growth during periods of increased development in each focal tissue. As explored above, this could result in positive or negative relationships between body growth and telomere length, if body growth tracks resource availability or reflects increasing resource competition within the body, respectively (Ricklefs, 2003). In this study, we find that brain telomere length is better predicted by peak growth instead of later growth. Altricial birds experience extraordinary postnatal brain growth (Bennett & Harvey, 1985), much of which occurs during the peak of postnatal growth (Iyengar & Pilo, 1981a; Iyengar & Pilo, 1981b; Pilo & Iyengar, 1981). Therefore, it follows that brain telomere length covaries best with body growth occurring during its own maximal development, although we cannot rule out delayed effects of earlier developmental factors, such as genetic, maternal, or other environmental effects. Adrenocorticoid responses, on the other hand, can develop later into the postnatal period and even into adulthood (Rensel et al., 2010; Tilgar et al., 2009; Wada et al., 2007; Wada, 2008). Consistent with this timing of adrenal development, we found that adrenal telomere length was unrelated to peak growth at ~5–6 days old, but was instead positively correlated with later growth. To effectively link telomeres to variation in growth, we advocate for a shift from using overall postnatal growth to growth occurring during important developmental periods, the timing of which may vary among tissues.

While treatment of mothers with LPS had a negligible effect on maternal phenotypes and nestling growth, our results hint at treatment effects on telomeres in select tissues. Conditional model averaging suggests that maternal LPS treatment led to shorter telomeres and higher rates of telomere attrition in nestling blood; however, full model averages do not, meaning the effect is weak. While significant decreases in blood telomere length have been shown at similar timescales in developing birds (Nettle et al., 2015; Stier et al., 2016; Wolf et al., 2021a), apparent telomere dynamics could also be driven by shifts in blood cell composition (Epel, 2012). Furthermore, a mother's change in mass, but not her treatment, predicted nestling growth. This suggests that maternal LPS did not robustly manipulate resource availability across nests, in contrast to other work (Dantzer et al., 2008; Palacios et al., 2011; Wolf et al., 2021a). For blood telomere length, top models included treatment, but not maternal mass or nestling growth, suggesting that at least in this tissue, telomere maintenance responds more strongly to some unmeasured aspect of stress exposure as opposed to resource availability. Indeed, past work shows that LPS treatment reduces adult body temperature and activity levels (Owen-Ashley et al., 2006), which could translate into stressful alterations in brooding and parental presence for chicks. Adrenal telomere length, on the other hand, was positively related to later growth, but only for chicks of immune-challenged mothers. This result suggests that covariation between telomere length and growth emerges in response to a challenge, even when that challenge does not directly impact growth per se. Similar interactions occur in jackdaws (*Corvus monedula*), in that body mass predicted telomere length only upon brood enlargement and presumably, with increased variation in resource availability among individuals (Boonekamp et al., 2014). The weak effects of LPS make it unclear whether mediation of telomeres occurred via stress or growth-related pathways, although this may differ by tissue. Regardless, these findings allude to complex interactions in which changes to resource availability via stress can muddy the links between growth and telomeres.

5 | CONCLUSION

As apparent biomarkers of health and lifespan, telomeres are often quantified using noninvasive sampling in longitudinal research. This body of work has been vital for testing formative hypotheses about the role of telomeres in survival and ageing in the wild (Monaghan, 2014; Olsson et al., 2018), but the general prioritization of only some taxon-specific "tissues of choice" limits our knowledge of tissue-specific telomere dynamics that set the trajectory for later success in wild animals. In the last decade, a large body of work has uncovered inconsistent patterns relating growth and telomere dynamics (Monaghan & Ozanne, 2018), and it has become increasing clear that the "rules" of ageing differ among tissues (Tarry-Adkins et al., 2021). While this study cannot directly address tissue-specific patterns, our work nevertheless suggests that telomere dynamics may be influenced by early-life growth. Furthermore, this work hints at complex relationships among developmental timing, resource availability, and telomeres that should not be ignored in future research. We therefore encourage greater focus on growth-telomere relationships in multiple tissues that may play pivotal roles in responding to early life conditions and setting ageing trajectories.

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CONFLICT OF INTERESTS

The authors declare that there are no conflict of interests.

DATA AVAILABILITY STATEMENT

Data are available from the Dryad Digital Repository (https://doi.org/10.5061/dryad.0vt4b8h1d) (Wolf & Rosvall, 2021).

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REFERENCES

Allsopp, R. C., Chang, E., Kashefi-Aazam, M., Rogaev, E. I., Piatyszek, M. A., Shay, J. W., & Harley, C. B. (1995). Telomere shortening is associated with cell division in vitro and in vivo. *Experimental Cell Research*, *220*(1), 194–200.

Angelier, F., Costantini, D., Blevin, P., & Chastel, O. (2018). Do glucocorticoids mediate the link between environmental conditions and telomere dynamics in wild vertebrates? A review. *General and Comparative Endocrinology*, *256*, 99–111.

Arendt, J. D. (1997). Adaptive intrinsic growth rates: An integration across taxa. *The Quarterly Review of Biology*, *72*(2), 149–177.

Barker, D. J. (2007). The origins of the developmental origins theory. *Journal of Internal Medicine*, *261*(5), 412–417.

Barton, K. (2019). MuMIn: Multi-Model Inference. Retrieved from https://CRAN.R-project.org/package=MuMIn

Bennett, P. M., & Harvey, P. H. (1985). Brain size, development and metabolism in birds and mammals. *Journal of Zoology*, *207*(4), 491–509.

Blanckenhorn, W. U. (2000). The evolution of body size: What keeps organisms small? *The Quarterly Review of Biology*, *75*(4), 385–407.

Bonter, D. N., & Bridge, E. S. (2011). Applications of radio frequency identification (RFID) in ornithological research: A review. *Journal of Field Ornithology*, *82*(1), 1–10.

Boonekamp, J. J., Bauch, C., Mulder, E., & Verhulst, S. (2017). Does oxidative stress shorten telomeres? *Biology Letters*, *13*(5), 20170164.

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 B: Biological Sciences*, *281*(1785), 20133287.

Burnham, K., & Anderson, D. (2002). *Model selection and multimodal inference: a practical information-theoretic approach* (2nd ed.). Springer.

Burnham, K. P., Anderson, D. R., & Huyvaert, K. P. (2011). AIC model selection and multimodel inference in behavioral ecology: Some background, observations, and comparisons. *Behavioral Ecology and Sociobiology*, *65*(1), 23–35.

Burraco, P., Díaz-Paniagua, C., & Gomez-Mestre, I. (2017). Different effects of accelerated development and enhanced growth on oxidative stress and telomere shortening in amphibian larvae. *Scientific Reports*, *7*(1), 1–11.

Cattan, V., Mercier, N., Gardner, J. P., Regnault, V., Labat, C., Mäki-Jouppila, J., Nzietchueng, R., Benetos, A., Kimura, M., & Aviv, A. (2008). Chronic oxidative stress induces a tissue-specific reduction in telomere length in CAST/Ei mice. *Free Radical Biology and Medicine*, *44*(8), 1592–1598.

Cawthon, R. M. (2009). Telomere length measurement by a novel monochrome multiplex quantitative PCR method. *Nucleic Acids Research*, *37*(3), e21.

Chatelain, M., Drobniak, S. M., & Szulkin, M. (2020). The association between stressors and telomeres in non-human vertebrates: A meta-analysis. *Ecology Letters*, *23*(2), 381–398.

Cherif, H., Tarry, J., Ozanne, S., & Hales, C. (2003). Ageing and telomeres: A study into organ-and gender-specific telomere shortening. *Nucleic Acids Research*, *31*(5), 1576–1583.

Criscuolo, F., Bize, P., Nasir, L., Metcalfe, N. B., Foote, C. G., Griffiths, K., Gault, E. A., & Monaghan, P. (2009). Real-time quantitative PCR assay for measurement of avian telomeres. *Journal of Avian Biology*, *40*(3), 342–347.

Criscuolo, F., Monaghan, P., Nasir, L., & Metcalfe, N. B. (2008). Early nutrition and phenotypic development: 'catch-up' growth leads to elevated metabolic rate in adulthood. *Proceedings of the Royal Society B: Biological Sciences*, *275*(1642), 1565–1570.

Dantzer, R., O'Connor, J. C., Freund, G. G., Johnson, R. W., & Kelley, K. W. (2008). From inflammation to sickness and depression: When the immune system subjugates the brain. *Nature Reviews Neuroscience*, *9*(1), 46–56.

Dmitriew, C. M. (2011). The evolution of growth trajectories: What limits growth rate? *Biological Reviews*, *86*(1), 97–116.

Epel, E. (2012). How "reversible" is telomeric aging? *Cancer Prevention Research*, *5*(10), 1163–1168.

Fox, J., & Weisberg, S. (2011). *An {R} Companion to Applied Regression*. Sage

Geiger, S., Le Vaillant, M., Lebard, T., Reichert, S., Stier, A.,Y., L. E. M., & 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.

Grueber, C. E., Nakagawa, S., Laws, R. J., & Jamieson, I. G. (2011). Multimodel inference in ecology and evolution: Challenges and solutions. *Journal of Evolutionary Biology*, *24*(4), 699–711.

Hamel, S., Côté, S. D., Gaillard, J. M., & Festa-Bianchet, M. (2009). Individual variation in reproductive costs of reproduction: High-quality females always do better. *Journal of Animal Ecology*, *78*(1), 143–151.

Harrison, X. A., Donaldson, L., Correa-Cano, M. E., Evans, J., Fisher, D. N., Goodwin, C. E., Robinson, B. S., Hodgson, D. J., & Inger, R. (2018). A brief introduction to mixed effects modelling and multi-model inference in ecology. *PeerJ*, *6*, e4794.

Haussmann, M. F., Winkler, D. W., Huntington, C. E., Nisbet, I. C., & Vleck, C. M. (2007). Telomerase activity is maintained throughout the lifespan of long-lived birds. *Experimental Gerontology*, *42*(7), 610–618.

Haussmann, M. F., Winkler, D. W., O'Reilly, K. M., Huntington, C. E., Nisbet, I. C., & Vleck, C. M. (2003). Telomeres shorten more slowly in long-lived birds and mammals than in short-lived ones. *Proceedings of the Royal Society of London. Series B: Biological Sciences*, *270*(1522), 1387–1392.

Haywood, S., & Perrins, C. M. (1992). Is clutch size in birds affected by environmental conditions during growth? *Proceedings of the*

Royal Society of London. Series B: Biological Sciences, *249*(1325), 195–197.

Herborn, K. A., Heidinger, B. J., Boner, W., Noguera, J. C., Adam, A., Daunt, F., & Monaghan, P. (2014). Stress exposure in early post-natal life reduces telomere length: An experimental demonstration in a long-lived seabird. *Proceedings of the Royal Society B: Biological Sciences*, *281*(1782), 20133151.

Iyengar, S., & Pilo, B. (1981a). Glucose-6-phosphate dehydrogenase and NADP-linked malate dehydrogenase during posthatching development of brain of altricial birds. *Neurochemical Research*, *6*(1), 69–76.

Iyengar, S., & Pilo, B. (1981b). Monoamine oxidase in brain maturation: a histochemical study during posthatching development in altricial birds. *Journal fur Hirnforschung*, *22*(2), 195–204.

Jennings, B., Ozanne, S., Dorling, M., & Hales, C. (1999). Early growth determines longevity in male rats and may be related to telomere shortening in the kidney. *FEBS Letters*, *448*(1), 4–8.

Kesäniemi, J., Lavrinienko, A., Tukalenko, E., Boratyński, Z., Kivisaari, K., Mappes, T., Milinevsky, G., Møller, A. P., Mousseau, T. A., & Watts, P. C. (2019). Exposure to environmental radionuclides associates with tissue-specific impacts on telomerase expression and telomere length. *Scientific Reports*, *9*(1), 850.

Lee, W.-S., Monaghan, P., & Metcalfe, N. B. (2013). Experimental demonstration of the growth rate–lifespan trade-off. *Proceedings of the Royal Society B: Biological Sciences*, *280*(1752), 20122370.

Lendvai, A. Z., Akçay, Ç., Weiss, T., Haussmann, M. F., Moore, I. T., & Bonier, F. (2015). Low cost audiovisual playback and recording triggered by radio frequency identification using Raspberry Pi. *PeerJ*, *3*, e877.

Ludlow, A. T., Witkowski, S., Marshall, M. R., Wang, J., Lima, L. C., Guth, L. M., Spangenburg, E. E., & Roth, S. M. (2012). Chronic exercise modifies age-related telomere dynamics in a tissue-specific fashion. *Journals of Gerontology Series A: Biomedical Sciences and Medical Sciences*, *67*(9), 911–926.

Magrath, R. D. (1991). Nestling weight and juvenile survival in the blackbird, Turdus merula. *The Journal of Animal Ecology*, *60*, 335–351.

McCarty, J. P. (2001). Variation in growth of nestling tree swallows across multiple temporal and spatial scales. *The Auk*, *118*(1), 176–190.

McCarty, J. P. (2002). The number of visits to the nest by parents is an accurate measure of food delivered to nestlings in tree swallows. *Journal of Field Ornithology*, *73*(1), 9–14.

McLennan, D., Armstrong, J., Stewart, D., Mckelvey, S., Boner, W., Monaghan, P., & Metcalfe, N. (2016). Interactions between parental traits, environmental harshness and growth rate in determining telomere length in wild juvenile salmon. *Molecular Ecology*, *25*(21), 5425–5438.

Metcalfe, N. B., & Monaghan, P. (2003). Growth versus lifespan: Perspectives from evolutionary ecology. *Experimental Gerontology*, *38*(9), 935–940.

Monaghan, P. (2014). Organismal stress, telomeres and life histories. *Journal of Experimental Biology*, *217*(1), 57–66.

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.

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 B: Biological Sciences*, *282*(1798), 20141610.

Van Noordwijk, A. J., & de Jong, G. (1986). Acquisition and allocation of resources: Their influence on variation in life history tactics. *The American Naturalist*, *128*(1), 137–142.

Nowicki, S., Searcy, W., & Peters, S. (2002). Brain development, song learning and mate choice in birds: A review and experimental test of the"nutritional stress hypothesis". *Journal of Comparative Physiology A*, *188*(11-12), 1003–1014.

Olsson, M., Wapstra, E., & Friesen, C. R. (2018). Evolutionary ecology of telomeres: A review. *Annals of the New York Academy of Sciences*, *1422*(1), 5–28.

Owen-Ashley, N. T., Turner, M., Hahn, T. P., & Wingfield, J. C. (2006). Hormonal, behavioral, and thermoregulatory responses to bacterial lipopolysaccharide in captive and free-living white-crowned sparrows (*Zonotrichia leucophrys gambelii*). *Hormones and Behavior*, *49*(1), 15–29.

Owen-Ashley, N. T., & Wingfield, J. C. (2006). Seasonal modulation of sickness behavior in free-living northwestern song sparrows (*Melospiza melodia morphna*). *Journal of Experimental Biology*, *209*(16), 3062–3070.

Palacios, M. G., Winkler, D. W., Klasing, K. C., Hasselquist, D., & Vleck, C. M. (2011). Consequences of immune system aging in nature: A study of immunosenescence costs in free-living Tree Swallows. *Ecology*, *92*(4), 952–966.

Pilo, B., & Iyengar, S. (1981). Changes in lipid content during the post-hatching development of the brain of altricial birds. *The Histochemical Journal*, *13*(3), 453–460.

Power, M. L., Power, S., Bertelsen, M. F., Jones, G., & Teeling, E. C. (2021). Wing: A suitable nonlethal tissue type for repeatable and rapid telomere length estimates in bats. *Molecular Ecology Resources*, *21*(2), 421–432.

Prowse, K. R., & Greider, C. W. (1995). Developmental and tissue-specific regulation of mouse telomerase and telomere length. *Proceedings of the National Academy of Sciences*, *92*(11), 4818–4822.

Reichert, S., Criscuolo, F., Verinaud, E., Zahn, S., & Massemin, S. (2013). Telomere length correlations among somatic tissues in adult zebra finches. *PLoS One*, *8*(12), e81496.

Reichert, S., & Stier, A. (2017). Does oxidative stress shorten telomeres in vivo? A review. *Biology Letters*, *13*(12), 20170463.

Rensel, M. A., Boughton, R. K., & Schoech, S. J. (2010). Development of the adrenal stress response in the Florida scrub-jay (Aphelocoma coerulescens). *General and Comparative Endocrinology*, *165*(2), 255–261.

Ricklefs, R. E. (1979). Adaptation, constraint, and compromise in avian postnatal development. *Biological Reviews*, *54*(3), 269–290.

Ricklefs, R. E. (1983). Avian postnatal development. *Avian biology*, *7*, 1–83.

Ricklefs, R. E. (2003). Is rate of ontogenetic growth constrained by resource supply or tissue growth potential? A comment on West et al.'s model. *Functional Ecology*, *17*(3), 384–393.

Rollings, N., Friesen, C. R., Whittington, C. M., Johansson, R., Shine, R., & Olsson, M. (2019). Sex-And tissue-specific differences in telomere length in a reptile. *Ecology and Evolution*, *9*(11), 6211–6219.

Rollo, C. D. (2002). Growth negatively impacts the life span of mammals. *Evolution & Development*, *4*(1), 55–61.

RStudio Team (2019). *RStudio: Integrated Development for R*. RStudio, PBC. http://www.rstudio.com/

Salmón, P., Millet, C., Selman, C., & Monaghan, P. (2021). Growth acceleration results in faster telomere shortening later in life. *Proceedings of the Royal Society B*, *288*(1956), 20211118.

Schmidt, J. E., Sirman, A. E., Kittilson, J. D., Clark, M. E., Reed, W. L., & Heidinger, B. J. (2016). Telomere correlations during early life in a long-lived seabird. *Experimental Gerontology*, *85*, 28–32.

Smith, S. M., Nager, R. G., & Costantini, D. (2016). Meta-analysis indicates that oxidative stress is both a constraint on and a cost of growth. *Ecology and Evolution*, *6*(9), 2833–2842.

Stearns, S. C. (1992). *The evolution of life histories* (249). Oxford University Press.

Stier, A., Delestrade, A., Bize, P., Zahn, S., Criscuolo, F., & Massemin, S. (2016). Investigating how telomere dynamics, growth and life history covary along an elevation gradient in two passerine species. *Journal of Avian Biology*, *47*(1), 134–140.

Stier, A., Delestrade, A., Zahn, S., Arrivé, M., Criscuolo, F., & Massemin-Challet, S. (2014). Elevation impacts the balance between growth and oxidative stress in coal tits. *Oecologia*, *175*(3), 791–800.

Tarry-Adkins, J. L., Aiken, C. E., Dearden, L., Fernandez-Twinn, D. S., & Ozanne, S. (2021). Exploring telomere dynamics in aging male rat tissues: Can tissue-specific differences contribute to age-associated pathologies? *Gerontology*, *67*(2), 233–242.

Tilgar, V., Saag, P., & Moks, K. (2009). Development of stress response in nestling pied flycatchers. *Journal of Comparative Physiology A*, *195*(8), 799–803.

Tricola, G. M., Simons, M. J. P., Atema, E., Boughton, R. K., Brown, J. L., Dearborn, D. C., Divoky, G., Eimes, J. A., Huntington, C. E., Kitaysky, A. S., Juola, F. A., Lank, D. B., Litwa, H. P., Mulder, E. G. A., Nisbet, I. C. T., Okanoya, K., Safran, R. J., Schoech, S. J., Schreiber, E. A.,, … Haussmann, M. F. (2018). The rate of telomere loss is related to maximum lifespan in birds. *Philosophical Transactions of the Royal Society, B: Biological Sciences*, *373*(1741), 20160445.

Ulaner, G. A., Hu, J. F., Vu, T. H., Giudice, L. C., & Hoffman, A. R. (2001). Tissue-specific alternate splicing of human telomerase reverse transcriptase (hTERT) influences telomere lengths during human development. *International Journal of Cancer*, *91*(5), 644–649.

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.

Verhulst, S., Aviv, A., Benetos, A., Berenson, G. S., & Kark, J. D. (2013). Do leukocyte telomere length dynamics depend on baseline telomere length? An analysis that corrects for 'regression to the mean'. *European Journal of Epidemiology*, *28*(11), 859–866.

Wada, H. (2008). Glucocorticoids: Mediators of vertebrate ontogenetic transitions. *General and Comparative Endocrinology*, *156*(3), 441–453.

Wada, H., Hahn, T. P., & Breuner, C. W. (2007). Development of stress reactivity in white-crowned sparrow nestlings: Total corticosterone response increases with age, while free corticosterone response remains low. *General and Comparative Endocrinology*, *150*(3), 405–413.

Waddington, C. H. (1942). Canalization of development and the inheritance of acquired characters. *Nature*, *150*(3811), 563–565.

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.

Williams, G. C. (1966). Natural selection, the costs of reproduction, and a refinement of Lack's principle. *The American Naturalist*, *100*(916), 687–690.

Wolf, S. E., & Rosvall, K. A. (2021). Data from: A multi-tissue view on telomere dynamics and postnatal growth. *Dryad Digital Repository*. https://doi.org/10.5061/dryad.0vt4b8h1d

Wolf, S. E., Sanders, T. L., Beltran, S. E., & Rosvall, K. A. (2021a). The telomere regulatory gene POT1 responds to stress and predicts performance in nature: Implications for telomeres and life history evolution. *Molecular Ecology*, *n/a(n/a)*, *9*, 1.

Wolf, S. E., Stansberry, K. R., Content, K. R., & Rosvall, K. A. (2021b). A putative telomerase activator has tissue-specific effects on telomere length in a developing songbird. *Journal of Avian Biology*, *52*(2), 216.

Zakian, V. A. (2012). Telomeres: The beginnings and ends of eukaryotic chromosomes. *Experimental Cell Research*, *318*(12), 1456–1460.

von Zglinicki, T. (2002). Oxidative stress shortens telomeres. *Trends in Biochemical Sciences*, *27*(7), 339–344.

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