BIOLOGY
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Cite this article: Brown AM, Wood EM, Capilla-Lasheras P, Harrison XA, Young AJ. 2021 Longitudinal evidence that older parents produce offspring with longer telomeres in a wild social bird. *Biol. Lett.* **17**: 20210409. https://doi.org/10.1098/rsbl.2021.0409

Received: 2 August 2021
Accepted: 29 September 2021

Subject Areas:
evolution, ecology, molecular biology

Keywords:
parental age effects, telomere attrition, maternal effects, transgenerational effects, plasticity, senescence

Authors for correspondence:
Antony M. Brown
e-mail: antonymbrown1@gmail.com
Andrew J. Young
e-mail: a.j.young@exeter.ac.uk

[†]Present address: Institute of Biodiversity, Animal Health and Comparative Medicine, University of Glasgow, Glasgow, UK.

Electronic supplementary material is available online at https://doi.org/10.6084/m9.figshare.c.5658529.

THE ROYAL SOCIETY
PUBLISHING

Evolutionary biology

Longitudinal evidence that older parents produce offspring with longer telomeres in a wild social bird

Antony M. Brown, Emma M. Wood, Pablo Capilla-Lasheras[†],
Xavier A. Harrison and Andrew J. Young

Centre for Ecology and Conservation, College of Life and Environmental Sciences, University of Exeter, Penryn Campus, Penryn, Cornwall TR10 9FE, UK

AMB, 0000-0001-7514-5771; EMW, 0000-0002-3609-4817; PC-L, 0000-0001-6091-7089; XAH, 0000-0002-2004-3601; AJY, 0000-0003-0560-6549

As telomere length (TL) often predicts survival and lifespan, there is considerable interest in the origins of inter-individual variation in TL. Cross-generational effects of parental age on offspring TL are thought to be a key source of variation, but the rarity of longitudinal studies that examine the telomeres of successive offspring born throughout the lives of parents leaves such effects poorly understood. Here, we exploit TL measures of successive offspring produced throughout the long breeding tenures of parents in wild white-browed sparrow weaver (*Plocepasser mahali*) societies, to isolate the effects of within-parent changes in age on offspring TLs. Our analyses reveal the first evidence to date of a positive within-parent effect of advancing age on offspring TL: as individual parents age, they produce offspring with longer telomeres (a modest effect that persists into offspring adulthood). We consider the potential for pre- and post-natal mechanisms to explain our findings. As telomere attrition predicts offspring survival to adulthood in this species, this positive parental age effect could impact parent and offspring fitness if it arose via differential telomere attrition during offspring development. Our findings support the view that cross-generational effects of parental age can be a source of inter-individual variation in TL.

1. Introduction

Evidence that individual variation in mean telomere length (TL) can predict components of fitness has led to a surge of interest in the origins of variation in TL [1–3]. Telomeres are nucleoprotein complexes at the ends of eukaryotic chromosomes [4]. They consist of a repeating non-coding DNA sequence ($TTAGGG_n$ in vertebrates) and have an important function in maintaining chromosomal integrity [1,2,4]. Telomeres vary in length both within and among individuals, and average TLs shorten with advancing age in many taxa [1,2]. Telomeres can be shortened as a consequence of cell division and exposure to oxidative stress [3,5], although the importance of oxidative stress as a driver of telomere erosion *in vivo* remains unclear [6]. As critically short telomeres can trigger cellular senescence, excessive telomere shortening may have negative effects on performance [1,2,4,7–10]. Indeed, TL positively predicts survival and lifespan in many taxa (e.g. [7–9]) and is often considered a biomarker of somatic integrity [2]. Consequently, there is considerable interest in identifying the origins of inter-individual variation in TL within populations. There is mounting evidence that parental age at offspring conception (hereafter ‘parental age’) predicts offspring TL in both human and non-human animals (e.g. [11–13]). However, the vast majority of studies to date have considered only whether population-level variation in parental age predicts offspring TL (see below). Whether such patterns reflect within-parent effects of advancing age rather than effects arising from the selective disappearance of certain types of parents with advancing age [14–17] remains poorly understood.

In humans, population-level studies have consistently demonstrated that paternal (but not maternal) age at conception positively predicts offspring TL (e.g. [11,18,19]). In non-human animals, however, there is considerable variation among species in the nature of the population-level relationship between parental age and offspring TL. Studies in mammals, birds and reptiles have to date found positive [20–22], negative [12,13,23] and no [24,25] relationship, with some finding evidence suggestive of either maternal [20,22] or paternal age effects [13,21,23], or both [12]. In order to understand the mechanistic origins and life-history implications of these relationships, studies now need to establish whether these patterns reflect within-parent effects of advancing age on the phenotypes of their offspring, or arise instead from among-parent processes (e.g. selective disappearance). Just three studies to date have yielded evidence that within-parent changes in age predict offspring TL. First, a study of captive zebra finches (*Taeniopygia guttata*) demonstrated that as individual mothers age they produce offspring with shorter telomeres [26]. Similarly, recent studies of wild jackdaws (*Corvus monedula*) [16] and Seychelles warblers (*Acrocephalus sechellensis*) [17] found that as individual fathers age they produce offspring with shorter telomeres, while no maternal age effects were apparent. These longitudinal studies therefore suggest that advancing parental age can *negatively* impact offspring TL.

Here, we investigate parental age effects on offspring TL in a free-living population of the cooperatively breeding white-browed sparrow weaver (*Plocepasser mahali*). We use measures of mean blood cell relative TL (RTL) from successive offspring produced throughout the long breeding tenures of parents to isolate the effects of within-parent changes in age. White-browed sparrow weavers live in year-round territorial groups, comprising a single dominant male and female pair that monopolizes within-group reproduction (i.e. the ‘parents’), and 0–12 non-breeding subordinate ‘helpers’ that assist with nestling feeding and are typically closely related to the dominant pair [27–30]. As the rate of telomere attrition during the nestling period predicts survival to adulthood in this species [10], parental age effects on offspring RTL could impact parent and offspring fitness if they arise via differential telomere attrition during development.

2. Material and methods

(a) Study species and field methods

Approximately 40 groups of white-browed sparrow weavers have been monitored intensively since 2007 in Tswalu Kalahari Reserve (South Africa; 27°16′ S, 22°25′ E), so that most individuals have known life-history (see [27–29]). Each group's dominant breeding pair were identified using characteristic behavioural profiles and were considered the parents of all offspring born in their group during their reproductive tenures (see electronic supplementary material S1.1 for justification, and S1.5 and S3.1 for supporting analysis using genetic data). Nests were checked regularly throughout each breeding season (October to April inclusive) to determine egg lay and hatch dates, which allowed estimation of parental age (parent's age in days on the average date that the focal offspring's clutch was laid; see electronic supplementary material, S1.2) and offspring age at sampling (days since their hatch date). Blood samples for telomere assessments were routinely taken from nestlings and adult birds (see electronic supplementary material, S1.3). As helpers contribute to nestling feeding when present [27,28], we verified that our findings were unaffected by controlling for variation in an offspring's rearing group size (see electronic supplementary material, S3.2).

(b) Telomere length measurements

TLs were estimated from whole blood samples using qPCR analysis of RTL (following [31]). Full methods and quality control details can be found in electronic supplementary material, S1.4 (see also [10,32]). We analysed 765 blood samples from 356 offspring from 248 clutches, produced by 61 mothers and 60 fathers in 41 social groups between 2010 and 2015 (numbers of mothers and fathers exceed the number of groups owing to dominance turnovers during the study).

(c) Statistical analyses

We fitted linear mixed-effects models with the lme4 package [33] in R (v. 3.6.1) [34] and ranked models by corrected Akaike information criterion (AICc) (see electronic supplementary material, S1.6). We z-transformed RTL for analysis, to facilitate comparison with other studies [35]. The following fixed-effect predictors were fitted alongside parental ages in our models: sample storage duration prior to DNA extraction (accurate to the day for each sample), offspring sex and offspring age class (less than 11 days (post-hatching), 11–89 days (later dependence) and greater than or equal to 90 days (independence & adulthood)). Mean ± s.e. offspring age at sampling = 1.40 years ± 24.3 days (range 3 days–7.35 years). These offspring age classes were chosen to identify whether any parental age effects were present in offspring soon after hatching or arose later in offspring development (we have found evidence of modest telomere attrition (on average) within nestlings [36], but have found no evidence of telomere attrition (on average) over the long term within adulthood, despite extensive longitudinal sampling [32]). Interactions between each parental age variable and offspring age class were also included. Offspring ID, clutch ID nested within mother ID, qPCR plate ID and sampling period (a factor identifying the breeding season and calendar year in which the blood sample was taken) were fitted as random intercepts. We did not fit father ID as it typically estimated zero or near-zero variance when alongside mother ID, likely owing to its strong correlation with mother ID (dominant pairs can produce many clutches together); our findings do not change if it is included. While global models contained both maternal and paternal age as fixed effects, models containing both were not AICc-ranked because maternal and paternal age were correlated (Pearson's $r = 0.76$, $t = 31.97$, d.f. = 763, $p < 0.001$).

In order to isolate the effects of within-parent changes in age on offspring RTL, we used within-subject centring [15] to partition the parental age variables into two components and fitted both components as fixed effects: (i) the parent's ‘mean age’ across all sampled clutches for that parent, and (ii) the parent's ‘Δ age’, the difference between the parental age value for the focal clutch and the parent's mean age. The Δ age variables allow the models to estimate the effect of within-parent changes in age on offspring RTL, while the mean age variables estimate among-parent effects. As parents first encountered as fledglings or adults were assigned a minimum age (see electronic supplementary material, S1.2), any parental age relationships detected in the population-level analysis should be interpreted with caution. However, following within-subject centring, the parental Δ age values will be accurate for all parents.

3. Results

Analysis of the predictors of offspring RTL yielded evidence of a positive *within-parent* effect of advancing parental age

Table 1. Top models within 6 AICc points of the best-supported model after implementation of the nesting rule [37] for the *within-parent* analysis of z transformed offspring RTL. Estimates are given with standard errors (s.e.) in parentheses and 95% CI. For the top model, marginal-$R^2 = 0.117$ and conditional-$R^2 = 0.651$. Predictors absent from top model set: maternal mean age, offspring sex, interactions between maternal age terms and offspring age class. Paternal Δ age effect estimates in top-ranked model: for offspring age less than 11 days = −0.087 (s.e. = 0.053, CI: −0.191, 0.018); 11–89 days = 0.013 (s.e. = 0.048, CI: −0.082, 0.109); greater than or equal to 90 days = 0.096 (s.e. = 0.040, CI: 0.016, 0.175). See electronic supplementary material S2, table S1 for random effect estimates from the top-ranked model.

intercept	offspring age class	paternal Δ age	paternal mean age	maternal Δ age	sample storage duration	offspring age class: paternal Δ age	offspring age class: paternal mean age	d.f.	logLik	AICc	ΔAICc	adjusted Akaike weight
+	+	+	+		−0.198 (0.047) CI: −0.299, −0.103	+	+	16	−861.63	1756.0	0.00	0.385
0.295 (0.162) CI: −0.031, 0.638				0.065 (0.029) CI: 0.007, 0.123	−0.126 (0.046) CI: −0.227, −0.031			9	−869.23	1756.7	0.70	0.271
0.309 (0.162) CI: −0.019, 0.651		0.063 (0.031) CI: 0.000, 0.125			−0.135 (0.046) CI: −0.234, −0.040			9	−869.69	1757.6	1.62	0.171
+	+				−0.161 (0.048) CI: −0.266, −0.063			10	−869.52	1759.3	3.35	0.072
0.349 (0.162) CI: 0.021, 0.692					−0.153 (0.045) CI: −0.252, −0.060			8	−871.61	1759.4	3.43	0.069
−0.027 (0.122) CI: −0.274, 0.217				0.080 (0.029) CI: 0.022, 0.137				8	−872.41	1761.0	5.02	0.031

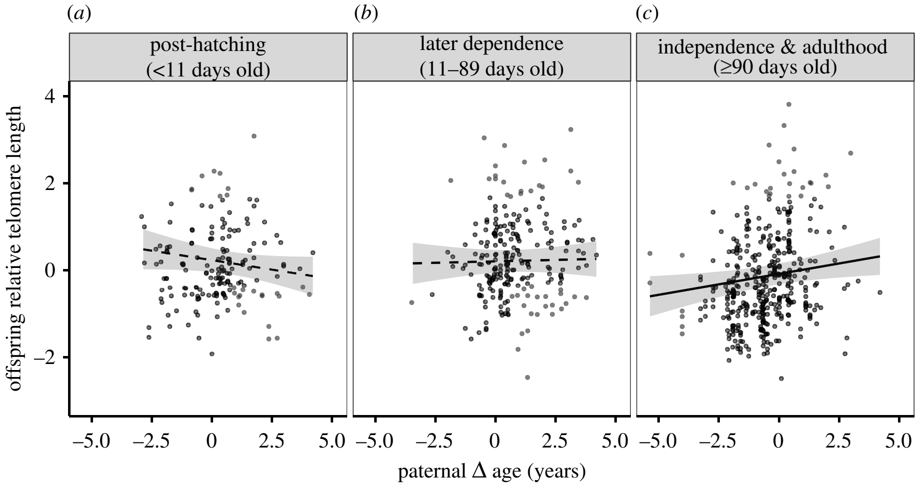

Figure 1. Relationship between paternal Δ age and offspring RTL (*z*-transformed) as captured by the top-ranked model following within-parent centring (table 1), which contained an interaction between paternal Δ age and offspring age class (whose three factor levels are depicted in the three figure panels). Lines represent model predictions from the top-ranked model (solid if the paternal Δ age effect size estimate for the focal offspring age class was greater than 1.96 × s.e.; dashed if not). Shaded areas represent the upper and lower CIs (1.96 × s.e.). Paternal Δ age effect sizes ± s.e.: (*a*) post-hatching = −0.087 ± 0.053 (*n* = 164 samples from 164 offspring); (*b*) later dependence = 0.013 ± 0.048 (*n* = 225 samples from 169 offspring); and (*c*) independence & adulthood = 0.096 ± 0.040 (*n* = 376 samples from 165 offspring).

(table 1); as individual parents get older, they produce offspring with longer telomeres. The top model contained an interaction between paternal Δ age and offspring age class, in which the effect size for paternal Δ age increased as offspring proceeded through development (figure 1 and table 1), only becoming non-zero (as judged by the confidence interval (CI) not spanning zero) during offspring 'independence & adulthood' (when a positive paternal age effect was apparent; figure 1). This interaction attracted only modest support, being absent from the second-best model just 0.70 AICc points below. The second-best model (ΔAICc = 0.70) contained a simple positive effect of maternal Δ age (i.e. with no offspring age interaction), while the third-best model (ΔAICc = 1.62) contained a simple positive effect of paternal Δ age (table 1). Overall, positive *within-parent* effects of advancing parental age were present within all top models above ΔAICc = 3.35 (table 1). The analysis yielded comparable support for positive effects of maternal and paternal Δ age (table 1), indicating that the observed parental age effect could be driven by effects of maternal or paternal age or both. The parental age effect was only a modest source of (inter-individual) variation in offspring RTL: in combination, the fixed effects in our top model for the within-parent analysis explained 11.7% of the total variation in offspring RTL, while the entire top model (including the random effects) explained 65.1% of the total variation in offspring RTL (table 1). Additional analyses suggest that these parental age relationships cannot be attributed to correlated variation in social group size (and hence helper number) or uncertainty regarding parental age (see electronic supplementary material, S3.1 and S3.2), and that they persist into offspring adulthood (see electronic supplementary material, S3.3). These parental age relationships were also apparent when repeating the analysis using a full model approach (rather than a model selection approach; see electronic supplementary material, S3.4). Repeating the entire analysis without the use of within-parent centring to isolate within-parent effects revealed that similar parental age relationships are apparent at the population level (see electronic supplementary material, S3.5).

4. Discussion

Our analyses reveal that within-parent changes in parental age positively predict offspring TL, a relationship that cannot be readily attributed to confounding effects of variation among parents. A within-parent effect of parental age on offspring TL has only been reported in three other studies (also in birds), all of which reported a negative effect [16,17,26]. Our findings therefore constitute the first evidence to our knowledge of a positive within-parent effect of advancing parental age on offspring TL. The modest positive effect reported here could arise via effects of maternal age, paternal age or both acting in concert.

Parental age effects could arise via both pre-natal mechanisms (e.g. gamete-mediated epigenetic effects) and post-natal mechanisms (including age-related changes in post-natal care), as has been underscored by evidence of biological parent and foster parent age effects on offspring TL [12,16]. Our top model following within-parent centring yielded some statistical support for an interactive effect of paternal Δ age and offspring age class on offspring TL (table 1 and figure 1), in which the positive effect of paternal Δ age was not apparent post-hatching, and only became apparent within the oldest offspring age class (greater than or equal to 90 days of age; figure 1). While statistical support for the offspring age class interaction is not strong, the form of the interaction is nevertheless more consistent with a role for post-natal than pre-natal mechanisms [38]. Several plausible post-natal mechanisms exist. For example, the quality of post-natal parental care could conceivably increase as parents age (e.g. via effects of breeding experience [39]), leading to slower rates of telomere attrition among nestlings [40]. While negative parental age effects (e.g. [12,16]) might be expected during parental senescence, the senescent period may not be well represented within our data as sampled mothers were not known to exceed 8 years of age and the species can breed beyond 12 years of age [10] (i.e. parental age effects over the entire maximum reproductive lifespan have not yet been explored).

The positive parental age effect detected here could conceivably be related to our study species' cooperative breeding system, as sparrow weaver helpers appear to lighten the nestling-feeding workloads of parents and reduce associated oxidative stress [29,41,42], processes that could temper age-related deterioration in parental performance [32,43] (indeed, we find no evidence of long-term age-related declines in the mean TLs of adults in this population; [32]). Under such circumstances, any beneficial effects of accrued breeding experience (with parental age; see above) might be more likely to yield an overall net positive effect of parental age on offspring TL.

Offspring produced by older parents often have reduced fitness and lifespan (the 'Lansing effect' [44,45]). By contrast, our finding of a positive effect of parental age on offspring TL highlights the possibility in this species of parental age-related increases in offspring performance, given that offspring TL often positively predicts their downstream performance [10]. Indeed, recent work on another social vertebrate has yielded striking evidence of a positive maternal age effect on offspring performance [46]. These apparent benefits of advanced parental age highlight the wider need for analyses of the fitness implications of reproductive senescence to consider parental age-related changes in offspring quality as well as offspring production, given the potential for the former to partially offset the latter. Indeed, if the parental age effect reported here did reflect a parental age-related increase in offspring quality, such a relationship might strengthen selection for investment in dominance retention and survival once dominant, helping to explain the long dominance tenures (up to 11.8 years) and maximum lifespan (greater than 12 years in both sexes; [32]) of our study species.

To conclude, we present the first evidence to our knowledge that within-parent changes in age positively predict offspring TL. Our findings, coupled with those of other recent studies [16,17,26], suggest that parental age at reproduction can indeed contribute to inter-individual variation in TL in animal populations and that these effects can persist into adulthood. Work is now needed to elucidate the mechanisms driving these parental age effects and to establish the causes of their variation across taxa.

Ethics. The protocols followed in this study were approved by University of Pretoria Animal Ethics Committee (EC023-07 and EC100-12).

Data accessibility. Data are available on Dryad: https://doi.org/10.5061/dryad.2jm63xsng [47].

Authors' contributions. A.M.B., E.M.W. and A.J.Y. designed the study. E.M.W. conducted the fieldwork and performed the TL analyses. A.J.Y. led the long-term field study. A.M.B., P.C.-L. and X.A.H. conducted the microsatellite genotyping analyses. A.M.B. analysed the data and wrote the manuscript. All authors commented on the manuscript and approved the final version.

Competing interests. We declare no competing interests.

Funding. This work was supported by the Biotechnology and Biological Sciences Research Council. A.J.Y. and the long-term field study were funded by a BBSRC David Phillips Research Fellowship (BB/H022716/1) to A.J.Y. A.M.B., E.M.W. and P.C.-L. were supported by the BBSRC South West Biosciences Doctoral Training Partnership (training grant references: A.M.B. BB/M009122/1; E.M.W. BB/J014400/1; P.C.L. BB/M009122/1).

Acknowledgements. We thank the many sparrow weaver team members for their contributions to the collection of our long-term life-history data, Nigel Bennett for logistical support, Northern Cape Conservation for permission to carry out the research, and E. Oppenheimer & Son, the Tswalu Foundation, and all at Tswalu Kalahari Reserve for their support in the field. We also thank the four anonymous reviewers for their insightful contributions, and Joshua Lynton-Jenkins for helpful discussion during manuscript revisions.

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