Received: 2 October 2020 | Revised: 27 February 2021 | Accepted: 22 March 2021

DOI: 10.1111/mec.15905

SPECIAL ISSUE

MOLECULAR ECOLOGY WILEY

Parental age does not influence offspring telomeres during early life in common gulls (*Larus canus*)

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Funding information
H2020 European Institute of Innovation and Technology, Grant/Award Number: 701747; Estonian Research Council, Grant/Award Number: IUT21-1, IUT34-8, PSG458 and PSG653; European Regional Development Fund, Grant/Award Number: MOBJD344

Abstract

Parental age can affect offspring telomere length through heritable and epigenetic-like effects, but at what stage during development these effects are established is not well known. To address this, we conducted a cross-fostering experiment in common gulls (*Larus canus*) that enabled us distinguish between pre- and post-natal parental age effects on offspring telomere length. Whole clutches were exchanged after clutch completion within and between parental age classes (young and old) and blood samples were collected from chicks at hatching and during the fastest growth phase (11 days later) to measure telomeres. Neither the ages of the natal nor the foster parents predicted the telomere length or the change in telomere lengths of their chicks. Telomere length (TL) was repeatable within chicks, but increased across development (repeatability = 0.55, intraclass correlation coefficient within sampling events 0.934). Telomere length and the change in telomere length were not predicted by post-natal growth rate. Taken together, these findings suggest that in common gulls, telomere length during early life is not influenced by parental age or growth rate, which may indicate that protective mechanisms buffer telomeres from external conditions during development in this relatively long-lived species.

KEYWORDS
ageing, maternal effects, qPCR, seabirds, telomere dynamics, telomere length

1 | INTRODUCTION

Parental age often has long-term effects on offspring phenotype in humans (Carslake et al., 2019) and wild animals (Bowers et al., 2017). In some cases, increased parental age has positive long-term effects on offspring (Bradley & Safran, 2014), possibly because older parents are more experienced breeders than younger parents and/or because reproductive investment increases with age as future reproductive opportunities decline (e.g., terminal investment) (Clutton-Brock, 1984). Alternatively, parental age may have negative long-term effects on offspring if older parents experience senescent declines in condition and/or reproductive function relative

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to younger parents (Bock et al., 2019; Maklakov & Chapman, 2019; Velarde & Menon, 2016). Regardless, the mechanisms that underlie these long-term effects of parental age on offspring phenotype are not well understood, although several candidate mechanisms have been proposed, including epigenetic factors (Bock et al., 2019).

One mechanism that may mediate cross-generational effects of parental age on offspring fitness are telomeres, highly conserved, repetitive, non-coding sequences of DNA that form protective caps at chromosome ends that enhance genome stability (Blackburn, 1991). Some evidence suggests that telomere length (TL) is largely inherited (Horn et al., 2011; Njajou et al., 2007; Nordfjäll et al., 2005; Olsson et al., 2011; Reichert et al., 2015), but TL can also be influenced by a complex interaction of genetic effects and environmental factors during an organism's life (Dugdale & Richardson, 2018; Monaghan, 2010). Telomeres typically shorten with age in somatic cells, but telomere elongation has also been described, and is thought to mainly be due to telomerase, an enzyme that can extend telomeres via the addition of terminal telomeric repeats (Cong et al., 2002). Telomerase is variably active in different cell types and at different life stages, and in different taxonomic groups (Gomes et al., 2011). The effect of parental age on the length of offspring telomeres is currently intensively studied (Bauch et al., 2019; Criscuolo et al., 2017; Froy et al., 2017; Heidinger et al., 2016; Noguera & Velando, 2020). The costs and benefits associated with maintaining the length of telomeres are particularly interesting when considering the adaptive role of telomeres in the evolution of life histories, as it is suggested that telomeres could play a proximate causal role in current–future life-history trade-offs (Young, 2018). Optimal life-history strategies are both inherited and shaped by environmental effects (Stearns, 1992), and accordingly, telomere dynamics are a plausible physiological mechanisms that may be related to variation in life-history strategies (Giraudeau et al., 2019).

Although an increasing number of studies have reported parental age effects on offspring telomeres, when during offspring development these effects are occuring is still unknown. Parental age could impact offspring telomeres at many different stages (Haussmann & Heidinger, 2015; Heidinger & Young, 2020). First, age-associated changes in parental gametes may directly affect the TL that offspring inherit. Parental age may negatively influence offspring TL if older parents produce gametes with shorter telomeres, as has been reported in mice (*Mus musculus*) (de Frutos et al., 2016). However, in humans the opposite pattern has been reported, as older fathers produce offspring with longer telomeres (Broer et al., 2013; Unryn et al., 2005), possibly due to active telomerase in sperm cells (Kimura et al., 2008). Such inconsistences among studies, and among species, could stem from differences in life-history strategies, probably via mechanisms related to spermatogonial stem cell telomere retention with increasing age, or selective attrition/survival of spermatogonia (Eisenberg & Kuzawa, 2018; Kimura et al., 2008). Studies in humans have mostly found a link between paternal age and offspring TL (Broer et al., 2013; Unryn et al., 2005); however, the effect of maternal age has been shown to be even stronger than paternal age for some species (e.g., great reed warbler (*Acrocephalus arundinaceus*) Asghar, Bensch, et al., 2015). While maternal reproductive cells develop at a very early age, after which they are retained throughout life without further cell divisions, associations between mother age and offspring TL could be explained by other mechanisms, for example age- and/or condition-dependent telomerase activity in the ovaries (Asghar, Bensch, et al., 2015; Kinugawa et al., 2000).

Parental age may also influence offspring telomeres through prenatal effects, for example if there are age-associated changes in the amounts of glucocorticoid or androgen hormones transferred from the mother to the developing embryo with increasing age of the mother (Haussmann & Heidinger, 2015; Heidinger & Young, 2020; Stier et al., 2019). This could in turn activate the production of reactive oxygen species (ROS), as well as decrease telomerase activity in the offspring (Haussmann & Heidinger, 2015), potentially leading to telomere erosion. Although there are now a number of studies linking prenatal stress exposure to offspring telomeres (reviewed for example, in Dantzer et al., 2020; Haussmann & Heidinger, 2015, Heidinger & Young, 2020), these studies have not considered the potential effects of parental age. Parental age may also influence offspring telomeres during post-natal development, as age-related variation in parental care and the characteristics of the post-birth environment could also influence offspring telomeres (Tarry-Adkins et al., 2009). For example, more experienced parents may provide better care (Beamonte-Barrientos et al., 2010), or older parents may become less capable of providing a high quality environment due to senescence effects (Torres et al.,2011). The quality of parental care during the growth phase may hasten or reduce telomere shortening (Criscuolo et al., 2017) with long-lasting effects on the aging rate and life-history trajectories of the offspring (Young, 2018).

Distinguishing when parental age influences offspring telomeres is difficult in natural populations, however this information is critical for understanding how these effects occur and what the fitness consequences are likely to be for parents and offspring (Dugdale & Richardson, 2018). Birds are a promising model system for addressing this question because embryonic development takes place within a sealed system, the egg, which can be cross-fostered among nests. This makes it possible to determine to what degree the effects of parental age on offspring telomeres are due to epigenetic like changes to parental gamete telomeres and/or pre-natal effects that would be influenced by the age of the natal parents or due to changes in offspring TL during post-natal development that would be influenced by the age of the foster parents. Few studies have used a cross-fostering approach to separate these effects. In jackdaws (*Corvus monedula*), there was a negative effect of the age of the natal father on offspring telomeres (Bauch et al., 2019), suggesting that in this species, the effects of parental age on offspring TL is due to epigenetic like changes to the telomeres that offspring inherit. Whereas in alpine swifts (*Apus melba*), offspring TL was negatively related to both the age of the natal father and the age of the foster mother (Criscuolo et al., 2017), suggesting that in this species, the effects of parental age on offspring telomeres are probably due to both epigenetic like changes to the telomeres that offspring inherit as well as differences in parental care during post-natal development. However,

neither of these studies took repeated telomere measures during chick growth. Measuring chicks repeatedly during development makes it possible to directly assess changes in TL during growth and to differentiate within- and between-individual variance to calculate repeatability (Stoffel et al., 2017). In a recent cross-fostering study of king penguins (*Aptenodytes patagonicus*), repeated measures of TL were taken, indicating that chick TL was positively related to a foster mother's TL at both 10 and 105 days after hatching; however, this study did not include information about parental age (Viblanc et al., 2020). Our study combines both of these approaches, applying a cross-fostering manipulation between differently aged parents with repeated measures of offspring TL in a wild population of long-lived birds. We cross-fostered whole clutches of common gull (*Larus canus*) eggs shortly after laying within and between age classes of young and old parents, and assessed TL and the change in TL during the time of the fastest chick growth (from hatching to approximately two weeks old) in red blood cells. This time period was based on the feasibility of repeated trapping of precocial bird chicks. The red blood cells (a major component of the blood) of birds have a maximum lifespan of about 30–40 days (Rodnan et al., 1957), and accordingly, it is reasonable to expect to see an effect on telomere shortening in this time period. We predicted that if there are epigenetic-like effects on the telomeres that offspring inherit and/or prenatal effects of parental age on offspring telomeres, there would be a relationship between the age of the natal parents and offspring telomeres. Alternatively, if there post-natal effects of parental age on offspring telomeres, there would be a relationship between the age of the foster parents and offspring telomeres.

2 | MATERIALS AND METHODS

2.1 | Field methods

We conducted the study between the end of May–early June 2017 on a free-living, known-age breeding population of common gulls located on Kakrarahu islet in Matsalu National Park on the west coast of Estonia (58°46′ N, 23°26′ E). Based on studies of offspring recruitment rate, the reproductive success in common gulls increases up to the tenth breeding year (12–13 years of age) and decreases thereafter (Rattiste, 2004). Common gulls undergo senescence in a number of physiological functions (Rattiste et al., 2015; Sepp et al., 2017; Urvik et al., 2018). This species is characterized by biparental care and a low frequency of extra-pair mating (Bukacinska et al., 1998), making it possible to study the effects of age of the parent of both sexes, and the age-dependent quality of parental care on offspring telomeres. All of the birds included in the study were banded as chicks and their exact age was therefore known. Common gulls typically lay clutches of three eggs, independent of maternal age.

A total of 40 nests were included in the experiment. Nests and experimental groups were chosen based on the age of the mother, but the father's age was also known. Common gulls mate assortatively with respect to age, and the ages of the parents were highly positively correlated (Spearman $r = .74$, $p < .0001$). Half of the breeders ($n = 20$ females) were young, on their first to third breeding event (age exactly five years). Another half ($n = 20$ females) were middle-aged or older (15–30 years old, average age 18 ± 3.37 [SD] years). In total, 19 males were 5–7 years old (average age 5.52 ± 0.80 [SD] years) and were grouped as "young", 21 males were 10+ years (average age 16.29 ± 5.58 [SD] years) old and were grouped as "old". The distribution of the ages of parent birds are shown in Figures S1–S4.

TABLE 1 Cross-fostering experiment sample sizes. A total of 72 chicks from 40 nests were included in the experiment

Parent gender	Manipulation	Number of nests	Number of chicks
Female	Young to young	10	12
	Old to old	10	18
	Young to old	10	26
	Old to young	10	16
Male	Young to young	8	14
	Old to old	12	18
	Young to old	11	24
	Old to young	9	16

We cross-fostered whole clutches immediately after the clutch was completed both within and between maternal age classes (Table 1), so that all of the chicks included in this experiment hatched in the nests of foster parents. Half of the clutches were cross-fostered between age classes (to test for the effects of parental age – young vs. old – on offspring TL) and half of the clutches within age classes (to exclude the possibility that we are measuring the effect of cross-fostering). We opted for cross-fostering whole clutches instead of a split-nest design for the following reasons: (i) swapping half clutches would have required very careful and logistically complicated monitoring of hatching order within nests, to know which chick hatched from which egg, (ii) swapping part of the clutch would have required very synchronous timing between nests to avoid age differences between "own" and "foster" chicks, (iii) if a split-nest cross-fostering design was used, the nest could no longer be included in the analyses if one of the chicks died and (iv) since the main approach was to swap between parental age categories, cross-fostering whole clutches was the most feasible study design. The 72 chicks that were successfully caught for second sampling were included in the study. From the nests of "old" parents, we recaptured on average 1.6 chicks, from "young" parents, 2.0 chicks (t test $t = -1.7$, $p = .1$). Within two days from hatching, we collected the first blood sample (10–30 µl taken, from brachial vein) for telomere measurement (average age 0.61 ± 0.09 [SE] days), individually marked the chicks for identification, and measured chick head size (the distance from the tip of the bill to the back of the head) with a calliper to the nearest 0.1 mm. Chicks were blood sampled again near their nests sites between 5 and 20 days after hatching (mean age: 10.62 days ± 0.35 SE). Blood samples were kept on ice in an insulated box while on the islet, and stored at −20°C until the end of the field work period, when they were transferred to −80°C and

maintained until analyses. The experimental protocol was approved by the Ministry of Rural Affairs of the Republic of Estonia (licence no. 106, issued 24.05.2017) and was performed in accordance with relevant Estonian and European guidelines and regulations.

2.2 | Telomere measurement and molecular sexing

DNA was extracted from whole blood samples using Macherey Nagel Nucleospin Blood kits following the manufacturer's protocol. Avian red blood cells are nucleated and are well suited for longitudinal telomere analysis (Nussey et al., 2013, 2014). DNA concentration was assessed with a Nanodrop 8000 (Thermo Scientific), all our samples had high purity. Relative TL was measured using qPCR (quantitative polymerase chain reaction) on an Mx3000P (Stratagene) as described in Cawthon (2002) and modified for use in common gulls (Rattiste et al., 2015). The relative telomere length (T/S) of the samples was calculated as the ratio of the telomere repeat copy number (T) to that of a single copy control gene (S), relative to the reference sample. Glyceraldehyde-3-phosphate dehydrogenase (GAPDH) was used as the single copy control gene. We used the following gull specific GAPDH forward and reverse primers (Integrated DNA Technologies): 5′-CGGAGCACCGCTTACAATTT-3′ and 5′-GCATCTCCCCACTTGATGTTG-3′ respectively (concentration in reaction mixture 200 nM). Amplified samples were run on a 3% agarose gel to verify that the amplification was a single product, which yielded a single band at 77 bp as expected. We used the following telomere primers (Quanta Bio, with final concentration of 200 nM): TEL 1b: 5′-CGGTTTGTTTGGGTTTGGGTTTGGGTT-3′ and TEL 2b: 5′-GGCTTGCCTTACCCTTACCCTTACCCT-3′. The qPCR reactions for GAPDH and telomeres were run on separate plates. The cycling parameters for telomere plate were: one cycle for 10 min at 95°C, 27 cycles for 15 s at 95°C, 30 s at 58°C, 30 s at 72°C, one cycle of dissociation curve (melt curve). The cycling parameters for the GAPDH plate were: one cycle for 10 min at 95°C, 40 cycles for 30 s at 95°C, 30 s at 60°C, one cycle of dissociation curve (melt curve). All reactions used 20 ng of DNA in a final volume of 25 µl containing 12.5 µl of SYBR green Master Mix, 0.25 µl forward and reverse primer, 6 µl water, and 6 µl of DNA sample. A negative control of water was run on each plate. All samples were run in duplicate, and average values were used to determine the T/S ratio. Treatment groups were distributed approximately equally between plates. Technical replicates of each sample, and the first and second sample for each bird were run on the same plate. Duplicates that had a SD higher of 0.25 were rerun again as duplicates. In order to assess the efficiencies of each plate, samples were run against a standard curve of 40, 20, 10, 5, and 2.5 ng produced by serially diluting a reference sample. In all cases, plate efficiencies were in the accepted range (i.e., 100+/−15%) and all of the samples fell within the bounds of the standard curve. Average plate efficiencies and standard errors for GAPDH and telomere plates were 102.34 ± 3.00, and 98.94 ± 3.00, respectively. The average intraplate variation of the C_t values was 0.82% for the telomere assays and 0.19% for the GAPDH assays, andthe interplate CV-s of the C_t values for telomere and GAPDH assays were 0.64% and 0.34%, respectively. The same individual was also included on every plate and the coefficient of variation of the T/S ratio across plates was 8.12%. Standards and Golden (reference sample) were from birds not included in the experiment. A subset of samples (45) were also run across two plates to calculate the intraclass correlation coefficient (ICC) for the T/S ratio (single measurements ICC = .876, 95% CI 0.785 ± 0.930, $p < .0001$).

The CHD gene (chromo helicase DNA-binding gene) was use as a molecular marker for sexing the birds. PCR was used to amplify the CHD genes in DNA extracted from the red blood cells. The primers used in the PCR were as follows: 2550F (5′-GTTACTGATTCGTCTACGAGA-3′) and 2718R (5′-ATTGAAATGATCCAGTGCTTG-3′) described by Fridolfsson and Ellegren (1999). The PCR products were visualized by gel electrophoresis and a FluorChem FC2 imaging system. One band on the gel indicates a male and two bands indicates a female.

2.3 | Statistical methods

To test for an effect of parental age on offspring TL and the change in offspring TL between the first and second sampling, and offspring headsize, we used repeated measures mixed models and the R package lmerTest (Kuznetsova et al., 2017). Brood ID and the sex of the chick were included in our analysis to account for the common origin of the chicks from the same brood. Models also included the age groups (young or old) of either natal mother and foster mother or natal father and foster father as categorical predictors. Similar models were also run with age as a continuous factor (Table S1). Separate models were run for the effects of the mother's and father's ages, because the age of the parents were strongly correlated and could not be entered in the same models. Both models with and without nonsignificant interactions (Table 2 and Table S2) were run. The sampling interval between the collection of the first and second telomere sample varied between individuals, but this was not included in these models as it was not significantly related to TL (Figure S5, Table S3, $p = .805$). Models examining offspring headsize (Tables S4 and S5) also included chick age as a covariate. Similar models were also run with age as a continuous variable (Table S1). Markov Chain Monte Carlo (MCMC) multivariate generalised linear mixed models where used to assess the continuous effects of parental age on offspring TL and the change in TL. Repeatabilites were calculated using R package rptR (Stoffel et al., 2017). Heritabilities were calculated for telomeres using a one-way ANOVA of the full sib design and using generalised linear mixed models of the repeated measures design (R package MCMCglmm) (de Villemereuil et al., 2018; Hadfield, 2010; Kuznetsova & Hadfield, 2010). The models were run for both telomere measurements (first and second) as well as telomere change as a dependent variable. The models included parent nest as random effect and the same fixed effects as the repeated measures mixed model for the effect of TL change. Both repeatability (Stoffel et al., 2017) and heritability measures are reported with 95% coefficient

TABLE 2 Repeated measures mixed model of telomere length dynamics (separate models for mother and father age groups). Final models without nonsignificant interactions are presented in Table S2

Predictor	Estimates	CI	*p*	Estimates	CI	*p*
Second – first measurement	0.11	0.02–0.20	**.022**	0.09	−0.01 to 0.18	.066
Chick sex	−0.02	−012 to 009	.749	−0.01	−0.11 to 0.09	.796
Mother age group	0.04	−0.10 to 0.17	.592			
Foster mother age group	0.11	−0.02 to 0.25	.099			
Time × mother age group	−0.03	−0.14 to 0.08	.550			
Time × foster mother age group	−0.03	−0.14 to 0.08	.552			
Father age group				0.09	−0.04 to 0.22	.179
Foster father age group				0.09	−0.04 to 0.21	.199
Time × father age group				−0.02	−0.12 to 0.09	.737
Time × foster father age group				0.01	−0.10 to 0.11	.899
Random effects						
σ^2		0.03			0.03	
τ_{00}		$0.02_{chick\_ID:parent\_nest}$ 0.02_{parent_nest}			$0.02_{chick\_ID:parent\_nest}$ 0.01_{parent_nest}	
ICC		.59			.58	
N		72_{chick_ID} 40_{parent_nest}			72_{chick_ID} 40_{parent_nest}	
Observations		144			144	

Abbreviations: ICC, intraclass-correlation (variance partition coefficient);σ^2, within-group (residual) variance; τ_{00}, between-group-variance.

intervals (CI). The traits were normally distributed and models were run on untransformed values (Figures S6–S8). Power analyses were conducted based on Monte Carlo simulations (Green & MacLeod, 2015) (Table S6). The R code for the statistical analyses is accessible in the data repository (Sepp et al., 2021a).

3 | RESULTS

There were no significant effects of the ages of the natal or foster parents on offspring TL at hatching or the change in offspring TL from hatching to the end of post-natal development, in male or female chicks (Figure 1, see Table 2 and Table S2 for results with age groups, young vs. old, and Table 3 and Table S1 for results with parental age as a continuous factor). The parental age effect did not become significant after the nonsignificant interactions and chick sex were removed from the model (Table S2). These results were not changed by replacing the parental age term with a parental age squared term in the models (Tables S7–S9). Telomere length was individually repeatable between the first and second sampling, separated by on average 11 days (repeatability r = .55, CI = 0.368–0.69, p < .0001, Figure 2). Chick TL between the first and second telomere measurements increased rather than decreased (the estimated change in relative TL was 0.08 ± 0.05, p = .003, Table 2; there was one outlier, a chick with the greatest increase in TL; however, the results were quantitatively and qualitatively similar when this individual was removed (estimated change 0.06 ± 0.04, p = .005). The rate of growth during post-natal development was not significantly related to the change in TL (measured as change of head size, r < .0001; p = .97, Figure 3). Chick growth rate was also not significantly related to the age of the natal or foster parents (all parental effect p-values > .31, Tables S7 and S8). The power for detecting the potential effects of parental age on offspring TL and the change in offspring TL in our sample is reported in Table S6. The study design also allowed us to assess the wide-sense heritability of TL, although these results must be taken with caution, since we did not use a split-nest cross-fostering design. Chick TL at both first (h^2 = 0.27 CI = 0.05–0.50) and second (h^2 = 0.30 CI = 0.08–0.54) measurement as well as the change of TL (calculated as TL 2 − TL 1, h^2 = 0.14 CI = 0.03–0.29) were heritable.

4 | DISCUSSION

Parental age often has long-term consequences for offspring phenotype and fitness, but the mechanisms that mediate these effects are not well understood. Previous research suggest that telomeres may be an important mechanism mediating these effects, but the routes by which these effects occur remain unclear. The cross-fostering experiment used here allowed us to distinguish whether any effects of parental age on offspring telomeres occur as a result of epigenetic-like changes to the telomeres that offspring inherit and/or prenatal effects versus post-natal parental effects. Previous research in this colony of common gulls (*Larus canus*) has demonstrated that

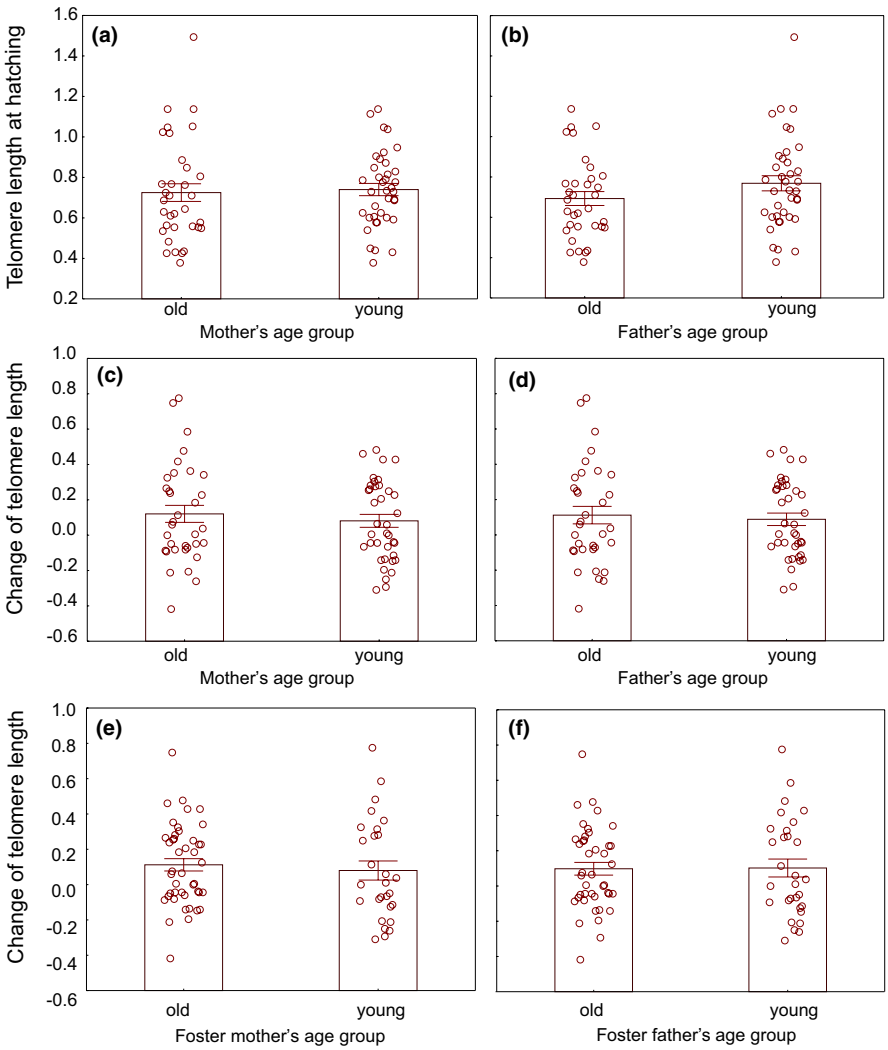

FIGURE 1 Natal parents' and foster parents' age group did not affect offspring telomere length (a, b) and telomere dynamics (c–f). Mother and father's age groups were concordant for most, but not all chicks. Columns denote group means, whiskers are standard errors

offspring fitness decreases with increasing parental age. Offspring produced by older parents (12–13 years of age) have lower recruitment (Rattiste, 2004) and some of these effects may be mediated by pre-natal maternal effects as older gulls allocate less nutrients to their eggs (Urvik et al., 2018). However, despite these demonstrated effects of parental age on offspring fitness in this species, we did not find any evidence of the effect of parental age on offspring telomeres at hatching or the change in telomere length during post-natal development.

Although parental age has been shown to have both negative and positive effects on offspring TL (Table 4, extended version Table S10), this has not been found in all studies. For example, no effect of parental age on offspring TL was found in a study of Soay sheep (*Ovis aries*) despite a large sample size (Froy et al., 2017). Studies in birds have generally found a negative effect of parental age on offspring telomeres (see for example studies in alpine swifts, (Criscuolo et al., 2017), and European shags *Phalacrocorax aristotelis*, (Heidinger et al., 2016)), but this pattern is not a universal (for example, older great reed warbles *Acrocephalus arundinaceus* produced offspring with longer telomeres, (Asghar, Hasselquist, et al., 2015)). Interestingly, the opposite pattern is observed in humans, as older fathers sire offspring with longer telomeres than younger fathers (Broer et al., 2013; Eisenberg, 2014). Discrepancies among studies could be due to interesting biological variation among species, but could also be due to differences in study designs (Eisenberg, 2014; Heidinger & Young, 2020). For example, within-individuals effects of age can be obscurred by cross-sectional sampling due to differences in cohort effects or past selection events. Accordingly, we cannot exclude the possibility that a longitudinal study design (examing age-related changes within rather than across parents) would have revealed an effect of parental age on offspring telomeres in our study. It is also possible that the relationship between parental age and offspring TL was too weak to be detected, despite having relatively good test power (Table S3). It is also possible that TL in common gulls is relatively buffered from environmental influence.

We may also have failed to detect an effect of parental age on offspring telomeres if our experimental time scale (approximately 2 weeks) was too short to detect effects on telomeres. However, the maximum lifespan of red blood cells of birds is estimated to be 30–45 days (Rodnan et al., 1957), which suggests that we should have been able to detect an effect of parental age on the change in TL in this study. Indeed, several studies have detected telomere loss

TABLE 3 Markov Chain Monte Carlo (MCMC) multivariate generalised linear mixed models of parental age (hatch year) effects on telomere length and telomere length dynamics

	Mother's hatch year	Foster mother's hatch year	Random effect (parent nest)	Father's hatch year	Foster father's hatch year	Random effect (parent nest)
Telomere length at hatching						
Mean (95% CI)	0.004 (−0.005 to 0.012)	0.007 (−0.001 to 0.015)	0.016 (0.003–0.032)	0.006 (−0.003 to 0.015	0.006 (−0.004 to 0.016)	0.016 (0.003–0.032)
pMCMC	0.41	0.10		0.23	0.25	
Telomere length at second trapping						
Mean (95% CI)	0.001 (−0.010 to 0.014)	0.003 (−0.007 to 0.014)	0.021 (0.004–0.043)	0.009 (−0.002 to 0.020)	0.009 (−0.003 to 0.020)	0.019 (0.004–0.039)
pMCMC	0.85	0.56		0.11	0.16	
Change of telomere length						
Mean (95% CI)	−0.003 (−0.013 to 0.006)	−0.005 (−0.014 to 0.005)	0.011 (0.002–0.023)	−0.031 (−0.174 to 0.103)	0.001 (−0.01 to 0.012)	0.011 (0.002–0.023)
pMCMC	0.50	0.33		0.65	0.093	

Abbreviation: CI, confidence interval.

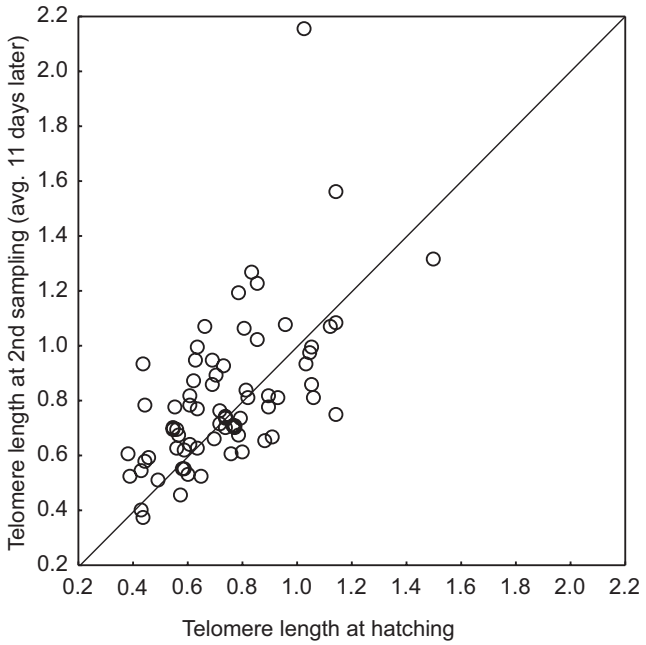

FIGURE 2 Telomere length was individually repeatable between first (average 0.61 days after hatching) and second blood sampling (average 10.62 days after hatching). *X* = *Y* line is depicted to illustrate the direction of individual increase of telomere length

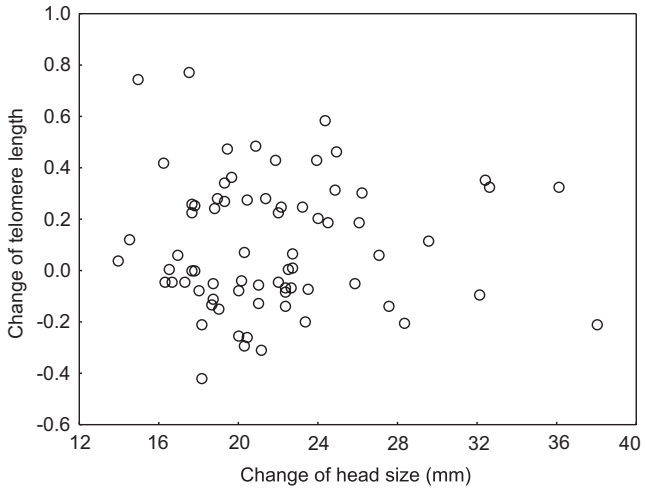

FIGURE 3 The change in relative telomere length between the first and second sampling (average time difference 10.62 days) were not related to chick growth rate (change of head size). Since head size and telomere length were measured at the same time points for individual birds, time is not included as a factor here

within 30 days or less (Boonekamp et al., 2014; Foote et al., 2011; Heidinger et al., 2016). It is also unlikely that the selective dissappearance of chicks contributed to our findings as the probability of trapping chicks from old and young parents was not significantly different. During the early age, recapture rate mainly depends on nest location – it is more difficult to trap chicks from nests that are close to water or surrounded by dense vegetation. In our colony, about half of the chicks die before fledging, mainly due to common hazards of the environment. Parental care quality might play a small role in chick mortality, but it is very likely that this role is eclipsed by the effect of random mortality. Accordingly, we consider the role of selective dissappearance to be negligible in our results.

Although telomere length was repeatable during post-natal growth, it significantly increased between the first and second sampling period and this change in TL was not related to the pace of growth. Some vertebrates show increased telomerase activity during development (reviewed by Larsson et al., 1997; Monaghan & Haussmann, 2006). In several bird species, telomerase activity appears to be highest in the hatchling age-class, when the proliferative demands of most organs are the highest (Haussmann et al., 2007). It has even been suggested that in free-living, long-lived organisms, evolution should favour mechanisms that maintain the longest possible telomeres at the end of the most active growth period (Chan, & Blackburn, 2004). Studies in the

TABLE 4 Overview of studies in nonhuman vertebrates (also excluding laboratory rodents) investigating the effect of parental age on offspring telomere length. For a review on human studies, please see Froy et al. (2017). Extended version of the table including study design and offspring developmental stage available in Supporting Information Materials (Table S10).

Class	Species	Sample size	Studied parental effects	Telomere method	Parental age effect (−/+)	References
Teleost fish	Atlantic salmon (*Salmo salar*)	84 HSF	M & P	qPCR (RTL)	Complex P_{fry} (+)	McLennan et al. (2016)
	Atlantic salmon (*Salmo salar*)	60 HSF	M & P	qPCR (RTL)	Complex P_{fry} (−)	McLennan et al. (2018)
Reptiles	Sand lizard (*Lacerta agilis*)	N_{sons} = 12	P	TRF (TL in bp)	P_{sons} (−)	Olsson et al. (2011)
Birds	Great reed warbler (*Acrocephalus arundinaceus*)	N_{ind} = 139 N_{broods} = 46	M & P	qPCR (RTL)	M (+)	Asghar, Bensch, et al. (2015)
	European shag (*Phalacrocorax aristotelis*)	N_{ind} = 311 N_{broods} = 134	M & P	qPCR (RTL) and ΔRTL	$M_{\Delta RTLoffspring}$ (−) $P_{\Delta RTLoffspring}$ (−)	Noguera et al. (2018)
	Alpine swift (*Apus melba*)	N_{ind} = 95 N_{broods} = 54	M & P biological and foster	qPCR (RTL)	$P_{biological}$ (−) M_{foster} (−)	Heidinger et al. (2016)
	Zebra finch (*Taeniopygia guttata*)	N_{ind} = 139 $N_{HSbroods}$ = 64	P	qPCR (RTL)	P (−)	Criscuolo et al. (2017)
	Black-browed albatross (*Thalassarche melanophrys*)	N_{ind} = 51	Mean M & P	TRF (TL in bp)	Par (+)	Dupont et al. (2018)
	Common tern (*Sterna hirundo*)	*N* = 142	M & P	TRF (TL in bp)	P (−)	Bouwhuis et al. 2018
	Jackdaw (*Corvus monedula*)	N_{ind} = 715 N_{nests} = 298 N_{ind} = 61 N_{nests} = 31	M & P biological and foster	TRF (TL in bp)	P_{biol} (−)	Bauch et al. (2019)
	Common gull (*Larus canus*)[Curr. study]	N_{ind} = 72 N_{nests} = 40	M & P	qPCR (RTL and ΔRTL)	No effect	Sepp et al. (current study)
Mammals	Soay sheep (*Ovis aries*)	N_{ind} = 318	M & P	qPCR (RTL)	No effect	Froy et al. (2017)
	Chimpanzee (*Pan troglodytes*)	N_{ind} = 40	P	qPCR (RTL)	P (+)	Eisenberg et al. (2016)

Abbreviations: (−), negative association; (+), positive association; HSF, half sib families; ind, individuals; M, maternal age effect; P, paternal age effect; Par, parental age effect; RTL, relative telomere length; TL, telomere length; TRF, terminal restriction fragment.

wild have shown that telomeres elongate in at least some life stages in Seychelles warblers (*Acrocephalus sechellensis*: Spurgin et al., 2018), edible dormouse (*Glis glis*: Hoelzl et al., 2016), Soay sheep (Fairlie et al., 2016), Magellanic penguins (*Spheniscus magellanicus*: Cerchiara et al., 2017), and Atlantic salmon (*Salmo salar*: (McLennan et al., 2018). However, telomere shortening during development has been observed in other gull species. For example, in lesser black-backed gull (*Larus fuscus*), telomeres shortened within the first 10 days of hatching (Foote et al., 2011), and in black-tailed gull chicks (*Larus crassirostris*), telomere loss occured in chicks with siblings, but not in chicks reared without siblings (Mizutani et al., 2016). The latter study suggests that under favourable growing conditions, telomere loss might be prevented.

TL and the change in TL were not predicted by growth rate (Figure 3). In general, a trade-off between rapid growth and telomere maintenance is expected, because a greater number of cell divisions are required to attain a larger size, and/or because of the conditions required for fast growth (e.g., higher metabolic rate and ROS production, reviewed by Monaghan & Ozanne, 2018). The current study adds to the increasing number of studies suggesting a different pattern in long-lived seabirds (Mizutani et al., 2016; Young et al., 2017). Seabirds are distinguished from most other species by a long time period between the end of the relatively fast somatic growth and the beginning of reproduction (stretching several years). More studies applying comparable methodological approaches are needed for a comparative study, including patterns of growth and life-history strategies of different species to determine if this phenomenon of delayed reproduction is causally linked with the lack of an association between fast growth and telomere shortening in seabirds.

In conclusion, our results suggest that the age of the parents at the time of offspring conception does not influence offspring

TL or the change in TL in common gulls. The physiological mechanisms mediating the link between parental age and offspring recruitment rate in common gulls remains unexplained and deserves further attention. An important area of future research is to identify other mechanisms that mediate the long-term effects of parental age on offspring and to better understand the factors that contribute to the variation in the influence of parental age on offspring telomeres across species. Future studies applying this design should also consider measuring parental telomere length. Additionally, studies testing the same hypothesis over years with different breeding conditions could indicate to what degree the links between parental age and offspring quality are dependent on environmental conditions.

ACKNOWLEDGEMENTS

We thank Mari-Ann Lind, Ulvi Karu, and other field assistants. We also thank three anonymous reviewers who gave valuable suggestions for improving the manuscript. This work was supported by the Estonian Research Council (IUT21-1, IUT34-8, PSG458, PSG653), Marie Sklodowska-Curie grant agreement no. 701747 to TS, and Mobilitas Pluss from European Regional Development Fund (MOBJD344 to KN).

AUTHOR CONTRIBUTIONS

Tuul Sepp, Mathieu Giraudeau, Peeter Hõrak and Kristina Noreikiene conceived the ideas and designed methodology; Janek Urvik, Kalev Rattiste, Richard Meitern and Lauri Saks collected the data; Kalev Rattiste provided demographic data; Tuul Sepp, Britt Heidinger, Jeffrey Kittilson and Mathieu Giraudeau analysed the samples; Peeter Hõrak, Richard Meitern and Tuul Sepp performed statistical analyses. All authors contributed critically to the drafts and gave final approval for publication.

DATA AVAILABILITY STATEMENT

Data are available from the Dryad Digital Repository (Sepp et al., 2021b, https://doi.org/10.5061/dryad.2ngf1vhn5).

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SUPPORTING INFORMATION

Additional supporting information may be found online in the Supporting Information section.

How to cite this article: Sepp T, Meitern R, Heidinger B, et al. Parental age does not influence offspring telomeres during early life in common gulls (*Larus canus*). *Mol Ecol*. 2021;00:1–11. https://doi.org/10.1111/mec.15905