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Telomere length is heritable and genetically correlated with lifespan in a wild bird

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Abstract

Telomeres are protective caps at the end of eukaryotic chromosomes that shorten with age and in response to stressful or resource-demanding conditions. Their length predicts individual health and lifespan across a wide range of animals, but whether the observed positive association between telomere length and lifespan is environmentally induced, or set at conception due to a shared genetic basis, has not been tested in wild animals. We applied quantitative genetic "animal models" to longitudinal telomere measurements collected over a 10-year period from individuals of a wild seabird (common tern; *Sterna hirundo*) with known pedigree. We found no variation in telomere shortening with age among individuals at the phenotypic and genetic level, and only a small permanent environmental effect on adult telomere length. Instead, we found telomere length to be highly heritable and strongly positively genetically correlated with lifespan. Such heritable differences between individuals that are set at conception may present a hitherto underappreciated component of variation in somatic state.

KEYWORDS
ageing, biomarker, birds, heritability, senescence, telomeres

1 | INTRODUCTION

Telomeres are repetitive nucleotide sequences at the ends of eukaryotic chromosomes (Meyne et al., 1989). They maintain chromosome integrity by serving as disposable buffers that protect the chromosome ends from deterioration and fusion with other chromosomes (Blackburn, 1991). Telomeres shorten with cell replication (Olovnikov, 1973; Stewart et al., 2003) and, without active restoration, become progressively shorter with age (Harley, 1991; Monaghan, 2010). A wide range of environmental variables that reflect stressful or energetically demanding circumstances have been reported to accelerate telomere shortening with age (Angelier et al., 2018; Chatelain et al., 2020), and studies on humans, other mammals and birds typically find individuals with short telomeres, for their age, to have an increased risk of mortality and a reduced lifespan (Boonekamp et al., 2013; Wilbourn et al., 2018). As such, telomere length may act as a biomarker for an individual's accumulated somatic stress, and thereby be positively associated with lifespan, irrespective of whether telomere length has a causal role in determining lifespan.

Studies on humans, however, suggest that between-individual differences in telomere length are maintained from early life onwards and that effects of adult lifestyle are comparatively small (Benetos et al., 2013; Graakjaer et al., 2004; Pepper et al., 2018; Verhulst et al., 2016). Studies on wild mammals and birds are equivocal in this respect. Studies on jackdaws (*Corvus monedula*) and

common terns (*Sterna hirundo*) corroborate these results (Bichet et al., 2020; Boonekamp et al., 2014), while studies on Soay sheep (*Ovis aries*), Seychelles warblers (*Acrocephalus sechellensis*) and European badgers (*Meles meles*) suggest between-individual differences in telomere length to be little consistent throughout life (Fairlie et al., 2016; van Lieshout et al., 2019; Spurgin et al., 2018). As these latter studies used quantitative polymerase chain reaction (qPCR) rather than terminal restriction fragment (TRF) analysis to estimate telomere length, it is, however, unclear to what extent these differences are methodological, rather than due to differences in strong and variable environmental effects between species (also see Bichet et al., 2020).

Consistent between-individual differences in telomere length, combined with telomere length predicting individual lifespan, can potentially be explained by a large role of the early-life environment in determining both telomere length and lifespan. Several studies suggest that environmental conditions during pre- and post-natal development can affect telomere shortening (e.g., Boonekamp et al., 2014; Herborn et al., 2014; Pineda-Pampliega et al., 2020; Vedder et al., 2018) and several studies indeed find lifespan to be predicted by telomere length early in life (Asghar, Hasselquist, et al., 2015; Eastwood et al., 2019; Fairlie et al., 2016; Heidinger et al., 2012; van Lieshout et al., 2019).

Alternatively, individual telomere length and its correlation with lifespan may already largely be determined at conception. In humans, there is strong evidence for genetic inheritance of telomere length (Broer et al., 2013; Dugdale & Richardson, 2018; Hjelmborg et al., 2015), and genome-wide association studies suggest genes involved in telomere maintenance to be associated with health and lifespan (Atzmon et al., 2010; Deelen et al., 2013; Soerensen et al., 2012). Apart from humans, estimates of heritability of telomere length have mostly been reported for birds and are variable (Dugdale & Richardson, 2018), with some studies suggesting low (i.e., 0.04–0.09) (Becker et al., 2015; Voillemot et al., 2012), and others high (i.e., 0.48–0.99) (Asghar et al., 2015; Atema et al., 2015; Belmaker et al., 2019), heritability. Here too, it is unknown whether this variation represents biological variation, or is due to differences arising from measurement techniques. The few heritability studies in birds that have measured telomeres using TRF analysis, which is considered more reliable than qPCR (Morinha et al., 2020; Nettle et al., 2019; Nussey et al., 2014), reported very high heritability estimates (Atema et al., 2015; Belmaker et al., 2019). As one of these studies was performed on captive zebra finches (*Taeniopygia guttata*), the extremely high heritability reported in this study (0.99; Atema et al., 2015) may stem from a stable and benign environment providing little scope for environmental effects on telomere length (Dugdale & Richardson, 2018). However, studies using TRF analysis suggest that environmental effects on telomere length in the wild are small too (Bauch et al., 2020; Bichet et al., 2020; Boonekamp et al., 2014; Chatelain et al., 2020; Vedder et al., 2017). As such, the option that telomere length is also highly heritable under natural conditions, and thus largely determined at conception, deserves further attention.

To our knowledge, the only quantitative genetic study that has tested for a genetic association between telomere length and survival was performed in dairy cattle (*Bos taurus*). In that study, telomere length measured shortly after birth was heritable and positively genetically correlated with survival to 4 years of age (Ilska-Warner et al., 2019), providing support for the telomere length–lifespan correlation to be determined at conception. However, in dairy cattle mortality and selection are typically the result of motives that may only partly reflect factors (e.g., disease) that would also influence mortality and selection in the wild. The genetic basis of the association between telomere length and lifespan should therefore also be tested in the wild, where mortality is natural and the genetic architecture of traits has been shaped by natural eco-evolutionary processes.

In the present study, we investigated whether a positive correlation between adult telomere length and lifespan we observed in a wild seabird—the common tern (Bichet et al., 2020)—is likely to be caused by the environment having a shared effect on both, or set at conception due to a common genetic basis. To do so, we used 619 telomere measurements from 387 individuals, aged 2–24 years, collected across a 10-year period, and combined a series of uni- and bivariate mixed models with random regressions and quantitative genetic techniques, including relatedness information from a detailed pedigree (i.e., an "animal model," Kruuk, 2004).

2 | MATERIAL AND METHODS

2.1 | Study species and population

Common terns are Holarctic migratory seabirds that share incubation and chick provisioning between the sexes, and raise zero to three chicks annually. The data for this study are described in detail in Bichet et al. (2020), and are part of a long-term study of common terns in a breeding colony located at the "Banter See" in Wilhelmshaven, on the German North Sea coast (53°30′40″N, 08°06′20″E). As part of the long-term study, all newly hatched chicks are ringed at their first encounter, between 0 and 2 days old, and receive a subcutaneously implanted transponder (model ID 100; TROVAN) shortly before fledging. New clutches are located within 0–2 days of first egg laying, and transponder-marked parents are linked to their clutch by placing an antenna (model EUR-3110; Euro I.D.), which reads transponder codes every 5–10 s at a distance of ≤11 cm, around all clutches for at least 24 hr. The sex of transponder-marked breeders is molecularly determined from a feather sample, collected shortly before fledging, since 1998 (Becker & Wink, 2003), and was determined by behavioural observation before that. In addition, 44 resting platforms equipped with antennae that automatically record the presence of transponder-marked individuals are distributed throughout the colony. This allows the identification of subadults, nonbreeding and breeding adults, and a very accurate assessment of annual adult survival rate (~0.90) and individual lifespan (Szostek & Becker, 2012). For this study, we assumed a breeder had

died if it was not registered in the colony for at least two consecutive years, until the breeding season of 2020. This is an accurate assumption, because 97% of transponder-marked breeders never skipped registration for more than two consecutive years (Bouwhuis et al., 2015). Common terns in our research colony start breeding on average 3.7 years after fledging (Zhang et al., 2015), and breeders have a lifespan of on average 10 years (Ezard et al., 2006). In the years that blood samples for telomere analysis were collected (see below) the colony size varied between 380 and 650 breeding pairs.

2.2 | Blood sampling

Blood samples were collected from breeders of both sexes in 2007, 2008, 2013, 2014 and 2016. To collect a sample from a specific bird, a larval instar of the blood-sucking bug *Dipetalogaster maximus* was placed in a hollow artificial egg, which was temporarily placed in the nest of the target bird during incubation (for details see Becker et al., 2006). Visual observation combined with the antenna system was used to confirm the continuous presence of the target bird. After 15–20 min, the artificial egg was retrieved and the blood extracted from the bug and stored in 2% EDTA buffer. Blood samples were kept at 4°C until transferred to a 40% glycerol buffer within 3 weeks after sampling and then snap-frozen at −80°C until analysis. It was previously confirmed that blood-sampling with bugs does not affect the telomere measurements, as compared to blood-sampling with a needle (Bauch et al., 2013).

2.3 | Telomere measurements

From each blood sample, average erythrocyte telomere length was measured by in-gel TRF analysis without DNA denaturation (following Bauch et al., 2013; Vedder et al., 2018), which excludes interstitial telomeric sequences. The buffer was removed and the blood cells were washed with 2% EDTA. DNA was isolated from erythrocytes (7 µl cells) using a CHEF Genomic DNA plug kit (Bio-Rad) and subsequently digested simultaneously with *Hind*III (60 U), *Msp*I (60 U) and *Hinf*I (30 U) in NEB2 buffer (New England Biolabs) at 37°C for ~18 hr. One third of the DNA from every sample was added in a 0.8% non-denaturing agarose gel (Pulsed Field Certified Agarose, Bio-Rad) and separated by pulsed field electrophoresis for 22 hr at 14°C (3 V cm^{-1}, initial switch time 0.5 s, final switch time 7.0 s). Two differently sized ^{32}P-labelled ladders were added for size calibration on every gel: (i) a 1-kb DNA ladder (range 1–10 kb; New England Biolabs) (once per gel) and (ii) DNA Molecular Weight Marker XV (range 2–48 kb; Roche Diagnostics) (twice per gel). In addition, we added a standard sample (DNA extracted from a single blood sample of a single chicken; once per gel) as a control. Gels were dried with a gel dryer (model 538, Bio-Rad) and hybridized overnight at 37°C with a ^{32}P-labelled oligonucleotide (5′-C3TA2-3′)4 that binds to the telomeric single-strand overhang. Subsequent washing of the gel with 0.25× SSC buffer at 37°C removed unbound oligonucleotides. The gel was then exposed to a phosphor screen for 4 hr (MS, PerkinElmer) to detect the radioactive signal, which was visualized by a phosphor imager (Cyclone Storage Phosphor System, PerkinElmer). Telomere length distributions were quantified using IMAGE J (version 1.38×, open source) and the average telomere length (in base pairs) was calculated for every sample following the method described in Bauch et al. (2013). The coefficient of variation of the standard sample run on all gels (n = 58) was 0.06. Using the same method, we previously found an intrablood sample repeatability (with multiple DNA extractions per sample) of average telomere length of 0.86 when measuring telomere length of common tern chicks (Vedder et al., 2018).

In total, we collected 619 telomere measurements from 387 individuals, aged 2–24 years. Among them, 220 individuals had one measurement of telomere length, 122 individuals had two measurements in different years, 27 had three, 16 had four, and two individuals had five. In total, 233 of these individuals, with 371 telomere measurements, were considered to have died by 2020 and were used in the analyses involving lifespan.

2.4 | Pedigree data

The pedigree was constructed by assigning all fledged offspring to their observed parents. This social pedigree is a good approximation of the genetic pedigree because common terns exhibit very low levels of extra-pair paternity (González-Solís et al., 2001). For estimating the heritability of telomere length, we pruned the pedigree to individuals with observed phenotypes and their known ancestors. Hence, the social pedigree to test for telomere length heritability comprised 473 records. The maximum depth was four generations, and the number of maternities and paternities 152 and 190, respectively. The number of full, maternal and paternal sibs was 44, 113 and 142, respectively. The mean pairwise relatedness was 0.003.

To estimate the heritability of lifespan and the additive genetic covariance between telomere length and lifespan, we only included those individuals that were considered to have died by 2020 (see above). The pruned pedigree used in these analyses comprised 297 records. The maximum depth was four generations, and the number of maternities and paternities 84 and 103, respectively. The number of full, maternal and paternal sibs was 25, 56 and 31, respectively. The mean pairwise relatedness was 0.004.

2.5 | Quantitative genetic models

We first investigated whether the previously observed shortening of telomeres with age within individuals (Bichet et al., 2020) varied among individuals and was heritable. We applied a random regression modelling framework where we first fitted a "phenotypic model" to test for individual variation in reaction norms (i.e., telomere shortening with age). We then fitted an "animal model" (Kruuk, 2004) to split the phenotypic variance associated with the reaction norms across individuals into additive genetic and permanent environment

components (Table 1). This second random regression model was implemented using a framework that allowed additive genetic variance to vary with age and tested whether variation in shortening across individuals harboured a genetic component.

In both random regression models, we fitted telomere length as the response variable, modelled assuming a Gaussian error distribution (Figure 1a). As fixed effects we fitted laboratory identity (factor with two levels), sex (factor with two levels: male or female) and age (continuous linear variable, mean-centred and variance-standardized, Pettay et al., 2008). The "laboratory effect" was added to capture the fact that the measurements of samples collected in 2016 were done with different equipment and by a different person, which may have led to slight modifications in outcome (also see Bichet et al., 2020). In this model, and all other models, only linear effects of age were modelled, because visual inspection of the data and models that tested quadratic age effects do not suggest the age effect to be nonlinear (Bichet et al., 2020).

For the phenotypic random regression model, we fitted random intercept and slope effects as continuous functions of age for individual identity, and random intercepts for gel identity. Telomere length Z of individual i of gel j at age x was thus modelled as:

$$Z_{ij\,x} = \mu + (\textit{fixed effects}) + f\left(ind_i,\ x\right) + gel_j + e_{ij\,x}, \quad (1)$$

where μ denotes the fixed effect for the overall population mean, $f(ind_i, x)$ is the random regression function of individual values over age (x); gel_j is the among-gel variation effect; and $e_{ij\,x}$ is the random residual value for individual i of gel j at age x. Age was modelled as a continuous linear variable (range = 2–24 years) and standardized to a mean of zero and a variance of one, such that the variance in intercepts can be interpreted as the among-individual variance at the average breeding age. As such, we tested for among-individual variance in the intercepts ($V_{\text{intercepts}}$) and slopes of telomere length on age (V_{slopes}), while also testing for the covariance and correlation between individuals' intercept and slope ($\text{COV}_{\text{intercepts-slopes}}$ and $r_{\text{intercepts-slopes}}$, respectively). Inappropriate modelling of residual variance (e.g., assuming residual homogeneity) might lead to erroneous inferences of slope variance in random regression models (Charmantier et al., 2014). Here, we assumed residual effects

TABLE 1 Posterior modes, means and 95% credible intervals (CI) for fixed effects and variance components of telomere length shortening obtained from two separate random regression models

	Phenotypic model			Animal model		
	Mode	Mean	95% CI	Mode	Mean	95% CI
Fixed effects						
Intercept	10.221	10.214	[10.084, 10.341]	10.228	10.220	[10.096, 10.364]
Age	−0.161	−0.164	[−0.220, −0.104]	−0.164	−0.155	[−0.212, −0.101]
Sex [female]	−0.097	−0.114	[−0.223, 0.018]	−0.113	−0.112	[−0.228, 0.001]
Laboratory [2]	0.729	0.781	[0.470, 1.083]	0.798	0.759	[0.478, 1.069]
Random effects						
Individual/additive genetic						
$V_{\text{intercepts}}$	0.206	0.209	[0.159, 0.262]	0.173	0.194	[0.141, 0.247]
$V_{\text{slopes (age)}}$	0.000	0.004	[0.000, 0.015]	0.000	0.003	[0.000, 0.012]
$\text{COV}_{\text{intercepts-slopes}}$	0.001	0.004	[−0.014, 0.026]	0.000	0.005	[−0.014, 0.024]
$r_{\text{intercepts-slopes}}$	.187	.145	[−.619, .860]	.247	.180	[−.639, .839]
Permanent environmental						
$V_{\text{intercepts}}$	—	—	—	0.000	0.010	[0.000, 0.035]
$V_{\text{slopes (age)}}$	—	—	—	0.000	0.003	[0.000, 0.013]
$\text{COV}_{\text{intercepts-slopes}}$	—	—	—	0.000	0.000	[−0.005, 0.006]
$r_{\text{intercepts-slopes}}$	—	—	—	−.079	.005	[−.795, .808]
Gel	0.105	0.126	[0.067, 0.190]	0.113	0.129	[0.067, 0.191]
Residual [2–6]	0.231	0.238	[0.156, 0.321]	0.212	0.230	[0.147, 0.313]
Residual [7–10]	0.152	0.154	[0.111, 0.200]	0.151	0.149	[0.107, 0.191]
Residual [11–15]	0.137	0.136	[0.094, 0.182]	0.127	0.129	[0.093, 0.172]
Residual [16–19]	0.238	0.263	[0.153, 0.424]	0.201	0.256	[0.139, 0.387]
Residual [20–24]	0.395	0.705	[0.153, 1.605]	0.444	0.702	[0.153, 1.647]

Note: Estimates shown are among-individual, additive genetic or permanent environmental variance in intercepts ($V_{\text{intercepts}}$), among-individual or additive genetic variance in slopes (V_{slopes}), and associated intercepts–slopes covariance ($\text{COV}_{\text{intercepts-slopes}}$) and intercepts–slopes correlation ($r_{\text{intercepts-slopes}}$). Residual effects were assumed to be age-specific (and estimated for each of five age classes: 2–6, 7–10, 11–15, 16–19, 20–24) and uncorrelated across ages (i.e., diagonal residual error structure). Terms not estimated are marked with "—".

to be age-specific (and estimated for each of five age classes: 2–6, 7–10, 11–15, 16–19, 20–24) and uncorrelated across ages (i.e., diagonal residual error structure) (Pettay et al., 2008). Although the choice of the age classes for the residual structure is arbitrary, this structure allows residual variance to vary with age without greatly increasing the number of parameters to be estimated.

For the genetic random regression model, we extended the phenotypic model to separate the individual variance component of the reaction norms into its additive genetic and permanent environmental effects. To do so, we fitted random intercepts and slopes for individual identity linked to the pedigree (allowing estimation of the interaction between additive genetic variance and age), and for individual identity (not linked to the pedigree) to model permanent environmental effects, while we also included random intercepts for gel identity. Telomere length Z of individual i of gel j at age x was thus modelled as:

$$Z_{ij\,x} = \mu + (\textit{fixed effects}) + f\left(a_i,\ x\right) + f\left(pe_i,\ x\right) + gel_j + e_{ij\,x}, \quad (2)$$

where μ denotes the fixed effect for the overall population mean, $f(a_i, x)$ is the random regression function of how additive genetic values vary with age (x); $f(pe_i, x)$ is the equivalent for permanent environment effects; gel_j captures the among-gel variation effect; and $e_{ij\,x}$ is the random residual value for individual i of gel j at age x. Age was again modelled as a continuous linear variable (range = 2–24 years) and standardized to mean of zero and variance of one, such that variance in the intercepts can be interpreted as the among-individual variance at the average breeding age. Thus, with this random regression animal model, we estimated a variance–covariance matrix for the intercept and the slope of the additive genetic and permanent environmental effects (Table 1).

To decompose the total phenotypic variance in telomere length and lifespan into additive genetic (V_A), permanent environmental (V_{PE}) and residual (V_R) variances, we built two univariate animal models. For the univariate animal model of telomere length, we fitted random intercepts for individual identity linked to the pedigree, allowing us to estimate the additive genetic variance (V_A), individual identity (not linked to the pedigree) to control for repeated measures of individuals across years (permanent environmental effects, V_{PE}), and gel identity to control for differences among gels (V_{GEL}). Telomere length was modelled assuming a Gaussian error distribution (Figure 1a). As fixed effects, we fitted age (continuous covariate, mean-centred and variance-standardized), laboratory identity (factor with two levels) and sex (factor with two levels: male or female). For the univariate animal model of lifespan, we fitted random intercepts for individual identity linked to the pedigree (V_A), and sex (factor with two levels) as a fixed effect. Lifespan was modelled assuming a Gaussian error distribution (Figure 1b).

Finally, we fitted a bivariate animal model that allowed the estimation of the genetic correlation between telomere length and lifespan. This model included both telomere length and adult lifespan as response variables, random intercepts for individual identity linked to the pedigree (V_A), and individual identity not linked to the pedigree controlling for repeated measures (V_{PE}). However, because lifespan is observed only once per individual, V_{PE} and V_R in lifespan are not separable. We therefore fixed the residual variance of lifespan to a very small value (i.e., 0.001). By doing so, we were able to effectively partition phenotypic variance in lifespan into additive genetic and permanent environmental effects. Additionally, the simultaneous estimation of additive genetic and permanent environmental variances for telomere length and lifespan allowed us to estimate additive genetic and permanent environmental covariances (COV_A and COV_{PE}, respectively). However, deviance information criterion (DIC) model comparison showed that the model with COV_{PE} was not fitting the data better and we therefore did not model such covariance (DIC of model with COV_A and COV_{PE} = 9,324.55, DIC of model with COV_A but without COV_{PE} = 9,323.45). We also did not fit the covariance between residual components, as the residual variance of lifespan was fixed to 0.001. To control for differences among gels, we fitted gel identity as a random effect associated only with telomere length. We included trait-specific intercepts and sex as fixed effects. We modelled age (continuous covariate) and laboratory (factor with two levels) as fixed effects associated with telomere length only. Both telomere length and individual lifespan were modelled assuming a Gaussian

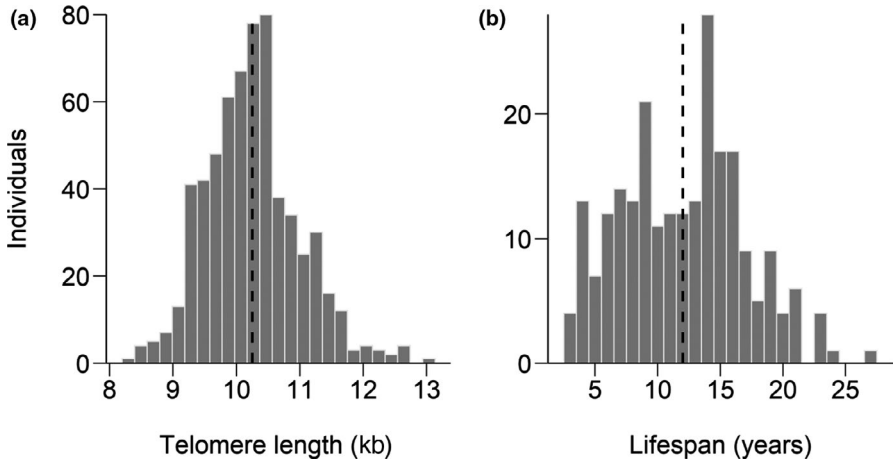

FIGURE 1 Phenotypic distributions of (a) adult telomere length (Kb), and (b) lifespan (years). Dashed vertical lines represent trait means

TABLE 2 Posterior modes, means and 95% credible intervals (CI) for fixed effects, variance components and heritability estimates of telomere length (Kb) and lifespan (years) obtained from two separate univariate animal models

	Telomere length (Kb)			Lifespan (years)		
	Mode	Mean	95% CI	Mode	Mean	95% CI
Fixed effects						
Intercept	10.200	10.215	[10.090, 10.364]	11.414	11.523	[10.677, 12.414]
Age	−0.150	−0.152	[−0.202, −0.106]	—	—	—
Sex [female]	−0.119	−0.102	[−0.221, 0.009]	0.839	0.878	[−0.384, 2.082]
Laboratory [2]	0.746	0.808	[0.493, 1.131]	—	—	—
Random effects						
V_A	0.223	0.229	[0.167, 0.292]	0.047	2.161	[0.000, 7.385]
V_{PE}	0.001	0.015	[0.000, 0.051]	—	—	—
V_{GEL}	0.120	0.137	[0.080, 0.208]	—	—	—
V_R	0.121	0.119	[0.097, 0.142]	22.964	22.618	[16.686, 29.257]
Standardized variance metrics						
h^2	0.464	0.458	[0.343, 0.557]	0.002	0.085	[0.000, 0.279]
h^2 (ex. V_{GEL})	0.650	0.628	[0.495, 0.738]			

Abbreviations: h^2, heritability; V_A, additive genetic variance;V_{GEL}, among-gel variance; V_{PE}, permanent environmental variance; V_R, residual variance.

error distribution (Figure 1a,b). Heritability (h^2) of each trait, conditional on the variance explained by the fixed effects, was estimated as the proportion of the total phenotypic variance explained by its additive genetic variance. In principle, by fitting a bivariate model of telomere length and lifespan, we could have avoided fitting the single univariate animal model of telomere length described above. However, doing so would reduce our sample size for the telomere model, as the analyses with lifespan were restricted to those individuals known to be dead by 2020. Reassuringly, however, estimates of additive genetic variance for telomere length were very similar in both analyses (Table 2; Table S1).

Quantitative genetic models were fitted using a Bayesian framework implemented in the statistical environment R (version 3.6.1, R Core Team 2019) using the R-package *MCMCglmm* (Hadfield, 2010). For all models, we used parameter-expanded priors with an inverse Gamma distribution, chosen to be noninformative (Hadfield, 2010). We fitted alternative priors and found that the quantitative genetic parameters were reasonably robust (see Appendix S1). The number of iterations and thinning interval were chosen for each model to ensure that the minimum Markov chain Monte Carlo (MCMC) effective sample sizes for all parameters were 1,000. Burn-in was set to a minimum of 5,000 iterations. The retained effective sample sizes yielded absolute autocorrelation values lower than 0.1 and satisfied convergence criteria based on the Heidelberger and Welch convergence diagnostic (Heidelberger & Welch, 1981). We drew inferences from the posterior modes, means and 95% credible intervals (95% CI) because inferences drawn from posterior modes and means might differ, particularly when model parameters are near their boundary (e.g., variance close to zero). Near the boundary, the posterior mode tends to converge toward zero while the posterior mean tends to shift away from the boundary due to the broadening of the posterior distributions (He & Hodges, 2008).

3 | RESULTS

The overall mean telomere length was 10.25 Kb (Figure 1a). As demonstrated previously (Bichet et al., 2020), telomere length did not differ between the sexes, but was negatively associated with age. DIC model comparison showed that there was no statistical support for among-individual or additive genetic variation in individual telomere shortening with age (DIC of model with random intercepts and slopes for individual effects = 883.4 vs. DIC of model with only random intercepts = 881.8; DIC of model with random intercepts and slopes for additive genetic and permanent environmental effects = 847.4 vs. DIC of model with only random intercepts = 845.9). This result is in concordance with the low variance explained by the reaction norm slopes, for both the among-individual and the additive genetic components (among-individual and additive genetic variance for slopes = ~0, Table 1). This lack of variance in slopes precluded the possibility of substantial covariance between intercept and slope, which was indeed estimated to be close to zero with wide 95% CI. Hence, we conclude that telomere length similarly shortened with age in all individuals.

There were, however, large heritable differences in telomere length. The posterior mean and mode for the additive genetic variance in telomere length were substantial and of similar magnitude, with the lower 95% CI limit not converging towards zero (Table 2, Figure 2a). The proportion of the total phenotypic variance in adult telomere length explained by additive genetic effects was around 46% (Table 2, Figure 3). Permanent environmental effects accounted for very little (3%) of the total phenotypic variance, while gel identity accounted for 28% (Table 2, Figure 3). Hence, if we assume that the random variance among gels represents measurement error, the heritability (h^2) of telomere length increases from 0.46 to 0.63 (with associated 95% CI = 0.50–0.73).

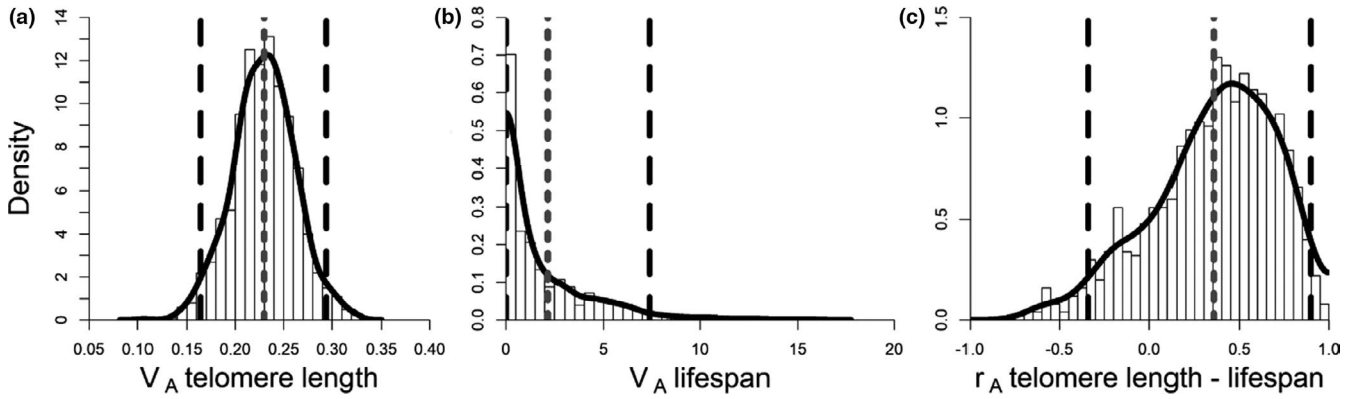

FIGURE 2 Posterior MCMC samples (bars), kernel density estimation (solid black line), posterior mean (grey dotted line) and 95% credible intervals (black dashed lines) for the additive genetic variances (V_A) in telomere length (a) and lifespan (b) extracted from the univariate animal models, and the additive genetic correlation (r_A) between them extracted from the bivariate animal model (c)

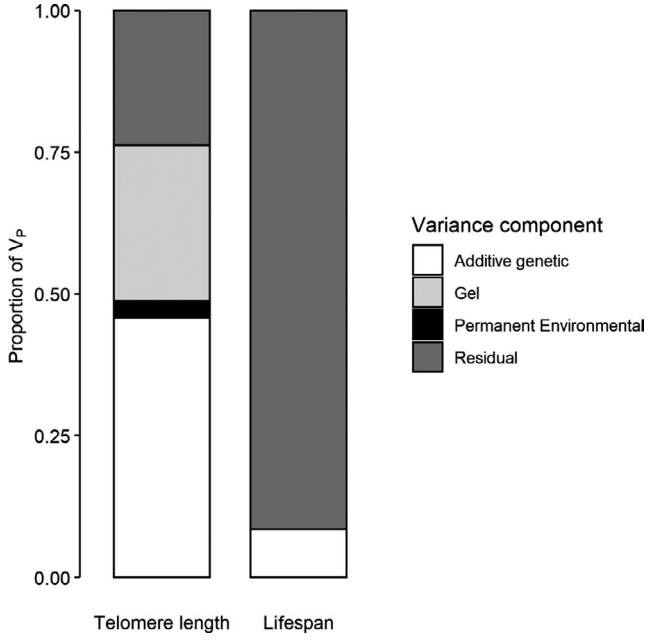

FIGURE 3 Mean proportion of total phenotypic variance (V_P) explained by the different variance components of telomere length and lifespan. Additive genetic effects are shown in white, permanent environmental effects in black, gel effects in light grey and residual variance components in dark grey

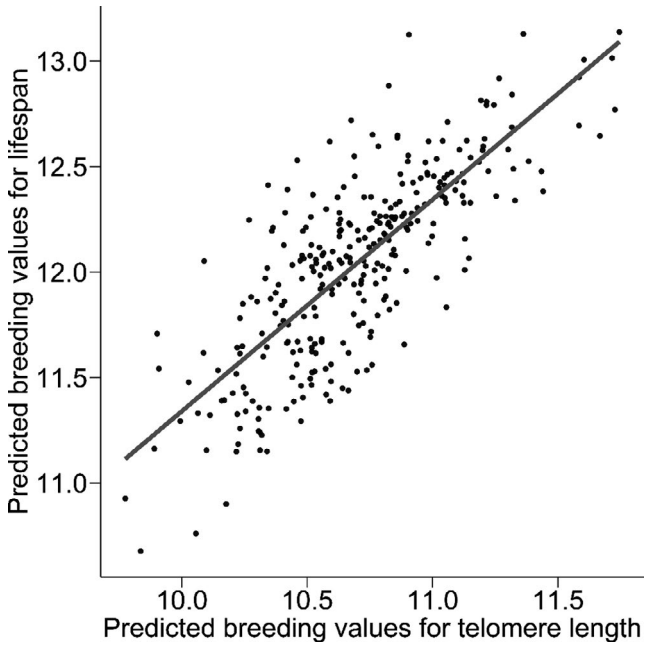

FIGURE 4 Predicted breeding values for lifespan plotted against those for telomere length, as extracted from the bivariate animal model (Table 2). Note that the predicted breeding values for telomere length and lifespan are expressed in their corresponding units (i.e., Kb and years) by adding the population mean trait value to the predicted breeding value of each individual. This figure is drawn for illustrative purposes only

Excluding individuals without a complete lifespan (i.e., still alive) left a data set consisting of 371 telomere measurements from 233 individuals. The lifespan of these individuals ranged from 3 to 27 years, with an average of 12.01 years (Figure 1b), and did not differ between the sexes (Table 2). There were genetic differences in lifespan: the posterior mode of V_A for lifespan was 0.05, whereas the posterior mean was 2.16 (Table 2). Consequently, the posterior mode and mean of the heritability of lifespan were also divergent (0.002 vs. 0.085, Table 2). This difference is due to the right-skewed posterior distribution for lifespan (Figure 2b), which is commonly observed in fitness-related traits (Bonnet et al., 2019; de Villemereuil et al., 2019; Wolak et al., 2018).

A bivariate "animal model" revealed that there was a positive additive genetic correlation between telomere length and lifespan (r_A ~ .4, Table S1; Figures 2c and 4). The posterior mode and mean for the genetic correlation were of similar magnitude. However, the 95% CIs were relatively wide and the lower 95% CI limit overlapped slightly with zero (Table S1; Figure 2c). Nevertheless, 89% of the posterior samples were above zero, suggesting that a shared genetic basis is the most likely explanation for the previously reported phenotypic correlation between adult telomere length and lifespan (Bichet et al., 2020). This conclusion is supported by DIC model comparison, which

showed a model including the additive genetic covariance between telomere length and lifespan outperforming a model without the covariance (DIC of model with additive genetic covariance = 9,323.45 vs. DIC of model without additive genetic covariance = 9,835.45).

4 | DISCUSSION

We here used a quantitative genetic "animal model" approach to reveal a high heritability of telomere length and a positive genetic correlation between telomere length and lifespan. This is the first report of such a genetic correlation in a wild population, in which mortality is natural and the genetic architecture of traits has been shaped by natural eco-evolutionary processes. It corroborates the result of a study on dairy cattle (Ilska-Warner et al., 2019) and fits with findings of genome-wide association studies in humans, which suggest genetic variation in the telomere maintenance pathway to be associated with lifespan (Atzmon et al., 2010; Deelen et al., 2013; Soerensen et al., 2012).

Our longitudinal sampling approach showed that among-individual variance in telomere shortening with age is virtually absent in adult common terns, at both the phenotypic and the genetic level. At the phenotypic level, such variation was also absent during growth in common tern chicks (Vedder et al., 2017). Hence, if variation in genes involved in telomere maintenance were to underlie the positive genetic correlation between telomere length and lifespan in common terns, as suggested for humans (Atzmon et al., 2010; Deelen et al., 2013; Soerensen et al., 2012), it probably involves maintenance of germline telomere length, or maintenance of telomeres during embryonic development, rather than during postnatal life.

In general, a genetic correlation between telomere length and lifespan could be due either to linkage disequilibrium between genes involved in determining telomere length and genes involved in determining lifespan, or to a direct causal effect of telomere length on lifespan. The latter option has remained contentious, as it is difficult to establish *in vivo* (Simons, 2015). If telomere length were to directly influence lifespan, the source of variation in telomere length should not affect its relationship with lifespan, and we would expect the heritable variation in telomere length to affect lifespan. Our results therefore do not disagree with a causal effect of telomere length on lifespan, while an absence of a genetic correlation with lifespan, in combination with a high heritability of telomere length, would have. Regardless, the fact that heritable genetic variation in telomere length is associated with lifespan implies that telomere length acts as a biomarker of lifespan already set at conception, and that therefore, at least in common terns, individual differences in adult somatic state are partly determined at the start of life.

The high heritability of telomere length may appear at odds with the common premise of evolutionary theory that traits with a strong link to fitness display little heritability (Fisher, 1930; Mousseau & Roff, 1987). Indeed, in common terns, which continue to reproduce at old age, the individuals with the longest lifespan, on average, also have greatest lifetime reproductive success (Bouwhuis et al., 2015). However, with an estimated mean heritability of lifespan of 0.085, and a coefficient of determination of its mean genetic correlation with telomere length of 0.126, only a very small proportion of phenotypic variance in lifespan (i.e., 0.085 × 0.126 = 0.011) is determined by additive genetic variance in telomere length. And even this is probably an overestimate, as the posterior mode of the heritability of lifespan was considerably smaller than the mean. Telomere length may thus represent a heritable metric trait that underlies fitness, but be less subjected to the environmental or stochastic sources of variation to which fitness itself is subjected, providing scope for a higher heritability (Price & Schluter, 1991). Moreover, recent studies suggest that germline telomere length may not be completely shielded against somatic ageing (Bauch et al., 2019; Bouwhuis et al., 2018; Monaghan & Metcalfe, 2019), which could act as a perpetual source of genetic variance over generations. The high heritability of telomere length is, however, not due to a comparatively high V_A. The additive genetic coefficient of variation (CV_A, Houle, 1992) is only 4.7%, which, across wild species, is more similar to that of morphological, rather than physiological or life-history traits (Postma, 2014). Possibly, the low CV_A for telomere length is explained by it being a linear trait, which can only vary across one dimension, and not necessarily reflect a signature of selection (Hansen et al., 2011).

Although our analyses show additive genetic variance for telomere length to account for the majority of phenotypic variation in telomere length and its association with lifespan, this does not contradict the overwhelming evidence for stressful or resource-demanding conditions to affect telomere length (Angelier et al., 2018; Chatelain et al., 2020). In common terns such effects are also apparent. We previously found environmentally induced faster embryonic development and more successful reproduction to shorten telomeres (Bauch et al., 2013; Bichet et al., 2020; Vedder et al., 2018). With a permanent environmental effect that accounts for ~4% of the phenotypic variation in adult telomere length (not including among-gel variation), and no significant variation in adult shortening rates, such effects may simply be comparatively small and not consistent enough to provide sufficient scope for a statistically detectable environmental correlation with lifespan. This, however, does not prevent telomere length from functioning as a biomarker in an optimality approach to individual behaviour and life history. If telomere length indeed has a causal effect on lifespan, an individual that inherited short telomeres at conception should still balance further telomere loss against life-history traits, such as growth or reproduction, to maximize fitness. Yet, the implication that individuals do not start with the same somatic state at conception has far reaching ramifications for life-history evolution and senescence, which requires consideration from a theoretical and empirical point of view (Galipaud & Kokko, 2020).

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AUTHOR CONTRIBUTIONS

O.V., S.B., P.H.B. and S.V. designed the study; S.B., C.Ba and O.V. collected blood samples; C.Ba. measured telomere lengths; M.M. and C.Bi. analysed the data; O.V. and M.M. wrote the paper with contributions from all other authors.

DATA AVAILABILITY STATEMENT

Data are available from the Dryad Digital Repository – https://doi.org/10.5061/dryad.wpzgmsbm2.

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SUPPORTING INFORMATION

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

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