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Older paternal ages and grandpaternal ages at conception predict longer telomeres in human descendants

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Telomere length (TL) declines with age in most human tissues, and shorter TL appears to accelerate senescence. By contrast, men's sperm TL is positively correlated with age. Correspondingly, in humans, older paternal age at conception (PAC) predicts longer offspring TL. We have hypothesized that this PAC effect could persist across multiple generations, and thereby contribute to a transgenerational genetic plasticity that increases expenditures on somatic maintenance as the average age at reproduction is delayed within a lineage. Here, we examine TL data from 3282 humans together with PAC data across four generations. In this sample, the PAC effect is detectable in children and grandchildren. The PAC effect is transmitted through the matriline and patriline with similar strength and is characterized by a generational decay. PACs of more distant male ancestors were not significant predictors, although statistical power was limited in these analyses. Sensitivity analyses suggest that the PAC effect is linear, not moderated by offspring age, or maternal age, and is robust to controls for income, urbanicity and ancestry. These findings show that TL reflects the age at the reproduction of recent male matrilineal and patrilineal ancestors, with an effect that decays across generations.

1. Introduction

Telomeres are repeating nucleotide sequences found at the ends of chromosomes that are implicated in health and ageing. Telomeres shorten with each round of cell replication and correspondingly shorten with age in most human tissues. When telomeres are too short a cell can no longer replicate. Thus, tissues containing cells with critically short telomere length (TL) are less able to be maintained, which subsequently may impair health [1,2].

Contrary to the telomere shortening that occurs in most proliferating tissues with ageing in humans, sperm TL appears to increase with age (reviewed in [3]). Accordingly, in humans and chimpanzees, the offspring of older fathers tend to have longer telomeres. We previously reported evidence that this paternal age at conception (PAC) effect persists across at least two generations in a Filipino population [4]. In this sample, the father's age at one's own conception and paternal grandfather's age at the father's conception independently and cumulatively predicted TL. We have hypothesized that this multi-generational PAC effect may represent a case of adaptive intergenerational genetic plasticity in which the TLs that are passed from fathers to their descendants are adjusted based on the father's own age at reproduction [3,5]. In this manner, offspring might inherit TLs that better fit the environment that they are likely to find themselves in. Key to

Table 1. Comparing sample sizes with partially overlapping 2012 study of this cohort. Abbreviations: paternal age at conception (PAC), grand-paternal age at conception (GPAC), great-GPAC (GGPAC), great-great-GPAC (GGGPAC).

	PAC	maternal GPAC	paternal GPAC	GGPAC	GGGPAC
2012	2023	342	234	0	0
current	3282	1476	1437	1278	32

this hypothesis is the notion that the PAC effect persists across multiple generations and thereby provides a more reliable signal of average ages at male reproduction in recent generations. That is, a single PAC effect includes considerable stochasticity (e.g. being firstborn versus the last born). If PAC effects are also conveyed from one's two grandfathers, and four great-grandfathers (and likely with diminishing impacts from an expanding lineage of more distal paternal relatives), a more extensive sampling of recent environments could be provided and thus a better estimation of the mean age at reproduction of recent male ancestors.

Using an expanded dataset from the Philippines, we further examine the intergenerational dynamics of the PAC effect on descendants' TLs across four generations. This expanded dataset contains over five times more individuals with known TL and grand-paternal age at conception (GPAC) than our previous analysis of this population [4] and the first examination of great-grandfather and great-great-grandfather age at conception effects (GGPAC and GGGPAC respectively; table 1). Our prior work suggested an equal magnitude of PAC effects and paternal grandfather age at conception effects [4]. However, based on the semi-conservative nature of DNA inheritance through meiosis, we expected the magnitude of the PAC effect to be halved with each successive generation (e.g. effect of grandfather's age at father's birth on grandchild should have half the effect of father's age on offspring's TL). Since prior work of ours suggested a paternal, but not maternal GPAC effect [4], here we also tested whether TL is passed on equally from mothers and fathers. Finally, we examine whether the PAC effect is linear or driven by other potential confounding factors.

2. Material and methods

This study builds on past work from the Cebu Longitudinal Health and Nutrition Survey (CLHNS) [6]. The project began with the enrolment of a sample of 3327 pregnant women in 1983–1984 in Cebu, Philippines (electronic supplementary material, figure S1). The mothers and 1983–1984 born offspring have been surveyed many times since. In 2005, venous blood was drawn from available mothers ($n = 1881$) and offspring ($n = 1759$) and TL later measured in these. In 2016, a subset of the mothers ($n = 713$) and fathers ($n = 712$) of the 1983–1984 born offspring was interviewed and donated venous blood from which we measured TL ($n = 641$ and 640, respectively). The 2016 mothers and fathers were recruited based on the inclusion criteria that the mothers donated blood samples in 2005 with matched offspring who also donated blood in 2005, for which biological fathers lived with these mothers. Our sensitivity analyses (see Results) suggest that the PAC effects that we document are unlikely to be biased by the non-random nature of our sample. Biological paternity analyses were not conducted, and we expect any non-paternity events would act to attenuate observed PAC associations.

We note that the current analyses partially overlap with our previous analysis of PAC and GPAC in this population [4]. This manuscript incorporates additional and improved TL measures [7] and an expanded, more accurate dataset of ancestral ages spanning two more generations (table 1). Importantly, this dataset allows an analysis of GPAC effects with over five times the sample size of our previous publication from this sample [4]. For future meta-analyses, the current results supersede any past analyses.

(a) Family member birth dates

Family member birth date data used to calculate parental ages at conception came from two complementary sources. First, at all surveys, household rosters were gathered. From these, we gathered the birth dates of family members who happened to be living in households together with the focal studied individuals. Second, in 2016, we administered family tree surveys to 713 of the mothers and 712 of the fathers of the 1983–1984 born cohort members.

(b) Telomere length analysis

DNA was extracted from venous blood and TLs were measured using the monochrome multiplex quantitative polymerase chain reaction assay as described previously [7,8]. Since the coefficient of variation (CV) has recently been recognized to be an invalid statistic to compare TL measurement reliability across studies, we instead used the intraclass correlation coefficient (ICC; [9,10]). Specifically, we assayed samples twice and calculated inter-assay ICC values using their within-run mean values (the same sample run in triplicate within runs). In the analyses of data from 2005, 873 samples were run separately in triplicate on two separate runs because of initially high intra-assay CVs. Using mean T/S values from the first and second run, resulted in an ICC(1) of 0.81 (95% CI: 0.79–0.84). For the 2016 samples, a plate of samples ($n = 95$) was assayed an additional time, which yielded an ICC(1) of 0.79 (95% CI: 0.70, 0.86). Of these 95 samples, 61 were fathers with an ICC(1) of 0.79 (95% CI: 0.68–0.87) and 34 were mothers with an ICC(1) of 0.79 (95% CI: 0.63–0.89). Intra-assay CV measures for 2016 father and mother samples were 0.09 and 0.10 respectively. See electronic supplementary material for additional details.

(c) Statistical methods

TL measures were z-score standardized within each group (offspring 2005 blood collection, mother 2005 blood collection, mother 2016 blood collection, and father 2016 blood collection) to make results across groups more comparable. All PAC values are in years, so β values of PAC predicting TL can be interpreted as standard deviation changes in TL per year change in PAC. All regression models that examined PAC effects on descendant TLs controlled for any/all intermediate male ancestors PAC. For example, we controlled for the father's age at conception when examining the paternal grandfather's GPAC effect on his grandchild. We did this to be more confident that the PAC effect of older ancestors was independent and additive to PAC effects of intermediate generations. In the case of

maternal grandfather's PAC effect, his age at the time of the birth of the mother was included as the main predictor but since there are no male intermediate ancestors, no additional controls were included. When statistical interactions or quadratic effects were included in models, these variables were first centred before transformation to remove non-essential collinearity. To control for potential population structure effects, we included principal components (PCs) of genome-wide genetic variation as controls (described in more detail in electronic supplementary material). Beta values from different regression models were compared with Wald tests using a sandwich estimator for the variance–covariance matrix (using Stata's suest followed by test commands; [11]). Meta-analysis of effects used fixed effects models with Stata's metan command and random-effects meta-regressions with the metareg command. To calculate statistical differences between correlation coefficients, we used the Fisher transformation technique [12,13].

All statistical models, unless otherwise noted (electronic supplementary material), were pre-registered at the Open Science Framework (OSF; https://osf.io/h47us/). Models were designed and coded with the outcome measure, TL, replaced with random numbers to allow designs to be attentive to missingness. Only after we posted analysis methods to OSF did we add real TL into the analysis script.

3. Results

This study builds on past work from a longitudinal health study of humans in Cebu, Philippines [6]. In 2005, venous blood was drawn from mothers ($n = 1881$, age 48.7 ± 6.1) and offspring ($n = 1759$, age 21.7 ± 0.3) and TL later measured in these. In 2016, 713 of the mothers and 712 of the fathers of the 1983–1984 born offspring were interviewed. Of the parents, 641 mothers (age 59.1 ± 5.3) and 640 fathers (61.1 ± 5.6) donated venous blood from which we measured TL. TL measures from 2005 negatively correlated with age and male offspring had shorter TLs than females [7,14]. Similarly, in 2016 mother, and father samples TL showed negative associations with age ($\beta = -0.034$, $p < 0.0001$ and $\beta = -0.038$, $p < 0.0001$, respectively). Mother TL measures from 2005 and 2016 were correlated at $r = 0.47$ ($p < 0.0001$; although it is important to note that qPCR analysis methods were adjusted to maximize this correlation—see electronic supplementary material).

(a) Does TL inheritance vary by the sex of parent?

Pearson correlations between parent–offspring TL were calculated after residualizing on age, and for offspring also on sex and age by sex interaction. Offspring and mother 2005 TL measures showed a correlation of 0.296 ($n = 1495$, 95% CI 0.249–0.342, $\beta = 0.297$, s.e. = 0.025). Using 2016 parental TL measures only for which complete case trio TL data were available ($n = 617$), mother–offspring pairs showed a non-significantly greater ($p = 0.085$) correlation of 0.190 (95% CI 0.113–0.265, $\beta = 0.195$, s.e. = 0.041) than father–offspring pairs correlation of 0.094 (95% CI 0.015–0.172, $\beta = 0.098$, s.e. = 0.042).

(b) Generational depth of PAC association

Estimates of PAC associations with descendant's TL are shown in table 2. In the overall estimates, illustrated in figure 1, we found a highly significant PAC association ($p < 0.00001$), a significant GPAC ($p = 0.036$), and no GGPAC ($p = 0.515$) or GGGPAC ($p = 0.947$) associations. The overall paternal versus maternal (FF versus MF in table 2) GPAC estimates were virtually identical.

Based on the semi-conservative nature of DNA inheritance, we expected that the PAC association would decrease in half for each more distant generation (e.g. that GPAC effect would be half of PAC effect). To assess the hypothesized dilution in PAC association across generations, and if this varies by the sex of intermediate ancestors, we conducted a meta-regression using the number of intermediate male ancestors and number of generations as predictors. The semi-conservative nature of inheritance patterns was modelled as $\frac{1}{2}^{\text{generational depth}}$ as a predictor of PAC effect size (e.g. $\text{PAC} = \frac{1}{2}^1 = \frac{1}{2}$, $\text{GPAC} = \frac{1}{2}^2 = \frac{1}{4}$, $\text{GGPAC} = \frac{1}{2}^3 = 1/8$). Number of intermediate male ancestors were transformed in the same manner, since we expected that any difference in the strength of inheritance patterns between males and females would also exponentially decrease with each generation and thus this transformation would better encompass this multiplicative (as opposed to additive) relationship. In this meta-regression model predicting PAC effect size, generational depth was a significant predictor ($\beta = 0.0472$, $p = 0.008$) while the number of intermediate male ancestors was not ($\beta = -0.0079$, $p = 0.340$). Since the intermediate male ancestor term was not significant, we re-ran our analyses without this term and found a similar effect of generational depth ($\beta = 0.0376$, $p = 0.007$; 95% CI 0.0117–0.0635, see electronic supplementary material, figure S2). This implies a predicted PAC effect of 0.0127 and a GPAC effect of 0.00328—a 3.87-fold decrease in effect size between generations. Similarly, using effect estimates from table 2, the GPAC effect was threefold smaller than the PAC effect.

To estimate the consistency of the observed dilution in the PAC effect with our predicted twofold reduction, we ran a simulation analysis. Briefly, in our simulations, we assumed the observed PAC effect size in our data, a GPAC effect half of the observed PAC effect and the same sample sizes as our analyses (table 2: $n = 3282$ in PAC and 2913 in GPAC analysis; see electronic supplementary material for more details). After 10 000 repetitions of this simulation, a halving of the PAC effect yielded a wide 95% confidence interval of 0.96–9.61-fold reduction in PAC effect with each generation. Thus, our observed threefold reduction in effect between PAC and GPAC is well within what we would expect to find if the actual effect were twofold (although given the low power, also consistent with many other possible effect sizes).

(c) Potential confounding of PAC effect

Although there is no definitive evidence that PAC is a direct cause of the longer TL in offspring, the evidence is converging across studies in support of this interpretation (see below). To continue to explore these and other nuanced issues of the PAC effect, we added in various additional variables to the regression models shown in table 2.

(i) Is the PAC effect moderated by age of offspring?

Since the PAC associations were least evident in the oldest cohort of individuals, the fathers (table 2), we examined if the association strength reduced with age within the mother and father cohorts (the offspring cohort all had such similar ages that power is minimal for this test in this

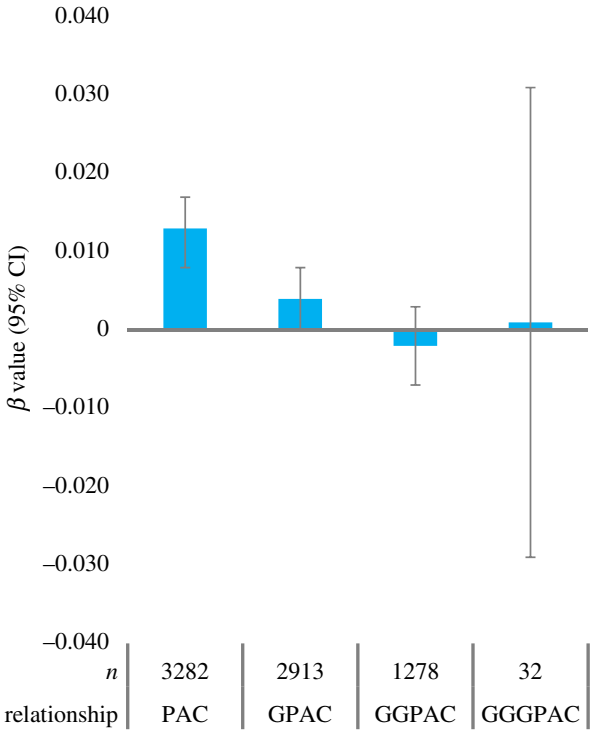

Figure 1. Paternal age at conception (PAC) associations with descendants' telomere lengths (TL). Summarizes results from table 2. GPAC signifies grand-paternal age association with grandchildrens' TL, GGPAC great-grand-paternal, GGGPAC great-great grand-paternal.

subgroup). There was no evidence for variability in PAC association by own age in either group (p-values for interaction terms greater than 0.318). Additionally, for the subset of mothers who had TL measured twice, we examined if PAC varied in predicting baseline versus follow-up TL 11.2 years later and found no difference ($n = 613$; baseline $\beta = 0.0179$, $p = 0.001$, 95% CI 0.0078–0.0280 versus follow-up $\beta = 0.0110$, $p = 0.029$, 95% CI 0.0011–0.0208; p for difference = 0.216).

(ii) Does PAC influence TL attrition?

It is possible that the PAC association is due to PAC influencing TL attrition rates in offspring [15–17]. For example, perhaps older fathers provide a better environment that helps to buffer the rate of TL attrition in their offspring compared to younger fathers. Longitudinal measures of early childhood TL dynamics, which are likely to be most informative about early life-rearing effects, are unavailable in this study. Still, it is possible that rates of TL attrition continue to be influenced by PAC later in life. For a subset of mothers for whom TL was measured longitudinally over 11.1 years (±0.15 s.d.), we examined whether PAC or GPAC predicted ΔTL values. Neither PAC ($n = 613$, $\beta = -0.00056$, $p = 0.25$), paternal GPAC ($n = 273$, $\beta = 0.00053$, $p = 0.34$), nor maternal GPAC ($n = 249$, $\beta = -0.00036$, $p = 0.53$) predicted ΔTL.

Table 2. Paternal ages predicting descendants' telomere lengths[a]. Numbers in italics represent significant values at $p < 0.10$ and in bold $p < 0.05$.

	cohort 1[b]		cohort 2[c]		cohort 3[d]		overall[e]		
ancestor[f]	β	n	β	n	β	n	β	n	I^2
PAC	**0.014*****	1738	**0.019*****	913	0.003	631	**0.013*****	3282	**73.1***
FF	**0.010***	833	–0.001	307	0.000	297	0.004	1437	40.1
MF	0.004	906	0.005	276	0.002	294	0.004	1476	0
GPAC	**0.007***	1739	0.002	583	0.001	591	**0.004***	2913	0.0
FFF	*0.008*†	325	*0.059*†	9	–0.020	9	*0.008*†	343	*65.8*†
FMF	–0.006	326	–0.032	6	—	—	–0.007	332	0
MFF	–0.008	306	—	—	–0.026	6	–0.009	312	0
MMF	–0.004	278	0.008	7	–0.034	6	–0.005	291	10.0
GGPAC	–0.001	1235	0.019	22	**–0.026***	21	–0.002	1278	*44.0*†
FFFF	0.017	10	—	—	—	—	—	—	
MFFF	–0.082	9	—	—	—	—	—	—	
MFMF	0.020	6	—	—	—	—	—	—	
MMMF	–0.051	7	—	—	—	—	—	—	
GGGPAC	0.001	32	—	—	—	—	0.001	32	**55.5****

[a]Each β and coupled n value are from independent regression models controlling for age and intermediate paternal ancestors as applicable. More specific statistics including standard error values are given in electronic supplementary material, table S7.

[b]Cohort born in 1983–1984 and TL measured in 2005. Models additionally controls for sex and age × sex.

[c]Mothers of 1983–1984 born cohort members with TL measured in 2005. Models control for age.

[d]Fathers of 1983–1984 born cohort members with TL measured in 2016. Models control for age.

[e]Fixed-effect meta-analysis combined estimates across the three cohorts.

[f]Ancestor or ancestor group (e.g. PAC indicates paternal age at conception, GPAC: grand-paternal age at the conception of intermediate ancestor; MF: mothers father's age at mothers conception; MMF: mother's mother's father's age at mother's conception). GPAC, GGPAC or GGGPAC ancestor groups are meta-analysis combined estimates across ancestors within that ancestor group (e.g. GPAC combines FF and MF).

$^{\dagger} < 0.10$; $^{*}p < 0.05$; $^{**}p < 0.01$; and $^{***}p < 0.001$. '—' indicates cells intentionally left blank because of insufficient data to model.

(iii) Is the PAC effect linear?

A linear versus curvilinear PAC effect may indicate different underlying biological or demographic processes [3]. No evidence for nonlinearities was found from testing all regression models from table 2 (p-values of squared terms greater than 0.10; see electronic supplementary material, table S4).

(iv) Maternal versus paternal age

Previous analyses suggest that, when PAC and maternal age at conception (MAC) are included in the same models, PAC effects generally increase in magnitude compared to models not including MAC [3]. MAC effects on TL when controlling for PAC are generally slightly negative [3]. Supported by simulation analyses (electronic supplementary material) these past results suggest that MAC has a slightly negative effect on TL which, when not controlled for, slightly attenuates the observed PAC effect. Where data were available, we added in MAC, GMAC and GGMAC to all regression models. Contrary to expectations, overall, PAC, GPAC and GGPAC beta values tended to decrease slightly, but non-significantly when MAC, GMAC and GGMAC were, respectively, added to regression models (electronic supplementary material, figure S3). Consistent with expectations, when we included both PAC and MAC (and grandparental equivalents) in the same model, PAC beta values were non-significantly larger than MAC, and GPAC than GMAC (electronic supplementary material, figure S4). However, GGPAC values were non-significantly smaller than GGMAC.

(v) Confounding by income, urbanicity or ancestry?

The PAC effect could be due to social or other factors that influence both TL and PAC. PAC effects showed virtually no change when adding in controls for log-household income and urbanicity nor PCs of genome-wide genetic variation (electronic supplementary material, table S5).

(vi) Do men with different reproductive pacing have different TLs?

Since longer TL predicts better health and longevity, it is plausible that the PAC effect is due to men with longer TL being more likely to reproduce at later ages and pass on their longer TLs to their offspring [17,18]. Another possibility is that sperm TL does not change with age but that men with shorter TL have worse overall health and longevity prospects. These men might value current mating potential more than future mating potential and be more likely to sire children at a younger age (for instance, if they exhibit 'fast' life-history characteristics more generally). In an effort to assess these possibilities, we included the paternal age at youngest sibling's conception (PAYC) or paternal age at oldest sibling's conception (PAOC) in all regression models for which these data are available. That is, we examined whether PAC, PAYC or PAOC are stronger predictors of an offspring's TL.

As PAC is substantially correlated with PAYC and PAOC, which could limit the ability to distinguish the relative causal role of these three variables, we ran a series of simulation models. These models generally suggested that our statistical power to distinguish between these alternative causal scenarios was quite limited. Thus, while our results are generally consistent with PAC having more of an effect than PAYC or PAOC, a more definitive assessment of this question will require a substantially larger sample size (electronic supplementary material for more details).

4. Discussion

Using a multi-generational dataset from the Philippines, this study helps to clarify inheritance patterns of human TL. In addition to examining parent–offspring correlations in TL, and the PAC effect on TL, we were able to more rigorously evaluate the GPAC effect on TL and begin to examine GGPAC and GGGPAC effects.

Several past studies have examined the strength of correlations between mother–offspring versus father–offspring TLs in humans, but these results have been inconsistent [19–21]. Using complete mother–father–offspring trio data, we showed a non-significantly ($p = 0.085$) stronger association in mother–offspring pairs than father–offspring pairs. These results are unlikely to be influenced by differences in measurement error between mothers and fathers since mother and father 2016 TL measures showed virtually identical levels of internal reliability (see Material and methods).

Previous analyses in humans have consistently found that the offspring of older fathers have longer telomeres. In our past, more limited analysis of this Filipino population (table 1), we showed evidence for a paternal grandfather age effect on TL which was similar in size to the PAC effect, but no evidence for maternal grandfather age effect [4]. Using an expanded dataset, here we report evidence for a grand-paternal age (GPAC) effect that does not differ between maternal and paternal grandfathers. The GPAC effect (overall across maternal and paternal grandfathers) was an estimated threefold smaller than the PAC effect. While this reduction in PAC effect is greater than the twofold expected reduction based on the semi-conservative nature of DNA inheritance, our simulation analysis suggests that we had limited power to precisely estimate this effect size and that our estimated effect is well within the broad 95% confidence interval for a twofold reduction effect.

We did not find significant evidence for GGPAC or GGGPAC effects. However, this was likely due to limited statistical power. Assuming a halving of the PAC association with each successive generation and the mean effect size observed for PAC in table 2, we had 94.6% power to detect a GPAC association, 49.2% power to detect a GGPAC association, and only 5.4% power to detect a GGGPAC association. It is important to note that the positive PAC and GPAC effects do not imply an inevitable increase in TL across generations. The more likely and evolutionarily stable scenario is that TLs will tend to shorten when PACs are below the average PAC in the population and lengthen when above this average age [3,22].

As we discuss elsewhere in more detail, there is strong converging evidence that the PAC association in humans is due to continual increases in sperm TL with age [3]. Measured in the same men cross-sectionally, sperm TL shows an increase with age while blood TL decreases [23], suggesting longer sperm TL observed in older men is not due to a selection bias (e.g. men with longer TL being more likely to donate sperm). Contrary to mortality selection biases, which would lead to the expectation of nonlinearities in the PAC effect, the PAC effect has been found to be linear

in this and other studies [3]. Additionally, the PAC effect is robust to a variety of controls in this and other studies, the PAC effect is similar in diverse populations, and it does not vary with offspring age in this or other studies [3]. In this study, we also examined whether a father's age at the conception of his first and last child influences his offspring's TL more than the PAC of that offspring. Simulation analyses suggested that this analysis had limited power, but results were slightly more consistent with a PAC effect than a PAC of first or last child effect. Our findings are broadly consistent with converging evidence that the PAC effect reflects the lengthening of sperm TL with age.

Despite the strong converging evidence for the human PAC effect being caused by continual increases in sperm TL with age, other research designs could shed further light on this topic. For example, the examination of sperm TL from the same men over time could help to more clearly establish a longitudinal change. Similarly, comparing the TL of siblings sired by the same father at different ages could also support longitudinal changes in sperm TL. Non-human studies permit experimental manipulations to address the nature of PAC effects [16–18,24]. However, telomere biology varies considerably across species [25,26] and the direction and magnitude of PAC effects on TL appear to vary considerably as well [3,17,27]. Thus, even strong experimental evidence of the biological mechanisms accounting for the PAC effect in one species should not be extrapolated uncritically to other species.

We have suggested that the PAC effect on TL may represent a case of adaptive intergenerational genetic plasticity in which the TL fathers pass onto their descendants varies based on their own age at reproduction [3,5]. The evidence here shows that the PAC effect persists across at least two generations and is transmitted with equal strength via male and female intermediate ancestors. While we fail to demonstrate evidence of the PAC effect persisting across more than two generations, statistical power was limited for these analyses and we predict that future, better-powered studies will demonstrate these effects. Should the PAC effect be conveying information about past environments, the multigenerational nature of the effect could allow a more reliable signal. Instead of a child receiving TLs that are influenced by just the PAC, their TLs are also influenced by both their maternal and paternal grandfathers, and possibly also more distant ancestors. We note that while the PAC effect of each individual male ancestor decreases with generational distance, the number of male ancestors doubles in parallel. Thus, it is possible that each generation has a roughly similar cumulative effect on progeny TL, with more distal generations effectively sampling a wider array of ancestors. Future research should consider whether the PAC effect is modified by physiological experiences of the father and work to trace the phenotypic and fitness effects of PAC influenced changes in TL on descendants.

Data accessibility. This article has no additional data.

Authors' contributions. D.T.A.E. conducted all statistical analyses and wrote the manuscript. D.T.A.E. and C.W.K. co-wrote the grant for and designed the 2016 data and sample collection protocols. M.G.H. and D.T.A.E. supervised the 2005 and 2016 telomere length analyses, respectively. D.T.A.E. and P.H.R. conducted the 2005 and 2016 telomere length analyses, respectively. N.R.L. supervised the implementation of all 2016 survey and sample collections. All authors commented on and approved this manuscript.

Competing interests. We declare we have no competing interests.

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