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Intergenerational effects of maternal lifetime stressor exposure on offspring telomere length in Black and White women

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Abstract

Background. Although maternal stressor exposure has been associated with shorter telomere length (TL) in offspring, this literature is based largely on White samples. Furthermore, timing of maternal stressors has rarely been examined. Here, we examined how maternal stressors occurring during adolescence, pregnancy, and across the lifespan related to child TL in Black and White mothers.

Method. Mothers (112 Black; 110 White; $M_{age} = 39$) and their youngest offspring ($n = 222$; $M_{age} = 8$) were part of a larger prospective cohort study, wherein mothers reported their stressors during adolescence (assessed twice during adolescence for the past year), pregnancy (assessed in midlife for most recent pregnancy), and across their lifespan (assessed in midlife). Mother and child provided saliva for TL measurement. Multiple linear regression models examined the interaction of maternal stressor exposure and race in relation to child TL, controlling for maternal TL and child gender and age. Race-stratified analyses were also conducted.

Results. Neither maternal adolescence nor lifespan stressors interacted with race in relation to child TL. In contrast, greater maternal pregnancy stressors were associated with shorter child TL, but this effect was present for children of White but not Black mothers. Moreover, this effect was significant for financial but not social pregnancy stressors. Race-stratified models revealed that greater financial pregnancy stressors predicted shorter telomeres in offspring of White, but not Black mothers.

Conclusions. Race and maternal stressors interact and are related to biological aging across generations, but these effects are specific to certain races, stressors, and exposure time periods.

Introduction

Racial disparities in health have reached a critical juncture, particularly between Black/African and White individuals, with lower life expectancy and earlier onset of chronic diseases of aging for Black relative to White individuals (Mays, Cochran, & Barnes, 2007; Murray et al., 2006; Williams & Mohammed, 2009). Evidence points to the influence of racism-related stressor exposures and coping strategies that can have long-term health consequences (Turner, 2013). In terms of stressor exposure, African Americans experience more acute and chronic life stressors as compared to their White counterparts, even after adjusting for socioeconomic status (SES) (Brown, Mitchell, & Ailshire, 2020; Sternthal, Slopen, & Williams, 2011). In terms of coping, Black Americans may have adopted unique coping resources to withstand chronic stressor exposures that are the result of oppressive socio-historical contexts.

One commonly used strategy by Black Americans is termed John Henryism, a style characterized by a high-effort, active coping that might have detrimental physical health effects due to the amounting physiological burden (Robinson & Thomas Tobin, 2021). Another coping mechanism at the intersection of race and gender is the Superwoman Schema, which reflects a desire on the behalf of Black women to present an image of strength, suppress emotions, avoid vulnerability, succeed regardless of resources, and help others (Woods-Giscombé, 2010). Although initially conceptualized as a positive coping mechanism to defy gendered racism, this may have negative health consequences (Woods-Giscombé et al., 2019). Formulations such as the 'weathering' hypothesis suggest that the rate of biological aging of African American women may be accelerated as a consequence of the cumulative impact of

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racism-related stressor exposures and the need for the development of unique coping mechanisms that may have long-term health consequences (Forde, Crookes, Suglia, & Demmer, 2019).

The negative health effects of experiencing major life stressors can also span multiple generations. Maternal stressor exposure during the perinatal period can have important long-term health consequences for the offspring, which has led to models such as the Developmental Origins of Health and Disease (DOHaD) hypothesis (Barker, 2007). Black mothers report more pregnancy stressors as compared to White mothers (Liu, Giallo, Doan, Seidman, & Tronick, 2016; Lu & Chen, 2004). The prenatal period is thus an important time during which racial health disparities might originate, though research is scarce in marginalized communities (Conradt, Carter, & Crowell, 2020). Furthermore, maternal stressor exposure during her own childhood/adolescence or during her lifetime can confer health risks on her offspring such as child psychopathology (Bödeker et al., 2019; Rijlaarsdam et al., 2014). Understanding when during the life course (adolescence *v.* pregnancy *v.* lifespan) maternal stressors are most impactful for offspring health is an important research question. Here, we focused on immune cellular age as a key outcome.

Telomere dynamics have been proposed to be a key mechanism in translating psychosocial stressors into biological changes that increase risk for health problems within and across generations (Epel, 2020). In this context, telomere length (TL) may be a potentially useful biomarker for understanding intergenerational stress effects and race/racism-related health disparities (Selvaraju, Phillips, Fouty, Babu, & Geetha, 2021). Telomeres are DNA-protein complexes that serve as protective caps at the ends of chromosomes, maintaining chromosomal integrity. With each cell division, telomeres are not fully replicated and shorten with age, constituting a biological marker for aging (Blackburn, Epel, & Lin, 2015). Critically short telomeres can trigger apoptosis or cellular senescence cascades. However, the enzyme telomerase, a ribonucleoprotein reverse transcriptase, can add repetitive nucleotide sequences to the ends of the DNA, thereby counteracting telomere shortening (Blackburn et al., 2015).

Deterioration of the telomere/telomerase maintenance system is implicated in physical (Haycock et al., 2014; Ma et al., 2011) and psychiatric diseases (Darrow et al., 2016), and has been linked to stressor exposure, particularly for childhood stressors (Epel & Prather, 2018). Notably, race-based differences in telomere dynamics have been shown, with most studies finding that Black individuals have longer telomeres (Brown, Needham, & Ailshire, 2017; Codd et al., 2021; c.f. Geronimus et al., 2010) and higher levels of telomerase (Kroenke et al., 2012).

Research also has suggested the presence of intergenerational stressor effects on TL, with most studies primarily investigating the sensitive period of pregnancy. In particular, maternal stressors occurring during pregnancy have been associated with shorter TL in newborns (Entringer et al., 2013) and young adults (Entringer et al., 2011). These effects likely occur via stress-related maternal-placental-fetal biological (e.g. oxidative, immune, endocrine, metabolic) pathways that may exert a 'programming' effect on the developing telomere biology system (Entringer, de Punder, Buss, & Wadhwa, 2018). Several independent cohort studies have now replicated the effects of maternal pregnancy stress on offspring TL (Carroll, Mahrer, Shalowitz, Ramey, & Schetter, 2020; Marchetto et al., 2016; Send et al., 2017; Verner et al., 2021), though some studies have not found an association (Ämmälä *et al.* 2020; Slykerman *et al.* 2019). A striking feature of this literature is the scarcity of non-White, particularly Black samples. Indeed, the three largest studies conducted to date included all-White, European participants (Ämmälä et al., 2020; Slykerman et al., 2019; Verner et al., 2021). The few studies that have enrolled Black participants either had small samples (Carroll et al., 2020; Entringer et al., 2013; Marchetto et al., 2016) or did not test for pregnancy stress-by-race interactions (Enlow et al., 2018; Esteves et al., 2020).

It has been proposed that stressors interact with race to shape offspring health outcomes, though this is rarely examined (Giscombé & Lobel, 2005). Racial differences in prenatal stressor exposure (Liu et al., 2016; Lu & Chen, 2004), culturally relevant coping mechanisms (John Henryism; Superwoman Schema), and racial differences in TL (Brown et al., 2017; Codd et al., 2021) may yield different associations. Examining the complex interactive effects of maternal race and stressor exposure with offspring TL may advance our understanding of racial disparities in offspring health and longevity.

Furthermore, maternal stressor exposure occurring outside the pregnancy period has rarely been examined in relation to offspring TL, despite new developments that extended the DOHaD hypothesis to the preconception period (Keenan, Hipwell, Class, & Mbayiwa, 2018). The DOHaD extension argues that pregnancy health depends on the integrity of parental physiological systems prior to conception and thus includes maternal childhood/adolescence or cumulative experiences across the lifespan. Adolescence, particularly late adolescence, is an important developmental period during which time stressor exposures can shape the responsiveness of stress-related physiological systems that may in turn impact maternal-fetal processes in pregnancy (Keenan et al., 2018). Similarly, maternal lifelong stressor exposure may contribute to fetal programming of the offspring's telomere biology system via long-term alterations of stress response systems as well as oocyte quality and mitochondrial function across oocyte development (Entringer et al., 2018). To our knowledge, however, no study has examined the cumulative effects of maternal stressor exposure occurring across the entire lifespan on offspring TL.

One study examined maternal stress occurring in the year prior to conception and did not find associations with offspring buccal TL (Carroll et al., 2020). In addition, two studies have investigated the effects of childhood adversity on offspring TL, showing that maternal childhood adversity was associated with shorter infant TL across infancy (Esteves et al., 2020). Furthermore, retrospective reports of mother's familial emotional support and sexual abuse in childhood have been associated with newborn TL in male infants (Enlow et al., 2018). It is important to replicate these findings and to widen the stressor assessment window to span the entire life course. Lastly, when possible, it is important to test how the intersection of race and stressor exposure impacts offspring TL. Black women are not only exposed to more cumulative stressors, but they also perceive and cope with them differently, which might have differential effects on offspring TL.

To address these issues, we examined how maternal stressor exposure occurring during late childhood/adolescence, pregnancy, and across the life course related to child TL in Black and White mothers. Based on the literature summarized above, we hypothesized that maternal stressor exposure occurring during adolescence, pregnancy, and across the life course would be associated with shorter offspring TL, particularly for offspring of Black mothers.

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Method

Overview of the National Heart, Lung and Blood Institute Growth & Health Study

The National Heart, Lung and Blood Institute Growth & Health Study (NGHS) is a prospective cohort study that recruited Black and White girls at 9–10 years of age from Richmond (CA, USA), Cincinnati (OH, USA), and Washington (D.C., USA). A total of 2379 girls (1209 Black; 1166 White) were enrolled in 1987–88 and followed prospectively from 9/10 to 19/20 years old (for an overview of the NGHS, see Morrison, 1992). The original aim of the NGHS was to examine factors associated with racial disparities in the onset and development of obesity and cardiovascular disease, specifically in Black and White females. After the initial ten-year study, a follow-up study was conducted in midlife ($M_{age} = 39$) that re-enrolled participants from the Richmond (CA) site, which was chosen based on census data showing approximately equal percentages of Black and White children with the least degree of income and occupational disparity between the races. We re-recruited 624 (307 Black; 317 White) of the original 887 participants (459 Black; 428 White) from the Richmond site (73.8% retention rate among eligible women). The follow-up study was further enriched by inviting biological children to participate with their mothers (the original participants) to examine the intergenerational transmission of stress and disease risk.

Participants

Eligibility criteria for adult women were (a) an original NGHS participant from the Richmond, CA site; (b) not pregnant at the time of recruitment and had not experienced a pregnancy, miscarriage, or abortion within the last three months; and (c) neither living abroad nor incarcerated or otherwise institutionalized. Participants' biological children were eligible if they were between the ages of 2 and 17. Of the 645 children who participated, 262 provided valid TL data. For the present study, we focused on data of the youngest (most recent born) child, for which we had measures of stress exposure during pregnancy. Of the 262 children with valid TL data, 222 children were the youngest. In total, there were 222 mother-youngest child dyads with offspring TL data.

Study procedures

Multiple recruitment strategies were used to re-recruit original NGHS participants from the Richmond site (e.g. mailing and telephone follow-up, social media outreach). Eligible participants provided written informed consent and participated in a baseline survey and home/clinic visit, including saliva collection for TL assessment. This study was approved by the Institutional Review Board.

Self-report measures

Sociodemographic information

The survey obtained in midlife ($M_{age} = 39$) assessed sociodemographic information, including maternal race ('Black' or 'White'), ethnicity, age, highest level of education completed, annual household income, child age, and child gender.

Maternal stressor measures

For a timeline of maternal stressor assessments in NGHS, see Fig. 1. Briefly, major life stressors that occurred over the past 12 months were assessed at age 17/18 and 19/20, indicating maternal stressor exposure during adolescence. In addition, during midlife ($M_{age} = 39$), mothers retrospectively reported on the major life stressors that occurred during their most recent pregnancy as well as major life stressors that had occurred across their lifespan.

Maternal adolescence stressors

Prospective measures of maternal life stressors occurring during adolescence were available as part of the historical NGHS. Specifically, a Life Events Scale (LES; Franko et al., 2004) was administered at age 17/18 (40 items) and 19/20 (45 items), assessing if the participant experienced a variety of stressors over the past 12 months. A total LES score was calculated by summing all items across both time points.

Maternal pregnancy stressors

Stressors occurring during the most recent pregnancy were measured retrospectively in midlife ($M_{age} = 39$) using a list of 14 major life stressors (Centers for Disease Control and Prevention, 2005). A total sum was computed for all 14 stressors. Stressors were further categorized into total social stressors and financial stressors.

Maternal lifespan stressors

Mothers' exposure to major stressors occurring over the entire life course was assessed in midlife ($M_{age} = 39$) using the Stress and Adversity Inventory for Adults (Adult STRAIN), which is an online system for assessing cumulative lifetime stressor exposure that has demonstrated very good validity and excellent test-retest reliability (Slavich & Shields, 2018).

Maternal depressive symptoms

Current mood may influence retrospective reports of maternal stressor exposure (Maughan & Rutter, 1997), so sensitivity analyses were conducted controlling for depressive symptoms at the time of stressor assessment. Depressive symptoms were assessed in midlife ($M_{age} = 39$) using the 20-item Center for Epidemiological Studies Depression Scale (CES-D), which measured symptoms over the past week on a 0–3 scale (Radloff, 1977).

Maternal and offspring telomere length measurement

The TL measurement assay was adapted from the published original method by Cawthon (Cawthon, 2002; Lin, Epel, & Blackburn, 2012). Additional details can be found in the online Supplement. The inter-assay coefficient of variation for this study was 2.2% ± 1.6%.

Statistical analysis

All analyses were conducted with IBM SPSS Statistics version 27. Maternal TL was natural log transformed, which improved skewness (Statistic = −0.38, S.E. = 0.17) and kurtosis (Statistic = 1.27, S.E. = 0.33). Offspring TL values above mean + 3 × S.D. were winsorized (6 participants) and then ln transformed, which improved skewness (Statistic = 0.59, S.E. = 0.17) and kurtosis (Statistic = 1.74, S.E. = 0.33).

Although there is no clear guidance on how to model the intersection of maternal race and stressor exposure best quantitatively, the most common statistical methods have been regression models with

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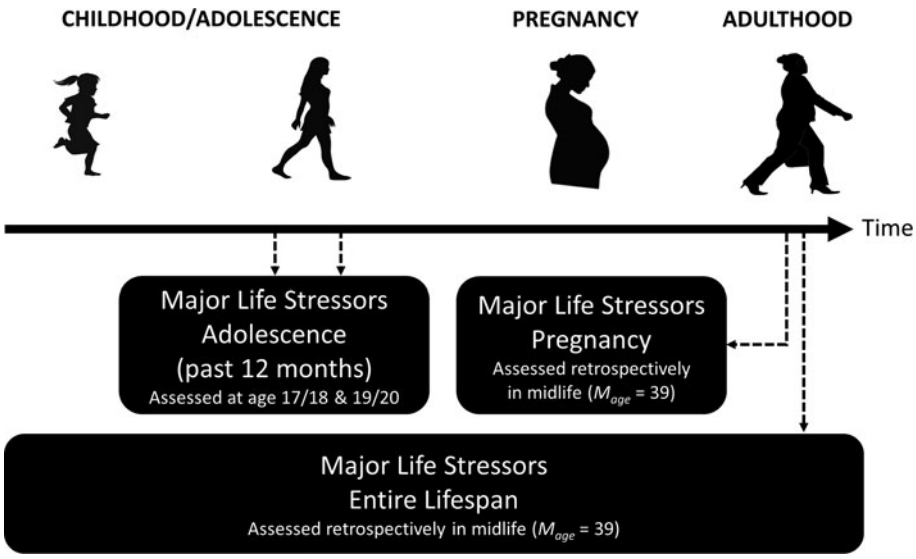

Fig. 1. Timeline of maternal stressor assessments. Note: Dotted arrows indicate the assessment time.

interaction terms and stratified analyses (Bauer et al., 2021; Guan et al., 2021; Wu, 2021). A series of multiple linear regression models examined the interaction of maternal stress exposure and race on offspring TL, controlling for covariates that are known to predict offspring TL, including child age and gender (Factor-Litvak et al., 2016), and maternal TL (Broer et al., 2013). Specifically, we controlled for maternal TL to examine environmental exposure effects while controlling for heritable factors such as genetic variants or direct transmission from germ line telomeres (Haussmann & Heidinger, 2015). Given race-related experiential theories and race-based TL differences, we also examined the effect of maternal stressors on offspring TL in models stratified by maternal race (Shenassa, Rossen, Cohen, Morello-Frosch, & Payne-Sturges, 2017; Williams, Shenassa, Slopen, & Rossen, 2018).

Results

Sample characteristics

Descriptive statistics are shown in Table 1. Mothers had a narrow age range (37–42) due to the cohort design. The majority of participants (63%) experienced at least one pregnancy stressor, with 57% experiencing at least one social stressor and 38% reporting at least one financial stressor. Lower maternal education and household income were associated with greater maternal adolescence, pregnancy, and lifespan stressor exposure (all $ps < 0.05$). Offspring were on average 8 years old (range: 2–17) with the majority of children (70%) being 10 years old or younger. Thus, children clustered around a similar developmental stage.

Notably, demographic characteristics differed across races (Table 1), such that Black mothers reported lower SES as compared to White mothers. However, unlike other studies that are often characterized by structural confounding of race and SES, in the present sample, there was a sufficient representation of higher-SES Black participants and lower SES White participants. Black mothers were exposed to more pregnancy stressors but had longer telomeres than White mothers.

Effects of sociodemographic variables on offspring telomere length

As hypothesized, maternal TL was positively correlated with child TL, $r(208) = 0.27$, $p < 0.001$. There were no significant relations between child TL with maternal race, $t(220) = -1.30$, $p = 0.194$, age, $r(220) = -0.001$, $p = 0.990$, education, $t(220) = -1.89$, $p = 0.060$, and annual household income, $r(210) = 0.005$, $p = 0.940$. Child age was not related to child TL, $r(220) = -0.054$, $p = 0.425$, which was expected given that children clustered around a similar developmental stage. Female gender was marginally associated with longer child TL, $t(220) = -1.91$, $p = 0.058$.

Effects of maternal stressors and race on offspring telomere length

Linear regression models and statistics are presented in Table 2 (models with stress-by-race interactions)[†1] and Table 3 (models stratified by maternal race). First, we examined the effect of *maternal adolescence stressors* and race on child TL (Table 2, Model a). Results showed that count of stressors occurring during mothers' adolescence did not interact with maternal race in relation to offspring TL ($p = 0.720$). Stratified analyses by maternal race (Table 3) also showed no effect of maternal adolescence stressors on child TL within Black and White mothers separately.

Next, we examined the effect of *maternal pregnancy stressors* and race on child TL. Results showed that stressors occurring during mothers' pregnancy and race significantly interacted in predicting offspring TL ($p = 0.044$; Table 2, Model b), such that more maternal pregnancy stressors were associated with shorter offspring TL but only for children of White mothers (Fig. 2). Stratified analyses by maternal race (Table 3) paralleled the overall pattern of a negative association for offspring of White mothers ($p = 0.116$), but not for children of Black mothers ($p = 0.308$), although the effect in offspring of White mothers was not statistically significant.

Based on prior research showing differential effects for financial *v.* social stressors (Puterman et al., 2016), we investigated whether the above-described findings differed by pregnancy stressor type. Only maternal financial pregnancy stressors (Table 2, Model d), and not social pregnancy stressors (Table 2, Model c), significantly interacted with maternal race in predicting offspring TL ($p = 0.024$). Stratified analyses by maternal race (Table 3) showed that experiencing more financial stressors

[†]The notes appear after the main text.

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Table 1. Descriptive statistics for sociodemographic information, adolescence, pregnancy, and lifespan stressors, as well as telomere length (TL) of mother-child dyads

			Entire sample ($n = 222$)	Black mothers ($n = 112$)	White mothers ($n = 110$)
	Category	Variable	Mean (s.d.) or No. (%)	Mean (s.d.) or No. (%)	Mean (s.d.) or No. (%)
Maternal	Sociodemographics	Hispanic, Latino, or Spanish origin, No. (%) yes	8 (3.6)	3 (2.7)	5 (4.6)
		Age, years	39.31 (1.14)	39.37 (1.09)	39.25 (1.20)
		Education, No. (%) less than college degree	138 (62%)*	87 (78%)	51 (46%)
		Annual household income, No. (%) < $ 60 000	90 (43%)*	63 (60%)	27 (25%)
	Adolescence stressors	Total count of stressors during adolescence	14.81 (8.29)	15.64 (8.83)	13.98 (7.65)
	Pregnancy stressors	Total count of stressors during pregnancy	1.87 (2.07)*	2.43 (2.25)	1.31 (1.72)
		Total count of social stressors during pregnancy	1.28 (1.52)*	1.72 (1.68)	0.85 (1.22)
		Total count of financial stressors during pregnancy	0.59 (0.86)*	0.72 (0.92)	0.46 (0.78)
	Lifespan stressors	Total count of stressors across the lifespan	26.89 (15.68)	27.90 (16.40)	25.80 (14.89)
	Cellular aging marker	TL (T/S ratio)	1.19 (0.24)*	1.24 (0.24)	1.13 (0.22)
Child	Sociodemographics	Age, years	8.12 (3.98)	8.41 (4.05)	7.83 (3.91)
		Gender, No. (%) female	119 (54%)	58 (52%)	61 (56%)
	Cellular aging marker	TL (T/S ratio)	1.57 (0.44)	1.60 (0.47)	1.53 (0.39)

* indicates significant race differences ($p < 0.05$).

during pregnancy was associated with shorter TL for children of White mothers ($p = 0.036$) but not Black mothers ($p = 0.410$). Sensitivity analyses further stratified by both maternal race and child gender showed that greater exposure to financial pregnancy stressors was associated with shorter TL only for boys of White mothers ($p = 0.010$; online Supplementary Table S1).

Finally, we examined the effect of *maternal lifespan stressors* and race on offspring TL (Table 2, Model e). Mother's stressors occurring over the entire life course did not significantly interact with maternal race in predicting offspring TL ($p = 0.693$). Stratified analyses by maternal race (Table 3) also did not show an effect of maternal lifespan stressors on child TL within each race group.

Other sensitivity analyses

The models above that used retrospective maternal stressor questionnaires were re-run while controlling for maternal depressive symptoms at the time of stressor assessment to account for possible mood-dependent reporting (Maughan & Rutter, 1997). Importantly, controlling for depressive symptoms did not alter any of the findings (see online Supplemental sensitivity analysis and Table S2). Furthermore, non-significant results for the associations between maternal adolescence and lifespan adversity with race on offspring TL remained non-significant when maternal TL was not included in the regression models (online Supplementary Table S3).

Discussion

This is the first study we are aware of to examine stressor exposure timing effects and race in the transmission of maternal life stressor exposure on offspring TL. Results showed that experiencing more maternal pregnancy stressors was related to shorter offspring TL but only for children of White mothers. Post-hoc analyses further revealed that this effect was only significant for financial pregnancy stressors and for boys of White mothers. Neither maternal adolescence nor lifespan stressors interacted with race in predicting offspring TL. Race-stratified models paralleled the overall pattern, showing that greater financial pregnancy stressors predicted shorter telomeres in offspring of White, but not Black mothers.

Our findings point to pregnancy stressors as influential social-environmental exposures that may shape TL for offspring of White women. It is under intrauterine conditions – when there is the closest overlap between the mother's and the child's biological and psychosocial environment – that maternal stressor effects may be more impactful for offspring health relative to maternal stress occurring prior to conception. This finding has important public health implications as adult TL, which is a function of the initial set point and attrition over time (Aviv, 2008), has been linked to stress-related diseases (Darrow et al., 2016; Haycock et al., 2014; Ma et al., 2011). Therefore, the foundation for later disease susceptibility might be laid during the prenatal period (Entringer et al., 2018). Reducing maternal stressor exposure occurring during the sensitive period of pregnancy may promote lifelong health, and this may occur in part by protecting offspring telomere shortening.

Multiple mechanisms may link maternal prenatal stressors and offspring TL. Maternal epigenetic patterning/marking does likely not transmit strongly, so transmission is not likely due to epigenetics (Entringer et al., 2018). Since all analyses controlled for observed maternal TL – a proxy for biological inheritance – our findings are less likely to be the results of mothers' genetics,

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Table 2. Maternal adolescence, pregnancy, and lifespan stressors predicting offspring telomere length (TL)

Model	Variable	*B*	*Std. Error*	*Beta*	*t*	*p*	r^2_{part}
Adolescence stressors[a]	Adolescence stressors	−0.002	0.00	−0.065	−0.62	0.538	0.002
	Maternal race (0 = White; 1 = Black)	0.014	0.03	0.033	0.48	0.634	0.001
	Adolescence stressors-by-race interaction	0.001	0.00	0.038	0.36	0.720	0.001
	Maternal TL	0.262	0.07	0.262	3.78	**<0.001**	0.064
	Child age	0.000	0.004	−0.005	−0.07	0.947	0.000
	Child gender (0 = boy; 1 = girl)	0.047	0.03	0.114	1.67	0.096	0.013
Total pregnancy stressors[b]	Total pregnancy stressors	−0.022	0.01	−0.217	−1.80	0.074	0.015
	Maternal race (0 = White; 1 = Black)	0.018	0.03	0.044	0.61	0.544	0.002
	Total pregnancy stressors-by-race interaction	0.031	0.02	0.237	2.02	**0.044**	0.019
	Maternal TL	0.259	0.07	0.257	3.67	**<0.001**	0.062
	Child age	0.000	0.004	0.008	0.12	0.907	0.000
	Child gender (0 = boy; 1 = girl)	0.046	0.03	0.112	1.64	0.104	0.012
Social pregnancy stressors[c]	Social pregnancy stressors	−0.017	0.02	−0.128	−1.03	0.307	0.005
	Maternal race (0 = White; 1 = Black)	0.014	0.03	0.034	0.47	0.641	0.001
	Social pregnancy stressors-by-race interaction	0.028	0.02	0.164	1.36	0.176	0.009
	Maternal TL	0.258	0.07	0.255	3.62	**<0.001**	0.061
	Child age	0.000	0.004	−0.009	−0.13	0.898	0.0001
	Child gender (0 = boy; 1 = girl)	0.049	0.03	0.119	1.73	0.085	0.014
Financial pregnancy stressors[d]	Financial pregnancy stressors	−0.062	0.03	−0.256	−2.33	**0.021**	0.025
	Maternal race (0 = White; 1 = Black)	0.016	0.03	0.040	0.57	0.573	0.001
	Financial pregnancy stressors-by-race interaction	0.079	0.04	0.249	2.27	**0.024**	0.024
	Maternal TL	0.255	0.07	0.253	3.64	**<0.001**	0.060
	Child age	0.001	0.004	0.022	0.32	0.751	0.0005
	Child gender (0 = boy; 1 = girl)	0.040	0.03	0.097	1.42	0.158	0.009
Lifespan stressors[e]	Lifespan stressors	0.000	0.002	−0.038	−0.32	0.748	0.001
	Maternal race (0 = White; 1 = Black)	0.019	0.03	0.048	0.61	0.545	0.002
	Lifespan stressors-by-race interaction	0.001	0.002	0.047	0.40	0.693	0.001
	Maternal TL	0.273	0.08	0.275	3.47	**<0.001**	0.070
	Child age	−0.001	0.004	−0.027	−0.34	0.731	0.001
	Child gender (0 = boy; 1 = girl)	0.051	0.03	0.125	1.63	0.105	0.016

Outcome: Offspring TL (winsorized and natural log transformed).
Note: r^2_{part} = Squared semipartial (or 'part') correlation (individual predictor).
Bold values indicate $p < .05$.
[a]Model fit $R = 0.300$; $R^2 = 0.090$; $F_{(6,\ 202)} = 3.33$; $p = 0.004$.
[b]Model fit $R = 0.320$; $R^2 = 0.103$; $F_{(6,\ 195)} = 3.72$; $p = 0.002$.
[c]Model fit $R = 0.304$; $R^2 = 0.092$; $F_{(6,\ 195)} = 3.30$; $p = 0.004$.
[d]Model fit $R = 0.333$; $R^2 = 0.111$; $F_{(6,\ 195)} = 4.04$; $p < 0.001$.
[e]Model fit $R = 0.330$; $R^2 = 0.109$; $F_{(6,\ 152)} = 3.09$; $p = 0.007$.

and of direct transmission of shorter TL through the germline. This strengthens our interpretation that the transmission occurred through the indirect impact of maternal stress exposure on offspring telomere biology via non-heritable pathways (Haussmann & Heidinger, 2015), such as through maternal health behaviors that might affect offspring stress regulation and ultimately impact telomere biology, or through multiple stress-related endocrine, inflammatory, oxidative and metabolic pathways during intrauterine life (Entringer et al., 2018). Evidence for the latter comes from studies examining stress-related biological processes that mediate the effect of stress on telomere biology during adult life. For example, biological markers of inflammation, such as C-Reactive Protein, Interleukin-6, and tumor necrosis factor-α have been linked to telomere shortening (Bekaert et al.,

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Table 3. The impact of maternal adolescence, pregnancy, and lifespan stressors on offspring telomere length (TL), stratified by maternal race

Stratified by	Model	Variable	*B*	*Std. Error*	*Beta*	*t*	*p*	r^2_{part}
White mothers	Adolescence stressors[a]	Adolescence stressors	−0.001	0.003	−0.043	−0.44	0.663	0.002
		Maternal TL	0.256	0.10	0.248	2.56	**0.012**	0.060
		Child age	−0.004	0.01	−0.077	−0.76	0.450	0.005
		Child gender (0 = boy; 1 = girl)	0.032	0.04	0.075	0.77	0.444	0.005
	Total pregnancy stressors[b]	Total pregnancy stressors	−0.020	0.01	−0.158	−1.59	0.116	0.023
		Maternal TL	0.271	0.10	0.262	2.70	**0.008**	0.066
		Child age	−0.003	0.01	−0.048	−0.47	0.643	0.002
		Child gender (0 = boy; 1 = girl)	0.035	0.04	0.082	0.85	0.399	0.007
	Social pregnancy stressors[c]	Social pregnancy stressors	−0.015	0.02	−0.086	−0.87	0.386	0.007
		Maternal TL	0.266	0.10	0.257	2.62	**0.010**	0.064
		Child age	−0.004	0.01	−0.075	−0.74	0.461	0.005
		Child gender (0 = boy; 1 = girl)	0.039	0.04	0.090	0.92	0.360	0.008
	Financial pregnancy stressors[d]	Financial pregnancy stressors	−0.060	0.03	−0.214	−2.13	**0.036**	0.040
		Maternal TL	0.263	0.10	0.254	2.66	**0.009**	0.063
		Child age	−0.001	0.01	−0.027	−0.26	0.794	0.001
		Child gender (0 = boy; 1 = girl)	0.025	0.04	0.058	0.59	0.553	0.003
	Lifespan stressors[e]	Lifespan stressors	−0.0003	0.002	−0.020	−0.17	0.867	0.0004
		Maternal TL	0.239	0.11	0.244	2.13	**0.037**	0.057
		Child age	−0.007	0.01	−0.126	−1.07	0.290	0.014
		Child gender (0 = boy; 1 = girl)	0.034	0.05	0.084	0.74	0.464	0.007
Black mothers	Adolescence stressors[a]	Adolescence stressors	−0.0004	0.002	−0.017	−0.17	0.864	0.0003
		Maternal TL	0.257	0.10	0.253	2.62	**0.010**	0.063
		Child age	0.003	0.00	0.054	0.56	0.574	0.003
		Child gender (0 = boy; 1 = girl)	0.055	0.04	0.138	1.40	0.164	0.018
	Total pregnancy stressors[b]	Total pregnancy stressors	0.009	0.01	0.101	1.03	0.308	0.010
		Maternal TL	0.235	0.10	0.229	2.31	**0.023**	0.052
		Child age	0.003	0.005	0.056	0.57	0.569	0.003
		Child gender (0 = boy; 1 = girl)	0.055	0.04	0.136	1.37	0.174	0.018
	Social pregnancy stressors[c]	Social pregnancy stressors	0.011	0.01	0.091	0.92	0.360	0.008
		Maternal TL	0.236	0.10	0.230	2.32	**0.022**	0.052
		Child age	0.002	0.005	0.051	0.51	0.608	0.003
		Child gender (0 = boy; 1 = girl)	0.056	0.04	0.138	1.39	0.166	0.019
	Financial pregnancy stressors[d]	Financial pregnancy stressors	0.018	0.02	0.083	0.83	0.410	0.007
		Maternal TL	0.235	0.10	0.229	2.30	**0.023**	0.051
		Child age	0.003	0.005	0.062	0.63	0.532	0.004
		Child gender (0 = boy; 1 = girl)	0.054	0.04	0.133	1.34	0.183	0.017
	Lifespan stressors[e]	Lifespan stressors	0.0003	0.00	0.025	0.23	0.816	0.001
		Maternal TL	0.302	0.11	0.290	2.69	**0.009**	0.083
		Child age	0.002	0.01	0.045	0.42	0.673	0.002
		Child gender (0 = boy; 1 = girl)	0.058	0.04	0.144	1.33	0.187	0.020

Outcome: Offspring TL (winsorized and natural log transformed).
Note: r^2_{part} = squared semipartial (or 'part') correlation (individual predictor).
Bold values indicate $p < .05$.
[a]Model fit: White Mothers $R = 0.289$; $R^2 = 0.084$; $F_{(4,\ 100)} = 2.28$; $p = 0.066$; Black Mothers: $R = 0.304$; $R^2 = 0.093$; $F_{(4,\ 99)} = 2.53$; $p = 0.046$.
[b]Model fit: White Mothers $R = 0.329$; $R^2 = 0.108$; $F_{(4,\ 98)} = 2.97$; $p = 0.023$; Black Mothers: $R = 0.304$; $R^2 = 0.093$; $F_{(4,\ 94)} = 2.40$; $p = 0.055$.
[c]Model fit: White Mothers $R = 0.304$; $R^2 = 0.092$; $F_{(4,\ 98)} = 2.49$; $p = 0.048$; Black Mothers: $R = 0.301$; $R^2 = 0.091$; $F_{(4,\ 94)} = 2.35$; $p = 0.060$.
[d]Model fit: White Mothers $R = 0.354$; $R^2 = 0.126$; $F_{(4,\ 98)} = 3.52$; $p = 0.010$; Black Mothers: $R = 0.299$; $R^2 = 0.089$; $F_{(4,\ 94)} = 2.30$; $p = 0.064$.
[e]Model fit: White Mothers $R = 0.313$; $R^2 = 0.098$; $F_{(4,\ 72)} = 1.96$; $p = 0.111$; Black Mothers: $R = 0.344$; $R^2 = 0.118$; $F_{(4,\ 77)} = 2.58$; $p = 0.044$.

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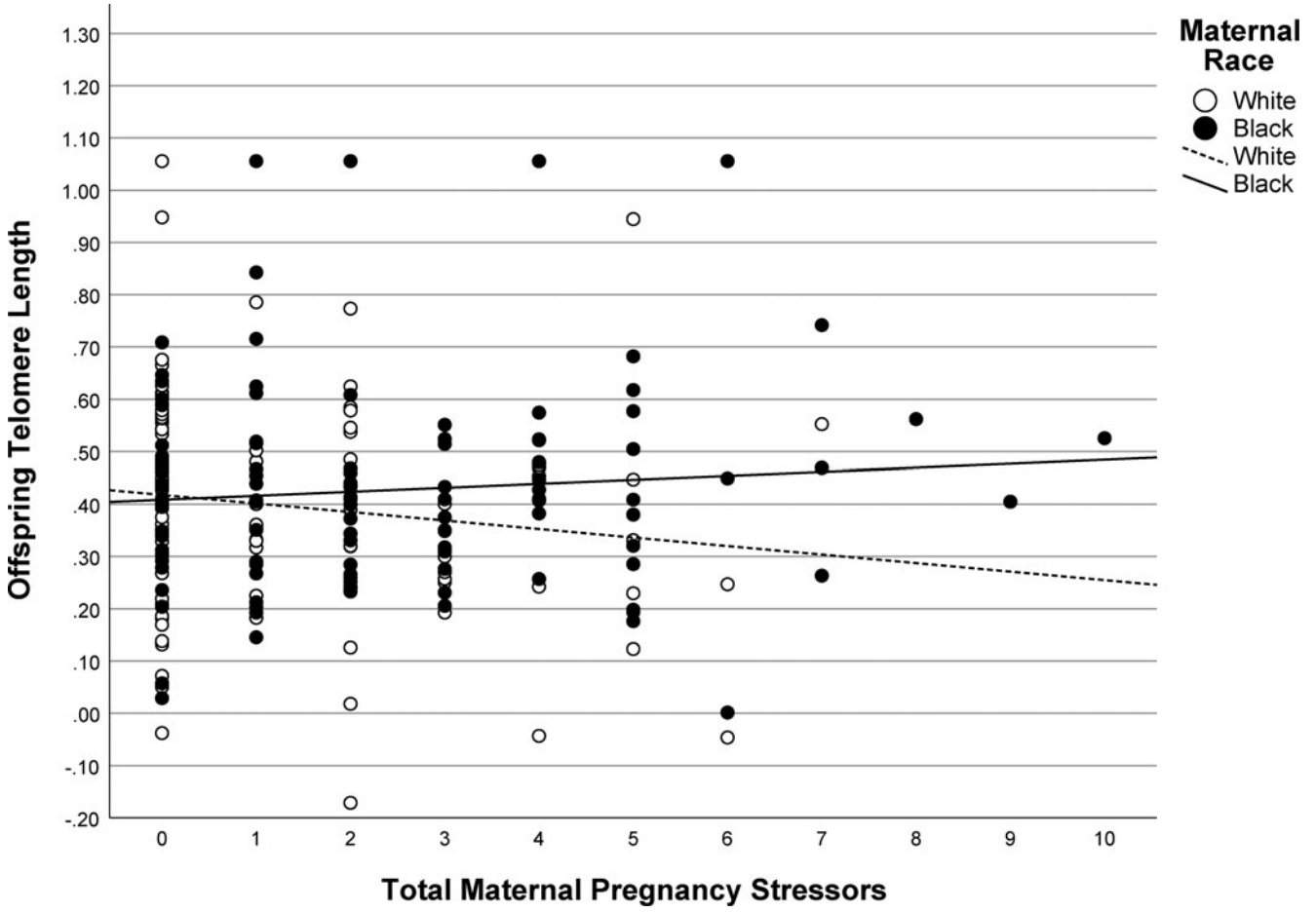

Fig. 2. Association between maternal stressors occurring during pregnancy and offspring telomere length (TL) in children of Black and White mothers. Mothers who experienced more stressors during pregnancy had offspring with shorter TL, but this relation was only significant for children of White but not Black mothers. Note: Offspring TL was winsorized and natural log transformed.

2007; Kiecolt-Glaser et al., 2011; Osler, Bendix, Rask, & Rod, 2016) and telomerase activity (Akiyama et al., 2004). Furthermore, studies that have examined associations in the context of pregnancy reported links between dehydroepiandrosterone sulfate (DHEAS), proinflammatory states (Lazarides et al., 2019), reactive oxygen species (Liu et al., 2017), and maternal estrogen levels (Entringer et al., 2015) with newborn TL.

Our results further highlight the need for a deeper understanding of the intergenerational transmission of stressor effects in Black mothers. The relation between pregnancy stressor exposure and shorter offspring TL was not observed in the offspring of Black mothers, nor maternal stressors at any other time point. Prior studies that have reported links have been primarily performed in White/European samples (Entringer et al., 2011; Send et al., 2017; Verner et al., 2021). Prior studies that included Black participants were either underpowered (e.g. Entringer *et al.* 2013; Marchetto *et al.* 2016) or did not examine interactions between pregnancy stressors and race (Enlow et al., 2018; Esteves et al., 2020). Clearly, further research on race effects in the transmission of pregnancy stressor effects across generations is needed.

We can only speculate about the lack of findings for offspring of Black mothers. Race-based experiential theories may offer an explanation. It is possible that Black mothers – despite their greater stressor exposure – did not perceive the stressors as threatening or upsetting (see Brown et al., 2020), which might buffer the effects of maternal stress on offspring TL. Furthermore, the development of culturally relevant coping strategies adapted by Black women, such as John Henryism (Robinson & Thomas Tobin, 2021) and Superwoman Schema (Woods-Giscombé, 2010), could have mitigated maternal stress effects. These coping styles are likely a 'double-edged sword' (Perez et al., 2022). Although prior studies have found detrimental effects of John Henryism on *physical* health and allostatic load (Robinson & Thomas Tobin, 2021), this coping style has also been shown to be protective for Black women's *mental* health (Bronder, Speight, Witherspoon, & Thomas, 2014), which could have exerted protective effects. Similarly, Superwoman Schema has been linked to detrimental health (Woods-Giscombé et al., 2019), but recent research suggests that effects vary based on the specific Superwoman Schema dimensions, with some aspects being protective (e.g. presenting strength, emotion suppression) and others exacerbating the impact of racial discrimination stress on allostatic load (motivation to succeed, obligation to help others; Allen et al., 2019). Unfortunately, race-based experiential theories that could explain the lack of finding in offspring of Black women could not be directly tested in these data. Examining stress perceptions (in addition to exposures), protective factors and unique coping resources and experiences of Black women warrant further investigation.

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Another explanation for the lack of findings for offspring of Black mothers is drawing on race-based differences in telomere dynamics. Most studies have found that Black individuals have longer TL (e.g. Brown *et al.* 2017; Codd *et al.* 2021). Black American individuals have been shown to have a higher genetic loading for longer telomeres, in part through genes for telomerase (Hamad, Tuljapurkar, & Rehkopf, 2016). Black individuals also have been found to have higher levels of telomerase, tested in at least one study (Kroenke et al., 2012). Therefore, Black mothers in the present study may not only have longer telomeres, but also higher telomerase, which might be protective in the face of stressor exposure. Assessing both TL and telomerase as well as tracking trajectories over time may provide insights into racial differences in the intergenerational transmission of maternal stressor effects.

Post-hoc analyses examining type of pregnancy stressors in relation to offspring TL showed that maternal financial (but not social) stressors were associated with shorter TL in children of White mothers. These findings advance prior studies that did not distinguish between financial and social stressors occurring during pregnancy or that assessed overall stress perceptions. The three most common financial pregnancy stressors in our sample involved partner's job loss, self unwanted job loss, and inability to pay bills. Therefore, our results are consistent with a systematic review that concluded that lower parental SES (often measured by household income) is associated with shorter TL in children (Coimbra, Carvalho, Moretti, Mello, & Belangero, 2017). The stressors assessed here represented chronic financial difficulties and acute life events (job loss), which can both induce a physiological chronic stress response during pregnancy. The resulting financial and employment insecurity and related difficulties to provide for the child may have contributed to a chronic threat state for the pregnant mother, worried about stability and wellbeing of herself and her child – although this interpretation is speculative as the subjective severity of financial difficulties was not directly measured. Furthermore, financial and employment-related stressors might pose long-term chronic stressors that cannot be offset easily by psychosocial resources. Our findings suggest that financial stressors (e.g. financial insecurity, unemployment) may be particularly influential in shaping offspring health and thus important to address in interventions. Future research may also investigate the subjective severity of financial and employment difficulties in addition to whether the stressor occurred or not.

Post-hoc analyses revealed that maternal pregnancy stressors may have sex-specific effects on offspring telomere biology. Specifically, the relation between financial pregnancy stressors and offspring TL was significant for boys, but not girls, of White mothers. Females have longer telomeres than males (Gardner et al., 2014), perhaps due to the protective effects of endogenous estrogen on biomarkers of aging (e.g. upregulating telomerase activity; Lulkiewicz, Bajsert, Kopczynski, Barczak, and Rubis, 2020). Greater exposure to estrogen during pregnancy might make girls more resistant to maternal stress exposure effects. Males are also exposed to higher cortisol levels across trimesters, perhaps mediating sex-specific effects of maternal stressor exposure on newborn TL (Enlow et al., 2019). In sum, our finding is consistent with prior studies showing that males may be more susceptible to maternal exposure effects on offspring TL (Enlow et al., 2018; Enlow, Petty, Hacker, & Burris, 2021), but conclusions are limited by our relatively small cell sizes. Examining sex effects in the maternal transmission of stress on telomere biology remains an open research area.

We did not find an effect of maternal stressors occurring during late childhood/adolescence or across the life course on offspring TL. One explanation is that maternal exposures might have the greatest impact on offspring TL in utero – a sensitive period when the shared socio-biological environment is maximized. Alternatively, it is possible that maternal adolescence and lifespan adversity are less relevant for offspring TL and that preconception stressors in closer proximity to conception (e.g. within 3 months) should be assessed in future studies (Carroll et al., 2020). Although this is the first study to examine the effects of maternal lifespan stressor exposure, prior studies have seen a relation of maternal childhood adversity with offspring TL (Enlow et al., 2018; Esteves et al., 2020). Given evidence that particularly the early developmental years are important for TL (Mayer et al., 2019), it is also possible that our prospective measure captured stressor exposure too late in childhood/adolescence (stressors before age 16 were not assessed). Nevertheless, both adolescence and lifespan stressor exposure can shape stress physiology and ultimately impact pregnancy health (Entringer et al., 2018; Keenan et al., 2018). Therefore, maternal stressors occurring during adolescence or across the lifespan might still directly influence offspring TL mediated by effects on parental germline telomeres pre-fertilization and its effects on the inherited TL by the offspring (Entringer et al., 2018; Haussmann & Heidinger, 2015). In this case, controlling for maternal TL might have obscured effects. Nevertheless, non-significant results for the associations between maternal adolescence and lifespan adversity with race on offspring TL remained when maternal TL was not included in the regression models. In sum, this remains an open research question and our null findings require replication in future studies.

Limitations and strengths

Limitations include that pregnancy and lifetime stressor exposure was based on retrospective self-report, which can be influenced by recall and reporting biases (Hardt & Rutter, 2004), and current mood states (Maughan & Rutter, 1997). Therefore, it is possible that the maternal stressor measures may measure negative biases in autobiographical memory or current affective states rather than actual stressor exposure (Danese & Widom, 2020). However, the stress assessment instruments used both measure stressors that are moderate-to-severe in nature, which are less impacted by recall biases (Krinsley, Gallagher, Weathers, Kutter, & Kaloupek, 2003). Furthermore, lifespan stressor count assessed by the STRAIN has been shown to be robust to recall bias as well as mood and social desirability effects (Slavich & Auerbach, 2018; Slavich & Shields, 2018). Furthermore, to account for mood biases with retrospective measures at least partly, we conducted sensitivity analyses that adjusted for depressive symptoms at the time of assessment, which did not affect the results. Moreover, prior research has shown that interview-based assessments of major life stressors can be reliably recalled across the lifespan (Brown & Harris, 1978). Nevertheless, assessment measures have been largely developed and normed on White samples, so it is possible that unique stressors and experiences of Black women are not fully captured by these state-of-the-art instruments.

Another limitation is that we did not have information regarding the timing of pregnancy stressors, despite evidence that the effect of pregnancy stress on child development is time dependent (e.g. see Project Ice Storm findings; Cao, Laplante, Brunet, Ciampi, & King, 2014; King & Laplante, 2005). Specifically,

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prior research showed that higher maternal perceived stress in the 3rd trimesters was predictive of shorter child buccal TL (Carroll et al., 2020), so this remains an open research question.

In addition, this study only enrolled individuals who identified as Black and White, capitalizing on the original aims of the NGHS to examine factors associated with racial disparities in the onset and development of obesity and cardiovascular disease, specifically in Black and White females. This design provided a unique opportunity to understand the intersection of maternal race and stressor exposure leveraging a population-based study that was designed to enroll a relatively large sample of Black and White women who were within a similar range of SES (sufficient representation of higher-SES Black participants and lower SES White participants). Nevertheless, given the dearth of research in non-White samples (Conradt et al., 2020) and the complexities involved in the analysis of the intertwined effects of social, historical and biological factors on health (Kaufman & Cooper, 2008), future studies designed to do so should specifically examine the intergenerational transmission of stress within Black samples as well as other understudied racial/ethnic communities. Understanding how racial disparities in health originate and transmit across generations is a critical public health problem.

A further limitation is that TL was assessed from saliva samples. This may limit direct comparison with findings obtained from other tissue types given that telomere attrition rates vary by cell types. However, TL obtained from salivary DNA is frequently used in population-based cohorts and correlated with TL obtained from blood samples (Mitchell et al., 2014; Stout et al., 2017). Furthermore, telomeres were measured using quantitative PCR, which may be vulnerable to measurement error. However, we extracted DNA from all samples in the same batch and assayed samples in the same assay batch to minimize potential. The coefficient of variation for this study was therefore low. Another limitation is that TL was only assessed at one time point, limiting conclusions about how maternal stressor exposure and race are associated with offspring telomere attrition rates over time. Furthermore, we did not have a measure of TL at birth. It is plausible that differences in TL were present at birth (e.g. in offspring of Black mothers), but compensated over time through telomere repair mechanisms (Salvador et al., 2016; Zhao, Pan, Liu, & Liu, 2014).

Nevertheless, this study had several strengths including a comprehensive, multi-time point stressor assessment that enabled us to examine how different types of maternal stressors, occurring during different time periods, were related to offspring TL. Additionally, the large sample size of Black and White women enabled us to test stressor-by-race interactions in the intergenerational transmission. Moreover, this study is among the few that controlled for maternal TL (c.f. Send et al., 2017), decreasing the likelihood that mothers directly transmitted shorter TL to their offspring. Finally, we collected data on life stressors in later childhood/adolescents prospectively, before pregnancy, contributing to existing literature using retrospective reports of maternal childhood adversity.

Conclusion

In conclusion, these data show that maternal stressors occurring during pregnancy, and in particular financial stressors occurring during this time period, are associated with offspring TL, but only for children of White mothers. Given the scarcity of research with diverse samples, understanding the intersection of race and the transmission of stress effects across generations remains an important area of research. In terms of targeting interventions, it may be beneficial to identify women with elevated life stressor exposure occurring during the critical period of pregnancy. Interventions could target food insecurity, financial strain and housing instability, and increase social support. Such intervention efforts could improve both maternal and child health outcomes across the lifespan, including aging-related diseases.

Supplementary material. The supplementary material for this article can be found at https://doi.org/10.1017/S0033291722003397

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Note

1 Linear regression models using Hayes (2017) PROCESS Model 1 (with 5000 bootstrap resamples) yielded the same results.

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Conflict of interest. All authors declare no conflict of interest.

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