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Maternal Childhood Adversity Accelerates Epigenetic Aging of Children

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Objective: Although early adversity is strongly related to lifelong health disparities, it is unclear how adversity might confer risk across generations. To investigate, we tested the hypothesis that mothers' childhood adversity was associated with their epigenetic aging and that of their children and examined whether associations differed for Black and White mothers. ***Method:*** Dyads ($N = 215$) of mothers (52% White, 48% Black, $M_{age} = 39.2$, $SD = 1.1$) and children ($N = 215$, 55% female, $M_{age} = 8.3$, $SD = 4.0$, range 2–17) provided saliva samples to assay the Horvath clock and pace of aging calculated from the epigenome epigenetic aging measures. Linear regressions were used to estimate the associations of maternal early adversity measures with the outcomes of maternal and child Horvath clock epigenetic age, as moderated by race. ***Results:*** For Black, but not White mothers, any abuse before age 13, $b = 0.81$, $p = .007$, physical abuse before age 18, $b = 1.69$, $p = .001$, and sexual abuse before age 18, $b = 1.17$, $p = .02$, were associated with significantly greater Horvath age acceleration in their children. In contrast, there was no relation between maternal childhood adversity and mothers' epigenetic aging, and no significant findings for the pace of aging calculated from the epigenome. ***Conclusions:*** Maternal childhood adversity appears to have a greater effect on the epigenetic aging of the children of Black mothers. The effects of systemic racism on Black Americans may interact with maternal childhood adversity to confer additional risk for Black children.

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Public Significance Statement
Black mothers who had experienced abuse in childhood had children with accelerated cellular aging. Childhood adversity in concert with being a member of a minoritized community may influence intergenerational health and aging. Interventions to reduce childhood abuse and systemic racism could improve future health equity.

Keywords: biological aging, health disparities, race, social factors influencing health, intergenerational transmission

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Early life adversity increases the risk for nine out of the 10 leading causes of death in the United States (Hughes et al., 2017; Merrick et al., 2019), as well as all-cause and disease-specific mortality (Galobardes et al., 2004). However, the mechanisms by which adversity confers risk for disease are not well understood and likely involve complex interactions between social, psychological, behavioral, and physiological pathways. Evidence has also linked early life adversity to increased risk of outcomes that exhibit disparities between the health of Black and White Americans, including hypertension, diabetes, stroke, and coronary heart disease (Gilbert et al., 2015; Su et al., 2015). To reduce racial health disparities, there is a need to understand the specific pathways by which adversity in early life increases the risk of disease and results in earlier mortality.

Adverse childhood experiences (ACEs) have not traditionally been conceptualized as a factor contributing directly to health disparities; however, recent research points toward the need to understand the role of childhood adversity and how it may interact with other risk factors to generate health disparities. The Social Hallmarks of Aging framework (Crimmins, 2020) draws from literature on social determinants of health (Braveman et al., 2011) and life course approaches to health disparities. These literature evaluate the impact of adversity across levels (e.g., individual, interpersonal, household, community, population, and societal) and describe the intergenerational transmission of disparities (Jones et al., 2019). The Social Hallmarks of Aging are: (a) lower socioeconomic status, (b) minority status, (c) adverse health behaviors (e.g., smoking), (d) adverse psychological states, and adversity in childhood (e.g., abuse, neglect) and adulthood. The current study addresses the intersectional impact of childhood adversity and minoritized racial background on age-related health outcomes.

Early life adversity may lead to health disparities in part by activating physiological processes that accelerate aging in cells and tissues, especially when such stressors involve the threat of social or physical harm (Furman et al., 2019; Slavich et al., 2023). For example, the weathering hypothesis posits that cumulative exposure to adversity, especially systemic race-based adversity, "weathers" the body, accelerates cellular aging, and leads to early health deterioration (Geronimus, 1992). For Black American women, higher levels of adversity in early life may help to explain well-established disparities in cardiovascular and metabolic health that persist even when accounting for social and economic factors (Bleil et al., 2017).

Epigenetic Age Acceleration as a Pathway to Adult Health Disparities

Epigenetic mechanisms may underlie associations between early life adversity and health disparities between Black and White Americans (Kuzawa & Sweet, 2009). Accelerated cellular aging is reliably indexed by epigenetic clocks, based on the methylation of specific sites in the genome, which reliably predict morbidity and mortality (Horvath & Raj, 2018). Accounting for chronological age, exposure to early adversity has been related to older epigenetic age as indexed by such clocks in both children and adults (Hamlat et al., 2021, Hamlat, Neilands, et al., 2023; Sumner et al., 2019; Wolf et al., 2018). Moreover, studies have found that Black Americans have greater accelerated epigenetic aging than White Americans (Hamlat et al., 2022) and, additionally, among postmenopausal women, Black women have been found to demonstrate accelerated epigenetic aging compared to White women and epigenetic aging mediated the association between race and mortality (Liu et al., 2019). However, other studies have found that Black Americans have decelerated epigenetic aging compared to White Americans, indicating that such effects may depend in part on the specific epigenetic clock used (Horvath et al., 2016).

Maternal Adversity and the Biological Aging of Children

In addition to life adversity shaping the epigenetic aging of an individual, research has found that maternal adversity occurring years before conception can influence the health outcomes of offspring (Bale, 2014; DeSocio, 2018; Keenan et al., 2018). For example, Harville et al. (2010) found that the mother's stress during adolescence had the largest impact on childbirth weight, which suggests that adolescence may be a sensitive period for the effects of maternal adversity on offspring health (Entringer et al., 2018; Keenan et al., 2018). Maternal childhood adversity (i.e., low family support and sexual abuse) has been linked with shorter telomere length in male newborns (Enlow et al., 2018). Maternal ACEs, such as early-life abuse, neglect, and family dysfunction, have also been found to predict shorter telomere length across infancy for offspring (Esteves et al., 2020). Moreover, in a sample of Latina mothers, maternal ACEs were associated with greater epigenetic age acceleration of offspring aged 7 through 14 (Nwanaji-Enwerem et al., 2021). Such evidence suggests that abuse during the mother's childhood, a critical period of biological embedding of trauma, may accelerate the biological aging of offspring.

The Effect of Racialized Maternal Adversity on Child Epigenetic Aging

Whether the influence of maternal adversity on epigenetic aging of offspring differs for Black and White Americans has not been empirically examined, because most of the research on this topic has been conducted with primarily White samples (Mayer et al., 2023). Kuzawa and Sweet (2009) propose a model in which prenatal stress leads to epigenetic changes that ultimately result in an elevated risk of cardiovascular disease in Black adults. Studies of intergenerational health risk do not usually include adversities that Black women frequently face, such as social marginalization and experiences of

structural racism (McKenna et al., 2021), which may lead to greater stress-related health risks among Black women and their children (Giscombé & Lobel, 2005). It is unknown if there is a differential impact of maternal childhood adversity on the epigenetic aging of children of Black mothers when compared to children of White mothers.

The Present Study

To address the gaps in knowledge described above, we investigated if mothers' childhood adversity was related to the epigenetic aging of her and her children and, if so, if there were differences in this association by race. We used the social construct of race as a proxy for race-based adversity and discrimination experiences that may infiltrate the childhood of Black women. To examine longitudinal associations between maternal early adversity and epigenetic aging in children, we used data from a well-characterized longitudinal cohort study, the National Heart, Lung, and Blood Institute Growth and Health Study (NGHS, 1992). To assess epigenetic aging, we used the original Horvath clock (Horvath, 2013) and a newer rate of epigenetic aging index, pace of aging calculated from the epigenome (DunedinPACE; Belsky et al., 2022). Based on the literature summarized above, we hypothesized that greater abuse during childhood experienced by mothers would be associated with faster epigenetic aging of their children. In addition, we explored the extent to which there were differences in these associations between children of Black mothers and children of White mothers.

Method

Participants and Procedure

The initial aims of NGHS were to track cardiovascular risk factors and other health-related variables annually from childhood through young adulthood in Black and White girls. In 1987–1988, the NGHS Contra Costa County cohort (887 girls) was recruited at ages 9 and 10 from public and parochial schools in the Richmond Unified School District area. The original investigators chose Richmond, CA based on census data that showed less income and occupational disparity between Black and White families. More details about the initial study sample recruitment are available (NGHS, 1992). Retention across the 10-year study period was 89% (NGHS, 1992).

In 2016, we began a follow-up of the NGHS Contra Costa County cohort to assess health in early midlife (ages 39–42). Eligibility criteria for the follow-up study included: (a) being an original NGHS participant; (b) not pregnant at the time of recruitment, and had not experienced a pregnancy, miscarriage, or abortion within the last 3 months; and (c) not living abroad, incarcerated, or otherwise institutionalized. As a result of extensive recruitment efforts, over 73% of eligible women were reenrolled in the study: 307 Black and 317 White women met the criteria and were eligible for follow-up. As part of the follow-up study, women reported on childhood adversities occurring before age 18.

The follow-up study also recruited the biological children (ages 2–17) of the women in the NGHS cohort to participate with their mothers. Mothers and their children were asked to provide saliva samples to assess epigenetic age acceleration. If NGHS mothers had more than one child, the youngest child was asked to provide a saliva sample. After exclusion due to quality control for saliva samples (see epigenetic age acceleration in Measures), there were 215 dyads in which both the child and mother were both able to provide saliva, and in which mothers had reported their childhood adversities.

The sample for the present study consisted of 215 dyads of mothers (111 White, 104 Black, $M_{age} = 39.2$, $SD = 1.1$) and their youngest child (119 female, 96 male, $M_{age} = 8.3$, $SD = 4.0$). Of the 215 women, 13.5% reported that they were current smokers (of tobacco), 55.8% reported that they had never smoked, and 30.7% reported that they had smoked in the past but did not smoke currently. Seven women (3.3%) reported that they had been diagnosed with diabetes, 8.4% reported that they had diabetes but only during pregnancy, and 6.5% reported having "prediabetes." Four women in the sample (2%) reported a diagnosis of heart disease. About 30.7% of the women reported that they had experienced depression and 12.6% reported that they were taking antidepressants.

Across the sample, 80% of the mothers had college degrees and 58% had current annual incomes of at least $60,000. There were no significant race differences in the likelihood of having a college degree (76% of Black mothers vs. 84% of White mothers); however, compared to White mothers, Black mothers were significantly less likely to have an annual income of at least $60,000 (38% of Black mothers vs. 77% of White mothers).

The 668 mothers who were part of the original NGHS study and not part of the present study, did not significantly differ in race, $\chi^2(1, 883) = 1.13$, $p = .29$, childhood income, $\chi^2(3, 837) = 3.13$, $p = .37$, or parental education, $\chi^2(881) = 0.74$, $p = .69$, from the 215 mothers in the present study.

Measures

Maternal Childhood Adversities

General Abuse. We used the Stress and Adversity Inventory (STRAIN; Slavich & Shields, 2018) to retrospectively measure the number of abuse-related adversities mothers experienced before age 13. The STRAIN assesses a person's cumulative exposure to stressors over the life course by systematically inquiring about a diverse array of acute life events and chronic difficulties. Cumulative stressor exposure assessed with the STRAIN has been linked to poor metabolic health and mental health in young adulthood (Toussaint et al., 2016), in addition to markers of biological aging (Mayer et al., 2023). The general abuse variable included stressors involving emotional abuse, sexual abuse, physical abuse, and prolonged harsh parental discipline. Respondents were also asked at what age the stressor occurred. The abuse variable represented the total number of stressors that occurred to the mothers between 0 and 12 years old.

Physical Abuse and Sexual Abuse. Adapted from Felitti et al. (1998), mothers were asked if they had experienced physical abuse or sexual abuse before age 18. Physical abuse was dichotomized into 0, if no physical abuse occurred before age 18, or 1, if physical abuse occurred. Sexual abuse was dichotomized into 0, if no sexual abuse occurred before age 18, or 1, if sexual abuse occurred.

Substance Abuse by Parent. Adapted from Felitti et al. (1998), participants were asked if one or both of their parents abused substances during the participant's childhood (before age 18). Substance abuse by parent was dichotomized into 0, if no parental substance abuse occurred before age 18, or 1, if parental substance abuse occurred.

Epigenetic Age Acceleration and Pace of Aging

Deoxyribonucleic acid (DNA) methylation analyses with saliva samples were performed at the Semel Institute University of California, Los Angeles Neurosciences Genomics Core using the

Illumina Infinium HumanMethylation450 BeadChip (Illumina, Inc.). Genomic DNA was isolated using temperature denaturation and subjected to bisulfite conversion, polymerase chain reaction amplification, and DNA sequencing (EZ DNA Methylation-Gold Kit, Zymo Research).

Horvath Clock. Epigenetic aging for both mother and child was assessed using the original Horvath clock (Horvath, 2013). This DNA methylation clock is based on 353 CpG sites and has been validated with both adult and pediatric samples across the different cell and tissue types. The Horvath clock is generally considered to be the most accurate epigenetic clock for use with child samples across most human tissues (Horvath, 2013; Nwanaji-Enwerem et al., 2021). Methylation profiles were input to Horvath's online calculator https://dnamage.genetics.ucla.edu/, which automatically imputes any missing CpGs. With selection of the advanced analysis option and normalization based on the beta-mixture quantile method (Teschendorff et al., 2013), output files contain the estimated epigenetic age of each participant and measures of predictive accuracy and data quality. Before data analysis began, a subset of participants ($n = 26$) was excluded due to quality control issues.

To assess epigenetic age acceleration, we used a variable representing epigenetic age adjusted for chronological age, calculated by obtaining the residuals of a linear regression. Positive residual values reflect an individual being older biologically than chronological age and negative residual values reflect the reverse. To account for confounding due to blood cell composition, we included cell abundance estimates of naïve CD8+ T and exhausted cytotoxic CD8+ T cells (CD8+ CD28− CD45−) as covariates (Horvath et al., 2016). As child saliva samples fell into one of two assay batches, a dummy variable was included in all models to control for batch effects.

DunedinPACE. To assess the epigenetic pace of aging, we used DunedinPACE. DunedinPACE (Belsky et al., 2022) was created from longitudinal data on 19 indicators of organ function and blood chemistry tracked on four occasions over 20 years. Through elastic-net regression, DNA methylation data were trained on within-individual decline in these indicators. Only CpG sites meeting a test-retest reliability threshold were included in the final algorithm for DunedinPACE.

We used publicly available code https://github.com/danbelsky/DunedinPACE-eLife-2022 (archived at swh:1:rev:e38233555b70c34f84f0b73e18f1a6bc4cb0852e, Belsky et al., 2022) to calculate epigenetic pace of aging for mothers and their children.

Data Analysis

We tested whether Black and White mothers differed on levels of maternal childhood adversities (i.e., abuse before age 13, sexual and physical abuse before age 18, and substance abuse by parent), or mother and child Horvath clock or DunedinPACE (Table 1). Next, we conducted zero-order correlations among these variables for the whole sample (Table 2) and stratified by race (Tables 3 and 4).

If there was a significant correlation between any of the maternal childhood adversities and either epigenetic biomarker (Horvath clock or DunedinPACE), we conducted linear regressions between maternal adversities and outcome, moderated by race. Significant Childhood Adversity × Race interactions ($p < .05$) were followed up by simple effects analyses. Regressions were not conducted for maternal adversities that did not have significant correlations with epigenetic biomarkers in either the whole sample or the sample stratified by race.

Covariates included child age, child sample batch, child cell type counts, and mother epigenetic Horvath clock or DunedinPACE.

Studies of maternal and child aging biomarkers, including those examining telomeres and epigenetic clocks, commonly include child age and the relevant maternal aging biomarker as covariates as these are usually associated with the child aging biomarker (Mayer et al., 2023). As we were primarily interested in the effects of exposure to adversity in the environment apart from heritable factors, we controlled for maternal epigenetic aging (Haussmann & Heidinger, 2015).

The few studies on maternal transmission of adversity to child epigenetic aging had mixed results for the association between child sex and child epigenetic aging (Dye et al., 2023; Nwanaji-Enwerem et al., 2021). To preserve power, we included child sex as a covariate in our analyses only if it was significantly associated with child epigenetic aging. The association was nonsignificant, so child sex was not included as a covariate. Sensitivity analyses included sex as a

Table 1
Descriptives of Study Variables (Dyads N = 215)

Study variables	All	Black	White	*t*	*d*
Age of child, years, *M*(*SD*)	8.27 (3.96)	8.92 (4.08)	7.67 (3.76)	2.35*	0.32
Mother education, % college degree, *n*	80.0 (172)	78.4 (80)	83.8 (93)	2.05 (χ^2)	
Annual income, % ≥ $60,000, *n*	57.7 (124)	38.2 (39)	76.6 (85)	33.59*** (χ^2)	
Mother epigenetic age acceleration, years, *M*(*SD*)	0.03 (4.38)	−1.08 (4.21)	1.06 (4.30)	3.69***	0.50
Mother's pace of aging	1.26 (0.17)	1.29 (0.15)	1.24 (0.17)	2.58*	0.35
Child epigenetic age acceleration, years, *M*(*SD*)	−0.11 (2.12)	−0.36 (2.32)	0.13 (1.89)	1.68	0.23
Child pace of aging	1.22 (0.20)	1.21 (0.20)	1.22 (0.21)	0.26	0.04
Maternal adversity					
Abuse events before age 13, *M*(*SD*) *N* = 170, Range 0 to 4	0.57 (0.78)	0.51 (0.70)	0.64 (0.86)	1.08	0.17
Sexual abuse, %, *n*	20.7 (43)	18.8 (19)	22.4 (24)	0.42 (χ^2)	
Physical abuse, %, *n*	24.7 (0.53)	17.3 (18)	31.5 (35)	5.85* (χ^2)	
Substance abuse by a parent, %, *n*	36.0 (76)	30.0 (30)	41.4 (46)	2.99 (χ^2)	

* $p < .05$. *** $p < .001$.

Table 2
Correlations of Study Variables

Variable	1	2	3	4	5	6	7	8	9	10	11
1. Female child	—										
2. Mother college	.14*	—									
3. Income, 60K+	.12	.16*	—								
4. Mother epiage	.03	.09	.04	—							
5. Mother pace	.08	−.03	−.16*	−.05	—						
6. Child epiage	−.01	.04	.08	.16*	−.08	—					
7. Child pace	.03	.07	.10	−.02	.16*	−.06	—				
8. Abuse > age 13	−.10	−.07	−.03	−.09	−.04	.05	−.06	—			
9. Sex abuse	.04	−.08	−.02	.01	.05	<.01	−.06	.56***	—		
10. Physical abuse	−.01	−.12	.03	.11	−.01	.10	−.06	.49***	.45***	—	
11. Substance abuse	−.02	.08	.05	.04	−.12	.09	−.08	.21**	.18*	.28**	—

* $p < .05$. ** $p < .01$. *** $p < .001$.

covariate and study findings remained supported (Table S1 in the online supplemental materials).

Results

Descriptive statistics are reported in Table 1. Spearman correlations are reported in Table 2. There was no significant difference in abuse before age 13, sexual abuse before age 18, or parental substance abuse before age 18 between Black and White mothers. Significantly more White mothers (31.5%) than Black mothers (17.3%) reported physical abuse before age 18.

Comparison of Epigenetic Aging for Black Versus White Mothers and Their Children

Horvath Clock

Black mothers had significantly slower epigenetic age acceleration (i.e., epigenetic age relative to chronological age) than White mothers, $t(213) = 3.69$, $p < .001$, $d = 0.50$. Black mothers were approximately 1 year (−1.08) younger epigenetically than their chronological age on average and White mothers were approximately 1 year (1.06) older epigenetically than their chronological age on average. There was also a significant difference in Horvath clock epigenetic age acceleration for the children of Black mothers compared to the children of White mothers, $t(213) = 1.68$, $p < .05$, $d = 0.23$. Children of Black mothers were just over 4 months (0.36 years) younger epigenetically than their chronological age and children of White mothers were approximately 6 weeks (0.13 years) older epigenetically than their chronological age.

DunedinPACE

Black mothers had significantly faster DunedinPACE than White mothers, $t(213) = 2.58$, $p = .01$, $d = 0.35$. Black mothers aged 1.29 epigenetic years for each chronological year and White mothers aged 1.24 epigenetic years for each chronological year. There was no significant difference in the epigenetic pace of aging for the children of Black mothers compared to the children of White mothers, $t(213) = 0.26$, $p = .80$, $d = 0.04$. Children of Black mothers aged 1.21 epigenetic years for each chronological year and children of White mothers aged 1.22 epigenetic years for each chronological year.

Zero-Order Correlations Between Maternal Adversity and Child Epigenetic Aging

Across the sample of mothers (Table 2), there were no significant associations between any of the maternal adversities with their own Horvath or DunedinPACE epigenetic measures.

We found small significant associations between mother and child epigenetic age acceleration and between mother and child pace of aging. There were no significant correlations between epigenetic age acceleration and pace of aging. Income was not significantly associated with epigenetic age acceleration but did correlate with maternal pace of aging, in that having

Table 3
Correlations of Study Variables—Black Women

Variable	1	2	3	4	5	6	7	8	9	10	11
1. Female child	—										
2. Mother college	.09	—									
3. Income 60K+	.03	.02	—								
4. Mother epiage	−.04	.19	−.03	—							
5. Mother pace	.14	.10	−.12	<.01	—						
6. Child epiage	−.11	<.01	.01	.10	−.13	—					
7. Child pace	.04	−.02	.02	−.01	.21*	.01	—				
8. Abuse > age 13	−.03	.08	.03	<.01	−.08	.25*	−.08	—			
9. Sex abuse	.16	.03	−.02	.04	.01	.20*	.01	.53***	—		
10. Physical abuse	−.02	−.04	−.09	.08	.04	.31**	<.01	.46***	.53***	—	
11. Substance abuse	.03	.16	−.02	.10	−.13	.14	−.11	.27*	.26*	.34***	—

* $p < .05$. ** $p < .01$. *** $p < .001$.

Table 4
Correlations of Study Variables–White Women

Variable	1	2	3	4	5	6	7	8	9	10	11
1. Female child	—										
2. Mother college	.18	—									
3. Income 60K+	.18	.28**	—								
4. Mother epiage	.06	−.06	−.10	—							
5. Mother pace	.05	−.13	−.08	−.02	—						
6. Child epiage	.10	.06	.08	.18	.01	—					
7. Child pace	.02	.15	.17	−.04	.13	−.15	—				
8. Abuse > age 13	−.16	−.25*	−.16	−.20	.01	−.18	−.05	—			
9. Sex abuse	−.07	−.21*	−.07	−.05	.10	−.26**	−.12	.60***	—		
10. Physical abuse	−.02	−.23*	.01	.06	<.01	−.13	−.11	.50***	.40***	—	
11. Substance abuse	−.07	−.03	.03	−.06	−.09	.02	−.07	.16	.11	.22*	—

* $p < .05$. ** $p < .01$. *** $p < .001$.

an income of at least $60,000 annually was associated with a slower epigenetic pace of aging for mothers (nonsignificant for children).

Zero-Order Correlations of Adversity and Aging Variables Stratified by Race

Black Mothers (Table 3). Maternal general abuse before age 13, maternal sexual abuse before age 18, and maternal physical abuse before age 18, were significantly related to the epigenetic age acceleration of the children of Black mothers. Conversely, substance abuse of maternal parent was unrelated to child epigenetic age, and there were no significant associations between any of the maternal childhood adversities and children's pace of epigenetic aging.

White Mothers (Table 4). Maternal sexual abuse before age 18 was associated with the epigenetic age deceleration in the children of White mothers. For White mothers, there were no other significant associations between maternal adversities and child epigenetic age acceleration or pace of aging.

For both Black and White mothers, there were no significant associations between child gender or socioeconomic status (income or education) and the child epigenetic biomarkers.

Does Race Moderate the Association Between Maternal Childhood Abuse and Child Horvath Epigenetic Age Acceleration?

As there were significant associations between child epigenetic acceleration and maternal childhood adversity, and because these relations differed by race, we formally tested the association between three maternal adversities (i.e., all abuse before age 13, sexual abuse before age 18, and physical abuse before age 18) and child Horvath age acceleration with the regression models described above. Covariates included child age, child sample batch, child cell type counts, and mother epigenetic age acceleration.

Table 5
Maternal Childhood Adversity Predicting Offspring Epigenetic Age Acceleration

Variables	*b*	*SE*	*t*	*p*
CD8+ CD28− CD45− (cell counts)	−0.02	0.06	−0.28	.78
CD8+ T (cell counts)	0.002	0.01	0.30	.77
Sample batch effects (*Batch 1* = 0, *Batch 2* = 1)	0.58	0.48	1.21	.23
Child age	−0.21	0.04	−5.00	<.001
Mother epigenetic age acceleration	0.07	0.03	2.12	.04
Race (0 = *Black*, 1 = *White*)	0.56	0.39	1.46	.15
Abuse before age 13	0.81	0.30	2.73	.007
Race × Abuse Before 13	−0.83	0.39	−2.14	.03
CD8+ CD28− CD45− (cell counts)	0.01	0.06	−0.22	.82
CD8+ T (cell counts)	−0.003	0.01	−0.52	.60
Sample batch effects (*Batch 1* = 0, *Batch 2* = 1)	1.00	0.44	2.29	.02
Child age	−0.17	0.04	−4.44	<.001
Mother epigenetic age acceleration	0.05	0.03	1.67	.10
Race (0 = *Black*, 1 = *White*)	0.47	0.31	1.51	.13
Sexual abuse before age 18	1.17	0.48	2.44	.02
Race × Sexual Abuse Before Age 18	−1.60	0.67	−2.40	.02
CD8+ CD28− CD45− (cell counts)	−0.01	0.06	−0.23	.82
CD8+ T (cell counts)	0.002	0.01	0.44	.66
Sample batch effects (*Batch 1* = 0, *Batch 2* = 1)	0.64	0.45	1.44	.15
Child age	−0.20	0.04	−5.18	<.001
Mother epigenetic age acceleration	0.06	0.03	1.87	.06
Race (0 = *Black*, 1 = *White*)	0.61	0.32	1.88	.06
Physical abuse before age 18	1.69	0.50	3.34	.001
Race × Physical Abuse Before Age 18	−2.09	0.65	−3.22	.002

All Abuse Before Age 13

There was a significant interaction between maternal abuse and race (Table 5), $b = -0.83$, $p = .03$, 95% confidence interval, CI [−1.59, −0.06], revealing that Black mothers who experienced greater abuse before age 13 had children with greater epigenetic age acceleration, $b = 0.81$, $p = .007$, 95% CI [0.22, 1.40]. For White mothers, there was no significant relation between maternal abuse before age 13 and child age acceleration, $b = -0.02$, $p = .94$, 95% CI [−0.51, 0.48].

Sexual Abuse Before Age 18

Similarly, there was a significant interaction between maternal sexual abuse and race (Table 5), $b = -1.60$, $p = .02$, 95% CI [−2.92, −0.29], revealing that Black mothers who experienced sexual abuse before age 18 had children with greater epigenetic age acceleration, $b = 1.17$, $p = .02$, 95% CI [0.22, 2.11]. For White mothers, there was no relation between maternal childhood sexual abuse and child age acceleration, $b = -0.43$, $p = .34$, 95% CI [−1.33, 0.47].

Physical Abuse Before Age 18

Finally, there was a significant interaction between maternal physical abuse and race (Table 5), $b = -2.09$, $p = .002$, 95% CI [−3.38, −0.81], revealing that Black mothers who experienced physical abuse before age 18 had children with greater epigenetic age acceleration, $b = 1.69$, $p = .001$, 95% CI [0.69, 2.68]. For White mothers, there was no relation between maternal childhood physical abuse and child age acceleration, $b = -0.41$, $p = .31$, 95% CI [−1.20, 0.39].

Discussion

Although maternal adversity has been found to affect children's health outcomes, the biological mechanisms underlying this association remain unknown. In the present study, we examined how maternal childhood adversity influences the biological aging of their children, which can provide insight into intergenerational vulnerability to aging-related outcomes. For Black mothers, maternal childhood abuse was associated with greater epigenetic age acceleration of their children. For White mothers, there were no significant associations between maternal childhood abuse and child epigenetic aging. Moreover, we found significant associations between maternal adversity of Black mothers and child epigenetic age acceleration, but not for child epigenetic pace of aging.

Using a continuous variable of any abuse before age 13, as well as separate categorical variables of sexual abuse and physical abuse before age 18, we found significant associations between all three variables of maternal childhood abuse and offspring epigenetic age acceleration. It is notable that we found significant associations for maternal childhood abuse, but not for maternal substance abuse. Maternal substance abuse has been characterized as an ACE, but it is not usually included as a form of childhood abuse. Childhood abuse is usually conceptualized as emotional abuse, sexual abuse, physical abuse, and prolonged harsh parental discipline.

Our findings are in line with a meta-analysis that found childhood adversity specifically characterized as abuse may be associated with accelerated biological aging (Colich et al., 2020). Childhood abuse may contribute to increased disease and early mortality through the child's perception of a lack of safety, a perception which may heighten responses to threat, upregulate inflammation, and influence health behaviors (Furman et al., 2019).

Prior research has found that mother's stressor exposure during adolescence had the largest impact on offspring's weight at birth compared to other developmental periods, which suggests that adolescence may be a critical period for the effects of maternal adversities on the health of future children (Entringer et al., 2018; Keenan et al., 2018). We found significant associations between offspring epigenetic age acceleration and three forms of maternal childhood abuse: all abuse before age 13, maternal sexual abuse before age 18, and physical abuse before age 18. Consistent with the present study, Nwanaji-Enwerem et al. (2021) found that greater maternal ACEs were associated with the epigenetic age acceleration (also using Horvath's clock) of the children of Latina mothers. Our study indicates that the relation between maternal childhood adversity and offspring epigenetic aging may extend to Black mothers.

An earlier study using the Horvath clock and multiple data sets found that Black Americans had lower epigenetic age acceleration compared to White Americans (Horvath et al., 2016). In the present study, Black mothers had significantly less epigenetic age acceleration and a faster pace of aging than White mothers. Such evidence suggests a similar pattern as to that found with telomeres as an index of biological aging, in that at younger ages, Black women may have longer telomeres but more rapid telomere attrition when compared to White women (Hunt et al., 2008; Needham et al., 2020). By ages 49–55, Black women have been found to have shorter telomere length than White women (Geronimus et al., 2010). Telomere attrition may speed up around age 50 (Frenck et al., 1998), and epigenetic biomarkers should be assessed throughout the life course to see if findings are replicated.

Epigenetic clocks appear to be reliable markers of social factors that accelerate aging and contribute to later health disparities. In the present study, epigenetic clocks help support the notion that experiencing early adversity as a member of a minoritized community accelerates cellular aging. Evidence suggests that epigenetic clocks may capture the biological and physiological processes set in motion by early adversity and other adverse social factors better than other aging-related biomarkers (Hamlat et al., 2021).

Significant work is needed to standardize metrics across different clocks as each clock functions essentially as a unique biomarker and each clock may measure distinct aging processes (Theodoropoulou et al., 2019). Along with standardization, studies need to integrate multiple aging biomarkers as multisystem phenotypes of aging that may better reflect whole-body aging (Hamlat, Neilands, et al., 2023; Hamlat, Zannas, & Epel, 2023).

Strengths and Limitations

The study has several significant strengths, including a sample with equivalent numbers of Black and White women and the use of several well-validated markers of biological aging. We accounted for maternal epigenetic aging to better isolate the effects of maternal adversity on offspring aging. Several limitations should be considered. First, maternal neglect was not examined in the current study as a priori we focused on childhood abuse as a risk factor. As the study questionnaire was structured to obtain dichotomized variables for physical, sexual, and substance abuse, we were not able to assess

if there was an effect of frequency of physical, sexual, and substance abuse on outcomes.

Second, maternal childhood abuse was solely assessed retrospectively in adulthood. Only modest agreement has been documented between prospective and retrospective reports of adverse childhood experiences (Baldwin et al., 2019; Reuben et al., 2016). Compared to prospective reporting, retrospective reporting of childhood maltreatment has shown stronger associations with subjective outcomes, such as self-report of symptoms, and weaker associations with more objective measures, such as biomarkers (Danese & Widom, 2020; Reuben et al., 2016). Consequently, the association between abuse and epigenetic aging may be underestimated in the current study (Reuben et al., 2016). Ideally, future research would include both prospective and retrospective reports of childhood adversity (Newbury et al., 2018).

Third, we did not include men or women of other racial and ethnic backgrounds, and paternal early adversity has been found to influence offspring DNA methylation (Merrill et al., 2021). With individuals of different racial and ethnic backgrounds, future studies should examine associations between both maternal and paternal childhood adversity and offspring epigenetic aging. Additionally, this study should be replicated using other samples of Black women. Fourth, unexpectedly, maternal childhood adversity was not correlated with maternal epigenetic aging. One study (Dye et al., 2023) found that maternal childhood adversity accelerated both maternal and offspring epigenetic aging, and another study (Nwanaji-Enwerem et al., 2021) found that maternal ACEs predicted offspring epigenetic age acceleration but did not examine the association between maternal adversity and maternal epigenetic aging. Finally, due to power concerns, we did not examine sex differences. Dye et al. (2023) found the relation between maternal adversity and offspring epigenetic age acceleration was present for male offspring but not for female offspring; however, Nwanaji-Enwerem et al. (2021) did not find sex differences in this association. In the present study, we did not find an association between offspring sex and either of our outcomes (epigenetic age association and pace of aging); consequently, sex was not included as a covariate.

Conclusion

In conclusion, the present findings add to the body of literature examining how early adversity exposure in mothers influences the health of their offspring and suggest a differential impact of maternal childhood adversity on the epigenetic aging of children of Black versus White mothers. Maternal childhood adverse experiences may be transmitted through epigenetic pathways to affect offspring biological aging. Racial discrimination has also been associated with accelerated aging (Brody et al., 2016). We did not specifically measure race-related adversity, but the Black women in our study are exposed to systemic racism in the United States, which contributes to health disparities between Black and White Americans (Hooten et al., 2022). Maternal adversity and systemic racism may result in greater levels of physiological changes that impact the biological aging of offspring. That social conditions become biologically embedded has been supported in prior work showing that socioeconomic changes predict longitudinal changes in epigenetic aging in Black women (Simons et al., 2021, 2022).

The present results extend this body of work and highlight the potential importance of prevention and early intervention for childhood adversities. Programs such as California's screening and referral system for ACEs are important (McBain et al., 2023); however, what is also crucially needed are specific interventions for children who have experienced adverse events. Family-level interventions hold promise, as supportive family environments have been found to buffer against further epigenetic age acceleration for Black youth (Brody et al., 2016). Changes at the institutional level that address social justice and racism may also lead to a deceleration of epigenetic aging (Phelan & Link, 2015; Simons et al., 2021). Social policies and programs that reduce childhood adversities and racial and economic inequalities could slow down accelerated aging and improve health equity.

Resumen

Objetivo: Aunque la adversidad temprana está fuertemente relacionada con las disparidades de salud a lo largo de la vida, no está claro cómo la adversidad podría conferir riesgo entre generaciones. Para investigar, probamos la hipótesis de que la adversidad infantil de las madres estaba asociada con su envejecimiento epigenético y el de su hijo y examinamos si las asociaciones diferían entre las madres Blancas y Negras. ***Métodos:*** Diadas ($N = 215$) de madres (52% Blancas, 48% Negras, edad media = 39.2, DE = 1.1) y niños ($N = 215$, 55% mujeres, edad media = 8.3, DE = 4.0, rango 2 a 17) proporcionaron muestras de saliva para analizar las medidas de envejecimiento epigenético de Horvath y DunedinPACE. Se utilizaron regresiones lineales para estimar las asociaciones de las medidas de adversidad materna temprana con los resultados de la edad epigenética de Horvath materna e infantil, moderada por la raza. ***Resultados:*** Para las madres Negras, pero no Blancas, cualquier abuso antes de los 13 años, $b = 0.81$, $p = .007$, abuso físico antes de los 18 años, $b = 1.69$, $p = .001$, y abuso sexual antes de los 18 años, $b = 1.17$, $p = .02$, se asociaron con una aceleración significativamente mayor de la edad de Horvath en sus hijos. Por el contrario, no hubo relación entre la adversidad materna en la infancia y el envejecimiento epigenético de las madres, y no hubo hallazgos significativos para DunedinPACE. ***Conclusiones:*** La adversidad materna en la infancia parece tener un mayor efecto en el envejecimiento epigenético de los hijos de madres Negras. Los efectos del racismo sistémico en los Estadounidenses Negros pueden interactuar con las adversidades maternas infantiles para conferir un riesgo adicional a los niños Negros.

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