HARSH AND DISENGAGED PARENTING AS A MECHANISM BETWEEN MATERNAL DEPRESSION AND ADOLESCENT ACCELERATED EPIGENETIC AGE CHANGE

by

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(Under the Direction of Kalsea Koss)

ABSTRACT

Depression in adults can negatively impact both those afflicted and those around them. Parental depression is associated with negative developmental outcomes in children. Parenting practices have been supported as a mechanism in parental depression's effect on child psychosocial outcomes, but it is less clear whether the same is true for long-term health outcomes. The current study examined whether parenting practices, specifically physical and psychologically aggressive parenting and level of parental engagement, served as a mechanism linking maternal depression and adolescents' epigenetic aging, a predictor of long-term health using data from 1,971 mother-child dyads from the Future of Families and Child Wellbeing study. Epigenetic aging was measured using three algorithms: PhenoAge, GrimAge, and Dunedin PACE. Results indicated that increased maternal physical aggression mediated the effect of maternal depression on greater changes in accelerated epigenetic age between ages 9 and 15 years when measured with the GrimAge clock.

INDEX WORDS*:* maternal depression, harsh parenting, maternal engagement, epigenetic age, parental aggression.

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Abstract

Depression in adults can negatively impact both those afflicted and those around them. Parental depression is associated with negative developmental outcomes in children. Parenting practices have been supported as a mechanism in parental depression's effect on child psychosocial outcomes, but it is less clear whether the same is true for long-term health outcomes. The current study examined whether parenting practices, specifically physical and psychologically aggressive parenting and level of parental engagement, served as a mechanism linking maternal depression and adolescents' epigenetic aging, a predictor of long-term health using data from 1,971 mother-child dyads from the Future of Families and Child Wellbeing study. Epigenetic aging was measured using three algorithms: PhenoAge, GrimAge, and Dunedin PACE. Results indicated that increased maternal physical aggression mediated the effect of maternal depression on greater changes in accelerated epigenetic age between ages 9 and 15 years when measured with the GrimAge clock.

Index words: maternal depression, harsh parenting, maternal engagement, epigenetic age, parental aggression

CHAPTER 1

INTRODUCTION AND LITERATURE REVIEW

Depression is among the most prevalent mental health disorders in the United States, and its prevalence has greatly increased over the past two decades (Substance Abuse and Mental Health Services Administration, 2020). In 2006, 14.2 million adults reported having had a major depressive episode during the past year (Substance Abuse and Mental Health Services Administration, 2007). Since then, this number has increased to 19.4 million in 2019, and to 22.5 million, or 8.8% of the US adult population, in 2022 (Han et al., 2023; Substance Abuse and Mental Health Services Administration, 2020). According to the fifth edition of the Diagnostic-Statistical-Manual (DSM-5), symptoms associated with depression include depressed mood, feelings of worthlessness, and loss of interest or pleasure in doing things (American Psychiatric Association, 2013). An adult's functioning across the social, emotional, and occupational domains can be greatly impacted by these symptoms. Parents with depression are no exception to these effects, and as a result their ability to meet the needs of their children may become more limited.

1.1 Maternal depression and child outcomes

Maternal mental health can be a particularly salient source of stress in a child or adolescent's environment (Goodman & Gotlib, 1999). High stress environments can have severe negative impacts on children and adolescents (Gunnar & Quevedo, 2007; Schneiderman et al., 2005). Therefore, it is unsurprising that children of depressed parents tend to have poorer physical and mental health outcomes than those of non-depressed parents (National Research

Council and Institute of Medicine, 2009). Still largely unknown, however, are the mechanisms through which this association may occur. One possible mechanism is through increased negativity and unpredictability in parenting that has been associated with maternal depression (Cummings & Davies, 1994). For example, depressed parents often exhibit inconsistency, withdrawal, and aggression in their parenting (Downey & Coyne, 1990; Lovejoy et al., 2000). Children of these parents tend to have higher rates of anxiety and aggression when compared to children of non-depressed parents (Jaser et al., 2005; Langrock et al., 2002).

1.2 Maternal depression and child biological age

Importantly, maternal depression has also been found to impact children's biological processes. For example, maternal depression has been associated with elevated cortisol levels during infancy, childhood, and adolescence, which is reflective of disrupted functioning of the stress response system (Ashman et al., 2002; Halligan et al., 2007a; 2007b). This finding may extend to children's biological age, which is the extent to which one's physical and physiological functioning resembles a typical person at a chronologically older or younger age. Research has suggested that those who have been exposed to high amounts of stress tend to appear physically and physiologically older than what is typical for their age, a phenomenon known as biological age acceleration (Ferrucci et al., 2020). There are multiple approaches to measuring this phenomenon which may be used to investigate the impact of maternal depression. Telomere length, for example, is a measure of biological age based on the shortening of protective DNA structures at the ends of chromosomes which shorten with each cell replication until cell death (Shammas et al., 2011). Exposure to maternal depression in early and middle childhood has been associated with shorter telomeres (Mitchell et al., 2014; Wojcicki et al., 2015). This implies that maternal depression may be a source of stress that accelerates the biological aging of children.

1.2.1 Epigenetic age as a measure of biological age

Another measure of biological age acceleration in children and adolescents is to estimate their epigenetic age. This measure reflects patterns of DNA methylation markers that tend to strongly correlate with chronological age (Horvath, 2013) and biomarkers of health (Levine et al., 2018; Lu et al., 2019; Ryan, 2021). Numerous algorithmic approaches to determining epigenetic age have been developed over the past decade, referred to as "epigenetic clocks" (Ryan, 2021). While many such clocks exist, they are generally grouped across multiple "generations", reflecting advances in research and shifting goals in their development. First-generation epigenetic clocks were developed to primarily reflect deviations from metrics of chronological age. Two well-known clocks in this category include the Hannum Clock (Hannum et al., 2013) and the Horvath Clock (Horvath, 2013). Second-generation clocks, however, were developed to reflect biomarkers and health outcomes in relation to epigenetic age (Ryan, 2021). Examples of epigenetic clocks in this group include PhenoAge (Levine et al., 2018), GrimAge (Lu et al., 2019), Dunedin PoAm (Belsky et al., 2020), and Dunedin PACE (Belsky et al., 2022). Measurements using these epigenetic clocks reflect one's epigenetic age at a given time point, and the difference between epigenetic age and chronological age is one's "epigenetic age acceleration". The degree of this acceleration may vary over time, reflecting longitudinal change in epigenetic aging, however few studies to date have investigated epigenetic aging in this way (e.g.: Aikins et al., 2024; Mak et al., 2023; Mastrotheodoros et al., 2024).

1.2.2 Maternal depression and epigenetic age

As a measure of biological age and biological age acceleration, epigenetic clocks from both generations have demonstrated the ability to capture accelerated epigenetic age, which is when an individual's epigenetic age is higher than that which is expected for their chronological

age. Accelerated epigenetic age in second generation clocks has been associated with numerous health related outcomes, including cardiovascular disease, type-II diabetes, and earlier all-cause mortality (Lu et al., 2019). Much of this research uses adult samples, however adolescence is an important developmental period to investigate, as doing so provides insight into current and future health trajectories during a point at which these trajectories tend to diverge (Belsky et al., 2015). Examinations during adolescence could allow for early detection and intervention with risk factors that predict poor health outcomes. Notably, exposure to psychosocial stressors such as abuse (Lawn et al., 2018), violence (Jovanovich et al., 2017; Sumner et al., 2019), and low socioeconomic status (George et al., 2021) during childhood and adolescence have been linked to accelerated epigenetic age. As such, accelerated epigenetic age could very well mediate the well documented association between maternal depression and poor health outcomes (Grummitt et al., 2021). There is, however, a dearth in research linking maternal depression and accelerated epigenetic age specifically. It is therefore necessary to investigate this association as well as its potential mechanisms.

1.3 Parenting as a mechanism between maternal depression and child biological age

1.3.1 Harsh parenting

Disruptions in parenting may serve as an environmental mechanism linking maternal depression and youth's accelerated epigenetic age, as evidence has suggested that parenting behaviors may be disrupted by maternal depression. Harsh parenting, characterized by physical or psychological aggression toward the child (Marcal, 2021), has been widely associated with maternal depression and has been strongly linked to negative psychological outcomes for children (NRCIM, 2009). A possible reason for the association between maternal depression and harsh parenting is that the increased irritability due to depression may reduce a parent's capacity

to serve a child's needs (Downey & Coyne, 1990; Lovejoy et al., 2000). The associations between harsh parenting and child outcomes have been well documented and associated with a range of physical and psychological issues in children and adolescents. Examples of these include increased internalizing and externalizing symptoms (Pinquart, 2021), increased BMI (Lei et al., 2021), and altered region-specific brain morphology (Cortes Hidalgo et al., 2022). At the same time, harsh parenting has also been associated with accelerated epigenetic age in young adults (Brody et al., 2016), and older individuals based on the recollection of their early life experience (Mrug et al., 2023). There is evidence suggesting harsh parenting is a mechanism through which maternal depression affects a child's mental and physical health outcomes (Lim et al., 2008). Specifically in regards to epigenetic age, Brody et al. (2016) found that harsh parenting practices served as a mediator between parental depression and accelerated epigenetic age in a sample of rural African American emerging adult males. Such an effect may also occur before emerging adulthood, though to my knowledge no such study has investigated this relation in an adolescent sample.

1.3.2 Disengaged parenting

In addition to harsh parenting, maternal depression has also been associated with withdrawn or disengaged parenting (Downey & Coyne, 1990; Lovejoy, 2000). Engaged parenting is characterized by active involvement, warmth, and communication in the child's life, as well as sufficient attention being paid to their needs. Disengaged parenting, conversely, is characterized by a lack of, or deficit in, these traits. Disengaged parenting may occur due to fatigue, a common symptom of depression that may serve as a barrier to engaging with their child (Field et al., 1998). That is, depressed parents may lack the energy necessary to focus on their child's needs. Disengaged parenting has been associated with a variety of outcomes in

children and adolescents including externalizing behaviors, depressive symptoms, and reduced self-esteem (Hoeve et al., 2009; Knutson et al., 2005; Milevsky et al., 2008; Simons et al., 2002; Steinberg et al., 2006). Research on the mediating role disengaged parenting as a mediator between maternal depression and child developmental outcomes is relatively scarce, however some evidence does suggest potential for maternal engagement as mediator. For example, in a study on mother-infant interaction, Jones et al. (1997) found that withdrawn parenting behavior in depressed mothers was associated with both increased frontal EEG asymmetry as well as lower Bayley development scores. Similarly, low engagement parenting in depressed parents has been associated with increased internalizing and externalizing symptoms during late childhood as well as adolescence (Gruhn et al., 2016; Wood et al., 2004). However, to my knowledge no research has investigated parental engagement as a mediator between maternal depression and youth's epigenetic age.

1.4 The current study

The current study sought to address this gap by investigating whether cumulative exposure to maternal depression across childhood predicts accelerated epigenetic age in adolescence, and whether harsh and disengaged parenting serve as mediators in this relation. While harsh parenting and low engagement parenting both occur in depressed parents (NRCIM, 2009), some research has suggested that harsh and disengaged parenting each have separate and unique effects on a child's physiological response to stress (Doom et al., 2022). In light of such findings, the current study investigated both harsh and disengaged parenting as separate mechanisms in the association between cumulative maternal depression and the epigenetic aging process across early to middle adolescence.

Three second-generation epigenetic clocks were utilized in this study, as these have been found to be related to differences in psychosocial stressors (Ecker & Beck, 2019; Li et al., 2020; Maddock et al., 2020; Zhao et al., 2019). I hypothesized that those with a greater cumulative exposure to maternal depression would have significantly higher accelerated epigenetic age change than those exposed less cumulative depression. I also hypothesized that both harsh parenting and disengaged parenting would serve as mediators between maternal depression and adolescent epigenetic aging. The conceptual model is shown in Figure 1.

CHAPTER 2

METHOD

2.1 Participants

The sample drew from The Future of Families and Child Wellbeing Study (FFCWS), a multi-ethnic birth cohort study consisting of 4,898 families with children born between 1998 and 2000 (Reichman et al., 2001). Participants were recruited at the birth of the focal child in hospitals located across 20 major U.S. cities with populations above 200,000. Unmarried mothers were oversampled by a three to one ratio; 76% of mothers were unmarried at baseline. As a result of this sampling approach, a large proportion of participants in the sample are from minority or low-income families. Survey data was collected across six waves starting at birth, and subsequently when children were approximately one, three, five, nine, and fifteen years old. Surveys were completed by the biological mothers, biological fathers, primary caregivers, and focal children. The majority of primary caregivers were biological mothers (87.88%).

Saliva samples from which biological data were derived were collected when children were ages nine and fifteen years old. The current study used data from a subsample of participants who had provided these saliva samples, resulting in a total $N = 1{,}971$. Among this subsample, 50.33% are male and 49.77% are female; 47.44% are Black, 26.23% are Hispanic, 25.31% are White, and 3.55% are of other racial/ethnic backgrounds.

2.2 Saliva sampling procedure

To assess epigenetic age from DNA methylation, DNA was first extracted from saliva under the Oragene prepIT·L2P laboratory protocol. Samples then underwent bisulfite conversion to determine methylation (EZ-96 DNA Methylation Kit; Zymo Research) and were then

analyzed using either the Illumina Infinium Human Methylation450K (N = 1,144) or Illumina Infinium MethylationEPIC (N = 829). Individual samples from each age were each analyzed using the same array on the same plate to minimize technical variation. Quality control procedures for the methylation data were performed in the EWAStools package. Epithelial cell proportion estimates were included in analyses using a reference panel estimate for saliva in children and adolescence through EWAStools (Middleton et al., 2022). For more information on the specimen collection procedure, see The Future of Families and Child Wellbeing Study Biomarker Appendage (2023).

2.3 Measures

2.3.1 Maternal depression

Maternal depression was measured using the major depression section of the Composite International Diagnostic Interview-Short Form (CIDI-SF) (Kessler et al., 1998). Survey questions assessed whether the respondent had experienced dysphoria or anhedonia for more than two weeks within the past year, and if so, responded to items about additional depressive symptoms. This resulted in binary classification indicating whether mothers were or were not likely to receive a depression diagnosis. The CIDI-SF interviews were conducted when children were aged one, three, five, and nine years old. The cumulative number of time points between birth and age nine at which the mother met criteria for a probable depression diagnosis were summed to form an indicator of maternal depression chronicity and used throughout subsequent analyses.

2.3.2 Harsh parenting

Harsh parenting was measured using the psychological aggression and physical aggression subscales of an abbreviated 10-item version of the Parent-Child Conflict Tactics

Scale (CTSPC) (Straus et al., 1998). Primary caregivers reported on their own parenting behaviors when children were nine years old. They were asked about the frequency of each behavior during the past year using the following scale: never, once, twice, 3-5 times, 6-10 times, 11-20 times, more than 20 times. A weighted score was then computed to reflect these frequencies. Responses were coded to represent the midpoints of each option in accordance with the recommendations of Straus et al. (2003) (i.e.: "3-5 times" was coded as 4, "6-10 times" as 7, "11-20" as 15, and "more than 20 times" as 25). Means of the weighted scores were computed for two dimensions of harsh parenting: physical aggression and psychological aggression. Reliability analyses indicated high internal consistency among all both dimensions (psychological aggression $\alpha = .70$; physical aggression $\alpha = .70$).

2.3.3 Maternal engagement

Maternal engagement was measured using survey items originating from the Future of Families and Child Wellbeing Study regarding each parent's involvement with their child (Doom et al., 2022; Lee & Schoppe-Sullivan, 2023; Reichman et al., 2001; Turney, 2011). When the child was approximately nine years old, parents were asked how many times in the past month they participated in certain activities with the child, such as playing outdoor sports or discussing current events. Items were scored on a 5-point Likert scale reflecting how frequently parents engaged in each activity. Scale responses included "not in the past month", "1-2 times in the past month", "once a week", "several times a week", and "every day". Reliability analysis indicated acceptable internal consistency ($\alpha = .71$).

2.3.4 Change in epigenetic age acceleration

Three epigenetic clocks, PhenoAge (Levine et al. 2018), GrimAge (Lu et al. 2019), and Dunedin PACE (Belsky et al., 2022) were used to assess epigenetic age. PhenoAge (Levine et

al., 2018) was derived from 513 CpG sites trained on several clinical health biomarkers. GrimAge (Lu et al., 2019) was derived from 30,084 CpG trained on methylation sites predicting mortality as well as chronological age, sex, and smoking pack years. The Dunedin Pace of Aging Clock from the Epigenome (PACE) (Belsky et al., 2022) differs from the other two in that it reflects the rate at which individuals are epigenetically aging, rather than their epigenetic age at the time of measurement. The PACE clock was trained on biomarker data collected across 20 years and derived from 173 CpG sites.

In accordance with the aims of this study, these second-generation clocks all reflect epigenetic age as indicated by methylation patterns which correlate with both chronological age and long-term health outcomes. In order to compute the acceleration of epigenetic age, each epigenetic clock was regressed on chronological age and unstandardized residuals were used as a measure of acceleration at each point. Positive residuals indicated a faster rate of epigenetic aging compared to chronological age. A latent change score model (McArdle, 2009) was then estimated to represent change in the residuals between age nine and fifteen so as to quantify the change in accelerated epigenetic age across early to mid-adolescence.

2.4 Data analytic plan

One-way ANOVAs were conducted to compare sex differences in accelerated epigenetic age change, as well as differences in accelerated epigenetic age between children of depressed and non-depressed mothers. To test the mediation models, age nine data from each of the three parenting measures, physical aggression, psychological aggression, and parental engagement were tested as mediators between maternal depression chronicity and change in epigenetic age acceleration between ages nine and fifteen. Separate models were tested using each of the three epigenetic clocks. Covariates included child sex, and race/ethnicity, family household income at

birth, marital status measured at birth, maternal smoking status at birth, saliva epithelial cell proportions, and array type (Illumina 450K or Illumina EPIC). Descriptive statistics and preliminary analyses were conducted in SPSS and the mediation analyses were conducted using MPlus 8.9 (Muthen & Muthen, 2017).

CHAPTER 3

RESULTS

3.1 Descriptive statistics

Descriptive statistics are displayed in Tables 1 and 2, bivariate correlations are displayed in Table 3. The majority of mothers (59.64%) did not receive a probable diagnosis of depression at any time point. Percentages of mothers with depression at each focal child age were: 15.4% at age one, 21.9% at age three, 17.2% at age five, and 18.2% at age nine. Total number of time points when depression was indicated were added to form a cumulative indicator of chronicity of maternal depression across childhood. Overall, 22.4% of mothers met depression criteria at only one time point, 10.3% met criteria at two time points, 5.4% met criteria at three time points, and 2.5% met criteria at all four time points. Chronicity of maternal depression was significantly and positively correlated with both physical ($r = .11$, $p < .001$) and psychological aggression ($r = .17$, $p < .001$). Maternal physical aggression significantly correlated with psychological aggression ($r = .53$, $p < .001$). Maternal depression and maternal engagement did not significantly correlate ($r = .01$, $p > .05$). Neither physical nor psychological aggression were significantly correlated with maternal engagement.

All accelerated epigenetic age measures positively correlated with one another, both between and within ages ($r = .15$-$.70$, all ps $< .001$). Assay type did not significantly correlate with any epigenetic age measures. Epithelial cell proportions, however, were significantly correlated with all epigenetic clocks (all ps $<.001$). See Table 3 for additional correlations among study variables and sociodemographic variables. Correlations among sociodemographic variables and study variables were small in magnitude (r range -.20 to .20), with the exception of

the correlation between child sex and age nine accelerated epigenetic age using the PhenoAge clock ($r = .34$, $p < .001$).

3.2 Sex differences in epigenetic age acceleration

To test for child sex differences in accelerated epigenetic age, three one-way analyses of variance (ANOVAs) were conducted. Girls displayed more accelerated epigenetic age when measured with PhenoAge and Dunedin PACE. Boys had more accelerated epigenetic age when measured with GrimAge. Results are displayed in Table 4.

3.3 Epigenetic age acceleration in children of depressed and non-depressed mothers

Three additional ANOVAs were conducted to test whether there were differences in accelerated epigenetic age among children with and without a depressed mother. There were no differences in accelerated epigenetic age among children with depressed and non-depressed mothers (see Table 5). The effect of maternal depression may still occur though in a dose-dependent manner which was addressed in the mediation analyses using maternal depression chronicity.

3.4 Mediation Analyses

Three mediational regression models were tested to examine the indirect effect of chronicity of maternal depression during childhood on change in accelerated epigenetic age, through maternal parenting. To compute change in accelerated epigenetic aging between ages 9 and 15, a latent change score (McArdle, 2009) was estimated. Child sex, marital status at birth, household income at time of birth, and mother smoking status during pregnancy were included as covariates. Two additional covariates of accelerated epigenetic age were included: assay type (i.e.: Illumina450k or IlluminaEPIC) and the estimated proportion of epithelial cells in each

saliva sample. Separate models were tested for each epigenetic clock (e.g., PhenoAge, GrimAge, PACE).

3.4.1 PhenoAge Model

First, analyses were conducted testing the mediation model with the PhenoAge clock to estimate accelerated epigenetic age change (see Figure 2 and Table 6). The model had adequate fit to the data CFI = .88, RMSEA = .08, χ^2 (120) = 6336.02, $p < .001$. Maternal depression chronicity was associated with physical aggression ($\beta = .11$, $p < .001$) and psychological aggression ($\beta = .17$, $p < .001$) such that greater chronicity in maternal depression was associated with higher levels of both physical and psychological aggression. However, maternal depression was not associated with maternal engagement ($\beta = .01$, $p = .70$). Longitudinal change in accelerated epigenetic aging was not associated with maternal parenting (physical aggression: $\beta = .01$, $p = .46$, psychological aggression: $\beta = .00$, $p = .94$, maternal engagement: $\beta = .02$, $p = .46$). Additionally, there were no direct associations between maternal depression chronicity and longitudinal change in accelerated epigenetic aging as measured by PhenoAge clock ($\beta = .02$, $p = .46$).

3.4.2 GrimAge Model

Next, analyses were conducted testing the mediation model with the GrimAge clock to estimate longitudinal change in adolescent accelerated epigenetic age. The model had an acceptable fit (CFI = .90, RMSEA = .08, and χ^2 (120) = 7659.98, $p < .001$; see Figure 3 and Table 7). Maternal depression chronicity was not directly associated with change in accelerated epigenetic age using the GrimAge clock ($\beta = .02$, $p = .50$). More maternal depression chronicity was associated with higher levels of physical aggression ($\beta = .11$, $p < .001$) and psychological aggression ($\beta = .17$, $p < .001$), but not maternal engagement ($\beta = .01$, $p = .71$). Increased maternal

physical aggression was associated with greater change in accelerated epigenetic age change using the GrimAge clock (β = .06, p = .02). There was a significant indirect effect of maternal depression chronicity on change accelerated epigenetic age through maternal physical aggression (β = .01, p = .02). This suggests that mothers who were more chronically depressed used higher rates of physically harsh parenting behaviors and in turn these higher rates of maternal physical aggression were associated with greater increases in accelerated epigenetic aging during adolescence. Increased maternal engagement was also associated with greater change in epigenetic age acceleration when measured using the GrimAge clock (β = .05, p = .02). This finding suggests that children of more engaging mothers were more likely to experience a greater change in accelerated epigenetic age. Lastly, there was no association between psychological aggression and change in accelerated epigenetic age. (β = -.01, p = .74).

3.4.3 Dunedin PACE Model

Lastly, analyses were conducted testing the mediation model with the Dunedin PACE clock to estimate longitudinal change in adolescent accelerated epigenetic age. The model had adequate fit to the data (CFI = .89, RMSEA = .08, $p < .001$, and χ^2 (120) = 6817.34, $p < .001$; See Figure 3 and Table 9). Maternal depression chronicity was significantly associated with higher levels of physical aggression (β = .11, $p < .001$) and psychological aggression (β = .17, p <.001), but not maternal engagement (β = .01, p = .74) nor to change in accelerated epigenetic age using the PACE clock (B = .01, p = .87). There was no association between physical aggression (β = .00, p =.35) nor psychological aggression (β = .03, p = .91) with change in accelerated epigenetic age as measured by the Dunedin PACE clock. Maternal engagement was significantly associated with change in PACE acceleration, (β = .00, $p < .001$). This finding

suggests that children of more engaging mothers were more likely to experience a greater change in the pace of epigenetic age acceleration.

CHAPTER 4

DISCUSSION

In this study, I sought to investigate whether more chronic exposure to maternal depression across childhood was associated with greater change in accelerated epigenetic age between ages nine and fifteen using three different epigenetic clocks, and whether this association was mediated by distinct aspects of maternal parenting including physical aggression, psychological aggression, and engagement.

While I expected maternal depression to be directly associated with change in epigenetic age acceleration, this was not the case in the current study. This outcome was surprising considering the nature of maternal depression as a highly proximal and salient stressor (Cummings & Davies, 1994; Goodman & Gotlib, 1999). Theoretically, the effects of maternal depression may operate through more proximal behavioral mechanisms, such as parenting, that directly impacts youth which is supported in part the current study through the tests of parenting dimensions as mediators.

As expected, maternal physical aggression mediated the association between cumulative maternal depression and change in epigenetic age acceleration as measured by the GrimAge clock. To my knowledge, this was the first study to demonstrate that maternal physical aggression may serve as a parenting behavioral mechanism linking maternal depression with greater changes in accelerated epigenetic aging across adolescence. This finding was consistent with prior literature indicating detrimental effects of maternal depression on parenting practices (NCRIM, 2009), and with literature indicating that harsh parenting practices affect epigenetic age (e.g., Creasey et al., 2024; Mastrothodoros et al., 2023; Mrug et al., 2023). This finding is

consistent with aspects of Life History Theory (Del Giuduce & Belsky, 2010) which posits that harsh and unpredictable environments lead to accelerated development. Physical aggression in parenting may constitute a threatening or unpredictable environment for youth, resulting in greater change in biological age acceleration during adolescence.

The fact that this association was found using GrimAge bears important implications. Firstly, Grimage, PhenoAge, and Dunedin PACE are all second-generation clocks designed with health risk indicators as factors in their estimation. Each of these clocks have demonstrated a greater ability to predict health outcomes than first generation clocks (Belsky et al., 2022; Levine et al., 2018; Lu et al, 2019). Moreover, all three have been shown to be associated with variations in childhood adversity with greater sensitivity than first generation clocks (Ecker & Beck, 2019; Maddock et al., 2020; Li et al., 2020; Zhou et al., 2019). However, the mediation findings in this study are only found with respect to estimates of accelerated epigenetic age change using the GrimAge clock. This may be due to GrimAge using the most CpG sites of all currently developed epigenetic clocks, and that GrimAge allows individual CpG sites to predict epigenetic age through multiple health biomarkers (Li et al., 2020). Additionally, while all three of these clocks have all demonstrated capability of reflecting early experiences, GrimAge appears to be more sensitive to environmental factors than the other two (Oblak et al., 2021), and therefore may be more strongly associated with variations in early life experiences better than the other two clocks. This has been supported by limited emerging research (Aikins et al., 2024; Graf et al., 2022; Petrovic et al., 2023; Suglia et al., 2024), although there have been contrasting findings as well (e.g. Farina et al., 2024; Rampersaud et al., 2022). Future research is needed to make comparisons among the second-generation clocks. Further, it is noteworthy that GrimAge generally outperforms PhenoAge, and to a lesser extent DunedinPACE, in predicting health

outcomes and time-to-death (Belsky et al., 2022; Hillary et al., 2019; Lu et al., 2019; Maddock et al., 2019; McCrory et al., 2022). Collectively, this emerging empirical research supports the use of GrimAge in lifespan models connecting environmental stressors and health outcomes.

Psychological aggression in maternal parenting did not mediate the association between maternal depression and change in epigenetic age acceleration as measured by any of the three clocks. This was unexpected considering previous research demonstrating evidence for an association between emotional abuse in youth and accelerated epigenetic age during in later adulthood using GrimAge (Tang et al., 2020) and PhenoAge (Joshi et al., 2023), as well as previous research which has shown a significant indirect association between maternal depression and epigenetic age acceleration during emerging adulthood through a general measure of harsh parenting using the first generation Horvath Clock (Brody et al., 2016; Horvath, 2013). The contrasting findings to Brody et al. (2016) may be due to their use of a cumulative metric of both physical and psychological behaviors that comprise their harsh parenting metric, whereas in the current study these were modeled as separate factors and analyses controlling for their unique effects. It may be that the effect of physical harsh parenting in the Brody et al. (2016) study was responsible for the majority of variance in epigenetic aging. The current study's contrasting findings to Joshi et al. (2023) and Tang et al. (2020) may be due to the fact that both used single dichotomous indicators for emotional abuse, based on the Adverse Childhood Experiences (ACEs) self-report questionnaire (Felitti et al., 1998), whereas the current study used the average of five items from the Conflict Tactics Scale at child age nine as indicators. Additionally, ACEs were retrospectively indicated and measured later in adulthood. Research finds differences in reports of prospective versus retrospective measures of adversity (Reuben et al., 2016). Another important difference is that the current study uses

longitudinal change in epigenetic age acceleration as an outcome, whereas all three of these other studies used epigenetic age at a single time point, and in the case of Joshi et al. (2023) and Tang et al. (2020) at a much later age into adulthood. It may be that the effect of psychological aggression takes a longer time to manifest as accelerated epigenetic age, and therefore may be undetectable during childhood and adolescence despite appearing in later adulthood.

Also contrary to expectations, maternal engagement did not significantly mediate the association between maternal depression chronicity and change in accelerated epigenetic age in any of the three clocks. The lack of association between maternal depression and maternal engagement was a contradictory to much of the prior literature and considering the consistency with which negative outcomes are reported in adolescents who experience low maternal engagement (Lovejoy, 2000). This may be due to measuring maternal depression as a cumulative score across childhood. This approach removes the variability of the time-varying changes in depression. Also notable was the result that maternal engagement was positively associated with change in epigenetic age acceleration using the GrimAge clock, suggesting that more maternal engagement led to acceleration in biological aging. To my knowledge, this has been the first study to investigate the association between maternal engagement and epigenetic age acceleration in adolescents, and further research will be necessary to replicate this counterintuitive finding. Little research has examined longitudinal changes in accelerated epigenetic age. Future research is needed to understand how the timing of changes at various points in the lifespan correspond to both environmental stressors and later health outcomes.

This study was limited by the fact that probable diagnosis of depression was used to measure maternal depression at each time point using the Composite International Diagnostic Interview - Short Form (CIDI-SF) (Kessler et al., 1998). This limited the amount of variability in

mother depression scores such that symptomology that only reached sub diagnostic levels was not accounted for in this measure. Future research should consider using metrics of depressive symptoms.

4.1 Conclusion

This study provided evidence for a mediating role of maternal physical aggression as a parenting behavioral mechanism linking maternal depression chronicity and change in epigenetic age acceleration between ages nine and fifteen years using the GrimAge clock. Evidence from the current study suggests that maternal depression may lead to increased use of physical aggression in parenting, which in turn may lead to greater increase in in accelerated epigenetic age across early adolescence. To my knowledge, this is the first study in which this association has been investigated using second generation epigenetic clocks, and the first to investigate an association between maternal depression and longitudinal change in accelerate epigenetic age. This bears important implications for developmental science as it provides evidence to identify environmentally mediated mechanisms of parenting impacting biological age change during the complex period of early adolescence.

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TABLES AND FIGURES

Table 1. Descriptive statistics for study variables

	n	Min	Max	*M*	*SD*
Maternal Depression Chronicity	1969	0.00	4.00	0.69	1.02
Physical Aggression	1842	0.00	20.00	1.33	2.31
Psychological Aggression	1842	0.00	25.00	3.73	3.95
Maternal Engagement	1883	0.00	28.00	15.11	4.77
Age 9 Epigenetic Age – PhenoAge Clock	1971	-26.43	21.63	-4.99	6.53
Age 9 Epigenetic Age – GrimAge Clock	1971	-19.63	28.88	5.51	6.45
Age 9 Epigenetic Age – PACE Clock	1971	.80	1.76	1.22	0.16
Age 15 Epigenetic Age – PhenoAge Clock	1974	.78	1.91	1.27	0.17
Age 15 Epigenetic Age – GrimAge Clock	1974	-5.26	43.30	16.37	5.70
Age 15 Epigenetic Age – PACE Clock	1974	5.27	46.17	22.55	6.14
Age 9 Accelerated Epigenetic Age – PhenoAge Clock	1971	-21.56	24.18	0.00	5.82
Age 9 Accelerated Epigenetic Age – GrimAge Clock	1971	-18.12	22.18	0.00	3.92
Age 9 Accelerated Epigenetic Age – PACE Clock	1971	-0.41	0.52	0.00	0.16
Age 15 Accelerated Epigenetic Age – PhenoAge Clock	1906	-22.08	21.61	0.00	5.80
Age 15 Accelerated Epigenetic Age – GrimAge Clock	1906	-13.87	20.70	0.00	4.32
Age 15 Accelerated Epigenetic Age – PACE Clock	1906	-0.49	0.62	0.00	0.18
Household Income (ln)	1971	0.00	11.80	9.92	1.28

Note. Household income is represented by the natural log of income reported at time of birth. Epigenetic age measures reflect raw scores on each clock measure; accelerated epigenetic age measures reflect ages scores residuals after removing the effect of chronological age.

Table 2. Frequencies of binary and categorical demographics and study variables

	n	%
Child Sex - Male	992	50.33
Child Sex - Female	979	49.67
Parental Marital Status at Birth - Married	486	24.66
Parental Marital Status at Birth - Unmarried	1485	75.34
Prenatal Maternal Smoking - Yes	376	19.10
Prenatal Marital Status - No	1592	80.90
Race/Ethnicity - Black	935	47.50
Race/Ethnicity - Hispanic	517	26.28
Race/Ethnicity - White	445	22.62
Race/Ethnicity - Other	70	3.56
Maternal Depression – Yes	800	40.63
Maternal Depression - No	1169	59.37

Table 3. Bivariate correlations for all variables

	1	2	3	4	5	6	7	8	9	10	11	12	13	14	15	16	17
1. Maternal Depression Chronicity	1																
2. Maternal Engagement	.01	1															
3. Physical Aggression	.11**	.00	1														
4.Psychological Aggression	.17**	-.12**	.53**	1													
5. Age 9 PhenoAge Clock	.01	.01	.03	.01	1												
6 Age 9 GrimAge Clock	.02	.01	.06*	.03	44**	1											
7. Age 9 PACE Clock	.02	.04	.04	.01	.41**	.67**	1										
8. Age 15 PhenoAge Clock	.02	.02	.02	.01	.56**	.19**	.15**	1									

9. Age 15 GrimAge Clock	.01	.05	.06*	.02	.18**	.42**	.22**	.48**	1								
10. Age 15 PACE Clock	.00	.07**	.03	.00	.12**	.15**	.32**	.43**	.70**	1							
11. Assay Type	-.06**	-.02	-.06*	-.07**	.00	.00	.00	.00	.00	.00	1						
12. Age 9 Epithelial Cell Proportions	-.01	.01	.06*	.02	.36**	.63**	.55**	.08**	.12**	.10**	-.07**	1					
13. Age 15 Epithelial Cell Proportions	-.02	.03	.04	.00	.00	.08**	.12*	.36**	.69**	.57**	-.08**	.15**	1				
14. Child Sex	-.01	.07**	-.07**	-.08**	.14**	-.15**	.02	.19**	-.06**	.14**	.00	-.04	-.02	1			
15. Marital Status at Birth	-.10**	-.07**	-.04	-.07**	-.06**	-.06**	-.09**	-.04	-.04	.10**	.02	.02	-.02	-.04	1		
16. Household Income at Birth (ln)	-.11**	-.03	-.06*	-.03	-.02	-.05*	-.08**	-.01	-.09**	-.09**	.04*	.01	-.03	-.02	.35**	1	
17. Maternal Smoking at Birth	.14**	.04	.01	.10**	.00	-.04	-.06**	.01	.00	.00	-.03	-.04	.00	.02	-.14**	-.13**	1

Note. * = $p < .05$, ** = $p < .001$. Binary variables were coded as follows. Assay Type: 0 = Illumina Methylation450K, 1 = IlluminaEPIC; Child Sex: 0 = Boy, 1 = Girl; Marital Status at Birth: 0 = unmarried, 1 = married; Maternal Smoking at Birth: 0 = non-smoking, 1 = smoking.

Table 4. Results from ANOVAs testing for sex differences in accelerated epigenetic age within each epigenetic
clock at age 9 and 15

	Boys *M (SD)*	Girls *M (SD)*	F	*df*	*p*
Age 9 Accelerated Epigenetic Age – PhenoAge Clock	-0.80 (5.84)	.081 (5.68)	38.28	1, 1969	<.001***
Age 15 Accelerated Epigenetic Age – PhenoAge Clock	-1.11 (5.85)	1.07 (5.55)	69.17	1, 1904	<.001***
Age 9 Accelerated Epigenetic Age – GrimAge Clock	-.059 (4.03)	-0.59 (3.72)	45.31	1, 1969	<.001***
Age 15 Accelerated Epigenetic Age – GrimAge Clock	0.24 (4.45)	-0.30 (4.71)	7.45	1, 1904	.006**
Age 9 Accelerated Epigenetic Age – PACE Clock	0.00 (0.16)	0.00 (0.15)	0.01	1, 1969	.52
Age 15 Accelerated Epigenetic Age – PACE Clock	-0.03 (0.18)	0.02 (0.17)	1.11	1, 1904	<.001***

Note. * = $p < .05$, ** = $p < .005$, *** = $p < .001$

Table 5. Results from ANOVAs comparing epigenetic age acceleration between children of depressed and non-depressed mothers by age and epigenetic clock

	Depression at any point	No Depression at any point	*F*	*df*	*p*
Age 9 Accelerated Epigenetic Age – PhenoAge Clock	0.10 (5.84)	-0.07 (5.81)	0.413	1, 1967	.52
Age 15 Accelerated Epigenetic Age – PhenoAge Clock	0.19 (5.80)	-.17 (5.81)	1.73	1, 1902	.19
Age 9 Accelerated Epigenetic Age – GrimAge Clock	0.06 (3.85)	-0.04 (3.98)	0.30	1, 1967	.58
Age 15 Accelerated Epigenetic Age – GrimAge Clock	0.00 (0.16)	-0.09 (4.33)	0.61	1, 1902	.44
Age 9 Accelerated Epigenetic Age – PACE Clock	0.00 (0.17)	0.00 (0.16)	0.15	1, 1967	.70
Age 15 Accelerated Epigenetic Age – PACE Clock	0.00 (0.18)	0.00 (0.17)	0.11	1, 1902	.75

Note. * = $p < .05$, ** = $p < .005$, *** = $p < .001$

Table 6. Mediation path analysis model examining maternal parenting as mediators between maternal depression chronicity and change in adolescent accelerated epigenetic age using the PhenoAge clock.

Direct Effects Estimates	*B* (*SE*)	*ß*	*p*
Maternal depression chronicity →Accelerated epigenetic age change	0.10 (0.14)	0.02	.46
Maternal depression chronicity →Maternal physical aggression	0.25 (0.05)	0.11	<.001***
Maternal depression chronicity →Maternal psychological aggression	0.64 (0.08)	0.17	<.001***
Maternal depression chronicity →Maternal engagement	0.04 (0.10)	0.01	.70
Maternal physical aggression → Accelerated epigenetic age change	0.03 (0.07)	0.01	.71
Maternal psychological aggression → Accelerated epigenetic age change	0.00 (0.04)	0.00	.94
Maternal engagement → Accelerated epigenetic age change	0.02 (0.03)	0.02	.46
Indirect Effect Estimates			
Maternal depression chronicity → Maternal physical aggression→ Accelerated epigenetic age change	0.01 (0.03)	0.00	.71
Maternal depression chronicity → Maternal psychological aggression → Accelerated epigenetic age change	0.00 (0.02)	0.00	.94
Maternal depression chronicity → Maternal engagement → Accelerated epigenetic age change	0.00 (0.00)	0.00	.73
Covariate estimates in relation to study variables			
Child Sex ↔ Maternal depression chronicity	-0.02 (0.03)	-0.02	.59
Black ↔ Maternal depression chronicity	0.10 (0.03)	0.10	<.001***
Hispanic ↔ Maternal depression chronicity	-0.11 (0.03)	-0.10	.001**
Other ↔ Maternal depression chronicity	-0.11 (0.06)	-0.10	0.09

Marital status at birth ↔ Maternal depression chronicity	-0.14 (0.03)	-0.14	<.001***
Household Income ↔ Maternal depression chronicity	-0.15 (0.03)	-0.11	<.001***
Mother smoking status at birth ↔ Maternal depression chronicity	0.19 (0.03)	0.18	<.001***
Maternal psychological aggression ↔ Maternal physical aggression	4.69 (0.15)	0.53	<.001***
Maternal engagement ↔ Maternal physical aggression	-0.00 (0.29)	-0.00	.97
Child Sex ↔ Maternal physical aggression	-0.19 (0.06)	-0.08	.002**
Black ↔ Maternal physical aggression	0.57 (0.06)	0.25	<.001***
Hispanic ↔ Maternal physical aggression	-0.44 (0.09)	-0.19	<.001***
Other ↔ Maternal physical aggression	-0.11 (0.12)	-0.05	.34
Marital status at birth ↔ Maternal physical aggression	-0.07 (0.05)	-0.03	.17
Household Income ↔ Maternal physical aggression	-0.13 (0.06)	-0.05	.03
Mother smoking status at birth ↔ Maternal physical aggression	-0.01 (0.08)	-0.00	.93
Maternal engagement ↔ Maternal psychological aggression	-2.35 (0.37)	-0.23	<.001***
Child Sex ↔ Maternal psychological aggression	-0.41 (0.11)	-0.11	<.001***
Black ↔ Maternal psychological aggression	0.67 (0.11)	-0.17	<.001***
Hispanic ↔ Maternal psychological aggression	-0.93 (0.13)	-0.24	<.001***
Other ↔ Maternal psychological aggression	-0.23 (0.22)	-0.06	.30
Marital status at birth ↔ Maternal psychological aggression	-0.28 (0.12)	-0.07	.02
Household Income ↔ Maternal psychological aggression	-0.04 (0.12)	-0.01	.77
Mother smoking status at birth ↔ Maternal psychological aggression	0.44 (0.13)	0.11	<.001***

Child Sex ↔ Maternal engagement	0.40 (0.14)	0.08	.004**
Black ↔ Maternal engagement	0.80 (0.13)	0.17	<.001***
Hispanic ↔ Maternal engagement	-0.81 (0.14)	-0.17	<.001***
Other ↔ Maternal engagement	0.02 (0.27)	0.00	.95
Marital status at birth ↔ Maternal engagement	-0.44 (0.16)	-0.09	.005**
Household Income ↔ Maternal engagement	-0.19 (0.14)	-0.03	.17
Mother smoking status at birth ↔ Maternal engagement	0.30 (0.15)	0.06	.05
Child Sex ↔ Accelerated epigenetic age change	1.18 (0.14)	-0.20	<.001***
Black ↔ Accelerated epigenetic age change	0.09 (0.14)	0.02	.51
Hispanic ↔ Accelerated epigenetic age change	0.34 (0.16)	0.06	.04
Other ↔ Accelerated epigenetic age change	0.01 (0.30)	0.03	.96
Marital status at birth ↔ Accelerated epigenetic age change	-0.36 (0.11)	-0.07	.02
Household Income ↔ Accelerated epigenetic age change	-0.11 (0.15)	-0.01	.49
Mother smoking status at birth ↔ Accelerated epigenetic age change	0.05 (0.17)	-0.01	.77
Estimates for latent change in epigenetic age acceleration			
Intercept	-.048 (0.51)	-0.88	.35
Residual variance	29.40 (0.77)	1.00	<.001***
Coefficients of determination for study variables			
Maternal engagement R^2	0.00	-	-
Physical aggression R^2	0.01	-	-
Psychological aggression R^2	0.03	-	-
Change in epigenetic age acceleration R^2	0.00	-	-

Table 7. Mediation path analysis model examining maternal parenting as mediators between maternal depression chronicity and change in adolescent accelerated epigenetic age using the GrimAge clock.

	B (*SE*)	*ß*	*p*
Maternal Depression→ Accelerated epigenetic age change	0.07 (0.10)	0.02	0.50
Maternal depression → Physical aggression	0.25 (0.05)	0.11	<.001***
Maternal depression → Psychological aggression	0.64 (0.08)	0.17	<.001***
Maternal depression → Maternal engagement	0.04 (0.10)	0.01	0.71
Physical aggression → Accelerated epigenetic age change	0.12 (0.05)	0.06	0.02*
Psychological aggression → Accelerated epigenetic age change	-0.10 (0.03)	-0.01	0.74
Maternal engagement → Accelerated epigenetic age change	0.05 (0.02)	0.05	0.02*
Indirect Effect Estimates			
Maternal depression → Physical aggression→ Accelerated epigenetic age change	0.03 (0.01)	0.01	0.02*
Maternal depression → Psychological aggression→ Accelerated epigenetic age change	-0.01 (0.02)	0.00	0.74
Maternal depression → maternal engagement→ Accelerated epigenetic age change	0.00 (0.01)	0.00	0.72
Covariances between study variables and covariates			
Child Sex ↔ Maternal depression chronicity	-0.02 (0.03)	-0.02	.59
Black ↔ Maternal depression chronicity	0.10 (0.03)	0.10	<.001***
Hispanic ↔ Maternal depression chronicity	-0.11 (0.03)	-0.10	0.001**

Other ↔ Maternal depression chronicity	-0.11 (0.06)	-0.10	0.09
Marital status at birth ↔ Maternal depression chronicity	-0.14 (0.03)	-0.14	<.001***
Household Income ↔ Maternal depression chronicity	-0.15 (0.03)	-0.11	<.001***
Mother smoking status at birth ↔ Maternal depression chronicity	0.19 (0.03)	0.18	<.001***
Maternal psychological aggression ↔ Maternal physical aggression	4.69 (0.15)	0.53	<.001***
Maternal engagement ↔ Maternal physical aggression	-0.01 (0.29)	-.00	.97
Child Sex ↔ Maternal physical aggression	-0.18 (0.06)	-0.08	.003**
Black ↔ Maternal physical aggression	0.57 (0.06)	0.25	<.001***
Hispanic ↔ Maternal physical aggression	-0.44 (0.09)	-0.19	<.001***
Other ↔ Maternal physical aggression	-0.11 (0.12)	-0.05	.34
Marital status at birth ↔ Maternal physical aggression	-0.08 (0.05)	-0.03	.15
Household Income ↔ Maternal physical aggression	-0.14 (0.06)	-0.05	.03
Mother smoking status at birth ↔ Maternal physical aggression	-0.00 (0.08)	-0.00	.97
Maternal engagement ↔ Maternal psychological aggression	-2.35 (0.37)	-0.23	<.001***
Child Sex ↔ Maternal psychological aggression	-0.41 (0.11)	-0.11	<.001***
Black ↔ Maternal psychological aggression	0.67 (0.11)	-0.17	<.001***

Hispanic ↔ Maternal psychological aggression	-0.93 (0.13)	-0.24	<.001***
Other ↔ Maternal psychological aggression	-0.23 (0.23)	-0.06	.30
Marital status at birth ↔ Maternal psychological aggression	-0.28 (0.14)	-0.07	.02
Household Income ↔ Maternal psychological aggression	-0.04 (0.13)	-0.01	.78
Mother smoking status at birth ↔ Maternal psychological aggression	0.44 (0.13)	0.11	<.001***
Child Sex ↔ Maternal engagement	0.41 (0.14)	0.09	.003**
Black ↔ Maternal engagement	0.81 (0.13)	0.17	<.001***
Hispanic ↔ Maternal engagement	-0.81 (0.14)	-0.14	<.001***
Other ↔ Maternal engagement	0.02 (0.27)	0.01	.94
Marital status at birth ↔ Maternal engagement	-0.45 (0.16)	-0.09	.004
Household Income ↔ Maternal engagement	-0.20 (0.14)	-0.03	.15
Mother smoking status at birth ↔ Maternal engagement	0.30 (0.15)	0.06	.04
Child Sex ↔ Accelerated epigenetic age change	-0.57 (0.31)	-0.15	<.001***
Black ↔ Accelerated epigenetic age change	0.78 (0.09)	0.20	<.001***
Hispanic ↔ Accelerated epigenetic age change	-0.19 (0.11)	-0.05	.08
Other ↔ Accelerated epigenetic age change	-0.29 (0.17)	-0.07	.10
Marital status at birth ↔ Accelerated epigenetic age change	-0.44 (0.10)	-0.11	<.001***

Household Income ↔ Accelerated epigenetic age change	-0.34 (0.09)	-0.07	<.001***
Mother smoking status at birth ↔ Accelerated epigenetic age change	-0.14 (0.11)	-0.04	.22
Estimates for latent change in epigenetic age acceleration			
Intercept	-0.93 (0.37)	-0.21	.01*
Residual variance	19.63 (0.46)	0.99	<.001***
Coefficients of determination for study variables			
Maternal engagement R^2	0.00	-	-
Physical aggression R^2	0.01	-	-
Psychological aggression R^2	0.03	-	-
Change in epigenetic age acceleration R^2	0.01	-	-

Note. * = $p < .05$, ** = $p < .005$, *** = $p < .001$

Table 8. Mediation path analysis model examining maternal parenting as mediators between maternal depression chronicity and change in adolescent accelerated epigenetic age using the Dunedin PACE clock.

	B (*SE*)	*ß*	*p*
Maternal Depression→ Accelerated epigenetic age change	0.00 (.00)	0.00	0.87
Maternal depression → Physical aggression	0.25 (0.05)	0.11	<.001***
Maternal depression → psychological aggression	0.65 (0.08)	0.17	<.001***
Maternal depression → Maternal engagement	0.04 (0.11)	0.01	.71
Physical aggression → Accelerated epigenetic age change	0.00 (0.00)	0.03	.35
Psychological aggression → Accelerated epigenetic age change	0.00 (0.00)	0.00	.91
Maternal engagement → Accelerated epigenetic age change	0.00 (0.00)	0.00	.001**
Indirect Effect Estimates			
Maternal depression → Physical aggression→ Accelerated epigenetic age change	0.00 (0.00)	0.00	0.27
Maternal depression → Psychological aggression→ Accelerated epigenetic age change	0.00 (0.00)	0.01	0.79
Maternal depression → Maternal engagement→ Accelerated epigenetic age change	0.00 (0.00)	0.00	0.12
Covariances among study variables and covariates			
Child Sex ↔ Maternal depression chronicity	-0.02 (0.03)	-0.01	.67
Black ↔ Maternal depression chronicity	0.10 (0.03)	0.11	<.001***
Hispanic ↔ Maternal depression chronicity	-0.11 (0.03)	-0.11	<.001***
Other ↔ Maternal depression chronicity	-0.11 (0.06)	-0.14	0.09
Marital status at birth ↔ Maternal depression chronicity	-0.14 (0.03)	-0.15	<.001***
Household Income ↔ Maternal depression chronicity	-0.15 (0.03)	-0.12	<.001***
Mother smoking status at birth ↔ Maternal depression chronicity	0.19 (0.03)	0.19	<.001***

Maternal psychological aggression ↔ Maternal physical aggression	4.69 (0.15)	0.53	<.001***
Maternal engagement ↔ Maternal physical aggression	-0.01 (0.29)	.00	.99
Child Sex ↔ Maternal physical aggression	-0.19 (0.06)	-0.08	.002**
Black ↔ Maternal physical aggression	0.57 (0.06)	0.25	<.001***
Hispanic ↔ Maternal physical aggression	-0.44 (0.09)	-0.19	<.001***
Other ↔ Maternal physical aggression	-0.11 (0.12)	-0.05	.33
Marital status at birth ↔ Maternal physical aggression	-0.08 (0.05)	-0.03	.16
Household Income ↔ Maternal physical aggression	-0.13 (0.06)	-0.05	.04
Mother smoking status at birth ↔ Maternal physical aggression	-0.00 (0.08)	-0.00	.96
Maternal engagement ↔ Maternal psychological aggression	-2.35 (0.37)	-0.13	<.001***
Child Sex ↔ Maternal psychological aggression	-0.41 (0.11)	-0.11	<.001***
Black ↔ Maternal psychological aggression	0.67 (0.11)	-0.17	<.001***
Hispanic ↔ Maternal psychological aggression	-0.93 (0.13)	-0.24	<.001***
Other ↔ Maternal psychological aggression	-0.23 (0.22)	-0.06	.30
Marital status at birth ↔ Maternal psychological aggression	-0.28 (0.12)	-0.07	.02
Household Income ↔ Maternal psychological aggression	-0.04 (0.12)	-0.01	.77
Mother smoking status at birth ↔ Maternal psychological aggression	0.44 (0.13)	0.11	.001**
Child Sex ↔ Maternal engagement	0.43 (0.14)	0.09	.002**
Black ↔ Maternal engagement	0.82 (0.14)	0.17	<.001***
Hispanic ↔ Maternal engagement	-0.81 (0.14)	-0.17	<.001***
Other ↔ Maternal engagement	0.01 (0.27)	0.00	.97
Marital status at birth ↔ Maternal engagement	-0.44 (0.16)	-0.09	.004**
Household Income ↔ Maternal engagement	-0.19 (0.14)	-0.03	.15

Mother smoking status at birth ↔ Maternal engagement	0.01 (0.15)	0.07	.04*
Child Sex ↔ Accelerated epigenetic age change	0.01 (0.00)	0.09	<.001***
Black ↔ Accelerated epigenetic age change	0.03 (0.00)	0.18	<.001***
Hispanic ↔ Accelerated epigenetic age change	0.00 (0.00)	0.01	.75
Other ↔ Accelerated epigenetic age change	-0.02 (0.01)	-0.15	.001**
Marital status at birth ↔ Accelerated epigenetic age change	-0.02 (0.00)	-0.12	<.001***
Household Income ↔ Accelerated epigenetic age change	-0.02 (0.00)	-0.09	<.001***
Mother smoking status at birth ↔ Accelerated epigenetic age change	-0.01 (0.00)	-0.05	.05
Estimates for latent change in epigenetic age acceleration			
Intercept	-0.05 (0.02)	-0.25	.002**
Residual variance	0.04 (0.00)	0.99	<.001***
R^2 values for study variables			
Maternal engagement R^2	0.00	-	-
Physical aggression R^2	0.01	-	-
Psychological aggression R^2	0.03	-	-
Change in epigenetic age acceleration R^2	0.01	-	-

Note. * = $p < .05$, ** = $p < .005$, *** = $p < .001$

Figure 1. Conceptual model for current study investigating physical aggression, psychological aggression, and engagement as parenting practices that mediate the association between chronicity of maternal depression and change in epigenetic age acceleration.

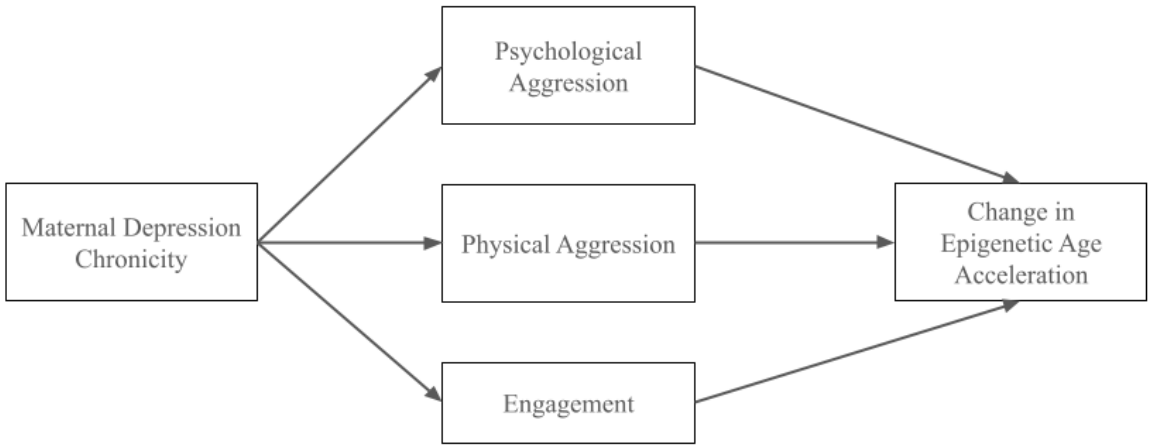
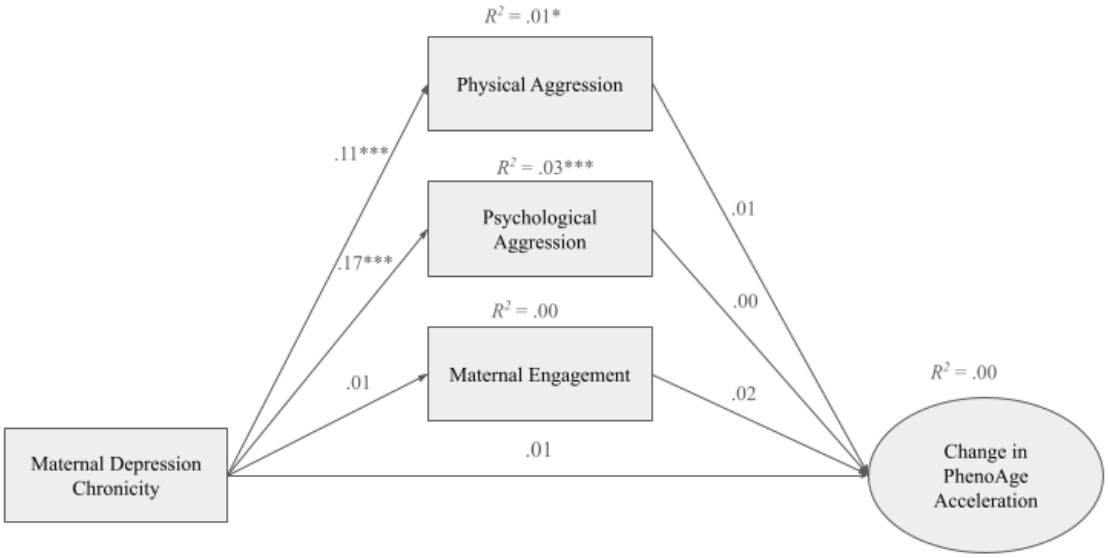
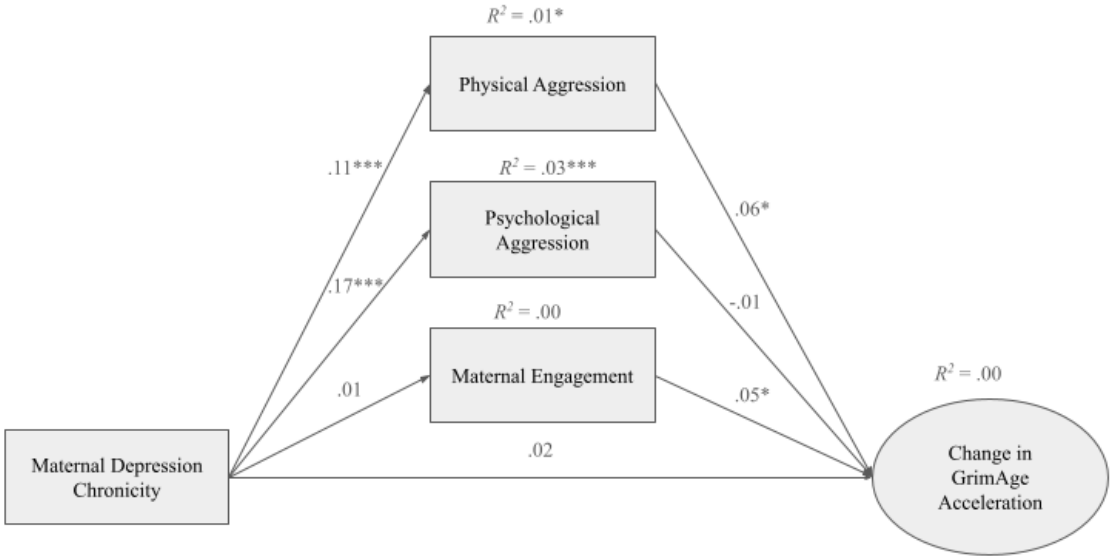


Figure 2. Model examining maternal parenting as mediators between matenral depression chronicity and accelerated epigenetic age using the PhenoAge clock.



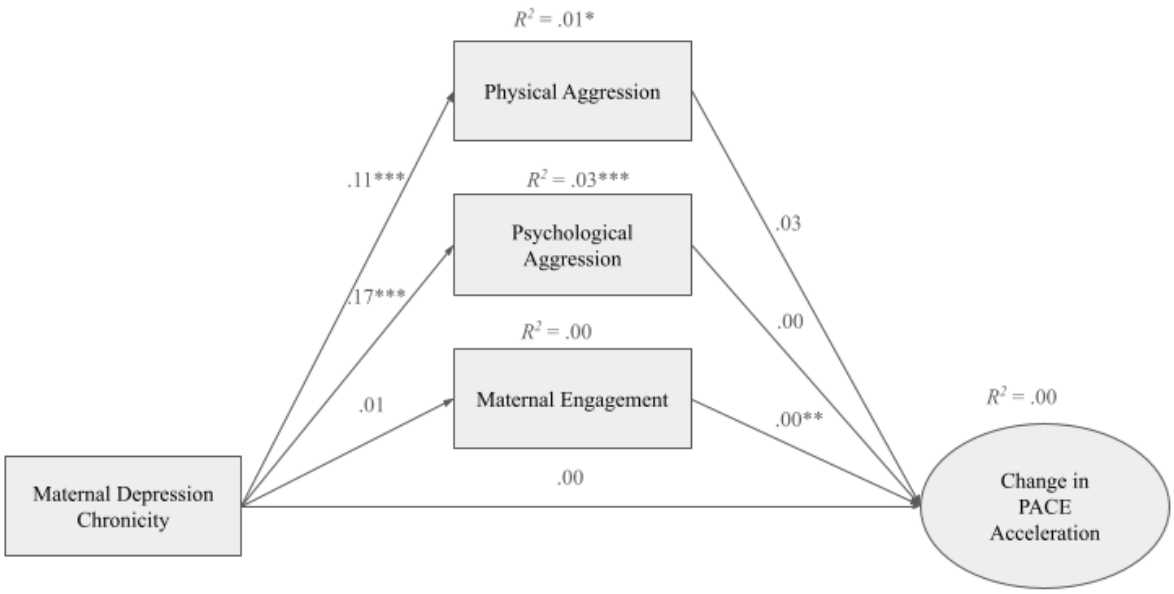
Note. * = $p < .05$, ** = $p < .005$, *** = $p < .001$. All coefficients are standardized.

Figure 3 Model examining maternal parenting as mediators between matenral depression chronicity and accelerated epigenetic age using the GrimAge clock.



Note. * = $p < .05$, ** = $p < .005$, *** = $p < .001$. All coefficients are standardized.

Figure 4. Model examining maternal parenting as mediators between matenral depression chronicity and accelerated epigenetic age using the Dunedin PACE clock.



Note. * = $p < .05$, ** = $p < .005$, *** = $p < .001$. All coefficients are standardized.