

Associations among prenatal stress, socioeconomic status, and infant epigenetic aging

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ABSTRACT

This pre-registered study investigated the associations between prenatal stress and socioeconomic disadvantage with epigenetic aging in one-month-old infants. We hypothesized that exposure to greater maternal perceived stress, maternal physiological stress, and family socioeconomic disadvantage would be associated with accelerated epigenetic aging among infants. A socioeconomically and racially diverse sample of mothers were recruited during their last 5 weeks of pregnancy, when they completed surveys on their family income, education, and perceived stress levels, and provided a hair sample to index hair cortisol concentration. At 1-month postpartum, saliva samples were collected from their infants to assess genome-wide DNA methylation ($n = 159$). Epigenetic age was assessed using the Horvath, PedBE, PhenoAge, GrimAge, and DunedinPACE clocks. There was no consistent association across socioeconomic and stress factors with infant epigenetic aging. Findings are situated within effect sizes reported from previous studies, and we consider the validity of applying epigenetic clocks to infant populations. The diverging effects of family income, education, perceived stress, and physiological stress on development are considered.

1. Introduction

Children who experience early life stress and socioeconomic disadvantage are more likely to experience behavioral, cognitive, and physical health challenges across the lifespan (Shonkoff et al., 2012). Examination of the potential biological mediators of these developmental effects has implicated epigenetic mechanisms, such as DNA methylation, which are sensitive to adverse experiences and may also lead to stable transcriptional changes that impact physiological, neurobiological, and immune pathways with implications for health and well-being (Scorza et al., 2019). Variation in DNA methylation within the genome may also indicate biological aging. Increasing evidence suggests that acceleration in this molecular measure of aging is predictive of chronic disease and mortality in later life (Horvath and Raj, 2018). The present study examines the associations among prenatal stress, socioeconomic disadvantage, and epigenetic aging in newborns.

We seek to examine whether biological indicators of prenatal socioeconomic disadvantage and stress are detectable at the beginning of postnatal life.

1.1. Developmental origins of behavior, health, and disease

Exposure to early life stress and socioeconomic disadvantage are robust predictors of worse health and well-being across multiple domains. Indeed, a recent meta-analysis found that prenatal stress robustly predicted emotional and behavioral problems in childhood, above and beyond postnatal levels of distress (Tung et al., 2024). Similarly, decades of research have documented that children born into poverty are prone to demonstrate lower performance on measures of cognitive and socioemotional functioning, as well as worse physical and mental health, compared to their higher-income peers (Noble et al., 2021). Experimental studies in rodent samples show that adverse exposures cause

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similar downstream effects. For example, offspring of rats experimentally exposed to stress during pregnancy demonstrate behavioral and cognitive changes much like those observed in human offspring, such as greater internalizing symptoms and declines in cognitive functioning (Weinstock, 2017).

Epigenetic modifications such as DNA methylation are one potential pathway by which these prenatal experiences may exert long-term effects. The Developmental Origins of Behavior, Health and Disease (DOBHAD) hypothesis proposed by Van Den Bergh (2011) argues that prenatal insults, such as poor nutrition, toxic stress, and environmental toxicants, can shape developing fetal systems via epigenetic pathways. For example, families residing in low-income neighborhoods tend to have reduced access to healthy foods (Vilar-Compte et al., 2021), and poor prenatal nutrition has subsequently been linked to epigenetic alterations in offspring (Koemel and Skilton, 2022). Similarly, poorer mental health and greater levels of perceived stress during pregnancy have also been linked to alterations in infant DNA methylation, possibly explained by changes to placental regulation of glucocorticoids (Champagne et al., 2024).

It is possible these effects emerge even prior to conception: experimental evidence from rodent models and correlational work in humans suggests that maternal preconception experiences, such as adverse early childhood experiences, may also impact future offspring's development via epigenetic pathways (e.g., Dye et al., 2024). However, causal evidence to support these intergenerational pathways remains scarce and highly contested in humans (see Scorza et al., 2019 for greater discussion). Importantly, these factors are interconnected and tend to co-occur within individuals: mothers experiencing social disadvantage tend to also experience greater negative life events, report higher levels of distress (Lefmann and Combs-Orme, 2014) and demonstrate increased chronic physiological stress (Ursache et al., 2017). Furthermore, cumulative lifetime stress impacts both modern-day stress appraisals and HPA axis functioning, shaping an individual's psychological and physiological experience, and ultimately, their health and wellbeing (Epel et al., 2018).

These fetal systems subsequently interact with the postnatal environment to produce individual susceptibility to disease. Importantly, though these adaptations may carry risks for overall health and behavior, they may also co-occur with improved performance in ecologically relevant domains, such as emotion detection, attention, and memory (see Ellis et al., 2020 for further discussion). The extent to which epigenetic biomarkers of stress and socioeconomic disadvantage are detectable at the beginning of postnatal life, versus later in childhood, may provide insight regarding the extent to which prenatal versus postnatal experiences shape epigenetic variation.

1.2. Epigenetic predictors of biological aging in childhood

Biological age reflects an individual's age at a cellular level, and this exhibits individual variation across the population. Recent analytic approaches have leveraged variation in genome-wide DNA methylation to predict chronological age and other age-related outcomes as a measure of biological age via epigenetic "clocks." An accelerated epigenetic age – a more advanced epigenetic age relative to one's chronological age – is a robust predictor of deleterious health and disease in adulthood (Oblak et al., 2021). Accumulating evidence suggests that an accelerated epigenetic age reflects a physiological weathering on the body as a consequence of adverse experiences (Horvath and Raj, 2018). For example, numerous studies report associations between adverse experiences in childhood – such as trauma and domestic violence (Dye et al., 2024), sexual abuse (Lawn et al., 2018), and poverty (McCrory et al., 2022) – and accelerated aging in adulthood. Importantly, these findings are inconsistent across correlational studies and rely on retrospective reporting, highlighting the need for prospective designs to improve temporal inference.

Emerging evidence suggests that the association between adverse

experiences and epigenetic age may be detectable in childhood. For instance, Raffington and colleagues (2021) found that greater socioeconomic disadvantage and body mass index were independently associated with accelerated aging during childhood, suggesting a link between early adverse exposures and long-term health disparities. Similarly, Del Toro et al. (2024) observed that greater racially-intrusive encounters with police and poorer self-reported health was associated with accelerated aging in racially minoritized youth. Recent research has extended these investigations even earlier in life, but with mixed findings. McGill et al. (2022a, 2022b) found that prenatal maternal anxiety was associated with accelerated aging across early childhood, but this effect was primarily seen in boys. In contrast, a multi-cohort study of nearly 4000 newborns reported no association between prenatal perceived stress and epigenetic aging (Murgatroyd et al., 2024). Differences in the types of epigenetic clocks used may partially explain these divergent findings.

1.3. Application of epigenetic clocks during infancy

A remaining question for the field is identifying *when* these disparities in epigenetic aging are detectable in development. Identifying the emergence of epigenetic signatures that are robustly associated with both adverse exposures and poor outcomes in adults may improve the ability to identify those at risk of developing adversity-related health problems. Furthermore, it may provide insight in how early experience shapes later life outcomes via the biological embedding of disadvantage. However, applying adult-based epigenetic clocks to pediatric samples introduces several methodological challenges.

First, it is unclear if the expected directionality of these effects will persist in very young infants. For example, adult research has observed a consistent pattern of *accelerated* epigenetic aging following exposure to trauma (Boks et al., 2015), childhood adversity (Sumner et al., 2019), childhood economic disadvantage (Hughes et al., 2018), and cumulative life stress (Zannas et al., 2015). In contrast, results seem less consistent in infant samples: although some studies reported associations between infant accelerated aging and prenatal exposures such as psychological distress and socioeconomic disadvantage (e.g., Euclides et al., 2022; Javed et al., 2016; McGill et al., 2022a, 2022b), other studies found that the same exposures predicted *decelerated* aging (Appleton et al., 2022; Folger et al., 2024; Katrinli et al., 2023; Laubach et al., 2024; McKenna et al., 2021; Pilkay et al., 2024; Suarez et al., 2018). Still, other studies reported null associations (Khouja et al., 2018; McKenna et al., 2021; Murgatroyd et al., 2024; Popovic et al., 2021). Additional research is needed to clarify the relationship between adverse prenatal exposures and infant epigenetic aging.

In addition, verifying the validity of these clocks to infant samples remains contested. For example, there is a relatively small correlation between epigenetic age and chronological age during early childhood (e.g., Bozack et al., 2023; dedeSteiguer et al., 2023; Laubach et al., 2024; Simpkin et al., 2017), which limits the ability of researchers to confirm the accurate measurement of epigenetic age in this population. Pediatric-specific clocks have since been created to increase accuracy (e.g., PedBE; McEwen et al., 2020), but the extent to which these pediatric-specific clocks are accurate in newborns is less clear due to limited research in this age range.

1.4. The present study

The present study examines the extent to which prenatal maternal stress and prenatal family socioeconomic disadvantage are associated with epigenetic aging in newborn infants. Hypotheses and analyses were preregistered at Open Science Framework (OSF; <https://osf.io/5e4kc>). Due to previous correlational work in children and adults, we hypothesized higher levels of maternal stress and lower socioeconomic status during pregnancy would be associated with accelerated epigenetic age among 1-month old infants. Given the nascent state of the literature in

newborn populations, we explored these associations using multiple measures of epigenetic age. This allowed us to examine the convergent validity of epigenetic aging measures in infants, in addition to exploring how these measures relate to important indicators of child environmental experiences. We pre-registered the Horvath and PedBE clocks as our primary outcomes of interest due to the extant literature using first-generation clocks and our use of a pediatric sample. We also report non-preregistered analyses examining associations with PhenoAge, GrimAge, and DunedinPACE as secondary outcomes, given more recent correlations reported with these constructs in second- and third-generation clocks.

2. Methods

2.1. Participants

We recruited 209 participants from New York City during their last trimester of pregnancy to participate in a prospective, longitudinal study of child development. Participants were recruited via local prenatal clinics, online advertising, flyers, and community events. To be included, mothers had to: 1) be fluent in either English or Spanish; 2) have a fetus with no known developmental disorders; 3) be at least 35 weeks pregnant with a singleton pregnancy; 4) live within a 1-hour radius of the laboratory by public transportation; and 5) be at least 18 years of age. Due to restrictions related to the COVID-19 pandemic, participants were recruited in two cohorts. Cohort 1 ($n = 93$) was recruited between June 2019 and March 2020, and Cohort 2 was recruited between August 2021 and September 2022 ($n = 116$).

2.2. Procedure

Upon providing informed consent, eligible participants completed a prenatal visit during which they completed surveys and provided a hair sample. After childbirth, participants were invited to complete a second visit at approximately 1-month postpartum, at which time a saliva sample was collected from their infant to assess epigenetic age. At this point, 3 mothers withdrew from the study and 1 was deemed ineligible due to birth complications. All study procedures were approved by the Institutional Review Board of [blinded for review] and were aligned with the ethical standards put forth by the Declaration of Helsinki.

2.3. Measures

2.3.1. Perceived stress component

We utilized principal component analysis (PCA) to reduce two measures of stress into a single “perceived stress” component (analyses examining each measure separately are also reported in the Supplement). These measures are described below. The measures loaded onto a single component with an eigenvalue of 1.53 and factor loadings of 0.71. This component explained 77 % of the variance and was extracted for use in analysis ($n = 201$).

Perceived Stress Scale (Cohen and Williamson, 1988). The PSS is a 10-item scale assessing the degree to which participants have perceived situations as stressful within the last month. Participants responded to each item using a 5-point Likert scale, ranging from 1 (*never*) to 5 (*very often*). Four items were positively stated and thus reverse scored before summing the items together. Participants needed to respond to at least 80 % of items to receive a valid score. Mean imputation was used to account for missingness on individual items.

Perceived Impact of Negative Life Events (Sarason et al., 1978). Participants were presented with 44 life events and asked if any had occurred within the past year. For each event that participants endorsed as occurring, participants then rated the perceived impact of the event on their life on a 7-point Likert scale, ranging from -3 (extremely negative) to $+3$ (extremely positive). The absolute value was calculated for events endorsed as negative and these values were summed together

to generate a Perceived Impact of Negative Life Events score.

2.3.2. Physiological stress

Hair cortisol concentration is a measure of cumulative cortisol secretion over an extended period and is traditionally considered a measure of chronic physiological stress. One centimeter of hair is associated with approximately 1 month of cortisol output, with the 3 cm closest to the root used to index cortisol output over the last three months (i.e., about the last trimester of pregnancy in this sample; Meyer and Novak, 2021). Upon providing informed consent, a trained research assistant cut a 3 cm segment of hair from the posterior vertex of the mother’s scalp ($n = 161$). Samples were frozen at -80°C then sent to either University of Massachusetts-Amherst or Yale Child Study Center for analysis. At this stage, 39 samples were excluded from processing due to too small of a sample being retained. The remaining samples were analyzed using previously described methods (see Meyer et al., 2014). Briefly, samples were weighed, washed twice with isopropanol and allowed to air dry, ground to a powder, and hormone was extracted with methanol. The methanol extract was evaporated, redissolved in an assay buffer, and samples were analyzed by an enzyme-linked immunosorbent assay according to standard quality controls (DetectX® Cortisol enzyme immunoassay kit #K003-H1W/H5W, Arbor Assays, Ann Arbor, MI). Values were set to missing if the participant reported using a corticosteroid (e.g., cream, medication) or applying one to another person within the last 3 months ($n = 7$). Values were also set to missing if they were deemed biologically implausible in line with previous research (≤ 0 or ≥ 750 pg/mg; $n = 2$; Magnuson et al., 2024) or greater than three standard deviations (SDs) from the mean ($n = 1$). To address the skew in the raw data and normalize the distribution, a total of 112 hair cortisol values were log-transformed ($\ln$) for linear analysis.

Due to cultural differences in values surrounding touching/cutting hair and challenges obtaining a large enough hair sample for analysis, we observed high levels of missingness on hair cortisol concentration values in the analytic sample ($n = 47$ missing). This missingness was not completely random according to Little’s MCAR test ($X^2(17) = 29.97$, $p = 0.03$). Mothers who identified as Black ($\beta = 0.39$, $\text{SE} = 0.09$, $p < 0.001$) were more likely to have a missing hair cortisol value. To adjust for this in analysis, we include auxiliary variables for maternal race in imputation models and utilize chained equations to allow for more flexible imputation estimation. For transparency, we report non-imputed results in the Supplement, which align with the imputed results reported below.

2.3.3. Socioeconomic status

Income-to-Needs. Participants reported their annual household income and the number of adults and dependent children living in their household at the prenatal visit. These data were used to calculate an income-to-needs ratio, which reflects a household’s economic well-being relative to the federal poverty line. For example, a family income-to-needs ratio of 2 means that a family has an annual income of 200 % or twice the federal poverty threshold. Due to a large right skew, values greater than 3 SDs from the mean were winsorized to match the next highest value within 3 SD of the mean ($n = 3$). We added \$1 to participants who reported zero dollars in income before log-transforming ($\ln$) values for analysis. Several participants ($n = 17$) declined to report their income at the prenatal visit.

Maternal Education. Participants reported the years of formal education that they had attained at the prenatal visit. All participants provided this information; there were no missing data.

2.3.4. Epigenetic age calculation

Sample Collection and Processing. At 1-month of age, infants were brought into the lab and a total of 185 saliva samples were collected using Oragene DNA kits. Upon confirming that the child had not eaten for at least 30 min prior to the appointment, a research assistant swabbed the inside of the infant’s mouth for 20 s to collect 1 mL of

saliva. Samples were frozen at -80°C and then shipped for processing at The University of Texas at Austin. DNA from saliva was extracted using the MagMAX™ DNA Multi-Sample Ultra 2.0 Kit by following the high-throughput automated DNA purification workflow on the KingFisher™ Flex System. DNA from samples was extracted and eluted to 90 μL . At this stage, several samples ($n = 21$) were deemed unusable due to a low concentration of DNA in the sponge ($<6 \text{ ng}/\mu\text{L}$). Retained samples underwent sodium bisulfite conversion and processed on the Beadchip using the Illumina Infinium HD Methylation Assay protocol (Moran et al., 2016; Pidsley et al., 2016). Next, DNA methylation levels were assayed using the EPIC (850 K) Infinium Methylation Beadchip Array (Illumina, Inc., San Diego, CA, USA) and stored as intensity data (IDAT) files. Beadchips were scanned with the Illumina NextSeq 550 at the Genomic Sequencing and Analysis Facility (GSAF) to assess fluorescence intensities and an average methylation value for each CpG site was obtained.

Pre-processing Pipeline. Methylation data was pre-processed using the *minifi* Bioconductor package (Aryee et al., 2014) and examined for quality control using the *ewastools* package according to Illumina quality control metrics (Just and Heiss, 2023). After ensuring each sample passed the bisulfite conversion threshold of 0.08, samples were examined for predicted sex against maternal report data and examined for genetic relatedness. At this stage, 3 samples were excluded due to a discrepancy between predicted and reported sex, and 2 samples were excluded as they were determined to be duplicate samples. Next, methylation beta values (via IDAT files) were filtered for background fluorescence using detection p-values and number of hybridizing beads at each CpG site (Heiss and Just, 2019) and dye-bias correction was implemented using *preprocessNoob* (Triche et al., 2013). Sex chromosome probes and cross-reactive probes were removed. PCA was used to extract the proportion of cell type to account for heterogeneity in epithelial and immune cells in saliva using previously established methods (Middleton et al., 2022). Ultimately, 159 samples were retained for the calculation of epigenetic age.

Epigenetic Clocks. The pre-registered outcomes of interest were the Horvath and PedBE epigenetic clocks, which were calculated using the *methclock* package (Pelegi-Sisó et al., 2021). We also chose to include the PhenoAge, GrimAge, and DunedinPACE clocks as secondary, non-preregistered outcomes of interest due to their association with relevant variables in previous studies. These were calculated using the *dnaMethyAge* package in R (Wang et al., 2023). After calculating the clocks, each value was residualized for technical artifacts (i.e., slide, array, plate) and cell type composition (i.e., proportion of leukocytes vs. epithelial cells).

Horvath Clock. The Horvath (2013) multi-tissue clock has been trained to predict chronological age using thousands of samples across the lifespan. Developed using elastic net regression, it examines 353 CpG sites to estimate an individual's epigenetic age. Though it is the most widely-researched epigenetic clock, the Horvath clock has an error rate of 3.6 years, and therefore may not be suitable for pediatric populations. In line with our preregistration, large values were winsorized to the 99th percentile ($n = 1$).

PedBE Clock. The Pediatric-Buccal-Epigenetic (PedBE; McEwen et al., 2020) clock was trained on a sample of children aged 0–20 years using buccal cells and is strongly correlated with chronological age across this age range. It utilizes 94 CpG sites to calculate epigenetic age and has demonstrated robustness with saliva samples. The PedBE clock is preferred in pediatric populations due to its low error rate of 0.35 years. In line with our preregistration, large values were winsorized to the 99th percentile ($n = 1$).

PhenoAge. The PhenoAge clock is a second-generation clock designed to use blood samples to predict phenotypic age (Levine et al., 2018), which reflects differences in health and lifespan of similarly-aged individuals. Using a set of clinical biomarkers collected from thousands of adults, the PhenoAge clock accurately estimates all-cause mortality, cognition, physical health, and lifestyle factors. We calculated a

principal components version of the clock, which has been shown to improve reliability in technical replicates (Higgins-Chen et al., 2022). Due to a large left skew, small values were winsorized to the 5th percentile ($n = 7$).

GrimAge. The GrimAge clock is a second-generation clock designed to use blood samples to predict mortality risk (Lu et al., 2019). The algorithm was developed using DNA methylation-based biomarkers of smoking pack-years and plasma proteins previously associated with stress and physiological risk factors, such as C-reactive protein. The algorithm was validated on a sample of older adults (mean age = 66) and scores reflect an individual's estimated biological age. While a robust predictor of lifespan, little research has applied this clock to pediatric populations. We calculated a principal components version of the clock, which is shown to improve reliability in technical replicates (Higgins-Chen et al., 2022). No adjustments were made to the GrimAge distribution.

DunedinPACE. The DunedinPACE is a third-generation clock designed to predict the rate of biological aging (Belsky et al., 2022). Developed from blood samples collected over two decades from the Dunedin Birth Cohort, DunedinPACE was trained to predict decline in organ system integrity using 19 clinical indicators, including biomarkers from cardiovascular, metabolic, and immune systems. The clock is scaled such that a value of 1 reflects one year of biological aging for each chronological year. The validation sample consists entirely of adults, though subsequent studies have applied this clock to pediatric populations. No adjustments were made to the DunedinPACE distribution.

2.4. Covariates

We preregistered a set of demographic variables to examine as potential confounders. Variables that were associated with either the PedBE or Horvath clock with a Pearson's correlation coefficient ≥ 0.10 were retained in linear analyses to maximize degrees of freedom. Ultimately, the demographic covariates retained for analyses included: maternal race and ethnicity, gestational age at birth, whether the infant was exclusively breastfed or not, and maternal report of alcohol consumption during pregnancy. In analyses where hair cortisol was examined, we also included a covariate for the mother's week of gestation at the time the hair sample was collected, and a categorical variable reflecting the lab in which the hair sample was processed; these covariates were not listed in the preregistration plan. Greater detail on the covariates examined are reported in the preregistration document (<https://osf.io/5e4kc>).

2.5. Analytic plan

Our pre-registered analytic plan utilizes linear regression with robust standard errors to estimate the associations among prenatal stress, prenatal SES, and infant epigenetic aging. We deviate from our preregistration plan in several ways. First, we pre-registered the cell type-adjusted epigenetic clock calculated by the *methclock* package as our dependent variable (i.e., AgeAccel3). Upon further investigation, we observed that this package did not fully adjust for cell composition in our data. To correct for this, we manually calculated the cell-adjusted residuals and discarded the package-generated variables. At this stage, we opted to further adjust for batch effects (i.e., slide, array, and plate) using partial correlations, which allowed us to interpret the associations between our variables of interest without further adjustment for technical characteristics. Beyond these adjustments, we follow our preregistration plan's approach of utilizing regression to adjust for demographic characteristics.

The first set of models estimated the association between each independent variable of interest and epigenetic aging, residualized for cell composition and technical artifacts (Model 1). Next, we covary for child chronological age (Model 2) and demographic characteristics (Model 3). Finally, in line with our preregistration, we conducted a multiple

regression analysis to examine the association between all predictors and epigenetic aging (Model 4). Perceived stress, hair cortisol concentration, income-to-needs, and maternal education were entered simultaneously as predictors of epigenetic aging, while adjusting for child age and demographic covariates. The perceived stress component, maternal education, and epigenetic age variables were standardized for linear analyses, while income-to-needs and hair cortisol concentrations were log-transformed (ln).

Analyses were restricted to those infants with a valid epigenetic age estimate ($n = 159$). Multiple imputation with chained equations (ICE) with 25 iterations was used to account for missing data on independent variables and covariates. Non-imputed estimates are reported in the Supplement and align with those reported below. Power analyses (calculated using G*Power) suggested the current sample is 80 % powered to detect an effect size of 0.22. We processed the DNA methylation data and calculated epigenetic age in RStudio. Subsequent analyses were conducted in Stata v.18.

3. Results

3.1. Descriptive statistics

Descriptive statistics for the analytic sample are reported in Table 1. At birth, infants were of normal weight ($M = 7.4$ lbs, $SD = 1.0$) and full term ($M = 39.4$ weeks, $SD = 1.0$). Approximately 43 % of the infants were male. Mothers identified as Asian (8 %), Black (24 %), Other/Mixed/Unknown Race (30 %), and White (38 %). About half of the sample (44 %) identified as Hispanic.

On average, mothers were 32.4 years old ($SD = 5.7$), attained 15.3

years of formal education ($SD = 3.5$), and reported an income-to-needs ratio of 6.8 ($SD = 9.3$). Mothers' perceived stress levels as assessed by the PSS ($M = 13.3$, $SD = 7.0$) aligned with normative scores identified for adult females ($M = 13.7$, $SD = 6.6$; Cohen and Williamson, 1988). Similarly, hair cortisol values for the sample (pg/mg; $M = 12.7$; $SD = 19.5$) fell within the expected values for pregnant women in the third trimester (Marceau et al., 2020).

The average epigenetic age of the sample according to the pan-tissue Horvath clock was 17.2 months ($SD = 3.7$), while the epithelial-based PedBE clock estimated 3.4 months ($SD = 2.5$). In contrast, the blood-based PhenoAge ($M = 47.8$ yrs, $SD = 2.4$) and GrimAge ($M = 42.6$ yrs, $SD = 1.6$) clocks provided epigenetic ages that extremely overestimated the chronological age of the sample, which was an average of 1.4 months ($SD = 0.6$; range 0–3 months). This divergence is likely due to differences in the tissues used to generate these clocks and those examined by the present study. Indeed, PCA analysis of cell composition revealed that samples consisted largely of epithelial cells (98 %), which aligns with other infant research (Kobor, 2024).

3.2. Correlations among key variables

Supplemental Table 1 reports the bivariate correlations among the key variables of interest. Lower family income-to-needs, but not maternal education, was weakly correlated with higher perceived stress ($r = -0.17$, $p = 0.04$). Notably, hair cortisol was not significantly associated with any of the predictors, including perceived stress.

Although each epigenetic age measure overestimated the mean age of the infants in our sample, all the measures were significantly correlated with infant chronological age, though with varying magnitudes. The strongest correlations were detected for the PedBE and PhenoAge clocks (r 's range 0.42 – 0.47, p 's < 0.001). The Horvath, DunedinPACE, and GrimAge clock each demonstrated a weak association with chronological age (r 's range 0.16 – 0.17, p 's = 0.04). This result with the Horvath clock aligns with a previous study in newborns (Bozack et al., 2023), while the DunedinPACE result aligns with work conducted in both adults (Raffington et al., 2023) and older children (dedeSteiguer et al., 2023).

Interestingly, we observed few significant associations among the epigenetic clocks themselves, possibly due to the few overlapping CpG's across clocks. As clocks trained to predict chronological age, PedBE and Horvath were moderately correlated with each other ($r = 0.50$, $p < 0.001$). The only other clock to significantly correlate with every other clock was PhenoAge (r 's range 0.20 – 0.35, p 's < 0.05).

3.3. Linear regression results

3.3.1. Perceived stress and epigenetic aging

Table 2 reports the coefficients from linear regression models, and Fig. 1 presents these fully adjusted coefficients in a Forest plot. Non-imputed estimates are reported in Supplemental Table 2. Overall, we observed little evidence of a robust association between the perceived stress component and epigenetic aging. Effect sizes across models ranged from -0.07 – 0.15 , with the majority indicating that greater perceived stress was associated with accelerated aging. Only 1 of the 6 pre-registered models emerged as statistically significant: greater perceived stress was associated with an accelerated Horvath age only when adjusting for demographic characteristics ($\beta = 0.15$, $SE = 0.07$, $p = 0.03$). One of the 9 models examined of the non-preregistered clocks demonstrated a similarly sized magnitude: higher perceived stress was associated with an accelerated GrimAge when adjusting for chronological age ($\beta = 0.15$, $SE = 0.07$, $p = 0.05$), but this did not maintain significance when adjusting for demographics. Analyses examining each measure of the perceived stress component separately are reported in Supplemental Table 3.

Table 1
Descriptive statistics of analytic sample.

	N	Mean	SD	Min	Max
<i>Maternal Characteristics</i>					
Age	159	32.37	5.69	19	45
Income-to-Needs Ratio	146	6.82	9.23	0	44
(winsorized)					
Years of formal education	159	15.26	3.46	6	22
Perceived Stress Scale	158	13.25	6.98	0	33
Total					
Perceived Impact of	154	5.22	6.74	0	46
Negative Life Events					
Score Total					
Hair Cortisol	112	12.69	19.46	0.10	160
Concentration					
(pg/mg; winsorized)					
<i>Birth Characteristics</i>					
Birth Weight (in grams)	159	3348.99	464.79	2268	4508
Gestational age at birth	158	39.41	1.03	37	42
(in weeks)					
Infant Chronological Age	159	1.37	0.58	0	3
(in months)					
Infant is Male	68	43 %			
<i>Maternal Ethnicity & Race</i>					
Hispanic	70	44 %			
Asian	13	8 %			
Black	38	24 %			
Other/Mixed/Unknown	47	30 %			
Race					
White	61	38 %			
<i>Epigenetic Age Estimates</i>					
Horvath Clock (in months;	159	17.23	3.67	10	31
winsorized)					
PedBE Clock (in months;	159	3.43	2.52	1	22
winsorized)					
PhenoAge (in years;	159	47.84	2.44	43	54
winsorized)					
GrimAge (in years)	159	42.59	1.60	34	46
DunedinPACE (rate of aging	159	1.34	0.07	1	2
per year)					
Observations	159				

Table 2

Linear association between biological clocks and outcomes of interest.

	First Generation Clocks (Primary Outcome)		Second & Third Generation Clocks (Secondary Outcome)		
	(1) Horvath	(2) PedBE	(3) PhenoAge	(4) GrimAge	(5) DunedinPACE
Perceived Stress Component					
1. Partial Correlation	0.11 (0.06)	−0.06 (0.07)	0.09 (0.06)	0.15 (0.08)	0.09 (0.08)
2. Adjusts for Age	0.11 (0.06)	−0.07 (0.07)	0.08 (0.06)	0.15* (0.07)	0.08 (0.08)
3. Adjusts for Demographics	0.15* (0.07)	−0.00 (0.07)	0.02 (0.06)	0.12 (0.08)	0.13 (0.09)
Hair Cortisol Concentration					
1. Partial Correlation	0.02 (0.09)	0.14 (0.08)	0.17* (0.09)	−0.04 (0.10)	0.19* (0.09)
2. Adjusts for Age ^a	−0.01 (0.11)	0.03 (0.09)	0.04 (0.09)	−0.10 (0.12)	0.17 (0.11)
3. Adjusts for Demographics ^a	−0.04 (0.11)	−0.01 (0.09)	0.02 (0.09)	−0.12 (0.12)	0.19 (0.12)
Income-to-Needs					
1. Partial Correlation	−0.01 (0.03)	−0.04 (0.03)	−0.03 (0.02)	−0.03 (0.02)	−0.03 (0.02)
2. Adjusts for Age	−0.01 (0.03)	−0.05* (0.02)	−0.04* (0.02)	−0.03 (0.02)	−0.03 (0.02)
3. Adjusts for Demographics	−0.02 (0.04)	−0.06* (0.03)	−0.03 (0.02)	−0.02 (0.03)	−0.04 (0.03)
Maternal Education					
1. Partial Correlation	0.02 (0.08)	−0.07 (0.08)	−0.17* (0.08)	−0.15 (0.09)	−0.10 (0.07)
2. Adjusts for Age	0.02 (0.08)	−0.05 (0.08)	−0.15* (0.07)	−0.14 (0.09)	−0.09 (0.07)
3. Adjusts for Demographics	0.06 (0.10)	−0.06 (0.09)	−0.07 (0.09)	−0.12 (0.12)	−0.17 (0.12)
Observations	159				

Note. * $p < 0.05$. Model 1 reflects partial correlations between the outcome of interest and each biological clock, residualized for technical artifacts (slide, array, and plate) and cell composition. Model 2 adjusts for infant chronological age at the time of sample collection. Model 3 adjusts for the following demographic characteristics: gestational age at birth, maternal race and ethnicity, whether the infant was exclusively breastfed, and maternal report of alcohol consumption during pregnancy. Coefficients reflect standardized betas. Robust standard errors are reported in parentheses. ^a Hair cortisol estimates in Models 2 and 3 further adjusts for week of pregnancy when hair sample was collected from the parent and the lab that processed each hair sample.

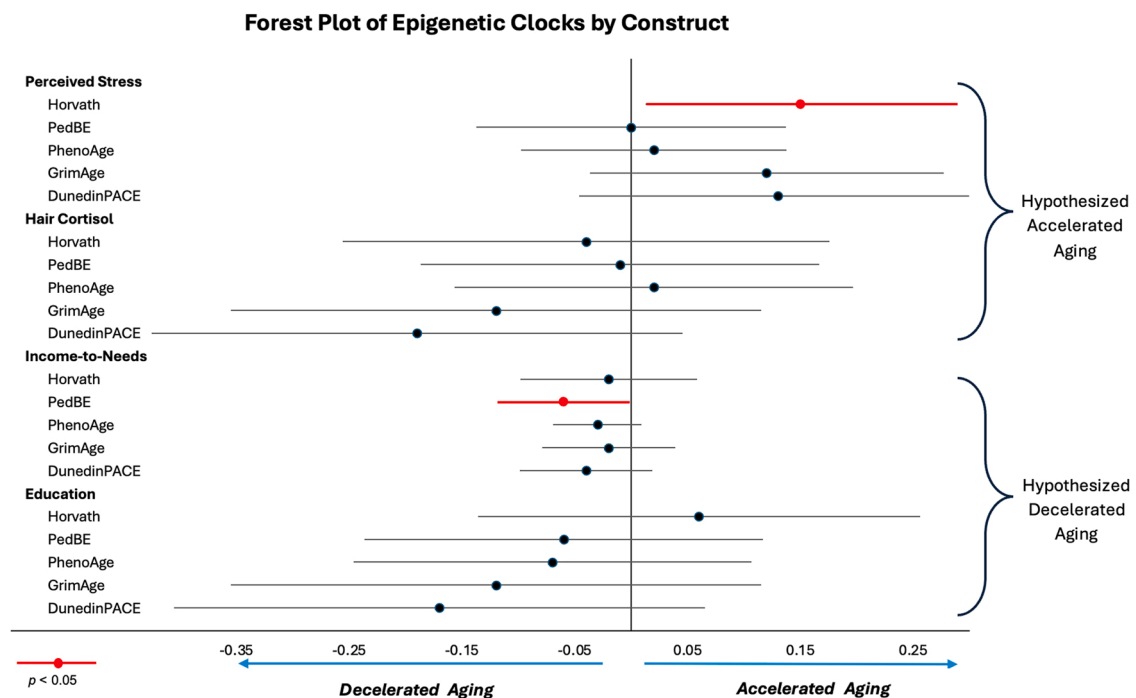


Fig. 1. Note. Significant effects indicated by red line ($p < 0.05$). Effect sizes reflect the association between each construct and the epigenetic clock of interest, adjusted for technical factors, chronological age, and demographic covariates (i.e., Model 3 in Table 3).

3.3.2. Hair cortisol and epigenetic aging

We observed little evidence of an association between maternal prenatal hair cortisol concentration and infant epigenetic aging. The magnitude and directionality of estimates varied widely across models (β 's range -0.12 – 0.19), and none of the pre-registered outcomes were statistically significant. Of the non-preregistered clocks, 2 of the 9 models examined suggested that greater hair cortisol concentration was associated with an accelerated PhenoAge ($\beta = 0.17$, $SE = 0.09$, $p = 0.05$) and DunedinPACE ($\beta = 0.19$, $SE = 0.09$, $p = 0.04$) in Model 1. While the PhenoAge coefficient dropped by more than half when adjusting for chronological age in Model 2, the DunedinPACE coefficient maintained trend-level significance when adjusting for demographics in Model 3 ($\beta = 0.19$, $SE = 0.12$, $p = 0.10$).

3.3.3. Socioeconomic status and epigenetic aging

We did not observe robust evidence of an association between prenatal income-to-needs and epigenetic aging. Across all models, coefficients were small in magnitude but negative in directionality (β 's range -0.06 to -0.01), suggesting that lower income-to-needs was associated with accelerated aging. Out of the 6 pre-registered models, two emerged as statistically significant: lower income-to-needs was associated with accelerated PedBE age when adjusting for chronological age ($\beta = -0.05$, $SE = 0.02$, $p = 0.05$) and demographics ($\beta = -0.06$, $SE = 0.03$, $p = 0.03$). Among the non-preregistered clocks, 1 out of the 9 models was statistically significant such that lower income-to-needs was associated with accelerated epigenetic aging as measured by the PhenoAge clock ($\beta = -0.04$, $SE = 0.02$, $p = 0.02$); however, this effect became statistically nonsignificant with adjustment for demographic characteristics.

Similarly, few models demonstrated significant associations between epigenetic age and maternal education. Effect sizes ranged from -0.17 – 0.06 , with the majority of models indicating that lower maternal education was associated with accelerated epigenetic aging. None of the pre-registered clocks were significantly associated with maternal education across the 6 models tested. Of the non-preregistered clocks, 2 of the 9 models examined emerged as statistically significant or marginally significant: lower maternal education was associated with accelerated PhenoAge in Model 1 ($\beta = -0.17$, $SE = 0.08$, $p = 0.04$) and Model 2 ($\beta = -0.15$, $SE = 0.07$, $p = 0.05$), but this effect was nonsignificant after adjusting for demographic covariates (Model 3). Models examining maternal education using a binary indicator of college degree attainment are reported in [Supplemental Table 4](#).

3.4. Multiple regression results

[Table 3](#) presents multiple regression results, and [Supplemental Table 5](#) reports non-imputed estimates. When all predictors were entered into the model, greater perceived stress continued to predict

trend-level accelerated Horvath aging at a similar magnitude as OLS models above ($\beta = 0.14$, $SE = 0.08$, $p = 0.08$). Additionally, lower income-to-needs continued to predict an accelerated PedBE age ($\beta = -0.06$, $SE = 0.03$, $p = 0.04$). However, none of the other predictors maintained statistical significance for any other clock.

4. Discussion

The present study examined the associations among prenatal stress, socioeconomic status, and epigenetic aging in 1-month-old infants. Overall, we find little evidence of consistent associations among stress and socioeconomic circumstances during pregnancy with epigenetic aging in newborns. These findings suggest that variation in epigenetic age in early infancy does not clearly reflect prenatal disparities in socioeconomic circumstance and stress. The magnitude and directionality of some of the detected effects align with those reported in other studies, but we likely lack the power to detect smaller effects that could have been present. Future research with larger samples, longitudinal data, and meta-analytic work is necessary to parse apart these associations.

4.1. Perceived and physiological stress

The participants included in this study exhibited generally low levels of stress, with PSS values in the low-to-moderate range. Nonetheless, we observed that higher perceived stress during pregnancy was significantly associated with accelerated aging according to the Horvath clock, but only when controlling for demographic characteristics. Although this finding aligns with our initial hypothesis, we did not detect an association with the PedBE clock, which was designed for pediatric samples. In contrast to the Horvath clock, the PedBE clock provided a much-improved estimated sample age and demonstrated a notably higher correlation with chronological age. Furthermore, previous work with a large, multi-cohort study reported a significant association between prenatal anxiety and accelerated aging in the first few months of life with the PedBE clock, but not the Horvath clock ([McGill et al., 2022a, 2022b](#)). These discordant findings, in addition to the poor estimate of chronological age, raises questions as to the robustness of this detected effect with the Horvath clock. Nonetheless, it is important to situate these findings within the broader construct of epigenetic aging: the detected effect of perceived stress on the Horvath clock aligns with those reported for the non-preregistered GrimAge and DunedinPACE clocks (though these later estimates do not reach statistical significance). Additional research with larger samples is necessary to build greater confidence in these results, as well as meta-analytic work to clarify the consistency of results across studies.

We found no consistent evidence of an association between maternal hair cortisol concentration during pregnancy and epigenetic aging in newborns. Estimates varied widely across different epigenetic clocks

Table 3
Multiple regression coefficients.

	(1) Horvath	(2) PedBE	(3) PhenoAge	(4) GrimAge	(5) DunedinPACE
Perceived Stress Component	0.14 (0.08)	−0.03 (0.08)	−0.00 (0.06)	0.12 (0.08)	0.12 (0.09)
Hair Cortisol Concentration ^a	−0.04 (0.11)	−0.01 (0.09)	0.02 (0.09)	−0.12 (0.12)	0.19 (0.12)
Income-to-Needs	−0.02 (0.05)	−0.06* (0.03)	−0.02 (0.02)	−0.01 (0.03)	−0.02 (0.03)
Maternal Education	0.10 (0.12)	0.03 (0.11)	−0.03 (0.10)	−0.11 (0.12)	−0.14 (0.13)
Observations	159				

Note. * $p < 0.05$. All predictors were simultaneously entered into the model to predict biological age. Epigenetic clock variables were residualized for cell type composition and technical artifacts (slide, array, and plate). Each model adjusts for infant chronological age at the time of sample collection and the following demographic characteristics: gestational age at birth, maternal race and ethnicity, whether the infant was exclusively breastfed, and maternal report of alcohol consumption during pregnancy. Coefficients reflect standard betas. Robust standard errors are reported in parentheses. ^a Hair cortisol estimates further adjust for week of pregnancy when hair sample was collected from the parent and the lab that processed each hair sample.

and model specifications. To our knowledge, this is the first study to examine these associations using this particular indicator of prenatal cortisol exposure. Previous work using a DNA methylation-based biomarker of glucocorticoid exposure finds that greater exposure to these stress hormones is associated with *decelerated* epigenetic aging in infants (Appleton, 2025; Euclydes et al., 2022; McGill et al., 2022a, 2022b). These differential findings may be due to differences in adjustment for cell-type compositions, as immune cells are particularly sensitive to glucocorticoids.

Accumulating evidence finds that perceived and physiological stress operate via different mechanisms to impose their effects, potentially explaining the diverging effects observed here. Indeed, our study finds no correlation between perceived stress and hair cortisol, which aligns with the results from several meta-analyses (Kim et al., 2020; Stalder et al., 2017). An important area for future research is identifying the pathways by which perceived stress may exert adverse effects on the developing fetus if not via glucocorticoids. Some evidence suggests that the interactive effects of cortisol with other adrenal hormones like dehydroepiandrosterone (DHEA) may be an improved indicator of stress-related outcomes. Indeed, these hormones impart opposing regulatory functions on the stress-response system, and emerging evidence suggests an imbalance in the ratio of these hormones is associated with developmental psychopathology (Kamin and Kertes, 2017).

Alternatively, the placenta may serve an essential role in explaining the molecular link between perceived stress and epigenetic alterations as observed in the present study (Champagne et al., 2024). Previous work found that perceived stress, but not cortisol, was associated with altered placental DNA methylation in genes responsible for glucocorticoid regulation (Monk et al., 2016), though the application of this site-specific work to epigenetic clocks is unclear. Importantly, little is known about the effects of prenatal stress on epigenetic aging across the lifespan. Longitudinal work is necessary to understand whether the present findings are due to a lack of sensitivity of epigenetic clocks during infancy, or a reflection of an overall null effect of prenatal stress on epigenetic aging broadly.

4.2. Maternal education and family income

Across all the epigenetic clocks, we observed little evidence of an association between maternal education and biological aging. Notably, the association between low maternal education and accelerated aging for the DunedinPACE, PhenoAge, GrimAge, and PedBE clocks were similar in size and magnitude in fully adjusted models. For income-to-needs, one significant effect emerged such that lower income-to-needs was associated with accelerated PedBE aging. Importantly, coefficients across all epigenetic clocks and income-to-needs were similar in magnitude and directionality. This suggests there may be some true effect of socioeconomic status on epigenetic aging that we are underpowered to detect. This aligns with the DoHAD hypothesis and with previous research in older children (e.g., Raffington et al., 2021), though it will be important to examine the persistence of these associations over time.

It is possible that more robust disparities in epigenetic aging will emerge overtime with the accumulation of postnatal experiences. The weathering hypothesis posits that cumulative experience with social inequality, such as that experienced by African-Americans, results in the deterioration of physical health overtime (Geronimus, 1992). For example, persistent hardships like reduced access to nutritious foods, less responsive parent-child interactions, discrimination, and systemic racism steadily deteriorate the body overtime and ultimately impact physical well-being. The lack of a detectable effect in the present study signals that these exposures may not have left a permanent imprint at birth, leaving room for possible early intervention. Identifying the emergence of socioeconomic disparities in these biomarkers is important for understanding the biological embedding of early life experience. Overall, we interpret these results as positive: the epigenetic effects of

social disadvantage are not robustly detectable at birth.

Importantly, this study was designed to examine the effect of prenatal exposures on the developing fetus during the last trimester of pregnancy. While we cannot rule out the effect of earlier gestational or preconception experiences on offspring epigenetic aging, it's important to consider the stability of the experiences assessed here. For example, experiencing poverty during early childhood is highly correlated with adult attainment (Duncan et al., 2010). Similarly, chronic stress shapes psychological appraisals of stress and puts one at greater risk of experiencing additional stressors (see Epel et al., 2018 for greater discussion). The confounding nature of these co-occurring phenomenon makes it difficult to parse apart the impact of life course experiences from more proximal events. Quasi-experimental research with large samples is necessary to parse apart the impact of these various pre- and post-conception experiences on infant epigenetic aging.

4.3. Validity in newborn samples

To date, few studies have critically examined the validity of applying epigenetic clocks designed for adult populations to infant samples. This concern is underscored by the poor chronological age estimates of Horvath, PhenoAge, and GrimAge in the present study, and reflected in the non-significant correlations among several of the epigenetic clocks. Additionally, numerous studies find that the correlation between biological and chronological age among first-generation clocks, such as Horvath and PedBE, are weak early in childhood and strengthen over time (Bozack et al., 2023; dedeSteiguer et al., 2023; Laubach et al., 2024; Simpkin et al., 2017). Even less work has critically examined second and third-generation clocks, such as PhenoAge, GrimAge, and DunedinPACE, in pediatric samples. Furthermore, the majority of epigenetic clocks were developed using blood samples, which demonstrate low to moderate cross-tissue correlations with saliva (Apsley et al., 2025; Raffington et al., 2023). These issues highlight the need to further consider the validity of applying epigenetic clocks designed for adults at such an early moment in a child's life. Further attention should also be paid to developing saliva-specific clocks for pediatric samples during infancy.

Some of our detected estimates were aligned in directionality and magnitude with those produced by other studies, providing some positive indication for the application of these epigenetic clocks in newborn populations. For example, the association between lower maternal education and faster DunedinPACE in the present study align with previously reported correlations in adult (Effect Size [ES] = 0.15; Maunakea et al., 2024) and pediatric samples (ES = 0.18; Raffington et al., 2021). Nonetheless, additional work is necessary to validate epigenetic clocks in this age range.

4.4. Strengths and limitations

This study has several strengths, including a pre-registered analysis plan, a racially and ethnically diverse sample, and multiple measures of prenatal stress and socioeconomic status. In addition, this is one of the first studies to examine the relationship between maternal hair cortisol concentration and newborn epigenetic aging and to investigate multiple epigenetic clocks within an infant sample.

Several limitations should also be noted, including our relatively small sample size, and the use of saliva samples with epigenetic clocks that were predominantly validated using blood and other tissues. Previous research has observed low-to-moderately sized cross-tissue correlations among epigenetic estimates obtained via blood and oral-based tissues (Apsley et al., 2025; Raffington et al., 2023). Additionally, we utilized a cell-type reference panel specifically designed for pediatric saliva samples to improve cell composition estimation (Middleton et al., 2022). Nonetheless, greater research on the validity of epigenetic clocks in pediatric samples, as well as the creation of saliva-specific clocks, are necessary to improve the generalizability of this work. In addition,

future work should leverage large, longitudinal samples to generate greater confidence in the reported estimates.

4.5. Conclusion and future directions

Broadly, we find little evidence of robust associations among prenatal stress and socioeconomic status and infant epigenetic aging. On the one hand, these null findings may be due to a lack of statistical power or the poor reliability of epigenetic aging measures in infancy. It is also possible that stronger associations may emerge later in development. We plan to follow the present sample throughout the first few years of life to examine whether these associations emerge in early childhood, and to assess their association with other indicators of development. Finally, it may be that these clocks are not appropriate indicators of biological aging during infancy. The mixed findings to date using these measures warrants caution, and we urge additional validation and meta-analytic approaches to disambiguate these findings.

Author note

The analyses for this study were preregistered at Open Science Framework (<https://osf.io/5e4kc>).

CRediT authorship contribution statement

Kimberly G. Noble: Project administration, Supervision, Conceptualization, Writing – review & editing, Funding acquisition. **Jessica F. Sperber:** Visualization, Funding acquisition, Conceptualization, Writing – original draft, Investigation, Data curation, Writing – review & editing, Software, Formal analysis. **Emma R. Hart:** Conceptualization, Writing – review & editing. **Sonya V. Troller-Renfree:** Writing – review & editing, Conceptualization. **Tyler W. Watts:** Writing – review & editing, Conceptualization, Supervision. **Melissa Miller:** Writing – review & editing, Data curation, Formal analysis. **Ariel Bellatin:** Writing – review & editing, Data curation, Investigation. **Jerrold Meyer:** Methodology, Writing – review & editing. **Amanda M. Dettmer:** Methodology, Writing – review & editing. **Frances A. Champagne:** Writing – review & editing, Methodology, Resources, Supervision, Conceptualization.

Declaration of Competing Interest

The authors declare the following financial interests/personal relationships which may be considered as potential competing interests: Jessica F. Sperber reports financial support was provided by National Institute of Child Health and Human Development. Kimberly G. Noble reports financial support was provided by National Institute of Child Health and Human Development. Sonya V. Troller-Renfree reports financial support was provided by National Institute of Child Health and Human Development. Emma R. Hart reports financial support was provided by National Science Foundation. If there are other authors, they declare that they have no known competing financial interests or personal relationships that could have appeared to influence the work reported in this paper.

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Appendix A. Supporting information

Supplementary data associated with this article can be found in the online version at [doi:10.1016/j.psyneuen.2025.107575](https://doi.org/10.1016/j.psyneuen.2025.107575).

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