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Health insurance and epigenetic aging: Trends in a United States adult population

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ABSTRACT

Background: Health insurance plays an important role in reducing morbidity and mortality. Still, there is limited data examining the relationships of health insurance with biomarkers of aging that reflect morbidity and mortality risk.
Methods: We conducted a cross-sectional study of United States adults using data from the National Health and Nutrition Examination Survey (NHANES) to examine the relationships of health insurance with seven DNA methylation-based biomarkers of aging (epigenetic age): HannumAge, HorvathAge, SkinBloodAge, PhenoAge, GrimAge2, DNAm Telomere Length, and DunedinPoAm.
Results: Our analyses included 2315 participants with available health insurance and epigenetic aging data (mean [sd] age, 65.1 [9.3] years). Compared to the uninsured, having health insurance was associated with a 2.25-year lower GrimAge2 (95 %CI: -3.49, −1.02, $P = 0.001$) and a slower DunedinPoAm pace of aging ($\beta = -0.04$, 95 % CI: -0.06, −0.02, $P < 0.001$) in basic demographic-adjusted models. GrimAge2 ($\beta = -1.42$, 95 %CI: -2.75, −0.09, $P = 0.04$) and DunedinPoAm ($\beta = -0.03$, 95 %CI: -0.06, −0.01, $P = 0.02$) relationships were attenuated after additional adjustments for general health, body mass index (BMI), education, occupation, and poverty-to-income ratio. Model estimates were larger if insurance plans were more comprehensive and included dental coverage and/or single service plans. When considering categories of insurance, similar trends were observed with private insurance and public insurance plans (i.e. Medicare, Medicaid/CHIP, and other government plans), although private insurance relationships were more often statistically significant.
Conclusion: Our findings suggest that epigenetic aging measures may be useful for examining the relationship between health insurance and population health, with potential implications for policy decisions.

1. Introduction

Evidence shows that having health insurance provides broad benefits and positively impacts individuals (Woolhandler & Himmelstein, 2017). Lacking health insurance is associated with increased morbidity and mortality (Ap et al., 2009; Franks et al., 1993). This relationship has been attributed to many factors that result in decreased access to healthcare and lower quality care when systems are accessed. For instance, uninsured individuals may be less likely to receive recommended preventive services and more likely to delay care for serious symptoms due to financial concerns (Franks et al., 1993). Given this evidence, several United States (U.S.) policy efforts have aimed to reduce uninsurance to improve healthcare access. Despite progress, it is estimated that approximately 26 million Americans remain uninsured (Robin & Cha, 2022). This statistic underscores the ongoing need for research on the health impacts of insurance as a public health priority. Epigenetic clocks, also known as epigenetic age biomarkers, have emerged as DNA methylation-based biomarkers of healthspan and lifespan that are associated with several social, environmental, and lifestyle factors (Belsky et al., 2020; Hannum et al., 2013; Horvath, 2013;

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Horvath et al., 2018; Levine et al., 2018; Lu et al., 2019, 2022; Pearce et al., 2021a). Nevertheless, there is a paucity of research examining epigenetic age relationships with health insurance. Notably, changes in epigenetic age measures may precede clinical illness and mortality thus offering the opportunity of informing interventions or lifestyle changes before a person is ill or dead. Many of these interventions and changes may be more achievable with health insurance-facilitated healthcare access.

In the present study, we examine the cross-sectional relationship of having health insurance with epigenetic age in the National Health and Nutrition Examination Survey (NHANES), a nationally representative sample of people in the U.S. that includes demographic, questionnaire, and laboratory data (NHANES - National Health and Nutrition Examination Survey Homepage, 2024). Based on the evidence showing that insurance is linked to improved healthspan and lifespan (Ap et al., 2009; Franks et al., 1993; Woolhandler & Himmelstein, 2017), and using the framework in which epigenetic age acceleration (being biologically older than expected) is considered harmful while deceleration (being biologically younger than expected) is viewed as beneficial in adults, we hypothesize that having health insurance will be associated with lower epigenetic ages, while a lack of insurance will be associated with higher epigenetic ages.

2. Methods

2.1. Study population

The National Center for Health Statistics conducts NHANES to assess the health of the noninstitutionalized U.S. population (NHANES - National Health and Nutrition Examination Survey Homepage, 2024).The NHANES sample is a representative sample of the U.S. population and includes data from interviews, physical examinations, and laboratory tests. Considering that our study is focused on understanding the relationships of insurance with DNA methylation-based epigenetic age measures, we utilized data from the 1999–2000 and 2001–2002 cycles of NHANES for which epigenetic aging data was publicly available. This subsample was composed of data from 2532 adult study participants who were surveyed in 1999–2000 and 2001–2002. Participants in this sample were at least 50 years of age. However, as a measure to protect participant identities, all NHANES study participants older than 85 years were top-coded as 85 years. Since we were unable to determine these participants' true ages and there was potential for this to introduce systematic bias in the determination of epigenetic age measures, all participants $\geq$85 years (n = 130) were removed prior to analyses. Additionally, to reduce misclassification error in epigenetic age measures, we excluded participants whose DNAm-predicted sex differed from their self-reported sex (n = 56). 2315 remaining participants had overall health insurance data, although data on the categories of insurance was more sparse. 1829 participants had information on all covariates (Fig. S1). All participants provided written informed consent, and the study protocols were approved by the NCHS Research Ethics Review Board (protocol #98-12).

2.2. Health insurance

Health insurance information was collected via self-report. Participants answered the question, "Are you covered by health insurance or some other kind of health care plan? [Include health insurance obtained through employment or purchased directly as well as government programs like Medicare and Medicaid that provide medical care or help pay medical bills.]" Participant responses were coded as "yes", "no", or "missing." Participants with missing responses were not included in the analyses. Furthermore, if participants answered "yes" to having insurance, they could provide further information on their type of insurance: private, Medicare, Medicaid/Children's Health Insurance Program (CHIP), other government insurance, or single service plan. Participants were also asked, "Does the insurance you have cover any part of dental care?". Responses were again coded as "yes", "no", or "missing."

We created mutually exclusive categories of insurance for our subanalyses. Participants reporting only having private insurance were characterized as having private insurance. Participants reporting having Medicare, Medicaid/Children's Health Insurance Program (CHIP), and/or other government insurance but not private insurance were characterized as having public insurance. We also created variables to denote if private and public insurance plans additionally included dental coverage, single service plan coverage, or both.

2.3. DNA methylation and epigenetic age

We downloaded epigenetic age measures and DNA methylation-based leukocyte proportion estimates from the NHANES website (https://www.cdc.gov/nchs/nhanes/index.htm). Additional information regarding DNA methylation data analysis and processing can also be found on the NHANES website. In short, the methodology involved extracting DNA from whole blood in a subset of NHANES 1999–2000 and 2001–2002 participants $\geq$50 years of age. The Illumina EPIC BeadChip array was then used to measure genome-wide DNA methylation. Our analysis included the HannumAge, HorvathAge, SkinBloodAge, PhenoAge, GrimAge2, and DunedinPoAm epigenetic age measures as well as DNA methylation Telomere Length (DNAmTL) as they each capture different elements of the biological aging process (Belsky et al., 2020; Hannum et al., 2013; Horvath, 2013; Horvath et al., 2018; Levine et al., 2018; Lu et al., 2019, 2022; Pearce et al., 2021a).

2.4. Statistical analysis

All analyses accounted for the complex survey design of NHANES, including oversampling, survey non-response, and post-stratification, by applying the appropriate sample weights that were created by NHANES for use with the epigenetic clock sample (CDC/National Center for Health Statistics, 2025). These weights help make the study sample representative of the U.S. civilian non-institutionalized population. We used the R 'Survey' package to perform generalized linear regression models accounting for participant sample weights (Lumley et al., 2024). The associations of health insurance (dichotomous: yes vs. no, independent variable) with each epigenetic age measure (continuous, dependent variable) were evaluated using the *svyglm* R function accounting for the survey design. Model covariates/confounders were identified *a priori* (Ap et al., 2009; Franks et al., 1993; Woolhandler & Himmelstein, 2017). Our primary models adjusted for the basic demographics of chronological age in years (continuous), chronological age in years squared (continuous), sex (dichotomous: female vs. male), and self-identified ethnicity/race (categorical; Non-Hispanic White, Mexican American, Other Hispanic, Non-Hispanic Black, Other Race). Given that some study participants were missing covariates, we conducted secondary analyses with imputed missing covariates. Imputation was achieved using the *MICE* function in R (10 imputations). Estimates from each imputed dataset were pooled using the *pool* function in R (Buuren et al., 2023). Covariates in the imputed models include the aforementioned in addition to general health condition (categorical; good, fair, poor, n = 3 missing), body mass index (BMI) (continuous, n = 83 missing), poverty-to-income ratio (PIR) (continuous, n = 238 missing), smoking status (categorical; never, former, current, n = 5 missing), alcohol intake (categorical; abstainer, moderate drinker, heavy drinker, n = 110 missing), physical activity (dichotomous; moderate/vigorous activity in the last 30 days: yes vs. no, n = 1 missing), education (categorical; less than high school, high school diploma or GED, greater than high school education, n = 1 missing), and occupation (categorical; white-collar and high-skill work, white-collar and semi-routine work, blue-collar and high-skill work, blue-collar and semi-routine work, or no work, n = 130 missing).

We performed a series of sensitivity analyses including [a] models

adjusting for estimated leukocyte proportions by using epigenetic age measures from the residuals of regressions of estimated leukocyte proportions (CD8 cells, CD4 cells, NK cells, B cells, monocytes, and neutrophils) on epigenetic age to examine if associations were independent of cell proportions; [b] "working age" models only including participants younger than 65 years of age given the employer-based health insurance model in the U.S.; [c] models examining the associations of categories of insurance (e.g. private versus public with or without dental coverage and single service plans) with epigenetic age compared to no insurance to get a more nuanced understanding of our results; and [d] models including our *a priori* determined covariates minus modifiable lifestyle behaviors (e.g. alcohol intake, physical activity, smoking) considering that some of these behaviors could be mediators of the health insurance and epigenetic age relationship.

All statistical analyses were performed using R Version 4.4.1 (R Core Team, Vienna, Austria). The multiple hypothesis testing threshold for statistical significance in the primary analyses was established using a Bonferroni-adjusted p-value $<0.05/7$ ($P < 0.007$), with the number of independent tests set at 7 for the seven epigenetic clocks examined. Findings with p-values greater than the Bonferroni-adjusted threshold but <0.05 are discussed as marginal.

3. Results

3.1. Participant characteristics

Table 1 describes the characteristics of the study participants prior to the application of survey weights. 79 % of the participants had complete covariate data. Participants with complete covariates were chronologically younger with a mean (sd) age of 64.9 (9.3) years than their counterparts missing covariates with a mean (sd) age of 65.7 (9.3) years ($P = 0.11$). Complete case participants were also generally epigenetically younger with the largest difference observed in GrimAge2. Complete cases had a mean (sd) GrimAge2 of 71.3 (8.4) years compared to 72.3 (8.6) years in participants missing demographic data ($P = 0.02$). Having insurance was slightly more common in compete case participants (89 %) compared to participants missing data (86 %, $P = 0.11$). Participants were predominantly male (52 %), physically active (54 %), moderate drinkers (53 %), and in good general health (69 %). The largest groups within other categories were those with less than a high school education (41 %), semi-routine blue-collar workers (40 %), Non-Hispanic White (42 %), and never smokers (44 %). Participants had a mean (sd) PIR and BMI of 2.7 (1.6) and 28.9 (5.9) respectively. We observed strong correlations between epigenetic age and chronological age (Fig. 1). SkinBloodAge demonstrated the strongest correlation with chronological age ($r = 0.87$, Median Absolute Error [MAE] = 3.43-years, $P < 0.001$). DNAmTL was negatively correlated with chronological age ($r = -0.58$, $P < 0.001$).

Table S1 presents the characteristics of the study participants by insurance status and type prior to the application of survey weights. On average, uninsured participants were chronologically and epigenetically younger than their insured counterparts. Given the non-mutually exclusive nature of single service insurance and dental coverage in this analysis, Table S2 presents data on the number of study participants with overlapping insurance coverage in these two categories.

3.2. Relationships of any health insurance with epigenetic age

Table 2 presents the results from our demographic-adjusted and imputed covariate-adjusted models examining relationships of any health insurance with epigenetic age. In our demographic-adjusted models, having any health insurance coverage versus not having health insurance was associated with a lower GrimAge2 ($\beta = -2.25$, 95 %CI: -3.49, −1.02, $P = 0.001$) and a slower DunedinPoAm pace of aging ($\beta = -0.04$, 95 %CI: -0.06, −0.02, $P < 0.001$). We observed similar model estimates for GrimAge2 ($\beta = -2.43$, 95 %CI: -3.71, −1.14, $P <$

Table 1
Unweighted study sample characteristics.

	Full Sample	Missing Demographic Data	Complete Cases	*P*-value[a]
	n = 2315	n = 486	n = 1829	
Variables				
Aging Variables				
Age (years), mean (sd)	65.1 (9.3)	65.7 (9.3)	64.9 (9.3)	0.11
Epigenetic Age/Clocks, mean (sd)				
HannumAge *years*	66.3 (9.2)	66.8 (9.4)	66.2 (9.1)	0.17
HorvathAge *years*	66.1 (8.6)	66.2 (8.7)	66.1 (8.6)	0.85
SkinBloodAge *years*	63.6 (9.1)	64.1 (9.5)	63.5 (8.9)	0.18
PhenoAge *years*	54.9 (10.1)	55.6 (10.1)	54.7 (10.1)	0.10
GrimAge2 *years*	71.5 (8.5)	72.3 (8.6)	71.3 (8.4)	0.02
DNAm Telomere Length (TL) *kb*	6.6 (0.3)	6.6 (0.3)	6.6 (0.3)	0.87
DunedinPoAm	1.11 (0.09)	1.12 (0.10)	1.11 (0.09)	0.02
Health Insurance				
Health Insurance, n (%)				
Yes	2050 (89 %)	420 (86 %)	1630 (89 %)	0.11
No	265 (11 %)	66 (14 %)	199 (11 %)	
Mutually Exclusive Health Insurance Category, n (%)				
Private	803 (35 %)	141 (29 %)	662 (36 %)	0.01
Public[b]	602 (26 %)	148 (30 %)	454 (25 %)	0.99
Medicare	470 (20 %)	106 (22 %)	364 (20 %)	0.53
Medicaid/CHIP	88 (4 %)	30 (6 %)	58 (3 %)	0.12
Other Government	44 (2 %)	12 (2 %)	32 (2 %)	0.88
Health Insurance Additions, n (%)				
Single Service Plan	63 (3 %)	3 (1 %)	60 (3 %)	0.003
Dental Coverage	1065 (46 %)	213 (44 %)	852 (47 %)	0.91
Additional Demographic Variables				
Education, n (%)				
Less Than High School			761 (41 %)	
High School Diploma (including GED)			398 (22 %)	
More Than High School			670 (37 %)	
Occupation, n (%)				
Blue-collar (high-skill)			254 (14 %)	
Blue-collar (semi-routine)			739 (40 %)	
White-collar (high-skill)			458 (25 %)	
White-collar (semi-routine)			332 (18 %)	
No Work			46 (3 %)	
Poverty to Income Ratio, mean (sd)			2.7 (1.6)	
Race/Ethnicity Category, n (%)				
Mexican American			510 (28 %)	
Other Hispanic			104 (6 %)	
Non-Hispanic White			768 (42 %)	

(*continued on next page*)

Table 1 (*continued*)

	Full Sample	Missing Demographic Data	Complete Cases	*P*-value[a]
	n = 2315	n = 486	n = 1829	
Non-Hispanic Black			387 (21 %)	
Other Race			60 (3 %)	
Sex, n (%)				
Male			982 (52 %)	
Female			847 (48 %)	
Health Variables				
Physically Active, n (%)				
Yes			953 (54 %)	
No			876 (46 %)	
Alcohol Intake, n (%)				
Abstainer			802 (44 %)	
Moderate Drinker			964 (53 %)	
Heavy Drinker			63 (3 %)	
Body Mass Index, mean (sd)			28.9 (5.9)	
General Health Condition, n (%)				
Good			1261 (69 %)	
Fair			442 (24 %)	
Poor			126 (7 %)	
Smoking, n (%)				
Current			286 (16 %)	
Former			734 (40 %)	
Never			809 (44 %)	

[a] *P*-values from T-tests or chi square tests comparing complete cases with participants missing demographic data.

[b] Public insurance category comprised of Medicare, Medicaid, and other government insurance.

0.001) and DunedinPoAm (β = −0.04, 95 %CI: -0.07, −0.02, $P < 0.001$) when we restricted the study sample to participants with any insurance that included dental coverage. GrimAge2 and DunedinPoAm model estimates remained statistically significant and were greater in magnitude in models restricted to participants whose insurance plans included single service coverage or single service and dental coverage. After adjusting for additional health and lifestyle variables in imputed covariate models, we continued to observe trends of lower GrimAge2 and DunedinPoAm values in insured participants compared to the uninsured. Only with insurance plans that included a single service coverage (β = −0.05, 95 %CI: -0.10, −0.0001, $P = 0.0496$) or both single service and dental coverage (β = −0.06, 95 %CI: -0.11, −0.003, $P = 0.04$) was health insurance marginally associated with a lower DunedinPoAm. We also observed that participants with insurance had marginally higher SkinBloodAges in our primary analysis (β = 1.34, 95 %CI: 0.14, 2.54, $P = 0.03$) in our demographic-adjusted models. Nevertheless, no marginal or statistically significant associations with SkinBloodAge were observed in imputed covariate-adjusted models. Overall, our leukocyte-adjusted model and working age sensitivity analyses showed similar statistically significant GrimAge2, DunedinPoAm, and SkinBloodAge relationships as our primary analysis (Table S3).

Given the association of any health insurance with GrimAge2, we examined relationships with each of the GrimAge2 components (Table 3). Compared to uninsured participants, participants with insurance had significantly lower estimated cigarette packyears (β = −6.48, 95 %CI: -9.94, −3.02, $P < 0.001$) and marginally lower A1c (β = −0.01, 95 %CI: -0.02, −0.002, $P = 0.02$) and GDF15 (β = −25.01, 95 % CI: -49.44, −0.58, $P = 0.045$) levels in demographic-adjusted models. Marginal associations were also observed for ADM, CRP, cystatin C, and TIMP1 in participants with more comprehensive insurance that included dental and/or single service plan coverage. All relationships were attenuated and no longer statistically significant in the imputed covariate-adjusted models (Table 3).

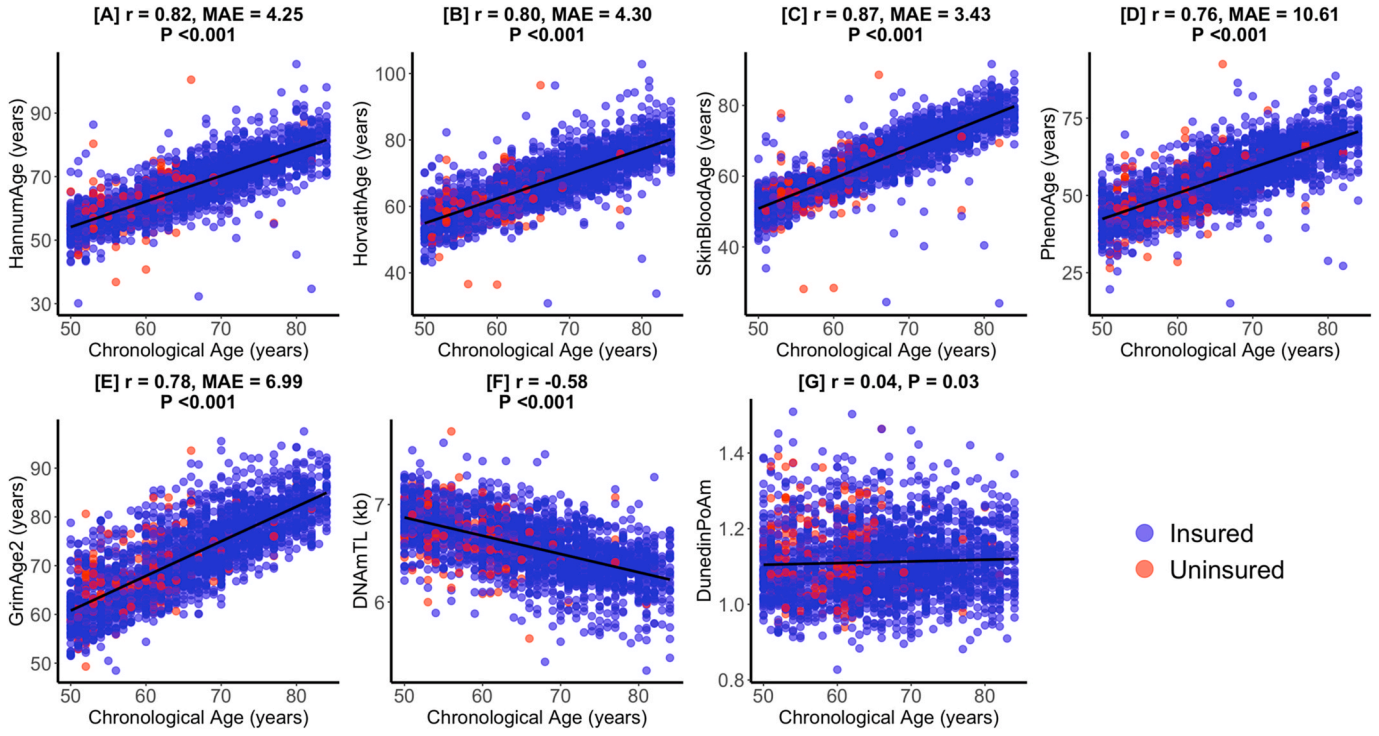

Fig. 1. Pearson Correlations (r) and Median Absolute Error (MAE) of Epigenetic Age with Chronological Age. Fig. 1 presents the chronological age and epigenetic age correlation coefficients and median absolute errors for the study sample (n = 2315) for Hannum DNAmAge [**A**], Horvath DNAmAge [**B**], SkinBloodAge [**C**], PhenoAge [**D**], GrimAge2 [**E**], DNAmTL [**F**], and DunedinPoAm [**G**]. Insured participants are shown in blue while uninsured participants are shown in red.

Table 2
Relationships of health insurance with epigenetic age in adjusted models.

Insurance	Biomarker	Demographic-Adjusted Models		Imputed Covariate-Adjusted Models		
		Estimate (95 % CI)	*P*-value	Estimate (95 % CI)	*P*-value	n
Any Insurance	HannumAge (years)	0.72 (−0.39, 1.84)	0.19	1.14 (−0.40, 2.69)	0.12	2315
Any Insurance	HorvathAge (years)	0.32 (−0.72, 1.36)	0.53	0.48 (−0.83, 1.79)	0.40	2315
Any Insurance	SkinBloodAge (years)	1.34 (0.14, 2.54)	0.03	1.45 (−0.13, 3.02)	0.07	2315
Any Insurance	PhenoAge (years)	−0.10 (−1.46, 1.26)	0.88	0.94 (−0.76, 2.63)	0.22	2315
Any Insurance	GrimAge2 (years)	−2.25 (−3.49, −1.02)	0.001	−0.23 (−1.42, 0.96)	0.65	2315
Any Insurance	DNAmTL (kb)	0.02 (−0.04, 0.07)	0.52	−0.03 (−0.09, 0.04)	0.31	2315
Any Insurance	DunedinPoAm	−0.04 (−0.06, −0.02)	<0.001	−0.01 (−0.04, 0.01)	0.28	2315
Insurance Additions						
+Dental Coverage	HannumAge (years)	0.52 (−0.63, 1.68)	0.36	0.76 (−0.94, 2.47)	0.31	1330
+Dental Coverage	HorvathAge (years)	0.07 (−1.05, 1.18)	0.90	−0.03 (−1.50, 1.45)	0.97	1330
+Dental Coverage	SkinBloodAge (years)	1.18 (−0.09, 2.45)	0.07	1.05 (−0.77, 2.87)	0.20	1330
+Dental Coverage	PhenoAge (years)	−0.25 (−1.69, 1.19)	0.72	0.68 (−1.19, 2.56)	0.40	1330
+Dental Coverage	GrimAge2 (years)	−2.43 (−3.71, −1.14)	<0.001	−0.42 (−1.73, 0.89)	0.46	1330
+Dental Coverage	DNAmTL (kb)	0.02 (−0.04, 0.07)	0.53	−0.03 (−0.09, 0.04)	0.33	1330
+Dental Coverage	DunedinPoAm	−0.04 (−0.07, −0.02)	<0.001	−0.02 (−0.04, 0.01)	0.16	1330
+Single Service Plan	HannumAge (years)	0.80 (−1.02, 2.62)	0.37	1.96 (−0.93, 4.85)	0.14	328
+Single Service Plan	HorvathAge (years)	0.64 (−0.97, 2.25)	0.42	0.85 (−1.97, 3.66)	0.47	328
+Single Service Plan	SkinBloodAge (years)	2.08 (0.40, 3.75)	0.02	3.06 (−0.15, 6.28)	0.06	328
+Single Service Plan	PhenoAge (years)	−0.93 (−2.88, 1.02)	0.33	0.69 (−3.19, 4.57)	0.66	328
+Single Service Plan	GrimAge2 (years)	−4.28 (−5.94, −2.62)	<0.001	−1.63 (−3.65, 0.40)	0.09	328
+Single Service Plan	DNAmTL (kb)	−0.02 (−0.11, 0.07)	0.62	−0.11 (−0.25, 0.04)	0.11	328
+Single Service Plan	DunedinPoAm	−0.08 (−0.11, −0.05)	<0.001	−0.05 (−0.10, −0.0001)	0.0496	328
+ Dental & SSP	HannumAge (years)	0.66 (−1.29, 2.61)	0.49	1.78 (−1.25, 4.80)	0.19	321
+ Dental & SSP	HorvathAge (years)	0.66 (−1.00, 2.31)	0.42	0.74 (−2.36, 3.85)	0.56	321
+ Dental & SSP	SkinBloodAge (years)	2.03 (0.29, 3.78)	0.02	3.16 (−0.23, 6.56)	0.06	321
+ Dental & SSP	PhenoAge (years)	−1.16 (−3.11, 0.79)	0.23	0.30 (−3.92, 4.52)	0.86	321
+ Dental & SSP	GrimAge2 (years)	−4.32 (−6.06, −2.58)	<0.001	−2.00 (−4.18, 0.18)	0.06	321
+ Dental & SSP	DNAmTL (kb)	−0.04 (−0.12, 0.05)	0.38	−0.12 (−0.27, 0.02)	0.08	321
+ Dental & SSP	DunedinPoAm	−0.07 (−0.11, −0.04)	<0.001	−0.06 (−0.11, −0.003)	0.04	321

Model estimates are for insured participants (uninsured participants are the reference group). SSP = Single Service Plan.
Demographic Model Adjustments: chronological age, chronological age^2, sex, and race/ethnicity.
Imputed Covariate Model Adjustments: chronological age, chronological age^2, sex, race/ethnicity, alcohol, BMI, education, general health, occupation, physical activity, PIR, and smoking.
$P < 0.007$: statistically significant.
$P < 0.05$: marginally significant.

3.3. Relationships of health insurance with epigenetic age stratified by private and public insurance

To better understand the associations of health insurance with epigenetic age, we focused on the biomarkers that were associated with any insurance but stratified our analysis by private and public health insurance (Table 4). In demographic adjusted models, compared to being uninsured, having private health insurance was marginally associated with a 1.32-year higher SkinBloodAge (95 %CI: 0.08, 2.55, $P = 0.04$) and significantly associated with a lower GrimAge2 ($\beta = -2.70$-years, 95 %CI: -3.95, −1.46, $P < 0.001$) and DunedinPoAm ($\beta = -0.05$, 95 %CI: -0.07, −0.02, $P < 0.001$). Similar positive trends for SkinBloodAge and negative trends for GrimAge2 and DunedinPoAm were observed for public insurance but only SkinBloodAge results reached the threshold for marginal significance. When we examined the more comprehensive insurance plans including dental and single service coverage, the model estimates were greater in magnitude, remained statistically significant for private insurance, and the DunedinPoAm results reached the threshold for statistical significance for public insurance ($\beta = -0.09$, 95 %CI: -0.14, −0.03, $P = 0.004$). The SkinBloodAge model estimates were greater in imputed covariate-adjusted models while the GrimAge2 and DunedinPoAm estimates were attenuated. Marginally significant associations were observed for SkinBloodAge with private insurance including a single service plan ($\beta = 3.48$-years, 95 %CI: 0.08, 6.88, $P = 0.047$) and for GrimAge2 with private insurance including both dental and single service coverage ($\beta = -2.56$-years, 95 %CI: -5.08, −0.03, $P = 0.048$). Similar significant trends were observed in imputed covariate-adjusted models not including adjustments for modifiable behaviors (Table S4).

In demographic-adjusted models, having private insurance coverage was associated with marginal or significantly lower levels of many GrimAge2 estimated components previously associated with having any insurance including, A1c, ADM, CRP, cystatin C, GDF15, packyears, and TIMP1 (Table S5). Public insurance with various additions was marginally associated with ADM and CRP. After adjusting for additional health and lifestyle variables, private insurance was marginally associated with have a lower estimated A1c ($\beta = -0.01$, 95 %CI: -0.02, −0.001, $P = 0.03$) while public insurance with dental coverage was marginally associated with a higher estimated ADM ($\beta = 5.90$, 95 %CI: 0.35, 11.45, $P = 0.04$). Still, there were no notable significance trends as either public or private health insurance became more comprehensive in the imputed covariate-adjusted models. A1c, CRP, packyear, and other GrimAge2 component relationships were again marginally significant in imputed covariate-adjusted models that did not include adjustments for modifiable behaviors (Table S6).

4. Discussion

In this cross-sectional study of U.S. adults aged 50–84 years participating in NHANES, we examined relationships of self-reported

Table 3
Relationships of health insurance with estimated GrimAge2 components in adjusted models.

Insurance	Biomarker	Demographic-Adjusted Model		Imputed Covariate-Adjusted Model		
		Estimate (95 % CI)	*P*-value	Estimate (95 % CI)	*P*-value	n
Any Insurance	A1c	−0.01 (−0.02, −0.002)	0.02	−0.01 (−0.01, 0.002)	0.12	2315
Any Insurance	ADM	−0.54 (−4.17, 3.09)	0.76	1.00 (−3.68, 5.68)	0.61	2315
Any Insurance	B2M	−6383.81 (−25817.41, 13049.79)	0.50	7257.52 (−17262.30, 31777.35)	0.49	2315
Any Insurance	CRP	−0.06 (−0.16, 0.03)	0.19	0.02 (−0.09, 0.13)	0.62	2315
Any Insurance	Cystatin C	−3044.97 (−8067.74, 1977.81)	0.22	−70.39 (−6303.34, 6162.56)	0.98	2315
Any Insurance	GDF15	−25.01 (−49.44, −0.58)	0.045	−3.53 (−33.87, 26.82)	0.78	2315
Any Insurance	Leptin	−103.72 (−451.63, 244.19)	0.54	−18.42 (−422.56, 385.73)	0.91	2315
Any Insurance	Packyears	−6.48 (−9.94, −3.02)	<0.001	−0.87 (−3.37, 1.62)	0.42	2315
Any Insurance	PAI1	−428.43 (−1051.92, 195.06)	0.17	−143.84 (−834.2, 546.51)	0.62	2315
Any Insurance	TIMP1	−94.89 (−216.89, 27.12)	0.12	−42.19 (−195.94, 111.57)	0.52	2315
Insurance Additions						
+Dental Coverage	A1c	−0.01 (−0.02, −0.002)	0.01	−0.01 (−0.01, 0.001)	0.09	1330
+Dental Coverage	ADM	−0.94 (−4.84, 2.96)	0.62	0.60 (−4.74, 5.94)	0.79	1330
+Dental Coverage	B2M	−6043.80 (−25977.89, 13890.29)	0.53	8523.17 (−18088.79, 35135.13)	0.46	1330
+Dental Coverage	CRP	−0.06 (−0.17, 0.04)	0.22	0.04 (−0.09, 0.17)	0.47	1330
+Dental Coverage	Cystatin C	−4447.66 (−9640.03, 744.72)	0.09	−1952.88 (−8552.11, 4646.36)	0.49	1330
+Dental Coverage	GDF15	−22.60 (−48.41, 3.21)	0.08	−1.21 (−38.65, 36.23)	0.94	1330
+Dental Coverage	Leptin	−51.32 (−416.22, 313.59)	0.77	−70.69 (−519.66, 378.28)	0.71	1330
+Dental Coverage	Packyears	−6.87 (−10.50, −3.24)	<0.001	−1.48 (−4.29, 1.33)	0.24	1330
+Dental Coverage	PAI1	−411.35 (−973.60, 150.90)	0.14	−177.96 (−805.60, 449.68)	0.51	1330
+Dental Coverage	TIMP1	−104.05 (−220.87, 12.77)	0.08	−64.93 (−218.60, 88.74)	0.33	1330
+Single Service Plan	A1c	−0.02 (−0.03, −0.01)	0.005	−0.01 (−0.03, 0.004)	0.11	328
+Single Service Plan	ADM	−5.54 (−10.41, −0.68)	0.03	−5.68 (−16.09, 4.73)	0.22	328
+Single Service Plan	B2M	−22310.60 (−55846.85, 11225.65)	0.18	−14152.22 (−64993.23, 36688.80)	0.50	328
+Single Service Plan	CRP	−0.21 (−0.40, −0.02)	0.03	−0.11 (−0.40, 0.18)	0.37	328
+Single Service Plan	Cystatin C	−6602.48 (−13218.74, 13.78)	0.05	−2792.37 (−11137.54, 5552.81)	0.42	328
+Single Service Plan	GDF15	−35.41 (−73.43, 2.62)	0.07	−16.73 (−84.18, 50.72)	0.54	328
+Single Service Plan	Leptin	−357.81 (−1003.21, 287.60)	0.26	−678.25 (−1493.98, 137.49)	0.08	328
+Single Service Plan	Packyears	−10.18 (−14.3, −6.05)	<0.001	−2.01 (−5.51, 1.50)	0.19	328
+Single Service Plan	PAI1	−939.37 (−1894.34, 15.61)	0.05	−767.00 (−1899.93, 365.93)	0.14	328
+Single Service Plan	TIMP1	−167.44 (−330.95, −3.92)	0.045	−201.40 (−508.20, 105.40)	0.15	328
+ Dental & SSP	A1c	−0.02 (−0.03, −0.01)	0.01	−0.01 (−0.03, 0.004)	0.10	321
+ Dental & SSP	ADM	−5.83 (−10.92, −0.75)	0.03	−6.46 (−17.47, 4.56)	0.19	321
+ Dental & SSP	B2M	−22675.95 (−56941.40, 11589.51)	0.18	−16675.83 (−70558.62, 37206.96)	0.46	321
+ Dental & SSP	CRP	−0.25 (−0.44, −0.05)	0.01	−0.18 (−0.46, 0.09)	0.15	321
+ Dental & SSP	Cystatin C	−7361.12 (−13873.11, −849.13)	0.03	−3521.25 (−12286.29, 5243.79)	0.34	321
+ Dental & SSP	GDF15	−33.95 (−72.13, 4.23)	0.08	−22.76 (−95.21, 49.68)	0.44	321
+ Dental & SSP	Leptin	−267.29 (−871.95, 337.37)	0.37	−573.26 (−1327.52, 181.00)	0.11	321
+ Dental & SSP	Packyears	−9.49 (−13.74, −5.24)	<0.001	−1.78 (−5.61, 2.06)	0.28	321
+ Dental & SSP	PAI1	−920.27 (−1934.75, 94.21)	0.07	−866.79 (−2101.91, 368.33)	0.13	321
+ Dental & SSP	TIMP1	−171.09 (−329.83, −12.36)	0.04	−237.21 (−552.26, 77.83)	0.11	321

Model estimates are for insured participants (uninsured participants are the reference group). SSP = Single Service Plan.
Demographic Model Adjustments: chronological age, chronological age^2, sex, and race/ethnicity.
Imputed Covariate Model Adjustments: chronological age, chronological age^2, sex, race/ethnicity, alcohol, BMI, education, general health, occupation, physical activity, PIR, and smoking.
$P < 0.007$: statistically significant.
$P < 0.05$: marginally significant.

health insurance with epigenetic aging biomarkers. In models adjusted for demographic factors, we observed that having health insurance was associated with a lower GrimAge2, a slower pace of aging in the DunedinPoAm biomarker, and a higher SkinBloodAge. Our model estimates were greater in magnitude with more comprehensive forms of insurance (i.e. including dental and/or single service plan coverage) and we observed similar trends when comparing private and public health insurance, although the associations with private insurance were more often statistically significant. Furthermore, several estimated GrimAge2 components were associated with health insurance, with cigarette packyears demonstrating the strongest association. Still, the majority of our findings were attenuated and did not meet the thresholds for marginal or statistical significance after adjusting for additional health and lifestyle factors.

When hypothesizing about how health insurance could be related to epigenetic aging measures of morbidity and mortality risk (Dhingra et al., 2018; Horvath, 2013; Levine et al., 2018; Lu et al., 2022), we considered the broader impact of insurance on overall health. Specifically, health insurance plays a pivotal role in facilitating healthcare access that is essential for both disease treatment and lifelong health maintenance (Ap et al., 2009; Woolhandler & Himmelstein, 2017). By enabling earlier interventions, consistent medical care, and better management of chronic conditions, health insurance contributes to improved long-term health outcomes that should be reflected in lower morbidity and mortality risk. Thus, we hypothesized that individuals with health insurance should be epigenetically younger than their uninsured counterparts. In line with this hypothesis, in demographic-adjusted models, we observed lower GrimAge2 and DunedinPoAm epigenetic ages in individuals with health insurance. In contrast, we also observed that individuals with insurance had higher

Table 4
Relationships of health insurance with epigenetic aging stratified by private versus public insurance.

Insurance	Biomarker	Demographic-Adjusted Models						Imputed Covariate-Adjusted Models					
		Private Insurance			Public Insurance			Private Insurance			Public Insurance		
		Estimate (95 % CI)	*P*-value	n	Estimate (95 % CI)	*P*-value	n	Estimate (95 % CI)	*P*-value	n	Estimate (95 % CI)	*P*-value	n
Main Insurance	SkinBloodAge (years)	1.32 (0.08, 2.55)	0.04	1068	1.64 (0.16, 3.11)	0.03	867	1.60 (−0.07, 3.27)	0.06	1068	1.45 (−0.32, 3.22)	0.09	867
Main Insurance	GrimAge2 (years)	−2.70 (−3.95, −1.46)	<0.001	1068	−0.18 (−1.75, 1.40)	0.82	867	−0.40 (−1.77, 0.98)	0.50	1068	0.52 (−1.07, 2.10)	0.45	867
Main Insurance	DunedinPoAm	−0.05 (−0.07, −0.02)	<0.001	1068	−0.01 (−0.04, 0.02)	0.42	867	−0.01 (−0.04, 0.02)	0.32	1068	0.001 (−0.03, 0.03)	0.94	867
Insurance Additions													
+Dental Coverage	SkinBloodAge (years)	1.13 (−0.10, 2.36)	0.07	822	2.15 (0.34, 3.96)	0.02	478	1.40 (−0.40, 3.20)	0.10	822	1.69 (−0.32, 3.71)	0.08	478
+Dental Coverage	GrimAge2 (years)	−3.09 (−4.36, −1.82)	<0.001	822	1.17 (−0.23, 2.56)	0.10	478	−0.60 (−2.16, 0.97)	0.38	822	1.12 (−0.32, 2.56)	0.10	478
+Dental Coverage	DunedinPoAm	−0.05 (−0.08, −0.03)	<0.001	822	−0.004 (−0.03, 0.03)	0.79	478	−0.02 (−0.05, 0.02)	0.24	822	−0.003 (−0.04, 0.03)	0.81	478
+Single Service Plan	SkinBloodAge (years)	2.18 (0.41, 3.94)	0.02	306	1.37 (−4.69, 7.42)	0.64	273	3.48 (0.08, 6.88)	0.047	306	4.03 (−6.47, 14.52)	0.30	273
+Single Service Plan	GrimAge2 (years)	−4.88 (−6.85, −2.91)	<0.001	306	−0.85 (−6.12, 4.42)	0.74	273	−2.33 (−4.89, 0.24)	0.06	306	0.16 (−5.98, 6.30)	0.94	273
+Single Service Plan	DunedinPoAm	−0.08 (−0.11, −0.05)	<0.001	306	−0.08 (−0.14, −0.03)	0.01	273	−0.05 (−0.11, 0.01)	0.07	306	−0.08 (−0.19, 0.03)	0.09	273
+ Dental & SSP	SkinBloodAge (years)	2.01 (0.16, 3.87)	0.03	305	1.50 (−4.52, 7.51)	0.61	271	3.32 (−0.20, 6.85)	0.06	305	4.12 (−6.46, 14.71)	0.29	271
+ Dental & SSP	GrimAge2 (years)	−4.90 (−6.91, −2.89)	<0.001	305	−0.90 (−6.46, 4.67)	0.74	271	−2.56 (−5.08, −0.03)	0.048	305	0.20 (−6.34, 6.73)	0.93	271
+ Dental & SSP	DunedinPoAm	−0.08 (−0.11, −0.04)	<0.001	305	−0.09 (−0.14, −0.03)	0.004	271	−0.05 (−0.11, 0.01)	0.07	305	−0.08 (−0.19, 0.02)	0.09	271

Model estimates are for insured participants (uninsured participants are the reference group). Public insurance includes coverage by Medicare and/or Medicaid/CHIP and/or other government insurance. SSP = Single Service Plan.
Demographic Model Adjustments: chronological age, chronological age^2, sex, and race/ethnicity.
Imputed Covariate Model Adjustments: chronological age, chronological age^2, sex, race/ethnicity, alcohol, BMI, education, general health, occupation, physical activity, PIR, and smoking.
$P < 0.007$: statistically significant.
$P < 0.05$: marginally significant.

SkinBloodAges. Some may interpret this difference in the direction of GrimAge2 and DunedinPoAm relationships versus SkinBloodAge relationships as contradictory, but we beg to differ. Second and third generation epigenetic clocks like GrimAge2 and DunedinPoAm were trained on chronological age and clinical variables and therefore may better reflect biological processes related to health (Belsky et al., 2020; Lu et al., 2022). In contrast, earlier clocks like SkinBloodAge were primarily trained to predict chronological age and therefore might not capture biologically meaningful relationships (Horvath et al., 2018). To further support this notion, in our study sample, we observe that SkinBloodAge is the clock most strongly correlated with chronological age. As such, we believe the SkinBloodAge results simply represent our observation that insured individuals are often chronologically older than their uninsured counterparts. Additionally, a prior NHANES study found no significant association between directly measured telomere length and health insurance status (Baltrus et al., 2020). Given the good correlation between DNAmTL and directly measured telomere length (Lu et al., 2019), we expected—and subsequently observed—a similar lack of association between health insurance status and DNAmTL. Although telomere length has long served as a marker of cellular aging, its measurement methods at times yield high variability and have produced conflicting results in relation to age-related pathology, particularly when compared to newer epigenetic age biomarkers (Behrens et al., 2017; Daios et al., 2022; Mather et al., 2011; Pearce et al., 2021b). Together, these findings suggest that telomere length measures—even those estimated via epigenetic methods—may not be the most sensitive indicators for exploring the relationship of health insurance with biological aging.

Compared to the other epigenetic aging biomarkers analyzed, GrimAge2 and DunedinPoAm are the newest and may be more sensitive to social factors like health insurance. As previously noted, these two measures were most extensively trained on clinical variables. This may explain why they yield the most robust health-related findings in our study. More specifically, GrimAge2 is the epigenetic aging measure that most accurately predicts mortality risk. GrimAge2 is calculated using a weighted combination of data for chronological age, sex, and DNA

methylation surrogates for smoking packyears and nine plasma proteins, including ADM, B2M, cystatin C, GDF15, leptin, CRP, hemoglobin A1C, PAI1, and TIMP1 (Lu et al., 2022). Our findings suggest that health insurance is associated with a lower risk of mortality as assessed by GrimAge2 and is in agreement with the broader health insurance mortality literature (Goldin et al., 2021). Examining the relationships of health insurance with the GrimAge2 components offers deeper insights into the associations we observed. Of all the GrimAge2 components, having a lower estimated cigarette packyears was most associated with health insurance. This observation supports our hypothesis that behavioral (e.g. smoking cessation) changes that participants may experience via health insurance-facilitated healthcare provider access may explain the beneficial association of health insurance with epigenetic aging (Agency for Healthcare Research and Quality, 2021). Similarly, health insurance-facilitated chronic disease management could explain marginal observations of lower estimated GrimAge2 components like CRP (a measure of inflammation) and A1c (a 3-month average of blood sugar levels used to diagnose and monitor diabetes in insured participants) (Mukonda et al., 2025; Yeh, 2004).

DunedinPoAm estimates an individual's rate of aging relative to others of the same chronological age (Belsky et al., 2020). DunedinPoAm is calculated using data from 18 biomarkers of organ system integrity, including A1c, apolipoprotein B100 to apolipoprotein A1 ratio, BMI, blood urea nitrogen, cardiorespiratory fitness, HDL cholesterol, total cholesterol, creatinine, CRP, respiratory function (e.g. FEV1/FVC ratio and FEV1), gum health, lipoprotein a, mean arterial pressure, telomere length, triglycerides, and white blood cell count. DunedinPoAm was developed because the length of the human lifespan often makes studying longevity-impacting interventions more onerous. Measuring the pace of aging – rather than absolute aging – theoretically allows one to assess the impact of interventions or policies quicker (Belsky et al., 2020). Our findings are in line with other evidence demonstrating that adverse social factors (e.g. food insecurity) are associated with increased rates of DunedinPoAm aging while protective social factors (e.g. upward social mobility) demonstrate decreased rates of aging (Graf et al., 2022; Ja & Y, 2024; Raffington et al., 2021; Simons et al., 2022; Y et al., 2022). Moreover, some of the estimated GrimAge2 components like A1c and CRP are also used in calculating DunedinPoAm, possibly explaining why DunedinPoAm and GrimAge2 demonstrate significant relationships with health insurance.

Although the overall impact of having insurance versus being insured was the primary focus of this manuscript, our modeling framework also allowed us to examine the impact of more comprehensive forms of insurance. Knowing that dental insurance is linked to better oral health (Borrell et al., 2023), and that previous research has shown that poor oral health is associated with accelerated epigenetic aging (Chen et al., 2024), we hypothesized that more comprehensive forms of insurance (including dental coverage) may further reduced health risks reflected by epigenetic aging. In general, lower epigenetic ages were observed in individuals with insurance that included dental and/or single service plan coverage. Single service plans are insurance plans that only cover one type of service (e.g. vision care, prescriptions, rehabilitation care). They can be added on to existing insurance plans to help a plan better meet an individual's needs. Our results could suggest that more comprehensive insurance plans are more beneficial to the health of the insured, but these findings could also be driven by the higher socioeconomic status of individuals with single service plans. For instance, individuals with single service plans in our study sample had the highest incomes and were most likely to hold white-collar high-skill occupations. Nevertheless, we continue to observe marginal associations of health insurance with lower epigenetic age after adjusting for these and other health and lifestyle variables in imputed covariate-adjusted models, suggesting that the association is not completely driven by these factors. Importantly, we have no information regarding what single service each plan is covering for the study participants. Moreover, the sample size for persons holding single service plan insurance was the smallest among all insurance categories giving additional impetus for having caution when making generalized conclusions from these results. Still, it is worth discussing the concept of more comprehensive insurance being more beneficial. While approximately 8 % of American adults are uninsured, it is estimated that 23 % are underinsured, meaning that the health insurance that a person holds does not offer them financial protections that make healthcare accessible (Collins & Gupta, 2024). Hence, these individuals, despite having some insurance coverage, may still experience some of the same challenges as the uninsured. We did not have specific data on if a participant's health insurance was meeting his/her healthcare needs, but this could explain some of our null findings and merits further investigation in future studies where underinsurance can be measured.

Our modeling framework also allowed us to consider the differences between public and private forms of insurance. Both public and private insurance plans showed trends where health insurance was linked to higher SkinBloodAges and lower GrimAge2 and DunedinPoAm biomarkers, and more comprehensive forms of insurance yielded larger model estimates; yet, the associations for private insurance were more frequently statistically significant. One explanation for this difference could be greater differences in the characteristics of individuals with public versus private insurance. More specifically, participants must meet certain characteristics to qualify for government-based insurance. Medicare is generally available to individuals 65 years of age or older; however, people with disabilities, end-stage renal disease, or amyotrophic lateral sclerosis may also qualify. In our study sample, we find that Medicare recipients are chronologically and epigenetically older than the uninsured when compared to other insurance categories. Similarly, to qualify for Medicaid/CHIP, individuals must be low income, children, pregnant women, elderly, and/or living with disabilities. Other forms of government insurance may include Veteran's Administration health benefits or Consolidated Omnibus Budget Reconciliation Act (COBRA) insurance, which enables eligible workers to retain their group health insurance for a limited period after experiencing a change in eligibility. Participants with other forms of government insurance were chronologically and epigenetically older than the uninsured in our study sample, surpassed only by those insured through Medicare. It is possible that health insurance must make up for a greater social vulnerability of the public insurance population; hence, we do not observe benefits as large as we see in the healthier, younger, and wealthier private insurance beneficiaries from our study sample. As extensive literature has documented the benefits of government insurance (Barnes et al., 2021; Creedon et al., 2022), we do not believe that our results suggest this form of insurance is less beneficial. Differences in factors such as patient satisfaction, wait times, and access to specialists can vary both within and between private and public insurance (Allen et al., 2021; Pizer & Prentice, 2011; Resneck et al., 2004; Wray et al., 2021). These differences, particularly in relation to epigenetic aging, warrant further investigation.

We observed similar trends of associations in sensitivity analysis models additionally adjusted for estimated leukocyte proportions and restricted to participants of working age (<65 years old). Similar or stronger observations in models adjusted for leukocytes importantly suggests that health insurance is impacting aging on a cellular level rather than simply impacting cell composition (Zhang et al., 2024). Likewise, given the age of our study sample (50–84 years), similar findings in our working age models provides some reassurance that our findings are not being driven by an older cohort of retirees and may be somewhat generalizable to middle age or younger working adults. Nevertheless, many of our findings were no longer significant and had attenuated model estimates after adjusting for health and lifestyle covariates. One explanation for the null and attenuated results after additional adjustments is that the relationship between health insurance and epigenetic aging is influenced by health and demographic confounders, and also partially mediated by behavioral modifications such as smoking cessation (as previously mentioned), alcohol intake, and

physical activity. This theory is supported by our observation of significant associations of health insurance with epigenetic aging in a sensitivity analysis that did not include adjustments for these modifiable behaviors but did include all other confounders. Importantly, other factors that we do not include in our study like stress or environmental exposures can in some circumstances be viewed as modifiable lifestyle factors and may also play a role in health insurance and epigenetic aging relationships (Dhingra et al., 2018; Nwanaji-Enwerem et al., 2021, 2023).

Strengths of our study include its use of molecular markers of biological aging in a study sample that is representative of the U.S. adult population. Still, our study does have some limitations. First, we performed a cross-sectional analysis that cannot rule out reverse causation and cannot speak to longitudinal insurance coverage. People can transition between employer-based coverage, public insurance, and may at times have temporary coverage gaps. Given that these transitions could significantly impact healthcare access, utilization, and subsequent aging-related processes, it will be important that future studies consider longitudinal trends when possible. Second, NHANES has extensive demographic and lifestyle factor variables, but some of these important measures were missing for our study sample. To help limit the impact of selection bias, we performed basic demographic-adjusted models using the entire study sample. Of note, although there were unweighted trends of older chronological aging and greater uninsurance in participants missing other lifestyle and health variables compared to complete case participants, these differences were neither statistically nor marginally significant. We thus performed imputed-covariate models and observed similar trends seen in our complete study sample basic demographic-adjusted models. Additionally, after adjusting for multiple hypothesis testing, many of our observed relationships did not meet the threshold for statistical significance. Nevertheless, we performed extensive sensitivity analyses with stable trends. Still, if data were not missing at random and missingness was associated with socioeconomic status (SES), this could bias our estimates and limit the generalizability of our findings. Specifically, associations observed in the analytic sample may not fully reflect those in lower SES populations, potentially underestimating disparities or differential effects related to SES. Third, although our data on broad categorizations of insurance can provide important insights, smaller samples sizes for some categories could lead to outlier results. Future studies with other large datasets and with more complete data will be helpful for further characterizing these epigenetic aging relationships. Fourth, our study involves a study sample of U.S. adults 50–84 years of age and uses data collected approximately two decades ago. These factors may impact the generalizability of our results as they pertain to geography, younger populations, and temporality, respectively. Nonetheless, these remain the most current methylation data available within NHANES. Despite these constraints, our study provides valuable insights by exploring novel research questions, and its findings from a nationally representative sample can guide future research utilizing more recent data sources. Lastly, although we attempt to adjust for important covariates in our models, we cannot fully rule out the possibility of unknown or residual confounding in our analyses.

In conclusion, the present study describes trends between health insurance and epigenetic aging in U.S. adults. If substantiated, these findings suggest that epigenetic aging measures may have some utility in studying health insurance and population health relationships with potentially important policy implications. Among these policy considerations may be the ethics of using epigenetic aging for assessing health risks and determining health insurance premiums. There have already been reports of some life insurance companies using epigenetics for insurance underwriting to more precisely predict policy-holders' life expectancies (Dupras et al., 2019). One can imagine a situation where health insurance companies could use epigenetic age to determine premiums or outright deny coverage – similar to what was experienced with pre-existing conditions prior to protections under the Affordable Care Act (ACA) (Zhao et al., 2022). Since legislation like the Genetic Information Nondiscrimination Act (GINA) does not explicitly address epigenetics (Davidson, 2023), strengthening health insurance protections for epigenetic information remains a critical goal for future efforts.

CRediT authorship contribution statement

Jamaji C. Nwanaji-Enwerem: Writing – review & editing, Writing – original draft, Formal analysis, Conceptualization. **Dennis Khodasevich:** Writing – review & editing, Formal analysis. **Nicole Gladish:** Writing – review & editing, Data curation. **Hanyang Shen:** Writing – review & editing, Data curation. **Saher Daredia:** Writing – review & editing. **Belinda L. Needham:** Writing – review & editing, Funding acquisition, Data curation. **David H. Rehkopf:** Writing – review & editing, Supervision, Funding acquisition. **Andres Cardenas:** Writing – review & editing, Supervision, Funding acquisition.

Availability of data and material

The datasets analyzed in the current study are available from the NHANES website.

Ethical statement

This study used de-identified, secondary, publicly available data collected by the United States Centers for Disease Control and Prevention (CDC) National Center for Health Statistics.

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Declaration of competing interest

The authors 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. Supplementary data

Supplementary data to this article can be found online at https://doi.org/10.1016/j.ssmph.2025.101806.

Data availability

The data is already publicly available and this statement is included in the manuscript.

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Glossary

DNAmAge =: DNA Methylation Age

DNAmTL =: DNA Methylation Telomere Length