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Research Article

The environmental chemical exposome and health insurance: Examining associations and effect modification of epigenetic aging in a representative sample of United States adults

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Abstract

Environmental exposures are contributors to morbidity and mortality, yet the potential protective role of health insurance in mitigating these effects remains underexplored. We used data from the 1999-2000 and 2001-2002 cycles of the National Health and Nutrition Examination Survey (NHANES) to examine whether health insurance status is associated with the chemical exposome and whether being insured modifies relationships of environmental exposures with epigenetic aging biomarkers of morbidity and mortality (GrimAge2 and DunedinPoAm). Among 2315 adults aged 50 - 84 years, we evaluated 64 chemical exposures. In covariate, socioeconomic status-adjusted models, being insured compared to being uninsured was marginally associated with 0.21-SD lower blood lead levels (95% CI: −0.39, −0.04, $P = 0.03$) and 0.29-SD higher PCB180 levels (95% CI: 0.003, 0.57, $P = 0.048$). In leukocyte- and covariate-adjusted models, insurance attenuated the relationship of lead with GrimAge2 (Insured: $\beta = 0.08$-years, 95% CI: −0.08, 0.24; Uninsured $\beta = 0.65$-years, 95% CI: 0.11, 1.20; $P_{interaction} = 0.04$) and DunedinPoAm (Insured: $\beta = 0.001$, 95% CI: −0.002, 0.003; Uninsured $\beta = 0.01$, 95% CI 0.0001, 0.02; $P_{interaction} = 0.047$). Similar trends were also observed for cadmium, cotinine, and PCB180 but not statistically different between insurance categories. These findings suggest that health insurance may serve as a protective factor against the biological aging impacts of certain environmental exposures, possibly through improved access to exposure monitoring and preventive/therapeutic care. While not a substitute for environmental policy or exposure remediation, insurance may represent a small, complementary, and more immediately actionable tool to help reduce harm. However, given the small magnitude of model estimates, results should be interpreted with caution regarding their practical significance.

Key words: DNA methylation age; NHANES; health insurance; lead; PCB180; GrimAge; DunedinPoAm.

Introduction

Environmental exposures are significant contributors to global morbidity and mortality. In 2019 alone, ozone and fine particulate air pollution were implicated in an estimated 8.34 million excess deaths worldwide, with over half of this burden attributed to cardiometabolic diseases such as stroke, ischemic heart disease, and type 2 diabetes mellitus.[1] In the United States (U.S.), data from a nationally representative sample revealed that exposure to heavy metals was associated with at least a 38% increase in relative risk for all-cause, cardiovascular, and cancer-related mortality.[2] Although environmental exposures play a critical role in disease development, they often interact with other influential factors such as genetic predisposition, physical activity, diet, and socioeconomic factors. Among these, health insurance is widely recognized as a protective factor because it facilitates access to preventive and therapeutic healthcare.[3-5] However, despite its importance, there is limited evidence showing that health insurance can also protect against diseases linked to environmental exposures. This question is especially important because individuals often have limited ability to shield themselves from environmental risks.[6] For example, while someone may install air filters in their home, it is nearly impossible to avoid outdoor air pollution, as most people cannot easily relocate their residence or workplace.[7] Similarly, although water filters can be used at home, individuals typically have little control over the source of their water, such as wells or municipal pipes.[8] Because of these

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limitations, environmental health protections often rely on broader policy interventions.[9-11] Unfortunately, implementing such policies can take time, leaving many people vulnerable to ongoing environmental harms. If supported by evidence, health insurance could serve as an additional strategy to help protect individuals in the near term. It would not replace the need for policy-level protections but could complement them as part of a broader public health response. Furthermore, given estimates that approximately 26 million Americans remain uninsured, the public health potential for insurance-focused interventions remains significant.[12]

Understanding how health insurance may protect people from harmful environmental exposures is important. Unlike interventions such as air or water filtration, where the way exposure is reduced is clear, the role of health insurance is more complex. Based on existing research, we suggest two main pathways. First, health insurance makes it easier to monitor exposures by improving access to healthcare services. Primary care providers, in particular, play a key role in screening for many environmental and workplace exposures.[13-15] Second, health insurance increases the chances of receiving preventive and therapeutic care that can reduce harm from environmental pollutants. For example, environmental factors such as metals and air pollution contribute to conditions like hypertension (high blood pressure), along with lifestyle and behavioral factors.[16-18] By improving access to medications such as those for blood pressure control, health insurance can help reduce the combined effects of these risks, including those linked to the environment. These pathways offer a new way to think about environmental protection. They also highlight the complexity of these relationships, which reflect broader social and political realities often overlooked in favor of more visible solutions like filtration systems. While less obvious than physical interventions, these pathways provide a practical framework that may inform public health, policy, and clinical discussions. Health insurance-facilitated exposure monitoring can help identify risks that may be reduced through measures like filtration, but even when exposures such as widespread air pollution cannot be fully eliminated, health insurance-facilitated therapeutic interventions can still play an important role in managing disease processes. Ultimately, this analysis aims to spark dialogue. To our knowledge, no previous studies have examined these health insurance pathways while accounting for socioeconomic factors.

To investigate this possibility of observing evidence of health insurance as an environmental exposure harm moderator, we use data from the National Health and Nutrition Examination Survey (NHANES), a nationally representative sample of the U.S. population, to examine whether health insurance modifies associations of chemical environmental exposures with epigenetic aging biomarkers that are linked to morbidity and mortality. In previous exposome-wide analyses of 64 chemical exposures, we found that higher serum levels of cadmium and cotinine were significantly associated with greater GrimAge2 and DunedinPoAm, two DNA methylation-based biomarkers of aging.[19] The GrimAge2 epigenetic clock is a strong predictor of mortality risk that integrates chronological age, sex, and DNA methylation surrogates for cigarette pack-years and plasma protein markers, including adrenomedullin (ADM), beta-2-microglobulin (B2M), C-reactive protein (CRP), cystatin C, growth differentiation factor-15 (GDF-15), hemoglobin A1c (A1c), leptin, plasminogen activator inhibitor-1 (PAI1), and tissue inhibitor metalloproteinase-1 (TIMP1).[20] The DunedinPoAm epigenetic aging biomarker estimates the pace of aging and was built using longitudinal data on 18 organ function biomarkers from individuals of the same chronological age.[21]

In a separate analysis, we found that individuals with health insurance had significantly lower GrimAge2 and DunedinPoAm values compared to those without insurance.[22] These findings suggest a protective role of health insurance, again likely by facilitating access to preventive and therapeutic healthcare. Building on this evidence, the present study uses the same study sample to investigate whether any of the previously examined 64 chemical exposures are associated with health insurance status and whether insurance modifies the relationship of these exposures, including cadmium and cotinine, with epigenetic aging biomarkers. Our goal is to provide evidence to help determine whether health insurance could be considered among the suite of protections available to individuals facing environmental harms.

Methods

Study population

We conducted a cross-sectional study using data from The National Health and Nutrition Examination Survey (NHANES). NHANES is conducted by the National Center for Health Statistics (NCHS) and is designed to assess the health and nutritional status of the noninstitutionalized U.S. population. NHANES collects data through structured interviews, physical examinations, and laboratory testing. For this study, we used publicly available data from the 1999-2000 and 2001-2002 NHANES cycles to examine the associations between chemical environmental exposures, health insurance status, and epigenetic aging biomarkers.[23,24]

Our initial sample included 2532 adults aged 50 years and older, the subset of NHANES participants with existing DNA methylation measurements. To protect participant privacy, NHANES top-coded the ages of individuals aged 85 years and older as 85 years (n = 130). Because exact chronological ages were not available for these participants, they were excluded to avoid potential misclassification in epigenetic age calculations. We also excluded participants whose DNA methylation-predicted sex did not match their self-reported sex (n = 56) to ensure data integrity. After these exclusions, the final analytical sample consisted of 2315 participants with health insurance data. All participants provided written informed consent, and the study protocols were approved by the NCHS Research Ethics Review Board (protocol #98-12). A detailed flowchart of the sample selection process is provided in Figure S1 of this and the original publication.[22]

Environmental exposures

Our environmental exposure assessment has been previously described,[19] but in brief, laboratory data on chemical exposures were obtained from the NHANES website for both the 1999-2000 and 2001-2002 survey cycles. We included datasets measuring chemical concentrations in biological samples, specifically: (1) phthalates, phytoestrogens, and polycyclic aromatic hydrocarbons (PAHs) in urine; (2) current-use pesticides in urine; (3) metals in urine; (4) dioxins, furans, and coplanar polychlorinated biphenyls (PCBs) in blood; (5) cadmium, lead, and cotinine in blood; and (6) volatile organic compounds (VOCs) in blood. Nutrition-related compounds and mercury measures were excluded due to limited population coverage and potential confounding.

Similar to the previous exposome NHANES study, we excluded exposures that were measured in only one of the two survey cycles, measured in pooled samples, or assessed in non-biological media such as dust or water.[19] For dioxins, furans, and coplanar PCBs, we focused exclusively on lipid-adjusted concentrations. Additionally, we excluded any exposure with a detection frequency below 50% in the study population, based on previously compiled NHANES compound detection codes and supplementary data available on Kaggle,[25] leaving 64 of the original 111 chemical exposures for analysis. All exposure variables were log2-transformed to normalize their distributions, then centered and Z-score scaled to improve interpretability of model coefficients. Detection frequencies and distributions have been shown previously.[19] Sample sizes for environmental exposures ranged from n = 110 for chloroform to n = 2313 for cadmium.

Health insurance

Health insurance information was obtained through self-report surveys. Participants were asked, "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.]" Responses were categorized as "yes," "no," or "missing." Participants with missing responses (n = 31) were excluded from all analyses involving insurance status.

DNA methylation and epigenetic age

Epigenetic age measures and DNA methylation-based leukocyte proportion estimates were obtained from publicly available data on the NHANES website.[26] Additional details regarding DNA methylation data processing and analysis are also available through NHANES documentation. Briefly, DNA was extracted from whole blood samples collected from a subset of participants aged 50 years and older during the 1999-2000 and 2001-2002 NHANES cycles. Genome-wide DNA methylation was measured using the Illumina EPICv1 BeadChip array.

For this analysis, we focused on two DNA methylation-based biomarkers of aging: GrimAge2 and DunedinPoAm. These measures were selected because they have previously been shown to be associated with environmental exposures and health insurance status in NHANES.[19,22] GrimAge2 is primarily a measure of mortality risk and integrates chronological age, sex, and DNA methylation surrogates for smoking history and plasma protein markers.[20] DunedinPoAm estimates the pace of aging based on longitudinal data from multiple organ systems and serves as a robust indicator of aging pace.[21]

Statistical analysis

All analyses accounted for the complex survey design of NHANES, including oversampling, survey non-response, and post-stratification. We applied the sample weights developed by NHANES for use with the epigenetic clock subsample to ensure that our findings are representative of the noninstitutionalized civilian U.S. population aged 50-84.[27] Survey-weighted generalized linear regression models were conducted using the R "survey" package, which incorporates participant sample weights and survey design features.[28]

We first evaluated associations between health insurance status (dichotomous: yes *Versus* no) and each log2-transformed, centered, and scaled environmental exposure using the *svyglm* function. Covariates were selected *a priori* based on prior literature.[19,22] Models adjusted for demographic variables without missingness, including chronological age (continuous), age squared (continuous), sex (female Versus male), and self-identified race/ethnicity (Non-Hispanic White, Mexican American, Other Hispanic, Non-Hispanic Black, Other Race—Including Multiracial). Additional covariates with missing data included general health condition (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 (never, former, current; n = 5 missing), alcohol intake (abstainer, moderate drinker, heavy drinker; n = 110 missing), physical activity (moderate/vigorous activity in the past 30 days: yes Versus no; n = 1 missing), education level (less than high school, high school diploma or GED, greater than high school; n = 1 missing), and occupation (white-collar and professional, white-collar and semi-routine, blue-collar and high-skill, blue-collar and semi-routine, or no work; n = 130 missing).[29] Missing covariates were imputed using the MICE package in R with 10 imputations. Estimates from each imputed dataset were pooled using the pool function.[30]

Environmental exposures that were marginally (unadjusted *P*-values < 0.05) or significantly (false discovery rate [FDR]-adjusted *P*-value < 0.05) associated with health insurance status were selected for further analysis, along with serum cadmium and cotinine, which had previously been linked to the epigenetic aging biomarkers GrimAge2 and DunedinPoAm.[19] To evaluate whether health insurance modified the relationship between these exposures and epigenetic aging, we applied the same modeling framework and covariates used in earlier analyses. We additionally adjusted for estimated leukocyte proportions, including CD8 T cells, CD4 T cells, natural killer cells, B cells, monocytes, and neutrophils.[31] This adjustment was made by using residuals from regressions of leukocyte proportions on epigenetic age, given prior evidence that leukocyte composition can influence epigenetic aging measures.[32] Interaction terms between health insurance status and each exposure were included to assess potential effect modification.

We conducted sensitivity analyses to assess the robustness of our findings. First, to examine if estimates from models examining epigenetic aging relationships were impacted by the additional adjustment of leukocyte proportions, we ran models not adjusted for leukocyte proportions. Second, we repeated the analyses not adjusted for leukocytes and using only participants with complete data to evaluate the impact of imputation.

All statistical analyses were performed using R version 4.4.1 (R Core Team, Vienna, Austria). Again, statistical significance in primary analyses was determined using an FDR-adjusted *P*-value threshold of less than 0.05. Findings with unadjusted *P*-values below 0.05 are described as marginal.

Results

Study sample characteristics

The unweighted study sample characteristics have been previously described for this sample,[22] but are also presented in Table 1. In brief, participants had a mean (SD) chronological age of 65.1 (9.3) years and 89% of the participants reported having health insurance. Just over half of the participants reported being male (51%). Non-Hispanic White was the most frequently reported ethnicity (40%). 55% of the participants had at least a high school diploma. With respect to lifestyle behaviors, 49% of participants reported being physically active, 48% were moderate drinkers, and 45% were never smokers. Table S1 describes the

Table 1. Unweighted study sample characteristics (n = 2315).

Aging variables	
Age (years), mean (sd)	65.1 (9.3)
Epigenetic age/clocks, mean (sd)	
GrimAge2 (years)	71.5 (8.5)
DunedinPoAm	1.11 (0.09)
Health insurance	
Health insurance, n (%)	
Yes	2050 (89)
No	265 (11)
Demographic variables	
Education, n (%)	
Less than high school	1043 (45)
High school diploma (including GED)	484 (21)
More than high school	787 (34)
Missing	1 (0)
Occupation, n (%)	
Blue-collar (high skill)	310 (13)
Blue-collar (semi-routine)	913 (39)
White-collar (high skill)	514 (22)
White-collar (semi-routine)	389 (17)
No Work	59 (3)
Missing	130 (6)
Poverty to income ratio, mean (sd)	2.6 (1.6)
Missing	238
Race/ethnicity category, n (%)	
Mexican American	666 (29)
Other Hispanic	147 (6)
Non-Hispanic White	917 (40)
Non-Hispanic Black	505 (22)
Other race	80 (3)
Sex, n (%)	
Male	1188 (51)
Female	1127 (49)
Health behavior variables	
Alcohol intake, n (%)	
Abstainer	997 (43)
Moderate drinker	1125 (48)
Heavy drinker	83 (4)
Missing	110 (5)
Body mass index (kg/m^2), mean (sd)	28.8 (5.8)
Missing	83
Self-rated health, n (%)	
Good	1542 (67)
Fair	589 (25)
Poor	181 (8)
Missing	3 (0)
Smoking, n (%)	
Current	370 (16)
Former	894 (39)
Never	1046 (45)
Missing	5 (0)
Physically Active, n (%)	
Yes	1131 (49)
No	1183 (51)
Missing	1 (0)

unadjusted mean values and corresponding standard errors (± SEM) for the 64 environmental exposures by insurance status. The 19 exposures that were significantly different between insurance status groups (FDR-adjusted *P*-values < 0.05) are shown in Figure 1. Bars represent unadjusted group means, and error bars indicate the precision of these estimates. Among the 19 environmental exposures, only lead, cadmium, and cotinine levels had higher unadjusted mean values in the uninsured when compared to participants with insurance. The remaining 16 exposures had significantly higher unadjusted mean values among insured participants.

Relationships of health insurance with environmental exposures

Table S2 presents the relationships of health insurance with the 64 environmental exposures in imputed, fully-adjusted models. Of these exposures, only polychlorinated biphenyl 180 (PCB180) and lead were marginally associated ($P < 0.05$) with health insurance (Table 2). Compared to uninsured participants, participants with insurance had 0.21-SD lower lead levels (95% CI: −0.39, −0.04, $P = 0.03$) and 0.29-SD higher PCB180 levels (95% CI: 0.003, 0.57, $P = 0.048$). We observed similar model estimates for both lead ($\beta = -0.18$, 95% CI: −0.35, −0.01, $P = 0.04$) and PCB180 ($\beta = 0.23$, 95% CI: −0.04, 0.51, $P = 0.08$) in the complete case sensitivity analysis.

Effect modification of the exposure and epigenetic age relationship by health insurance

Figure 2 presents the relationships of health insurance with SD increases in 4 environmental exposures previously associated with either health insurance (*ie*, lead and PCB180) or epigenetic age (*ie*, cadmium and cotinine). For both lead and PCB180, the relationships of the exposures with both GrimAge2 and DunedinPoAm were mitigated in the insured group in both the main analysis and sensitivity analyses. Specifically, the interaction for lead reached marginal significance in the imputed, leukocyte-adjusted analyses for both GrimAge2 ($\beta_{insured} = 0.08$, 95% $CI_{insured}$: −0.08, 0.24, $\beta_{uninsured} = 0.65$, 95% $CI_{uninsured}$: 0.11, 1.20, $P_{interaction} = 0.04$) and DunedinPoAm ($\beta_{insured} = 0.001$, 95% $CI_{insured}$: −0.002, 0.003, $\beta_{uninsured} = 0.01$, 95% $CI_{uninsured}$: 0.0001, 0.02, $P_{interaction} = 0.047$). The interactions of cotinine and insurance on GrimAge2 followed a similar pattern albeit not statistically or marginally significant.

Discussion

In this analysis of a nationally representative cross-sectional sample of U.S. adults aged 50 to 84 years, we examined the associations of health insurance status with 64 chemical environmental exposures. We found marginally significant associations for lead and PCB180, with insured individuals exhibiting higher blood levels of PCB180 and lower blood levels of lead compared to those without insurance. Building on previous work that identified associations between serum cadmium and cotinine levels and the epigenetic aging biomarkers GrimAge2 and DunedinPoAm,[19] we explored whether health insurance could modify the relationships of cadmium, cotinine, lead, and PCB180 exposures with epigenetic aging. In models adjusted for leukocyte proportions and imputed covariates, we observed a marginally significant interaction suggesting that health insurance may reduce the strength of the association of lead exposure with both GrimAge2 and DunedinPoAm. To our knowledge, this is the first study to present evidence that health insurance may help protect individuals from the adverse health effects of specific environmental chemicals—potentially through the facilitation of exposure monitoring or access to preventive and therapeutic interventions. Similar trends were observed for PCB180, cotinine, and cadmium, but these relationships did not meet thresholds for statistical significance.

One may immediately assume that any relationships of health insurance and environmental exposures may be due to socioeconomic status (SES); hence, we designed our analysis to consider and control for many of these variables. Nevertheless, given the longstanding evidence that individuals of lower SES tend to experience a higher burden of environmental exposures, we believe it

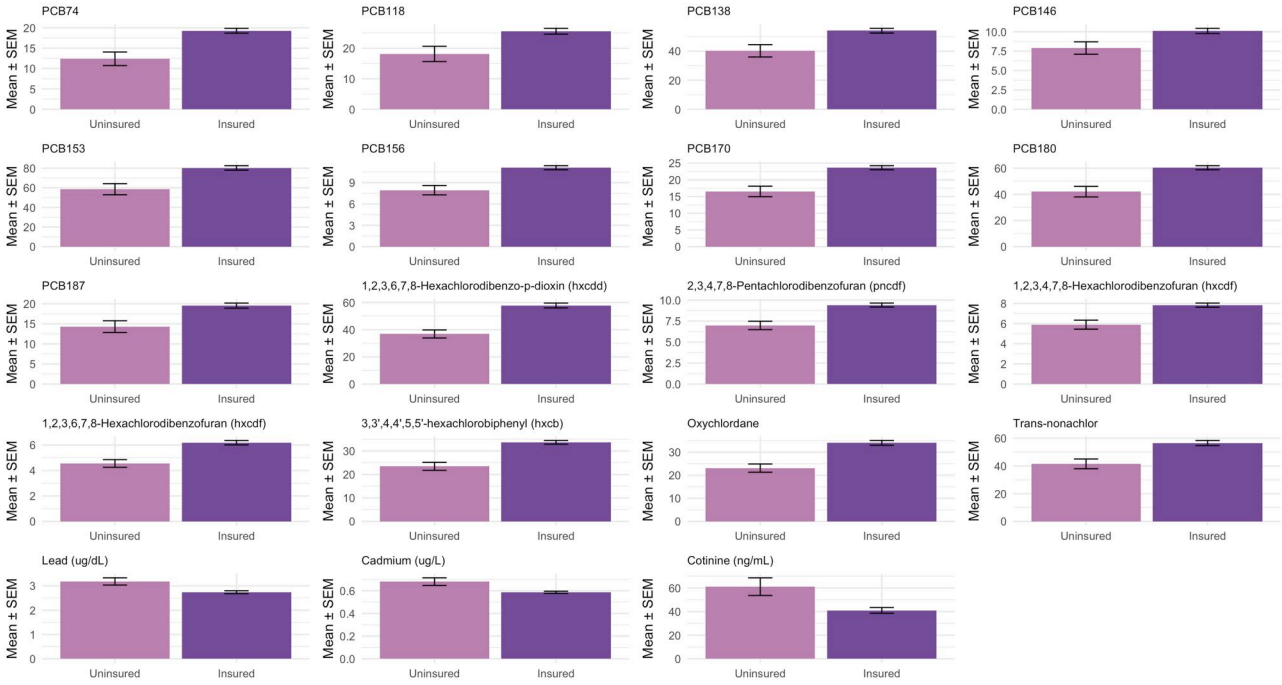

Figure 1. Unadjusted mean chemical environmental exposure levels by insurance status. Figure 1 presents unadjusted mean values and corresponding standard errors (±SEM) for environmental exposures among insured and uninsured individuals. Only exposures with false discovery rate (FDR)-adjusted *P*-values < 0.05 are shown. Bars represent group means, and error bars indicate the precision of these estimates. Comparisons were made using two-sample t-tests.

Table 2. Adjusted associations of health insurance with chemical environmental exposures.[a]

Exposure	Estimate SD (95% CI)	*P*-value	FDR-adjusted *P*-value	N
Imputed models				
PCB180	0.29 (0.003, 0.57)	0.048	0.78	748
Lead	−0.21 (−0.39, −0.04)	0.03	0.78	2313
Complete case models				
PCB180	0.23 (−0.04, 0.51)	0.08	0.59	597
Lead	−0.18 (−0.35, −0.01)	0.04	0.59	1828

Model estimates are for insured participants (uninsured participants are the reference group). Only associations with unadjusted *P*-values < 0.05 are shown.

[a]Models adjusted for chronological age, chronological age2, sex, race/ethnicity, alcohol intake, BMI, education, occupation, physical activity, PIR, smoking, and general health status.

is important to highlight some of these univariate relationships.[33-35] In our study, uninsured participants appeared to reflect this pattern, as indicated by a lower poverty-to-income ratio (PIR) compared to insured participants (1.7 versus 2.8, respectively; see Table S1 of the original publication).[22] Based on this, we anticipated that uninsured individuals would consistently show higher levels of environmental exposures. However, the relationships between insurance status and chemical exposures proved to be more complex. Lifestyle factors that influence contact with environmental chemicals must also be considered. Among the 19 chemicals that showed statistically significant univariate associations with health insurance, only three—lead, cadmium, and cotinine—had higher mean levels in the uninsured population. Notably, lead and cadmium were the only metals among the significant exposures. Both cadmium and cotinine are associated with cigarette smoking, and their associations with insurance status were nonsignificant after adjustment for covariates including smoking. Of note, we observed a higher number of current smokers in our uninsured study participants compared to insured individuals (27% versus 14%, respectively; see Table S1 of the original publication).[22]

Lead and cadmium are toxic heavy metals, and although exposure levels have been decreasing in the U.S.,[36,37] they still pose significant health risks. Lead exposure commonly occurs through deteriorating paint in older homes, contaminated soil, drinking water from aging infrastructure, and certain occupational settings.[38] Cadmium exposure is primarily dietary, with sources including leafy vegetables, grains, and shellfish, as well as tobacco smoke and industrial emissions.[39,40] Both metals are associated with a range of adverse health effects. Lead can impair cognitive development and contribute to behavioral problems in children, while also increasing the risk of hypertension and kidney dysfunction in adults.[41] Cadmium has been linked to kidney damage, bone demineralization, and cancer.[42,43] Cotinine is the primary metabolite of nicotine and is widely used as a biomarker to assess exposure to tobacco smoke.[44] While cotinine itself is not considered harmful at typical levels, elevated concentrations signal increased nicotine intake, which is associated with serious health risks such as cardiovascular disease, respiratory problems, and the harmful effects of secondhand smoke.[45,46] Importantly, these exposures are not evenly distributed. Lower SES has been consistently associated with higher rates of cigarette smoking and elevated levels of cotinine.[47] With respect to metals, low-income and racially marginalized communities are disproportionately affected due to historical patterns of residential segregation, aging infrastructure, and proximity to industrial sites.[34,35] Events such as the Flint water crisis have underscored how systemic neglect can amplify exposure risks in vulnerable populations.[48] Given this context, we were not surprised to observe higher levels of lead, cadmium, and cotinine in uninsured participants. Consistent with this, we found that health

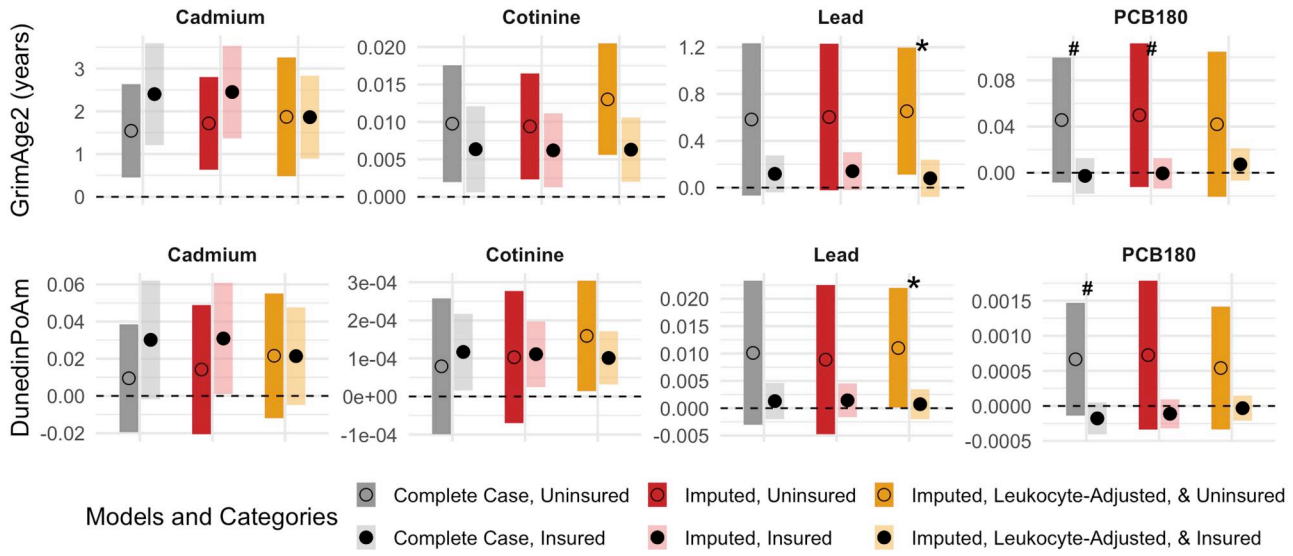

Figure 2. Relationships of environmental exposures with epigenetic age by insurance status. Figure 2 presents model estimates and corresponding 95% confidence intervals for exposure (SD unit increase) (cadmium [n = 2269], cotinine [n = 2313], lead [n = 2313] and PCB180 [n = 748]) and epigenetic age relationships by insurance status. *Interactions of lead and insurance status with both GrimAge2 and DunedinPoAm in imputed, leukocyte-adjusted models reached the thresholds for marginal statistical significance (unadjusted *P*-values < 0.05) and are indicated with asterisks. #Interactions with unadjusted *P*-values < 0.10 are indicated with hash marks.

insurance, which may be associated with higher SES, was associated with lower lead levels in fully-adjusted models, although this relationship was only marginally significant.

Most other chemicals with significant unadjusted, univariate associations showed higher levels in insured participants. The most prominent among these were polychlorinated biphenyls (PCBs), which accounted for more than half of the significant associations. PCBs are synthetic organic compounds that were widely used in industrial and commercial applications until their ban in the late 1970s.[49] Despite the ban, PCBs persist in older equipment and building materials and continue to enter the environment through improper disposal and degradation. They are highly stable and bioaccumulate in the food chain, with the primary route of exposure being ingestion of contaminated food such as fish, meat, and dairy. Additional exposure can occur through inhalation of indoor air in older buildings and dermal contact with contaminated materials. PCBs have been associated with a range of adverse health effects in humans. Occupational exposure has been linked to skin conditions like chloracne, liver enzyme abnormalities, and respiratory issues. In the general population, prenatal and early-life exposure has been associated with developmental delays, reduced birth weight, and long-term cognitive and behavioral deficits. PCBs are also classified as probable human carcinogens and may affect the immune, reproductive, and endocrine systems even at low levels of exposure.[50,51] Our finding that insured individuals had higher levels of PCBs may reflect dietary patterns associated with higher SES, such as increased consumption of seafood, which is a known source of PCB exposure.[52,53] This pattern was also observed in our fully-adjusted imputed models, where insurance was associated with lower lead levels and higher PCB180 levels, although both relationships were marginally significant.

As noted earlier, our fully-adjusted models account for many socioeconomic variables, so we believe the observed association between health insurance and lower lead levels may reflect insurance-related exposure monitoring and access to preventive or therapeutic care. However, these explanations do not seem plausible for the association between health insurance and higher PCB180 levels. A more likely explanation for PCB180, and possibly for lead as well, is residual confounding by socioeconomic factors that our models did not fully capture. It is also worth noting that both associations are only marginally significant and have relatively small model estimates, which further underscores the need for additional research to better understand these relationships. Such work would help determine whether these associations are biological or clinically meaningful.

Still, the central aim of this study was to evaluate whether health insurance modifies the relationship between environmental exposures and epigenetic aging. Our findings suggest that, in many cases, insurance may attenuate the associations between harmful exposures and epigenetic aging biomarkers. In leukocyte-adjusted models for both GrimAge2 and DunedinPoAm, the relationships between cadmium, cotinine, lead, and PCB180 and epigenetic aging were weaker among insured participants compared to those without insurance. The interaction between insurance and lead exposure reached the threshold for marginal significance. These results support the hypothesis that health insurance may play a protective role not only in disease processes driven by genetics or lifestyle factors, but also in those influenced by environmental exposures. Again, we hypothesize that this may be through insurance-facilitated exposure monitoring and preventive/therapeutic care. For example, PCB180 has been linked to obesity and related conditions such as type 2 diabetes,[54,55] which can also result from genetic and lifestyle factors. Environmental exposures may not be the primary driver for every individual, but they can be for some. Regardless of the underlying cause, clinicians are often effective at managing these disorders through lifestyle and therapeutic interventions. Because epigenetic aging reflects morbidity and mortality risk, it is reasonable to expect that access to preventive and therapeutic care facilitated by health insurance could help reduce these risks from environmental and other contributors. Furthermore, research consistently shows that uninsured individuals are less likely to receive recommended health screenings.[56,57] Even among adults with higher incomes, lacking health

insurance is associated with significantly lower use of recommended health services.[58] This makes it plausible that insurance coverage increases the likelihood of screenings, including those for environmental exposures, thereby improving detection of harmful agents and enabling timely intervention.

Although our findings demonstrate consistent trends and marginal statistical significance, they should be interpreted with caution regarding practical significance. The estimate sizes observed in our models were relatively small and may not translate into meaningful differences in real-world health outcomes. Nevertheless, we believe these findings are important to share because, although the model estimates may appear modest in the general population, they could be more biologically and clinically meaningful for vulnerable groups such as children or individuals with greater morbidity. For this reason, we suggest these areas as important priorities for further investigation. Future studies with larger sample sizes and longitudinal designs will also be critical for confirming our observed associations and enabling subgroup analyses. Such analyses may help identify populations for whom health insurance offers more substantial protective benefits against the biological impacts of environmental exposures, potentially reinforcing its role as a complementary intervention alongside broader exposure reduction efforts.

This study offers several notable strengths. First, it utilizes molecular biomarkers of biological aging, specifically GrimAge2 and DunedinPoAm, within a nationally representative sample of middle-aged and older adults in the U.S. This enhances the generalizability of our findings and allows for population-level insights into the intersection of environmental exposures, health insurance, and aging. Second, our analysis focused exclusively on exposures measured in biological media, providing direct and individualized assessments of chemical burden. Third, the NHANES dataset offers a rich array of publicly available questionnaire and laboratory data collected from the same participants, enabling a comprehensive and systematic evaluation of factors influencing epigenetic aging.

Despite these strengths, several limitations should be acknowledged. Our analysis was restricted to 64 chemical exposures, which represents only a small portion of the broader exposome. Nonetheless, this is a substantial number for a novel investigation and provides a strong foundation for future research. The study design was cross-sectional, limiting our ability to infer causality or assess changes in insurance status over time. Individuals may transition between types of coverage or experience gaps in coverage, which could influence healthcare access and aging-related outcomes. Additionally, our sample was limited to adults aged 50-84 years, and the data were collected approximately two decades ago. These factors may affect the relevance of our findings to younger populations and current environmental conditions. However, these remain the most recent DNA methylation data available within NHANES, and the insights gained can inform future studies using more contemporary datasets. We also recognize the potential for residual confounding, despite adjusting for a wide range of socioeconomic and health-related variables. Factors such as dietary patterns and geographic location may still influence both exposure levels and aging outcomes. Furthermore, sample sizes varied across exposures, and while most analyses were adequately powered, some chemicals had limited representation, which may have affected the stability of those estimates. It is also important to note that the health insurance landscape in the United States is primarily employer-based, which may limit the generalizability of our findings to settings with national health insurance schemes or universal health care. In systems where everyone has coverage and basic access to care, health insurance may not serve as a meaningful complementary strategy for addressing environmental harms. Taken together, this study provides an important early contribution to understanding how health insurance may interact with environmental exposures to influence biological aging. Future research should aim to build on these findings using longitudinal designs, younger cohorts, different national health insurance structures, and expanded exposure panels including mixtures to better characterize periods of vulnerability and long-term health impacts.

In conclusion, this study of U.S. adults aged 50-84 years provides early evidence that health insurance may play a protective role in the context of environmental exposures and biological aging. We found that health insurance status was marginally associated with blood levels of lead and PCB180, and that insurance may weaken the relationship between lead exposure and epigenetic aging as measured by GrimAge2 and DunedinPoAm. While similar trends were observed for other exposures, including cotinine and PCB180, these did not reach statistical significance. Although these findings suggest that health insurance, likely through its facilitation of exposure monitoring and access to preventive and therapeutic care, could be considered among the suite of strategies used to protect individuals from the health impacts of environmental exposures, they should be interpreted with caution. The observed effect sizes were modest and may not translate into meaningful differences in real-world health outcomes. Future research with larger samples and longitudinal designs will be essential to confirm these associations and to better understand the mechanisms through which insurance may buffer environmental risks. If these relationships prove to be robust, clinically meaningful, and practically significant, individuals who work with populations affected by environmental exposures—including clinicians, policy makers, and environmental advocates—should consider routinely inquiring about insurance status as part of their efforts to support and protect those they serve. Understanding and addressing insurance coverage may help guide more comprehensive strategies for environmental relief, especially in communities facing disproportionate exposure burdens.

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Author contributions

Jamaji C. Nwanaji-Enwerem(Conceptualization [Lead], Data curation [Lead], Formal analysis [Lead], Investigation [Lead], Visualization [Lead], Writing—original draft [Lead], Writing—review & editing [Lead]), Dennis Khodasevich (Data curation [Supporting], Investigation [Supporting], Methodology [Supporting], Writing—review & editing [Supporting]), Nicole Gladish(Data curation [Supporting], Writing—review & editing [Supporting]), Hanyang Shen (Data curation [Supporting], Writing—review & editing [Supporting]), Anne Bozack (Data curation [Supporting], Writing—review & editing [Supporting]), Saher Daredia (Writing—review & editing [Supporting]), Belinda L. Needham (Funding acquisition [Equal], Supervision [Supporting], Writing—review & editing [Supporting]), and David H Rehkopf (Funding acquisition [Equal], Supervision [Supporting], Writing—review & editing [Supporting]), Andres Cardenas (Funding

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Supplementary material

Supplementary material is available at *Exposome* online.

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Conflicts of interest

The authors declare no conflicts of interest.

Data availability

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

Competing financial interests

None.

Clinical trial registration

Not Applicable.

Institutional review board (human subjects)

All NHANES participants gave written informed consent, and the study protocols received approval from the NCHS Research Ethics Review Board (protocol #98-12).

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