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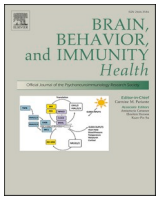

Multi-discrimination exposure and biological aging: Results from the midlife in the United States study

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ABSTRACT

Discrimination is a social determinant of health and health disparities for which the biological mechanisms remain poorly understood. This study investigated the hypothesis that discrimination contributes to poor health outcomes by accelerating biological processes of aging. We analyzed survey and blood DNA methylation data from the Midlife in the United States (MIDUS) study (N = 1967). We used linear regression analysis to test associations of everyday, major, and workplace discrimination with biological aging measured by the DunedinPACE, PhenoAge, and GrimAge2 epigenetic clocks. MIDUS participants who reported more discrimination tended to exhibit a faster pace of aging and older biological age as compared to peers who reported less discrimination. Effect-sizes for associations tended to be larger for the DunedinPACE pace-of-aging clock (effect-size range r = 0.1–0.2) as compared with the PhenoAge and GrimAge2 biological-age clocks (effect-sizes r < 0.1) and for experiences of everyday and major discrimination as compared with workplace discrimination. Smoking status and body-mass index accounted for roughly half of observed association between discrimination and biological aging. Reports of discrimination were more strongly associated with accelerated biological aging among White as compared with Black participants, although Black participants reported more discrimination overall and tended to exhibit older biological age and faster biological aging. Findings support the hypothesis that experiences of interpersonal discrimination contribute to accelerated biological aging and suggest that structural and individual-level interventions to reduce discrimination and promote adaptive coping have potential to support healthy aging and build health equity.

1. Introduction

Discrimination, irrespective of its attribution (e.g., race, sex, weight), is a substantial social determinant of health, exerting a profound negative impact on various health outcomes (Williams et al., 2019). People exposed to discrimination experience increased risk for cardiovascular disease, hypertension, self-reported health deterioration, mortality, depression, psychological distress, and suicidal ideation (Williams et al., 2019). Despite these well-documented associations and behavioral pathways (e.g., obesity and smoking (Unger, 2018; Hunte, 2011)), the precise biological mechanisms underlying the influence of discrimination on the development of these diseases remain poorly understood. Nevertheless, one potential component of this mechanism appears to be the chronic activation of the stress response (Lawrence et al., 2022).

The biological weathering hypothesis posits that the persistent exposure to discrimination and other psychosocial stressors accelerate the aging process, particularly among Black Americans, elevating their susceptibility to disease and premature mortality (Forde et al., 2019). This hypothesis finds strong support in empirical data revealing that aging-related diseases manifest at younger ages for Black Americans (Geronimus et al., 2006; Boen, 2016), Black-White disparities intensify

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with advancing age, (Geronimus et al., 2006; Crimmins et al., 2003) and are conspicuously present across a wide spectrum of age-related health conditions (Boen, 2016). While strides have been taken in elucidating how discrimination heightens disease risk, our comprehension of the underlying biological mechanisms remains incomplete.

DNA methylation (DNAm) has emerged as a promising marker of the impacts of the social environment on our biology. The process of DNA methylation involves the addition of a methyl group to cytosine residues within the DNA sequence. Environmental chemical exposures can directly affect DNAm while other environmental features may impact DNAm indirectly through effects on behavior or the physiological cascade induced by psychological stress (Belsky and Baccarelli, 2023). DNA methylation is also affected by the aging process (Horvath, 2013). Over the past decade, biomarkers derived from analysis of blood DNAm have emerged as the leading measurement technology to quantify the pace and progress of biological aging in epidemiological studies of humans (Moqri et al., 2023).

The first generation of DNAm biomarkers of aging were developed using machine learning tools to create algorithms that could predict how old a person was (Horvath, 2013). These biomarkers came to be known as "clocks", partly for the complexity of their construction and partly for their striking accuracy. However, the first generation of DNAm clocks proved to be poor predictors of healthspan and lifespan and were not consistently sensitive to risk exposures, including social determinants of health, that showed other indications of accelerating the aging process. In response, a second generation of clocks were developed by applying machine learning tools to develop algorithms to predict survival time (Levine et al., 2018; Liu et al., 2018). The resulting second-generation clocks proved more predictive of morbidity and mortality and more sensitive to exposure histories, including social determinants of health. Most recently, clocks were developed to predict the rate of physiological deterioration, termed "pace of aging" (Belsky et al., 2022). Whereas the first and second generation clocks function like odometers in a car, recording the amount of aging experienced across a lifespan, the pace of aging clocks function like speedometers, reading out how fast aging is occurring.

Accelerated biological aging plays a profound role in the onset of various diseases, including cardiovascular diseases, cancer, and neurodegenerative disorders (López-Otín et al., 2013, 2023; Campisi et al., 2019). "Odometer" epigenetic clocks measure accelerated biological aging as the difference between clock-calculated epigenetic age and chronological age. "Speedometer" epigenetic clocks measure accelerated biological aging as the ratio of clock-calculated years of biological aging per calendar year. Individuals exposed to a range of adverse life conditions exhibit epigenetic-clock signs of accelerated biological aging. Childhood adversity, economic hardship across life, and experiences of psychological distress are all associated with older biological age/faster pace of aging as measured by epigenetic clocks (Raffington and Belsky, 2022). Emerging evidence suggests that race-related adversities, including discrimination, may also contribute to accelerated biological aging (Simons et al., 2021, 2023).

The existing body of research in the field of discrimination and health predominantly focuses on a single aspect of discrimination, namely everyday discrimination. Everyday discrimination refers to subtle and minor instances of disrespect experienced in daily life (Williams, 1999). However, discrimination can also manifest with acute intensity, resembling other significant life events such as severe accidents or the loss of a loved one. Acute forms of discrimination (e.g., being physically threatened by police officers, being denied a promotion), often referred to as "major discrimination," are linked with increased risk for hypertension (Beatty et al., 2016), premature mortality (Obaoye et al., 2023), and cardiovascular disease (Lewis et al., 2014). Discrimination can also manifest in the workplace, where individuals spend a significant portion of their lives. Workplace discrimination encompasses unjust practices, stunted opportunities, and punitive actions (Cheung et al., 2016). Workplace discrimination is also associated with adverse health outcomes, including hypertension (Li et al., 2023) and long-term illness-related absence (Clark et al., 2021). Given the multifaceted ways in which discrimination manifests, research is needed to understand how different forms of discrimination may contribute to biological aging.

The study of discrimination and biological aging can shed light on discrimination as a social determinant of healthy aging, while also revealing a potential root cause of racial disparities. Black-White disparities in aging-related morbidity and mortality are large and have persisted for decades (Quiñones et al., 2019). Currently, Black adults have a higher burden of many aging-related chronic diseases and higher death rates for the leading causes of death—including heart disease, stroke, and diabetes—compared to their White counterparts (Quiñones et al., 2019). Socioeconomic and health-behavior factors only partly explain these differences, suggesting that psychosocial aspects of racial identity, including experiences of discrimination, may constitute an important mechanism driving disparities.

According to a national survey, (National Public Radio NPR, 2018) approximately 74% of Black individuals reported experiencing discrimination, in significant contrast to just 25% of White individuals. This statistic underscores the pervasive nature of discrimination for Black Americans and may play a crucial role in differential disease risk between Black and White Americans. Building understanding of how discrimination may contribute to accelerated biological aging is therefore a critical step toward elucidating mechanisms through which systemic inequities become embodied and to uncovering solutions for promoting health equity.

This study aimed to investigate the relationship between various forms of discrimination (everyday, major, and workplace) and biological aging, using multiple epigenetic clocks. We also tested whether smoking and BMI might serve as mediator in the association between discrimination and biological aging. We hypothesized that higher levels of discrimination would be associated with accelerated biological aging. Furthermore, we hypothesized that this association would be more pronounced among Black participants compared to their White counterparts. By examining these relationships, we sought to contribute to a better understanding of the impact of discrimination on biological aging processes and its potential differential effects across racial groups.

2. Methods

2.1. Data and analytic sample

Participants in the present study enrolled in the Study of Midlife in the United States (MIDUS) Biomarker Project (n = 1255; 2004–2009) (Dienberg et al., 2010) or MIDUS Refresher Biomarker Study (n = 863; 2012–2016) (Weinstein et al., 2019). Participants underwent a comprehensive assessment covering sociodemographic, psychosocial, and behavioral factors before participating in the biomarker assessments. DNA methylation (DNAm) profiling was conducted on whole blood DNA samples and data were released in 2023. Currently, epigenetic age scores are available for 1310 participants; all of whom participated in any one of the biomarker assessments. Data collection was approved by Institutional Review Boards at University of Wisconsin, and all participants provided informed consent. The epigenetic age scores are found in the MIDUS data portal. (Home).

2.2. Measures

Biological aging. Biological aging is the progressive loss of integrity and resilience capacity in our cells, tissues, and organs that occurs as we grow older. Biological aging arises from the accumulation of molecular damage resulting in cellular-level changes or "hallmarks" that compromise functioning and damage health, leading to many different chronic diseases. While there is no gold standard measure of biological aging in humans, the current state-of-the-art are a family of algorithms known as

"epigenetic clocks" that estimate the pace and progress of biological aging from the DNA methylation states of white blood cells. To date, there have been three distinct generations of these epigenetic clocks. A first generation was developed to predict chronological age by comparing the genomes of older and younger people. A second generation was developed to predict mortality risk based on analyses of blood-test and survival-time data. A third generation was developed from analyses of longitudinal change in panels of organ-system integrity indicators. In this study, we concentrated our analysis on the second and third-generation clocks, given their robust predictive potential for morbidity and mortality risks, along with their notable sensitivity to the influence of social determinants of health: DunedinPACE (Belsky et al., 2022), PhenoAge (Levine et al., 2018), and GrimAge2 (Lu et al., 2019). The values of the DunedinPACE clock reflect rates of aging relative the reference norm of one year of biological change per calendar year and therefore require no further transformation. For analysis, the PhenoAge and GrimAge2 clocks were regressed on chronological age to compute residual values that index how much older or younger a person is biologically relative to their chronological age.

Everyday discrimination. Everyday discrimination will be assessed using the Everyday Discrimination Scale (Williams et al., 1997). The scale includes items such as being treated with less courtesy or respect than others; receiving poorer service than others at restaurants or stores; being called names, insulted, threatened, or harassed; and having people act afraid of the respondent. This scale employs four frequency response codes (1 = often, 2 = sometimes, 3 = rarely, 4 = never). A final score (ranging from 9 to 36) was calculated by taking the sum of the values of reverse-coded items, whereby higher scores indicate greater reports of everyday discrimination.

Major discrimination. Major Experiences of Discrimination Scale asks participants how many times in their lives they have experienced acute forms of discrimination (Kessler et al., 1999). The scale includes occurrences in different settings, such as academics (discouraged from continuing education), employment (being denied promotion), financial services (prevented from renting or buying a home), and experiences of social hostility (hassled by the police). A final score (ranging from 0 to 10) for major discrimination was constructed by taking the sum of endorsed questions irrespective of frequency (i.e., 1 = event occurred one or more times, 0 = event never occurred), where higher scores reflect greater experiences of major discrimination.

Workplace Discrimination. Workplace discrimination was measured using an adapted version of the Chronic Work Discrimination and Harassment scale (Bobo and Suh, 1995; McNeilly et al., 1996). The scale asks participants how often they experienced different forms of discrimination in the workplace in the last 12 months. Items include being unfairly given the jobs that no one else wanted to do, being watched more closely than other workers, and supervisor or boss using ethnic, racial, or sexual slurs or jokes. Items were rated on a 5-point scale (1 = once a week or more, 2 = a few times a month, 3 = a few times a year, 4 = less than once a year, 5 = never). A final score (ranging from 6 to 30) was calculated by taking the sum of the values of reverse-coded items, such that higher scores reflect higher workplace discrimination.

2.3. Covariates

The following variables were included as covariates in the primary models: participant's race (categorical: Black and White), age (continuous); sex (categorical: male, female); and educational attainment (categorical: less than high school, high school degree/GED, some college and above). Employment status (categorical: employed, unemployed) was included in the analyses of workplace discrimination.

2.4. Potential mediators

Smoking status (categorical: currently or not smoking) and body mass index (BMI; continuous) are commonly included as covariates in studies assessing social determinants and biological aging. Nonetheless, these health behaviors play a role within the pathways linking discrimination to health outcomes. Thus, we incorporated them in a separate model, recognizing their potential role in the association between discrimination and biological aging.

2.5. Data analytic procedures

For analysis, discrimination and biological aging measures were standardized to have mean = 0, standard deviation = 1. This transformation allows for direct comparison of effect sizes across models and interpretation of coefficient magnitudes on a Pearson r scale.

We regressed each of the three discrimination exposures (everyday, major, and workplace) on each of the three epigenetic clocks (DunedinPACE, PhenoAge, and GrimAge2) in separate models. For each discrimination measure-epigenetic clock pair, we fitted three regression models. The first model included covariates for chronological age and sex. The second model added covariates for racial identity category (Black, White), level of education, and employment status. The third model added covariates for body-mass index and smoking status. Finally, we performed stratified analyses by racial-identity category and formally tested for effect modification by race for all discrimination measures using interaction terms. Analyses were conducted using R (R Core Team R. R, 2013).

3. Results

Our analysis sample comprised 1967 participants (55% women, mean age 53 years) who identified as Black (19%, n = 376) or White (81%, n = 1591; Table 1) and had data on any of the predictors or outcomes of interest. The majority of participants had completed at least some college (77%) and most were employed (67%). Participants exhibited an average everyday discrimination score of 13.01 (SD = 4.87), a major discrimination score of 1.15 (SD = 1.78), and a workplace discrimination score of 10.87 (SD = 4.79).

3.1. Main analyses

Adjusting for age and sex, participants who reported higher levels of everyday discrimination tended to have faster DunedinPACE values (r = 0.19, 95% CI = 0.14, 0.25), PhenoAge (r = 0.03, 95% CI = 0.004, 0.06), and GrimAge2 (r = 0.08, 95% CI = 0.06, 0.11; Table 2). After adjusting for race and educational attainment, the association between everyday discrimination and the clocks remained statistically different from zero (see Fig. 1; Table 2).

Higher levels of major discrimination were also associated with

Table 1
Descriptive characteristics of full sample.

Characteristic	Overall N = 1967 (Williams et al., 2019)
Age (years)	53 (12)
Race	
White	1591 (81%)
Black	376 (19 %)
Sex (n = 1967)	
Male	887 (45%)
Female	1080 (55%)
Education (n = 1936)	
< High School	84 (4.3%)
High School/GED	359 (19%)
Some College and Above	1493 (77%)
Employment Status (n = 1889)	
Employed	1262 (67%)
Unemployed	627 (33%)
Daily Discrimination (n = 1949)	13.01 (4.87)
Major Discrimination (n = 1926)	1.15 (1.78)
Workplace Discrimination (n = 1324)	10.87 (4.79)

Table 2
Relationship between discrimination and biological aging.

	Everyday Discrimination		Major Discrimination		Workplace Discrimination	
	r	95% CI	r	95% CI	r	95% CI
DunedinPACE						
M1	**0.19**	**0.14, 0.25**	**0.21**	**0.15, 0.26**	**0.11**	**0.05, 0.18**
M2	**0.13**	**0.08, 0.18**	**0.14**	**0.09, 0.20**	**0.07**	**0.004, 0.13**
M3	**0.08**	**0.02, 0.14**	**0.10**	**0.04, 0.16**	0.00	−0.07, 0.07
PhenoAge						
M1	**0.03**	**0.004, 0.06**	0.03	−0.001, 0.05	0.01	−0.02, 0.04
M2	**0.03**	**0.01, 0.06**	**0.04**	**0.01, 0.07**	0.01	−0.02, 0.04
M3	0.01	−0.02, 0.05	0.02	−0.01, 0.05	−0.01	−0.05, 0.03
GrimAge2						
M1	**0.08**	**0.06, 0.11**	**0.07**	**0.04, 0.10**	**0.04**	**0.01, 0.08**
M2	**0.06**	**0.03, 0.08**	**0.05**	**0.03, 0.08**	0.02	−0.01, 0.05
M3	**0.04**	**0.01, 0.07**	0.03	−0.004, 0.06	−0.02	−0.05, 0.02

Note: Bold signifies significance at $p < 0.05$.
Model 1. Covariates include chronological age and sex.
Model 2. Covariates include those from Model 1 in addition to race, educational attainment, and employment status.
Model 3. Covariates include those from model 2 in addition to smoking and body-mass index.

faster DunedinPACE (r = 0.21, 95% CI = 0.15, 0.26) and GrimAge2 (r = 0.07, 95% CI = 0.04, 0.10) in the age and sex-adjusted models (see Fig. 2). The relationships remained statistically significant after adjusting for race and educational attainment. In the age and sex-adjusted model, there was no significant association between major discrimination and biological aging for PhenoAge (r = 0.03, 95% CI = −0.001, 0.05). However, after accounting for race and educational attainment, major discrimination showed a significant association with biological aging (b = 0.03, 95% CI = 0.001, 0.06).

Workplace discrimination was positively associated with DunedinPACE (r = 0.11, 95% CI = 0.04, 0.177) and GrimAge2 (r = 0.05, 95% CI = 0.02, 0.08), adjusting for age and sex (see Fig. 3). After adjusting for race, educational attainment, and employment status, the association remained statistically significant for DunedinPACE (r = 0.07, 95% CI = 0.004, 0.13), but not for GrimAge2 (r = 0.02, 95% CI = −0.01, 0.05). Participant reports of workplace discrimination showed no significant association with PhenoAge, in both age and sex-adjusted (r = 0.01, 95% CI = −0.03, 0.04) and models further adjusting for race, educational attainment, and employment status (r = 0.01, 95% CI = −0.02, 0.04).

3.2. Adjusting smoking status and BMI

After adjusting for smoking status and BMI, most associations were attenuated below the level of statistical significance. Higher levels of everyday discrimination remained positively associated with DunedinPACE (r = 0.08, 95% CI = 0.02, 0.14) and GrimAge2 (r = 0.04, 95% CI = 0.01, 0.08. Major discrimination was positively associated with DunedinPACE values (r = 0.10, 95% CI = 0.04, 0.16).

3.3. Race as a moderator

We investigated whether discrimination could have different consequences for biological aging in White and Black participants, adjusting for race, age, sex, and educational attainment. In stratified analyses, we observed positive associations between both major and everyday discrimination and biological aging for White participants across all clocks, while there was no association or weaker associations for Black participants (seeSupplemental Tables 1–3). Workplace discrimination was associated with the DunedinPACE measure of biological aging for White participants, but not for Black participants. In formal tests of effect-measure-modification, we found these differences were

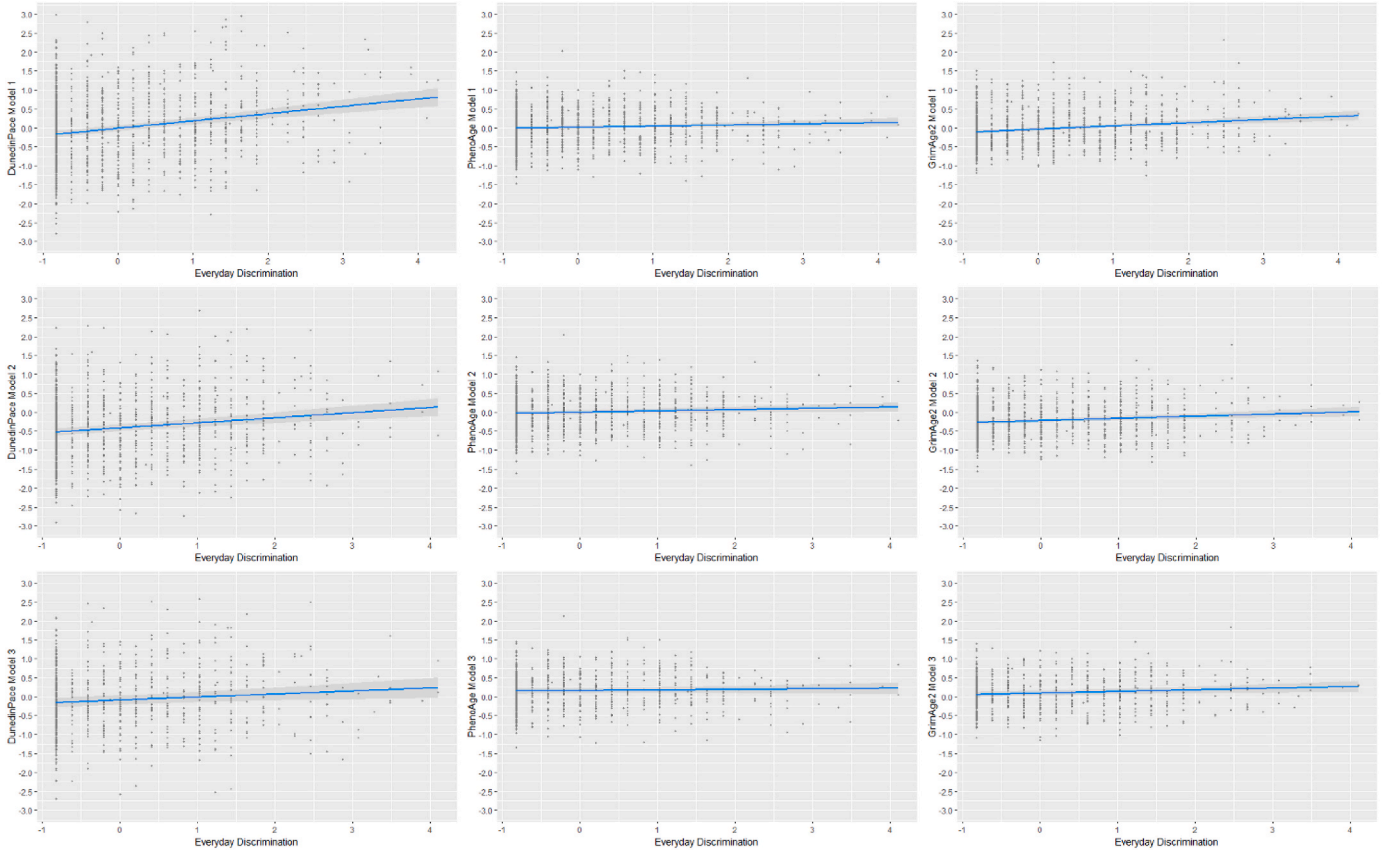

Fig. 1. Everyday discrimination and biological aging.

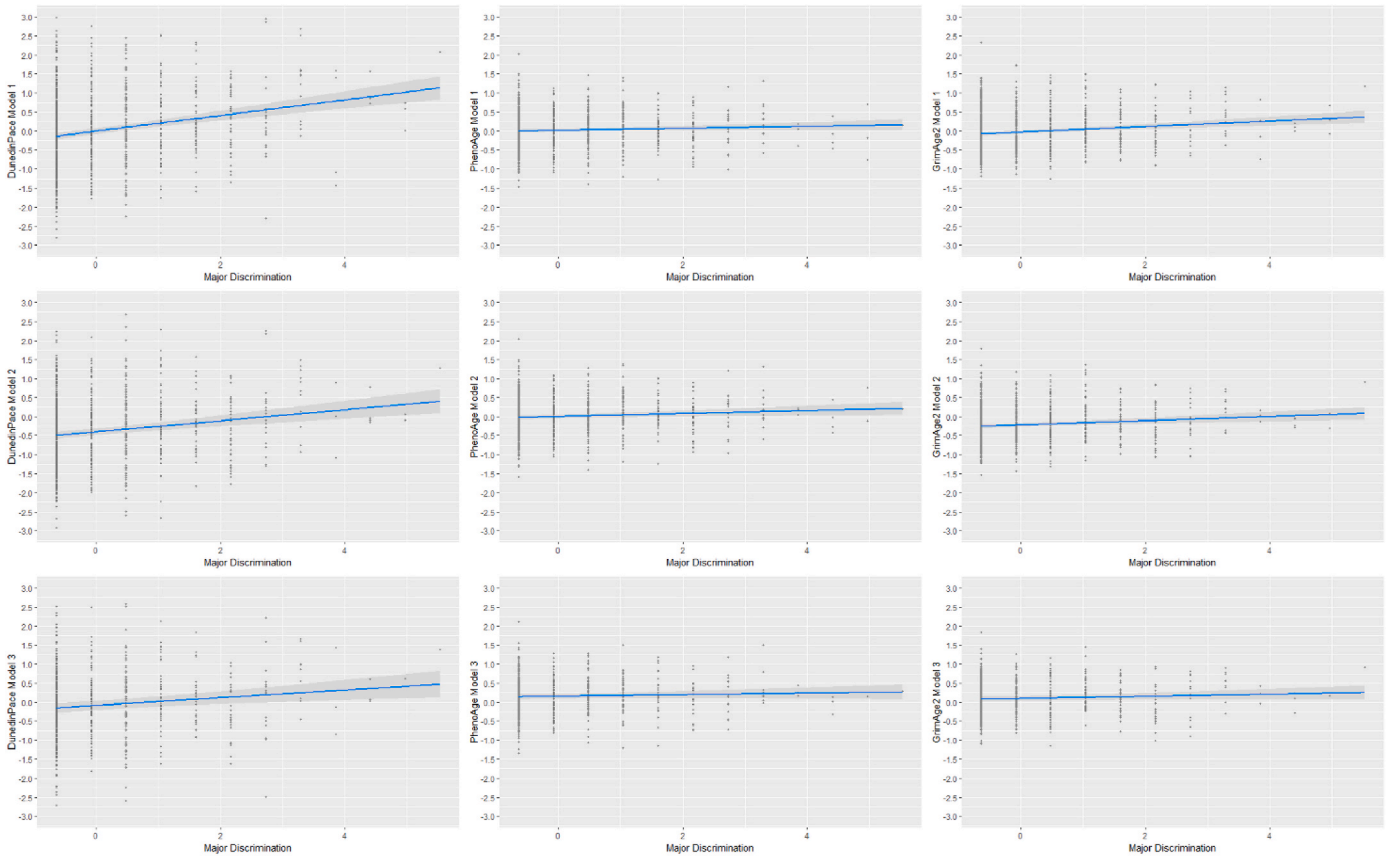

Fig. 2. Major discrimination and biological aging.

statistically significant for workplace discrimination (r = −0.17, 95% CI = −0.30, −0.04) and everyday discrimination with DunedinPACE (r = −0.17, 95% CI = −0.27, −0.06). Race x major discrimination interaction was significant with GrimAge2 (r = −0.069, 95% CI = −0.13, −0.01).

4. Discussion

We investigated relationships between three forms of discrimination and three epigenetic-clock measures of biological aging. There were three main findings. First, midlife adults who report experiencing more discrimination tend to exhibit a faster pace of aging and older biological age, as measured by epigenetic clocks. Second, experiences of everyday and major discrimination were more strongly associated with biological aging as compared with workplace discrimination. Third, effect-sizes for associations were to some extent larger for the "speedometer" clock DunedinPACE as compared with the "odometer" clocks PhenoAge and GrimAge2. In exploratory analyses, we found roughly half of the relationship between discrimination and biological aging was accounted for by smoking and body mass index. Overall, the associations between discrimination and biological aging varied by race, whereby discrimination was stronger for White participants compared to Black participants.

There is growing interest in the biological pathways that underlie discrimination and disease. One mechanism that warrants attention is accelerated biological aging, as examined in this study. We found that greater reports of discrimination were associated with accelerated biological aging. Specifically, everyday and major discrimination consistently exhibited association with biological aging across all three aging clocks. These distinct forms of discrimination encapsulate diverse facets of mistreatment—chronic and acute, respectively. Studies have shown the far-reaching effects of both forms of discrimination on disease risk (Williams et al., 2019). Our empirical evidence posits that these discriminatory experiences may exert their influence at the molecular level, thereby hastening the process of aging. It is noteworthy to highlight that exposure to discrimination in the workplace can also accelerate aging, even though it was not associated with the biological clocks, with the exception of DunedinPACE. It is plausible that workplace discrimination, although detrimental, may be comparatively less severe or enduring than everyday and major discrimination. Workplace discrimination constitutes just one facet of an individual's life and may not accrue to the same extent as other forms of discrimination. This underscores, however, the need for future research to replicate study findings, examining whether the duration of employment plays a role in the association between workplace discrimination and biological aging.

While the mechanism linking discrimination and aging remains unclear, we have some ideas regarding its pathways. The effect sizes were attenuated by half after adjusting for smoking and BMI, which was expected as these variables likely reflect a range of behavioral processes linking discrimination and biological aging. Nevertheless, exposure to discrimination can also trigger the release of physiological responses (e.g., release of cortisol) and other behavioral responses (e.g., poor sleep) that can lead to accelerated aging (Slavich, 2016). Different forms of discrimination can elicit distinct physiological and behavioral reactions, necessitating further investigation. Nevertheless, our findings suggest that indices of biological aging can provide a summary of the downstream consequences of these physiological and behavioral responses to the stresses of discrimination. As such, they represent useful measures for population surveillance and for evaluation of interventions designed to reduce discrimination or to mitigate its health consequences.

In our analysis, effect-sizes for associations of discrimination with biological aging were larger for the DunedinPACE clock as compared with the other two clocks. A key difference between DunedinPACE and the other two clocks is that DunedinPACE is designed to measure the

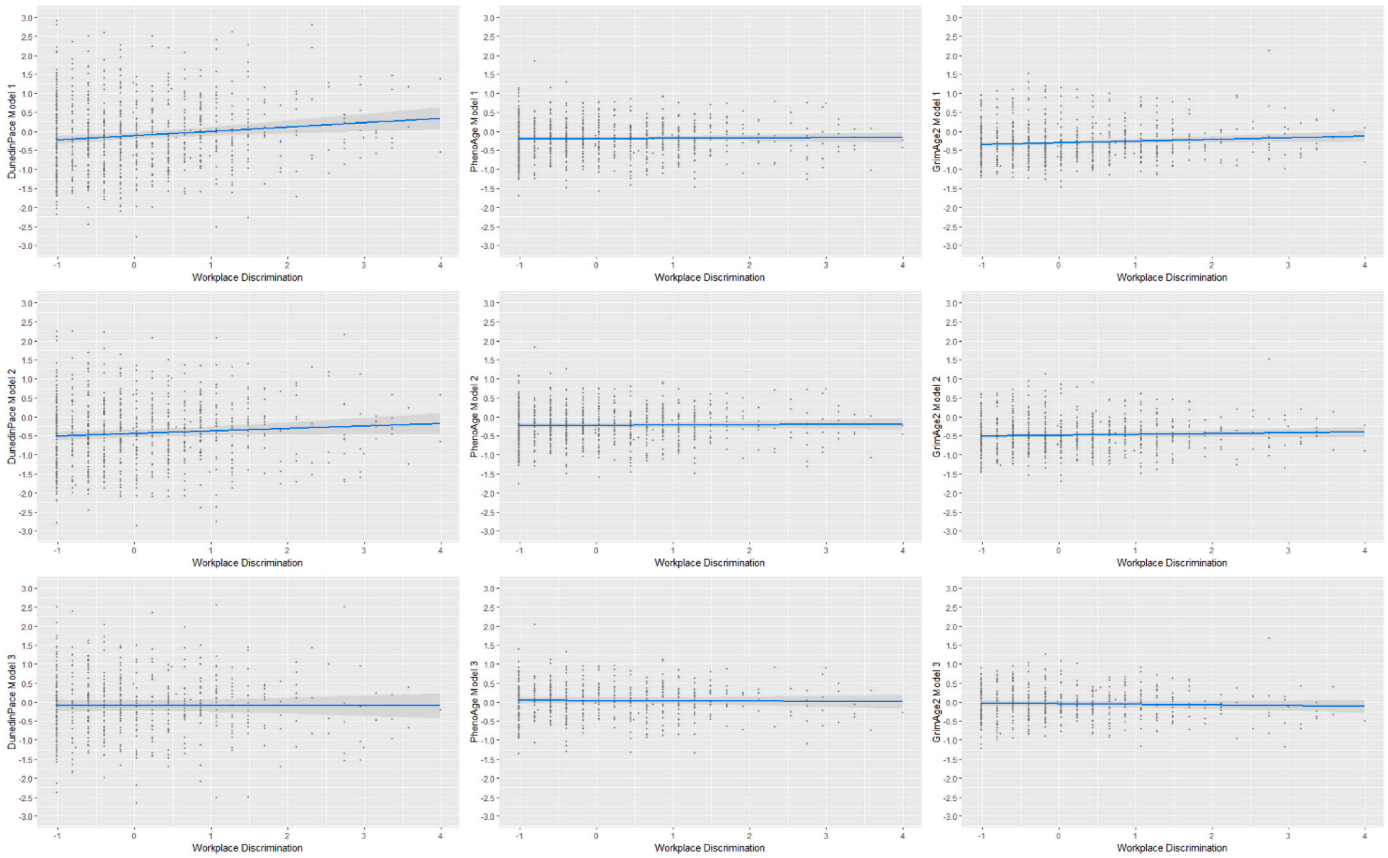

Fig. 3. Workplace discrimination and biological aging.

current pace of aging whereas the other clocks are designed to measure the cumulative effects of aging across the entire lifecourse. To the extent that experiences of discrimination are only beginning to accelerate aging processes by midlife, DunedinPACE may be more sensitive to these just-emerging effects. However, replication in other samples is needed to confirm this interpretation.

We found that associations between discrimination with biological aging tended to be stronger among White-as compared to Black-identifying participants. This pattern aligns with prior research in which associations between discrimination and various adverse health outcomes, including mortality risk, are stronger for White-as compared with Black-identifying participants (Barnes et al., 2008; Ayalon and Gum, 2011; Fuller-Rowell et al., 2012). It is possible that Black Americans, a group consistently exposed to discrimination throughout their lives, have developed more resilient coping strategies to manage this stressor, whereas White Americans, who experience discrimination less frequently, could be more susceptible to its impacts (Ayalon and Gum, 2011). Gaining insight into how Black and White Americans cope with discrimination can help shed light into our findings. While the association between discrimination and biological aging may be more pronounced among White participants, it is important to mention that Black Americans consistently face higher levels of discrimination throughout their lives. Therefore, it is particularly important to identify potential psychosocial resources that may mitigate the impact of discrimination on epigenetic aging for this group.

We acknowledge limitations. There is no gold-standard measure of biological aging. We focused on three well-studied epigenetic clocks because of robust evidence that these clocks are (a) predictive of future morbidity and mortality; and (b) sensitive to social determinants of health. Findings should be replicated with alternative biomarkers of aging as they pass validation testing. The MIDUS sample with DNA methylation data is not representative of the US population and we were not able to include analysis of race/ethnic subgroups other than Black- and White-identifying Americans. Extension of analyses to other race/ethnic groups is a priority. Discrimination was assessed at a single time point. Exposure to discrimination may not be stable over the course of an individual's life. This could be particularly evident in the context of workplace discrimination, as individuals who become unemployed, change jobs, or retire from work may experience less exposure to workplace discrimination compared to those who remain in the same work environment. Potential fluctuations in discrimination exposure may have significant implications for downstream epigenetic aging processes. Future research should aim to examine how cumulative exposure to discrimination across multiple time points influences epigenetic aging. In addition, the current assessments of discrimination primarily focus on the interpersonal level, relying on self-reported experiences. However, interpersonal discrimination is a multidimensional construct that goes beyond these self-reports. Individuals who are victims of discrimination may not always be aware of the discriminatory events they experience, yet they can still suffer from its adverse health effects. The current study design may underestimate the true impact of interpersonal discrimination. Moreover, discrimination is not limited to the interpersonal level. Discrimination can take form at the neighborhood-level by way of limited educational and economic opportunities, inadequate access to resources that promote health, and suboptimal air and water quality. Given the potent health effects of neighborhood-level discrimination, particularly for Black Americans, it may exert additional influences on biological aging that go beyond the impact of interpersonal forms of discrimination. Understanding the multidimensional nature of discrimination is crucial for a comprehensive examination of its effects on epigenetic aging and its broader implications for health disparities.

5. Conclusion

The findings of this study shed light on a potential mechanism underlying the relationship between discrimination exposure and disease. To the best of our knowledge, this study is one of the first to examine multiple forms of discrimination while considering multiple epigenetic clocks within a large community-based cohort of adults. Our results demonstrate that everyday and major discrimination are associated with accelerated biological aging. Workplace discrimination is also positively associated with biological aging, but only for DunedinPACE. These findings lay a foundation for future research to delve into the pathways linking discrimination with biological aging, examine potential protective factors, and investigate additional dimensions of discrimination.

CRediT authorship contribution statement

Adolfo G. Cuevas: Conceptualization, Writing – original draft. **Steven W. Cole:** Conceptualization, Data curation, Investigation, Writing – original draft. **Daniel W. Belsky:** Conceptualization, Writing – original draft, Writing – review & editing. **Anna-Michelle McSorley:** Methodology, Writing – original draft, Writing – review & editing. **Jung Min Shon:** Formal analysis, Investigation, Writing – original draft, Writing – review & editing. **Virginia W. Chang:** Supervision, Writing – original draft, Writing – review & editing.

Declaration of competing interest

DWB is listed as an inventor on the Duke University and University of Otago invention DunedinPACE, which is licensed to TruDiagnostic.

Data availability

All data are publicly available from MIDUS.

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Appendix A. Supplementary data

Supplementary data to this article can be found online at https://doi.org/10.1016/j.bbih.2024.100774.

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