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Research Article

Relationship Between Accelerated Biological Aging, Race, Perceived Discrimination, and Limitations in Activities of Daily Living

Maria Teresa Brown[1*], Miriam Mutambudzi[2], Nai-Wei Chen[3]

[1]School of Social Work, Syracuse University, New York, USA

[2]Department of Public Health, Syracuse University, New York, USA

[3]Biomedical Informatics, Biostatistics and Medical Epidemiology, School of Medicine, University of Missouri, New York, USA

***Corresponding author:** Maria Teresa Brown, School of Social Work/Aging Studies Institute/Syracuse University, 314 Lyman Hall, Syracuse, New York, 13244, USA

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Abstract

This cross-sectional study aimed to evaluate the relationship between accelerated aging (PhenoAge) and limitations in Activities of Daily Living (ADLs) in White, Black, and Hispanic older adults (≥ 50 years). We further aimed to explore how perceived discrimination may differentially impact this association. We ran multivariable logistic regression models to evaluate the strength of the association between accelerated aging, ADLs, and perceived discrimination using 2014/2016 Health and Retirement Study (HRS) data for White (n=2107), Black (n=435), and Hispanic (n=351) adults aged 50 and older who contributed epigenetic clock data during the 2016 HRS Venous Blood Study. We hypothesized that accelerated aging would be associated with greater levels of functional limitations, and that perceived discrimination would impact this relationship with evident disadvantage among Blacks and Hispanics relative to Whites. We found that more Blacks and Hispanics reported ADLs (23.68% and 23.08% respectively) than Whites (12.48%, $p<0.01$). Blacks reported more discrimination (M 3.71, SD 4.26, $p<0.01$) than Hispanics (M 2.69, SD 3.84) or Whites (M 2.86, SD 3.52). ADLs were associated with accelerated aging (OR=1.42, 95% confidence interval = 1.13,1.78) and discrimination (OR=1.07, 95% confidence interval = 1.04,1.10). Our research examining how exposure to discrimination differentially affects the biological aging-ADL association across racial and ethnic groups contributes to efforts addressing health disparities associated with functional decline. This work is part of a broader body of research aiming to understand the impact of discrimination on biological outcomes and their consequences for health and loss of independence.

Key words: epigenetic aging; epigenetic clocks; activities of daily living

Introduction

Biological aging, defined based on patterns of DNA methylation (DNAm) in the human genome [1], is a relatively new field of study. Based on biological differences in aging, adults who are the same chronological age may have different levels of vulnerability to age-related health concerns and mortality [2]. Disparities between chronological and biological aging may contribute to nuanced variations in health outcomes, with cumulative effects across the life course. Advanced chronological age is associated with increased risk of chronic health outcomes [3], functional and cognitive decline [4], and reduced quality of life [5]. When a person's biological age is greater than their chronological age, they can be considered as experiencing accelerated aging [6].

Previous research has linked biological aging with various indicators of adversity across the life span, and with various health behaviors [6]. Social scientists have not identified all effects of biological aging on health in later life or fully explored the life course experiences and social determinants that contribute to variations in biological aging. This cross-sectional study aimed to evaluate the relationship between accelerated aging (PhenoAge) and limitations in Activities of Daily Living (ADLs) in White,

Black, and Hispanic older adults (≥ 50 years. We further aimed to explore how perceived discrimination may differentially impact this association and assess whether this impact varies by race or ethnicity.

Second-Generation Epigenetic Clocks

Rather than being trained on chronological age, as were the first generation of epigenetic clocks, second-generation epigenetic clocks were constructed using clinical measures of phenotypic age and their relation to novel CpG sites, which may make them better predictors of biological aging [2]. GrimAge and PhenoAge are two commonly used second generation epigenetic clocks, which have different developmental backgrounds. GrimAge was derived from data of the predominantly white Framingham Heart Study Offspring sample. [7]. In contrast, PhenoAge was developed using clinical data from the third National Health and Nutrition Examination Survey (NHANES III), which represents a racially diverse, nationwide probability sample (NHANES III; Centers for Disease Control and Prevention, 2024). Consequently, PhenoAge is more suitable for analyzing racially diverse populations.

PhenoAge has been linked to health behaviors (e.g. smoking) and lifestyle and demographic factors like education, income, and exercise, negatively correlated with "good" HDL cholesterol, and positively correlated with C-reactive protein, insulin, glucose, triglycerides, and waist to hip ratio [2]. PhenoAge has also been linked to chronic health conditions, all-cause mortality, and aging-related morbidity [2, 8, 9] and is a more accurate predictor of 10- and 20-year survival than first generation epigenetic clocks [10]. DNAm summary measures like PhenoAge have been suggested as potentially improving our understanding of health disparities, disease, and age-associated functional decline [6]. To date however, it is unclear whether accelerated biological age may impact one's ability to live independently and carry out activities of daily living (ADLs).

Discrimination

Social scientists have only recently begun investigating the role of biological age as a proxy for cumulative inequality, the result of the lifetime accumulation of exposure to structural inequalities that can differentially impact people of color, and a potential predictor of health and social outcomes in epidemiological studies of aging [11]. One experience or range of experiences that may contribute to cumulative inequality and health disparities for non-Whites in the United States is discrimination. Individual experiences of discrimination have been linked to racial differences in quality of education and employment opportunities for individual members of minority groups and in turn adversely affect socioeconomic status for members of minority groups [12].

The association between socioeconomic status and health is well-documented and there is growing body of research indicating that experiencing multiple forms of discrimination can indirectly affect health regardless of race, but that this impact is worse for Blacks [12-14]. Results from studies exploring the relationship between perceived discrimination and various health outcomes – body mass index, obesity, poorer health, cardiovascular disease – and between discrimination and indicators of poor health – hypertension, markers of inflammation, allostatic load – provide some evidence of an association, although results can vary by gender, race, and the indicators being studied [13]. Our understanding of these associations would benefit from additional research.

Limitations in Activities of Daily Living

Limitations in Activities of Daily Living (ADLs) in older adults have been associated with onset of disability, loss of independence, greater morbidity, and greater mortality due to cardiovascular disease [15-17]. For example, ADL dependence has been associated with higher rates of 30-day hospital readmissions among older adults undergoing hip or knee replacement [18]. Studies have found that retaining ADL independence may be a critical factor in aging successfully among those 80 and older [19]. As many older adults desire to remain healthy and independent, and to exert control over their individual aging experience, it is important to understand what factors affect functional independence and, conversely, ADL limitations [20].

The Current Study

The overarching objective of this cross-sectional study was to explore the association between biological age and functional limitations and to assess the differential impact of perceived discrimination on this association in Black, Hispanic, and White older adults. To achieve this objective, the project used the 2014 and 2016 Health and Retirement Study (HRS) data on adults aged 50 and older including PhenoAge epigenetic clock data from the 2016 HRS Venous Blood Study (VBS) [21].

This study attempted to answer the research questions: 1) How is accelerated aging, as measured by PhenoAge, associated with functional limitations in older adults? and 2) Is this association differentially impacted by perceived discrimination or race/ethnicity? We hypothesized that: 1) accelerated aging would be associated with greater levels of functional limitations, and 2) perceived discrimination would impact this relationship with evident disadvantage among Blacks and Hispanics relative to Whites.

Materials and Methods

Data

The Health and Retirement Study (HRS) is a longitudinal cohort study of retirement and health in non-institutionalized Americans, which uses a mixed-mode design of telephone and face-to-face interviews, to collect data on a wide range of factors including sociodemographic, social, behavioral, and physical and mental health. Hispanics and non-Hispanic Blacks were oversampled at a 2:1 rate and maintained a high follow-up rate [22]. During the 2016 interview, 78.5% of all HRS respondents consented to have their blood drawn for the HRS VBS [21], and collection was completed for 82.9% or 9,934 cases (65% of eligible HRS cases). Thirteen epigenetic clocks were constructed using the 2016 VBS

DNA methylation data (n = 4,018, 40.4% of 2016 respondents), and 3,966 of these participants provided all data needed for regression analyses to estimate epigenetic clocks. Specific details of the processing of these DNA methylation data can be found in [6].

In 2014 and 2016 respectively, in-person interviews were conducted with 50% mutually exclusive study samples, who were also given a mail-in self-administered Psychosocial Leave Behind Questionnaire (PLBQ) (90% response rate). Data on perceived discrimination were obtained from the 2014 and 2016 PLBQs for those respondents with whom VBS collection was completed. Approximately 1,735 participants in 2014 and 1,557 participants in 2016 completed the PLBQ and have epigenetic clock data.

Sample Inclusion and Exclusion: Participants were included in this study if they were (1) 50 years and older in 2016, (2) had 2016 VBS DNA methylation data, and (3) had completed the PLBQ in 2014 or 2016, including perceived discrimination items. Participants were excluded if their HRS interviews were completed via proxy. Our final sample consisted of 2,893 HRS participants: 2,107 non-Hispanic Whites, 435 non-Hispanic Blacks, and 351 Hispanics.

Measures

Outcome Measure: The dependent variable for this pilot was limitations in activities of daily living (ADLs), as measured in the HRS in 2016. The HRS identified functional limitations using a modified version of the Katz Activities of Daily Living (ADL) scale from the Rand files [23], which includes six tasks (i) getting out of bed, (ii) bathing, (iii) dressing, (iv) eating, (v) toileting, and (vi) walking across a room, resulting in a 0–6 point scale of ADL limitations [24]. Consistent with previous studies on biological aging and to facilitate clearer interpretation of results, we opted to use a binary variable in our analysis [8]. While this may sacrifice some nuance, it aligns with established practices in the field and enhances clarity of our results.

Independent Variables: The primary independent variable is accelerated aging, which was derived from the second-generation epigenetic clock "PhenoAge" developed and trained by Levine and colleagues (2018). To assess the presence of accelerated aging for each participant, we regressed PhenoAge on chronological age [6, 25] and calculated the residual from this regression [25]. We then constructed a binary variable with 1 representing older predicted epigenetic age or accelerated aging (positive residual) and 0 representing younger predicted epigenetic age (negative residual). All regressions with the independent variable accelerated aging included a control for chronological age.

Moderating Variables: The moderator of interest, perceived discrimination, is measured by the Everyday Discrimination Scale. This five-item scale measures chronic and routine interpersonal discrimination and unfair treatment in everyday life in the past 12 months on a five-point Likert scale (never to almost every day [26]: (1) being treated with less courtesy or respect, (2) receiving poorer service at restaurants or stores; (3) people acting as if they think you are not smart; (4) people acting as if they are afraid of you; and (5) being threatened or harassed [27]. The scale is made up of the average of the five items (range = 0-25, higher scores reflect greater exposure to discrimination). Using these five items, we generated a dichotomous discrimination variable (none vs. any).

Control Variables: Regression models include measures for chronological age, race (Black, Hispanic, and White), sex (female=1, male=0), and years of education (ranged from 0-17). Household income is an integer variable representing total household income of respondent and spouse. Health controls included currently smoking and self-rated health. Currently smoking was defined by responses to the question "about how many cigarettes or packs do you usually smoke in a day now?" with scores of 1 for one or more and 0 for none; missing cases (n=16) were assigned the modal value of 0. Self-rated health scores ranged from excellent (1) to poor (5), with larger values indicating worse self-rated health.

Analytic Plan

We ran descriptive analyses by race to assess differential distributions of ADLs, PhenoAge, and controls for demographic, behavioral, social, and health factors within the study sample. To test our first hypothesis, we ran bivariate analyses (difference of means tests and cross-tabulations with chi-square tests of significance) to examine the association between ADLs and the PhenoAge clock. Multivariable logistic regression models evaluated the strength of the association between ADLs and PhenoAge. Model 1 regressed the presence of at least one ADL on the binary indicator for accelerated aging while controlling for chronological age and additional demographic, social, behavioral, and health factors. To test our second hypothesis, we added perceived discrimination to Model 2, an interaction between accelerated aging and discrimination to Model 3, and an interaction between accelerated aging and race in Model 4.

Results

As seen in Table 1, there were racial differences in any ADL limitations (12.5% of Whites compared to 23.1% of Hispanics and 23.7% of Blacks, $p<0.01$) and in the mean number of ADL limitations (0.50, SD 1.10 for Blacks, 0.50, SD 1.16 for Hispanics, and 0.23, SD 0.76 for Whites, $p<0.001$). Whites had higher mean chronological age (71.05, SD 9.34, $p<0.01$) compared to Blacks (66.67, SD 7.97) or Hispanics (66.39, SD 8.35), and their epigenetic age, as indicated by the PhenoAge clock, was older (58.29, SD 0.86, $p<0.01$) compared to Blacks (55.18, SD 9.36) and Hispanics (55.36, SD8.85). The proportions of Whites (46.18%), Blacks (48.74%), and Hispanics (50.71%) with accelerated aging were similar ($p=0.221$) (Table 1).

	Full Sample		Black (n=435)		Hispanics (n=351)		White (n=2107)		
Characteristic	N / Mean	% / SD	N/Mean	% / SD	N / Mean	% / SD	N / Mean	% / SD	Sign.
Chronological Age (mean, SD)	69.83	9.25	66.67	7.97	66.39	8.35	71.05	9.34	***
PhenoAge (mean, SD)	57.47	9.76	55.18	9.36	55.36	8.85	58.29	0.86	***
Accelerated Aging (N, %)	1363	47.11%	212	48.74	178	50.71	973	46.18	
Female (N, %)	1719	59.42%	300	68.97	208	59.26	1211	57.48	***
Household Income (mean, SD)	$73,474	$101,623	$45,064	$53,109	$39,515	$48,043	$84,996	$112,813	***
Years of Education (mean, SD)	13.10	3.02	12.77	2.73	9.95	4.47	13.69	2.37	***
Self-Rated Health (mean, SD)	2.81	1.00	3.03	1.00	3.25	1.01	2.69	0.97	***
Health Behaviors									
Currently Smoking (N, %)	288	9.96%	66	15.17%	34	9.69%	188	8.92%	***
Body Mass Index (mean, SD)	28.84	6.10	31.05	6.88	30.32	6.55	29.01	6.15	***
Functional Limitations									
Number of ADLs (mean, SD)	0.306	0.88	0.499	1.10	0.504	1.16	0.233	0.755	***
Any ADLs (N, %)	447	15.45%	103	23.68%	81	23.08%	263	12.48%	**
Perceived Discrimination (mean, SD)	2.97	3.69	3.71	4.26	2.69	3.84	2.86	3.52	**

Table 1: Sample Characteristics by Race, 2014/2016 Health and Retirement Study

A larger proportion of Blacks in this sample were female (68.97%, p<0.0001) relative to Hispanics (59.26%) and Whites (57.48%). Whites had significantly greater incomes on average ($85,000) compared to $45,000 and $40,000 for Blacks and Hispanics respectively. Whites had significantly better self-rated health (2.69 – good to very good) relative to Blacks (3.03 - good) and Hispanics (3.25 - fair to good), lower body mass index (29.10 vs 31.05 and 30.32), and were less likely to be current smokers (8.92% vs 15.17% and 9.69%). Unexpectedly, Whites had higher perceived discrimination scores (2.86) than Hispanics (2.69), and lower than Blacks (3.71).

Bivariate analyses of associations between ADL limitations and the PhenoAge clock are presented in Table 2. A greater proportion of participants with accelerated aging (19.15%) reported any ADL limitations relative to those with no accelerated aging (12.16%, p<0.0001). The association between specific numbers of ADL limitations and the experience of accelerated aging was also significant and varied based on the number of ADL limitations. For example, participants with accelerated aging were more likely to report one limitation than those without it (10.56% vs 5.82%, p<0.0001), but the differences between participants with and without accelerated aging were much smaller when looking at four or more ADL limitations (1.89% vs 2.65%). Finally, the mean number of ADL limitations significantly varied based on whether a participant had experienced accelerated aging (0.36, SD 0.03, p<0.0001) or not (0.26, SD 0.84) (Table 2).

	No Accelerated Aging[a]	Accelerated Aging[a]	Sig.
Number of ADL Limitations – mean (SD)	0.26 (0.84)	0.36 (0.030)	***
Any ADL Limitations	12.16%	19.14%	***
One ADL Limitation	6.20%	10.41%	***
Four or more ADL Limitations	2.20%	2.30%	***

*p<0.05, **p<0.01, ***p<0.001
[a]Accelerated aging indicated by a binary variable representing the presence (1) or absence (0) of PhenoAge residual values exceeding chronological age.

Table 2: Bivariate Associations Between ADL Limitations and Accelerated Aging, 2014/2016 Health and Retirement Study

In Model 1 of the multivariate logistic regressions, accelerated aging was associated with greater odds of having at least one ADL limitation. Each year of accelerated aging increased the risk of ADLs by 37% (OR 1.37, 95% CI 1.10-1.71). Other factors associated with greater risk of at least one ADL were older chronological age (OR 1.03, 95% CI 1.02-1.04), being Black (OR 1.86, 95% CI 1.39-2.48), and poorer self-rated health (OR 2.70, 95% CI 2.38-3.07). Being Hispanic was not associated with greater risk of ADLs (OR 1.33, 95% CI 0.93-1.90) (Table 3).

	Model 1		Model 2		Model 3		Model 4	
Variable	OR	95% CI	OR	95% CI	OR	95% CI	OR	95% CI
Accelerated Aging[a]	1.37	[1.10,1.71]	1.42	[1.13,1.78]	1.41	[1.05,1.89]	1.41	[1.07,1.88]
Chronological Age	1.03	[1.02,1.04]	1.03	[1.02,1.05]	1.03	[1.02,1.045	1.03	[1.02,1.05]
Demographic Characteristics								
Female	1.24	[0.98,1.56]	1.30	[1.03,1.65]	1.30	[1.03,1.65]	1.30	[1.03,1.65]
Household Income	1.00	[1.00, 1.00]	1.00	[1.00, 1.00]	1.00	[1.00, 1.00]	1.00	[1.00, 1.00]
Years of Education	0.97	[0.94,1.01]	0.97	[0.94,1.01]	0.97	[0.94,1.01]	0.97	[0.94,1.01]
Race / Ethnicity (Ref: Non-Hispanic White)								
Non-Hispanic Black	1.86	[1.39,2.48]	1.83	[1.37,2.45]	1.83	[1.37,2.45]	1.99	[1.31,3.04]
Hispanic	1.33	[0.93,1.90]	1.43	[1.00,2.04]	1.43	[1.00,2.05]	1.26	[0.76,2.10]
Health Behaviors								
Currently Smoking	1.00	[0.71,1.43	1.01	[0.71,1.43]	1.01	[0.71,1.43]	1.00	[0.70,1.43]
Self-Rated Health	2.70	[2.28,2.07]	2.62	[2.30,2.97]	2.62	[2.30,2.97]	2.62	[2.30,2.98]
Perceived Discrimination			1.07	[1.04,1.10]	1.07	[1.03,1.11]	1.07	[1.04,1.10]
Interaction Effects								
Accelerated Aging with Discrimination					1.00	[0.95,1.06]		
Accelerated Aging with Race								
Non-Hispanic White							(omitted)	

Non-Hispanic Black							0.85	[0.49,1.50]
Hispanic							1.25	[0.67,2.32]
Intercept	-6.83		-7.42		-7.42		-7.43	
[a]Accelerated aging indicated by a binary variable representing the presence (1) or absence (0) of PhenoAge residual values exceeding chronological age.								

Table 3: Presence of at Least One ADL Limitation in 2016 (N=2,869), 2014/2016 Health and Retirement Study

In Model 2 we added perceived discrimination, which increased the odds of having at least one ADL limitation by 7% (OR 1.07, 95% CI 1.04-1.10). Including discrimination in the models increased the association between accelerated age and ADLs (OR 1.42, 95% CI 1.13-1.78). At the same time, it slightly reduced the risk of ADLs based on being Black (by 3%, OR 1.83, 95% CI 1.37-2.45), and increased the risk based on being Hispanic (by 10%, OR 1.43, 95% CI 1.00-2.04). The risk of ADLs because of self-rated health also decreased (by 8%, OR 2.62, 95% CI 2.30-2.97). In Models 3 and 4 we added interactions between accelerated aging and discrimination and accelerated aging and race. None of these interactions were significant, nor did they change the relationship between accelerated aging and the odds of having ADL limitations.

Discussion

This study contributes to the literature on biological aging by exploring the relationship between biological aging and the presence of ADL limitations in older adults. We found evidence that accelerated aging, as measured by Levine's PhenoAge epigenetic clock, was associated with greater risk of ADL limitations at age 50 or older. Our first hypothesis was supported by the significant association between accelerated aging and risk of one or more ADL limitations and between race and ethnicity and risk of ADL limitations. Our second hypothesis was partially supported by evidence that perceived discrimination was significantly associated with ADLs and accounting for discrimination increased the odds of having at least one ADL limitation by 5% for individuals with accelerated aging. The addition of discrimination to the model partially explained the odds of ADL limitations associated with being Black. Counterintuitively, being Hispanic was not initially associated with the risk of ADL limitations, and accounting for discrimination increased the odds of ADL limitations among Hispanics. However, the cross-sectional design of the study precludes any determination of causation between discrimination and other factors. Finally, we found no significant effects for interactions between accelerated aging and discrimination or between accelerated aging and race.

Experiences of racial discrimination at different points in the life course have previously been linked with accelerated or biological aging [28-30]. The weathering hypothesis posits that social inequity, and the repeated exposure to socially structured stressors resulting from that inequality, leads to health deterioration early in the life course for African Americans but not for Caucasians [31], and that this repeated exposure can lead to life-course accumulation of disease vulnerability in marginalized groups [32]. This process of physical weathering leads to disparities in allostatic load [33], biological aging [34], risk of hypertension [35,36], and in combination with genomics leads to racial disparities in breast cancer risk [37].

Our findings provide partial support for this pattern among Black respondents in our sample, in that we have linked perceived discrimination with greater risk of ADL limitations among Black respondents. However, our results do not show a similar pattern for Hispanics in our sample, as accounting for discrimination increased the odds of ADL limitations among Hispanics, rather than explaining that association. Contrary to prior research, Hispanics in our sample also reported lower exposure to discrimination than Black or White respondents [38]. Our findings related to the Hispanic subgroup in our sample indicate a broader diversity in Hispanic experiences of ADL limitations and discrimination, and a need to disaggregate Hispanic samples to capture their range of experiences [39]. Future research should incorporate larger samples of Hispanics to enable disaggregation of this population.

Chronological aging occurs at the same rate for everyone and has been identified as a strong risk factor for various aging-related diseases and death. However, due to genetic variation and the accumulation of lifetime exposures, experiences, and health factors, adults of similar chronological age may be experiencing different rates of biological aging [40-42]. Enhancing our comprehension of the efficient utilization of biological age as a potential indicator for health and social consequences that contribute cumulative inequality over the life course [11] is necessary.

Numerous policies rely on chronological age, posing a latent challenge for adults experiencing health or independent living declines before becoming eligible for specific benefits or assistance. Assessing health and social needs based on biological age could enable the fine-tuning of population-level health and social policies. Further, identifying health disparities related to biological age can contribute to targeted public health interventions. This refinement is crucial for mitigating aging related health challenges, dependency, and diminished quality of life, in particular for those with accelerated aging. By exploring the relationship between biological aging and functional limitations, the findings from this study can improve our understanding of the potential need for services and supports among older adults who would benefit, but may currently be excluded, from age-tested retirement, social support, income support, or health and long-term care policies.

Limitations

These findings should be evaluated in the context of several limitations. First, the first-generation epigenetic clocks were derived using primarily white European samples of older adults, which restricts their usability with non-White and non-Western populations. PhenoAge was developed using NHANES III, which was a more diverse sample and included oversampling of Mexican Americans and Non-Hispanic Blacks [43,44]. However, as a second-generation clock, it does have roots in first generation clock data and may still be limited in its ability to explain variation in health among non-white older adults. Second, the data employed in this analysis were cross-sectional, and therefore we do not know how functional limitation may change over time based on biological aging. Future studies should incorporate ADL measures from subsequent waves of the HRS to evaluate differences in trends over time. Third, measures for perceived discrimination in these data only account for experiences in the prior 12 months, and therefore do not account for discrimination experienced across the life course; further research should include lifetime measures of discrimination. Fourth, the use of a binary measure for accelerated aging may introduce threshold effects and eliminate the ability to detect the different associations with ADL limitations that we might find between individuals with a biological age ten years older than their chronological age and those with a biological age only one year older than chronological age. However, the distribution of the residual values for PhenoAge was so broad (-27.83 to 42.29), and the mean value so small (8.19838E-15, SD 6.74), that using those residual values to capture the rate of accelerated aging resulted in nonsignificant results. Finally, the HRS data did not capture within-person factors that may influence functional limitations over time. Other data should be incorporated to assess the influence of intrapersonal factors that may explain some of the variation in functional limitation seen in these data.

Implications

Our findings contribute to knowledge about how biological age, in spite of chronological age, may be associated with functional limitations differently for those exposed to any discrimination compared to none, in mid to later life. Research examining how discrimination affects the biological aging-functional limitations association for each racial group can better inform efforts to address disparities in health and well-being associated with loss of independence due to functional decline. The findings from this study can inform the design and use of targeted structured interventions to prevent or reduce ADL limitations among higher-risk older adults, such as older adults who have been exposed to discrimination or have experienced accelerated aging.

Conclusions

The ability to measure biological age provides a significant opportunity to rethink processes of aging and policy and health interventions to address health disparities across the life course [45]. Previous research has shown how social factors can result in accelerated aging, with subsequent unfavorable health outcomes [29,46]. It is important to understand whether and how chronological and biological age may be incongruent, as this may provide important insights into which adults may need supports earlier than expected and are often unable to access resources reserved for adults beyond a certain chronological age.

The current study identified accelerated aging and perceived discrimination as risk factors for ADL limitations. Understanding differing levels of risk among older adults based on history of discrimination or biological age can inform preventive measures and design and implementation of interventions to address these risks. This knowledge can be used to evaluate and modify existing social and economic support programs to acknowledge the different levels of needs older adults may experience when their biological age is greater than their chronological age.

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Ethical Guidelines

This study analyzed secondary, de-identified data, and was determined to qualify for an exemption by the Syracuse University Institutional Review Board.

Conflict of Interest

We have no conflicts of interest to disclose.

References

1. Moore LD, Le T, Fan G (2013) DNA methylation and its basic function. Neuropsychopharmacology Reviews 38: 23-38.
2. Levine ME, Lu AT, Quach A, Chen BH, Assimes TL, et al. (2018) An epigenetic biomarker of aging for lifespan and healthspan. Aging 10: 573-591.
3. Lakatta EG (2002) Age-associated cardiovascular changes in health: Impact on cardiovascular disease in older persons. Heart Fail Rev 7: 29-49.
4. Tucker-Dobb EM, Johnson KE, Jones RN (2009) The cognitive reserve hypothesis: A longitudinal examination of age-associated declines in reasoning and processing speed. Developmental Psychology 45: 431-446.
5. Trombetti A, Reid KF, Hars M, Hermann FR, Pasha E, et al. (2016) Age-associated declines in muscle mass, strength, power, and physical performance: Impact on fear of falling and quality of life Osteoporosis International 27: 463-471.
6. Crimmins EM, Thyagarajan B, Levine ME, Weir DR, Faul JD (2021) Associations of age, sex, race/ethnicity, and education with 13 epigenetic clocks in a nationally representative U.S. Sample: The health and retirement study. J Gerontol A Biol Sci Med Sci 76: 1117-1123.
7. Lu AT, Quach A, Wilson JG, Reiner AP, Aviv A, et al. (2019) DNA methylation grimage strongly predicts lifespan and healthspan. Aging 11: 303-327.

8. Mutambudzi M, Brown MT, Chen NW (2024) Association of epigenetic age and everyday discrimination with longitudinal trajectories of chronic health conditions in older adults. The Journals of Gerontology, Series A, Biological Sciences and Medical Sciences 79: glae005.

9. Stephan Y, Sutin AR, Luchetti M, Terracciano A (2021) An older subjective age is related to accelerated epigenetic aging. Psychology and Aging, 36: 767-772.

10. Ware EB, Faul JD (2021) Genomic data measures and methods: A primer for social scientists. Handbook of aging and the social sciences.49-62.

11. Ferraro KF, Shippee TP (2009) Aging and cumulative inequality: How does inequality get under the skin? Gerontologist 49: 333-343.

12. Williams DR (1999) Race, socioeconomic status, and health: The added effects of racism and discrimination. Annals of the New York Academy of Sciences 1999: 173-188.

13. Williams DR, Lawrence JA, Davis BA, Vu C (2019) Understanding how discrimination can affect health. Health Services Research 54: 1374-1388.

14. Williams DR, Mohammed SA (2013) Racism and health i: Pathways and scientific evidence. American Behavioral Scientist 57: 1152-1173.

15. Newman AB, Simonsick EM, Naydeck BL, Boudreau RM, Kritchevsky SB, et al. (2006) Association of long-distance corridor walk performance with mortality, cardiovascular disease, mobility limitation, and disability. Journal of the American Medical Association 295: 2018-2026.

16. Shaw LJ, Olson MB, Kip K, Kelsey SF, Johnson BD, et al. (2006) The value of estimated functional capacity in estimating outcome: Results from the NHBLI-sponsored women's ischemia syndrome evaluation (wise) study Journal of the American College of Cardiology 47: S36-S43.

17. Studenski S, Perera S, Patel K, Rosano C, Faulkner K, et al. (2010). Gait speed and survival in older adults. Journal of the American Medical Association, 305: 50-58.

18. Falvey JR, Bade MJ, Hogan C, Foster JE, Stevens-Lapsley JE (2020) Preoperative activities of daily living dependency is associated with higher 30-day readmission risk for older adults after total join arthroplasty. Clin Orthop Relat Res 478: 231-237.

19. Kasper R, Wahl HW, Diehl M, Zank S (2022) Subjective views of aging in very old age: Predictors of 2-year change in gains and losses. Psychol Aging 37: 503-516.

20. Layne AS, Hsu FC, Blair SN, Shyh-Huei C, Dungan J, et al. (2016) Predictors of change in physical function among older adults in response to long-term, structured, physical activity: The life study. Archives of Physical Medicine and Rehabilitation 98: 11-24.

21. Crimmins EM, Faul JD, Thyagarajan B, Weir D (2017) Venous blood collection and assay protocol in the 2016 health and retirement study 2016 Venous Blood Study (VBS) (pp. 72). Ann Arbor, MI: University of Michigan.

22. Sonnega A, Faul JD, Ofstedal MB, Langa KM, Phillips JWR, et al. (2014) Cohort profile: The health and retirement study (HRS). International Journal of Epidemiology 43: 576-585.

23. Katz S, Ford AB, Moskowitz RW, Jackson BA, Jaffe MW (1963) Studies of illness in the aged. The index of ADL: A standardized measure of biological and psychosocial function. JAMA 185: 914-919.

24. Bugliaria D, Campbell N, Chan C, Hayden O, Hurd M, et al. (2016) Rand HRS data documentation, Version P. Santa Monica, CA: RAND Center for the Study of Aging.

25. Faul JD, Kim JK, Levine ME, Thyagarajan B, Weir DR, et al. (2023) Epigenetic-based age acceleration in a representative sample of older Americans: Associations with aging-related morbidity and mortality. Proc Natl Acad Sci U S A 120.

26. Williams DR, Yan YN, Jackson JS, Anderson NB (1997) Racial differences in physical and mental health: Socio-economic status, stress and discrimination. Journal of Health Psychology 2: 335-351.

27. Zahodne LB, Kraal AZ, Sharifian N, Zaheed AB, Sol K (2019) Inflammatory mechanisms underlying the effects of everyday discrimination on age-related memory decline. Brain Behavior and Immunity 75: 149-154.

28. Carter SE, Ong ML, Simons RL, Gibbons FX, Lei MK, et al. (2019) The effect of early discrimination on accelerated aging among African Americans. Health Psychol 38: 1010-1013.

29. Geronimus AT (2001) Understanding and eliminating racial inequalities in women's health in the United States: The role of the weathering conceptual framework. J Am Med Womens Assoc (1972) 56: 133-136.

30. Simons RL, Lei MK, Klopack E, Beach SRH, Gibbons FX, et al. (2021) The effects of social adversity, discrimination, and health risk behaviors on the accelerated aging of African Americans: Further support for the weathering hypothesis. Social Science & Medicine 282.

31. Geronimus AT (1996) Black/white differences in the relationship of maternal age to birthweight: A population-based test of the weathering hypothesis. Soc Sci Med 42: 589-597.

32. Geronimus AT (2013) Deep integration: Letting the epigenome out of the bottle without losing sight of the structural origins of population health. Am J Public Health 103: 56-63.

33. Geronimus AT, Pearson JA, Linnenbringer E, Eisenberg AK, Stokes C, et al. (2020) Weathering in Detroit: Place, race, ethnicity, and poverty as conceptually fluctuating social constructs shaping variation in allostatic load. Milbank Q 98: 1171-1218.

34. Geronimus AT, Hicken MT, Pearson JA, Seashols SJ, Brown KL, et al. (2010) Do US Black women experience stress-related accelerated biological aging? Hum Nat 21: 19-38.

35. Geronimus AT, Bound J, Keene D, Hicken MT (2006) "Weathering" and age patterns of allostatic load scores among blacks and whites in the United States. Am J Public Health 96: 826-833.

36. Geronimus AT, Bound J, Keene D, Hicken MT (2007) Black-white differences in age trajectories of hypertension prevalence among adult women and men, 1999-2002. Ethn Dis 17: 40-48.

37. Linnenbringer E, Gehlert S, Geronimus AT (2017) Black-white disparities in breast cancer subtype: The intersection of socially patterned stress and genetic expression. AIMS Public Health 4: 526-556.

38. Cano M, Perez Portillo AG, Figuereo V, Rahman A, Reyes-Martínez J, et al. (2021) Experiences of ethnic discrimination among US Hispanics: Intersections of language, heritage, and discrimination setting. Int J Intercult Relat 84: 233-250.

39. Roth KB, Musci RJ, Eaton WW (2019) Heterogeneity of Latina/os' acculturative experiences in the national Latino and Asian American study: A latent profile analysis. Annals of Epidemiology 38: 48-56.e42.

40. Elliott ML, Avshalom C, Houts RM, Ambler A, Broadbent JM, et al. (2021) Disparities in the pace of biological aging among midlife adults of the same chronological age have implications for future frailty risk and policy. Nature Aging, 1: 295-308.

41. Kennedy BK, Berger SL, Brunet A, Campisi J, Cuervo AM, et al. (2014) Geroscience: Linking aging to chronic disease. Cell 159: 709-713.

42. McCartney DL, Stevenson AJ, Walker RM, Gibson J, Morris SW, et al. (2018) Investigating the relationship between DNA methylation age acceleration and risk factors for Alzheimer's disease. Alzheimer's & Dementia 10: 429-437.

43. Levine ME, Higgins-Chen A, Thrush K, Minteer C, Niimi P (2022) Clock work: Deconstructing the epigenetic clock signals in aging, disease, and reprogramming. bioRxiv.

44. Centers for Disease Control and Prevention (2024). NHANES III (1998-1994).

45. Gee GC, Hing A, Mohammed S, Tabor DC, William DR (2019) Racism and the life course: Taking time seriously. Am J Public Health 109: S43-S47.

46. McEwen BS (1998) Stress, adaptation, and disease: Allostasis and allostatic load. Annals of the New York Academy of Sciences 840: 33-44.