Which Types of Stress Are Associated With Accelerated Biological Aging? Comparing Perceived Stress, Stressful Life Events, Childhood Adversity, and Posttraumatic Stress Disorder

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ABSTRACT

Objective: Stress and stressful events are associated with poorer health; however, there are multiple ways to conceptualize and measure stress and stress responses. One physiological mechanism through which stress could result in poorer health is accelerated biological aging. This study tested which types of stress were associated with accelerated biological aging in adulthood.

Methods: Studying 955 participants from the Dunedin Longitudinal Study, we tested whether four types of stress assessed from ages 32 to 45 years—perceived stress, number of stressful life events, adverse childhood experiences, and posttraumatic stress disorder—were associated with accelerated biological aging.

Results: Higher levels of all four measures of stress were significantly associated with accelerated aging in separate models. In a combined model, more perceived stress and more stressful life events remained associated with faster aging, and the stress measures explained 6.9% of the variance in aging. The magnitudes of the associations between the four measures of stress and biological aging were comparable to associations for smoking and low education, two established risk factors for accelerated aging. People with high levels of perceived stress, numerous adverse childhood experiences (4+), high stressful life event counts, or posttraumatic stress disorder were aging an additional estimated 2.4 months, 1.1 additional months, 1.4 months, and 1.4 months per year, respectively.

Conclusions: Assessing stress, particularly perceived stress, could help identify people at risk of accelerated aging. Intervening to treat stress or the health-relevant sequelae of stress could potentially slow the rate at which people are aging, improving their health as they age.

Key words: biological aging, perceived stress, stressful life events, posttraumatic stress disorder.

INTRODUCTION

People who experience higher levels of stress are at a greater risk of poor health. Health consequences of stress include poorer immune function (1,2), a greater risk of developing chronic disease (3–6), and an increased risk of premature death (7,8). Recent empirical work has highlighted links between higher levels of psychological stress and accelerated biological aging (9,10). This work has included a wide array of outcomes, from telomere length (11,12) to systemic inflammation (1), oxidative stress (13), and epigenetic clocks (14–16). People with higher levels of psychological stress are theorized to marshal more biological resources when responding to threatening stressors. Drawing on more biological resources is thought to accelerate cellular aging (15–17), which is associated with increased risk of chronic disease and premature death (18–20). Accelerated aging could be one pathway through which stress results in poorer health (21). Intervening to treat stress is a promising avenue to improve people's health if doing so slows the rate at which people biologically age. However, future intervention studies would first require evidence as to which types of stress are most relevant to biological aging.

ACEs = adverse childhood experiences, **CI** = confidence interval, **PTSD** = posttraumatic stress disorder, **SD** = standard deviation

There are a number of ways to conceptualize and measure psychological stress (22,23), and the measurement of stress can vary widely in terms of timescale, period across the life span, and type

SDC Supplemental Digital Content

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of stress or stress response (24,25). In the context of this complexity, it remains an open question which measures of stress are most strongly associated with accelerated aging. For example, are people who feel higher levels of subjective stress or people who experience more childhood adversity or a higher number of stressful life events more likely to have accelerated rates of biological aging? Although people who experience more stressful events—whether earlier or later in the life span—might be expected to report more subjective stress, these constructs are only moderately correlated (1) and could show distinct associations with aging. Alternatively, perhaps it is the presence of mental health sequelae in response to stressful life events and trauma, such as posttraumatic stress disorder (PTSD) symptoms, that is most relevant to aging. Central characteristics of PTSD, such as hyperarousal and reexperiencing symptoms, could result in ongoing physiological strain that could promote accelerated aging. Perceived stress, stressful life events, adverse childhood experiences (ACEs), and PTSD have been shown to predict poor health in separate studies (1,4,26–29). The variation in measurement (23,24) highlights the need for studies that examine multiple constructs of stress and biological aging, ideally assessed across different periods of the life span and dimensions of stress and stress responses.

Present Study

The current study used data from 955 participants of the Dunedin Longitudinal Study (30) to examine associations between biological aging and four types of stress assessed in adulthood. At multiple occasions from ages 32 to 45 years, participants reported on their perceived stress on a checklist, described the stressful life events they experienced on a life-history calendar, reported the ACEs they experienced in childhood, and underwent a standardized diagnostic interview to determine PTSD status. Notably, each of these stress measures can be assessed at a single clinical encounter or research visit in adulthood. We tested the associations between these four types of stress and biological aging across adulthood and midlife, as assessed by the Pace of Aging (31,32). We hypothesized that each of the measures of stress would be associated with accelerated aging in midlife. We also expected that PTSD would show the strongest association, as PTSD diagnosis represents psychological sequelae of experiencing a traumatic event and establishes that stress was at a severe enough level to warrant clinical treatment.

METHODS

Participants and Study Design

Participants were members of the Dunedin Study, a longitudinal investigation of health and behavior in a birth cohort followed until age 45 years. The full cohort comprised all individuals born between April 1, 1972, and March 31, 1973, in Dunedin, New Zealand, who were eligible based on residence in the province of Otago and participation in the first assessment at 3 years of age (30). The cohort represents the full range of socioeconomic status in the general population of New Zealand's South Island. As adults, the cohort matches the results from the New Zealand National Health and Nutrition Survey on key adult health indicators (30), as well as the distribution of educational attainment among citizens of the same age from the New Zealand Census (30). The cohort self-reported as predominantly White (93%), matching South Island demographics. Assessments were performed at birth; at ages 3, 5, 7, 9, 11, 13, 15, 18, 21, 26, 32, and 38 years; and, most recently, at age 45 years (completed April 2019). The timing of study occasions and measurement are included in Figure S1, Supplemental Digital Content, http://links.lww.com/PSYMED/A924. In the cohort, 955 (92.1% of the original cohort, 95.8% of the cohort who remained alive at age 45 years) met the inclusion criteria by having their biological aging measured by the Pace of Aging and by being assessed for at least two measures of stress. Written informed consent was obtained from participants. Study protocols were approved by the Southern Health and Disability Ethics Committee at the New Zealand Ministry of Health and Duke University Health System Institutional Review Board.

Preregistration information for the study is accessible at https://sites.duke.edu/moffittcaspiprojects/files/2022/03/Bourassa_2022_PTSD-and-Biological-Aging.pdf). In short, the study covariates, inclusion and exclusion criteria, and the majority of the original primary and sensitivity analyses were preregistered. Benchmarking of effect sizes was exploratory and conducted after preregistered analyses. Analyses using childhood adversity and depression, as well as assessments of each stress measure at each study occasion, were added in response to peer review. Custom code that supports the findings of this study is available from the corresponding author on request. The Dunedin Study data sets are available on request by qualified scientists. Requests require a concept paper describing the purpose of data access, ethical approval at the applicant's university, and provision for secure data access (https://moffittcaspi.trinity.duke.edu/research-topics/dunedin). We offer secure access on the Duke, Otago, and King's College campuses.

Measures

Biological Aging

Biological aging is defined as a gradual physiological decline (20) that simultaneously involves multiple-organ systems and is progressive over the course of years (19,31). Biological aging was assessed using the Pace of Aging, a previously validated and well-established measure that assesses the rate at which people biologically age over time (31,32). The Pace of Aging was measured using repeated assessments of a panel of 19 biomarkers at ages 26, 32, 38, and 45 years (31,32). The 19 biomarkers were body mass index, waist-hip ratio, glycated hemoglobin, leptin, mean arterial pressure, cardiorespiratory fitness, forced expiratory volume in 1 second, forced expiratory volume in 1 second to forced vital capacity ratio, total cholesterol, triglycerides, high-density lipoprotein cholesterol, apolipoprotein B100/A1 ratio, lipoprotein(a), creatinine clearance, urea nitrogen, C-reactive protein, white blood cell count, periodontal disease, and caries-affected tooth surfaces. Linear change with increasing age in each biomarker for each study member was assessed using mixed modeling; the 19 resulting slopes were then summed and scaled so that 1 year of chronological age equated roughly to 1 year of average change in physiological functioning in the sample (mean [standard deviation {SD}] = 1.0 [0.3]). People with a Pace of Aging greater than 1.0 are aging faster over each given chronological year than the cohort average, and vice versa for those with values less than 1.0. Detailed description of the Pace of Aging measure can be found elsewhere (31,32).

Perceived Stress

Perceived stress was assessed using the Perceived Stress Scale (33,34), a validated 10-item measure assessing the degree to which people appraise their life as stressful. The scale in the Dunedin Study included three response options ("almost never" = 0; "sometimes" = 1; "a lot" = 2) for each item assessing the prior year. Responses at ages 32 and 45 years were averaged (this measure was not administered at age 38 years). Higher scores represented relatively higher levels of subjective stress (mean [SD] = 5.6 [3.3]).

Stressful Life Events

The number of stressful life events that participants experienced from ages 32 to 45 years was assessed using a life history calendar (35), as previously described (1,36). Participants reported events occurring between study assessments (ages 32–38 years; ages 38–45 years), which were coded to provide a total count of events. The coding evidenced strong interrater reliability: percent agreement = 92.2% of events, $\kappa = 0.91$ (1,36). The count was truncated to a maximum of 30 events and summed across the study periods. A higher count represented a relatively greater number of stressful life events (mean [SD] = 11.8 [9.0]).

Adverse Childhood Experiences

Retrospective ACEs were measured using structured interviews conducted when Dunedin Study participants were adults, largely at age 38 years. Similar to the Centers for Disease Control and Prevention ACE Study (37), the Childhood Trauma Questionnaire (38)—which assesses physical, sexual, and emotional abuse, physical neglect, and emotional neglect—was assessed at age 38 years. A category of harm was assessed as present if the Study member had a moderate to severe score in line with the Childhood Trauma Questionnaire manual and prior work in this sample (39). Participants were also interviewed about memories of exposure to family substance abuse, mental illness, and incarceration during childhood via the Family History Screen (40), as well as parental loss (due to separation, divorce, death, or removal from home). Exposure to parental partner violence was assessed by asking participants, "Did you ever see or hear about your mother/father being hit or hurt by your father/mother/stepfather/stepmother?" In line with Centers for Disease Control and Prevention study methods, ACE counts greater than 4 were truncated to 4. Across the sample, participants had an average (SD) of 1.4 (1.4) ACEs.

Posttraumatic Stress Disorder

PTSD status was assessed at ages 32, 38, and 45 years using the Diagnostic Interview Schedule (41) and diagnosed according to then current versions of *Diagnostic and Statistical Manual of Mental Disorders* (Fourth Edition) (*DSM-IV*; ages 32 and 38 years) and *DSM-5* (age 45 years). Participants were first asked whether they experienced a Criterion A trauma and were interviewed on their symptoms related to the traumatic experience that affected them most during the reporting period of since the last assessment. Diagnostic status assessed whether participants had met the criteria for PTSD as a result of this trauma and resulting symptoms. The number of occasions at which participants had met the criteria for PTSD was summed across occasions to measure PTSD diagnostic status, with higher scores representing more occasions at which the participant met the criteria for PTSD. In total, 849 participants (88.9%) did not have PTSD at any occasion, 84 (8.8%) had PTSD at one assessment occasion, and 22 (2.3%) had PTSD at multiple occasions. Three participants had PTSD at three occasions, and these scores were recoded to 2 to reduce skew.

Additional Measures

Tobacco smoking and educational attainment were included in the study as established predictors of accelerated biological aging (42–44) to benchmark the magnitudes of the associations for the measures of stress. High levels of lifetime smoking was defined as being diagnosed with tobacco dependence at two or more study occasions ($n = 247$ [25.9%]). Low educational attainment defined as having not completed the New Zealand equivalent of a high school degree by age 45 years ($n = 134$ [14.0%]).

Data Analysis

We used multiple regression models to test associations between the measures of stress and biological aging, as assessed by the Pace of Aging. First, we tested the associations between each type of stress and biological aging in four independent models. Second, we tested a multivariable model that included all four stress measures simultaneously predicting biological aging. Finally, we conducted sensitivity analyses to provide additional context to our results by further probing the nature of the association between PTSD and Pace of Aging, as well as by comparing the stress measure associations to benchmarks of smoking and educational attainment. All models were run in MPLUS version 8.3 (45) using full-maximum likelihood estimation to account for missing data and included sex as a covariate. All β values reported reflect standardized effect sizes, whereas B values reflect unstandardized values. An independent data analyst checked the analyses for reproducibility using code created from the manuscript and applied to a copy of the original data.

RESULTS

Of the 1037 original Dunedin Study members, 955 (95.8% of living cohort members, 49.2% women) met the inclusion criteria by having at least two measures of stress and a Pace of Aging score. The four measures of stress were moderately correlated, $0.17 < r$ values < 0.43, p values $< .001$. Full correlations among study variables of interest are reported in Table 1. Attrition analyses comparing the full cohort, those alive at age 45 years, and the study sample showed no differences in childhood IQ and childhood socioeconomic status among these groups, suggesting that differential attrition was not responsible for observed associations, as shown in Supplemental Figure 2, http://links.lww.com/PSYMED/A924.

Stress Measures and Accelerated Aging

All four types of stress were associated with faster biological aging. People with more perceived stress ($\beta = 0.24$, 95% confidence interval [CI], 0.18–0.30, $p < .001$), more ACEs ($\beta = 0.14$, 95% CI = 0.08–0.20, $p < .001$), and more stressful life events ($\beta = 0.19$, 95% CI = 0.13–0.25, $p < .001$), and PTSD ($\beta = 0.15$, 95% CI = 0.09–0.21, $p < .001$) were aging faster than same-age cohort peers. For each of these measures of stress, a 1-SD increase in perceived stress, ACEs, stressful life events, and PTSD corresponded to 0.8, 0.5, 0.7, and 0.5 months of accelerated aging each year, respectively, and explained 5.4%, 2.0%, 3.5%, and 2.1% of the variance in biological aging in the sample.

TABLE 1. Correlation Matrix of Study Variables

N = 955	(1)	(2)	(3)	(4)	(5)	(6)	(7)	(8)
Pace of Aging (1)	1.0							
Perceived stress (2)	0.23*	1.0						
Childhood adversity (3)	0.14*	0.29*	1.0					
Stressful life events (4)	0.19*	0.43*	0.28*	1.0				
PTSD (5)	0.15*	0.31*	0.17*	0.36*	1.0			
Smoking (6)	0.25*	0.19*	0.27*	0.24*	0.14*	1.0		
Educational attainment (7)	0.25*	0.14*	0.14*	0.12*	0.14*	0.28*	1.0	
Sex (8)	0.00	−0.13*	−0.06	−0.10*	−0.09*	0.01	0.10*	1.0

PTSD = posttraumatic stress disorder.

* $p < .05$.

In a combined model, perceived stress ($\beta = 0.17$, 95% CI = 0.10 to 0.24, $p < .001$) and stressful life event count ($\beta = 0.08$, 95% CI = 0.01 to 0.15, $p = .026$) were independently associated with faster biological aging (Table 2). ACEs ($\beta = 0.06$, 95% CI = −0.00 to 0.13, $p = .058$) and PTSD status ($\beta = 0.06$, 95% CI = −0.00 to 0.13, $p = .082$) were not significantly associated with faster aging. In total, this combined model explained 6.9% of the variance in biological aging in the sample. The results suggest that the association between perceived stress and biological aging remained the strongest compared with the associations with the other measure, particularly ACE count and PTSD.

Sensitivity Analyses

We expected that PTSD would evidence the strongest associations with biological aging, as it represented psychological consequences after experiencing a Criterion A trauma. However, our results did not reflect this expectation. We conducted a series of sensitivity analyses to provide context to these unexpected results, particularly the measurement and timing of PTSD. PTSD was assessed using a count of occasions when participants met the diagnostic criteria (a sum of three dichotomized assessments), whereas the other measures were assessed as continuous dimensions, which could have attenuated associations. Thus, we first examined the rate of biological aging among people who had a) PTSD, b) trauma exposure but no PTSD, or c) no PTSD or trauma exposure across the life span. Second, we examined our primary associations when assessing perceived stress and stressful life events using cutoffs for a high level of stress. Third, we examined associations for PTSD when using dimensional symptom counts rather than diagnostic status. We also conducted two additional analyses to provide context to our main results. First, at the request of reviewers, we assessed the association between PTSD and aging while controlling for depression. Second, we benchmarked effect sizes for the stress measures by comparing them with two well-established predictors of accelerated aging—tobacco smoking and low educational attainment.

PTSD and Trauma Exposure

The Dunedin age-45 visit included measures assessing whether participants had experienced a Criterion A trauma in their lifetime. This allowed us to categorize participants as having experienced a Criterion A trauma and developed PTSD, having experienced a Criterion A trauma without developing PTSD, or neither PTSD nor trauma exposure across the life span. People who experienced trauma and were diagnosed with PTSD were aging most quickly (1.07 biological years per chronological year, 95% CI = 1.03–1.12 years). People with trauma exposure who did not develop PTSD (1.01 biological years per chronological year, 95% CI = 0.98–1.05 years) were aging more slowly than those with PTSD, but faster than those with no trauma or PTSD (0.96 biological years per chronological year, 95% CI = 0.94–0.98 years). Figure 1 illustrates the three groups' average rates of aging.

TABLE 2. Associations Between Stress and Midlife Aging

Outcome = Pace of Aging	Unadjusted Associations		Multivariable Associations	
N = 955	β	95% CI	β	95% CI
Perceived stress	0.24**	0.18 to 0.30	0.17**	0.10 to 0.24
Stressful life events	0.19**	0.13 to 0.25	0.08*	0.01 to 0.15
ACEs	0.14**	0.08 to 0.20	0.06	−0.00 to 0.13
PTSD	0.15**	0.09 to 0.21	0.06	−0.00 to 0.13

CI = confidence interval; ACEs = adverse childhood experiences; PTSD = posttraumatic stress disorder.

Unadjusted associations show estimates for each stress measure and the Pace of Aging in separate models, whereas the multivariable associations show estimates from the model including all stress measures. All models were adjusted for sex.

* $p < .05$.

** $p < .001$.

Assessing High Perceived Stress, Childhood Adversity, and Stressful Life Events Using Cutoffs

We next used cutoffs to descriptively represent high levels of perceived stress (mean scores ≥10, n = 105 [11.0%]), ACEs (counts ≥4, n = 141 [14.8%]), and stressful life events (sum of events ≥20, n = 145 [15.2%]). In total, 637 participants (66.7% of the sample) did not have high levels of perceived stress, ACEs, stressful life event, or PTSD, whereas 196 (20.5%) of the sample had high levels of one of the types of stress, and 122 (12.8%) had multiple types of high stress. When using the dichotomized measures, higher levels of perceived stress (B = 0.20, 95% CI = 0.14–0.25, p < .001), higher levels of ACEs (B = 0.09, 95% CI = 0.04–0.14, p < .001), and higher stressful life event counts (B = 0.12, 95% CI = 0.07–0.18, p < .001) remained associated with accelerated aging. The sizes of these associations were similar to the sizes of the associations when assessing perceived stress, childhood adversity, and stressful life events continuously.

Assessing PTSD Using Symptom Counts

The Dunedin Study included symptom criterion counts for the occasions when PTSD was assessed. We tested whether the association between the count of PTSD symptoms that participants met the criteria for and aging was stronger than the association when using the sum of occasions with PTSD. The associations with aging were almost identical (symptom criteria count: β = 0.14, 95% CI = 0.08–0.21, p < .001; PTSD diagnostic status: β = 0.15, 95% CI = 0.09–0.21, p < .001), suggesting that the size of the association was not attenuated by using diagnostic status rather than symptoms criteria count.

Controlling for Depression

We next assessed the association between PTSD and biological aging while controlling for depression. We measured depression in a similar way to PTSD using a count of the assessment occasions during the study in which participants met the criteria for major depressive disorder. As shown in Supplemental Analysis 1, Supplemental Digital Content, http://links.lww.com/PSYMED/A924, PTSD remained associated with accelerated aging when controlling for depression (β = 0.12, 95% CI = 0.06–0.18, p < .001). The full results of the primary study analyses testing the association between the stress measures and aging while controlling for depression are presented in Table S1, Supplemental Digital Content, http://links.lww.com/PSYMED/A924.

Benchmarking Stress-Measure Effect Sizes

To provide additional context, we compared the associations of the four types of stress with associations for two well-established predictors of accelerated aging—high levels of lifetime smoking (B = 0.17, 95% CI = 0.13–0.21, p < .001) and low educational attainment (B = 0.22, 95% CI = 0.17–0.27, p < .001). As shown in Figure 2, the four types of stress evidenced associations with accelerated aging that were comparable in sizes to these two traditional risk factors. When looking at the Pace of Aging measure, people with high levels of perceived stress, high number of ACEs, high stressful life event counts, and PTSD were aging an estimated 2.4, 1.1, 1.4, and 1.4 additional months per year, respectively. These values were similar to those for smoking (2.0 additional months) and low educational attainment (2.6 additional months of aging). For people with high levels of perceived stress, high stressful life event counts, and PTSD, this acceleration corresponded to an estimated difference of 2.6, 1.2, 1.5, and 1.5 additional years of aging over the 13-year period covered by this study.

Contextualizing the Frequency and Timing of Stress Measurement

Our measures of stress combined several assessments during adulthood, and the timing or frequency of stress measurement might have influenced the observed pattern of associations. In this context, we assessed correlations between the stress measures at

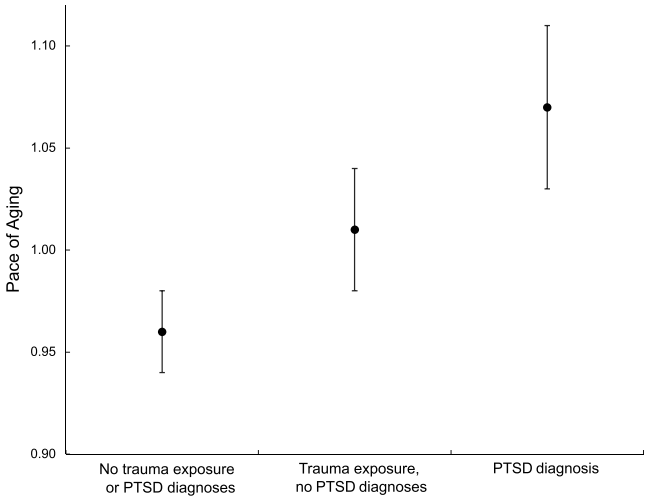

FIGURE 1. Comparing the Pace of Aging for people with no trauma exposure or PTSD diagnosis (n = 458 [49.7%]), people with trauma exposure but no PTSD (n = 290 [31.5%]), and those with trauma exposure and a diagnosis of PTSD (n = 174 [18.9%]) across the lifetime. Error bars represent 95% confidence intervals. PTSD = posttraumatic stress disorder.

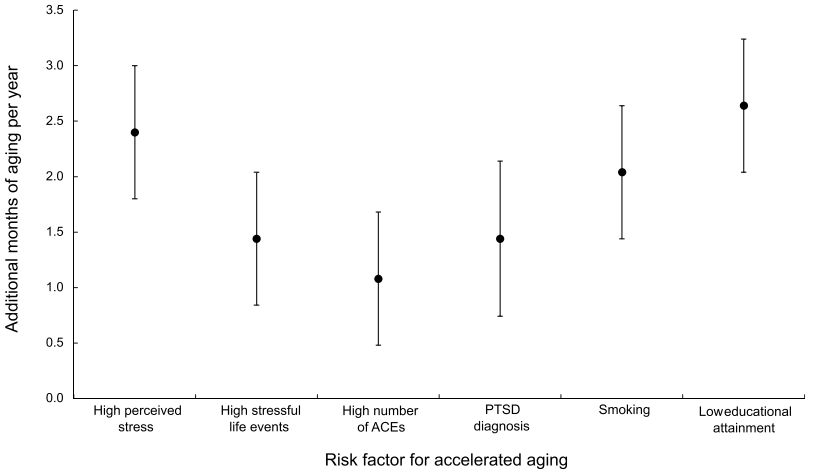

FIGURE 2. Additional months of aging per year associated with the four types of stress (high perceived stress, high number of ACEs, high stressful life events, and PTSD diagnosis) and two traditional risk factors for accelerated aging (smoking and low educational attainment). Additional months of aging are in comparison to the associated reference group (e.g., nonsmoking for smoking). Error bars represent 95% confidence intervals. ACEs = adverse childhood experiences; PTSD = posttraumatic stress disorder.

each individual assessment, as well as the association for each measure at each study occasion with biological aging. The full results are presented in Table S2, Supplemental Digital Content, http://links.lww.com/PSYMED/A924. In short, the measures of stress were moderately correlated over time and between measures. In addition, the associations with biological aging were largely consistent across the measurement occasions. Notably, each individual measure of stress at each time point was significantly associated with accelerated aging independently.

DISCUSSION

This study used participants ($N = 955$) from a longitudinal birth cohort to investigate the associations between biological aging and four types of stress—perceived stress, ACEs, stressful life events, and PTSD. We found that the people with more stress on any of the four measures were aging faster over the course of adulthood into midlife. Notably, the independent associations between the four types of stress and aging were comparable to the effect sizes for two established predictors of accelerated aging—tobacco smoking and low educational attainment. These associations remained significant for perceived stress and number of stressful life events when the stress measures were included in the same model (Table 2). Perceived stress evidenced descriptively stronger associations with accelerated aging, although the CIs of the associations for the four stress measures were overlapping and the associations for all four of the measures were attenuated. Overall, these results suggest that having higher levels of perceived stress, experiencing childhood adversity, experiencing more stressful life events, and developing PTSD could each contribute to accelerated biological aging.

We also conducted sensitivity analyses to explore the association between PTSD and accelerated aging. First, we found that assessing PTSD as a count of diagnostic statuses was not responsible for attenuating the magnitude of the association with aging. Second, when examining PTSD and trauma status independently, people with PTSD showed the fastest aging, people without PTSD or trauma exposure showed the slowest aging, and people with trauma exposure without PTSD had rates of aging between the two. This matches well with prior evidence linking trauma exposure and aging (46), as well as linking PTSD with more rapid aging and poorer health (47,48). One possible explanation is that people with PTSD might be more likely to receive mental health treatment than people with high levels of stress broadly, which could attenuate associations with accelerated aging. However, empirical evidence would be needed to determine whether participants receiving efficacious PTSD treatment might have slower biological aging. Regardless of the ultimate explanation, these results suggest that experiencing trauma and developing a mental health condition in response are associated with accelerated aging.

These findings have theoretical and clinical implications. Theoretically, the results suggest that stress is a construct that can be assessed in many ways (22,24,25), each of which might have unique relevance to health and aging. Often studies of stress and health test associations with a single type or measure of stress, such as one of the four included in this study. Our study is notable in combining four ways to conceptualize stress in the same model to examine whether associations with aging might vary for each measure. The results highlight the importance of assessing stress multiple ways (22,25) when studying mechanisms linking stress to health and aging. Different types of stress might affect outcomes via several different biopsychosocial mechanisms, which would suggest different interventions to address these pathways. For example, it is possible that perceived stress or stressful life events might accelerate aging through proinflammatory processes (1), whereas PTSD avoidance behaviors might influence aging through changes in social or health behaviors after PSTD (49). Alternatively, hyperarousal or reexperiencing symptoms observed in PTSD could contribute to poor health through changes in cardiovascular physiology (28,50). Future studies of stress and aging would benefit from investigating the biopsychosocial pathways that might explain the associations observed in this study to better untangle how different types of stress might accelerate aging.

These results also could have implications for clinical practice. First, the results suggest that there is potential in studying whether

interventions aiming to reduce the psychological impact of stress could improve health by slowing aging. All the measures of stress included in our study could be assessed during a single clinical visit—although some might be more simple and efficient to include in practice. Because each type of stress showed independent associations, presumably each kind of stress could be a potentially modifiable target to slow aging. Notably, the current study is correlational and future causal evidence of reversibility would be needed before firm conclusions could be drawn. Given the links between accelerated aging and poorer health (19,20), however, interventions that slow aging would likely improve health outcomes broadly. For example, a faster DunedinPACE—a measure of biological aging derived using methylation data from the Dunedin and applied in other cohorts—has been shown to predict functional and cognitive decline, multimorbidity, and mortality in a number of established older cohorts (51–54). This is particularly relevant given the size of the observed associations; people with higher stress were aging approximately 2 months faster per year than people with lower levels of stress. Alternatively, interventions could aim to improve the psychological or behavioral sequelae of stress that are relevant to aging (52). The results of this study suggest that measures of perceived stress could be particularly good as frontline screening tools to determine who might benefit from interventions treating stress to slow aging. For example, the Perceived Stress Scale (34) is a relatively inexpensive and quick self-report measure of stress that could be used for screening purposes, particularly given that perceived stress explained the most variance in aging outcomes independently and within the combined model. As a subjective measure, it likely captures the extent to which a person is at risk of negative psychological and physical health outcomes associated with traits, such as personality. Alternatively, existing electronic medical records that include diagnosis of PTSD could provide efficient evidence as to who might be at risk of accelerated aging as a result of traumatic stress. If different mechanisms are implicated in the health relevance of different types of stress in the future, using multiple measures of stress could guide providers in promoting interventions relevant to their patient's specific type of stress.

The results of the current study should be understood within the context of its limitations. First, the study participants are from a birth cohort representing one country and are predominantly White. The current results should be replicated in different samples, with different ancestral backgrounds, and over different historical periods to determine whether these results generalize. Second, although the life history calendar captured many types of stressful life events—including work stress and relationship dissolution—there are additional measures of specific stress (e.g., work, relational, or environmental) that could also be relevant to aging. Future studies of aging would benefit from additional measures of stress. Third, the study assessed biological aging and the four types of stress over a similar measurement period. It is possible that accelerated aging might result in higher levels of stress rather than stress leading to accelerated aging. Future studies that provide additional temporal ordering would be valuable to disentangle how these associations operate over time, particularly if they include outcomes such as the development or progression of chronic health conditions. Similarly, the measurement of the four types of stress varied in their timing, frequency, method of assessment, and scaling. Although we believe that this heterogeneity is beneficial in representing the types of measures commonly used in clinical practice and research as well, a more consistent measurement of stress across different dimensions might produce a different pattern of results from those observed here. Finally, the current study did not examine what mechanisms might explain the association between stress and accelerated aging. Future work should prioritize studying how stress might accelerate aging, which could inform interventions to slow aging and improve health.

CONCLUSIONS

In a birth cohort of 955 adults assessed in adulthood and midlife, people were more likely to show accelerated aging if they reported higher perceived stress, experienced childhood adversity in the form of ACEs, experienced more stressful life events, or developed PTSD. The sizes of these associations were comparable to effect sizes from other well-established risk factors for accelerated aging, specifically tobacco smoking and low educational attainment. The results suggest that people experiencing higher levels of stress might benefit from interventions designed to slow biological aging and improve health.

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