Health Psychology

In the public domain
ISSN: 0278-6133

2023, Vol. 42, No. 3, 172–181
https://doi.org/10.1037/hea0001276

Lifespan Psychosocial Stressors, Optimism, and Hemodynamic Acute Stress Response in a National Sample

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Objective: To understand the association between psychosocial stressors and cardiovascular health by evaluating: (a) lifespan patterns of childhood and adulthood stressors in relation to hemodynamic acute stress reactivity and recovery and (b) the role of optimism in these associations. ***Method:*** Participants (n = 1,092, 56% women, 21% racial/ethnic minority, M_{age} = 56.2) were from the Midlife in the United States Study II Biomarker Project. Lifespan profiles of psychosocial stressor exposure (low lifespan exposure, high childhood only, high adulthood only, persistent exposure) were constructed from responses to the Childhood Trauma Questionnaire and a life events inventory. Optimism was measured with the Life Orientation Test-Revised. Hemodynamic acute stress reactivity to and recovery from cognitive stressors were assessed using a standardized laboratory protocol involving continuous measurements of systolic and diastolic blood pressure (BP) and baroreflex sensitivity (BRS). ***Results:*** Compared with the low lifespan exposure group, the high childhood- and persistent-exposure groups showed lower BP reactivity, and to a lesser extent, slower BP recovery. Persistent exposure was also associated with slower BRS recovery. Optimism did not modify the association between stressor exposure and any hemodynamic acute stress responses. However, in exploratory analyses, greater stressor exposure across all developmental periods was indirectly associated with reduced BP acute stress reactivity and slower recovery via lower optimism levels. ***Conclusions:*** Findings support childhood as a unique developmental period wherein high adversity exposure may exert an enduring influence on adulthood cardiovascular health by limiting individuals' capacity to cultivate psychosocial resources and altering hemodynamic responses to acute stressors.

Keywords: childhood adversity, stress, cardiovascular reactivity, blood pressure, optimism

Supplemental materials: https://doi.org/10.1037/hea0001276.supp

Numerous studies have documented the association of early adversity with poor cardiovascular health (Hughes et al., 2017; Pierce et al., 2020), but knowledge of the underlying mechanisms driving this relationship is incomplete. Abnormal patterns of cardiovascular acute stress response have been operationalized as above- or below-average short-term change (i.e., reactivity) and slowed recovery in blood pressure (BP) or heart rate (HR) when confronting a stressor. Such responses may signal poor physiological calibration to challenges and serve as harbingers of subsequent disease development (Chida & Hamer, 2008; Chida & Steptoe, 2010). In particular, exposure to high levels of childhood adversity is linked to reduced cardiovascular acute stress reactivity (e.g., Bourassa et al., 2021; Lovallo, 2013), but its relationship with acute stress recovery is less well-investigated. Moreover, while processes by which stress "gets under the skin" and contribute to disease development are unlikely to occur during only one developmental period, studies have not yet considered the joint roles of childhood and adulthood stressors on cardiovascular acute stress response. Evaluating relations between stressor exposure and health-relevant biological processes at multiple times in the life course may provide insight into whether and how developmental

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This study was supported by the grants from the National Institutes of Health (Grants K08AG048221, RF1AG064006, UL1TR001430, and 4K00AG068431). The views expressed in this paper are those of the authors and do not necessarily represent the views of the U.S. Department of Veterans Affairs or other support institutions.

Lewina O. Lee served as lead for conceptualization, formal analysis, funding acquisition, methodology, visualization, writing–original draft, and writing–review and editing. Ruijia Chen served in a supporting role for software and writing–review and editing. Wendy Berry Mendes served in a supporting role for methodology and writing–review and editing. Laura D. Kubzansky served as lead for supervision and served in a supporting role for conceptualization, funding acquisition, methodology, writing–original draft, and writing–review and editing.

We have no known conflict of interest to disclose.

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timing (i.e., when stressor exposures occur) affects the stress-health association. As not all stressful exposures are avoidable, identifying modifiable, positive psychosocial resources that limit the effects of stress on pathophysiological processes may inform interventions targeting downstream diseases.

Meta-analytic studies have linked lower cardiovascular acute stress reactivity and slower recovery to higher levels of psychosocial stressor exposure (Chida & Hamer, 2008) and subsequent cardiovascular health (Chida & Steptoe, 2010). Growing evidence further supports an association of childhood adversity with lower acute stress reactivity as assessed by cardiovascular and cortisol markers (e.g., McLaughlin et al., 2014; Voellmin et al., 2015). In a prospective study, higher early adversity levels were associated with reduced BP acute stress reactivity at age 32, which in turn was linked to worse self-rated health and higher inflammation levels at age 45. These findings were replicated in a cross-sectional adult sample (Bourassa et al., 2021). Across studies, effect sizes of psychosocial adversity tend to be larger for BP than HR, and fewer studies consider acute stress recovery than reactivity as an outcome. Despite common references to the acute stress response as being "exaggerated" or "blunted," no clinical threshold or normative data underlie such classifications; rather, they are based on within-study comparisons of individual responses, resulting in limited cross-study generalizability. Nonetheless, individual differences in cardiovascular reactivity (and to a lesser extent, recovery) consistently predict health outcomes across diverse populations (Whittaker et al., 2021).

Closely related to BP is baroreflex sensitivity (BRS), an understudied hemodynamic marker that may be sensitive to the effects of psychosocial stressors (Lucini et al., 2005) and predicts cardiovascular health (La Rovere et al., 1998). Upon detecting BP changes, arterial baroreceptors maintain BP homeostasis by altering interbeat intervals (Gianaros et al., 2012). Baroreflex inhibition during stress may be adaptive given it seems to enhance biological readiness for the challenge, facilitate task engagement, and filter environmental distraction (Anderson et al., 2016). Initial evidence links lower resting BRS to chronic stress (Lucini et al., 2005), post-traumatic stress disorder (Hughes et al., 2007), and anxiety (Virtanen et al., 2003), but whether higher psychosocial stressor exposure influences BRS acute stress response is unknown. Given findings suggesting acute stress response in BP versus HR is more closely related to both psychosocial stressors and future cardiovascular health, and initial evidence hinting at connections between chronic stressor exposure and BRS, in this study we examined hemodynamic acute stress response (systolic [SBP] and diastolic [DBP] blood pressures, and BRS) in relation to psychosocial stressors.

Positive psychosocial resources, such as optimism, are associated with salubrious health outcomes (Kubzansky et al., 2018) and may mediate or moderate associations between psychosocial stressor exposure and health. Dispositional optimism refers to having generalized expectations for positive outcomes (Scheier & Carver, 2018). Rigorous epidemiologic evidence has prospectively linked optimism to reduced risk of cardiovascular events (Rozanski et al., 2019), even after adjusting for psychological distress. Cardiovascular acute stress response may be one mechanism underlying cardioprotective effects of optimism, but findings on associations of optimism with cardiovascular acute stress reactivity and recovery are inconclusive (e.g., Bajaj et al., 2019; Puig-Perez et al., 2015; Solberg Nes et al., 2005). A key limitation is the predominant use of small undergraduate samples, which limits the generalizability of findings to middle-aged and older populations.

Optimism may have "stress-buffering" properties by serving as a resource bank, or *reserve capacity*, that dampens the potentially harmful impact of stressors on health (Gallo & Matthews, 2003). For example, Boylan et al. (2020) reported that low childhood socioeconomic status (SES) was associated with weaker cardiovascular acute stress recovery only among individuals with lower, but not higher, optimism levels. Alternatively, early adversity may elevate risks for poor health by hindering the accrual of optimism and other health-promoting resources from a young age (Lee et al., 2019; Non et al., 2020); this suggests a mediating rather than moderating role for optimism, although they are not mutually exclusive. Optimism's role in linkages between early adversity and later cardiovascular health is largely uninvestigated.

Current Study

Allostasis theory posits abnormal patterns of physiological acute stress response as the sequelae of cumulative stressor exposure (McEwen, 1998), yet studies rarely considered if childhood and adulthood stressors synergistically influence hemodynamic acute stress response. Such synergistic effects are commonly evaluated with a cumulative scoring approach that sums total adversity exposure across one's lifetime (e.g., Lovallo et al., 2012). This approach may obscure differing effects in direction or magnitude of childhood versus adulthood stressors. The current study addressed the following questions on the developmental timing of stressor exposure in relation to hemodynamic acute stress response: Does early adversity leave a residue above and beyond the more proximal influence of adulthood stressors? Does early adversity sensitize individuals resulting in stronger effects of adulthood stressors (Hammen, 2005)? Alternatively, are the effects of early adversity and adulthood stressors additive?

Using data from a national adult sample, we evaluated lifespan patterns of childhood and adulthood stressors in relation to hemodynamic acute stress reactivity and recovery, and the role of optimism in the associations. We hypothesized greater childhood adversity exposure would be independently associated with lower hemodynamic acute stress reactivity and slower recovery, beyond any hemodynamic effects of adult stressors. We expected stress-buffering by optimism, with greater attenuation of the association of lifespan stressor exposure with acute stress response among more versus less optimistic adults. Given evidence suggesting stressor exposure can erode accrual of psychosocial resources, we also evaluated the indirect association from life course stressor exposure via optimism to acute stress response.

Our study used data from the same parent study described in two recent reports examining related questions. Bourassa et al. (2021) considered linkages among early adversity (abuse, neglect, SES), BP and HR acute stress reactivity, and health-related outcomes. Keogh et al. (2022) tested depression as a mediator of the association between childhood abuse and neglect and cardiovascular (BP and HR) acute stress reactivity. Our study extends prior work by: (a) broadening the conceptualization of childhood stressors to include negative life events beyond abuse, neglect, and SES; (b) testing synergistic effects of childhood and adulthood stressor exposure; (c) considering acute stress recovery as well as reactivity; (d) adding BRS as a novel marker of acute stress response; and (e) testing the role of optimism in the association between lifespan stressor exposure and adulthood acute stress response.

Method

Design and Sample

Data are from the Midlife in the United States (MIDUS) study, a national longitudinal study on psychosocial and biological factors accounting for age-related variations in health and well-being. For Wave I (1995–1996), eligible participants were non-institutionalized, English-speaking adults aged 25–74 ($n = 7{,}108$; including 950 siblings and 957 twin pairs). MIDUS II (2004–2006) was a follow-up study which included 81% of MIDUS I participants (Radler & Ryff, 2010). To include more Black participants, MIDUS II recruited a new sample of 592 Black adults from Milwaukee. All MIDUS II participants responded to a phone (main MIDUS II) or in-person (Milwaukee) interview, and a survey. Survey participants were eligible for the Biomarker Project if they were able to travel to the study clinic. The Biomarker Project entailed a 2-day clinic visit including a laboratory-based psychophysiology protocol, biomarker data collection, medical history interview, and additional psychosocial questionnaires ($n = 1{,}255$; Dienberg Love et al., 2010; see Document S1 in the online supplemental materials for more details). The current study used data from the overall MIDUS II interview and survey and Biomarker Project. Our analytic sample included 1,092 individuals (262 siblings or twins) with data on childhood trauma, childhood and adulthood negative life events, optimism, and at least one acute stress response variable. The current study qualified for an exemption determination under the Boston University Medical Campus Institutional Review Board due to the use of a limited dataset under the HIPAA Privacy Rule.

Psychophysiology Protocol

Acute physiological stress response was assessed with a standard laboratory protocol (see Document S1 in the online supplemental materials). Psychophysiological parameters were recorded while participants performed two validated cognitive stressor tasks: the Stroop Color-Word task and a mental arithmetic task. Task order was counter-balanced across participants.

Continuous SBP and DBP were recorded using a Finometer (Finapres Medical Systems, Amsterdam, Netherlands; Schutte et al., 2003). SBP and DBP were assessed as the mean value in each protocol period (i.e., baseline, stressor #1, recovery #1, stressor #2, recovery #2). BRS was calculated with the sequence method approach (Voss et al., 1999), which derives a mean beta for each sequence, defined as a series of 3+ consecutively increasing or decreasing SBP values. BRS in each period was the average of mean betas from usable sequences. Higher BRS scores represent greater baroreceptor feedback on HR.

Measures

Childhood Trauma

Childhood trauma was assessed at the Biomarker Project with the Childhood Trauma Questionnaire (CTQ; Bernstein & Fink, 1998), which has demonstrated convergent validity with clinician ratings of abuse and good reliability (see Document S1 in the online supplemental materials). CTQ domains include physical abuse, emotional abuse, sexual abuse, physical neglect, and emotional neglect. For each 5-item subscale, we handled missing values with mean substitution if ≥4 items were completed. Following cutoffs in the CTQ manual (Bernstein & Fink, 1998), we first dichotomized each subscale score into none/low versus moderate/severe categories and then computed a childhood trauma count score representing the number of subscales scoring in the moderate/severe range (possible range: 0–5). Missing data was minimal (<1%).

Childhood and Adulthood Negative Life Events

Negative life events were assessed in the MIDUS II survey with a checklist that included seven childhood-specific events and 20 events that could happen at any age. Respondents were asked to check items they experienced, report the age(s) of occurrence, and rate the long-term impact of the event from −2 (*very negatively*) to +2 (*very positively*). *Negative* life events were defined as events with long-term impact <0. For each of childhood (cNLEs) and adulthood negative life events (aNLEs), we computed a count score if participants had completed ≥50% items; <3% of the sample had incomplete data on 2+ cNLE or 3+ aNLE items. Four childhood items with CTQ content overlap were excluded from the cNLE count score, which represented the number of negative life events occurring prior to age 18 (range: 0–12; see Table S1 in the online supplemental materials). The aNLE count score represented the number of negative life events occurring after age 18 (range: 0–20).

Lifespan Stressor Exposure

Following prior conceptual and empirical work (Power et al., 2013; Winning et al., 2015), we constructed four mutually exclusive profiles of lifespan stressor exposure by combining information on high versus low exposure to early adversity and aNLEs. We operationalized early adversity using the measure of childhood traumas and of cNLEs. High (vs. low) early adversity exposure was defined as having moderate/severe scores on ≥2 CTQ subscales and/or having ≥2 cNLEs. High (vs. low) exposure to adult stressors was operationalized as having ≥2 events on the measure of aNLEs. We then categorized participants into "low lifespan (i.e., childhood and adulthood) exposure," "high childhood exposure," "high adulthood exposure," and "persistent (i.e., high childhood and adulthood) exposure." In supplemental analyses, we operationalized the stressor exposure as continuous variables. To derive a continuous early adversity score (score range: 0–17), we summed the number of CTQ subscales with moderate/severe scores and cNLE count score. We operationalized continuous adult stressor exposure as the aNLE count score.

Optimism

Dispositional optimism was assessed in the MIDUS II survey with the 6-item Life Orientation Test-Revised which has good validity and reliability (LOT-R; Scheier et al., 1994). Internal consistency was high in this sample (Cronbach's $\alpha = .80$). Following prior psychometric recommendations (Segerstrom et al., 2011), we computed an overall optimism score (range: 0–24) using both negatively and positively framed items. We first reverse-coded negatively framed items (e.g., "I hardly expect things to go my way"), then summed all item scores, and converted the total score into a z-score metric, such that each additional unit corresponds to 1 standard deviation (*SD*) higher in optimism level. Following the MIDUS II convention, we handled missing values with mean substitution if ≥3 items were

completed. After mean substitution, 2% of the Biomarker Project participants had missing optimism data.

Hemodynamic Acute Stress Response

Hemodynamic acute stress response was operationalized as reactivity and recovery scores for SBP, DBP, and BRS from the psychophysiology protocol. A healthy pattern of acute stress response is generally characterized by a moderate increase in BP from baseline to stressor presentation, followed by a BP decrease to baseline levels during the recovery period. For BRS, a healthy response involves a decline from baseline to stressor due to stress-related BRS inhibition, followed by an increase during the recovery period signaling an end of the inhibition response.

Following standard practice (e.g., Bibbey et al., 2013; Gianaros et al., 2012) and given no baseline differences in the physiological parameters by stressor exposure categories, we calculated a reactivity score for each physiological parameter in each stressor task by subtracting the parameter value obtained at baseline from the value obtained in the stressor task, and averaging reactivity scores across stressor tasks. Positive reactivity scores represent an increase in the parameter from baseline to the cognitive stressor task periods. Recovery scores for SBP and DBP were calculated by subtracting BP during recovery from BP obtained in the stressor task, and then averaging recovery scores across stressor tasks. Positive values represent a decrease in BP from stressor to recovery periods. To facilitate interpretation, we calculated the BRS recovery score by subtracting BRS in each stressor period from BRS obtained in the recovery period and then averaging the two scores. Positive BRS recovery scores represent a BRS increase from stressor to recovery, consistent with termination of baroreflex inhibition. In the Biomarker Project, 71% of participants had data on all six hemodynamic acute stress response variables, 12% had data on 4–5 variables, 7% had data on 1–3 variables, and 11% had no data on these variables.

Covariates

All covariates were self-reported and assessed during the MIDUS II interview or survey unless otherwise indicated. They were selected based on their role as potential confounders in the exposure-outcome relations. Covariates included age at the Biomarker Project visit, sex (1 = *female*, 0 = *male*), race (White, Black and/or African American, Native American/Alaskan natives/Asians/native Hawaiian or Pacific Islanders/others), highest education level (0 = *no school to some high school*, 1 = *GED or high school graduate*, 2 = *some college*, 3 = *college graduate or equivalent*, 4 = *postgraduate education*), current use of BP-altering medications at the Biomarker Project (yes/no; see Document S1 in the online supplemental materials), and lifetime chronic diseases (count score based on yes/no response to 13 conditions, top-coded at 5 due to skewness). Following prior work in this sample (Keogh et al., 2022), depression status was assessed at the Biomarker Project with the depressive affect subscale of the Center for Epidemiologic Studies Depression Scale (CES-D; Radloff, 1977). Our manipulation check for the psychophysiology protocol considered participant stress ratings "at the moment" on a scale from 1 (not at all) to 10 (extremely) during each protocol period.

Analytic Strategy

We examined distributions of demographics, health variables, and optimism by lifespan stressor exposure profiles using one-way analysis of variance and χ^2 tests. To test hypotheses regarding associations of lifespan stressor exposure with hemodynamic acute stress response, we used ordinary least squares (OLS) regression with acute stress reactivity and recovery in SBP, DBP, and BRS as outcomes in separate models. We entered the main effects of lifespan stressor exposure categories while considering two levels of covariate of adjustment: Model 1 adjusted for demographics (age, sex, education, and race). Model 2 added chronic diseases and BP-altering medications as potential health confounders. To test the stress-buffering hypothesis, we added the main effect of optimism (Model 3) and the interaction between optimism and each lifespan stressor exposure profile (Model 4).

Exploratory analyses evaluated the role of optimism in the associations between life course stressor exposure and acute stress response. For each acute stress response outcome, we specified a path model comprising the direct effects of each lifespan stressor exposure profile on the outcome and all indirect effects via optimism. Paths predicting optimism adjusted for demographics and chronic diseases; paths predicting acute stress response variables additionally adjusted for BP-altering medications. Each indirect effect was computed as the product of its component paths (Hayes, 2017). Models used maximum likelihood estimation and missing covariate data were handled using multiple imputations with 30 imputed datasets.

We conducted five sets of sensitivity analyses. First, we compared stress ratings during the cognitive stressors to those during the baseline and recovery periods as a manipulation check. Second, we examined intraclass correlations (ICCs) of the acute stress response for evidence of clustering due to twin or sibling status, as defined by ICC $>$ .05. ICCs were 37.4%, 40.3%, 24.5%, 13.2%, $<$1%, and $<$1% for reactivity and recovery in SBP, DBP, and BRS, respectively. To account for familial clustering in SBP and DBP outcomes, we re-estimated the main effects and interaction of lifespan stressor exposure and optimism using multilevel regression (which included family-level intercepts) with multiple imputations. Because the multilevel regression results (Table S2 in the online supplemental materials) were highly similar to those from main analyses, we report findings without adjustment in the Results section for simplicity. Third, because lifespan stressor exposure was operationalized using binary variables for childhood trauma, cNLEs and aNLEs, and specific cutpoints have not been validated, we ran OLS regression using continuous stressor variables to test the total versus unique effects of childhood and adulthood stressor exposure (Models A–C), and to evaluate the additive (Model D) versus multiplicative effects (Model E) of childhood and adulthood stressors. Fourth, in light of ongoing debate regarding the dimensionality of dispositional optimism and evidence suggesting a stronger association of pessimism than optimism with physical health outcomes (Scheier et al., 2021), we re-estimated the main effects of lifespan stressor exposure and optimism by replacing the LOT-R total score with optimism and pessimism subscale scores (Cronbach's αs: .70 and .80, respectively). Fifth, given Keogh et al.'s (2022) findings suggesting depressive affect mediated the association between early adversity and reduced cardiovascular reactivity (Keogh et al., 2022), we re-estimated the indirect effects via optimism in the

path models while further adjusting for an indirect path via depressive affect.

Transparency and Openness

As this study involved secondary analyses of existing data, sample size was pre-determined by data availability. We reported all data exclusions and manipulations. Analyses were conducted with SAS Version 9.4 and Mplus Version 8.5; analytic code and output are available upon request. This study was not pre-registered; however, data and measures used are maintained at the National Archive of Computerized Data on Aging website.

Results

Descriptive Statistics

The sample included 56% women, 21% racial/ethnic minority, and was on average 56.2 years old ($SD = 11.3$) with some college education. There were notable differences in demographics, health, and optimism by lifespan stressor exposure categories (Table 1). For example, individuals with persistent stressor exposure were more likely to be women, Black, have the lowest education attainment, and highest number of chronic diseases. Optimism levels were highest in the low lifespan stressor exposure group and lowest in the persistent exposure group. Manipulation check indicated stress ratings were higher during both stressor tasks relative to baseline and recovery, as expected (Document S2 in the online supplemental materials).

Lifespan Stressor Exposure and Hemodynamic Acute Stress Response

Our hypothesis that greater childhood adversity exposure would be associated with weaker hemodynamic acute stress reactivity and slower recovery was partially supported (Table 2 and Figure 1). Relative to individuals with low lifespan stressor exposure, those with high childhood stressor exposure alone had 2.36 mmHg, 95% CI [−4.56, −0.15] less SBP rise from baseline to stressor. Those with persistent stressor exposure had 3.72 mmHg [−6.50, −0.94] less SBP rise, adjusted for demographics and health confounders. Having high adulthood stressor exposure alone was unrelated to SBP reactivity (Model 2). For SBP recovery, relative to low lifespan exposure, persistent stressor exposure was associated with 2.95 mmHg [−5.40, −0.50] less SBP decline from stressor to recovery and childhood stressor exposure alone was weakly related to 1.87 mmHg [−3.84, 0.11] less SBP decline. For reference, each additional decade of age was associated with 2.62 mmHg [1.87, 3.36] and 0.78 mmHg [0.49, 1.06] greater SBP and DBP rise, respectively, in the above models.

A similar pattern was observed for DBP reactivity. Both high childhood exposure and persistent exposure groups showed attenuated DBP reactivity relative to the low lifespan exposure group. High childhood exposure alone was also linked to slower DBP recovery, with a similar and marginally significant association observed for persistent stressor exposure.

No stressor variables were associated with BRS reactivity. Persistent stressor exposure was associated with slower BRS recovery (0.43 ms, 95% CI [−0.80, −0.07] less IBI change per SBP unit change from the stressor tasks to the recovery periods) relative to the low lifespan exposure group, adjusted for demographics and health confounders.

Role of Optimism

We found no evidence for stress-buffering by optimism. Higher optimism was associated with higher SBP (Table 2, Model 3: $B = 1.37$, 95% CI [0.61, 2.14]) and DBP ($B = 0.57$, [0.28, 0.86]) reactivity, and with faster SBP ($B = 0.99$, [0.30, 1.68]) and DBP ($B = 0.51$, [0.26, 0.75]) recovery. Optimism was unrelated to any BRS acute stress response. Notably, after adding the main effect of optimism, associations of high childhood exposure alone and persistent exposure with acute stress reactivity and recovery were attenuated by 12%–46%. No interaction term between optimism and the stressor

Table 1
Sample Characteristics by Exposure to Childhood and Adulthood Stressors, N = *1,092*

M (*SD*) unless otherwise noted	Low lifespan exposure $n = 682$	High childhood exposure only $n = 147$	High adulthood exposure only $n = 177$	Persistent exposure $n = 86$	χ^2/F ($df = 3$)	*p*
Age	56.71 (11.79)	53.93 (10.83)	56.42 (10.47)	55.35 (8.93)	2.66	.047
Female, *n* (%)	351 (51.5)	94 (64.0)	106 (59.9)	63 (73.3)	20.93	.0001
Education (0–4)	2.48 (1.16)	2.07 (1.21)	2.12 (1.08)	1.90 (1.35)	11.84	<.0001
Race, *n* (%)						
White	585 (85.8)	109 (74.7)	118 (66.7)	51 (59.3)	57.08	<.0001
Black	79 (11.6)	29 (19.9)	51 (28.8)	30 (34.9)	51.18	<.0001
Other[a]	18 (2.6)	8 (5.5)	8 (4.5)	5 (5.8)	4.98	.17
Number of chronic diseases (0–10)	1.41 (1.28)	2.01 (1.58)	1.90 (1.52)	2.13 (1.56)	15.57	<.0001
Blood pressure-altering medications use: *N* (%)	183 (29.9)	46 (34.1)	74 (45.7)	32 (40.5)	16.06	.001
Optimism (1–24)	18.44 (4.35)	16.06 (5.04)	17.08 (4.88)	14.34 (5.62)	27.63	<.0001

Note. % represents the percent of participants having a particular characteristic (e.g., being female) within a stressor exposure group.
[a]"Other" racial category includes Native American or Alaskan natives, Asians, native Hawaiian or Pacific Islanders, and others. Group differences were evaluated using one-way analysis of variance for continuous variables and using χ^2 tests for categorical variables. Childhood stressor exposure was classified as high based on having 2+ Childhood Trauma Questionnaire subscales in the moderate or severe range and/or having 2+ childhood negative life events. Adulthood stressor exposure was classified as high based on having 2+ adulthood negative life events. Education was coded as 0 = *no school to some high school*, 1 = *GED or high school graduate*, 2 = *some college*, 3 = *college graduate or equivalent*, 4 = *postgraduate education*.

Table 2
Association of Lifespan Psychosocial Stressor Exposure and Optimism With Hemodynamic Acute Stress Reactivity and Recovery

	Model 1 demographics		Model 2+ health		Model 3+ optimism main effect	
	B	95% CI	*B*	95% CI	*B*	95% CI
			DV: SBP reactivity ($n = 921$)			
Low lifespan exposure	Ref.	—	Ref.	—	Ref.	—
High childhood exposure	**−2.52**	**[−4.71, −0.33]**	**−2.36**	**[−4.56, −0.15]**	*−1.87*	*[−4.08, 0.34]*
High adulthood exposure	−0.39	[−2.47, 1.69]	−0.29	[−2.38, 1.80]	0.04	[−2.05, 2.12]
Persistent exposure	**−3.87**	**[−6.64, −1.10]**	**−3.72**	**[−6.50, −0.94]**	*−2.70*	*[−5.52, 0.12]*
Optimism (*z*-score)	—	—	—	—	**1.37**	**[0.61, 2.14]**
			DV: SBP recovery ($n = 989$)			
Low lifespan exposure	Ref.	—	Ref.	—	Ref.	—
High childhood exposure	*−1.90*	*[−3.86, 0.06]*	*−1.87*	*[−3.84, 0.11]*	−1.47	[−3.46, 0.51]
High adulthood exposure	0.08	[−1.76, 1.92]	0.07	[−1.78, 1.93]	0.29	[−1.56, 2.14]
Persistent exposure	**−2.98**	**[−5.41, −0.54]**	**−2.95**	**[−5.40, −0.50]**	*−2.21*	*[−4.71, 0.28]*
Optimism (*z*-score)	—	—	—	—	**0.99**	**[0.30, 1.68]**
			DV: DBP reactivity ($n = 921$)			
Low lifespan exposure	Ref.	—	Ref.	—	Ref.	—
High childhood exposure	**−1.17**	**[−2.00, −0.34]**	**−1.12**	**[−1.96, −0.29]**	**−0.92**	**[−1.75, −0.09]**
High adulthood exposure	−0.33	[−1.12, 0.46]	−0.30	[−1.09, 0.49]	−0.16	[−0.95, 0.62]
Persistent exposure	**−1.11**	**[−2.16, −0.07]**	**−1.07**	**[−2.12, −0.02]**	−0.65	[−1.72, 0.42]
Optimism (*z*-score)	—	—	—	—	**0.57**	**[0.28, 0.86]**
			DV: DBP recovery ($n = 989$)			
Low lifespan exposure	Ref.	—	Ref.	—	Ref.	—
High childhood exposure	**−0.85**	**[−1.55, −0.16]**	**−0.84**	**[−1.54, −0.14]**	*−0.64*	*[−1.34, 0.07]*
High adulthood exposure	−0.11	[−0.76, 0.54]	−0.12	[−0.78, 0.53]	−0.01	[−0.67, 0.64]
Persistent exposure	*−0.83*	*[−1.70, 0.03]*	*−0.82*	*[−1.69, 0.05]*	−0.44	[−1.33, 0.44]
Optimism (*z*-score)	—	—	—	—	**0.51**	**[0.26, 0.75]**
			DV: BRS reactivity ($n = 1{,}054$)			
Low lifespan exposure	Ref.	—	Ref.	—	Ref.	—
High childhood exposure	0.10	[−0.21, 0.41]	0.12	[−0.19, 0.43]	0.10	[−0.21, 0.41]
High adulthood exposure	0.16	[−0.13, 0.44]	0.17	[−0.12, 0.46]	0.16	[−0.13, 0.45]
Persistent exposure	0.26	[−0.13, 0.66]	0.29	[−0.10, 0.69]	0.26	[−0.15, 0.66]
Optimism (*z*-score)	—	—	—	—	−0.05	[−0.16, 0.06]
			DV: BRS recovery ($n = 1{,}045$)			
Low lifespan exposure	Ref.	—	Ref.	—	Ref.	—
High childhood exposure	−0.02	[−0.31, 0.27]	−0.03	[−0.32, 0.27]	0.00	[−0.29, 0.30]
High adulthood exposure	−0.20	[−0.47, 0.07]	−0.21	[−0.48, 0.07]	−0.19	[−0.46, 0.08]
Persistent exposure	**−0.42**	**[−0.79, −0.06]**	**−0.43**	**[−0.80, −0.07]**	**−0.38**	**[−0.75, −0.01]**
Optimism (*z*-score)	—	—	—	—	0.07	[−0.03, 0.17]

Note. DV = dependent variable; CI = confidence interval; SBP = systolic blood pressure; DBP = diastolic blood pressure; BRS = baroreflex sensitivity. Bold indicates $p < .05$. Italics indicates $.05 \leq p < .10$. Model 1 adjusted for age, sex, education, and race. Models 2 and 3 additionally adjusted for number of diseases and use of medications affecting blood pressure. The sizes of the stressor exposure groups were: low lifespan exposure, $n = 682$; high childhood exposure, $n = 147$; high adulthood exposure, $n = 177$; and persistent exposure, $n = 86$.

variables was statistically significant (Table S3 in the online supplemental materials).

In exploratory analyses, each stressor exposure category was indirectly associated with reduced SBP and DBP reactivity and slower recovery through lower optimism levels (Table S4 in the online supplemental materials). We observed a dose-response association between stressor exposure and optimism: High levels of childhood exposure alone, adulthood exposure alone, and persistent exposure were associated, respectively, with 0.39, 95% CI [−0.56, −0.23]; 0.23 [−0.39, −0.07]; and 0.77 [−0.98, −0.55] *SD* lower on optimism. For reference, each unit higher on education was associated with 0.16 [0.11, 0.20] *SD* lower on optimism. In turn, lower optimism was associated with reduced BP reactivity and slower recovery. The indirect effects of each stressor variable through optimism to SBP and DBP reactivity and recovery were significant. For example, persistent stressor exposure was associated with 1.06 mmHg lower [0.62, 2.14] SBP acute stress reactivity due to its association with lower optimism. Indirect effects to BRS acute stress response were not statistically significant. In sensitivity analyses that added an indirect path through depressive affect (Table S5 in the online supplemental materials), indirect effects of each stressor variable through optimism to SBP reactivity and DBP reactive and recovery were somewhat attenuated but remained significant. Indirect effects of each stressor variable through optimism to SBP recovery became nonsignificant, likely due to a nonsignificant path from optimism to SBP recovery.

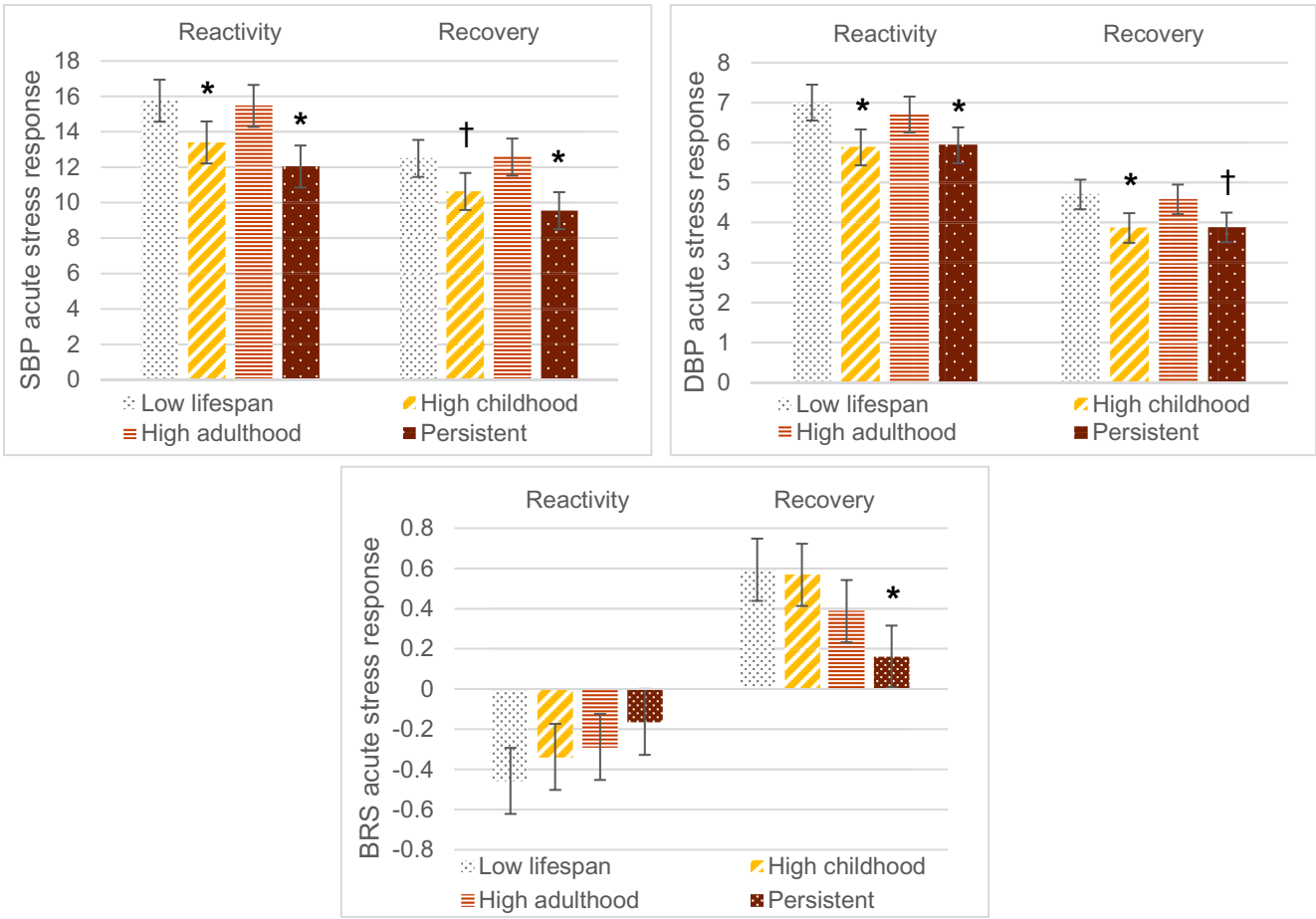
The overall pattern of findings for the main effect of optimism on acute stress response was similar when considering separate associations with the optimism and pessimism subscale scores (Table S6 in the online supplemental materials). For SBP and DBP reactivity, effect sizes of optimism and pessimism were similar. For SBP and DBP recovery, effect sizes were larger for pessimism than optimism but all effects were in the expected directions.

Sensitivity Analyses Using Continuous Stressor Scores

When stressor exposure was modeled as continuous variables (Table S7 in the online supplemental materials), higher levels of

Figure 1

Predicted Hemodynamic Acute Stress Response by Stressor Exposure Category, Adjusted for Demographics and Health Confounders



Note. Asterisks (*) and obelisks (†) represent differences from the low lifespan stressor exposure category (reference group) at $p < .05$ and $.05 \leq p < .10$, respectively. Values were predicted for White male participants at the sample mean values of age, education, number of chronic diseases, and without use of blood pressure-altering medications. See the online article for the color version of this figure.

childhood stressor exposure were associated with lower reactivity and slower recovery in SBP and DBP, but unrelated to BRS acute stress response (Model A). Adulthood stressor exposure alone was unrelated to any acute stress response variable (Model B). This pattern of findings was unchanged when both stressor variables were entered simultaneously (Model C). The additive effect of childhood and adulthood stressor exposures was linked to lower reactivity and slower recovery in both SBP and DBP (Model D). The interaction of child and adult stressor exposures was not statistically significant for all outcomes (Model E).

Discussion

In a large, national sample of middle-aged and older adults, high exposure to psychosocial stressors in childhood was associated with lower BP reactivity and slower recovery from acute stressors, above and beyond the influence of adulthood stressors. Irrespective of childhood adversity exposure, associations of adulthood stressor exposure with BP acute stress response were not evident. Our study provides initial evidence linking persistent stressor exposure across childhood and adulthood to slower BRS recovery. While we found little support for a stress-buffering role of optimism, in exploratory analyses greater lifespan stressor exposure was indirectly linked to lower BP reactivity and slower BP recovery through lower optimism levels. Our findings contribute knowledge on the developmental timing of stressor exposure and on the role of optimism in the association between early adversity and hemodynamic acute stress response.

High childhood exposure alone and persistent stressor exposure were both associated with lower reactivity and slower recovery in BP. For SBP reactivity and recovery, effect sizes of persistent stressor exposure versus high childhood exposure were 58% higher, suggesting the combined impact of adversity across childhood and adulthood may be stronger than childhood exposure alone. Results using continuous stressor variables did not support a multiplicative effect of childhood and adulthood stressor exposures, thus there was little evidence for stress sensitization. The magnitude of associations between adulthood stressor exposure and BP acute stress response were attenuated

by more than half when childhood stressors were included, whereas childhood exposure effect sizes were robust to adjustment for adulthood stressors. These findings generally support the idea that childhood may be a developmental period wherein exposures have more potent effects than those of adulthood exposures (Power et al., 2013), perhaps via alterations in critical biological processes involved in long-term stress response differences that culminate in elevated chronic disease risks (Hertzman, 2012). However, the broad age range (0–18) of our early adversity measures precluded formal tests of exposure effects during more focused developmental periods. Our findings also parallel earlier findings that childhood psychological distress is uniquely associated with midlife cardiometabolic risk, even if such distress remitted by adulthood (Winning et al., 2015). The stronger association of the stressor variables with systolic compared with diastolic BP acute stress response aligns with findings that SBP is a stronger determinant of adverse cardiovascular outcomes than BP (Flint et al., 2019). While the effect sizes of the stressor variables on BP reactivity and recovery are small, they are comparable or somewhat higher than those reported for life events in a meta-analysis (Chida & Hamer, 2008).

Another explanation for the differential association of childhood versus adulthood stressors with BP acute stress response is that adverse childhood events are more severe than adulthood stressors. This could be due to methodological differences in item content (e.g., assessing abuse only in childhood) or to true severity differences in circumstances. Childhood stressors, particularly maltreatment and the conditions under which they occur, could be more enduring than some of the more episodic adulthood stressors included in the life events checklist. Moreover, children who experience multiple psychosocial stressors are likely to grow up in environments plagued by other stressors like poverty and physical risks (Evans & English, 2002) and encounter more stressors as adults (Lee et al., 2019). Thus, the "high childhood exposure" and "persistent exposure" groups may be more similar in their developmental histories than to the "high adult exposure" group.

For BRS acute stress response, we observed slower recovery in individuals with persistent stressor exposure, suggesting both distal and proximal stressors may be important for its recovery. Given stressor exposure was unrelated to BRS reactivity, acute stress reactivity and recovery may not relate to exposures in a synchronized manner and their underlying mechanisms may not entirely overlap. The overall pattern in which lifespan stressors related to BRS versus BP acute stress response differed; BRS may be more tightly regulated than BP and therefore less susceptible to external influences. Given scarce research on psychosocial correlates of BRS acute stress response, we consider the current findings preliminary.

Early adversity may contribute to below-average hemodynamic acute stress response via neural mechanisms underlying emotion and motivational processes. Early adversity has been linked to global brain structural variation, and dysregulation in neuroendocrine stress response systems and neural circuits underlying fear learning and emotion regulation (Gehred et al., 2021; McLaughlin et al., 2019), which may explain poorly calibrated responses to challenges. Ginty (2013) proposed blunted cardiovascular and cortisol acute stress responses may signal a general "biological disengagement from life challenges" involving inadequate processing of psychologically stressful stimuli, ineffective coordination of motivated behaviors, diminished response to rewards, and dysregulated reward-seeking behaviors. These deficits are also overrepresented among individuals with early adversity (Hughes et al., 2017). Alternatively, some researchers have wondered if reduced acute stress response may confer short-term cognitive and emotional benefits in the immediate aftermath of stressors (Phillips et al., 2013). Others suggest depression (Keogh et al., 2022) and cognitive function (Bourassa et al., 2021) may be intermediaries linking early adversity with adult stress physiology, but evidence for associations of stressor exposure, neuropsychiatric processes, stress physiology, and health outcomes is often cross-sectional, which may yield biased estimates of longitudinal processes. Research clarifying directionality among these complex developmental processes is needed.

Our results do not suggest a stress-buffering effect of optimism on adult hemodynamic acute stress response, but in path analyses lower optimism at least partially accounted for associations of lifespan stressor exposure with diminished SBP and DBP reactivity and slower recovery. We also observed a main effect of higher optimism on higher BP reactivity and faster recovery, suggesting optimism is associated with a more flexible hemodynamic system that engages with and bounces back from environmental demands. Findings on optimism were largely maintained after adjustment for depression, suggesting the observed associations were not due primarily to a lack of negative affect. Failure to detect an optimism-by-stressor exposure interaction could be due to having few individuals who developed high optimism despite having high stressor exposure. Of participants in the highest optimism quartile, 74% had low lifespan stressor exposure, compared with 4% who had high lifespan stressor exposure. Perhaps the physiological benefits of optimism were not limited to individuals with high stressor exposure levels. The dose-response association between stressor exposure and optimism suggests that stressful life circumstances may limit opportunities to cultivate confidence in one's ability to overcome challenges and achieve positive outcomes over the life span (Lee et al., 2019).

Several limitations should be considered. First, our data were cross-sectional and reverse causality is possible. Second, our measures of stressor exposure were retrospective and can be susceptible to the influence of later-life circumstances and potential bias by unreliable or inaccurate recall, or current mood. However, a comparison study suggested that associations between childhood exposures and midlife outcomes were highly similar between prospective birth cohort data and retrospective life history data (Jivraj et al., 2020). Third, as our acute stressor tasks were laboratory-based and represent situations requiring a high cognitive load, they may have limited ecological validity and generalizability to other psychologically stressful situations. Individual differences in task engagement, for which we do not have a measure, may influence acute stress response. Fourth, we did not distinguish between stressor type or severity. However, compared with prior studies using MIDUS II data (Bourassa et al., 2021; Keogh et al., 2022), we augmented our early adversity measure with negative life events to increase content coverage and representativeness, and observed the same pattern of findings for BP outcomes. Moreover, Keogh et al. (2022) did not observe differential associations of childhood trauma subtypes with BP acute stress reactivity. Finally, our study did not include a replication sample, thus findings are not yet conclusive. We hope these findings motivate additional research that replicates and extends them, by leveraging existing data and materials or by designing studies to address the research questions directly.

In summary, the current study informs theory on the developmental timing of stressor exposure in relation to hemodynamic acute

stress response. We offer novel evidence on the sensitivity of the baroreflex mechanism to background and acute stressors. Interventions that promote psychosocial resources to manage stressors and their aftermath may be relevant for fostering both optimism and more adaptive hemodynamic stress responses. By addressing psychological and physiological factors upstream to cardiovascular disease development, this study informs the identification of potential targets for primordial intervention efforts aimed at mitigating the lifelong impact of stress on health.

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Received August 27, 2022
Revision received December 28, 2022
Accepted December 30, 2022 ■