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Lifetime Trauma, Perceived Control, and All-Cause Mortality: Results From the Midlife in the United States Study

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Objective: To investigate whether lifetime-trauma exposure predicts all-cause mortality and whether this association is mediated or moderated by perceived control. ***Method:*** A sample of middle-aged and older adults (N = 4,961) who participated in the second wave of the Midlife in the United States Study (MIDUS) provided data. Lifetime trauma was operationalized using the reported number of potentially traumatic experiences spanning childhood through adulthood. Both the perceived constraints and mastery dimensions of perceived control were examined. Cox regression models tested main effects and interactions of lifetime trauma with mastery and constraints predicting 10-year mortality risk. ***Results:*** There was a significant main effect of lifetime trauma, b = .06, hazard ratio (HR) = 1.07, p = .032, and an interaction of trauma with mastery, b = −.08, p = .004. A greater number of traumatic experiences was associated with increased mortality risk at below-average levels of mastery, −1 *SD*; HR = 1.14, p < .001, but not at above-average levels, +1 *SD*; HR = 0.97, p = .48. This interaction persisted after further adjustment for health status, psychological, and behavioral covariates. An association of constraints with elevated mortality risk, HR = 1.33, p = .008, was attenuated in a fully adjusted model, HR = 1.06, p = .26. ***Conclusion:*** A strong sense of mastery may buffer elevated mortality risk associated with exposure to traumatic experiences. Findings extend evidence that mastery may foster resilience to the adverse health effects of traumatic stressors, whereas constraints may show stronger independent associations with health outcomes.

Keywords: trauma, stress, perceived control, mastery, mortality

Exposure to potentially traumatic stressors is common and associated with higher morbidity and mortality (Felitti et al., 1998; Hendrickson et al., 2013; Krause, Shaw, & Cairney, 2004). Individuals exposed to traumatic stressors such as abuse or assault, military combat, or natural disasters are at greater risk for medical and psychiatric conditions, including cardiovascular disease, cancer, diabetes, pain disorders, and depression (Felitti et al., 1998; Kendall-Tackett, 2009; Pietrzak, Goldstein, Southwick, & Grant, 2011), as well as earlier mortality (Brown et al., 2009; Chen, Turiano, Mroczek, & Miller, 2016; Hendrickson et al., 2013; Kelly-Irving et al., 2013). In addition to associations of medical comorbidity and mortality with child abuse and other adverse childhood experiences (ACEs; Brown et al., 2009; Felitti et al., 1998), studies have reported associations with cumulative lifetime exposure to traumatic experiences, independent of posttraumatic stress disorder (PTSD) status (Hendrickson et al., 2013; Pietrzak et al., 2011). These studies support an accumulation-of-risk model, in which exposure to each additional potentially traumatic stressor increases risk for a given health outcome in an additive, cumulative fashion (Appleton, Holdsworth, Ryan, & Tracy, 2017).

There is growing interest in identifying factors associated with reduced susceptibility to the adverse health consequences of ACEs and other traumatic stressors. One factor conceptually and empirically linked to such resilience is high perceived control, or, beliefs in one's ability to influence circumstances and attain goals (Benight & Bandura, 2004; Dunkel Schetter & Dolbier, 2011; Ong, Bergeman, & Boker, 2009). Conceptual models of stress, coping, and health (Folkman, 1984; Pearlin, Menaghan, Lieberman, & Mullan, 1981; Taylor & Stanton, 2007) posit that greater perceived

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control may reduce perceived threat in appraisals of stressful events, as well as promote more adaptive coping responses (e.g., problem-solving, support-seeking). This may, in turn, reduce the severity or chronicity of negative cognitive–emotional states, as well as stress-related physiological alterations, evoked by potentially traumatic events and experiences. Trauma exposure is linked to affective and physiological states such as depression, inflammation, and shortened telomeres, which predict health and mortality risk (Cuijpers et al., 2014; Felitti et al., 1998; O'Donovan, Neylan, Metzler, & Cohen, 2012; O'Donovan et al., 2011; Puterman et al., 2016). In earlier studies, perceived control mediated or moderated associations of traumatic and other stressors with negative affect and depressive symptoms (Jang, Chiriboga, & Small, 2008; King, Wardecker, & Edelstein, 2015; Turiano, Silva, McDonald, & Hill, 2017), and moderated associations of lifetime trauma with the inflammatory marker, C-reactive protein (CRP) (Elliot, Mooney, Infurna, & Chapman, 2017). In addition to these pathways, positive coping behaviors fostered by greater perceptions of control may reduce the likelihood of engagement in risky behaviors as means of coping with distress associated with trauma exposure. Such behaviors potentially linking trauma and mortality include smoking and alcohol and/or substance abuse (Bellis, Lowey, Leckenby, Hughes, & Harrison, 2014; Feldner, Babson, & Zvolensky, 2007; Felitti et al., 1998).

Potentially operating through these behavioral, psychological, or biological pathways, perceived control moderated associations of chronic stressors and low socioeconomic status (SES) with self-report and objective health outcomes, including mortality (Elliot & Chapman, 2016; Mausbach et al., 2007; Pudrovska, Schieman, Pearlin, & Nguyen, 2005; Turiano, Chapman, Agrigoroaei, Infurna, & Lachman, 2014). As for traumatic stressors, control buffered associations of childhood sexual abuse with self-rated health in pregnant women (King et al., 2015) and of physical abuse with self-reported physical health in middle-age and older adults (Pitzer & Fingerman, 2010). Perceived control also directly predicts mortality and other health conditions (Infurna, Ram, & Gerstorf, 2013; Infurna & Mayer, 2015) and may play a mediating role in their associations with trauma exposure. Beginning in childhood and continuing through the life span, traumatic stressors may sensitize individuals to external threats and undermine global perceptions of control (Bolger & Patterson, 2001; Shaw & Krause, 2002). In previous studies, perceived control was found to mediate associations of child maltreatment and various stressors in adulthood (e.g., chronic stressors, discrimination) with mental and physical health status (Bolger & Patterson, 2001; Jang et al., 2008; Mooney, Elliot, Douthit, Marquis, & Seplaki, 2016; Shaw & Krause, 2002). Although on average, individuals with greater exposure to traumatic or other stressors report lower perceptions of internal or overall control, those able to maintain high perceived control may experience better health. Indeed, in several studies, perceived control both mediated and moderated associations between life-span stress exposures and physical and mental health outcomes (Bolger & Patterson, 2001; Jang et al., 2008; Pitzer & Fingerman, 2010).

In the present study, we investigated whether perceived control mediates or moderates an association between cumulative lifetime exposure to potentially traumatic experiences and mortality risk. We examined two conceptually and empirically distinct dimensions of control, mastery (perceived capacity to achieve goals) and perceived constraints (belief that outcomes are determined by uncontrollable factors rather than individual actions; Infurna & Mayer, 2015; Lachman & Weaver, 1998). We hypothesized that mastery and constraints would partly explain associations of greater lifetime trauma with higher mortality (mediation), and that high mastery and low constraints would also buffer mortality risk associated with trauma (moderation). In addition, we expected that these interactions would be partly explained by physical health status, psychological factors, and health-related behaviors. We further investigated whether any mediation or moderation by mastery or constraints was specific to trauma experienced in childhood versus in adulthood.

Method

Participants

In the present study, we used data from the Midlife Development in the United States Study (MIDUS), a national longitudinal study that commenced in 1995, with the first follow-up (MIDUS 2) conducted between 2004 and 2006 (Radler & Ryff, 2010). The initial sample consisted of approximately 7,000 adults ages 25 to 74, of whom 4,963 participated in MIDUS 2 (mortality-adjusted response rate of 75%). The present study was restricted to the MIDUS 2 sample because of availability of survey items comprising the measure of lifetime trauma. Whites, women, and individuals with higher levels of education, better self-rated health, and fewer functional limitations were more likely to participate in MIDUS 2 (Radler & Ryff, 2010). Two participants were excluded because of missing survival data. We also performed sensitivity analyses using a more restricted sample with complete data on all study variables (N = 3,809). Relative to those excluded, participants included in this sample had higher levels of perceived control, higher SES, were in better health, and were more likely to be male and of minority race/ethnicity. The present study was exempt from institutional review board requirements, as the selected MIDUS data are publicly available through the Interuniversity Consortium for Political and Social Research (ICPSR).

Measures

Lifetime trauma exposure. As in other studies testing accumulation-of-risk models, lifetime trauma exposure was operationalized using a count of reported potentially traumatic experiences, out of 12 total (prevalence shown in Table 1). These items were drawn from a larger subset of stressful experiences assessed in the MIDUS 2 survey, along with a measure of childhood abuse assessed in the MIDUS 1 survey. Selected items were based on those included in other surveys of lifetime-trauma exposure in community samples (Krause et al., 2004; Turner & Lloyd, 1995). Six items referred to experiences that participants may have had during childhood and are commonly included in measures of ACEs: parental physical and emotional abuse, parental alcohol and substance abuse, parental divorce (Bellis et al., 2014; Brown et al., 2009; Hostinar, Lachman, Mroczek, Seeman, & Miller, 2015). We also included parental death during childhood. Childhood abuse was measured using the validated Conflict Tactics Scale (Straus, 1979), which was administered only at MIDUS 1. Physical abuse was considered present if it was reported to have occurred *sometimes* or *often* (Chen et al., 2016), and emotional abuse, if it occurred *often*. In addition to moderate physical abuse (e.g.,

Table 1
Descriptive Statistics for the Analytic Sample (N = 3,809)

Variables	Percent/Mean (*SD*)	Range
Sociodemographic covariates		
Age	55.4 (12.45)	28–84
Female gender	53.3%	
White race	86.0%	
Minority race/ethnicity	10.9%	
Parental/personal education:	5.71 (2.88)/7.20 (2.52)	1–12
Less than high school	25.7%/7.5%	
High school degree	35.5 %/25.5%	
Some college	15.8%/29.9%	
Graduated 4-year college	23.0 %/37.1%	
Participant SES	.0 (1.00)	−3.46–3.90
Lifetime trauma	1.15 (1.41)	0–8
Parental alcohol abuse[a]	22.9%	
Parental drug use[a]	1.6%	
Parent died[a]	6.3%	
Parental divorce[a]	16.6%	
Emotional abuse (often)[a]	11.2%	
Moderate/severe physical abuse[a]	27.0%/12.0%	
Child died[b]	14.2%	
Child life-threatening illness[b]	12.2%	
Physical assault[b]	14.2%	
Sexual assault[b]	14.2%	
Lost home to fire, flood, etc.[b]	6.7%	
Experienced combat[b]	8.2%	
Perceived Control	7.6%	
Mastery	5.74 (1.03)	1–7
Perceived constraints	2.58 (1.18)	1–7
Other covariates		
Chronic disease	2.43 (2.38)	0–13
ADL limitations	1.56 (.73)	1–4
Self-rated health	3.54 (1.02)	1–5
Depression	10.0%	
Neuroticism	2.07 (.63)	1–4
Social support	3.46 (.46)	1–4
Pack-years smoked	16.8 (28.24)	0–244.80
Alcoholic drinks per week	11.4 (20.62)	0–83
Alcohol problems	3.5%	

Note. SES = socioeconomic status; ADL = activities of daily living.
[a] Survey items asked whether these experiences occurred during childhood. [b] Survey items asked whether these experiences occurred at any point in life.

pushed, grabbed, slapped), severe physical abuse (e.g., hit with fist or object, choked, burned) was considered an additional traumatic exposure. A measure of physical or emotional abuse in adulthood was not available. The remaining six experiences were based on survey items asking about experiences that participants may have had at any point in the life span. To increase comparability with other studies (e.g., Elliot et al., 2017), including those using narrower criteria for traumatic events (Hendrickson et al., 2013; O'Donovan et al., 2012), we selected only those experiences commonly regarded as potentially traumatic in nature (e.g., abuse, assault, natural disaster, life-threatening illness, accident).

The MIDUS questionnaire also asked participants to report the age at which a given stressful event occurred. Based on reported ages we also created separate counts of potentially traumatic experiences in childhood (before age 18) and adulthood (age 18 and over). Participants also rated how the given experience affected them both *initially* and *in the long-run* on a 5-point scale, from *very negatively* to *very positively*.

Perceived control. The measure of perceived control was developed by Lachman and Weaver (1998). Personal mastery (α = .73) was measured using four items (e.g., "I can do just about anything I really set my mind to"), and perceived constraints (α = .85) using eight items (e.g., "What happens in my life is often beyond my control"), rated on a 7-point Likert scale.

Sociodemographic covariates. Sociodemographic covariates included gender and race (White vs. minority race/ethnicity). We controlled for parental education (highest level achieved by either parent, measured on a 12-point scale from *no school/some grade school* to *advanced graduate/professional degree*) given that lower family SES is associated with greater odds of various ACEs (Lee, Coe, & Ryff, 2017). Similarly, we adjusted for adult SES as a potential confounder. To derive a comprehensive measure of SES, we constructed a composite by averaging standardized scores (*z* scores) for education (measured on the same 12-point scale), household income, household wealth, and occupational prestige (Hauser & Warren, 1996).

Health-status covariates. Disease burden was measured using a count of the number of reported medical conditions (out of 29 total, e.g., diabetes, high blood pressure, stroke, ulcer). Functional health was assessed using a measure of limitations in basic and instrumental activities of daily living (ADLs). Participants rated the extent to which their health limited them in performing each of 10 total activities (e.g., bathing or dressing, climbing stairs, lifting or carrying groceries), with scores on a 4-point scale (*not at all* to *a lot*) averaged to generate an overall limitations score. Self-rated physical health, measured on a 5-point scale from *poor* to *excellent*, was also included, as it predicts mortality over and above objective measures of health status (DeSalvo, Bloser, Reynolds, He, & Muntner, 2006).

Psychosocial covariates. Depressive symptoms were measured using the reported number of symptoms (up to seven in total, e.g., loss of interest, low energy, trouble concentrating) among participants who reported feeling *sad, blue, or depressed* most of the day or all day for 2 weeks or more in the past 12 months. We also included the Big-5 personality trait, neuroticism (α = .74), as well as social support as potential confounders, given observed associations with both perceived control (Judge, Erez, Bono, & Thoresen, 2002; Taylor, Lehman, Kiefe, & Seeman, 2006) and mortality (Chapman, Fiscella, Kawachi, & Duberstein, 2010; Holt-Lunstad, Smith, & Layton, 2010). For neuroticism, participants rated themselves on four trait adjectives (i.e., moody, worrying, nervous, calm), on a 4-point scale (*not at all* to *a lot*). Support from family, friends, and spouse/partner were each measured with between four and six items (e.g., "How much do your family members really care about you?"), rated on the same 4-point scale and averaged for a total network support score (α = .86).

Behavioral covariates. Behavioral covariates included pack-years of smoking (number of packs of cigarettes smoked per day times the number of years smoking) and multiple variables assessing alcohol use/abuse. Participants were asked questions about their alcohol consumption "during the period in your life, now or in the past, when you drank the most," from which we constructed a continuous variable measuring the number of drinks per week. We also included an indicator variable for whether participants reported at least one alcohol-related problem (e.g., psychological problems stemming from use, development of tolerance) in the past 12 months.

Mortality. Mortality follow-up was conducted through October 2015. Mortality data on participants was obtained using several methods. First, a National Death Index (Centers for Disease Control and Prevention, 2017) search conducted in 2009 confirmed 173 participant deaths. Second, 322 deaths were recorded during tracing and mortality closeout interviews conducted by the University of Wisconsin Survey Center as part of MIDUS 3 (2013–2015) survey fielding (University of Wisconsin Madison, 2017). Last, 57 deaths were recorded during the course of normal longitudinal sample maintenance.

Statistical Analysis

We used Cox regression to evaluate an association of trauma with all-cause mortality and to examine potential mediation or moderation by mastery and perceived constraints (Infurna & Mayer, 2015; Lachman & Weaver, 1998). We used age at death (attained age) as the time scale, as it provides a natural metric for longitudinal observational studies while still adjusting for age effects (Pencina, Larson, & D'Agostino, 2007). We also screened for nonproportionality (hazards that change with increasing survival time) by testing interactions of predictors with survival time (attained age in these models). We first tested main effects of lifetime trauma, mastery, and constraints in separate models adjusted for sociodemographic covariates. To test for mediation, we used VanderWeele's (2011) "difference method," which measures the indirect or mediated effect as the difference in the coefficient for the exposure variable (lifetime trauma) in models with and without the mediator (mastery or constraints). To evaluate potential moderation, a second set of models tested interactions of lifetime trauma with each dimension of control in separate models, and if significant, we examined the effect of trauma at varying levels of the given control variable. To facilitate these analyses, mastery and constraints variables were standardized (z-scored).

Next, we examined the extent to which additional health status, psychosocial, and behavioral covariates statistically accounted for any observed interactions (Turiano et al., 2014). To do so, we tested a fully adjusted model that included all covariates and their interactions with trauma, if significant. Additional analyses included repeated tests of mediation and moderation for separate measures of trauma in childhood and adulthood. We also performed various sensitivity analyses detailed in the results section. Missing values in the MIDUS 2 (longitudinal) sample were imputed using multiple imputation with chained equations (MICE). Following recommendations for imputation in Cox models (White & Royston, 2009), in addition to covariates, we included the event indicator (deceased status) as well as the Nelson–Aalen estimator of the baseline hazard function in the imputation model. We performed 10 imputations.

Results

Descriptive statistics are shown in Table 1. Participants ranged in age from 28 to 84, with an average age of 55.4 (SD = 12.5). The majority (86%) was White and around two thirds had completed some college. More than half of the sample (60%) reported at least one potentially traumatic experience, and nearly a third (30%) reported two or more. In the analytic sample there were 552 deaths (11.1%). The mean age at death was 74.9 years (SD = 10.8), with 99 deaths (18%) before age 65, 135 (24%) between 65 and 74, 200 (36%) between 75 and 85, and 118 (21%) at age 85 and over. mastery was uncorrelated with lifetime trauma ($r = -.02$, $p = .30$) and trauma in childhood ($r = -.01$, $p = .52$) and adulthood ($r = -.03$, $p = .11$), specifically. The constraints dimension was significantly correlated with lifetime trauma ($r = .08$, $p < .001$), and showed similar correlations with trauma exposure in childhood ($r = .06$, $p < .001$) and adulthood ($r = .08$, $p < .001$). When we examined specific experiences (analyses not shown), we found that only physical assault was significantly associated with lower mastery. Perceived constraints were higher for those reporting emotional abuse, moderate and severe physical abuse, physical and sexual assault, and having lost a home. Mastery and constraints were moderately correlated ($r = -.50$, $p < .001$).

Table 2 shows coefficients and corresponding hazard ratios (HRs) for the series of Cox regression models. In the baseline model (Model 1), greater lifetime trauma predicted higher all-cause mortality in a dose–response fashion (HR = 1.07, 95% CI [1.01, 1.13], $p = .032$). The hazard associated with lifetime trauma was proportional (i.e., it did not change with increasing survival time). Mastery and perceived constraints, however, showed nonproportional hazards: their associations with lower and higher mortality, respectively, diminished with increasing survival time. As shown in Table 2, nonproportional hazards for mastery and constraints, expressed as significant interactions with survival time (attained age), were retained in all models. When mastery and constraints were added to the model with lifetime trauma, the coefficient for trauma was largely unchanged (HR = 1.07, $p = .038$). As such, there was no evidence for mediation. When examined separately, mortality was not significantly associated with childhood trauma (HR = 1.06, $p = .14$) or adulthood trauma (HR = 1.09, $p = .10$).

With regard to moderation, we observed a significant interaction of trauma with mastery ($b = -.08$, 95% CI [−.14, −.03], $p = .004$), whereby the trauma–mortality association decreased in magnitude with increasing mastery. Simple slopes revealed a significant association at low mastery (−1 SD, HR = 1.14, 95% CI [1.06, 1.23], $p < .001$) but not at average mastery (HR = 1.05 [0.99 – 1.12], $p = .10$) or high mastery (+1 SD: HR = .97, 95% CI [.89 – 1.06], $p = .48$). The association was statistically significant at a level of mastery just below the mean (−.25 SD, HR = 1.07, 95% CI [1.01, 1.14], $p = .018$). Figure 1 shows the relative mortality hazard across the range of observed trauma scores at low, average, and high mastery. The interaction of trauma with constraints was not significant ($p = .57$)[1]. When examined separately, interactions of mastery with separate measures of trauma occurring in childhood and in adulthood were both significant ($b = -.09$, $p = .031$; $b = -.14$, $p = .004$, respectively), with each showing significant effects at a low level of mastery (−1 SD, HR = 1.14, $p = .011$; HR = 1.24, $p = .011$, respectively. When included in the same model, the interaction of childhood trauma and mastery was attenuated to nonsignificance ($b = -.07$, $p = .089$) whereas the interaction for adulthood trauma persisted ($b = -.12$, $p = .013$). Last, we tested a fully adjusted model (Table

[1] When included in the same model, the interaction term for mastery was attenuated by less than 20% and approached significance ($p = .058$), whereas the interaction term for constraints was more than 50% attenuated and nonsignificant ($p = .53$), suggesting that the interaction effect was largely specific to mastery.

Table 2
Coefficients and 95% Confidence Intervals for Cox Regression Models Predicting All-Cause Mortality

Variable	Model 1			Model 2			Model 3		
	b	HR	*p*	*b*	HR	*p*	*b*	HR	*p*
Female gender	−.42	.66	**<.001**	−.43	.65	**<.001**	−.36	.70	**<.001**
Minority race	.08	1.08	.553	.09	1.09	.520	.01	1.01	.922
Parental education	−.01	.99	.726	−.01	.99	.704	.01	1.01	.728
Participant SES	−.39	.67	**<.001**	−.39	.68	**<.001**	−.15	.86	.054
Lifetime trauma	.06	1.07	**.032**	.05	1.05	.100	.06	1.06	.124
Mastery[a]	−.11	.89	**.010**	.01	1.01	.089	−.07	.93	.859
Mastery × Attained age	.01	–	**.011**	.01	—	**.038**	.00	—	.410
Constraints	.28	1.33	**.008**	.26	1.29	**.020**	.06	1.06	.264
Constraints × Attained Age	−.01	—	**.045**	−.01	—	.076	−.00	—	.271
Trauma × Mastery				−.08	—	**.004**	−.08	—	**.012**
Trauma × Constraints[b]				.01	—	.568	—	—	—
Chronic disease							.03	1.03	.113
Self-rated health							−.32	.73	**<.001**
ADL limitations							.34	1.41	**<.001**
Trauma × ADL							−.08	.93	**.003**
Depression							−.01	.99	.768
Neuroticism							−.07	.93	.435
Social support							−.10	.90	.364
Pack-years smoked							.00	1.00	**<.001**
Alcohol problem							.18	1.20	.540
Alcoholic drinks per week							.00	1.00	.988

Note. HR = hazard ratio ($\exp_b$); SES = socioeconomic status; ADL = activities of daily living. *p* values less than .05 are boldfaced.
[a] Represents coefficient for mastery at average attained age of 65. [b] The interaction with constraints was tested in a separate model.

2, Model 3) including all additional covariates and any significant interactions with lifetime trauma, which applied only to functional limitations. The interaction with mastery was minimally attenuated (<5%) and the HR for trauma at low mastery (HR = 1.14), average mastery (HR = 1.05) and high mastery (HR = .98) were essentially unchanged.

In a sensitivity analysis using mastery scores averaged across MIDUS 1 and MIDUS 2 assessments (possibly reflecting average levels of mastery over a longer period of time), the interaction with lifetime trauma was significant and slightly larger ($b = -.09$, $p < .001$). This interaction also remained significant in a sensitivity analysis examining measures of lifetime trauma weighted by participants' ratings of both the initial and the long-term impact ($b = .09$, $p = .013$; $b = -.10$, $p = .006$, respectively). Complete case analysis ($N = 3{,}809$) revealed a similar interaction of lifetime trauma and mastery ($b = -.09$, $p = .003$), although there appeared to be some selection effects involving a somewhat larger main effect of trauma ($b = .09$, HR = 1.09, $p = .008$), as well as effects at varying levels of mastery. We also imputed missing values and tested associations in the entire initial MIDUS cohort ($N = 7{,}049$). The interaction of lifetime trauma with mastery scores averaged across MIDUS 1 and MIDUS 2 was significant ($b = -.042$, $p = .036$), with lifetime trauma again predicting mortality at just below the mean level of mastery (−.25 *SD,* HR = 1.05, $p = .028$).

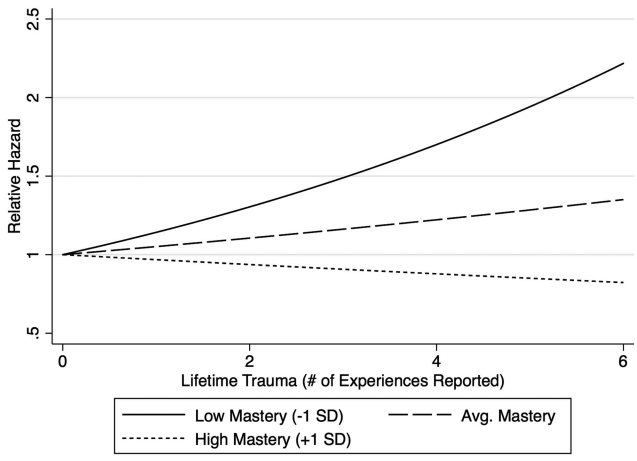

Figure 1. Relative hazards associated with lifetime trauma at varying levels of mastery.

Discussion

Using a national sample of middle-aged and older adults, we found that a greater number of potentially traumatic experiences was associated with elevated risk for all-cause mortality. Neither mastery nor constraints mediated the trauma-mortality association. However, mastery moderated the association: the effect of trauma was weaker with increasing mastery and no longer evident at above-average levels of mastery. In prior studies perceived control mediated associations of childhood maltreatment, as well as various stressors in adulthood, with measures of mental and physical health (Bolger & Patterson, 2001; Jang et al., 2008; Mooney et al., 2016; Pudrovska et al., 2005; Shaw & Krause, 2002). Here, higher perceived constraints was associated with trauma exposure in childhood (including child maltreatment) and adulthood, but did not explain associations of lifetime trauma with mortality. With regard to moderation, the interaction of lifetime trauma and mastery observed here adds to a growing body of evidence implicating mastery or perceived control as a key psychological factor asso-

ciated with resilience, broadly defined as positive or better-than-expected outcomes despite exposure to significant risk or adversity (Ong et al., 2009). Prior studies have found weaker associations of traumatic or other stressors with self-reported mental and physical health symptoms or conditions at high levels of perceived control (Jang et al., 2008; King et al., 2015; Pitzer & Fingerman, 2010; Pudrovska et al., 2005). We extend these findings by showing buffering effects of mastery/control involving a measure of cumulative stress exposure and predicting an objective endpoint in mortality. It is unclear whether and why the role of perceived control (mediation, moderation, or both) in the health effects of traumatic or other stressors might differ across specific mental or physical health outcomes. Further research examining pathways underlying stress-buffering and main effects of control variables may help to shed light on this question.

Notably, at lower levels of mastery, lifetime trauma was related to mortality in a dose-dependent fashion. An earlier study of cardiovascular patients (Hendrickson et al., 2013) found elevated mortality risk only at the upper end of the lifetime trauma distribution, a discrepancy that may be attributable to the clinical population, as well as a narrower diagnostic conceptualization of trauma. With regard to ACEs Brown et al. (2009) found an association with mortality only at a high level of exposure, whereas Kelly-Irving et al. (2013) found a graded association in women, but not men. In this study, potentially traumatic stressors experienced in childhood (similar to the ACE measures) showed a graded association with mortality at lower levels of mastery, although effects of trauma exposure in adulthood appeared to be stronger. Prior studies have also found associations of biomarkers and self-report health outcomes with cumulative trauma or adversity occurring in both childhood and adulthood (Hostinar et al., 2015; Krause et al., 2004; Lin, Neylan, Epel, & O'Donovan, 2016; Puterman et al., 2016).

In this study, lifetime trauma interacted with mastery but not constraints, which exhibited an independent association with mortality. These results parallel recent reports of differential effects for mastery and constraints—including a stronger main effect of constraints on mortality (Duan-Porter, Hastings, Neelon, & Van Houtven, 2017) and a moderating effect of mastery in relation to CRP (Elliot et al., 2017)—and further suggest examining these constructs separately. Constraints may be an independent risk factor, whereas mastery may buffer excess risk associated with stress exposure (viewed alternatively, it may be protective only at higher levels of stress exposure). Constraints may reflect circumstances leading to a perceived lack of contingency between actions and outcomes (i.e., the belief that desired outcomes are thwarted by limitations beyond one's control), whereas mastery may reflect beliefs regarding personal competence (i.e., the perceived ability to perform the requisite actions; Infurna & Mayer, 2015; Skinner, 1996).

Social–cognitive models (Bandura, 1997; Benight & Bandura, 2004) conceptualize perceived control as central to self-efficacy, which is thought to play an instrumental role in recovery from trauma and is related to better self-reported health in trauma survivors (Luszczynska, Benight, & Cieslak, 2009). Higher mastery may be accompanied by greater "coping self-efficacy," or the perceived ability to perform specific coping behaviors in response to stressors, including problem-solving, managing negative thoughts and emotions, and obtaining support (Bandura, 1997). These coping behaviors may serve to blunt adverse behavioral, psychological, or physiological impacts of traumatic experiences. With regard to health behaviors, we found little evidence that the moderating effect of mastery was explained by less alcohol use/abuse or smoking, although we were unable to examine diet, exercise, or obesity. The interaction of lifetime trauma and mastery also persisted after adjustment for various health and psychological covariates, indicating that it was not merely an artifact of better health status, less depression (although the measure was limited in capturing duration and severity of symptoms), lower neuroticism, or greater social support among individuals with higher mastery. The lack of attenuation upon adjustment for health status and depression was surprising, as these are also plausible mediators. Nevertheless, this result is in line with prior studies of ACEs and lifetime-trauma exposure (Brown et al., 2009; Hendrickson et al., 2013; Kelly-Irving et al., 2013) showing minimal or modest attenuation of mortality effects in similar analyses.

Another possibility is that mastery modulates biological processes such as systemic inflammation or telomere length, through which trauma increases mortality risk. Preliminary support for this possibility emerged in a recent study (Elliot et al., 2017) in which mastery buffered an association of greater lifetime trauma with elevated CRP. Studies that directly test potential biological mediators as well as validated measures of behavioral and psychological factors are needed to identify mechanisms underlying the moderating effect of mastery. Future researchers should also investigate the role of stress reactivity. Unexpectedly, a recent community study (Infurna, Rivers, Reich, & Zautra, 2015) found that childhood trauma-exposed adults with high perceived control were more rather than less emotionally reactive to negative daily events.

Findings of the present study suggest that individuals with substantial lifetime trauma exposure and low mastery are at the greatest risk for earlier death. It is possible that enhancing mastery/control, a modifiable characteristic (Lachman, Neupert, & Agrigoroaei, 2011; Zautra et al., 2012), could reduce excess mortality risk in individuals with histories of trauma exposure. For example, cognitive–behavioral interventions, which have been widely used in the treatment of PTSD and are potentially feasible for implementation in primary care, may impact perceptions of control and self-efficacy (Benight & Bandura, 2004), and have improved mental health and reduced risk behaviors among individuals exposed to ACEs (Korotana, Dobson, Pusch, & Josephson, 2016).

Findings of the present study should be interpreted in consideration of both the strengths and limitations of the design. The sample was predominantly White and relatively well-educated, and further research is needed to determine whether findings extend to more ethnically diverse, lower SES populations. Regarding mortality assessment, a greater number of decedents may have been missed through sample maintenance and fielding procedures than if a National Death Index search had been repeated in 2015. This may have resulted in some additional error in mortality classification. We were also unable to examine cause-specific mortality.

The measure of lifetime trauma used in this study also has various limitations, including its retrospective nature and the assessment of childhood abuse and other traumatic experiences occurring many years apart. However, evidence supports the validity and cross-time stability of retrospective reports of childhood abuse (Hardt & Rutter, 2004; Yancura & Aldwin, 2009), including in the MIDUS survey (Lee et al., 2017). As noted, the measure of cumulative trauma exposure is similar to measures in several

health-related investigations (Krause et al., 2004; Puterman et al., 2016; Turner & Lloyd, 1995) but reflects a broader conceptualization and less precision than in others that have employed structured clinical interviews (Hendrickson et al., 2013). There is ongoing debate as to how to conceptualize and measure trauma exposure (Saunders & Adams, 2014). The use of a count variable also has limitations: It does not capture differences associated with repeated or prolonged exposure to the same type of stressor (Appleton et al., 2017). In addition, it makes the assumption that each type of traumatic stressor impacts risk to a similar degree. To address this limitation, we also examined a measure weighted by subjective impact ratings (Appleton et al., 2017), which showed similar results. In sum, here as in other studies (Lin et al., 2016), there were limitations in the measurement of trauma resulting from the use of an existing population-based dataset. New studies would ideally include more extensive and repeated measures of trauma, and prospective designs are needed to address important questions concerning the temporal nature of associations between trauma exposure (including specific types of experiences), perceived control, and health status over the life span. The MIDUS data used here also had advantages, including a national sample that is more representative than smaller single-site or convenience samples. As well, though retrospective, the data permits a life-span view in connecting these childhood and adulthood experiences with a key health endpoint in midlife and older adulthood. Ultimately, multiple types of research designs and measures of trauma are needed to achieve a comprehensive understanding of relationships between trauma, perceived control, and health. Other strengths of the present study include the examination of separate dimensions of control and several potential explanatory variables.

In the present study, we found that a strong sense of mastery buffered excess mortality risk associated with lifetime exposure to potentially traumatic experiences in middle-aged and older adults. These findings extend evidence implicating mastery or perceived control in health-related resilience to stress, and call for further investigation of underlying mechanisms. Further research on trauma and perceived control has the potential to inform programs and interventions to reduce health-related risk in persons with histories of trauma exposure.

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