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Social Integration: Implications for the Association Between Childhood Trauma and Stress Responsivity

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Objective: Childhood trauma is linked to the dysregulation of physiological responses to stress, particularly lower cardiovascular reactivity (CVR) to acute stress. The mechanisms that explain this association, however, are not yet fully understood. Method: Using secondary data from the Midlife in the United States (MIDUS) Biomarker Project (N = 1,148; n = 652 females), we examine whether social integration can help explain the association between childhood trauma and lower CVR. Participants completed a standardized laboratory stress paradigm which involved completing executive functioning (Stroop) and mental arithmetic (MATH) tasks. Cardiovascular measurements were continuously assessed using electrocardiogram (ECG) and Finometer equipment. The Social Well-Being Scale (Keyes, 1998) and the Childhood Trauma Questionnaire (CTQ; Bernstein et al., 2003) measured social integration and trauma, respectively. Results: Regression analyses demonstrated that childhood trauma was associated with lower systolic (SBP; $\beta = -.14$, p < .001) and diastolic (DBP; $\beta = -.11$, p < .001) blood pressure reactivity but not heart rate (HR) reactivity. Mediation analyses, using Hayes PROCESS Model 4, showed that higher levels of trauma were associated with less social integration and in turn linked to lower reactivity across all biological indices. Moreover, sensitivity analyses showed that this indirect effect via social integration was evident for emotional and physical abuse, emotional and physical neglect, but not sexual abuse. Conclusion: Overall, the results indicated that dysregulated cardiovascular stress responses owing to childhood trauma may be shaped by a lack of social integration. The implications of this, as well as the findings for the individual types of trauma, are discussed.

Clinical Impact Statement

These findings highlight the importance of social integration for the health of those made vulnerable by adverse childhood experiences. This knowledge offers a potentially useful and inexpensive approach to support trauma-exposed individuals, potentially reducing some of its longer-lasting impacts on physical health. Although experimental research is needed to provide evidence for socially-based interventions for those who have experienced trauma, this research sets the foundation for further investigation, and points to the implications for policy and practice.

Keywords: cardiovascular reactivity, childhood trauma, social integration, stress

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The experience of trauma and adverse life events during child-hood has been shown to have widespread consequences for psychological and physical health (Danese et al., 2007, 2008; Moffitt et al., 2013). A range of traumatic stressors, including sexual

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abuse, physical abuse, exposure to violence, parental substance abuse, and neglect, have not only been linked to greater risk of mental health disorders, including PTSD (Alisic et al., 2014; Pratchett & Yehuda, 2011) and depression (Kessler et al., 2010) but also to physical health outcomes, such as cancer, liver disease, respiratory disease (Felitti et al., 1998; Gilbert et al., 2015; Hughes et al., 2017; Kessler et al., 2010; Wegman & Stetler, 2009) and, in particular, the development of cardiovascular disease (CVD; for reviews, see Basu et al., 2017; Jakubowski et al., 2018; Loria et al., 2014; Su et al., 2015; Suglia et al., 2020). Specifically, it is hypothesized that the experience of trauma during childhood, a sensitive developmental period, can cause long-term changes in a range of interrelated biological systems such as neural, endocrine, immune, and cardiovascular systems (al'Absi et al., 2021; Danese &

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McEwen, 2012). Research has shown that individuals who report adverse childhood experiences demonstrated altered patterns of physiological responses to stress (McLaughlin et al., 2014, 2015); however, the social factors that might explain, or indeed mitigate, the effect of childhood trauma on such adverse physical health outcomes are yet to be fully examined.

Physiological Reactivity to Stress

The importance of examining physiological responses to stress is underscored by the well-established cardiovascular reactivity (CVR) hypothesis, which suggests that individuals who show physiological responses to stress outside of normative ranges are more likely to experience negative physical health outcomes, such as CVD, later in life (Krantz & Manuck, 1984; Obrist, 1976, 1981; Turner et al., 2020). Indeed, longitudinal research has demonstrated that exaggerated and prolonged CVR to stress (particularly excessive blood pressure responses) is a risk factor for future CVD (Carroll et al., 2012; Chida & Steptoe, 2010). In addition to this, and consistent with the principles of homeostatic regulation (McEwen, 1998; McEwen & Seeman, 1999), individuals who exhibit CVR below normal operating ranges are also thought to be at risk of negative health outcomes. Comparatively lower levels of CVR than what would typically be seen during stress (often termed "blunted reactivity"; Phillips et al., 2013) has been linked to a range of negative health outcomes including depression, obesity, eating disorders, addiction, greater illness frequency, poorer self-reported health, greater physical disability, lower bone mass, and greater musculoskeletal pain (Carroll, 2011; Carroll et al., 2007, 2008; Ginty et al., 2012; Phillips, 2011; Phillips et al., 2012, 2013; Turner et al., 2020) as well as, importantly, PTSD (e.g., Zimmerman et al., 2020).

Trauma and Physiological Responses to Stress

Overall, research has implicated the experience of early adversity in dysregulated responses to stress (al'Absi et al., 2021; Alastalo et al., 2009; Appelmann et al., 2021; Carpenter et al., 2007, 2011; Felitti et al., 1998; Iob et al., 2021; Trickett et al., 2014). In particular, childhood trauma has been linked to lower cardiovascular responses to laboratory stress in both adolescents (Gooding et al., 2016; Murali & Chen, 2005) and adults (Bourassa et al., 2021; Ginty et al., 2017; Heim et al., 2000; Lovallo et al., 2012; Voellmin et al., 2015). Although it is not yet fully understood why childhood trauma is implicated in lower physiological responses, some researchers have speculated that trauma can lead to a "burn out" phenomenon, such that consistently elevated levels of arousal during early development results in long term changes in biological systems and dampened responses to future stress (Carpenter et al., 2011; De Bellis & Zisk, 2014; Murali & Chen, 2005; Tarullo & Gunnar, 2006). In essence, it can be seen as the body's way of adapting to, or regulating, prolonged arousal over time. Although these hypotheses are often discussed in terms of cortisol responding (i.e., the stress hormone), overall, it appears that experience of childhood adverse events translates to dysregulated patterns of stress responsivity across multiple biological systems including the cardiovascular system. Given its implications for physical health outcomes, it is important to identify the mediating pathways by which childhood trauma is linked to maladaptive cardiovascular responses to stress. Indeed, drawing on the social psychological literature may shed light on the process by which this occurs.

Social Support, Social Integration, and Health

One of the most commonly documented psychosocial factors to influence health is social support (i.e., the provision and receipt of psychological and tangible resources from social networks; Uchino et al., 2012), as evidenced by multiple reviews on the topic over the past 4 decades (e.g., Berkman & Glass, 2000; Cohen & Syme, 1985; Holt-Lunstad et al., 2010; Uchino, 2004; Uchino et al., 2018). The association between social support and better cardiovascular functioning (see Uchino, 2006, for review) is often explained by the fact that when support is available to us during stressful periods it can help us to reappraise the stressor in a more positive or manageable way, facilitating coping and reducing distress (Rueger et al., 2016).

Although social support has also been implicated in the reduction of PTSD and the adverse effect of trauma (e.g., John-Henderson & Ginty, 2020; Robinaugh et al., 2012; Sippel et al., 2015), this research often does not consider the value of community and group-based factors, particularly for trauma-exposed individuals. Social support theory is not synonymous with perspectives pertaining to collective or group-based resources (such as social identity theory [SIT]; Tajfel & Turner, 2004); however, moving from an individualized perspective on support to incorporate perceived belonging and social integration to the community may provide a unique understanding of how social processes may alleviate the adverse physiological effect of stress, and indeed, trauma.

Although not entirely unlike social support theory, the integrated social identity model of stress (ISIS; Haslam et al., 2005; Haslam & Reicher 2006) suggests that the groups we identify with and integrate into can act as a collective resource that can alter the meaning and experience of stress (in a more positive way), as well as offer social support, thus buffering the negative effect of stress on health. ISIS also suggests, however, that social identity processes are central to understanding the implications of stress. Of distinct relevance here, Haslam et al. (2005) state that it is key that individuals perceive they belong to the group to acquire the potential health benefits of such resources.

Importantly, research has highlighted the risk of reduced social integration for morbidity and mortality (Schwarzer & Rieckmann, 2002). For example, a lack of social integration has been linked to poor heart rate variability and increased risk of CVD (Gouin et al., 2015), as well as cardiovascular related mortality (Horsten et al., 2000). Indeed, researchers have argued that social integration can have stress-buffering properties, thus extending our understanding of processes linking social integration to health (Barrera, 1986; Uchino, 2004), and pointing to the potential importance of integration for cardiovascular responsivity to stress. Albeit limited, there is growing experimental evidence to suggest that social processes can influence how individuals appraise and physiologically respond to stress (e.g., Gallagher et al., 2014; Scheepers & Ellemers, 2005). In essence, if a lack of social integration can be detrimental for health, maintaining high or adequate levels can be protective for health, particularly in terms of how we physiologically respond to stress.

Trauma and Social Integration

Although the implications of social integration for physiological health have been established, it is important to note that trauma can also have repercussions for social integration. Social integration, and the valuable resources offered by group memberships, can be thought of as a buffer against the negative effects of trauma (e.g., supporting and helping individuals to cope with trauma); however, traumatic experiences often impact people's capacity to engage with and gain support from relevant social and occupational groups (Haslam et al., 2012; Muldoon et al., 2019). As well as undermining people's faith and trust in others, the experience of trauma can reduce the willingness of those affected by traumatic abuse to engage with others (Ozer et al., 2003). In addition, many traumatic experiences including child abuse and neglect are highly stigmatized, and as such survivors are often positioned on the margins of society, reducing their ability to access social, psychological and economic resources (Muldoon et al., 2021). As such, altered cardiovascular reactivity indices among trauma-exposed individuals (e.g., Bourassa et al., 2021; McLaughlin et al., 2014, 2015) may be shaped by poorer social integration and reduced ability to access group-based resources in support of health.

The Present Study

In summary, the link between childhood trauma, maladaptive physiological responses to stress (e.g., Bourassa et al., 2021; Voellmin et al., 2015), and adverse health outcomes (Jakubowski et al., 2018) is relatively well established within the health psychology literature. In parallel, social psychological research has shown that trauma can influence social integration and in particular people's sense of belonging with groups and communities (Muldoon et al., 2021). Although each of these pathways has been explored independently, here, we merge these paradigms to examine whether social integration acts as a mediating factor in explaining how trauma can lead to maladaptive cardiovascular responses to stress. This is an important avenue for research given the potential for improved social integration and social resources to support the health of those made vulnerable by adverse childhood experiences.

Method

Design

This study presents research on secondary data from the Midlife in the United States (MIDUS); a national, longitudinal survey of more than 7,000 Americans. The MIDUS study, which is funded by National Institute on Aging, was conducted by the University of Wisconsin Institute on Aging to explore the behavioral, psychological, and social factors for understanding age-related differences in mental and physical health. The present study focused on the Biomarker Project (Project 4) of MIDUS 2 and adapts a cross sectional, observational design. The purpose of the Biomarker Project (Project 4) was to include biometric assessments of a subsample of MIDUS respondents, with the view to identifying biopsychosocial pathways that may influence health outcomes. Biometric data were collected at University of California, Los Angeles, Georgetown University, and the University of Wisconsin and processed at the Columbia University Medical Center (CUMC) in the laboratory of Dr. Richard Sloan. Data collection was approved by the Education and Social/Behavioral Sciences and the Health Sciences institutional review boards at the University of Wisconsin-Madison. Further information on MIDUS and access to the data can be obtained via http:// midus.wisc.edu/.

Participants

The Biomarker Project contains data from 1,255 participants (n = 713 [65.8%] females) ranging in age from 34 to 84 ($M_{\rm age} = 54.09$, SD = 1.59). Participants were excluded from participating in the protocol if baseline blood pressure measures during the termination criteria phase were greater than 180/100 based on three readings taken 5 minutes apart (See Psychophysiological Blood Pressure Termination Criteria available at http://midus.wisc.edu/) because this is indicative of hypertension. From this sample, a number of participants were removed owing to missing data (n = 82) or for having completed a different task (PASAT) that was subsequently changed from the original protocol (n = 25); a final sample of 1148, with 496 males (43.2%) and 652 (56.8%) females ($M_{\rm age} = 56.94$; SD = 11.45), is analyzed. Table 1 includes further details of participant characteristics.

Materials and Apparatus

Childhood Trauma Questionnaire

Childhood trauma was assessed using the 28-item self-report Childhood Trauma Questionnaire (CTQ; Bernstein et al., 2003), a commonly used validated measure for retrospectively evaluating childhood trauma in adults. This measure assessed trauma scores across five subscales: emotional abuse (EA), physical abuse (PA), sexual abuse (SA), physical neglect (PN), and emotional neglect (EN), as well as a summed total trauma score. Items were reported on a Likert scale ranging from 1 (Never True) to 5 (Very Often True) with higher scores indicating higher levels of trauma. Reliability analyses for the overall scale yielded a Cronbach's α of .85. Although not central to the main analyses, good reliability for each of the individual subscales was also noted (emotional abuse $\alpha = .87$; physical abuse $\alpha = .79$; sexual abuse $\alpha = .94$; physical neglect $\alpha = .70$; and emotional neglect $\alpha = .89$). Descriptive statistics on the CTQ, included in Table 2, are relatively similar, or slightly above, normative data on the CTQ (Scher et al., 2001). Frequencies are also provided (See Table 3 of the online supplemental materials) for those who did or did not experience each type of trauma. Notably, 98.3% of the participants who completed the questionnaire reported experiencing at least one type of trauma. Sexual abuse was the most commonly reported type of trauma, with 76.3% of the sample reported experiencing it (regardless of how frequently it occurred).

Table 1Participant Characteristics

Characteristic	N	%	M	SD
Sex				
Male	496	43.20		
Female	652	56.80		
Ethnicity				
White	889	77.40		
Black, African American	25	2.20		
Native American, Inuit, Aleut	13	1.10		
Asian	3	0.30		
Other	28	2.50		
Age			56.94	11.45
Body mass index			29.80	6.59
Household total income (\$US)			77,833.82	61,276.62

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 Table 2

 Descriptive Statistics on Childhood Trauma

	Total		Ma	ale	Female	
Childhood trauma	M	SD	M	SD	M	SD
Emotional abuse	8.03	4.18	7.40	3.37	8.50	4.66
Physical abuse	7.03	3.12	6.94	2.74	7.09	3.38
Sexual abuse	6.59	3.97	5.66	2.32	7.31	4.75
Emotional neglect	9.76	4.59	9.62	4.24	9.87	4.83
Physical neglect	6.94	2.80	6.80	2.47	7.04	3.01
Total CTQ	38.84	14.29	36.94	11.28	40.29	16.08

Note. CTQ = Childhood Trauma Questionnaire. Range for subscales = 5–25; Range for total scale = 20–114.

Social Integration

Social integration was assessed using the 'Social Integration' subscale of the Social Well-being Scale (Keyes, 1998). Items, which include "I do not feel I belong to anything I'd call a community," "I feel close to other people in my community," and "My community is a source of comfort" were reported on a scale of 1 (Strongly Agree) to 7 (Strongly Disagree); higher scores refer to higher levels of social integration. Total scores were calculated as the sum of all items following reverse coding. Good internal reliability was evident within the present sample (Cronbach's $\alpha = .75$).

Stress Testing Protocol

The stress-testing protocol lasted approximately 90 minutes and involved an 11-minute baseline period followed by the first six-minute psychological stress task. Subsequently, there was a recovery period that lasted 6 minutes, and finally the second six-minute psychological stress task. The order in which these tasks were presented to participants were counterbalanced. The stress tasks used in the protocol were tests of executive function and mental arithmetic which are commonly used to elicit a cardiovascular stress response (e.g., Ginty et al., 2017; Howard et al., 2011; McLaughlin et al., 2014; Voellmin et al., 2015). Mental arithmetic was assessed using the Morgan and Turner Hewitt (MATH) task (Turner et al., 1986). Details and validation of the tasks are reported elsewhere (Ryff et al., 2019), and further information on the protocol is available in the Psychophysiology Protocol Documentation available from MIDUS (http://midus.wisc.edu/). In brief, this is a computerized task where a math problem is presented on screen, followed by the word "equals," with a solution to the problem finally appearing for up to 1.0 second. The participants were required to press one of two keys (corresponding to yes or no) on a keypad to indicate whether the presented solution is correct or not. The task varied in level of difficulty, beginning at level 3, and subsequent levels of difficulty were determined by response accuracy on the previous trial. Note that the mental arithmetic task was changed from the PASAT to the MATH three months after Project 4 data collection began (n = 25 participants) to reduce respondent burden, confusion, and frustration; these participants were removed and only participants who completed the MATH task are assessed in the current sample. In addition, a modified version of the Stroop task was also presented to participants where they were shown one of four color name words (blue, green, yellow, or red) in a font color that was either congruent or incongruent with the name. Participants were instructed to select one of four keys on the keyboard that corresponded to the color of the letters in the word, not the color name.

Psychological Responses to Stress

To measure psychological stress appraisal to the tasks, participants reported their stress levels verbally on a scale from 1 (*not stressed at all*) to 10 (*extremely stressed*) at baseline and during each stressor.

Cardiovascular Assessment

Physiological data were assessed continuously throughout the protocol using electrocardiogram (ECG) and Finometer equipment. Systolic and diastolic blood pressure (SBP; DBP) were recorded noninvasively using a Finometer (Finapres Medical Systems, Amsterdam, Netherlands); a finger cuff was placed on the middle finger of the nondominant hand, and a blood pressure arm cuff was placed on the upper arm of the same arm. Heart rate (HR) was measured using ECG. Beat-to-beat analog ECG signals were collected and then digitized at a sampling rate of 500 Hz by a 16-bit National Instruments analog-to-digital (A/D) board installed in a microcomputer. ECG electrodes were placed on the left and right shoulders, as well as the left lower quadrant. Heart rate (HR) was calculated as an average of all valid RR intervals and has been converted to beats per minute.

Statistical Analyses

For ease of interpretation, and consistent with previous research examining MIDUS data (e.g., Bibbey et al., 2013; Coyle et al., 2020; Creaven et al., 2020), CVR was computed as the difference between average baseline measures and average stress measures (across the two stress tasks). Prior to the main analyses, a series of repeated-measures t tests were conducted to test for differences between baseline measures of psychological stress levels and reported levels of stress during the task, as well as differences between baseline and task measures of SBP, DBP, and HR. This was to confirm that participants perceived the stressor to be psychologically stressful and that they physiologically reacted to the stressor. The main analyses first consisted of a series of linear regression analyses to examine the association between childhood trauma and each CVR parameter. Following this, a series of mediation analyses to test social integration as a mediator between childhood trauma (independent variable) and CVR (individual dependent variables) using Hayes's PROCESS (Model 4) were conducted. Analyses were conducted using IBM SPSS Version 26.

Results

Preliminary Analyses

To ensure that the task elicited a physiological stress response, results from a series of paired-samples t tests showed a statistically significant difference between baseline and task period for SBP, t(1017) = 34.99, p < .001, d = 1.35, DBP, t(1017) = 43.85, p < .001, d = .63, and HR, t(1073) = 31.76, p < .001, d = .33. Table 3 presents all means (and SDs) for each cardiovascular parameter during resting and task phases, as well as reactivity scores. An additional t test also demonstrated that participants perceived the task to be psychologically stressful; there was a statistically significant increase from baseline stress ratings (M = 2.03, SD = 1.49, Range 1–10) to stress ratings for the overall task period (M = 4.59, SD = 1.89), t(1121) = 49.04, p < .001, d = 1.50.

Main Analyses

Regression analyses show that childhood trauma is negatively associated with SBP ($\beta = -.14$, t = -4.42, p < .001, 95% CI [-.15, -.06]) and DBP ($\beta = -.11, t = -3.59, p < .001, [-.05, p]$ -.02]) reactivity, but not HR ($\beta = -.06$, t = -1.86, p = .06, [-.03, .001]) reactivity. This suggests that an increase in childhood trauma is associated with lower blood pressure reactivity. Following this, a series of mediation analyses were conducted to examine social integration as a mediating variable explaining the association between childhood trauma and each cardiovascular parameter. Although a total and direct effect was only evident for SBP and DBP reactivity (See c and c' pathways in Figure 1), analysis confirmed a significant indirect effect between childhood trauma and SBP reactivity ($\beta = -.01$, SE = .006, [-.03, -.002]), DBP reactivity ($\beta = -.005$, SE = .002, [-.01, -.002]) and HR reactivity ($\beta =$ -.005, SE = .002, [-.01, -.002]) via social integration.¹ These findings suggest that higher levels of childhood trauma are associated with less social integration (see A paths in Figure 1), which in turn is linked to lower SBP, DBP, and HR reactivity in response to stress (see B paths in Figure 1).

Sensitivity Analyses: Trauma Type

Additionally, given that the childhood trauma questionnaire assesses trauma across a range of different trauma types (emotional, physical, and sexual abuse, and physical and emotional neglect), additional sensitivity analyses examined social integration as a mediator between each of the aforementioned traumatic experiences and SBP, DBP, and HR reactivity to stress. Interestingly, an indirect effect via social integration was evident for emotional abuse (SBP: $\beta = -.04$, SE = .02, [-.08, -.004]; DBP: $\beta =$ -.05, SE = .007, [-.03, -.004]; HR: $\beta = -.02$, SE = .006, [-.03, -.004]-.007]), physical abuse (SBP: $\beta = -.03$, SE = .02, [-.07, -.006]; DBP: $\beta = -.01$, SE = .007, [-.03, -.003]; HR: $\beta = -.01$, SE = .007.006, [-.03, -.003]), emotional neglect (SBP: $\beta = -.05, SE = .02$, [-.10, -.005]; DBP: $\beta = -.02$, SE = .009, [-.01, -.004]; HR: $\beta = -.02$, SE = .008, [-.04, -.009]), and physical neglect (SBP: $\beta = -.06$, SE = .03, [-.12, -.01]; DBP: $\beta = -.02$, SE = .01, $[-.05, -.007]^2$; HR: $\beta = -.02$, SE = .009, [-.04, -.005]; see Figures 1.2, 1.3, 1.5, and 1.6 in the online supplemental materials, where experiencing more of these types of trauma was related to lower levels of social integration and, in turn, lower stress reactivity. Interestingly, no significant indirect effect of social integration for sexual abuse on SBP ($\beta = -.01$, SE = .01, [-.04, .003]) or DBP ($\beta = -.006$, SE = .005, [-.02, .001]) was noted; however, a statistically significant indirect effect via integration was evident for HR ($\beta = -.006$, SE = .006, [-.02, -.0001]), although this is a small effect (see Figure 1.4 in the online supplemental materials). Overall, these results from the individual pathways showed that whereas lower social integration was linked to lower blood pressure reactivity (SBP, DBP), sexual abuse was not associated with social integration. For HR, however, higher rates of sexual abuse were associated with decreased social integration, and this in turn was associated with lower CVR. Overall, the lack of significance for two of the three key biological indices suggests a weaker effect for social integration as a mediating pathway by which childhood trauma is linked to adverse CV responses with regards to sexual abuse, over other forms of trauma.

Discussion

Summary of Findings

Using data from the MIDUS national survey, the current study examined whether the relationship between childhood trauma and altered patterns of physiological responses to stress is mediated by social integration. First, as predicted, the results indicated that experiences of childhood trauma were linked to lower reactivity. Second, in line with our expectations, the results demonstrate that social integration mediates this relationship; respondents that reported increased levels of childhood trauma also reported lower levels of social integration, and this in turn was associated with lower levels of cardiovascular responses to acute stress.

Childhood Trauma and Lower CVR

We found evidence of a direct association between childhood trauma and lower levels of reactivity which corroborates a wealth of existing literature showing that adverse childhood experiences are linked to lower CVR to laboratory stress (Bourassa et al., 2021; Heim et al., 2000; Lovallo et al., 2012; Voellmin et al., 2015). Indeed, the present study supports the hypothesis that childhood trauma is linked to dysregulated patterns of stress responsivity as highlighted by McLaughlin et al. (2014, 2015). Our findings related to the more recent "blunted" (i.e., lower) reactivity hypothesis, and contributes to better understanding the psychosocial factors that precede such responses, and how lower cardiovascular responses to stress occurs. The present study extends this line of enquiry and implicates social integration (or lack thereof) in the

¹ When controlling for body mass index and medication use, results remained the same. Regression analyses showed that CTQ was associated with SBP ($\beta = -.13$, p < .001) and DBP ($\beta = -.10$, p = .001) but not HR ($\beta = -.05$, p = .10), and mediation analyses showed a significant indirect effect via social integration for SBP ($\beta = -.01$, [-.03, -.001]), DBP ($\beta = -.001$, [-.002, -.001]) and HR ($\beta = -.005$, [-.009, -.001]).

² All subgroup analyses withstood the adjustment of the Bonferroni correction on the overall model significance, with the exception of social integration mediating the effect of physical neglect on DBP reactivity (p = .004).

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Table 3Descriptive Statistics for Baseline, Task, and Reactivity Scores for Each CVR Parameter

	Baseline scores		Task scores		Change scores (Reactivity)	
Parameter	M	SD	M	SD	M	SD
SBP (mm hg)	123.77	18.38	136.31	21.58	12.54	11.43
DBP (mm hg)	61.24	11.75	67.26	12.16	6.02	4.38
HR (mm hg)	72.95	10.76	76.56	11.22	3.61	3.72

Note. SBP = systolic blood pressure; DBP = diastolic blood pressure; HR = heart rate.

relationship between childhood trauma and maladaptive physiological responses to stress.

Social Integration as a Mediator

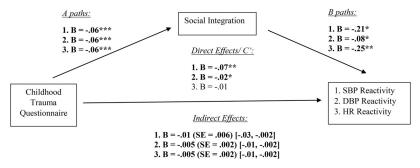
First, the results suggest that childhood trauma can adversely impact social integration. Indeed, this is in line with previous literature demonstrating that childhood trauma often isolates individuals from their own communities of support (Haslam et al., 2012; Muldoon et al., 2021), alters how people engage and respond to their social groups, and undermines levels of trust and connection with others (Muldoon et al., 2019). This lack of social integration is then, in turn, linked to lower levels of cardiovascular reactivity. These findings support the notion that social factors are important correlates of biological indices of physiological health. Indeed, it extends existing research that has demonstrated that social processes can influence how individuals appraise and physiologically respond to stress (e.g., Gallagher et al., 2014; Scheepers & Ellemers, 2005) while also highlighting the damaging effect that a lack of social integration, in particular, can have on stress responsivity. Most importantly, our findings indicate that it is plausible that the altered cardiovascular reactivity metrics brought about by childhood trauma may be shaped by an associated lack of social integration. In essence, the ability for individuals who have experienced greater childhood trauma to access resources to protect physiological health is reduced.

Trauma Type

Given that the CTQ assess trauma across five different trauma types (i.e., physical abuse, emotional abuse, physical neglect, emotional neglect, and sexual abuse), follow-up subgroup analyses explored these specific types of trauma as separate predictors. Emotional and physical abuse, and emotional and physical neglect, were all associated with less social integration, which in turn was linked to lower reactivity across all three biological parameters. These effects are as anticipated, and in line with related findings that some specific types of trauma, such as emotional and physical abuse, has more adverse effects on social integration (Barnett, 1997; Elliott et al., 2005; Loos & Alexander, 1997). It is worth acknowledging the results for sexual abuse, in particular.

The indirect effect of childhood sexual abuse on CVR via social integration was only significant for HR reactivity, but not for SBP or DBP reactivity: increased sexual abuse was associated with decreased social integration and, in turn, lower HR reactivity. The lack of statistical significance for two of the three parameters (SBP and DBP) may suggest that a socially based factor may not be enough to buffer or mediate the impact that sexual abuse may have on physiological biomarkers. Sexual abuse is a trauma that remains highly stigmatized, and such traumas can marginalize individual and reduce their ability to access social, psychological and economic resources (Muldoon et al., 2021). Thus, overall low or limited levels of social integration may not counteract the damaging health impact of such a stigmatized trauma (Lee, 2019; cf. Stanley et al., 2019). It is also worth noting that the mechanism by which sexual abuse impacts CVR may differ from other types of trauma. Indeed, scholars (Carpenter et al., 2011; Flory et al., 2009; Weissbecker et al., 2006) have reported varying physiological responses to stress across the different subgroups of those affected by childhood trauma. Similarly, previous research has highlighted that when examining the relationship between social variables

Figure 1
Simple Mediation Diagram Demonstrating Social Integration as a Mediating
Factor Between CTQ and all CVR Parameters



^{*} p < .05. ** p < .01. *** p < .001.

(i.e., social support) and health outcomes among people who have experienced sexual abuse, there are overlapping and inconsistent findings, highlighting the need for research directly examining this type of trauma (e.g., Steine et al., 2020) and indeed the support and integration of those exposed to sexual abuse. Given these mixed findings and the stigmatizing nature of sexual abuse, this is an avenue of research that warrants further investigation.

Strengths, Limitations, and Future Directions

The limitations of the "blunted" reactivity literature, particularly with regard to determining a threshold of what constitutes a lower and "blunted" response in comparison to a lower and "adaptive" responses (in terms of changes in mm hg) should be noted. The change in blood pressure and heart rate from baseline to task within this sample (SBP = 12.54 mm hg, DBP = 6.02 mm hg, HR = 3.61 bpm) is consistent with existing research concluding a "blunted" response (e.g., Brindle et al., 2017); however, without a predetermined threshold, the use of the term "blunted" should be used tentatively and should be noted as a limitation of both this study, and the field as a whole. Interestingly, in a bid to address this issue, researchers have adapted a data-driven approach and examined hypo- and hyper- reactivity with quartiles (e.g., Lang et al., 2016). Although this approach does not allow us to investigate the main objective of this article (i.e., mediational pathways), we have reported logistical regression analyses on trauma types using this approach to CVR in the online supplemental materials (Table 4). Until a consensus for hypo- and hyper- reactivity response thresholds is established, however, future research could consider adapting this statistical approach where possible, even as additional analyses.

Moreover, although the MIDUS dataset is longitudinal, only one wave was included in the current study because of the Biomarker Project linked to Wave 2. As such, this study is cross-sectional in nature, and we cannot infer causality. However, we present associational patterns to highlight mechanisms that may link childhood trauma, social processes, and biomarkers of physiological health. Future research should consider experimentally manipulating social resources such as social integration or social identification within a laboratory setting, with the view to developing socially based interventions that may reduce the adverse health effects of childhood trauma.

Although there are benefits to using publicly available secondary data (e.g., large sample size, reduced participant burden), this can also come with limitations. Of note to this study, the stress tasks used within this protocol are tasks of executive functioning (cognitive stress tasks) with no salient emotional aspect to the task. While these tasks are commonly used within CVR research, it is worth noting that past research examining the effect of trauma on CVR have observed differences in responses with respect to the type of task used. For example, Bourassa and Sbarra (2022) showed that a task with low personal salience (e.g., serial subtraction math stressor) was associated with a lower blood pressure response among people with a history of more traumatic experiences, whereas a task with higher personal emotional salience (e.g., a divorce-recall task) was associated with a higher blood pressure response. Although significant effects are noted within the current sample, perhaps more pronounced effects may be evident if emotional-provocation tasks were used.

Despite the limitations of the design, constrained by secondary data analysis, this is a large sample, particularly for physiological data, and includes both a diverse and difficult to reach population. Moreover, it uses existing, publicly available data while maintaining rigorous methodological stress-testing protocols to assess objective, biological markers of health.

Conclusion

Taken as a whole, these findings point to the potential for social integration to support the health of those made vulnerable by adverse childhood experiences and have important theoretical and clinical implications. From a theoretical standpoint, this research merges health, social and developmental psychology paradigms to demonstrate that social psychological processes contribute to our understanding of how trauma impacts physical health. Although research at the intersection of health and social psychology is not new, it reinforces the value of drawing on what we typically see as different schools of thought to shed light on unanswered questions and provide new perspectives on existing ideas.

With respect to clinical implications, the knowledge that social resources are at least one potential mechanism that facilitates the association between trauma and physical health is valuable for those working with trauma exposed individuals. In fact, this knowledge offers a potentially useful and inexpensive approach to support those at risk (by virtue of their experience of childhood trauma) and a way to potentially ameliorate some of its longer-lasting impacts on physical health. Although further experimental research is undoubtedly needed to provide evidence for socially based interventions among trauma-exposed individuals, this research sets the foundation for further investigation, and points to the implications for policy and practice.

References

Al'Absi, M., Ginty, A. T., & Lovallo, W. R. (2021). Neurobiological mechanisms of early life adversity, blunted stress reactivity and risk for addiction. *Neuropharmacology*, 188, 108519. https://doi.org/10.1016/j .neuropharm.2021.108519

Alastalo, H., Räikkönen, K., Pesonen, A. K., Osmond, C., Barker, D. J., Kajantie, E., Heinonen, K., Forsen, T. J., & Eriksson, J. G. (2009). Cardiovascular health of Finnish war evacuees 60 years later. *Annals of Medicine*, 41(1), 66–72. https://doi.org/10.1080/07853890802301983

Alisic, E., Zalta, A. K., van Wesel, F., Larsen, S. E., Hafstad, G. S., Hassanpour, K., & Smid, G. E. (2014). Rates of post-traumatic stress disorder in trauma-exposed children and adolescents: Meta-analysis. *The British Journal of Psychiatry*, 204(5), 335–340. https://doi.org/10.1192/bjp.bp.113.131227

Appelmann, H. M., Manigault, A. W., Shorey, R. C., & Zoccola, P. M. (2021). Childhood adversity and cortisol habituation to repeated stress in adulthood. *Psychoneuroendocrinology*, 125, 105118. https://doi.org/10.1016/j.psyneuen.2020.105118

Barnett, O. W., Miller-Perrin, C. L., & Perrin, R. D. (1997). Family violence across the lifespan: An introduction. Sage.

Barrera, M. (1986). Distinctions between social support concepts, measures, and models. American Journal of Community Psychology, 14(4), 413–445. https://doi.org/10.1007/bf00922627

Basu, A., McLaughlin, K. A., Misra, S., & Koenen, K. C. (2017). Childhood maltreatment and health impact: The examples of cardiovascular disease and type 2 diabetes mellitus in adults. *Clinical Psychology: A Publication* S140 McMAHON ET AL.

- of the Division of Clinical Psychology of the American Psychological Association, 24(2), 125–139. https://doi.org/10.1111/cpsp.12191
- Berkman, L. F., & Glass, T. (2000). Social integration, social networks, social support, and health. *Social Epidemiology*, 1, 137–173. https://doi.org/10.4135/9781412952576.n192
- Bernstein, D. P., Stein, J. A., Newcomb, M. D., Walker, E., Pogge, D., Ahluvalia, T., Stokes, J., Handelsman, L., Medrano, M., Desmond, D., & Zule, W. (2003). Development and validation of a brief screening version of the Childhood Trauma Questionnaire. *Child Abuse & Neglect*, 27(2), 169–190. https://doi.org/10.1016/S0145-2134(02)00541-0
- Bibbey, A., Carroll, D., Roseboom, T. J., Phillips, A. C., & de Rooij, S. R. (2013). Personality and physiological reactions to acute psychological stress. *International Journal of Psychophysiology*, 90(1), 28–36. https://doi.org/10.1016/j.ijpsycho.2012.10.018
- Bourassa, K. J., Moffitt, T. E., Harrington, H., Houts, R., Poulton, R., Ramrakha, S., & Caspi, A. (2021). Lower cardiovascular reactivity is associated with more childhood adversity and poorer midlife health: Replicated findings from the Dunedin and MIDUS cohorts. *Clinical Psychological Science*, 9(5), 961–978. https://doi.org/10.1177/2167702621993900
- Bourassa, K. J., & Sbarra, D. A. (2022). Cardiovascular reactivity, stress, and personal emotional salience: Choose your tasks carefully. *Psychophysiology*, 59(8), e14037. https://doi.org/10.1111/psyp.14037
- Brindle, R. C., Whittaker, A. C., Bibbey, A., Carroll, D., & Ginty, A. T. (2017). Exploring the possible mechanisms of blunted cardiac reactivity to acute psychological stress. *International Journal of Psychophysiology*, 113, 1–7. https://doi.org/10.1016/j.ijpsycho.2016.12.011
- Carpenter, L. L., Carvalho, J. P., Tyrka, A. R., Wier, L. M., Mello, A. F., Mello, M. F., Anderson, G. M., Wilkinson, C. W., & Price, L. H. (2007). Decreased adrenocorticotropic hormone and cortisol responses to stress in healthy adults reporting significant childhood maltreatment. *Biological Psychiatry*, 62(10), 1080–1087. https://doi.org/10.1016/j.biopsych .2007.05.002
- Carpenter, L. L., Shattuck, T. T., Tyrka, A. R., Geracioti, T. D., & Price, L. H. (2011). Effect of childhood physical abuse on cortisol stress response. *Psychopharmacology*, 214(1), 367–375. https://doi.org/10.1007/ s00213-010-2007-4
- Carroll, D. (2011). A brief commentary on cardiovascular reactivity at a crossroads. *Biological Psychology*, 86(2), 149–151. https://doi.org/10 .1016/j.biopsycho.2010.07.006
- Carroll, D., Ginty, A. T., Der, G., Hunt, K., Benzeval, M., & Phillips, A. C. (2012). Increased blood pressure reactions to acute mental stress are associated with 16-year cardiovascular disease mortality. *Psychophysiology*, 49(10), 1444–1448. https://doi.org/10.1111/j.1469-8986.2012.01463.x
- Carroll, D., Phillips, A. C., & Der, G. (2008). Body mass index, abdominal adiposity, obesity, and cardiovascular reactions to psychological stress in a large community sample. *Psychosomatic Medicine*, 70(6), 653–660. https://doi.org/10.1097/PSY.0b013e31817b9382
- Carroll, D., Phillips, A. C., Hunt, K., & Der, G. (2007). Symptoms of depression and cardiovascular reactions to acute psychological stress: Evidence from a population study. *Biological Psychology*, 75(1), 68–74. https://doi.org/10.1016/j.biopsycho.2006.12.002
- Chida, Y., & Steptoe, A. (2010). Greater cardiovascular responses to laboratory mental stress are associated with poor subsequent cardiovascular risk status: A meta-analysis of prospective evidence. *Hypertension*, 55(4), 1026–1032. https://doi.org/10.1161/HYPERTENSIONAHA.109.146621
- Cohen, S. E., & Syme, S. L. (1985). Social support and health. Academic Press
- Coyle, D. K. T., Howard, S., Bibbey, A., Gallagher, S., Whittaker, A. C., & Creaven, A. M. (2020). Personality, cardiovascular, and cortisol reactions to acute psychological stress in the Midlife in the United States (MIDUS) study. *International Journal of Psychophysiology*, 148, 67–74. https://doi.org/10.1016/j.ijpsycho.2019.11.014
- Creaven, A. M., Higgins, N. M., Ginty, A. T., & Gallagher, S. (2020).Social support, social participation, and cardiovascular reactivity to stress

- in the Midlife in the United States (MIDUS) study. *Biological Psychology*, 155, 107921. https://doi.org/10.1016/j.biopsycho.2020.107921
- Danese, A., & McEwen, B. S. (2012). Adverse childhood experiences, allostasis, allostatic load, and age-related disease. *Physiology & Behavior*, 106(1), 29–39. https://doi.org/10.1016/j.physbeh.2011.08.019
- Danese, A., Moffitt, T. E., Pariante, C. M., Ambler, A., Poulton, R., & Caspi, A. (2008). Elevated inflammation levels in depressed adults with a history of childhood maltreatment. *Archives of General Psychiatry*, 65(4), 409–415. https://doi.org/10.1001/archpsyc.65.4.409
- Danese, A., Pariante, C. M., Caspi, A., Taylor, A., & Poulton, R. (2007). Childhood maltreatment predicts adult inflammation in a life-course study. Proceedings of the National Academy of Sciences of the United States of America, 104(4), 1319–1324. https://doi.org/10.1073/pnas.0610362104
- De Bellis, M. D., & Zisk, A. (2014). The biological effects of childhood trauma. *Child and Adolescent Psychiatric Clinics of North America*, 23(2), 185–222, vii. https://doi.org/10.1016/j.chc.2014.01.002
- Elliott, G. C., Cunningham, S. M., Linder, M., Colangelo, M., & Gross, M. (2005). Child physical abuse and self-perceived social isolation among adolescents. *Journal of Interpersonal Violence*, 20(12), 1663–1684. https://doi.org/10.1177/0886260505281439
- Felitti, V. J., Anda, R. F., Nordenberg, D., Williamson, D. F., Spitz, A. M., Edwards, V., Koss, M. P., & Marks, J. S. (1998). Relationship of childhood abuse and household dysfunction to many of the leading causes of death in adults. The Adverse Childhood Experiences (ACE) Study. *American Journal of Preventive Medicine*, 14(4), 245–258. https://doi. org/10.1016/S0749-3797(98)00017-8
- Flory, J. D., Yehuda, R., Grossman, R., New, A. S., Mitropoulou, V., & Siever, L. J. (2009). Childhood trauma and basal cortisol in people with personality disorders. *Comprehensive Psychiatry*, 50(1), 34–37. https://doi.org/10.1016/j.comppsych.2008.05.007
- Gallagher, S., Meaney, S., & Muldoon, O. T. (2014). Social identity influences stress appraisals and cardiovascular reactions to acute stress exposure. *British Journal of Health Psychology*, 19(3), 566–579. https://doi.org/10.1111/bjhp.12056
- Gilbert, L. K., Breiding, M. J., Merrick, M. T., Thompson, W. W., Ford, D. C., Dhingra, S. S., & Parks, S. E. (2015). Childhood adversity and adult chronic disease: An update from ten states and the District of Columbia, 2010. American Journal of Preventive Medicine, 48(3), 345–349. https://doi.org/10.1016/j.amepre.2014.09.006
- Ginty, A. T., Masters, N. A., Nelson, E. B., Kaye, K. T., & Conklin, S. M. (2017). Cardiovascular reactions to psychological stress and abuse history: The role of occurrence, frequency, and type of abuse. *Anxiety*, *Stress, and Coping*, 30(2), 155–162. https://doi.org/10.1080/10615806 .2016.1210791
- Ginty, A. T., Phillips, A. C., Higgs, S., Heaney, J. L. J., & Carroll, D. (2012). Disordered eating behaviour is associated with blunted cortisol and cardiovascular reactions to acute psychological stress. *Psychoneur-oendocrinology*, 37(5), 715–724. https://doi.org/10.1016/j.psyneuen.2011.09.004
- Gooding, H. C., Milliren, C. E., Austin, S. B., Sheridan, M. A., & McLaughlin, K. A. (2016). Child abuse, resting blood pressure, and blood pressure reactivity to psychosocial stress. *Journal of Pediatric Psychology*, 41(1), 5–14. https://doi.org/10.1093/jpepsy/jsv040
- Gouin, J. P., Zhou, B., & Fitzpatrick, S. (2015). Social integration prospectively predicts changes in heart rate variability among individuals undergoing migration stress. *Annals of Behavioral Medicine*, 49(2), 230–238. https://doi.org/10.1007/s12160-014-9650-7
- Haslam, S. A., O'Brien, A., Jetten, J., Vormedal, K., & Penna, S. (2005).
 Taking the strain: Social identity, social support, and the experience of stress. *British Journal of Social Psychology*, 44(Part 3), 355–370. https://doi.org/10.1348/014466605X37468
- Haslam, S. A., & Reicher, S. (2006). Stressing the group: Social identity and the unfolding dynamics of responses to stress. *Journal of Applied Psychology*, 91, 1037–1052. https://doi.org/10.1037/0021-9010.91.5.1037

- Haslam, S. A., Reicher, S. D., & Levine, M. (2012). When other people are heaven, when other people are hell: How social identity determines the nature and impact of social support. In *The social cure: Identity, health and well-being* (1st Ed., pp. 157–174). Psychology Press.
- Heim, C., Newport, D. J., Heit, S., Graham, Y. P., Wilcox, M., Bonsall, R., Miller, A. H., & Nemeroff, C. B. (2000). Pituitary-adrenal and autonomic responses to stress in women after sexual and physical abuse in childhood. *JAMA*, 284(5), 592–597. https://doi.org/10.1001/jama.284.5.592
- Holt-Lunstad, J., Smith, T. B., & Layton, J. B. (2010). Social relationships and mortality risk: a meta-analytic review. *PLoS Medicine*. Advance online publication. https://doi.org/10.4016/19865.01
- Horsten, M., Mittleman, M. A., Wamala, S. P., Schenck-Gustafsson, K., & Orth-Gomér, K. (2000). Depressive symptoms and lack of social integration in relation to prognosis of CHD in middle-aged women. *The Stockholm Female Coronary Risk Study. European Heart Journal*, 21(13), 1072–1080. https://doi.org/10.1053/euhj.1999.2012
- Howard, S., Hughes, B. M., & James, J. E. (2011). Type D personality and hemodynamic reactivity to laboratory stress in women. *International Jour*nal of Psychophysiology, 80(2), 96–102. https://doi.org/10.1016/j.ijpsycho .2011.02.006
- Hughes, K., Bellis, M. A., Hardcastle, K. A., Sethi, D., Butchart, A., Mikton, C., Jones, L., & Dunne, M. P. (2017). The effect of multiple adverse childhood experiences on health: A systematic review and meta-analysis. *The Lancet. Public Health*, 2(8), e356–e366. https://doi.org/10.1016/S2468-2667(17)30118-4
- Iob, E., Baldwin, J. R., Plomin, R., & Steptoe, A. (2021). Adverse child-hood experiences, daytime salivary cortisol, and depressive symptoms in early adulthood: A longitudinal genetically informed twin study. *Translational Psychiatry*, 11(1), 420. https://doi.org/10.1038/s41398-021-01538-w
- Jakubowski, K. P., Cundiff, J. M., & Matthews, K. A. (2018). Cumulative childhood adversity and adult cardiometabolic disease: A meta-analysis. *Health Psychology*, 37(8), 701–715. https://doi.org/10.1037/hea0000637
- John-Henderson, N. A., & Ginty, A. T. (2020). Historical trauma and social support as predictors of psychological stress responses in American Indian adults during the COVID-19 pandemic. *Journal of Psychosomatic Research*, 139, 110263. https://doi.org/10.1016/j.jpsychores.2020 110263
- Kessler, R. C., McLaughlin, K. A., Green, J. G., Gruber, M. J., Sampson, N. A., Zaslavsky, A. M., Aguilar-Gaxiola, S., Alhamzawi, A. O., Alonso, J., Angermeyer, M., Benjet, C., Bromet, E., Chatterji, S., de Girolamo, G., Demyttenaere, K., Fayyad, J., Florescu, S., Gal, G., Gureje, O., . . . Williams, D. R. (2010). Childhood adversities and adult psychopathology in the WHO World Mental Health Surveys. *The British Journal of Psychiatry*, 197(5), 378–385. https://doi.org/10.1192/bjp.bp.110.080499
- Keyes, C. L. M. (1998). Social well-being. Social Psychology Quarterly, 61(2), 121–140. https://doi.org/10.2307/2787065
- Krantz, D. S., & Manuck, S. B. (1984). Acute psychophysiologic reactivity and risk of cardiovascular disease: A review and methodologic critique. *Psychological Bulletin*, 96(3), 435–464. https://doi.org/10.1037/0033-2909.96.3.435
- Lang, P. J., McTeague, L. M., & Bradley, M. M. (2016). RDoC, DSM, and the reflex physiology of fear: A biodimensional analysis of the anxiety disorders spectrum. *Psychophysiology*, 53(3), 336–347. https://doi.org/ 10.1111/psyp.12462
- Lee, J. S. (2019). Perceived social support functions as a resilience in buffering the impact of trauma exposure on PTSD symptoms via intrusive rumination and entrapment in firefighters. *PLoS ONE*, 14(8), e0220454. https://doi.org/10.1371/journal.pone.0220454
- Loos, M. E., & Alexander, P. C. (1997). Differential effects associated with self-reported histories of abuse and neglect in a college sample. *Journal of Interpersonal Violence*, 12(3), 340–360. https://doi.org/10 .1177/088626097012003002

- Loria, A. S., Ho, D. H., & Pollock, J. S. (2014). A mechanistic look at the effects of adversity early in life on cardiovascular disease risk during adulthood. *Acta Physiologica*, 210(2), 277–287. https://doi.org/10.1111/ apha.12189
- Lovallo, W. R., Farag, N. H., Sorocco, K. H., Cohoon, A. J., & Vincent, A. S. (2012). Lifetime adversity leads to blunted stress axis reactivity: Studies from the Oklahoma Family Health Patterns Project. *Biological Psychiatry*, 71(4), 344–349. https://doi.org/10.1016/j.biopsych.2011.10.018
- McEwen, B. S. (1998). Stress, adaptation, and disease. Allostasis and allostatic load. *Annals of the New York Academy of Sciences*, 840(1), 33–44. https://doi.org/10.1111/j.1749-6632.1998.tb09546.x
- McEwen, B. S., & Seeman, T. (1999). Protective and damaging effects of mediators of stress. Elaborating and testing the concepts of allostasis and allostatic load. *Annals of the New York Academy of Sciences*, 896(1), 30–47. https://doi.org/10.1111/j.1749-6632.1999.tb08103.x
- McLaughlin, K. A., Sheridan, M. A., Alves, S., & Mendes, W. B. (2014).
 Child maltreatment and autonomic nervous system reactivity: Identifying dysregulated stress reactivity patterns by using the biopsychosocial model of challenge and threat. *Psychosomatic Medicine*, 76(7), 538–546. https://doi.org/10.1097/PSY.00000000000000098
- McLaughlin, K. A., Sheridan, M. A., Tibu, F., Fox, N. A., Zeanah, C. H., & Nelson, C. A., III. (2015). Causal effects of the early caregiving environment on development of stress response systems in children. Proceedings of the National Academy of Sciences of the United States of America, 112(18), 5637–5642. https://doi.org/10.1073/pnas.1423363112
- Moffitt, T. E., & Tank, T. K.-G., & The Klaus-Grawe 2012 Think Tank. (2013). Childhood exposure to violence and lifelong health: Clinical intervention science and stress-biology research join forces. *Develop*ment and Psychopathology, 25(4 Part 2), 1619–1634. https://doi.org/10 .1017/S0954579413000801
- Muldoon, O. T., Lowe, R. D., Jetten, J., Cruwys, T., & Haslam, S. A. (2021). Personal and political: Post-traumatic stress through the lens of social identity, power, and politics. *Political Psychology*, 42(3), 501– 533. https://doi.org/10.1111/pops.12709
- Muldoon, O. T., Walsh, R. S., Curtain, M., Crawley, L., & Kinsella, E. L. (2019). Social cure and social curse: Social identity resources and adjustment to acquired brain injury. *European Journal of Social Psychology*, 49(6), 1272–1282. https://doi.org/10.1002/ejsp.2564
- Murali, R., & Chen, E. (2005). Exposure to violence and cardiovascular and neuroendocrine measures in adolescents. *Annals of Behavioral Medicine*, 30(2), 155–163. https://doi.org/10.1207/s15324796abm3002_8
- Obrist, P. A. (1976). Presidential Address, 1975. The cardiovascular-behavioral interaction—As it appears today. *Psychophysiology*, *13*(2), 95–107. https://doi.org/10.1111/j.1469-8986.1976.tb00081.x
- Obrist, P. A. (1981). Cardiovascular psychophysiology: A perspective. Plenum Press. https://doi.org/10.1007/978-1-4684-8491-5
- Ozer, E. J., Best, S. R., Lipsey, T. L., & Weiss, D. S. (2003). Predictors of posttraumatic stress disorder and symptoms in adults: A meta-analysis. *Psychological Bulletin*, 129(1), 52–73. https://doi.org/10.1037/0033-2909 129 1 52
- Phillips, A. C. (2011). Blunted cardiovascular reactivity relates to depression, obesity, and self-reported health. *Biological Psychology*, 86(2), 106–113. https://doi.org/10.1016/j.biopsycho.2010.03.016
- Phillips, A. C., Ginty, A. T., & Hughes, B. M. (2013). The other side of the coin: Blunted cardiovascular and cortisol reactivity are associated with negative health outcomes. *International Journal of Psychophysiol*ogy, 90(1), 1–7. https://doi.org/10.1016/j.ijpsycho.2013.02.002
- Phillips, A. C., Roseboom, T. J., Carroll, D., & de Rooij, S. R. (2012). Cardiovascular and cortisol reactions to acute psychological stress and adiposity: Cross-sectional and prospective associations in the Dutch Famine Birth Cohort Study. *Psychosomatic Medicine*, 74(7), 699–710. https://doi.org/10.1097/PSY.0b013e31825e3b91
- Pratchett, L. C., & Yehuda, R. (2011). Foundations of posttraumatic stress disorder: does early life trauma lead to adult posttraumatic stress disorder?

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Development and psychopathology, 23(2), 477–491. https://doi.org/10.1017/S0954579411000186

- Robinaugh, D. J., Marques, L., Bui, E., & Simon, N. M. (2012). Recognizing and treating complicated grief. *Current Psychiatry*, 11(8), 30–35.
- Rueger, S. Y., Malecki, C. K., Pyun, Y., Aycock, C., & Coyle, S. (2016).
 A meta-analytic review of the association between perceived social support and depression in childhood and adolescence. *Psychological Bulletin*, 142(10), 1017. https://doi.org/10.1037/bul0000058
- Ryff, C. D., Seeman, T., & Weinstein, M. (2019). Midlife in the United States (MIDUS 2): Biomarker Project, 2004–2009. *Inter-university Con*sortium for Political and Social Research [distributor], 2019-03-27. https://doi.org/10.3886/ICPSR29282.v9
- Scheepers, D., & Ellemers, N. (2005). When the pressure is up: The assessment of social identity threat in low and high status groups. *Journal of Experimental Social Psychology*, 41(2), 192–200. https://doi.org/10.1016/j.jesp.2004.06.002
- Scher, C. D., Stein, M. B., Asmundson, G. J., McCreary, D. R., & Forde, D. R. (2001). The childhood trauma questionnaire in a community sample: psychometric properties and normative data. *Journal of Traumatic Stress*, 14(4), 843–857. https://doi.org/10.1023/A:1013058625719
- Schwarzer, R., & Rieckmann, N. (2002). Social Support, Cardiovascular Disease and Mortality. Heart disease: *Environment, Stress, and Gender*, 327, 185.
- Sippel, L. M., Pietrzak, R. H., Charney, D. S., Mayes, L. C., & Southwick, S. M. (2015). How does social support enhance resilience in the traumaexposed individual? *Ecology and Society*, 20(4), art10. https://doi.org/10 .5751/ES-07832-200410
- Stanley, I. H., Hom, M. A., Chu, C., Dougherty, S. P., Gallyer, A. J., Spencer-Thomas, S., Shelef, L., Fruchter, E., Comtois, K. A., Gutierrez, P. M., Sachs-Ericsson, N. J., & Joiner, T. E. (2019). Perceptions of belongingness and social support attenuate PTSD symptom severity among firefighters: A multistudy investigation. *Psychological Services*, 16(4), 543–555. https://doi.org/10.1037/ser0000240
- Steine, I. M., Winje, D., Krystal, J. H., Milde, A. M., Bjorvatn, B., Nordhus, I. H., Grønli, J., & Pallesen, S. (2020). Longitudinal relationships between perceived social support and symptom outcomes: Findings from a sample of adult survivors of childhood sexual abuse. *Child Abuse & Neglect*, 107, 104566. https://doi.org/10.1016/j.chiabu.2020.104566
- Su, S., Jimenez, M. P., Roberts, C. T. F., & Loucks, E. B. (2015). The role of adverse childhood experiences in cardiovascular disease risk: A review with emphasis on plausible mechanisms. *Current Cardiology Reports*, 17(10), 88. https://doi.org/10.1007/s11886-015-0645-1
- Suglia, S. F., Campo, R. A., Brown, A. G. M., Stoney, C., Boyce, C. A., Appleton, A. A., Bleil, M. E., Boynton-Jarrett, R., Dube, S. R., Dunn, E. C., Ellis, B. J., Fagundes, C. P., Heard-Garris, N. J., Jaffee, S. R., Johnson, S. B., Mujahid, M. S., Slopen, N., Su, S., & Watamura, S. E. (2020). Social determinants of cardiovascular health: Early life adversity as a contributor to disparities in cardiovascular diseases. *The Journal of Pediatrics*, 219, 267–273. https://doi.org/10.1016/j.jpeds.2019.12.063
- Tajfel, H., & Turner, J. C. (2004). The social identity theory of intergroup behavior. In *Political Psychology* (pp. 276–293). Psychology Press.

- Tarullo, A. R., & Gunnar, M. R. (2006). Child maltreatment and the developing HPA axis. *Hormones and Behavior*, 50(4), 632–639. https://doi.org/10.1016/j.yhbeh.2006.06.010
- Trickett, P. K., Gordis, E., Peckins, M. K., & Susman, E. J. (2014). Stress reactivity in maltreated and comparison male and female young adolescents. *Child Maltreatment*, 19(1), 27–37. https://doi.org/10.1177/ 1077559513520466
- Turner, A. I., Smyth, N., Hall, S. J., Torres, S. J., Hussein, M., Jayasinghe, S. U., Ball, K., & Clow, A. J. (2020). Psychological stress reactivity and future health and disease outcomes: A systematic review of prospective evidence. *Psychoneuroendocrinology*, 114, 104599. https://doi.org/10.1016/j.psyneuen.2020.104599
- Turner, J. R., Hewitt, J. K., Morgan, R. K., Sims, J., Carroll, D., & Kelly, K. A. (1986). Graded mental arithmetic as an active psychological challenge. *International Journal of Psychophysiology*, 3(4), 307–309. https://doi.org/10.1016/0167-8760(86)90039-5
- Uchino, B. N. (2004). Social support and physical health: Understanding the health consequences of relationships. Yale University Press.
- Uchino, B. N. (2006). Social support and health: a review of physiological processes potentially underlying links to disease outcomes. *Journal of Behavioral Medicine*, 29(4), 377–387. https://doi.org/10.1007/s10865-006-9056-5
- Uchino, B. N., Bowen, K., Carlisle, M., & Birmingham, W. (2012). Psychological pathways linking social support to health outcomes: A visit with the "ghosts" of research past, present, and future. Social Science & Medicine, 74(7), 949–957. https://doi.org/10.1016/j.socscimed.2011.11.023
- Uchino, B. N., Trettevik, R., Kent de Grey, R. G., Cronan, S., Hogan, J., & Baucom, B. R. (2018). Social support, social integration, and inflammatory cytokines: A meta-analysis. *Health Psychology*, 37(5), 462. https://doi.org/10.1037/hea0000594
- Voellmin, A., Winzeler, K., Hug, E., Wilhelm, F. H., Schaefer, V., Gaab, J., La Marca, R., Pruessner, J. C., & Bader, K. (2015). Blunted endocrine and cardiovascular reactivity in young healthy women reporting a history of childhood adversity. *Psychoneuroendocrinology*, 51, 58–67. https://doi.org/10.1016/j.psyneuen.2014.09.008
- Wegman, H. L., & Stetler, C. (2009). A meta-analytic review of the effects of childhood abuse on medical outcomes in adulthood. *Psychosomatic Medicine*, 71(8), 805–812. https://doi.org/10.1097/PSY.0b013e3181bb2b46
- Weissbecker, I., Floyd, A., Dedert, E., Salmon, P., & Sephton, S. (2006). Childhood trauma and diurnal cortisol disruption in fibromyalgia syndrome. *Psychoneuroendocrinology*, 31(3), 312–324. https://doi.org/10.1016/j.psyneuen.2005.08.009
- Zimmerman, A., Halligan, S., Skeen, S., Morgan, B., Fraser, A., Fearon, P., & Tomlinson, M. (2020). PTSD symptoms and cortisol stress reactivity in adolescence: Findings from a high adversity cohort in South Africa. *Psychoneuroendocrinology*, 121, 104846. https://doi.org/10.1016/j.psyneuen .2020.104846

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