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Authors: Tracey Mary Keogh, Siobhán Howard, and Stephen Gallagher

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Early life adversity and blunted cardiovascular reactivity to acute psychological stress: The role of current depressive symptoms

Tracey M. Keogh, BSc^{1,2}, Siobhán Howard, PhD^{1,2}, and Stephen Gallagher, PhD^{1,2}

¹Department of Psychology, Centre for Social Issues Research, Study of Anxiety, Stress and Health Laboratory, University of Limerick, Limerick, Ireland

²Health Research Institute, University of Limerick, Castletroy, Limerick, Ireland

Corresponding author

Tracey Keogh

Department of Psychology

University of Limerick

Castletroy

Limerick

Ireland

Contact: tracey.keogh@ul.ie/ +353 (0) 61 23 3618

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Abstract

Objective: The pathways underlying the early life adversity and cardiovascular reactivity association remain unclear. The current study examined the role of current depressive symptoms on this relationship.

Methods: Mediation analyses were conducted using data from 639 participants drawn from the Midlife Development in the United States (MIDUS II) Biomarker Project. Responses were derived from the Childhood Trauma Questionnaire [CTQ] and Center for Epidemiologic Studies Depression Scale [CES-D]). Participants had their systolic and diastolic blood pressure (SBP, DBP) and heart rate (HR) monitored throughout a standardized stress testing protocol.

Results: The association between early life adversity and reactivity was mediated by current depressive symptoms; all adversity factors were linked to higher levels of current depressive symptoms, which in turn, were associated with lower cardiovascular reactivity. For emotional abuse, this was noted for SBP ($\beta = -.06$, 95% CI [-.13,-.01]; and DBP: $\beta = -.04$, [-.07,-.01]), physical abuse (SBP: $\beta = -.05$, [-.11,-.01]; DBP: $\beta = -.03$, [-.06,-.01]), sexual abuse (SBP: $\beta = -.04$, [-.09,-.01]; DBP: $\beta = -.02$, [-.05,-.01]), emotional neglect (SBP: $\beta = -.04$, [-.09,-.01]; DBP: $\beta = -.02$, [-.05,-.01]), physical neglect (SBP: $\beta = -.09$, [-.17,-.02]; DBP: $\beta = -.05$, [-.09,-.02]) and total CTQ (SBP: $\beta = -.02$, [-.03,-.00]; DBP: $\beta = -.01$, [-.02,-.00]).

Conclusions: The present findings extend research and demonstrate that depression is an underlying mechanism linking early life adversity and blunted cardiovascular reactivity.

Key words: Cardiovascular reactivity, blunted reactivity, early life adversity, depression, mediation

ANOVA = Analysis of variance, **CESD** = Center for Epidemiologic Studies Depression Scale, **CHD** = Coronary heart disease, **CTQ** = Childhood Trauma Questionnaire, **CTQ-SF** = Childhood trauma questionnaire short form, **CVD** = Cardiovascular disease, **CVR** = Cardiovascular reactivity, **DBP** = Diastolic blood pressure, **EA** = Emotional abuse, **EN** = Emotional neglect, **ECG** = electrocardiogram, **ELA** = Early life adversity, **HR** = Heart rate, **MATH** = Morgan and Turner Hewitt, **MBSR** = Mindfulness-based stress reduction, **MIDUS** = Midlife Development in the United States, **PA** = Physical abuse, **PN** = Physical neglect, **SA** = Sexual abuse, **SBP** = Systolic blood pressure, **SLEs** = Stressful life events

INTRODUCTION

Coronary heart disease (CHD) is the leading cause of global deaths annually and has been in the US for over 90 years (1). Since 2007, there has been a 21.1% mortality increase attributable to cardiovascular disease (CVD), and an estimated 82.6 million Americans are living with cardiac related illnesses (2). The focus in research is moving beyond conventional risk factors such as family history, physical inactivity, obesity, and smoking (3-5), to examining psychological factors such as depression. Hopelessness, a core symptom of depression, is associated with atherosclerosis (6), and is an established risk factor for CHD (7). Prospective studies suggest depression is predictive of subsequent hypertension (8), associated with increased risk of CHD (9), and mortality (10). Furthermore, greater depressive symptoms predict stroke in men (11), with the risk of having a stroke increasing two-to-four-fold, even after controlling for confounds (12).

Stress is proposed to link depression and CVD (13), with maladaptive cardiovascular responses indicating adverse outcomes (7, 14). Specifically, the cardiovascular reactivity (CVR) hypothesis suggests exaggerated responses to stress signify a vulnerability to disease, such as hypertension (15), atherosclerosis (16), and mortality (17). Blunted cardiovascular reactions, often considered to be protective, also lead to poorer outcomes, for instance, addiction (7, 14), eating disorders (18) and depression (19). Studies indicate depression is associated with increased systemic vascular resistance (20) and heightened stress responses (21), while others report no relationship (22, 23). In contrast, depression was found to be associated with blunted heart rate reactivity five years later (19), with several studies reporting an inverse relationship (19, 24-29). Moreover, a recent systematic review found hypo-stress reactivity to be a hallmark

of depression (30).

Stressful life events (SLEs), be it proximal (recent events) or distal (early life adversity; ELA), have been identified as a pathway potentially related to dysfunctional stress responses (29, 31-32). Chronic exposure to ELA during critical periods when physiological systems are developing, can result in alterations in stress response systems implicated in blunted CVR (33). For example, studies found blunted reactivity was associated with greater ELA (31), in women exposed to ELA (34), and in those with greater experience to emotional adversity prior to the age of 15 (35).

There are also a number of proximal and distal factors that influence the probability of developing depression (36). A longitudinal study of twins reported that recent life events (previous 12 months) were associated with symptom severity and may be the strongest determinant of the disorder (36). In contrast, it is well documented that ELA increases the risk of depression (37), is related to earlier onset, symptom severity, duration, more episodes, and impairment (38-40). ELA is attributed to over 30% of mental disorders in the US (41), with emotional abuse (EA) and emotional neglect (EN) increasing the risk of lifetime depression (42, 43); and more reliable than physical abuse (PA) or sexual abuse (SA; 44). EA has been linked to low self-esteem, depression, and eating disorders (45). Additionally, the prevalence estimate for EA (36%) is considerably higher than PA (8%) and for SA (2%) (46, 47). Furthermore, there is a significant association between ELA and adult depression, even when controlling for mediating factors (48, 49).

While depression, be it historical, current, or clinically diagnostic, confers a risk for cardiac events and mortality (26), and may be the strongest psychosocial risk factor for CVD (50), the mechanistic association is still poorly understood. The majority of studies on depression and blunted reactivity focus on current depressive symptoms. Furthermore, ELA is reportedly a source of variation in reactivity among those with depression (51). Therefore, it is plausible that depression, given its links with blunted reactivity (28) and ELA (43, 52) mediates the ELA-CVR relationship.

Therefore, based on the above evidence and using data from the Midlife Development in the United States 2 (MIDUS II), the aim of the present study was to identify if current depression influenced the relationship between ELA and CVR. In line with previous research, we hypothesized that individuals with depression would exhibit blunted reactivity to stress. Based on theoretical and empirical reports linking ELA with depression and CVR, we predicted that those with greater ELA would also report higher symptoms of depression and exhibit a blunted cardiovascular response to the stress task.

METHOD

Participants

Data were drawn from the 1,255 respondents who completed the MIDUS II Biomarker Project, which consisted of 1,054 from the longitudinal survey sample and 201 from the Milwaukee sample. At the time of the clinic visit, participants ranged in age from 35 to 86 ($M = 57.32$, $SD = 11.55$), with 56.8% females ($n = 713$) in the sample. A decision was made *a priori* to exclude the total number of twins in the sample ($n = 388$), due to genetic determinants of

reactivity (53), as well as considerations of the assumption of independence in analyses. Furthermore, an additional 16 participants were excluded as they had completed an extended version of the stress task (54), with an additional 26 outliers, 7 who had pacemakers, and an additional 179 participants who were missing data on study variables. This left a total of 639 participants and demographic information for the sample can be found in Table 1. There were no differences in reactivity or ELA due to socioeconomic status (all $ps > .05$). Furthermore, no association was observed for participants who reported taking medication for depression ($n = 55$) and CVR. Over two-thirds (70.1%) of the sample reported taking prescription medication of some kind. Given taking prescription medication was in effect normative for this sample; these participants were included and controlled for as a covariate in all mediation analyses. The Biomarker Project (P4) ran from 2004 to 2009 and data collection took place during a 24-hour stay at a clinical research centre and informed consent was obtained prior to beginning study procedures.

[INSERT TABLE 1 ABOUT HERE]

Procedure

Participants from the original MIDUS survey study were invited to complete the MIDUS II Project 1 phone interview and self-administered questionnaire. Upon completion, those who were not part of the city oversamples and who lived in the US were eligible for participation in the Biomarker Project. Participants underwent comprehensive biological assessment and completed a standardized laboratory-based stress task, where indicators of beat-to-beat blood pressure and heart rate were obtained. To enhance self-reported data collected in Project 1,

participants completed a medical history and self-administered questionnaires for psychosocial assessment.

Measures

Depression Measure

Depressive symptoms were assessed using the Center for Epidemiologic Studies Depression Scale (CES-D; 55). It comprises of 20 items pooled from previously validated scales to measure current state of depressive symptomology (55). Seven items measure depressive affect, four items measure positive affect, seven items measure somatic complaints, and two items are an interpersonal subscale. Items are scored from 0 (*rarely or none of the time*) to 3 (*most or all of the time*) and responses are based on occurrence in the past week. Higher scores indicate higher symptoms of depression. The CES-D has excellent concurrent validity and acceptable psychometric properties (55). Only the depressive affect subscale was used in the present study, as it has been previously reported to hold the highest proportion of variance (55, 56) and in the current study yielded a Cronbach's α of .85. It is also reported that the CES-D can be reduced in size with low impact on reliability and validity (57, 58).

Early Life Adversity

The original Childhood Trauma Questionnaire (CTQ) is a 70-item retrospective tool that assesses childhood abuse and neglect (59). A short form of the scale (CTQ-SF) was developed to reduce participant burden and this 28-item self-report questionnaire contains five clinical scales which measure occurrence of EA, PA, SA, EN and physical neglect (PN), with an additional three item minimization-denial scale to detect underreporting of abuse (60). Items are rated on a

5-point Likert scale with response options ranging from 1 (*never true*) to 5 (*very often true*). The CTQ has good psychometric properties and is a validated tool for psychiatric populations (61-63). For this study, both the total score and the five clinical scales were analysed. The total CTQ scale achieved an overall α (.62) and the Cronbach's alpha for the each of the five scales exceeded .73.

Cardiovascular Assessment

Beat-to-beat systolic blood pressure (SBP) and diastolic blood pressure (DBP) were measured using a Finometer monitor (Finapres Medical Systems, Amsterdam, Netherlands). Stemming from the volume-clamp method (64), measurements of finger arterial pressure were obtained through an inflatable finger cuff, which adjusts to increases in arterial pressure, reflecting changes in blood pressure (65). The Finometer accurately measures continuous blood pressure and detects minor changes in cardiovascular function (66). It is a widely used apparatus for measuring cardiac function in CVR research (4). Heart rate (HR) was recorded using electrocardiogram (ECG) signals digitized at 500 Hz and installed in a microcomputer. With ECG waveforms submitted to detection software to identify R waves, MIDUS research staff then visually reviewed all ECG waveforms to amend software errors in identifying normal R waves. HR was calculated from the resulting series of normal RR intervals and converted to beats per minute (54).

Stress Task

The stress tasks consisted of the Stroop colour/word task and the Morgan and Turner Hewitt (MATH; 67) mental arithmetic task which were presented in random order, each at six

minutes duration. The laboratory stress session ran for approximately 90 minutes and is described in detail elsewhere (54, 65). In brief, for the Stroop interference task, one of four colour name words were presented in either a font that was congruent or incongruent to the name on a computer screen (e.g., the word blue was written in blue font), and participants used one of four keys on a keypad which corresponded to the colour of the letters but not the colour name. The rate of presentation was based on task performance, with greater accuracy leading to faster presentation rates.

For the MATH task, participants were required to complete a series of addition and subtraction mental arithmetic tasks. The problem was presented on the computer screen for 2.0 sec, followed by the word equals for 1.5 sec, and then the answer which was displayed for 1.0 sec. Using the keypad, participants pressed one of two keys which corresponded to yes or no to indicate whether the answer presented to the problem was correct. Problem difficulty varied across five levels, from level 1 (*1-digit ± 1-digit numbers*) to level 5 (*3-digit ± 3-digit numbers*), and difficulty level was determined by response accuracy to each trial.

Participants completed practice trials for the stress task, after which a formal 11-minute resting baseline period began. They then completed the first of two cognitive stress tasks. A six minute recovery period followed after which the second cognitive task began. Stressor measures for cardiovascular variables are averaged across the two stress tasks (Stroop and MATH), as previously reported elsewhere (65).

Statistical analyses

Data were screened for outliers and assumption of fit prior to analyses. Mean levels of SBP, DBP, and HR were computed for baseline, task, and recovery. Given our *a priori* hypothesis was concerned with ELA and CVR, we only examined this and not recovery. Reactivity scores were calculated by subtracting the mean baseline levels from the mean task levels. Independent *t*-tests were conducted to test for differences in reactivity values, due to potential confounds of sex and smoking. A series of repeated measures ANOVAs were used to determine if the task was successful in eliciting a stress response; partial η^2 is reported for the effect size.

Correlational analyses evaluated the association between ELA, current depressive symptoms, and cardiovascular outcomes. Simple mediation analyses (Model 4) examined whether the relationship between ELA and CVR was mediated by current depression; using the overall total from the CTQ, and then specifically investigating each individual subscale (EA, PA, SA, EN, and PN), while controlling for covariates mentioned above, along with BMI, taking of prescription medication, hypertension, and baseline cardiovascular measures. The data were analysed using the PROCESS macro (v3.1) in IBM SPSS Statistics version 25.0 (68).

RESULTS

Descriptive statistics

The descriptive statistics and correlational analysis of study variables can be seen in Table 2.

[INSERT TABLE 2 ABOUT HERE]

Manipulation check

Results from a series of repeated measures (baseline, task) ANOVAs confirmed an increase in baseline to task on each cardiovascular measure for; SBP, $F(2,1248) = 517.84$, $p < .001$, $\eta^2 = .45$, DBP, $F(2,1248) = 822.56$, $p < .001$, $\eta^2 = .57$; and HR, $F(2,1266) = 555.80$, $p < .001$, $\eta^2 = .47$, demonstrating the task was physiologically stressful (Table 3). Furthermore, independent t -tests revealed differences in reactivity due to sex for SBP, $t(637) = 2.40$, $p = .017$, with higher SBP responses in males ($M = 13.00$, $SD = 11.85$), compared to females ($M = 10.81$, $SD = 11.10$), and a similar pattern observed for HR, $t(637) = 6.29$, $p < .001$. Additionally, analyses revealed differences in reactivity due to smoking, with smokers exhibiting lower SBP and DBP reactivity compared to non-smokers ($ps < .001$). These potential confounds were also associated with baseline cardiovascular measures. Thus, these were controlled for in our main analyses.

[INSERT TABLE 3 ABOUT HERE]

Associations between ELA, depressive symptoms and CVR

As can be seen in Table 2, there were significant associations between exposure to ELA and current depressive symptoms, such that those scoring higher on all measures of abuse and neglect scored higher on depression. Furthermore, as expected for CVR, depression was negatively associated with reactivity values; those with higher depressive symptoms had blunted SBP, DBP and HR responses to stress. Lastly, ELA factors were also associated in a negative

manner with CVR variables, such that those scoring higher on total CTQ, EA, PA, SA, EN and PN displayed blunted SBP and DBP responses to stress. Those who had a greater number of total CTQ, and greater experiences of SA also exhibited blunted HR reactivity.

Mediation analyses

Our overall analyses aimed to test if current depression (past week) was mediating the relationship between ELA and CVR. A simple mediation analysis was conducted while controlling for confounds sex, age, smoking, prescription medication, BMI, hypertension, and baseline cardiovascular measures. Ordinary least square path analysis and 10000 bootstrap samples were used to examine whether the effect of ELA factors on cardiovascular reactivity was mediated by current depressive symptoms. In our mediation analyses, no total, direct or indirect effects were observed for HR reactivity. Therefore, this is excluded from further reporting.

We observed several associations between ELA and SBP reactivity (please refer to Figure 1). Higher EA was significantly related to blunted SBP reactivity with a total effect; $\beta = -.20$, $t(631) = -2.00$, $p = .04$, 95% CI [-.40, -.00] being detected. EA was also related to greater depressive symptoms ($a = .20$, $p < .001$), 95% CI [.15, .26]. A 95% bias corrected CI based on 10000 bootstrap samples indicated that the indirect effect ($ab = -.06$) was entirely below zero [-.13, -.01]. EA is indirectly related to blunted SBP reactivity through its relationship with current depression. A similar pattern emerged for EN.

[INSERT FIGURE 1 ABOUT HERE]

We also observed several associations for ELA factors and DBP (please refer to Figure 2). Higher EN was significantly related to blunted DBP reactivity with a direct effect; $\beta = -.08$, $t(631) = -2.40$, $p = .02$, 95% CI [-.15, -.01] being observed. EN was also related to higher levels of depression ($a = .14$, $p < .001$), 95% CI [.09, .19]. A 95% bias corrected CI based on 10000 bootstrap samples indicated that the indirect effect ($ab = -.02$) was entirely below zero [-.05, -.01].

As can be seen in Table 4, a broadly analogous pattern emerged for SBP and DBP reactivity, with total and indirect effects observed for total CTQ and PA; and total, direct, and indirect effects for EN. These outcome variables only differed on EA, with total and indirect effects observed for SBP and only indirect effects for DBP. The relationship between ELA factors (childhood SA and PN), and blunted SBP and DBP reactivity only existed through the mediating role of current depression (refer to Figure 3a and 3b).

[INSERT FIGURE 2 ABOUT HERE]

[INSERT TABLE 4 ABOUT HERE]

[INSERT FIGURE 3A AND 3B ABOUT HERE]

DISCUSSION

The present study sought to examine associations between ELA, depression and CVR to acute stress. As predicted, an inverse relationship was observed between depression and CVR; those reporting higher depressive symptoms displayed blunted responses to stress. The findings

are in line with previous research reporting a negative relationship between depression and CVR (19, 25-27). More importantly, and in line with our own expectations, depression mediated the association between ELA and blunted reactivity. These findings were evident for SBP and DBP reactivity and withstood adjustment for several confounding factors, providing support for research reporting blunted SBP and DBP in patients with MDD (69). While no significant interaction was observed for HR, associations were observed in our regression models. Moreover, it is worth noting that not all studies find depressed participants have alterations in HR (70).

This study extends knowledge on the ELA-CVR relationship and supports the proposition that ELA increases susceptibility to depression and influences stress regulation (29). Carroll et al. (29) proposed that the association between ELA and blunted reactivity was linked to motivational factors, and that blunted reactivity may potentially be a marker of motivation dysregulation, implying that motivation may be a key mediating pathway. Our findings extend on this work by suggesting the ELA-blunted reactivity relationship is mediated by current depression; this association became less pronounced through the mediational pathway. More importantly, it may be that external factors like ELA interact with motivational factors and depression to influence blunted stress responses. For example, ELA influences physiological responses that result in changes to stress response systems associated with depression and motivated behaviour (71, 72). Furthermore, repeated or enduring exposure to ELA can have a long lasting impact on brain systems responsible for stress regulation and mood (29, 30). Thus, together these studies offer some support for this notion that ELA may influence both depression and motivated behaviour which affects our physiological response to stress; a combination that

could be examined in future studies.

Moreover, our study is in keeping with emerging evidence that supports the importance of preventative and intervention care. In fact, it is worth noting a recent study found women with ELA in a mindfulness-based stress reduction (MBSR) group; displayed improved cortisol responses and reduced depression relative to those in the control group (73). Additionally, another study reported that MBSR reduced depressive symptoms over a 2 month period in adults who reported childhood SA (74, 75). Considering the present findings suggest the relationship between ELA and blunted reactivity is mediated through current depression, future research may benefit from cultivating MBSR in samples with a history of ELA to reduce depression and somatic complaints (74) and improve mental and physical outcomes (75).

There are several limitations to the present study. First, due to the cross-sectional nature and reliance on self-report measures, inferences about causality and pathology cannot be drawn (76). Although the CES-D is a valid and reliable measure of depression, it only measures symptom severity experienced in the past week (55, 77). Second, due to the age of the sample, the majority of participants had at least one chronic health condition which may be co-morbid with depression, albeit we did control for these, and the effects were still evident. Furthermore, retrospective assessment of ELA does not capture the effects of stress during critical periods of development (78, 79), is subject to recall bias and issues of underreporting (80, 81). Future research may benefit by adding a measure of age of occurrence, this may offer a more comprehensive assessment on the critical periods of stress during sensitive developmental epochs. Nevertheless, the severity of events makes bias unlikely, and evidence suggests recollection of ELA in adulthood is relatively reliable (82), strongly associated with adult

depression (83) and blunted reactivity (84, 85). Finally, multiple comparisons were analysed and as such future research should replicate our findings. Strengths of the present study are its relatively large sample size, and inclusion of several covariates. Considering the wealth of research that shows blunted reactivity predicts the development of depression [19], an alternative avenue of investigation is that blunted reactivity and higher experiences of ELA may predict the development of depression. While this was beyond the scope of the current paper, it may prove useful for future research.

To conclude, the present study provides confirmation that ELA is associated with blunted CVR. Additionally, it provides empirical demonstration that depression is a key factor in this story; in particular we have found that the magnitude of the ELA-blunted reactivity relationship became less pronounced through the mediational pathway of current depression. Although SA and PA have reportedly lower prevalence estimates (46, 47), with both EA and EN considered more reliable risk factors for depression (42, 43), it is interesting that our findings suggest that those with greater exposure to childhood adversity, irrespective of type, are more susceptible to depression in adulthood. Given that ELA signifies a vulnerability to depression in adulthood (88), raises the frequency of depressive symptoms (48, 86-87), and is reportedly a source of variation in reactivity among those with depression, it is not surprising that those reporting greater exposure to adversity in childhood had higher symptoms of current depression and blunted cardiovascular responses to stress. Considering the negative health implications associated with blunted CVR, ELA and depression, our findings have important implications for tailored and targeted interventions. While there is no consistent evidence to suggest one particular type of ELA induces exaggerated or blunted reactivity; or has a differential impact in

later life (35), it is clear that the harmful effects of ELA persist long into adulthood (89) and is linked to mental health issues (90).

ACCEPTED

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List of Figures:

Figure 1: Mediation path diagram: Early life adversity and SBP reactivity.

Note: Each mediation analysis includes sex, age, smoking, taking prescription medication and baseline SBP as covariates. Statistics refer to unstandardized betas (B) and 95 % confidence intervals at the lower and upper limit for indirect effects. The A path represents the association between each independent variable and the mediator. Path B reflects the association between the mediator and SBP reactivity. Path C' represents the direct effect between each independent variable and SBP reactivity while holding the mediator and covariates constant. Finally, Path ab represents the indirect effect of each independent variable on SBP reactivity through current depressive symptoms. Significant effects are highlighted in bold text. $*p < 0.05$, $**p < 0.01$.

Figure 2: Mediation path diagram: Early life adversity and DBP reactivity.

Note: Each mediation analysis includes age, smoking, body mass index and baseline DBP as covariates. Statistics refer to unstandardized betas (B) and 95 % confidence intervals at the lower and upper limit for indirect effects. The A path represents the association between each independent variable and the mediator. Path B reflects the association between the mediator and DBP reactivity. Path C' represents the direct effect between each independent variable and DBP reactivity while holding the mediator and covariates constant. Finally, Path ab represents the indirect effect of each independent variable on DBP reactivity through current depressive symptoms. Significant effects are highlighted in bold text. $*p < 0.05$, $**p < 0.01$.

Figure 3. Simple mediation diagram. Current depressive symptoms as a mediator between ELA (sexual abuse) and SBP reactivity (A) and DBP reactivity (B).

* $p < 0.05$ level

** $p < 0.01$ level

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Figure 1

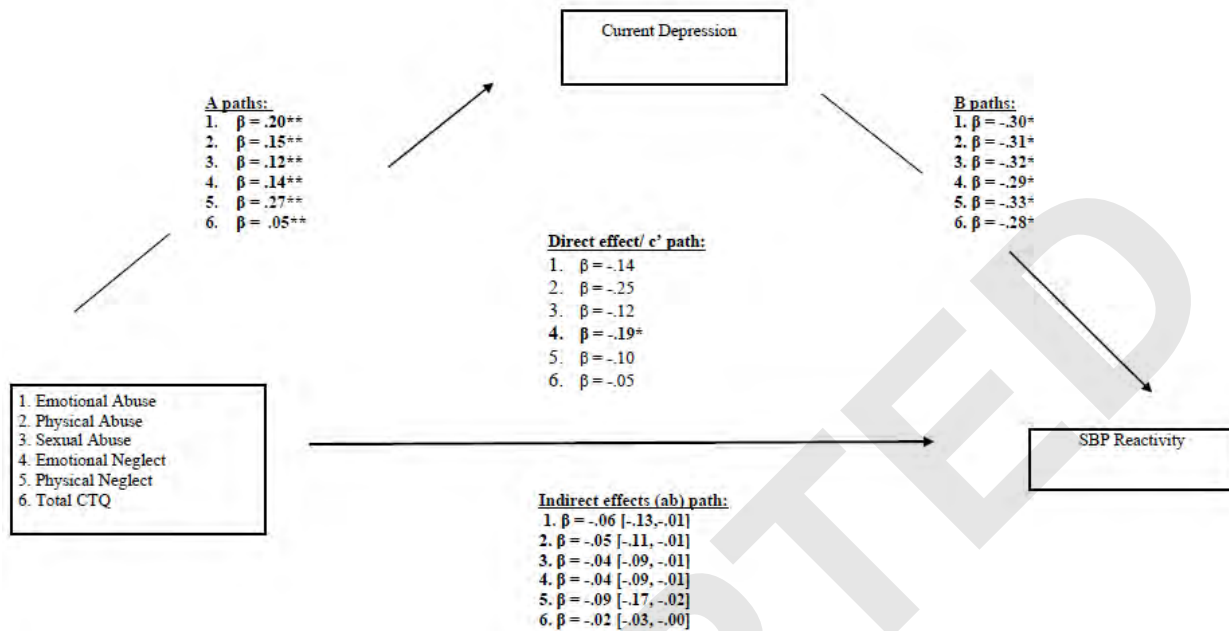


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Figure 2

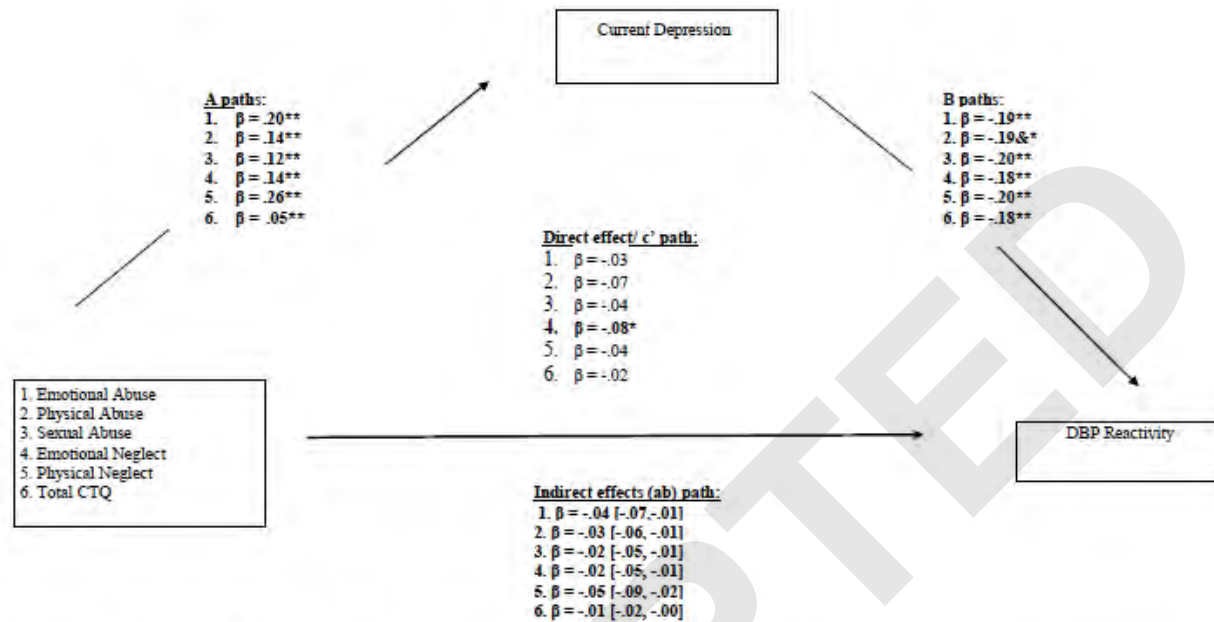


Figure 2: Mediation path diagram: Early life adversity and DBP reactivity.

Note: Each mediation analysis includes age, smoking, body mass index and baseline DBP as covariates. Statistics refer to unstandardized betas (β) and 95% confidence intervals at the lower and upper limit for indirect effects. The A path represents the association between each independent variable and the mediator. Path B reflects the association between the mediator and DBP reactivity. Path C' represents the direct effect between each independent variable and DBP reactivity while holding the mediator and covariates constant. Finally, Path ab represents the indirect effect of each independent variable on DBP reactivity through current depressive symptoms. Significant effects are highlighted in bold text. * $p < 0.05$, ** $p < 0.01$.

Figure 3

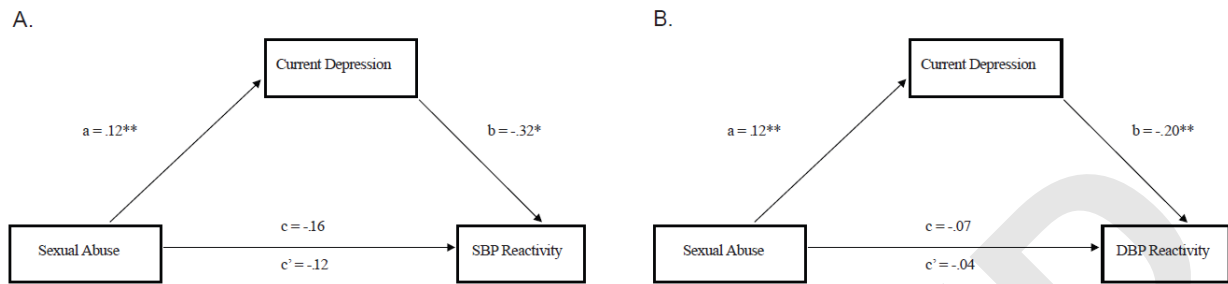


Table 1*Participant characteristics and demographics*

Items	N	Mean (SD)	Range
Age (years)	639	56.00 (11.10)	35 - 85 years
Body mass index (kg/m ²)	639	30.28 (6.81)	
Sex			
Male	279	56.39 (11.34)	
Female	360	55.70 (10.91)	
Ethnicity			
Caucasian	429		
African American	153		
Native American	3		
Asian/Pacific Islander	1		
Multiracial	7		
Other	12		
Employment status			
Employed	332		
Unemployed	152		
Education level			
High school	121		
College (attending)	106		
Associates degree	35		
Bachelor's degree	121		
Some graduate school	18		
Masters	64		
PhD	20		

Note: For sex, age, and BMI, $N = 639$. For ethnicity, $N = 605$ and for socioeconomic measures, education level, $N = 485$ and employment status, $N = 484$.

Table 2*Descriptive statistics and correlational analysis for study variables*

	Mean (SD)	Range	CES-D DA	CTQ-EA	CTQ-PA	CTQ-SA	CTQ-EN	CTQ-PN	CTQ- Total	SBP	DBP	HR
1. CES-D DA	2.1(3.24)	0-20	-	.326**	.210**	.193**	.253**	.282**	.319**	-.187**	-.218**	-.090**
2. CTQ-EA	8.4(4.4)	5-25	-	-	.728**	.470**	.669**	.547**	.870**	-.156**	-.134**	-.066
3. CTQ-PA	7.2(3.4)	5-25	-	-	-	.521**	.541**	.534**	.826**	-.158**	-.140**	-.059
4. CTQ-SA	6.8(4.4)	5-25	-	-	-	-	.326**	.385**	.690**	-.089*	-.093*	-.093*
5. CTQ-EN	10.1(4.8)	5-25	-	-	-	-	-	.643**	.816**	-.155**	-.167**	-.054
6. CTQ-PN	7.1(3.1)	5-21	-	-	-	-	-	-	.758**	-.097*	-.100*	-.055
7. CTQ Total	39.7(15.9)	25-114	-	-	-	-	-	-	-	-.167**	-.163**	-.083*
8. SBP reactivity	11.8(11.5)	-	-	-	-	-	-	-	-	-	.824**	.161**
9. DBP reactivity	5.8(4.3)	-	-	-	-	-	-	-	-	-	-	.260**
10. HR reactivity	3.5(3.3)	-	-	-	-	-	-	-	-	-	-	-

Note: The sample size was $N = 639$ on all measures except CES-D depressive affect ($N = 638$).

* $p < 0.05$ level.

** $p < 0.01$ level.

Table 3

Mean (SD) values of systolic blood pressure (SBP), diastolic blood pressure (DBP), heart rate (HR), during baseline, in response to stress task and recovery by sex.

	Male	Female
SBP (mmHg)		
Baseline	127.25 (16.63)	121.38 (17.67)
Task	140.26 (20.16)	132.35 (20.06)
Recovery	129.36 (17.42)	122.46 (17.56)
DBP (mmHg)		
Baseline	62.51 (10.43)	59.75 (10.43)
Task	68.46 (10.86)	65.49 (10.81)
Recovery	64.21 (10.71)	61.55 (10.72)
HR (mmHg)		
Baseline	70.55 (10.53)	74.59 (10.22)
Task	73.88 (10.73)	78.28 (10.57)
Recovery	71.32 (10.46)	74.99 (10.21)

Note: Number of males ($n = 274$) and females ($n = 349$) on all cardiovascular measures.

Table 4

Mediational pathways on cardiovascular variables

	SBP Reactivity									DBP Reactivity									HR Reactivity								
	<u>Total effect (c)</u>			<u>Direct effect (c')</u>			<u>Indirect effect (ab)</u>			<u>Total effect (c)</u>			<u>Direct effect (c')</u>			<u>Indirect effect (ab)</u>			<u>Total effect (c)</u>			<u>Direct effect (c')</u>			<u>Indirect effect (ab)</u>		
	β	<i>p</i>	95% CI	β	<i>p</i>	95% CI	β	95% CI	β	<i>p</i>	95% CI	β	<i>p</i>	95% CI	β	95% CI	β	<i>p</i>	95% CI	β	<i>p</i>	95% CI	β	<i>p</i>	95% CI	β	95% CI
EA	-.20	.05*	-.40, -.00	-.14	.176	-.34, .06	-.06	-.13, -.01	-.07	.067	-.15, .00	-.03	.429	-.11, .05	-.04	-.07, -.01	-.10	.313	-.29, .09	-.06	.582	-.26, .14	-.04	-.11, .01			
PA	-.30	.02*	-.55, -.05	-.25	.053	-.50, .00	-.05	-.11, -.01	-.10	.04*	-.20, -.00	-.07	.139	-.17, .02	-.03	-.06, -.01	-.17	.184	-.42, .08	-.14	.285	-.39, .11	-.03	-.09, .01			
SA	-.16	.116	-.35, .04	-.12	.248	-.31, .08	-.04	-.09, -.01	-.07	.078	-.14, .01	-.04	.260	-.12, .03	-.02	-.05, -.01	-.10	.278	-.30, .08	-.08	.429	-.27, .11	-.03	-.07, .01			
EN	-.24	.009*	-.41, -.06	-.19	.03*	-.38, -.01	-.04	-.09, -.01	-.11	.002**	-.18, -.04	-.08	.02*	-.15, -.01	-.02	-.05, -.01	-.07	.446	-.25, .11	-.04	.691	-.22, .14	-.03	-.08, .01			
PN	-.19	.187	-.46, .09	-.10	.495	-.38, .19	-.09	-.17, -.02	-.09	.092	-.20, .01	-.04	.478	-.15, .07	-.05	-.09, -.02	-.06	.663	-.34, .21	-.002	.991	-.28, .28	-.06	-.15, .01			
Total CTQ	-.07	.01*	-.12, -.02	-.05	.056	-.11, .00	-.02	-.03, -.00	-.03	.006	-.05, -.01	-.02	.081	-.04, .00	-.01	-.02, -.00	-.03	.234	-.09, .02	-.02	.463	-.08, .03	-.01	-.03, .00			

Note. Path a represents the pathway from ELA factors to depression in the mediation model, while path b represents the pathway from depression to CVR parameters. The indirect pathway (ab) represents the effects of ELA on CVR through current depressive symptoms and was significant for SBP and DBP reactivity, as it does not pass through zero.

* $p < 0.05$ level.

** $p < 0.01$ level.