Journal of Pediatric Psychology, 41(1), 2016, 5–14
doi: 10.1093/jpepsy/jsv040
Advance Access Publication Date: 15 May 2015
Original Research Article

Child Abuse, Resting Blood Pressure, and Blood Pressure Reactivity to Psychosocial Stress

Holly C. Gooding,[1,2] MD, MSc, Carly E. Milliren,[3] MPH, S. Bryn Austin,[1,2,4] ScD, Margaret A. Sheridan,[2,5] PhD, and Katie A. McLaughlin,[6] PhD

[1]Division of Adolescent and Young Adult Medicine, Boston Children's Hospital, [2]Department of Pediatrics, Harvard Medical School, [3]Clinical Research Center, Boston Children's Hospital, [4]Department of Social and Behavioral Sciences, Harvard School of Public Health, [5]Division of Developmental Medicine, Boston Children's Hospital, and [6]Department of Psychology, University of Washington

All correspondence concerning this article should be addressed to Holly C. Gooding, MD, MSc, Division of Adolescent and Young Adult Medicine, Boston Children's Hospital, 300 Longwood Avenue, Boston, MA 02115, USA. E-mail: holly.gooding@childrens.harvard.edu

Received November 10, 2014; revisions received April 8, 2015; accepted April 13, 2015

Abstract

Objective Childhood trauma is associated with hypertension in adults. It is unknown whether childhood trauma predicts elevated blood pressure earlier in development. We investigated whether the trauma of child abuse was associated with blood pressure in adolescents. **Methods** The sample included 145 adolescents aged 13–17 years, 40% with exposure to child abuse. The mean age of participants was 14.93 years ($SD = 1.33$); 58% were female. The majority self-identified as non-Hispanic White (43%), with the remainder identifying as non-Hispanic Black (17%), Hispanic (17%), or other/mixed race (23%). We used established age/sex/height-specific cutoffs to determine the prevalence of prehypertension and hypertension in the sample. We used two-sample *t* tests to examine associations of abuse with resting systolic blood pressure (SBP) and diastolic blood pressure (DBP) and blood pressure reactivity to the Trier Social Stress Test and a frustration task. We used linear regression to adjust for potential confounders including socio-demographic variables, body mass index, smoking, and psychopathology. **Results** Mean resting SBP and DBP were 114.07 mmHg and 61.35 mmHg in those with a history of abuse and 111.39 mmHg and 56.89 mmHg in those without a history of abuse. This difference was significant for DBP only. Twelve percent of participants met criteria for prehypertension or hypertension based on resting blood pressure values; this did not differ between those with and without an abuse history. Child abuse was associated with lower DBP and SBP reactivity to laboratory stress tasks and reduced DBP reactivity to frustration. These associations were robust to adjustment for potential confounders. **Conclusions** Child abuse is associated with higher resting DBP and blunted DBP and SBP reactivity to laboratory stress in adolescence. These findings suggest a potential pathway by which child abuse leads to hypertension.

Key words: adolescent; child abuse/psychology; public health.

Introduction

Epidemiologic research has consistently identified an association between childhood trauma exposure—including child abuse—and cardiovascular disease in adults (Afifi, Mota, Macmillan, & Sareen, 2013; Felitti et al., 1998). The relationship between childhood trauma and cardiovascular disease appears to be mediated by traditional cardiovascular risk factors, including obesity, diabetes, and hypertension (Danese et al., 2009; Rich-Edwards et al., 2012). Hypertension in particular has been consistently related to early childhood trauma in middle-aged adults (Lehman, Taylor, Kiefe, & Seeman, 2009; Riley, Wright, Jun, Hibert, & Rich- Edwards, 2010; Stein et al., 2010; Suglia, Clark, Boynton-Jarrett, Kressin, & Koenen, 2014). Hypertension is one of the strongest risk factors for the development of cardiovascular disease (Lewington, Clarke, Qizilbash, Peto, & Collins, 2002), and there is growing evidence that long-term fluctuations in blood pressure are associated with cardiovascular mortality as well (Suchy-Dicey et al., 2013). Thus further understanding of the relationship between childhood trauma and dynamic blood pressure changes has important implications for chronic disease prevention.

Several physiologic mechanisms might link childhood trauma to alterations in blood pressure. Individuals with a history of child abuse demonstrate changes in both the hypothalamic-pituitary-adrenal (HPA) axis and the sympathetic nervous system (Taylor, 2010), both of which are critical for blood pressure regulation (Brooks & Osborn, 1995; Phoon, Tam, Brown, & Whitworth, 1997). While activation of these systems is part of the normal physiological response to stress, excessive stimulation can lead to salt retention and vasoconstriction via increases in renin and aldosterone and via direct effects on the vasculature. Furthermore, childhood trauma is associated with elevations in pro-inflammatory cytokines (Slopen, Kubzansky, McLaughlin, & Koenen, 2013), which over time could produce vascular inflammation and elevations in blood pressure. Repeated activation of these hormonal and inflammatory systems is postulated to occur in those with a history of child abuse owing to heightened attention to and perception of threat over time (Pollak & Tolley-Schell, 2003; Pollak, Vardi, Putzer, Bechner, & Curtin, 2005), leading to chronic vigilance. While this activation could cause short-term elevations in blood pressure with each perceived threat, it may also lead to long-term changes in blood pressure reactivity owing to downregulation of receptors in the pituitary following exposure to excessive levels of corticotropin-releasing hormone from chronic threat perception (Heim, Newport, Bonsall, Miller, & Nemeroff, 2001).

Evidence that child maltreatment might influence blood pressure and other vascular responses to stress comes from recent work from our group. We applied a theoretical model developed in social psychology—the biopsychosocial model of challenge and threat (Mendes, Major, McCoy, & Blascovich, 2008)—to differentiate between adaptive and maladaptive patterns of physiological reactivity to stress in youth with and without a history of childhood maltreatment. We found that adolescents with a history of child abuse demonstrated reduced cardiac output reactivity and elevated peripheral resistance reactivity in response to a standard laboratory stress task (McLaughlin, Sheridan, Alves, & Mendes, 2014), consistent with a pattern of elevated threat perception and reduced cardiovascular efficiency (Mendes, Major, McCoy, & Blascovich, 2008). As blood pressure is a reflection of both cardiac output and total peripheral resistance, in this article we further focus on how this combination of physiologic changes affects both resting blood pressure and blood pressure response to the laboratory stress task in the same sample of adolescents.

Behavioral patterns that emerge in those with a history of child abuse are also likely to play a role in the development of hypertension. Individuals with a history of child abuse are more likely to be obese (Midei & Matthews, 2011; Vamosi, Heitmann, & Kyvik, 2009), to smoke (Topitzes, Mersky, & Reynolds, 2010), and to use alcohol to excess (Widom, White, Czaja, & Marmorstein, 2007), each of which are known risk factors for hypertension. These maladaptive patterns often emerge in adolescence (Burke, Hellman, Scott, Weems, & Carrion, 2011; Jun et al., 2008, 2012), which is already a vulnerable time for the initiation of behavioral changes that have long-term implications for cardiovascular health (Shay et al., 2012). Finally, child abuse is a known risk factor for depression and anxiety (Lindert et al., 2014; McLaughlin et al., 2012) both of which have been associated with hypertension later in life (Stein et al., 2014).

The current study aimed to investigate associations between child abuse history and blood pressure parameters in adolescents, a key developmental period for the convergence of heightened physiologic reactivity to psychosocial stressors (Gunnar & Quevedo, 2007; Stroud et al., 2009) and emergence of cardiovascular risk factors (Shay et al., 2012). We aimed first to characterize the relationship between child abuse exposure—including physical, sexual, and emotional abuse—and systolic blood pressure (SBP) and diastolic blood pressure (DBP) measured at rest. We then aimed to characterize differences in the magnitude of the SBP and DBP response to a standard laboratory stress task between adolescents with and without a history of child abuse. Finally, we aimed to investigate whether any associations seen were attenuated after accounting for body mass index (BMI), smoking status, or psychopathology, including depression, anxiety, and posttraumatic stress disorder (PTSD) symptoms.

Methods

Study Population

A community-based sample of 168 adolescents aged 13–17 years was recruited for participation in a parent study of stress reactivity after childhood maltreatment. Adolescents were recruited using flyers from schools and after-school programs in Boston and Cambridge, MA. Adolescents were also identified via a previously established research registry in a general adolescent medicine clinic and via chart review in a general psychiatry clinic. Recruitment efforts were targeted at recruiting a sample with high racial/ethnic diversity as well as variability in exposure to adversity. Participants were told this was a study of emotional experiences, emotions, and health and were reimbursed $50 for their participation. No participant was currently experiencing maltreatment, and the proper authorities were contacted in cases where there were safety concerns. Informed consent was obtained from the parent/guardian who attended the session, and assent was provided by adolescents. This study was approved by the Boston Children's Hospital Office of Clinical Investigation.

Equipment malfunctions resulted in loss of all autonomic data from eight participants. An additional 15 participants were missing data on key covariates including self-reported height and/or weight or race. The final sample for the current analysis included 145 participants from the parent study. The mean age of participants in the analytic sample was 14.93 years ($SD = 1.33$) and 58% ($n = 84$) were female. The majority self-identified as non-Hispanic White ($n = 63$, 43%) with the remainder identifying as non-Hispanic Black ($n = 24$, 17%), Hispanic ($n = 24$, 17%), or other/mixed race ($n = 34$, 23%).

Child Abuse Exposure

Child abuse was assessed using a self-report questionnaire and an interview. First, we administered the Childhood Trauma Questionnaire (CTQ; Bernstein, Ahluvalia, Pogge, & Handelsman, 1997). The CTQ is a 28-item scale that assesses the frequency of maltreatment exposure during childhood and adolescence, including physical, sexual, and emotional abuse and physical and emotional neglect. Items are rated on a Likert scale from 1 (*never true*) to 5 (*very often true*); the scale range is from 28 to 140. The CTQ has excellent psychometric properties including internal consistency, test–retest reliability, and convergent and discriminant validity with interviews and clinician reports of maltreatment (Bernstein et al., 1997; Bernstein, Fink, Hondelsman, Foote, & Lovejoy, 1994) Second, one trained research assistant administered the Childhood Experiences of Care and Abuse (CECA) interview (Bifulco, Brown, & Harris, 1994; Bifulco, Brown, Lillie, & Jarvis, 1997). The CECA assesses multiple aspects of caregiving experiences, including abuse and neglect. Interrater reliability for maltreatment reports is excellent, and multiple validation studies suggest high agreement between siblings on reports of caregiver behaviors and maltreatment (Bifulco et al., 1994, 1997). Participants who reported any physical or sexual abuse during the CECA interview or who had a score on the CTQ abuse subscales of ≥ 8 (for physical and sexual abuse) or ≥ 10 (for emotional abuse) were classified as having experienced abuse. These CTQ subscale cut points were chosen because they have previously been shown to maximize sensitivity and specificity for the abuse types when compared with a gold-standard clinical interview (Walker et al., 1999).

Physiological Measures

Participants were instructed to abstain from caffeine intake for 4 hr before the study and from all food intake for 1 hr before the study. All physiologic measures were obtained between 1 and 7 pm. A Colin Prodigy II oscillometric blood pressure machine (Colin Medical Instruments, San Antonio, TX) was used to obtain blood pressure recordings at predetermined times during the study protocol, as follows. Baseline physiological data were first collected during a 5-min period in which participants were asked to sit quietly. Adolescents then completed questionnaire and interview measures and resting blood pressures were again taken. The average of the second, third, and fourth SBP and DBP taken during these two periods comprised the resting SBP and DBP variables; as is customary in analyses of repeated blood pressures, the first readings were discarded owing to concerns they may be falsely elevated as participants adjust to the study environment. Resting SBP and DBP are presented as z-scores for each participant's age, sex, and height as recommended by the National Heart, Lung, Blood Institute Fourth Report on the diagnosis, evaluation, and treatment of high blood pressure in children and adolescents (National High Blood Pressure Education Program Working Group on High Blood Pressure in Children and Adolescents, 2004).

Participants with either SBP or DBP between the 90th and 95th percentile for age, sex, and height were classified as prehypertensive, and participants with either SBP or DBP $\geq$ 95th% for age, sex, and height were classified as hypertensive (National High Blood Pressure Education Program Working Group on High Blood Pressure in Children and Adolescents, 2004). For example, a 15-year-old girl at the median height for age would be classified as pre-hypertensive at a blood pressure of 123/79 mmHg, and as hypertensive at a blood pressure of 127/83 mmHg. A 15-year-old boy at the median height for age would be classified as pre-hypertensive at a blood pressure of 127/79 mmHg and as hypertensive at a blood pressure of 131/83 mmHg.

Procedure

Participants completed the Trier Social Stress Task (TSST; Kirschbaum, Pirke, & Hellhammer, 1993), a stress induction procedure that has been used with children and adolescents (Buske-Kirschbaum et al., 1997; Stroud et al., 2009). The TSST involves three periods. After being told that they would be delivering a speech in front of trained evaluators who would judge their performance, participants were given 5-min to prepare their speech. In the current study, participants were asked to talk about the qualities of a good friend and which of those characteristics they did and did not possess. Next, participants delivered a 5-min speech in front of two evaluators. Evaluators were trained to provide neutral and mildly negative feedback (e.g., appearing bored) during the speech. Participants then completed a mental subtraction task out loud in front of the evaluators for 5 min. Specifically, participants were asked to count backward in steps of seven from a three-digit number and were stopped and asked to start again each time they made a mistake. Following the TSST, participants completed a 5-min rest period to facilitate a return to baseline for all physiological markers. Participants then completed a frustration task. They were told that they would be playing a game in which a number appeared on a computer screen, and that they needed to press that same number on the keyboard as quickly as possible. Correct responses provided within the time allowed for a response were followed by a green smiling face and a positive noise; incorrect responses and those that occurred outside the allowable time window were followed by red frowning face and an irritating noise. The task had two distinct phases. During the first phase, participants were given accurate feedback about their performance and had an 800-ms window in which to provide a response. During the second phase, participants had a shortened window in which to respond (600 ms) and received inaccurate feedback about their performance on 30% of trials (i.e., correct responses were given feedback indicating an inaccurate response). Together, this resulted in a high proportion of feedback indicating incorrect responses during the second phase. Blood pressure recordings were sampled during the first and fourth minutes of each period and are presented as a percent change from the baseline blood pressure for each individual participant.

Covariates and Potential Mediators

Participants self-reported their race and whether they were of Hispanic/Latino origin. Parents provided information on the highest parental education achieved by the primary caregiver/guardian and partner. Participants were asked "Are you a current smoker, ex-smoker, or have you ever smoked?" and were designated smokers if they reported any current smoking. Participants were asked to self-report their height in inches and weight in pounds. The BMI for each participant was calculated as [(weight in pounds/(height in inches)^2 × 703]. Age- and sex-specific BMI z-scores were created using Centers for Disease Control standard growth charts (Kuczmarski et al., 2002). Depressive symptoms were assessed via the Children's Depression Inventory (CDI; Kovacs, 1985) and anxiety symptoms were assessed via the Multidimensional Anxiety Scale for Children (MASC; March, Parker, Sullivan, Stallings, & Conners, 1997). PTSD symptoms were assessed using the PTSD subscale of the Youth Self Report form of the Child Behavior Checklist (YSR/CBCL) (Achenbach, 1991; Achenbach & Rescorla, 2001).

Statistical Analysis

We first examined the distribution of sociodemographic variables, baseline physiologic characteristics, and scores on the psychopathology scales by abuse history using chi-square tests and two-sample *t* tests. For our first aim, we used two-sample *t* tests to test for associations between abuse history and resting SBP and DBP. For our second aim, we used two-sample *t* tests to test for associations between abuse history and percent change in SBP and DBP in response to the TSST and the frustration task. For our third aim, we used multivariate linear regression to test for associations between abuse history and resting SBP and DBP z-scores as well as SBP and DBP reactivity adjusting for sociodemographic characteristics (age, sex, race/ethnicity, and parental education, Model 1), for continuous BMI and smoking status (Model 2), and for continuous scores on the CDI, MASC, and YSR/CBCL (Model 3). Because participants were included even if missing physiologic data from one part of the TSST, the total sample size for each comparison was allowed to vary. All analyses were conducted in STATA version 13 (College Station, TX).

Results

Descriptive Statistics

The mean age of participants was 14.93 years ($SD = 1.33$) and 58% ($n = 86$) were female. Additional sociodemographic, physiologic, and psychological characteristics of the total sample are found in the first column of Table I. BMI z-score, depressive symptoms, and PTSD symptoms were each associated with both resting SBP z-score ($r = .55$, $p < .001$; $r = .20$, $p = .02$; and $r = .20$, $p = 0.01$, respectively) and resting DBP z-score ($r = .34$, $p < .001$; $r = .26$ $p = .001$; and $r = .22$, $p = .007$, respectively). Resting SBP z-score was also associated with race/ethnicity

Table I. Distribution of Sociodemographic, Physiologic, and Psychological Characteristics by Child Abuse History in Adolescents Aged 13–17 Years ($n = 145$)

Participant characteristic	*n* (%) or mean (*SD*)			*p*-value*
	Total sample (*N* = 145)	Reported any abuse 58 (40%)	No abuse reported 87 (60%)	
Age (years)	14.93 (1.33)	15.26 (1.31)	14.71 (1.31)	.02
Gender				.13
Male	61 (42%)	20 (33%)	41 (67%)	
Female	84 (58%)	38 (45%)	46 (55%)	
Race/ethnicity				.04
White	63 (43%)	17 (27%)	46 (73%)	
Black	24 (17%)	13 (54%)	11 (46%)	
Hispanic	24 (17%)	12 (50%)	12 (50%)	
Other	34 (23%)	16 (47%)	18 (53%)	
Parental education				.003
Less than high school	63 (44%)	14 (22 %)	49 (78%)	
High school grad/equivalent	21 (15%)	13 (62%)	8 (38%)	
Some college	25 (17%)	13 (52%)	12 (48%)	
College grad	29 (20%)	14 (48%)	15 (52%)	
Unknown	7 (5%)	4 (57%)	3 (43%)	
Smoker				.001
No	111 (77%)	36 (32%)	75 (68%)	
Yes	34 (23%)	22 (65%)	12 (35%)	
BMI	22.26 (5.01)	22.77 (4. 35)	21.91 (5.41)	.32
BMI z-score	0.44 (0.95)	0.55 (0.92)	0.36 (0.97)	.24
Child Depression Inventory	9.17 (7.47)	13.05 (8.78)	6.59 (5.05)	<.001
Multidimensional Anxiety Scale for Children	37.73 (14.02)	40.55 (14.40)	35.85 (13.52)	.05
PTSD subscale of YSR/CBCL	56.90 (7.25)	59.47 (7.97)	55.20 (6.21)	<.001

Note. Chi-square test, two-sample *t* test, where appropriate.
PTSD = posttraumatic stress disorder; YSR/CBCL = youth self-report form of Child Behavior Checklist.
**p*-values from testing for association with dichotomous abuse.

($p = .008$), and resting DBP z-score was also associated with anxiety symptoms ($r = .20$, $p = .02$).

Forty percent ($n = 58$) of the sample reported any type of abuse, 18% of the sample reported physical abuse, 30% reported emotional abuse, and 12% reported sexual abuse. The three types of abuse commonly co-occurred, with 10% of those reporting abuse (4% of the sample overall) reporting all three types. Any abuse history was more common in those who were older, those with non-White race/ethnicity, and those whose parent had completed high school or beyond. Sixty-five percent of smokers reported abuse compared with 32% of non-smokers. Participants reporting abuse also had higher depression and PTSD scores than those who did not report any abuse.

Child Abuse and Resting Blood Pressure

We first compared the resting blood pressure of adolescents with and without a history of abuse. Mean resting SBP and DBP values, z-scores, and percentiles are reported in Table II for the overall sample and for those with and without an abuse history. No differences in resting SBP values were observed according to child abuse history. In contrast, mean resting DBP, DBP z-score, and DBP percentile were higher in participants exposed to any type of child abuse.

Six percent of participants met criteria for prehypertension and 7% met criteria for hypertension based on age-, sex-, and height-specific resting SBP or DBP percentile. Adolescents with an abuse history were no more likely to meet criteria for prehypertension or hypertension using these standard clinical cutoffs (National High Blood Pressure Education Working Group on High Blood Pressure in Children and Adolescents, 2004).

Child Abuse and Blood Pressure Reactivity to Laboratory Stress

We then compared differences in the magnitude of the SBP and DBP response to a standard laboratory stress task between adolescents with and without a history of child abuse. The majority of participants demonstrated the anticipated increase in SBP and DBP with each of the three portions of the laboratory stress task and the frustration task. Mean percent change in SBP and DBP from baseline for the sample and by abuse history are found in Table II. History of any child abuse was associated with a smaller increase in SBP from baseline in response to the speech and math tasks compared with nonabused youth (Table II). History of child abuse was associated with a smaller increase from baseline DBP in

Table II. Systolic and Diastolic Blood Pressures at Rest and With Each Task by Child Abuse History

Blood pressure parameter	Mean (*SD*)			*p*-value*
	Total sample (*N* = 145)	Reported any abuse 58 (40%)	No abuse reported 87 (60%)	
Systolic				
Resting (mmHg)	112.46 (11.44)	114.07 (11.90)	111.39 (11.06)	.17
Resting z-score	0.01 (1.02)	0.18 (1.11)	−0.09 (0.94)	.11
Resting percentile	49.47 (29.43)	55.09 (30.98)	45.74 (27.90)	.06
Speech preparation (% change from resting)	13.58 (11.30)	12.20 (10.15)	14.44 (11.94)	.26
Speech (% change from resting)	25.70 (16.20)	**20.66 (15.47)**	**28.97 (15.90)**	**.003**
Math (% change from resting)	21.43 (12.55)	**17.85 (12.54)**	**23.85 (12.04)**	**.005**
Frustration (% change from resting)	7.82 (12.01)	6.41 (10.66)	8.75 (12.81)	.27
Diastolic				
Resting (mmHg)	58.67 (7.91)	**61.35 (7.89)**	**56.89 (7.45)**	**<.001**
Resting z-score	−0.61 (0.70)	**−0.38 (0.72)**	**−0.76 (0.64)**	**.001**
Resting percentile	30.35 (20.89)	**36.88 (22.53)**	**25.99 (18.61)**	**.002**
Speech preparation (% change from resting)	14.84 (14.15)	**10.98 (12.04)**	**17.24 (14.88)**	**.01**
Speech (% change from resting)	26.84 (20.99)	**18.19 (22.22)**	**32.43 (18.20)**	**<.001**
Math (% change from resting)	22. 36 (18.55)	**16.15 (14.85)**	**26.48 (19. 66)**	**.001**
Frustration (% change from resting)	4.51 (19.59)	**−2.88 (13.72)**	**9.53 (21.40)**	**<.001**

Note. Values in bold differ significantly between groups with and without abuse exposure in unadjusted analyses.
**p*-values from two-sample *t*-test testing for association with child abuse.

Table III. Associations Between any Child Abuse, Resting Blood Pressure, and Blood Pressure Response to a Laboratory Stress Task in a Sample of Adolescents Aged 13–17 years ($n = 145$)

	Model 1			Model 2[a]			Model 3[b]		
	B	SE	*p*-value	β	SE	*p*-value	β	SE	*p*-value
Systolic blood pressure[c]									
Resting z-score	0.30	0.20	0.113	0.10	0.20	0.586	−0.03	0.20	.861
% Change speech preparation	−2.20	2.00	0.256	−2.30	2.20	0.294	−1.20	2.40	.605
% Change speech task	**−8.30**	**2.80**	**0.003**	**−7.00**	**3.00**	**0.022**	−5.00	3.20	.129
% Change math task	**−6.00**	**2.10**	**0.005**	**−6.80**	**2.20**	**0.003**	**−5.40**	**2.40**	**.03**
% Change frustration task	−2.30	2.20	0.274	−2.50	2.30	0.280	−1.60	2.50	.521
Diastolic blood pressure[c]									
Resting z-score	**0.40**	**0.10**	**0.001**	**0.30**	**0.10**	**0.013**	0.20	0.10	.066
% Change speech preparation	**−6.30**	**2.40**	**0.010**	**−6.80**	**2.60**	**0.011**	**−5.80**	**2.80**	**.042**
% Change speech task	**−14.20**	**3.50**	**<0.001**	**−13.10**	**3.80**	**0.001**	**−11.80**	**4.20**	**.006**
% Change math task	**−10.30**	**3.10**	**0.001**	**−9.40**	**3.30**	**0.005**	−6.40	3.60	.076
% Change frustration task	**−12.40**	**3.30**	**<0.001**	**−11.80**	**3.60**	**0.001**	**−9.00**	**3.80**	**.021**

Note. β = change per one unit in blood pressure z-score (resting variables) or percent change from resting (laboratory stress variables).
SE = standard error.
[a]Adjusted for age, sex, race/ethnicity, parental education, body mass index z-score, and smoking status.
[b]Adjusted for age, sex, race/ethnicity, parental education, Child Depression Inventory, Multidimensional Anxiety Scale for Children, and Posttraumatic Stress Disorder Subscale of the Youth Self Report form of the Child Behavior Checklist.
[c]Referent group is nonabused. Adjusted for age, sex, race/ethnicity, parental education. Values in bold differ significantly the referent group at $p < .05$.

response to all laboratory stress tasks and the frustration tasks compared with nonabused youth (Table II).

Potential Confounders of the Relationship Between Child Abuse and Blood Pressure

Finally, we assessed the impact of controlling for sociodemographic variables, BMI, smoking status, and psychopathology in our model. The associations between any child abuse history and higher resting DBP z-score and any child abuse history and decreased DBP reactivity were still observed after controlling for sociodemographic variables (Table III, Model 1). After additional adjustment for BMI and smoking (Table III, Model 2) and depression, anxiety, and PTSD symptoms (Table III, Model 3), decreased DBP reactivity was still observed during speech preparation, speech, and frustration tasks, although the associations were somewhat attenuated. The effects were no longer significant for resting DBP z-score and DBP response to the math task after adjustment for psychological factors. Decreased reactivity in SBP in response to the math task was robust to controls for these potential confounders, but decreased SBP reactivity to the speech task was no longer significant after adjustment for the psychological factors.

Discussion

Child trauma exposure is associated with a wide range of adverse physical health outcomes in adulthood, including elevated rates of hypertension (Riley et al., 2010; Stein et al., 2010) and higher risk for developing cardiovascular disease (Afifi et al., 2013; Dong et al., 2004). However, it is unknown when differences in risk for cardiovascular disease as a function of child trauma emerge in the life course and whether early markers of cardiovascular disease risk such as hypertension are evident by adolescence. In the current study, exposure to child abuse was associated with higher resting DBP and lower SBP and DBP reactivity to several distinct laboratory stressors. Many of these associations remained significant after controlling for sociodemographic factors and potential confounders, including smoking, BMI, depression, anxiety, and PTSD symptoms. Our findings suggest that maltreated youth may experience both chronically elevated DBP and loss of the flexible variation in blood pressure that is typical during states of heightened arousal. Together, these findings have implications for understanding pathways linking child trauma to cardiovascular health.

The more robust association found between child maltreatment and DBP as opposed to SBP is consistent with the epidemiology of elevated blood pressure in youth, as well as studies of other types of childhood adversity and blood pressure in this age-group (Su et al., 2014). Diastolic hypertension is more common than systolic hypertension in younger individuals (Franklin, Jacobs, Wong, L'Italien, & Lapuerta, 2001). While some have debated the significance of isolated diastolic hypertension, there is good evidence that the majority of people with diastolic hypertension will develop systolic hypertension over time (Franklin et al., 2005) and that diastolic hypertension itself is associated with increased risk of stroke (Fang et al., 2006). Although the higher resting DBP z-scores seen in those with a history of child abuse in our study did not translate into higher rates of diastolic hypertension in this cohort, likely owing to the relatively young age of our sample, persistently elevated DBP over time would be expected to result in progression to hypertension and elevated cardiovascular risk in later adulthood for a number of individuals. Whether changes in blood pressure variability in adolescence lead to cardiovascular disease is less certain (Stabouli et al., 2015; Suchy-Dicey et al., 2013).

There are several possible mechanisms for our findings. Su and colleagues recently demonstrated a graded association of the number of childhood adversities and levels of the vasoconstrictor plasma endothelin-1 in healthy adolescents and young adults (Su et al., 2014). Childhood adversity is associated with elevated markers of inflammation in adolescents and adults (Danese, Pariante, Caspi, Taylor, & Poulton, 2007; Slopen et al., 2013), which are in turn linked to arterial stiffness and hypertension (Virdis, Ghiadoni, Plantinga, Taddei, & Salvetti, 2007). Some evidence suggests that youth who are exposed to maltreatment experience blunted physiological responses to stress including in the HPA axis and sympathetic nervous system (Gordis, Granger, Susman, & Trickett, 2008; Gunnar & Vazquez, 2001; Trickett, Gordis, Peckins, & Susman, 2014), a plausible mechanism for the decreased blood pressure variability seen in our study. Notably many of the associations between child abuse and blood pressure reactivity were attenuated after controlling for symptoms of internalizing psychopathology, suggesting a possible psychological link between abuse and hemodynamic changes. Finally, behavioral factors such as more sedentary lifestyles or greater intake of high-sodium foods in those with a history of abuse might play a role.

Notable strengths of the study include the use of well-validated measures of abuse exposure, multiple measures of blood pressure (both resting and in response to multiple types of laboratory stressors), a racially and ethnically diverse community sample of participants, and our focus on adolescence, a developmental period in which the association between child trauma and blood pressure has not previously been examined. However, our findings should also be interpreted in light of several important limitations. Our sample is small and may be underpowered to detect clinically meaningful differences in blood pressure z-scores. We did not have data on other variables related to both abuse and blood pressure, including drug use, family history of hypertension, and social support. These would be important inclusions in future studies. Importantly, the cross-sectional nature of the study does not allow us to conclude that child abuse causes an increase in resting blood pressure over time. Finally, the young age of the sample may have itself contributed to difficulty detecting variation in blood pressure owing to their relatively healthy state.

Chronic elevations in resting blood pressure and decreased blood pressure reactivity in the face of repeated stressors, as suggested by our study, might be an important mechanism underlying the well-described epidemiologic association between early child abuse and adult hypertension and heart disease. Raising awareness among clinicians on both sides of the equation—those who care for victims of abuse and those who screen for and treat hypertension—is necessary to begin to tackle this issue. While stopping ongoing abuse and attending to acute psychological needs are paramount, practitioners should be aware of the later life effects of child abuse and be sure victims are connected to primary medical care services. Similarly, clinicians who provide primary and cardiology care should be trained to take sensitive histories of early life adversities that impact the current care of their patients. While providing this type of trauma-informed care may be unfamiliar or uncomfortable for many physicians (Starecheski, 2015), it is essential for helping patients with the lifestyle changes and medication adherence necessary for treating hypertension. Finally, public health measures to address the root causes of child trauma and to minimize its many behavioral, psychological, and physiological outcomes are also needed.

In conclusion, exposure to physical, sexual, or emotional abuse was associated with elevated resting DBP and blunted SBP and DBP reactivity to a laboratory stress in this adolescent sample. Children with a history of trauma, including abuse, appear to be at increased risk for sustained blood pressure elevations in adolescence and, ultimately, the development of hypertension and cardiovascular disease in adulthood. There is a growing recognition of the long-term psychological and physical consequences of many types of childhood trauma, and this study supports the importance of considering vascular health in these frameworks. Future work aimed at modulating the impact of child trauma on vascular health is needed to prevent the long-term physiological effects of child abuse and promote cardiovascular health for all youth.

Acknowledgments

The authors thank Currie Touloumtzis for assistance with manuscript preparation and the research subjects and their families for their time.

Funding

This work was supported by the Mary A. and John M. McCarthy Foundation (to H.C.G.) and the National Institutes of Mental Health and the National Institutes of Health (K01-MH092526 to K.A.M.).

Conflicts of interest: None declared.

References

Achenbach, T. M. (1991). *Integrative guide for the 1991 CBCL/4-18, YSR and TRF Profiles*. Burlington, VT: Department of Psychiatry, University of Vermont.

Achenbach, T. M., & Rescorla, L. (2001). *The manual for the ASEBA school-age forms and profiles*. Burlington, VT: University of Vermont, Research Center for Children, Youth, and Families.

Afifi, T. O., Mota, N., Macmillan, H. L., & Sareen, J. (2013). Harsh physical punishment in childhood and adult physical health. *Pediatrics*, *132*(2),e333–e340.

Bernstein, D. P., Ahluvalia, T., Pogge, D., & Handelsman, L. (1997). Validity of the Childhood Trauma Questionnaire in an adolescent psychiatric population. *Journal of the American Academy of Child and Adolescent Psychiatry*, *36*, 340–348.

Bernstein, D. P., Fink, L., Hondelsman, L., Foote, J., & Lovejoy, M. (1994). Initial reliability and validity of a new retrospective measure of child abuse and neglect. *American Journal of Psychiatry*, *151*, 1132–1136.

Bifulco, A., Brown, G. W., & Harris, T. O. (1994). Childhood experiences of care and abuse (CECA): A retrospective interview measure. *Journal of Child Psychology and Psychiatry*, *35*, 1419–1435.

Bifulco, A., Brown, G. W., Lillie, A., & Jarvis, J. (1997). Memories of childhood neglect and abuse: Corroboration in a series of sisters. *Journal of Child Psychology and Psychiatry*, *38*, 365–374.

Brooks, V. L., & Osborn, J. W. (1995). Hormonal-sympathetic interactions in long-term regulation of arterial pressure: An hypothesis. *American Journal of Physiology*, *268*(6 Pt 2), R1343–R1358.

Burke, N. J., Hellman, J. L., Scott, B. G., Weems, C. F., & Carrion, V. G. (2011). The impact of adverse childhood experiences on an urban pediatric population. *Child Abuse and Neglect*, *35*, 408–413.

Buske-Kirschbaum, A., Jobst, S., Psych, D., Wustmans, A., Kirschbaum, C., & Rauh, W. (1997). Attenuated free cortisol response to psychosocial stress in children with atopic dermatitis. *Psychosomatic Medicine*, *59*, 419–426.

Danese, A., Moffitt, T. E., Harrington, H., Milne, B. J., Polanczyk, G., Pariante, C. M., … Caspi, A. (2009). Adverse childhood experiences and adult risk factors for age-related disease: Depression, inflammation, and clustering of metabolic risk markers. *Archives of Pediatrics and Adolescent Medicine*, *163*, 1135–1143.

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*, 1319–1324.

Dong, M., Giles, W. H., Felitti, V. J., Dube, S. R., Williams, J. E., Chapman, D. P., & Anda, R. F. (2004). Insights into causal pathways for ischemic heart disease: Adverse childhood experiences study. *Circulation*, *110*, 1761–1766.

Fang, X. H., Zhang, X. H., Yang, Q. D., Dai, X. Y., Su, F. Z., Rao, M. L., & Li, S. C. (2006). Subtype hypertension and risk of stroke in middle-aged and older Chinese: A 10-year follow-up study. *Stroke, 37*, 38–43.

Felitti, V. J., Anda, R. F., Nordenberg, D., Williamson, D. F., Spitz, A. M., Edwards, V., … 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*, 245–258.

Franklin, S. S., Jacobs, M. J., Wong, N. D., L'Italien, G. J., & Lapuerta, P. (2001). Predominance of isolated systolic hypertension among middle-aged and elderly US hypertensives: Analysis based on National Health and Nutrition Examination Survey (NHANES) III. *Hypertension*, *37*, 869–874.

Franklin, S. S., Pio, J. R., Wong, N. D., Larson, M. G., Leip, E. P., Vasan, R. S., & Levy, D. (2005). Predictors of new-onset diastolic and systolic hypertension: The Framingham Heart Study. *Circulation*, *111*, 1121–1127.

Gordis, E. B., Granger, D. A., Susman, E. J., & Trickett, P. K. (2008). Salivary alpha amylase-cortisol asymmetry in maltreated youth. *Hormones and Behavior*, *53*, 96–103.

Gunnar, M., & Quevedo, K. (2007). The neurobiology of stress and development. *Annual Review of Psychology*, *58*, 145–173.

Gunnar, M. R., & Vazquez, D. M. (2001). Low cortisol and a flattening of expected daytime rhythm: Potential indices of risk in human development. *Development and Psychopathology*, *13*, 515–538.

Jun, H. J., Corliss, H. L., Boynton-Jarrett, R., Spiegelman, D., Austin, S. B., & Wright, R. J. (2012). Growing up in a domestic violence environment: Relationship with developmental trajectories of body mass index during adolescence into young adulthood. *Journal of Epidemiology and Community Health*, *66*, 629–635.

Jun, H. J., Rich-Edwards, J. W., Boynton-Jarrett, R., Austin, S. B., Frazier, A. L., & Wright, R. J. (2008). Child abuse and smoking among young women: The importance of severity, accumulation, and timing. *Journal of Adolescent Health*, *43*, 55–63.

Heim, C., Newport, D. J., Bonsall, R., Miller, A. H., & Nemeroff, C. B. (2001). Altered pituitary-adrenal axis responses to provocative challenge tests in adult survivors of childhood abuse. *American Journal of Psychiatry*, *158*, 575–581.

Kirschbaum, C., Pirke, K.-M., & Hellhammer, D. (1993). The 'Trier Social Stress Test'—a tool for investigating psychobiological stress responses in a laboratory setting. *Neuropsychobiology*, *28*, 76–81.

Kovacs, M. (1985). The Children's Depression, Inventory (CDI). *Psychopharmacology Bulletin*, *21*, 995–998.

Kuczmarski, R. J., Ogden, C. L., Guo, S. S., Grummer-Strawn, L. M., Flegal, K. M., Mei, Z., & Johnson, C. L. (2002). 2000 CDC Growth Charts for the United States: Methods and development. *Vital and Health Statistics. Series 11: Data from the National Health Survey*, *246*, 1–190.

Lehman, B. J., Taylor, S. E., Kiefe, C. I., & Seeman, T. E. (2009). Relationship of early life stress and psychological functioning to blood pressure in the CARDIA study. *Health Psychology*, *28*, 338–346.

Lewington, S., Clarke, R., Qizilbash, N., Peto, R., & Collins, R. (2002). Age-specific relevance of usual blood pressure to vascular mortality: A meta-analysis of individual data for one million adults in 61 prospective studies. *Lancet*, *360*, 1903–1913.

Lindert, J., von Ehrenstein, O. S., Grashow, R., Gal, G., Braehler, E., & Weisskopf, M. G. (2014). Sexual and physical abuse in childhood is associated with depression and anxiety over the life course: Systematic review and meta-analysis. *International Journal of Public Health*, *59*, 359–372.

March, J. S., Parker, J. D., Sullivan, K., Stallings, P., & Conners, C. K. (1997). The Multidimensional Anxiety Scale for Children (MASC): Factor structure, reliability, and validity. *Journal of the American Academy of Child and Adolescent Psychiatry*, *36*, 554–565.

McLaughlin, K. A., Greif Green, J., Gruber, M. J., Sampson, N. A., Zaslavsky, A. M., & Kessler, R. C. (2012). Childhood adversities and first onset of psychiatric disorders in a national sample of US adolescents. *Archives of General Psychiatry*, *69*, 1151–1160.

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*, 538–546.

Mendes, W. B., Major, B., McCoy, S., & Blascovich, J. (2008). How attributional ambiguity shapes physiological and emotional responses to social rejection and acceptance. *Journal of Personality and Social Psychology*, *94*, 278–291.

Midei, A. J., & Matthews, K. A. (2011). Interpersonal violence in childhood as a risk factor for obesity: A systematic review of the literature and proposed pathways. *Obesity Reviews*, *12*, e159–e172.

National High Blood Pressure Education Program Working Group on High Blood Pressure in Children and Adolescents. (2004). The fourth report on the diagnosis, evaluation, and treatment of high blood pressure in children and adolescents. *Pediatrics*, *114*(Suppl. 2, 4th Report), 555–576.

Phoon, R. K., Tam, S. H., Brown, M. A., & Whitworth, J. A. (1997). The role of the hypothalamic-pituitary-adrenal (HPA) axis in the regulation of blood pressure. *Clinical and Experimental Hypertension*, *19*, 417–430.

Pollak, S. D., & Tolley-Schell, S. A. (2003). Selective attention to facial emotion in physically abused children. *Journal of Abnormal Psychology*, *112*, 323–338.

Pollak, S. D., Vardi, S., Putzer Bechner, A. M., & Curtin, J. J. (2005). Physically abused children's regulation of attention in response to hostility. *Child Development*, *76*, 968–977.

Rich-Edwards, J. W., Mason, S., Rexrode, K., Spiegelman, D., Hibert, E., Kawachi, I., & Wright, R. J. (2012). Physical and sexual abuse in childhood as predictors of early-onset cardiovascular events in women. *Circulation*, *126*, 920–927.

Riley, E. H., Wright, R. J., Jun, H. J., Hibert, E. N., & Rich-Edwards, J. W. (2010). Hypertension in adult survivors of child abuse: Observations from the Nurses' Health Study II. *Journal of Epidemiology and Community Health*, *64*, 413–418.

Shay, C. M., Ning, H., Allen, N. B., Carnethon, M. R., Chiuve, S. E., Greenlund, K. J., ... Lloyd-Jones, D. M. (2012). Status of cardiovascular health in US adults: Prevalence estimates from the National Health and Nutrition Examination Surveys (NHANES) 2003–2008. *Circulation*, *125*, 45–56.

Slopen, N., Kubzansky, L. D., McLaughlin, K. A., & Koenen, K. C. (2013). Childhood adversity and inflammatory processes in youth: A prospective study. *Psychoneuroendocrinology*, *38*, 188–200.

Stabouli, S., Papakatsika, S., Kotronis, G., Papadopoulou-Legbelou, K., Rizos, Z., & Kotsis, V. (2015). Arterial stiffness and SBP variability in children and adolescents. *Journal of Hypertension*, *33*, 88–95.

Starecheski, L. (2015). "10 Questions Some Doctors are Afraid to Ask" in What Shapes Health? from National Public Radio. http://www.npr.org/blogs/health/2015/03/03/377569539/even-some-doctors-fear-these-10-questions.

Stein, D. J., Aguilar-Gaxiola, S., Alonso, J., Bruffaerts, R., de Jonge, P., Liu, Z., ... Scott, K. M. (2014). Associations between mental disorders and subsequent onset of hypertension. *General Hospital Psychiatry*, *36*, 142–149.

Stein, D. J., Scott, K., Haro Abad, J. M., Aguilar-Gaxiola, S., Alonso, J., Angermeyer, M., … Von Korff, M. (2010). Early childhood adversity and later hypertension: Data from the World Mental Health Survey. *Annals of Clinical Psychiatry*, *22*, 19–28.

Stroud, L. R., Foster, E., Papandonatos, G. D., Handwerger, K., Granger, D. A., Kivlighan, K. T., & Niaura, R. (2009). Stress response and the adolescent transition: Performance versus peer rejection stressors. *Development and Psychopathology*, *21*, 47–68.

Su, S., Wang, X., Kapuku, G. K., Treiber, F. A., Pollock, D. M., Harshfield, G. A., & Pollock, J. S. (2014). Adverse childhood experiences are associated with detrimental hemodynamics and elevated circulating endothelin-1 in adolescents and young adults. *Hypertension*, *64*, 201–7.

Suchy-Dicey, A. M., Wallace, E. R., Mitchell, S. V., Aguilar, M., Gottesman, R. F., Rice, K., & Longstreth, W. T. Jr. (2013). Blood pressure variability and the risk of all-cause mortality, incident myocardial infarction, and incident stroke in the cardiovascular health study. *American Journal of Hypertension*, *26*, 1210–1217.

Suglia, S. F., Clark, C. J., Boynton-Jarrett, R., Kressin, N. R., & Koenen, K. C. (2014). Child maltreatment and hypertension in young adulthood. *BMC Public Health*, *14*, 1149.

Taylor, S. E. (2010). Mechanisms linking early life stress to adult health outcomes. *Proceedings of the National Academy of Sciences of the United States of America*, *107*, 8507–8512.

Topitzes, J., Mersky, J. P., & Reynolds, A. J. (2010). Child maltreatment and adult cigarette smoking: A long-term developmental model. *Journal of Pediatric Psychology*, *35*, 484–498.

Trickett, P. K., Gordis, E., Peckins, M. K., & Susman, E. J. (2014). Stress reactivity in maltreated and comparison male and female young adolescents. *Child Maltreat*, *19*, 27–37.

Vamosi, M., Heitmann, B. L., & Kyvik, K. O. (2009). The relation between an adverse psychological and social environment in childhood and the development of adult obesity: A systematic literature review. *Obesity Reviews*, *11*, 177–184.

Virdis, A., Ghiadoni, L., Plantinga, Y., Taddei, S., & Salvetti, A. (2007). C-reactive protein and hypertension: Is there a causal relationship?. *Current Pharmaceutical Design*, *13*, 1693–1698.

Walker, E. A., Unutzer, J., Rutter, C., Gelfand, A., Saunders, K., VonKorff, M., … Katon, W. (1999). Costs of health care use by women HMO members with a history of childhood abuse and neglect. *Archives of General Psychiatry*, *56*, 609–613.

Widom, C. S., White, H. R., Czaja, S. J., & Marmorstein, N. R. (2007). Long-term effects of child abuse and neglect on alcohol use and excessive drinking in middle adulthood. *Journal of Studies on Alcohol and Drugs*, *68*, 317–326.