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Child maltreatment and blood pressure in young adulthood☆

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ABSTRACT

Adverse childhood experiences are associated with hypertension in older adults. This study assessed whether an association between child maltreatment and blood pressure is detectable in young adults and whether any association differs by sex or is modified by genetic polymorphisms known to be involved in stress sensitivity. We examined these patterns in a sample of 12,420 young adults ages 24–32 years who participated in Wave IV of the National Longitudinal Study of Adolescent Health. Participants retrospectively reported history of physical, emotional, or sexual abuse before age 18 years. Participants with a systolic blood pressure (SBP) ≥140 mmHg or a diastolic blood pressure (DBP) ≥90 mmHg were classified as hypertensive. We used sex-stratified linear and logistic regression models to assess associations between each type of childhood maltreatment and SBP, DBP, and hypertension. We created interaction terms to assess for effect modification of any relationship between maltreatment and blood pressure by sex or SLC64A genotype. Fifteen percent of females and 31.5% of males were hypertensive. Frequent physical abuse in childhood was reported by 5%, frequent emotional abuse by 12%, and any sexual abuse by 5%. No association was observed between abuse history and blood pressure in either males or females, nor was effect modification present by SLC64A genotype. Child maltreatment exposure was not associated with blood pressure or hypertension in young adults in this study. Future studies should investigate additional critical windows for the effect of child maltreatment on cardiovascular health.

Abbreviations: SBP, systolic blood pressure; DBP, diastolic blood pressure; CVD, cardiovascular disease; DNA, deoxyribonucleic acid; BMI, body mass index; OR, odds ratio; CI, confidence interval.

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Introduction

Hypertension is a well-documented risk factor for the development of cardiovascular disease (CVD), the leading cause of death in U.S. adults (Hoyert & Xu, 2012) and is also linked to morbidity related to stroke, chronic kidney disease, and heart failure (Cushman, 2003). Recent research has identified an association between adverse childhood experiences – including child abuse – and hypertension in middle-aged adults (Afifi, Mota, Macmillan, & Sareen, 2013; Danese et al., 2009; Lehman, Taylor, Kiefe, & Seeman, 2009; Riley, Wright, Jun, Hibert, & Rich-Edwards, 2010; Stein et al., 2010). The mechanisms for this association remain unclear and may represent a higher co-morbidity of risk factors for hypertension among those who experienced child abuse (e.g., obesity (Midei & Matthews, 2011; Vamosi, Heitmann, & Kyvik, 2009), smoking (Jun et al., 2008), or alcohol use (Widom, White, Czaja, & Marmorstein, 2007)). In addition, those with an abuse history may have increased physiologic and/or emotional response to stressors (Miller, Chen, & Parker, 2011). Some studies have found a stronger relationship between child maltreatment and various CVD risk factors in women compared to men (Afifi et al., 2013; Batten, Aslan, Maciejewski, & Mazure, 2004). This finding raises the question of whether women respond differently to child maltreatment or are simply more likely to be exposed to certain forms of maltreatment, such as sexual abuse, that may have more direct effects on blood pressure.

In addition to potential sex differences in the response to child maltreatment, genes that influence changes in blood pressure, heart rate, and vascular endothelial function in response to psychological stress may also play a role. Individuals with a long promoter region for the serotonin transporter gene SLC6A4 have greater transcription of this gene and therefore greater serotonin uptake in platelets compared to those with a short promoter region for SLC6A4 (Greenberg et al., 1999). Experimental studies have generally found greater cardiovascular reactivity in response to acute stressors in those with the short promoter region (McCaffery, Bleil, Pogue-Geile, Ferrell, & Manuck, 2003; Way & Taylor, 2011). However, studies of the moderating effect of SLC64A polymorphisms on the relationship between child maltreatment and adult health outcomes such as depression have shown mixed results (Banny, Cicchetti, Rogosch, Oshri, & Crick, 2013; Brown et al., 2012; Klauke et al., 2011; Uher et al., 2011). Whether any increased cardiovascular reactivity associated with SLC64A polymorphisms results in chronic hypertension due to frequent stressful exposures in those with a history of child maltreatment or conversely, lower blood pressures in those who experienced supportive environments, is unknown. The latter may be true if certain SLC6A4 alleles confer a differential susceptibility to both positive and negative environments (Belsky & Pluess, 2009). We are unaware of previous studies examining the effect of the SLC6A4 gene on the relationship between child maltreatment and blood pressure.

Early identification and treatment of hypertension is a cornerstone of CVD prevention (Chobanian et al., 2003), and clarifying any association between child maltreatment and blood pressure in young adults could assist with screening and prevention programs before end-organ effects occur. As the majority of the studies of child maltreatment and hypertension have looked at this outcome in mid-life, we aimed to assess for relationships between physical, sexual, and emotional abuse and elevated blood pressure in young adults aged 24–32 years in the National Longitudinal Study of Adolescent Health (Add Health). We hypothesized that any association found would be stronger for females compared to males and in those with a short compared to a long promoter region for SLC64A.

Methods

We used data from the fourth wave of Add Health ($n = 15{,}701$), a nationally representative school-based study of adolescents enrolled in grades 7–12 at initial recruitment (Wave I) in 1994–1995. Wave IV data were collected in 2007–2008 when the participants were ages 24–32 years. Informed consent was obtained at Wave I and the study was approved by the Institutional Review Board at the University of North Carolina at Chapel Hill (Harris et al., 2009); additional analyses were approved by the Boston Children's Hospital Office of Clinical Investigation.

The sample for this study contained 12,420 young adults (79% of Wave IV participants). We excluded participants with missing data for sample weights or region ($n = 904$) because it was not possible to take into account the complex survey design for these individuals. We excluded those currently pregnant ($n = 487$) because of concern that their blood pressure might be influenced by factors different than the non-pregnant population. We also excluded those with systolic blood pressure (SBP) <80 mmHg or >200 mmHg ($n = 9$) and diastolic blood pressure (DBP) <40 mmHg or >120 mmHg ($n = 15$) because of concerns about measurement error. We excluded those currently on blood pressure lowering medications in the past four weeks as inventoried in the home by the Add Health field interviewer ($n = 528$) but also performed a sensitivity analysis with the included individuals. In addition, we excluded those who had missing data for either the outcome variable or for any key independent variables ($n = 1{,}361$). Because there was a high non-response rate for household income (~7%), we imputed income by Gaussian normal regression to avoid selection bias.

Measures

Outcome variables

Blood pressure was measured by trained Add Health field interviewers using an appropriately sized arm cuff and an automatic oscillometric monitor approved by the British Hypertension Society (BP 3MC1-PC_IB; MicroLife USA, Inc., Dunedin,

FL; Nguyen et al., 2011). After five minutes of rest, three blood pressure measurements were taken from the right arm with the patient in the resting, seated position, each separated by 30 s. The second and third measurements were double-entered and then averaged to give the final blood pressure recorded. We classified participants as hypertensive if they had an average measured SBP ≥140 mmHg or an average measured DBP ≥90 mmHg.

Primary exposure variables

Participants were asked at Wave IV to report retrospectively on mistreatment by adults during their childhood or adolescence using validated questions adapted from the Conflict Tactics Scale (Straus & Gelles, 1990). For emotional abuse, participants were asked, "Before your 18th birthday, how often did a parent or other adult caregiver say things that really hurt your feelings or made you feel like you were not wanted or loved?"; for physical abuse, "Before your 18th birthday, how often did a parent or adult caregiver hit you with a fist, kick you, or throw you down on the floor, into a wall, or down stairs?"; and for sexual abuse, "How often did a parent or other adult caregiver touch you in a sexual way, force you to touch him or her in a sexual way, or force you to have sexual relations?" Participants indicated *never*, *1 time*, *2 times*, *3–5 times*, *6–10 times*, or *more than 10 times* for each type of abuse. We categorized frequent emotional abuse as occurring >10 times, frequent physical abuse as occurring ≥6 times, and any sexual abuse as occurring ≥1 time to correspond with prevalence of emotional, physical and sexual abuse reported by studies using validated scales of child maltreatment (Green et al., 2010; McLaughlin et al., 2012) and used these as our primary exposure variables. We also assessed ordinal abuse scores for each of the three abuse questions (range: 0–5) and a composite abuse score created by summing the categorical responses from all three abuse questions (range: 0–15) in secondary analyses.

Genetic variables

Salivary buccal cell DNA was collected from Add Health participants using a standardized protocol (Smolen et al., 2012). We categorized individuals with 14 repeat units in the 5′ regulatory region of the serotonin transporter gene SLC6A4 as having the short form of the HTTLPR promoter and individuals with 16 or more repeat units as having the long form (Heils et al., 1996). We also categorized individuals with a G in the rs25531 single nucleotide polymorphism position as having the short form given evidence of transcriptional activity similar to the short form with this polymorphism (Hu et al., 2006). We assessed for Hardy–Weinberg equilibrium of these alleles in the overall population and in each racial/ethnic group.

Covariates

We constructed five racial/ethnic categories based on participant responses to two questions regarding racial identity and whether participants were of Hispanic/Latino origin: Asian/Pacific Islander, black/African American, Hispanic, multiracial/other, Native American/American Indian, and white. We used participants' self-reported household income and household size to create income as a percentage of the poverty level and then categorized based on standards used in NHANES reporting (Centers for Disease Control and Prevention, 2012). We collapsed the highest level of parental education achieved reported by either parent at Wave I to create three categories: high school graduate or less, some college or trade school training beyond high school, and college graduate or beyond. We calculated body mass index (BMI = kg/m^2) from Wave IV measured weight and height when available (98%) and from self-report in the small number of participants missing measured height and weight. We considered participants smokers at Wave IV if they reported currently smoking tobacco on more than 10 days in the proceeding 30 days. We categorized participants as moderate users of alcohol at Wave IV if they reported consuming alcohol on one or more days per week in the past 12 months and as heavy users if they reported consuming more than five drinks (men) or four drinks (women) in a row at least monthly in the past 12 months. We used the 10-item subscale of the Center for Epidemiologic Studies Depression Scale (CES-D; Radloff, 1977) to assess the psychological functioning of participants. Scores on the subscale ranged from 0 to 30 and those who scored greater than or equal to 10 were considered possibly depressed, consistent with prior research (Musliner & Singer, 2014).

Statistical analysis

All analyses were performed using STATA SE 12.1 (Stata Corporation, College Station, TX). Survey sampling weights were applied to account for the unequal likelihood of certain subpopulations being sampled. Bivariate analyses of the covariates of interest with the three exposures variables and the three outcome variables were conducted to test for significant relationships with simple linear regression for continuous variables and chi-square tests for categorical variables. We then created linear and logistic regression models to identify any association among physical abuse, emotional abuse, or sexual abuse and the outcomes of SBP, DBP, and hypertension. We first controlled only for age and sex and then controlled for potential confounders and mediators based on a priori findings from the literature using dummies for categorical variables (fully adjusted models). We assessed for effect modification by SLC64A genotype by creating genotype-by-maltreatment interaction terms for each of the abuse variables and adding these to the models. We also assessed for effect modification by sex.

Table 1
Characteristics of 12,420 young adults in the National Longitudinal Study of Adolescent Health (Wave IV) by child maltreatment history.

Characteristic	Sample *N*	Frequent emotional abuse % (SE) (*n* = 1,487)	Frequent physical abuse % (SE) (*n* = 611)	Any sexual abuse % (SE) (*n* = 647)
Age at Wave IV				
24–28 years	6,059	11.7 (0.6)	4.6 (0.4)	5.1 (0.4)
29–32 years	6,361	12.4 (0.7)	5.2 (0.4)	5.5 (0.4)
Sex				
Male	6,003	9.8 (0.5)	5.0 (0.4)	2.6 (0.3)
Female	6,417	14.4 (0.7)	4.7 (0.4)	8.1 (0.5)
Race/ethnicity				
White	6,702	12.4 (0.5)	4.4 (0.3)	4.6 (0.4)
Black/African-American	2,497	9.6 (0.9)	4.8 (0.7)	6.6 (0.9)
Hispanic	2,016	10.5 (1.1)	5.6 (0.8)	5.8 (0.9)
Asian/Pacific Islander	725	15.8 (3.4)	7.8 (2.0)	4.2 (1.3)
Native American	98	19.8 (4.6)	7.0 (3.5)	14.1 (4.0)
Other/Multiracial	382	17.5 (3.4)	7.9 (2.1)	8.6 (0.9)
Income at Wave I				
<100% FPL	1,920	13.0 (1.2)	6.2 (0.7)	7.6 (0.9)
100–299% FPL	5,916	12.0 (0.6)	5.4 (0.4)	6.2 (0.5)
300–499% FPL	3,290	12.4 (0.8)	4.0 (0.5)	3.3 (0.4)
≥500% FPL	1,294	9.9 (1.0)	2.6 (0.6)	2.3 (0.6)
Parental education at Wave I				
High school or less	4,427	11.1 (0.6)	5.1 (0.5)	6.2 (0.6)
Some college or trade	4,412	14.2 (0.8)	5.6 (0.5)	5.8 (0.5)
College graduate	3,581	10.4 (0.6)	3.5 (0.4)	3.3 (0.4)
BMI Wave IV				
Underweight (BMI < 18.5 kg/m^2)	192	12.3 (2.9)	3.1 (1.6)	5.3 (1.9)
Normal (BMI ≥ 18.5 & <25 kg/m^2)	4,054	12.2 (0.7)	4.6 (0.4)	4.6 (0.5)
Overweight (BMI ≥ 25 & <30 kg/m^2)	3,774	12.0 (0.8)	4.8 (0.5)	5.3 (0.5)
Obese (BMI ≥ 30 kg/m^2)	4,400	11.8 (0.7)	5.2 (0.4)	5.8 (0.5)
Alcohol consumption Wave IV				
None	6,467	12.4 (0.6)	5.0 (0.4)	5.9 (0.5)
Moderate	3,487	11.8 (0.8)	4.6 (0.5)	4.9 (0.5)
Heavy	2,466	11.5 (0.9)	5.0 (0.6)	4.2 (0.5)
Tobacco smoking Wave IV				
≥10 days per month	3,590	14.5 (0.8)	6.6 (0.6)	6.1 (0.6)
<10 days per month	8,830	10.8 (0.5)	4.0 (0.3)	4.8 (0.6)
Depression score Wave IV				
Mean (SE)	12,421	10.9 (0.19)	10.6 (0.26)	11.3 (0.27)
≥10	4,697	16.7 (0.8)	6.9 (0.5)	7.5 (0.6)
<10	7,723	9.2 (0.5)	3.6 (0.3)	3.9 (0.3)
SLC64A genotype				
Short/short	3,519	12.8 (0.8)	4.6 (0.4)	5.5 (0.5)
Short/long	6,010	11.5 (0.6)	4.8 (0.4)	5.3 (0.4)
Long/long	2,891	12.1 (0.8)	5.2 (0.6)	4.9 (0.5)

All analyses accounted for complex-survey design using sampling weights. Weighted estimates are presented.

Results

Characteristics of the study population by child maltreatment history are presented in Table 1. Female participants reported higher rates of frequent emotional abuse and of any sexual abuse compared to males. Tobacco smoking and having a depression score ≥10 were each associated with greater prevalence of all 3 types of abuse ($p < .001$ for all comparisons). Alcohol consumption and BMI were each associated with frequent sexual abuse ($p < .05$).

Females had lower mean blood pressures compared to males (SBP: 119.9 vs. 129.6 mmHg; DBP: 77.0 vs. 81.5 mmHg) and corresponding lower rates of hypertension also (15.3% vs. 31.5%). Higher SBP, DBP, and prevalence of hypertension was found in those with greater alcohol consumption, tobacco smoking, and higher BMI ($p < .001$ for all comparisons). Reporting a depression score of ≥10 was associated with lower systolic blood pressure ($p = .001$).

We did not find a significant association among frequent emotional abuse, frequent physical abuse, or any sexual abuse and systolic blood pressure, diastolic blood pressure, or hypertension in models adjusted for age and sex (Table 2, Model 1) or fully adjusted for covariates and potential mediators (Table 2, Model 2). Because no effect modification by sex was found, we report models for males and females together. A significant relationship was not found between SLC64A genotype and blood pressure (data not shown in table). Also, no effect modification of a relationship between maltreatment and blood pressure by genotype identified was found (Table 2, Model 3). Results did not differ when we modeled ordinal responses to each of the abuse variables or the composite abuse score (data not shown).

Table 2
Associations between abuse types and blood pressure in 12,420 participants from the National Longitudinal Study of Adolescent Health (Wave IV).

Predictor	Systolic blood pressure			Diastolic blood pressure			Hypertension		
	Model 1 β (95% CI)	Model 2 β (95% CI)	Model 3 β (95% CI)	Model 1 β (95% CI)	Model 2 β (95% CI)	Model 3 β (95% CI)	Model 1 OR (95% CI)	Model 2 OR (95% CI)	Model 3 OR (95% CI)
Emotional abuse									
Frequent emotional abuse[a]	0.26 (−0.59, 1.12)	0.35 (−0.39, 1.10)	0.44 (−0.99, 1.86)	0.48 (−0.27, 1.23)	0.44 (−0.26, 1.14)	0.43 (−0.71, 1.57)	1.11 (0.93, 1.33)	1.09 (0.92, 1.30)	1.01 (0.73, 1.41)
Short/long[d]	–	–	0.93 (0.19, 1.67)	–	–	0.23 (−0.38, 0.83)	–	–	1.03 (0.87, 1.21)
Long/long[d]	–	–	0.34 (−0.50, 1.17)	–	–	−0.47 (−1.11, 0.16)	–	–	1.05 (0.88, 1.24)
Short/long × emotional abuse[e]	–	–	−0.97 (−2.81, 0.86)	–	–	−0.67 (−2.07, 0.74)	–	–	0.96 (0.66, 1.41)
Long/long × emotional abuse[e]	–	–	1.68 (−0.46, 3.82)	–	–	1.41 (−0.29, 3.11)	–	–	1.45 (0.90, 2.32)
Physical abuse									
Frequent physical abuse [b]	−0.55 (−1.94, 0.84)	−0.78 (−2.06, 0.50)	0.36 (−2.08, 2.80)	−0.42 (−1.41, 0.57)	−0.67 (−1.61, 0.27)	−0.31 (−2.42, 1.81)	1.06 (0.81, 1.40)	0.99 (0.75, 1.31)	1.38 (0.81, 2.34)
Short/long[d]	–	–	0.90 (0.18, 1.63)	–	–	0.19 (−0.41, 0.79)	–	–	1.05 (0.90, 1.23)
Long/long[d]	–	–	0.58 (−0.29, 1.46)	–	–	−0.32 (−0.97, 0.33)	–	–	1.11 (0.94, 1.30)
Short/long × emotional abuse[e]	–	–	−1.90 (−4.77, 0.97)	–	–	−0.92 (−3.47, 1.64)	–	–	0.58 (0.32, 1.05)
Long/long × emotional abuse[e]	–	–	−0.89 (−3.89, 2.12)	–	–	0.39 (−2.00, 2.78)	–	–	0.75 (0.38, 1.50)
Sexual abuse									
Any sexual abuse[c]	−0.20 (−1.47, 1.07)	−0.69 (−1.82, 0.45)	−0.62 (−2.90, 1.67)	−0.06 (−1.14, 1.03)	−0.47 (−1.50, 0.56)	−0.74 (−2.35, 0.88)	1.10 (0.86, 1.41)	0.99 (0.76, 1.29)	1.02 (0.64, 1.65)
Short/long[d]	–	–	0.81 (0.09, 1.52)	–	–	0.13 (−0.45, 0.72)	–	–	1.02 (0.88, 1.19)
Long/long[d]	–	–	0.56 (−0.31, 1.42)	–	–	−0.35 (−1.00, 0.30)	–	–	1.10 (0.93, 1.30)
Short/long × emotional abuse[e]	–	–	0.09 (−3.07, 3.25)	–	–	0.20 (−1.85, 2.25)	–	–	1.01 (0.54, 1.90)
Long/long × emotional abuse[e]	–	–	−0.52 (−3.82, 2.78)	–	–	0.73 (−1.63, 3.09)	–	–	0.84 (0.37, 1.90)

β indicates change per 1 mmHg blood pressure difference. OR, odds ratio; CI, confidence interval.
All analyses accounted for complex survey design using sampling weights.
Model 1 is adjusted for age and sex.
Model 2 is adjusted for age, sex, race/ethnicity, parental income as percentage of federal poverty level, highest parental education, young adult BMI, alcohol drinking, frequent smoking, and depression score.
Model 3 is adjusted for age, sex, race/ethnicity, parental income as percentage of federal poverty level, highest parental education, young adult BMI, alcohol drinking, frequent smoking, and depression score and the interaction between emotional abuse and the SLC64A genotype.
Referent groups as follows:

[a] No/less than frequent emotional abuse.
[b] No/less than frequent physical abuse.
[c] No sexual abuse.
[d] Short/short genotype.
[e] Short/short genotype/no/less than frequent emotional abuse.

Discussion

Child maltreatment and hypertension are important public health issues and were prevalent in this nationally representative sample of young adults. However, we did not find an association between reports of physical, emotional, or sexual abuse in childhood and blood pressure in young adulthood in either males or females participating in Add Health. Furthermore, our findings neither support a relationship between SLC64A genotype and blood pressure in young adults generally nor any differential susceptibility (Belsky & Pluess, 2009) to abusive vs. non-abusive environments by SLC64A genotype with regards to young adult blood pressure.

There are several possible explanations for our null findings. We excluded 21% of Add Health participants in the complete case analysis because of missing data on a variety of variables. There were, however, no differences between the included and excluded participants on the blood pressure outcomes or prevalence of abuse exposure. Given the higher rates of measured hypertension in Add Health (19%) compared to participants of similar age in the National Health and Nutrition Examination Survey (4%; Nguyen et al., 2011), some individuals in Add Health may have been misclassified as hypertensive. However, the Add Health protocol for measuring blood pressure has been well-validated (Nguyen et al., 2011), and we do not have reason to suspect that any overestimation effect would differentially affect the maltreated and non-maltreated groups. Although the prevalence of hypertension is quite high in young adults in this sample, childhood exposures may have latent effects that are not yet evident by early adulthood. For example, studies have indicated steeper weight gain trajectories among women with a history of abuse (Noll, Zeller, Trickett, & Putnam, 2007) or posttraumatic stress disorder (Kubzansky et al., 2014); it may be that blood pressure increments are a downstream effect that follows overweight and obesity. Others have found a lack of relationship between child maltreatment and young adult blood pressure when psychosocial factors are accounted for (Lehman et al., 2009); unmeasured confounders related to emotional functioning may also have obscured any relationship in this sample. Because we did not test for any relationship between supportive or resilient environments in childhood on young adulthood blood pressure, differential susceptibility conferred by SLC64A genotype on young adult blood pressure cannot fully be excluded.

We were also unable to quantify the duration of exposure to maltreatment over time. It is plausible that child maltreatment causes changes in the autonomic nervous system or hypothalamic–pituitary–adrenal response to future stressors (Carpenter et al., 2007; Heim et al., 2000; MacMillan et al., 2009) and that only repeated exposures to future stressors over long periods of time result in chronically elevated blood pressures. This may also partially explain our null findings related to SLC6A4 genotype, as we hypothesized that those with the short promoter region would have higher blood pressures if exposed to child abuse due to increased cardiovascular reactivity in response to stress over time. Finally, we used retrospective reports of abuse based on a limited number of questions adapted from the validated Conflict Tactics Scale (Straus & Gelles, 1990). Retrospective reports are subject to under-reporting of abuse (Hardt & Rutter, 2004), and we were not able to measure severity of abuse. It is possible that by combining these rarer, severe events with more prevalent mild events, we obscured associations that would have been evidence for severe maltreatment.

Conclusion

Studies of cohorts of older adults have identified child maltreatment as a risk factor for ischemic cardiovascular events in adulthood (Batten et al., 2004; Dong et al., 2004; Rich-Edwards et al., 2012). Repeated but transient elevations in blood pressure in response to future stressors in those with a history of maltreatment may indeed contribute to these findings in older populations. We did not find any association between child maltreatment and the rates of hypertension in younger adults, nor any effect modification by a common genetic polymorphism associated with increased cardiovascular reactivity to stress. However, given the high rates of hypertension and child maltreatment found in young adults in Add Health, these remain important public health issues and areas for future study and intervention.

Conflict of interest

The authors have no conflicts of interest to disclose. Dr. Gooding drafted the manuscript; none of the authors were paid an honorarium for this work.

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References

Afifi, T. O., Mota, N., Macmillan, H. L., & Sareen, J. (2013). Harsh physical punishment in childhood and adult physical health. *Pediatrics*, *132*, e333–e340. http://dx.doi.org/10.1542/peds.2012-4021

Banny, A. M., Cicchetti, D., Rogosch, F. A., Oshri, A., & Crick, N. R. (2013). Vulnerability to depression: A moderated mediation model of the roles of child maltreatment, peer victimization, and serotonin transporter linked polymorphic region genetic variation among children from low socioeconomic status backgrounds. *Development and Psychopathology*, *25*, 599–614. http://dx.doi.org/10.1017/S0954579413000047

Batten, S. V., Aslan, M., Maciejewski, P. K., & Mazure, C. M. (2004). Childhood maltreatment as a risk factor for adult cardiovascular disease and depression. *Journal of Clinical Psychiatry*, *65*, 249–254.

Belsky, J., & Pluess, M. (2009). Beyond diathesis stress: Differential susceptibility to environmental influences. *Psychological Bulletin*, *135*, 885–908. http://dx.doi.org/10.1037/a0017376

Brown, G. W., Ban, M., Craig, T. K., Harris, T. O., Herbert, J., & Uher, R. (2012). Serotonin transporter length polymorphism, childhood maltreatment, and chronic depression: A specific gene–environment interaction. *Depression and Anxiety*, *30*, 5–13. http://dx.doi.org/10.1002/da.21982

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*, 1080–1087. http://dx.doi.org/10.1016/j.biopsych.2007.05.002. S0006-3223(07)00431-3

Centers for Disease Control and Prevention. (2012). Vital signs: Awareness and treatment of uncontrolled hypertension among adults – United States, 2003–2010. *MMWR. Morbidity and Mortality Weekly Report*, *61*, 703–709.

Chobanian, A. V., Bakris, G. L., Black, H. R., Cushman, W. C., Green, L. A., Izzo, J. L., Jr., Jones, D. W., Materson, B. J., Oparil, S., Wright, J. T., Jr., & Roccella, E. J. (2003). The seventh report of the Joint National Committee on prevention, detection, evaluation, and treatment of high blood pressure: The JNC 7 report. *Journal of the American Medical Association*, *289*, 2560–2572. http://dx.doi.org/10.1001/jama.289.19.2560

Cushman, W. C. (2003). The burden of uncontrolled hypertension: Morbidity and mortality associated with disease progression. *Journal of Clinical Hypertension (Greenwich)*, *5*(3 Suppl. 2), 14–22.

Danese, A., Moffitt, T. E., Harrington, H., Milne, B. J., Polanczyk, G., Pariante, C. M., Poulton, R., & 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. http://dx.doi.org/10.1001/archpediatrics.2009.214

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. http://dx.doi.org/10.1161/01.CIR.0000143074.54995.7F01

Green, J. G., McLaughlin, K. A., Berglund, P. A., Gruber, M. J., Sampson, N. A., Zaslavsky, A. M., & Kessler, R. C. (2010). Childhood adversities and adult psychiatric disorders in the national comorbidity survey replication I: Associations with first onset of DSM-IV disorders. *Archives of General Psychiatry*, *67*, 113–123. http://dx.doi.org/10.1001/archgenpsychiatry.2009.186

Greenberg, B. D., Tolliver, T. J., Huang, S. J., Li, Q., Bengel, D., & Murphy, D. L. (1999). Genetic variation in the serotonin transporter promoter region affects serotonin uptake in human blood platelets. *American Journal of Medical Genetics*, *88*(1), 83–87. http://dx.doi.org/10.1002/(SICI)1096-8628(19990205)88:1<83::AID-AJMG15>3.0.CO;2-0

Hardt, J., & Rutter, M. (2004). Validity of adult retrospective reports of adverse childhood experiences: Review of the evidence. *Journal of Child Psychology and Psychiatry and Allied Disciplines*, *45*, 260–273.

Harris, K. M., Halpern, C. T., Whitsel, E., Hussey, J., Tabor, P., Entzel, P., & Udry, J. R. (2009). *The National Longitudinal Study of Adolescent Health: Research design*. Retrieved from http://www.cpc.unc.edu

Heils, A., Teufel, A., Petri, S., Stober, G., Riederer, P., Bengel, D., & Lesch, K. P. (1996). Allelic variation of human serotonin transporter gene expression. *Journal of Neurochemistry*, *66*(6), 2621–2624.

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. *Journal of the American Medical Association*, *284*, 592–597 (joc92111).

Hoyert, D. L., & Xu, J. (2012). . *Deaths: Preliminary data for 2011 national vital statistics reports* (61) Atlanta: Centers for Disease Control.

Hu, X. Z., Lipsky, R. H., Zhu, G., Akhtar, L. A., Taubman, J., Greenberg, B. D., Xu, K., Arnold, P. D., Richter, M. A., Kennedy, J. L., Murphy, D. L., & Goldman, D. (2006). Serotonin transporter promoter gain-of-function genotypes are linked to obsessive–compulsive disorder. *American Journal of Human Genetics*, *78*, 815–826. http://dx.doi.org/10.1086/503850. S0002-9297(07)63816-6

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. http://dx.doi.org/10.1016/j.jadohealth.2007.12.003. S1054-139X(07)00662-3

Klauke, B., Deckert, J., Reif, A., Pauli, P., Zwanzger, P., Baumann, C., Arolt, V., Glöckner-Rist, A., & Domschke, K. (2011). Serotonin transporter gene and childhood trauma – a G × E effect on anxiety sensitivity. *Depression and Anxiety*, *28*, 1048–1057. http://dx.doi.org/10.1002/da.20840

Kubzansky, L. D., Bordelois, P., Jun, H. J., Roberts, A. L., Cerda, M., Bluestone, N., & Koenen, K. C. (2014). The weight of traumatic stress: A prospective study of posttraumatic stress disorder symptoms and weight status in women. *JAMA Psychiatry*, *71*(1), 44–51. http://dx.doi.org/10.1001/jamapsychiatry.2013.2798

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. http://dx.doi.org/10.1037/a0013785

MacMillan, H. L., Georgiades, K., Duku, E. K., Shea, A., Steiner, M., Niec, A., Tanaka, M., Gensey, S., Spree, S., Vella, E., Walsh, C. A., De Bellis, M. D., Van der Meulen, J., Boyle, M. H., & Schmidt, L. A. (2009). Cortisol response to stress in female youths exposed to childhood maltreatment: Results of the youth mood project. *Biological Psychiatry*, *66*(1), 62–68. http://dx.doi.org/10.1016/j.biopsych.2008.12.014. S0006-3223(08)01596-5

McCaffery, J. M., Bleil, M., Pogue-Geile, M. F., Ferrell, R. E., & Manuck, S. B. (2003). Allelic variation in the serotonin transporter gene-linked polymorphic region (5-HTTLPR) and cardiovascular reactivity in young adult male and female twins of European-American descent. *Psychosomatic Medicine*, *65*, 721–728.

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. http://dx.doi.org/10.1001/archgenpsychiatry.2011.2277

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*(5), e159–e172. http://dx.doi.org/10.1111/j.1467-789X.2010.00823.x

Miller, G. E., Chen, E., & Parker, K. J. (2011). Psychological stress in childhood and susceptibility to the chronic diseases of aging: Moving toward a model of behavioral and biological mechanisms. *Psychological Bulletin*, *137*, 959–997. http://dx.doi.org/10.1037/a0024768

Musliner, K. L., & Singer, J. B. (2014). Emotional support and adult depression in survivors of childhood sexual abuse. *Child Abuse and Neglect*, *38*, 1331–1340. http://dx.doi.org/10.1016/j.chiabu.2014.01.016

Nguyen, Q. C., Tabor, J. W., Entzel, P. P., Lau, Y., Suchindran, C., Hussey, J. M., Halpern, C. T., Harris, K. M., & Whitsel, E. A. (2011). Discordance in national estimates of hypertension among young adults. *Epidemiology*, *22*, 532–541. http://dx.doi.org/10.1097/EDE.0b013e31821c79d2

Noll, J. G., Zeller, M. H., Trickett, P. K., & Putnam, F. W. (2007). Obesity risk for female victims of childhood sexual abuse: A prospective study. *Pediatrics*, *120*(1), e61–e67. http://dx.doi.org/10.1542/peds.2006-3058

Radloff, L. (1977). The CES-D scale: A self-report depression scale for research in the general population. *Applied Psychological Measurement*, *1*, 385–401.

Rich-Edwards, J. W., Mason, S., Rexrode, K., Spiegelman, D., Hibert, E., Kawachi, I., Jun, H. J., & Wright, R. J. (2012). Physical and sexual abuse in childhood as predictors of early-onset cardiovascular events in women. *Circulation*, *126*, 920–927. http://dx.doi.org/10.1161/CIRCULATIONAHA.111.076877

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. http://dx.doi.org/10.1136/jech.2009.095109

Smolen, A., Whitsel, E. A., Tabor, J. W., Killeya-Jones, L. A., Cuthbertson, C. C., Hussey, J. M., Halpern, C. T., & Harris, K. M. (2012). Add health wave IV documentation. In C. P. Center (Ed.), *Candidate genes*. Chapel Hill, NC: University of North Carolina at Chapel Hill.

Stein, D. J., Scott, K., Haro Abad, J. M., Aguilar-Gaxiola, S., Alonso, J., Angermeyer, M., Demytteneare, K., de Girolamo, G., Iwata, N., Posada-Villa, J., Kovess, V., Lara, C., Ormel, J., Kessler, R. C., & Von Korff, M. (2010). Early childhood adversity and later hypertension: Data from the World Mental Health Survey. *Annals of Clinical Psychiatry*, *22*(1), 19–28.

Straus, M. A., & Gelles, R. J. (1990). *Physical violence in American families: Risk factors and adaptations to violence in 8,145 families*. New Brunswick: Transaction Publishers.

Uher, R., Caspi, A., Houts, R., Sugden, K., Williams, B., Poulton, R., & Moffitt, T. E. (2011). Serotonin transporter gene moderates childhood maltreatment's effects on persistent but not single-episode depression: Replications and implications for resolving inconsistent results. *Journal of Affective Disorders*, *135*(1–3), 56–65. http://dx.doi.org/10.1016/j.jad.2011.03.010. S0165-0327(11)00093-0

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. http://dx.doi.org/10.1111/j.1467-789X.2009.00645.x

Way, B. M., & Taylor, S. E. (2011). A polymorphism in the serotonin transporter gene moderates cardiovascular reactivity to psychosocial stress. *Psychosomatic Medicine*, *73*(4), 310–317. http://dx.doi.org/10.1097/PSY.0b013e31821195ed

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.

Appendix A. Supplementary data

Supplementary data associated with this article can be found, in the online version, at http://dx.doi.org/10.1016/j.chiabu.2014.08.019.