HHS Public Access

Author manuscript

Prev Med. Author manuscript; available in PMC 2024 July 03.

Published in final edited form as:

Prev Med. 2016 June ; 87: 132–137. doi:10.1016/j.ypmed.2016.02.031.

Intimate Partner Violence in Late Adolescence and Young Adulthood and Subsequent Cardiovascular Risk in Adulthood

Cari Jo Clark, Sc.D., M.P.H. [Assistant Professor, Department of Medicine Adjunct Assistant Professor],
Division of Epidemiology and Community Health, Program in Health Disparities Research, University of Minnesota, 717 Delaware Street, SE, Ste 166 Minneapolis, MN 55414, United States

Alvaro Alonso, M.D., Ph.D.,
Division of Epidemiology and Community Health, University of Minnesota, Minneapolis, MN

Susan A. Everson-Rose, Ph.D., M.P.H.,
Department of Medicine, Program in Health Disparities Research and Center for Health Equity, University of Minnesota, Minneapolis, MN

Rachael A. Spencer, M.P.A.,
Independent Gender Based Violence Specialist, Atlanta, GA

Sonya S. Brady, Ph.D.,
Division of Epidemiology and Community Health, University of Minnesota, Minneapolis, MN

Michael D. Resnick, Ph.D., FSAHM,
Department of Pediatrics, University of Minnesota, Minneapolis, MN

Iris W. Borowsky, M.D., Ph.D.,
Department of Pediatrics, University of Minnesota, Minneapolis, MN

John E. Connett, Ph.D.,
Division of Biostatistics, Biostatistical Design and Analysis Center, University of Minnesota, Minneapolis, MN

Robert F. Krueger, Ph.D.,
Department of Psychology, University of Minnesota, Minneapolis, MN

Viann N. Nguyen-Feng, M.P.H.,
Department of Psychology, University of Minnesota, Minneapolis, MN

Steven L. Feng, M.D.,
Department of Internal Medicine, Hennepin County Medical Center, Minneapolis, MN

Shakira F. Suglia, Sc.D.
Department of Epidemiology, Columbia University, New York, NY

Corresponding Author: Cari Jo Clark, Sc.D., M.P.H. Assistant Professor, Department of Medicine, Adjunct Assistant Professor, Division of Epidemiology and Community Health, Program in Health Disparities Research, University of Minnesota, 717 Delaware Street, SE, Ste 166 Minneapolis, MN 55414, United States, Fax: 612.626.6782; cjclark@umn.edu.

Conflict of Interest Statement: The authors declare that there are no conflicts of interest.

Abstract

Background—Childhood maltreatment has been linked to adulthood cardiovascular disease (CVD). Little is known about the relationship between intimate partner violence (IPV) in late adolescence and young adulthood and CVD risk later in adulthood.

Purpose—To examine whether IPV perpetration and victimization experienced in late adolescence and young adulthood are associated with CVD risk among adults in the United States and whether this relationship differs by sex.

Methods—Data include 9,976 participants (50% female) in the National Longitudinal Study of Adolescent to Adult Health. Physical and sexual IPV were measured at wave 3 (2001/02) with items from the revised Conflict Tactics Scales. Participants' 30-year risk of CVD was calculated at wave 4 (2008/09) using a Framingham prediction model. Linear regression models adjusted for confounders and IPV by sex interaction terms were tested to examine the relationship.

Results—The mean CVD risk score was 13.18% (95% CI: 12.71, 13.64). A one-standard deviation increase in the victimization score was associated with a 0.28% (95% CI: 0.03, 0.54) increase in CVD risk. Perpetration was similarly positively associated with CVD risk (beta: 0.33, 95% CI: 0.03, 0.62). When measured as a composite, all violence types were associated with increased CVD risk but only prior exposure to both victimization and perpetration reached statistical significance (0.62%, 95% CI: 0.01, 1.22). No differences by sex were detected.

Conclusions—Effect sizes are not large, but early detection of increased CVD risk in this relatively young population is notable and worthy of further study to inform the clinical response.

Keywords

cardiovascular diseases; domestic violence; adolescent health services; National Longitudinal Study of Adolescent Health

Introduction

Cardiovascular disease (CVD) primarily affects individuals of middle and old age; however risk factors for CVD are apparent at a much younger age (Eaton et al., 2010; Kavey et al., 2003; Skinner and Skelton, 2014). Adverse childhood experiences, particularly child maltreatment, have been associated with CVD in adulthood (Batten et al., 2004; Dong et al., 2004; Wegman and Stetler, 2009). Less is known, however, about the potential relationship between highly prevalent forms of violence in adolescence and young adulthood, such as intimate partner violence (IPV) and CVD risk. Ten percent of US high school students in the 2013 Youth Risk Behavior Survey reported physical partner violence in the prior year (Kann et al., 2014). Further, the majority of the 29% of men and 36% of women who reported lifetime exposure to IPV in the 2010 National Intimate Partner and Sexual Violence Survey were victimized for the first time before the age of 25 (Breiding et al., 2014). Greater attention to the role of partner violence exposure during adolescence and young adulthood in the development of CVD risk is warranted to identify primary prevention strategies.

Existing research on the relationship between IPV and CVD risk has focused almost exclusively on adult female populations and has generated inconclusive findings (Suglia

et al., 2015). IPV has been positively associated with self-reported diagnosis of CVD (Matthew J. Breiding et al., 2008; Coker et al., 2000b; Gass et al., 2010; Lown and Vega, 2001), smoking (Crane et al., 2013), diabetes (Mason et al., 2013), hypertension (Gass et al., 2010; Mason et al., 2012), use of antihypertensive medication (Stene et al., 2013), and weight gain, higher body mass index and obesity (Clark et al., 2014b; Garcia et al., 2014; Sato-DiLorenzo and Sharps, 2007; Yount and Li, 2011). Alternatively, studies have found a lack of association with CVD and many of these same risk factors (M. J. Breiding et al., 2008; Coker et al., 2000b; Gass et al., 2010; Lown and Vega, 2001; Stene et al., 2013). In addition to disparate findings, most prior research examines individual risk factors associated with IPV despite the fact that risk factors cluster in individuals (Freedman et al., 1999) and few studies have examined the relationship between CVD risk and exposure to both victimization and perpetration (see (Clark et al., 2014a; Crane et al., 2014) for examples) despite the prominence of bi-directionally violent relationships, especially among adolescents and young adults (Gray and Foshee, 1997; Whitaker et al., 2007).

CVD risk manifests over the life course and is prominent in adolescence and young adulthood.(Clark et al., 2014a) This fact, with growing evidence of the cost effectiveness of primary prevention,(Weintraub et al., 2011) has prompted increasing attention to the extent of early life CVD risk and its contributing factors to inform prevention strategies. The present study addresses this need using a nationally representative, longitudinal study of adolescents to examine whether IPV victimization and perpetration experienced during adolescence and young adulthood is associated with increased CVD risk in adulthood and to determine whether this relationship differs by sex.

Methods

The study sample includes participants of the National Longitudinal Study of Adolescent to Adult Health (Add Health) which is a longitudinal study of a nationally representative sample of adolescents in grades 7–12 in the U.S. during the 1994–95 school year (Harris et al., 2009) who have been followed into adulthood (2008–09). Student rosters were used to select a sample of 20,745 adolescents who were interviewed at baseline (wave 1, 1995, participant age range 11–21). A parental figure was also interviewed at this time. A year later, a second wave of data collection occurred. In 2001 and 2002, approximately 6 years after baseline, a third wave of data was collected (age range of participants, 18–28). In 2008 and 2009, approximately 13 years since baseline and approximately 7 years since wave 3, a fourth wave of data was collected (age range of participants, 24–34). Response rates across the 4 waves of data collection were 79%, 89%, 77% and 80%, respectively, and the parental response rate for wave 1 was 85%. The present study is restricted to respondents who had valid sampling weights (n=12,288), reported on a relationship at wave 3 (first time point in which IPV perpetration was asked in addition to victimization) in which IPV was assessed (n=10,171), and were free from CVD and cancer at the time of CVD risk factor assessment at Wave 4 (n=9,976). Participants provided written informed consent, and the parent study was approved by the Institutional Review Board (IRB) of the University of North Carolina, Chapel Hill. The University of Minnesota IRB determined that the present analysis did not meet the regulatory definition of human subjects' research due to the sole use of de-identified data.

Measures

Exposure—IPV was retrospectively assessed at the wave 3 interview in reference to relationships that occurred between the summer of 1995 (average age 16) and the wave 3 interview (2001/02, average age 22) representing up to 7 years of recall. Four items measured victimization (threatened by partner with violence, pushed or shoved, or had something thrown at you that could hurt; partner slapped, hit or kicked you; partner made you have sexual relations when you did not want to; you had an injury, such as a sprain, bruise, or cut because of a fight with your partner); these items were based on the Revised Conflict Tactics Scales (Straus et al., 1996), the most frequently used scale to assess IPV. The same 4 items also were used to assess perpetration. The scale was self-administered using a laptop computer to protect the participant's privacy and to reduce the likelihood of socially desirable responses.

Continuous scales of IPV victimization and perpetration were created using Rasch modeling based on the conditional probabilities of giving a positive response to each item given its severity and the true but unobserved violence exposure level of each person. Because acts of low/moderate violence severity are most frequently perpetrated by U.S. couples in intimate relationships (Black et al., 2011), items that were more commonly reported were considered less severe (i.e., being threatened with violence versus being forced to have sexual relations). The model is generalized to account for whether the event occurred once or more than once in the same relationship and whether the event occurred in more than one relationship (Suglia et al., 2008). Separate scales for men and women were created; however, the analytic procedure generates scores with similar distributions enabling them to be combined into one perpetration and one victimization scale. Each score was scaled by its standard deviation. To assess whether IPV predicted CVD risk differently accordingly to the dynamics of the prior experience, a nominal composite variable was created with the following categories: no IPV experience, victimization only, perpetration only, and bi-directional (both victimization and perpetration).

Outcomes—The risk of developing CVD over a 30-year time frame was estimated using a prediction model developed by Pencina et al. (Pencina et al., 2009). According to guidelines from the American Heart Association and the American College of Cardiology, long-term risk prediction is an appropriate assessment tool in individuals as young as 20 (Goff et al., 2014) and average long-term risk CVD in the Add health population has been shown to be high (Clark et al., 2014a). Data used in the prediction model were collected at wave 4 when the participations were on average 29 years of age (2008/09). The function predicts the risk of occurrence of CVD (coronary death, myocardial infarction, coronary insufficiency, angina pectoris, stroke, transient ischemic attack, intermittent claudication, and congestive heart failure) over a 30-year time frame (Pencina et al., 2009). Risk factors used to calculate the 30-year FRS include sex, age, systolic blood pressure (SBP), use of antihypertensive medication, smoking, diabetes, and body mass index (BMI), all of which were ascertained from interview data, anthropometric measurements, and biological specimens taken at wave 4. Standardized approaches to height and measurements were used (Entzel et al., 2009). SBP was measured after a 5-min seated rest; 3 measurements were obtained at 30-second intervals, and the latter 2 readings were averaged for resting SBP (Entzel et al., 2009).

Use of antihypertensive medication in the prior 4 weeks was assessed by the interviewer through a medication inventory (Tabor and Whitsel, 2010). Cigarette smoking in the 30 days preceding the interview was ascertained by self-report. Diabetes mellitus was considered present if the respondent had: a fasting glucose ≥ 126 mg/dl, a non-fasting glucose ≥ 200 mg/dl, an HbA1c ≥ 6.5%, self-reported a health provider diagnosis of diabetes except during pregnancy, or used anti-diabetic medication in the prior four weeks (Whitsel et al., 2012).

Potential Confounders—Socio-demographic variables were included in the analysis because they have been associated with CVD risk (Go et al., 2013; Murray et al., 2010) and IPV experience (Cunradi et al., 2000; Field and Caetano, 2005; Renner and Whitney, 2010). Socio-demographics collected at wave 3 include age, sex (male, female), race/ethnicity (Non-Hispanic White, Black, Native American/American Indian, Asian/Pacific Islander, Mixed race (i.e., report of two or more races), and Hispanic), educational attainment (number of years of education past the 6th grade), and financial stress (a positive response to any of 7 items indicating an inability to pay for basics). Indicators of early life socioeconomic status included educational attainment of the parental figure at wave 1 (1994/95) and neighborhood poverty defined as the proportion of families in the census block group with incomes in 1989 below the poverty line.

Frequency of parent/adult-caregiver perpetrated abuse and neglect that occurred prior to the 6th grade was assessed at wave 3 and was included in the analysis because experiencing childhood maltreatment is independently associated with later IPV experience (Renner and Whitney, 2012) and CVD risk in adulthood (Wegman and Stetler, 2009). Abuse and neglect that occurred prior to the 6th grade was retrospectively assessed at wave 3 using modified versions of existing scales (Finkelhor and Dziuba-Leatherman, 1994; Hussey et al., 2006; Straus et al., 2008). Each type of maltreatment was defined similarly to prior analyses of these data (Fang and Corso, 2007). Inadequate supervision was considered present if the respondent had been left alone more than 5 times when an adult should have been present, and physical neglect was considered present if the respondent did not have his/her basic needs met at least once. Physical abuse included being slapped, hit, or kicked more than 5 times. Sexual abuse was defined as being forced to have sexual relations or being touched or being forced to touch in a sexual way at least once.

Analysis

Descriptive statistics were calculated by sex. Differences in study variables by sex were examined using *t*-tests and Chi-square tests. Restricted cubic spline functions (Desquilbet and Mariotti, 2010) were used to examine whether the victimization score, the perpetration score, neighborhood poverty, and the measures of respondent and parental educational attainment were linearly associated with CVD risk. All were determined to be linear associations with the exception of neighborhood poverty. A quadratic term for this variable was included in subsequent regression models.

The relationship between IPV and CVD risk was tested with linear regression models adjusted for sex, race/ethnicity, and wave 3 age, respondent educational attainment, financial stress, and child maltreatment and wave 1 indicators of parental education and neighborhood

poverty. Separate models were computed using the victimization score and the perpetration score. To assess whether IPV differentially predicted CVD risk by the type of IPV exposure (i.e. victimization only, perpetration only, and bi-directional violence), the model was re-run using the composite variable. A sex by IPV interaction term was tested in all models; none were detected (all p-values between ≥ 0.19).

All descriptive statistics and regression models incorporated survey design and unequal probability of selection per Add Health user guidance (Chantala and Tabor, 2010). Means, percents, and regression results reflect this weighting process. Multiple imputation was performed to address missing data which ranged from 0% to 5% for all variables except childhood neglect and parental educational attainment which had 6.64% and 13.18% missing values, respectively. In addition to the study variables, several auxiliary variables were included in the imputation process to improve power and reduce non-response bias (Enders, 2010) including individual CVD risk score components, exposure to other forms of interpersonal violence, depressive symptoms, family history of obesity, and whether the respondent was born in the United States. Twenty-five datasets were generated using PROC MI in SAS 9.3 and descriptive statistics and regression models were subsequently computed using SUDAAN 11.0. Analyses were finalized in November 2014.

Results

Table 1 indicates the socio-demographic characteristics of participants (4,472 men and 5,504 women) by sex. At wave 3, the sample was, on average, 21.82 (95% CI: 21.59, 22.06) years old and the participants were 67.07% White (n=5,533), 14.37% Black (n=1,907), 0.67% American Indian/Native American (n=66), 3.02% Asian/Pacific Islander (n=576), 3.40% Mixed (n=372), and 11.46% Hispanic (n=1,522). Mean 30-year CVD risk for the sample was 13.18% (95% CI: 12.71, 13.64) and was higher among men (Table 1). Overall, 39.84% (n=4,056) reported IPV exposure. Women were more likely to report any IPV exposure and more frequently reported perpetration only and a prior history of both victimization and perpetration. Women also had more years of education and were more likely to report financial stress, but were less likely than men to report being physically neglected as a child and were statistically, but not meaningfully, younger than the men in the sample.

The results of the regression analyses for IPV perpetration and IPV victimization are presented in Table 2. A one-standard deviation increase in victimization score was associated with a 0.28% increase in CVD risk score (95% CI: 0.03, 0.54). Similarly, a one standard deviation change in perpetration was associated with a 0.33% increase in the CVD risk score (95% CI: 0.03, 0.62). Male sex, Black race, older age, financial stress, higher neighborhood poverty, and physical neglect were also associated with higher long-term CVD risk. Conversely, Asian/Pacific Islanders and Hispanics and individuals with higher levels of education had lower risk of CVD. When categorized by type of exposure, all forms demonstrated increased CVD risk compared to those not exposed to IPV [victimization only: 0.62% (95% CI: −0.12,1.36); perpetration only: 0.76% (95% CI: −0.04, 1.57), both victimization and perpetration: 0.62% (95% CI: 0.01, 1.22)]; only prior exposure to both IPV types reached statistical significance ($p<0.05$).

Discussion

This study indicates that recalled exposure to IPV in late adolescence and young adulthood is associated with increased risk of CVD 7 to 14 years post exposure. This association is consistent for perpetrators and victims of IPV and did not differ by sex. To our knowledge, this is the first study to investigate whether IPV experienced during adolescence or young adulthood predicts long-term CVD risk. The study's contribution to the literature is strengthened by the use of a large, nationally representative sample with behavioral markers of IPV, an investigation of sex differences, and inclusion of victimization, perpetration, and exposure to both types of violence. Further, an objectively defined marker of long-term cardiovascular risk was used to simultaneously account for multiple risk behaviors and competing causes of death, making it a more realistic estimate of the future risk of CVD, a feature of particular importance to assessing CVD risk in younger adults.

Overall, individuals who reported exposure to IPV in late adolescence and young adulthood in this study have a higher risk for experiencing a CVD event in later adulthood compared to those not exposed to IPV. While the effect sizes are not large, the early detection of increased CVD risk in the present study is notable, especially given the young age at which it is being measured (average age 29) and is consistent with prior research indicating a positive relationship between IPV and self-reported diagnosis of CVD (Matthew J. Breiding et al., 2008; Coker et al., 2000b; Gass et al., 2010; Lown and Vega, 2001) and individual CVD risk factors (Clark et al., 2014a; Clark et al., forthcoming; Crane et al., 2013; Crane et al., 2014; Gass et al., 2010; Mason et al., 2012; Mason et al., 2013; Stene et al., 2013). Complex interrelationships among behavioral, psychological, physiological, and inter-relational mechanisms may undergird this association (Ehlert, 2013; Schnurr and Green, 2004; Scott-Storey, 2013; Taylor, 2010). IPV has been associated with smoking (Crane et al., 2013), weight promoting health behaviors (physical inactivity, poor diet, disordered eating) (Ackard and Neumark-Sztainer, 2002; Campbell, 2002; Heise and Garcia-Moreno, 2002; McNutt et al., 2002; Plichta, 2004), and IPV is bi-directionally associated with depressive symptoms (Campbell, 2002; Golding, 1999; Lehrer et al., 2006). Dysregulation following early adversity has been linked to alterations in inflammatory processes, neural regulation, and hypothalamic-pituitary-adrenal (HPA) and metabolic functioning (Bertone-Johnson et al., 2012; Danese and McEwen, 2012; Ehlert, 2013; Fagundes et al., 2013; Johnson et al., 2013; Taylor, 2010). Higher levels of C-reactive protein, a marker of inflammation, and an established risk factor for CVD (Ridker et al., 2000), have been found in adult victims of IPV compared to controls (Fernandez-Botran et al., 2011). Individuals exposed to IPV in adolescence also are at increased risk of later exposure to IPV (Gómez, 2010) and may be sensitized to develop depression subsequent to future stressors (Fogarty et al., 2008; Hammen et al., 2000; McLaughlin et al., 2010) via dysregulation of the HPA axis and persistent sensitization of the stress response (Heim et al., 2008).

The present study did not find bi-directionally violence to be especially cardiotoxic compared to victimization or perpetration only, but this may be due to the crude categorization of bi-directionally violence. A more nuanced examination of patterns of bi-directionally violent relationships is warranted to test for profiles that might be especially

cardiotoxic and for gender and racial-ethnic differences given prior research finding Blacks to be at greater risk of being in a bi-directionally violent relationship (Caetano et al., 2005; Langhinrichsen-Rohling et al., 2012) compared to Whites and gender differences in types of perpetration and victimization (Black et al., 2011; Renner and Whitney, 2010; Spencer et al., 2015).

The study's findings are tempered by several factors. Self-reports of IPV could not be confirmed because most incidents are not reported to verifiable sources such as the police (Coker et al., 2000a; Flicker et al., 2011; Tjaden and Thonnes, 2000) and reliance on formal reports underestimates the occurrence of violence. Recall may is minimized since assessment is at most 7 years from the encounter and the measure is behavior-based reducing the likelihood that events would be perceived differently as the participant matured. Social desirability bias is possible given the sensitive nature of the topic; however, prior research has found its impact on reports of IPV victimization and perpetration to be minimal (Fernandez-Gonzalez et al., 2013; Visschers et al., 2015). A limited number of items were used to measure IPV and individuals might be misclassified if they experienced alternative forms of violence. The study's ability to detect different patterns of IPV experience may be limited by the presence of same-method variance since the same individual reported on their own and their partner's behavior (Visschers et al., 2015). Also, this study did not examine psychological abuse and severe psychological abuse has been demonstrated to be a stronger predictor of incident hypertension (Mason et al., 2012) and diabetes (Mason et al., 2013) than physical and sexual IPV. Rasch modeling was used to better differentiate among individuals experiencing IPV, and took into consideration the respondent's relationship history and salient features of these relationships; however, doing so might have masked potential gender differences in patterns of IPV exposure. Finally, the full complement of biomarkers needed to calculate the 30-year risk score was only available at wave 4, so change in long-term CVD risk relative to exposure could not be examined. However, prior research using Add Health data has found that exposure to IPV is associated with greater increases in BMI and greater likelihood of smoking, especially among women (Clark et al., 2014b; Exner-Cortens et al., 2013). Subsequent waves of Add Health data collection are needed to more elucidate this question.

Conclusion

Given the extent of IPV in late adolescence and young adulthood and its link to increased risk of the nation's leading cause of death, much greater clinical effort is needed to prevent its occurrence. There is a growing awareness of the role of the health provider in violence prevention and the long term health implications of early adversity (Garner and Shonkoff, 2012; Johnson et al., 2013). Ignoring abuse history may compromise risk mitigation, if the underlying issues are not addressed (Edwards et al., 2007; Lopes et al., 2013). Guidance already exists to assist health care providers to educate, assess, and refer for violence (American Academy of Pediatrics, 2006; Moyer, 2013) and there are a number of primary care-based IPV interventions that improve patient well-being (Bair-Merritt et al., 2014) and growing guidance on how to build effective health care responses to IPV (Miller et al., 2015). While more evidence is needed on the link between IPV and CVD risk to inform clinical practice, the growing body of research suggests that addressing violence may

improve the success of health promotion and maintenance efforts and stymy the erosion of health attributable to violence exposure.

ACKNOWLEDGEMENTS

This research uses data from Add Health, a program project directed by Kathleen Mullan Harris and designed by J. Richard Udry, Peter S. Bearman, and Kathleen Mullan Harris at the University of North Carolina at Chapel Hill, and funded by grant P01-HD31921 from the Eunice Kennedy Shriver National Institute of Child Health and Human Development, with cooperative funding from 23 other federal agencies and foundations. Special acknowledgment is due Ronald R. Rindfuss and Barbara Entwisle for assistance in the original design. Information on how to obtain the Add Health data files is available on the Add Health website (http://www.cpc.unc.edu/addhealth). No direct support was received from grant P01-HD31921 for this analysis.

Funding Sources:

Dr. Cari Jo Clark was supported by National Center for Advancing Translational Sciences of the NIH Award Number 8UL1TR000114–02/ KL2TR000113 and grant number R03HD068045 from the Eunice Kennedy Shriver National Institute of Child Health and Human Development. Additional support was provided by the Program in Health Disparities Research and the Applied Clinical Research Program at the University of Minnesota. Dr. Susan A. Everson-Rose was supported in part by grant 1P60MD003422 from the National Institute on Minority Health and Health Disparities and by the Program in Health Disparities Research and the Applied Clinical Research Program at the University of Minnesota. Study sponsors had no role in study design; collection, analysis, or interpretation of data; writing the report; or the decision to submit the report for publication. The content is solely the responsibility of the authors and does not necessarily represent the official views of the National Institutes of Health.

REFERENCES

Ackard DM, Neumark-Sztainer D, 2002. Date violence and date rape among adolescents: associations with disordered eating behaviors and psychological health. Child Abuse & Neglect 26:455–73. [PubMed: 12079084]

American Academy of Pediatrics, 2006. Connected Kids: Safe, Strong, Secure Clinical Guide, in: Spivak, H., Sege, R., Flanigan, E., Licenziato, V. (Eds.). American Academy of Pediatrics, Elk Grove Village, IL.

Bair-Merritt MH, Lewis-O'Connor A, Goel S, Amato P, Ismailji T, Jelley M, Lenahan P, Cronholm P, 2014. Primary care-based interventions for intimate partner violence: a systematic review. Am J Prev Med 46:188–94. [PubMed: 24439354]

Batten SV, Aslan M, Maciejewski PK, Mazure CM, 2004. Childhood maltreatment as a risk factor for adult cardiovascular disease and depression. J Clin Psychiatry 65:249–54. [PubMed: 15003081]

Bertone-Johnson ER, Whitcomb BW, Missmer SA, Karlson EW, Rich-Edwards JW, 2012. Inflammation and early-life abuse in women. American journal of preventive medicine 43:611–20. [PubMed: 23159256]

Black MC, Basile KC, Breiding MJ, Smith SG, Walters ML, Merrick MT, Chen J, Stevens MR, 2011. The national intimate partner and sexual violence survey (NISVS): 2010 summary report. National Center for Injury Prevention and Control, Centers for Disease Control and Prevention, Atlanta, GA.

Breiding M, Chen J, Black M, 2014. Intimate Partner Violence in the United States - 2010. National Center for Injury Prevention and Control, Centers for Disease Control and Prevention, Atlanta, GA.

Breiding MJ, Black MC, Ryan GW, 2008. Chronic disease and health risk behaviors associated with intimate partner violence-18 U.S. states/territories, 2005. Annals of epidemiology 18:538–44. [PubMed: 18495490]

Breiding MJ, Black MC, Ryan GW, 2008. Chronic Disease and Health Risk Behaviors Associated with Intimate Partner Violence—18 U.S. States/Territories, 2005. Annals of epidemiology 18:538–44. [PubMed: 18495490]

Caetano R, Ramisetty-Mikler S, Field CA, 2005. Unidirectional and bidirectional intimate partner violence among White, Black, and Hispanic couples in the United States. Violence and victims 20:393–406. [PubMed: 16250407]

Campbell JC, 2002. Health consequences of intimate partner violence. Lancet 359:1331–6. [PubMed: 11965295]

Chantala K, Tabor J, 2010. Strategies to Perform a Design-Based Analysis Using the Add Health Data. Carolina Population Center, University of North Carolina at Chapel Hill, p. 19.

Clark CJ, Alonso A, Spencer RA, Pencina M, Williams K, Everson-Rose SA, 2014a. Predicted long-term cardiovascular risk among young adults in the National Longitudinal Study of Adolescent Health. American Journal of Public Health 104:e108–15.

Clark CJ, Everson-Rose SA, Alonso A, Spencer RA, Brady SS, Resnick MD, Borowsky IW, Connett JE, Krueger RF, et al. , 2014a. Effect of partner violence in adolescence and young adulthood on blood pressure and incident hypertension. PloS one 9:e92204.

Clark CJ, Spencer RA, Everson-Rose SA, Brady SS, Mason SM, Connett JE, Henderson KM, To M, Suglia SF, 2014b. Dating Violence, Childhood Maltreatment, and BMI From Adolescence to Young Adulthood. Pediatrics 134:678–85. [PubMed: 25201793]

Clark CJ, Spencer RA, Everson-Rose SA, Brady SS, Mason SM, Connett JE, Henderson KM, To M, Suglia SF, 2014b. Dating violence, childhood maltreatment, and BMI from adolescence to young adulthood. Pediatrics 134:678–85. [PubMed: 25201793]

Clark CJ, Spencer RA, Everson Rose SA, Brady SS, Mason SM, Connett JE, Henderson KM, To M, Suglia SF, forthcoming. Dating Violence, Childhood Maltreatment, and Body Mass Index from Adolescence to Young Adulthood. . Pediatrics.

Coker AL, Derrick C, Lumpkin JL, Aldrich TE, Oldendick R, 2000a. Help-seeking for intimate partner violence and forced sex in South Carolina. American journal of preventive medicine 19:316–20. [PubMed: 11064237]

Coker AL, Smith PH, Bethea L, King MR, McKeown RE, 2000b. Physical health consequences of physical and psychological intimate partner violence. Arch Fam Med 9:451–7. [PubMed: 10810951]

Crane CA, Hawes SW, Weinberger AH, 2013. Intimate partner violence victimization and cigarette smoking: a meta-analytic review. Trauma, violence & abuse 14:305–15.

Crane CA, Pilver CE, Weinberger AH, 2014. Cigarette smoking among intimate partner violence perpetrators and victims: Findings from the National Epidemiologic Survey on Alcohol and Related Conditions. The American Journal on Addictions:n/a-n/a.

Cunradi CB, Caetano R, Clark C, Schafer J, 2000. Neighborhood Poverty as a Predictor of Intimate Partner Violence Among White, Black, and Hispanic Couples in the United States: A Multilevel Analysis. Annals of epidemiology 10:297–308. [PubMed: 10942878]

Danese A, McEwen BS, 2012. Adverse childhood experiences, allostasis, allostatic load, and age-related disease. Physiology & behavior 106:29–39. [PubMed: 21888923]

Desquilbet L, Mariotti F, 2010. Dose-response analyses using restricted cubic spline functions in public health research. Stat Med.

Dong M, Giles WH, Felitti VJ, Dube SR, Williams JE, Chapman DP, Anda RF, 2004. Insights into causal pathways for ischemic heart disease: adverse childhood experiences study. Circulation 110:1761–6. [PubMed: 15381652]

Eaton DK, Kann L, Kinchen S, Shanklin S, Ross J, Hawkins J, Harris WA, Lowry R, McManus T, et al. , 2010. Youth risk behavior surveillance - United States, 2009. MMWR Surveill Summ 59:1–142.

Edwards VJ, Anda RF, Gu D, Dube SR, Felitti VJ, 2007. Adverse childhood experiences and smoking persistence in adults with smoking-related symptoms and illness. The Permanente journal 11:5–13.

Ehlert U, 2013. Enduring psychobiological effects of childhood adversity. Psychoneuroendocrinology 38:1850–7. [PubMed: 23850228]

Enders CK, 2010. Applied missing data analysis. The Guilford Press, New York.

Entzel P, Whitsel EA, Richardson A, Tabor J, Hallquist S, Hussey J, Halpern CT, Mullan Harris K, 2009. Add Health Wave IV Documentation: Cardiovascular and Anthropometric Measures. <http://www.cpc.unc.edu/projects/addhealth/data/guides/Wave%20IV%20cardiovascular%20and%20anthropometric%20documentation%20110209.pdf>, p. <http://www.cpc.unc.edu/projects/addhealth/data/guides/Wave%20IV%20cardiovascular%20and%20anthropometric%20documentation%20110209.pdf>.

Exner-Cortens D, Eckenrode J, Rothman E, 2013. Longitudinal associations between teen dating violence victimization and adverse health outcomes. Pediatrics 131:71–8. [PubMed: 23230075]

Fagundes CP, Glaser R, Kiecolt-Glaser JK, 2013. Stressful early life experiences and immune dysregulation across the lifespan. Brain Behav Immun 27:8–12. [PubMed: 22771426]

Fang X, Corso PS, 2007. Child maltreatment, youth violence, and intimate partner violence: developmental relationships. Am J Prev Med 33:281–90. [PubMed: 17888854]

Fernandez-Botran R, Miller JJ, Burns VE, Newton TL, 2011. Correlations among inflammatory markers in plasma, saliva and oral mucosal transudate in post-menopausal women with past intimate partner violence. Brain Behav Immun 25:314–21. [PubMed: 20888902]

Fernandez-Gonzalez L, O'Leary KD, Munoz-Rivas MJ, 2013. We are not joking: need for controls in reports of dating violence. J Interpers Violence 28:602–20. [PubMed: 22948169]

Field CA, Caetano R, 2005. Longitudinal Model Predicting Mutual Partner Violence Among White, Black, and Hispanic Couples in the United States General Population. Violence and victims 20:499–511. [PubMed: 16248487]

Finkelhor D, Dziuba-Leatherman J, 1994. Children as victims of violence: a national survey. Pediatrics 94:413–20. [PubMed: 7936846]

Flicker SM, Cerulli C, Zhao X, Tang W, Watts A, Xia Y, Talbot NL, 2011. Concomitant forms of abuse and help-seeking behavior among white, African American, and Latina women who experience intimate partner violence. Violence against women 17:1067–85. [PubMed: 21821618]

Fogarty CT, Fredman L, Heeren TC, Liebschutz J, 2008. Synergistic effects of child abuse and intimate partner violence on depressive symptoms in women. Prev Med 46:463–9. [PubMed: 18207563]

Freedman DS, Dietz WH, Srinivasan SR, Berenson GS, 1999. The relation of overweight to cardiovascular risk factors among children and adolescents: the Bogalusa Heart Study. Pediatrics 103:1175–82. [PubMed: 10353925]

Garcia L, Qi L, Rasor M, Clark CJ, Bromberger J, Gold EB, 2014. The Relationship of Violence and Traumatic Stress to Changes in Weight and Waist Circumference: Longitudinal Analyses From the Study of Women's Health Across the Nation. Journal of interpersonal violence 29:1459–76. [PubMed: 24212978]

Garner AS, Shonkoff JP, 2012. Early childhood adversity, toxic stress, and the role of the pediatrician: translating developmental science into lifelong health. Pediatrics 129:e224–31. [PubMed: 22201148]

Gass JD, Stein DJ, Williams DR, Seedat S, 2010. Intimate partner violence, health behaviours, and chronic physical illness among South African women. S Afr Med J 100:582–5. [PubMed: 20822646]

Go AS, Mozaffarian D, Roger VL, Benjamin EJ, Berry JD, Borden WB, Bravata DM, Dai S, Ford ES, et al. , 2013. Heart disease and stroke statistics−−2013 update: a report from the American Heart Association. Circulation 127:e6–e245. [PubMed: 23239837]

Goff DC Jr., Lloyd-Jones DM, Bennett G, Coady S, D'Agostino RB Sr., Gibbons R, Greenland P, Lackland DT, Levy D, et al. , 2014. 2013 ACC/AHA guideline on the assessment of cardiovascular risk: a report of the American College of Cardiology/American Heart Association Task Force on Practice Guidelines. J Am Coll Cardiol 63:2935–59. [PubMed: 24239921]

Golding JM, 1999. Intimate partner violence as a risk factor for mental disorders: a meta-analysis. Journal of Family Violence 14:99–132.

Gómez AM, 2010. Testing the Cycle of Violence Hypothesis: Child Abuse and Adolescent Dating Violence as Predictors of Intimate Partner Violence in Young Adulthood. Youth & Society.

Gray HM, Foshee V, 1997. Adolescent Dating Violence: Differences Between One-Sided and Mutually Violent Profiles. Journal of Interpersonal Violence 12:126–41.

Hammen C, Henry R, Daley SE, 2000. Depression and sensitization to stressors among young women as a function of childhood adversity. J Consult Clin Psychol 68:782–7. [PubMed: 11068964]

Harris KM, Halpern CT, Whitsel E, Hussey J, Tabor P, Entzel P, Udry JR, 2009. The National Longitudinal Study of Adolescent Health: Research Design [WWW document].

Heim C, Newport DJ, Mletzko T, Miller AH, Nemeroff CB, 2008. The link between childhood trauma and depression: insights from HPA axis studies in humans. Psychoneuroendocrinology 33:693–710. [PubMed: 18602762]

Heise L, Garcia-Moreno C, 2002. Violence by intimate partners, in: Krug, E.G., Dahlberg, L.L., Mercy, J.A., Zwi, A.B., Lozano, R. (Eds.), World Report on Violence and Health. World Health Organization, Geneva, pp. 87–122.

Hussey JM, Chang JJ, Kotch JB, 2006. Child maltreatment in the United States: prevalence, risk factors, and adolescent health consequences. Pediatrics 118:933–42. [PubMed: 16950983]

Johnson SB, Riley AW, Granger DA, Riis J, 2013. The science of early life toxic stress for pediatric practice and advocacy. Pediatrics 131:319–27. [PubMed: 23339224]

Kann L, Kinchen S, Shanklin SL, Flint KH, Kawkins J, Harris WA, Lowry R, Olsen EO, McManus T, et al. , 2014. Youth risk behavior surveillance--United States, 2013. MMWR Surveill Summ 63 Suppl 4:1–168.

Kavey RE, Daniels SR, Lauer RM, Atkins DL, Hayman LL, Taubert K, 2003. American Heart Association guidelines for primary prevention of atherosclerotic cardiovascular disease beginning in childhood. J Pediatr 142:368–72. [PubMed: 12712052]

Langhinrichsen-Rohling J, Selwyn C, Rohling ML, 2012. Rates of Bidirectional Versus Unidirectional Intimate Partner Violence Across Samples, Sexual Orientations, and Race/Ethnicities: A Comprehensive Review. Partner Abuse 3:199–230.

Lehrer JA, Buka S, Gortmaker S, Shrier LA, 2006. Depressive symptomatology as a predictor of exposure to intimate partner violence among US female adolescents and young adults. Arch Pediatr Adolesc Med 160:270–6. [PubMed: 16520446]

Lopes CS, Sampaio MM, Werneck GL, Chor D, Faerstein E, 2013. Influence of psychosocial factors on smoking cessation: longitudinal evidence from the Pro-Saude Study. Rev Saude Publica 47:732–9. [PubMed: 24346664]

Lown EA, Vega WA, 2001. Intimate partner violence and health: self-assessed health, chronic health, and somatic symptoms among Mexican American women. Psychosom Med 63:352–60. [PubMed: 11382262]

Mason SM, Wright RJ, Hibert EN, Spiegelman D, Forman JP, Rich-Edwards JW, 2012. Intimate partner violence and incidence of hypertension in women. Annals of epidemiology 22:562–7. [PubMed: 22717307]

Mason SM, Wright RJ, Hibert EN, Spiegelman D, Jun HJ, Hu FB, Rich-Edwards JW, 2013. Intimate partner violence and incidence of type 2 diabetes in women. Diabetes Care 36:1159–65. [PubMed: 23248189]

McLaughlin KA, Conron KJ, Koenen KC, Gilman SE, 2010. Childhood adversity, adult stressful life events, and risk of past-year psychiatric disorder: a test of the stress sensitization hypothesis in a population-based sample of adults. Psychol Med 40:1647–58. [PubMed: 20018126]

McNutt LA, Carlson BE, Persaud M, Postmus J, 2002. Cumulative abuse experiences, physical health and health behaviors. Ann Epidemiol 12:123–30. [PubMed: 11880220]

Miller E, McCaw B, Humphreys BL, Mitchell C, 2015. Integrating intimate partner violence assessment and intervention into healthcare in the United States: a systems approach. J Womens Health (Larchmt) 24:92–9. [PubMed: 25606823]

Moyer VA, 2013. Screening for intimate partner violence and abuse of elderly and vulnerable adults: U.S. preventive services task force recommendation statement. Ann Intern Med 158:478–86. [PubMed: 23338828]

Murray ET, Roux AVD, Carnethon M, Lutsey PL, Ni H, O'Meara ES, 2010. Trajectories of Neighborhood Poverty and Associations With Subclinical Atherosclerosis and Associated Risk Factors The Multi-Ethnic Study of Atherosclerosis. American journal of epidemiology:kwq044.

Pencina MJ, D'Agostino RB Sr., Larson MG, Massaro JM, Vasan RS, 2009. Predicting the 30-year risk of cardiovascular disease: the framingham heart study. Circulation 119:3078–84. [PubMed: 19506114]

Plichta SB, 2004. Intimate partner violence and physical health consequences: policy and practice implications. J Interpers Violence 19:1296–323. [PubMed: 15534333]

Renner LM, Whitney SD, 2010. Examining symmetry in intimate partner violence among young adults using socio-demographic characteristics. Journal of Family Violence 25:91–106.

Renner LM, Whitney SD, 2012. Risk factors for unidirectional and bidirectional intimate partner violence among young adults. Child abuse & neglect 36:40–52. [PubMed: 22269774]

Ridker PM, Hennekens CH, Buring JE, Rifai N, 2000. C-reactive protein and other markers of inflammation in the prediction of cardiovascular disease in women. N Engl J Med 342:836–43. [PubMed: 10733371]

Sato-DiLorenzo A, Sharps PW, 2007. Dangerous intimate partner relationships and women's mental health and health behaviors. Issues Ment Health Nurs 28:837–48. [PubMed: 17729169]

Schnurr PP, Green BL, 2004. Understanding relationships among trauma, posttraumatic stress disorder and health outcomes, in: Schnurr PP, Green BL (Eds.), Trauma and health: physical health consequences of exposure to extreme stress. American Psychological Association, Washington, D.C., pp. 247–75.

Scott-Storey KA, 2013. Abuse as a Gendered Risk Factor for Cardiovascular Disease A Conceptual Model. Journal of Cardiovascular Nursing 28:E1–E8.

Skinner AC, Skelton JA, 2014. Prevalence and Trends in Obesity and Severe Obesity Among Children in the United States, 1999–2012. JAMA Pediatr.

Spencer RA, Renner LM, Clark CJ, 2015. Patterns of Dating Violence Perpetration and Victimization in U.S. Young Adult Males and Females. J Interpers Violence.

Stene LE, Jacobsen GW, Dyb G, Tverdal A, Schei B, 2013. Intimate partner violence and cardiovascular risk in women: a population-based cohort study. J Womens Health (Larchmt) 22:250–8. [PubMed: 23428282]

Straus M, Hamby SL, Boney-McCoy S, Sugarman DB, 1996. The Revised Conflict Tactics Scales (CTS2): Development and Preliminary Psychometric Data. Journal of Family Issues 17:283–316.

Straus MA, Kinard EM, Williams LM, 2008. The Multidimensional Neglectful Behavior Scale, Form A: Adolescent and Adult-Recall Version, 6 March 2008 ed. University of New Hampshire, Family Research Laboratory, Durham, NH.

Suglia SF, Ryan L, Wright RJ, 2008. Creation of a community violence exposure scale: accounting for what, who, where, and how often. Journal of traumatic stress 21:479–86. [PubMed: 18956446]

Suglia SF, Sapra KJ, Koenen KC, 2015. Violence and cardiovascular health: a systematic review. Am J Prev Med 48:205–12. [PubMed: 25599905]

Tabor J, Whitsel EA, 2010. Add Health Wave IV Documentation: Precription Medication Use. http://www.cpc.unc.edu/projects/addhealth/data/guides/medication_documentation.pdf.

Taylor SE, 2010. Mechanisms linking early life stress to adult health outcomes. Proceedings of the National Academy of Sciences of the United States of America 107:8507–12. [PubMed: 20442329]

Tjaden P, Thonnes N, 2000. Full Report of the Prevalence, Incidence, and Consequences of Violence Against Women: Findings From the National Violence Against Women Survey. National Institute of Justice and the Centers for Disease Control and Prevention.

Visschers J, Jaspaert E, Vervaeke G, 2015. Social Desirability in Intimate Partner Violence and Relationship Satisfaction Reports: An Exploratory Analysis. J Interpers Violence.

Wegman HL, Stetler C, 2009. A meta-analytic review of the effects of childhood abuse on medical outcomes in adulthood. Psychosom Med 71:805–12. [PubMed: 19779142]

Weintraub WS, Daniels SR, Burke LE, Franklin BA, Goff DC Jr., Hayman LL, Lloyd-Jones D, Pandey DK, Sanchez EJ, et al. , 2011. Value of primordial and primary prevention for cardiovascular disease: a policy statement from the American Heart Association. Circulation 124:967–90. [PubMed: 21788592]

Whitaker DJ, Haileyesus T, Swahn M, Saltzman LS, 2007. Differences in frequency of violence and reported injury between relationships with reciprocal and nonreciprocal intimate partner violence. American journal of public health 97:941–7. [PubMed: 17395835]

Whitsel EA, Tabor JW, Nguyen QC, Cuthbertson CC, Wener MH, Potter AJ, Killeya-Jones LA, Mullan Harris K, 2012. Measures of Glucose Homeostasis: Add Health Wave IV Documentation. Carolina Population Center, University of North Carolina at Chapel Hill, Chapel Hill, NC.

Yount KM, Li L, 2011. Domestic violence and obesity in Egyptian women. J Biosoc Sci 43:85–99. [PubMed: 20809993]

Table 1.

Participant socio-demographic characteristics by sex in the National Longitudinal Study of Adolescent to Adult Health (1995–2008/09), weighted (N=9,976).

	Males (N=4,472)	Females (N=5,504)	p-value
Age, Wave 1 mean (range)	15.53 (11, 21)	15.32 (11, 21)	<.01
Age, Wave 3 mean (range)	21.92 (18, 27)	21.72 (18, 27)	<.01
Age, Wave 4 mean (range)	28.94 (25, 34)	28.73 (24, 34)	<.01
Race/Ethnicity, Wave 3 n (%)			0.88
White	2,512 (66.99)	3,021 (67.15)	
Black	764 (13.93)	1,143 (14.81)	
American Indian/Native American	29 (0.65)	37 (0.68)	
Asian / Pacific Islander	280 (3.12)	296 (2.93)	
Mixed	171 (3.53)	201 (3.28)	
Hispanic	716 (11.78)	805 (11.15)	
Years of education past 6th grade, Wave 3 mean (95% CI)	6.98 (6.79, 7.18)	7.26 (7.08, 7.45)	<.01
Financial stress, Wave 3 n (%)	1,328 (29.73)	1,951 (36.41)	<.01
Parental education, Wave 1 n (%)[a]			
Less than high school	655 (15.01)	959 (16.49)	0.12
High school	1290 (30.89)	1619 (32.89)	
Some college	1353 (29.43)	1566 (27.73)	
College / university graduate	1174 (24.66)	1361 (22.88)	
Neighborhood poverty, Wave 1 mean (95% CI)	0.12 (0.10, 0.13)	0.12 (0.10, 0.13)	0.95
Child abuse and neglect, Wave 3 n (%)			
Inadequate supervision	543 (11.46)	587 (10.56)	0.29
Physical neglect	603 (14.09)	488 (8.53)	<.01
Physical abuse	424 (8.73)	483 (8.56)	0.85
Sexual abuse	234 (5.52)	289 (5.12)	0.55
Victimization score, Wave 3 mean (95% CI)	0.87 (0.79, 0.94)	0.98 (0.91, 1.04)	Na
Perpetration score, Wave 3 mean (95% CI)	0.65 (0.59, 0.71)	0.86 (0.80, 0.92)	Na
Intimate partner violence by type, Wave 3 n (%)			<.01
None	2982 (66.16)	2938 (54.17)	

	Males (N=4,472)	Females (N=5,504)	p-value
Victimization only	550 (12.54)	448 (8.45)	
Perpetration only	217 (4.28)	670 (11.58)	
Both victimization and perpetration	723 (17.02)	1448 (25.80)	
30-year CVD risk score, Wave 4 mean (95% CI)	17.32 (16.74, 17.90)	9.03 (8.64, 9.43)	<.01

[a]Categorized for descriptive purposes.

Table 2.

Relationship Between Partner Violence Victimization and Perpetration and Thirty-year Framingham Risk Score in the National Longitudinal Study of Adolescent to Adult Health (2008–2009), weighted (N=9,976).

	Victimization		Perpetration	
	Beta (95% CI)	**p-value**	**Beta (95% CI)**	**p-value**
Violence score	0.28 (0.03, 0.54)	0.03	0.33 (0.03, 0.62)	0.03
Sex				
Male	7.92 (7.49, 8.34)	<.01	7.96 (7.52, 8.39)	<.01
Female	Ref			
Age	1.29 (1.16, 1.41)	<.01	1.29 (1.16, 1.41)	<.01
Race/Ethnicity				
White	Ref			
Black	0.80 (0.07, 1.53)	0.03	0.75 (0.00, 1.50)	0.05
American Indian/Native American	3.22 (−1.11, 7.54)	0.14	3.16 (−1.19, 7.50)	0.15
Asian / Pacific Islander	−1.36 (−2.47, −0.24)	0.02	−1.37 (−2.48, −0.25)	0.02
Mixed	0.95 (−0.37, 2.28)	0.16	0.98 (−0.34, 2.29)	0.14
Hispanic	−1.06 (−1.76, −0.36)	<.01	−1.09 (−1.79, −0.38)	<.01
Respondent education	−0.52 (−0.66, −0.38)	<.01	−0.52 (−0.66, −0.37)	<.01
Financial stress	1.09 (0.59, 1.60)	<.01	1.09 (0.60, 1.58)	<.01
Parental education	−0.10 (−0.21, 0.02)	0.10	−0.09 (−0.21, 0.02)	0.11
Neighborhood poverty, per 10% change	0.65 (0.17, 1.14)	0.01	0.65 (0.17, 1.14)	0.01
Neighborhood poverty quadratic term	−0.07 (−0.16, 0.01)	0.09	−0.07 (−0.16, 0.01)	0.09
Child abuse and neglect				
Inadequate supervision	0.29 (−.40, 0.98)	0.41	0.29 (−0.40, 0.98)	0.41
Physical neglect	0.97 (0.08, 1.86)	0.03	0.95 (0.06, 1.84)	0.04
Physical abuse	−0.31 (−1.03, 0.42)	0.40	−0.31 (−1.04, 0.42)	0.40
Sexual abuse	0.38 (−0.83, 1.59)	0.54	0.30 (−0.89, 1.48)	0.62

Note: Table presents results of linear regression models accounting for survey design and unequal probability of selection. Separate models were computed for victimization and perpetration and each was adjusted for the variables listed.