Preventive Medicine 121 (2019) 47–54

Contents lists available at ScienceDirect

Preventive Medicine

journal homepage: www.elsevier.com/locate/ypmed

The impact of intimate partner violence, depressive symptoms, alcohol dependence, and perceived stress on 30-year cardiovascular disease risk among young adult women: A multiple mediation analysis☆

Elizabeth Novack Wright*, Alexandra Hanlon, Alicia Lozano, Anne M. Teitelman

University of Pennsylvania School of Nursing, Philadelphia, PA, United States of America

ARTICLE INFO

Keywords:
Domestic violence
Cardiovascular disease
Stress
Depression
Disease prevention

ABSTRACT

Intimate partner violence (IPV), the physical, sexual, psychological abuse or control by a former or current intimate partner, affects almost one-third of women in the United States. IPV exposure can result in many negative outcomes including physical injury, increased stress, and depression. Currently, there is a small, but, growing body of literature examining the link between IPV victimization and increased cardiovascular disease (CVD) risk among young adult women. To better prevent this negative outcome, it is imperative to understand what factors associated with IPV victimization may be increasing this risk. A secondary analysis of Wave IV of the Add Health study was conducted to examine possible factors mediating past year IPV exposure and 30-year CVD risk score including perceived stress, depressive symptoms, and alcohol dependence among a representative sample of young adult women in the United States. Multiple mediation analyses were run to examine the possible mediating factors in the relationship between IPV and CVD risk. In a multiple mediation model, the indirect effect of perceived stress became insignificant when depressive symptoms were introduced. The findings of this study reveal that 30-year CVD risk in the context of IPV victimization should continue to be examined among this population. The mediation models suggested the importance of stress and depression in the context of IPV and heart health. Screening for depression among women exposed to IPV should be considered as an important intervention point, not only to mitigate mental health issues, but to also help prevent the development of cardiovascular disease.

1. Introduction

Intimate partner violence (IPV), the physical, sexual, psychological abuse or control by a former or current intimate partner, has been associated with negative health outcomes and coping behaviors (Breiding et al., 2014). Almost one in three women will experience some type of IPV in their lifetime with young women most at risk as a majority of victims will experience their first abuse before the age of 25 (Breiding et al., 2014). Researchers are investigating the connection between IPV and cardiovascular disease (CVD) among women, especially younger women, as IPV has been called a gendered-risk factor for CVD, however this link is not well understood (Scott-Storey, 2013). While the overall death rate from CVD has declined over the past 10 years, as of 2013, CVD still accounts for almost one in three deaths among women in the U.S. (Mozaffarian et al., 2016). CVD related deaths are occurring among younger ages. (Mozaffarian et al., 2016). In a sample of young adults ages 20 to 45 years, 59% had either coronary heart disease (CHD), a CHD equivalent, or one or more risk factors for CHD, yet CHD screening rates among the age group were low (Kuklina et al., 2010). Women ages 35 to 44 years have seen an increase in CHD mortality rates with an average increase in mortality rate of 1.3% between the years 1997–2002 (Mosca et al., 2011). When examining the link between IPV and CVD among young women, examining CVD *risk* may allow us to better understand how to prevent CVD from occurring among this population. A 30-year CVD risk model, based on the Framingham Heart Study, was used to calculate 30-year CVD risk in young adults using well-known risk factors including blood pressure, diabetes diagnosis, and smoking status (Pencina et al., 2009). One study used this risk calculation among a sample of young adults and found a one-standard deviation increase in IPV victimization score was associated with a

☆ The authors do not have any pertinent disclosures to make including any real or perceived conflicts of interests. Dr. Wright wrote the first draft of the manuscript with all co-authors contributing to the editing of the final manuscript.

* Corresponding author at: 418 Curie Blvd, Philadelphia, PA 19104, United States of America.
E-mail addresses: novack@nursing.upenn.edu (E.N. Wright), alhanlon@nursing.upenn.edu (A. Hanlon), alozano@nursing.upenn.edu (A. Lozano), teitelm@nursing.upenn.edu (A.M. Teitelman).

https://doi.org/10.1016/j.ypmed.2019.01.016
Received 21 August 2018; Received in revised form 1 December 2018; Accepted 24 January 2019
Available online 26 January 2019
0091-7435/

0.28% increase in 30-year CVD risk (Clark et al., 2014).

While the causal links between IPV and CVD risk are still not clear, there may be a possible pathway through well-studied coping mechanisms, behaviors, and outcomes associated with IPV such as risky alcohol use, stress, and depression (Ashare et al., 2011; Bosch et al., 2015; Calvete and Corral, 2007; Sullivan et al., 2015; Ullman and Sigurvinsdottir, 2015). High alcohol use can increase total cholesterol, narrow blood vessels, and cause decreased blood flow (McEwen and Seeman, 2009). Increased or heavy alcohol use, > 20 g per day, can cause an increased risk of hypertension in women, and alcohol use of 15 g or more can cause an increased risk for Type 2 diabetes (Mekary et al., 2011; Witteman et al., 1990).

Women experiencing IPV have reported high mental health service use (Rivara et al., 2007). Not only has IPV been associated with depression, but also young women experiencing depression can be at increased risk for subsequent IPV (Chuang et al., 2012; Connelly et al., 2013; Devries et al., 2013; Lehrer et al., 2006). Depression, separate from IPV, disproportionately affects women compared to men in the U.S. and has been noted as one of the most significant health risks for women (Glied and Kofman, 1995). Depressive symptoms have also been associated with left ventricular dysfunction (Gustad et al., 2016). Similarly, a longitudinal population-based sample of Australian women found depression to be a long-term indicator of 18-year coronary heart disease incidence independent of typical and atypical risk factors (O'Neil et al., 2016).

Post-traumatic stress disorder and chronic stress, outcomes associated with IPV, have been connected to CVD risk (Coughlin, 2011; Edmondson and Cohen, 2013). Chronic stress can impact the body's ability to regulate its stress-response system leading to increased heartrate and blood pressure, development of atherosclerosis, and insulin resistance, which are risk factors for CVD development. (McEwen and Seeman, 2009).

The connection between IPV, its associated outcomes and CVD risk factors highlight an important need to examine CVD and CVD risk among those who experience IPV. Understanding the connection between IPV among young women and their CVD risk will allow for the development of interventions that can improve this already victimized population's quality of life.

This study examines potential mediators, including depressive symptoms, perceived stress, and alcohol dependence, on the relationship between IPV and CVD risk among young adult women from the National Longitudinal Study of Adolescent to Adult Health (Add Health).

2. Methods

This study was a cross-sectional, secondary analysis of Add Health, a longitudinal, comprehensive, nationally representative sample of adolescents to adults in the U.S. (Harris et al., 2009). Wave I Add Health data was collected from adolescents in grades 7–12 beginning in 1995. Please refer to Harris et al. (2009) for further explanation of recruitment. This current study uses the set of responses (Wave IV) of adults ages 24–32 (Harris et al., 2009). Incorporating systematic sampling methods and implicit stratification into the Add Health study design ensured this sample is representative of U.S. schools with respect to region of country, urbanity, school size, school type, and ethnicity (Harris et al., 2009). For each subsequent wave (Wave II-IV) participants were sampled from Wave I. Due to the focus on female IPV victimization, those who self-identified as male in the survey were excluded from this analysis. Participants with a history of cancer or current CVD were also excluded from the study. All remaining participants who responded to Wave IV questions pertaining to the romantic partner relationship questions and had valid sample weights (N = 7392) were included in the current analysis. See Fig. 1 for the sample size flow chart. This study was determined exempt from review by the University of Pennsylvania Institutional Review Board.

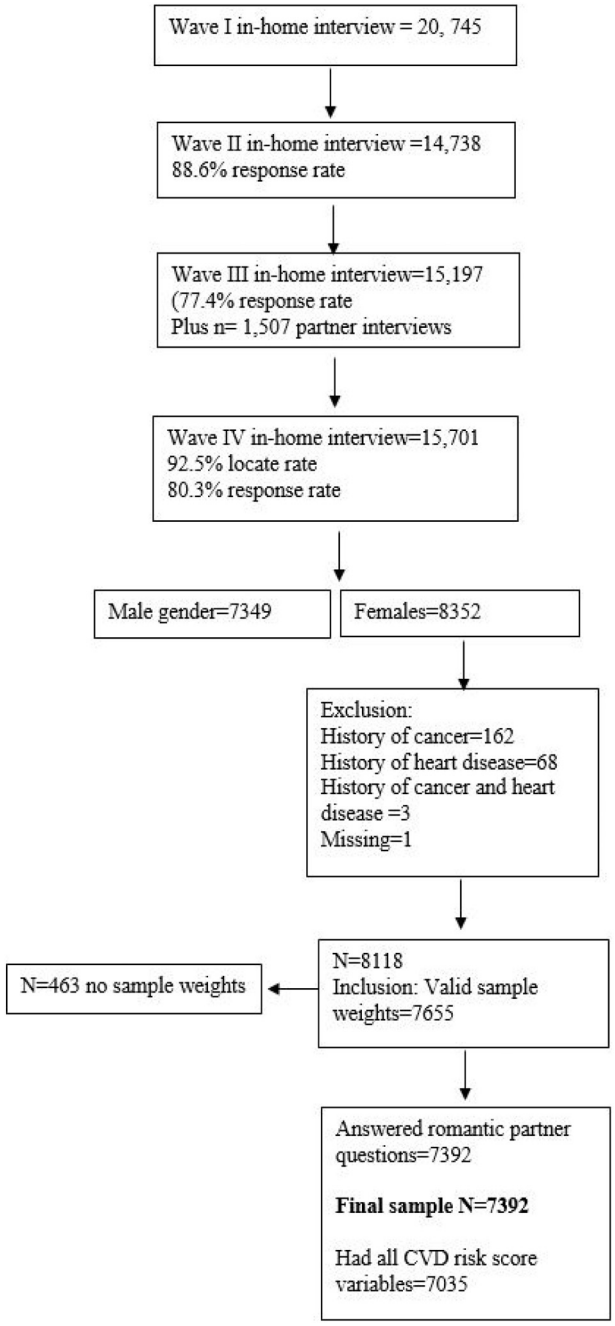


Fig. 1. Sample size flow chart.
Add Health.

2.1. Measures

The outcome variable, 30-year CVD risk score, was calculated based off of the prediction model of Pencina et al. (2009), which uses a Cox proportional hazards model that has been modified to account for competing causes of death. This score estimated "general" CVD (coronary death, myocardial infarction, coronary insufficiency, angina pectoris, stroke, transient ischemic attack, intermittent claudication and congestive heart failure) risk for each participant in this study, with scores ranging from 0% to 100%. The risk factors in the 30-year CVD risk score are age, gender, systolic blood pressure, use of antihypertensive medications, diabetes diagnosis, body mass index (weight in kilometers divided by height in centimeters squared) and status. Please refer to Wright et al. (2018) for full description of variable

construction.

The primary predictor variable in this present study was exposure to intimate partner violence (IPV) in the past year. The Add Health Wave IV survey assessed physical and sexual IPV using four questions from the Revised Conflict Tactic Scales (CTS2) (Cronbach's alpha = 0.76) (Cui et al., 2013; Harris et al., 2009; Straus et al., 1996). Each question had Likert-scale responses ranging from 0 = this never happened to 7 = > 20 times in the past year. A response of "1" indicated "this has not happened this year, but has happened in the past". The participants were asked to answer these questions about their most current partner over the last year. Example of questions included: "How often has your partner threated you with violence, pushed or shoved you, or thrown something at you that could hurt?" and "How often have they insisted on or made you have sexual relations with them when you didn't want to?" Participants who provided any affirmative response to a victimization question were coded as having IPV exposure; those who did not were coded as no.

Depression was measured by an adapted validated 5-item version of the Center for Epidemiologic Studies Depression (CES-D) Scale (Radloff, 1977). Responses were scored and summed. Scores range from 0 to 15 with a higher score indicating more depressive symptoms. Depression was measured as a continuous variable.

Perceived stress was measured from a validated four item 5-level scale adapted from the Cohen Perceived Stress Scale (Cohen et al., 1983). The questions assessed respondents' feelings of lack of control and stress over the past month with summed scores of the questions ranging from 0 to 16. Responses were scored and summed, with the positive phrases questions reversed scored. A higher scored indicated higher perceived stress. Perceived stress was measured as a continuous variable. These scores can also be classified into categorizes such as into low (0–3), medium (4–6), and high (7–16) perceived stress (Dowd et al., 2014).

Alcohol Dependence was measured by the Alcohol Dependence measure from the *DSM-IV*. Diagnosis of a 12-month alcohol dependence requires that respondents satisfy three or more *DSM-IV* criteria for dependence in the past year or during any year before the past year (Hingson et al., 2006). An 8-item questionnaire with responses as *yes* or *no* was used to calculate alcohol dependence. This was used as a continuous variable in the mediation models. The literature states that scores higher than 3 or more is considered alcohol dependence (Hingson et al., 2006).

Covariates were selected based on the literature and have been used in prior studies examining partner violence victimization (Ahmed and McCaw, 2010; Basu et al., 2013; Cerulli et al., 2010; Cheng and Lo, 2014; Cho and Kim, 2012; Connelly et al., 2013; Flicker et al., 2011;Fox and Benson, 2006; Humphreys et al., 2012; Kothari et al., 2009). These covariates included: health insurance status, history of childhood abuse, race, ethnicity, sexual orientation, education, midpoint household income, financial stress, health status and pregnancy status. All data on insurance status was collected prior to the implementation of the Affordable Care Act. See Table 1 for covariate responses.

2.2. Statistical analysis

Descriptive statistics were used to characterize all variables. Frequencies and percentages were used to describe categorical variables, and measures of central tendency (mean, median) and variation (standard deviation, interquartile range, range) were used to describe continuous variables. Chi-square tests and two-sample *t*-tests, as appropriate, were used to examine differences in all variables between female survey participants with exposure to intimate partner violence victimization versus those without.

Simple mediation using the Baron and Kenny (1986) method and multiple mediation using the multivariate delta method were used to examine mediation of depressive symptoms, perceived stress, and alcohol dependence on the relationship between past year IPV and 30-year CVD risk. Simple mediation involves examining one mediator at a time, while multiple mediation allows for two or more mediators to be included in the same model to examine whether a mediator's effect is independent on the effect of the other mediators (Preacher & Hayes, 2008).

Per the Baron & Kenney method, three separate linear regression models were generated to demonstrate mediation. In the first model, 30-year CVD risk was regressed on past year IPV. In the second model, a single mediator of interest was regressed on past year IPV. Lastly, in the third model, 30-year CVD risk was regressed on past year IPV and controlled for the mediator of interest. Full or partial mediation was determined using three conditions; first, past year IPV was statistically significant in the first model. Next, past year IPV was significant predictor of the mediator in the second model. For full mediation, there was a reduction in the magnitude of the estimate for past year IPV in the third model (compared to the first model), and past year IPV was no longer a statistically significant predictor of 30-year risk when controlling for the mediator of interest. For partial mediation, there was a reduction in the magnitude of the estimate for past year IPV in the third model (compared to the first model), and past year IPV remained statistically significant after controlling for the mediator of interest.

For the multiple mediation, all three mediators of interest (depression, perceived stress, alcohol dependence) were simultaneously included in the model. The multivariate delta method approximates standard errors of the total indirect effect and specific indirect effects and is appropriate to use in large sample sizes (Preacher & Hayes, 2008). Partial and full mediation were determined in a similar fashion as the Baron & Kenny method for the multiple mediation model. Standardized estimates and their standard errors were provided for all mediation models. All analyses included sample weights to reflect the complex sampling of the study. Survey weights ensure all estimates are unbiased and results are generalizable in samples with complex survey design and unequal probability of selection (Chantala & Tabor, 2010). Statistical significance was taken at the 0.05 level. Descriptive statistics were generated used SAS version 9.4 (SAS Institute Inc., Cary, NC), and mediation analyses were performed using MPlus 7 (Muthén and Muthén, 2012).

3. Results

3.1. Sample characteristics

Table 2 displays the sample characteristics. The mean age of the women was 29 years (SD = 9.34), with a mean 30-year CVD risk score of 8.2%. Of the women in the sample, 15% reported any IPV in the past year. In terms of mediators, on average, participants demonstrated medium perceived stress levels (Mean = 5.05, SD = 0.07), few depressive symptoms (Mean = 2.83, SD = 0.05), and little to no alcohol dependence (Mean = 0.67, SD = 0.03). Participants exposed to IPV in the past year had statistically significantly higher perceived stress (Mean = 6.30 vs. Mean = 4.79, $p < .01$), increased depressive symptoms (Mean = 3.90 vs Mean = 2.64, $p < .01$), and higher 30-year CVD risk (Mean = 9.6% vs. Mean = 8.7%, $p < .01$) compared those who did not. No significant differences in alcohol dependence were observed between the two groups (p = .24) (Fig. 2).

3.2. Mediation analysis

3.2.1. Simple mediation analyses

Fig. 3 displays the standardized regression coefficients for the impact of past year IPV on 30-year CVD risk score through perceived stress. IPV was significantly associated with perceived stress (B = 0.49, SE = 0.05, $p < .01$) and perceived stress was significantly associated with 30-year CVD risk score (B = 0.10, SE = 0.02 $p < .01$,). Since past year IPV continued to be significantly associated with 30-year CVD risk score when controlling for the mediator, perceived stress partially

Table 1
Descriptive variables.

Covariates	Collection tool	Measurement	Recoding notes
Health insurance status	Add health developed	No health insurance insured	
Childhood abuse	Add health developed	Childhood abuse No childhood abuse	Binary variables were created using cutoff points for each type of abuse as analyzed in previous research to represent moderate to severe abuse; childhood neglect cutoff was > 10 times, childhood physical abuse cutoff was > 6 times, and childhood sexual abuse was 1 or more times (Gooding et al., 2014). Child abuse was defined as having positive response to any of the binary childhood variables
Race/ethnicity	Add health developed	White, black/African American, American Indian/native American, Asian/Pacific islander or other. Hispanic origin	Collected at wave I
Sexual orientation/sexual identity	Add health developed	Heterosexual Sexual minority	Response options were: 100% heterosexual, mostly heterosexual, bisexual, mostly homosexual, 100% homosexual, and not sexually attracted to males or females. We created a binary variable that grouped any response besides *100% heterosexual* in to a sexual minority women category.
Education level	Investigator developed	No college degree College degree	
Income	Mid-point household income	Range from < 20,000 to > $75,000	
Financial stress	Add health developed	Financial stress No financial stress	Financial stress was affirmative response to any of the following questions: In the past 12 months, was there a time when you were without phone service because you didn't have enough money? In the past 12 months, was there a time when you didn't pay the full amount of the rent or mortgage because you didn't have enough money? In the past 12 months, was there a time when you were evicted from your house or apartment for not paying the rent or mortgage? In the past 12 months, was there a time when you didn't pay the full amount of a gas, electricity, or oil bill because you didn't have enough money? In the past 12 months, was there a time when you had the service turned off by the gas or electric company, or the oil company wouldn't deliver, because payments were not made? In the past 12 months, was there a time when you were worried whether food would run out before you would get money to buy more?
Health status	Add health developed	Excellent Very good Good Fair Poor	
Pregnancy status	Add health developed	Pregnant Not pregnant	

Add Health Wave IV.

mediates the relationship between IPV and 30-year CVD risk (B = 0.10, SE = 0.04, p < .01,). The indirect effect via perceived stress was statistically significant (B = 0.047, SE = 0.010, p < .01). Specifically, the standardized mediated effect of IPV in the past year through perceived stress is equal to a 0.05 increase in 30-year CVD risk as mediated by perceived stress.

Fig. 4 displays the standardized regression coefficients for the impact of past year IPV on 30-year CVD risk score through depressive symptoms. IPV was significantly associated with depressive symptoms (B = 0.47, SE = 0.05, p < .01) and depressive symptoms were also significantly associated with 30-year CVD risk score (B = 0.12, SE = 0.02, p < .01). Depressive symptoms partially mediate the relationship between IPV and 30-year CVD risk score as the relationship between IPV and 30-year CVD remained statistically significant when controlling for depressive symptoms (B = 0.10, SE = 0.04 p < .01). The indirect effect of the mediator in the model was 0.054 (SE = 0.012, p < .01). Specifically, the standardized mediated effect of IPV in the past year through depressive symptoms is equal to a 0.05 increase in 30-year CVD risk. Fig. 5 displays the standardized regression coefficients for the impact of past year IPV on 30-year CVD risk score through alcohol dependence. In this analysis, IPV was not significantly associated with alcohol dependence (B = 0.04, SE = 0.04, p = .33) and alcohol dependence was not significantly associated with 30-year CVD risk (B = 0.03, SE = 0.02, p = .07). The indirect effect via alcohol dependence was also not statistically significant (B = 0.001, SE. = 0.001, p = .39). Therefore alcohol dependence does not mediate the relationship between IPV and 30-year CVD risk score.

3.2.2. Multiple mediation analyses

While alcohol dependence was not significant in the simple mediation model, it was included in the multiple mediation model based on the literature that identifies heavy alcohol use as a possible coping mechanism of IPV (Ashare et al., 2011; Ullman and Sigurvinsdottir, 2015). Fig. 6 displays the standardized regression coefficients for the multiple mediation model including perceived stress, depressive symptoms, and alcohol dependence. When all three mediators are included in a mediation model at once, partial mediation occurred only through only depressive symptoms as the relationship between IPV and 30-year CVD risk was statistically significant (p < .05). Specifically, past year IPV is associated with a 0.05 increase in depressive symptoms compared to no past year IPV (p < .01), while a one standard deviation increase in depressive symptoms is associated with a 0.09 increase in 30-year CVD risk score (p < .01). That is on average, women with past year IPV had more depressive symptoms compared to those without past year IPV, and as depressive symptoms increase, 30-year CVD risk also increases. The specific indirect effect via depressive symptoms was statistically significant (B = 0.04, SE = 0.01, p = .003), where the standardized mediated effect of IPV in the past year through depressive symptoms is equal to a 0.04 increase in 30-year CVD risk. Overall, the total indirect effect of all three mediators in the model is

Table 2
Sample characteristics (n = 7392).

Variable	Total sample (N = 7392)	No past year IPV (N = 6231)	Past year IPV (N = 1161)	p-Value
Age (years), mean (SD)	28.8 (9.340)	28.89 (9.23)	28.84 (5.14)	**p < .01**
Race (n = 7379), n (%)				**p < .01**
White	4368 (59.2)	3812 (61.3)	574(49.6)	
African American	1589(21.5)	1234 (19.8)	355(30.7)	
American Indian	81(1.1)	70(1.1)	11 (0.9)	
Asian/Pacific islander	398(5.4)	343 (5.5)	55 (4.7)	
Mixed/other	925(12.5)	762(12.2)	163 (14.1)	
Ethnicity (n = 7367)				**p < .05**
Hispanic	1161(15.8)	980 (15.8)	181(15.6)	
Non-Hispanic	6206(84.2)	5228 (84.2)	978 (84.3)	
Sexual identity (n = 7379)				**p < .01**
Heterosexual	5919 (80.2)	5067 (81.5%)	852 (73.5)	
Sexual minority	1460 (19.8)	1153 (18.5%)	307 (26.5)	
College degree				**p < .01**
Yes	2688(36.4)	2403 (38.6)	285(24.5)	
No	4704(63.6)	3828 (61.4)	876(75.5)	
Midpoint household income(n = 6979)				**p < .01**
< $20,000	879(12.6)	653(11.1)	226(20.8)	
$20–000-$40,000	1466(21)	1194(20.3)	272(25)	
$40,000–$75,000	2551(36.6)	2201 (37.4)	350(32.2)	
$ > 75,000	2083(29.8)	1843 (31.3)	240(22.1)	
Financial stress				**p < .01**
Yes	1971(26.7)	1486 (23.8)	485(41.8)	
No	5421(73.3)	4745(76.2)	676 (58.2)	
Insured (n = 7384)				**p < .01**
Yes	6130(83)	5242(84.2)	888(76.6)	
No	1254(17)	982(15.8)	272(23.4)	
Self-reported health status				**p < .01**
Excellent	1367(18.5)	1220(19.6)	147(12.7)	
Very good	2834(38.3)	2434(39.1)	400(34.5)	
Good	2495(33.8)	2048 (32.9)	447(38.5)	
Fair	614(8.3)	467 (7.5)	147 (12.7)	
Poor	82(1.2)	62(0.9)	20(1.7)	
Currently pregnant(n = 7358)				**p < .05**
Yes	479(6.5)	413(6.7)	66(5.7)	
No	6879(93.4)	5788 (93.3)	1091(94.3)	
Childhood abuse (n = 7285)				**p < .01**
Yes	1457(20)	1140(18.6)	317(27.7)	
No	5828(80)	5000(81.4)	828 (72.3)	
Perceived stress level (n = 7385)				**p < .01**
Low	2485(33.6)			
Med	2677(36.2)			
High	2223(30.1)			
Mean	5.05 (0.068)	4.794 (5.531)	6.3075 (4.631)	
Depressive symptoms (n = 7389)				**p < .01**
Yes	2293(31)			
No	5096(69)			
Mean	2.829 (0.0479)	2.6409 (4.009)	3.8998 (4.055)	
Alcohol dependence (n = 7390)				p = .241
Yes	741(10)			
No	6649(90)			
Mean	0.665 (0.0299)	6777 (2.351)	0.7294 (1.989)	
Past year IPV				
Yes	1158(15.7)			
No	6221(84.2)			
30-year CVD risk (mean) (n = 7035)	0.0824 (0.056)	0.0869(0.1309)	0.0955(0.0831)	**p < .01**
Systolic blood pressure (mean mmHg) (SE) (n = 7136)	119.859	119.267(0.458)	119.967 (0.458)	**p < .01**
Medication treatment for blood pressure				
Yes	257(3.5)	42 (3.6)	215 (3.4)	
No	7135(96.5)	1119 (96.4)	6016 (96.6)	p = .77
Body mass index (mean) (SE) (n = 7279)	28.97	29.77(0.31)	28.93 (0.21)	**p < .01**
Current smoker (n = 7360)				
Yes	2269(30.1)	484 (41.9)	1785 (28.8)	
No	5091(69.2)	670 (58.1)	4421 (71.2)	**p < .01**
Diabetes				
Yes	520 (6.3)	89 (7.7)	431 (6.9)	
No	6872 (93.7)	1072 (92.3)	5800 (93.1)	p = .77

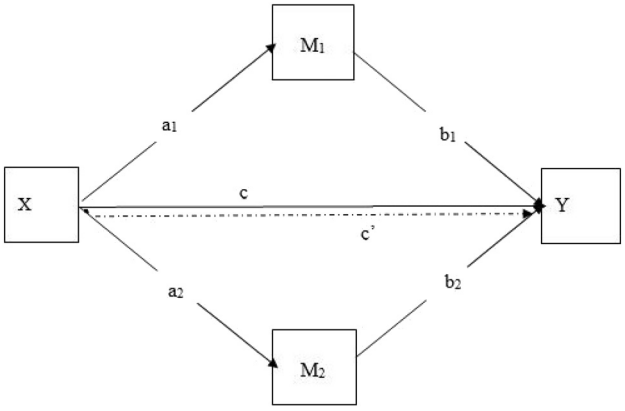


Fig. 2. Multiple mediation.
Add Health Wave IV.

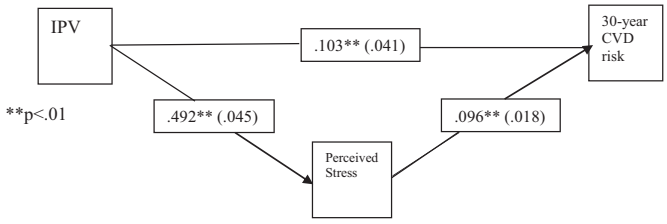


Fig. 3. Standardized regressions coefficients for impact of past year IPV on 30-year CVD score through perceived stress.
Add Health Wave IV.
Total indirect effect = 0.047 (SE = 0.010, p < .01).

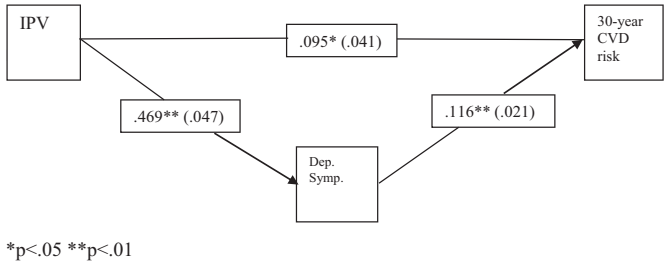


Fig. 4. Standardized regression coefficients for impact of past year IPV on 30-year CVD score through depressive symptoms.
Add Health Wave IV.
*p < .05 **p < .01
Total indirect effect: 0.054 (SE = 0.012, p < .01).

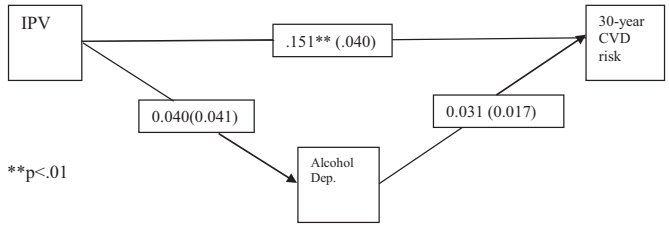
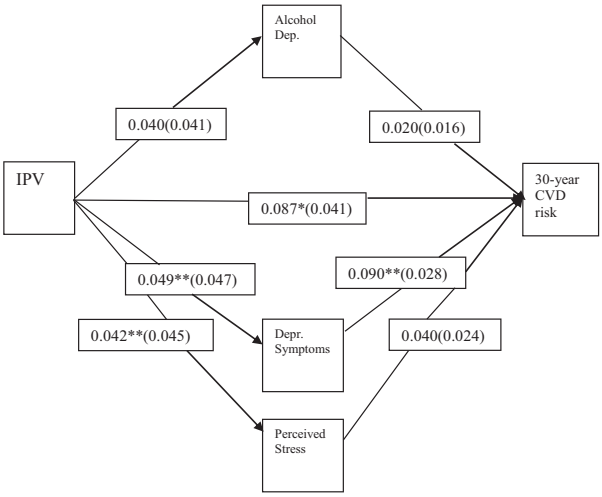


Fig. 5. Standardized regression coefficients for impact of past year IPV on 30-year CVD score through alcohol dependence.
Add Health Wave IV.
**p < .01
Total indirect effect: 0.001 (SE. = 0.001, p = .392).



**p<.01

Fig. 6. Standardized regression coefficients for impact of past year IPV on 30-year CVD score through perceived stress, depressive symptoms, and alcohol dependence.
Add Health Wave IV.
**p < .01
Total indirect effect: 0.063 (SE = 0.012, p < .01).

equal to 0.063 (SE = 0.01, p < .01).

4. Discussion

In our current sample, 15% reported any past year IPV, which is lower than other nationally representative samples (Breiding et al., 2014). The highest risk group for IPV are generally women ages 18–24 years, which may explain the lower proportion among Add Health Wave IV participants (Breiding et al., 2014). Additionally, emotional victimization was not assessed and self-disclosure of IPV is often underreported as well (Ruiz-Pérez et al., 2007).

The simple mediations revealed that perceived stress and depressive symptoms were independent partial mediators of the relationship between IPV and 30-year CVD risk score. These findings are consistent with previous literature that states perception of stress and depressive symptoms are associated with both IPV victimization and subsequent health outcomes including CVD (Chuang et al., 2012: Connelly et al., 2013; Devries et al., 2013; Kendall-Tackett, 2007; Martinez-Torteya et al., 2009; Sabri et al., 2013). These findings also reflect that stress and either the ability or inability to cope with stress may mediate health outcomes (Lazarus & Folkman, 1984). However, in contrast with research that has found heavy alcohol use as an outcome of IPV and a risk factor for CVD, alcohol dependence was not significant in any of the mediation models (Ullman and Sigurvinsdottir, 2015; Witteman et al., 1990). Low rates of alcohol dependence in the sample may have contributed to these insignificant findings.

The multiple mediation model with the proposed mediators of perceived stress, depressive symptoms, and alcohol dependence revealed that when all three variables are included, only depressive symptoms remain as a partial mediator on the relationship between past year IPV and 30-year CVD risk score. The link between stress and depression has been well studied in the literature (Fassett-Carman et al., 2018; Hill and Hoggard, 2018). The perception of stress can impact how one implements a specific coping mechanism (Lazarus & Folkman, 1984). Perceived controllability of a stressor has been shown to impact internalizing symptoms such as depression (Fassett-Carman et al., 2018) Active and positive coping mechanisms to deal with stress may lead to better mental health outcomes. Both the coping mechanism and the effectiveness of coping can contribute to outcomes caused by the

stressor, in this study, IPV. Coping with stress may act as a mediator for mental health outcomes among IPV victims as alexithymia, depression, and attachment issues are negatively correlated with a women's ability to cope with IPV (Craparo et al., 2014). Thus, depression may be the outcome of the inability to cope with a perceived stressor, and therefore may have a more direct effect on CVD risk. However, due to the cross-sectional nature of the study, temporality of these mediators and, therefore, causality cannot be concluded. It is also important to note that there were some significant differences among the specific CVD risk scores between IPV and non-IPV participants. Namely, BMI was higher and current smokers were more likely in the IPV group of participants. Thus, further analysis can should be done that examines differences between IPV and non IPV individuals and their CVD risk. Similarly, there were many significant differences in IPV exposure between different population groups in this study. While many of these differences have been well studied, further examination of these subgroups and their CVD risk should be done to ensure all different subgroups CVD risks can be addressed and managed.

Findings from this multiple mediation model confirm that depressive symptoms play an important role in the relationship between IPV and 30-year CVD risk score. Depression is a well-studied outcome of IPV; and there is body of literature examining the effects of depression on heart health. Depression in otherwise healthy populations is associated with an increased risk of coronary heart disease, heart rate variability, and coronary artery disease (Jangpangi et al., 2016; Lett et al., 2004; Lichtman et al., 2008; Whooley and Wong, 2013). The literature examining depression and heart health cite biological factors such as systemic inflammation and increased cortisol levels as well as behavioral factors such as physical inactivity, smoking, medication non-adherence, and social isolation as contributing to the relationship between depression and poor heart health (Whooley and Wong, 2013). IPV victimization may be considered a contributing factor in the relationship between depression and heart health. IPV victimization has also been shown to be associated with biological factors such as increased inflammation and cortisol levels as well all social factors such as smoking and social isolation (Ashare et al., 2011; Matheson et al., 2015; Newton et al., 2011; Pico-Alfonso et al., 2004). The relationship between IPV, depression, and CVD risk should be further explored. Further research should include physiologic measures of stress.

To our knowledge, this the first study to examine possible mediating factors impacting the relationship between IPV and 30-year CVD risk score. Many of the key variables in this study also used well-known validated measures (Cohen et al., 1983; Harris et al., 2009; Radloff, 1977; Straus et al., 1996). The large, representative sample and the inclusion of sampling weights in the analysis allow for generalizable results. While the mean age of the sample was relatively young to develop measurable risk factors for CVD, the ability to detect any increase in risk at this age should be noted. Further analysis that examines this groups CVD risk overtime as they age should be examined.

Limitations of the study include the measure used to assess IPV victimization; our past year IPV variable did not allow for the measurement of chronicity of violence, emotional abuse, or coercive control all of which may have led to underestimating the health effects of IPV. The rate of alcohol dependence was also relatively low in this sample. A better measure to asses drinking as both a coping mechanism and risk factor for CVD should be used in this population. The Pencina et al. (2009) model has its own measuring limitations including difficulty differentiating between incidence and mortality, this is one of the only risk measurements developed to examine CVD risk in young populations. Lastly, the cross-sectional nature of this study does not allow casualty to be determined.

There is evidence that exposure to IPV can increase CVD risk, even among young adult women. Coping effectiveness may be important in mediating the relationship between IPV and CVD risk through mental health outcomes. Thus, proper mental health services and support should continue to be of importance when working with survivors of IPV. In order to better the health among survivors of IPV and to prevent long term health complications, further research is needed.

Acknowledgments

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. The study was also received funding from the Ortner Center for Family Violence student research fund. The funding source had no involvement in the study design, data collection, analysis, interpretation, write-up or any decisions regarding the publication of results.

Conflict of interest

The authors declare no conflict of interest.

References

Ahmed, A.T., McCaw, B.R., 2010. Mental health services utilization among women experiencing intimate partner violence. Am. J. Manag. Care 16 (10), 731–738 (doi:12724 [pii]).

Ashare, R.L., Weinberger, A.H., McKee, S.A., Sullivan, T.P., 2011. The role of smoking expectancies in the relationship between PTSD symptoms and smoking behavior among women exposed to intimate partner violence. Addict. Behav. 36 (12), 1333–1336. https://doi.org/10.1016/j.addbeh.2011.07.022.

Baron, R.M., Kenny, D.A., 1986. The moderator–mediator variable distinction in social psychological research: conceptual, strategic, and statistical considerations. J. Pers. Soc. Psychol. 51 (6), 1173. https://doi.org/10.1037/0022-3514.51.6.1173.

Basu, A., Levendosky, A.A., Lonstein, J.S., 2013. Trauma sequelae and cortisol levels in women exposed to intimate partner violence. Psychodyn. Psychiatry 41 (2), 247–275. https://doi.org/10.1521/pdps.2013.41.2.247.

Bosch, J., Weaver, T.L., Arnold, L.D., Clark, E.M., 2015. The impact of intimate partner violence on women's physical health: findings from the Missouri behavioral risk factor surveillance system. J. Interpers. Violence. https://doi.org/10.1177/0886260515599162.

Breiding, M.J., Smith, S.G., Basile, K.C., Walters, M.L., Chen, J., Merrick, M.T., 2014. Prevalence and characteristics of sexual violence, stalking, and intimate partner violence victimization—National intimate partner and sexual violence survey, United States, 2011 [pdf]. Retrieved from. http://www.cdc.gov/ViolencePrevention/pdf/NISVS_Executive_Summary-a.pdf.

Calvete, E., Corral, S., Estévez, su A., 2007. Cognitive and coping mechanisms in the interplay between intimate partner violence and depression. Anxiety Stress Coping 20 (4), 369–382. https://doi.org/10.1080/10615800701628850.

Cerulli, C., Edwardsen, E.A., Duda, J., Conner, K.R., Caine, E., 2010. Protection order petitioners' health care utilization. Violence Against Women 16 (6), 679–690. https://doi.org/10.1177/1077801210370028.

Chantala, K., Tabor, J., 2010. National Longitudinal Study of Adolescent Health: Strategies to perform a design-based analysis using the add-health data. Retrieved from. http://www.cpc.unc.edu/projects/addhealth/data/guides/weight1.pdf.

Cheng, T.C., Lo, C.C., 2014. Domestic violence and treatment seeking: a longitudinal study of low-income women and mental health/substance abuse care. Int. J. Health Serv. Plan. Adm. Eval. 44 (4), 735–759. https://doi.org/10.2190/HS.44.4.d.

Cho, H., Kim, W.J., 2012. Intimate partner violence among Asian Americans and their use of mental health services: comparisons with white, black, and Latino victims. J. Immigr. Minor. Health 14 (5), 809–815. https://doi.org/10.1007/s10903-012-9625-3.

Chuang, C.H., Cattoi, A.L., McCall-Hosenfeld, J.S., Camacho, F., Dyer, A.M., Weisman, C.S., 2012. Longitudinal association of intimate partner violence and depressive symptoms. Ment Health Fam Med 9 (2), 107–114.

Clark, C.J., Alonso, A., Spenceras, R.A., Pencina, M., Williams, K., Everson-Rose, S.A., 2014. Predicted long-term cardiovascular risk among young adults in the national longitudinal study of adolescent health. Am. J. Public Health 104 (12), e108–e115. https://doi.org/10.2105/AJPH.2014.302148.

Cohen, S., Kamarck, T., Mermelstein, R., 1983. A global measure of perceived stress. J. Health Soc. Behav. 24 (4), 385–396. https://doi.org/10.2307/2136404.

Connelly, C.D., Hazen, A.L., Baker-Ericzen, M.J., Landsverk, J., Horwitz, S.M., 2013. Is screening for depression in the perinatal period enough? The co-occurrence of depression, substance abuse, and intimate partner violence in culturally diverse

pregnant women. J. Women's Health 22 (10), 844–852. https://doi.org/10.1089/jwh.2012.4121.
Coughlin, S.S., 2011. Post-traumatic stress disorder and cardiovascular disease. Open Cardiovasc. Med. J. 5 (1), 164–170. https://doi.org/10.2174/1874192401105010164.
Craparo, G., Gori, A., Petruccelli, I., Cannella, V., Simonelli, C., 2014. Intimate partner violence: relationships between alexithymia, depression, attachment styles, and coping strategies of battered women. J. Sex. Med. 11 (6), 1484–1494. https://doi.org/10.1111/jsm.12505.
Cui, M., Ueno, K., Gordon, M., Fincham, F.D., 2013. The continuation of intimate partner violence from adolescence to young adulthood. J. Marriage Fam. 75 (2), 300–313. https://doi.org/10.1111/jomf.12016.
Devries, K.M., Mak, J.Y., Bacchus, L.J., Child, J.C., Falder, G., Petzold, M., ... Watts, C.H., 2013. Intimate partner violence and incident depressive symptoms and suicide attempts: a systematic review of longitudinal studies. PLoS Med. 10 (5), e1001439. https://doi.org/10.1371/journal.pmed.1001439.
Dowd, J.B., Palermo, T., Chyu, L., Adam, E., McDade, T.W., 2014. Race/ethnic and socioeconomic differences in stress and immune function in The National Longitudinal Study of Adolescent Health. Soc. Sci. Med. 115, 49–55. https://doi.org/10.1016/j.socscimed.2014.06.011.
Edmondson, D., Cohen, B.E., 2013. Posttraumatic stress disorder and cardiovascular disease. Prog. Cardiovasc. Dis. 55 (6), 548–556. https://doi.org/10.1016/j.pcad.2013.03.004.
Fassett-Carman, A., Hankin, B.L., Snyder, H.R., 2018. Appraisals of dependent stressor controllability and severity are associated with depression and anxiety symptoms in youth. Anxiety Stress Coping 1–18. https://doi.org/10.1080/10615806.2018.1532504.
Flicker, S.M., Cerulli, C., Zhao, X., Tang, W., Watts, A., Xia, Y., Talbot, N.L., 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 (8), 1067–1085. https://doi.org/10.1177/1077801211414846.
Fox, G.L., Benson, M.L., 2006. Household and neighborhood contexts of intimate partner violence. Publ. Health Rep. 121 (4), 419–427 (Washington, D.C.: 1974).
Glied, S., Kofman, S., 1995. Women and Mental Health: Issues for health reform background paper. The Commonwealth Fund, Commission on Women's Health, New York.
Gustad, L.T., Bjerkeset, O., Strand, L.B., Janszky, I., Salvesen, O., Dalen, H., 2016. Cardiac function associated with previous, current and repeated depression and anxiety symptoms in a healthy population: the HUNT study. Open Heart 3 (1) (e000363-2015-000363. eCollection 2016. doi:10.1136/openhrt-2015-000363 [doi]).
Harris, K.M., Halpern, C.T., Whitsel, E., Hussey, J., Tabor, P., Entzel, P., Udry, J.R., 2009. The National Longitudinal Study of Adolescent to Adult Health: Research design [WWW document]. URL. http://www.cpc.unc.edu/projects/addhealth/design.
Hill, L.K., Hoggard, L.S., 2018. Active coping moderates associations among race-related stress, rumination, and depressive symptoms in emerging adult African American women. Dev. Psychopathol. 30 (5), 1817–1835. https://doi.org/10.1017/S0954579418001268.
Hingson, R.W., Heeren, T., Winter, M.R., 2006. Age at drinking onset and alcohol dependence: age at onset, duration, and severity. Arch. Pediatr. Adolesc. Med. 160 (7), 739–746 (doi:160/7/739).
Humphreys, J., Epel, E.S., Cooper, B.A., Lin, J., Blackburn, E.H., Lee, K.A., 2012. Telomere shortening in formerly abused and never abused women. Biol. Res. Nurs. 14 (2), 115–123. https://doi.org/10.1177/1099800411398479.
Jangpangi, D., Mondal, S., Bandhu, R., Kataria, D., Gandhi, A., 2016. Alteration of heart rate variability in patients of depression. J. Clin. Diagn. Res. 10 (12), CM04–CM06. https://doi.org/10.7860/JCDR/2016/22882.9063.
Kendall-Tackett, K.A., 2007. Inflammation, cardiovascular disease, and metabolic syndrome as sequelae of violence against women: the role of depression, hostility, and sleep disturbance. Trauma Violence Abuse 8 (2), 117–126. https://doi.org/10.1177/1524838007301161.
Kothari, C.L., Cerulli, C., Marcus, S., Rhodes, K.V., 2009. Perinatal status and help-seeking for intimate partner violence. J Womens Health (2002) 18 (10), 1639–1646. https://doi.org/10.1089/jwh.2008.1310.
Kuklina, E.V., Yoon, P.W., Keenan, N.L., 2010. Prevalence of coronary heart disease risk factors and screening for high cholesterol levels among young adults, United States, 1999–2006. Ann. Fam. Med. 8 (4), 327–333. https://doi.org/10.1370/afm.1137.
Lazarus, R.S., Folkman, D., 1984. Stress, appraisal, and coping. Springer, New York.
Lehrer, J.A., Buka, S., Gortmaker, S., Shrier, L.A., 2006. Depressive symptomatology as a predictor of exposure to intimate partner violence among US female adolescents and young adults. Arch. Pediatr. Adolesc. Med. 160 (3), 270–276. https://doi.org/10.1001/archpedi.160.3.
Lett, H.S., Blumenthal, J.A., Babyak, M.A., Sherwood, A., Strauman, T., Robins, C., Newman, M.F., 2004. Depression as a risk factor for coronary artery disease: evidence, mechanisms, and treatment. Psychosom. Med. 66 (3), 305–315. https://doi.org/10.1038/nrcardio.2016.181.
Lichtman, J.H., Bigger Jr., J.T., Blumenthal, J.A., Frasure-Smith, N., Kaufmann, P.G., Lesperance, F., ... American Psychiatric Association, 2008. Depression and coronary heart disease: recommendations for screening, referral, and treatment: a science advisory from the american heart association prevention committee of the council on cardiovascular nursing, council on clinical cardiology, council on epidemiology and prevention, and interdisciplinary council on quality of care and outcomes research: endorsed by the american psychiatric association. Circulation 118 (17), 1768–1775. https://doi.org/10.1161/CIRCULATIONAHA.108.190769.
Martinez-Torteya, C., Bogat, G.A., von Eye, A., Levendosky, A.A., Davidson 2nd, W.S., 2009. Women's appraisals of intimate partner violence stressfulness and their relationship to depressive and posttraumatic stress disorder symptoms. Violence Vict. 24 (6), 707–722. https://doi.org/10.1891/0886-6708.24.6.707.
Matheson, F.I., Daoud, N., Hamilton-Wright, S., Borenstein, H., Pedersen, C., O'Campo, P., 2015. Where did she go? The transformation of self-esteem, self-identity, and mental well-being among women who have experienced intimate partner violence. Womens Health Issues 25 (5), 561–569. https://doi.org/10.1016/j.whi.2015.04.006.
McEwen, B., Seeman, T., 2009. Allostatic load and allostatsis. In: MacArthur Research Network on Socioeconomic Status and Health: Allostatic Load Notebook, . http://www.macses.ucsf.edu/research/allostatic/allostatic.php.
Mekary, R.A., Rimm, E.B., Giovannucci, E., Stampfer, M.J., Willett, W.C., Ludwig, D.S., Hu, F.B., 2011. Joint association of glycemic load and alcohol intake with type 2 diabetes incidence in women. Am. J. Clin. Nutr. 94 (6), 1525–1532. https://doi.org/10.3945/ajcn.111.023754.
Mosca, L., Barrett-Connor, E., Wenger, N.K., 2011. Gender differences in cardiovascular disease prevention. Circulation 124, 2145–2154. https://doi.org/10.1161/CIRCULATIONAHA.110.968792.
Mozaffarian, D., et al., 2016. On behalf of the American Heart Association statistics committee and stroke statistics subcommittee. Heart disease and stroke statistics—2016 update: a report from the American Heart Association. Circulation 133, e38–e360. https://doi.org/10.1161/CIR.0000000000000350.
Muthén, L.K., Muthén, B.O., 2012. Mplus Version 7 user's guide. Muthén & Muthén, Los Angeles, CA.
Newton, T.L., Fernandez-Botran, R., Miller, J.J., Lorenz, D.J., Burns, V.E., Fleming, K.N., 2011. Markers of inflammation in midlife women with intimate partner violence histories. J. Women's Health (2002) 20 (12), 1871–1880. https://doi.org/10.1089/jwh.2011.2788.
O'Neil, A., Fisher, A.J., Kibbey, K.J., Jacka, F.N., Kotowicz, M.A., Williams, L.J., Stuart, A.L., Berk, M., Lewandowski, P.A., Taylor, C.B., Pasco, J.A., 2016. Depression is a risk factor for incident coronary heart disease in women: An 18-year longitudinal study. J. Affect. Disord. 196, 117–124. https://doi.org/10.1016/j.jad.2016.02.029.
Pencina, M.J., D'Agostino, R.B., Larson, M.G., Massaro, J.M., Vasan, R.S., 2009. Predicting the 30-year risk of cardiovascular disease the Framingham heart study. Circulation 119 (24), 3078–3084. https://doi.org/10.1161/CIRCULATIONAHA.108.816694.
Pico-Alfonso, M.A., Garcia-Linares, M.I., Celda-Navarro, N., Herbert, J., Martinez, M., 2004. Changes in cortisol and dehydroepiandrosterone in women victims of physical and psychological intimate partner violence. Biol. Psychiatry 56 (4), 233–240. https://doi.org/10.1016/j.biopsych.2004.06.001.
Preacher, K.J., Hayes, A.F., 2008. Asymptotic and resampling strategies for assessing and comparing indirect effects in multiple mediator models. Behav. Res. Methods 40, 879–891. https://doi.org/10.3758/BRM.40.3.879.
Radloff, L.S., 1977. The CES-D scale a self-report depression scale for research in the general population. Appl. Psychol. Meas. 1 (3) (385-40).
Rivara, F.P., Anderson, M.L., Fishman, P., Bonomi, A.E., Reid, R.J., Carrell, D., Thompson, R.S., 2007. Healthcare utilization and costs for women with a history of intimate partner violence. Am. J. Prev. Med. 32 (2), 89–96 (doi:S0749-3797(06)00423-5).
Ruiz-Pérez, I., Plazaola-Castaño, J., Vives-Cases, C., 2007. Methodological issues in the study of violence against women. J. Epidemiol. Community Health 61 (Suppl. 2), ii26–ii31. https://doi.org/10.1136/jech.2007.059907.
Sabri, B., Bolyard, R., McFadgion, A.L., Stockman, J.K., Lucea, M.B., Callwood, G.B., ... Campbell, J.C., 2013. Intimate partner violence, depression, PTSD, and use of mental health resources among ethnically diverse black women. Soc. Work Health Care 52 (4), 351–369. https://doi.org/10.1080/00981389.2012.745461.
Scott-Storey, K.A., 2013. Abuse as a gendered risk factor for cardiovascular disease: a conceptual model. J. Cardiovasc. Nurs. 28 (6), E1–E8. https://doi.org/10.1097/JCN.0b013e318279e372.
Straus, M.A., Hamby, S.L., Boney-McCoy, S., Sugarman, D.B., 1996. The revised conflict tactics scales (CTS2) development and preliminary psychometric data. J. Fam. Issues 17 (3), 283–316. https://doi.org/10.1177/019251396017003001.
Sullivan, T.P., Flanagan, J.C., Dudley, D.N., Holt, L.J., Mazure, C.M., McKee, S.A., 2015. Correlates of smoking status among women experiencing intimate partner violence: substance use, posttraumatic stress, and coping. Am. J. Addict. 24 (6), 546–553. https://doi.org/10.1111/ajad.12261.
Ullman, S.E., Sigurvinsdottir, R., 2015. Intimate partner violence and drinking among victims of adult sexual assault. J. Aggress. Maltreat. Trauma 24 (2), 117–130. https://doi.org/10.1080/10926771.2015.996312.
Whooley, M.A., Wong, J.M., 2013. Depression and cardiovascular disorders. Annu. Rev. Clin. Psychol. 9, 327–354. https://doi.org/10.1146/annurev-clinpsy-050212-185526.
Witteman, J.C., Willett, W.C., Stampfer, M.J., et al., 1990. Relation of moderate alcohol consumption and risk of systemic hypertension in women. Am. J. Cardiol. 65 (9), 633–663. https://doi.org/10.1016/0002-9149(90)91043-6.
Wright, E.N., Hanlon, A., Lozano, A., Teitelman, A.M., 2018. The association between intimate partner violence and 30-year cardiovascular disease risk among young adult women. J. Interpers. Violence. https://doi.org/10.1177/0886260518816324. (in press).