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Intimate Partner Violence And 30-Year Cardiovascular Disease Risk Among Young Adult Women In The United States

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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 growing body of literature examining the link between IPV victimization and poor heart health. However, there is little known on how IPV impacts cardiovascular disease (CVD) risk among young adult women and what outcomes associated with IPV victimization may be increasing this risk. Using a physiologic framework and a stress and coping framework, a secondary analysis of the National Longitudinal Study of Adolescent to Adult Health (Add Health) was conducted to examine the association between past year IPV exposure and 30-year CVD risk score among a representative sample of young adult women in the United States. Regression analyses were run to examine the relationship between IPV and 30-year CVD risk score. Multiple mediation analyses were run to examine possible mediating factors in the relationship between IPV and CVD risk including perceived stress, depressive symptoms, alcohol dependence, and high sensitivity C-reactive protein levels. The results of the bivariate analyses suggested that past year IPV exposure may have a small impact on 30-year risk score, however this finding becomes insignificant when important covariates are introduced into the model highlighting the complexity of IPV and its co-occurring phenomenon. The mediation analyses revealed that perceived stress and depressive symptoms were partial independent mediators of the relationship between IPV and 30-year CVD risk score. 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.

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Dedication

To my family, it takes a village.

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lives. And lastly, to the survivors of IPV and those who continue to experience violence, your strength and courage is humbling and inspiring. You are the true heroes.

ABSTRACT

INTIMATE PARTNER VIOLENCE AND 30-YEAR CARDIOVASCULAR DISEASE RISK AMONG YOUNG ADULT WOMEN IN THE UNITED STATES

Elizabeth Novack

Anne Teitelman

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 growing body of literature examining the link between IPV victimization and poor heart health. However, there is little known on how IPV impacts cardiovascular disease (CVD) risk among young adult women and what outcomes associated with IPV victimization may be increasing this risk. Using a physiologic framework and a stress and coping framework, a secondary analysis of the National Longitudinal Study of Adolescent to Adult Health (Add Health) was conducted to examine the association between past year IPV exposure and 30-year CVD risk score among a representative sample of young adult women in the United States. Regression analyses were run to examine the relationship between IPV and 30-year CVD risk score. Multiple mediation analyses were run to examine possible mediating factors in the relationship between IPV and CVD risk including perceived stress, depressive symptoms, alcohol dependence, and high sensitivity C-reactive protein levels. The results of the bivariate analyses suggested that past year IPV exposure may have a small impact on 30-year risk score, however this finding becomes insignificant when important covariates are introduced into the model highlighting the complexity of IPV and its co-

occurring phenomenon. The mediation analyses revealed that perceived stress and depressive symptoms were partial independent mediators of the relationship between IPV and 30-year CVD risk score. 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.

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CHAPTER 1

BACKGROUND AND SIGNIFICANCE

Introduction

There is a small, growing body of literature that is examining the link between women who experience intimate partner violence (IPV) and cardiovascular disease (CVD) (Scott-Storey, 2013). IPV, the physical, sexual, psychological abuse or control by a former or current intimate partner, has been associated with many negative health outcomes including mental health issues, stress, and chronic disease, however, the physiologic link between IPV and cardiovascular disease is not well understood (Breiding, Black, & Ryan, 2008; Breiding et al., 2014; Scott-Storey, 2013). Scott-Storey (2013) has defined abuse as a gendered risk factor for cardiovascular disease; almost one in three women will experience IPV in their lifetime, and one in ten women experience rape by an intimate partner compared to one in forty-five men experiencing rape by an intimate partner (Breiding et al., 2014). Similarly, 24.8% of women have reported severe physical violence by an intimate partner compared to 13.8% of men (Breiding et al., 2014). Young females are most at risk for IPV as 71% of women who experience IPV will fall victim to abuse before the age of 25 (Breiding et al., 2014). Some types of IPV victimization can be conceptualized as a chronic stressor impacting CVD development through direct pathways such as physical changes from chronic stress or indirect pathways such as negative coping behaviors like smoking or high alcohol use that are associated with IPV victimization and also increase risk for CVD (Basu, Levendosky, & Lonstein, 2013; Kendall-Tackett, 2007; Scott-Storey, 2013). Since CVD is a major cause of morbidity and mortality in the United States, it is important to understand how this

disease is associated with IPV victimization, with those experiencing partner violence already at risk for negative health outcomes (Campbell et al., 2002). It also is crucial to intervene and manage one's health to prevent the development of CVD *risk*, especially among young adults as CVD risk is becoming more prevalent earlier in life. While there has been a plethora of literature focusing on diet, lifestyle, and other factors increasing the risk of CVD, there is a scant amount of literature that incorporates trauma, specifically IPV, in the list of CVD risk factors. Thus, this study seeks to fill a gap in the literature by examining the connections between IPV and CVD risk by further assessing the possible direct or indirect pathways between IPV and CVD among young adult women. We will examine these gaps using data from the National Longitudinal Study of Adolescent to Adult Health (Add Health). Add Health is a longitudinal, comprehensive, nationally representative sample of adolescents to adults in the United States (Harris et al., 2009). Add Health has collected data on adolescents in grades 7-12 beginning in 1995 (Wave I). The newest set of responses from this data (Wave IV) consists of adults ages 24-32 (Harris et al., 2009). This data has information on relevant topics including demographics, social and intimate relationships, health behaviors, and biomarker levels. Chapter One will introduce the conceptual frameworks guiding this study as well as literature on cardiovascular disease and intimate partner violence and their shared outcomes. This chapter will also review risk calculations for cardiovascular disease. Chapter Two will consist of a literature review on the current state of the science, discuss the measurement issues of IPV, and will introduce the aims of the study. Chapter Three will outline the methods and statistical plan for this study. Chapter Four will summarize

the results of this study while Chapter Five will discuss the main findings and provide implications for research, nursing practice, and policy.

Physiologic Framework

A physiologic framework was one of the frameworks used to guide this study. There is an area of literature that examines the body's response to chronic stress and the health outcomes of this response, known as the allostatic load response (McEwan & Seeman, 2009). Stress is linked to changes in the body. Exposure to external stressors can cause the body to have a physiologic response to adapt to the stressor. The common explanation of the body's response to stress is the "fight or flight" scenario, where a person is confronted with a stressor and the body releases adrenalin and adrenocortical hormones to help respond. The term "allostasis" refers to the body's process of returning to homeostasis, or stability, during this fight or flight response. When the body is constantly exposed to stress causing a physiologic response and allostasis, the body begins to develop an "allostatic load". Allostatic load refers to the body's "wear and tear" of repeating cycles of stress and the constant turning on and off of the neurotransmitter stress responses (McEwan & Seeman, 2009).

The stress response can be affected by an individual's perception of the stressor (similar to one's perception of the violence they experienced), genetic predisposition of certain physiologic responses, and past experiences such as child abuse or neglect that may cause the body to over react to an external stressor (McEwan & Seeman, 2009). As the individual is exposed to stress for longer periods, the allostatic mechanisms of the body become inefficient and the regulation of the neuroendocrine responses are affected as they are constantly trying to achieve homeostasis. Aside from experiences and genetic

background, learned behaviors such as smoking, high alcohol use, high fat diet, and inadequate exercise can also affect the physiologic reactions to stress; these behaviors can increase total cholesterol, narrow blood vessels, and cause decreased blood flow (McEwan & Seeman, 2009).

The mediators of allostasis consists of the adrenal steroids, catecholamines, and other hormones related to the immune system response such as cytokines. When either the adrenal steroids or catecholamines are released, short-term and long-term effects are seen throughout the target cell processes in the body such as increased heartrate and chronic inflammation. Each system in the body sees effects from both short-term allostasis and long-term allostatic load (McEwan & Seeman, 2009).

The development of CVD is partly due through reactions in the oxidative and inflammation processes in the body (McEwan & Seeman, 2009). During the allostasis process in the cardiovascular system, catecholamines are released to increase heart rate and blood pressure, but repeated releases of these hormones and the inability to inhibit them due to chronic stress exposure can increase the development of atherosclerosis (McEwan & Seeman, 2009). Adrenal steroids, which regulate food-seeking behavior and control energy input and expenditure, in the face of allostatic load can lead to insulin resistance and type II diabetes, abdominal obesity, atherosclerosis, and hypertension. A constant stress can cause the adrenal steroids, such as glucocorticoids, to be released leading to the elevation of the deposition of body fat and further insulin resistance.

Allostatic load has been divided into four groups. They consist of: experiencing repeatedly new stressors, the inability to adapt to stress, prolonged response to stress due

to delayed shut down of the response, and, lastly, inadequate responses that lead to hyperactivity of other hormonal mediators (McEwan & Seeman, 2009). The constant experience of new stressors can consist of multiple stressors over time. For example, experiencing an unstable and unsafe childhood may lead to difficulty in school leading to difficulty in employment, and further economic hardship. The inability for one to adapt to the same stressor is due to the body's inability to decrease the hormonal response to a repeated event, experienced in anxiety before exams. Prolonged reaction to stress can be related to the inability to mediate the stress response, possibly due to other predisposed abnormalities within the body. A common example would be an individual with a family history of hypertension who has difficulty lowering blood pressure when experiencing stressors (McEwan & Seeman, 2009). Inadequate stress responses occur when the hormonal response is not sufficient to meet the needs of the individual causing excessive activity in the body leading to elevated catecholamines and cortisol levels (McEwan & Seeman, 2009).

While there is little research addressing the pathway between IPV victimization and CVD risk, there is substantial evidence in the child abuse and maltreatment literature that abuse and chronic stress can be considered a causal pathway to chronic conditions like heart disease (Dong et al., 2004; Felitti et al., 1998). The Adverse Childhood Experiences (ACE) study followed over 17,000 adults for ten years to examine the link between child maltreatment and adult health outcomes (Felitti et al., 1998). The ACEs study found multiple, graded relationships between number of adverse childhood experiences (such as homelessness, abuse, divorce) and poorer morbidity and mortality as

adults that could not be explained by behaviors, such as high fat diet and poor exercise, alone (Felitti et al., 1998). Specifically, a graded, causal pathway was found between number of ACEs and ischemic heart disease (IHD) later in life (Dong et al., 2004). Thurston et al. (2014) found that women with a history of childhood sexual abuse had higher carotid intima media thickness (IMT), a measure of subclinical CVD, compared to those without childhood abuse. This link between past abuse and CVD development may be due to psychological factors, such as mental health sequelae, the physiological response of the altered hypothalamic pituitary adrenal axis function, altered peripheral adrenergic function, and inflammatory dysfunction (Girdler et al., 2003; Heim et al., 2000; Thurston et al., 2014). Research has found these poor outcomes are a result of a disruption in the stress response system in the body previously discussed which can affect brain development and immune system development as well as dysregulate other important functions of the body (Gunna & Quevedo, 2007; Tyrka et al., 2012). A similar study examined adults (32 years of age) and the impact of childhood maltreatment and social isolation on their current health status (Danese et al., 2009). This study found that children who were maltreated and socially isolated were at greater risk of elevated inflammation levels at 32 years (Danese et al., 2009). Interventions that foster support and nurturing among children who are currently experiencing or have experienced maltreatment have been effective in reversing some of the inappropriate regulatory responses in the body. This highlights the importance of intervention development in preventing chronic conditions later in life (Bick et al., 2015). The physiologic model and

child maltreatment literature provide a structure for examining the pathway between IPV victimization and CVD risk and development.

Stress and Coping Framework

As previously mentioned, IPV victimization can be a chronic stressor in one's life. Aside from the physiologic response to stress, one's social reaction to stress and their coping mechanisms can also drive health outcomes. Lazarus and Folkman's (1984) theory, the Transactional Model of Stress and Coping, can help explain the process of coping among women experiencing IPV. Some of the negative outcomes associated with IPV have also been associated with poor coping skills and resources such as depression and PTSD (Evans, Dowling, & Shapiro, 2011). Those experiencing IPV over a long period of time may often use maladaptive coping; these include self-blame, self-medication, accepting the violence, and isolation (Meyer, Wagner, & Dutton, 2010). Thus, the phenomenon of coping, the effort to manage stressful demands, is an important factor in understanding the outcomes IPV.

The Transactional Model of Stress and Coping emphasizes the process of evaluating stress, coping efforts, and outcomes. The idea of coping as a "process" rather than a deliberate choice is an important distinction for survivors of IPV. This theory does not follow the path that people necessarily "choose" maladaptive coping mechanisms. Similarly, those in abusive relationships may not feel that they have many choices in their life. The major concepts in this model are stress, cognitive appraisal, coping, and adaptive outcomes (Lazarus & Folkman, 1984).

Appraisal

Cognitive appraisal, including primary and secondary appraisal, is the evaluative process that examines why and how much a certain event or person is perceived as stressful (Lazarus & Folkman, 1984). Primary and secondary often work together at the same time to appraise the situation (Lazarus & Folkman, 1984). An important aspect of cognitive appraisal is that it is evaluative. Primary appraisal determines what exactly the stimuli is: benign-positive, stress-threat, harm/loss, or challenge (Lazarus & Folkman, 1984). A begin-positive stimulus is evaluated as either a positive situation or neither positive or negative. A threat is a possibility of stress. Harm/loss is a stress that will result in someone losing something such as control or money, or something that will cause harm. Challenge is a stimulus that can cause growth. Secondary appraisal evaluates what is to be done with the stress, what resources are there, and what will the attempt to overcome this stress accomplish. Consequently, reappraisal is a new appraisal with the introduction of new information about the stimuli, whereby, the perception of the stressor is important in determining how to handle that particular event (Lazarus & Folkman, 1984). In an IPV relationship, women who do not perceive an episode of partner violence as stressful may not need to find a way to manage that stress, thus reducing the risk of engaging in maladaptive coping mechanisms.

Personal Factors Influencing Appraisal

Due to the definition of appraisal as an evaluative process, evaluation occurs uniquely for each person with the influence of many factors (Lazarus & Folkman, 1984). Influential personal factors may include; commitment and beliefs can influence the appraisal process. Commitments are what is important to someone that may influence

how they approach the situation. Beliefs often deal with the idea of personal control and a feeling of control over a situation. Control can play a major role in abusive relationships, as the perpetrator's goal can be to exert control over their partner. The experience of loss of control over one's life in an abusive relationship may lead a woman to use "giving in" to her partner as a way to cope (Vatnar & Bjorkly, 2014).

Situational Factors Influencing Appraisal

Beyond personal factors, situational factors influence how an individual evaluates a stressor; these factors include novelty, predictability, event uncertainty, and temporality (Lazarus & Folkman, 1984). If a stress is completely novel, then it will not be appraised as a threat or challenge because the individual has no basis to expect a stress to occur. In contrast, predictability will allow an individual to engage in anticipatory coping. Predictability can also allow the use of personal control as a way to anticipatory cope. In IPV relationships often the top priority is to keep oneself safe, therefore, women may decide to pursue certain actions in order to stay safe (Vatnar & Bjorkly, 2014). Being able to predict an abusive outburst by choosing a specific anticipatory coping mechanism is one of such protective tactics. Event uncertainty is defined as trying to determine the likelihood of an event occurring. Event uncertainty has an "immobilizing effect" on anticipatory coping. Not knowing if an event will occur can lead to internal conflict and can create feelings of helplessness or loss of control. Temporal factors are defined as imminence, duration, temporal uncertainty and ambiguity. Imminence is the length of time before an event occurs, which again can lead to anticipatory coping. When there is ambiguity, the person's factors shape the appraisal, making the appraisal a result of the

person not necessarily just the stress (Lazarus & Folkman, 1984). In IPV relationships, one may live in uncertainty of the next violence outburst and be unable to predict a violent event, which can lead to a chronic stress experience.

Coping

Lazarus & Folkman (1984) define coping as the changing process where an individual manages a stimuli appraised as stressful and exceeding one's resources as well as the emotions created from that stimuli. A goal of coping is to manage the situation, not master it. In the Transactional Model of Stress and Coping, coping has two functions: problem-focused and emotion-focused coping. Problem-focused coping manages the environment that has caused the stress and emotion-focused coping regulates an emotional response (Lazarus & Folkman, 1984). In the context of IPV, emotion-focused coping may include avoidance, distancing, casual attributions, employment, and resiliency (Beecham, 2014). Employment, as a way of coping, can create a separation from home and work and help compartmentalize the violence (Beecham, 2014). Resiliency can also be an important characteristic for women to persevere through the violent relationship. For problem-focused coping, the decision to stay or leave in a relationship and resisting violence occurs in IPV relationships (Anderson, Renner, & Bloom, 2014; Kelly, 2009). Survivor-focused coping is a term from the IPV literature which explains how women in poverty cope with violence including constant negotiations and short term planning (Goodman, Dutton, Weinfurt, & Cook, 2003).

Coping resources and constraints

Lazarus & Folkman (1984) identified coping resources as health, energy, existential beliefs, control, commitments, problem solving skills, social skills, social support, and material resources. These resources can be of aid in managing stress in a positive way. Constraints can include cultural values and psychological deficits. Lazarus & Folkman (1984) explained that a constraint, such as cultural values, can hinder someone from making certain decisions regarding the management of stress. Environmental constraints are identified as demands in the environment that compete for the same recourses as well as something that causes high levels of threat to a community or group of individuals (Lazarus & Folkman, 1984).

Appraisal, Coping and Adaptational Outcomes.

Although Lazarus & Folkman (1984) explained that the coping effort is more important than the actual outcome, the outcomes of coping management have been conceptualized. Social functioning is an important outcome of managing stress. Morale and self-esteem are also important outcomes that can lead to further positive results. Those in abusive relationships can be isolated from family and friends and often experience lack of self-esteem, which can impact effective coping (Matheson et al., 2015). Coping effectiveness occurs when problems and emotions are managed. Coping must match with a person's personal commitment and beliefs, when there is a mismatch the effectiveness is reduced.

Coping can act as a mediator for mental health outcomes among IPV victims. Alexithymia, depression, and attachment issues are negatively correlated with a women's ability to cope with IPV (Craparo, Gori, Petruccelli, Cannella & Simoneli, 2014).

Avoidance coping can mediate the relationships between IPV and PTSD, depression, and drug use problems (Flanagan, Jaquier, Overstreet, Swan, & Sullivan, 2014; Krause, Kaltman, Goodman & Dutton, 2008). Avoidance coping can also exacerbate negative women's health issues for victims of IPV (Flanagan et al., 2014). Emotion focused coping, compared to problem-focused coping, is associated with higher PTSD symptoms and is used among those with higher IPV exposure (Lilly & Graham-Berman, 2010). Other maladaptive coping strategies seen to mediate mental health outcomes include distancing, accepting responsibility, and confrontive coping (Mitchell et al., 2006). Disengagement is also associated with maladaptive coping schemes as well as depression and PTSD (Calvete, Corral & Estevez, 2007; Flicker, Cerulli, Swogger & Talbot, 2012). While the specific mechanisms of stress and coping are not the main focus of this study, the transactional model of stress and coping provides a possible pathway in examining the impact of intimate partner violence on CVD risk.

Cardiovascular Disease

Cardiovascular disease (CVD) is a major cause of morbidity and mortality in the United States, as the death rate for CVD in 2013 for men was 269.8 per 100,000 and 184.8 per 100,000 for women (Mosca, Barrett-Connor, & Wenger, 2011; Mozaffarian, et al., 2016). However, the absolute number of women living and dying CVD and stroke exceed the number among men (Mosca, et al., 2011; Mozaffarian, et al., 2016). CVD is referred to as the narrowing or blockage of blood vessels leading to myocardial infarction, angina, stroke, and peripheral vascular disease (Scott, 2004). There are numerous risk factors for CVD including: high cholesterol (hypercholesterolemia),

hypertension, and smoking (Scott, 2004). These risk factors have been well characterized in the literature and specifically by the Framingham Heart Study, a longitudinal cohort study examining risk factors for CVD (Kannel, Feinleib, McNamara, Garrison, & Castelli, 1979).

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 United States (Mozaffarian et al., 2016). CVD related deaths are occurring among younger ages as almost 35% of deaths attributable to CVD were among Americans younger than 75 years of age and 155,000 Americans under age 65 died of CVD in 2013 while the current average life expectancy is 78.8 years (Mozaffarian et al., 2016). A 2010 study found that 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, Yoon, & Keenan, 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). The financial burden of this disease is quite high with the total direct and indirect cost for CVD and stroke in the U.S. in 2010 was $315.4 billion compared to 2008 the cost of cancer and benign neoplasms was $201.5 billion (Mozaffarian et al., 2016).

Risk Factors

Risk factors for CVD are often behavioral factors or physiological factors with differences seen between women and men. It is important to acknowledge that differences between men and women in biological pathways such as the activation of the

stress response and behavioral risk factors such as inadequate physical activity or high fat diet should not be solely attributed to sex (anatomical differences between reproductive systems and secondary sex characteristics among females and males). Rather gender differences among men and women play a critical role. The social construction of gender and related social gender norms shape perception and presentation of gender in society and differentially affect men and women (Butler, 1988). However, physiologic risk factors such as biomarker levels, measures of biological processes and responses in the body, and adipocyte (fat cells) sizes can differ based on one's sex at birth: male and female. Thus, it is important to consider both sex-based differences in CVD risk and more behavioral-based, gendered-risks, but the broader literature on CVD often does not distinguish between sex and gender, and may use the two interchangeably. Gendered life experiences also have the ability to influence both the behavioral aspects as well as the physiologic response. For example, in the patriarchal society, women have been seen as "less than" their male counterparts, thus increasing their risk for partner violence. This gendered experience of inequality and violence can lead to harm-causing coping strategies such as the excessive use of alcohol, as well as a stress response; both have potential for causing poor health outcomes. The woman's performance of her gender as well as a physiologic response to stress combine to increase risk for poor health outcomes. The same can be true for individuals not performing gender "correctly" compared to traditional gender norms, with those individuals at risk for further violence and stress. It is necessary to understand the importance of gender and the gendered experience when examining behavior and physiological responses.

Furthermore, more than one in three women adults ages greater than 20 years in the U.S. have some form of CVD (Go et al., 2013). While men and women share similar risk factors for CVD, women traditionally present with more advanced stages of the disease due to lack of early recognition and treatment as well as differences in prevalence of certain risk factors (Scott, 2004). While women can present with similar symptoms for cardiovascular related events as men, such as myocardial infarction (MI), women are less likely to experience other typical signs of MI compared to men such as indigestion, shortness of breath, and back pain. Physician bias in perceiving women as "low risk" for CVD despite women reporting a high risk score or multiple CVD risk factors has also impacted CVD diagnosis and treatment among women (Mosca et al., , 2011). Part of this bias may be explained by the fact that women have been historically under-sampled from CVD research (Mosca et al., 2011). This under sampling may also explain why some of the original CVD risk function calculators could underestimate CVD risk in women (Pencina et al., 2009).

Research has shown that the prevalence of high blood pressure is greater among women older than 65 years of age compared to men and the diagnosis of diabetes mellitus is higher among women than men over 20 years of age (Mosca et al., 2011). While smoking rates are still higher among men than women, women report higher rates of physical inactivity compared to men; women also have higher rates of metabolic syndrome, a group of risk factors that increase susceptibility to heart disease and diabetes (Mosca et al., 2011). Some research has revealed that hypertension and diabetes may be stronger predictors of coronary artery disease in females compared to males (Scott,

2004). Metabolic syndrome is also a strong predictor of CVD risk for females (Mosca et al., 2011; Scott, 2004). To be diagnosed with metabolic syndrome, one must have at least three of the following risk factors: a large waistline measurement (35 in. or more for females, 40 in. or more for males), high triglycerides (150 mg/dl), low HDL levels (< 50 mg/dl), hypertension (blood pressure 130/85 mmHg or higher), and a high fasting blood sugar (>100mg/dL) (National Heart, Lung and Blood Institute [NHLBI], 2015). Similarly, a body mass index (BMI), a measurement of obesity, of 25.0 or greater is a risk factor for CVD and is often associated with metabolic syndrome. Research has found possible differences in dysglycemia (poor blood sugar regulation leading to high fasting blood sugar), body fat distribution associated with large waistlines, adipocyte size impacting body mass index (BMI), and hormonal impacts on body weight between sexes (Pradhan, 2014). Differences in blood sugar regulation may be attributed to muscle mass and visceral adiposity. Females experience more fat distribution in the lower body compared to males who tend to have more fat tissue in the waist (visceral adipose tissue [VAT]) (Manson & Bassuk, 2015). Females often have less VAT and a smaller VAT to total body fat ratio which provides less accurate results regarding the impact of BMI and waist circumference on CVD risk in this group (Manson & Bassuk, 2015). Females have smaller sizes of adipocytes (fat cells) in the waist area (including the sides of the abdomen) than their front abdominal subcutaneous adipocytes, where males have adipocytes of equal diameter throughout the body (Manson & Bassuk, 2015). Adipocytes increase in size as BMI increases which can increase lipolysis rates and proinflammatory adipokine secretions impacting glucose and lipid metabolism and causing insulin

resistance (Manson & Bassuk, 2015). Additionally estrogen levels may impact adipocyte biology as well as glucose and lipid metabolism. Low estrogen levels may cause an increase in visceral adiposity (Manson & Bassuk, 2015). This impact on lipid metabolism and visceral adiposity can result in weight gain and increased BMI, putting an individual at risk for poorer health outcomes.

In the clinical setting, typical factors such as smoking, low physical activity, high fat diet, high blood pressure and lipid levels are used in recognizing CVD risk (Greenland et al., 2010). Moderate alcohol consumption has been found to be a protective factor in the development of CVD related outcomes; however, studies have found alcohol intake and CVD outcomes occur within a dose response relationship, with an increased risk for negative outcomes among heavy drinkers (Chomistek et al., 2015; Wittman et al., 1990). Increased or heavy alcohol use, more than 20 g per day, has been shown to 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).

The role of new novel biomarkers, aside from lipid levels, have been incorporated to gain a better physiologic understanding of CVD risk. Common biomarkers used in the clinical setting are: C-reactive protein (CRP), fibrinogen (Factor 1) B-type natriuretic peptides (BNP), D-dimer, lipid levels including total cholesterol and high density lipoprotein (HDL), Apolipoprotein A (ApoA) and Apolipoprotein B (ApoB), see Table 1 for definitions and clinical cut points (Hochholzer, Morrow, & Giugliano, 2010; van Holten et al., 2013). C- reactive protein (CRP), a measure of inflammation and a proxy measure of chronic stress, has clinical cut points that determine CVD risk (Table 1)

(Ridker, 2003). While women who take hormone therapy have higher levels of CRP, studies have found little clinical value in separate CRP clinical cut-points by sex or hormone use (Ridker, 2003).

Table 1

Biomarkers in Cardiovascular Health

Biomarker	Relation to CVD	Clinically significant levels
C-reactive protein	Marker of inflammation	Low risk for CVD: <1.0 mg/L Average risk for CVD: between 1.0 mg/L and 3.0 mg/L High risk for CVD: >3.0 mg/L
Fibrinogen (Factor 1)	Blood clotting and impacts blood flow	Reference values: Males: 200-375 mg/dL Females: 200-430 mg/dL Above normal range values may be indicative of blood clot leading to stroke, CHD and MI
Cholesterol (total cholesterol). high density lipoprotein + low density lipoprotein= total cholesterol	Fat-like substance that causes a hardening and narrowing of the arteries impacting circulation and blood flow	CVD risk increases as cholesterol levels increase. Desirable: Less than 200 mg/dL Borderline High: 200-239 mg/dL High: > 240 mg/dL
high density lipoprotein	Prevents cholesterol	CVD risk increases as

	from building up in the wall of the arteries	HDL levels decrease. Low: < 40 mg/dL Hi:> 60 mg/dL
Apolipoprotein A (ApoA)	The major protein of HDL	Desirable level: > 123 mg/dL
Apolipoprotein B (ApoB)	Major protein found in cholesterol	< 100 mg/dL for those with low or intermediate CVD risk <80 mg/dL for high risk individuals
BNP	Secreted from ventricles in the heart in response to changes in pressure, often indicative of heart failure (HF)	No HF: <100 pg/mL Presence of HF: 100-300 pg/mL Mild HF: > 300 pg/mL Moderate HF: > 600 pg/mL Severe HF: >900 pg/mL
D-Dimer	Indicative of pulmonary embolism or deep vein thrombosis	Positive D-dimer test may indicate blood clot

(Cleveland Clinic, 2013; Hochholzer, Morrow, & Giugliano, 2010; Mahajan, & Jarolim, 2011; Morrison et al.,2002; NHLBI, 2005; Ridker, Libby, & Buring,2015; van Holten et al., 2013)

Aside from the physiologic risk factors and biomarkers associated with CVD and CVD risk, more general, behavioral risk factors are examined. In the clinical setting, biomarkers and more behavioral risk factors are commonly clustered together to create an overall CVD risk score for individuals (Greenland et al., 2010). These risk factors include cigarette smoking, limited physical activity, poor nutrition, increased alcohol consumption, and high stress (Åkesson, Weismayer, Newby, & Wolk, 2007; Heinrich &

Maddock, 2011). These behavioral risk factors have also been associated with IPV victimization, thus creating a possible association between IPV victimization and subsequent CVD and CVD risk.

Intimate Partner Violence

IPV has been considered a major public health problem from its high prevalence in society and devastating outcomes (Breiding et al., 2014). However, it is important to understand that there are different types of partner violence that one may experience that fall under the overarching IPV umbrella. Kelly & Johnson (2008) differentiated between the types of IPV by providing more description of the context of the abuse regardless of the form (physical, sexual, psychological) of violence. Coercive Controlling Violence (formerly known as battering) is the emotional and coercive control of an individual and while physical violence in this type of relationship is often severe, many individuals experiencing coercive control report multiple psychological symptoms due to fear and manipulation (Kelly & Johnson, 2008). Situational Couple Violence is the most common form of physical aggression and does not include fear or control tactics seen in coercive controlling violence. This form of violence is less likely to increase in severity overtime and may occur only once in a relationship (Kelly & Johnson, 2008). Violent Resistant is seen as the "fighting back" violence when a victim of IPV is responding to their abuser. Lastly, Separation Instigated Violence occurs as couples are divorcing or separating with no prior history of violence (Kelly & Johnson, 2008). In measuring and assessing IPV, it is crucial to understand the context in which the violence occurred or the "type" of IPV that was experienced. Similarly, the perception or meaning of the violence by the person

experiencing it can also be a determinant of subsequent outcomes. If the individual experiencing the violence is not fearful or feeling manipulated, then one could expect that individual to have fewer negative outcomes than an individual fearful and worried. Time frame of IPV is also a point of interest, as a single episode of IPV may be traumatic but that individual may not experience the chronic stress response like an individual living with ongoing abuse.

Sexual minority women (SMW), those identifying as lesbian, bisexual, asexual, queer, women having sex with women or women having sex with women and men, have been found to experience equal or higher rates of IPV compared to their heterosexual counterparts as well as more negative health behaviors and outcomes separate from IPV victimization (Breiding et al., 2014; Diamant & Wold, 2003;McCauley et al., 2015; Matthews, Hughes, Johnson, Razzano, & Cassidy, 2002; Steele et al., 2017; Ward, Jestl, Galinsky, & Dahlamer, 2015). Research has found lesbian women to report higher rates of verbal, emotional and psychological abuse—abuse that might not be apparent from an outsider at first glance (Renzetti, 1992). SMW can experience unique stressors such as stigma and homophobia that perpetuate negative health outcomes and create barriers to health care in general (Weisz, 2009). Fears of "outing", disclosure of sexual identity and battering in a homophobic context can cause SMW to have difficulty in recognizing abuse, disclosing abuse, finding appropriate services and having others believe their experiences (Hassouneh & Glass, 2008).

IPV and Its Coping Mechanisms

The association between specific maladaptive coping behaviors such as frequent cigarette smoking and increased and risky alcohol use among women experiencing IPV has been well studied in the literature (Ashare, Weinberger, McKee, Sullivan, 2011; Ullman, & Sigurvinsdottir, 2015). Similarly, these maladaptive coping strategies have also been linked to increased depressive symptoms which could further compound negative health issues and such coping strategies (Bosch, Weaver, Arnold & Clark, 2015; Calvete et al., 2007; Sullivan et al., 2015). Mental health outcomes associated with IPV victimization such as depression, post-traumatic stress disorder (PTSD), and increased overall stress levels have been researched as well (Kendall-Tackett, 2007; Martinez-Toteya et al., 2009; Sabri et al., 2013). Women experiencing IPV have reported high mental health service use, which can increase their health care costs (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, Buka, Gortmaker, & Shrier, 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 & Kofman, 1995). Gustad et al. (2016) has found associations with increased depressive symptoms and left ventricular dysfunction. 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). SMW, specifically, have reported higher rates of anxiety and depression compared to heterosexual women, thus they should be included

when examining the physical health outcomes associated with depression (Caceres, Brody, & Chyun, 2016; Diamont & Wold, 2003; Matthews et al., 2002; Steele et al., 2017).

IPV and the Stress Response

Post-traumatic stress disorder and chronic stress, outcomes associated with IPV, have been connected to CVD risk (Coughlin, 2011; Edmonson & Cohen, 2013). Using the stress response framework, experiencing chronic partner violence may cause the body's regulatory systems to respond inappropriately. Thus, those experiencing long-term partner abuse, multiple abuse experiences, or elevated stress from an isolated abusive behavior may experience this allostatic stress response, impacting their physical health. Similarly, coping mechanisms associated with IPV such as smoking and high alcohol use, can further increase the body's detrimental response to stress as well as directly augment negative physiologic changes and enable the development of poor health outcomes, such as cardiovascular disease. The connection between IPV, its associated outcomes such as maladaptive coping mechanisms and stress, and CVD risk factors highlight an important need to examine CVD and CVD risk among those who experience IPV. There is little known specifically about young adult women, the most at risk group for IPV victimization, and their CVD risk (Breiding et al., 2014). In terms of general heart health, younger women, (ages 55 and younger) compared to older women (ages greater than 55 years) have demonstrated less knowledge on risk factors for heart disease in women and the signs and symptoms of heart attacks (Mochari-Greenberger, Miller, & Mosca, 2012). Younger women (ages 55 and younger) are also less likely to

talk to their doctors about heart disease prevention compared to older women (ages greater than 55 years) (Mochari-Greenberger et al., 2012). Intervening and aiding in preventing behaviors that can increase CVD risk later in life is crucial. Understanding the connection between IPV among young women and their CVD risk will allow for the development of clinical guidelines and interventions that can improve this already victimized populations quality of life.

Figure 1 (below) represents the path model of the hypothesis of the study.

The first hypothesis: *exposure to IPV victimization will be associated with a higher 30-year CVD risk,* is shown in the Figure 1a. The solid line between the two variables represents the total effect of IPV victimization on CVD risk. Below this figure is a sub hypothesis that a *higher severity of IPV victimization will be associated with a higher 30-year CVD risk score among young women.*

The second hypothesis, *perceived stress levels, depressive symptoms, and alcohol dependence mediate the relationship between IPV exposure and higher 30-year CVD score among young women,* is shown in Figure 1b using a multiple mediator model. This multiple mediation model will allow us to examine the direct effect of the IPV exposure on the 30-year CVD risk score, the specific indirect effects of IPV exposure on the 30-year CVD risk score through each specific mediator (*alcohol dependence, perceived stress, and depressive symptoms),* and the total indirect effect of IPV exposure on the 30-year CVD risk score through the sum of the specific effects (Preacher & Hayes, 2008).

The third hypothesis, *increased C-reactive protein levels (a proxy measure of stress) and increased perceived stress levels will mediate the relationship between IPV*

exposure and higher 30-year CVD score among young women, is shown in Figure 1c using a multiple mediator model. This multiple mediation model will allow us to examine the direct effect of IPV exposure on the 30-year CVD risk score, specific indirect effects of IPV exposure on the 30-year CVD risk score through each specific mediator (*C-reactive protein levels (a proxy measure of stress) and increased perceived stress levels*), and the total indirect effect of IPV exposure on the 30-year CVD risk score through the sum of the specific effects (Preacher & Hayes, 2008). Important covariates in this relationship are: health insurance status, history of childhood abuse, race/ethnicity, sexual orientation, education, income, financial stress, health status, and pregnancy status.

Figure 1: Path Model

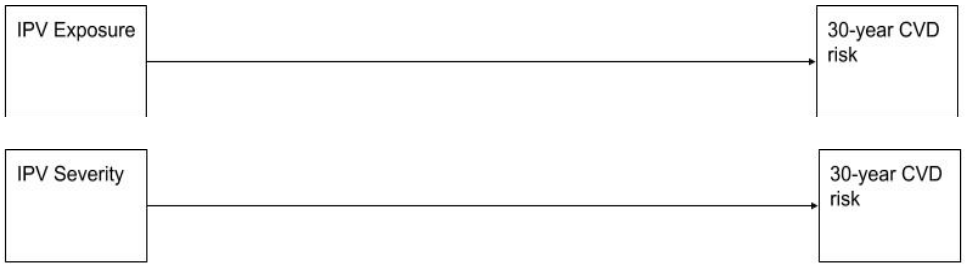


Figure 1a

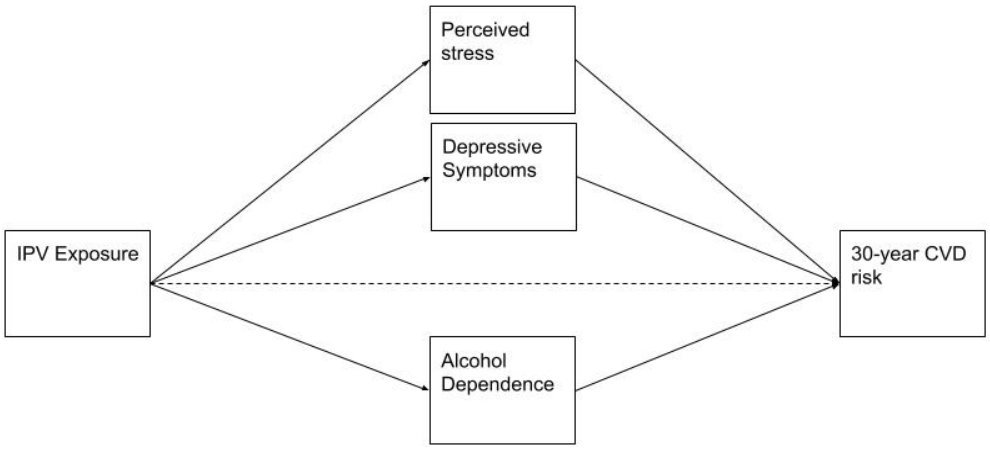


Figure 1b

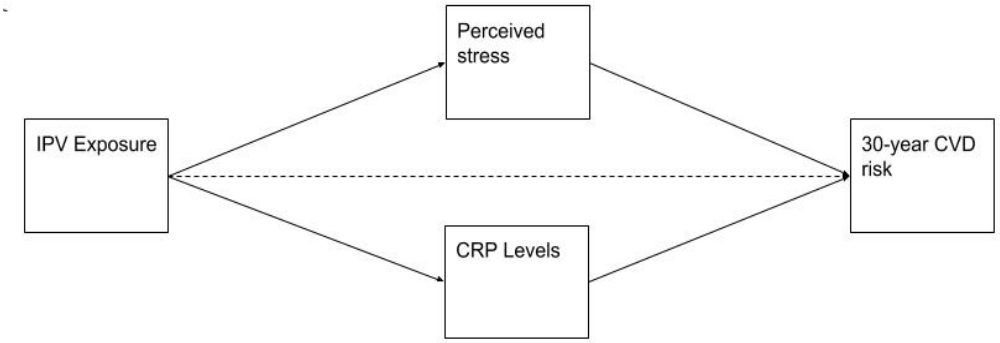


Figure 1c

Risk Calculation

This next section discusses the background literature related to how CVD risk was calculated in this present study. CVD risk can include any factor including characteristics or exposures of an individual that increases their likelihood of developing cardiovascular disease. Preventing, examining, and managing CVD risk factors allows for the reduction of the likelihood of one eventually developing CVD. The American College of Cardiology and the American Heart Association recommend the use of specific risk calculations that can be used in both research and clinical practice as a way to identify an

individual's CVD risk (Greenland et al., 2010). These risk calculations focus on the clustering of individual risk factors that have been associated with later development of cardiovascular disease. One of the most prominent risk scores is the Framingham 10-year risk score, which calculates an individual's 10-year risk of developing cardiovascular disease. This risk score was developed through the Framingham Heart Study, a longitudinal cohort study meant to examine the risk factors for CVD overtime (D'Agostino, Pencina, Massaro, & Coady, 2013; Kannel et al., 1979). The Framingham Heart Study began in 1948 with a cohort of n=5209 and in 1971, n=5124 offspring and spouses were enrolled (Kannel et al., 1979). As statistical methods evolved, the ability to calculate a more accurate risk score was achieved. Currently, the risk score estimates the risk for general coronary heart disease (CHD) which includes angina, myocardial infarction and coronary death, hard CHD (coronary death or non-fatal MI) and more recently a global CVD score which includes CVD death, general CHD, stroke, intermittent claudication and congestive heart failure (D'Agostino et al., 2008, D'Agostino et al., 2013). The standard risk factors included in the score are sex, age, blood pressure, smoking, diabetes, total cholesterol and HDL levels (D'Agostino et al., 2013).

Time-to-event survival models are used to calculate the risk score (D'Agostino et al., 2013). Sex-specific model (males and females) are also incorporated as sex can impact CVD development. The C-statistic, a method of discrimination which refers to the functions ability to discriminate cases from non-cases, for the risk functions for males and females are 0.79 and 0.83, respectively (D'Agostino et al., 2013). Nam-D'Agostino

chi-square tests, used to calibrate the time-event analysis and the chi-square results, are x^2=3.3 and x^2=3.7 for males and women, respectively. Both the C-statistic results and chi-square results reveal excellent calibration (D'Agostino & Nam, 2003; D'Agostino et al., 2013). The Framingham Study itself was conducted among a homogenous group of middle class white individuals, however this model has been validated among African Americans, and with re-calibration adjustments, Asian and Puerto Rico men (D'Agostino Grundy, Sullivan, & Wilson, 2001; Grundy et al., 2001). The developers of the scale recommended the development of a model that predicts longer-term risk among a younger generation as the development of CVD risk behaviors and predictors are becoming apparent at increasingly younger ages (D'Agostino et al., 2013).

In response to this need and the added fact that 10-year risk functions may underestimate risk, specifically in young women, a 30-year risk function model was developed (Pencina et al., 2009). This model was developed from the Framingham Heart Study and its epidemiologic results from the start of the study until 2006. For the 30-year risk model, the primary outcome is hard CVD (coronary death, MI and stroke) and the secondary outcome is full or general CVD (hard CVD plus coronary insufficiency, angina pectoris, TIA, intermittent claudication and CHF) (Pencina et al., 2009). The risk factors included in this model are sex, age, systolic blood pressure, antihypertensive treatment, smoking, diabetes mellitus, total cholesterol, HDL and BMI. Because this model predicts risk over a longer period of time, both Cox-regression and the Anderson et al. (1993) risk model were used in order to adjust for the competing risk of non-CVD mortality (Putter, Fiocco, & Geskus, 2007; Rosthøj, Andersen, & Abildstrom, 2004). The

30-year model showed excellent discrimination with the cross validated C-statistic (0.803), internally validated C-statistic (0.802), cross validated Nam-D'Agostino chi-square (x^2=4.25), and internally validated Nam-D'Agostino chi-square (x^2=3.98). Time-dependent analysis was used to update all variables approximately every four years (Pencina et al., 2009). In the first use of this 30-year model in the Framingham study, the differences in risk compared to the 10-year function model were almost three times higher for women and men using the 30-year risk model, demonstrating its ability to detect long-term CVD risk in women perhaps more accurately than the 10-year function model (Pencina et al., 2009). The standard risk variables, male sex, systolic blood pressure, antihypertensive treatment, total lipids and HDL, smoking, and diabetes mellitus were significant in relation to hard CVD in the time dependent analysis. BMI was weakly significant in the 30-year model and may be mediated through other factors. However, a 30-year model was developed including BMI in replacement of lipids to be more easily used in a clinical setting and still provides a valid risk score (Pencina et al., 2009).

A handful of studies have used the 30-year risk prediction model among late adolescents and young adults (Clark et al., 2014b; Clark et al., 2015; Clark et al., 2016). These studies, focused on individuals ages 24 to 32, found in general that average 30-year risks for hard and general CVD were 10.4% and 17.3% among men and 4.4% and 9.2% among women (Clark et al., 2014b). Risks for hard and general CVD were higher among American Indians and Blacks than among Whites and lower among Asian/Pacific Islander women than White women (Clark et al., 2014b). One study found 4% of women

had a 20% risk of general CVD, which is often deemed “high risk” (Clark et al., 2014b). After adjusting for socioeconomic status, racial differences were not detected. Among the same sample, compared to heterosexual females, mostly heterosexual females (0.8%) and mostly homosexual females (2.8%) had a significantly higher CVD risk (Clark et al., 2015). In one study, a one-standard deviation increase in IPV victimization score was associated with a 0.28% increase in 30-year risk score (Clark et al., 2016). The use of the 30-year risk score in these studies provide evidence that cardiovascular risk can begin to be detected in the young adult population (Clark et al., 2014b; Clark et al., 2015; Clark et al., 2016). It is important to note that the Clark et al. (2016) study did not differentiate between genders in terms of IPV victimization and CVD risk, thus the impact of IPV on CVD risk among women specifically is not known. The high internal and external validity and excellent discrimination of the 30-year risk model gives promise of this model’s ability to provide valid and accurate results and should be incorporated into future research and clinical practice guidelines. This current study will further expand the understanding of the relationship between IPV and CVD by looking at the impact on young women specifically, as well as possible mediators that may increase one’s CVD risk.

CHAPTER 2
LITERATURE REVIEW

Introduction

A review of the literature that focuses on CVD risk and young adult female victims of IPV was conducted to understand the current state of the science. Analysis does not only include the results of the studies, but a focus on the measurements of both IPV victimization and cardiovascular disease risk was included to assess the rigor of the studies. This review will highlight what is known as well as the gaps in the literature regarding IPV and CVD risk among young adult women.

Methodology

This literature review focused on IPV victimization and CVD or CVD risk among young adult women. Inclusion criteria were: published in last 10 years, peer-reviewed, young adult women sample, IPV victimization, occurred in the U.S or Western country. Exclusion criteria were: focus on male sex or statistical analysis did not separate males from females, did not occur in the United States or Western country, and does not examine cardiac disease or cardiac risks. This first review yielded few results thus inclusion criteria was expanded to include adult women. Both PubMed MeSH terms and CINHAL databases were used for this search. Search terms included: cardiovascular disease risk, cardiovascular disease, young women, young adult women, intimate partner violence, partner violence, domestic violence. A total of 27 articles were found. After title and abstract review, 15 articles remained. After full text review and reference list review, the literature revealed 7 articles (Appendix) that assessed IPV victimization and

CVD risks or associated risk behaviors. The findings were mixed as well as a variety of measurements were used to asses both IPV victimization and CVD variables.

Summary and Analysis

IPV and Hypertension

Two studies specifically examined IPV victimization and blood pressure levels, with the assumption that hypertension is a known risk factor for cardiovascular disease (Clark et al., 2014a; Mason et al., 2012). Clark et al. (2014a) used secondary analysis to assess IPV victimization, using the Revised Conflict Tactic Scale, and blood pressure readings. For women who reported IPV victimization, there was no significant association between IPV and hypertension (Clark et al., 2014a). Blood pressure was measured at time of data collection and hypertension was defined as systolic blood pressure (SBP) greater or equal to 140 mmHg, diastolic blood pressure (DBP) greater or equal to 90 mmHg or taking antihypertensive medication. IPV victimization was assessed in relationships occurring during an 8-year time span. The lack of association between IPV and hypertension among women may be due to the younger age of the women (mean age at baseline was 21.72 years) and the low number of women who experienced a high severity of violence in the study (Clark et al., 2014a). The authors found, although not significant, there was a possible relationship between elevated blood pressure and women who experienced severe physical or sexual violence, however a small number of women who experienced such violence were included making it difficult to determine significance (Clark et al., 2014a). The sample was also relatively young to have hypertension, rather than risk of developing hypertension. In contrast, Mason et al. (2012)

found differences between types of abuse experienced and hypertension. Using the Nurses' Health Study, women, with mean ages ranging from 45.6-47 years, reporting extreme emotional abuse had a 24% increased rate of hypertension compared with women who had no emotional abuse (Mason et al., 2012). This study assessed women's relationship violence who were in ongoing relationships at the first wave of data collection in 2001. Physical and sexual IPV were assessed and coded into dichotomous variables of "yes" and "no" in response to *have you experience this type of violence*. Severity or frequency was not taken into account (Mason et al., 2012). Emotional abuse was assessed using the Women's Experience of Battering Scale and results were coded into three groups that increased in severity of emotional abuse based on the scores with each group (Mason et al., 2012). While Clark et al. (2014a) found no associations among women, it is important to note that emotional abuse was not assessed in that study, which may have impacted the results. In the Mason et al. (2012) article, the sample was non-representative as women were mostly white and blood pressure levels were self-report.

IPV and Multiple CVD Risks

The remaining articles assed IPV victimization and varying cardiovascular diseases risks. The findings were mixed and the methods of the studies should also be examined. Veteran women often report higher rates of IPV victimization than civilian women (Dichter, Cerulli & Bossarte, 2011). Dichter et al. (2011) found IPV victimization among veteran women was associated with depression, smoking and heavy drinking while no association with IPV and exercise or weight was found after controlling for demographics and veteran status. In this study, 61% of the veteran women were under the

age of 45 years with only 50% of non-veteran women under 45 years of age. Lifetime IPV was assessed as *ever have experiencing actual or the threat of physical violence or unwanted sex by an intimate partner* (Dichter et al., 2011).

Scott-Storey, Wuest, & Ford-Gilboe (2009) sampled from women who had already left an abusive partner. This study found a positive association between severity of abuse, measured by the Index of Spouse Abuse, and smoking (Scott-Storey et al., 2009). Yet, IPV and smoking behaviors were not able to explain CVD risk symptoms such as BMI, blood pressure readings and self-report of CVD diagnosis or self-report of CVD medication use (Scott-Storey et al., 2009). One study collected biomarkers from women who had experienced both IPV and acute coronary syndrome (Symes et al., 2010). Eleven biomarkers were examined to test a psychological and biological pathway of IPV to chronic illness. A moderate effect size was found for vascular cell adhesion molecule-1(Symes et al., 2010). Stene, Jacobsen, Dyb, Tverdal, & Schei, (2013) were able to use the 10-year Framingham Risk Calculator as well as drug prescription filling information in their study. The 10-year risk calculator includes age, sex, diabetes diagnosis, current smoker, systolic blood pressure, total cholesterol and HDL levels. IPV was assessed using questions developed by the research team that assessed emotional abuse, physical abuse, and sexual abuse. Women, ages 30 -60, who reported IPV had a slightly higher 10-year risk score and were more often smokers compared to women with no IPV history (Stene et al.,2013). Women who reported physical and/or sexual violence were associated with having low HDL, abdominal obesity, and elevated triglycerides (Stene et al., 2013).

Vijayaraghaven et al. (2011) specifically looked at CVD risk and IPV among homeless women. By using health care providers' diagnosis of diabetes, hypertension, or obesity as CVD risk indicators, they found no statistical differences between women who experienced IPV and those who did not in terms of their cardiovascular risk (Vijayaraghaven et al., 2011).

Discussion

The studies used different measures of IPV victimization ranging from validated scales to self-report of physical assault as well as different measures of cardiovascular risk. When analyzing results regarding IPV victimization, measurement is ctitical. Studies that only ask about physical assault may not be able to understand the entire IPV experience as compared to studies that cover multiple types of IPV victimization (Clark et al., 2014a; Clark et al., 2016; Dichter et al., 2011). One of the studies in the review that found significant results found an association between hypertension among only emotional abuse, a type of violence that some validated abuse scales do not thoroughly capture (Mason et al., 2012). In general, emotional abuse has been associated with stress and poor health in general (Mason et al., 2012). Time frame of abuse is also an important facet in understanding the context of abuse. Many of the studies did not differentiate abuse experience by frequency (Dichter et al., 2011; Mason et al., 2012; Stene et al., 2013; Symes et al., 2010; Vijayaraghaven et al., 2011). While even one episode of IPV victimization is a valid traumatic experience, understanding the chronicity of violence is important when linking violence to a chronic stress response. Future studies should

clarify the context of violence by understanding the type of violence, the severity and frequency of violence.

For the cardiovascular measurements, relying solely on self-report may provide lower validity than standardized cardiovascular risk scales that measure the risk factors during data collection. Only one study included lipid levels as part of the risk score, yet did not find overwhelmingly significant results (Stene et al., 2013). The age ranges in the studies were mostly middle age women, but often women present CVD symptoms later in life and women are more likely to have coronary episodes without previous symptoms (Go et al., 2013). Only collecting individually identified CVD risk factors and analyzing them separately may not be an effective way to calculate risk. In terms of CVD risk, one risk alone may not be a direct pathway to CVD development; rather, multiple, clustered risk factors working together to create a synergistic effect may be more indicative of CVD or CVD risk. While one study used the 10-year Framingham risk calculator as a way to cluster risk factors, the women's ages were still relatively young to predict CVD in the next 10 years (Pencina et al., 2009; Stene et al., 2013). This 10-year risk score has also been found to underestimate CVD risk in women and younger individuals. Thus, a 30-year risk score was developed to be used in a younger population and developed to more accurately measure risk in women (Pencina et al., 2009).

Currently, there is only one study that has examined IPV victimization and perpetration on 30-year CVD risk among late adolescents and young adults (Clark et al., 2016). However, this study did not differentiate between genders in the final model examining IPV and CVD risk, thus the paper did not meet criteria for this this review

(Clark et al., 2016). While the study demonstrated the ability to detect an increase in 30-year CVD risk among those who experience IPV, the study does not allow one to look at the impact of IPV on CVD risk among women alone. Based on this review, the need for more research focusing on young adult women who experience IPV and their CVD risk is evident. This review also highlights the need to understand other possible factors associated with IPV that might also increase the risk of developing CVD risk factors such as hypertension, diabetes and increased BMI.

IPV Measurement

The literature review revealed the multiple measurements used to identify intimate partner violence in research. Currently, there is no true consensus among IPV researchers on the "best" way to conceptualize, define, and measure IPV in health care and research (Nicolaidis & Paranjape, 2009). The term intimate partner violence was initially derived from the feminist movement in the 1970's with an emphasis on relationship dynamics and power and control imbalances (Nicolaidis & Paranjape, 2009). In the feminist framework of IPV, this violence often occurs in isolation and there is a focus on the *intent* of the violence and the *consequences* for the victims (Nicolaidis & Paranjape, 2009). Family conflict researchers use the viewpoint that IPV evolves from conflict, not necessarily to exert power and control (Nicolaidis & Paranjape, 2009). In the family violence research setting, IPV often does not include the contextual information about relationship dynamics, the antecedents of violence, and the consequences of the violence for the victim (Nicolaidis & Paranjape, 2009). Legal frameworks conceptualize IPV as it relates to specific penal codes such as simple assault or aggravated assault with

non-criminal behavior often omitted in this framework (Nicolaidis & Paranjape, 2009). Public health frameworks tend to focus on IPV and its subsequent health-related outcomes and again omit contextual information (Nicolaidis & Paranjape, 2009).

Furthermore, research studies have used various inclusion criteria for the term "intimate partner". Definitions can range from married couples only, married or cohabitating partners, or any romantic partner (Nicolaidis & Paranjape, 2009). Standard definitions prove difficult as the context of violence may differ depending on the relationship one has with their "intimate partner" (Nicolaidis & Paranjape, 2009). Nicolaidis & Paranjape (2009) use the example of a woman who is raped on a first date. Would that be considered an intimate partner or an acquaintance rape? Similarly, one who experiences partner violence from a partner with whom they have been with for an extended period of time may share more intimacies and shared experiences than a shorter relationship, yet both can experience traumatizing violence (Nicolaidis & Paranjape, 2009).

Seeking consensus on how to define abusive behaviors can also lead to difficulty in standardizing IPV measurements. While family researchers, for example, may define certain behaviors as partner violence, feminist researchers focus more on power and control tactics, as well as intent and motivation behind the behaviors (Nicolaidis & Paranjape, 2009). Measures of sexual assault vary as sexual coercion is not always included in both research and legal realms (Nicolaidis & Paranjape, 2009). Power and control, an important contextual element in feminist frameworks, is often omitted from family violence research measurement (Nicolaidis & Paranjape, 2009).

Many of the discrepancies among the IPV researchers are a result of different theoretical and conceptual viewpoints that guide the development of measurement tools for research and clinical practice. Therefore, when choosing measurement tools to use in IPV research, the etiology of the IPV scale as well as its limitations must be understood.

The Conflict Tactics Scale

The Conflict Tactics Scale is considered the most widely used instrument to both measure and identify IPV in research with over 200 published papers reporting results based off of this scale (Straus et al., 1996; Straus, 2007; Straus & Douglas, 2004). The Revised Conflict Tactics Scale (CTS2) was developed to measure victimization and perpetration in dating or martial relationships. The types of partner violence measured are physical assault, psychological aggression, negotiation, injury, and sexual coercion (Straus, 2007). Developed from family violence researchers, the CTS2 was derived from the notion that conflict in relationships is inevitable and part of problem solving. However, this conflict becomes harmful when coercion or violence are used to solve the problem (Straus, 2007). The scale focuses on conflict management rather than the topic of the conflict itself (Straus, 2007).

The CTS2 is made up of a list of behaviors (78 questions) that fall under the five previously mentioned categories: physical assault, psychological aggression, negotiation, injury, and sexual coercion. The scale focuses solely on behaviors directed towards a partner rather than including attitudes, emotions and cognitive appraisals associated with those behaviors and the context surrounding them (Straus, 2007). The CTS2 identifies if a certain behavior has occurred and then identifies the frequency of that behavior (never

to more than 20 times in the past year). This can help establish a chronicity of violence in a relationship over a period of a year. Severity of violence is established through subscales under each category, an example of such subscales is; minor physical assault versus major physical assault (Straus, 2007).

Strengths of this scale in research is that testing time of the full CTS2 ranges from 12 to 15 minutes, which may not be available to every participant or patient, therefore a short form of the CTS2 was developed and includes only 20 questions (Straus, 2007; Straus & Douglas, 2004). Similarly, the design of the questions and responses allow for multiple types of scoring. For example, prevalence of specific behaviors such as severe injury or sexual assault can be identified easily from responses that report any occurrence of those behaviors (Straus, 2007). Understanding the frequency of a behavior through the numbered responses can establish chronicity of violence (Straus et al., 1996). This scale also allows one to understand the overlap of multiple types of violence occurring: physical only, physical and sexual violence, or aggression and physical violence, for example. Severity level through the subscales can allow researchers to better understand the level of violence. Similarly, because victimization and perpetration questions are included, respondent-only violence, partner-only violence or mutual violence can be established (Straus, 2007; Straus et al., 1996). The scale also has high content validity and sensitivity (Straus & Douglas, 2004).

One of the biggest critiques about the CTS2 is that is does not account for intent, motivation and consequences related to the violence (Kimmel, 2002). Intention and motivation are crucial in understanding the full context of IPV and how this violence

impacts the overall well-being of the victim (Kimmel, 2002). Historically, the CTS2 has found gender symmetry among rates of violence and perpetration (Kimmel, 2002; Straus, 2007). However, the argument for this symmetry is the omission of contextual information regarding violence, especially consequences of violence. In terms of physical assault, men are more likely to be more violent and injury causing as opposed to women (Kimmel, 2002). The scale also only covers a limited number of violent acts and the examples are mostly conflict related, not control related (Kimmel, 2002). Control-motivated violence is extremely gender asymmetric with men often using violence or manipulation to exert control (Kimmel, 2002). Control-motivated violence can occur as a sense of control is being lost, often used among ex-partners or ex-spouses (Kimmel, 2002). However, the CTS2 does not include ex-partners and only recounts violence in the past year, decreasing the opportunity to examine violence between ex-partners as well as difficulty in examining patterns of violence over a longer period of time (Kimmel, 2002). Recall bias and self-report also hinder the accuracy of the data from this scale. Kimmel (2002) explains that due to gender norms and the normalization of violence, men often underestimate their use of violence against women. Similarly, women may overestimate the violence experiences, or they may underestimate due to a "normalization" of violence. (Kimmel, 2002).

While the CTS2 allows researchers to quantify different aspects of IPV, there are important theoretical constructs missing such as power and control, intent, and motivation. The Women's Experience of Battering Scale (WEB) however can be used to assess the experiences of abuse and more specifically the gendered experience of

battering (Smith, Earp, & DeVellis, 1994). This scale can be used in accordance with scales like the CTS2 to connect experiences and perceptions about abuse with the actual behaviors of abuse encountered (Smith et al., 1994).

The ability to use multiple scoring methods to examine the data support the notion that the CTS2 is the most widely used scale in IPV research. However, results using this scale should be interpreted with caution as important, telling aspects of the IPV experience may be missing.

IPV Measurement in Add Health

The Add Health data set, the data set used for this present study, includes questions surrounding partner violence (Harris et al., 2009). The waves that include partner violence questions (Waves II-IV) use questions derived from the Revised Conflict Tactics Scale (CTS2). However, each wave does not include the full or short-form version of the CTS2; rather, specific behavior questions were chosen leaving interpretation and scoring in the hands of the researcher causing much variance in how this scale is used to identify partner violence in Add Health.

All three waves have different inclusion criteria for whom the partner violence questions are asked, as well as different time frames for when the violence would be occurring (Harris et al., 2009). Wave II (grades 7-12) partner violence questions were asked to participants for up to three romantic and sexual (non-romantic) partners within the past 18 months. Five questions were asked assessing: *if they have been insulted at, sworn at, threatened with violence, pushed or shoved, and had something thrown at them*. Each question had a response of *yes this has happened* or *no this has not happened*. A

positive response then elicited a question of what month and year this behavior occurred in.

Wave III included partner violence information on as many partner relationships an individual desired to list beginning in 1995 up to Wave III (ages 18-26) data collection (Harris et al., 2009). This wave asked four questions focused on victimization*:*

Victimization	How often (has/did) {initials} (threatened/threaten) you with violence, (pushed/push) or (shoved/shove) you, or (thrown/throw) something at you that could hurt?	How often (has/did) {initials} (slapped/slap), hit or (kicked/kick) you?	How often (have/did) you (had/have) an injury, such as a sprain, bruise, or cut because of a fight with {initials}?	How often (has/did) {initials} (insisted/insist) on or (made/make) you have sexual relations with (him/her) when you didn't want to?
Perpetration	How often (have/did) you (threatened/threaten) {initials} with violence, (pushed/push) or (shoved/shove) {initials}, or (thrown/throw) something at {initials} that could hurt?	How often (have/did) you (slapped/slap), hit or (kicked/kick) {initials}?	How often (has/did) {initials} (had/have) an injury, such as a sprain, bruise, or cut because of a fight with you?	How often (have/did) you (insisted/insist) on or (made/make) {initials} have sexual relations with you when {initials} didn't want to?

Each question was followed with a Likert-scale response ranging from 0=never to 7=more than 20 times in the past year, also including the response of 1= this has not happened in the past year but has happened before then (Harris et al., 2009).

While Wave IV (ages 24-32) uses the same eight questions and response options as Wave III, Wave IV participants only reported information about their most current partner over a 12-month period (Harris et al., 2009). The variation in questions between Wave II and Waves III/IV make it difficult to following partner violence longitudinally.

Similarly, although the questions are pulled from the CTS2, many of the questions surrounding negotiation are missing in the Add Health questionnaire. There is also no standardized method of scoring these items apart from the full or short form CTS2 scale, resulting in a wide range of scoring methods using the Add Health data set.

In Wave II, due to the binary response of the violence questions, studies created overall prevalence variables for the 5 items asked with a positive response indicating that partner violence occurred (Exner-Cortens, Eckenrode, & Rothman, 2013; Gehring, & Vaske ,2015). Studies that have used cross sectional studies of either Wave III or Wave IV have used multiple different scoring methods. Some studies used any affirmative response to a victimization or perpetration question to create binary variables for type of violence: no violence, threatening violence, slapped, or injury or minor violence, major violence, rape/sexual coercion, and injury, for example (Barnes, TenEyck, Boutwell, & Beaver, 2013; Manlove, Welti, & Karpilow, 2015; Milner & Baker, 2015; Notwotny & Graves, 2013). However not every study included both victimization and perpetration questions, with many Add Health studies focusing solely on victimization (Barnes et al., 2013; Milner & Baker, 2015; Notwotny & Graves, 2013).

Rather than focusing on the type of violence occurring, studies have used the questions to identify the perpetrator of any IPV, victim of any IPV, mutual IPV and instigator of mutual IPV (Hess et al., 2013; Kuhl, Warner, & Warner, 2015; Manlove et al., 2015; Tillyer & Wright, 2013). It also important to note that studies have omitted the sexual coercion questions and focused solely on physical partner violence (Manlove et al., 2015; Tillyer & Wright, 2013; Ulloa & Hammett, 2014).

Manlove et al. (2015) combined multiple scoring methods to identify type of IPV using any affirmative response to a question to indicate the violence had occurred and created the following variable categories: no violence, threatening violence, slapped, or injury. A separate frequency variable was created using the Likert-scale to determine how often violence occurred using the categories: 0 times, 1 time, 2 times, 3 to 10 times, and more than 10 times in the past year. This same study also identified perpetrator of violence variables using affirmative responses to any of the victimization or perpetration questions resulting in the following categories: no violence, respondent only, partner only, reciprocal violence: respondent-dominant, partner-dominant, common-couple (Manlove et al., 2015).

Aside from using the Likert-scale response to create binary responses, some of the Add Health studies have created victimization and perpetration scores by summing the Likert-responses for each question (Ulloa & Hammett, 2014; Ulloa & Hammett, 2015). Clark et al. (2016) used Rach modeling to create overall scores based on conditional probabilities of giving a positive response given its severity and the true but unobserved violence exposure of a person. Items most commonly reported were weighted as less severe. However, the summed scores may be difficult to interpret as the 8-item questions do not have clear scoring instructions and are missing valid IPV measures such as emotional abuse, negation and power and control. However, using binary variables such as 0=no violence and 1=IPV has occurred missing important aspects of the violence including severity and frequency. These questions in Add Health, Wave IV specifically, lack the ability to examine the chronicity of violence as questions are limited to past 12

months or most current partner (Harris et al., 2009). Of the biggest limitations is the omission of emotional abuse and power and control. The questions in the Add Health only allow a small amount of violence to be captured which could skew results and associations between IPV and the outcome variable under study. The variance in measurement and scoring among the Add Health studies are also of concern as there are no true guidelines to score these 8 items leaving these decisions up to the researchers which may cause researchers to score and interpret to achieve their desired results. The self-report nature of this survey always allows the possibility that participants may be miss-reporting both victimization and perpetration, which can be a commonality in IPV surveys (Kimmel, 2002).

Conclusion

The IPV literature reveals the limited rigorous data on cardiovascular disease risk among women who experience IPV. Research on young women, specifically, who experience IPV and their long term CVD risks is scant. Studying individual risk factors for CVD separately may not provide the full context of the health risk. Clustering risk factors can provide a more accurate description of one's cardiovascular risk, yet choosing the appropriate model depending on the age and demographic information of the sample is crucial. This literature review highlights an important topic that needs to be further studied in order to intervene in terms of IPV victimization, but also to better understand preventable health outcomes later in life.

There has been a call to examine CVD risk in younger populations as the risk factors for CVD are developing earlier in life. Young women, ages 18 to 25, are also at

most increased risk for IPV victimization compared to older women. However, the link between IPV among young adult women and CVD risk later in life is just beginning to be explored. Using appropriate risk scores, such as the Framingham 30-year risk score, can help examine CVD risk at this age.

Nursing and nursing care can also play a major role in this topic area, specifically the prevention of cardiovascular disease among young adult female victims of IPV. While there is large emphasis of the primary prevention IPV, secondary and tertiary levels of prevention should not be forgotten as IPV is still widely prevalent in society. Nurses have the ability to screen for IPV and create dialogue surrounding IPV victimization with a patient. The unique nurse-patient relationship can better the health care experience for victims of IPV using a trauma informed care approach when working with patients on preventing chronic disease and promoting health management.

Aims

This study will begin to close the gap surrounding young adult female victims of IPV and cardiovascular disease risk by exploring the specific pathways in which IPV victimization is linked to cardiovascular disease risk. The study will use data from the National Longitudinal Study of Adolescents to Adult Health (Add Health), a longitudinal, comprehensive, nationally representative sample of adolescents in the United States (Harris et al., 2009). Add Health has collected data on adolescents in grades 7 to 12 beginning in 1995 (Wave I). The newest set of responses from this data (Wave IV) consists of ages 24-32, ages at which females will have been likely exposed to IPV (Breiding et al., 2014; Harris et al., 2009). Wave IV was chosen as the wave under

analysis in this current study because of its focus on the young adult time period. The purpose of this study is to determine the relationship between female IPV victimization, and 30-year CVD risk as well as the impact of perceived stress level, alcohol dependence, depressive symptoms, and C-reactive protein (CRP) levels on the relationship between IPV victimization and 30-year CVD risk using multiple mediator models.

We will test for relationships among IPV victimization and a 30-year cardiovascular risk score as well as the impact of perceived stress levels, alcohol dependence, depressive symptoms, and C-reactive protein (CRP) levels on the increase of 30-year CVD risk using a cross-sectional secondary data analysis from Wave IV of the National Longitudinal Study of Adolescent to Adult Health (Add Health) and we will control for potential confounders. Potential confounders include insurance status, history of childhood abuse, pregnancy status, race/ethnicity, sexual orientation, income, financial stress, and education level.

We will examine these relationships under these specific aims:

1: To examine the impact of exposure to IPV in the past year on 30-year CVD risk score of young adult females in the sample compared to female peers who have not been exposed to IPV in the past year.

1a: To examine the impact of the high severity of IPV exposure in the past year among young adult women in the sample on 30-year CVD risk score compared to female peers who have been exposed to low IPV severity in the past year.

2: To examine if perceived stress levels, depressive symptoms, and alcohol dependence among young adult females in the sample mediates the relationship between exposure to IPV and 30-year CVD risk score using a multiple mediation model

Exploratory aim: To examine if perceived stress levels and increased C-reactive protein levels (a proxy measure of chronic stress) among young adult females in the sample mediates the relationship between exposure to IPV and 30-year CVD risk score using a multiple mediation model.

CHAPTER 3
METHODS

Introduction

This chapter addresses the methodological aspects of the study, which aim to understand the relationship between IPV victimization and 30-year cardiovascular risk score among young adult women in the United States. After a broad overview of the study methodology, this chapter will describe the parent study where the data were collected, the dataset subset used for this current study, measurement strategies for the variables of interest and the analytic plan for the proposed aims.

Overview

This study was a cross-sectional secondary analysis of the National Longitudinal Study of Adolescent to Adult Health (Add Health), a longitudinal study of a nationally representative sample of adolescents in the United States. The study examined the relationship between exposure to intimate partner violence victimization and 30-year cardiovascular risk using the Framingham 30-year CVD risk score (Pencina et al., 2009). Covariates included in the analysis, derived from the literature, were: health insurance status, history of childhood abuse, race/ethnicity, sexual orientation, education, income, financial stress, health status, and pregnancy status.

Lastly, mediators, also derived from the literature, were added to a multiple mediation model to examine their impact on the relationship between IPV exposure and CVD risk. The mediators included alcohol dependence, depressive symptoms, perceived stress, and high sensitivity C-reactive protein (hsCRP) levels.

Parent Study

Add Health is a longitudinal, comprehensive, nationally representative sample of adolescents in the United States (Harris et al, 2009). Add Health data collection began in 1995 with adolescents in grades 7-12 (Wave I) and the latest data come from the 4th wave (Wave IV) in which participants were ages 24-32. The Add Health study has collected data on health behaviors and risks, cognitive functioning and non-cognitive personality traits, decision-making, expectations, risk preferences and family support, relationship quality and ties of obligation (Harris et al., 2009). A sample of 80 high schools and 52 middle schools from the US were selected with unequal probability of selection. Incorporating systematic sampling methods and implicit stratification into the Add Health study design ensured this sample is representative of US schools with respect to region of country, urbanity, school size, school type, and ethnicity (Harris et al., 2009). Of the 80 high schools selected, 52 were eligible to participate and the remaining 28 schools were replaced by similar schools using the sampling methods previously established. Each participating high school identified one junior high or middle school that would provide at least 5 students to the entering class of the high school. Parental consent was required for students to participate in the study. Add Health participants provided written informed consent for participation in all aspects of Add Health in accordance with the University of North Carolina School of Public Health Institutional Review Board guidelines that which based on the Code of Federal Regulations on the Protection of Human Subjects 45CFR46: http://www.hhs.gov/ohrp/humansubjects/guidance/45cfr46.html (Harris et al., 2009). A rigorous security system was implemented to protect the identities of the participants and prevent the linkage of respondent's answers to their name or identity.

Wave I

In the first wave, 90,118 students completed a 45-minute questionnaire while each school was also asked to complete a School Administrator questionnaire (Harris et al., 2009). From the participating students, random sampling that ensured representative samples was conducted and students were chosen to participate in an in-home interview. The adolescents were interviewed at Wave I and then a year later at Wave II. A Computer-Assisted Personal Interview (CAPI)/Audio Computer-Assisted Self Interview (ACASI) were used to administer survey. Sensitive questions were asked in the self-administered section of the interview (Harris et al., 2009).

A parent or guardian of the adolescent was interviewed during Wave I (Harris et al., 2009). The in-home sample included an oversampled number of black adolescents with college-educated parents, an oversample of Cuban and Puerto Rican adolescents, an oversample of Chinese adolescents and an oversample of physically disabled adolescents. Genetic supplements included twins, siblings of twins, other full siblings, half-siblings and non-related siblings. The total sample size of Wave I was n=20,745 (Harris et al., 2009).

Wave II

Wave II occurred in 1996 with n=14,738 students in grades 8 to 12 (88.6% follow-up) (Harris et al., 2009). Participants were mostly drawn from Wave I participants. Twelfth-graders who exceeded grade requirements were removed aside from those who were part of a genetic pair. Disabled participants from Wave I were not re-

interviewed. Wave II included a small number of participants who did not participate in the Wave I. No parent interview was conducted in Wave II (Harris et al., 2009).

Wave III

Wave III was collected in 2002 with a 77.4% follow-up rate resulting in n=15, 197 for the in-home interview (Harris et al., 2009). In this wave, young adults were ages 18-26 and partners of the participants were also interviewed during a partner questionnaire. New information also included anthropometric measures weight and height as well as STI/HIV testing and buccal cell testing. Residential longitude and latitude of participants were recorded. High school transcripts of participants were also available (Harris et al., 2009).

Wave IV

In the most recent wave, Wave IV, there was a follow-up rate of 80.3% yielding n=15,701 participants (Harris et al., 2009). Data was collected in 2006 where participants were ages 24-32 years. Height, weight, waist circumference, metabolic biomarkers, immune markers, inflammation markers, cardiovascular markers, medications history were also collected in this wave (Harris et al., 2009).

Sample

The present study used data from Wave IV of the Add Health study. 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 excluded from the study. All remaining participants who answered the romantic partner

relationship questions in Wave IV and had valid sample weights (N=7392) were included in the analysis.

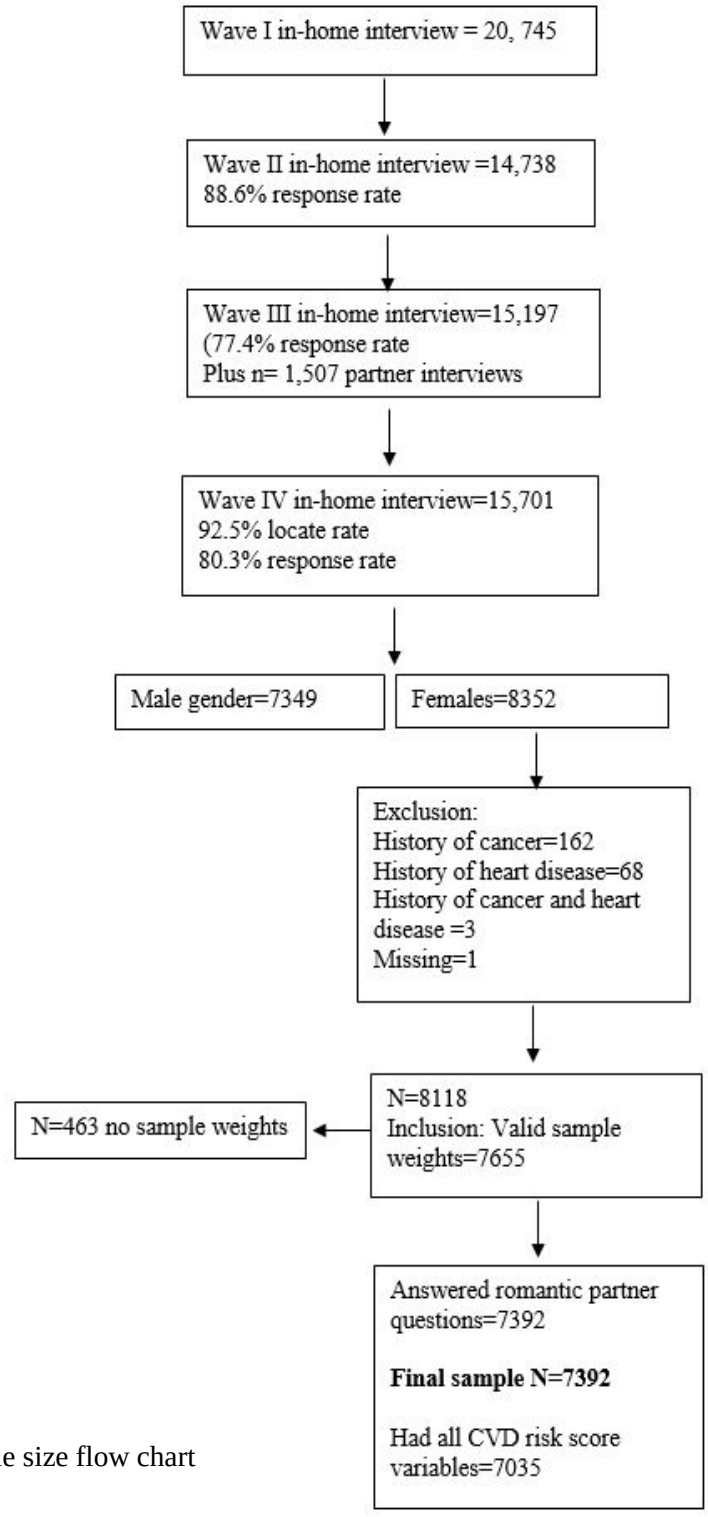


Figure 2: Sample size flow chart

Measurement

Outcome Variable

CVD Risk

A 30-year cardiovascular 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. While this score can be used to estimate both "hard" CVD (coronary death, myocardial infarction, and fatal and non-fatal stroke) and "general" CVD (coronary death, myocardial infarction, coronary insufficiency, angina pectoris, stroke, transient ischemic attack, intermittent claudication and congestive heart failure), this study focused on general CVD risk. While there is no validated cut point for the 30-year risk score, previous literature using this score, which can range from 0% to 100%, on the Add Health sample deemed a 20% score as "high risk" for developing CVD in the next 30 years (Clark et al., 2014; Pencina et al., 2009). Due to the young age of the sample (mean age 29 years), 20% risk was also deemed clinically relevant.

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 smoking status. Participants' systolic blood pressures were measured after a five-minute rest and three measurements were collected at 30-second intervals. The last two measurements were averaged to calculate systolic blood pressure. Medication history was collected to asses for antihypertensive medication use in the past four weeks (Tabor & Whitsel, 2010). A

diagnosis of diabetes was present if participants had a fasting glucose level of 126 milligrams per decliner or above, a non-fasting glucose level of 200 milligrams per deciliter or above, had ever been diagnosed with diabetes from a health care provider aside from during pregnancy or had taken any antidiabetic medication in the past four weeks (Whitsel et al., 2012). Standardized approaches were taken to ascertain height and weight measurements (Entzel et al., 2009). Smoking status was based on self-report of cigarette smoking in the last 30 days.

Predictor Variable

Past Year IPV

The predictor variable in this present study was exposure to intimate partner violence (IPV) in the past year. The Add Health Wave IV survey assessed IPV using questions from the Revised Conflict Tactic Scales (CTS2) (Cronbach's alpha =0.76) (Cui, Ueno, Gordon, & Fincham, 2013; Harris et al., 2009; Straus et al., 1996). As an important note, the Add Health IPV questions do not ask specific emotional IPV questions, which may be a limitation in the study. Each question had Likert-scale responses ranging from 0=this never happened to 7=more than 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. The four victimization questions were:

Victimization	How often (has/did) {initials} (threatened/threaten) you with violence, (pushed/push) or (shoved/shove) you, or (thrown/throw)	How often (has/did) {initials} (slapped/slap), hit or (kicked/kick) you?	How often (have/did) you (had/have) an injury, such as a sprain, bruise, or cut because of a	How often (has/did) {initials} (insisted/insist) on or (made/make) you have sexual relations with

	something at you that could hurt?		fight with {initials}?	(him/her) when you didn't want to?

A binary IPV exposure variable was created by coding any affirmative response to a victimization question as "1". To create an IPV severity measure, each IPV variable was coded as low severity (reported violence less than 3 times in past year) or high severity (3 or more instances of IPV in the past year). Those who had experienced high severity of at least one of the IPV variables was considered "high severity." Those who only reported low severity IPV variables were coded as "low severity."

Mediators

Depressive Symptoms

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

Perceived stress was measured from a validated four item 5-level scale adapted from the Cohen Perceived Stress Scale (Cohen, Kamarck & Mermelstein, 1983). The questions assessed respondents' feelings of lack of control and stress over the past month with summed scores of the questions ranging from 0-16. 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

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, Heeren & Winter, 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 model. The literature states that scores higher than 3 or more is considered alcohol dependence (Hingson et al., 2006).

C - reactive protein

High sensitivity C - reactive protein (hs-CRP), a measure of chronic inflammation and a proxy measure of stress was measures at Wave IV using dried blood samples. Collection, documentation and quality control measures regarding these samples are available through Add Health (Whitsel et al., 2013). The sensitivity of the CRP assay was 0.035 mg/L, the within-assay coefficient was 8.1%, and the between-assay coefficient of variation was 11.0%. Comparison of hs-CRP values from the dried blood spot and plasma was conducted in a sample of 87 participants, linear correlations were high with a Pearson's R= .98 (see Table 1; Whitsel et al., 2013). Any hsCRP levels higher than 6.25 mg/L are considered to be an acute infection and were excluded from analysis.

Covariates of Interest

The covariates in this study were health insurance status, history of childhood abuse, race/ethnicity, sexual orientation, education, income, financial stress, health status, and pregnancy status. These covariates were derived from the literature and have been used in prior studies examining partner violence victimization (Ahmed, & McCaw, 2010; Basu et al., 2013; Cerulli et al., 2010; Cheng & Lo, 2014; Cho & Kim, 2012; Connelly et al., 2013; Flicker et al., 2011;Fox & Benson, 2006; Humphreys, Cooper& Miaskowski, 2010; Humphreys et al., 2012; Kothari, Cerulli, Marcus, & Rhodes, 2009).

Health Insurance

Health insurance status was measured via self-report as binary variable reflecting any or no insurance. All data on insurance status was collected prior to the implementation of the Affordable Care Act.

History of Childhood Abuse

History of child hood abuse is an important variable related to experiencing IPV victimization later in life (Whitfield, Anda, Dube & Felitti, 2003). Add Health screened for three types of childhood abuse:

Type	Screening Question	Binary variable definition
Childhood Neglect	Before your 18th birthday, how often did a parent or other adult caregiver say things that really hurt your feelings or made you feel like you were not wanted or loved?	Greater than 10 times
Childhood Physical Abuse	Before your 18th birthday, how often did a parent or adult caregiver hit you with a fist, kick you, or throw you down on the floor, into a wall, or down stairs?	Greater than 6 times

Childhood Sexual Abuse	How often did a parent or other adult caregiver touch you in a sexual way, force you to touch him or her in a sexual way, or force you to have sexual relations?	Any experience

Responses ranged from *this never happened* to *more than 10 times before your 18th birthday*. Binary variables were created using cutoff points for each type of abuse as analyzed in previous research; childhood neglect cutoff was greater than 10 times, childhood physical abuse cutoff was greater than 6 times, and childhood sexual abuse was 1 or more times (see table above) (Gooding et al., 2014). Using these created binary variables, a single binary child abuse variable of childhood abuse: "yes" or "no" was created. "Yes" was defined as having positive response to any of the binary childhood variables

Race/ethnicity

Race and ethnicity data were collected at Wave I. Participants were asked to identify the category that best reflected their racial background and could choose more than 1 category (White, Black/African American, American Indian/Native American, Asian/Pacific Islander or other) (Harris et al., 2009). Ethnicity was measured through self-report of Hispanic origin (yes/no).

Sexual orientation/sexual identity

Sexual orientation data were collected at Wave IV (Harris et al., 2009). 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

Education data was collected at Wave IV. A binary variable of college degree obtainment was used to measure educational attainment in the present study.

Income

Income was measured by midpoint household income and separated into meaningful categories using Add Health responses.

Financial Stress

Financial stress has been previously examined as a covariate with IPV (Clark et al., 2014). Financial stress was measured using a binary variable of 1= financial stress by an affirmative response to any of the following questions:

In the past 12 months, was there a time when {YOU/YOUR HOUSEHOLD} was without phone service because you didn't have enough money?	In the past 12 months, was there a time when {YOU/YOUR HOUSEHOLD} 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/YOURHOUSEHOLD} 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/YOUR HOUSEHOLD} 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/YOUR HOUSEHOLD} 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/YOUR HOUSEHOLD WERE/WAS} worried whether food would run out before you would get money to buy more?

Self-Reported Health Status

Participants' current health status was measured by self-report using a Likert-scale response ranging from excellent (1) to poor (5).

Pregnancy Status

Current pregnancy status was measured via self-report as pregnancy has shown to increase stress as well as increase risk for IPV victimization (Kothari et al., 2009).

Table 2: Variables in Present Study

Dependent Variable	**Collection Tool**	**Variable Type**	**No. Items**	**Measurement**	**Psychometrics**	**Aims**
30-Year Cardiovascular risk	30-Year Framingham Risk Score	Continuous	7	Range 0%-100%	cross validated c-statistic=0.803, internally validated c-statistic=0.802, cross validated Nam-D'Agostino chi-square =x2=4.25, and internally validated Nam-D'Agostino chi-square=x2=3.98	All aims
Predictors of Interest	**Collection Tool**	**Variable Type**	**No. Items**	**Measurement**	**Psychometrics**	**Aims**
Intimate Partner Violence Exposure	Adapted from Revised-Conflict Tactics Scale	Binary	2	8- point Likert Scale. Any positive response coded to 1= IPV exposure.	$\alpha = 0.76$ for Wave IV	Primary aim, secondary aim, exploratory aim
Severity of Intimate Partner Violence Exposure	Adapted from Revised-Conflict Tactics Scale	Binary	2	8-point Likert Scale. Coded into low and high	$\alpha = 0.76$ for Wave IV	Primary sub aim

				severity		
Mediators	**Collection Tool**	**Variable Type**	**No. Items**	**Measurement**	**Psychometrics**	**Aims**
Perceived Stress Level	4-item Cohen Perceived Stress Scale	Continuous	4-item	4 questions with 8-point Likert-scale. Results summed and scored with positive phrases reversed scored.	$\alpha = 0.72$	Secondary aim, exploratory aim
Depressive symptoms	Abbreviated version of Center for Epidemiologic Studies Depression Scale (CES-D).	Continuous	5-iten	5 questions with 4-point Likert scale. Results were scored and summed with a higher scores indicating greater depressive symptoms.	$\alpha = 0.79$	secondary aim, exploratory aim
Alcohol Dependence	DSM IV Criteria for Alcohol Dependence	Continuous	8 items	8 questions with binary yes/no responses to create overall score.	n/a	Secondary Aim
C-reactive protein levels	Biomarker collection	Continuous	1	Range 0-6.25 mg/L	The sensitivity of the CRP assay was 0.035 mg/L, the within-assay coefficient was 8.1%, and the	exploratory aim

					between-assay coefficient of variation was 11.0% . Comparison of hs-CRP values from the dried blood spot and plasma was conducted in a sample of 87 participants; linear correlations were high with a Pearson's R= .98	
Covariates	**Collection Tool**	**Variable Type**	**No. Items**	**Measurement**	**Psychometrics**	**Aims**
Health insurance status	Add Health developed	Binary	3	0=no health insurance 1Insured	n/a	Aim 1
Childhood abuse	Add Health developed	binary	2	Combined 3 childhood abuse variables into overall childhood abuse variable	n/a	Aim 1
Race/ethnicity	Add Health developed	Nominal	5	White, Black/African American, American Indian/Native American, Asian/Pacific Islander or other. Hispanic	n/a	Aim 1

				origin		
Sexual orientation/sexual identity	Add Health developed	Binary	5	Heterosexual, sexual minority	n/a	Aim 1
Education level	Investigator developed	Binary	4	No college degree and College degree	n/a	Aim 1
Income	Mid-point household income	Ordinal Categorical	4	Range from <20,000 to >$75,000	n/a	Aim 1
Financial stress	Add Health developed	binary	2	A positive to response to any of the 7 financial stress questions. Coded to 1=financial stress	n/a	Aim 1
Health status	Add Health developed	ordinal	5	Responses ranging from 1=excellent to 5=poor	n/a	Aim 1
Pregnancy status	Add Health developed	binary	2	1= currently pregnant	n/a	Aim 1

Preliminary Analysis

Power Analysis

A power analysis was run to estimate the sample sizes needed to detect a significant effect size for Aim 1 and Aim 1a. Both aims test 1 independent variable and control for the same amount of independent variables. Table 3 below highlights multiple

power tests that estimated sample sizes for Aim 1 and Aim 1a, using 90% power, to detect different ranges of R-squared attributable to a single predictor variable of interest (IPV exposure and IPV severity) using an F-Test with a significance level set to 0.01. For example, a sample size of 1328 is needed to detect, with 90% power, an R-squared of < 1% attributable to a single predictor variable of interest (IPV exposure) using an F-Test with a significance level of 0.01. The estimated sample size for Aim 1 was approximately n=7000. Therefore, according to the power analysis, this study will be adequately powered to detect a small effect size for Aim 1. Aim1a had a sample of n=1166. Therefore, this sample size, with 90% power, can detect R-squared of < 2% attributable to a single predictor variable of interest (IPV severity) using an F-Test with a significance level of 0.01. As the analysis including the full sample size is overpowered, Cohen's d effect sizes were estimated to help accommodate for the large sample size (Cohen, 1988). Effect sizes help to interpret the magnitude of effect independent of sample size (Cohen, 1988).

Table 3

Power Analysis Table

Aim 1 and Aim 1a							
Power	N	Alpha	Beta	Ind. Variables Tested		Ind. Variables Controlled	
				Cnt	R2	Cnt.	R2
0.9001	1328	0.01	0.0999	1	0.010	11	0.10
0.9042	108	0.01	0.0995	1	0.020	11	0.10
0.9238	21	0.01	0.0993	1	0.050	11	0.10
0.9001	1179	0.01	0.0999	1	0.010	11	0.20

0.90020	584	0.01	0.0998	1	0.020	11	0.20
0.90054	227	0.01	0.0996	1	0.050	11	0.20
0.9003	1031	0.01	0.0997	1	0.010	11	0.30
0.9005	510	0.01	0.0995	1	0.020	11	0.30
0.9000	197	0.01	0.1000	1	0.050	11	0.30
0.90026	733	0.01	0.0998	1	0.010	11	0.50
0.90047	361	0.01	0.0996	1	0.020	11	0.50
0.90137	138	0.01	0.0989	1	0.050	11	0.50

Missing Data

Prior to analysis of the aims, preliminary analysis identified missing data in this secondary analysis. If missing data was excessive or arbitrary (>10%) the multiple imputation procedure, developed by the Survey Methodology Program at the University of Michigan was to be run (Chen & Chantala, 2014). Multiple imputation is warranted when missing data occurs in a random or arbitrary pattern (Yuan, 2010). Multiple imputation (MI) uses the Markov chain Monte Carlo (MCMC) algorithm known as fully conditional specification or chained equations imputation (IBM, 2012). This algorithm allows the imputation of incomplete variables one at a time, using the filled-in variable from one step as a predictor in all subsequent steps. After data was examined, missing data was not excessive or arbitrary and all data analyses was used with complete data.

Descriptive statistics

Using Add Health user guidance, all data analysis incorporated grand sample survey 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). Data was analyzed using SAS Version 9.4 for the descriptive statistics and regression models while Mplus 7 was used for the multiple mediation analysis.

We performed visual data analysis, including histograms and scatterplots on the data to better understand patterns and relationships between covariates and the predictor variable. Descriptive statistics were run on the outcome variable, predictor of interest, possible mediators and covariates. For nominal variables, descriptive statistics included distribution frequencies and percentages. For continuous variables and nominal variables with five or more categories, descriptive statistics included measures of central tendency and variation such as mean, median, standard deviation, minimum and maximum values and ranges. We performed two sample t-tests and chi-square testes of all variables to the predictor of interest variable, exposure to intimate partner violence victimization.

Aims

Aim 1: To examine the impact of exposure IPV in the past year on 30-year CVD risk among young adult females compared to female peers who have not been exposed to IPV in the past year. The 30-year risk score includes age, sex, systolic blood pressure, use of antihypertensive medication, smoking status, diabetes diagnosis, and BMI.

To test the primary aim, inferential associations between intimate partner violence exposure and 30-year cardiovascular risk score were examined using general linear modeling (GLM) which relies on weighted least squares to estimate model parameters (IBM,2012). CVD risk scores was regressed over IPV exposure while controlling for covariates (see Table 2) in order to identify statistically meaningful associations between

IPV exposure and CVD risk at the .05 significance level. The underlying assumptions of the GLM are linearity, statistical independence of the errors, homoscedasticity and normality of the error distribution (Koerts, & Abrahamse, 1969; Nau, 2016). Due to the categorical nature of the predictor variables, linearity is not a concern. Similarly, the study design insures that participants are independent of one another. The CVD risk score within the IPV exposure groups will follow a Gaussian distribution and have homogenous variances. The CVD risk distributions will be tested for normality using the Shapiro-Wilks test. To protect against potential heterogeneous variances, a model will be generated using robust variance estimation (Nau, 2016). If the normality assumptions are violated, transformation of the variables will be considered.

Aim 1a: To examine the impact of the high severity of IPV exposure in the past year among young adult women in the sample on 30-year CVD risk compared to female peers who have been exposed to low IPV severity in the past year.

Associations between severity of intimate partner violence exposure and 30-year cardiovascular risk score was examined using general linear modeling. CVD risk scores were regressed over severity of IPV exposure while controlling for covariates (see Table 2). The model tested statistical assumptions highlighted in the previous aim.

Aim 2: To examine if perceived stress levels, depressive symptoms, and alcohol dependence among young adult females mediates the relationship between exposure to IPV in the past year and 30-year CVD risk score using a multiple mediation model.

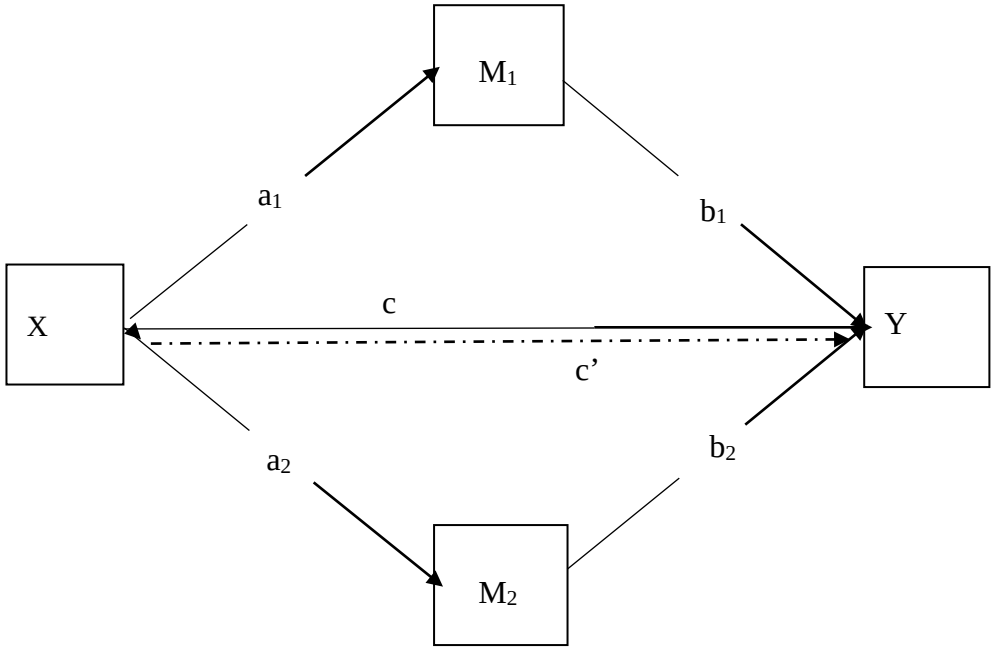


Figure 3: Multiple mediation model
Figure 3 displays a standard multiple mediation model (Preacher & Hayes, 2008). The total effect of *X* on *Y*, independent of the mediators, is represented by *c*. The indirect effect of *X* on *Y*, determined once mediators are included in the model, is represented by *c'*. The specific indirect effect of *X* on *Y* through each mediator (M_1 and M_2) is the product of the respective unstandardized *ab* coefficients (a_1b_1, a_2b_2). The total indirect effect of the mediators can be calculated by *c*-*c'* (Preacher & Hayes, 2008). Multiple mediation models report specific the indirect effects of M_1 dependent on M_2.

The multivariate delta method, also known as the Sobel test, in Mplus 7 assessed the mediating effects of adverse coping mechanisms and behaviors between intimate partner violence exposure and 30-year CVD risk score. 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). This allowed us to test the following: (a) to the total indirect effect of exposure to IPV on 30-year CVD risk through the set of mediators described in Aim 2, (b) the specific indirect effect of the IPV exposure on 30 –year CVD risk score mediated by perceived stress, (c) the specific

indirect effect of the IPV exposure on 30 –year CVD risk score mediated by depressive symptoms, and (d) the specific indirect effect of the IPV exposure on 30 –year CVD risk score mediated by alcohol dependence (Preacher & Hayes, 2008). All mediation analyses included sample weights and provided both unstandardized and standardized indirect effects for interpretation and comparison. Simple mediations were run prior to multiple mediation using the Baron and Kenny (1986) method for mediation to aid in interpretation.

Exploratory Aim (Aim 3): To examine if perceived stress levels and increased C-reactive protein levels (a proxy measure of stress) among young adult females mediates the relationship between exposure to IPV in the past year and 30-year CVD risk score using a multiple mediation model.

The multivariate delta method assessed the mediating effects between intimate partner violence exposure and 30-year CVD risk score in Mplus 7 to test the following: (a) to the total indirect effect of exposure to IPV on 30-year CVD risk through the set of mediators described in the exploratory aim, (b) the specific indirect effect of the IPV exposure on 30 –year CVD risk score mediated by perceived stress levels and (c) the specific indirect effect of the IPV exposure on 30 –year CVD risk score mediated by C-reactive protein levels (Preacher, & Hayes, 2008). All mediation analyses included sample weights and provided both unstandardized and standardized indirect effects. Simple mediations were run prior to multiple mediation using the Baron and Kenny (1986) method for mediation.

Data Integrity and Security

Data was sent to the University Of Pennsylvania School Of Nursing via mail on a CD-ROM from the Add Health Researchers. All data was previously de-identified before being sent to the University of Pennsylvania. A data contract was signed between Add Heath researchers and the current study team. When the contract for this analysis ends, the CD-ROM will be returned to Add Health and all data will be cleared from the computer used to analyze data during this present study. Add Health data access was restricted to key study personnel only using security features such as login via username and strong passwords. Encryption software for directories containing secure data was installed and analysis software was configured to point temporary work files to the encrypted Add Health data directory. A secure erasure program was run monthly and after secure data has been removed from the computer. Data was protected from unauthorized access across the wire. The security protocols developed by the IT department of the School of Nursing were followed to ensure the integrity and security of the data and to prevent deductive disclosure.

Humans Subjects

Due to the nature of secondary analysis, there was no contact with human subjects in the present study. The parent study received consent from parents and participants throughout the waves of the study.

IRB and Add Health approval

This study was determined exempt from review by the University of Pennsylvania Institutional Review Board.

CHAPTER 4
RESULTS

Introduction

This chapter reports the analysis and findings of the study. This cross-sectional secondary analysis from Add Health examined relationships between IPV exposure in the past year and 30-year CVD risk score among a representative sample of young adult women in the United States. The major aims of this study were:

1: To examine the impact of exposure IPV in the past year on 30-year CVD risk score of young adult females compared to female peers who have not been exposed to IPV in the past year. The 30-year risk score includes age, sex, systolic blood pressure, use of antihypertensive medication, smoking status, diabetes diagnosis, and BMI.

1a: To examine the impact of the high severity of IPV exposure in the past year among young adult women in the sample on 30-year CVD risk score compared to female peers who have been exposed to low IPV severity in the past year.

2: To examine if perceived stress levels, depressive symptoms, and alcohol dependence among young adult females mediates the relationship between exposure to IPV in the past year and 30-year CVD risk using a multiple mediation model.

3: To examine if perceived stress levels and increased C-reactive protein levels (a proxy measure of stress) among young adult females mediates the

relationship between exposure to IPV in the past year and 30-year CVD risk score using a multiple mediation model.

All analysis incorporated the sample weights and complex survey analysis as recommended by Add Health to ensure results are representative (Chantala & Tabor, 2010). SAS Version 9.4 and Mplus 7 were used for the analysis. The final sample included 7392 females who answered the partner relationship questions, had no history of cancer or heart disease, and had valid sample weights at Wave IV. First, descriptive statistics of the sample including chi-square analysis and comparison of means are discussed. Then the regression models and mediations models for each aim are discussed.

Descriptive Statistics

Table 4 displays the overall characteristics of the sample. The mean age of the women was 29 years. Of the women in the sample, 15% reported any IPV in the past year. Over half the sample where white (59.2%) and 21.5% of the sample were African American. Almost 20% of the sample identified as something other than not 100% heterosexual. Only about one-third of the sample had completed a college degree. Approximately 12% of the women reported a midpoint household income of less than $20,000 a year while over a quarter (26.7%) reported financial stress. A majority of the sample had health insurance (83%) and almost 90% of the sample reported good to excellent health status. Only 7% of the sample was currently pregnant. Almost a quarter of the sample (20%) had experienced some type of childhood abuse.

In terms of mediators, perceived stress levels had a mean score of 5.05 (medium stress level). Mean depressive symptoms score was 2.829 (score of 4 or more indicated

depressive symptoms) while mean alcohol dependence score was .665 (score of 3 or greater indicated alcohol dependence). The mean hsCRP level was 2.12 mg/L (avg risk for CVD). The mean 30-year CVD risk score was 8.2%. Mean systolic blood pressure was 119.9 mmHg while mean BMI was 28.97. A large majority of the sample was not taking medication treatment for high blood pressure and had no diagnosis of diabetes mellitus. Approximately one-third of the sample were current smokers.

Table 3: **Descriptives (n=7392)**	
Variable	**n(%) or mean**
Age (years)	28.8
Race (n=7379)	
White	4368 (59.2)
African American	1589(21.5)
American Indian	81(1.1)
Asian/Pacific Islander	398(5.4)
Mixed/Other	925(12.5)
Ethnicity (n=7367)	
Hispanic	1161(15.8)
Non-Hispanic	6206(84.2)
Sexual identity (n=7379)	
Htersoexual	5919 (80.2)
Sexual minority	1460 (19.8)
College Degree	
Yes	2688(36.4)
No	4704(63.6)
Midpoint Household Income(n=6979)	
<$20,000	879(12.6)
$20-000-$40,000	1466(21)
$40,000-$75,000	2551(36.6)
$>75,000	2083(29.8)
Financial Stress	
Yes	1971(26.7)

No	5421(73.3)
Insured (n=7384)	
Yes	6130(83)
No	1254(17)
Self-Reported Health Status	
Excellent	1367(18.5)
Very Good	2834(38.3)
Good	2495(33.8)
Fair	614(8.3)
Poor	82(1.2)
Currently Pregnant(n=7358)	
Yes	479(6.5)
No	6879(93.4)
Childhood Abuse (n=7285)	
Yes	1457(20)
No	5828(80)
Perceived Stress Level (n=7385)	
Low	2485(33.6)
Med	2677(36.2)
High	2223(30.1)
Mean	5.05 (0.068)
Depressive Symptoms (n=7389)	
Yes	2293(31)
No	5096(69)
Mean	2.829 (.0479)
Alcohol Dependence (n=7390)	
Yes	741(10)
No	6649(90)
Mean	.665 (.0299)
Past Year IPV	
Yes	1158(15.7)
No	6221(84.2)
hsCRP Levels (mean mg/L) (n=6735)	6.12
30-Year CVD risk (mean) (n=7035)	0.0824

Systolic Blood Pressure (mean mmHg) (n=7136)	119.859
Medication Treatment for Blood Pressure	
Yes	257(3.5)
No	7135(96.5)
Body Mass Index (mean) (n=7279)	28.97
Current Smoker (n=7360)	
Yes	2269(30.1)
No	5091(69.2)

Descriptive Statistics by Any IPV in Past Year

Table 5a displays the descriptive statistics of all variables by any past year IPV using t-tests and chi-square analysis. Although the statistics revealed a statistically significant difference in age between groups, the mean age for both group were 29 years. Being African American (30.7% vs 19.8 %, p<.01) or mixed race (14.1% vs 12.2 %, p<.01) was associated with IPV in the past year compared to whites. Over one fourth (26%) of those who reported past year IPV were categorized as a sexual minority woman compared to only 18.9% of those who did not report past year IPV (p<.01). Not having a college degree (75.5% vs. 61.4%, p<.01) and reporting a lower midpoint household income was also significantly associated with past year IPV. Those who reported past year IPV were significantly associated with being uninsured compared to those who did not report past year IPV (23.4% to 15.8%, p<.01). Those who reported past year IPV were also significantly associated with fair (12.7% vs 7.5%, p<.01) or poor health status (1.7% vs .9%, p<.01) as well as history of childhood abuse (27.7% vs 18.6%, p<.01). Of the mediators, high perceived stress (6.30 vs. 4.79, p<.01) and increased depressive

symptoms (3.899 vs 2.6409, p<.01) were significantly associated with past year IPV, however alcohol dependence was not statistically different between groups. hsCRP levels (2.134 vs 2.092 mg/L, p<.01) were higher among those who did not reported past year IPV (p<.01). 30-year CVD risk was significantly higher among those woho reported past year IPV (9.6% vs. 8.7%, p<.01).

Cohen's *d* effect sizes were also calculated due to the large sample size. Effect sizes can be used to examine the size of the difference between two groups without confounding with sample size (Cohen, 1988). Effect sizes greater than 0.20 are deemed significant effect sizes in this study. Cohen defines small effect sizes as less than 0.20, medium effect sizes as 0.20 to 0.50 and large effect sizes as greater than 0.50 (Cohen, 1988). The only variables with effect sizes larger than 0.20 are two of the proposed mediators, perceived stress level (.2967) and depressive symptoms (.312). Notably, the outcome variable, 30-year CVD risk score, had a relatively small effect size (-0.0784) compared to other significant predictors in the table.

Table 5a

Descriptive Statistics by Any IPV in Past Year (n=7392)

	No Past Year IPV n=6231	Past Year IPV n=1161	p-value	Effect size
Age (mean; SD)	28.89 (9.23)	28.84 (5.14)	**p<.001**	0.006
Race n(%) (n=7379)			**p<.001**	0.106
White	3812 (61.3)	574(49.6)		
African American	1234 (19.8)	355(30.7)		
American Indian	70(1.1)	11 (.9)		
Asian/Pacific Islander	343 (5.5)	55 (4.7)		
Mixed/Other	762(12.2)	163 (14.1)		

Sexual Identity n (%) (n=7379)			**p<.001**	0.060
Heterosexual	5067 (81.5%)	852 (73.5)		
Sexual minority	1153 (18.5%)	307 (26.5)		
Ethnicity n(%) (n=7367)			**p<.05**	0.029
Hispanic	980 (15.8)	181(15.6)		
Non-Hispanic	5228 (84.2)	978 (84.3)		
College Degree n (%)			**p<.001**	0.117
Yes	2403 (38.6)	285(24.5)		
No	3828 (61.4)	876(75.5)		
Midpoint Household Income n(%)(n=6979)			**p<.001**	0.133
<$20,000	653(11.1)	226(20.8)		
$20,000-$40,000	1194(20.3)	272(25)		
$40,000-$75,000	2201 (37.4)	350(32.2)		
$>75,000	1843 (31.3)	240(22.1)		
Financial Stress n(%)			**p<.001**	0.137
Yes	1486 (23.8)	485(41.8)		
No	4745(76.2)	676 (58.2)		
Insured n(%) (n=7384)			**p<.001**	0.0767
Yes	5242(84.2)	888(76.6)		
No	982(15.8)	272(23.4)		
Self-Reported Health Status n(%)			**p<.001**	0.095
Excellent	1220(19.6)	147(12.7)		
Very Good	2434(39.1)	400(34.5)		
Good	2048 (32.9)	447(38.5)		
Fair	467 (7.5)	147 (12.7)		
Poor	62(.9)	20(1.7)		

Currently Pregnant n(%) (n=7358)			**p<.05**	0.033
Yes	413(6.7)	66(5.7)		
No	5788 (93.3)	1091(94.3)		
Childhood Abuse n(%) (n=7285)			**p<.001**	0.080
Yes	1140(18.6)	317(27.7)		
No	5000(81.4)	828 (72.3)		
Perceived Stress Level mean(SD) (n=7385)	4.794 (5.531)	6.3075 (4.631)	**p<.001**	**-0.297**
Depressive Symptoms mean(SD) (n=7389)	2.6409 (4.009)	3.8998 (4.055)	**p<.001**	**-0.312**
Alcohol Dependence mean(SD) (n=7390)	.6777 (2.351)	.7294 (1.989)	p=.241	-0.024
CRP Levels (mean; SD) (n=4786)	2.134 (2.04)	2.092 (2.23)	**p<.001**	0.021
30-Year CVD risk (mean; SD) (n=7035)	.0869(0.1309)	.0955(0.0831)	**p<.001**	-0.078
Note: Percentages may not sum to 100% due to rounding				

Descriptive Statistics by Severity of IPV

Table 5b displays the descriptive statistics of each variable by severity of IPV in the past year using t-tests and chi-square analysis. This sub-sample consists of those who reported any past year IPV. The mean age for both groups was 29 years. The only statistically significance differences between groups were completing college, depressive symptoms, hsCRP levels, and CVD risk score. Those who reported high IPV severity had a slightly higher frequency (79.5% vs 73.4%) of not completing college and this relationship was significant at the 0.05 level (p<.05). Of those who reported high IPV

severity, the mean score for depression was 4.242 compared to a mean score of 3.694 for those who reported low IPV severity. High severity IPV participants had a significantly higher hsCRP (mg/L) level (2.270 vs. 1.98, p<0.01). The high severity group also had a higher 30 -year CVD risk score (9.98% vs. 9.32%, p<0.01). No variables had an effect size greater than 0.20 though depressive symptoms and hsCRP levels had effect sizes of approximately 0.13.

Table 5b

Descriptive Statistics by Severity of IPV (n=1166)

	Low Severity n=747	High Severity n=419	p-value	Effect size
Age (mean; SD)	28.81 (0.1678)	28.91 (0.1811)	**p<.001**	-0.024
Race n(%) (n=1163)			p=.400	0.075
White	357(47.9)	221(52.9)		
African American	240(32.2)	115(27.5)		
American Indian	9(1.2)	2 (.5)		
Asian/Pacific Islander	35 (4.7)	20(4.8)		
Mixed/Other	104(14.0)	60 (14.4)		
Ethnicity n(%) (n=1164)			p=.565	0.028
Hispanic	124(16.6)	60(14.3)		
Non-Hispanic	621(83.4)	359(85.7)		
Sexual Identity n (%) (n=1163)			p=.4846	0.023
Heterosexual	548 (73.6%)	308 (73.7%)		
Sexual minority	197 (26.4%)	110 (26.3%)		
Completed College n(%)			**p<.05**	0.069
Yes	199(26.6)	86(20.5)		
No	548(73.4)	333(79.5)		
Midpoint Household Income n(%) (n=1092)			p=.343	0.078
<$20,000	126(18)	102 (26)		
$20-000-$40,000	169 (24.1)	103(26.3)		
$40,000-$75,000	239(34.1)	113 (28.8)		

$>75,000	166 (23.7)	74(18.9)		
Financial Stress n(%)			p=.753	0.013
Yes	302(40.4)	185(44.2)		
No	445(59.6)	234(55.8)		
Insured n(%) (n=1165)			p=.885	0.006
Yes	576(77.2)	316(75.4)		
No	170((22.8)	103(24.6)		
Self-Reported Health Status n(%)			p=.24	0.095
Excellent	100(13.4)	47(11.2)		
Very Good	258(34.5)	143(34.1)		
Good	283 (37.9)	166 (39.6)		
Fair	98 (13.1)	51(12.2)		
Poor	8(1.1)	12(2.9)		
Currently Pregnant n(%)(n=1162)			p=.989	0.001
Yes	40(5.4)	26(6.2)		
No	705 (94.6)	391(93.8)		
Childhood Abuse n(%)(n=1150)			p=.413	0.035
Yes	196(26.6)	121(29.4)		
No	542(73.4)	291 (70.6)		
Perceived Stress Level (mean; SD) (n=1165)	6.236 (.151)	6.442(.241)	p=.670	-0.045
Depressive Symptoms (mean; SD)	3.694 (.165)	4.242 (.1764)	**p<.001**	-0.134
Alcohol Dependence (mean; SD)	.7481 (.0739)	.6900 (.0778)	p=.489	0.032
CRP Levels (mean; SD) (n=738)	1.98 (2.39)	2.270 (1.94)	**p<.001**	-0.130
30-Year CVD risk (mean; SD) (n=1109)	.0932 (0.00304)	.0998 (.00432)	**p<.01**	-0.079
Note: Percentages may not sum to 100% due to rounding				

Analysis of Aim 1

Regression Results on the Association between Past Year IPV and 30-year CVD risk

30-year CVD risk score was regressed over past year IPV in a bivariate model (Model 1, Table 6a). Those who reported past year IPV had a 0.009-unit increase in their 30-year CVD risk score ($p<0.01$). However the strength of the model was small ($F=13.40$, $R^2=0.00295$). We then ran a multivariate model (Model 2) including the previously identified predictor variables from the literature ($R^2=.2269$, $F=47.40$, $p<.01$). In this model, the relationship between past year IPV and 30-year CVD risk score became insignificant once predictors were introduced. Women who reported being Asian/Pacific Islander saw a statistically significant 0.02-unit decrease in 30-year CVD risk compared to White women. Being Hispanic ($B=-0.010756$, $p<.01$), having a college degree ($B=-0.01399$, $p<.01$), having health insurance ($B=-0.00852$, $p<.01$), and being currently pregnant ($B=-0.0089$, $p<.01$) all significantly decreased 30- year CVD risk score compared to their respective reference groups. Identifying as 100% heterosexual also decreased CVD risk, compared to identifying as any other sexual orientation ($B= -0.00542$, $p<.01$).

Table 6a

Regression Models for the Association between Past Year IPV and 30-year CVD Risk Score

	Model: 1 Bivariate				Model 2: Full model with all predictors (n=6700)			
	B	β	SE	P	B	β	SE	P
Age					0.008695	0.27696	0.0004712	<**0.0001**
Race (reference: White)								
African American					0.00212	0.01349	0.00258	0.3944
American Indian					0.00185	0.00278	0.00721	0.7974
Asian/Pacific Islander					-0.02041	-0.0633	0.003006	<**0.001**

Mixed/other					0.00159	0.00836	0.00335	0.6350
Ethnicity (reference: Non-Hispanic)								
Hispanic					-0.01037	-0.0568	0.0027782	<**0.001**
Sexual orientation (reference: sexual minority)								
Heterosexual					-0.00542	-0.0386	0.0021397	**0.011**
College Degree (reference: No)								
Yes					-0.01401	-0.1194	0.001523	<**0.001**
Midpoint Household Income (reference: >$75,000)								
<&20,000					0.01149	0.06804	0.003218	<**0.001**
$20,000-$40,000					0.00518	0.03786	0.00240	**0.0309**
$40,000-$75,000					0.00350	0.02986	0.0018370	0.0536
Financial Stress (reference: No)								
Yes					0.00801	0.06345	0.002242	<**0.001**
Insured (reference: No)								
Yes					-0.00831	-0.0553	0.0026	**0.02**
Health Status (reference: Very Good)								
Excellent					-0.01111	-0.0766	0.001755	<**0.001**
Good					0.01475	0.12352	0.00187	<**0.001**
Fair					0.04109	0.18860	0.00499	<**0.001**
Poor					0.05744	0.10653	0.01676	<**0.001**
Currently Pregnant (reference: No)								
Yes					-0.0088	-0.0394	0.00244	<**0.001**

Childhood Abuse (reference: no)								
Yes					-0.00042	-0.0030	0.0021	0.8421
Past Year IPV (reference: No)								
Yes	0.0085	0.0543	0.002335	**<0.001**	-0.00174	-0.0111	0.00230	0.4491
R-square	0.002951				0.2285			
Root MSE	0.05651				0.04959			
F-statistic (p-value)	**13.40 (0.0003)**				**45.86 (<.001)**			

Table 6b displays the results of Aim 1a, which includes all participants who reported past year IPV. Model 3 is a bivariate analysis of 30-year CVD risk regressed over those who experienced high IPV severity (36%) with low IPV severity (64%) as the reference category. In this model, there was no statistically significant relationship between IPV severity and 30-year CVD risk. Model 4, which included other predictors, was significant at the $p<0.01$ level ($F=6.81$, $R^2=.2187$). Having a college degree (B=-0.01314, p<.01), having health insurance (B=-0.01, p<.05), and having "excellent" self-reported health status (B=-0.01568, p<.01) were all significantly associated with a decrease in 30-year CVD risk score.

Table 6b

Regression Models for the Association between Severity of Past Year IPV and 30-year CVD Risk Score

	Model 3: Bivariate				Model 4: Full model with all predictors			
	B	β	SE	P	B	β	SE	P
Age					0.0093668	0.30464	0.00131	**<0.001**
Race (reference: White)								

African American					-0.0029725	-0.0220	0.01533	0.5765
American Indian					0.0016873	0.0026	0.0148	0.9124
Asian/ Pacific Islander					-0.0150624	-0.0437	0.00826	0.0686
Mixed/other					0.004549	0.0275	0.0071	0.523
Ethnicity (reference: Non-Hispanic)								
Hispanic					-0.0069768	-0.0415	0.00706	0.314
Sexual orientation (reference: sexual minority)								
Heterosexual					0.0006714	0.0052	0.00377	0.858
College Degree (reference: No)								
Yes					-0.0131543	-0.0960	0.00389	**<0.001**
Midpoint Household Income (reference: >$75,000)								
<&20,000					0.0132183	0.0981	0.00679	0.052
$20,000-$40,000					0.0011036	0.0084	0.0059	0.851
$40,000-$75,000					0.0021874	0.0180	0.0051	0.667
Financial Stress (reference: No)								
Yes					0.0024976	0.0218	0.00439	0.569
Insured (reference: No)								
Yes					-0.0100114	-0.0765	0.00494	**0.043**
Health Status (reference: Very Good)								
Excellent					-0.0157420	-0.0960	0.00456	**<0.001**

Good					0.0062045	0.0528	0.00436	0.155
Fair					0.0383635	0.2157	0.00869	**<0.001**
Poor					0.0154111	0.0413	0.0160	0.336
Currently Pregnant (reference: No)								
Yes					-0.0130952	-0.0466	0.0078	0.094
Childhood Abuse (reference: No)								
Yes					-0.0074147	-0.0583	0.0042	0.079
IPV Severity (reverence: Low Severity)								
High Severity	0.0066495	0.056	0.00456	0.1453	0.0042855	0.03622	0.00426	0.315
R-square	0.003172				0.1303			
Root MSE	0.05669				0.05346			
F-statistic (p-value)	2.12 (0.1453)				5.55 **(<0.001)**			

Analysis of Aim 2 and Aim 3

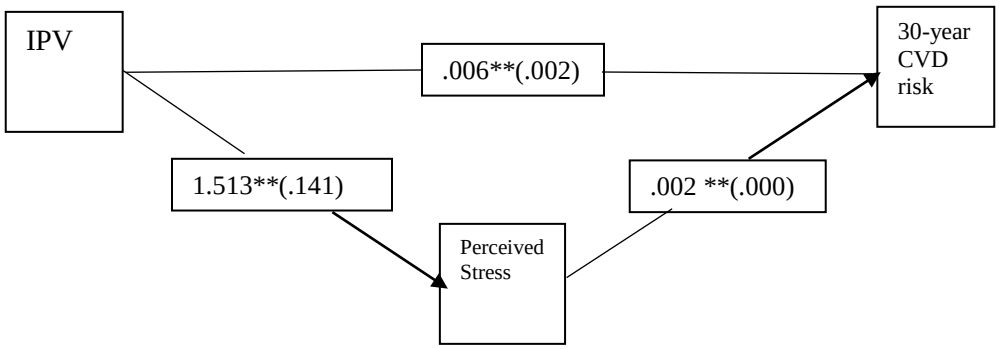
We first ran simple mediation analyses on each mediators to better understand their relationships with IPV and 30-year CVD risk score.

Simple Mediation Analyses

Perceived Stress

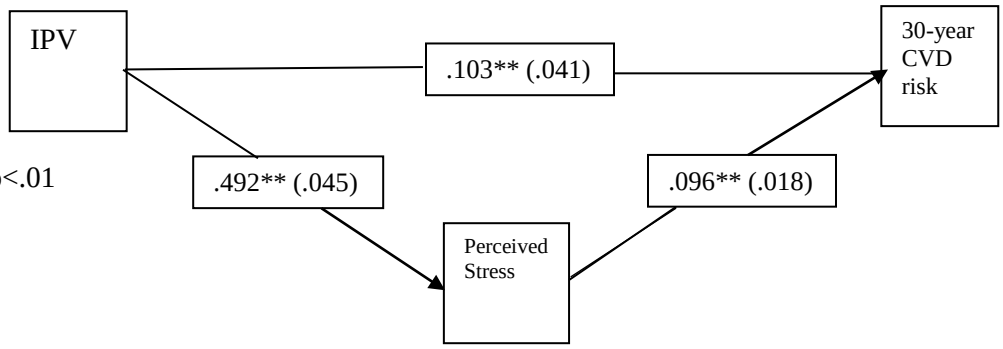
Figure 4a displays the unstandardized regression coefficients and Figure 4b displays the standardized regression coefficients for the impact of past year IPV on 30-year CVD risk score through perceived stress. Using the Baron & Kenny (1986) method, IPV was significantly associated with perceived stress (B=1.513, p<.01, Figure 4a) while perceived stress was also significantly associated with 30-year CVD risk score (B=0.002,

p<.01, Figure 4a). Perceived stress partially mediates the relationship between IPV and 30-year CVD risk score as the direct relationship between IPV and 30-year CVD remains significant with perceived stress in the model.



**p<.01

Figure 4a: Unstandardized Regression Coefficients for Impact of Past Year IPV on 30-year CVD Score through Perceived Stress



**p<.01

Figure 4b: Standardized Regressions Coefficients for Impact of Past Year IPV on 30-year CVD Score through Perceived Stress

Table 7 summarizes the standardized and unstandardized indirect, direct, and total effects of perceived stress on the effect of IPV on 30-Year CVD risk score. The total standardized effect of IPV to CVD risk was 0.150 (p<.01, 95% CI 0.071, 0.229). The direct standardized effect of IPV to CVD was 0.103 (p<.01, 95% CI 0.023, 0.182) leaving

the specific standardized indirect effect of perceived stress to be 0.047 (p<.01, 95% CI 0.027, 0.067).

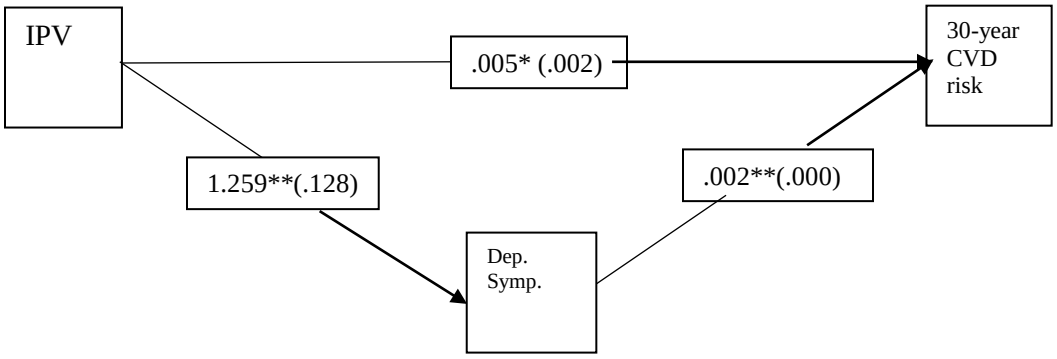
Table 7

Standardized and Unstandardized Indirect, Direct, and Total Effects of Perceived Stress on the Effect of IPV on 30-Year CVD Risk

	Standardized				
	Effect	S.E	Est./S.E	p-value	95% CI
Perceived Stress	0.047	0.010	4.608	0.000	0.027, 0.067
Total indirect (IPV –CVD)	0.047	0.010	4.608	0.000	0.027, 0.067
Total direct(IPV to CVD)	0.103	0.041	2.531	0.011	0.023, 0.182
Total (IPV-CVD)	0.150	0.040	3.728	0.000	0.071, 0.229
	Unstandardized				
	Effect	S.E	Est./S.E	p-value	95% CI
Perceived Stress	0.003	0.001	4.541	0.073	0.002, 0.004,
Total indirect (IPV –CVD)	0.003	0.001	4.541	0.000	0.002, 0.004
Total direct(IPV to CVD)	0.006	0.002	2.535	0.011	0.001, 0.010
Total (IPV-CVD)	0.009	0.002	3.725	0.000	0.004, 0.013

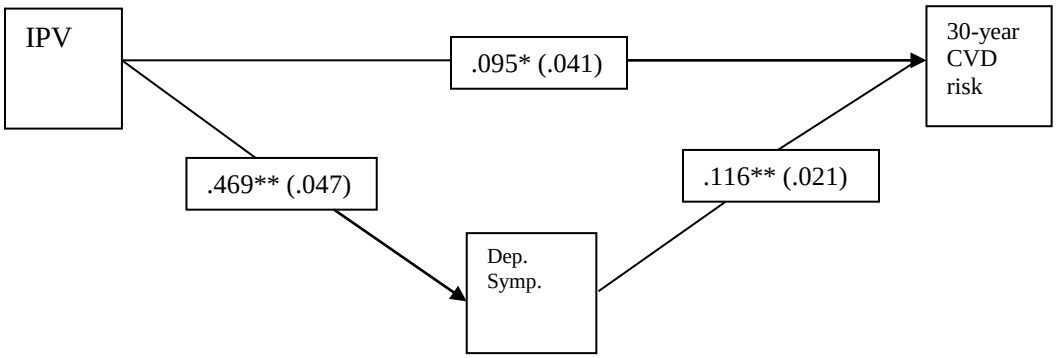
Depressive Symptoms

Figure 5a displays the unstandardized regression coefficients and Figure 5b 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=1.259, p<.01, Figure 5a) and depressive symptoms were also significantly associated with 30-year CVD risk score (B=0.002, p<.01, Figure 5a). Depressive symptoms partially mediate the relationship between IPV and 30-year CVD risk score as the relationship between IPV and 30-year CVD remains significant with depressive symptoms in the model.



*p<.05 **p<.01

Figure 5a: Unstandardized Regression Coefficients for Impact of Past Year IPV on 30-year CVD score through Depressive Symptoms



*p<.05 **p<.01

Figure 5b: Standardized Regression Coefficients for Impact of Past Year IPV on 30-year CVD score through Depressive Symptoms

Table 8 summarizes the standardized and unstandardized indirect, direct, and total effects of depressive symptoms on the effect of IPV on 30-Year CVD risk. The total standardized effect of IPV to CVD risk was 0.149 (p<.01, 95% CI 0.071, 0.228). The direct standardized effect of IPV to CVD was 0.095(p<.05, 95% CI 0.015, 0.175), leaving the specific standardized indirect effect of depressive symptoms to be 0.054 (p<.01, 95% CI 0.031, 0.078).

Table 8:

Standardized and Unstandardized Indirect, Direct, and Total Effects of Depressive Symptoms on the Effect of IPV on 30-Year CVD Risk

	Standardized				
	Effect	S.E	Est./S.E	p-value	95% CI
Depressive Symptoms	0.054	0.012	4.527	0.000	0.031, 0.078
Total indirect (IPV –CVD)	0.054	0.012	4.527	0.000	0.031, 0.078
Total direct(IPV to CVD)	0.095	0.041	2.327	0.020	0.015, 0.175
Total (IPV-CVD)	0.149	0.040	3.718	0.000	0.071, 0.228

	Unstandardized				
	Effect	S.E	Est./S.E	p-value	95% CI
Depressive Symptoms	0.003	0.001	4.473	0.000	0.002, 0.004
Total indirect (IPV –CVD)	0.003	0.001	4.473	0.000	0.002, 0.004
Total direct(IPV to CVD)	0.005	0.002	2.331	0.020	0.001, 0.010
Total (IPV-CVD)	0.008	0.002	3.716	0.000	0.004, 0.013

Alcohol Dependence

Figure 6a displays the unstandardized regression coefficients and Figure 6b 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 is not significantly associated with alcohol dependence and alcohol dependence is not significantly associated with 30-year CVD risk. Therefore, in this model, alcohol dependence does not mediate the relationship between IPV and 30-year CVD risk score.

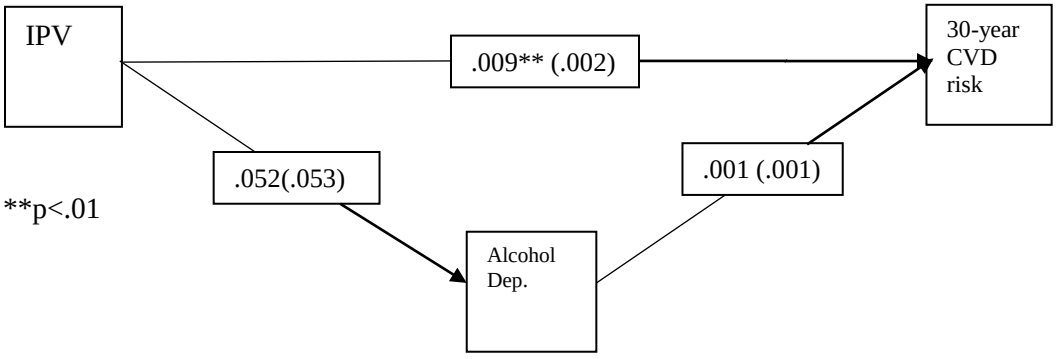


Figure 6a: Unstandardized Regression Coefficients for Impact of Past Year IPV on 30-year CVD score through Alcohol Dependence

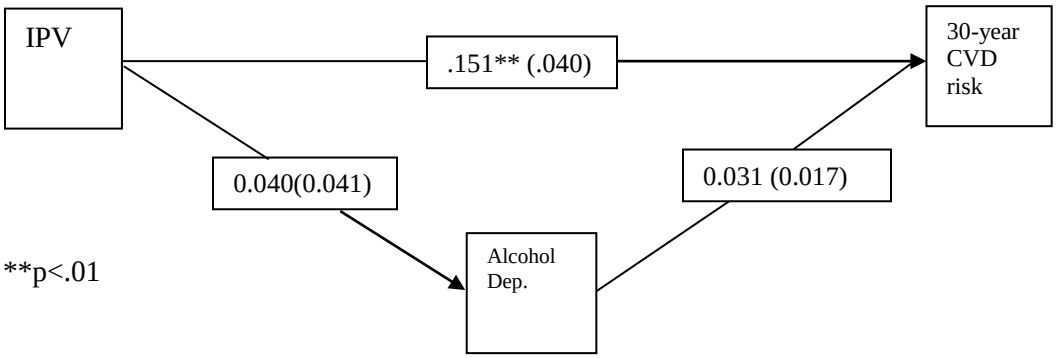


Figure 6b: Standardized Regression Coefficients for Impact of Past Year IPV on 30-year CVD score through Alcohol Dependence

Table 9 summarizes the standardized and unstandardized indirect, direct, and total effects of alcohol dependence on the effect of IPV on 30-Year CVD risk. The total standardized effect of IPV to CVD risk was 0.152 (p<.01, 95% CI 0.073, 0.230). The direct standardized effect of IPV to CVD was 0.150 (p<.01, 95% CI 0.072, 0.229), leaving the specific standardized indirect effect of alcohol dependence to be 0.001 (p=.392), however this indirect effect is insignificant.

Table 9

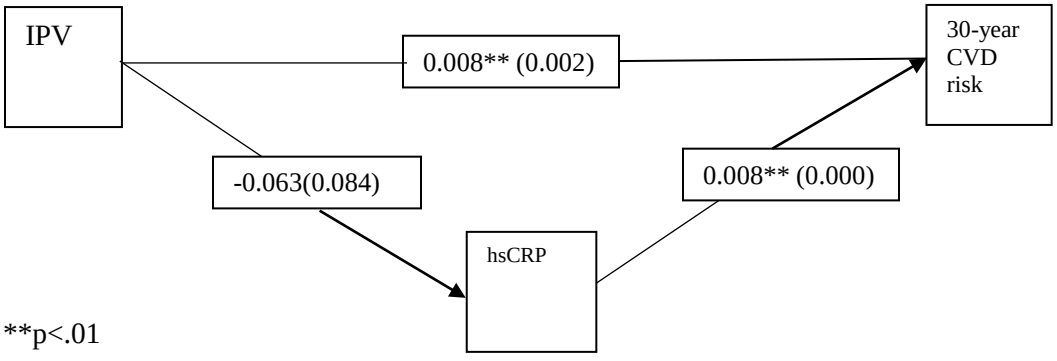
Standardized and Unstandardized Indirect, Direct, and Total Effects of Alcohol Dependence on the Effect of IPV on 30-Year CVD Risk

	Standardized				
	Effect	S.E	Est./S.E	p-value	95% C.I.
Alcohol Dependence	0.001	0.001	0.856	0.392	-0.002, 0.004
Total indirect (IPV –CVD)	0.001	0.001	0.856	0.392	-0.002, 0.004
Total direct(IPV to CVD)	0.150	0.040	3.745	0.000	0.072, 0.229
Total (IPV-CVD)	0.152	0.040	3.771	0.000	0.073, 0.230
	Unstandardized				

	Effect	S.E	Est./S.E	p-value	95% CI
Alcohol Dependence	0.00	0.000	0.854	0.393	0.000, 0.000
Total indirect (IPV –CVD)	0.000	0.000	0.854	0.393	0.000, 0.000
Total direct(IPV to CVD)	0.009	0.002	3.743	0.011	0.004, 0.013
Total (IPV-CVD)	0.009	0.002	3.767	0.000	0.004, 0.013

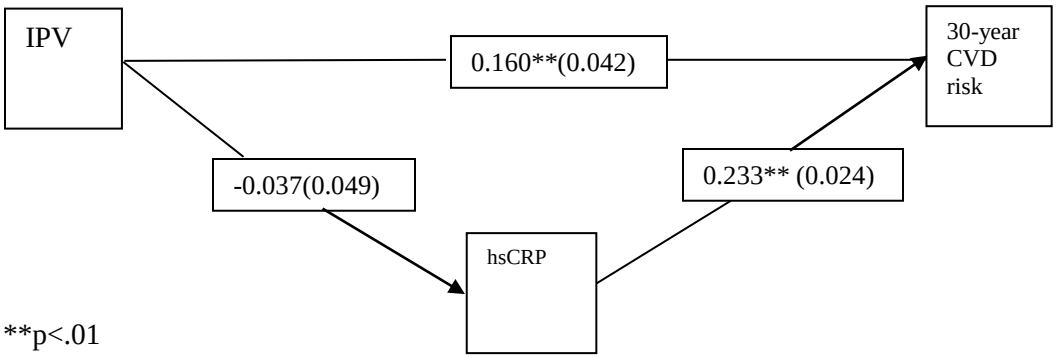
hsCRP Levels

Figure 7a displays the unstandardized regression coefficients and Figure 7b displays the standardized regression coefficients for the impact of past year IPV on 30-year CVD risk score through hsCRP levels. In this analysis, IPV is not significantly associated with hsCRP levels (Figure 7a) while hsCRP levels are significantly associated to 30-year CVD risk (B=.008, p<.01). hsCRP levels cannot be considered a true mediator due to the insignificant relationship between IPV and hsCRP levels. However, hsCRP levels will be considered in the multiple mediation model due to its significance with CVD.



**p<.01

Figure 7a: Unstandardized Regression Coefficients for Impact of Past Year IPV on 30-year CVD score through hsCRP Levels



**p<.01

Figure 7b: Standardized Regression Coefficients for Impact of Past Year IPV on 30-year CVD score through hsCRP Levels

Table 10 summarizes the standardized and unstandardized indirect, direct, and total effects of hsCRP levels on the effect of IPV on 30-Year CVD risk. The total standardized effect of IPV to CVD risk was 0.151 (p<.01, 95% CI 0.072, 0.230). The direct standardized effect of IPV to CVD was 0.160 (p<.01, 95% CI. 0.078, 0.241) leaving the specific standardized indirect effect of hsCRP to be -0.009, however this indirect effect is not significant in the model.

Table 10

Standardized and Unstandardized Indirect, Direct, and Total Effects of hsCRP Levels on the Effect of IPV on 30-Year CVD Risk

	Standardized				
	Effect	S.E	Est./S.E	p-value	95% CI
hsCRP	-0.009	0.012	-0.738	0.460	-0.032, 0.014
Total indirect (IPV -CVD)	-0.009	0.012	-0.738	0.460	-0.032, 0.014
Total direct(IPV to CVD)	0.160	0.042	3.835	0.000	0.078, 0.241
Total (IPV-CVD)	0.151	0.040	3.738	0.000	0.072, 0.230
	Unstandardized				

	Effect	S.E	Est./S.E	p-value	95% CI
hsCRP	0.000	0.001	-0.737	-0.737	-0.002, 0.001
Total indirect (IPV- CVD)	0.000	0.001	-0.737	-0.737	-0.002, 0.001
Total direct(IPV to CVD)	.009	0.002	3.825	0.000	0.004, 0.014
Total (IPV-CVD)	0.009	0.002	3.738	0.000	0.004, 0.013

Multiple Mediation Analyses

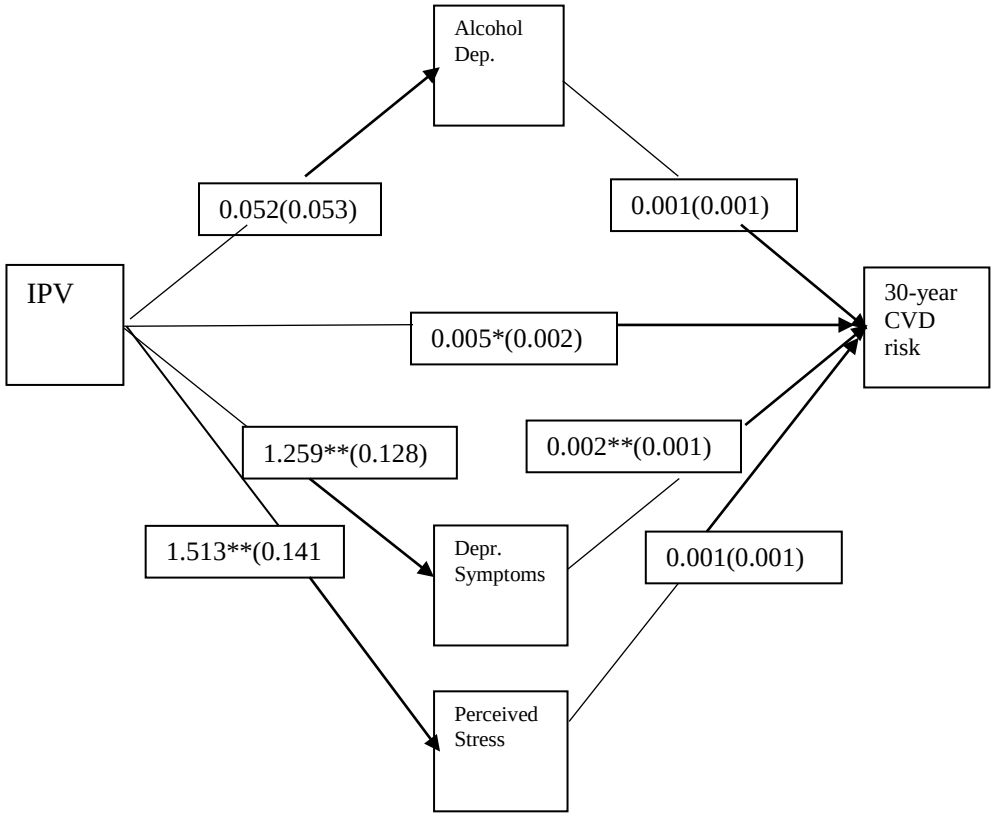
We construed multiple mediation models based on the evidence from the literature that identified specific coping mechanisms and responses to trauma could mediate the relationship between IPV and CVD risk.

Multiple Mediation Model 1: Perceived Stress, Depressive Symptoms, and Alcohol Dependence

Regression coefficients

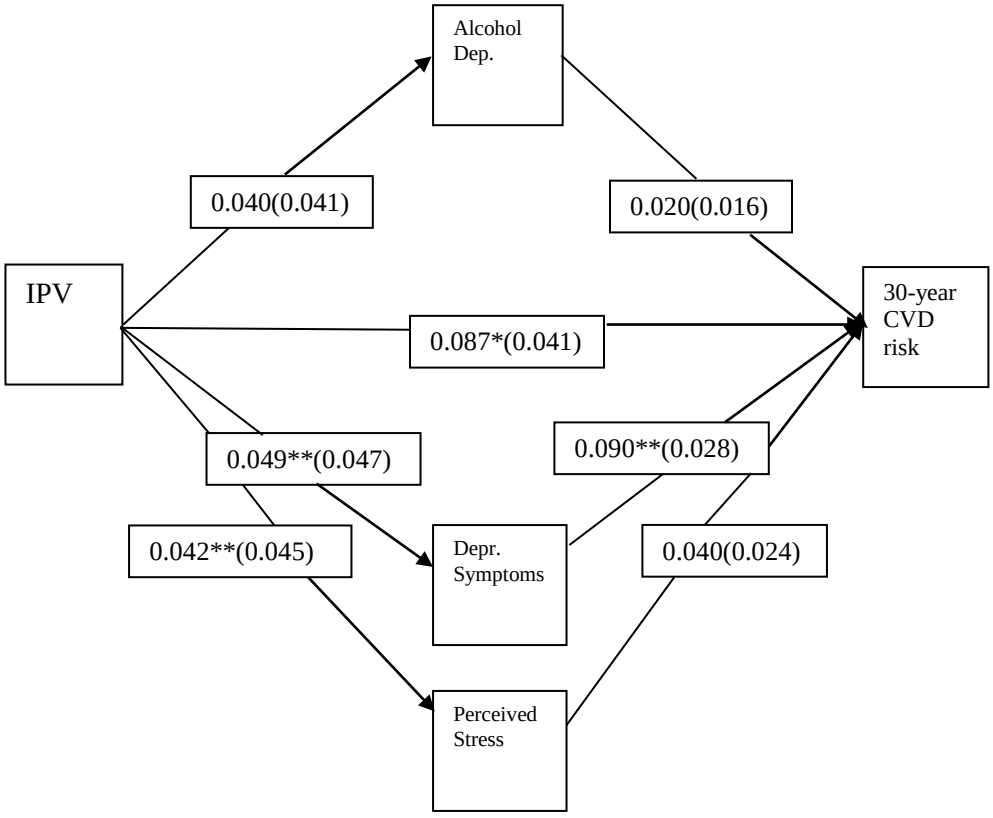
Figures 8a and 8b display the unstandardized and standardized regression coefficients for the Aim 2 multiple mediation model including perceived stress, depressive symptoms, and alcohol dependence. While alcohol dependence was not significant in the simple mediation model, it was included into the multiple mediation model based on the literature that identifies heavy alcohol use as a possible coping mechanism of IPV (Ashare et al., 2011; Ullman & Sigurvinsdottir, 2015). When all three mediators are included in a mediation model, partial mediation occurs through only depressive symptoms as the relationship between IPV and 30-year CVD risk remains significant ($p<.05$). A one standard deviation increase in past year IPV is associated with

a 0.049 (p<.01) standard deviation increase in depressive symptoms while a one-unit increase in depressive symptoms is associated with a 0.090 (p<.01) standard deviation increase in 30-year CVD risk score.



**p<.01

Figure 8a: Unstandardized Regression Coefficients for Impact of Past Year IPV on 30-year CVD score through Perceived Stress, Depressive Symptoms, and Alcohol Dependence



**p<.01

Figure 8b: Standardized Regression Coefficients for Impact of Past Year IPV on 30-year CVD score through Perceived Stress, Depressive Symptoms, and Alcohol Dependence

Indirect and Direct Effects

Table 11 shows the standardized and unstandardized indirect, direct, and total effects of perceived stress, depressive symptoms, and alcohol dependence on the effect of IPV on 30-Year CVD Risk score. The standardized specific indirect effect of the significant mediator, depressive symptoms, is 0.042 (p<.01, 95% CI .012, .070). Perceived stress and alcohol dependence were not significant in this model, thus depressive symptoms have the largest and most significant impact on the relationship between IPV and 30-year CVD risk among these mediators. The total indirect effect of

IPV on 30-year CVD risk score was 0.063 and was statistically significant (p<.01, 95% CI .039, .087). The direct effect of IPV to CVD was .087 with a p-value of 0.032 (95% CI .007, .167), thus, depressive symptoms is considered to partially mediate the relationship between IPV and 30-year CVD risk score dependent of perceived stress and alcohol dependence. Perceived stress was a mediator of IPV and CVD in the simple mediation model (Figure 3a and 3b), but insignificant in the multiple mediation model.

Table 11

Standardized and Unstandardized Indirect, Direct, and Total Effects of Perceived Stress, Depressive Symptoms, and Alcohol Dependence Levels on the Effect of IPV on 30-Year CVD Risk

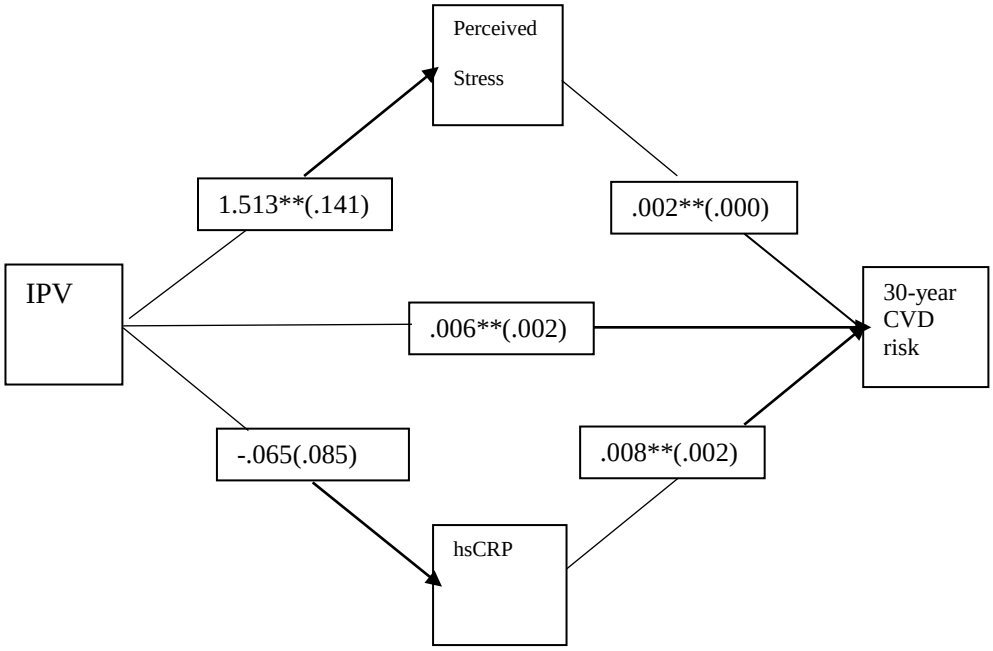
	Standardized				
	Effect	S.E	Est./S.E	p-value	95% CI
Perceived Stress	0.020	0.012	1.637	0.102	-0.004, 0.043
Depressive Symptoms	0.042	0.014	2.994	0.003	0.013, 0.070
Alcohol Dependence	0.001	0.001	0.771	0.440	-0.001, 0.003
Total indirect (IPV – CVD)	0.063	0.012	5.148	0.000	0.039, 0.087
Total direct(IPV to CVD)	0.087	0.041	2.142	0.032	0.007, 0.167
Total (IPV-CVD)	0.150	0.040	3.719	0.000	0.071, 0.229
	Unstandardized				
	Effect	S.E	Est./S.E	p-value	95% CI

Perceived Stress	0.001	0.001	1.635	0.102	0.000, 0.002
Depressive Symptoms	0.002	0.001	2.982	0.003	0.001, 0.004
Alcohol Dependence	0.000	0.000	0.770	0.440	0.000, 0.000
Total indirect (IPV – CVD)	0.004	0.001	5.078	0.000	0.002, 0.005
Total direct(IPV to CVD)	0.005	0.002	2.145	0.032	0.000, 0.009
Total (IPV-CVD)	0.008	0.002	3.717	0.000	0.004, 0.013

Multiple Mediation Model 2: Perceived Stress and hs CRP Levels

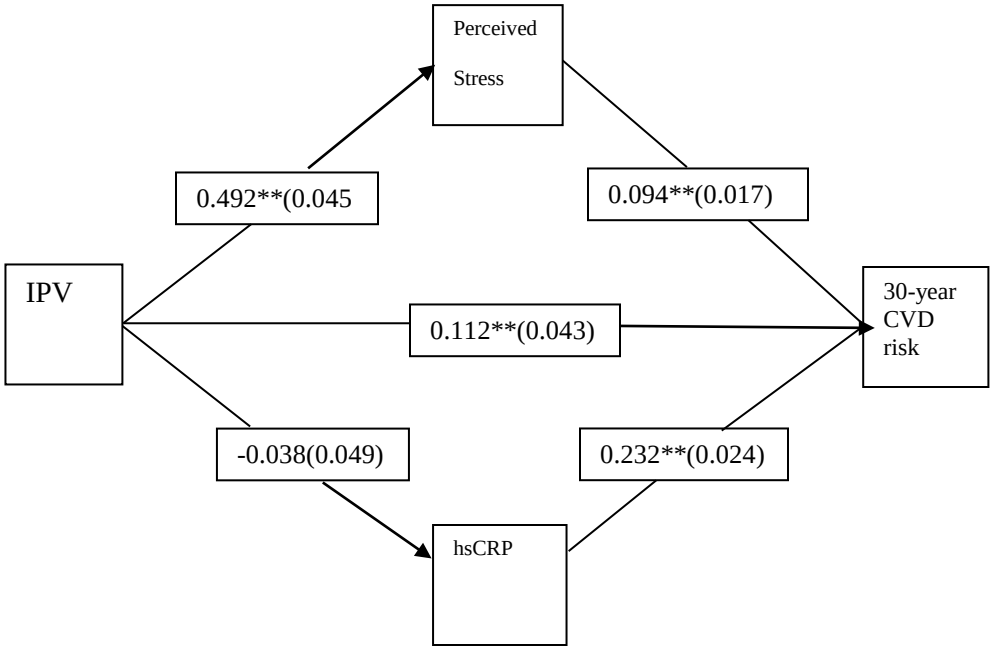
Regression Coefficients

Figures 9a and 9b display the unstandardized and standardized regression coefficients for the multiple mediation model including perceived stress and hsCRP levels. With these two mediators in the model, partial mediation occurs through perceived stress as the relationship between IPV and 30-year CVD risk score remains significant ($p<.01$). A one standard deviation increase in past year IPV is associated with a 0.492 ($p<.01$) standard deviation increase in perceived stress level while a one standard deviation increase in perceived stress level is associated with a 0.094 ($p<.01$) standard deviation increase in 30-year CVD risk score. While hsCRP levels are not significantly associated with IPV, hsCRP levels are significantly associated with an increased CVD risk score.



**p<.01

Figure 9a. Unstandardized Regression Coefficients for Impact of Past Year IPV on 30-year CVD score through Perceived Stress and hsCRP levels



**p<.01

Figure 9b: Standardized Regression Coefficients for Impact of Past Year IPV on 30-year CVD score through Perceived Stress and hsCRP levels

Indirect and Direct Effects

Table 12 displays the standardized and unstandardized indirect, direct, and total effects of perceived stress and hsCRP levels on the effect of IPV on 30-Year CVD risk score. The standardized specific indirect effect of the significant mediator, perceived stress, is 0.046 (p<.01, 95% CI 0.027, 0.066). The indirect effect of hsCRP was statistically insignificant. The direct effect of IPV on 30-year CVD risk score was 0.112 (p<.01, 95% CI 0.028, 0.196), thus, perceived stress is considered to partially mediate the relationship between IPV to 30-year CVD risk of hsCRP.

Table 12

Standardized and Unstandardized Indirect, Direct, and Total Effects of Perceived Stress and hsCRP Levels on the Effect of IPV on 30-Year CVD Risk

	Standardized				
	Effect	S.E	Est./S.E	p-value	95% CI
Perceived Stress	0.046	0.010	4.685	0.000	0.027, 0.066
hsCRP	-0.009	0.012	-0.752	0.452	-0.039, 0.014
Total indirect (IPV – CVD)	0.038	0.017	2.189	0.029	0.004, 0.071
Total direct(IPV to CVD)	0.112	0.043	2.626	0.009	0.028, 0.196
Total (IPV-CVD)	0.150	0.040	3.727	0.000	0.071, 0.229
	Unstandardized				
	Effect	S.E	Est./S.E	p-value	95% CI
Perceived	0.003	0.001	4.631	0.000	0.002, 0.004

Stress					
hsCRP	0.000	0.001	-0.751	0.453	-0.002, 0.004
Total indirect (IPV – CVD)	0.002	0.001	2.188	0.029	0.000, 0.004
Total direct(IPV to CVD)	0.006	0.002	2.626	0.009	0.002, 0.011
Total (IPV-CVD)	0.008	0.002	3.726	0.000	.004, .013

CHAPTER 5
DISCUSSION

The purpose of this study was to examine the relationship between past year intimate partner violence (IPV) and 30-year cardiovascular disease (CVD) risk score and to test possible mediating factors using a nationally representative sample of young adult women in the United States. The results of this study suggested that past year intimate partner violence might have a relatively small, but statistically significant effect on 30-year CVD risk score. However, this effect became insignificant when other important covariates were introduced, highlighting the complexity of IPV, its associated outcomes, and co-occurring phenomena.

The mediation analyses demonstrated that of two of the four proposed mediators, perceived stress and depressive symptoms were significant partial mediators in simple mediations. However, perceived stress became insignificant once depressive symptoms were introduced into a multiple mediation model. Alcohol dependence and hsCRP levels showed no evidence of mediation.

This chapter discusses the main findings of the study as well as strengths and limitations. Implications for nursing practice, future research, and policy will conclude this chapter.

Principal Findings

In our sample of women between 24 and 32 years old, 15% reported any past year IPV, which is lower than other nationally representative samples (Breiding et al., 2014). However, the highest risk group for IPV are generally women ages 18-24 years, which may explain the lower proportion among Add Health participants (Breiding et al., 2014).

Additionally, emotional victimization was not assessed for in this current study. Self-disclosure of IPV is often underreported as well (Ruiz-Pérez, Plazaola-Castaño, & Vives-Cases, 2007).

In terms of other important demographics, a substantial proportion of the women in this study were categorized as a sexual minority. There were high rates of stress levels and depressive symptoms among the sample. The mean 30-year CVD risk score of the sample was 8.2%, which is comparable to 30-year CVD risk scores in similar populations (Clark et al., 2016). Previous literature in this age group has noted that a 30-year risk score of 20% is high risk in a young adult population (Clark et al., 2016). Any increases in risk score at this young age should be noted and considered.

Past Year IPV and 30-year CVD risk

Several demographic variables showed significant associations with experiencing past year IPV including race, not having a college degree, lower household income, financial stress, history of childhood abuse, and being categorized as a sexual minority. Previous IPV research has found that these variables are associated with IPV victimization (Breiding et al., 2014; Cunradi, Caetano, & Schafer, 2002; Cerulli et al., 2010; Flicker et al., 2011; Fox & Benson, 2006; Manchikanti Gómez, 2011). No demographic variables showed significant effect sizes (<.20).

However, two of the proposed mediators in this study, perceived stress and depressive symptoms, both showed significant effect sizes (>.20) in relation to past year IPV. Stress-related mental health outcomes, including PTSD, have been thoroughly researched as outcomes of IPV (Ahmed & McCaw, 2010; Sullivan et al., 2016).

Similarly, depression is not only higher among women in general, but has been linked to IPV victimization (Chuang et al., 2012; Connelly et al., 2013; Devries et al. 2013). In this study, alcohol dependence was not significantly related to IPV although alcohol has been previously found to be a coping mechanism for IPV (Overup et al., 2015; Sugg, 2015; Sullivan et al., 2016). The low rates of alcohol dependence in this analysis may have contributed to this outcome. In contrast to the literature, those who did not experience past year IPV had significantly higher mean hsCRP levels compared to those who did experience IPV (Newton et al., 2011). The lower levels of hsCRP among those with past year IPV may be a result of the measurement of IPV in this study. The questions were unable to capture the larger context of violence and a longer history of coercive controlling abuse that may increase this chronic stress and inflammation response (Danese et al., 2009; Kelly & Johnson, 2008; McEwan & Seeman, 2009).

In the chi-square analysis, those who reported past year IPV had almost a 1% higher mean 30-year CVD risk score compared to those without past year IPV (9.6% vs 8.7%). Although the effect size was small, this difference in risk score should be highlighted. A bivariate regression model revealed that those who reported past year IPV had a slight, but statistically significant, increase in 30-year CVD risk score. Other research that has examined CVD risk score among IPV victimization in this age group also found small, but statistically significant increases (Clark et al, 2016). However, this is the first study to examine the impact of IPV on 30-year CVD risk score in a sample of only women. Women tend to have a lower 30-year CVD risk score compared to men, with men in this age group reporting a mean score of 17% (Clark et al., 2016). Thus, this

small increase in 30-year CVD risk score at a young age may be important to consider in the larger context of IPV and long-term health for women. The difference in 30-year CVD risk score for women exposed to IPV and not exposed to IPV may only grow due to older age, continued victimization due to lack of intervention or support, and longer use of mal-adaptive CVD-related coping mechanisms.

The multivariate model assessing the impact of past year IPV on 30-year CVD risk revealed that past year IPV becomes insignificant when its covariates are introduced. Many of these covariates significantly increased in 30-year CVD risk, including lower self-reported health status, lower income, experiencing financial stress, and identifying as a sexual minority woman (SMW). SMW have previously been found to report worse mental health outcomes such as increased rates of stress and depression as well as higher rates of smoking and alcohol abuse compared to heterosexual women (Burgard, Cochran, & Mays, 2005; Caceres, Brody, & Chyun, 2016; Diamont & Wold, 2003; Matthews et al., 2002; Przedworski, McAlpine, Karaca-Mandic, & VanKim, 2014; Ryan, Wortley, Easton, Pederson, & Greenwood, 2001; Steele et al., 2017). These outcomes may also increase CVD risk. However, research has been limited on CVD risk among young SMW (Caceres et al., 2016). Thus this novel finding should be further explored. Having a college degree and heaving health insurance were significantly associated with a decrease in 30-year CVD risk score. Being African American, while associated with past year IPV in the chi-square analysis, was not associated with 30-year CVD risk score in this regression model.

These findings further highlight the complexity of IPV. As previously stated, these significant covariates in the regression model often co-occur in the context of IPV, especially among women who may experience economic abuse or social isolation. Women who experience IPV may also experience difficulty keeping steady employment because of their abuser, becoming economically dependent on their abuser, and many other social problems that could increase risk factors related to cardiovascular disease (Sanders, 2015). Financial stress, lower income and educational attainment, and lack of health insurance have shown to have deleterious effects on health (Marmot & Allen, 2014: McWilliams, 2009; Pickett & Wilkinson, 2015; Schaller & Stevens, 2015; Yen & Moss, 1999). Thus, these finding suggest that IPV alone may have an impact on CVD risk, but its co-occurring challenges may further strengthen that impact.

Severity of IPV and 30-year CVD Risk

Of those in the sample who reported past year IPV, 36% reported experiencing high severity IPV. Those who had high severity IPV were more likely to not have a college degree, report greater depressive symptoms, have higher hsCRP levels, and a higher 30-year CVD risk score. Severe IPV has been found to lead to worse mental and physical health outcomes (Kelly & Johnson, 2008; Mason et al., 2012). By separating IPV by severity, we were able to provide more depth regarding the intensity of IPV victimization which may explain the increase in hsCRP levels in this group compared to the findings with the general IPV sample. The physiologic stress response framework can also help explain why more severe violence may cause a more prominent physiologic response in hsCRP levels as this severe violence may trigger a more constant physiologic

response. However, due to the inability to measure emotional abuse and coercive control, the overall severity of violence may have been underestimated thus impacting the findings.

The bivariate regression model of 30-year CVD risk on severity of IPV was insignificant suggesting that the severity levels assessed in this study were not strong enough to impact 30-year CVD risk score. While previous literature has documented increases in hypertension among older women experiencing severe emotional abuse, this is the first study that examined 30-year CVD risk among young women experiencing both high and low severity IPV (Mason et al., 2012). The multivariate regression model revealed that among women who experienced IPV, not having a college degree and increased depressive symptoms were associated with an increase in 30-year CVD risk score. These findings support previous findings that educational attainment and depressive symptoms can impact one's physical health.

Mediation Models

Simple Mediations

The simple mediation models revealed that perceived stress and depressive symptoms were independent partial mediators of the relationship between intimate partner violence 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 cardiovascular disease (Chuang et al., 2012: Connelly et al., 2013; Devries et al., 2013; Kendall-Tackett, 2007; Martinez-Toteya et al., 2009; Sabri et al., 2013). These findings also reflect the

Transactional Model of Stress and Coping, which theorizes that stress and either the ability or inability to cope with stress can 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 cardiovascular disease, alcohol dependence was not significant in any of the mediation models (Ullman & Sigurvinsdottir, 2015; Witteman et al., 1990). Low rates of alcohol dependence in the sample may have contributed to these insignificant findings. While hsCRP levels were significantly related to 30-year CVD risk score in a simple mediation model, hsCRP levels were not significantly related to past year IPV, thus was not considered a mediator. As previously mentioned, the past year IPV variable may have been unable to capture the chronic stress of experiencing abuse for longer periods of time.

Multiple Mediation

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. According to the Transactional Model of Stress and Coping, the perception of stress can impact how one implements a specific coping mechanism (Lazarus & Folkman, 1984). Both the coping mechanism and the effectiveness of coping contribute to outcomes caused by the stressor. 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.

Findings from this multiple mediation model confirm the preliminary analysis that suggests 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, Mondal, Bandhu, Kataria, & Gandhi, 2016; Lett et al., 2004; Lichtman et al., 2008;Whooley & 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 & 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 (Asahre et al., 2011; Matheson et al., 2015; Newton et al., 2011; Pico-Alfonso, Garcia-Linares, Celda-Navarro, Herbert, & Martinez, 2004). The relationship between IPV, depression, and CVD risk should be further explored.

The multiple mediation model including perceived stress and hsCRP levels demonstrated that the perception of stress is an important factor in the relationship between IPV and 30-year CVD risk score. Both the Transactional Model of Stress and Coping framework and the stress-response framework can support this finding. An

increase in the perception of stress may mediate the negative health outcome of experiencing a stressor (Lazarus & Folkman, 1984). While elevated hsCRP levels would be expected with increased perceived stress levels, in accordance with the physiological stress framework, hsCRP levels were not significant in this model.

As previously stated, the past IPV variable used in this analysis did not collect information on emotional abuse or chronicity of IPV, both of which can often coincide with increased stress and poorer health outcomes (Kelly & Johnson, 2008; Mason et al., 2012). While hsCRP levels can be used to measure chronic inflammation as a proxy for chronic stress, cortisol levels may have provided a better picture of the stress response due to the cross sectional nature of the study (Young, Tolman, Witkowski, & Kaplan, 2004). Elevated cortisol levels occur during times of increased stress or when the body in unable to adapt to stress (McEwan & Seeman, 2009). Elevated cortisol levels have also been associated with recent IPV exposure compared to those who experienced IPV in the past (Pico-Alfonso et al., 2004; Yong et al., 2004). Since increased cortisol levels have been linked with depression as well, the significant impact of depression on CVD risk in this sample may have may have better reflected in cortisol levels than hsCRP (Goodyer, Herbert, Tamplin, & Altham, 2000; Herbert, 2003; Tse & Bond, 2004).

This study had many strengths including the ability to detect a small change in CVD risk in a relatively young sample of women. To our knowledge, this is the first study to examine IPV and 30-year CVD risk score among a sample of solely young women. This is also 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.

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. Lastly, the cross-sectional nature of this study does not allow casualty to be determined.

Implications

This study revealed a small, but statistically significant increase in 30-year CVD risk score among young women who experienced IPV in the past year. As these women age and if they continue to experience IPV, their CVD risk may only increase over time. CVD risk is already growing among young women in the U.S.; these findings highlight the need to examine CVD risk factors among women in this age group who experience IPV (Mozaffarian, et al., 2016). This study also supported the theory that perception of stress in the context of IPV and the effectiveness of coping with the stress can affect both mental and physical health, specifically measured by depression and CVD risk.

Nursing Considerations when Working with IPV Survivors

Intimate partner violence is a complex phenomenon and nursing professionals must acknowledge these complexities when working with this population. Safety is often

a top priority among those experiencing partner violence, even long after an abusive relationship has ended. Those in abusive relationships often know the best actions that will keep them safe and those actions may evolve and change over time. Actions to keep themselves safe may be to leave a relationship or to stay in a relationship. Attempting to leave the relationship or leaving the relationship increases risks for violence and may not always be a safe or advisable plan (Campbell et al., 2003). If someone decides to leave an abusive relationship, nurses caring for them must acknowledge that leaving is an evolving process and women may leave and return multiple times before they are able to leave permanently. As nurses, we must collaborate with IPV survivors to create plans on how to keep them both safe and healthy. As IPV relationships may evolve over time, our plans of care with this population should evolve to meet the needs our patients.

Nursing Practice

Nurses are seen as the front line in health care and have the ability to create trusting relationships with our patients. This trust can allow for safe dialogue between nurses and patients on sensitive issue such as IPV victimization. Not only should nurses participate in screening for intimate partner violence among women of childbearing age as recommended by the U.S. Preventive Services Task Force, but nurses are also responsible for discussing heart health with young women and assessing their individual CVD risk (Nelson, Bougatsos, & Blazina, 2012). Our findings suggest that screening for depression among young women, especially those who experience IPV, could be an important intervention point in preventing the development of cardiovascular disease.

Trauma-informed care should be incorporated into care planning for all patients, but especially when IPV victimization and mental health issues, such as depression, are discussed. Trauma-informed care is a universal service delivery approach that acknowledges and understands the impact of trauma, emphasizes the safety of both survivors and provider, and allows for survivors to regain a sense of control and empowerment (Hopper, Bassuk, & Olivet, 2010). Trauma-informed care also attempts to avoid actions or process that could be re-traumatizing to an individual (Hopper et al., 2010). Nurses should also be trained on how to respond to an IPV disclosure and be kept up to date on tangible referrals and options for those who experience IPV in their specific geographical area. The findings of the current study suggest that nurses who see young women with increased CVD risk should also screen for IPV using a trauma informed care approach.

Future Research

IPV research should focus on CVD risk and overall heart health as important health related outcomes in this population. Since women have been historically under-sampled in CVD research, more CVD research is also needed that focuses on women and heart health, and, more specifically, on the development of CVD risk among young women (Mosca et al., 2011).

IPV research should further examine the biological response to IPV by including cortisol levels when working with women recently exposed to IPV. Research should also employ IPV measures that provide the context of the violence and the victim's perception of the violence. Including emotional IPV and coercive control as well as more qualitative

measures such as fear and self-esteem in an abusive relationship may strengthen our understanding of the connection between IPV and the stress and coping response.

Future research should examine the longitudinal impact of chronic IPV victimization on the biological stress response systems in relation to CVD risk. Measuring IPV longitudinally will also allow for causality to be determined between IPV, perceived stress, subsequent depression, and increased CVD risk over time.

Future intervention research should also examine the impact of physical activity intervention among young women who have experienced IPV. Not only can physical activity decrease CVD risk, but also it can improve self-esteem and reduce social isolation and stress (Eime, Young, Harvey, Charity, & Payne, 2013; Penedo &Dahn, 2005; Vankim & Nelson, 2013; Warburton, Nicol, & Bredin, 2006). Physical activity also can improve depressive symptoms (Eime et al., 2013; Hiles, Lamers, Milaneschi, & Pennix, 2017; Lee & Kim, 2010; Pasco et al., 2011; Penedo & Dahn, 2005; Warburton et al., 2006). Exercise has been found to increase both serotonin and brain-derived neurotrophic factor (BDNF) and decrease depressive behavior (Masrais, Stein, & Daniels, 2009; Neeper, Gomez-Pinilla, Choi, & Cotman, 1996; Whiteman et al., 2014). BDNF and serotonin activate signal pathways and transportation factors that help to regulate stress resistance, cell survival, and neural plasticity, all of which help improve brain function (Maraus et al., 2009).

Lastly, research examining the effects of IPV should include and specifically analyze sexual minority women as they have been largely excluded from IPV research (Simpson & Helfrich, 2014). SMW have been found to experience high rates of IPV as

well as poorer health outcomes compared to their heterosexual counterparts (Black et al., 2014; McCauley et al., 2015). With these increased IPV rates, unique institutional and societal challenges, this population and their risk for CVD and other stress-related health issues should be further examined (Caceres et al., 2016).

Policy Implications

The findings of this study reveal the importance of providing primary, secondary, and tertiary care to survivors of IPV. Policy that provides resources in this area is greatly needed, as the need for more resources, including space in domestic violence shelters, is growing. Continued funding for the Violence Against Women Act (VAWA), should be a priority. VAWA, enacted in 1994, was the first federally funded legislation to declare domestic violence, sexual assault, stalking, and dating violence as crimes (The United States Department of Justice [DOJ], 2017). Resources provided by VAWA aim to empower communities to respond and combat violence (DOJ, 2017). VAWA supports domestic violence shelters, rape crisis centers, legal assistance programs, training for law enforcement, and other relevant legislation (DOJ, 2017). Re-authoring this legislation will continue to strengthen communities ability to in support safe and healthy relationships. Without this legislation, funding for services, such as domestic violence shelters, could reach critically low levels.

Affordable health insurance can increase this population's access to treatment for many of the acute and chronic health conditions associated with IPV. Supportive, accessible, and affordable mental health services and mental health screenings are also a necessity. Screening and counseling for IPV should be included as a free preventative

service under both public and private health insurance. Under the Affordable Care Act (ACA), screening for IPV is covered as a preventative health service and insurance companies are prohibited from denying coverage to IPV survivors on the basis of a pre-existing condition (United States Department of Health and Human services [HHS], 2013). Recent survivors of domestic violence are also exempt from paying a penalty if they cannot afford insurance (HHS, 2013). These tenets of the ACA can greatly affect the overall well-being of IPV survivors and should be part of any future health insurance legislation.

Governmental policies should also provide funding and support for community and public health nurses to make frequent visits to domestic violence shelters in order to assess the health needs of the residents. These visits would allow for those staying in shelter to talk about their health issues within the context of IPV in a space that is safe and supportive. Visiting nurses can also provide health education on the various health risks associated with IPV victimization as well as screen for mental health issues and mal-adaptive coping.

Conclusion

Both IPV and CVD remain prominent health issues in the United States. There is growing evidence that exposure to IPV can increase CVD risk, even among young adult women. The physiological stress response and individual coping effectiveness may play important roles in mediating the relationship between IPV and CVD risk through mental health outcomes such as depression. 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, we must continue to explore this connection and develop appropriate interventions.

APPENDIX: Table of Evidence

	Finding	IPV measure	CVD Measure	N	Sample	Author
IPV and blood pressure	IPV victimization was not significantly associated with BP among women. Men who reported both severe victimization and perpetration of IPV had 59% higher odds of HTN compared to men who had never experienced IPV exposure.	IPV victimization and perpetration measured from questions derived from Revised Conflict Tactic Scales. Did not include emotional abuse. Victimization score was compiled to help measure severity. IPV was assessed in relationships that occurred during an 8 year time frame.	Blood Pressure and Hypertension. BP taken 3 times with 30 second intervals, HTN defined at SBP>140, BDP > 90 or antihypertensive medication being taken	n=9,157 men and women	Wave 3 and Wave 4 of Add Health, nationally representative sample of young adults in the U.S. with 46% of females reporting Ivy exposure. No victimization associated with less financial distress.Mean age of sample at Wave 3 was 21 years.	Clark et al., 2014

IPV and blood pressure	Physical and sexual abuse were not associated with hypertension. Women experiencing extreme emotional abuse had 24% increased rate of hypertension compared with women who had no emotional abuse	Women's Experience of Battering Scale and Violence Questionnaire derived from CFT2. Severity and frequency of physical and sexual violence was not included in analysis, these experiences were coded as "yes" or "no". IPV was assessed in relationships that were ongoing in 2001.	Hypertension was self reported on biennial questionnaire	n=51,434 women from Nurses Health Study II	Mean age in 2001 was 45.6-47. Most of sample was white. Child abuse was strongly correlated with IPV. 22% of sample reported physical, 10% sexual, and 1.2% scored serious abuse on WEB	Mason et al., 2012
	Finding	IPV measure	CVD Measure	N	Sample	Author
IPV CVD risk	Veteran women more likely than non-veteran women to report IPV victimization. IPV exposure associated with depression, smoking and heavy drinking. No association with IPV and exercise or weight after controlling for veteran status and demographics	BFRSS: Lifetime IPV victimization reported ever experiencing actual or threatened physical violence, or unwanted sex, from an intimate partner.	CVD Risk factors: depression (>10 on Patient Health Questionnaire Scale), current smoking, binge or heavy drinking, lack of exercise (no regular exercise in past 30 days) and being overweight or obese	n=21,162 women	Veteran women more likely to be non-White or Hispanic. 50% of both groups has annual income less than $50,000 and more than 60% had not graduated college. 62.1% of veterans were under the age of 45 while 50% non-veterans were <45.	Dichter, Cerulli, Boassarte, 2011

IPV and CVD risks	Severity of IPV and smoking behaviors were not statistically significant in explaining CVD symptoms. Positive association between severity of past abuse and smoking.	Severity of IPV assessed using Index of Spouse Abuse. Relationships were assessed from women who had separated from abusive partner 3 months to 3 years	Smoking status: do you currently smoke? How many cigarettes do you smoke in a day. CVD risk: partner abuse symptoms scale BMI, BP readings, self-report CVD diagnosis and self-report use of CVD medications. CVD symptoms measured: 4-item cardio-respiratory symptoms scale of the PASS	n=309 women from the Women's Health Effects Study	Mean age 39 years. 44.1% were smokers, 53.2% were overweight or obese, 54.7 had BP above normal range, 50.8% reported CVD symptoms	Scott-Storey, Wuest, & Ford-Gilboe, 2009
	Finding	IPV measure	CVD Measure	N	Sample	Author

IPV and CVD risks	Women who reported physical and/or sexual IPV and psychological IPV alone more likely to be smokers than women with no IPV exposure. Physical and/or sexual violence was associated with abdominal obesity, low HDL and elevated triglycerides and more likely to receive anti-hypertensives, Women who reported IPV had slightly higher risk score	Lifetime IPV: Have you ever been systematically intimidated, degraded, or humiliated over a long period of time? 2. Have you ever experienced threats to harm you or someone close to you? 3. Have you ever been physically attacked/abused? 4. Have you ever been forced into sexual activities? 5. Has anyone ever raped you or tried to rape you? All questions were followed by questions identifying perpetrator. Women were flagged for IPV if they identified their partner as the perpetrator.	Drug prescription filing used for cardiovascular drug use. CVD risk used Framingham 10-year risk calculator: age, sex, DM, smoking, SBP, total cholesterol and HDL	n=5593 women without CVD at baseline. 13.4% had IPV experience	Sample taken from population based cohort of the Olso Health Study and prescription records from Norwegian Prescription Database. 13.4% of women reported lifetime IPV, 7.4% reported physical and/or sexual, and 6% reported psychological alone. Women ages 30-60 years	Stene et al., 2013
	Finding	IPV measure	CVD Measure	N	Sample	Author

IPV and CVD risks	11 biomarkers were examined in relation to women experiencing IPV with ACS to test a psychological and biological pathway of IPV to chronic illness. A moderate effect size was found for vascular cell adhesion molecule-1	Lifetime Trauma and Victimization History Questionnaire	Neuroendocrine Biomarkers, Proinflammatory cytokines, cell adhesion molecules and chemotactic cytokine	n=45 women	Sample taken from women hospitalized at urban care facilities and diagnosed with Acute Coronary Syndrome (ACS). Average age 57 years and 75% African American	Symes et al., 2010
IPV and CVD risks	Cardiovascular risks did not statistically differ between women who experience IPV and women who did not	Respondents self-reported history of or on-going physical assault by intimate partner	health care providers diagnosis of diabetes, hypertension or obesity	n=329	Data from the HIV Risk Among Homeless Women Study. Women from homeless shelters in NYC. 31.6% cardiovascular symptoms, 73.5% had health insurance. 50% were current smokers. Mean age 37.9	Vijayaraghavan et al., 2011

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