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Influence of Intimate Partner Violence (IPV) Exposure on Cardiovascular and Salivary Biosensors: Is There a Relationship?

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Abstract

Background/purpose: Intimate partner violence (IPV) is a global public health epidemic that initiates/exacerbates health consequences affecting a victim's lifespan. IPV can significantly predispose women to a lifetime risk of developing cardiovascular disease (CVD) due to the effects

of stress and inflammation. This study investigates the correlation among IPV exposure, in-vivo CVD events, and inflammatory biomarkers as predictor indices(s) for CVD in female dental patients.

Methods: Of 37 women enrolled in this study, 19 were African-American (AA) and 18 non-African-American (non-AA) and their ages ranged from 19 to 63 years. IPV-exposure and stress-induced in-vivo CVD events such as Chest Pain (CP) and Heart palpitations were recorded from all enrolled subjects. Cardiovascular events were obtained through surveys by patient self-report. Saliva specimens were obtained from all women and were analyzed for CVD biomarkers using multiplex-ELISA.

Results: The prevalence of IPV was 51% (19/37) and statistically equivalent for AA and non-AA. The results show differences in experience of **1**) CP ($p < 0.01$) and **2**) heart palpitations ($p < 0.02$) when IPV + participants are compared with IPV-AA and non-AA cohorts. Of 10 CVD biomarkers analyzed, significant correlations between IPV+ and IPV- subjects were observed for biomarkers that include Interleukin-1 β /sCD40L; TNF α /sCD40L; Myoglobin/IL-1 β ; CRP/sCD40L; CRP/IL-6; CRP/TNF α ; TNF α /sICAM; CRP/MMP9; TNF- α /Adiponectin ($p < 0.01$).

Discussion/Implications: Analysis of *in vivo* CVD status showed that significant race/health disparities exist in IPV + cohorts, as well as increased expression of inflammatory mediators, specifically CRP, IL-1 β , IL-6, MMP9. Women who have experienced IPV may be a target cohort for primary prevention of CVD. The use of salivary biomarkers and our protocol may provide a less invasive method to help increase identification of victims at risk for IPV and CVD and potentially decrease other health injuries associated with IPV exposure.

Keywords

Intimate partner violence (IPV); cardiovascular disease (CVD); Saliva; Health disparities; Facial injuries; Questionnaires

INTRODUCTION

Intimate partner violence (IPV) is a global public health epidemic that can initiate/exacerbate the prevalence of health conditions affecting a victim's lifespan.^{1–30} Studies indicate a strong association between IPV and an increase in the risk of cardiovascular disease (CVD) because persistent stress contributes directly to the pathogenesis of cardiovascular function.^{2,3,6–12} CVD is the number one silent killer in women but only 13% perceive it as a health threat.^{1,3,5,10} Women who have experienced IPV engage in coping strategies that pose negative health effects, such as smoking and poor nutrition, which potentiate the long-term negative impact/risk for CVD.^{3,6–9} Although some researchers theorize that inflammatory/hormonal cascades are pivotal in the development of significant health disparities such as CVD in victims of IPV, the exact mechanism(s) are still undetermined.^{3–8,16,17} Women who have been exposed to IPV may be more susceptible to the deleterious effects of pro-inflammatory cytokines and impairment of endothelial function with subsequent cardiovascular breakdown.^{1,4}

Salivary factors have been applied as a “diagnostic alphabet” to monitor disease progression in stress-related events.^{4,16–20} Studies using these risk predictors of adverse clinical

outcomes associated with IPV and CVD include C-reactive protein (CRP), Interleukin-1 and 6, and other inflammatory peptides.^{6,20–25} As such, saliva can provide an accurate sensitive vehicle for biomarker profiling of CVD inflammatory mediators to assess the mechanisms in cardiovascular function, platelet aggregation, and inflammatory responses to endothelial dysfunction.^{24,25}

A previous pilot study conducted by our group indicated that IPV prevalence is greater in African-American women patients presenting to our dental center, and that facial injuries, and other health consequences are more common among those with IPV positivity ($p < 0.05$)¹⁶. This study's purpose was to investigate the correlation among IPV exposure, in-vivo CVD outcomes, and salivary biomarkers in order to identify potential mechanisms that may predispose African-American (AA) and non-AA female dental patients to an increased risk for CVD. Our specific aims were 1) to identify IPV victims with stress-related CVD outcomes using two well-validated screening questionnaires and 2) to evaluate differences between inflammatory biomarkers associated with cardiovascular disease in those with and without exposure to IPV.

MATERIALS AND METHODS

Study design/sample

Participants were recruited for this analyses from the Meharry Medical College (MMC) School of Dentistry (SOD) Oral Surgery Clinic as a cross-sectional study that was Institutional Review Board (IRB) approved. Enrollees were female, ages 19-63 yrs. The total number enrolled for this pilot study were $N = 37$, including 19 African-Americans (AA), and 18 non-African-Americans (Non-AA). Inclusion criteria: The participants were required to be female, to speak, read, and comprehend English, and be willing to consent. Exclusion criteria: Patients that were unable to speak English were not eligible. Patients were recruited during computer-generated random clinic times over a period of 10 months during the clinic hours of 8:00 a.m. to 12 Noon, Monday -Friday. The study was divided into two visits. The first visit was used to complete the questionnaire about demographics, health history, including history of injury to head, neck and face, and to complete the Partner Violence Screen (PVS).³¹ The participants also provided a sample of their saliva (below). The second visit was a one-week follow-up whereby the participants responded to a second questionnaire, the Partner Abuse Symptom Scale (PASS).^{32,33}

Study variables

Questionnaire predictor variables.—1. Injury location and Partner Violence Screen (PVS) and 2. Partner Abuse Symptom Scale (PASS). Injury location was divided into 2 categories: head, neck or face (HNF) or injuries to the chest, abdomen, pelvis or extremities (termed 'other'). If the participant had HNF and other injuries, it was counted as a positive for head, neck and facial injuries. The PVS asked 3 questions: 1. *"Have you ever been hit, kicked or punched in a relationship?"* 2. *Have you ever felt unsafe in a relationship?* 3. *Do you feel unsafe in your present relationship?"* A yes response to any of the 3 questions constitutes a positive screen for IPV (Feldhaus et al., 1997).^{28,30,31} Based on national averages, we estimated that 40% would have been exposed to IPV (Halpern and Dodson

2006; Halpern et al., 2009; Halpern et al., 2016). Participants were also asked to respond to the Partner Abuse Symptom Scale (PASS; Ford-Gilboe et al., 2009; McHorney et al., 1994).^{32,33} This instrument measures long-term injuries resulting from abuse and assesses acute and chronic physical symptoms (McHorney et al., 1994; Miller and Campbell 1993).^{33,34} This self-report scale lists 25 injuries, conditions, symptoms, and illnesses related to IPV and asks whether the woman visited a physician or nurse for the problem in the past 12 months. Women rated 44 symptoms related to 6 domains of health (gastrointestinal, reproductive, neurological, cardio-respiratory, pain, and mental health) according to the method of Ford-Gilboe et al.³² These were then compared with the injury location/PVS questionnaire to assign subjects to IPV + or IPV– cohort.^{32,33} The confidentiality of each participant was protected by calibrating the evaluators as to methodology of questioning and questioning behind closed doors. *3. Primary Outcome Predictor Variable:* the outcome variable of interest was self-report of IPV for 2 categories: recent/past IPV (+) or IPV– (fall, occupational injury, sports, assault by non-intimate partner). For our purposes IPV was defined as a woman's self-report of an assault by a spouse/non-spouse using the PVS screening tool (see discussion).^{28,30,31} *4. Secondary predictor variables:* The *in vivo* CVD parameters measured included chest pain (CP), heart palpitations (HP) and Hypertension as evidenced with a yes or no answer and review of medications. Stress was measured by self-reported: 1) anxiety, and 2) post-traumatic stress disorder. Other variables included age (years), race, social habits (tobacco, alcohol, and substance use), and marital status. *5. Salivary specimens:* Saliva specimens were obtained from women using the passive drool method (unstimulated whole saliva; UWS) for saliva collection according to Foley et al., 2012 and Out et al., 2012.^{25,26} The samples were centrifuged immediately at 10,000 r/min for 5 min at 4 °C. Aliquots (0.5 mL) of each saliva sample were put into a 1-mL EP tube and stored at –80 °C (Out et al., 2012). Ten Biomarkers were selected to aid in evaluating the clinical utility for risk of CVD according to the method of Foley et al.²⁵ Specimens were analyzed by multiplex-ELISA at Oral Health Research Laboratory (CDTL), Lexington, KY. Statistical significance ($p < 0.05$) *6. Statistical Analyses:* Data analyses included descriptive statistics, univariate and bivariate analyses using SPSS® (Chicago, IL). P-value of <0.05 of predictor and outcome variables were considered statistically significant. Based on the relationship between risks for IPV-related injuries (high-or low-risk) classified per our questionnaires and reported injury location we were able compute the likelihood of an IPV related injury with associated 95% confidence intervals (Halpern and Dodson 2006; Halpern 2009; Halpern et al., 2006; Halpern et al., 2016)

RESULTS

A total of 37 women ($N = 37$), ages 19-63 yrs (± 14.2) were enrolled in this pilot cross-sectional study. There were 19 African-American (AA) and 18 non-African American (non-AA) females. (Fig. 1). The prevalence of IPV positivity was 51% (19/37) versus 26%. According to race, 47% (9/19) of IPV + women were AA compared to 52% (10/19) for non-AA IPV + women ($p > 0.05$). Association of demographic variables with IPV + vs IPV– women were non-significant for age, race, and educational level ($p > 0.05$). A significant difference was observed for IPV + women for substance abuse ($p < 0.05$; Table 1). With respect to in-vivo CVD outcomes, significant differences were noted between IPV + and

IPV— that included anxiety, CP and hypertension ($p < 0.05$; Fig. 2). IPV + AA females differed significantly ($p < 0.05$) from their IPV + non-AA cohorts with respect to CP and heart palpitations ($p < 0.05$; Table 2).

Table 3 depicts the ten most common biomarkers association with CVD disease progression.^{17,20,21,25} Samples between the 2 groups were analyzed for 10 biomarkers associated with CVD to identify differences, if any, based upon exposure to IPV. The sample were evaluated based upon a cross correlation of pairs of inflammatory mediators. CVD biomarker cross-correlates of the inflammatory mediators chosen between IPV + and IPV – cohorts showed significant differences ($p < 0.001$; Table 4) Of the 10 CVD biomarkers (Table 3) analyzed from saliva samples, IPV + women showed significant correlations with respect to Interleukin-1 β /sCD40L; TNF α /sCD40L; Myoglobin/IL-1 β ; CRP/sCD40L; CRP/IL-6; CRP/TNF α ; TNF α /sICAM; CRP/MMP9; and TNF- α /Adiponectin when compared with their IPV– counterparts($p < 0.005$; Table 4). A coincident result was observed in Table 5 that shows a similar cross —correlation comparison between IPV + AA and IPV + non-AA women. Significant differences are seen for the cross —correlates of inflammatory mediators within the IPV + group. ($p < 0.05$)

DISCUSSION

The purpose of this cross-sectional cohort study was to use a number of innovative tools and techniques to assess the association between exposure to IPV and risk for cardiovascular disease (CVD) among female patients seen at the SOD of Meharry Medical College, TN.^{16,28,30} We hypothesized that our two validated protocols (PVS/PASS) and inflammatory biomarkers in saliva will identify women exposed to IPV and who may be at a higher risk for CVD. The goal of our study was to use both IPV exposure and salivary biomarkers to determine risk and identify potential mechanisms that may predispose victims to an increased risk for CVD. The ultimate goal is to apply salivary diagnostics for early identification of victims at risk for cardiovascular disease and initiate early interventions that will help decrease adverse stress and CVD outcomes. We suggest that these approaches can provide useful information that can be used to decrease future IPV injuries and improve the cardiovascular health of patients.

The prevalence of IPV positivity was 51% (19/37) versus 26%. According to race, 47% (9/19) of women IPV+ were AA compared to 52% (10/19) for non-AA women ($p > 0.05$). Our findings show that the prevalence rate for IPV exposure among the participants is higher than state and national averages at 51.1%.^{7,11–13,16} The IPV frequency measured for AA females was 47% compared to 52% for non-AA. These values obtained from the PVS questionnaire were significantly different from the standard operating procedure (SOP) with the one question; “Are you a victim of domestic violence?” (27%, IPV +; $p < 0.05$). Previous studies by Halpern et al. have shown that a diagnostic protocol using injury location as head, neck and face, along with the PVS screening tool exhibits internal and external validity to stratify risk of self-report of IPV-related injuries.^{16,28–31} The PVS specifically has comparable criterion, construct, and content validity when compared to the Conflict Tactics Scale (CTS) and Index of Spousal Abuse (ISA).^{28–31} Halpern and Dodson previously developed a predictive multivariate logistic regression model using this protocol

to stratify risk of self-report of IPV-related injuries.³⁰ These unambiguous variables can aid in early diagnosis and prompt rapid referrals for intervention. The difference in prevalence rate seen between the SOP and our protocol may have been a result of how the questions are asked and not whether or not the questions were presented.^{16,28–31}

Analyses of the demographic variables with IPV + vs IPV– women were non-significant for age, race, and marital status ($p > 0.05$). A significant difference was observed for IPV + women for substance abuse ($p < 0.05$; Table 1). With respect to in-vivo CVD outcomes, significant differences were noted between IPV + and IPV– cohorts ($p < 0.05$; Fig. 2). IPV + AA females differed significantly ($p < 0.05$) from their IPV + non-AA cohorts with respect to CP and heart palpitations ($p < 0.05$; Table 2). The significant finding of smoking in the IPV + group when compared with their IPV-counterparts is in agreement with other studies in the literature.^{5,7,9,16,35} IPV has been associated with smoking as well as weight promoting health behaviors; physical inactivity, poor diet, disordered eating habits and access to care.^{1,2,5,10,35} The three significant risk factors for CVD in women as reported in numerous studies are smoking, obesity and hypertension.^{5,10,35–38} The results seen within our population of African American women agree with other studies that characterize certain sociodemographic characteristics and health consequences that are more frequent among abused women compared to never-abused women. Furthermore, the findings from this study support other studies that have included ethnically diverse samples in terms of sociodemographic characteristics, risk factors for abuse, health consequences, and use of medical services by abused women compared to never abused women.^{39,40} Previous studies have shown that abuse of women is usually associated with the sociodemographic characteristics of income and employment status, marital status, and age, although intra-ethnic group relationships have not usually been examined.^{39,40} Future studies are needed to compare multiracial female populations across larger community centers in order to understand the dynamics of IPV and how it intersects with the long term health consequences among ethnic diversities, as well as how they relate to sociodemographic and environmental exposure which are often “intertwined”.⁴⁰

Several authors of retrospective and cross-sectional studies have hypothesized the temporal relationship(s) of exposure of IPV and adult CVD. Exposure to IPV by parents, and/or abuse during childhood and adolescence may precipitate coping behaviors that significantly affect cardiovascular health through indirect pathways such as PTSD, obesity from poor dietary habits, sleep disturbances and substance abuse.^{37,38} Several cross-sectional studies have used a prospective sub-group approach to examine the effect of childhood abuse on cardiovascular outcomes in adulthood.^{41,42} The adult exposure to violence and abuse may operate under different neuro-hormonal cascades that have their origin many decades earlier. These exposures can further lead to inflammatory cascades and neuroendocrine disruptions resulting in hypertension, MI, stroke, and/or angina.^{9,10,35–38,43} Although our sample size was small IPV + participants demonstrated CVD risk that differed significantly ($p < 0.05$) from their non-AA cohorts with respect to anxiety, chest pain, heart palpitations and PTSD. Future studies are required, to more closely connect adverse health outcomes as a result of life-time exposure to stress-relate/abuse or IPV within our community in order to implement effective interventions and prospective surveillance. By doing so, it may be possible to more

closely isolate vulnerable time periods of IPV exposure and cardiovascular problems so that interventions can be monitored for successful healthy outcomes.

The use of saliva in assessing systemic disease is a well established noninvasive method to provide insight into the biologic mechanisms of CVD disease identification as well as prognosis with intervention.^{9,20,36} The CVD biomarkers we chose are associated with inflammation, atherosclerosis, myocardial damage and plaque stability.^{20,25,36} The CVD biomarkers measured within our saliva samples show trends that are significantly different between IPV+ and IPV- participants, as well as IPV + AA and IPV + non-AA individuals. The results from this investigation support findings from other studies that correlates IPV exposure with immune dysfunction, chronic precipitation of inflammation and/or exacerbation of the metabolic syndromes. In addition, they show possible chronic cumulative effects along the inflammatory cascade which agrees with studies that suggests IPV as a severe stressor. The biomarkers of choice; adiponectin, BNP, CK-MB, CRP, IL1, IL6, MMP-9, TNF- α , sCD40-1 and sICAM-1 measured from whole unstimulated saliva (UWS) can be highly specific and has clinical utility as a baseline prognostic indicator for identification and measure of biomarkers that are most often implicated in cardiac dysfunction and myocardial damage in other studies.^{3-6,9,10,20,43} Foley et al. have utilized assays with unstimulated whole saliva (UWS) to test whether oral fluids can detect severity of CVD in patients that undergo interventional therapy such as ablation or percutaneous coronary perfusion.²⁰ Changes from baseline and post intervention in this study support the premise that stress can specifically impair endothelial architecture due to circulating inflammatory cytokines Interleukin-6, CRP, Tumor necrosis factor (TNF) - α and platelet activation factors. The inflammatory profile of the patient was shown to resolve after interventional therapy as measured by Unstimulated Saliva (UWS).²⁰

The differences seen with respect to the cross correlates of inflammatory mediators between the IPV + and IPV- cohort salivary samples follow findings seen in other studies.^{4,6,19-21} Fernandez-Botran et al. suggest that elevated levels of CRP are more likely to be the result of a chronic cumulative effect of other mediators including the interleukins. Previous exposure to traumatic stress events such as facial trauma can result not only in elevated individual mediators, but their relationship to each other.^{6,10,21,44} Stressors whether they be physical or psychological can both increase the systemic levels of acute phase reactants such as IL-6 and CRP as well as act as regulators of other mediators within the inflammatory cascades. This has its basis at the level of the Hypothalamic-Adrenal — Axis which then provides feedback loops that orchestrate inflammatory cascades. The exposure of IPV whether it be as an adult or through temporal timing from early trauma sets up the rhythm for disease progression. Our results with respect to the mediators measured also suggests a potential trend of heightened inflammatory status. The significant differences seen between IPV + cohorts and controls in our study with respect to stress/PTSD combined with the above differences support the paradigm of a chronic cumulative effect as seen in other data published. Elevated cytokines have been reported in association with numerous health-related psychosocial factors such as depression, low socioeconomic environments, and chronic stress from unhealthy life behaviors.^{45,46} Several systematic reviews have indicated that depression is a major risk factor in the development of coronary artery disease either directly or indirectly via a complex pathway of inflammation, and psychosocial risk

predictors.⁴⁷ As such, IPV can impact cardiovascular health both directly and indirectly by disrupting a cascade of physiological mechanisms including modification of behaviors that lead to poor health outcomes. Information derived from these studies supporting a relation between IPV exposure, inflammation and CVD are significant given that CVD is the leading cause of mortality in women (23.2% in African American (AA) females and 22.3% in white females) [CDC].

CVD in Tennessee is the number one cause of death with higher rates among African-American (AA) women (7.5%) compared to their Caucasian women cohorts (5.4%). [BRFSS Fact Sheet: TN Board of Health] and is the 5th in the nation with respect to the number of women who are murdered each year from violence/abuse.^{11,12} TN loses at least \$10 million/year in paid work time, as well as, \$33 million in healthcare costs due to ipv.^{13,18,19} As such, early diagnosis of IPV exposure and related injuries would significantly improve the health of victims, and save billions of dollars in healthcare costs^{14,15} Meharry Medical College (MMC) School of Dentistry (SOD) serves a community that lacks proper access to care in the Nashville region of TN. IPV is a frequent cause of facial injuries in victims between the ages of 18-64 with 75% of injuries to the head, neck, mouth.^{26,27} With >50% of adults/children visiting our center, the SOD provides a pivotal point of contact for IPV victims.²⁷ The significant differences seen between African American and non-African American IPV + women in this study is of interest, as with many minority and disadvantaged populations, they continue to suffer disproportionately from chronic diseases that includes significant cardiovascular morbidity and mortality. Our previous study characterized significant differences in health consequences between races. Low socioeconomic status, partner unemployment, and a lack of access to care in the community are significant risk factors for partner abuse and poor health.^{40,48} This is further complicated by basic shortcomings of the health care delivery system, especially in metropolitan communities with higher poverty. Even with improvements in education and job opportunity there still exists a higher rate of IPV disparity among AA women compared to non-AA cohorts^{40,48} Additional studies in more ethnically diverse populations have the potential to tease out even more IPV-related effects on poor health outcomes.

Our study although quite encouraging does have limitations in data interpretation. The study design is cross-sectional; cause and effect studies can be prohibitive and. the small sample size (N = 37) is of insufficient power to make any generalizations about CVD health and biomarker risk indices. Although there were significant differences between the number of IPV + participants compared with their IPV- cohorts, due to the fear and stigma associated with IPV, the study may have susceptible to false negative responses from participants. Also, while questionnaires, have been shown to be valid and have good test-retest reliability victims may still report no abuse. In a study of this nature, victim misclassification is expected and should be taken into consideration as a potential confounder.⁴⁹ The health consequences from IPV may or may not exist after the abuse is discontinued and therefore our estimates may have missed a “window of opportunity”. This may explain why we did not see significant differences in IPV and hypertension that is a significant risk factor for development of CVD. Our data comparisons between IPV+ and IPV- participants with respect to hypertension may not have been significant due to the fact that totally different underlying mechanisms are involved in the processes and/or the measure for hypertension

may need to be captured over time with temporal exposure of IPV, and inherent concerns with recall bias related to exposure during earlier ages. Several other studies have suggested such a model of exposure to IPV and hypertensive predisposition in adult women who are prescribed anti-hypertensive medication prior to being questioned about present or past IPV exposure^{10,35,39} Suglia et al. performed a systematic review that concluded evidence of how the developmental process of CVD may operate under different biologic mechanisms compared to exposure of abuse decades earlier, suggesting that the timing of exposure and diagnosis of hypertension occurs within a window of opportunity. Evidence of this assumption was based upon five studies that characterized a positive relationship between child abuse and adult hypertension.^{10,39} Data from the Nurse's Health study concluded that severe abuse and forced sex was associated with increased risk of stroke, however, forced sex in childhood led to nonsignificant increase in MI among women victims.⁵⁰ All authors conclude that there may exist sensitive/vulnerable timelines relating exposure to this stress-event and cardiovascular health along the lifespan of victims.

Although saliva has provided promising use as a sensitive and specific risk predictor for disease identification, progression and resolution, several issues need to be addressed. Timing of sampling may influence concentrations of inflammatory mediator identification. Studies using plasma have suggested that IL-6 levels may be more susceptible to change in response to physiological and psychosocial stimuli compared with CRP. Other cytokines can together influence synthesis of CRP and IL-6. These intricate relationships can play a significant role in cross-correlation differences seen between our IPV+ and IPV- cohorts.^{6,48,50,51} Other sensitivity issues include preparation of samples. All samples are frozen and thawed prior to analysis, and the effect of temperature changes on concentrations of these proteins are not well understood. Time-effect analyses also need to be more carefully monitored with greater comparisons among populations that are not exposed to the stress-events described in our study.²⁰ Future studies will more closely examine the value of these oral fluids in monitoring CVD within larger sample sizes, as well as comparisons with sampling of plasma among patient's exposed to IPV.

Implications

The study presented provides a non-invasive approach using an innovative well-validated diagnostic tool to identify victims of IPV exposure and a possible link to cardiovascular disease (CVD). This is the first study undertaken in an oral and maxillofacial surgery clinic within a community dental school that treats a population that are predisposed to increased risk of CVD not only from IPV but other socio-demographic/health risk predictors. The application of salivary sampling along with our diagnostic protocols can provide a robust measurement to provide insights(s) in further expanding the relationship(s) between the etiology of CVD and exposure of IPV. These insights have the potential as a screening tool for clinicians in assessing cardiovascular risk in women with a history of IPV and provide a baseline for preventive strategies and better overall health related quality of life.

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Figure 1.
Demographic Distribution of the participants.

Figure 2. Cardiovascular variables in IPV vs non-IPV. P-value = 0.001, P-value = 0.002, P-value = 0.471.

Table 1.

Socio-demographics of study participants (N = 37).

Independent variable	IPV + (%)	IPV – (%)	p value
Income (<50,000)	52.6% (10/19)	44% (8/18)	0.62
Marital status (married, Divorced, widowed)	36.8% (7/19)	44.4% (8/18)	0.64
Race: African-American	47.3% (9/19)	55.5% (10/18)	0.62
Social history; (tobacco, alcohol, recreational drugs)	89.4% (17/19)	61.1% (11/18)	0.04
Was participant ever Asked before about IPV/abuse	21% (4/19)	16.6% (3/18)	0.73

Table 1: Socio-demographic data of participants that shows the frequencies and respective percentages. P-values have also been included (far right) to show significance.

Bolded p-values in corresponding IPV group represent significant correlation to that IPV group. Significance ($p < 0.05$).

Table 2.

Risk predictor variables of CVD within IPV + and IPV – cohorts.

Independent variable	IPV +	IPV –	p value
Anxiety	62.5% (12/19)	18.4% (4/18)	0.01
Chest Pain	20.6% (6/19)	6.7% (2/18)	0.01
Heart Palpitations	76.5% (15/19)	3.3% (1/18)	0.02
Stress (PTSD)	62.5% (12/19)	34.2% (6/18)	0.03
Hypertension ^a	50.7% (10/19)	48.9% (8/18)	0.71

Table 2: Previous studies showing the independent variables that were most significant to IPV. The center columns indicates IPV+ and IPV– with frequencies and their respective percentages. The column on the far right indicates the significance (p values) for each independent variables.

Bolded p-values in corresponding IPV group represent significant correlation to that IPV group. Significance ($p < 0.05$).

^aPatients that were given anti-hypertensive medication prior to being interviewed for participation in study.

Table 3.Cardiovascular disease (CVD) risk biomarkers^a.

CVD Biomarker	Known effect of CVD Biomarkers
1.sCD40L	Pro-inflammatory marker shown to promote atherosclerosis and plaque instability
2.IL-1 beta	Hypercholesterolemia
3.IL-6	Increases plaque instability driving expression of matrix metalloproteinases; TNF-alpha and MCP-1
4.TNF-alpha	Associated with myocardial dysfunction and remodeling after acute coronary events
5. Adiponectin	Adiponectin in CVD itself is debatable because high levels of Adiponectin have been associated with decreased CVD risk in asymptomatic individuals, whereas it can also predict poor Prognosis in patients with established CVD.
6. PAI-1	Contributes to the development of ischemic CVD
7. MMP9	Prognostic for CVD
8. Myoglobin	Protein of skeletal and cardiac muscle to aid in morphological changes in cardia event effects of muscle size
9. sICAM-1	Atherosclerosis promotion
10. CRP	Inflammatory mediator associated with atherogenesis

^a Adapted from Foley JD, Sneed J, Steinhubl SR et al. Oral fluids that detect cardiovascular disease biomarkers. Oral surg Oral Med, Oral Pathol Oral Radiol 2012; 114(2):207-214.

Table 4.

CVD biomarker IPV +\|- cross correlation.

IPV + Samples (Spearman's rho)	sCD40L (pg/ml)	IL-1b (pg/ml)	IL - 6 (pg/ml)	TNF-a (pg/ml)	Adiponectin (pg/ml)	PAI-1 (pg/ml)	MMP9 (pg/ml)	Myoglobin (ng/ml)	sICAM-1 (pg/ml)	CRP (mg/ml)
sCD40L (pg/ml)		0.137	0.270	0.056	0.696	0.413	0.706	0.766	0.468	0.788
IL - 1b (pg/ml)	0.031		0.020	0.045	0.002	0.013	0.000	0.044	0.294	0.026
IL - 6 (pg/ml)	0.166	0.000		0.014	0.016	0.088	0.100	0.795	0.908	0.969
TNF-a (pg/ml)	0.039	0.000	0.002		0.092	0.146	0.068	0.713	0.184	0.586
Adiponectin (pg/ml)	0.252	0.001	0.003	0.003		0.008	0.001	0.023	0.524	0.168
PAI-1 (pg/ml)	0.336	0.000	0.000	0.003	0.001		0.025	0.004	0.106	0.030
MMP9 (pg/ml)	0.128	0.006	0.047	0.002	0.014	0.000		0.081	0.086	0.057
Myoglobin (ng/ml)	0.863	0.013	0.006	0.025	0.000	0.007	0.064		0.185	0.024
sICAM-1 (pg/ml)	0.067	0.000	0.006	0.000	0.001	0.001	0.003	0.008		0.144
CRP (mg/ml)	0.036	0.000	0.000	0.001	0.060	0.000	0.022	0.183	0.001	

Cross comparison of Cross Correlated CVD Biomarkers in IPV+ and IPV- participants. Left side right triangle area represents the significance of Cross Correlated CVD Biomarkers among IPV+ participants represented in p-values. Right sided upside down triangle represents the significance of Cross Correlated CVD Biomarkers among IPV- participants represented in p-values. Bolded black p-values in corresponding IPV group represent significant correlation to that IPV group. Significance ($P < 0.05$).

Table 5.
CVD biomarker IPV + cross correlation amongst african-americans and non-african american participants.

IPV + Samples (Spearman's rho)	sCD40L (pg/ml)	IL-1b (pg/ml)	IL - 6 (pg/ml)	TNF-a (pg/ml)	Adiponectin (pg/ml)	PAI-1 (pg/ml)	MMP9 (pg/ml)	Myoglobin (ng/ml)	sICAM-1 (pg/ml)	CRP (mg/ml)
sCD40L (pg/ml)		0.934	0.243	0.9687	0.602	0.567	0.933	0.400	0.957	0.789
IL - 1b (pg/ml)	0.001		0.016	0.011	0.033	0.025	0.185	0.100	0.041	0.002
IL - 6 (pg/ml)	0.003	0		0.293	0.074	0.082	0.663	0.038	0.530	0.200
TNF-a (pg/ml)	0.006	0.005	0.005		0.187	0.162	0.192	0.281	0.004	0.011
Adiponectin (pg/ml)	0.061	0.025	0.010	0.025		0.048	0.179	0.014	0.074	0.150
PAI-1 (pg/ml)	0.001	0.004	0.013	0.001	0.125		0.007	0.024	0.074	0.038
MMP9 (pg/ml)	0.012	0.031	0.045	0.002	0.081	0.003		0.148	0.089	0.275
Myoglobin (ng/ml)	0.111	0.081	0.021	0.041	0.003	0.318	0.195		0.166	0.464
sICAM-1 (pg/ml)	0.001	0.007	0.002	0.001	0.003	0.009	0.013	0.009		0.016
CRP (mg/ml)	0.001	0.001	0.001	0.008	0.137	0.002	0.043	0.158	0.007	

Cross comparison of Cross Correlated CVD Biomarkers in IPV + AA and non-AA participants. Left side right triangle represents the significance of Cross Correlated CVD Biomarkers among IPV + AA participants represented in p-values. Right side upside down triangle represents the significance of Cross Correlated CVD Biomarkers among IPV + non-AA participants represented in p-values. Black bolded p-values in corresponding IPV group represent significant correlation to that IPV group. Significance ($p < 0.05$).