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Review Article

Intimate Partner Violence perpetration and cardiovascular risk: A systematic review

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ABSTRACT

Intimate Partner Violence (IPV) perpetration may induce cardiovascular reactivity and risk markers thereby precipitating early onset cardiovascular disease (CVD). However, this relationship has been largely under-researched in comparison to the health impacts of IPV victimisation. We therefore aimed to systematically review the current evidence investigating the relationship between IPV perpetration and CV risk. Six databases (CINAHL, Ovid MEDLINE, Pubmed, Scopus, ProQuest, Google Scholar) were searched between August 2016 and August 2017 using a predefined search strategy. Inclusion criteria were studies of cross sectional and longitudinal design published since 2010, presenting IPV status by perpetrators (as distinct from victims) and an outcome of CVD (e.g. cardiac disease, stroke), CV risk markers (e.g. blood pressure) and/or a composite CV risk score. Twenty two potentially eligible studies were identified and full texts recovered. After ineligible studies were excluded, four remained (total n = 10,665). Positive relationships were observed between IPV perpetration and (i) short term CV reactivity markers (higher heart rate, lower vagal ratios, shorter pre-ejection periods) and (ii) longer term CV risk factors and outcomes including greater systolic blood pressure, incident hypertension, elevated 30 year CV risk score and self-report cardiac disease. Despite being a neglected area of research characterised by a high degree of heterogeneity, the early evidence suggests that IPV perpetration may be associated with elevated risk of CVD. We discuss these findings in the context of CVD prevention from the individual, family and inter-generational perspectives and directions for future studies.

1. Introduction

Family violence (FV) is a pervasive source of environmental stress that affects one in three women, globally (World Health Organization, 2013). It is most commonly perpetrated by men against current or former intimate partners (Intimate Partner Violence; IPV) (Taft et al., 2001). IPV victimisation has been associated with a range of deleterious mental and physical health effects including cardiovascular (CV) risk behaviours and outcomes (Stene et al., 2013). The CV effects of IPV victimisation have been extensively researched and include higher rates of carotid atherosclerosis, Takotsubo cardiomyopathy ("broken heart syndrome"), obesity, high triglycerides, low HDL-C, and higher cigarette, drug and alcohol consumption compared with women who are not exposed (Stene et al., 2013).

In 2015, Suglia et al. conducted a systematic review investigating the long term association between exposure to violence in either childhood or adulthood and subsequent CV outcomes including hypertension, blood pressure, stroke, coronary heart disease or myocardial infarction (Suglia et al., 2015). The authors found a consistent and significant relationship between childhood exposure to physical or sexual violence and cardiovascular endpoints, documenting heterogeneity in exposure and endpoint measures. The association was less clear for violent exposure in adulthood and CV health.

By comparison, the cardiotoxic effects of IPV perpetration remain largely unknown, largely due to a lack of research. Research from the fields of personality and social psychology and criminology support the hypothesis that violent behaviours have negative effects on the CV system, particularly over the short term. For example, aspects of control (i.e., dominance) have been shown to predict higher systolic and diastolic blood pressure (SBP, DBP) in men (Baron et al., 2016). Others have demonstrated that high-hostile men show elevated and more prolonged increases in BP, forearm blood flow and vascular resistance, and norepinephrine compared with low-hostile men (Suarez et al., 1998). When compared with low-hostile men, high hostility independently predicts vascular sympathetic drive (Virtanen et al., 2003) and higher skin conductance responses (Carmona et al., 2008).

In 2010, Pinto et al., conducted a review of the literature that identified potential biological correlates of IPV perpetration more

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specifically, including physiological reactivity (e.g. changes in biomarkers in response to a stressor). The objective of the review of the evidence (generated between 1979 and 2010) was to investigate physiological reactivity, specifically arousal levels, in order to identify subtypes of IPV perpetration. Research assessing the relationship between IPV perpetration and CV risk and markers has rarely been considered in the context of CV disease progression. Indeed, it is plausible that chronic, maladaptive CV responses owing to the use of IPV perpetration could induce subclinical CV disease such as carotid atherosclerosis and peripheral vascular disease, CV risk factors such as hypertension or hyperlipidemia, or CV reactivity. Yet traditionally, this has been a neglected area of public health research.

The aim of this paper was therefore to systematically review the current evidence based investigating the relationship between IPV perpetration (distinct from victimisation) and CVD, discuss candidate pathophysiologic mechanisms and consider how better understanding the CV system of perpetrators may have utility in both IPV and CVD prevention.

2. Methods

2.1. Selection of studies

This review built on that of Pinto et al., published in 2010. Studies were considered for inclusion if they were: (i) published between 2010 and August 2017 (ii) full-text articles; (iii) cross sectional and longitudinal cohort study designs; (iv) examined associations between IPV perpetration (self-reported, identified through administrative records, partner reported) as distinct from victimisation; (v) included a CV outcome (e.g. cardiac disease, stroke), markers of CV risk or reactivity (e.g. blood pressure, heart rate variability) and/or a composite CV risk score identified by self-report, medical records, data linkage; and (vi) studies that defined IPV as form(s) of physical, verbal, emotional, sexual, religious, technological, and/or financial abuse where a current or previous intimate partner was victimised. We excluded studies which did not present the aforementioned CV outcomes, and randomised controlled trials.

2.2. Search strategy and data extraction

Between August 2016 and August 2017, six databases for medical, health, psychiatric and social sciences (CINAHL, Ovid MEDLINE, Pubmed, SCOPUS, Pro Quest and Google Scholar) were searched using a computer-generated search strategy. The full search strategy is provided in Appendix A. In order to identify grey literature, reference lists of relevant reviews and studies were searched. AO conducted the electronic search strategy, which was replicated by AJS. Once abstracts of potentially relevant papers were identified and full-text copies obtained, each author finalised the list of included articles based on predetermined inclusion and exclusion criteria. The review was conducted in accordance with the guidelines outlined in the preferred reporting items for systematic reviews and meta-analyses (PRISMA) statement 2009 (Moher et al., 2009).

3. Results

The initial search strategy yielded 520 citations. Of these, 22 were identified as potentially relevant. After removing five duplicates, 16 full text articles were retrieved. Of these, 12 were excluded, leaving four eligible studies for inclusion in this review (total n = 10,665). The consensus between authors was 100%. Fig. 1 displays a summary of the results of the systematic search.

3.1. Population and design

Key characteristics of the included studies are displayed in Table 1. One study investigating short term CV reactivity used a laboratory based, repeated measures study design of healthy males; half of whom had a criminal history of IPV (n = 17) and half did not (n = 17) (mean age: 36.88 ± 2.59 and 34.82 ± 1.47, respectively). Two of the included studies used data from the National Longitudinal Study of Adolescent to Adult Health; (n = 9976; n = 9699, respectively) a nationally representative sample of US adolescents in grades 7–12. Participants were interviewed across four waves of assessment between 1994–95 and 2008–09. The fourth study used a cross-sectional design investigating the association between IPV and physical health conditions in offenders with alcohol disorders (mean age 33.4 ± 10.9). This study treated cardiac and other conditions as a predictor of IPV perpetration.

3.2. Measures of IPV

Three of the four studies used a subset of questions from the revised Conflict Tactics Scales (Straus et al., 1996) to assess IPV perpetration, e.g. Have you threatened your partner with violence, pushed or shoved, or thrown something that could hurt; slapped, hit or kicked your partner; made your partner have sexual relations when they did not want to; caused an injury, such as a sprain, bruise, or cut because of a fight with your partner. Both Clark studies categorised IPV as follows: no IPV experience, victimisation only, perpetration only and bi-directional (both victimisation and perpetration) (Clark et al., 2014, 2016). Crane and Easton (2017) categorised participants as violent (any physical IPV) or non-violent (no physical IPV). The Romero-Martinez study identified IPV perpetration history through criminal records (Romero-Martínez et al., 2014).

3.3. Measures of CVD

Romero-Martínez et al.'s (2014) study used short term CV reactivity in response to a stressful task as the outcome of interest (heart rate (HR), vagal ratio and pre-ejection period (PEP) before, during and after exposure). Clark et al. (2014) used the primary endpoint of systolic blood pressure (SBP) as a continuous variables as well incident hypertension (as diagnosed by participant self-report). Clark et al. (2016) used the Framingham Risk Prediction model (Pencina et al., 2009) (calculated at wave 4 (2008/09) when participants were 29 years) to assess 30-year risk of developing incident CVD. Crane and Easton (2017) identified existing cardiac issues (CV or heart disease, angina, hypercholesterolemia, hypertension) by self-report during clinical interview, confirmed with review of medical records.

3.4. Relationship between IPV perpetration and CVD outcome

All four studies identified a positive association between IPV perpetration and the CV measure of interest. Romero-Martínez et al. (2014) found that men with a history of IPV had poorer CV reactivity compared with men with no history. A significant “time × group” interaction effect was found where IPV perpetrators had higher HR [0.68; F (2.05, 65.58). 3.17, p. 0.047] and lower vagal ratio [0.64, F (1.92, 61.49). 3.08, p. 0.050] during the recovery time, compared to controls. A significant “group” effect was found for PEP [F (1, 32). 3.93, p. 0.05], where IPV perpetrators had shorter PEP than controls. Those with a history of IPV perpetration and those without did not differ on any socio-demographic factors. Clark et al. (2014) found that men who identified as severe perpetrators (combined with victims) had a 2.66 mm Hg (95% CI: 0.05, 5.28) higher SBP and a 59% increased odds of incident hypertension. When looking at CVD risk scores, Clark et al. (2016) found that IPV perpetration, specifically in late adolescence and young adulthood, was associated with an increased risk of incident CVD in the ensuing 7–14 years (b 0.33 (95% CIs: 0.03, 0.62)). Both Clark et al. (2014, 2016) papers adjusted for socio-demographic factors, including age, ethnicity, educational attainment and financial stress.