Published in final edited form as:
Trauma Violence Abuse. 2013 October ; 14(4): 305–315. doi:10.1177/1524838013495962.

Intimate Partner Violence Victimization and Cigarette Smoking: A Meta-Analytic Review

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Abstract

The current meta analytic review represents the first comprehensive empirical evaluation of the strength of the relationship between intimate partner violence (IPV) victimization and cigarette smoking. Thirty-nine effect sizes, drawn from 31 peer reviewed publications, determined the existence of a small to medium composite effect size for the victimization-smoking relationship (*d* = .41, 95% CI = .35–.47). Results indicate that victims of IPV are at greater smoking risk than non victims. Subsequent moderator analyses indicated that the association between victimization and smoking is moderately stronger among pregnant compared to non pregnant victims. The strength of the victimization-smoking relationship did not differ by relationship type or ethnicity. More research is needed on the smoking behavior of male victims, victims of physical violence, and victims who identify as Latino/a. It would be useful for professionals working with IPV victims to assess for smoking and incorporate smoking prevention and cessation skills in intervention settings.

Introduction

Intimate partner violence (IPV) is defined as a means of intimidating or controlling an intimate partner through a coercive pattern of inflicting or threatening to inflict physical, sexual or emotional harm (Saltzman, Fanslow, McMahon, & Shelley, 2002). IPV victimization has a lifetime prevalence of 22.1% among women and 7.4% among men (Tjaden & Thoennes, 2004). IPV victims report a lower quality of life relative to non-victims in terms of mental and physical health (Laffeye, Kennedy, & Stein, 2003). Victimization is as a significant risk factor for posttraumatic stress disorder (PTSD) and subclinical levels of trauma symptomatology (Kaysen, Dillworth, Simpson, Waldrop, Larimer, & Resick, 2007) as well as additional adverse mental health outcomes such as mood disorders (Stein & Kennedy, 2000), general anxiety problems (Pico-Alfonso et al., 2006), and substance use and dependence (Coker et al., 2002), including cigarette smoking (El-Mohandes, El-Khorazaty, Kiely, & Gantz, 2011). Victimization often results in multiple comorbidities (Nixon et al., 2004). IPV experience is further associated with detrimental physical health consequences (e.g., Campbell et al., 2002)

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Cigarette smoking is the leading preventable cause of illness and mortality in the United States (Centers for Disease Control and Prevention (CDC), 2008). Nationally representative data finds that 19.3% of adults in the United States self-identify as current smokers with a higher proportion of male (21.5%) relative to female (17.3%) smokers (CDC, 2011). Decreases in smoking rates have slowed in recent years despite national efforts to reduce cigarette use through educational programs and advertisements educating smokers about the negative health and social consequences of tobacco addiction. Understanding and addressing specific factors that underlie initial and continued use remains an issue of critical importance (CDC, 2004).

The existing literature on the direct association between IPV victimization and cigarette smoking has demonstrated that victims are more likely to smoke than nonvictims (e.g., Ditcher, Ceruli, & Bossarte, 2011; Vest, Catlin, Chen, & Brownson, 2002) but the strength of this relationship has never been systematically quantified. Further, there is little data on the potential moderators of the relationship between IPV victimization and smoking. The present meta analytic investigation examined 31 published studies that reported the relationship between IPV and smoking as well as various potential moderators of the relationship. Moderators included pregnancy status, relationship type, ethnicity, and socioeconomic status (SES). To our knowledge, this is the first meta analytic review of the literature on IPV victimization and smoking behavior.

Etiology

Smoking has been attributed to attempts to cope with the experience of negative affect resulting from withdrawal, affective dysregulation, or traumatic events (Baker, Piper, McCarthy, Majeskie, & Fiore, 2004). IPV meets the criteria for a traumatic event or series of events as operationalized by the DSM-IV-TR (American Psychiatric Association, 2000) and has been accepted as a psychological stressor that places victims at increased risk for developing both depression and PTSD (Coker et al., 2002; Kaysen et al., 2007). Negative affect and stressful stimuli have been shown to promote substance abuse continuation, relapse, and increased subjective cravings across studies (e.g., Sinha, 2001). Feldner and colleagues (2007) recently published a review article in which they concluded that, across 7 studies, the prevalence of smoking was consistently elevated among samples diagnosed with PTSD compared to samples without the diagnosis.

Researchers specifically describe smoking as a maladaptive coping mechanism for relationship distress and IPV (Scott-Storey, Wuest, & Ford-Gilboe, 2009). Reinforcement models help clarify the potential relationship between IPV victimization and smoking through the intermediary of negative affect. The biphasic effects of nicotine initially result in alertness, a physiological response that represents immediate positive reinforcement (Pomerleau & Pomerleau, 1984). The second limb of nicotine's effect involves a calming response that serves as a negative reinforcer (Pomerleau & Pomerleau, 1984). Kassel and Unrod (2000) asserted that smoking diverts one's attention from stimuli that produce negative affect to less relevant stimuli, effectively reducing perceived anxiety and depressed mood through distraction. Thus, the initial effects of smoking may serve to temporarily remediate symptoms of depression while the secondary effects may reduce the anxious symptoms of trauma. The salience and immediacy of reinforcement increases the likelihood that smoking will be maintained to avoid the physiological or cognitive cues of negative affect. This learned method of coping with or suppressing undesirable emotional responses that arise from either physiological withdrawal or environmental stressors is a form of self-medication (Audrain-McGovern, Rodriguez, & Kassel, 2009; Khantzian, 1985).

Neurobiologically, nicotine is a physiologically addictive substance that stimulates nicotinic acetylcholine receptors, exerting a direct effect on the neurotransmitters and neural

pathways associated with negative affective states including depression, stress, and anxiety (e.g., Piccioto, Brunzell, & Caldarone, 2002). Nicotine use may result in short-term relief of anxiety and depression but long-term neurobiological adaptations in the catecholamine, GABA, glutamate, and serotonin neurotransmitters also make it difficult to stop smoking through enhanced negative affect during acute withdrawal (Markou, Kosten, & Koob, 1998). Such adaptations disrupt the natural reward system of the brain and promote the belief that substance use holds an unrealistically high value when compared to abstinence, thus tobacco use following or in anticipation of negative affect becomes more likely through both short term behavioral reinforcement and long term biological changes (Baker, Piper, McCarthy, Majeskie, & Fiore, 2004; Robinson & Berridge, 1993).

Moderators

While the majority of studies report a relationship between IPV victimization and smoking, there is heterogeneity of such magnitude as to suggest that the strength of the relationship may be moderated by other factors. Our final selection of potential moderators for the current analysis was derived from factors that influence the severity of IPV, the presence of additional stressors, or the ability to cope with existing stressors within the literature and limited by statistical power to included pregnancy status (pregnant or not pregnant), relationship type (married or dating), ethnicity (Caucasian American, African American, or Latino American), and SES (Unemployed / <$25,000 per year or employed / >$25,000 per year).

Pregnancy—Pregnancy is a period of increased emotional and physiological stress and a time during which women are at considerable risk for partner violence (McFarlane, Parker, & Soeken, 1996). Estimates of the prevalence of IPV victimization during pregnancy reach up to 81% of expectant mothers, depending on sample characteristics and study methodology (Bailey & Daugherty, 2007). Rates of smoking among pregnant victims exceed the general population (El-Mohandes, El-Khorazaty, Kiely, & Gantz, 2011). We would expect the relationship between victimization and smoking to be stronger among pregnant relative to non-pregnant participants due to higher baseline levels of physical and emotional distress as well as various characteristics that place them at greater risk for victimization.

Relationship Type—Distinguishing between married and dating IPV may account for some of the observed heterogeneity in the IPV-smoking association across studies. As Straus (2004) reported, a preponderance of studies have concluded that physical IPV occurs more frequently in dating rather than marital relationships. This pattern of violence may be related to age and life experience. The late adolescent and young adult years are ones of increasing independence, social change, and experimentation that make individuals vulnerable to antisocial behavior, including substance use and violence (Brown et al., 2008; Leonard, 2002). The availability of cigarettes and the absence of finely developed adaptive coping skills may contribute to the vulnerability of victimized youths or dating partners to smoke as a means of temporarily reducing the negative affect associated with IPV victimization. Older IPV victims are more likely to be married and may be more skilled than younger victims at coping through cognitive efforts, involvement in social roles, and social supports (Zink et al., 2006). Thus, we expected a stronger association between victimization and smoking among younger, dating participants relative to older, married victims.

Demographics—Research regarding *ethnicity* (i.e., Caucasian and African American) as a potential moderator of the victimization-smoking relationship is contradictory. While the majority of studies indicate that violence is more severe within minority populations (e.g. Lipsky et al., 2012), it has been reported that Caucasians generally experience greater

psychological sensitivity to victimization as evidenced by significantly more depression and trauma reporting among clinical samples (e.g., Sullivan & Rumptz, 1994). This may be an artifact of willingness to seek mental health services to cope with trauma (El-Khoury, Dutton, Goodman, Engel, Belamaric, & Murphy, 2004). Research also shows that the link between negative affect and smoking is stronger in Caucasian than African American adults (Gritz et al., 1998). We expected that the link between victimization and smoking would be strongest among Caucasian samples. Finally, low *SES* is a risk factor for both IPV and smoking (Vest et al., 2002). This has been established using educational attainment, income levels, and residential placement variables as proxy measures of SES (e.g., Novotny, Warner, Kendrick, & Remington, 1988). Given the overlapping risk factors for both smoking and IPV victimization, it is possible that SES may mask the strength of the relationship between the two behaviors within samples that report economic hardship.

Prior Reviews

The association between IPV victimization and substance use (e.g., alcohol, cocaine, and marijuana) has been evaluated in both qualitative (Riggs, Caulfield, & Street, 2000) and quantitative (Stith, Smith, Penn, Ward, & Tritt, 2003) reports. Most recently, Moore and colleagues (2008) collected 547 effect sizes from 96 studies to report the association between the use of illicit drugs and IPV victimization and perpetration. While the magnitude of the composite effect was small, analyses of specific substances concluded that cocaine use shared a strong association and medium effect with physical victimization based upon 6 effect sizes gathered from 2 studies and sexual victimization based upon 3 effect sizes from a single study. Marijuana use and IPV victimization shared a small association. The relationship between nicotine use and IPV victimization has not been evaluated.

The Current Review

We attempted to identify and review all published studies that have examined both smoking behavior and IPV victimization in the first meta analytic review of nicotine use as a correlate of partner violence. We first examined the overall effect size for IPV victimization and smoking. We then examined potential categorical moderators. Based on the literature reviewed above, we hypothesized that the relationship between smoking and IPV victimization would be stronger for: 1) pregnant compared to non-pregnant participants, 2) dating compared to marital violence, and 3) Caucasians compared to African Americans. We did not advance a hypothesis regarding SES due to inconsistencies in the literature and insufficient empirical information.

Method

Study Selection

Viable articles were retrieved through a search of the PsychInfo, Social Work Abstracts, Social Sciences Abstracts, Social Sciences Citation Index, and subsequent reference list searches. Our search was confined to articles published before February of 2012. Articles containing terms from each of two keyword groups (violence and smoking) were identified and examined for relevance. The violence keyword parameter included variations of victim, partner violence, IPV, aggression, domestic, and assault. The smoking keyword parameter included cigarette, smoke, tobacco, and nicotine. The initial search returned 102 viable articles.

Inclusion Criteria—All articles selected for inclusion in the current analysis were required to provide the necessary data to calculate an effect size. Sixty-six of the 102 identified studies were eliminated because they did not meet this criterion. Four articles were eliminated due to the high probability of duplicated samples. One article included

information on only perpetrators rather than victims. Each study was published in a peer reviewed journal and written in English. The final sample of articles included 31 studies.

Coding Procedures—Information regarding the study, the samples of interest, and effect size data was collected from each article using a structured codebook composed by the first author and reviewed by all study personnel. IPV group membership reflected the presence or absence of IPV victimization. Across studies, IPV data was drawn largely from self-report questionnaires or automated surveys (84.6%). The presence or absence of smoking behavior was gathered using diverse methods, primarily questionnaires (30.8%) and interviews (23.1%).

Victim data regarding pregnancy status, relationship type, ethnicity, and SES were gathered for moderator analyses. Regarding relationship type, samples were categorized as either dating or married depending upon the majority report (i.e. 60% or greater). Samples were categorized within a particular ethnicity if the majority (i.e. 60% or greater) of participants were identified as belonging to a specific ethnic group. Samples were categorized as low SES if the majority of participants reported correlates of economic hardship (i.e. failure to complete high school, a household income below $25,000 per year, or unemployment). The first two authors independently double coded 16 (50.0%) articles and obtained high overall inter-rater reliability across items (Kappa = 0.86). All inconsistencies in ratings were discussed and final decisions were mutually determined. The first author then coded the remaining 16 articles.

Sample description

The 31 articles included in the current analysis appeared in peer reviewed publications between the years 1991 and 2011 (see Table 1). Female victims were disproportionately represented with 37 (94.9%) effect sizes. Eighteen (46.2%) effect sizes represented pregnant victims. Thirty-two (82.1%) effects reported physical or sexual violence, 2 (5.1%) reported only psychological aggression, and 5 (12.8%) reported violence in an unspecified domain. Ten (25.6%) of the effects reflected married samples, 9 (23.1%) reflected dating samples and the remaining 20 (51.3%) specified no majority relationship type. Effect sizes were divided between Caucasian (48.7%), African American (17.9%), Latino (5.1%), and unspecified (28.2%) samples. Twelve (30.8%) effects were gathered from participants with economic hardship, 12 (30.8%) were gathered from participants with no reported economic hardship, and 15 (38.5%) effect sizes could not be associated with an SES variable. The majority of the effects related to smoking (92.3%) represented current smoking or smoking that occurred during pregnancy whereas 7.7% reflected a lifetime history of smoking.

Effect Size Data

The freeware version of the Microsoft Excel Effect Size Computation Program was used to calculate Cohen's *d* values as a measure of effect size in the current meta-analysis (Wilson, 2010). Cohen's *d* represents the degree to which two sample means differ divided by a pooled standard deviation. Small effects are reported when *d* falls between .2 and .5, medium effect sizes range from .5 to .8, and large effects are detected when *d* exceeds .8 (Cohen, 1992). Here, larger positive values of *d* indicated that victims of IPV were proportionally more likely to be smokers or to smoke more, on average, than non-victims.

When distinct data was presented for males and females, pregnant and non-pregnant samples, or various ethnic groups within a single study, we calculated and retained multiple, independent effect sizes for use in the meta analysis. Data that allowed for the calculation of multiple effect sizes within a distinct group were averaged to produce a single effect size and prevent violation of the independence assumption. Effect sizes reflect the presence or

absence of IPV and smoking. Each effect size was derived from reported *d*, proportions, or converted odds ratios (Chinn, 2000) and weighted by sample size according to the recommended Hedges and Olkin (1985) procedure. An overall effect size for the IPV-smoking relationship was then calculated using the weighted individual effect sizes and tested for homogeneity (Q_w; Lipsey & Wilson, 2001). Between group tests of homogeneity (Q_b) were conducted using random effects models to conservatively determine which moderators accounted for observed heterogeneity using the suggested ANOVA analogue methodology (Field, 2003). Significant results indicated that effect sizes significantly differed across the categories of the moderator.

Results

Grand Effect Size

Thirty-nine effect sizes gathered from 31 studies were used in a random effects analysis to calculate the overall effect size describing the relationship between IPV victimization and cigarette smoking (see Table 1). With $d = .41$ (k = 39, $p < .001$; 95% CI = .35–.47), the association was small to medium in magnitude (Table 1). A failsafe N of 43 was calculated to compensate for publication bias. Thus, the current effect size would be reduced to the $d = .2$ level if 43 investigations found an effect size of zero between victimization and smoking.

An analysis of homegeneity indicated significant variability among the effect sizes included within the analysis ($Q(38) = 740.79$, $p < 0.001$). We recalculated the overall effect size following the elimination of the two most extreme outliers ($d = .14$; Berenson, Stiglich, Wilkinson, & Anderson, 1991; $d = 1.63$; Berenson, San Miguel, & Wilkinson, 1992) to find that the magnitude of association remained small to moderate ($d = .41$, k = 37, $p < 0.001$; 95% CI = .35 – .47) and that heterogeneity changed only slightly ($Q(36) = 729.69$, $p < 0.001$). Ultimately, all 39 effect sizes were included in subsequent analyses to allow for an unbiased representation of the relationship between victimization and smoking.

Moderator Analyses

Pregnancy—After the elimination of two effect sizes that represented the relationship between victimization and smoking among males, the distinction between composite effect sizes for pregnant ($d = .49$, k = 18, $p < 0.001$; 95% CI = .38–.59) and non-pregnant (d = .37, k = 19, $p < .001$; 95% CI = .28–.46) participants approached significance as indicated by homogeneity testing ($Q_b(1) = 2.76$, $p = 0.09$). Thus, the strength of the association between victimization and smoking was marginally greater, approaching a medium effect size, within samples of pregnant compared to non-pregnant participants. Additionally, neither pregnant ($Q_w(17) = 26.52$, $p = .07$) nor non-pregnant ($Q_w(18) = 10.21$, $p = .92$) participants demonstrated significant individual effect size variability, though the pregnant group displayed marginal within group variability, suggesting that additional factors would be required to explain the variability across effect sizes in pregnant samples.

Relationship—Ten effect sizes indicated a small to medium overall relationship between victimization and smoking among married samples ($d = .39$, $p < 0.001$; 95% CI = .24–.53). Nine effect sizes also produced a small to medium composite effect size for the relationship between victimization and smoking among dating samples ($d = .41$, $p < 0.001$; 95% CI = .26–.57). Thus, victimization and smoking behavior were comparably associated in dating and married samples ($Q_b(1) = 0.08$, $p < 0.78$). Composite effect sizes for both married ($Q_w(9) = 6.12$, $p = 0.73$) and dating ($Q_w(8) = 10.58$, $p = 0.23$) victims failed to demonstrate significant heterogeneity.

Ethnicity—Nineteen studies were analyzed to estimate the composite relationship between victimization and smoking among Caucasians (d = .45, p < 0.001; 95% CI = .37–.54). The composite effect size for African Americans was calculated from 7 effect sizes (d = .31, p < 0.001; 95% CI = .16–.46). Only two effect sizes were used to calculate the Latino composite (d = 1.26, p < 0.001; 95% CI = .71–1.82). The effect sizes for the three ethnic categories differed from one another ($Q_b(2) = 11.52$, $p < 0.01$). There existed no significant variability within the Caucasian ($Q_w(18) = 23.96$, $p = 0.16$), African American ($Q_w(6) = 2.06$, $p = 0.91$), or Latino ($Q_w(1) = 1.08$, $p = 0.30$) categories, indicating homogeneity within respective subgroups. We then eliminated the Latino effect sizes and determined that the relationship between victimization and smoking remained marginally larger among primarily Caucasian samples compared to primarily African American samples ($Q_b(1) = 2.77$, $p = 0.09$).

Socioeconomic Status—Studies consisting primarily of participants with economic hardship produced a comparable composite effect size between victimization and smoking (d = .43, k = 12, p < 0.001; 95% CI = .31–.56) compared to the composite effect size generated by studies with participants lacking economic hardship (d = .38, k = 12, p < 0.001; 95% CI = .28–.49) as evidenced by a test of homogeneity (Q_b (1) = 0.33, p = 0.57). Hardship effect sizes were not homogeneous ($Q_w(11) = 23.78$, $p = 0.01$), unlike thee effect sizes for those without hardship ($Q_w(11) = 5.05$, $p < 0.93$).

Methodological Factors—We examined methodological factors and observed no effect size differences between past-year (d = .42, k = 24, p < .001, 95 % CI = .35–.50) and lifetime (d = .39, k = 14, p < .001, 95 % CI = .30–.47) IPV, Q_b (1) = 0.50, p = .48; or among assessment settings (medical facility, d = .41, k = 19, p < .001, 95 % CI = .32–.51; academic setting, d = .27, k = 6, p = .002, 95 % CI = .10–.45; survey d = .45, k = 13, p < .001, 95 % CI = .35–.55), Q_b (2) = 2.93, p = .23.

Discussion

The results of the current meta analytic review suggested that the overall relationship between IPV victimization and smoking is small to medium with a composite effect size of d = .41. Thus, across the collected literature, victims of IPV are significantly more likely than non-victims to engage in smoking behavior. The magnitude of the effect size is comparable to that of other victimization-substance use relationships found within the IPV literature (e.g. Moore et al., 2008). Moderator analyses detected a trend towards significance based upon pregnancy status and ethnicity. The magnitude of the relationship between victimization and smoking was not moderated by relationship type or SES. Our ability to examine the influence of gender and violence type was compromised due to limited data.

As discussed earlier, theory and preliminary evidence suggest that nicotine may serve as a maladaptive coping strategy associated with the reduction of negative affect and anxiety related to IPV as well as other stressors. Drawing upon previous threshold models (e.g. Klostermann & Fals-Stewart, 2006), IPV-related smoking may be more likely to result when victimization contributes to a constellation of stressors, exceeding the individual's ability to inhibit the urge to smoke. This stress threshold would be lower for individuals who lack alternative coping skills and higher for those who possess prosocial means to cope with stress. Other factors, such as previous experience with the anxiolytic or anti-depressant effects of nicotine, could also contribute to the level of stress that each individual requires to cross a smoking threshold.

Pregnancy is a particularly distressing period of physical change and psychological stress. The relationship between victimization and smoking is moderately stronger among pregnant

than non-pregnant victims. This association requires additional attention as smoking is associated with both negative health outcomes for mothers and an increased risk of multiple negative birth defects in the fetus, including low birth weight and poor lung development (Lannero, Wickman, Pershagen, & Nordvall, 2006). Further, IPV victims are more likely than non-victims to be socially isolated (e.g., Lanier & Maume, 2009). Recent research shows that pregnant smokers with low social support are more likely than those with high social support to report depression, experience complications, and give birth prematurely (Elsenbruch et al., 2007). It is important to investigate the influence of IPV on smoking patterns during pregnancy.

The association between victimization and smoking was marginally stronger within Caucasian compared to African American samples. Few studies reported samples that were ethnically distinct. It is possible that, with a greater number studies identifying risk within ethnic groups, ethnicity may significantly moderate the relationship between victimization and smoking. With only two effect sizes drawn from Latino samples, there were too little data from which to draw conclusions. Further research on ethnic differences in the relationship between experiencing IPV and smoking is recommended, particularly within the Latino community.

Unlike ethnicity, economic hardship did not share a trend toward significance, despite ethnic disparities in economic status. The above analyses suggest that the magnitude of the victimization-smoking relationship is robust across both ethnic and economic subgroups. The absence of an SES modifier indicates that low SES is not a spurious factor capable of explaining the relationship between IPV victimization and smoking (Chuang, Cubbin, Ahn, & Winkleby, 2005). While smoking is associated with lower SES (CDC, 2011), IPV may represent a sufficiently traumatic or distressing occurrence to prompt crossing the aforementioned smoking threshold or to maintain smoking behaviors regardless of SES status.

Factors contributing to the severity of IPV and potential stress resulting from victimization failed to affect the victimization-smoking relationship in the anticipated directions. For example, the magnitude of the relationship was comparable across dating and married samples. The strength of the relationship between victimization and smoking may partially result from the stress associated with IPV perpetration and remaining involved in a dissatisfactory relationship. Archer (2000) noted that IPV is often bidirectional and others have reported that female samples selected for victimization do not significantly differ from those selected for perpetration in terms of aggressive responding (Sullivan, Titus, Holt, Swan, Fisher, & Snow, 2010). Further, ample evidence suggests that partners in violent relationships report higher levels of relationship distress and dissatisfaction than non-violent dyads (for a review, see Stith, Green, Smith, & Ward, 2008). It should be noted that our literature review yielded only two studies that provided the necessary data to calculate an effect size between IPV perpetration and smoking behavior (Easton, Weinberger, & McKee, 2008; Rhodes et al., 2009) highlighting the need for research examining the relationship between IPV perpetration and smoking and how this relationship compares to that found for IPV victimization.

The directionality of the relationship between IPV and the various substances associated with increased victimization is unclear. Research and theory suggest that IPV may be a precipitant to cigarette smoking (e.g., Schneider, Burnette, Ilgen, & Timko, 2009). Unlike alcohol, cannabis, and cocaine intoxication, heavy nicotine use has not been causally linked to acute psychological difficulties and lacks the proximal effects that substantially impair inhibition or executive functioning (Giancola, 2000; Hoaken & Stewart, 2003). Research is needed to test causal links between IPV experience and cigarette smoking. It is evident,

however, that the relationship shared between cigarette use and IPV victimization is comparable to observed associations between victimization and other drugs of abuse as reported by Moore and colleagues (2008), such as cocaine (d = .59, CI = .48–.70) and marijuana (d = .18, CI = .02–.33).

The current review has the potential to impact IPV treatment in a number of ways. First, information about smoking should be added to current psychoeducational materials presented to victims of IPV through intervention programs. Victim advocates and medical providers are ideally suited to discuss the consequences of smoking, motivation to quit smoking, and smoking cessation treatments with IPV victims. In fact, it has been suggested that there are important benefits of smoking cessation services being provided by health care providers such as a strong therapeutic alliance and an on-going relationship that allows for the monitoring of smoking and mood during and after a quit attempt (Ziedonis et al., 2008). The Clinical Practice Guidelines for Treating Tobacco Use and Dependence (Fiore et al., 2008) advanced recommendations for first-and second-line smoking interventions that can be incorporated by health care professionals who routinely interact with IPV victims. The guidelines recommend that health care professionals administer the 5 A's: 1) *Ask* patients if they are current or former smokers, 2) *Advise* patients who are current smokers to quit, 3) *Assess* patients' motivation level for quitting, 4) *Assist* with quit attempts, and 5) *Arrange* follow-up contacts. Beyond preventative care, victims may also benefit from motivational enhancement efforts (e.g., Miller & Rollnick, 2002) that encourage both autonomy from perpetrators and future non-violent relationships along with smoking abstinence. Although the 5-As may represent the ideal healthcare intervention strategy across patients, IPV victims represent a special subset of the population whose heightened physical risk may require an adapted protocol. Care should be taken to not increase the victim's risk by removing a potential coping strategy. Without further study, healthcare workers should implement the 5-As only when the victim is no longer in immediate physical danger or when sufficient alternative coping strategies may be developed. Further, healthcare personnel must avoid alienating the victimized smoker, which may further harm self-esteem and prevent him or her from seeking medical services in the future (McFarlane et al., 1996).

The most significant limitation of the current review was a lack of studies that provided data on smoking and IPV victimization. Similarly, moderator analyses were limited due to the number of investigations that have reported researching IPV in male victims, victims of psychological abuse, and Latino victims. Additional research is needed to develop a better understanding of the relationship between smoking and IPV for these important populations. Although we examined several methodological variables that could moderate the association between victimization and smoking reported within individual studies, it should be noted that additional study-level factors may be of interest in better understanding the magnitude of the primary relationship. Study level data may also help explain the magnitude of moderator analyses. Age differences, in particular, may account for some of the variation in effect sizes between pregnant and non-pregnant samples; however, reporting inconsistencies across studies prevented the examination of the age effect in this study. The relationship of age to IPV and smoking in pregnant and non-pregnant samples would be an important area of future research. Additionally, based on the limited smoking data included in many published reports, we used a dichotomous variable to indicate smoking (smoker versus non-smoker) and were unable to examine differences by IPV experience related to other smoking variables such as smoking frequency, quantity of cigarettes consumed per day, or age of smoking onset.

In conclusion, smoking represents a significant long term health risk and is associated with IPV victimization. High base rates of IPV in the general population combined with a small to medium effect size between victimization and smoking translate into hundreds of

thousands at an increased risk of smoking in the United States alone. There exists a dearth of literature on the theoretical and practical association between victimization and smoking but this relationship is consistent with the hypothesis that smoking is secondary to insufficient coping following the stress, depressed mood, and anger associated with IPV victimization. Although the self-medication model may offer one theoretically-based interpretation of the observed relationship between victimization and smoking, the current design is incapable of evaluating the model. Alternative explanations, such as the possibility that the affective and behavioral correlates of nicotine addiction and withdrawal may increase the smoker's likelihood of victimization (e.g. Boles & Miotto, 2003), must be considered. Future research should further evaluate the self-medication hypothesis in the victimization-smoking relationship and work toward establishing a firm theoretical framework describing the relationships between IPV victimization, stress, coping skills, smoking, and poor health outcomes. Given the health problems associated with smoking, it is possible that cigarette use may moderate the relationship between IPV and many of the health issues that disproportionately affect victims. Exploring the precipitants to and consequences of smoking within victims may be essential to understanding the association between victimization and long term health problems.

Critical Findings and Implications for Practice, Policy, and Research

Victims of IPV are at greater smoking risk than non victims (d = .41, 95% CI = .35–.47). The victim-smoking association is marginally stronger in pregnant compared to non pregnant participants. Based on the articles available to the review, the strength of the victimization-smoking relationship did not differ by relationship type or ethnicity. Socioeconomic status does not moderate the IPV victimization-smoking relationship. The unavailability of data prevented the examination of gender and the type of violence perpetrated as potential moderators of the victimization-smoking relationship. Future research should investigate the influence of IPV on smoking cessation during pregnancy. Further research on ethnic differences in the relationship between experiencing IPV and smoking is needed, particularly within the Latino community. Adding screenings and information about smoking cessation to current materials and interventions presented to IPV victims may be useful for improving health outcomes. It is recommended that all health care professionals complete the 5 A's (Fiore et al., 2008) with IPV victims when the intervention is considered safe and help victims develop alternative and healthy coping strategies. Exploring the precipitants to and consequences of smoking, the leading preventable cause of U.S. mortality, within IPV victims may be essential to better understand the association between victimization and long term health problems and to direct efforts to improve the health of adults who have experienced IPV victimization.

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Table

Intimate Partner Violence Victimization and Cigarette Smoking

Study	Sample Size	Gender	Ethnicity	Pregnant	Effect Size *d*
Ackerson, Kawachi, Barbeau, & Subramanian, 2007	89,092	Female	---	No	.166
Bailey & Daugherty, 2007	104	Female	CA	Yes	.468
Berenson, San Miguel, & Wilkinson, 1992	153	Female	CA	Yes	.328
	73	Female	LA	Yes	1.632
Berenson, Stiglich, Wilkinson, & Anderson, 1991	238	Female	CA	Yes	.144
	141	Female	AA	Yes	.433
	120	Female	LA	Yes	1.028
Black & Breiding, 2008	27,590	Male	---	No	.355
	42,566	Female	---	No	.460
Bonomi et al., 2006	3,429	Female	CA	No	.463
Bonomi et al., 2009	1,928	Female	CA	Yes	.518
Coker, Sanderson, & Dong, 2004	755	Female	AA	No	.506
Cokkinides & Coker, 1998	6,244	Female	---	Yes	.634
Curry, 1998	1,208	Female	CA	No	.339
	393	Female	AA	No	.206
Dichter, Cerulli, & Bossarte, 2011	21,162	Female	CA	No	.569
Gao et al., 2010	828	Female	---	Yes	.283
Janssen et al., 2003	4,750	Female	CA	Yes	1.119
Jun, Rich-Edwards, Boynton-Jarrett, & Wright, 2008	13,731	Female	CA	No	.224
	15,535	Female	CA	No	.275
Lemon, Verhoek-Oftedahl, & Donnelly, 2002	1,687	Female	CA	No	.402
	1,494	Female	CA	No	.162
Leung, Leung, & Lam, 1999	631	Female	---	Yes	.365
Lipskey, Holt, Easterling, & Critchlow, 2003	3,478	Female	---	Yes	.473
Martin, English, Andersen-Clark, Cilenti, & Kupper, 1996	2,092	Female	AA	Yes	.259
Martin, Andersen-Clark, Lynch, & Kupper, 1999	604	Female	AA	Yes	.347
McFarlane, Parker, & Soeken, 1996	1,203	Female	---	Yes	.231
McNutt, Carlson, Persaud, & Postmus, 2002	557	Female	CA	No	.324
Pico-Alfonso, Garcia-linares, Celda-Navarro, Herbert, & Martinez, 2004	162	Female	---	No	.252
Rhodes, Houry, Cerulli, Straus, Kaslow, & McNutt, 2009	595	Male	AA	No	.206
Silverman, Raj, Mucci, & Hathaway, 2001	1,977	Female	CA	No	.690
	2,186	Female	CA	No	.444
Vest, Catlin, Chen, & Brownson, 2002	18,415	Female	CA	No	.445
Webster, Chandler, & Battistutta, 1996	772	Female	CA	Yes	.628
Weinbaum, et al., 2001	2,983	Female	CA	No	.435
Weimann, Agurcia, Berenson, Volk, & Rickert, 2000	724	Female	---	Yes	.476

Study	Sample Size	Gender	Ethnicity	Pregnant	Effect Size *d*
Widding-Hedin & Janson, 2000	207	Female	CA	Yes	.683
Woods, Page, O'Campo, Pugh, Ford, & Campbell, 2005	101	Female	AA	No	.223
Yoshihama, Horrocks, & Bybee, 2010	1,284	Female	---	No	.255
OVERALL	271,192	.409 (95% CI = .348–.470)			

Key: CA, Caucaisan; LA, Latino/a; AA, African-American

Note: --- refers to studies in which there was no clear ethnic majority in the samples (>60% belonging to one ethnic group), no effect size was calculated; studies with multiple rows indicate that >1 effect size was calculated from the data for that study.