# SUBSTANCE USE AND INTIMATE PARTNER VIOLENCE: A META-ANALYSIS

by

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B.S., Walla Walla University, 2004
M.Div., Andrews University, 2009
M.S., Northern Illinois University, 2012

AN ABSTRACT OF A DISSERTATION

submitted in partial fulfillment of the requirements for the degree

DOCTOR OF PHILOSOPHY

School of Family Studies and Human Services
College of Human Ecology

KANSAS STATE UNIVERSITY
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# Abstract

This meta-analysis used data from 285 studies (yielding 983 effect sizes and a combined sample size of 627,726) to quantitatively evaluate the link between substance use and physical intimate partner violence (IPV) perpetration and victimization. Results indicated that overall substance use, alcohol use, and drug use were significantly related to both IPV perpetration and victimization, with mean effect sizes ranging from *r* =.18 to .23. Moderator analyses also compared males and females for overall substance use, alcohol use, and drug use; subcategories of alcohol use and drug use; and different types of drugs, for males and females, and for IPV perpetration and for victimization. This is the first meta-analysis to compare alcohol versus drug use for IPV perpetration and IPV victimization. The analyses revealed drug use to be a significantly stronger risk marker for victimization, and a non-significantly stronger risk marker for perpetration, compared to alcohol use. Alcohol consequence measures (i.e., abuse and dependence) were significantly stronger risk markers than consumption measures for IPV victimization, but non-significantly different for IPV perpetration. Furthermore, more frequent alcohol use (few times a week, almost daily, and daily) was a significantly stronger risk marker for perpetration compared to other alcohol frequency measures. Drug consequence measures (abuse/dependence) were significantly stronger risk markers for perpetration than simply drug use measures. There were no significant differences between different drug types, and no significant difference between stimulants versus non-stimulants for IPV perpetration and victimization (though these smaller comparisons may have been underpowered, and thus unable to detect differences). The findings of this study are important because they provide the most comprehensive and detailed analysis of the link between substance use and IPV to date.

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# Chapter 1 - Introduction

Physical intimate partner violence (IPV) and substance use permeate all levels of society and are considered major health concerns worldwide (World Health Organization, 2014). Research has shown that IPV and substance use co-occur in almost every culture, class, region, and country (e.g., Allegra, 2012; Duvvury, Callan, Carney, & Raghavendra, 2013; Eng, Li, Mulsow, & Fischer, 2010; Pandey, Dutt, & Banerjee, 2009; Robbins, 2002; Shorey et al, 2012; Testa, 2012). Substance use and IPV each have costly emotional, physical, and psychological effects on individuals, families, communities, and the larger society (e.g., Boles & Miotto, 2003; Leonard, 2001; World Health Organization, 2014).

Physical intimate partner violence has been defined as any physical force which could harm, injure, or cause disability or death, and includes (but is not limited to) grabbing, pushing, shoving, scratching, slapping, punching, throwing, choking, shaking, burning, using a weapon, or forcefully restraining an intimate partner (Saltzman, Fanslow, McMahon, & Shelly, 1999; for a discussion as to the typologies of IPV, see Johnson, 2004). The World Health Organization estimates that almost a third of women across the globe will become victims of IPV over the course of their lifetime (World Health Organization, 2014). Although reported incidents of IPV per year in the United States decreased 25% (from 1 million to around 750,000) between 2004 to 2013, there was an 8% increase (from 334,620 to 360,820) in reported incidents of severe IPV per year during this same time period (Bureau of Justice Statistics, 2014). Furthermore, around 20% of all violent crime reported in the United States are acts of intimate partner violence (Bureau of Justice Statistics, 2014).

IPV negatively impacts the victim's (and family's) physical and psychological health, but there are also substantial economic consequences (Duvvury, Callan, Carney, & Raghavendra,

2013). Not only can recovery (e.g., physical, trauma) be costly for the victim and his/her community, but victims may temporarily miss work, which negatively affects their productivity (an important component of economic growth) and could limit their future work opportunities (Duvvury, Callan, Carney, & Raghavendra, 2013; World Health Organization, 2014). Thus, IPV contributes—especially in poorer communities—to current and future economic hardship for the victim, their family, their community, and their country (Duvvury, Callan, Carney, & Raghavendra, 2013; World Health Organization, 2014).

Substance use (which can range from use to addiction) is also a worldwide behavior resulting in severe socioeconomic consequences, is associated with over 200 different detrimental health conditions (World Health Organization, 2014a), and has been linked to IPV perpetration and victimization. Different substances may have differing effects on IPV based on a person's gender, history, psychology, cultural factors, etc. (e.g., Boles & Miotto, 2003). It is estimated that over one third of the global population has had an alcoholic drink in the past year, with the average consumption being 13.5 grams of pure alcohol (about one standard drink in the USA) consumed per person per day (World Health Organization, 2014a). Similarly, the United Nations Office on Drugs and Crime (UNODC) estimated that up to 250 million people globally used an illicit substance in 2007 (UNODC, 2009). Although not all substance users experience IPV, research suggests that 36% of victims seeking support for IPV and 61% of perpetrators in batterer intervention programs have substance abuse/dependence problems (Collins & Spencer, 2002). Furthermore targeting substance use has been shown to substantially reduce IPV recidivism (O'Farrell, Fals-Stewart, Murphy, & Murphy, 2003; Stuart et al., 2003), which suggests integrating substance abuse treatment would benefit batterer intervention programming (Stuart, Temple, & Moore, 2007). If we are to effectively intervene in the lives of perpetrators

and victims of IPV, the link between substance use and IPV warrants national and international attention.

In the last decade, research on substance use and IPV has grown exponentially, so an updated meta-analytic study has the potential to sharpen our understanding of the existing empirical evidence linking substance use with physical IPV. The purpose of this current study is to meta-analyze a variety of substances (cocaine, marijuana, heroin, alcohol, methamphetamine, stimulants, non-stimulants, and overall substance use) as risk markers for IPV male perpetration, female perpetration, female victimization, and male victimization.

# Chapter 2 - Literature Review

## Linking Substance Use and Intimate Partner Physical Violence

Although many people who use alcohol and drugs never perpetrate violence, substance use is prevalent among both perpetrators and victims (Boles & Miotto, 2003; Foran, 1990). Intimate partner violence (IPV) research has mainly focused on male's and female's substance use with male-to-female violence (Crowne et al., 2012; Murphy et al., 2001; Stuart & Holtzworth-Monroe, 2005; Ting et al., 2009), yet a growing number of studies have also linked substance use to female-to-male IPV (e.g., Stuart et al., 2008; Testa et al., 2012). However, it is recognized that various substances may have different linkages to violence (Lavine, 1997), which begs the question: Are there significant differences in the strength of the links between various substances (e.g., alcohol, drugs, drug types) and IPV perpetration or victimization?

### Alcohol Use and IPV

Compared to other substances, alcohol has been the most prominently linked to violence (e.g., Martin, 1993; Parker & Rebhun, 1995). Research suggests that roughly 20% to 50% of reported cases of IPV involve alcohol use (e.g., Leonard, 2001; Kaufman-Kantor & Straus, 1987, Roizen, 1993). High rates of alcoholism have been found among perpetrators of IPV (e.g., Greenberg, 1981; Reiss & Roth, 1993), and compared to others, alcoholics/problem drinkers are more likely to have histories of violence (e.g., Reiss & Roth, 1993, Schuckit & Russell, 1984). However some advocacy groups are leery of recognizing how alcohol might contribute to the perpetration of IPV, for fear of shifting the blame from the perpetrator onto the alcohol consumed (Foran & O'Leary, 2008).

Most research has followed the trend of linking alcohol use with male perpetration (Allegra, 2012; Kyriacou et al., 1999; Snow et al., 2006; Tong, 2003) or female victimization

(Buchanan, 2006; Cattaneo et al, 2007; Li, 2006; Nathanson et al., 2012). Yet, there are a substantial number of recent studies measuring the link between alcohol use and female perpetration (Keller et al., 2009; Kelly & Halford, 2006; Shorey et al, 2012) or male victimization (Drapkin et al., 2004; Heru et al., 2006; Mair et al., 2012; Testa, 2012). The strength of the link between alcohol use and IPV sometimes varies by gender (e.g., Foran & O'Leary, 2008) and sample type, as stronger associations have been found in clinical populations compared to non-clinical populations (e.g., Foran & O'Leary, 2008; Rosenbaum & O'Leary,1981). Furthermore, results from some alcohol intervention studies show that successfully reducing alcohol use is associated with a reduction in perpetration of IPV (O'Farrell, Fals-Stewart, Murphy, & Murphy, 2003; O'Farrell & Murphy, 1995), which suggests that attending to alcohol use may be beneficial for batterer intervention (e.g., Martin, 1993; Parker & Rebhun, 1995).

**Drug Use and IPV**

Compared to alcohol, the association between different drugs and IPV is much less understood, as many studies do not typically distinguish between different types of illicit drug use (e.g., Abrahams, 2006; Buchanan, 2006; Hines & Douglas, 2011; Kyriacou et al., 1999; Vieira et al., 2011; Zhang et al., 2012).

Similar to the research on alcohol use, the link between drug use and IPV has predominantly focused on female drug users as victims of IPV (e.g., Burke et al., 2005; Clark & Foy, 2000; Panchanadeswaran et al., 2010; Raghavan et al., 2006) and male drug users as perpetrators of IPV (e.g., Aldarondo & Kantor, 1997; Chan et al., 2008; Kaslow et al., 1998; Kyu & Kanai, 2005). However, a growing number of studies have also begun to link drug use with

female perpetration (Fergusson et al., 2008; Robbins, 2002; Shorey et al., 2012) and drug use with male victimization (Chase et al., 2003; Fergusson et al., 2008; Stuart et al., 2008).

Stimulants, such as cocaine or methamphetamines, and non-stimulants, such as heroin and marijuana, have been associated with men's and women's perpetration and victimization (Bennett et al, 1994; Brewer et al., 1998; Burke et al., 2005; Chase et al., 2003; Gilbert et al., 2012; Martino et al., 2005; Reingle et al., 2012; Schneider et al., 2009; Walton et al., 2009). Compared to other drugs, cocaine seems to have the strongest link to the perpetration of IPV (e.g., Afifi et al., 2012, Boles & Miotto, 2003; Foran et al., 2008), but part of this consensus may be due to the variability in findings associated with other drugs. For example, marijuana has been significantly, and other times non-significantly, associated with IPV (e.g., Goldstein, 1985, Kramer et al., 2012, Lockie, 2011, Moore & Stewart, 2005; Schneider et al., 2009). There is a possibility that other drugs (e.g., heroine) may actually decrease the likelihood of perpetration and victimization (e.g., Goldstein, 1985; Schneider et al., 2009), though this may not always be the case (e.g., Brewer et al., 1998, Burke et al., 2005). Because of the variability of findings for different drug types, there is still no empirical consensus regarding which drugs are most strongly linked to IPV. Thus, the linkage between substance use and IPV could differ by drug type (perhaps due to the resulting pharmacological, physiological, and behavioral effects; see Stuart et al., 2008), but also by gender and sample type (e.g., Afifi et al., 2012, Brewer et al., 1998; Foran et al., 2008, Moore & Stuart, 2005).

This lack of consensus regarding the link between different drugs and IPV, combined with the more prominently studied association between alcohol use and IPV, raises questions about whether drug use or alcohol use is the stronger risk marker for IPV perpetration and victimization. No meta-analytic studies have specifically compared alcohol use to drug use as

risk markers for IPV. A comprehensive empirical analysis of the link between substance use and IPV would be beneficial, especially if this analysis could compare alcohol use and drug use, explore gender differences, and test different drug types, and different ways of measuring substance use.

## Previous Substance Use and IPV Meta-Analyses

Six previous meta-analytic reviews (Ferrer et al., 2004, Foran & O'Leary, 2008; Golding, 1999; Hotaling & Sugarman, 1986; Moore, Stuart, Meehan, Rhatigan, Hellmuth, & Keen, 2008; Schumacher, Feldbau-Kohn, Smith Slep, & Heyman, 2001; Stith et al., 2004) have measured the link between substance use (predominantly alcohol) and physical intimate partner violence (see Table 1 in Appendix A for summary of previous meta-analyses' characteristics). The resulting effect sizes ranged from inconclusive to large, with large heterogeneity between studies, which suggests the presence of moderating variables.

The first meta-review looking at the empirical link between substances and IPV was conducted by Hotaling and Sugarman (1986) and included articles reporting a variety of risk markers associated with husband-to-wife violence. They found 3 of the 5 (60%) studies reporting wives' drug use showed a positive relationship to female victimization, whereas only 1 of 6 studies (17%) reported wives' alcohol use as positively related to their victimization, yet found 7 of 9 studies (78%) measuring husband's alcohol use reported a positive relationship to male perpetration (Hotaling & Sugarman, 1986).

In his meta-analysis looking at a variety of mental health factors associated with female victimization, Golding (1999) included 4 drug use/abuse effect sizes and found the women who used substances were over five and a half times more likely to be abused (OR 5.62, 95% CI = 3.55, 7.72) compared to women who did not use drugs. Similarly, using data from 10 alcohol

use/abuse effect sizes Golding (1999) found that alcohol using women were also five and a half times more likely (OR 5.56, 95% CI = 3.32, 9.31) to experience victimization compared to women who did not use alcohol.

Schumacher and colleagues' (2001) meta-analysis surveyed several factors associated with IPV, including alcohol problems and drug problems (14 studies and 19 effect sizes). The heterogeneous effect sizes for alcohol problems or drug problems ranged from $r$ = .21 to .65 for male perpetration and from $r$ = -.08 to .41 for female victimization (which are small to large effect sizes; Cohen, 1992). Due to their inclusion and exclusion criteria, no effect sizes were included for male victimization or female perpetration.

Ferrer and colleagues' (2004) meta-analysis included 12 total substance use studies (20 effect sizes) and found an effect size of $d$ = 0.57 for alcohol abuse/dependence and male perpetration of IPV (a medium effect size; Cohen, 1992). They also included six effect sizes for drug abuse/dependence with no distinction between drug type, and found the relationship with male perpetration of IPV to be $d$ = 0.51 (a medium effect size; Cohen, 1992; Ferrer et al., 2004).

Stith and colleagues' (2004) meta-analysis also reported a variety of risk markers associated with IPV perpetration and victimization, and were able to include 27 studies (38 effect sizes) that measured substance use. They found the link between male alcohol use and male perpetration to be $r$ =.24, $p$ < .001, female alcohol use and female victimization to be $r$ =.13, $p$ < .001, and male illicit drug use (5 studies) and male perpetration to be $r$ = .31, $p$ < .001 (small to medium effect sizes; Cohen, 1992; Stith et al, 2004). Because of the limited studies available, they only measured drug use with male perpetration and lacked enough effect sizes to meta-analyze alcohol use with female perpetration or male victimization.

The alcohol and IPV meta-analysis conducted by Foran and O'Leary (2008) was the first to include enough studies (50 studies and 55 effect sizes) to calculate a mean effect size for both male and female perpetrators. They found the mean effect size to be moderate for male perpetrators ($r$ = .23; 95% CI =.21, .24) and small for female perpetrators ($r$ =.14; 95% CI =.08, .20). Foran and O'Leary also found significantly stronger effect sizes from clinical vs. non-clinical samples ($r$ = .33) compared to community samples ($r$ = .19), and significantly stronger effect sizes when measuring alcohol abuse/dependence ($r$ = .33) compared to less acute alcohol usage ($r$ = .21; small to medium effect sizes).

The final meta-analysis is the most comprehensive to date and measures the link from various drugs used to psychological, physical, and sexual intimate partner violence (Moore et al., 2008). Moore and colleagues included 67 studies (319 effect sizes) and found the link between substance use and physical IPV to be $d$ = .34 (95% CI =.32, .37; a small effect size; Cohen, 1992). They also reported the effects of different types of drugs on physical IPV for each gender for victimization and perpetration. For example, they found that cocaine had the strongest association with perpetration of physical IPV, and discovered widely varying results for marijuana ($d$ = from -.52 to .35) and perpetration of IPV. They found that the link between substance use and physical IPV was significantly stronger for female victimization ($d$ = .49) than for male victimization ($d$ = .14), but that this link was significantly stronger for male perpetration ($d$ = .49) than female perpetration ($d$ = .26; small and medium effect sizes; Cohen, 1992, Moore et al., 2008). They also found the strength of the effect size of male cocaine use and male perpetration to be almost identical to male marijuana use and male perpetration. However because they lacked effect sizes linking specific drugs to female perpetration (only one effect size for female marijuana use and female perpetration), they were unable to test for gender

differences with regard to perpetration or the potential differences between female perpetration and female victimization (Moore et al., 2008).

**Previous Meta-Analyses' Limitations**

Due to limited studies available, these previous meta-analyses (with the exception of Moore et al., 2008) were not able to meta-analyze the strength of specific substances to male victimization and female perpetration. Therefore, they were unable to test for significant differences for substances used with male perpetration, female perpetration, male victimization, and female victimization. Because of their inclusion/exclusion criteria, some of these meta-analyses chose not to include unpublished studies and/or did not include many effect sizes from international studies—thus most of these findings are limited to the relationship between substance use and IPV in the United States. Furthermore, these meta-analyses measured alcohol use and drug use as two of many risk markers associated with IPV, or they only measured alcohol use or only drug use (but not both). Thus, we do not know the overarching relationship between substance use (alcohol and drug use) and IPV on a meta-analytic level, nor do we know whether alcohol use or drug use is the stronger risk marker.

In order to be more inclusive, Moore and colleagues (2008) meta-analysis included studies which only reported multivariate effect sizes for substance use and intimate partner violence (psychological, physical, and emotional). However, they found that the bivariate substance use effect sizes were significantly different from their included multivariate effect sizes (Moore et al., 2008). Thus, the inclusion of multivariate effect sizes which control for several other vairables may have significantly altered the aggregated "bivariate" effect size for various substances with physical IPV. Furthermore, Moore and colleagues included several studies representing dating adolescent/university samples (which is a related, yet slightly

different field of study compared to adults in a committed married/cohabiting relationship; Johnson & Ferraro, 2000; Lloyd & Emery, 2000), and this inclusion may have also affected their mean effect sizes. Finally, because of the limited number of female perpetration and male victimization effect sizes, most of the previous meta-analyses were unable to test for significant gender differences on the link between substance use and IPV.

## Theoretical Links Between Substance Use and IPV

Despite decades of research, there is no clear consensus about the precise nature of the causal link—and in some cases, whether there even is a link—between substance use and IPV (Fagan, 1990). An updated meta-analysis combining the current research, which is sensitive to different types of substances used by males and females, would further our understanding of the link between substances and IPV.

### Spurious Models

Some scholars have seriously questioned whether there really *is* a causal link between substance use and IPV. Those in support of a spurious model suggest that factors (other than substance use) at the individual, relational, or cultural levels are what drive the perpetration of IPV (Center for Substance Abuse Treatment, 1997; Harrison, Erickson, Adlaf, & Freeman, 2001; Osgood, Johnson, O'Malley, & Backman, 1988). For example, substance use and IPV are two co-occurring aspects of larger cognitive, behavioral, emotional, or cultural systems—yet these two do not always co-occur, so any other system or variable (e.g., intergenerational transmission of trauma, anger problems, antisocial personality disorder) may be what actually predicts the "relationship" between substance use and IPV (e.g., Morales, 1989). People in some cultures have virtually non-existent levels of violence, even when regularly intoxicated (Heath, 1983), while in other cultures, the sociocultural influences may have the opposite effect—as

perpetration may be somewhat expected during intoxication (e.g., Ahlstrom, 1981; Burns, 1980). The latter may lead some perpetrators to redirect the blame for their actions onto the substance, even though the substance did not cause the IPV (Center for Substance Abuse Treatment, 1997). Proponents of the spurious link would suggest that focusing on substance use would not reduce IPV recidivism. They suggest a more comprehensive approach, including the individual characteristics and predispositions as well as the emotional, behavioral, and sociocultural patterns (e.g., dominance and control) which characterize the perpetrator and victim's relationship (Zubretsky & Digirolamo, 1996).

**Integrative Theories**

Although substance use has been linked to IPV, there are a multitude of possible mediating and moderating factors which may simultaneously come into play. Thus, incorporating and/or integrating several theoretical perspectives may be most helpful when explaining the link between substance use and IPV. For example, the biopsychosocial model (Chermack & Giancola, 1997; Leonard, 1993; Moore & Stuart, 2005) highlights the interplay between various distal factors and proximal factors, which lead to IPV. Distal factors are relatively stable individual characteristics such as the individual's temperament, history of child abuse or witnessing parental violence, gender role expectations, peer influences, cultural norms, and social skills, which are always present when experiencing relationship conflict (Moore & Stuart, 2005). Proximal factors include the influences of acute substance use (e.g., pharmacological effects, impulsivity, emotional arousal), contextual influences (e.g., nature of the intimate relationship, assessment of threat in that particular setting/encounter), and the possible consequences of substance withdrawal (e.g., irritability, negative mood, information processing; Moore & Stuart, 2005). This biopsychosocial model suggests the pharmacological

effects may differ by substance, and that a substance may have differing effects depending on the dosage, and an individual's other proximal and distal factors, such as their gender roles and cultural expectations regarding aggressive behaviors. Integrative theory driven interventions adopt a multi-prong approach more sensitive to situational, individual, relational, and cultural factors.

Testing the impact of these integrating factors on the link between substance use and IPV is important, but challenging. Judging by the diverse array of empirical findings, individual quantitative studies sometimes lack sufficient power to adequately, accurately, or consistently measure how other factors may alter this link between substance use and IPV. As each new study adds empirical weight to the richness of our theoretical understanding, these seemingly diverse findings across studies also have the potential to muddy the theoretical waters—losing the forest for the trees.

In contrast, meta-analytic methods, which systematically pool together our field's empirical studies, increase the sensitivity (i.e. power) with which we can measure the link between substance use and IPV. Perhaps more importantly, meta-analyses can harness this combined power to test whether there are significant differences in the important interplay between substance types, the perpetrator's/victim's gender, and culture. The resulting field-wide, empirical benchmarks can help advance our understanding and point the field in important directions. Thus, when considering the link between substance use and IPV, we must continue to ask broad, important questions—some of which may only be adequately ascertained on a meta-analytic level.

### Important Theoretical Moderators

These theories highlight several important considerations which can guide a meta-analysis. Instead of just combining all substances into a conglomerate "alcohol & drug use" category, it would be beneficial to individuate by substance type (e.g., cocaine, heroin, marijuana) and then test for potential differences. Even though we do not know whether an individual perpetrated or was victimized while under the influence of a particular substance, differences in the strength of the link between each substance and IPV may hint at differing strengths associated with the substance's pharmacological or physiological effects.

Likewise, gender differences associated with each substance used, may (or may not) point to differing gender roles underlying the link between substances and IPV. Though, it may be that resulting pharmacological, physiological, and gender differences inform sociocultural factors, which reciprocally shape future gendered roles and behaviors. Finally, the way in which substance use is measured (e.g., frequency, quantity, criteria for abuse/dependence) may significantly impact the strength of the link between substances and IPV.

## Purpose and Unique Contributions of this Proposed Meta-analysis

This meta-analysis provides the most comprehensive and sensitive meta-analysis to date measuring the link between substance use and physical IPV. Due to the exponential increase in studies measuring substance use and physical IPV in the last decade, this study incorporates effect sizes from 285 studies to meta-analyze the link between distinct substances (alcohol, cocaine, marijuana, heroin, methamphetamines, stimulants, non-stimulants, and overall substance use) as risk markers for adult male perpetration/victimization and female perpetration/victimization. By testing for important moderators such as gender, direction of violence, substance type, and measurement type, this study also contributes empirical meta-

evidence to better understand the link between substance use and IPV. This meta-analysis measured the strength of: a) the link between overall substance use and IPV perpetration/victimization, b) the link between alcohol use and IPV perpetration/victimization, c) the link between various illicit drugs (cocaine, marijuana, heroin, methamphetamines, stimulants, non-stimulants, and non-differentiated drug use) and IPV perpetration/victimization. This meta-analysis also tested for various moderators which may affect the correlational link between substance use and IPV, such as gender (male vs. female), type of sample (clinical vs. non-clinical), and type of substance (e.g., cocaine, heroin, marijuana, amphetamines).

This study has the potential to make several important contributions to the literature. First, this study provides an updated, comprehensive, and detailed analysis of the link between substance use and IPV. The link between substance use and IPV may differ depending on substance type, which may point to how differing pharmacological or physiological effects are associated with violence. Significant differences in the link between a substance and IPV perpetration or IPV victimization would help us better understand whether substances have stronger links to a particular direction of violence. Second, this study could deepen our understanding of the differences between alcohol use and IPV, compared to drug use and IPV. Third, compared to previous meta-analyses which somewhat (or completely) lacked a substantial number of effect sizes to measure substance use with female perpetration or male victimization (see Appendix A), this meta-analyses has the power to more accurately test for gender differences in the link between substances and IPV perpetration or victimization, which may suggest different underlying gender roles or expectations. Fourth, this study has the potential to highlight a more nuanced and sensitive understanding of the link between different drug types

and IPV. Fifth, these findings could help us better understand how the manner in which we measure substance use affects the link between substance use and IPV.

# Chapter 3 - Method

## Literature Search

The identification and ultimate selection of studies to be included in this meta-analysis occurred in multiple phases. First, we reviewed 509 studies published from 1980 to 2000, which had been gathered for consideration in previous meta-analyses of IPV risk markers (Stith et al., 2000; 2004). These studies had been found through computer database searches (ERIC, Medline, PsychoLit, Social Sciences Abstracts, Sociological Abstracts, and Social Sciences Citations Index). The key words in this previous search had been: intimate partner and abuse, intimate partner and violence; spousal/spouse and violence, spousal/spouse abuse, spousal/spouse and aggression, family and violence, family and abuse, family and aggression, couple and violence, couple and abuse, couple and aggression, marital and violence, marital and abuse, and marital and aggression.

Second, we conducted a broad search using several computer databases (Web of Science, PROQUEST, and MEDLINE) for any study (including dissertations and theses) published from January 2001 to December 2012 using search terms related to couple (intimate partner, marital, spouse, husband, wife, or same-sex partner), partner aggression (abuse, aggression, domestic violence, batter, maltreatment, or violence), and risk markers (risk, factor, predictor, pathway, or correlate). Third, we conducted a more focused search (PsychINFO, Social Services Abstracts, and Sociological Abstracts) for any study (or dissertation/thesis) published before January 2014 which reported substance use and IPV using terms related to couple (intimate partner, marital, spouse, husband, or wife), partner aggression (abuse, aggression, domestic violence, batter,

maltreatment, or violence), and substances (cocaine, marijuana, heroin, stimulant, non-stimulant, amphetamine, methamphetamine, drug*[1], or alcohol).

Next we used an assortment of hand-picking methods to foster higher levels of study inclusion. We hand-picked through all available issues of journals which focused on family violence (*Violence and Victims, Journal of Family Violence, Journal of Interpersonal Violence, Partner Abuse, Journal of Aggression, Maltreatment, and Trauma, Violence Against Women, and Psychology of Violence*) and through abstracts from national conferences (National Council on Family Relations, National Association for Social Work, American Association for Marriage and Family Therapy, and International Family Violence and Child Victimization Research Conference) in order to contact presenters about potential unpublished findings. Prominent IPV researchers were also contacted about possible unpublished papers/presentations. Finally, we hand-picked reference lists from all included studies between 2009 and 2012 and from twelve comprehensive reviews/meta-analyses on IPV.

**Inclusion and Exclusion Criteria**

National and international studies were included in this substance use meta-analysis after adhering to inclusion and exclusion criteria through selection and screening stages (Stith et al., 2004). Titles and abstracts of potential studies were read to initially select them for further screening. If a screened study aligned with our inclusion criteria and did not meet our exclusion criteria, we included it in our substance use meta-analysis: Included studies (a) used physical IPV as the outcome, (b) were published in English, (c) included statistics sufficient for calculating at least one bivariate effect size, and (d) had an adult sample that was married or cohabitating. Studies were ultimately excluded if: (e) they used university samples or focused on

[1] The * in the search terms allows for the search results to include the words drug and drugs.

dating violence (as adolescent dating violence is considered a related, but different field of study compared to violence in adult, long-term committed relationships; Johnson & Ferraro, 2000; Lloyd & Emery, 2000), (f) they did not specifically report on physical IPV (i.e., they combined physical, psychological, and sexual IPV as one outcome), (g) they focused on intimate partner homicide, (h) their samples were solely a special population (such as those with traumatic brain injuries), or (i) they did not differentiate results by victimization or perpetration.

**Included Samples**

We located 18,798 studies through searches conducted for two previous meta-analyses (Stith et al., 2000; 2004; *n* = 509), through database searching (*n* =17,952), and through handpicking (*n* = 337). We excluded 1,731 duplicates, which resulted in 17,067 studies considered for the initial selection. Of these, we excluded 14,248 studies based on inclusion/exclusion criteria, which resulted in 2,819 studies selected for closer screening. A total of 2,215 of these studies were ultimately excluded because they focused on dating violence (8.3%), were not quantitative (7.4%), did not include relevant risk markers (5.6%), physical intimate partner violence was not the outcome variable (62.8%), included homicide as the outcome variable (3.3%), focused on a special population (1.3%), or were not written in English (0.4%). If a study of interest did not report univariate or bivariate data necessary to compute an effect size, we attempted to contact the authors, but only 11% of the contacted authors provided additional output in response to our requests. As a result, we had to exclude several studies because authors did not report usable data and did not respond/or were unable to help with our requests for more information. Finally, because we ultimately lacked enough same-sex intimate partner data to make meaningful comparisons for substance use, only data from studies with

participants who reported being in heterosexual intimate partner relationships were used in this meta-analysis.

**Coding Procedures**

We followed recommended coding procedures for conducting a meta-analysis (Card, 2012; Hunter & Schmidt, 2004). A graduate research coding team was trained by project leaders (two upper-level graduate students and one faculty member) to use a 37-item code sheet to capture study, data, and sample information from each included study (see Appendix E). Coders met weekly with project leaders to deliberate coding questions. Of the 287 studies included in this analysis, 58% were cross-coded by two separate coders. Coders collectively achieved a 96% agreement rate on their codesheets, but instead of calculating inter-rater reliability, the coders conferred with one-another to arrive at the correct answer if they experienced a coding discrepancy. However, when coders were unable to agree, they discussed the discrepancy with a project leader in order to arrive at a deeper understanding of the data (similar to the procedure used by Hawkins, Blanchard, Baldwin, & Fawcett, 2008). A lead researcher entered data into an Excel spreadsheet, and if he had any questions about the codesheet information, he would consult another leader to come to a clearer consensus.

**Other Study Characteristics**

Study and sample characteristics were coded such as the year of publication/presentation, whether the study was published or unpublished, type of publication (e.g., journal article, thesis or dissertation), dyadic or non-dyadic data, domestic or international sample, the instrument used to measure substance use, and non-clinical or clinical populations (a study was coded as clinical if it included participants from a women's shelter, hospital/emergency care, couples therapy,

batterer intervention or substance use program, psychologist/psychiatrist/outpatient mental health clinic, or prison).

## Statistical Method

The effect sizes between risk markers and adult IPV were analyzed using Comprehensive Meta-Analysis software (Borenstein, Hedges, Higgins, & Rothstein, 2005). A fixed-effect analysis for yielding aggregate effect sizes assumes there is one "true" population mean effect size across all studies, and thus, only accounts for within-study variance. As a result, the inferences from a fixed-effect analysis can only appropriately speak to those studies included in that particular meta-analysis (Card, 2012). It was theorized there would be real population differences between studies, therefore, a random-effects model was used to aggregate mean effect sizes. A random-effects approach accounts for both within-study variance and between-study variance, which allows for generalized inferences that can extend beyond the populations included in the studies in our meta-analysis (Card, 2012). A random-effects model was also used to combine subgroups (e.g., gender subgroups, substance use subgroups, measurement subgroups) which resulted in a fully random-effects analysis for testing significant group differences. A fully random-effects analysis accounts for real population differences between groups. Cohen's (1992) suggested criteria of evaluating trivial ($r < .01$), small ($r = .10$), medium ($r = .30$), and large ($r = .50$) were used to evaluate effect sizes.

## Plan of Analysis

For each of the analyses, I examined the link between substance use with IPV perpetration and IPV victimization separately. First, I conducted standard tests (Duval and Tweedie's trim and fill, classic fail-safe N, and Orwin's fail-safe N) to evaluate possible publication bias for (a) overall substance use (combined alcohol and drug) with IPV, (b) alcohol

use with IPV, and (c) drug use with IPV. Next, I conducted meta-regressions to test whether publication date was significantly linked to the magnitude of each study's overall substance use effect sizes. Third, I tested for significant study characteristic differences (e.g., published vs. non-published) on overall substance use. Fourth, I meta-analyzed the mean effect size for overall substance use, and then within overall substance use, I tested for differences between overall alcohol use vs. overall drug use. Fifth, I tested for gender differences for substance use, alcohol use, and drug use. Sixth, I grouped standardized and unstandardized alcohol measures into different categories of measurement. The overarching categories were alcohol consequence measures (abuse/dependence/problems) and alcohol consumption measures. The subcategories for alcohol consequence measures were abuse/dependence, diagnosis, and problem drinking. The subcategories for alcohol consumption measures were excessive drinking, frequency, quantity, quantity-frequency, and use, Seventh, I compared those categories of alcohol use combining females and males, for female alcohol use only, and then for male alcohol use only. Next, I compared various drug types (females and males combined), compared various drug types for females only, and then compared various drug types for males only. Finally, I grouped standardized and unstandardized measures of drug use into different categories of drug use measurement and compared those categories for females and males combined, for females only, and then for males only.

# Chapter 4 - Results

## Study Characteristics

A total of 287 studies were gathered which had a combined sample size of 627,726 and provided 994 unique effect sizes (ES) for this substance use meta-analysis (Table 2 and Table 3). All of these studies provided data for substance use linked with perpetration and/or victimization. Most studies (89%) were published in a journal or book chapter, and over 25% of the studies were published after 2009. International samples comprised 28% of the total studies, and 73% of the total studies reported a sample size less than 1,000 participants.

A function in the Comprehensive Meta-Analysis software was used to view residual *SD* to help identify potential outlying effect sizes (ES). Outlier ES were not included in the meta-analysis if they were more than 3 residual *SD*'s (in either direction) from the mean ES for substance use and perpetration or for substance use and victimization (Pukelsheim, 1994). A total of 11 outlier ESfrom five studies (Bevan & Higgins, 2002; Broach, 2004, Drapkin et al., 2004; Hastings & Hamberger, 1988; Mignone, 2006) were not included in the analyses. The mean ES for substance use, alcohol use, and drug use (for perpetration and for victimization) were then calculated with and without the outliers. The removal of the outlier ES did not significantly or substantially affect the mean ES for substance use, alcohol use, or drug use for perpetration or for victimization. However, the inclusion of these outlier ES could affect more specific analyses (e.g., when comparing smaller subgroups) by significantly and substantially altering the mean ES, and by significantly and substantially lengthening the confidence intervals (which would increase the likelihood of a Type II error when comparing differences between sub-groups). After not including these 11 outlier ES, a total of 983 unique ES from 285 different studies were used in meta-analyses (Table 3).

The 983 unique ES were grouped into alcohol/drug use (when the authors reported a collective alcohol & drug use variable), alcohol use, and drug use. These ES were further organized by gender of the user (male, female, or combined/undifferentiated gender report) and by the direction of violence (perpetration or victimization). Alcohol and drug ES were further categorized by the type of measure used (consequence or consumption), and then separated again into sub-categories of measure types (e.g., frequency of alcohol consumption was sub-divided into (a) occasional use, (b) use a few times a week, (c) use daily/almost daily, and (d) an unspecified frequency of use in the past 3 months, 6 months, 12 months, lifetime, or during a non-specified amount of time). Finally, drug ES were organized by drug type: undifferentiated drug use (a study's "drug use" variable which grouped various illicit drugs together) or amphetamines, cocaine, heroin/opioids, marijuana, stimulants, non-stimulants, or other drug types (pills, tranquilizers, etc.).

## Analyses of Publication Bias

All meta-analyses suffer from the "file drawer problem" of not including every possible study, many of them unpublished (Hunter & Schmidt, 1990). For this meta-analysis, three different tests were used to evaluate the possibility of publication bias: the trim and fill test (Duval & Tweedie, 2000), the fail-safe N (Rosenthal, 1979), and Orwin's fail-safe N (Orwin, 1983). The trim and fill test assesses for publication bias by using a funnel plot to evaluate the asymmetrical distribution of the included studies, and then plots the number of potential missing studies and provides a corrected mean effect size estimate based on the "inclusion" of these missing studies (Duval & Tweedie, 2000). Using random-effects, the trim and fill results for perpetration of IPV (Table 3) plotted 7 potential studies for overall substance use and perpetration of IPV (Figure 2), 7 potential studies for alcohol use and perpetration of IPV (Figure

3), and 2 potential studies for drug use and perpetration of IPV (Figure 4). Using random-effects, the trim and fill test (Table 3) estimated 26 potential studies for overall substance use with IPV victimization (Figure 5), 10 potential studies for alcohol use with IPV victimization (Figure 6), and 3 potential studies for drug use with IPV victimization (Figure 7). In each instance, the impact of these potential missing studies was trivial (Borenstein, Hedges, Higgins, & Rothstein, 2009), which suggests our mean effect sizes for overall substance use, alcohol use, and drug use are reasonably robust against publication bias.

Next, fail-safe Ns were conducted for overall substance use, alcohol use and drug use with perpetration and with victimization, to evaluate how many null studies would be needed to pull the significance of the mean effect size above the $p < .05$ threshold (Rosenthal, 1979). The resulting fail-safe Ns for overall substance use, alcohol use, and drug use far exceeded the recommended benchmark (5k + 10), which strongly indicates that our mean effect sizes are robust against publication bias (Table 3). Finally, an Orwin's fail-safe N, which calculates how many studies with a particular effect size would be needed to shift the mean effect size up/down to a specific magnitude (Orwin, 1983), was conducted for overall substance use, alcohol use, and drug use (with perpetration and then with victimization). The Orwin's fail-safe Ns were calculated by estimating how many missing studies having a value of $r = .00$ would be needed to pull the mean effect size down to $r = .10$ (the lower threshold of Cohen's small effect size; Cohen 1992). The results of these Orwin's fail-safe Ns reveal a substantial number of studies would be needed to pull our mean substance use, alcohol use, and drug use effect sizes down to $r = .10$ (Table 3). Thus, the results of all three publication bias tests indicate this meta-analysis is robust against publication bias.

## Analyses of Study Characteristics

The characteristics of each study shape the outcomes of that study, thus for a meta-analysis it is important to test the link between certain study characteristics and the resulting effect sizes. First, meta-regressions were conducted, using unrestricted maximum likelihood, to test the potential link between publication date and the magnitude of a study's overall substance use effect size (for perpetration and then for victimization) to see if the magnitude of ES had shifted over time. These meta-regressions resulted in a non-significant coefficient linking publication date with overall substance use and perpetration ($b$ = .0001, $p$ = .20) and victimization ($b$ = -.0004, $p$ = .81). This suggests that publication date was not significantly linked to the magnitude of each study's overall substance use effect size.

Next, moderator analyses were conducted for perpetration and for victimization to explore four other study characteristics which may affect each study's substance use effect sizes: published vs. unpublished study, clinical vs. non-clinical sample, dyadic vs. non-dyadic data, severe vs. moderate violence (for studies specifically reporting severe and moderate violence). Analyses revealed (Table 5) no significant differences for IPV perpetration based on study characteristics, but the overall substance use effect sizes for IPV victimization were significantly stronger ($Q^b$ (1) = 10.17, $p$ <.01) from non-dyadic data ($r$ = .21) compared to dyadic data ($r$ = .13).

## Analyses of Overall Substance Use and IPV

Using each study as the unit of analysis, the mean effect size (ES) for overall substance use and perpetration of IPV ($r$ = .22, $SE$ =.005, CI [0.20, 0.24], $p$ < .001, $k$ = 215; Table 6) was considered to be small (Cohen, 1992). Similarly, using each study as the unit of analysis, the mean ES for overall substance use and IPV victimization was also small ($r$ = .20, $SE$ =.004, CI

[0.18, 0.22], $p < .001$, $k = 146$). There was a substantial amount of heterogeneity within the perpetration studies ($Q^w = 5297.67$, $p < .001$, $I^2 = 95.96$) and within the victimization studies ($Q^w = 2365.31$, $p < .001$, $I^2 = 93.87$), which supports our decision to analyze the data using random-effects to account for real population differences between studies. The $I^2$ indicated that 95.96% of the variance in the ES of overall substance use and perpetration, and 93.87% of the variance in the ES of overall substance use and victimization, were due to between-study differences (which suggests that only 4.04% and 6.13% of the variance, respectively, was due to within-study measurement error). A deeper exploration of potential moderators, therefore, was warranted to help explain this between-study heterogeneity for substance use and IPV.

**Comparing Alcohol Use and Drug Use with IPV**

Next, alcohol use effect sizes and drug use effect sizes reported within studies were used as the units of analysis to compare the association between alcohol use with IPV and drug use with IPV (see Table 6). There was no significant difference between alcohol use ($r = .20$, CI [0.19, 0.22], $p < .001$) and drug use ($r = .23$, CI [0.20, 0.26], $p < .001$) for IPV perpetration. However, there was a significantly stronger ($Q^b (1) = 11.17$, $p < .001$) effect size for drug use ($r = .23$, CI [0.20, 0.25], $p < .001$) compared to alcohol use ($r = .17$, CI [0.14, 0.20], $p < .001$) and IPV victimization. This suggests that the association between substance use and being a victim of IPV is significantly stronger for drug users than for alcohol users. Furthermore, there was a substantial amount of heterogeneity within the alcohol use and perpetration effect sizes ($Q^w = 5040.47$, $p < .001$, $I^2 = 92.56$), the alcohol use and victimization effect sizes ($Q^w = 2055.29$, $p < .001$, $I^2 = 88.81$), the drug use and perpetration effect sizes ($Q^w = 1471.25$, $p < .001$, $I^2 = 89.90$), and the drug use and victimization effect sizes ($Q^w = 1113.84$, $p < .001$, $I^2 = 86.71$), which

suggests the presence of other moderators which may explain some of the between-effect sizes heterogeneity for alcohol use and drug use.

**Gender Differences in Substance Use and IPV**

Male and female subgroups were then used as the units of analysis to compare how gender was associated with the link between perpetration of IPV with substance use, alcohol use, and drug use (see Table 6). For overall substance use, there was a significantly stronger ($Q^b$ (1) = 12.07, $p < .001$) effect size for male substance use ($r = .23$, CI [0.21, 0.24], $p < .001$) than for female substance use ($r = .17$, CI [0.14, 0.20], $p < .001$) and IPV perpetration. This suggests that overall substance use is a significantly stronger risk marker for males perpetrating IPV, than it is for females perpetrating IPV. Similarly, there was a significantly stronger ($Q^b$ (1) = 14.36, $p < .001$) effect size for male alcohol use ($r = .22$, CI [0.21, 0.24], $p < .001$) than for female alcohol use ($r = .15$, CI [0.12, 0.18], $p < .001$) and IPV perpetration. This suggests that alcohol use is a significantly stronger risk marker for males perpetrating IPV, than it is for females perpetrating IPV. However, there were no significant gender differences for drug use and perpetration of IPV.

Male and female subgroups were then used as the units of analysis to compare how dichotomized gender was associated with the link between substance use, alcohol use, and drug use with being a victim of IPV (Table 6). There was a significantly stronger ($Q^b$ (1) = 4.17, $p < .05$) effect size for female substance use ($r = .21$, CI [0.19, 0.23], $p < .001$) than for male substance use ($r = .17$, CI [0.14, 0.20], $p < .001$) and IPV victimization. This suggests that overall substance use is a significantly stronger risk marker for females becoming victims of IPV, than it is for males becoming victims of IPV. However, there were no significant gender differences for alcohol use or for drug use and being a victim of IPV. For both males and females, there was a substantial amount of heterogeneity (Table 6) for substance use, alcohol use, and drug use with

IPV perpetration and victimization effect sizes, which again suggests the presence of other moderators which can help explain some of this heterogeneity.

## Analysis of Alcohol Use and IPV

### Subcategories of Alcohol Use with Perpetration of IPV

Next, various ways of measuring alcohol use were compared to test whether certain measures were more strongly related to perpetrating IPV (see Table 7). No significant difference was found between alcohol consequence (e.g., problem drinking and alcohol dependence) measures ($r$ = .22) and alcohol consumption measures ($r$ = .19) for IPV perpetration, as their confidence intervals somewhat overlapped ([0.20, 0.24] and [0.17, 0.21] respectively). When comparing ways of measuring alcohol consequences, no significant difference was found between abuse/dependence measures ($r$ = .21), an clinical DSM diagnosis of alcohol abuse/dependence diagnosis ($r$ = .24), and problem drinking ($r$ = .22) with perpetration of IPV. When comparing alcohol consumption measures, no significant differences were found between excessive drinking ($r$ = .21), frequency of alcohol use ($r$ = .15), quantity of alcohol ($r$ = .13), quantity-frequency measures ($r$ = .17), and use ($r$ = .24) with IPV perpetration. When comparing the sub-categories of excessive drinking, no significant differences were found between measures of heavy/binge drinking and frequency of drunkenness/binging for IPV perpetration. However, when comparing the sub-categories of different ways to measure frequency of alcohol use , there were significantly stronger effect sizes ($Q^b$ (3) = 22.47, $p$ < .001) for using alcohol a few times a week (1-3 days) *(*$r$ = .258, CI [0.15, 0.36], $p$ < .001, $k$ = 7) and using alcohol daily/almost daily (4-7 days; $r$ = .314, CI [0.15, 0.36], $p$ < .001, $k$ = 8) compared to "frequency" of alcohol use ($r$ = .026, CI [-.05, 0.10], $p$ < .001, $k$ = 18) and IPV perpetration (Table 7). This suggests that using alcohol a few times a week, almost daily, or daily are stronger risk markers

for perpetrating IPV, compared to alcohol use at an unspecified frequency. The unspecified frequency of alcohol use may be an unhelpful way to compare frequency of alcohol use, versus creating specific categories of frequency of alcohol use.

**Subcategories of Alcohol Use with IPV Victimization**

Various ways of measuring alcohol use were then compared to test whether certain measures were more strongly related to being a victim of IPV (Table 8). There was a significantly stronger ($Q^b$ (1) = 10.97, $p < .001$) effect size for measures of alcohol consequences ($r$ = .201, CI [0.18, 0.22], $p < .001$, $k$ = 113) than for alcohol consumption measures ($r$ = .139, CI [0.11, 0.17], $p$ <.001, $k$ = 100) and IPV victimization. This suggests that having met criteria for alcohol consequences (e.g., problem drinking and alcohol dependence) is a significantly stronger risk marker for being a victim of IPV, compared to consumption of alcohol. There were no significant differences between sub-categories of alcohol consequence measures, between sub-categories of alcohol consumption measures, between sub-categories of excessive drinking measures, or between sub-categories of frequency measures for being a victim of IPV (Table 8).

**Subcategories of Female Alcohol Use with Perpetration of IPV**

Next, the alcohol measure subgroups were dichotomized by gender (female or male) and various ways of measuring female alcohol use were compared to test whether certain measures were more strongly related to females being perpetrators of IPV (Table 9). No significant differences were found when comparing female alcohol consequence measures with female alcohol consumption measures. Likewise no significant differences were found between sub-categories of female alcohol consequence measures, or between sub-categories of female alcohol consumption measures, or between sub-categories of female excessive drinking measures, or between sub-categories of female frequency measures for females perpetrating IPV.

**Subcategories of Female Alcohol Use with IPV Victimization**

Then, various ways of measuring female alcohol use were compared to test whether certain measures were more strongly related to females being victims of IPV (Table 10). There was a significantly stronger ($Q^b$ (1) = 6.98, $p < .01$) effect size for measures of female alcohol consequences ($r$ = .211, CI [0.18, 0.24], $p < .001$, $k$ = 88) than for female alcohol consumption measures ($r$ = .152, CI [0.12, 0.18], $p$ <.001, $k$ = 100) and females being victims of IPV. This suggests that females having met criteria for alcohol consequences (e.g., problem drinking and alcohol dependence) is a significantly stronger risk marker for females also being a victim of IPV, compared to females who consume alcohol, but do not necessarily meet the threshold of alcohol abuse/dependence/problem drinking. However, there were no significant differences between sub-categories of female alcohol consequence measures, or between sub-categories of female alcohol consumption measures, or between sub-categories of female excessive drinking measures, or between sub-categories of female frequency of alcohol use with females being victims of IPV (Table 10).

**Subcategories of Male Alcohol Use with Perpetration of IPV**

Next, various ways of measuring male alcohol use were compared to test whether certain measures were more strongly related to males being perpetrators of IPV (Table 11). There was a significantly stronger ($Q^b$ (1) = 4.93, $p < .05$) effect size for measures of male alcohol consequences (e.g., problem drinking and alcohol dependence; $r$ = .246, CI [0.22, 0.27], $p <$ .001, $k$ = 136) than for male alcohol consumption measures ($r$ = .204, CI [0.18, 0.23], $p$ <.001, $k$ = 141) with males perpetrating IPV. This suggests that males having met the criteria for alcohol consequences (e.g., problem drinking and alcohol dependence) is a significantly stronger risk marker for males also perpetrating IPV, compared to males who consume alcohol, but do not

meet the alcohol abuse/dependence/problem drinking criteria. Likewise, when comparing sub-categories of frequency of male alcohol use, there were significantly stronger effect sizes for ($Q^b$ (3) = 16.52, $p < .001$) using alcohol a few times a week (1-3 days) *(*$r = .258$, CI [0.15, 0.36], $p < .001$, $k = 7$) and using alcohol daily/almost daily (4-7 days; $r = .314$, CI [0.15, 0.36], $p < .001$, $k = 8$) compared to an undifferentiated frequency of alcohol use measures (i.e., frequency of use in the past 3, 6, or 12 months; $r = .035$, CI [-.06, 0.13], $p < .001$, $k = 12$) and IPV perpetration. This suggests that for males, using alcohol a few times a week, almost daily, or daily are stronger risk markers for males perpetrating IPV, compared to male alcohol use at some unspecified frequency. However, there were no significant differences between sub-categories of male alcohol consequence measures, between sub-categories of male consumption measures, or between sub-categories of male excessive drinking measures with males being perpetrators of IPV (Table 11).

### Subcategories of Male Alcohol Use with IPV Victimization

Various ways of measuring male alcohol use were then compared to test whether certain measures were more strongly related to males being victims of IPV (Table 12). No significant differences were found between consumption versus consequence measures, between sub-categories of male alcohol consequence measures, between sub-categories of male consumption measures, between sub-categories of male excessive drinking measures, or between sub-categories of frequency with males being victims of IPV (Table 12).

## Analysis of Drug Use and IPV

### Different Drug Types and IPV Perpetration

Because it was theorized there might be differences in the link between specific drug types and IPV perpetration or victimization, drug types were compared amongst each other to

test whether different illicit drug types had a stronger link with the perpetration of IPV (Table 13). The overall effect sizes for perpetrating IPV and using amphetamines ($r$ = .198, $p$ < .01), cocaine ($r$ = .215, $p$ < .001), heroin ($r$ = .055, *ns*), marijuana ($r$ = .252, $p$ < .001), and other drugs (a combined group of pills, tranquilizers, injection drugs, etc.; $r$ = .126, $p$ > .05) were non-significantly different. Next, drug types were grouped into stimulant (cocaine and amphetamines) versus non-stimulant (all other drugs) categories and tested to see whether one category had a stronger relationship with perpetrating IPV. The effect sizes for perpetrating IPV and using stimulant drugs ($r$ = .214, $p$ < .001) or using non-stimulant drugs ($r$ = .205, $p$ < 0.01) were not significantly different.

**Different Drug Types and IPV Victimization**

Similarly, drug types were compared amongst each other to test whether different drug types had a stronger link with being a victim of IPV (Table 13). The overall effect sizes for victimization and using amphetamines ($r$ = .308, $p$ < .001), cocaine ($r$ = .284, $p$ < .001), heroin ($r$ = .039, *ns*), marijuana ($r$ = .229, $p$ < .001), and other drugs (a combined group of pills, tranquilizers, injection drugs, etc.; $r$ = .234, $p$ < .01) were non-significantly different. Stimulant drug use ($r$ = .247, $p$ < .001) was non-significantly different from non-stimulant drug use ($r$ = .200, $p$ < .001) and IPV victimization.

**Gender Comparison by Drug Type with IPV Perpetration and Victimization**

Next, drug types were compared by dichotomized gender (male or female) for IPV perpetration and victimization. Results indicated there were no significant gender differences based on each drug type for IPV perpetration or victimization (Table 14 and Table 15). However, these results should be taken with caution, because although some effect sizes seemed to be substantially larger (e.g., female cocaine use and perpetration, $r$ = .333, $p$ < .05, $k$ = 3, compared

to male cocaine use and perpetration, $r = .174$, $p < .05$, $k = 9$), these comparisons often lacked power due to the limited number of effect sizes for gender and drug subgroups.

### *Different Drug Types Compared for Females and IPV*

Various drug types used by females were then compared to test whether specific drug types had a stronger relationship with female perpetration and then with female victimization (Table 16). No significant differences were found between the use of amphetamines, cocaine, heroin, marijuana or other drugs for perpetration or victimization and female perpetration; the same was found for female victimization. The same was true for stimulant versus non-stimulant drugs and perpetration, and with victimization. Solely comparing the effect sizes, some drug types looked substantially different from one another (e.g., female cocaine use ($r = .326$) and female heroin use ($r = .064$) for female victimization). However, the confidence intervals for each effect size substantially overlapped (e.g., [0.15, 0.48] and [-.18, 0.30] respectively). Again, these results should be taken with caution because this comparison analysis of different female use by drug type may have been underpowered due to a lack of available effect sizes for specific illicit drug types.

### *Different Drug Types Compared for Males and IPV*

Various drug types used by males were then compared to test whether specific drug types had a stronger relationship with male perpetration and then with male victimization (Table 17). Similar to our findings for females, no significant differences were found between males' use of amphetamines, cocaine, heroin, marijuana or other drugs for male perpetration, or with male victimization. The same was true for male stimulant versus non-stimulant drugs with male perpetration or victimization. Although some drug types seemed substantially different (e.g., male marijuana use ($r = .261$) versus male heroin use ($r = .108$) for male perpetration), the lack of available effect sizes for each drug type resulted in the confidence intervals substantially

overlapping (e.g., [0.17, 0.35] and [-.06, 0.27] respectively). Thus, the non-significant differences between male drug type use and male IPV should be taken with caution.

**Subcategories of Drug Use with Perpetration of IPV**

Next, different ways of measuring drug use were compared to test whether certain drug measures were more strongly related to perpetration of IPV (Table 18). There was a significantly stronger ($Q^b$ (1) = 9.30, $p < .01$) effect size for measures of drug consequences ($r$ = .297, CI [0.25, 0.34], $p < .001$, $k$ = 42) than for drug use measures ($r$ = .203, CI [0.17, 0.24], $p$ <.001, $k$ = 110) with perpetrating IPV. This suggests that illicit drug abuse/dependence is a significantly stronger risk marker of perpetration, compared to simply using illicit drugs. Similarly, when comparing within the drug use measures, the sub-group of undifferentiated use of drugs ($r$ = .234, CI [0.20, 0.27], $p < .001$, $k$ = 77) was a significantly stronger ($Q^b$ (1) = 8.33, $p < .01$) risk marker for perpetration of IPV compared to frequency of drug use ($r$ = .130, CI [0.07, 0.19], $p <$ .001, $k$ = 33). This suggests that whether or not someone has used drugs is a stronger risk marker for perpetration of IPV, compared to counting up how frequently an individual uses drugs. Within the drug consequence category, no significant differences were found between the abuse/dependence/diagnosis subgroup ($r$ = .219) compared to drug-related problems subgroup ($r$ = .172).

**Subcategories of Drug Use with IPV Victimization**

Different ways of measuring drug use were then compared to test whether certain drug measures were more strongly related to IPV victimization(Table 18). When comparing within the drug use measures, the sub-group of drug use ($r$ = .259, CI [0.23, 0.29], $p < .001$, $k$ = 71) was again a significantly stronger ($Q^b$ (1) = 6.17, $p < .05$) risk marker for being a victim of IPV compared to frequency of drug use ($r$ = .179, CI [0.12, 0.23], $p < .001$, $k$ = 27). This suggests

that whether or not someone has used illicit drugs is a stronger risk marker for IPV victimization, compared to counting up how frequently an individual has used illicit drugs. No significant differences were found between drug consequence measures and drug use measures, or between the sub-groups of drug-related problems or abuse/dependence/diagnosis, with regard to being a victim of IPV (Table 18).

**Subcategories of Female Drug Use with IPV**

Next, different ways of measuring female drug use were compared to test whether certain measures were more strongly related to female perpetration and to IPV female victimization(Table 19). No significant differences were found between female drug consequence measures and female drug use measures for either IPV female perpetration or female victimization. Likewise, no significant differences were found between the sub-groups of drug-related problem measures versus abuse/dependence/diagnosis measures with regards to female perpetration, or female victimization. However, within the drug use measures, the sub-group of female's undifferentiated use of drugs ($r$ = .252, CI [0.18, 0.32], $p$ < .001, $k$ = 13) was a significantly stronger ($Q^b$ (1) = 5.19, $p$ < .05) risk marker for female perpetration, compared to measuring the frequency of female drug use ($r$ = .103, CI [0.00, 0.20], $p$ < .05, $k$ = 9). Likewise, the sub-group of female's undifferentiated use of drugs ($r$ = .287, CI [0.25, 0.33], $p$ < .001, $k$ = 49) was a significantly stronger ($Q^b$ (1) = 4.98, $p$ < .05) risk marker for female victimization, than measuring the frequency of female drug use ($r$ = .191, CI [0.12, 0.26], $p$ < .001, $k$ = 17). In other words, whether or not females have used illicit drugs is a stronger predictor for IPV perpetration and victimization, compared to how frequently females used illicit drugs.

**Subcategories of Male Drug Use with IPV**

Finally, different ways of measuring male drug use were compared to test whether certain measures were more strongly related to IPV male perpetration or victimization (Table 20). There was a significantly stronger ($Q^b$ (1) = 6.59, $p < .05$) effect size for measures of male drug use consequences ($r = .310$, CI [0.25, 0.37], $p < .001$, $k = 28$) than for male drug use measures ($r = .218$, CI [0.17, 0.24], $p < .001$, $k = 73$) with males perpetrating IPV. This suggests that males having met the criteria for drug abuse/dependence or drug problems is a significantly stronger risk marker for males perpetrating IPV, compared to males who simply use drugs. Furthermore, when comparing the subgroups within drug use measures for male perpetration, the sub-group of males' undifferentiated use of drugs ($r = .251$, CI [0.21, 0.29], $p < .001$, $k = 56$) was a significantly stronger ($Q^b$ (1) = 9.81, $p < .01$) risk marker for being a male perpetrator of IPV compared to frequency of drug use ($r = .128$, CI [0.05, 0.19], $p < .001$, $k = 17$). This suggests that whether or not males have used illicit drugs is a stronger risk marker for male perpetration of IPV, compared to counting up how frequently males have used illicit drugs. However, no significant differences were found between drug measures and male victimization (Table 20).

# Chapter 5 - Discussion

The purpose of this meta-analytic study was to measure the link between substance use and intimate partner violence (IPV). Using 983 unique effect sizes (ES) from 285 studies, the mean effect size (ES) for substance use and perpetration ($r$ = .22), substances use and victimization ($r$ = .20), alcohol use and perpetration ($r$ = .20), alcohol use and victimization ($r$ = .18), drug use and perpetration ($r$ = .23), and drug use and victimization ($r$ = .23) were all found to be significant. This confirms that across the available studies, the link between substance use, alcohol use, or drug use and IPV perpetration or victimization is significant, yet small in magnitude (Cohen, 1992). These overall findings are similar to those reported by other literature (e.g., Bushman & Cooper, 1990) and the three most recent IPV meta-analyses which included substance use (Foran & O'Leary, 2008; Moore et al., 2008; Stith et al., 2004), however there were several important findings which can significantly add to our understanding of the link between substance use and IPV.

This meta-analytic study is important because it is the first to analyze an overall effect size for substance use (not only drug use or only alcohol use) with IPV perpetration and victimization, for males and females. Furthermore this was the first meta-analysis to detect empirical differences between alcohol use and drug use with IPV. This study further adds to the literature by the substantial inclusion of female perpetration and male victimization ES, which allowed gendered comparisons for IPV perpetration and victimization for alcohol use, ways of measuring alcohol use, drug use, drug types, and ways of measuring drug use. These gender comparisons are important because in some cases gender may significantly affect the link between substance use and IPV, but not in other instances. These gender differences are helpful for identifying which substances or measures differ by gender so that future research can explore

why these gender differences are present in particular circumstances, but not in others. These meta-analytic findings also reveal a more nuanced understanding of the significant differences found among various ways of measuring alcohol use. Similarly, these findings reveal drug consequence measures were significantly stronger risk markers for IPV perpetration than simply drug use measures. These differences are important because certain ways of measuring substance use have a significantly stronger link to IPV, which could impact the manner in which IPV researchers decide to measure substance use. The last major contribution from this meta-analysis was finding no significant differences between stimulant and non-stimulant drug use for perpetration of IPV or IPV victimization. This is important because it suggests that non-stimulant drugs are not necessarily safer than stimulant drugs in terms of not experiencing violence in the intimate relationship.

## Study Characteristic Differences

Previous research has identified significant differences between non-clinical populations and clinical populations (or an ES derived from comparing a clinical sample to a non-clinical sample) in the linkage from substance use and IPV (e.g., Murphy & O'Farrell, 1996; Kaufman-Kantor & Straus, 1987). In fact, because of the pronounced differences between clinical and non-clinical populations, some meta-analyses have opted to not include data from clinical populations in their analysis (e.g., Schumacher, Feldbau-Kohn, Smith Slep, & Heyman, 2001) in order to avoid potentially skewing their meta-analytic results. Although, this current meta-analysis did find the ES for perpetration and victimization to be larger for clinical samples, these clinical sample ES were not significantly larger than ES derived from non-clinical samples (which is similar to findings in the meta-analysis by Foran & O'Leary, 2008). The overlapping confidence intervals are due to the heterogeneous dispersion of studies within each subgroup, and this

supports the concept that there is a substantial range of experiences within the clinical and non-clinical populations. Just because a population is deemed to be clinical, does not necessarily mean the link between substance use and IPV will automatically be stronger compared to a non-clinical population. In fact, the overlapping confidence intervals suggest that in certain instances, the non-clinical population ES may actually be stronger. Finally, there was a significant difference between ES for IPV victimization based on whether the study reported dyadic or non-dyadic data, but this difference is probably due to using each study as the unit of analysis which would have aggregated male victimization (which is a significantly weaker ES than female victimization) with female victimization in the dyadic study overall ES. Thus, the significant difference found between dyadic data and non-dyadic data is probably due to the aggregation of male victimization with female victimization (which, when combined, would result in an overall smaller ES for that particular study), compared to a study which only measured female victimization (which would typically report a comparatively stronger overall ES for that study).

### Differences Between Alcohol and Drug Use

Compared to alcohol use, drug use was found to be a non-significantly stronger risk marker for IPV perpetration and a significantly stronger risk marker for IPV victimization. The most obvious explanation, at first glance, is that this difference between alcohol and drug use with victimization may be due to differing physiological or pharmacological effects from drugs compared to alcohol—as these differing effects also seem to be present (just not as pronounced) for the perpetration ES. Yet, just because the physiological and pharmacological effects of drug use can be distinct from alcohol consumption, this still would not explain why the significant difference between alcohol and drugs is found only with victimization but not in perpetration. Comparing the confidence intervals (Cumming & Finch, 2005) between alcohol use with

perpetration of IPV and alcohol use with IPV victimization(Table 6), the ES for alcohol use victimization is significantly weaker. In contrast, when comparing drug use with perpetration and drug use with victimization, the drug use ES and confidence intervals are virtually identical. So either the alcohol physiological and pharmacological effects change depending on the direction of violence, while the illicit drug physiological and pharmacological effects do not change depending on the direction of violence, or there is another factor impacting this difference between alcohol use and drug use with regards to the direction of violence.

Another possibility is that this significant difference found between drug use and alcohol use with victimization can be attributed to the numerous female victimization ES (two to three times more numerous than male victimization ES), which are statistically pulling the ES in a certain direction. When comparing the confidence intervals (Cumming & Finch, 2005) between victimized female alcohol use and victimized female drug use, female drug use is also a significantly stronger risk marker for female victimization. In fact, when looking at the ES for perpetration and victimization (Table 6), both males and females reported stronger ES for drug use, compared to their alcohol use. So, although the sheer number of the female ES are undoubtedly pulling the mean overall victimization drug use and alcohol use in their direction, this does not necessarily explain why the drug use ES is a stronger risk marker than alcohol use for both males and females. So, what other factor could help explain how meta-analytically across studies (a) both males and females report a stronger link between drug use and IPV compared to alcohol use and IPV and (b) how, regardless of the direction of violence, the mean ES for overall drug use does not change depending on the direction of violence, even though the mean ES does significantly change for alcohol use depending on the direction of violence?

Integrated theoretical models, such as the tripartite conceptual framework (Goldstein, 1995) and biopsychosocial model (Leonard, 2001; McKenry, Julian, Gavazzi, 1995; Moore & Stuart, 2005), suggest a complex relationship between substance use and IPV. One component of their proposed multifaceted linkage is the sociocultural framework surrounding the substance use and IPV. The biopsychosocial model identifies cultural norms as stable (distal) components which are always present during relationship conflict and can affect the link between substance use and IPV (Moore & Stuart, 2005). Proponents of the tripartite framework point to the association between illicit drug use, illicit drug seeking and other illegal activities as behaviors embedded in larger systemic illicit drug market, the rules of which support violent behavior and habitual substance use (Goldstein, 1995; White, 1997).

Although it is culturally and legally acceptable to purchase and/or consume alcohol at almost any restaurant, plane ride, or convenience store, illicit drug use is not as widely available or accepted. Perhaps the drug user's unique, and possibly violent, sociocultural context is playing a significant role in this link between their drug use and their IPV perpetration or victimization. Obviously, it would be extremely difficult to accurately test the strength of the impact that illicit drug culture could have on the link between substance use and IPV. However, it should also be noted that there are distinct differences between sociocultural frameworks. Violence is not ubiquitous for every illicit drug market, as drug market and supply culture are a confluence of many characteristics, and therefore the violence component may result from the combination of the location, target population, and drug type (Coomber, 2015; Fagan & Chin, 1990). Thus, these meta-analytic results lend some empirical evidence in support of integrative theories, by suggesting that an outside factor, perhaps a sociocultural factor, is playing a significant role in

the relationship between drug use and IPV, because the drug use ES and confidence intervals do not change depending on the direction of violence.

### Differences Between Alcohol Measures

Although this meta-analysis did not find a significant difference between alcohol consequence measures and alcohol consumption measures for perpetration of IPV, results did reveal that alcohol consequence measures (i.e., alcohol abuse or drinking problems) were significantly stronger than alcohol consumption measures (i.e., alcohol use or frequency of alcohol use) for victimization. This differs from Foran and O'Leary's (2008) alcohol meta-analysis which found consequence/problem measures for perpetration to be significantly stronger than consumption measures for perpetration. Because this meta-analysis included a substantial amount of female alcohol use perpetration ES (and only found one measure, excessive drinking , to be above $r = .20$ for female perpetration), the inclusion of female ES in the overall comparison between consequence and consumption measures may have widened the overall confidence intervals for each measurement type. However, when solely comparing the male perpetration effect sizes (Table 9), the results from this meta-analysis supported Foran and O'Leary's (2008) previous findings, which had included mostly male-perpetration ES. Thus, the significant difference between alcohol consequence measures and alcohol consumption measures holds true for male perpetration, but may not for female perpetration.

Furthermore, when comparing different measures of frequency of alcohol use and perpetration, results from this meta-analysis indicate that measures reporting individuals who used alcohol a few times a week, almost daily, and daily had significantly stronger ES for perpetration of IPV, compared to other types of frequency measures (such as occasional use or frequency of use in the past 3, 6, or 12 months). Similar to conclusions proposed by previous

research (e.g., Leonard, 2001b; Testa, 2004), using alcohol in such high frequency may be associated with alcohol abuse, dependence, diagnosis, or problem drinking, which may be why these frequency measures were also significantly stronger for male perpetration, and why the ES and confidence intervals were the same ranges as the alcohol abuse/dependence/diagnosis /problem drinking measures. This finding is important because an "undifferentiated frequency of alcohol use" variable is not very helpful for predicting IPV or for comparing between different types of frequencies. Authors should strongly consider converting their "frequency of alcohol use" variables into subgroups of occasional, a few times a week, almost daily, and daily alcohol consumption, as this would allow for a more meaningful comparison between subcategories of frequency.

### Differences Between Drug Types

It was somewhat surprising to find no significant differences between drug types for IPV perpetration or for IPV victimization. Heroin was the only drug type in our analysis clearly not significantly related to IPV, yet this finding may be a result of our limited number of ES, or this non-significant relationship with IPV might be due to heroin's analgesic and sedative pharmacological properties (Goldstein, 1991; Martin & Fraser, 1961; Sawynok, 1986). The similarity between the ES for marijuana use, amphetamine use and cocaine use was also striking, because Moore and colleagues (2008) found in their drug use meta-analysis that cocaine had the strongest relationship with IPV. Marijuana has been reported as being less likely to result in activity and violence (Reiss & Roth, 1993; Boles & Miotto, 2003), having lower addiction potential (Van Amsterdam et al., 2010), and was recently heralded as being one of the least dangerous illicit drugs (Lachenmeier & Rehm, 2015). Yet, this does not necessarily mean marijuana use is less dangerous for the user's intimate partner. Marijuana withdrawal symptoms

have been linked with irritability, anger, and aggression (Boles & Miotto, 2003, Budney, Hughes, Moore, & Vandrey, 2004; Gold & Tullis, 1999; Smith, 2002), which could conceivably lead to IPV. While these meta-analytic findings do not account for whether the participants were using or were experiencing withdrawal symptoms at the time of perpetration or victimization, they do provide initial, new evidence that the strength of the link between marijuana use and perpetration or victimization is on par with substances more typically associated with IPV, such as alcohol, cocaine, or amphetamines.

Perhaps the most interesting result of the drug type comparison analyses was the non-significant difference between stimulants and non-stimulants for perpetration and victimization (though there seems to be more of a difference, albeit non-significant, with victimization). Given the statistical restraining effect of the non-significant heroin ES on the non-stimulant group mean, the similarity between stimulant and non-stimulant drugs may have been even closer after removing heroin from the non-stimulant group. This is surprising given the psychosis, irritability, increased activity, and aggression associated with stimulants (e.g., Fischman & Haney, 1999; Kosten & Singha, 1999; Mørland, 2000), which are effects not typically associated to the same degree (or at all) with other drugs (e.g., Boles & Miotto, 2003). These findings are important because the stimulant and non-stimulant ES are similar for perpetration, and for victimization, which suggests that regardless of whether a drug is a stimulant or non-stimulant, it is a risk marker for perpetrating or being a victim of IPV—and this holds true for both males and females.

The analysis comparing male and female perpetration and victimization by drug type resulted in no significant gender differences for *any* of the drug types. However, these non-significant drug type by gender findings should be taken with some caution. Similar to Moore

and colleagues (2008), this comparison analysis suffered from a lack of available ES from studies reporting specific drug types by gender with IPV, which may have increased the likelihood of a Type II error. However, when comparing males and females on overall drug use (several included studies reported an undifferentiated illicit drug use variable, for which participants reported use of one or more illicit drug types), the results also indicated no significant gender differences—though the overall results were trending toward the ES being stronger for male perpetration and for female victimization. Although the specific illicit drug type comparison for each gender was underpowered, the overall illicit drug use comparison by gender was not. This suggests that gender differences may play a role in certain situations, but gender differences may not always strengthen or weaken the link between specific drug types and IPV.

### Differences Between Drug Measures

Drug consequence measures were significantly stronger risk markers for perpetration than simply drug use measures, which suggests that those individuals who have developed a relationship with drugs characterized by abuse and dependence are more likely to perpetrate IPV, than individuals who do not fulfill the criteria for dependence or abuse. Individuals who develop a dependent relationship with drugs could conceivably be under the influence of the drugs more frequently, may use in higher dosages, and would experience withdrawals more often—any of which may contribute to a stronger link with IPV. Similarly, compared to individuals who reported a certain frequency of drug use, whether an individual reported any illicit drug use was a significantly stronger risk marker for both perpetration and for victimization. Studies typically measured the frequency of drug use, by asking about any drug use in the past 3, 6, or 12 months, thus more specific frequency measures, such as the number of uses daily or weekly, may provide

stronger effect sizes (as they have for alcohol use). This is a similar issue when measuring alcohol use, in that an "undifferentiated frequency of drug use" variable is less helpful for predicting IPV than comparing between different categories of drug use frequencies. Authors should strongly consider converting their continuous "frequency of drug use" variables into categorical subgroups of occasional, a few times a week, almost daily, and daily drug use, as this would allow for a more meaningful comparison between subcategories of drug use frequency.

### The "Underpowered" Dilemma

There is an inherent dilemma when comparing subgroups when one or more of these subgroups has a small number of ES: how should the non-significant differences be interpreted? A smaller number of ES results in a wider confidence interval for that particular subgroup, which means the confidence interval will significantly overlap with other subgroup confidence intervals, even though the mean estimate for that particular group is substantively different from the other subgroups. For example, the drug type comparison results found no significant differences between the different drug types for perpetration or for victimization—even though heroin clearly appeared to be substantially different. Does this mean (a) there is a real substantial difference between heroin and other drugs, and simply by adding more ES the analysis would have been able to detect it, or (b) that the included ES happen to heavily represent one side of the spectrum, but with the inclusion of more ES there would be a more equal distribution, which would shorten the confidence intervals, but also increase the magnitude of the ES—no longer revealing a substantive difference between heroin and other drug types? This dilemma is further amplified when comparing specific drug types between males and females, as these already underpowered drug type subgroups are divided again into males and females.

These smaller subgroup analyses can become seemingly meaningless, because the resulting confidence intervals are so wide, it is incredibly difficult to detect "significant" differences. This is especially challenging when using random-effects models within subgroups, which account for within-study variance and between-study variance, and random-effects between subgroups, which accounts for variance within-subgroups and between-subgroups (Borenstein, Hedges, Higgins, & Rothstein, 2009; Card, 2012). In comparison, a fixed effect approach within a subgroup or between subgroups accounts for only one type of variance (the within-study/within-subgroup variance; Borenstein et al.,2009; Card, 2012) leading to much smaller confidence intervals, which makes it easier to discover significant differences between groups (both of which have shorter confidence intervals), but also increases the risk for Type 1 errors.

In exchange for the ability to more easily detect differences (by accounting for only one type of variance), the fixed effect approach (which I did not use) sacrifices important theoretical assumptions, such as accounting for real population differences between studies or accounting for real differences between drug type subgroups. Thus, using random-effects to estimate the mean ES within a subgroup and random-effects to compare between subgroups is a more theoretically sound, yet a more statistically conservative approach. This means that subgroup comparisons are more easily underpowered when using random-effects, which results in wider confidence intervals, yet the ES estimate is more theoretically accurate (because it accounts for real possible differences between studies and subgroups). However, when significant differences are discovered using fully random-effects models, these differences are more likely to be "real," and less likely to be a result of Type 1 error. Thus, especially when using random-effects, it is important to not only focus on the confidence intervals for significant differences, but to also

compare the magnitude of the ES (e.g., for drug types or for gender differences) for substantive differences.

For example, the analyses revealed a significant difference between males and females for overall substance use and IPV victimization (see Table 6). However, when comparing males and females solely with alcohol, or solely with drug use and IPV victimization, no significant gender differences were found, even though there appears to possibly be real substantive gender differences. When looking at the similarity of the $I^2$ for males and females (which shows the percentage of the variance due to between study differences) for overall substance use, alcohol use, and drug use with IPV victimization, we see how the percentage of variance due to between study differences is very similar for females for substance use, alcohol use, and drug use, and the same for males. However, by combining alcohol use and drug use ES together when testing overall substance use this provided enough power to detect differences between males and females, even though no significant gender differences were detected with only alcohol or with only drug use (even though the mean ES point to possible gender differences). As future studies report specific drug types used by different genders, hopefully future meta-analyses will be better able to parse out both substantive differences and significant differences between drug types and significant gender differences on each drug type when using fully random-effects.

## Limitations

There are several limitations of this study that must be taken into account when interpreting these findings. Missing effect sizes decrease power and increase sampling error, and are the most prevalent limitation in meta-analyses (Peterson & Brown, 2005). Although this study implemented multiple search strategies and attempts were made to contact other authors, there were undoubtedly some studies never identified that could have provided usable effect

sizes which were not included in this bivariate meta-analysis. In fact, there were over 40 different studies not included in this analysis simply because they either (a) reported only multivariate results for the link between substance use and IPV or (b) they combined their physical IPV variable with sexual IPV or psychological IPV. Although attempts were made to contact authors of these studies, for various reasons, several bivariate effect sizes were not obtained and used in this meta-analysis.

Although the goal of this study was to harness the power of this field of research to measure the link between substance use and IPV, causation of IPV was never an anticipated finding—even though a better understanding of a potential causal link would be most desirable. This meta-analysis only measured the correlational link between substance use and IPV, and we cannot know how many of the total participants were intoxicated, inebriated, under the influence, or otherwise using substances at the time of perpetration or victimization. Furthermore, we do not know the circumstances of the substance use: Was the substance used socially? With the intimate partner? In a "responsible" manner? What was the quantity or frequency consumed? Was the substance used in combination with other substances? Some of these comparison analyses suffered from being underpowered due to a low number of available effect sizes, which increases the likelihood of Type II error. Those comparison analyses which included male victimization or female perpetration were especially limited when comparing sub-groups, such as certain drug types or specific measures of alcohol use. Thus, several of the comparison analyses were unable to detect potential differences between subgroups (see the "Underpowered" dilemma section for a deeper discussion of this issue).

Finally, meta-analytic findings are comprised from studies which each have their own methodological limitations (Card, 2012). While some studies included ES from validated

measures of substance use linked with validated measures of physical IPV, other studies measured their substance use and/or IPV variables with single-item, non-validated measures. Furthermore, studies measured or defined IPV in very different ways; as one study may include individuals who hit their partner only once in their lifetime, while another study only included individuals who met a certain threshold of IPV severity. Yet, in this meta-analysis, physical IPV was combined from all these studies even though the majority of studies did not differentiate between severities of violence or typologies of violence (see Johnson, 2004). Likewise, these findings are comprised of heterosexual couples, thus inferences should not be extended for those in same-sex relationships or adolescent/university dating couples.

## Clinical Implications

Because of the nature of a meta-analysis, we cannot make statements as to causality or directionality of substance use and IPV. For example, we do not know if individuals used substances and then perpetrated IPV or were victimized, likewise we do not know if individuals used substances as a coping mechanism after the perpetration or victimization. This meta-analysis still offers several implications for clinicians working with individuals, couples, or groups who use substances and/or have been in a violent relationship with an intimate partner, but these implications speak to the co-occurrence of substance use and IPV, not the causality or directionality of that link. A clinician who typically only conducts a cursory assessment of substance use, would benefit from understanding that if their clients meet the criteria for abuse, dependence, or other substance use problems, this is a higher risk marker for IPV, in that abuse/dependence is more likely to co-occur with IPV than non-dependence use. However, even if clients do not meet full criteria for abuse/dependence, clinicians should still assess for how

frequent the client consumes alcoholic beverages. For, compared to less frequent alcohol consumption, there is a stronger link between IPV perpetration and frequent alcohol use.

Similar to alcohol use frequency, for the clients' safety and well-being it is important to assess the frequency and quantity of illicit drug use. Some clinicians may be skeptical about whether or not clients downplay their recent frequency of illicit drug use, but simply whether or not clients report ever having used illicit drugs is a significantly stronger risk marker for IPV, compared to the frequency with which they have used illicit drugs. Furthermore, just because clients do not use stimulant drugs (i.e., amphetamines and cocaine), does not mean other "safer" illicit drugs are less strongly be linked to IPV. Marijuana, for example, has recently been legalized for recreational or medicinal use in several states, yet marijuana has just as strong a link with IPV as other stimulant drugs such as cocaine and amphetamines. Similarly, stimulants and non-stimulants have a very similar relationship with perpetration of IPV and a similar relationship with IPV victimization. Thus, regardless of the higher or lower risk associated with individual using a particular illicit drug (Lachenmeier & Rehm, 2015), that drug may have the same strength of association with IPV as more "dangerous" illicit drugs. Finally, clinicians may want to inquire whether the client has been exposed to violence (or threat of violence) as a result of being a participant in the larger systemic illicit drug market, as this could significantly impact the relationship between substance use and IPV.

## Future Directions & Conclusion

As the research on substance use and IPV continues to grow at an exponential rate, researchers are strongly encouraged to report bivariate data which can be used for future meta-analytic inquiry. Similarly, researchers should strongly consider including specific drug types (instead of an overarching, generic, illicit drug use variable) in their future studies so we can

learn more about the differences between drug types and IPV. Likewise, if researchers are going to measure frequency of substances use in the past 3, 6, or 12 months, it would be beneficial for that data to be converted to more detailed categories of frequency of use per week or per month to get a better sense of the differences between frequencies of use.

Most substance use and IPV research focuses on the effect of substance use and the outcome for the user, but the field may benefit from broadening our scope to explore the various effects substance use may have on the relationship. Considering how integrative theories suggest an interplay of factors present in the link between substance use and IPV, it may be beneficial to shift our focus from individual factors (the user's characteristics) to the relational factors (factors in the relationship and/or the partner's characteristics). Even though the bivariate link between substance use and IPV may be small in magnitude, substance use may have strong interaction effects with user, partner, and relationship characteristics. Perhaps there is an interaction effect between the user's marijuana use and their partner's emotional abuse which predicts the user's IPV perpetration or victimization. Perhaps the user's alcohol abuse/dependence significantly interacts with their partner's experience of violence in their family of origin with IPV, whereas there may not be any significant interaction with general alcohol use (not characterized by abuse/dependence). As the field continues to study the link between substance use and IPV, we would benefit from exploring the interacting actor and partner effects present in that violent relationship.

Longitudinal studies tracking the type, manner, frequency, and consequences of substance use would also be beneficial for finding potential periods of substance use when IPV is more likely to occur. Similarly, a longitudinal substance use and IPV meta-analysis might get closer to looking at the time-sequenced relationship between IPV and substance use. A

multivariate meta-analysis measuring substance use and IPV would also help test this link while accounting for other important contextual factors. Finally, a meta-analysis looking at substance use and IPV in same-sex relationships would be beneficial.

In conclusion, this meta-analysis enhances our understanding of the overall link between substance use, alcohol use, and drug use with IPV perpetration and victimization. These findings clearly establish an empirical link between substance use and IPV, however this link can be significantly moderated by a number of factors, such as substance type, measurement of substance use, gender, and direction of violence. By focusing on certain types of substances, how substance use is measured, and what relational characteristics may interact with the substance use, future research can increase our understanding of the specific context which may affect the link between substance use and IPV.

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# Appendix A - Characteristics of Previous Meta-Analyses

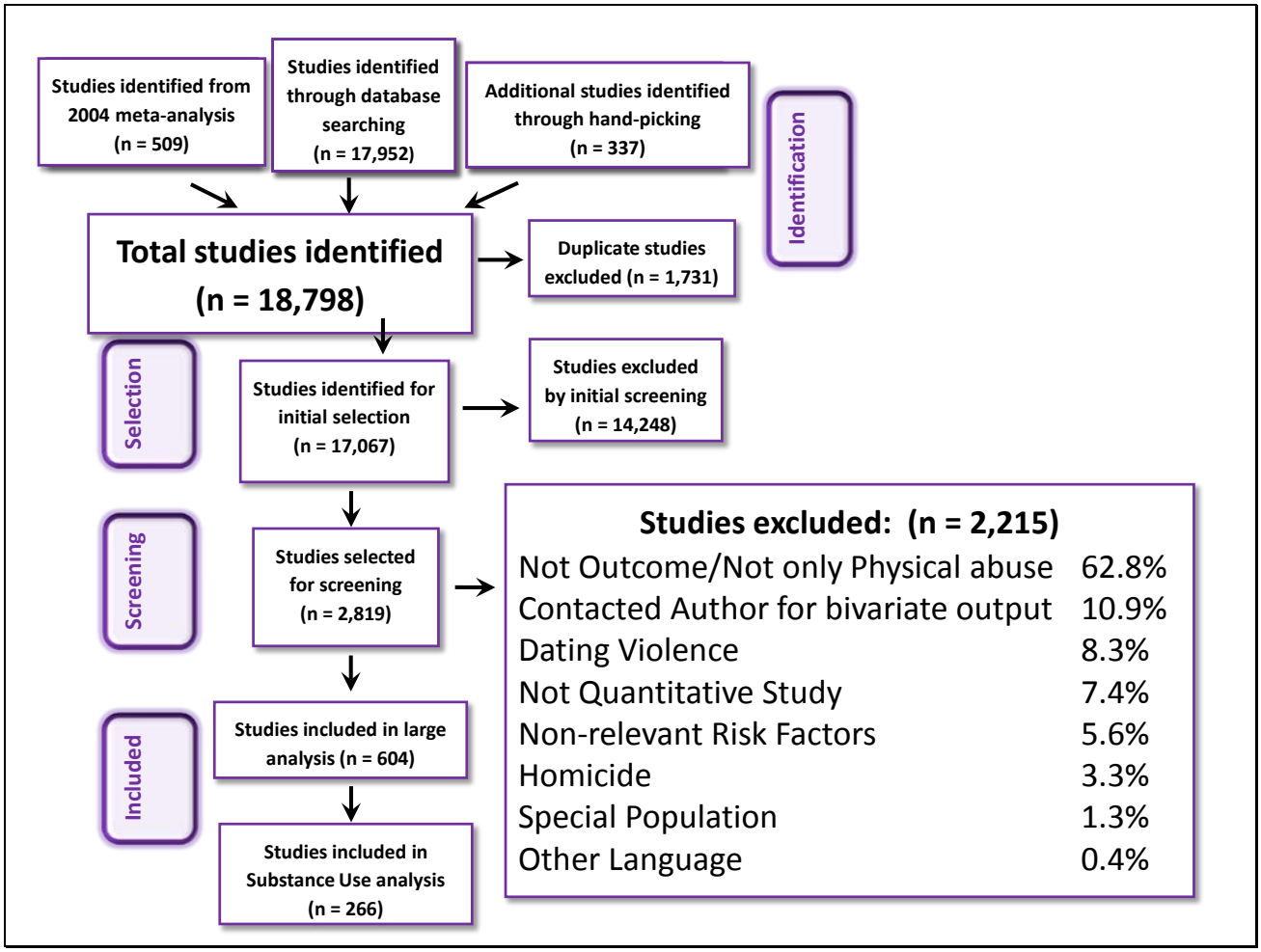
Table 1 Previous Meta-Analyses' Characteristics

| Author(s) | MP | MV | FP | FV | Alcohol Use | Drug Use | Different Drug types | # Substance *ES* (Physical IPV) | # of Studies (Physical IPV) |
|---|---|---|---|---|---|---|---|---|---|
| Hotaling & Sugarman, 1986 | X | | | X | X | X | | 20 | 13 |
| Golding, 1999 | | | | X | X | X | | 14 | 10 |
| Schumacher et al., 2001 | X | | | X | X | X | | 19 | 14 |
| Ferrer et al., 2004 | X | | | | X | X | | 20 | 12 |
| Stith et al., 2004 | X | | | X | X | X | | 38 | 27 |
| Foran & O'Leary, 2008 | X | | X | | X | | | 55 | 50 |
| Moore et al., 2008 | X | X | X | X | | X | X | 319 | 67 |
| **The current study** | **X** | **X** | **X** | **X** | **X** | **X** | **X** | **983** | **287** |

*MP* = Male substance use and male perpetration, *MV* = Male substance use and male victimization, *FP* = Female substance use and female perpetration, *FV* = Female substance use and female victimization, *ES*= Effect sizes

# Appendix B - Flowchart

Figure 1 Flowchart



# Appendix C - Trim and Fill Funnel Plots

Figure 2 Overall Substance Use and Perpetration (7 "Missing" Studies on the Right)

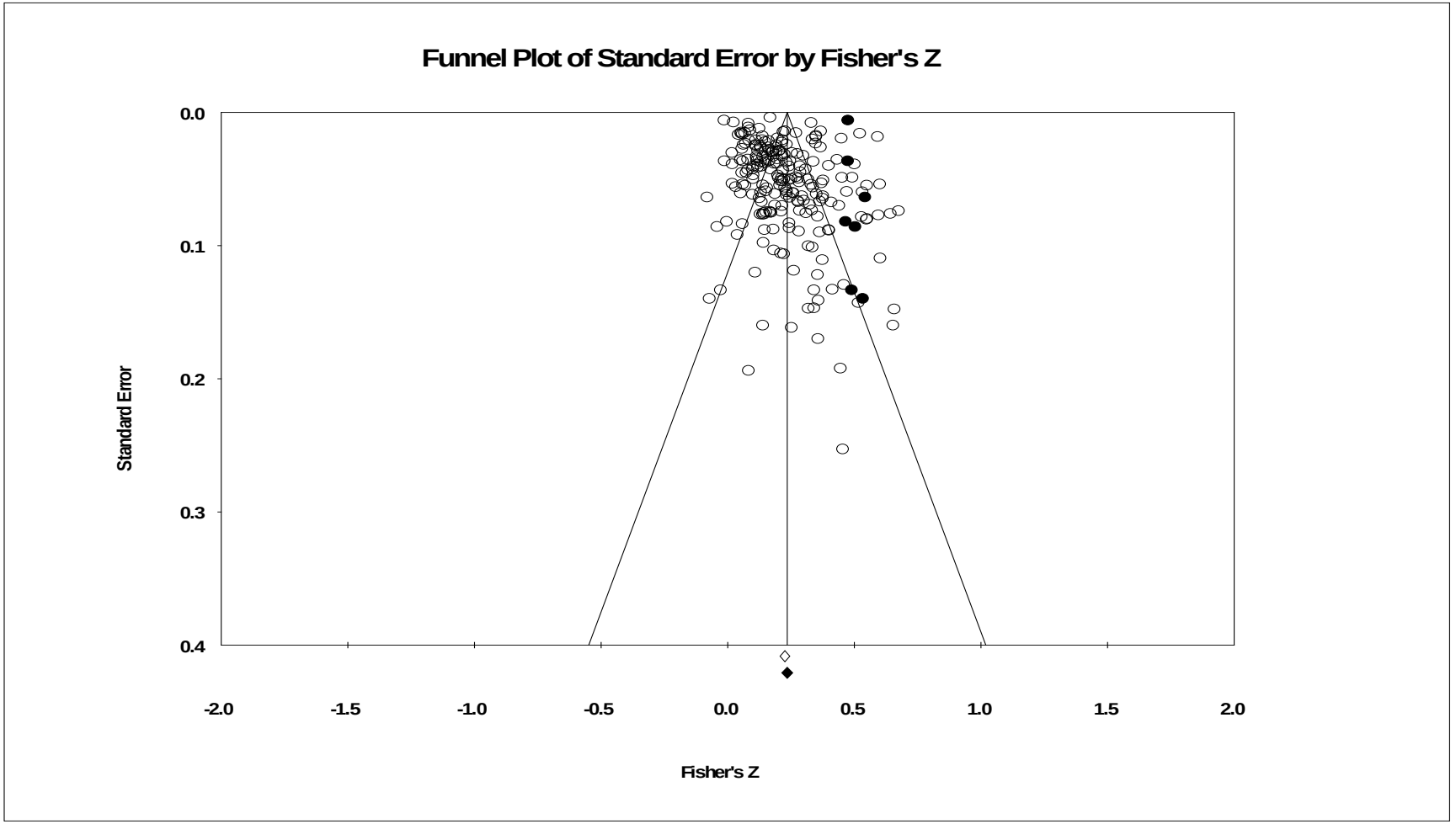


Figure 3 Alcohol Use and Perpetration (7 "Missing" Studies on the Right)

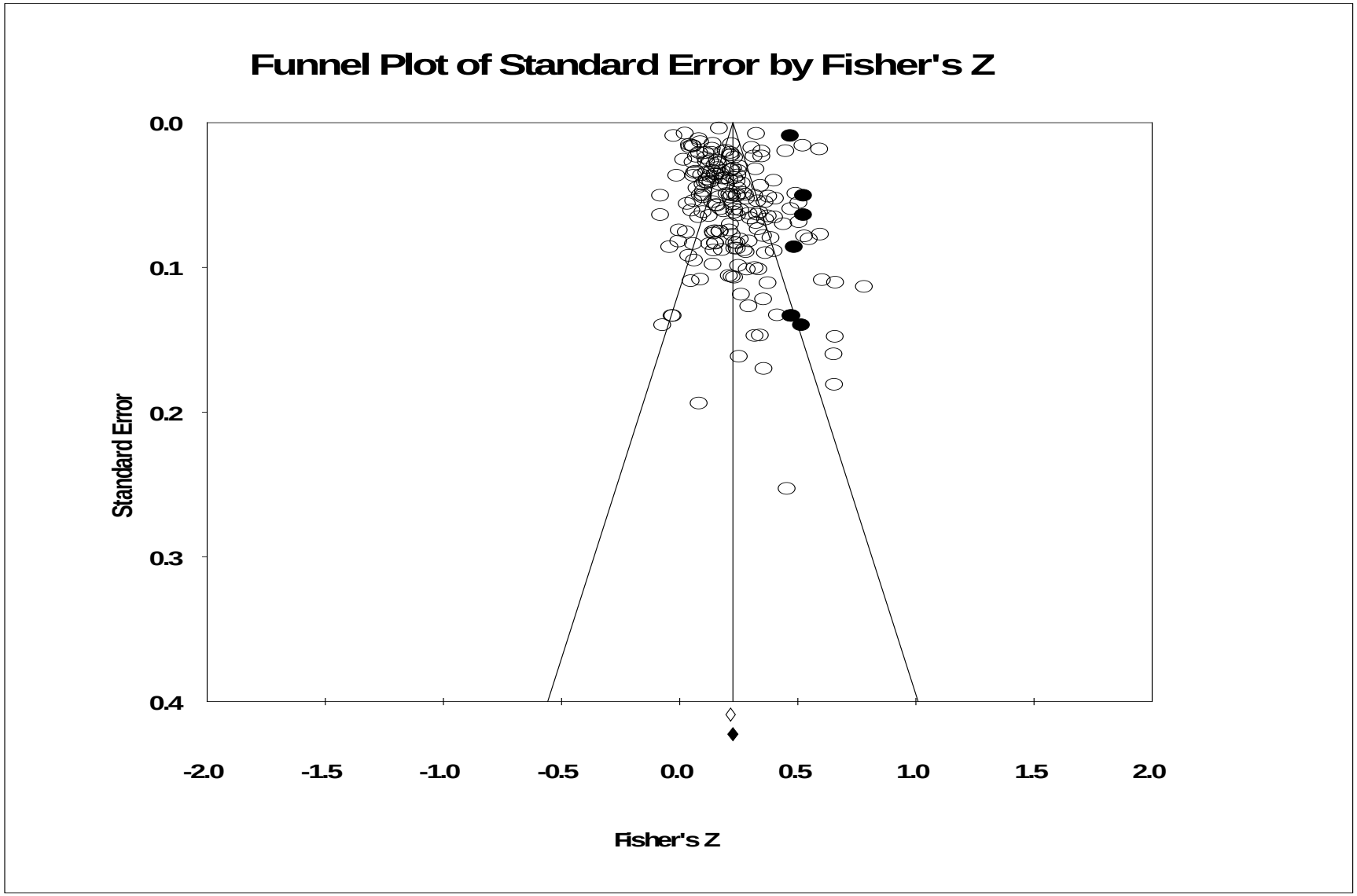


Figure 4 Drug Use and Perpetration (2 "Missing" Studies on the Right)

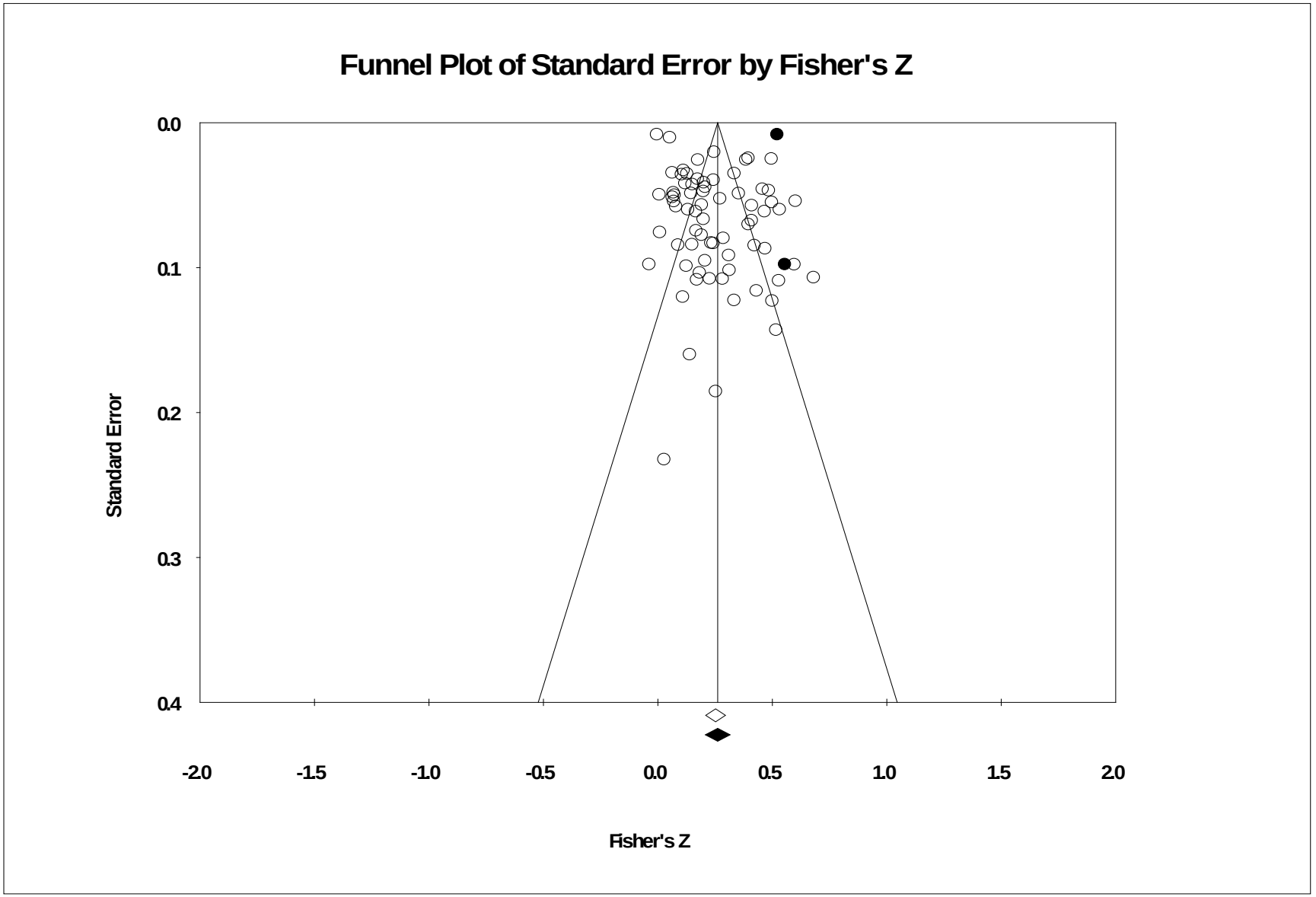


Figure 5 Overall Substance Use and Victimization (26 "Missing" Studies on the Left)

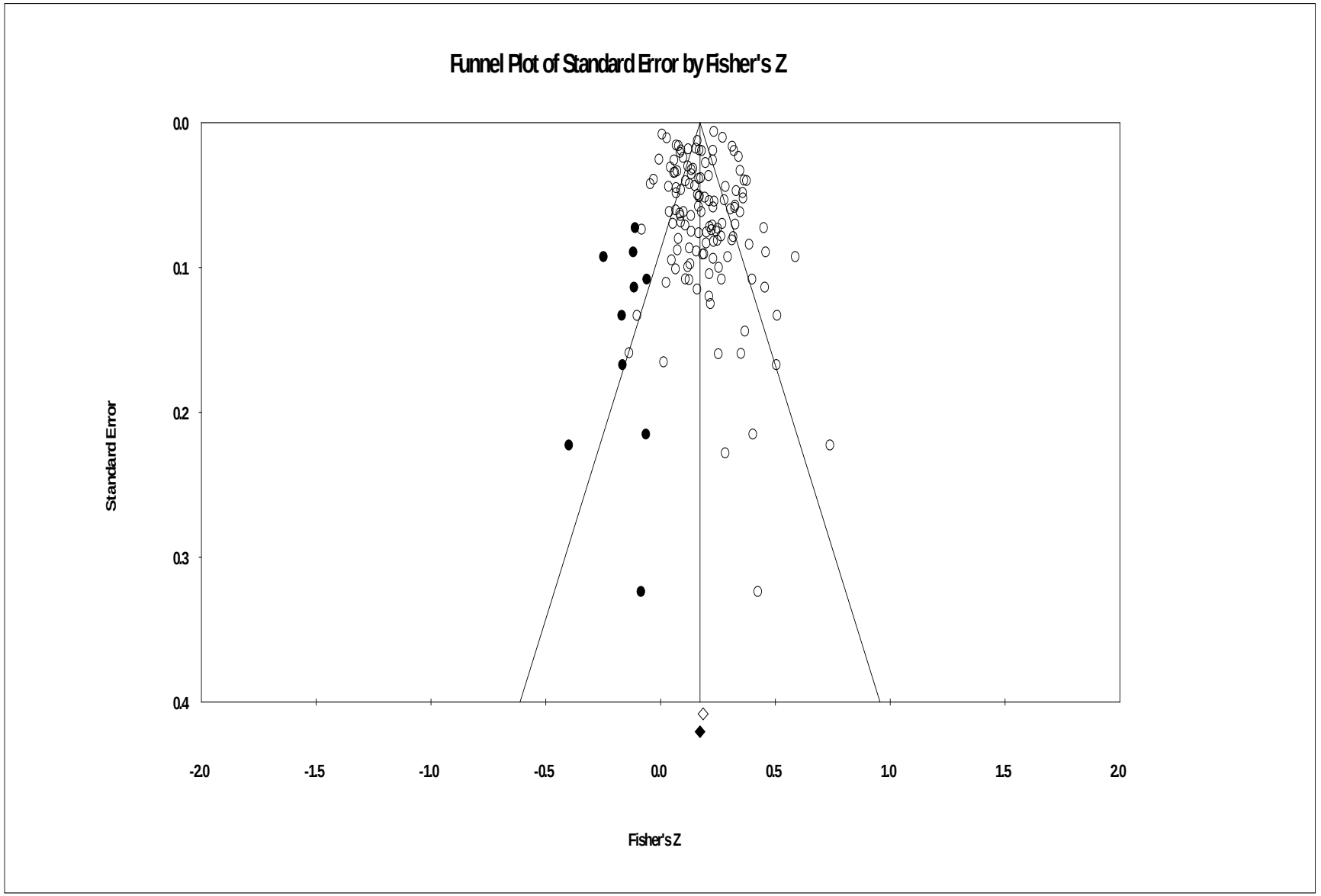


Figure 6 Alcohol Use and Victimization (10 "Missing" Studies on the Left)

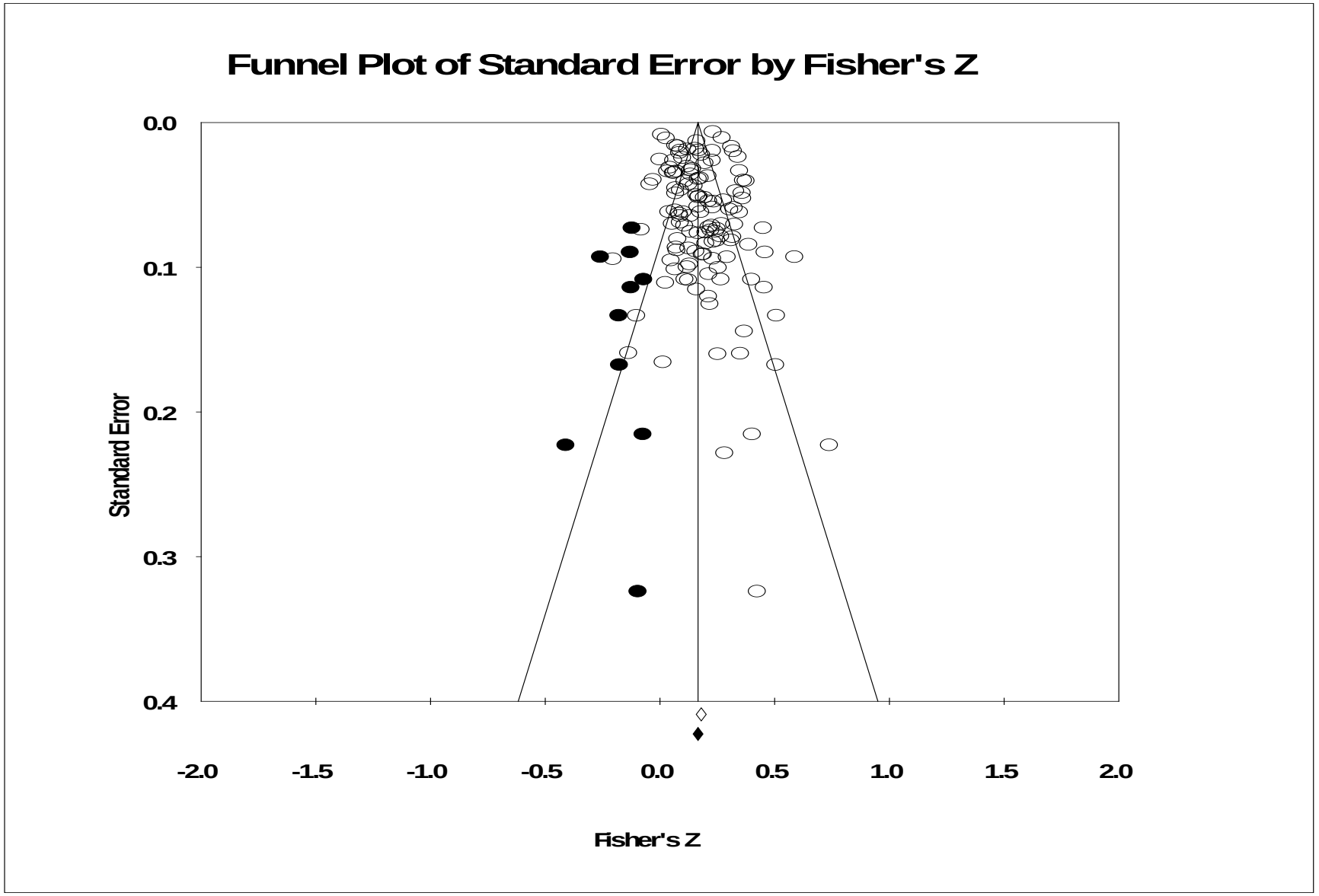
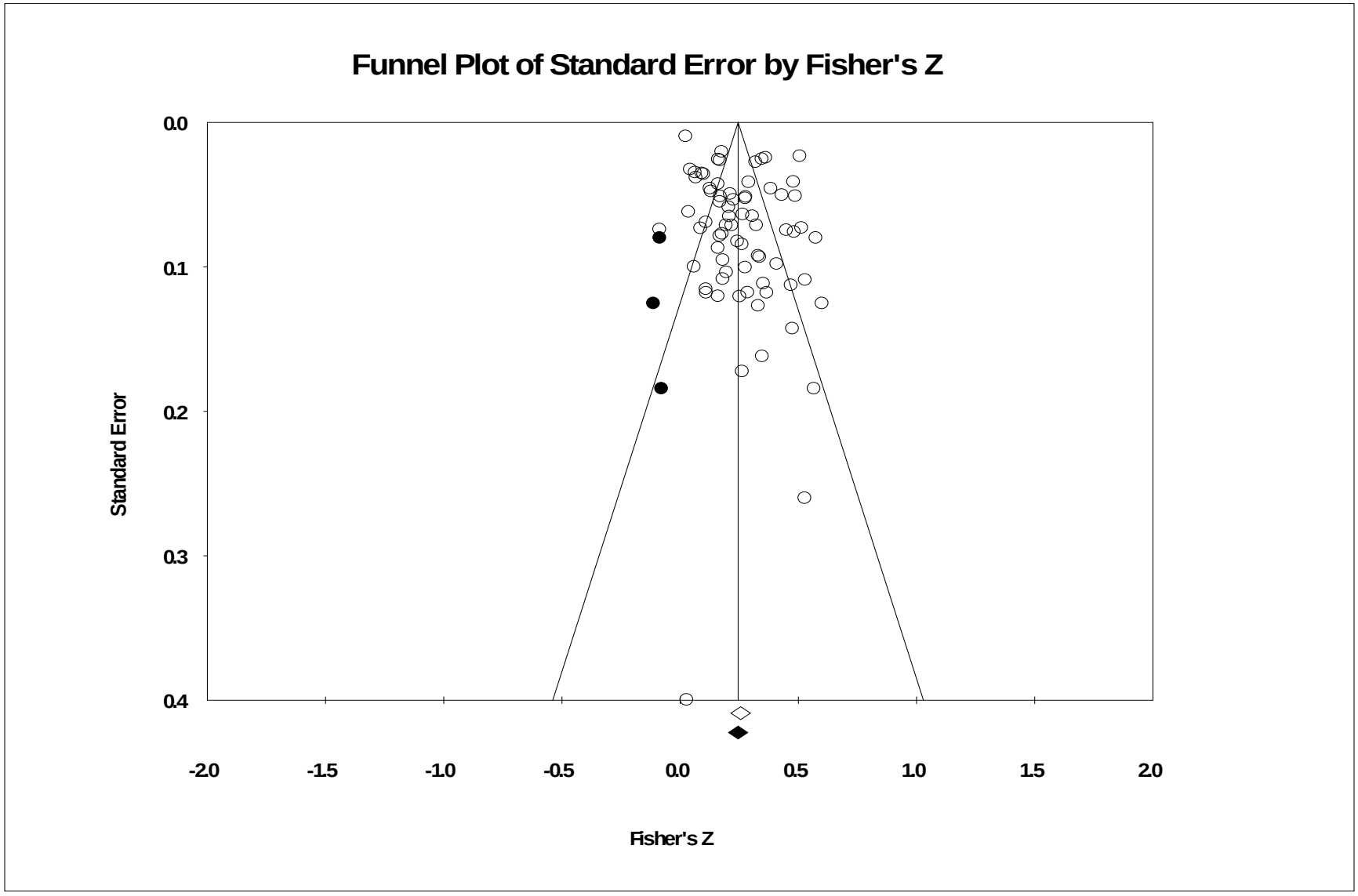


Figure 7 Drug Use and Victimization (3 "Missing" Studies on the Left)



# Appendix D - Results Tables

Table 2 Study and Sample Characteristics

| Characteristics | *Number of Studies (k)* |
|---|---|
| Source of data | |
| Journal Article/Book Chapter | 255 |
| Dissertation/Thesis/Unpublished/Presentation | 32 |
| Date of publication | |
| 1979-1989 | 19 |
| 1990-1999 | 55 |
| 2000-2009 | 139 |
| 2010-2013 | 74 |
| Sample size (total *N = 627,726*) | |
| < 100 | 39 |
| 100-249 | 74 |
| 250-499 | 51 |
| 500-999 | 46 |
| 1,000-2,999 | 44 |
| 3,000-9,999 | 20 |
| 10,000-30,000 | 9 |
| > 30,000 | 4 |
| Location of sample | |
| International | 81 |
| National (USA) | 206 |
| Sample type | |
| Military | 9 |
| National/Community | 97 |
| Social services | 2 |
| Hospital/PediaEmergency care | 27 |
| Gynecology/Pediatric clinic | 6 |
| Psychiatrist/Psychologist/Mental health clinic | 12 |
| Couples treatment | 3 |
| Women's shelter | 4 |
| Substance abuse treatment program | 19 |
| Batterer intervention program | 14 |
| Other/Unknown | 25 |
| Multiple sample types | 69 |

Table 3 Types of Effect Sizes for IPV Perpetration and Victimization

| Types of Effect Sizes | *# of Effect Sizes (k)* |
|---|---|
| Total # of Effect Sizes included for analysis | 983 |
| Substance use (alcohol/drug variable) and IPV | 39 |
| Female substance use | 15 |
| Male substances use | 18 |
| Undifferentiated gender substance use | 6 |
| Alcohol use and IPV | 637 |
| Female alcohol use | 234 |
| Male alcohol use | 367 |
| Undifferentiated gender alcohol use | 36 |
| Drug use and IPV | 306 |
| Female drug use | 130 |
| Drug type specified | 62 |
| Unspecified drug type | 68 |
| Male drug use | 134 |
| Drug type specified | 64 |
| Unspecified drug type | 71 |
| Undifferentiated gender drug use | 42 |
| Drug type specified | 28 |
| Unspecified drug type | 14 |

*Note:* Originally 994 unique effect sizes were gathered, but it was decided not to include 11 outlier effect sizes (more than 3 *SD* in either direction from the mean ES for perpetration or victimization) gathered from five studies (Bevan & Higgins, 2002; Broach, 2004, Drapkin et al., 2004; Hastings & Hamberger, 1988; Mignone, 2006) in the substance use meta-analysis.

Table 4 Duval and Tweedie's Trim and Fill (Random Effects), Classic Fail-Safe N, and Orwin's Fail-Safe N Tests for Substance use with IPV Perpetration and Victimization

| | | Trim and Fill | Classic | Orwin's Fail-Safe N |
|---|---|---|---|---|
| Risk Marker | *k* | Imputed Studies | Fail-Safe N | *r to .10* |
| *Perpetration* | | | | |
| Overall Substance Use | 214 | 7 | 376,246 | 146 |
| Alcohol Use | 192 | 7 | 245,682 | 140 |
| Drug Use | 71 | 2 | 26,037 | 34 |
| | | | | |
| *Victimization* | | | | |
| Overall Substance Use | 145 | 26 | 135,393 | 98 |
| Alcohol Use | 128 | 10 | 66,894 | 75 |
| Drug Use | 72 | 3 | 24,393 | 62 |

Table 5 Study Moderator Analysis for Substance use and IPV Perpetration and Victimization

| Moderator | *k* | Mean *r* | 95% CI | $Q^b$ | *p*-value |
|---|---|---|---|---|---|
| *Perpetration* | | | | | |
| Published vs. Unpublished Study | | | | | |
| Published | 188 | .216*** | [0.20, 0.24] | 3.21 | .073 |
| Unpublished | 26 | .268*** | [0.21, 0.32] | | |
| Clinical vs. Non-Clinical Sample | | | | | |
| Clinical | 68 | .247*** | [0.21, 0.28] | 2.68 | .102 |
| Non-Clinical | 146 | .213*** | [0.19, 0.24] | | |
| Dyadic vs. Non-Dyadic Data | | | | | |
| Dyadic | 50 | .196*** | [0.16, 0.23] | 2.64 | .105 |
| Non-Dyadic | 164 | .232*** | [0.21, 0.25] | | |
| Severe vs. Moderate Violence† | | | | | |
| Severe Violence | 20 | .210*** | [0.12, 0.29] | .402 | .526 |
| Non-Severe Violence | 15 | .168*** | [0.07, 0.26] | | |
| *Victimization* | | | | | |
| Published vs. Unpublished Study | | | | | |
| Published | 132 | .196*** | [0.17, 0.22] | .753 | .385 |
| Unpublished | 13 | .226*** | [0.16, 0.29] | | |
| Clinical vs. Non-Clinical Sample | | | | | |
| Clinical | 54 | .210*** | [0.14, 0.24] | .699 | .403 |
| Non-Clinical | 91 | .191*** | [0.17, 0.22] | | |
| Dyadic vs. Non-Dyadic Data | | | | | |
| Dyadic | 27 | .134*** | [0.09, 0.18] | **10.17** | **.001** |
| Non-Dyadic | 118 | .213*** | [0.19, 0.23] | | |
| Severe vs. Moderate Violence† | | | | | |
| Severe Violence | 24 | .207*** | [0.14, 0.27] | .240 | .624 |
| Non-Severe Violence | 17 | .182*** | [0.11, 0.25] | | |

*Note: k* = number of studies; *r* = point estimate of the effect size; CI = confidence interval; $Q^b$ = Between-Category Test of Homogeneity; † = subgroups within studies were used for this severe vs. moderate violence analysis.
* $p < .05$; ** $p < .01$; *** $p < .001$.

Table 6 Effect Sizes for the Link Between Substance Use and IPV Perpetration and Victimization

| Variable | *k* | Mean *r* | 95% CI | $Q^w$ | $I^2$ |
|---|---|---|---|---|---|
| *Perpetration* | | | | | |
| Substance Use and Perpetration† | 215 | .223*** | [0.20, 0.24] | 5297.67*** | 95.96 |
| Alcohol Use | 376 | .204*** | [0.19, 0.22] | 5040.47*** | 91.56 |
| Drug Use | 151 | .230*** | [0.20, 0.26] | 1471.25*** | 89.90 |
| | | | $Q^b$ (1) = 2.91, $p$ =.088 | | |
| Substance Use and Perpetration | | | | | |
| Female | 109 | .170*** | [0.14, 0.20] | 808.78*** | 86.65 |
| Male | 399 | .227*** | [0.21, 0.24] | 4175.78*** | 90.47 |
| | | | **$Q^b$ (1) = 12.07, $p$ <.001** | | |
| Alcohol Use and Perpetration | | | | | |
| Female | 77 | .149*** | [0.12, 0.18] | 494.92*** | 84.64 |
| Male | 277 | .223*** | [0.21, 0.24] | 3436.83*** | 91.97 |
| | | | **$Q^b$ (1) = 14.36, $p$ <.001** | | |
| Drug Use and Perpetration | | | | | |
| Female | 31 | .222*** | [0.16, 0.28] | 195.10*** | 84.62 |
| Male | 103 | .244*** | [0.21, 0.27] | 493.49*** | 79.33 |
| | | | $Q^b$ (1) = 0.32 $p$ =.571 | | |
| *Victimization* | | | | | |
| Substance Use and Victimization† | 146 | .197*** | [0.18, 0.22] | 2365.31*** | 93.87 |
| Alcohol Use | 231 | .175*** | [0.15, 0.19] | 2055.29*** | 88.81 |
| Drug Use | 149 | .227*** | [0.20, 0.25] | 1113.84*** | 87.34 |
| | | | **$Q^b$ (1) = 11.17, $p$ < .001** | | |
| Substance Use and Victimization | | | | | |
| Female | 262 | .209*** | [0.19, 0.23] | 2130.31*** | 87.92 |
| Male | 91 | .173*** | [0.14, 0.20] | 700.36*** | 86.55 |
| | | | **$Q^b$ (1) = 4.17, $p$ =.041** | | |
| Alcohol Use and Victimization | | | | | |
| Female | 162 | .181*** | [0.16, 0.20] | 1430.85*** | 88.75 |
| Male | 58 | .148*** | [0.11, 0.18] | 423.86*** | 86.55 |
| | | | $Q^b$ (1) = 2.53, $p$ =.112 | | |
| Drug Use and Victimization | | | | | |
| Female | 90 | .255*** | [0.22, 0.29] | 707.91*** | 87.42 |
| Male | 31 | .208*** | [0.14, 0.27] | 253.90*** | 88.18 |
| | | | $Q^b$ (1) = 1.61, $p$ =.203 | | |

*Note: k* = number of studies or effect sizes; *r* = point estimate of the effect size; CI = confidence interval; $Q^b$ = heterogeneity of between-group differences with k-1 degrees of freedom;; † = for the overall effect size each study was the unit analysis, but subgroups were used for all comparison analyses. * $p < .05$;*** $p < .001$

Table 7 Subcategories of Alcohol Use with Perpetration of IPV

| Measures for Alcohol Use | *k* | Mean *r* | 95% CI | $Q^b$ | *p*-value |
|---|---|---|---|---|---|
| *Alcohol Use and Perpetration of IPV* | | | | | |
| Comparison of Alcohol Measures | | | | | |
| Alcohol Consequence Measures | 195 | .221*** | [0.20, 0.24] | 3.57 | .059 |
| Alcohol Consumption Measures | 175 | .190*** | [0.17, 0.21] | | |
| Alcohol Consequence Measures | | | | | |
| Abuse/Dependence | 90 | .210*** | [0.18, 0.24] | | |
| Diagnosis | 26 | .249*** | [0.20, 0.30] | 1.98 | .371 |
| Problem Drinking | 79 | .214*** | [0.19, 0.24] | | |
| Alcohol Consumption Measures | | | | | |
| Excessive Drinking | 62 | .208*** | [0.16, 0.25] | | |
| Frequency | 40 | .153*** | [0.09, 0.21] | | |
| Quantity | 20 | .133** | [0.05, 0.22] | 6.67 | .154 |
| Quantity-Frequency | 18 | .172*** | [0.09, 0.26] | | |
| Use | 35 | .239*** | [0.18, 0.30] | | |
| Excessive Drinking | | | | | |
| Heavy/binge drinking | 27 | .182*** | [0.12, 0.24] | 1.19 | .275 |
| Frequency of drunkenness/binging | 35 | .229*** | [0.17, 0.28] | | |
| Frequency | | | | | |
| Occasional use (1-4 times a month) | 7 | .158** | [0.05, 0.27] | | |
| Few times a week (1-3 days) | 7 | .258*** | [0.15, 0.36] | **22.47** | **<.001** |
| Daily/Almost daily (4-7 days) | 8 | .314*** | [0.21, 0.41] | | |
| "Frequency" of use | 18 | .026 | [-.05, 0.10] | | |

*Note: k* = number of effect sizes; *r* = point estimate of the effect size; CI = confidence interval; $Q^b$ = heterogeneity of between-group differences with k-1 degrees of freedom.
** $p < .01$; *** $p < .001$

Table 8 Subcategories of Alcohol Use with IPV Victimization

| Measures for Alcohol Use | *k* | Mean *r* | 95% CI | $Q^b$ | *p*-value |
|---|---|---|---|---|---|
| *Alcohol Use and IPV Victimization* | | | | | |
| Comparison of Alcohol Measures | | | | | |
| Alcohol Consequence Measures | 133 | .201*** | [0.18, 0.22] | **10.97** | **< .001** |
| Alcohol Consumption Measures | 100 | .139*** | [0.11, 0.17] | | |
| Alcohol Consequence Measures | | | | | |
| Abuse/Dependence | 65 | .221*** | [0.18, 0.26] | 2.33 | .313 |
| Diagnosis | 22 | .170*** | [0.10, 0.24] | | |
| Problem Drinking | 46 | .188*** | [0.14, 0.23] | | |
| Alcohol Consumption Measures | | | | | |
| Excessive Drinking | 26 | .149*** | [0.10, 0.20] | 4.60 | 0.33 |
| Frequency | 24 | .136*** | [0.08, 0.19] | | |
| Quantity | 16 | .092* | [0.02, 0.16] | | |
| Quantity-Frequency | 9 | .100* | [0.1, 0.18] | | |
| Use | 25 | .176*** | [0.12, 0.23] | | |
| Excessive Drinking | | | | .036 | .849 |
| Heavy/binge drinking | 13 | .151*** | [0.10, 0.20] | | |
| Frequency of drunkenness/binging | 13 | .144*** | [0.09, 0.20] | | |
| Frequency | | | | 3.35 | .341 |
| Occasional use (1-4 times a month) | 7 | .159* | [0.04, 0.28] | | |
| Few times a week (1-3 days) | 1 | .212 | [-.13, 0.51] | | |
| Daily/Almost daily (4-7 days) | 2 | .301* | [0.07, 0.50] | | |
| "Frequency" of use | 14 | .088 | [-.01, 0.18] | | |

*Note: k* = number of effect sizes; *r* = point estimate of the effect size; CI = confidence interval; $Q^b$ = heterogeneity of between-group differences with k-1 degrees of freedom.
* $p < .05$; ** $p < .01$; *** $p < .001$

Table 9 Subcategories of Female Alcohol Use with Perpetration of IPV

| Measures for Female Alcohol Use | *k* | Mean *r* | 95% CI | $Q^b$ | *p*-value |
|---|---|---|---|---|---|
| *Female Alcohol Use and Perpetration* | | | | | |
| Comparison of Alcohol Measures | | | | | |
| Alcohol Consequence Measures | 52 | .156*** | [0.12, 0.19] | .634 | .426 |
| Alcohol Consumption Measures | 25 | .132*** | [0.08, 0.18] | | |
| Alcohol Consequence Measures | | | | | |
| Abuse/Dependence | 26 | .162*** | [0.11, 0.21] | | |
| Diagnosis | 8 | .146*** | [0.05, 0.24] | .093 | .955 |
| Problem Drinking | 18 | .156*** | [0.09, 0.22] | | |
| Alcohol Consumption Measures | | | | | |
| Excessive Drinking | 9 | .179*** | [0.10, 0.26] | | |
| Frequency | 5 | -.011 | [-.14, 0.12] | | |
| Quantity | 4 | .104 | [-.03, 0.24] | 6.67 | .154 |
| Quantity-Frequency | 2 | .124 | [-.07, 0.31] | | |
| Use | 5 | .166** | [0.05, 0.28] | | |
| Excessive Drinking | | | | | |
| Heavy/binge drinking | 4 | .238*** | [0.13, 0.34] | 2.22 | .136 |
| Frequency of drunkenness/binging | 5 | .128* | [0.03, 0.23] | | |
| Frequency | | | | | |
| Occasional use (1-4 times a month) | - | *n/a* | *n/a* | | |
| Few times a week (1-3 days) | - | *n/a* | *n/a* | *n/a* | *n/a* |
| Daily/Almost daily (4-7 days) | - | *n/a* | *n/a* | | |
| "Frequency" of use | 5 | .005 | [-.08, 0.09] | | |

*Note: k* = number of effect sizes; *r* = point estimate of the effect size; CI = confidence interval; $Q^b$ = heterogeneity of between-group differences with k-1 degrees of freedom.
** $p < .01$; *** $p < .001$

Table 10 Subcategories of Female Alcohol Use with IPV Victimization

| Measures for Female Alcohol Use | *k* | Mean *r* | 95% CI | $Q^b$ | *p*-value |
|---|---|---|---|---|---|
| *Female Alcohol Use and IPV Victimization* | | | | | |
| Comparison of Alcohol Measures | | | | | |
| Alcohol Consequence Measures | 88 | .211*** | [0.18, 0.24] | **6.98** | **.008** |
| Alcohol Consumption Measures | 74 | .152*** | [0.12, 0.18] | | |
| Alcohol Consequence Measures | | | | | |
| Abuse/Dependence | 42 | .231*** | [0.19, 0.27] | | |
| Diagnosis | 14 | .184*** | [0.10, 0.26] | 1.57 | .455 |
| Problem Drinking | 32 | .196*** | [0.15, 0.25] | | |
| Alcohol Consumption Measures | | | | | |
| Excessive Drinking | 17 | .141*** | [0.08, 0.21] | | |
| Frequency | 20 | .155*** | [0.09, 0.22] | | |
| Quantity | 10 | .118* | [0.02, 0.21] | 4.61 | .329 |
| Quantity-Frequency | 7 | .082 | [-.02, 0.19] | | |
| Use | 20 | .202*** | [0.14, 0.27] | | |
| Excessive Drinking | | | | | |
| Heavy/binge drinking | 9 | .147*** | [0.07, 0.22] | .070 | .791 |
| Frequency of drunkenness/binging | 8 | .132** | [0.05, 0.22] | | |
| Frequency | | | | | |
| Occasional use (1-4 times a month) | 7 | .158* | [0.03, 0.28] | | |
| Few times a week (1-3 days) | 1 | .212 | [-.13, 0.51] | 2.33 | .506 |
| Daily/Almost daily (4-7 days) | 2 | .302* | [0.07, 0.50] | | |
| "Frequency" of use | 10 | .109 | [-.00, 0.22] | | |

*Note: k* = number of effect sizes; *r* = point estimate of the effect size; CI = confidence interval; $Q^b$ = heterogeneity of between-group differences with k-1 degrees of freedom.
** $p < .01$; *** $p < .001$

Table 11 Subcategories of Male Alcohol Use with Perpetration of IPV

| Measures for Male Alcohol Use | *k* | Mean *r* | 95% CI | $Q^b$ | *p*-value |
|---|---|---|---|---|---|
| *Male Alcohol Use and Perpetration* | | | | | |
| Comparison of Alcohol Measures | | | | | |
| Alcohol Consequence Measures | 136 | .246*** | [0.22, 0.27] | **4.93** | **.027** |
| Alcohol Consumption Measures | 141 | .204*** | [0.18, 0.23] | | |
| Alcohol Consequence Measures | | | | | |
| Abuse/Dependence | 61 | .231*** | [0.20, 0.26] | | |
| Diagnosis | 18 | .307*** | [0.25, 0.36] | .093 | .955 |
| Problem Drinking | 57 | .234*** | [0.20, 0.27] | | |
| Alcohol Consumption Measures | | | | | |
| Excessive Drinking | 48 | .218*** | [0.17, 0.27] | | |
| Frequency | 34 | .176*** | [0.11, 0.24] | | |
| Quantity | 15 | .148** | [0.05, 0.24] | 4.62 | .392 |
| Quantity-Frequency | 15 | .179*** | [0.09, 0.27] | | |
| Use | 29 | .252*** | [0.19, 0.32] | | |
| Excessive Drinking | | | | | |
| Heavy/binge drinking | 19 | .194*** | [0.12, 0.26] | .771 | .380 |
| Frequency of drunkenness/binging | 29 | .235* | [0.17, 0.29] | | |
| Frequency | | | | | |
| Occasional use (1-4 times a month) | 7 | .158** | [0.04, 0.27] | | |
| Few times a week (1-3 days) | 7 | .258*** | [0.14, 0.37] | **16.52** | **< .001** |
| Daily/Almost daily (4-7 days) | 8 | .314*** | [0.21, 0.41] | | |
| "Frequency" of use | 12 | .035 | [-.06, 0.13] | | |

*Note: k* = number of effect sizes; *r* = point estimate of the effect size; CI = confidence interval; $Q^b$ = heterogeneity of between-group differences with k-1 degrees of freedom.
** $p < .01$; *** $p < .001$

Table 12 Subcategories of Male Alcohol Use with IPV Victimization

| Measures for Female Alcohol Use | *k* | Mean *r* | 95% CI | $Q^b$ | *p*-value |
|---|---|---|---|---|---|
| *Male Alcohol Use and IPV Victimization* | | | | | |
| Comparison of Alcohol Measures | | | | | |
| Alcohol Consequence Measures | 39 | .166*** | [0.12, 0.21] | 2.09 | .148 |
| Alcohol Consumption Measures | 19 | .113*** | [0.05, 0.17] | | |
| Alcohol Consequence Measures | | | | | |
| Abuse/Dependence | 17 | .172*** | [0.10 0.25] | | |
| Diagnosis | 8 | .149** | [0.04, 0.25] | .151 | .927 |
| Problem Drinking | 14 | .172*** | [0.10, 0.25] | | |
| Alcohol Consumption Measures | | | | | |
| Excessive Drinking | 7 | .173*** | [0.11, 0.24] | | |
| Frequency | 3 | .014 | [-.11, 0.14] | | |
| Quantity | 5 | .062 | [-.03, 0.16] | 6.70 | .152 |
| Quantity-Frequency | 2 | .146 | [-.01, 0.29] | | |
| Use | 2 | .083 | [-.07, 0.24] | | |
| Excessive Drinking | | | | | |
| Heavy/binge drinking | 2 | .205*** | [0.13, 0.28] | .070 | .791 |
| Frequency of drunkenness/binging | 5 | .146** | [0.09, 0.20] | | |
| Frequency | | | | | |
| Occasional use (1-4 times a month) | - | *n/a* | *n/a* | | |
| Few times a week (1-3 days) | - | *n/a* | *n/a* | *n/a* | *n/a* |
| Daily/Almost daily (4-7 days) | - | *n/a* | *n/a* | | |
| "Frequency" of use | 3 | .025 | [-.13, 0.18] | | |

*Note: k* = number of effect sizes; *r* = point estimate of the effect size; CI = confidence interval; $Q^b$ = heterogeneity of between-group differences with k-1 degrees of freedom.
** $p < .01$; *** $p < .001$

Table 13 Different Drug Types and IPV Perpetration and Victimization

| Drug Use and Perpetration/Victimization | *k* | Mean *r* | 95% CI | $Q^b$ | *p*-value |
|---|---|---|---|---|---|
| *Perpetration* | | | | | |
| Different Drugs and Perpetration | | | | | |
| Amphetamines | 6 | .198** | [0.06, 0.33] | | |
| Cocaine | 17 | .215*** | [0.13, 0.30] | | |
| Heroin/Opium | 6 | .055 | [-.10, 0.21] | 6.72 | .151 |
| Marijuana | 22 | .252*** | [0.19, 0.32] | | |
| Other | 3 | .126 | [-.04, 0.28] | | |
| Drug Types and Perpetration | | | | | |
| Stimulants | 31 | .214*** | [0.16, 0.27] | .062 | .802 |
| Non-Stimulants | 46 | .205*** | [0.16, 0.25] | | |
| *Victimization* | | | | | |
| Different Drugs and Victimization | | | | | |
| Amphetamines | 5 | .308*** | [0.13, 0.47] | | |
| Cocaine | 15 | .284*** | [0.19, 0.37] | | |
| Heroin/Opium | 5 | .039 | [-.13, 0.20] | 7.41 | .116 |
| Marijuana | 26 | .229*** | [0.16, 0.30] | | |
| Other | 6 | .234** | [0.09, 0.37] | | |
| Drug Types and Victimization | | | | | |
| Stimulants | 28 | .247*** | [0.18, 0.31] | 1.20 | .272 |
| Non-Stimulants | 47 | .200*** | [0.15, 0.25] | | |

*Note: k* = number of effect sizes; *r* = point estimate of the effect size; CI = confidence interval; $Q^b$ = heterogeneity of between-group differences with k-1 degrees of freedom.
* $p < .05$;*** $p < .001$.

Table 14 Gender Comparison by Drug Type with IPV Perpetration

| Drug Use Gender Differences and Perpetration | *k* | Mean *r* | 95% CI | $Q^b$ | *p*-value |
|---|---|---|---|---|---|
| *Perpetration* | | | | | |
| Cocaine | | | | | |
| Female Perpetration | 3 | .333* | [0.07, 0.56] | 1.06 | .303 |
| Male Perpetration | 9 | .174* | [0.01, 0.33] | | |
| Amphetamines | | | | | |
| Female Perpetration | 1 | .221* | [0.04, 0.37] | .001 | .968 |
| Male Perpetration | 5 | .208*** | [0.11, 0.30] | | |
| Marijuana | | | | | |
| Female Perpetration | 5 | .306*** | [0.21, 0.40] | .487 | .485 |
| Male Perpetration | 10 | .261*** | [0.18, 0.34] | | |
| Heroin/Opium | | | | | |
| Female Perpetration | 2 | -.062 | [-.22, 0.10] | 2.28 | .131 |
| Male Perpetration | 4 | .087 | [-.01, 0.19] | | |
| Stimulants | | | | | |
| Female Perpetration | 8 | .254*** | [0.14, 0.36] | .657 | .418 |
| Male Perpetration | 18 | .197*** | [0.12, 0.27] | | |
| Non-Stimulants | | | | | |
| Female Perpetration | 12 | .205*** | [0.11, 0.29] | .011 | .915 |
| Male Perpetration | 26 | .200*** | [0.14, 0.26] | | |

*Note: k* = number of effect sizes; *r* = point estimate of the effect size; CI = confidence interval; $Q^b$ = heterogeneity of between-group differences with k-1 degrees of freedom.
** $p < .01$; *** $p < .001$

Table 15 Gender Comparison by Drug Type with IPV Victimization

| Drug Use Gender Differences and Victimization | *k* | Mean *r* | 95% CI | $Q^b$ | *p*-value |
|---|---|---|---|---|---|
| *Victimization* | | | | | |
| Cocaine | | | | | |
| Female Victimization | 7 | .328** | [0.11, 0.52] | .037 | .848 |
| Male Victimization | 2 | .286 | [-.12, 0.61] | | |
| Amphetamines | | | | | |
| Female Victimization | 3 | .401 | [-.05, 0.72] | .048 | .826 |
| Male Victimization | 1 | .317 | [-.38, 0.78] | | |
| Marijuana | | | | | |
| Female Victimization | 13 | .281*** | [0.16, 0.40] | .418 | .518 |
| Male Victimization | 5 | .207* | [0.01, 0.39] | | |
| Heroin/Opium | | | | | |
| Female Victimization | 4 | .063 | [-.20, 0.32] | .165 | .680 |
| Male Victimization | 1 | -.055 | [-.51, 0.2] | | |
| Stimulants | | | | | |
| Female Victimization | 26 | .225*** | [0.15, 0.30] | .281 | .596 |
| Male Victimization | 12 | .188** | [0.07, 0.30] | | |
| Non-Stimulants | | | | | |
| Female Victimization | 14 | .271*** | [0.15, 0.38] | .127 | .722 |
| Male Victimization | 7 | .236** | [0.08, 0.38] | | |

*Note: k* = number of effect sizes; *r* = point estimate of the effect size; CI = confidence interval; $Q^b$ = heterogeneity of between-group differences with k-1 degrees of freedom.
** $p < .01$; *** $p < .001$

Table 16 Different Drug Types Compared for Female IPV Perpetration and Victimization

| Drug Use and Perpetration/Victimization | *k* | Mean *r* | 95% CI | $Q^b$ | *p*-value |
|---|---|---|---|---|---|
| *Female Perpetration* | | | | | |
| Different Drugs and Perpetration | | | | | |
| Amphetamines | 1 | .212 | [-.13, 0.51] | | |
| Cocaine | 3 | .340** | [0.14, 0.52] | | |
| Heroin/Opium | 2 | -.055 | [-.32, 0.22] | 6.03 | .197 |
| Marijuana | 5 | .291*** | [0.14, 0.43] | | |
| Other | 1 | .170 | [-.23, 0.51] | | |
| Various Drug Types and Perpetration† | | | | | |
| Stimulants | 8 | .205*** | [0.11, 0.30] | .372 | .542 |
| Non-Stimulants | 13 | .254*** | [0.13, 0.37] | | |
| *Female Victimization* | | | | | |
| Different Drugs and Victimization | | | | | |
| Amphetamines | 3 | .406** | [0.09, 0.65] | | |
| Cocaine | 7 | .326*** | [0.15, 0.48] | | |
| Heroin/Opium | 4 | .064 | [-.18, 0.30] | 4.43 | .351 |
| Marijuana | 13 | .282 | [0.15, 0.40] | | |
| Other | 4 | .345*** | [.11, 0.54] | | |
| Various Drug Types and Victimization† | | | | | |
| Stimulants | 26 | .226*** | [0.14, 0.31] | .392 | .531 |
| Non-Stimulants | 14 | .270*** | [0.16, 0.38] | | |

*Note: k* = number of effect sizes; *r* = point estimate of the effect size; CI = confidence interval; $Q^b$ = heterogeneity of between-group differences with k-1 degrees of freedom; † some studies reported effect sizes that were "stimulant" or "non-stimulant" and these were included in this particular analysis along with the specific drug types which were grouped in to stimulant and non-stimulant categories.
* $p < .05$;*** $p < .001$.

Table 17 Different Drug Types Compared for Male IPV Perpetration or Victimization

| Drug Use and Perpetration/Victimization | *k* | Mean *r* | 95% CI | $Q^b$ | *p*-value |
|---|---|---|---|---|---|
| *Male Perpetration* | | | | | |
| Different Drugs and Perpetration | | | | | |
| Amphetamines | 5 | .199** | [0.06, 0.33] | | |
| Cocaine | 9 | .180*** | [0.08, 0.28] | | |
| Heroin/Opium | 4 | .108 | [-.06, 0.27] | 4.43 | .351 |
| Marijuana | 10 | .261*** | [0.17, 0.35] | | |
| Other | 5 | .114 | [-.05, 0.27] | | |
| Various Drug Types and Perpetration† | | | | | |
| Stimulants | 18 | .200*** | [0.13, 0.27] | .000 | .997 |
| Non-Stimulants | 26 | .200*** | [0.14, 0.26] | | |
| *Male Victimization* | | | | | |
| Different Drugs and Victimization | | | | | |
| Amphetamines | 1 | .317 | [-.20, 0.69] | | |
| Cocaine | 2 | .285 | [-.08, 0.58] | | |
| Heroin/Opium | 1 | -.055 | [-.52, 0.44] | 1.53 | .821 |
| Marijuana | 5 | .207 | [-.03, 0.42] | | |
| Other | 1 | .098 | [-.40, 0.55] | | |
| Various Drug Types and Victimization† | | | | | |
| Stimulants | 7 | .236*** | [0.09, 0.37] | .277 | .599 |
| Non-Stimulants | 12 | .188*** | [0.18, 0.29] | | |

*Note: k* = number of effect sizes; *r* = point estimate of the effect size; CI = confidence interval; $Q^b$ = heterogeneity of between-group differences with k-1 degrees of freedom; † some studies reported effect sizes that were "stimulant" or "non-stimulant" and these were included in this particular analysis along with the specific drug types which were grouped in to stimulant and non-stimulant categories.
* $p < .05$;*** $p < .001$.

Table 18 Subcategories of Drug Use with IPV Perpetration and Victimization

| Various Measures for Drug Use | *k* | Mean *r* | 95% CI | $Q^b$ | *p*-value |
|---|---|---|---|---|---|
| *Drug Use and Perpetration of IPV* | | | | | |
| Drug Consequence Measures | 42 | .297*** | [0.25, 0.34] | **9.30** | **.002** |
| Drug Use Measures | 110 | .203*** | [0.17, 0.24] | | |
| Drug Use Measures | | | | | |
| Frequency | 33 | .130*** | [0.07, 0.19] | **8.33** | **.004** |
| Use | 77 | .234*** | [0.20, 0.27] | | |
| Drug Consequence Measures | | | | | |
| Abuse/Dependence/Diagnosis | 37 | .300*** | [0.24, 0.36] | .078 | .780 |
| Drug-related problems | 5 | .277*** | [0.12, 0.42] | | |
| *Drug Use and IPV Victimization* | | | | | |
| Drug Consequence Measures | 51 | .213*** | [0.17, 0.26] | .831 | .362 |
| Drug Use Measures | 98 | .239*** | [0.21, 0.27] | | |
| Drug Use Measures | | | | | |
| Frequency | 27 | .179*** | [0.12, 0.23] | **6.17** | **.013** |
| Use | 71 | .259*** | [0.23, 0.29] | | |
| Drug Consequence Measures | | | | | |
| Abuse/Dependence/Diagnosis | 48 | .219*** | [0.16, 0.27] | .170 | .680 |
| Drug-related problems | 3 | .172 | [-.05, 0.38] | | |

*Note: k* = number of effect sizes; *r* = point estimate of the effect size; CI = confidence interval; $Q^b$ = heterogeneity of between-group differences with k-1 degrees of freedom.
** $p < .01$; *** $p < .001$

Table 19 Subcategories of Female Drug Use with IPV

| Various Measures for Female Drug Use | *k* | Mean *r* | 95% CI | $Q^b$ | *p*-value |
|---|---|---|---|---|---|
| *Female Drug Use and Perpetration* | | | | | |
| Drug Consequence Measures | 10 | .278*** | [0.16, 0.39] | 1.35 | .244 |
| Drug Use Measures | 22 | .196*** | [0.12, 0.27] | | |
| Drug Use Measures | | | | | |
| Frequency | 9 | .103* | [0.00, 0.20] | **5.91** | **.015** |
| Use | 13 | .252*** | [0.18, 0.32] | | |
| Drug Consequence Measures | | | | | |
| Abuse/Dependence/Diagnosis | 8 | .296** | [0.11, 0.46] | .074 | .785 |
| Drug-related problems | 2 | .241 | [-.12, 0.55] | | |
| *Female Drug Use and Victimization* | | | | | |
| Drug Consequence Measures | 30 | .202*** | [0.14, 0.26] | 2.78 | .096 |
| Drug Use Measures | 66 | .265*** | [0.22, 0.31] | | |
| Drug Use Measures | | | | | |
| Frequency | 17 | .191*** | [0.12, 0.26] | **4.98** | **.026** |
| Use | 49 | .287*** | [0.25, 0.33] | | |
| Drug Consequence Measures | | | | | |
| Abuse/Dependence/Diagnosis | 27 | .210*** | [0.13, 0.29] | .103 | .749 |
| Drug-related problems | 3 | .172 | [-.05, 0.38] | | |

*Note: k* = number of effect sizes; *r* = point estimate of the effect size; CI = confidence interval; $Q^b$ = heterogeneity of between-group differences with k-1 degrees of freedom.
** $p < .01$; *** $p < .001$

Table 20 Subcategories of Male Drug Use with IPV

| Various Measures for Male Drug Use | *k* | Mean *r* | 95% CI | $Q^b$ | *p*-value |
|---|---|---|---|---|---|
| *Male Drug Use and Perpetration* | | | | | |
| Drug Consequence Measures | 28 | .310*** | [0.25, 0.37] | **6.59** | **.010** |
| Drug Use Measures | 73 | .218*** | [0.18, 0.26] | | |
| Drug Use Measures | | | | | |
| Frequency | 17 | .128*** | [0.05, 0.19] | **9.81** | **.002** |
| Use | 56 | .251*** | [0.21, 0.29] | | |
| Drug Consequence Measures | | | | | |
| Abuse/Dependence/Diagnosis | 25 | .310*** | [0.24, 0.37] | .001 | .972 |
| Drug-related problems | 3 | .307*** | [0.11, 0.48] | | |
| *Male Drug Use and Victimization* | | | | | |
| Drug Consequence Measures | 13 | .213*** | [0.12, 0.30] | 2.78 | .096 |
| Drug Use Measures | 18 | .203*** | [0.13, 0.28] | | |
| Drug Use Measures | | | | | |
| Frequency | 4 | .143** | [0.04, 0.24] | 2.08 | .149 |
| Use | 14 | .224*** | [0.18, 0.27] | | |
| Drug Consequence Measures | | | | | |
| Abuse/Dependence/Diagnosis | 13 | .217*** | [0.11, 0.31] | *n/a* | *n/a* |
| Drug-related problems | - | *n/a* | *n/a* | | |

*Note: k* = number of effect sizes; *r* = point estimate of the effect size; CI = confidence interval; $Q^b$ = heterogeneity of between-group differences with k-1 degrees of freedom.
** $p < .01$; *** $p < .001$

# Appendix E - Codesheet

This is the official (corrected) codesheet: NO ☐ YES ☐

Coding discrepancies on these items_____

Total number if coding discrepancies____

Coding discrepancies on these items_____

Total number if coding discrepancies____

DRAFT #7

IPV Code-Sheet

**Risk Assessment Meta-Analysis**

**01) Coder ID Number _____**

**02) Date Coded __/__/__ (mm/dd/yy)**

**03) Study ID Number _____**

**<u>Source Characteristics</u>**

**04) Last names of Author(s) _____**

**_____**

**05) Gender of first author? _____(#)**

**0. Unknown**
**1. Male**
**2. Female**

**06) Was the data collection process funded? _____ (0 = No/Unknown, 1 = Yes)**

**Page#**

**07) If funded, what was the source of funding? _____**

**0. Not Applicable**
**1. Internal funding**
**2. External funding**

**08) List source of funding:**

**Page#** **_____**

Page#

09) **Year of printed Publication _____**

10) **Type of Publication ___ (#)**

1. **Journal Article**
2. **Book Chapter**
3. **Dissertation/Thesis**
4. **Conference Presentation**
5. **Other _____**

Page#

11) **Journal/Book Title _____**

**_____**

Page# 12) **Article/Chapter Title_____**

**_____**

## Sample Characteristics

13) **From where were the participants recruited (clearly circle all that apply)?**

**0. Unknown**
**1. Military**
**2. National**
**3. University/Academic setting (non-clinical)**
**4. Social services**
**5. Hospital setting and Emergency Care**
**~~6. Emergency Care~~**
**7. Obstetrics/Gynecology clinic**

Page# **8. Pediatric clinic**
**9. Psychiatrist/Psychologist /Outpatient Mental Health/Clinic**
**10. Couples treatment**
**11. Women's shelter**
**12. Substance abuse treatment program**
**13. Batterers program**

Page# **14. Prison**
**15. Religious organization**
**16. Community**
**17. Other_____**

14) **Additional Type of Recruitment _____**

| | |
|---|---|
| Page# | **15) Which branch of the military? _____(#)**<br>**0. Not applicable**<br>**1. Army**<br>**2. Marine Corps**<br>**3. Navy**<br>**4. Air Force**<br>**5. Coast Guard**<br>**6. Unknown**<br><br>**16) What is the name of this data set (or brief description of data set)?**<br>**_____**<br><br>**17) What was the combined sample size for <u>this particular study</u>? N = _____**<br><br>**17a) What is the N for each racial/ethnic group in the study?**<br>**White/Caucasian_____ Black/African American_____**<br>**Latino/Hispanic_____ Asian _____ Native American_____ Other_____**<br><br>**18) From where was the sample collected? ____(#)**<br>**1. International**<br>**2. United States**<br>**3. Both**<br><br>**19) From which international country was the sample collected? _____**<br><br>**20) From which region within the United States was the study conducted? ___(#)**<br><br>**0. Not Applicable**<br>**1. Northeast**<br>**2. South**<br>**3. Midwest**<br>**4. West**<br>**5. Various regions**<br>**6. Nationwide**<br>**7. Unknown** |
| Page# | **21) Were immigrants purposefully included in this sample? _____(0= No, 1= Yes)** |
| Page# | **22) Immigrants composed _____% of this sample.** |

## Measurement Characteristics

**23) Who reported the data?_____(#)**

1. Females
2. Males
3. Males and Females
4. Clinicians (*regardless of gender*)

Page#

**24) This IPV data reflects:_____(#)**

| | |
|---|---|
| **Single Gender Data** | **1. Male perpetration/Female victimization**<br>**2. Female perpetration/Male victimization**<br>**3. Both male and female perpetration and victimization (bi-lateral IPV)** |
| **Unrelated, Mixed Gender Data** | **4. Male and female as perpetrators**<br>**5. Male and female as victims**<br>**6. Male and female as both perpetrators/victims**<br>**11. Male perpetration/Female victimization**<br>**12. Female perpetration/Male victimization** |
| **Couples Data** | **7. Male perpetration and female victimization**<br>**8. Female perpetration and male victimization**<br>**9. Both male and female perpetration and victimization (bi-lateral IPV)**<br>**10. Undifferentiated (mixed sample of couples-male and female)** |

Page#

Page#

**25) Were some couples identified as same sex couples?_____(No = 0, Yes = 1)**

**26) Were both partners who were in a relationship together surveyed so that the data reflects this relationship (dyadic)? _____(No = 0, Yes =1)**

Page#

**27) How was the data collected?___(#)**

0. Unknown
1. Internet survey
2. Survey &/or face-to-face interview (collapsed from 2-5)
6. Telephone interview
7. Clinician report
8. Two or more of the above

Page#

**28) Was partner present at the time that the data was reported?____(#)**

0. No/Unknown
1. Yes

| | |
|---|---|
| Page# | **29) Were participants rewarded for their participation?___(#)**<br>**0. No/Unknown**<br>**1. Yes**<br><br>**30) What was the nature of study conducted? ____(#)**<br>**0. Unknown**<br>**1. Longitudinal**<br>**2. Cross-sectional** |
| Page# | **31) Were established instruments used for <u>each</u> physical aggression outcome variable? ____ (0 = No, 1 = Yes, 2 = Both)** |
| Page # | **32) What instrument(s)/scale(s) were used?_____(#)**<br><br>**1. CTS (Straus)/Items from CTS (collapsed 1-2)**<br>**3. Other standardized instrument/Other method_____**<br><br>**33) How did the authors draw the sample? __ (#)**<br>**0. Unknown**<br>**1. Convenience**<br>**2. Representative (National)**<br>**3. Representative (other)**<br>**4. Random**<br>**5. Other_____** |
| Page # | **34) What is your subjective rating of this article? _____(Low 1---------4 High)**<br><br>**35) Need to contact the author? _____** |

**Effect Size: Specific # of risk factor _____ & Name of risk factor _____**

**Brief description of risk factor_____ Page____**

Name of Instrument/Scale_____ **[*N* = _____]**

What data will be used for the effect size?

___ (#)

1. Pearson *r* / Correlation Matrix
2. Hedges' *g*
3. Cohen's *d*
4. *o* (odds ratio)
5. *o* (odds ratio adjusted)
6. *F*-ratio
7. T-test
8. Chi-squared (X^2)
9. Z score

| Who reported |
|---|
| M F U |
| About Whom |
| M F U /// P V B |
| *p* |

| | |
|---|---|
| *Dependent* (or) *Independent* | Group 1 = _____ Mean = _____ SD = _____ *N* = _____<br>Group 2 = _____ Mean = _____ SD = _____ *N* = _____<br>Group 3 = _____ Mean = _____ SD = _____ *N* = _____<br>Group 4 = _____ Mean = _____ SD = _____ *N* = _____ |

**Effect Size: Specific # of risk factor _____ & Name of risk factor _____**

**Brief description of risk factor_____ Page____**

Name of Instrument/Scale_____ **[*N* = _____]**

What data will be used for the effect size?

___ (#)

1. Pearson *r* / Correlation Matrix
2. Hedges' *g*
3. Cohen's *d*
4. *o* (odds ratio)
5. *o* (odds ratio adjusted)
6. *F*-ratio
7. T-test
8. Chi-squared (X^2)
9. Z score

| Who reported |
|---|
| M F U |
| About Whom |
| M F U /// P V B |
| *p* |

| | |
|---|---|
| *Dependent* (or) *Independent* | Group 1 = _____ Mean = _____ SD = _____ *N* = _____<br>Group 2 = _____ Mean = _____ SD = _____ *N* = _____<br>Group 3 = _____ Mean = _____ SD = _____ *N* = _____<br>Group 4 = _____ Mean = _____ SD = _____ *N* = _____ |

**Effect Size: Specific # of risk factor _____ & Name of risk factor _____**

**Brief description of risk factor_____ Page_____**

Name of Instrument/Scale_____ **[*N* = _____]**

What data will be used for the effect size?

___ (#)

1. Pearson *r* / Correlation Matrix
2. Hedges' *g*
3. Cohen's *d*
4. *o* (odds ratio)
5. *o* (odds ratio adjusted)
6. *F*-ratio
7. T-test
8. Chi-squared (X^2)
9. Z score

| Who reported<br>M F U |
|---|
| About Whom<br>M F U /// P V B |
| *p* |

| | |
|---|---|
| *Dependent*<br>(or)<br>*Independent* | Group 1 = _____ Mean = _____ SD = _____ *N* = _____<br>Group 2 = _____ Mean = _____ SD = _____ *N* = _____<br>Group 3 = _____ Mean = _____ SD = _____ *N* = _____<br>Group 4 = _____ Mean = _____ SD = _____ *N* = _____ |

**Effect Size: Specific # of risk factor _____ & Name of risk factor _____**

**Brief description of risk factor_____ Page____**

Name of Instrument/Scale_____ **[*N* = _____]**

What data will be used for the effect size?

___ (#)

1. Pearson *r* / Correlation Matrix
2. Hedges' *g*
3. Cohen's *d*
4. *o* (odds ratio)
5. *o* (odds ratio adjusted)
6. *F*-ratio
7. T-test
8. Chi-squared (X^2)
9. Z score

| Who reported<br>M F U |
|---|
| About Whom<br>M F U /// P V B |
| *p* |

| | |
|---|---|
| *Dependent*<br>(or)<br>*Independent* | Group 1 = _____ Mean = _____ SD = _____ *N* = _____<br>Group 2 = _____ Mean = _____ SD = _____ *N* = _____<br>Group 3 = _____ Mean = _____ SD = _____ *N* = _____<br>Group 4 = _____ Mean = _____ SD = _____ *N* = _____ |

**Effect Size: Specific # of risk factor _____ & Name of risk factor _____**

**Brief description of risk factor_____ Page____**

Name of Instrument/Scale_____ **[*N* = _____]**

What data will be used for the effect size?

___ (#)

1. Pearson *r* / Correlation Matrix
2. Hedges' *g*
3. Cohen's *d*
4. *o* (odds ratio)
5. *o* (odds ratio adjusted)
6. *F*-ratio
7. T-test
8. Chi-squared (X^2)
9. Z score

| Who reported | |
|---|---|
| M F U | |
| About Whom | |
| M F U /// P V B | |
| *p* | |

| | |
|---|---|
| *Dependent* (or) *Independent* | Group 1 = _____ Mean = _____ SD = _____ *N* = _____<br>Group 2 = _____ Mean = _____ SD = _____ *N* = _____<br>Group 3 = _____ Mean = _____ SD = _____ *N* = _____<br>Group 4 = _____ Mean = _____ SD = _____ *N* = _____ |

**Effect Size: Specific # of risk factor _____ & Name of risk factor _____**

**Brief description of risk factor_____ Page____**

Name of Instrument/Scale_____ **[*N* = _____]**

What data will be used for the effect size?

___ (#)

1. Pearson *r* / Correlation Matrix
2. Hedges' *g*
3. Cohen's *d*
4. *o* (odds ratio)
5. *o* (odds ratio adjusted)
6. *F*-ratio
7. T-test
8. Chi-squared (X^2)
9. Z score

| Who reported | |
|---|---|
| M F U | |
| About Whom | |
| M F U /// P V B | |
| *p* | |

| | |
|---|---|
| *Dependent* (or) *Independent* | Group 1 = _____ Mean = _____ SD = _____ *N* = _____<br>Group 2 = _____ Mean = _____ SD = _____ *N* = _____<br>Group 3 = _____ Mean = _____ SD = _____ *N* = _____<br>Group 4 = _____ Mean = _____ SD = _____ *N* = _____ |

**Effect Size: Specific # of risk factor _____ & Name of risk factor _____**

**Brief description of risk factor_____ Page____**

Name of Instrument/Scale_____ **[*N* = _____]**

What data will be used for the effect size?

___ (#)

1. Pearson *r* / Correlation Matrix
2. Hedges' *g*
3. Cohen's *d*
4. *o* (odds ratio)
5. *o* (odds ratio adjusted)
6. *F*-ratio
7. T-test
8. Chi-squared (X^2)
9. Z score

| Who reported |
|---|
| M F U |
| About Whom |
| M F U /// P V B |
| *p* |

| | |
|---|---|
| *Dependent* (or) *Independent* | Group 1 = _____ Mean = _____ SD = _____ *N* = _____<br>Group 2 = _____ Mean = _____ SD = _____ *N* = _____<br>Group 3 = _____ Mean = _____ SD = _____ *N* = _____<br>Group 4 = _____ Mean = _____ SD = _____ *N* = _____ |

**Effect Size: Specific # of risk factor _____ & Name of risk factor _____**

**Brief description of risk factor_____ Page____**

Name of Instrument/Scale_____ **[*N* = _____]**

What data will be used for the effect size?

___ (#)

1. Pearson *r* / Correlation Matrix
2. Hedges' *g*
3. Cohen's *d*
4. *o* (odds ratio)
5. *o* (odds ratio adjusted)
6. *F*-ratio
7. T-test
8. Chi-squared (X^2)
9. Z score

| Who reported |
|---|
| M F U |
| About Whom |
| M F U /// P V B |
| *p* |

| | |
|---|---|
| *Dependent* (or) *Independent* | Group 1 = _____ Mean = _____ SD = _____ *N* = _____<br>Group 2 = _____ Mean = _____ SD = _____ *N* = _____<br>Group 3 = _____ Mean = _____ SD = _____ *N* = _____<br>Group 4 = _____ Mean = _____ SD = _____ *N* = _____ |

**Effect Size: Specific # of risk factor _____ & Name of risk factor _____**

**Brief description of risk factor_____ Page____**

Name of Instrument/Scale_____ **[*N* = _____]**

What data will be used for the effect size?

___ (#)

1. Pearson *r* / Correlation Matrix
2. Hedges' *g*
3. Cohen's *d*
4. *o* (odds ratio)
5. *o* (odds ratio adjusted)
6. *F*-ratio
7. T-test
8. Chi-squared (X^2)
9. Z score

| Who reported |
|---|
| M F U |
| About Whom |
| M F U /// P V B |
| *p* |

| | |
|---|---|
| *Dependent* (or) *Independent* | Group 1 = _____ Mean = _____ SD = _____ *N* = _____<br>Group 2 = _____ Mean = _____ SD = _____ *N* = _____<br>Group 3 = _____ Mean = _____ SD = _____ *N* = _____<br>Group 4 = _____ Mean = _____ SD = _____ *N* = _____ |

**Effect Size: Specific # of risk factor _____ & Name of risk factor _____**

**Brief description of risk factor_____ Page____**

Name of Instrument/Scale_____ **[*N* = _____]**

What data will be used for the effect size?

___ (#)

1. Pearson *r* / Correlation Matrix
2. Hedges' *g*
3. Cohen's *d*
4. *o* (odds ratio)
5. *o* (odds ratio adjusted)
6. *F*-ratio
7. T-test
8. Chi-squared (X^2)
9. Z score

| Who reported |
|---|
| M F U |
| About Whom |
| M F U /// P V B |
| *p* |

| | |
|---|---|
| *Dependent* (or) *Independent* | Group 1 = _____ Mean = _____ SD = _____ *N* = _____<br>Group 2 = _____ Mean = _____ SD = _____ *N* = _____<br>Group 3 = _____ Mean = _____ SD = _____ *N* = _____<br>Group 4 = _____ Mean = _____ SD = _____ *N* = _____ |

# Appendix F - References for Analyses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