# A LONGITUDINAL STUDY OF ALCOHOL USE AND INTIMATE PARTNER VIOLENCE AMONG MEN WITH HIV AND HAZARDOUS ALCOHOL USE IN VIETNAM

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#### **ABSTRACT**

Rebecca Beth Hershow: A Longitudinal Study of Alcohol Use and Intimate Partner Violence Among Men with HIV and Hazardous Alcohol Use in Vietnam (Under the direction of Vivian F. Go)

**Background:** Intimate partner violence (IPV) is an urgent global health problem that is associated with HIV. Alcohol use may lead to male-perpetrated IPV, although little is known about the alcohol-IPV relationship among men with HIV and hazardous alcohol use, a group at high risk for IPV perpetration and forward HIV transmission.

Methods: I conducted three studies using data from a randomized controlled trial in Thai Nguyen, Vietnam. The parent trial evaluated the effects of two alcohol reduction interventions (two-session Brief Intervention [BI]; six-session Combined Intervention [CoI]) on alcohol use as compared to the standard of care (SOC) among antiretroviral treatment patients with hazardous alcohol use. In Study 1, I examined the longitudinal effects of alcohol use (proportion of days alcohol abstinent, heavy drinking, and alcohol use disorder [AUD]) on IPV perpetration among male participants who were married or cohabitating (N=313) using multilevel modeling. In Study 2, I tested whether depressive symptoms moderated the alcohol-IPV relationship. In Study 3, I tested the effects of the BI and CoI on IPV perpetration and alcohol use as a mediator among male participants (N=426) using path analyses.

**Results:** In Study 1, reporting higher levels of alcohol use on average increased odds of IPV perpetration for all alcohol measures. Time-varying effects of alcohol use on IPV perpetration were observed for proportion of days alcohol abstinent and AUD. In Study 2, depressive symptoms weakened the time-varying effects of proportion of days alcohol abstinent and heavy drinking on IPV perpetration. Depressive symptoms did not moderate the relationship

between AUD and IPV perpetration; a main effect of depressive symptoms on IPV perpetration was observed. In Study 3, the BI and CoI reduced IPV perpetration at three months as compared to the SOC, although effects were not sustained at six and 12 months. Alcohol use was not a mediator.

**Conclusions:** Alcohol use is a risk factor for IPV perpetration among Vietnamese men with HIV and hazardous alcohol use. Depressive symptoms and alcohol use may not interact to exacerbate IPV perpetration. Enhanced alcohol reduction interventions addressing IPV and depression should be tested to improve and sustain effects on IPV perpetration.

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## LIST OF ABBREVIATIONS

AIC Akaike information criterion

aOR Adjusted odds ratio

ART Antiretroviral treatment

AUD Alcohol use disorder

AUDIT-C Alcohol Use Disorders Identification Test

AR Autoregressive

BCT Behavioral couples therapy

BI Brief Intervention

BIC Bayesian information criterion

CBT Cognitive Behavioral Therapy

CI Confidence Interval

Col Combined Intervention

CTS2 Conflict Tactics Scale 2

DSM IV Diagnostic and Statistical Manual of Mental Health Disorders, 4th edition

DV Law 2007 Law on Domestic Violence Prevention and Control

GE Law 2006 Law on Gender Equality

ICBI Integrated cognitive-behavioral intervention

ICC Intraclass correlation

ID Identification

IPV Intimate partner violence

IRB Institutional Review Board

I-StoP Integrated treatment for Substance abuse and Partner violence

MET Motivational Enhancement Therapy

MINI Mini International Neuropsychiatric Interview

MMT Methadone Maintenance Treatment

Neg 2 LL Negative 2 log likelihood

OR Odds ratio

U.S. President's Emergency Plan for AIDS Relief **PEPFAR** 

PEth Phosphatidylethanol

PHQ-9 Patient Health Questionnaire-9

People living with HIV **PLHIV** 

**PWID** People who inject drugs

Randomized controlled trial **RCT** 

Reducing Hazardous Alcohol Use & HIV Viral Load: A Randomized Controlled Trial in Antiretroviral Treatment Clinics in Vietnam **REDART** 

SD **Standard Deviation** 

SE Standard Error

SOC Standard of care

**TLFB** Timeline Followback

WHO World Health Organization

#### **CHAPTER 1: INTRODUCTION**

# 1.1. Specific Aims

Male-perpetrated intimate partner violence (IPV) is a major public health problem globally [1, 2]. IPV is defined as psychological, physical, or sexual abuse perpetrated towards an intimate partner [3]. Globally, 30% of women have experienced IPV in their lifetime [2]. IPV leads to numerous health issues and drives HIV infection [2, 4, 5]. Both empirical research and theory suggest a link between alcohol use and IPV perpetration [6-13], including three meta-analyses showing small to moderate effects of alcohol use on IPV perpetration [12-14]. The relationship between alcohol use and IPV perpetration is often explained using two theoretical models. First, alcohol use may lead to IPV perpetration due to immediate psychopharmacological effects of alcohol, such as lowered inhibitions or distorted perceptions of cues [12, 14, 15]. Second, alcohol use may lead to increased relationship conflict, in turn leading to IPV perpetration [9, 10, 16].

It is critical to investigate the impact of alcohol use on IPV perpetration among men with HIV and hazardous alcohol use. The prevalence of hazardous drinking is elevated (36%) among men living with HIV [17], which may be due to vulnerability to social pressure to drink and/or coping responses to HIV-related depressive symptoms [18]. Further, men with HIV and hazardous alcohol use are at especially high risk for forward HIV transmission, as hazardous drinking is associated with decreased viral suppression and antiretroviral treatment (ART) adherence [19-21].

There is a dearth of longitudinal research on the alcohol-IPV relationship among men living with HIV. Since cross-sectional studies cannot establish temporality and can only examine

proximal or between-person effects (mean differences in IPV perpetration across individuals), there is limited research examining the following: (1) the time-varying or within-person effects of alcohol use on IPV perpetration (the effect of an individual's change in alcohol use at one time point on IPV perpetration at the same time point); and (2) the effect of average alcohol use over time on IPV perpetration.

Further, research with men living with HIV has not examined theoretically important moderators of the alcohol-IPV relationship or whether alcohol use reduction interventions decrease IPV perpetration. Depressive symptoms may act as a moderator and has strengthened the association between alcohol use and IPV perpetration in other settings [22-27]. This factor may interact with alcohol use to exacerbate psychopharmacological effects and relationship problems that both lead to IPV perpetration. While alcohol and/or drug use reduction interventions in other settings have decreased IPV perpetration among men who use substances [28-31], none of this research has been done with men living with HIV. It is important to identify efficient prevention strategies that address multiple health issues at once.

The dissertation research was conducted within the context of the NIDA R01, Reducing Hazardous Alcohol Use & HIV Viral Load: A Randomized Controlled Trial (RCT) in ART Clinics in Vietnam (Grant No. 1R01DA037440-01). The parent study is a three-arm RCT among clinic patients with HIV and hazardous alcohol use in northern Vietnam comparing the effects of two alcohol reduction interventions (a two-session Brief Intervention; a six-session Combined Intervention) both against each other and compared with an assessment-only standard of care. Interviewer-administered surveys were collected at baseline, three, six, and 12 months. The dissertation research only used parent study data collected from male participants. The specific aims of the dissertation are:

**Aim 1:** Examine the proximal (between-person) and time-varying (within-person) effects of alcohol use on IPV perpetration over time, controlling for intervention exposure and other covariates.

**Aim 2:** Test whether time-varying (within-person) depressive symptoms moderates the relationship between time-varying (within-person) alcohol use and IPV perpetration over time, controlling for intervention exposure and other covariates.

**Aim 3:** Assess the effectiveness of two alcohol reduction interventions (as compared to standard of care) in reducing individuals' IPV perpetration over time, controlling for covariates.

**Sub-Aim 3A:** Compare the effectiveness of the two alcohol reduction interventions in reducing individuals' IPV perpetration over time compared with the standard of care.

**Sub-Aim 3B:** Examine whether alcohol use mediates the relationship between intervention exposure and individuals' IPV perpetration over time.

By examining the impact of alcohol use on IPV perpetration among men with HIV and hazardous alcohol use, this study will inform IPV, HIV, and alcohol use reduction interventions for vulnerable groups facing overlapping epidemics.

#### **CHAPTER 2: BACKGROUND AND LITERATURE REVIEW**

In this chapter, I present the literature related to my dissertation research and identify the gaps my dissertation research addresses. I start by discussing the literature related to IPV globally (Section 2.1.), including the prevalence of and risk factors for IPV, findings from IPV prevention intervention research, and evidence on the intersection between IPV and HIV. Then, I discuss the public health problem of alcohol globally (Section 2.2.), focusing on alcohol use among high-risk groups such as people living with HIV (PLHIV). Next, I examine the existing evidence base on alcohol use and IPV perpetration (Section 2.3.) by reviewing meta-analyses and potential moderators of the alcohol-IPV relationship. In Section 2.4., I present results from studies that evaluated the effect of alcohol reduction interventions on IPV outcomes. Finally, I examine the history, sociocultural context, and existing research on IPV, alcohol use, and HIV in the study setting, Vietnam (Section 2.5.).

## 2.1. IPV Globally

Globally, 30% of women have experienced IPV in their lifetime [2]. IPV is defined as psychological, physical, or sexual abuse perpetrated towards an intimate partner [1-3]. Psychological abuse includes insulting, yelling, or threatening a partner; physical abuse includes shoving, hitting, or kicking a partner; and sexual abuse includes coerced sex or rape of a partner [1-3]. Experiencing IPV has been shown to lead to a variety of short- and long-term health consequences, including HIV/STI infection, substance abuse, chronic pain, depression, physical injury, and death [2, 4]. In addition to causing detrimental effects for women's health and well-being, IPV also has a far-reaching negative impact on men, families, communities, and societies [32].

Research on IPV perpetration has been limited, as efforts have mainly focused on understanding the extent of the problem regarding the experience of IPV among women [7]. Some population-based studies have found the prevalence of male-perpetrated IPV ranges from 24 to 46% across various global regions [33, 34], with the prevalence ranging from 39 to 87% in Asia and the Pacific [7].

Cross-sectional research has identified multiple prominent risk factors for IPV perpetration among men. Common risk factors include: exposure to child maltreatment, including sexual abuse and intra-parental violence [6, 7, 35, 36], harmful use of alcohol and illicit drugs [6, 7, 28-31, 35, 37-40], depression [7, 35], gender-equitable attitudes [7], attitudes towards IPV [41, 42], and engagement in violence outside the home [6, 7, 35]. More research, especially longitudinal research, is needed to better understand the potential causal relationships between risk factors and IPV perpetration. This research will also help inform IPV prevention efforts targeting men.

There is limited evidence on effective IPV perpetration prevention interventions, especially in global settings. Most IPV prevention interventions have targeted women and aimed to reduce IPV victimization [43]. Some of these approaches, such as school-based programs to prevent violence in dating relationships, home visitation and parent education programs to prevent child maltreatment, and psychological therapies, have been found to be effective [1]. However, researchers, practitioners, and other key stakeholders recognize that men and boys need to be targeted by IPV prevention interventions to achieve large-scale, sustained improvements for IPV prevention [44]. As a result, there has been a more recent shift from implementing IPV prevention interventions that target girls and women to those that engage men and boys [32, 43, 44]. More rigorous intervention research is needed in global settings to identify male-targeted strategies for IPV perpetration prevention, especially among groups at high risk for IPV perpetration.

#### 2.1.1. Intersection of IPV and HIV

Research has clearly demonstrated a link between IPV and HIV [2, 4, 45]. In some regions of the world, such as Africa, women who have experienced IPV are 1.5 times as likely to contract HIV as compared to women who have not experienced IPV [2]. There are three main direct and indirect pathways in which exposure to IPV can increase women's HIV risk. The direct pathway between IPV and HIV is forced or coerced unprotected sex by a sexual partner with HIV [5]. One indirect pathway is limited ability to negotiate safer sex practices due to gender-related power imbalances in relationships; the other indirect pathway is increased HIV risk behaviors due to experience of childhood sexual abuse [5]. Research conducted in Africa has also shown a higher IPV prevalence among women living with HIV as compared to women not living with HIV [46]. Multiple studies have demonstrated that HIV disclosure to a partner can increase the risk for IPV among women living with HIV [47-49]. Additionally, women living with HIV may have become HIV-infected due to IPV and then continued to be in violent relationships post-HIV infection [5].

As in studies with women, research with men demonstrates the link between IPV perpetration and HIV. One study in South Africa found that physical IPV perpetration was associated with HIV sero-prevalence among young men [50]. Two other studies in the United States found a significant association between IPV perpetration and recent STI/HIV diagnosis among men [51, 52]. The link between IPV perpetration and HIV is understudied in Asia, although studies in India and Bangladesh found an association between STI or STI symptoms and IPV perpetration [53, 54]. The relationship between IPV and HIV among men may be explained by the co-occurrence of IPV perpetration with other forms of HIV risk behavior, such as multiple sexual partners, unprotected sex, and transactional sex [51, 52, 55-57]. This pattern of behavior may be shaped by gender inequality and sociocultural norms dictating that men should demonstrate their masculinity by controlling women and being sexually promiscuous [52, 57-60].

As a result of the research linking IPV and HIV, researchers and practitioners have moved to integrate IPV prevention activities into HIV prevention interventions [49]. These women-centered interventions have mostly focused on three main themes: HIV prevention among women at risk for experiencing IPV, prevention for women at risk for both HIV and IPV, and prevention focused on improving social conditions, such as poverty, that make women susceptible to both IPV and HIV [49]. These integrated interventions have utilized varied approaches, including community mobilization [61, 62], microfinance programs for women [63], and educational sessions for women, men, or couples [64, 65]. Only one intervention, an empowerment-based educational program for women, found significant effects on both HIV and IPV outcomes [65]. However, most interventions have led to reductions in IPV- or HIV-related attitudes and behaviors [49]. Future interventions should build on the successes of these interventions, but aim to target groups at high risk for IPV perpetration such as men living with HIV, address multiple factors influencing women's susceptibility to IPV and HIV, and better engage men [49, 66].

Despite the substantial evidence linking IPV and HIV, there is a dearth of research on IPV perpetration among men living with HIV, a group at high risk for IPV perpetration and forward HIV transmission [57, 67-70]. It is critical to understand the prevalence of and risk factors for IPV perpetration and to identify effective IPV prevention intervention approaches for this group.

# 2.2. Alcohol Globally

Alcohol consumption is a major global contributing factor to death, disease, and injury. It impacts over 200 health conditions, including cardiovascular disease, liver disease, tuberculosis, and HIV/AIDS [71]. Alcohol consumption also negatively impacts others through the actions of intoxicated individuals, such as violence [71]. Every year, 2.5 million deaths are attributed to the harmful use of alcohol [71]. Men are at particularly high risk for death due to harmful use of alcohol; 6.2% of male deaths are attributed to alcohol, compared to 1.1% of

female deaths [71]. While alcohol consumption is declining overall globally, it remains on the rise in low- and middle-income countries [71].

Alcohol consumption among vulnerable groups, such as PLHIV, is particularly harmful. For PLHIV, alcohol consumption is associated with premature mortality [72], decreased viral suppression [19], increased liver damage among those co-infected with hepatitis C virus [73], and reduced ART adherence [19, 21, 74]. A meta-analysis showed that PLHIV who drink alcohol were 50-60% as likely to be adherent as compared with those who abstain from alcohol or drink relatively little [20]. Furthermore, alcohol consumption among PLHIV has been associated with depression, which has in turn been associated with accelerated HIV disease progression [75, 76]. Understanding how to intervene and reduce alcohol use among PLHIV should be a priority in the fields of HIV and alcohol use. Alcohol use reduction efforts may not only help improve HIV-related outcomes among PLHIV, but also reduce alcohol-related harms to others, such as IPV.

# 2.3. Alcohol and IPV Perpetration

Alcohol use has been shown to be a risk factor for a multitude of violent and aggressive behaviors, such as homicide and IPV [6-11, 16, 77]. To date, several meta-analytic reviews on alcohol use and male-to-female IPV perpetration have been conducted [12, 13, 78]. The Ferrer et al. [78] meta-analysis included nine studies from 1988 to 1998 and compared batterers to non-batterers; the Stith et al. [13] meta-analysis included 22 studies published from 1980 to 2000; the Foran et al. [12] meta-analysis included 47 studies published from 1980 to 2006; and the Crane et al. [14] meta-analysis included 22 experimental studies published from 1981 to 2014. Authors across these meta-analytic reviews found heterogeneity in effect sizes across studies, concluding that the effect sizes for alcohol use and male-to-female aggression/IPV were in the small to moderate range [12-14, 78].

In an attempt to explain the large heterogeneity in effect sizes found in previous metaanalytic reviews, Foran et al. tested potential moderators of the alcohol-IPV relationship [12]. These moderators included type of sample (clinical vs. community), type of aggression (severe vs. any physical aggression), and type of alcohol measure (overall consumption vs. heavy/binge drinking) [12]. While results showed that type of aggression was not a moderator, the other two variables (type of sample and type of alcohol measure) were found to significantly moderate the alcohol-IPV relationship [12]. Type of sample was tested as a moderator by comparing effect sizes from studies with clinical samples only, community samples only, and both clinical and community samples [12]. Results showed that effect sizes in studies with clinical samples only and community samples only were significantly smaller than effect sizes in studies comparing clinical and non-clinical samples [12]. Although the prevalence of alcohol use and aggression was higher in clinical samples than in community samples, the effect sizes in studies with clinical samples only or community samples only were similar to each other [12]. Authors hypothesized that this similarity in effect sizes was likely due to restriction in range of alcohol and aggression within each study [12]. In contrast, the larger effect sizes observed in studies comparing clinical and non-clinical samples were likely due to a larger range of alcohol and aggression across both types of samples [12].

Additionally, the Foran et al. review found that the type of alcohol measure acted as a moderator of the alcohol-IPV relationship [12]. In alcohol and violence studies, there are a multitude of alcohol measures used, including problem drinking, alcohol abuse/dependence, frequency (i.e., number of days drinking in the past month), binge/heavy drinking (i.e., number of heavy drinking days in the past 30 days), or quantity (i.e., number of drinks consumed in the past month). In the review, measures of problem drinking and alcohol abuse/dependence were more strongly associated with aggression as compared to other alcohol measures [12]. Further, binge or heavy drinking measures were more strongly associated with aggression than measures of frequency [12]. Interestingly, the associations reported with quantity measures were not significantly different than those reported with binge drinking measures [12]. Overall, these findings provide support for two possible pathways in which alcohol may lead to IPV

perpetration. Binge/heavy drinking is hypothesized to immediately lead to IPV perpetration due to the effect of intoxication on disinhibition [12, 14, 15], whereas frequency, quantity, or alcohol abuse/dependence may measure drinking behavior that leads to more marital conflict and ultimately more aggression over time [9, 10, 16]. For example, if an individual is drinking away from home often, this could lead to relationship conflict. The authors concluded that the relationship between alcohol and IPV perpetration is complex and other individual and situational factors need to be tested as potential moderators in future research [12].

Despite the substantial evidence linking alcohol and IPV perpetration, there is limited research, especially longitudinal research, on alcohol and IPV perpetration among PLHIV. None of the studies included in the previous meta-analytic reviews included a sample of PLHIV only [12-14, 78]. As a result, there is limited research examining the time-varying (within-person) effects of alcohol use on IPV perpetration (the effect of an individual's change in alcohol use at one time point on IPV perpetration at the same time point) and the proximal (between-person) effects (associations between average alcohol use over time and IPV perpetration).

The current evidence base highlights that further research is needed to better understand and disentangle the complexity inherent in the alcohol-IPV relationship. PLHIV represent a marginalized and stigmatized group facing a unique set of economic, health, and social challenges [79, 80]; thus, the alcohol-IPV relationship needs to be studied to better understand how to tailor alcohol reduction and IPV prevention efforts for this group.

# 2.3.1. Potential psychosocial moderators of the alcohol-IPV relationship

Meta-analytic reviews of alcohol and IPV perpetration studies have found large heterogeneity in effect sizes, suggesting the need to identify and test potential moderators of the alcohol-IPV relationship [12-14, 78]. Previous meta-analytic reviews have explored whether study design factors, such as type of alcohol measures or type of sample, moderate the alcohol-IPV relationship [12, 14]. Individual-level or situational factors, such as psychosocial measures, need to be tested as moderators of the alcohol-IPV relationship [12, 14].

Depressive symptoms may moderate the relationship between alcohol use and IPV perpetration, as mental health and alcohol use often interact to influence health outcomes [81, 82]. Depressive symptoms are pervasive among men living with HIV and are a common correlate of IPV perpetration [7, 24, 83-85]. Depressive symptoms may interact with alcohol use to either exacerbate the immediate psychopharmacological effects of alcohol or drive relationship conflict over time, both of which lead to IPV perpetration. A nationally representative study in the United States found that the co-occurrence of a severe mental illness and substance abuse predicted violence, while reporting a severe mental illness alone did not [26]. Another United States study found that alcohol use strengthened the relationship between mental illness symptoms and perpetration of aggression [27]. In addition to this quantitative research, qualitative research suggests that alcohol use, depressive symptoms, and IPV intersect in intricate and complex ways [25, 38].

Depressive symptoms remain untested as a moderator of the association between alcohol use and IPV perpetration in research with PLHIV. Identifying potential moderators can provide critical insight into the complexity of the alcohol-IPV relationship and explain some of the heterogeneity found in the association. Further, moderated analyses will provide understanding on how to effectively intervene and reduce IPV perpetration among men living with HIV.

# 2.4. Alcohol Reduction Interventions and IPV

Due to the strong link between alcohol use and IPV, researchers and practitioners, including the World Health Organization (WHO), have been advocating for the use of alcohol interventions to reduce IPV [1, 49, 86-91]. Although there is limited rigorous intervention evaluation research, especially in low- and middle-income countries, there is promising emerging evidence that alcohol interventions can effectively reduce IPV [1, 86, 89, 91]. Alcohol and IPV intervention strategies have included individual-level interventions (i.e., substance use treatment), relationship-level interventions (i.e., couples-based counselling), community-level

interventions (i.e., alcohol sales restrictions), and policy-level interventions (i.e., alcohol taxation) [91]. Thus far, most research has focused on evaluating individual- and couples-based alcohol and IPV interventions [91]. Due to the focus of the dissertation research, I restricted my in-depth review to studies that evaluated clinic-based and/or psychosocial counselling interventions at the individual- or relationship-level.

Numerous studies have examined the effect of individual substance use treatment or "treatment-as-usual" on IPV. Stuart et al. [31] evaluated the effectiveness of an intensive partial hospitalization abstinence-oriented treatment program on alcohol use and IPV perpetration among heterosexual male patients and their partners in the northeastern United States. The cognitive-behavioral treatment comprised group therapy, daily individual psychotherapy sessions, and daily meetings with a psychiatrist over six weeks [31]. Results showed a decrease in alcohol use and male-perpetrated IPV from baseline to six- and 12-month follow-up [31]. O'Farrell et al. [92] also tested the effect of alcohol treatment involving eight individual and 16 group therapy sessions over a 12-week period on IPV perpetration among heterosexual couples in the northeastern United States. Findings demonstrated that male-perpetrated IPV decreased significantly when comparing the prevalence of IPV perpetration the year before treatment (56%) to the year following individual treatment (25%) [92].

Other intervention studies have evaluated individual-based interventions that directly address both substance use and IPV. Kraanen et al. [29] compared the effectiveness of Integrated treatment for Substance abuse and Partner violence (I-StoP) to a substance use disorders treatment program that included one session on IPV among substance abuse treatment facility attendants in Amsterdam, Netherlands. I-StoP involved 16 sessions addressing IPV and substance use; participants also received a workbook containing weekly assignments and diary cards to document substance abuse cravings and anger or IPV perpetration events [29]. While results showed that I-StoP participants perpetrated significantly less physical IPV post-intervention compared to pre-intervention, there were no significant

differences in IPV outcomes between treatment groups [29]. A similar trial in India compared the effectiveness of an integrated cognitive-behavioral intervention (ICBI) that addressed both alcohol use and IPV to the standard of care among alcohol dependent male inpatients [30]. The ICBI intervention comprised eight cognitive-behavioral sessions on the relationship between alcohol use and IPV, triggers for alcohol use and IPV, and consequences and prevention of IPV [30]. While participants in the ICBI group reported significantly lower IPV perpetration as compared to the standard of care group, alcohol consumption did not significantly differ between groups [30]. Stuart et al. [93] evaluated whether a batterer program combined with a brief alcohol intervention reduced IPV perpetration more effectively than a standard batterer program in Rhode Island. The brief alcohol intervention consisted of one 90-minute session with a therapist to discuss alcohol problems, develop a plan to reduce alcohol use, and raise awareness on the link between alcohol use and IPV [93]. Participants in the combined batterer and alcohol program showed improved alcohol and violence outcomes initially, but these improvements were not sustained at 12 months [93].

Behavioral couples therapy (BCT) is another substance use and IPV intervention approach commonly found in the literature. BCT is a type of substance abuse treatment involving the partner that teaches skills that promote partner support for abstinence and emphasizes healthy conflict mediation skills to deal with relationship problems. O'Farrell et al. [94] conducted a study evaluating BCT for married or cohabitating male alcoholic patients in Massachusetts. The BCT program consisted of 20 to 22 weekly couples-only and group sessions over a five- to six-month period focused on promoting sobriety and positive couple and family activities, and providing communication and negotiation skill training [94]. The year after completing BCT, IPV decreased from 60% to 24% among participants [94]. Fals-Stewart et al. [39] compared the effectiveness of BCT to individual treatment among male attendants in an outpatient substance abuse treatment facility in the northeastern United States. The BCT program included weekly individual sessions, drug abuse counseling therapy group sessions,

and couples sessions with a therapist over a 12-week period [39]. After the intervention, IPV perpetration reductions were significantly lower in the BCT group as compared to the individual treatment group [39]. Additionally, Easton et al. [28] compared effectiveness of BCT to a twelve-step facilitated substance abuse program among men in an outpatient substance abuse treatment facility in Connecticut. The BCT intervention comprised twelve weekly couples therapy sessions focused on substance use, IPV, and the relationship between substance use and IPV [28]. There was a greater reduction in the frequency of violent episodes over time in the BCT group as compared to the twelve-step facilitation group [28]. The first study to test a BCT intervention in a low-income setting was conducted recently with couples in India [95]. The three-arm pilot study compared the effectiveness of an intervention comprising four weekly BCT sessions plus monetary incentives for men's negative breathalyzer scores to an intervention for monetary incentives only to a control condition. Significant reductions in alcohol use and IPV were only observed in the BCT plus incentives arm [95].

The current evidence base demonstrates that individual- or relationship-based psychosocial interventions can significantly reduce alcohol use and IPV perpetration, although only two studies have been conducted in a low- or middle-income country [30]. None of the studies tested whether reductions in alcohol use mediated the effect of the intervention on IPV outcomes [91]. Additionally, most interventions tested in these studies were intensive multisession programs that were delivered over 12 weeks or six months. Low-cost interventions, such as brief one- or two-session interventions, should be evaluated as they are more feasible to sustain at scale, especially in low-resource settings [91, 96].

# 2.5. Study Context: Vietnam

Vietnam is a country located in Southeast Asia with an estimated population of 93 million people. Vietnam shares a border to the north with China, to the west with Laos, and to the southwest with Cambodia (Figure 2.1). Since 1976, Vietnam has been a socialist republic with its capital in the northern city of Hanoi. In 1986, to address economic distress due to previous

wars, Vietnam passed economic and political reforms moving the country to a market-based economy. This economic shift, called Đổi Mới ("New Change"), facilitated trading of goods and services with other countries, substantially spurring economic growth and development [80, 97]. As a result of the reforms, the World Bank now considers Vietnam to be a middle-income country [98].

Vietnam's health system is a vertical, three-tiered system that is overseen by the Ministry of Health. Vietnam is in the process of scaling up its social health insurance program and is striving to achieve universal coverage [99, 100]. In the past, HIV/AIDS services have been largely covered by external funding sources, such as U.S. President's Emergency Plan for AIDS Relief (PEPFAR); however, PEPFAR and other funders have scaled down their funding due to Vietnam's emergence as a middle-income country in 2010 [79]. As a result, the country is shifting to finance HIV services through the social health insurance program [100].

Vietnamese culture is influenced greatly by the philosophy and religion of Confucianism, which is characterized by patriarchy and gendered family roles [101-103]. The sociocultural norms derived from Confucianism shape family structure and dynamics in Vietnam [104]. Women are responsible for upholding family harmony and values by taking care of the home, children, and husband [103, 104]. They are also expected to be passive and subservient to their husbands [103, 104]. If a woman's husband is violent, it is seen as a failure on her part to maintain a happy family and she is expected to endure any violence [38, 103]. Conversely, men are responsible for lineage continuity and they are the key decision-makers and income earners of their households [101, 102]. Men are expected to be in control of their wife and family; as a result, being assertive and even aggressive or violent is seen as necessary to maintain a superior position [101, 102].

To some extent, the "Open Door" policies from the Đổi Mới period led to changes in value systems and traditional gender norms among Vietnamese people. Gender equity in the workforce and female workforce participation increased; however, women continue to retain

sole responsibility for household labor [102, 105]. Additionally, women's financial empowerment (wife contributing more to the household than her husband) is a risk factor for IPV [6]. Shifts towards gender equity at the country or institutional level may not be translating to improvements in gender equity in relationships or households. In fact, these institutional changes may have led to increases in IPV, as men may exercise violence to reinforce traditional gender norms that continue to be pervasive [6].

Alcohol is an important part of Vietnamese culture and is shaped distinctly by contemporary sociocultural and gender norms. Alcohol use is strongly encouraged through cultural practices and informal and formal social events, especially for men [18, 106-108]. Men's alcohol use is seen as closely tied to their masculinity and is a regular part of social life and professional networking [108]. It is seen as disrespectful for men to refuse a drink at a social event [18]. Alcohol, especially home-brewed rice wine, is also widely available and relatively cheap due to the ease and low-cost of traditional alcohol production, as well as limited regulation by the government [106]. The WHO found that annual per capita consumption of pure alcohol in Vietnam was 5.1 liters, of which 1.7 liters was unrecorded alcohol [71]. In comparison, global consumption of pure alcohol per capita has been recorded at 4.3 to 4.7 liters since 1990 [109].



*Note:* Vietnam-Guide.com/maps/

Figure 2.1. Map of Vietnam

#### 2.5.1. Intersection of IPV and alcohol use in Vietnam

The prevalence of IPV in Southeast Asia is one of the highest (37.7%) in the world [2]. Prior to 2010, the national prevalence of IPV in Vietnam was unknown [110]. A national study in 2010 showed that over half (58%) of married women reported experiencing IPV by their husbands, and about a third (34%) of these women reported experiencing physical and/or sexual violence [111]. While there are no estimates of national IPV perpetration prevalence among men in Vietnam, one recent cross-sectional study found that 37% of men reported having ever perpetrated IPV against their wife [36].

In Vietnam, several recent studies have identified risk factors for IPV among married couples. Prominent risk factors for experiencing IPV among women have included childhood experience or exposure to violence, history of experiencing sexual violence by other perpetrators, and first sexual experience being forced or coerced [6, 36, 112]. Several characteristics of the husband have also been shown to be risk factors for IPV, including young

age, alcohol use, extramarital relationships, exposure to or experience of family violence, fighting with other men, and favorable attitudes towards IPV [6, 42]. Further, sociocultural factors influencing IPV have been highlighted using qualitative research; this research has shown that the cultural definition of masculinity is used to minimize men's use of IPV [113].

In recent literature on IPV in Vietnam, alcohol use has been identified as a risk factor for IPV perpetration. One nationally representative study found that husband's frequency of alcohol consumption, ranging from daily alcohol use to alcohol use once a month, was significantly associated with experience of IPV among married couples [6]. Other qualitative research shows that many instances of IPV involved men's alcohol use [38, 102]. The strong link between alcohol use and IPV in Vietnam may be explained by sociocultural influences. Traditional gender norms suggest men have uncontrollable characteristics that lead to volatile tempers; alcohol is seen as a drink that can drive this aggressive or violent behavior in men [102]. As a result, men's use of violence under the influence of alcohol is often seen as out of their control and an innate demonstration of masculinity [101, 102, 108].

Over the past several decades, Vietnam has passed federal legislation to address IPV. The Law on Marriage and Family that was passed in 1959 and revised in 1986 gave men and women equal rights in marriage [114]. In 1989, the Penal Code of the Socialist Republic of Vietnam defined penalization for acts of sexual violence [115]. More recently, the 2006 Law on Gender Equality (GE Law) [116] and the 2007 Law on Domestic Violence Prevention and Control (DV Law) [117] were developed by the Vietnam Ministry of Health with support from the WHO. The GE Law was passed to promote gender equality with goals of eliminating gender discrimination and creating equity in the workforce as well as in social and family life [116]. In particular, it describes the responsibilities of individuals, families, organizations, and institutions to promote the health, economic, and social status of girls and women [116]. The DV Law defined domestic violence and outlined prevention strategies, such as education on family values, as well as strategies for assisting the victims of domestic violence [117]. It also specifies

a wide range of social, legal, and medical protection for people who experience violence within their family [117]. While these laws are promising, implementation has been delayed, public awareness around violence is limited, and gender inequality persists [102, 110, 118].

Beyond policy changes, few violence prevention interventions have been implemented in Vietnam. One pilot program in a coastal district in Vietnam, the Responsible Men Club, demonstrated promising results [41]. The Responsible Men Club was a men's empowerment program that aimed to encourage men to challenge inequality and violence [41]. The program was facilitated by the Vietnamese non-profit, Center for Creative Initiatives in Health and Population, and a district-level administrative body, Cualo People's Committee; it was funded by the Ford Foundation [41]. The program consisted of 14 sessions on topics including gender norms, violence, anger management, and fatherhood and also included holiday events for the men to attend with their wives [41].

Given the lack of research on IPV perpetration, studies to understand key drivers of men's IPV perpetration, such as alcohol use, are needed. Additionally, research with groups such as men living with HIV should be a priority due to their high risk for IPV perpetration and forward HIV transmission [57, 67-70]. By understanding the potential mechanisms driving IPV perpetration among men living with HIV, key stakeholders will be able to develop effective IPV prevention interventions.

## 2.5.2. Intersection of HIV and alcohol use in Vietnam

Although the HIV prevalence in the general population is low, Vietnam continues to have concentrated epidemics among key populations [119, 120]. The HIV prevalence among these groups, including men who have sex with men (4%), female sex workers (3%), and people who inject drugs (PWID) (10%), is substantially higher than among the general population (<1%) [119, 120]. The Vietnam Administration for AIDS Control has focused its attention on risk behaviors often associated with these populations, such as unprotected sex with sex workers and sharing needles and syringes [119]. However, alcohol use, a highly normative behavior in

Vietnam that is associated with higher rates of HIV infection and lower ART adherence, has been overlooked [17].

Estimates of hazardous alcohol use in Vietnam vary widely. Hazardous alcohol use refers to a spectrum of alcohol use behavior, including exceeding weekly drinking limits, heavy episodic/binge drinking, and alcohol use disorder (AUD). AUD is a "chronic relapsing brain disease characterized by compulsive alcohol use, loss of control over alcohol intake, and a negative emotional state when not using" [121]. The WHO estimated that 9% of males and 1% of females over the age of 15 years in Vietnam had an AUD [71]. Other estimates of hazardous drinking are higher among men, with one study in rural Vietnam finding the prevalence of alcohol consumption-related problems was 26% among men and 1% among women [122].

Hazardous alcohol use among PLHIV in Vietnam may be even more prevalent. In 2013, a Vietnam study found that 36% of male patients with HIV reported hazardous drinking, 28% reported binge drinking behavior, and male drug users with HIV were 1.11 times as likely to have AUD as compared to non-drug users (p<0.01) [17]. Additionally, research has shown that PWID who reduced their injection frequency were more likely to increase their alcohol consumption [123].

Due to the high prevalence of hazardous alcohol use among men living with HIV, research is needed to further investigate the intersections between alcohol use and HIV in Vietnam. In particular, it is important to understand how alcohol use and HIV interact to influence other health outcomes, such as IPV.

#### CHAPTER 3: THEORETICAL FRAMEWORK AND CONCEPTUAL MODEL

## 3.1. Conceptual Model for Dissertation Research

This dissertation research tests relationships as shown in the conceptual model (Figure 3.1). Aim 1 tests the association between alcohol use and IPV perpetration over time (from baseline to 12-month follow-up); Aim 2 tests whether the association between alcohol use and IPV perpetration will be stronger at times when participants report having versus not having depressive symptoms; and Aim 3 tests whether intervention exposure has an effect on IPV perpetration (Sub-Aim 3A) and whether alcohol use mediates the relationship between intervention exposure and IPV perpetration (Sub-Aim 3B).

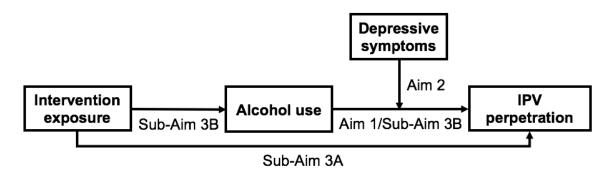


Figure 3.1. Conceptual model for dissertation research

The theoretical framework for the dissertation research draws from five theories. Each theory contributes uniquely to the dissertation research (Figure 3.2). The proximal effects model, indirect effects model, and social ecological model explain the relationship between alcohol use and IPV perpetration. The multiple threshold and disinhibition theories explain how

depressive symptoms may moderate the alcohol-IPV relationship. Next, I will describe each theory and how it informs the dissertation research.

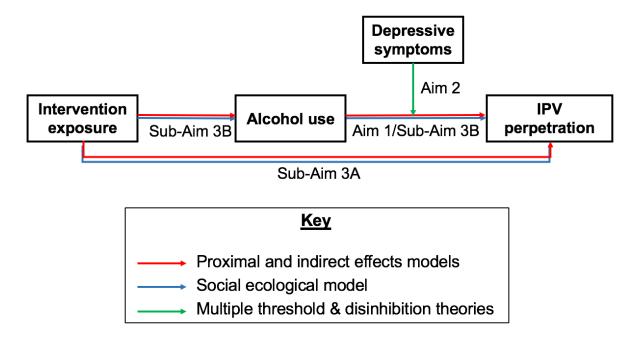


Figure 3.2. Map of theories on conceptual model

#### 3.2. Proximal Effects and Indirect Effects Models

Aims 1 and 3 are informed by the proximal and indirect effects models, two theoretical models commonly used to explain the relationship between alcohol use and IPV perpetration. Both models were developed based on empirical evidence and have been tested in a multitude of research studies on alcohol and aggressive behavior [8, 9, 12, 14-16, 22, 124-126]. However, these models have not been used in research with men with HIV and hazardous alcohol use.

The proximal effects model posits that alcohol consumption leads to psychopharmacological effects, such as lowered inhibitions or distorted perceptions of cues, which can lead to aggression towards a partner [12, 14, 15]. According to this model, alcohol increases an individual's focus on insult or anger and reduces focus on affection or empathy, leading to an inability to use nonaggressive conflict resolution strategies with a partner [14, 124]. In contrast to the proximal effects model, the indirect effects model implies that the causal

relationship between alcohol use and IPV perpetration needs to be assessed over a longer period of time. The indirect effects model posits that elevated alcohol use leads to poor relationship quality, which in turn leads to IPV perpetration over time [9, 10, 16]. Both the proximal and indirect effects models inform the hypotheses that time-varying (within-person) and proximal (between-person) effects of alcohol use on IPV perpetration will be statistically significant.

In addition to highlighting that alcohol use and IPV perpetration are strongly associated, both the proximal and indirect effects models suggest that intervening on alcohol use may also reduce IPV perpetration. Since both interventions evaluated in the parent study reduced alcohol use [127], I hypothesize that participants randomized to either intervention will have decreased IPV perpetration as compared to those in the standard of care arm.

## 3.3. Social Ecological Model

Aims 1 and 3 are also informed by the social ecological model. While the proximal and indirect effects models can help explain the immediate and longer-term effects of alcohol use on IPV perpetration, the social ecological model can help explain the influence of sociocultural factors on alcohol use and IPV perpetration [1, 128, 129]. The social ecological model is commonly used to understand the public health problem of IPV, as it highlights that there are both individual and environmental determinants of IPV [1, 128, 129]. The WHO has demonstrated that patriarchal and male dominance norms influence IPV by promoting the use of violence to control and overpower women [1]. These norms may also drive alcohol use, as alcohol use is often seen as masculine behavior and men face social pressure to drink [18, 108]. Further, men's alcohol use enhances masculine ideologies related to violence, as alcohol is seen as an acceptable driver of masculine behavior, such as aggression [101, 102, 108, 130].

### 3.4. The Multiple Threshold and Disinhibition Theories

The multiple threshold and disinhibition theories were used to select depressive symptoms as a potential moderator of the alcohol-IPV relationship. Both theories posit that each

individual has an aggressive threshold that is determined by their propensity for aggression or the extent to which their context encourages aggressive behaviour [131-133]. For each individual, IPV occurs when they surpass their aggressive threshold, meaning the strength of their aggressive motivations exceeds that of their inhibitions [131-133]. As depression may be an aggression-provoking factor, having depressive symptoms may synergistically interact with alcohol use to lower the threshold at which aggression will occur [131-133]. Depression often manifests as internalized anger and may lead to self-control impairment, making individuals who experience depressive symptoms more susceptible to the disinhibiting effects of intoxication on IPV perpetration [27, 134, 135].

#### **CHAPTER 4: METHODS**

# 4.1. Parent Study: REDART

# 4.1.1. Intervention setting

The study was conducted in Thai Nguyen, a semi-urban province in northern Vietnam located approximately 75 kilometers north of Hanoi with a population of 1 million (Figure 4.1). Thai Nguyen has the highest HIV prevalence among PWID (34%) in Vietnam [119]. There are 12 government-run outpatient ART clinics with a total of about 3150 adult patients on ART.



Figure 4.1. Map of Thai Nguyen province in Vietnam

## 4.1.2. Study design

This dissertation research addressed the Aims through secondary analysis of data from the NIDA R01, Reducing Hazardous Alcohol Use & HIV Viral Load: A Randomized Controlled Trial (RCT) in Antiretroviral Treatment (ART) Clinics in Vietnam [REDART] (PI: Vivian Go PhD: 1R01DA037440-01). The parent study is a three-arm RCT among clinic patients with HIV and

hazardous alcohol use (N=440) in northern Vietnam comparing the effects of two evidence-based, manually guided, individually delivered interventions (a two-session Brief Intervention and a six-session Combined Intervention) to reduce alcohol use and determine the impact on viral load (Figure 4.2). Interviewer-administered surveys and blood samples were collected at baseline, three, six, and 12 months, allowing for longitudinal analysis across four time points. Data collection was conducted from March 2016 to June 2018.

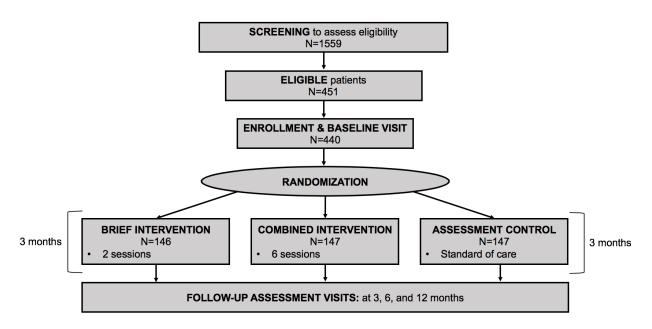


Figure 4.2. REDART study design

### 4.1.3. Description of interventions and study arms

In the REDART study, there were three arms of the RCT: Brief Intervention group, Combined Intervention group, and assessment-only standard of care group (Table 4.1).

Alcohol reduction interventions. In the formative phase of the trial, two alcohol reduction interventions were selected and culturally adapted, as described in detail elsewhere [136]. Briefly, the following intervention development activities were completed: (1) the alcohol content of commonly consumed drinks in Vietnam was assessed; (2) interviews with key stakeholders, including ART patients and providers, were conducted to culturally adapt

intervention manuals and workbooks; and (3) extensive training and supervision using the Yale Adherence and Competence Scale (YACS) [137] was conducted to ensure fidelity to the adapted intervention manuals.

Both the Brief and Combined Interventions were implemented by trained psychosocial counselors and were delivered within a three-month period after the baseline questionnaire was administered. Each team of counselors only delivered one of the interventions. The duration of each session was designed to be approximately 45 minutes. Both interventions were guided by the principles of Motivational Enhancement Therapy (MET) and Cognitive Behavioral Therapy (CBT). MET involves prompting motivation for behavior change and helping to develop a plan for change [138-140]. CBT uses problem-solving and coping skills to promote behavior change [141].

The Brief Intervention consisted of two individual counseling sessions and two "booster" phone sessions. Face-to-face sessions took place approximately one month apart and each phone session occurred approximately two to three weeks after each face-to-face session. The face-to-face sessions were designed to raise awareness of the health consequences of alcohol use and to strengthen the patient's commitment to change. The "booster sessions" reviewed progress and renewed motivation and commitment to change. The session content included a review of drinking patterns, harmful effects of drinking, and alcohol use behavior change strategies.

The Combined Intervention consisted of six mandatory counseling sessions and three optional group sessions. Individual face-to-face sessions occurred approximately one week apart. The mandatory sessions were designed to develop coping skills to assist the patient in managing alcohol cravings and high-risk moods and situations. The session content included a review of drinking patterns and harmful effects of drinking as well as skill-building on alcohol use behavior change. In particular, skills related to alcohol use behavior change focused on drinking refusal skills, skills to cope with managing cravings and triggers, and developing positive

thoughts and attitudes. The group sessions emphasized the skills learned in the one-on-one sessions and facilitated interactions between patients so that they could support one another in managing their drinking and share experiences around alcohol. Participants could attend the group sessions at any point after being randomized into the study.

**Standard of care.** Participants randomized to the standard of care group received referrals to alcohol treatment and infectious disease treatment.

Table 4.1. Summary of study arms

|                                    | Assessment-  | Combined     | Brief        |
|------------------------------------|--------------|--------------|--------------|
|                                    | Only Control | Intervention | Intervention |
| Standard of care services          | X            |              |              |
| Harm reduction services            | X            | X            | X            |
| ART at any CD4 count               | X            | X            | X            |
| Referrals for substance use        | X            | X            | X            |
| Referrals for diagnosis and        | X            | Χ            | X            |
| treatment of Hepatitis B Virus,    |              |              |              |
| Hepatitis C Virus, sexually        |              |              |              |
| transmitted infections, and        |              |              |              |
| tuberculosis                       |              |              |              |
| Face-to-face counseling sessions   |              | 6 sessions   | 2 sessions   |
| Phone sessions                     |              | _            | 2 sessions   |
| Group sessions (optional, up to 3) |              | X            |              |

## 4.1.4. Participant recruitment and data collection

Study participants were recruited from all seven ART community clinics in Thai Nguyen. All recruitment was exhausted in one clinic before moving on to recruit participants from the next clinic; the order of clinics to be recruited from was random (Table 4.2). After introducing the project, study interviewers administered baseline written informed consent to those who were interested and were 18 years of age or older. After obtaining consent, the interviewer conducted a screening survey with the patient including the WHO Alcohol Use Disorders Identification Test (AUDIT-C) survey items to determine eligibility [142]. Male patients with an AUDIT-C score  $\geq$  4 and female patients with an AUDIT-C score  $\geq$  3 were considered to have hazardous alcohol use. Inclusion criteria for eligibility included: (1) current patient on ART at the clinic; (2) AUDIT-C

score  $\geq$  4 (for men) or  $\geq$  3 (for women); (3) 18 years of age or older; and (4) plan on residing in Thai Nguyen for the next 24 months. Exclusion criteria for eligibility included: (1) unable to participate in study activities due to psychological disturbance, cognitive impairment or threatening behavior (assessed by study staff); (2) unwilling to provide locator information; (3) unwilling to provide informed consent; and (4) currently participating in other HIV, drug use, or alcohol program, study, or intervention.

Table 4.2. Number of participants enrolled by clinic

| Clinic<br>Number                | 1  | 2  | 3   | 4  | 5  | 6  | 7 | Total |
|---------------------------------|----|----|-----|----|----|----|---|-------|
| Number of participants enrolled | 76 | 60 | 114 | 89 | 46 | 55 | 1 | 441   |

Participants who were deemed eligible for enrollment were asked to consent to enroll in the study. Specifically, interviewers described the RCT study objectives, procedures, risks and benefits to the eligible participants, and answered any questions. If there were literacy reasons why a signature was not appropriate, individuals were allowed to mark the baseline or enrollment and follow-up consent forms with an "X." If the participant provided written informed consent, he/she completed the baseline questionnaire and Timeline Followback (TLFB) to measure daily alcohol use over the past 30 days. Once enrollment eligibility was confirmed, individuals were randomly assigned to one of three study arms in a 1:1:1 ratio. One hundred and forty-seven participants were randomly assigned to receive the Combined Intervention (N=145 males), 147 participants randomly assigned to receive the Brief Intervention (N=140 male participants), and 146 participants randomly assigned to the Control arm (N=141 males). Descriptive statistics for the study sample at baseline are presented below (Table 4.3).

Quantitative assessments were administered at baseline, three, six, and 12 months for all study participants; the duration of each visit was approximately two hours. Questionnaires

were administered through face-to-face interviews in a private room at an ART clinic by trained interviewers. At each follow-up study visit, participants confirmed informed consent for enrollment, and completed the follow-up questionnaire and TLFB. The behavioral assessment for the parent study collected quantitative data on the following topics: sociodemographics, HIV medical and treatment history, HIV clinic and treatment costs, alcohol use, drug use, sexual behavior, health utility, readiness to change, coping skills, stigma, opportunistic infections, patient trust in providers, mental health, involvement in community violence, exposure to violence as a child, and IPV. Participants also provided blood samples at every study visit for CD4 T-cell count, HIV viral load, hepatitis B surface antigen rapid test, and anti-HCV rapid test.

To maximize retention at every study visit, the following was done: (1) a computerized database was used to track scheduled and missed study appointments; (2) locator information was updated at every study visit; (3) within 24 hours of a missed appointment, participants were contacted via phone or home visit; and (4) if a participant was detained, hospitalized, or incarcerated, the follow-up visit was scheduled after release.

For participants randomized to the Brief or Combined Intervention study arms, attendance at intervention sessions was documented in two ways. First, the study staff checked in participants when they arrived at the clinic to receive an intervention session. Second, the psychosocial counselors completed a form to document every intervention session (including phone call sessions) they had with intervention participants.

**Table 4.3.** Descriptive statistics for study sample at baseline by gender

|  | Males<br>(N=426) | Females<br>(N=14) | Total<br>(N=440) |
|--|------------------|-------------------|------------------|
|  | N                | (%) or Mean (SE   | D)               |
| Age in years                             | 40.24 (5.68)     | 39.79 (8.55)      | 40.22 (5.78)     |
| Highest education level completed        |                  |                   |                  |
| Technical training/College or university | 41 (9.62)        | 3 (21.43)         | 44 (10.00)       |
| High school                              | 147 (34.51)      | 5 (35.71)         | 152 (34.55)      |
| Secondary school                         | 146 (34.27)      | 5 (35.71)         | 151 (34.32)      |
| Primary school                           | 54 (12.68)       | 1 (7.14)          | 55 (12.50)       |
| None                                     | 38 (8.92)        | 0 (0.00)          | 38 (8.64)        |
| Employment status                        |                  |                   |                  |
| Employed full- or part-time              | 344 (80.75)      | 13 (92.86)        | 357 (81.14)      |
| Unemployed/Retired                       | 82 (19.25)       | 1 (7.14)          | 83 (18.86)       |
| Marital status                           |                  |                   |                  |
| Married or living with a partner         | 313 (73.47)      | 5 (35.71)         | 318 (72.27)      |
| Single                                   | 66 (15.49)       | 0 (0.00)          | 66 (15.00)       |
| Widowed/Divorced/Separated               | 47 (11.03)       | 9 (64.29)         | 56 (12.73)       |

Note: SD=Standard Deviation

#### 4.1.5. Ethical considerations

Participants were assigned a unique identification (ID) number upon enrollment in the study. The research team maintained a separate list that linked the unique ID number with the personally identifying information in locked filing cabinets at the Center for Preventive Medicine in Thai Nguyen, Vietnam. De-identified electronic data files were password protected and maintained on a secure server at UNC-Chapel Hill. I conducted my statistical analyses in SAS and MPlus using a dataset with no individually identifying information. Since my secondary data analysis of the parent study's data was conducted with de-identified data, there were negligible potential risks to questionnaire participants.

#### 4.2. Dissertation Research

To examine the longitudinal and moderated effects of alcohol use on male-perpetrated IPV in Aims 1 and 2, the dissertation research used parent study data collected from males who

reported being married/living with a partner at baseline (N=313). The analysis used longitudinal multilevel modeling to: (a) account for the nested structure of the data (time nested within individuals); and (b) facilitate investigation on whether average alcohol use predicts *who* is at risk for IPV perpetration (proximal or between-person effects) and/or whether time-specific elevations in alcohol use predicts *when* risk of IPV perpetration is elevated (time-varying or within-person effects).

To evaluate the alcohol reduction intervention effects on IPV perpetration and test whether alcohol use is a mediator in Aim 3, the dissertation research used parent study data collected from males (N=426). The intervention effects analysis used path analyses to estimate longitudinal pathways between intervention assignment and recent IPV perpetration at each study visit. The mediation analysis then used lagged panel mediation models to test whether alcohol use mediated any significant relationships between intervention exposure and IPV perpetration.

## 4.2.1. Aims and hypotheses

The specific aims and hypotheses for my dissertation research are listed below. The study aims to:

**Aim 1** Examine the proximal (between-person) and time-varying (within-person) effects of alcohol use on IPV perpetration over time, controlling for intervention exposure and other covariates.

**H1:** For time-varying effects, men will report increased levels of IPV perpetration, above their usual baseline, at time points when they report increased levels of alcohol use.

**H2:** For proximal effects, men who report higher as compared with lower mean levels of alcohol use will report increased levels of IPV perpetration.

**Aim 2** Test whether time-varying (within-person) depressive symptoms moderates the relationship between time-varying (within-person) alcohol use and IPV perpetration over time, controlling for intervention exposure and other covariates.

**H3:** Depressive symptoms will moderate the time-varying effects of alcohol use on IPV perpetration, such that the association will be stronger at times when an individual screens positive for depressive symptoms as compared to negative.

**Aim 3** Assess the effectiveness of two alcohol reduction interventions (as compared to standard of care) in reducing individuals' IPV perpetration over time, controlling for covariates.

**Sub-Aim 3A** Compare the effectiveness of the two alcohol reduction interventions in reducing individuals' IPV perpetration over time as compared with the standard of care.

**H4:** Participants randomized to receive the Brief and Combined Interventions will report reduced IPV perpetration across the three- to 12-month follow-up period as compared to participants randomized to receive the standard of care.

**Sub-Aim 3B** Examine whether alcohol use mediates the relationship between intervention exposure and individuals' IPV perpetration over time.

**H5:** Alcohol use will mediate the relationship between intervention exposure and IPV perpetration such that exposure to the Brief and Combined interventions vs. standard of care will be associated with decreased alcohol use, and, in turn, alcohol use will be positively associated with IPV perpetration.

## 4.2.2. Key measures

Measures related to IPV perpetration, alcohol use, and depressive symptoms were collected at baseline, three, six, and 12 months (Table 4.4). Psychological, physical, and sexual IPV perpetration were measured using the widely used and validated Conflict Tactics Scale 2 (CTS2) [143-145]. Psychological IPV perpetration was measured using two items: (1) I insulted or swore or shouted or yelled at my partner; and (2) I destroyed something belonging to my partner or threatened to hit my partner. Physical IPV perpetration was measured using two

items: (1) I pushed, shoved, or slapped my partner; and (2) I punched or kicked or beat up my partner. Sexual IPV perpetration was measured using two items: (1) I insisted on sex when my partner did not want to or insisted on sex without a condom (but did not use physical force); and (2) I used force (like hitting, holding down, or using a weapon) to make my partner have sex with me. Recall periods varied based on the study visit. At baseline, participants were asked about any events of IPV in the past year. Response options included: "More than once in the past year", "Once in the past year", "Not in the past year, but it did happen before", or "This has never happened." At the following study visits, the recall period was the past three months.

Alcohol use was assessed using three variables: proportion of days alcohol abstinent in past 30 days, number of heavy drinking days in past 30 days, and alcohol use disorder (AUD) (Table 3). Proportion of days alcohol abstinent and heavy drinking were measured using the TLFB, a measure that has been shown to be valid and reliable across multiple settings and populations [146, 147]. The TLFB is an interviewer-administered assessment that reconstructs a daily behavior calendar to help prompt memory recall for alcohol consumption. A heavy drinking day was defined as having more than four drinks per day for men [148]. AUD was measured using the Mini International Neuropsychiatric Interview questionnaire (MINI) Diagnostic and Statistical Manual of Mental Health Disorders, 4th edition (DSM IV) tool [149]. The MINI comprises items that measure alcohol dependence and items that measure alcohol abuse by assessing level of drinking and mental or physical harm due to drinking [142]. The standard scoring method was used to create the binary AUD variable [149]. Composite scores were first calculated for each participant. Participants who scored three or more on the alcohol dependence items and/or one or more on the alcohol abuse items were categorized as having an AUD; those who did not, were categorized as not having an AUD.

Depressive symptoms were measured using the nine-item Patient Health Questionnaire-9 (PHQ-9) scale [150]. A composite PHQ-9 score was calculated for each participant; those who reported a score of zero to four on the PHQ-9 were categorized as screening negative for depressive symptoms and those who reported a score above four were categorized as screening positive for depressive symptoms [151]. Covariates were selected based on substantial theoretical or empirical evidence of potential confounding [6, 11, 35, 60, 123, 152, 153].

**Table 4.4.** Overview of measures for dissertation research

| Variable<br>type | Variable name   | Scale/Item                                | Wave(s) of data collection |
|------------------|---|---|----------------------------|
| Outcome          | Recent IPV perpetration   | CTS2 [143]                                | Baseline and all follow-up |
| Predictors       | Intervention exposure <sup>a</sup>                                | Results of<br>randomization<br>procedures | Baseline                   |
|                  | Proportion of days alcohol abstinent in past 30 days <sup>b</sup> | TLFB [146]                                | Baseline and all follow-up |
|                  | Number of heavy drinking days in past 30 days <sup>b</sup>        | TLFB [146]                                | Baseline and all follow-up |
|                  | Alcohol use disorder (AUD)  | MINI [149]                                | Baseline and all follow-up |
| Moderator        | Depressive symptoms   | PHQ-9 [150]                               | Baseline and all follow-up |
| Covariates       | Age   | Not applicable                            | Baseline                   |
|                  | Level of education  | Not applicable                            | Baseline                   |
|                  | Marital status  | Not applicable                            | Baseline and all follow-up |
|                  | Employment status   | Not applicable                            | Baseline and all follow-up |
|                  | Injection drug use  | Not applicable                            | Baseline and all follow-up |
|                  | Exposure to violence as a child                                   | Not applicable<br>[42]                    | Baseline and all follow-up |
|                  | Involvement in community violence                                 | Aggression<br>scale (two<br>items) [154]  | Baseline and all follow-up |

<sup>&</sup>lt;sup>a</sup>Intervention exposure acts as a predictor variable in Aim 3 and as a covariate in Aims 1 and 2. <sup>b</sup>All three alcohol measures act as predictor variables in Aims 1 and 2 and as mediating variables in Aim 3.

## 4.2.3. Sample size and power

At baseline, 426 males enrolled and 376 males completed 12-month follow-up. Thus, there is a minimum of 1,504 observations over the course of the study, which is sufficient for the dissertation analyses. With a conservative sample size estimate of 376 participants at four data

collection time points, I have 80% power to detect an effect size of 0.17, 0.18, and 0.20 (assuming an intraclass correlation (ICC) value of 0.10, 0.20, and 0.30, respectively), according to Raudenbush's Optimal Design Software [155]. These effect sizes are similar to those found in alcohol use and IPV perpetration meta-analytic reviews [12, 13] and intervention trials [28-31].

#### 4.2.4. Analysis plan

Statistical analysis was conducted in SAS 9.4 and MPlus8. I began by examining the pattern of missing data. Then, I conducted preliminary data analysis, including univariate descriptions of each key variable to assess whether they were normally distributed with homogeneous variances. I also explored whether alcohol use and intervention exposure were associated with IPV perpetration by analyzing the correlations between variables within each time point.

Aims 1 and 2. For Aims 1 and 2, the longitudinal effects of alcohol use on IPV perpetration were estimated using generalized linear mixed models. Due to the binary outcome variable, I used an approximation of maximum likelihood to deal with any missing data [156]. As there was not substantial missingness observed for covariates, I chose not to apply a multilevel multiple imputation strategy [157]. The models were specified at two levels in which repeated measure of the outcome (level one) was nested within individuals (level two). This approach allowed for the separation of the total variance in IPV perpetration into within-person (individual's variation in IPV perpetration over time) and between-person variation (mean differences in IPV perpetration).

First, I determined the best-fitting unconditional growth model for IPV perpetration. I tested linear and quadratic functional forms and removed any random effects that could not be estimated and any fixed effects that were non-significant. I then selected a residual error structure by conducting a likelihood ratio test (LRT) to compare the fit of the unconditional growth model with an unstructured covariance structure (allows variance and covariance of the residuals in IPV perpetration to vary over time) to one with a compound symmetry covariance

structure (assumes equal variance and covariance of the residuals in IPV perpetration over time) [158].

Then, I centered the alcohol use variables to allow for estimation of both time-varying and proximal effects. The alcohol use variable used to estimate proximal effects was grand-mean centered after the person-mean was calculated (average alcohol use across all time points). The alcohol use variable used to estimate time-varying effects was person-mean centered (individual's mean alcohol use subtracted from the individual's alcohol use at a specific time-point). I then added the grand-mean and person-mean centered alcohol use variables and covariates to the model (Model 2) to estimate the time-varying and proximal effects of alcohol use on IPV perpetration when adjusting for covariates.

To conduct the moderation analyses described in Aim 2, I built upon the models used in the Aim 1 analyses. I only included the person-mean centered alcohol use variable as my moderation hypotheses focused on the time-varying effects of alcohol use on IPV perpetration. To assess depressive symptoms as a time-varying moderator, I used the dummy coded depressive symptoms variable with person means included as controls. I added hypothesized two-way interactions between time-varying alcohol use and time-varying depressive symptoms to the model. Any non-significant interaction terms were removed for the final model. Significant interactions were probed by graphing the relationship between alcohol use and IPV perpetration at the two levels of depressive symptoms. After graphing these relationships, I examined the simple intercepts and slopes to assess the degree of effect measure modification. I used Dr. Kristopher Preacher's computational tools to guide these steps for testing moderation [159].

**Aim 3.** For Sub-Aim 3A, I used path analyses on MPlus8 to assess intervention effects at three, six, and 12 months. I estimated longitudinal pathways between intervention assignment and recent IPV perpetration at each time point, controlling for covariates.

The results of Sub-Aim 3A directly informed the analysis for Sub-Aim 3B. If significant intervention effects were observed, I tested the mediation hypothesis via a lagged panel

mediation model (Figure 4.3) [160-162]. First, I tested longitudinal pathways between predictor (X), mediating (M), and outcome (Y) variables at each time point (paths  $a_{x1}$ ,  $a_{x2}$ ,  $b_{T0}$ ,  $b_{T1}$ ,  $b_{T2}$ , and b<sub>T3</sub>). To address the potential for prior levels of the alcohol use and IPV perpetration variables to confound associations, I included autoregressive (AR) pathways (paths AR<sub>mT0</sub>, AR<sub>mT1</sub>, AR<sub>vT0</sub>,  $AR_{vT1}$ , and  $AR_{vT2}$ ). Direct pathways were estimated from  $X_0$  to all measures of Y at the following time points (c'<sub>Ti</sub>, c'<sub>T2</sub>, c'<sub>T3</sub>) and covariances (depicted by the curved arrows) were allowed between all variables assessed at the same time point. To assess mediation, estimates from the path analyses were used to calculate causally-defined mediation effects, including the natural indirect effect and the natural direct effect. The natural indirect effect, or mediated effect, estimates the reduction in IPV perpetration comparing the Brief and Combined Intervention groups to a hypothetical condition of intervention participation where the interventions' effects on alcohol use is blocked [163, 164]. The natural direct effect estimates the reduction in IPV perpetration comparing the hypothetical condition to the standard of care group [163, 164]. Finally, standard errors (SEs) and bootstrapped confidence intervals (CIs) for indirect effects were estimated based on 5,000 bootstrap resamples. Bootstrapping is a nonparametric method for estimating SEs and CIs; it does not make assumptions about the sampling distribution of the indirect effect, providing more accurate Type I error rates and greater power for detecting indirect effects than other methods [165, 166]. I concluded that mediation was present if the indirect effect of intervention exposure on IPV perpetration through alcohol use was found to be statistically significant [167].

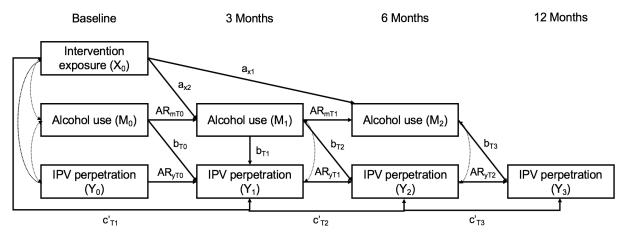


Figure 4.3. Lagged panel mediation model

# CHAPTER 5: LONGITUDINAL ANALYSIS OF ALCOHOL USE AND INTIMATE PARTNER VIOLENCE PERPETRATION AMONG MEN LIVING WITH HIV IN NORTHERN VIETNAM

#### 5.1. Introduction

Male-perpetrated intimate partner violence (IPV) is a pervasive and urgent public health problem. Globally, 30% of women have ever experienced IPV [2], defined as psychological, physical, or sexual abuse perpetrated towards an intimate partner [1-3]. Experiencing IPV leads to a range of health consequences, including HIV/STI infection, substance abuse, chronic pain, depression, physical injury, and death [2, 4]. In addition to causing detrimental effects for women's health and well-being, IPV also has a far-reaching negative impact on men, families, communities, and societies [32].

There is substantial evidence that alcohol use is a risk factor for IPV perpetration by men [6-13], including three meta-analyses showing small to moderate effects of alcohol use on IPV perpetration [12, 13, 78]. The relationship between alcohol use and IPV perpetration is often explained using two theoretical models. First, alcohol use may lead to IPV perpetration through immediate psychopharmacological effects of alcohol, such as lowered inhibitions or distorted perceptions of cues [12, 14, 15]. Second, alcohol use may lead to increased relationship conflict, in turn leading to IPV perpetration [9, 10, 16].

Although the relationship between alcohol use and IPV perpetration has been well-established, most of this research has been conducted in Western countries [12, 14, 78]. Few rigorous studies have examined the alcohol-IPV relationship in Vietnam. Alcohol use and IPV perpetration are largely shaped by sociocultural norms [1, 101, 102], suggesting that the association between alcohol use and IPV perpetration may differ across cultural contexts. This is supported by the results from a multi-country study in Asia and the Pacific which found that

alcohol use was only a risk factor for IPV perpetration in some countries and not others [7]. To develop effective alcohol and IPV reduction interventions, it is critical to understand whether the associations between alcohol use and IPV perpetration among men in Vietnam are similar or different to other settings.

Further, there is a dearth of research on alcohol use and IPV perpetration among men living with HIV globally. Research with men living with HIV is important as they are a group at particularly high risk for IPV perpetration and forward HIV transmission. Research has demonstrated that men living with HIV have an elevated prevalence of hazardous alcohol use [168]; one Vietnam study found that 36% of male patients with HIV reported hazardous drinking [17]. Hazardous alcohol use refers to a spectrum of alcohol use behavior, including exceeding weekly drinking limits, heavy episodic/binge drinking, and alcohol use disorder (AUD). Hazardous drinking is a known correlate of IPV perpetration [6, 7, 35] and other HIV transmission risk factors, such as poor antiretroviral treatment (ART) adherence [19, 21, 74, 169] and inconsistent condom use [170]. Research with sub-groups such as men living with HIV should be a priority to improve men's health and wellbeing [57, 70] and to prevent forward HIV transmission [57, 67-70].

In this study, I aim to estimate two different longitudinal effects of alcohol use on IPV perpetration among men with HIV and hazardous alcohol use in northern Vietnam. First, I estimate whether men report increased IPV perpetration, above their usual baseline, at those times when men also report increased alcohol use. Second, I estimate whether men who report higher average alcohol use across the study period in turn report increased IPV perpetration over time. Due to a dearth of longitudinal research on alcohol use and IPV perpetration, estimating both longitudinal effects in this study provides insight on the complex nature of the alcohol-IPV relationship in an understudied population and setting. As cross-sectional studies cannot examine time-varying effects or associations between alcohol use and IPV perpetration over time, study findings will address an important research gap.

Across these longitudinal analyses, I selected three measures of alcohol use that capture different dimensions of hazardous drinking based on the literature [12]: proportion of days alcohol abstinent, number of heavy drinking days, and alcohol use disorder (AUD).

Multiple measures of alcohol use were used in this study as a meta-analytic review found that the magnitude of the effect size for the association between alcohol use and IPV perpetration varied by alcohol measure [12]. Analyses will help test whether the nature of the alcohol-IPV relationship differs by drinking pattern. My specific hypotheses include:

- Men will report increased IPV perpetration, above their usual baseline, at time points when they report increased alcohol use.
- Men who report higher as compared with lower mean levels of alcohol use will report increased IPV perpetration over time.

Results will inform alcohol reduction and IPV and HIV prevention efforts for a group at high risk for forward HIV transmission.

#### 5.2. Methods

### 5.2.1. Study design

This study is a secondary analysis of data from a three-arm randomized controlled trial (RCT) among clinic patients with HIV and hazardous alcohol use (N=440) in Thai Nguyen province [127]. Thai Nguyen is a semi-urban province in northern Vietnam located approximately 75 kilometers north of Hanoi with a population of 1 million. The RCT compared the effects of two evidence-based, manually guided, individually delivered interventions to reduce alcohol use and determine the impact on viral load. There were three arms of the RCT: (1) a Brief Intervention (two in-person sessions and two phone call sessions); (2) a Combined Intervention (six in-person sessions and three optional group sessions); and (3) standard of care. Data collection was conducted from March 2016 to June 2018.

## 5.2.2. Participant recruitment and data collection

Study participants were recruited from all seven ART community clinics in Thai Nguyen. Once all ART patients were approached for recruitment in one clinic, recruitment efforts moved to the next clinic. The order of clinics that ART patients were recruited from was random. After introducing the project, study interviewers administered baseline written informed consent to those who were interested and were 18 years of age or older. After obtaining consent, the interviewer conducted a screening survey with the patient including the WHO Alcohol Use Disorders Identification Test (AUDIT-C) survey items to determine eligibility [142]. Male patients with an AUDIT-C score ≥ 4 were considered to have hazardous alcohol use. Inclusion criteria for eligibility included: (1) current patient on ART at the clinic; (2) AUDIT-C score ≥ 4; (3) 18 years of age or older; and (4) plan on residing in Thai Nguyen for the next 24 months. Exclusion criteria for eligibility included: (1) unable to participate in study activities due to psychological disturbance, cognitive impairment or threatening behavior (assessed by study staff); (2) unwilling to provide locator information; (3) unwilling to provide informed consent; and (4) currently participating in other HIV, drug use, or alcohol program, study, or intervention.

Participants who were deemed eligible for enrollment were asked to consent to enroll in the study. Specifically, interviewers described the RCT study objectives, procedures, risks and benefits to the eligible participants, and answered any questions. If there were literacy reasons why a signature was not appropriate, individuals could mark the baseline or enrollment and follow-up consent forms with an "X." If the participant provided written informed consent, he/she was assigned a unique identification number and completed the baseline questionnaire and Timeline Followback (TLFB) to measure daily alcohol use over the past 30 days [171]. Once enrollment eligibility was confirmed, individuals were randomly assigned to one of three study arms in a 1:1:1 ratio.

Quantitative assessments were administered at baseline, three, six, and 12 months for all study participants; the intervention was delivered to intervention participants between the

study visits at baseline and three months. Participants also provided blood samples at every study visit. The duration of each visit was approximately two hours. Questionnaires were administered through face-to-face interviews in a private room at an ART clinic by trained interviewers. At each follow-up study visit, participants confirmed informed consent for enrollment, and completed the follow-up questionnaire and TLFB. The behavioral assessment collected quantitative data on sociodemographics, alcohol and drug use, involvement in community violence, exposure to violence as a child, and IPV. Among those who did not become incarcerated (n=11/440; 2.5%) or die (n=15/440; 3.4%) during the course of the study, retention was 94% (405/430) at three months, 96% (410/427) at six months, and 94% (390/414) at 12 months. Eighty-five percent of participants (372/440) completed all four assessments, 9% (39/440) completed three assessments, 3% (13/440) completed two assessments, and 4% (16/440) completed one assessment.

The study protocol received ethical approval from the Institutional Review Boards at the University of North Carolina-Chapel Hill and Thai Nguyen Center for Preventive Medicine.

#### 5.2.3. Key measures

IPV perpetration. Recent psychological, physical, and sexual IPV perpetration among any current or previous partner was measured with the widely used and validated six-item shortened Conflict Tactics Scale 2 (CTS2) [143-145]. Psychological, physical, and sexual IPV perpetration were each measured using two items. Response options varied based on the study visit. At baseline, participants were asked about any events of IPV in the past year. At the following study visits, the recall period was the past three months. Due to the differences in recall periods across study visits, the outcome will be referred to as "recent IPV perpetration." For each item measuring psychological, physical, or sexual IPV perpetration, responses were dichotomized to those who reported recent IPV perpetration at least once and those who did not. IPV variables were dichotomized as the numbers of recent IPV events reported over the course of the study were skewed. Those who refused to answer or didn't know the answer were

marked as missing. Participants who reported recently perpetrating psychological, physical, and/or sexual IPV were categorized as reporting any form of recent IPV perpetration and those who reported not recently perpetrating psychological, physical, and/or sexual IPV were categorized as not reporting any form of recent IPV perpetration [172].

Alcohol use. Alcohol use was measured using the TLFB, a tool that has been shown to be valid and reliable across multiple settings and populations [146, 147]. The TLFB is an interviewer-administered assessment that reconstructs a daily behavior calendar to help prompt memory recall for alcohol consumption. Using the TLFB, alcohol use was assessed as proportion of days alcohol abstinent in past 30 days (0 to 1) and number of heavy drinking days in past 30 days. A heavy drinking day was defined as having more than four drinks per day for men [148].

While proportion of days alcohol abstinent and heavy drinking capture drinking behaviors that indicate hazardous alcohol use, I also wanted to assess patterns of severity of hazardous alcohol use. Therefore, AUD was measured using the Mini International Neuropsychiatric Interview questionnaire (MINI) Diagnostic and Statistical Manual of Mental Health Disorders, 4th edition (DSM IV) tool [149]. The MINI consists of a set of items that measures alcohol dependence and a set of items that measures alcohol abuse, which are generally defined by level of drinking and mental or physical harm due to drinking [142]. Composite scores for each participant were calculated. Based on the standard scoring method used for the MINI, participants who scored three or more on the alcohol dependence items and/or one or more on the alcohol abuse items were categorized as having an AUD; those who did not were categorized as not having an AUD [149].

**Covariates.** Covariates for the relationship between alcohol use and IPV perpetration were selected based on substantial theoretical or empirical evidence of potential confounding [6, 11, 35, 60, 123, 152, 153]. Exposure to violence as a child comprised three items asking about ever witnessing interparental violence as a child, experiencing physical abuse as a child, or

experiencing sexual abuse as a child. Responses were categorized as having been exposed to violence as a child if a participant answered yes to any of the three questions or no if a participant answered no to all three questions. Involvement in community violence ever was measured using two items from the Modified Aggression scale [154] that asked if they had ever been physically violent towards someone in their community or had ever experienced physical violence by someone in their community. As both experiencing and perpetrating community violence are known risk factors for IPV perpetration [6, 35, 173, 174], I created a composite variable for involvement in community violence ever.

The remaining covariates, including education (primary school or less/secondary school or less/high school or less/technical training or college or university or less), age, employment status (employed full- or part-time/unemployed or retired), and injection drug use in the past three months, were collected via self-report. As analyses have shown both alcohol reduction interventions significantly reduced alcohol use at each follow-up study visit as compared to the standard of care group [127], intervention arm was also included as a covariate to control for intervention effects on alcohol use and IPV perpetration. Intervention arm was documented during the computer-generated randomization process.

### 5.2.4. Data analysis

I restricted the analysis to male participants who reported being married or living with a partner at baseline (N=313) for two reasons. First, correlates for IPV perpetration differed for men who were married or living with a partner versus those who were single, divorced, separated or widowed at baseline [85]. These findings suggest that IPV operates differently for those in long-term relationships, which make up the large majority of the sample (77%) [85]. Second, a commonly used theoretical model in alcohol and IPV research posits that poor relationship quality may explain the alcohol-IPV relationship [9, 10, 16]. Those who are married or living with a partner have more opportunity for changes in relationship quality during the study window as compared to those who are not.

The longitudinal effects of alcohol use on IPV perpetration were estimated with multilevel growth models using maximum likelihood based on the Laplace estimation [156]. The models were specified at two levels in which the repeated measure of the outcome (level one) was nested within individuals (level two). This approach allowed for the separation of the total variance in IPV perpetration into variation in an individual's IPV perpetration over time and variation across individuals in IPV perpetration. As a result, I was able to examine both the time-varying (within-person) and proximal (between-person) effects of alcohol use on IPV perpetration.

Linear and quadratic functional forms were tested to determine the best-fitting unconditional growth model for IPV perpetration. If the variance component for the random effect could not be estimated, it was removed from the model; if the fixed effect was then found to be non-significant, it was also removed from the model. Additionally, a residual error structure was selected by conducting a likelihood ratio test (LRT) to compare the fit of the unconditional growth model with an unstructured covariance structure (allows variance and covariance of the residuals in IPV perpetration to vary over time) to one with a compound symmetry covariance structure (assumes equal variance and covariance of the residuals in IPV perpetration over time) [158].

Appropriate centering strategies were used with all variables included in the analysis [175]. The variable for wave of data collection was re-coded to start at zero so that the intercept represents the log odds of IPV perpetration at baseline. Alcohol use variables estimating time-varying effects were person-mean centered and alcohol use variables estimating proximal effects were grand-mean centered after calculating the person means. Other categorical covariates were dummy coded and age (years) was grand-mean centered.

Analysis was conducted using SAS 9.4. The same analytic approach was used for each of the three alcohol use measures. After determining the best-fitting unconditional growth model, the centered alcohol use variables and covariates were added to the model. The adjusted

conditional growth model tested the time-varying and proximal effects of alcohol use on IPV perpetration adjusting for covariates. As participants were recruited from different ART community clinics, recruitment site was initially tested as a fixed effect in analyses. However, as it did not change the results, it was removed to make models more parsimonious.

#### 5.3. Results

### 5.3.1. Descriptive statistics

The sample comprised 313 male participants who were married or living with a partner (Table 5.1). The mean age of participants was 40.8 years (SD=5.6) at baseline. Most participants had a high school education or less (N=189; 60.4%). At baseline, blood test results showed that 14.4% (N=45) of participants did not achieve viral suppression (less than 20 copies/ml).

Median percent days alcohol abstinent in the past 30 days increased over the study from 36.7% (SD=33.1) at baseline to 76.7% (SD=36.9) at 12-month follow-up. Median number of heavy drinking days in the past 30 days decreased over the study from 2.0 days (SD=10.1) at baseline to 1.0 days (SD=8.7) at 12-month follow-up. The proportion of participants with symptoms of AUD decreased from 39.6% (N=124) at baseline to 19.4% (N=54) at 12-month follow-up.

At baseline, 94 participants (30.0%) reported any form of IPV perpetration in the last 12 months; psychological IPV was the most prevalent (N=82; 26.2%; Table 5.2). At three-month follow-up, the prevalence of any form of recent IPV perpetration decreased to 15.4% (N=44), which was expected due to the shorter reference period. The prevalence estimates remained around this level throughout the rest of the study (six-month follow-up: N=47, 16.1%; 12-month follow-up: N=42, 15.0%). Participants who reported recent IPV perpetration at baseline (N=94) had higher IPV perpetration prevalence estimates across all subsequent study visits as compared to participants who reported no recent IPV perpetration at baseline (N=219; Figure 5.1).

Table 5.1. Characteristics of male participants who were married or living with a partner (N=313)<sup>a</sup>

|   | Baseline (N=313) | 3 months (N=287) | 6 months (N=292) | 12 months (N=279) |
|---|------------------|------------------|------------------|-------------------|
|   | N (%)            |                  |                  |                   |
| Sociodemographics   |                  |                  |                  |                   |
| Mean age in years (SD)                                    | 40.8 (5.6)       |                  |                  |                   |
| Highest level of education                                |                  |                  |                  |                   |
| Technical training/College or university or less          | 52 (16.6)        |                  |                  |                   |
| High school or less                                       | 189 (60.4)       |                  |                  |                   |
| Secondary school or less                                  | 48 (15.3)        |                  |                  |                   |
| Primary school or less                                    | 24 (7.7)         |                  |                  |                   |
| Employment status   |                  |                  |                  |                   |
| Employed full- or part-time                               | 262 (83.7)       | 243 (84.7)       | 255 (87.3)       | 245 (87.8)        |
| Unemployed/Retired  | 51 (16.3)        | 44 (15.3)        | 37 (12.7)        | 34 (12.2)         |
| Viral suppression <sup>b,c</sup>                          |                  |                  |                  |                   |
| No  | 45 (14.4)        | 39 (13.6)        | 35 (12.0)        | 42 (15.1)         |
| Yes   | 268 (85.6)       | 247 (86.4)       | 257 (88.0)       | 237 (85.0)        |
| Alcohol Use   |                  |                  |                  |                   |
| Median percent days abstinent in past 30 days (SD)        | 36.7 (33.1)      | 70.0 (35.6)      | 76.7 (37.3)      | 76.7 (36.9)       |
| Median number of heavy drinking days in past 30 days (SD) | 2.0 (10.1)       | 1.0 (8.7)        | 0.0 (8.6)        | 1.0 (8.7)         |
| Alcohol use disorder (AUD)                                |                  |                  |                  |                   |
| No  | 189 (60.4)       | 210 (73.2)       | 223 (76.4)       | 225 (80.7)        |
| Yes   | 124 (39.6)       | 77 (26.8)        | 69 (23.6)        | 54 (19.4)         |
| Psychosocial Variables                                    |                  |                  |                  |                   |
| Injection drug use in past 3 months <sup>c</sup>          |                  |                  |                  |                   |
| No  | 244 (78.0)       | 226 (79.0)       | 228 (78.1)       | 222 (79.6)        |
| Yes   | 69 (22.0)        | 60 (21.0)        | 64 (21.9)        | 57 (20.4)         |

| Exposed to violence as a child <sup>d</sup>      |            |            |            |            |
|--|------------|------------|------------|------------|
| No   | 199 (63.6) |            |            |            |
| Yes  | 114 (36.4) |            |            |            |
| Ever involved in community violence <sup>c</sup> |            |            |            |            |
| No   | 178 (56.9) | 176 (61.5) | 173 (59.3) | 140 (50.2) |
| Yes  | 135 (43.1) | 110 (38.5) | 119 (40.8) | 139 (49.8) |

<sup>&</sup>lt;sup>a</sup>Percentages may not sum to 100 due to rounding.

<sup>&</sup>lt;sup>b</sup>Achieving viral suppression was defined as having less than 20 copies/ml.

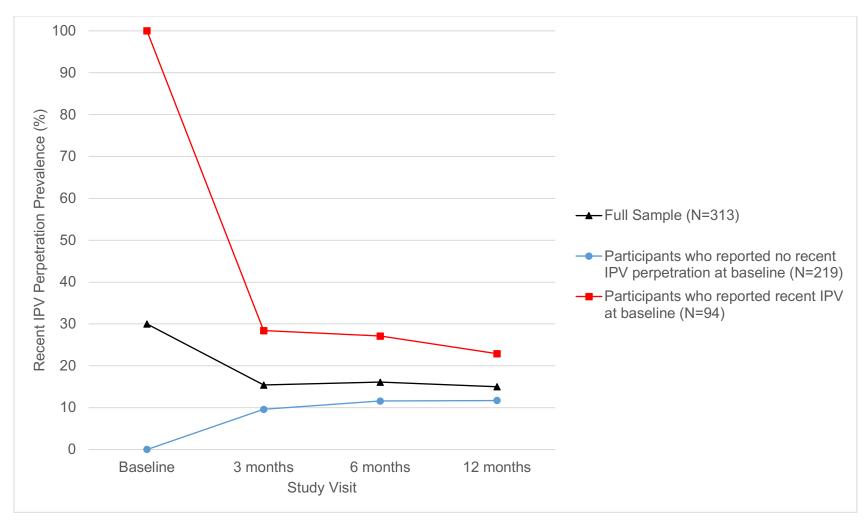
<sup>°</sup>Missing data due to refused to answer or don't know: Viral suppression at 3 months: N=1; Injection drug use at 3 months: N=1; Ever involved in community violence at 3 months: N=1.

<sup>&</sup>lt;sup>d</sup>Exposed to violence as a child includes having ever witnessed interparental violence as a child or experienced physical or sexual abuse as a child.

Table 5.2. Prevalence of intimate partner violence perpetration across study visits<sup>a</sup>

|  | Baseline (N=313) | 3 months (N=287) | 6 months (N=292) | 12 months (N=279) |
|--|------------------|------------------|------------------|-------------------|
|  | N (%)            |                  |                  |                   |
| Recent Psychological IPV Perpetration <sup>b</sup> |                  |                  |                  |                   |
| No   | 231 (73.8)       | 250 (87.4)       | 252 (86.3)       | 242 (86.7)        |
| Yes  | 82 (26.2)        | 36 (12.6)        | 40 (13.7)        | 37 (13.3)         |
| Recent Physical IPV Perpetration                   |                  |                  |                  |                   |
| No   | 298 (95.2)       | 282 (98.3)       | 285 (97.6)       | 275 (98.6)        |
| Yes  | 15 (4.8)         | 5 (1.7)          | 7 (2.4)          | 4 (1.4)           |
| Recent Sexual IPV Perpetration                     |                  |                  |                  |                   |
| No   | 293 (93.6)       | 276 (96.2)       | 285 (97.6)       | 270 (96.8)        |
| Yes  | 20 (6.4)         | 11 (3.8)         | 7 (2.4)          | 9 (3.2)           |
| Recent Physical/Sexual IPV Perpetration            |                  |                  |                  |                   |
| No   | 280 (89.5)       | 272 (94.8)       | 279 (95.6)       | 266 (95.3)        |
| Yes  | 33 (10.5)        | 15 (5.2)         | 13 (4.5)         | 13 (4.7)          |
| Any Form of Recent IPV Perpetration <sup>b</sup>   |                  |                  |                  |                   |
| No   | 219 (70.0)       | 242 (84.6)       | 245 (83.9)       | 237 (85.0)        |
| Yes  | 94 (30.0)        | 44 (15.4)        | 47 (16.1)        | 42 (15.0)         |

<sup>&</sup>lt;sup>a</sup>Percentages may not sum to 100 due to rounding.
<sup>b</sup>Missing data due to refused to answer or don't know: Recent psychological IPV perpetration at 3 months: N=1; Any form of recent IPV perpetration at 3 months: N=1.



**Figure 5.1.** Prevalence of recent intimate partner violence (IPV) perpetration among the full sample (N=313), among those who reported no recent IPV perpetration at baseline (N=219), and among those who reported recent IPV perpetration at baseline (N=94)

## 5.3.2. Trajectories of IPV perpetration: unconditional growth model

The best fitting unconditional model for IPV perpetration included a random intercept with random and linear fixed effects for time (Table 5.3). The LRT demonstrated that allowing for heteroscedasticity over time did not improve model fit. As a result, a compound symmetry model was used, which assumes equal variance and covariance in IPV perpetration over time. The linear parameter for time showed that the odds of IPV perpetration significantly decreased over time (OR=0.45, 95% CI 0.34, 0.59). There was also significant variance in IPV perpetration between participants at baseline (Intercept: b=-1.09, SE=0.14, p<0.0001).

**Table 5.3.** Unconditional growth model (N=313)

|                   | Coefficient (SE) or OR (95% CI), p-value |        |  |  |
|-------------------|--|--------|--|--|
| Fixed Effects     |  |        |  |  |
| Intercept         | -1.09 (0.14)                             | <.0001 |  |  |
| Time              | 0.45 (0.34, 0.59)                        | <.0001 |  |  |
| Random Effects    |  |        |  |  |
| Variance Componer | nts                                      |        |  |  |
| Covariance        | 0.53 (0.                                 | 54)    |  |  |
| Variance          | 0.12 (0.24)                              |        |  |  |
| Model Fit         |  |        |  |  |
| AIC               | 1090.0                                   | )5     |  |  |
| BIC               | 1105.04                                  |        |  |  |
| Neg 2 LL          | 1082.05                                  |        |  |  |

Note: SE=Standard error; OR=Odds ratio; AIC=Akaike information criterion; BIC=Bayesian information criterion; Neg 2 LL=Negative 2 log likelihood

# 5.3.3. Adjusted analyses

**Proportion of days alcohol abstinent.** As expected, the time-varying (within-person) and the proximal (between-person) effects of proportion of days alcohol abstinent on IPV perpetration were negative and statistically significant (Table 5.4). The time-varying effect demonstrates that at time points when participants reported higher proportion of days alcohol abstinent than average, they were 0.39 times as likely to report IPV perpetration (95% CI 0.17,

0.88). Additionally, the proximal effect shows that participants who reported higher as compared to lower average proportion of days alcohol abstinent across the study period were 0.43 times as likely to report IPV perpetration over time (95% CI 0.20, 0.91). As higher proportion of days alcohol abstinent indicate less alcohol use, these inverse relationships between proportion of days alcohol abstinent and IPV perpetration were expected.

**Heavy drinking.** Contrary to expectations, the time-varying effect of heavy drinking on IPV perpetration was non-significant (aOR=0.99, 95% CI 0.96, 1.02; Table 5.4). However, as expected, the proximal effect showed that reporting higher levels of heavy drinking over the study period was associated with higher odds of IPV perpetration over time (aOR=1.05, 95% CI 1.02, 1.08).

Alcohol use disorder (AUD). As hypothesized, the time-varying and proximal effects of AUD were associated with higher odds of IPV perpetration. Participants who screened positive for AUD across the study as compared to negative were 4.74 times as likely to report IPV perpetration (95% CI 2.31, 9.71). Controlling for the effect of average tendency to report AUD, participants were 2.95 times as likely to report IPV perpetration at times when they screened positive for AUD (95% CI 1.59, 5.46).

**Table 5.4.** Adjusted conditional growth models<sup>a</sup> (N=313)

|                     | Proportion of day abstiner                | •       | Heavy drin        | king    | Alcohol use disor | rder (AUD) <sup>b</sup> |  |  |
|---------------------|---|---------|-------------------|---------|-------------------|-------------------------|--|--|
|                     | Coefficient (SE) or aOR (95% CI), p-value |         |                   |         |                   |                         |  |  |
| Fixed Effects       |   |         |                   |         |                   |                         |  |  |
| Intercept           | -0.73 (0.41)                              | 0.08    | -0.55 (0.41)      | 0.09    | -0.94 (0.42)      | 0.03                    |  |  |
| Time                | 0.53 (0.41, 0.70)                         | <0.0001 | 0.50 (0.38, 0.65) | <0.0001 | 0.53 (0.40, 0.70) | <0.0001                 |  |  |
| Alcohol use         |   |         |                   |         |                   |                         |  |  |
| Time-varying        | 0.39 (0.17, 0.88)                         | 0.02    | 0.99 (0.96, 1.02) | 0.61    | 2.95 (1.59, 5.46) | 0.001                   |  |  |
| Proximal            | 0.43 (0.20, 0.91)                         | 0.03    | 1.05 (1.02, 1.08) | 0.002   | 4.74 (2.31, 9.71) | <0.0001                 |  |  |
| Random Effects      |   |         |                   |         |                   |                         |  |  |
| Variance Components |   |         |                   |         |                   |                         |  |  |
| Covariance          | -0.007 (0.2                               | 25)     | -0.10 (0.2        | 26)     | -0.10 (0.1        | 19)                     |  |  |
| Variance            | 0.56 (0.55)                               |         | 0.71 (0.58)       |         | 0.74 (0.47)       |                         |  |  |
| Model Fit           |   |         |                   |         |                   |                         |  |  |
| AIC                 | 1052.89                                   |         | 1052.37           |         | 1025.82           |                         |  |  |
| BIC                 | 1112.82                                   |         | 1112.31           |         | 1085.76           |                         |  |  |
| Neg 2 LL            | 1020.89                                   | 9       | 1020.37           | 1020.37 |                   | 993.82                  |  |  |

<sup>&</sup>lt;sup>a</sup>Controlling for education, grand-mean centered age, exposure to violence as a child, employment status, injection drug use, involvement in community violence ever, and intervention arm.

<sup>&</sup>lt;sup>b</sup>A random slope for person-mean centered AUD was added to this model as model fit improved when it was included. *Note:* SE=Standard error; aOR=Adjusted odds ratio; AIC=Akaike information criterion; BIC=Bayesian information criterion; Neg 2 LL=Negative 2 log likelihood

#### 5.4. Discussion

To my knowledge, this is the first study to examine the longitudinal relationship between alcohol use and IPV perpetration among men living with HIV. Study findings demonstrate that hazardous alcohol use is a risk factor for IPV perpetration in this population. IPV and HIV prevention interventions targeting this group should incorporate alcohol reduction services into HIV care and treatment. Clinic-based psychosocial counseling interventions targeting men with hazardous alcohol use in the United States have effectively reduced IPV perpetration [31, 92], offering a promising IPV prevention strategy to test in low-resource settings.

Differing types of longitudinal effects were found across alcohol measures, suggesting that the nature of the alcohol-IPV relationship varies by drinking pattern, as shown in other alcohol and violence research [12]. For proportion of days alcohol abstinent and AUD, increases in alcohol use at one time point increased the likelihood of IPV perpetration at the same time point. These observed effects may be explained by the proximal effects model, which is used to explain the acute effects of alcohol use on IPV perpetration [12, 14, 15, 176, 177]. According to this model, the immediate intoxication effects of alcohol increases an individual's focus on insult or anger and reduces focus on affection or empathy, leading to an inability to use nonaggressive conflict resolution strategies with a partner [14, 124]. Additionally, lower average proportion of days alcohol abstinent, and higher average heavy drinking and AUD across the study increased the likelihood of IPV perpetration. These observed effects may be explained by the indirect effects model, which posits that elevated alcohol use leads to poor relationship quality, which in turn leads to IPV perpetration over time [9, 10, 16]. Future research should measure anger and relationship quality to test if either or both of these variables explain the longitudinal effects of alcohol use on IPV perpetration. Understanding the mechanisms for which alcohol influences IPV perpetration in this population will help inform development of IPV prevention interventions.

The relationship between alcohol use and IPV perpetration may also be explained by unmeasured confounding. The spurious effects model posits that individuals with deviant characteristics, such as antisocial personality traits and hostility, may be prone to engaging in hazardous drinking and IPV perpetration, explaining the alcohol-IPV relationship [178]. However, studies have found that the association between alcohol use and IPV perpetration remains after controlling for these potential confounders [179, 180]. For men living with HIV, the spurious effects model may need to be adapted to consider unique characteristics in this population that may drive hazardous drinking and IPV perpetration, such as HIV-specific coping strategies, anger, hostility, or psychological distress [181, 182].

My hypotheses on the relationship between heavy drinking and IPV perpetration were not fully supported by study results. Increases in heavy drinking at one time point were not associated with increases in IPV perpetration at the same time point. Additionally, the magnitude of the effect for the significant association between higher average heavy drinking and higher odds of IPV perpetration was small. When compared to the results for the other measures of alcohol use, study findings suggest that heavy drinking may not be the strongest risk factor for IPV perpetration in this population. There are two potential explanations for these results. First, as alcohol use is considered a conduit for men's violent behavior, women may avoid or placate their husbands when they are intoxicated after drinking heavily, reducing the opportunity for IPV [38, 102]. Qualitative research in other settings has suggested women utilize such strategies to avoid experiences of alcohol-related IPV [130]. Secondly, men may not remember or accurately remember perpetrating IPV after drinking heavily, as they may recall the positive social consequences of heavy drinking over the negative [130, 183]. This is particularly possible among a socially isolated group such as men living with HIV, as heavy drinking often occurs at social events with other men, which act as key access points for social support or bonding [18, 108]. While reducing heavy drinking is often a primary aim for alcohol

and IPV reduction interventions [39, 93], findings suggest that reducing AUD and frequent drinking is also important for interventions targeting men living with HIV in Vietnam.

As all variables were measured using self-report and the questionnaire was interviewer-administered [150], responses may have been subject to social desirability bias. If present, the social desirability bias would have likely led to under-reporting of IPV perpetration [184, 185]. However, interviewers underwent extensive training on questionnaire administration, minimizing the likelihood of social desirability bias. Additionally, recall bias may have influenced alcohol use estimates as all three alcohol use variables were measured via self-report. However, sub-analyses of the study data conducted by Go et al. [127] validated the self-report alcohol measures using phosphatidylethanol (PEth), a direct metabolite of alcohol use that acts as a biomarker [186].

Alcohol use and recent IPV perpetration have different response frames in the questionnaire. The response frame for alcohol use measures is the last 30 days and the response frames for recent IPV perpetration varies from the last 12 months to the last three months, depending on the data collection time point (baseline: IPV perpetration in last 12 months; three-, six-, and 12-month follow-up: IPV perpetration in last three months). This mismatch in response frames is a limitation when examining the time-varying effects of alcohol use on recent IPV perpetration. However, the alcohol use variables are intended to measure alcohol use since the last data collection time point. When using the TLFB, research has shown that a recall period of 30 days can be used to estimate annual consumption [147]. Additionally, the three-month recall period for the IPV outcome at endline does not span the full lag between the study visits at six and 12 months. This suggests the magnitude of the proximal effects of alcohol use on IPV perpetration may be underestimated.

Despite these limitations, this longitudinal study contributes important findings on alcohol use and IPV perpetration in a high-risk, understudied population. Results suggest that the relationship between alcohol use and IPV perpetration may vary across drinking patterns,

underscoring the importance of measuring alcohol use in multiple ways and estimating multiple types of longitudinal effects. For all measures of alcohol use, reporting greater as compared to lower levels of alcohol use on average was significantly associated with higher odds of IPV perpetration over time. However, time-specific increases in alcohol use were only significantly associated with higher odds of IPV perpetration for measures of proportion of days alcohol abstinent and AUD, not for heavy drinking. To prevent IPV perpetration and forward HIV transmission among men living with HIV, psychosocial counseling interventions should address alcohol use with a focus on reducing frequent drinking and AUD.

# CHAPTER 6: ALCOHOL USE, DEPRESSIVE SYMPTOMS, AND INTIMATE PARTNER VIOLENCE PERPETRATION: A LONGITUDINAL ANALYSIS AMONG MEN LIVING WITH HIV IN NORTHERN VIETNAM

#### 6.1. Introduction

The link between alcohol use and male-perpetrated intimate partner violence (IPV) has been established [6-13]; however, meta-analytic reviews on alcohol and IPV perpetration have observed large heterogeneity in effect sizes across studies [12-14, 78]. Research is needed to test whether psychosocial factors, such as depressive symptoms, influence the strength of the alcohol-IPV relationship [12-14, 78]. These types of investigations will explain some of the heterogeneity found in the alcohol-IPV association, providing critical understanding on how to effectively intervene and reduce IPV perpetration among men.

Alcohol use may be associated with IPV perpetration for some individuals, but not for others. The multiple threshold and disinhibition theories posit that each individual has an aggressive threshold determined by their propensity for aggression or by the extent to which their context encourages aggressive behavior [131-133]. These theories state that for each individual, IPV occurs when they surpass their aggressive threshold, meaning the strength of their aggressive motivations exceeds that of their inhibitions [131-133]. Depressive symptoms may interact with alcohol use to drive individuals past their aggressive thresholds. In particular, researchers have posited that depression may lead to self-control impairment, making individuals who experience depressive symptoms more susceptible to the disinhibiting effects of alcohol intoxication on IPV perpetration [27, 134, 135].

Empirical evidence also suggests that the co-occurrence of alcohol use and mental health exacerbates violence outcomes. A nationally representative study in the United States

found that reporting a severe mental illness did not predict violence, but reporting both a severe mental illness and substance abuse did [26]. Another United States study found that alcohol use strengthened the relationship between mental illness symptoms and perpetration of aggression [27]. In addition to these quantitative studies, qualitative research has suggested that depressive symptoms intersect with alcohol use to drive IPV perpetration [25].

The influence of depressive symptoms on the alcohol-IPV relationship is unexplored among men living with HIV, a group at high risk for hazardous alcohol use, IPV perpetration, and forward HIV transmission [57, 67-70]. In Vietnam, depressive symptoms are highly prevalent and a known correlate of IPV perpetration among men living with HIV [85]. However, there is a dearth of research in Vietnam and other global settings examining how depressive symptoms and alcohol use interact to influence IPV perpetration.

In this study, I aim to test whether depressive symptoms influence the strength and/or direction of the alcohol-IPV relationship among men with HIV and hazardous alcohol use in Vietnam. I investigate whether the concurrent effect of alcohol use on IPV perpetration differs at times when an individual reports having depressive symptoms versus not having depressive symptoms. As a meta-analytic review found that the effect size for the association between alcohol use and IPV perpetration varied by alcohol measure [12], three measures of alcohol use were used in analyses: *proportion of days alcohol abstinent, number of heavy drinking days,* and *alcohol use disorder (AUD)*. My specific hypothesis is as follows:

 The time-varying association between alcohol use and IPV perpetration will be stronger at times when an individual screens positive for depressive symptoms as compared to negative.

This study will improve understanding on how to effectively intervene and reduce IPV perpetration among men living with HIV.

#### 6.2. Methods

# 6.2.1. Study design

This study is a secondary analysis of data from a three-arm randomized controlled trial (RCT) comparing the effects of two evidence-based, manually guided, individually delivered interventions to reduce alcohol use and determine the impact on viral load [127]. From March 2016 to June 2018, data collection was conducted with clinic patients with hazardous alcohol use and HIV (N=440) in northern Vietnam.

## 6.2.2. Participant recruitment and data collection

Recruitment of study participants took place at all seven antiretroviral treatment (ART) community clinics in Thai Nguyen. Recruitment was completed at one clinic before moving on to recruit potential participants at the next clinic. Study interviewers introduced the project before administering written informed consent to those who were interested in participating in the baseline questionnaire. Those who provided consent completed an interviewer-administered screening survey. The survey included the World Health Organization (WHO) Alcohol Use Disorders Identification Test (AUDIT-C) survey items to determine eligibility for enrollment. Male patients with an AUDIT-C score  $\geq 4$  were considered to have hazardous alcohol use [142]. Inclusion criteria for eligibility included: (1) current patient on ART at the clinic; (2) AUDIT-C score  $\geq 4$ ; (3) 18 years of age or older; and (4) plan on residing in Thai Nguyen for the next 24 months. Exclusion criteria for eligibility included: (1) unable to participate in study activities due to psychological disturbance, cognitive impairment or threatening behavior (assessed by study staff); (2) unwilling to provide locator information; (3) unwilling to provide informed consent; and (4) currently participating in other HIV, drug use, or alcohol program, study, or intervention.

Eligible participants were asked to consent a second time to enroll in the study after interviewers explained the RCT study objectives, procedures, risks and benefits to the participants, and answered any questions. Individuals were allowed to mark any study consent

forms with an "X" if a signature was not appropriate for literacy reasons. If the eligible participant provided written informed consent, he/she was assigned a unique identification number. He/she then completed the baseline questionnaire and Timeline Followback (TLFB) to measure daily alcohol use over the past 30 days [171]. Eligible participants were also randomly assigned to one of the three study arms in a 1:1:1 ratio.

At baseline, three, six, and 12 months, quantitative questionnaires and the TLFB were administered to all study participants. Each study visit lasted approximately two hours. Trained interviewers administered questionnaires through face-to-face interviews in a private room at an ART clinic. Participants confirmed informed consent for enrollment at each follow-up visit. The behavioral assessment collected quantitative data on sociodemographics, alcohol and drug use, mental health, involvement in community violence, exposure to violence as a child, and IPV. Among those who did not become incarcerated (n=11/440; 2.5%) or die (n=15/440; 3.4%) during the study, retention was 94% (405/430) at three months, 96% (410/427) at six months, and 94% (390/414) at 12 months. Eighty-five percent of participants (372/440) completed all four assessments, 9% (39/440) completed three assessments, 3% (13/440) completed two assessments, and 4% (16/440) completed one assessment.

The study protocol received ethical approval from the Institutional Review Boards at the University of North Carolina-Chapel Hill and Thai Nguyen Center for Preventive Medicine.

# 6.2.3. Key measures

IPV perpetration. The widely used and validated six-item shortened Conflict Tactics Scale 2 (CTS2) was used to measure recent psychological, physical, and sexual IPV perpetration among any current or previous partner [143-145]. Recall periods varied based on the study visit (baseline: IPV perpetration in last 12 months; three-, six-, and 12-month follow-up: IPV perpetration in last three months). The outcome will be referred to as "recent IPV perpetration" due to the varied recall periods across study visits. Due to skewed numbers of recent IPV events reported, responses to all six items were categorized as those who reported

recent IPV perpetration at least once and those who did not. Those who refused to answer or didn't know the answer were marked as missing. A binary outcome variable was created for any form of recent IPV perpetration, defined as reporting recent psychological, physical, and/or sexual IPV perpetration [172].

Alcohol use. The TLFB was used to measure alcohol use; the tool has been shown to be valid and reliable in multiple settings [146, 147]. The TLFB is an interviewer-administered assessment that reconstructs a daily behavior calendar to help prompt memory recall for alcohol consumption. The following two alcohol use variables were created using the TLFB: proportion of days alcohol abstinent in past 30 days (0 to 1) and number of heavy drinking days in past 30 days. A heavy drinking day was defined as having more than four drinks per day, which is a commonly used guideline for men [148].

In addition to measuring proportion of days alcohol abstinent and heavy drinking, it was important to capture severity of hazardous alcohol use using the Mini International Neuropsychiatric Interview questionnaire (MINI) Diagnostic and Statistical Manual of Mental Health Disorders, 4th edition (DSM IV) tool [149]. The MINI comprises items that measure alcohol dependence and items that measure alcohol abuse by assessing level of drinking and mental or physical harm due to drinking [142]. The standard scoring method was used to create the binary AUD variable [149]. Composite scores were first calculated for each participant. Participants who scored three or more on the alcohol dependence items and/or one or more on the alcohol abuse items were categorized as having an AUD; those who did not, were categorized as not having an AUD.

**Depressive symptoms.** Depressive symptoms were measured using the nine-item Patient Health Questionnaire-9 (PHQ-9) scale (Cronbach's alpha at baseline=0.83) [150]. A composite PHQ-9 score was calculated for each participant. Based on the standard scoring method used for the PHQ-9, those who scored zero to four were categorized as screening

negative for depressive symptoms and those who scored above four were categorized as screening positive for depressive symptoms [151].

Covariates. Theoretical and empirical evidence were used to select covariates for the relationship between alcohol use and IPV perpetration [6, 11, 35, 60, 123, 152, 153]. Exposure to violence as a child was measured using three items asking about ever witnessing interparental violence as a child, ever experiencing physical abuse as a child, or ever experiencing sexual abuse as a child. Responses were dichotomized; those who responded yes to any of the three questions were categorized as having been exposed to violence as a child and those who answered no to all three questions were categorized as not having been exposed to violence as a child. Involvement in community violence comprised two items asking about having ever been physically violent towards someone in their community or having ever experienced physical violence by someone in their community [154]. As both experiencing and perpetrating community violence increase the likelihood of IPV perpetration [6, 35, 173, 174], a composite variable was created for involvement in community violence.

Education (primary school or less/secondary school or less/high school or less/technical training or college or university or less), age, employment status (employed full- or part-time/unemployed or retired), and injection drug use in the past three months, were collected via self-report. Intervention arm was also selected as a covariate and was documented using a computer-generated randomization process.

## 6.2.4. Data analysis

In previous analyses, the longitudinal effects of alcohol use on IPV perpetration were estimated using multilevel growth models (CHAPTER 5: PAPER 1). Specifically, the timevarying or within-person effects (effect of an individual's change in alcohol use at one time point on IPV perpetration at the same time point) and proximal or between-person effects (effects of average alcohol use over the study on IPV perpetration) were examined. Results showed that the best fitting unconditional growth model included a random intercept and linear fixed effects

for time. Further, a compound symmetry model was used, meaning it was assumed that there was equal variance and covariance in IPV perpetration over time [158]. Results demonstrated that the odds of IPV perpetration decreased over time. At times when participants screened positive for AUD or reported lower proportion of days alcohol abstinent than usual, there were higher odds of IPV perpetration. In this study, I aim to build off of the previous analyses and test depressive symptoms as a potential moderator of the relationship between time-varying (or within-person) alcohol use and IPV perpetration.

For this study, analysis was restricted to male participants who reported being married or living with a partner at baseline (N=313). Previous research with men living with HIV in northern Vietnam has shown that the alcohol-IPV relationship operates differently for men who were married or living with a partner versus those who were single, divorced, separated or widowed [85].

Appropriate centering strategies were used with all variables included in the analysis [175]. The variable for wave of data collection was re-coded to start at zero to ensure the intercept represents the log odds of IPV perpetration at baseline. The alcohol use variables used to estimate time-varying (or within-person) effects were person-mean centered. For depressive symptoms, the person means were calculated and included in the model as controls along with the dummy coded depressive symptoms variable. Other categorical covariates were dummy coded and age (years) was grand-mean centered.

Moderation analyses were conducted in SAS 9.4 with multilevel growth models using maximum likelihood based on the Laplace estimation [156]. The same analytic approach was used for each of the three alcohol use measures. The adjusted conditional growth model tested the time-varying effects of alcohol use and depressive symptoms and the two-way interactions among depressive symptoms and alcohol use on IPV perpetration adjusting for covariates. Any non-significant interactions were removed from the final model. If an interaction term was significant, the effect of alcohol use on the predicted probability of IPV perpetration was graphed

at both levels of the depressive symptoms variable (0 or 1) to visualize the relationships. Standard errors (SEs) and p-values were generated to test the statistical significance of the intercepts and slopes represented in the graphs. Since participants were recruited from different ART community clinics, recruitment site was initially tested as a fixed effect to assess if it influenced results. As it did not change the results, it was removed to make models more parsimonious.

# 6.3. Results

# 6.3.1. Descriptive statistics

The sample comprised 313 male participants who were married or living with a partner at baseline (Mean age: 40.8 years, SD=5.6; Table 6.1). The proportion of participants who screened positive for depressive symptoms decreased over time (Baseline: N=68, 21.7%; 12-month follow-up: N=43, 15.4%).

The prevalence of any form of recent IPV perpetration was highest at baseline (N=94, 30.0%). At three-month follow-up, the prevalence dropped to 15.4% (N=44) and remained fairly stable throughout the remainder of the study period (six-month follow-up: N=47, 16.1%; 12-month follow-up: N=42, 15.0%; Table 6.2). This decrease in IPV perpetration prevalence from baseline to endline was expected due to the shorter recall periods at follow-up study visits as compared to baseline.

Table 6.1. Characteristics of male participants who were married or living with a partner (N=313)<sup>a</sup>

|   | Baseline (N=313) | 3 months (N=287) | 6 months (N=292) | 12 months (N=279) |
|---|------------------|------------------|------------------|-------------------|
|   |                  | N                | (%)              |                   |
| Sociodemographics   |                  |                  |                  |                   |
| Mean age in years (SD)                                    | 40.8 (5.6)       |                  |                  |                   |
| Highest level of education                                |                  |                  |                  |                   |
| Technical training/College or university or less          | 52 (16.6)        |                  |                  |                   |
| High school or less                                       | 189 (60.4)       |                  |                  |                   |
| Secondary school or less                                  | 48 (15.3)        |                  |                  |                   |
| Primary school or less                                    | 24 (7.7)         |                  |                  |                   |
| Employment status   |                  |                  |                  |                   |
| Employed full- or part-time                               | 262 (83.7)       | 243 (84.7)       | 255 (87.3)       | 245 (87.8)        |
| Unemployed/Retired  | 51 (16.3)        | 44 (15.3)        | 37 (12.7)        | 34 (12.2)         |
| Viral suppression <sup>b,c</sup>                          |                  |                  |                  |                   |
| No  | 45 (14.4)        | 39 (13.6)        | 35 (12.0)        | 42 (15.1)         |
| Yes   | 268 (85.6)       | 247 (86.4)       | 257 (88.0)       | 237 (85.0)        |
| Alcohol Use   |                  |                  |                  |                   |
| Median percent days abstinent in past 30 days (SD)        | 36.7 (33.1)      | 70.0 (35.6)      | 76.7 (37.3)      | 76.7 (36.9)       |
| Median number of heavy drinking days in past 30 days (SD) | 2.0 (10.1)       | 1.0 (8.7)        | 0.0 (8.6)        | 1.0 (8.7)         |
| Alcohol use disorder                                      |                  |                  |                  |                   |
| No  | 189 (60.4)       | 210 (73.2)       | 223 (76.4)       | 225 (80.7)        |
| Yes   | 124 (39.6)       | 77 (26.8)        | 69 (23.6)        | 54 (19.4)         |
| Psychosocial Variables                                    |                  |                  |                  |                   |
| Depressive symptoms                                       |                  |                  |                  |                   |
| Negative  | 245 (78.3)       | 239 (83.3)       | 243 (83.2)       | 236 (84.6)        |
| Positive  | 68 (21.7)        | 48 (16.7)        | 49 (16.8)        | 43 (15.4)         |

| 244 (78.0) | 226 (79.0)  | 228 (78.1)   | 222 (79.6)   |
|------------|---|--|--|
| 69 (22.0)  | 60 (21.0)   | 64 (21.9)  | 57 (20.4)  |
|            |   |  |  |
| 199 (63.6) |   |  |  |
| 114 (36.4) |   |  |  |
|            |   |  |  |
| 178 (56.9) | 176 (61.5)  | 173 (59.3)   | 140 (50.2)   |
| 135 (43.1) | 110 (38.5)  | 119 (40.8)   | 139 (49.8)   |
|            | 69 (22.0)<br>199 (63.6)<br>114 (36.4)<br>178 (56.9) | 69 (22.0) 60 (21.0)<br>199 (63.6)<br>114 (36.4)<br>178 (56.9) 176 (61.5) | 69 (22.0) 60 (21.0) 64 (21.9)  199 (63.6) 114 (36.4)  178 (56.9) 176 (61.5) 173 (59.3) |

<sup>&</sup>lt;sup>a</sup>Percentages may not sum to 100 due to rounding. <sup>b</sup>Achieving viral suppression was defined as having less than 20 copies/ml.

<sup>&</sup>lt;sup>c</sup>Missing data due to refused to answer or don't know: Viral suppression at 3 months: N=1; Injection drug use at 3 months: N=1; Ever involved in community violence at 3 months: N=1.

<sup>&</sup>lt;sup>d</sup>Exposed to violence as a child includes having ever witnessed interparental violence as a child or experienced physical or sexual abuse as a child.

Table 6.2. Prevalence of intimate partner violence perpetration across study visits<sup>a</sup>

|  | Baseline (N=313) | 3 months (N=287) | 6 months (N=292) | 12 months (N=279) |  |  |  |
|--|------------------|------------------|------------------|-------------------|--|--|--|
|  | N (%)            |                  |                  |                   |  |  |  |
| Recent Psychological IPV Perpetration <sup>b</sup> |                  |                  |                  |                   |  |  |  |
| No   | 231 (73.8)       | 250 (87.4)       | 252 (86.3)       | 242 (86.7)        |  |  |  |
| Yes  | 82 (26.2)        | 36 (12.6)        | 40 (13.7)        | 37 (13.3)         |  |  |  |
| Recent Physical IPV Perpetration                   |                  |                  |                  |                   |  |  |  |
| No   | 298 (95.2)       | 282 (98.3)       | 285 (97.6)       | 275 (98.6)        |  |  |  |
| Yes  | 15 (4.8)         | 5 (1.7)          | 7 (2.4)          | 4 (1.4)           |  |  |  |
| Recent Sexual IPV Perpetration                     |                  |                  |                  |                   |  |  |  |
| No   | 293 (93.6)       | 276 (96.2)       | 285 (97.6)       | 270 (96.8)        |  |  |  |
| Yes  | 20 (6.4)         | 11 (3.8)         | 7 (2.4)          | 9 (3.2)           |  |  |  |
| Recent Physical/Sexual IPV Perpetration            |                  |                  |                  |                   |  |  |  |
| No   | 280 (89.5)       | 272 (94.8)       | 279 (95.6)       | 266 (95.3)        |  |  |  |
| Yes  | 33 (10.5)        | 15 (5.2)         | 13 (4.5)         | 13 (4.7)          |  |  |  |
| Any Form of Recent IPV Perpetration <sup>b</sup>   |                  |                  |                  |                   |  |  |  |
| No   | 219 (70.0)       | 242 (84.6)       | 245 (83.9)       | 237 (85.0)        |  |  |  |
| Yes  | 94 (30.0)        | 44 (15.4)        | 47 (16.1)        | 42 (15.0)         |  |  |  |

<sup>&</sup>lt;sup>a</sup>Percentages may not sum to 100 due to rounding.
<sup>b</sup>Missing data due to refused to answer or don't know: Recent psychological IPV perpetration at 3 months: N=1; Any form of recent IPV perpetration at 3 months: N=1.

# 6.3.2. Adjusted analyses

Proportion of days alcohol abstinent. Depressive symptoms significantly moderated the negative relationship between proportion of days alcohol abstinent and IPV perpetration (Table 6.3). As higher proportion of days alcohol abstinent indicate less alcohol use, the inverse relationship between proportion of days alcohol abstinent and IPV perpetration was expected. However, the pattern of moderation did not support my hypothesis, as having depressive symptoms was found to weaken the relationship between proportion of days alcohol abstinent and IPV perpetration (Figure 6.1). At times when individuals screened negative for depressive symptoms, reporting higher proportion of days alcohol abstinent than usual was associated with decreased probability of IPV perpetration (Slope: b=-1.77, SE=0.49, p=0.0004; Table 6.4). In contrast, the time-varying effect of proportion of days alcohol abstinent on IPV perpetration became non-significant at times when individuals screened positive for depressive symptoms (Slope: b=1.45, SE=0.85, p=0.09).

**Heavy drinking.** Depressive symptoms significantly moderated the positive relationship between heavy drinking and IPV perpetration (Table 6.3), although the pattern of moderation diverged from the hypothesis. Having depressive symptoms was found to weaken the association between heavy drinking and IPV perpetration (Figure 6.2). At times when individuals screened negative for depressive symptoms, the relationship between heavy drinking and IPV perpetration became non-significant (Slope: b=0.028, SE=0.019, p=0.15; Table 6.5). At times when individuals screened positive for depressive symptoms, reporting more heavy drinking days than average was associated with decreased probability of IPV perpetration (Slope: b=-0.067, SE=0.032, p=0.04).

**Alcohol Use Disorder (AUD).** Contrary to the hypothesis, depressive symptoms did not moderate the relationship between AUD and IPV perpetration (Table 6.3). However, at times when individuals screened positive versus negative for depressive symptoms, they were 3.56 times as likely to report IPV perpetration (95% CI 1.89, 6.70).

**Table 6.3.** Final adjusted conditional growth model by alcohol use measure<sup>a</sup> (N=313)

|                                 | Proportion of days abstinent | alcohol | Heavy drink             | ing           | Alcohol use disc  | order (AUD) <sup>b</sup> |
|---------------------------------|------------------------------|---------|-------------------------|---------------|-------------------|--------------------------|
|                                 |                              | Coe     | efficient (SE) or aOR ( | (95% CI), p-v | value             |                          |
| Fixed Effects                   |                              |         |                         |               |                   |                          |
| Intercept                       | -1.19 (0.43)                 | 0.006   | -1.08 (0.42)            | 0.01          | -1.16 (0.45)      | 0.01                     |
| Time                            | 0.55 (0.41, 0.73)            | <0.0001 | 0.53 (0.40, 0.70)       | <0.0001       | 0.51 (0.37, 0.69) | <0.0001                  |
| Alcohol use                     | 0.17 (0.065, 0.45)           | 0.0004  | 1.03 (0.99, 1.07)       | 0.15          | 2.93 (1.52, 5.63) | 0.001                    |
| Depressive symptoms             | 3.96 (2.18, 7.20)            | <0.0001 | 3.62 (2.00, 6.55)       | <0.0001       | 3.56 (1.89, 6.70) | <0.0001                  |
| Alcohol use*Depressive symptoms | 25.02 (3.54, 176.78)         | 0.001   | 0.91 (0.85, 0.98)       | 0.01          |                   |                          |
| Random Effects                  |                              |         |                         |               |                   |                          |
| Variance Components             |                              |         |                         |               |                   |                          |
| Covariance                      | -0.0001 (0.25)               |         | -0.063 (0.26)           |               | -0.10 (0.19)      |                          |
| Variance                        | 0.53 (0.57)                  |         | 0.63 (0.59)             |               | 0.87 (0.53)       |                          |
| Model Fit                       |                              |         |                         |               |                   |                          |
| AIC                             | 1017.23                      |         | 1026.78                 |               | 1015.97           |                          |
| BIC                             | 1084.67                      |         | 1094.21                 |               | 1079.66           |                          |
| Neg 2 LL                        | 981.23                       |         | 990.78                  |               | 981.97            |                          |

<sup>&</sup>lt;sup>a</sup>Controlling for education, grand-mean centered age, exposure to violence as a child, employment status, injection drug use, involvement in community violence ever, and intervention arm.

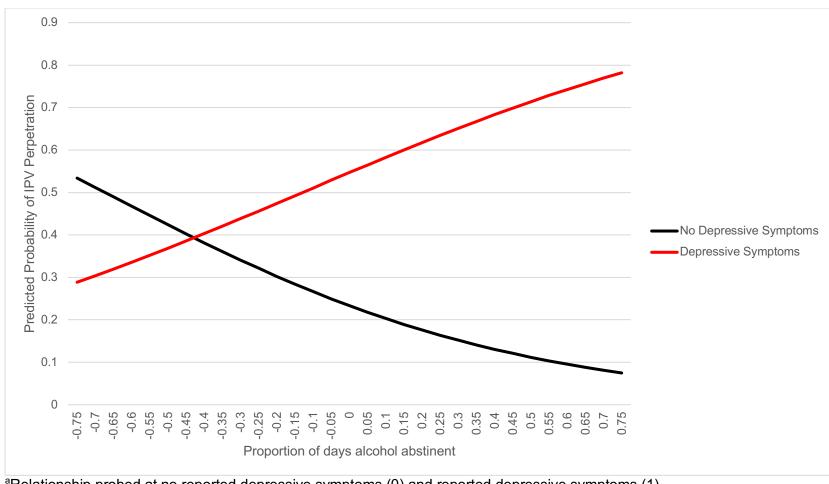
Note: SE=Standard error; aOR=Adjusted odds ratio; AIC=Akaike information criterion; BIC=Bayesian information criterion; Neg 2 LL=Negative 2 log likelihood

<sup>&</sup>lt;sup>b</sup>A random slope for WP AUD was added as it improved model fit.

**Table 6.4.** Simple intercepts and slopes, proportion of days alcohol abstinent\*depressive symptoms

|                        | Intercept    |         | Slope        |         |  |
|------------------------|--------------|---------|--------------|---------|--|
|                        | b (SE)       | p-value | b (SE)       | p-value |  |
| No Depressive Symptoms | -1.19 (0.43) | 0.006   | -1.77 (0.49) | 0.0004  |  |
| Depressive Symptoms    | 0.19 (0.52)  | 0.72    | 1.45 (0.85)  | 0.09    |  |

Note: SE=Standard error



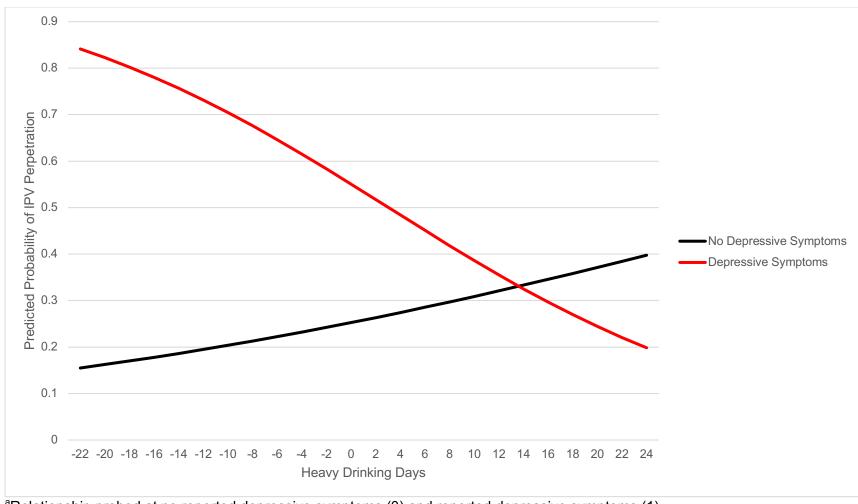
<sup>&</sup>lt;sup>a</sup>Relationship probed at no reported depressive symptoms (0) and reported depressive symptoms (1).

Figure 6.1. Estimated IPV perpetration as a function of depressive symptoms and proportion of days alcohol abstinent at times when participants screened positive and negative for depressive symptoms<sup>a</sup>

**Table 6.5.** Simple intercepts and slopes, heavy drinking\*depressive symptoms

|                        | Intercept    |         | Slope          |         |  |
|------------------------|--------------|---------|----------------|---------|--|
|                        | b (SE)       | p-value | b (SE)         | p-value |  |
| No Depressive Symptoms | -1.08 (0.42) | 0.01    | 0.028 (0.019)  | 0.15    |  |
| Depressive Symptoms    | 0.20 (0.51)  | 0.69    | -0.067 (0.032) | 0.04    |  |

Note: SE=Standard error



<sup>a</sup>Relationship probed at no reported depressive symptoms (0) and reported depressive symptoms (1).

**Figure 6.2.** Estimated IPV perpetration as a function of depressive symptoms and number of heavy drinking days at times participants screened positive and negative for depressive symptoms<sup>a</sup>

#### 6.4. Discussion

In this study, alcohol use and depressive symptoms did not act synergistically to increase the likelihood of IPV perpetration. At times when participants reported having depressive symptoms, the effects of proportion of days alcohol abstinent and heavy drinking on IPV perpetration became non-significant or reversed direction, showing that more alcohol use led to lower predicted probability of IPV perpetration. This finding diverges from empirical and theoretical evidence suggesting that alcohol use and depression interact to exacerbate violence outcomes [25-27, 38, 131-133]. One potential explanation for this unexpected finding may be the unique intersection between depressive symptoms, alcohol use, and social isolation in Vietnam. Men may respond to experiences of depression with social isolation [187, 188], leading to reduced alcohol use as drinking alone is uncommon [18, 108]. Instead, drinking centers around social interactions in Vietnam [18, 84, 108]. Those experiencing the cooccurrence of heightened alcohol use and depressive symptoms may have lower odds of IPV perpetration because they represent a particularly isolated group that is not engaging with their partner, leaving limited opportunity for IPV events to occur. Research is needed to understand the circumstances under which men living with HIV may or may not use alcohol as a coping strategy when experiencing depressive symptoms.

Unexpectedly, depressive symptoms did not influence the relationship between AUD and IPV perpetration. However, a main effect in this model showed that screening positive versus negative for depressive symptoms at one time point increased the odds of IPV perpetration at the same time point. These results enhance findings from a cross-sectional study conducted with men living with HIV in Vietnam [85] and studies in other global settings with men from the broader population [7, 189, 190]. There are several potential explanations for this relationship. As mental health issues may lead to self-control impairment, experiencing depressive symptoms may increase risk for aggressive behavior [27, 134, 135]. Relationship conflict may also increase risk for both depressive symptoms and IPV perpetration [134].

Additionally, the consequences of IPV perpetration may be incident depressive symptoms [191]. Future research should determine temporality to understand whether onset of depressive symptoms precedes IPV perpetration or vice versa.

Due to the high prevalence of depressive symptoms among men living with HIV globally and in Vietnam [84, 192], intervening on depression may be a promising IPV prevention strategy. Forty-four percent of HIV-infected men who inject drugs in Vietnam reported having severe depressive symptoms [84]. Men living with HIV experience a multitude of psychosocial stressors, such as unemployment, lack of social support, and stigma and discrimination [80, 192]. These stressors may make men living with HIV particularly vulnerable to developing depressive symptoms [192], thus increasing their risk of IPV perpetration. Intervening on depression by incorporating mental health services into HIV care and treatment may reduce both depression and IPV perpetration among men living with HIV. A recent systematic review found there was promising evidence that mental health treatments for common mental disorders could effectively reduce experience of IPV in low- and middle-income countries [193]. However, there is limited research on the impact of mental health treatment on IPV perpetration instead of IPV victimization [193].

As all variables were measured using self-report, responses may have been biased due to social desirability, leading to more conservative estimates of IPV perpetration and depressive symptoms [184, 185]. This is probable as the questionnaires were interviewer-administered, which may have made participants less likely to report depressive symptoms or IPV perpetration [150]. However, interviewers received extensive training on questionnaire administration, minimizing the likelihood of social desirability bias. Additionally, estimates of alcohol use may be subject to recall bias as data were collected via self-report. However, sub-analyses of the study data conducted by Go et al. [127] validated the self-report alcohol measures using phosphatidylethanol (PEth), a direct metabolite of alcohol use that acts as a biomarker [186].

This longitudinal study contributes important findings on the intersection between hazardous alcohol use, depressive symptoms, and IPV perpetration in a high-risk, understudied population. Unexpectedly, having depressive symptoms was found to weaken the alcohol-IPV relationship for measures of proportion of days alcohol abstinent and heavy drinking. These results suggest that men who reported higher alcohol use and depressive symptoms than usual may be experiencing extreme social isolation from an intimate partner, leading to reduced IPV perpetration. AUD and depressive symptoms were found to be independent risk factors for IPV perpetration, demonstrating that mental health interventions should be integrated into HIV care and treatment to reduce IPV perpetration. Previous mental health interventions aiming to reduce IPV perpetration have either targeted participants who misuse alcohol or those who screen positive for depressive symptoms [193]. This study suggests that mental health interventions for men living with HIV should target both groups - those who screen positive for hazardous drinking and/or depressive symptoms - to effectively reduce IPV perpetration.

CHAPTER 7: EVALUATING THE EFFECTS OF TWO ALCOHOL REDUCTION INTERVENTIONS ON INTIMATE PARTNER VIOLENCE PERPETRATION AND TESTING ALCOHOL USE AS A PATHWAY FOR EFFECTIVENESS: RESULTS FROM A THREE-ARM RANDOMIZED CONTROLLED TRIAL AMONG MEN LIVING WITH HIV IN NORTHERN VIETNAM

#### 7.1. Introduction

Evidence clearly demonstrates that alcohol use is a risk factor for male-perpetrated intimate partner violence (IPV), offering a promising leverage point for intervention [6-13]. Alcohol use may lead to IPV perpetration due to immediate psychopharmacological effects of alcohol, such as lowered inhibitions or distorted perceptions of cues [12, 14, 15]. Alternatively, alcohol use may lead to increased relationship conflict, resulting in IPV perpetration [9, 10, 16]. Due to the strong link between alcohol and IPV perpetration, researchers and practitioners, including the World Health Organization (WHO), have been advocating for the use of alcohol interventions to reduce IPV perpetration [1, 49, 86-91].

There is some evidence, mostly from the United States, that alcohol reduction interventions involving psychosocial counseling may reduce IPV perpetration among men who use substances [1, 86, 89, 91]. Researchers have proposed a direct and an indirect pathway for which alcohol reduction interventions may decrease IPV perpetration, although these pathways are largely untested [194]. For the direct pathway, researchers posit that intervening on alcohol use leads to reduced IPV perpetration [86, 89, 194], as the psychopharmacological effects of drinking alcohol result in aggressive behavior [12, 14, 15]. For the indirect pathway, researchers theorize that improved relationship functioning may be a potential mechanism that could produce intervention effects on IPV perpetration, as skill-building on couple communication and problem solving are common features of alcohol reduction interventions [194]. While there is

promising evidence demonstrating alcohol reduction interventions may be an effective IPV prevention strategy, there is limited research that is conducted in low- and middle-income countries and that is examining potential pathways for effectiveness.

Alcohol and IPV reduction interventions for men living with HIV are particularly important as they are a group at high risk for IPV perpetration and forward HIV transmission. Research with men shows that hazardous alcohol use, depression, sexual risk behaviors, and IPV perpetration co-occur [57, 70, 195, 196]. For men living with HIV, this pattern of overlapping behavior is associated with poor antiretroviral treatment (ART) adherence, condomless sex, and unsuppressed HIV viral load, all of which are factors that increase risk for HIV transmission and compromise men's health and wellbeing [67-69].

This study aims to: (1) assess the effectiveness of two alcohol reduction interventions (a two-session Brief Intervention [BI] and a six-session Combined Intervention [CoI]) compared to the standard of care (SOC) in reducing IPV perpetration among men living with HIV in northern Vietnam; and (2) examine whether alcohol use explains the relationship between intervention exposure and IPV perpetration (Figure 7.1). Previous analyses indicated that both the BI and CoI decreased alcohol use at three, six, and 12 months [127]. The current analysis expands on this work to examine whether the alcohol reduction interventions have an effect on IPV perpetration and whether alcohol use explains the relationship between intervention exposure and IPV perpetration. I hypothesize that:

- Participants randomized to receive the BI and CoI will report lower levels of IPV
  perpetration at three, six and 12-month follow-up as compared to participants
  randomized to receive the SOC.
- Alcohol use will mediate the relationship between intervention exposure and IPV
  perpetration such that exposure to the BI and CoI vs. SOC will be associated with
  decreased alcohol use, and, in turn, alcohol use will be positively associated with IPV
  perpetration.

Findings will provide evidence that alcohol reduction interventions implemented in understudied settings and populations can impact important health outcomes, such as IPV perpetration, in addition to hazardous alcohol use.

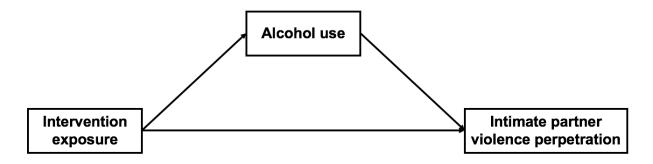


Figure 7.1. Conceptual model

# 7.2. Methods

# 7.2.1. Study design

This study is a secondary analysis of data from a three-arm randomized controlled trial (RCT) among ART patients with HIV and hazardous alcohol use (N=440) in Thai Nguyen, Vietnam [127]. Thai Nguyen is a semi-urban province in northern Vietnam located approximately 75 kilometers north of Hanoi with a population of 1 million. The RCT compared the effects of two evidence-based, manually guided, individually delivered interventions to reduce alcohol use and increase viral suppression. Data were collected from March 2016 to June 2018.

# 7.2.2. Study arms

There were three study arms: Brief Intervention (BI), Combined Intervention (CoI), and standard of care (SOC).

Alcohol reduction interventions. In the formative phase of the trial, two alcohol reduction interventions were selected and culturally adapted, as described in detail elsewhere [136]. The two alcohol reduction interventions were conducted by different teams of trained psychosocial counselors and were delivered within a three-month period after the baseline

questionnaire was administered. Both interventions were guided by the principles of Motivational Enhancement Therapy (MET) and Cognitive Behavioral Therapy (CBT). MET involves prompting motivation for behavior change and helping to develop a plan for change [138-140]. CBT uses problem-solving and coping skills to promote behavior change [141]. Neither the BI nor the Col directly addressed violence.

While both interventions drew from MET and CBT, the structure and focus of the interventions differed. The BI consisted of two individual counseling sessions and two booster phone sessions. Face-to-face sessions took place approximately one month apart and each phone session occurred approximately two to three weeks after each face-to-face session. The duration of the first counseling session was 50 minutes on average and the second was 30 minutes on average (range for both was 29-76 minutes); and each phone session lasted 8 minutes on average (range of 6-12 minutes). The face-to-face sessions were designed to raise awareness of the HIV-related health consequences of excessive alcohol use and to strengthen the patient's commitment to change their alcohol use behavior. The phone sessions reviewed progress and renewed motivation and commitment to change. The session content included a review of drinking patterns, harmful HIV-related effects of excessive drinking, and alcohol use behavior change strategies. Ninety-five percent (133/140) of BI participants attended at least one in-person session and 84% (117/140) attended all four sessions.

The CoI consisted of six mandatory counseling sessions and three optional group sessions. Individual face-to-face sessions occurred approximately one week apart. The duration of the individual sessions was 52 minutes on average (range of 29-76 minutes) and each group session was 65 minutes on average (range of 60-70 minutes). The mandatory sessions were designed to develop coping skills to assist the patient in managing alcohol cravings and high-risk moods and situations. The session content included a review of drinking patterns and harmful effects of drinking as well as skill-building on alcohol use behavior change. In particular, skills related to alcohol use behavior change focused on drinking refusal skills, skills to cope

with managing cravings and triggers, and development of positive thoughts and attitudes. The group sessions emphasized the skills learned in the one-on-one sessions and facilitated interactions between patients so that they could support one another in managing their drinking and share experiences around alcohol. Participants could attend the group sessions at any point after being randomized into the study. Ninety-eight percent (142/145) of the Col participants attended the first in-person session, 90% (131/145) attended at least three in-person sessions, and 76% (110/145) attended all six in-person sessions. Further, 83% (121/145) attended at least one optional group session and 76% (110/145) attended all three optional group sessions.

**Standard of care.** Participants randomized to the standard of care (SOC) group received referrals to alcohol treatment and infectious disease treatment.

# 7.2.3. Participant recruitment and data collection

Participant recruitment took place at all seven ART community clinics in Thai Nguyen. All recruitment was completed in one clinic before recruiting participants from the subsequent clinic. Clinics were approached for recruitment in a random order. After introducing the project, trained study interviewers administered baseline written informed consent to those who were interested in study participation and were 18 years of age or older. After providing consent, the interviewer administered a screening survey including the WHO Alcohol Use Disorders Identification Test (AUDIT-C) survey items to determine eligibility for study enrollment [142]. Male patients with an AUDIT-C score ≥ 4 were considered to have hazardous alcohol use. Inclusion criteria for eligibility included: (1) current patient on ART at the clinic; (2) AUDIT-C score ≥ 4; (3) 18 years of age or older; and (4) plan on residing in Thai Nguyen for the next 24 months. Exclusion criteria for eligibility included: (1) unable to participate in study activities due to psychological disturbance, cognitive impairment or threatening behavior (assessed by study staff); (2) unwilling to provide locator information; (3) unwilling to provide informed consent; and (4) currently participating in other HIV, drug use, or alcohol program, study, or intervention.

Participants who were found eligible for the study based on the screening survey were asked to consent to enroll in the study. Interviewers described the RCT study objectives, procedures, risks and benefits to the eligible participants, and answered any questions. If needed due to literacy issues, individuals were allowed to mark the baseline or enrollment and follow-up consent forms with an "X." If the participant provided written informed consent, he/she was assigned a unique identification number to be used throughout the study. Then, he/she completed the baseline questionnaire and Timeline Followback (TLFB) to measure daily alcohol use over the past 30 days. In a 1:1:1 ratio, eligible participants were randomly assigned to one of the three study arms.

Quantitative assessments were administered to participants at baseline, three, six, and 12 months. On average, each study visit lasted approximately two hours. In a private room at an ART clinic, assessments were administered through face-to-face interviews by trained interviewers. At each follow-up study visit, participants provided informed consent for study enrollment, and completed the follow-up questionnaire and TLFB. The quantitative assessment collected data on sociodemographics, alcohol and drug use, and violence. Excluding those who were incarcerated (n=13/440; 2.5%) and/or died (n=15/440; 3.4%) during the course of the study, retention was 94% (405/430) at three months, 96% (410/427) at six months, and 94% (390/414) at 12 months. Eighty-five percent (372/440) completed all four assessments, 9% (39/440) completed three assessments, 3% (13/440) completed two assessments, and 4% (16/440) completed one assessment.

The study protocol received ethical approval from the Institutional Review Boards at the University of North Carolina-Chapel Hill and Thai Nguyen Center for Preventive Medicine.

# 7.2.4. Key measures

**IPV perpetration.** Recent psychological, physical, and sexual IPV perpetration among any current or previous partner was measured with the widely used and validated shortened Conflict Tactics Scale 2 (CTS2) [143-145]. The scale comprised six total items and

psychological, physical, and sexual IPV perpetration were each measured using two of the items. At baseline, participants were asked about any events of IPV in the past year. At the following study visits, the recall period was the past three months. In this paper, due to the various recall periods used across study visits, the outcome variable will be referred to as "recent IPV perpetration." As the number of recent IPV events reported were skewed, responses were dichotomized to those who reported recent IPV perpetration at least once and those who did not. Those who refused to answer or didn't know the answer were marked as missing. The binary outcome variable for any form of recent IPV perpetration was created using all six items for the CTS2 [172].

Alcohol use. There were three alcohol use variables used in analyses that were measured the same way across all time points. Using the TLFB, alcohol use was assessed as proportion of days alcohol abstinent in past 30 days (0 to 1) and number of heavy drinking days in past 30 days. A heavy drinking day was defined as having more than four drinks per day for men [148]. TLFB is a tool that has been found to be valid and reliable across multiple settings and multiple populations [146, 147]. The TLFB is a daily behavior calendar; it is interviewer-administered to help prompt memory recall for alcohol consumption.

To capture patterns of problem drinking, alcohol use was also measured using the Mini International Neuropsychiatric Interview questionnaire (MINI) Diagnostic and Statistical Manual of Mental Health Disorders, 4th edition (DSM IV) [149]. The MINI questionnaire is a set of items that measures both alcohol abuse and alcohol dependence, which are generally defined by level of drinking and mental or physical harm due to drinking [142]. Composite scores for each participant were calculated. Based on the standard scoring method used for the MINI, participants who scored three or more on the alcohol dependence items and/or one or more on the alcohol abuse items were categorized as having alcohol use disorder (AUD); those who did not were categorized as not having AUD [149].

Covariates. Covariates were identified for inclusion in the analysis based on potential for confounding of the association between alcohol use and IPV perpetration [6, 11, 35, 60, 123, 152, 153]. Three questions from the baseline questionnaire were combined to create an index capturing exposure to violence as a child: ever witnessed interparental violence as a child, ever experienced physical abuse as a child, and ever experienced sexual abuse as a child.

Responses were categorized as having been exposed to violence as a child if a participant answered yes to any of the three questions or no if a participant answered no to all three questions. Two questions asked at all study visits were combined to measure involvement in community violence ever: ever been physically violent towards someone in their community; and ever experienced physical violence by someone in their community [154]. As both experiencing and perpetrating community violence are known risk factors for IPV perpetration [6, 35, 173, 174], a composite variable capturing time-varying involvement in community violence ever was used.

Additional time-varying covariates measured via self-report at all study visits were partnership status (married/living with a partner or not), employment status (employed full- or part-time/unemployed or retired), and injection drug use in the past three months. The remaining covariates were time-invariant and measured only at baseline: education (primary school or less/secondary school or less/high school or less/technical training or college or university or less) and age.

## 7.2.5. Data analysis

Previous analyses indicated that both the BI and CoI increased proportion of days alcohol abstinent and decreased number of heavy drinking days at three, six, and 12 months; intervention effects on AUD were untested [127]. In this study, AUD was also tested as a potential mediator due to results from a meta-analysis demonstrating that effect sizes for the alcohol-IPV association vary across measures of proportion of days alcohol abstinent, heavy drinking, and AUD [12].

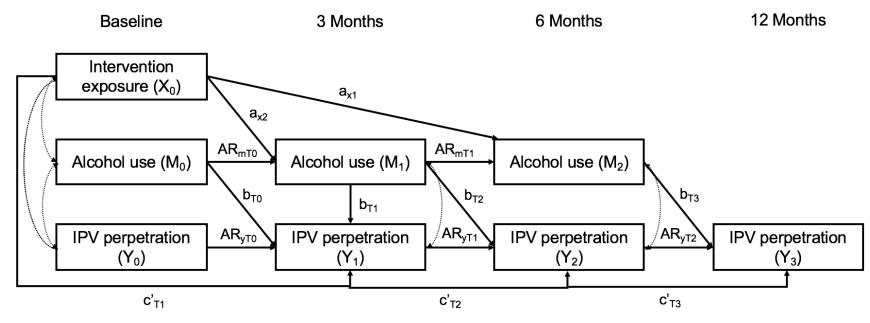
The analysis was restricted to male participants due to the focus of the study on male-to-female IPV perpetration. As the prevalence of IPV perpetration differs by marital status in this population and setting [85], sensitivity analyses with male participants who reported being married/living with a partner at baseline were conducted (Appendix B). As there were no substantive differences in results, the analyses with the full sample of male participants are presented. Descriptive and correlational statistical analyses were conducted using SAS 9.4. Frequencies and percentages for categorical variables and means with standard deviation (SD) for continuous variables were reported. Bivariable statistics, including one-way ANOVA for continuous variables and Wald  $X^2$  for categorical variables, were used to assess comparability across study arms at baseline.

To evaluate intervention effects on IPV perpetration at three, six, and 12 months, path analysis on MPlus8 was used. Longitudinal pathways between intervention assignment and recent IPV perpetration at each time point were estimated, controlling for covariates.

Lagged panel mediation models were used to test whether alcohol use mediates the relationship between intervention exposure and IPV perpetration, with separate models estimated for each alcohol use measure (Figure 7.2). In each model, the lagged relationship between intervention exposure and alcohol use, or the 'a path', was tested to evaluate intervention effects on alcohol use post-intervention. The relationship between alcohol use and IPV perpetration, or the 'b path', was tested to examine the lagged associations between alcohol use and IPV perpetration controlling for intervention exposure. Estimates from the path analysis were then used to calculate the causally-defined mediation effects, including: (1) the natural indirect effect, or mediated effect, which estimates the reduction in IPV perpetration comparing the BI and CoI interventions to a hypothetical condition of intervention participation where the intervention's effects on alcohol use is blocked; and (2) the natural direct effect, or the 'c' paths', which estimates the reduction in IPV perpetration comparing the hypothetical condition to the SOC group [163, 164]. Potential confounding was addressed by including: (1) autoregressive

(AR) pathways for prior levels of alcohol use and IPV perpetration; and (2) covariates for the alcohol-IPV relationship (age, education, marital status, employment status, injection drug use, exposure to violence as a child, and involvement in community violence ever). Additionally, covariances between all variables assessed at the same time point were included. Total, natural direct, and natural indirect effects odds ratios (ORs) were estimated using maximum likelihood [163, 164]. Standard errors (SEs) and bootstrapped confidence intervals (CIs) for indirect effects were estimated based on 5,000 bootstrap resamples to provide more accurate Type I error rates and greater power for detecting indirect effects than other methods [165, 166]. We concluded that mediation was present if the indirect effect of intervention exposure on IPV perpetration through alcohol use was found to be statistically significant [167].

All analyses were conducted as intention to treat. To address missingness, multiple imputation was conducted with MPlus8. As the findings had no substantive differences to those conducted using available cases (Appendix C), the results from the complete case analysis at three months (N=384), six months (N=372), and 12 months (N=368) are presented as the main results.



 $X_0$ = Predictor variable (intervention exposure) at baseline;  $M_0$ ,  $M_1$ ,  $M_2$ = Mediator variable (alcohol use) at baseline, three, and six months, respectively;  $Y_0$ ,  $Y_1$ ,  $Y_2$ ,  $Y_3$ = Outcome variable (IPV perpetration) at baseline, three, six, and 12 months, respectively;  $a_{x1}$ ,  $a_{x2}$ = a paths (intervention effects on alcohol use) at three and six months, respectively;  $b_{T0}$ ,  $b_{T1}$ ,  $b_{T2}$ ,  $b_{t3}$ = b paths (associations between alcohol use and IPV perpetration controlling for covariates) at baseline, three, six and 12 months, respectively;  $AR_{mT0}$ ,  $AR_{mT1}$  = AR paths for prior levels of alcohol use at three and six months, respectively;  $AR_{yT0}$ ,  $AR_{yT1}$ ,  $AR_{yT2}$ = AR paths for prior levels of IPV perpetration at three, six, and 12 months, respectively;  $c'_{yT1}$ ,  $c'_{T2}$ ,  $c'_{T3}$ = c' paths (direct effects of intervention exposure on IPV perpetration) at three, six, and 12 months, respectively.

Note: IPV= Intimate partner violence; AR= Autoregressive

Figure 7.2. Hypothesized lagged panel mediation model

### 7.3. Results

### 7.3.1. Study sample

There were 426 male participants enrolled in the study at baseline (N=141 SOC participants; N=140 BI participants; N=145 CoI participants; Table 7.1). The mean age of participants was 40.2 years (SD=5.7) and the majority reported being married or living with a partner (N=313, 73.5%). Most participants had completed high school or less (N=239, 56.1%) and were employed full- or part-time (N=344, 80.8%). Median percent days alcohol abstinent in the past 30 days was 33.3% (SD=33.5), median number of heavy drinking days in the past 30 days was 2.0 (SD=10.5), and 42.0% (N=179) screened positive for AUD. A quarter of participants reported perpetrating any form of IPV perpetration (N=106, 25.1%). No significant differences in characteristics were observed between groups at baseline.

**Table 7.1.** Characteristics of male participants at baseline by trial arm

|   | Total       | SOC         | BI               | Col         | p-value |
|---|-------------|-------------|------------------|-------------|---------|
|   | (N=426)     | (N=141)     | (N=140)          | (N=145)     |         |
|   |             | N           | l (%) or Mean (S | (D)         |         |
| Sociodemographics                                     |             |             |                  |             |         |
| Age in years (range: 25-60)                           | 40.2 (5.7)  | 40.2 (5.5)  | 39.9 (5.7)       | 40.6 (5.8)  | 0.604   |
| Partnership status <sup>a</sup>                       |             |             |                  |             | 0.901   |
| No  | 113 (26.5)  | 37 (26.2)   | 39 (27.9)        | 37 (25.5)   |         |
| Yes   | 313 (73.5)  | 104 (73.8)  | 101 (72.1)       | 108 (74.5)  |         |
| Highest level of education                            |             |             |                  |             | 0.748   |
| Technical training/College or university or less      | 63 (14.8)   | 16 (11.4)   | 24 (17.1)        | 23 (15.9)   |         |
| High school or less                                   | 239 (56.1)  | 85 (60.3)   | 73 (52.1)        | 81 (55.9)   |         |
| Secondary school or less                              | 82 (19.3)   | 24 (17.0)   | 26 (18.6)        | 32 (22.1)   |         |
| Primary school or less                                | 42 (9.9)    | 16 (11.4)   | 17 (12.1)        | 9 (6.2)     |         |
| Employment status                                     |             |             |                  |             | 0.409   |
| Employed full- or part-time                           | 344 (80.8)  | 109 (77.3)  | 114 (81.4)       | 121 (83.5)  |         |
| Unemployed/Retired                                    | 82 (19.3)   | 32 (22.7)   | 26 (18.6)        | 24 (16.6)   |         |
| Alcohol Use   |             |             |                  |             |         |
| Median percent days alcohol abstinent in past 30 days | 33.3 (33.5) | 36.7 (33.1) | 36.7 (33.6)      | 26.7 (33.9) | 0.700   |
| Median number of heavy drinking days in past 30 days  | 2.0 (10.5)  | 2.0 (10.4)  | 2.0 (10.2)       | 3.0 (10.9)  | 0.669   |
| Alcohol abuse/dependence                              |             |             |                  |             | 0.118   |
| No  | 247 (58.0)  | 83 (58.9)   | 72 (51.4)        | 92 (63.5)   |         |
| Yes   | 179 (42.0)  | 58 (41.1)   | 68 (48.6)        | 53 (36.6)   |         |
| Psychosocial Variables                                |             |             | · · · · · · ·    |             |         |
| Injection drug use in past 3 months <sup>b</sup>      |             |             |                  |             | 0.503   |
| No  | 308 (72.5)  | 98 (69.5)   | 101 (72.1)       | 109 (75.7)  |         |
| Yes   | 117 (27.5)  | 43 (30.5)   | 39 (27.9)        | 35 (24.3)   |         |

| Exposed to violence as a child               |            |            |            |            | 0.875 |
|--|------------|------------|------------|------------|-------|
| No   | 272 (63.9) | 89 (63.1)  | 88 (62.9)  | 95 (65.5)  |       |
| Yes  | 154 (36.2) | 52 (36.9)  | 52 (37.1)  | 50 (34.5)  |       |
| Ever involved in community violence          |            |            |            |            | 0.351 |
| No   | 230 (54.0) | 83 (58.9)  | 71 (50.7)  | 76 (52.4)  |       |
| Yes  | 196 (46.0) | 58 (41.1)  | 69 (49.3)  | 69 (47.6)  |       |
| Intimate partner violence (IPV) perpetration |            |            |            |            |       |
| Any form of recent IPV perpetration          |            |            |            |            | 0.659 |
| No   | 316 (74.9) | 104 (74.3) | 102 (72.9) | 110 (77.5) |       |
| Yes  | 106 (25.1) | 36 (25.7)  | 38 (27.1)  | 32 (22.5)  |       |

<sup>&</sup>lt;sup>a</sup>Those who reported being married or living with a partner were categorized as having a partner and those who reported being single, separated, divorced, or widowed were categorized as not having a partner.

<sup>b</sup>Missing data due to not knowing or refused to answer: Injection drug use (Col group): N=1; Any form of recent IPV perpetration: N=1

<sup>&</sup>lt;sup>b</sup>Missing data due to not knowing or refused to answer: Injection drug use (CoI group): N=1; Any form of recent IPV perpetration: N=1 SOC participant, N=3 CoI participants.

### 7.3.2. Intimate partner violence perpetration

The prevalence of any form of recent IPV perpetration at baseline was high across all groups (SOC: N=36, 25.7%; BI: N=38, 27.1%; CoI: N=32, 22.5%; Table 7.2). Psychological IPV perpetration was the most prevalent form of recent IPV perpetration reported at baseline (SOC: N=30, 21.4%; BI: N=31, 22.1%; CoI: N=29, 20.4%). As expected due in part to the shorter recall periods, the prevalence of any form of recent IPV perpetration decreased across all trial arms from baseline to endline (Figure 7.3). At each follow-up time point, the IPV perpetration prevalence was highest in the SOC group. At 12 months, the prevalence of recent IPV perpetration was 14.1% (N=17) in the SOC group, 12.3% (N=15) in the BI group, and 11.3% (N=15) in the Col group.

Table 7.2. Prevalence of recent intimate partner violence (IPV) perpetration by trial arm

|                               |                | Baseline      |                |                | 3 months      |                |                | 6 months      |                |                | 12 months     | 6              |
|-------------------------------|----------------|---------------|----------------|----------------|---------------|----------------|----------------|---------------|----------------|----------------|---------------|----------------|
|                               | SOC<br>(N=141) | BI<br>(N=140) | Col<br>(N=145) | SOC<br>(N=129) | BI<br>(N=127) | Col<br>(N=133) | SOC<br>(N=128) | BI<br>(N=127) | Col<br>(N=139) | SOC<br>(N=121) | BI<br>(N=122) | Col<br>(N=133) |
|                               |                | ,             | ,              | ,              |               | N (            | (%)            | ,             | ,              | ,              | ,             | ,              |
| Recent                        | 30             | 31            | 29             | 21             | 4             | 12             | 15             | 16            | 14             | 13             | 13            | 15             |
| psychological IPV             | (21.4)         | (22.1)        | (20.4)         | (16.3)         | (3.2)         | (9.0)          | (11.8)         | (12.6)        | (10.1)         | (10.7)         | (10.7)        | (11.3)         |
| perpetrationa                 |                |               |                |                |               |                |                |               |                |                |               |                |
| Recent physical               | 4              | 10            | 3              | 3              | 0             | 2              | 4              | 2             | 2              | 3              | 1             | 0              |
| IPV perpetration <sup>a</sup> | (2.9)          | (7.1)         | (2.1)          | (2.3)          | (0.0)         | (1.5)          | (3.1)          | (1.6)         | (1.4)          | (2.5)          | (8.0)         | (0.0)          |
| Recent sexual IPV             | 9              | 9             | 6              | 6              | 5             | 2              | 6              | 1             | 3              | 6              | 3             | 1              |
| perpetrationa                 | (6.4)          | (6.4)         | (4.2)          | (4.7)          | (3.9)         | (1.5)          | (4.7)          | (8.0)         | (2.2)          | (5.0)          | (2.5)         | (8.0)          |
| Any form of recent            | 36             | 38            | 32             | 24             | 9             | 13             | 20             | 16            | 18             | 17             | 15            | 15             |
| IPV perpetration <sup>a</sup> | (25.7)         | (27.1)        | (22.5)         | (18.6)         | (7.1)         | (9.8)          | (15.8)         | (12.6)        | (13.0)         | (14.1)         | (12.3)        | (11.3)         |

<sup>a</sup>Missing data due to not knowing or refused to answer: Recent psychological, physical, sexual, and any form of recent IPV perpetration at baseline in the SOC group: N=1; Recent psychological IPV perpetration and any form of recent IPV perpetration at 3 months in the SOC group: N=1; Recent psychological IPV perpetration and any form of IPV perpetration at 3 months in the BI group: N=1; Recent psychological, physical, sexual, and any form of recent IPV perpetration at baseline in the Col group: N=3.

Note: SOC=Standard of Care; BI=Brief Intervention; Col=Combined Intervention; IPV=intimate partner violence.

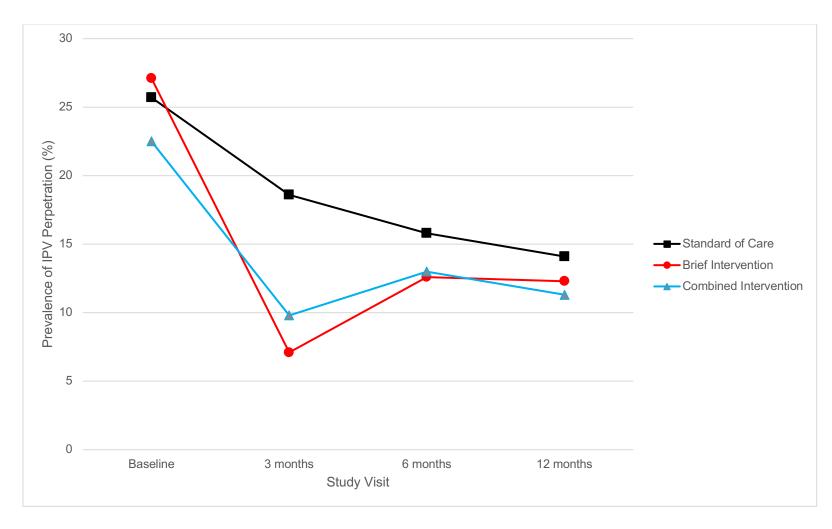


Figure 7.3. Prevalence of any form of intimate partner violence (IPV) perpetration over the study period by trial arm

### 7.3.3. Intervention effects

Compared to the SOC group, BI participants were significantly less likely to report any form of IPV perpetration at three months (aOR=0.27, 95% CoI 0.11, 0.65; Table 7.3). The CoI participants were also less likely to report any form of IPV perpetration at three months as compared to the SOC participants (aOR=0.50, 95% CoI 0.22, 1.13), although the association was not statistically significant. There were no significant intervention effects observed at six and 12 months. Due to the results of the intervention effects models, lagged panel mediation models were only used to test whether alcohol use mediated the significant relationship between intervention exposure and IPV perpetration at three months.

Table 7.3. Intervention effects on intimate partner violence (IPV) perpetration<sup>a</sup>

|                                     | 3 months          | s (N=384)         | 6 months          | s (N=372)         | 12 months (N=368) |                   |  |
|-------------------------------------|-------------------|-------------------|-------------------|-------------------|-------------------|-------------------|--|
|                                     | Brief             | Combined          | Brief             | Combined          | Brief             | Combined          |  |
|                                     |                   |                   | aOR (             | 95% CI)           |                   |                   |  |
| Any form of recent IPV perpetration | 0.27 (0.11, 0.65) | 0.50 (0.22, 1.13) | 0.91 (0.42, 1.98) | 0.94 (0.44, 1.98) | 0.94 (0.42, 2.14) | 0.84 (0.37, 1.90) |  |

<sup>&</sup>lt;sup>a</sup>Controlling for age, education, IPV perpetration (at previous time point), partnership status (at previous time point), employment status (at previous time point), injection drug use (at previous time point), exposure to violence as a child, and involvement in community violence ever (at previous time point).

Note: Estimates in bold are significant at p<0.05. aOR=Adjusted odds ratio; CI=Confidence Interval; IPV=Intimate partner violence

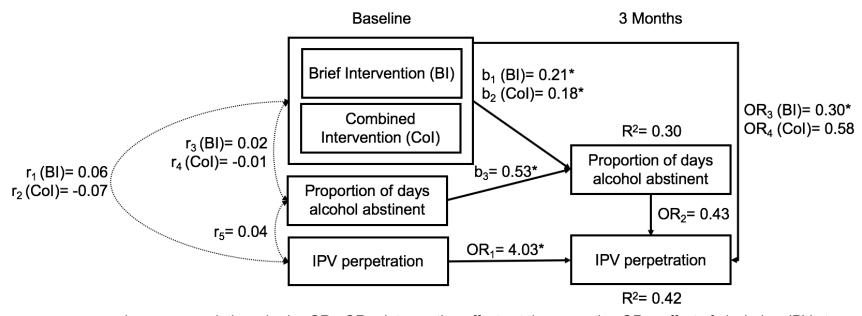
### 7.3.4. Mediation analyses

The results for the intervention effects on alcohol use, or the a paths, varied slightly across lagged panel mediation models (Figures 7.4-6). Both the BI and CoI participants reported significant increases in proportion of days alcohol abstinent at three months (BI: b=0.21, 95% CI 0.14, 0.29, SE=0.04; CoI: b=0.18, 95% CI 0.11, 0.26, SE=0.04). This result was expected as higher proportion of days alcohol abstinent corresponds to less alcohol use. Both the BI and CoI participants also reported significant decreases in number of heavy drinking days at three months as compared to SOC participants (BI: b=-3.52, 95% CI -5.40, -1.68, SE=0.95; CoI: b=-4.38, 95% CI -6.24, -2.53, SE=0.97). CoI and BI participants reported reduced likelihood of reported AUD at three months as compared to SOC participants, although results were only significant in the CoI group (BI: aOR=0.71, 95% CI 0.40, 1.23, SE=0.21; CoI: aOR=0.55, 95% CI 0.29, 0.98, SE=0.17).

Similarly, the results for the associations between alcohol use and IPV perpetration at three months, or the b paths, differed across models. The relationships between alcohol use and IPV perpetration at three months were non-significant in the models for proportion of days alcohol abstinent and heavy drinking. However, as expected, participants who screened positive versus negative for AUD were 2.34 times as likely to report IPV perpetration at three months (95% CI 1.13, 5.95, SE=0.97).

The direct and indirect effects used to test mediation were computed based on the path analysis for each lagged panel mediation model (Table 7.4). No alcohol use measures were found to mediate the relationship between intervention exposure and IPV perpetration, as all indirect effect estimates were non-significant. The natural direct effects of the BI intervention on recent IPV perpetration when blocking the pathway through alcohol use were significant across all three models (Proportion of days alcohol abstinent: OR=0.30, 95% CI 0.08, 0.79, SE=0.18; Heavy drinking: OR=0.28, 95% CI 0.08, 0.71, SE=0.16; AUD: OR=0.27, 95% CI 0.08, 0.66,

SE=0.15). The natural direct effects of the CoI intervention on recent IPV perpetration were non-significant across all models, suggesting the intervention was not effective when blocking the pathway through alcohol use.

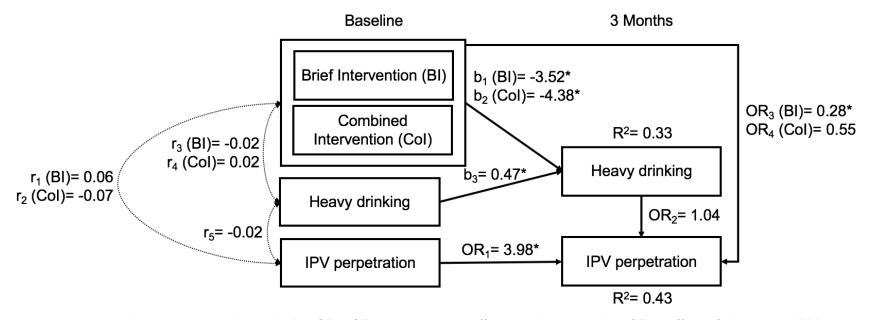


 $r_1$ ,  $r_2$ ,  $r_3$ ,  $r_4$ ,  $r_5$ = synchronous correlations;  $b_1$ ,  $b_2$ ,  $OR_3$ ,  $OR_4$ = intervention effects at three months;  $OR_2$ = effect of alcohol on IPV at three months;  $b_3$ ,  $OR_1$ = autoregressive (AR) pathways;  $R^2$ = variance explained. \*p<0.05

<sup>a</sup>Controlling for age, education, baseline partnership status, baseline employment status, baseline injection drug use, exposure to violence as a child, and baseline involvement in community violence ever.

Note: IPV=Intimate partner violence; OR=Odds ratio

**Figure 7.4.** Path analysis, proportion of days alcohol abstinent<sup>a</sup> (N=384)

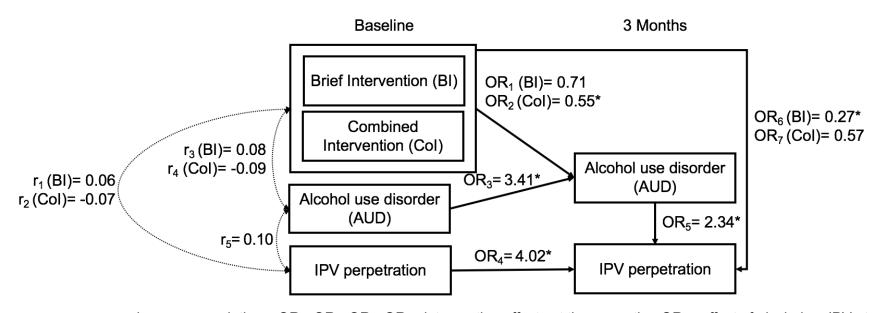


 $r_1$ ,  $r_2$ ,  $r_3$ ,  $r_4$ ,  $r_5$ = synchronous correlations;  $b_1$ ,  $b_2$ ,  $OR_3$ ,  $OR_4$ = intervention effects at three months;  $OR_2$ = effect of alcohol on IPV at three months;  $b_3$ ,  $OR_1$ = autoregressive (AR) pathways;  $R^2$ = variance explained. \*p<0.05

<sup>a</sup>Controlling for age, education, baseline partnership status, baseline employment status, baseline injection drug use, exposure to violence as a child, and baseline involvement in community violence ever.

Note: IPV=Intimate partner violence; OR=Odds ratio

Figure 7.5. Path analysis, number of heavy drinking days<sup>a</sup> (N=384)



 $r_1$ ,  $r_2$ ,  $r_3$ ,  $r_4$ ,  $r_5$ = synchronous correlations; OR<sub>1</sub>, OR<sub>2</sub>, OR<sub>6</sub>, OR<sub>7</sub>= intervention effects at three months; OR<sub>5</sub>= effect of alcohol on IPV at three months; OR<sub>3</sub>, OR<sub>4</sub>= autoregressive (AR) pathways. \*p<0.05

<sup>a</sup>Controlling for age, education, baseline partnership status, baseline employment status, baseline injection drug use, exposure to violence as a child, and baseline involvement in community violence ever.

Note: R<sup>2</sup> could not be calculated by MPlus8 due to the binary mediator. IPV=Intimate partner violence; OR=Odds ratio

**Figure 7.6.** Path analysis, alcohol use disorder (AUD)<sup>a</sup> (N=384)

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**Table 7.4.** Mediation results by alcohol measure<sup>a,b</sup> (N=384)

|  | Proportion of days alcohol abstinent |      | Heavy drinki      | Heavy drinking |                   | er (AUD) |
|--|--------------------------------------|------|-------------------|----------------|-------------------|----------|
| _  |                                      |      | OR (95% CI), S    | SE             |                   |          |
| Effect of BI on IPV through alcohol use  |                                      |      |                   |                |                   |          |
| Natural indirect effect                  | 0.84 (0.63, 1.04)                    | 0.10 | 0.88 (0.74, 1.01) | 0.07           | 0.95 (0.80, 1.04) | 0.06     |
| Natural direct effect                    | 0.30 (0.08, 0.79)                    | 0.18 | 0.28 (0.08, 0.71) | 0.16           | 0.27 (0.08, 0.66) | 0.15     |
| Total effect                             | 0.25 (0.07, 0.62)                    | 0.14 | 0.25 (0.07, 0.61) | 0.14           | 0.25 (0.07, 0.61) | 0.14     |
| Effect of Col on IPV through alcohol use |                                      |      |                   |                |                   |          |
| Natural indirect effect                  | 0.86 (0.66, 1.03)                    | 0.09 | 0.86 (0.70, 1.01) | 0.08           | 0.91 (0.73, 1.01) | 0.07     |
| Natural direct effect                    | 0.58 (0.21, 1.46)                    | 0.32 | 0.55 (0.20, 1.40) | 0.31           | 0.57 (0.21, 1.44) | 0.32     |
| Total effect                             | 0.50 (0.18, 1.16)                    | 0.25 | 0.48 (0.17, 1.14) | 0.25           | 0.52 (0.19, 1.25) | 0.28     |

<sup>&</sup>lt;sup>a</sup>As causally-defined mediation effects are computed differently than effect estimates in the path analyses, total effect and natural direct effect estimates may differ slightly than those shown in the path analyses.

*Note:* Estimates in bold are significant at p<0.05. OR=Odds ratio; CI=Confidence interval; SE=Standard error; BI=Brief Intervention; CoI=Combined Intervention; IPV=Intimate partner violence

<sup>&</sup>lt;sup>b</sup>Controlling for age, education, baseline IPV perpetration, baseline partnership status, baseline employment status, baseline injection drug use, exposure to violence as a child, and baseline involvement in community violence ever.

#### 7.4. Discussion

To my knowledge, this is the first study to evaluate alcohol reduction intervention effects on IPV perpetration among a sample of men living with HIV. Reductions in IPV perpetration were observed at three months in the BI and CoI groups as compared to the SOC group. BI and CoI intervention effects were not sustained at six and 12 months. Previous research in high-income countries has demonstrated that alcohol reduction interventions are an effective IPV prevention strategy [1, 86, 89, 91]. This study builds on this research and suggests that alcohol reduction interventions may be a promising IPV and HIV prevention strategy in low- or middle-income countries, although interventions should be enhanced to improve and sustain effects on IPV perpetration. As both alcohol reduction interventions had sustained effects on alcohol use at six and 12 months, additional intervention activities may need to be incorporated to intervene more effectively on IPV perpetration in addition to alcohol use [127].

In contrast to our study, alcohol reduction interventions involving psychosocial counseling implemented in the United States among men with alcohol dependence have observed sustained effects on IPV perpetration at six and 12 months [31, 92]. However, the interventions tested in these studies were far more intensive than the interventions tested in our study, with one intervention involving a partial hospitalization treatment program [31] and another involving eight individual and 16 group therapy sessions [92]. Further, the participants in these studies were at higher risk for IPV perpetration than those in my study as they comprised men with alcohol abuse or alcohol dependence, which is a more severe alcohol-related inclusion criterion than the one used in this trial (AUDIT-C score ≥ 4) [31, 92]. Adding booster psychosocial counseling sessions on alcohol use and related topics to maintain engagement with participants and reinforce messaging may be beneficial and sustain intervention effects on IPV perpetration.

Incorporating IPV-related components into alcohol reduction interventions may also improve and sustain intervention effects on IPV perpetration. Several trials in the United States

and India have evaluated individual-based psychosocial counseling interventions integrating alcohol and IPV reduction content and found mixed results for alcohol and IPV outcomes [29, 30, 93]. However, couples-based interventions involving behavioral couples therapy (BCT) have produced sustained effects on IPV perpetration when compared to individual-based alcohol reduction interventions in the United States [28, 39, 94]. A pilot study in India also found that a BCT intervention coupled with monetary incentives for men's negative breathalyzer scores significantly reduced IPV [95]. BCT is a type of substance use disorder treatment involving the partner that teaches skills that promote partner support for abstinence and emphasizes healthy conflict mediation skills to deal with relationship problems. While incorporating BCT offers a potential option to improve and sustain effects on IPV perpetration, cost and scalability of the interventions may be compromised [96]. Future research should assess cost-effectiveness of enhanced alcohol reduction interventions.

While the BI and Col reduced alcohol use, mediation analysis suggests that alcohol use reduction did not explain the impact of the BI or Col on IPV perpetration at three months. There may be alternative mechanisms through which the interventions impacted IPV perpetration. It may be that the BI and Col improved relationship functioning, thus leading to reduced IPV perpetration; this is consistent with research conducted in other settings [194]. For men living with HIV, gaining skills in problem solving and communication may be a crucial component for IPV prevention efforts. Men living with HIV are known to experience high levels of anger and hostility associated with their HIV status [181]. Thus, psychosocial counseling interventions improving anger management may be particularly effective in this population [134, 193, 197]. As this study suggests that other mechanisms beyond alcohol use may explain intervention effects, future research should consider testing conflict mediation skills and relationship conflict as potential mediators.

It is important to note that this study may have had low power to detect intervention and mediated effects. This is probable as IPV perpetration decreased across all study arms over

time and the prevalence of IPV perpetration in the study sample was lower than in other intervention trials [31, 92]. Additionally, there were strong associations between the CoI and AUD and between AUD and IPV perpetration, yet mediation was not supported; this further suggests I had low power to detect mediated effects. A reference period of three months may also be too short to capture exposure to IPV perpetration.

There are additional limitations to consider related to the mediation analyses. First, there may be potential unmeasured confounding between the mediator (alcohol use) and the outcome (IPV perpetration), such as partner characteristics. Secondly, there is difficulty establishing temporality between the mediator and the outcome as alcohol use and IPV perpetration were measured at the same study visit. Further complicating this point is that the recall period for proportion of days alcohol abstinent and heavy drinking (30 days) and IPV perpetration (three months) differed at three months follow-up. However, the alcohol use variables are intended to measure alcohol use since the last data collection time point. When using the TLFB, research has shown that a recall period of 30 days can be used to estimate annual consumption [147]. Further, the relationship between alcohol use and IPV perpetration is commonly explained by the immediate psychopharmacological effects of alcohol use on aggression [12, 14, 15], suggesting that it is appropriate to examine associations between alcohol use and IPV perpetration at the same time point. In the context of these limitations related to causal inference, mediation results should be considered with caution [163, 164]. Still, this study contributes important and novel findings on alcohol and IPV reduction interventions targeting an understudied group in a middle-income country.

Results demonstrate that alcohol reduction interventions should be considered an IPV and HIV prevention strategy for men with HIV and hazardous alcohol use. Future research with larger samples should test alternative explanations for intervention effects beyond alcohol use,

assess the cost-effectiveness of alcohol reduction interventions as an IPV reduction strategy, and evaluate enhanced interventions to examine whether effects on IPV perpetration are sustained over time.

#### **CHAPTER 8: DISCUSSION AND CONCLUSION**

The purpose of this dissertation was to examine the longitudinal effects of alcohol use on intimate partner violence (IPV) perpetration, assess whether the strength and direction of the alcohol-IPV relationship differed among those who reported having depressive symptoms versus those who did not, and evaluate whether alcohol reduction interventions decreased IPV perpetration among men with HIV and hazardous alcohol use in Thai Nguyen, Vietnam. The results from this dissertation provide understanding on the nature and magnitude of the relationship between alcohol use and IPV perpetration in an understudied population and setting. The findings also inform development of IPV prevention interventions for men with HIV and hazardous alcohol use and similar populations at high risk for alcohol use, IPV perpetration, and forward HIV transmission.

### 8.1. Summary of Aims and Findings

#### 8.1.1. Paper 1

In the first paper corresponding to the results of Aim 1 (Chapter 5), I estimated the longitudinal effects of alcohol use on IPV perpetration over time. To capture the multiple dimensions of hazardous drinking, alcohol use was measured in three ways: (1) proportion of days alcohol abstinent, (2) number of heavy drinking days (a heavy drinking day was defined as having more than four drinks per day), and (3) alcohol use disorder (AUD; AUD is a "chronic relapsing brain disease characterized by compulsive alcohol use, loss of control over alcohol intake, and a negative emotional state when not using" [121]). For all measures of alcohol use, results showed that reporting greater levels of alcohol use on average was associated with higher odds of IPV perpetration over time. However, time-specific increases in alcohol use were

only associated with increased odds of IPV perpetration for measures of proportion of days alcohol abstinent and AUD, not for heavy drinking. Findings demonstrated that heavy drinking may not be as strong a risk factor for IPV perpetration as compared to proportion of days alcohol abstinent and AUD in this population and setting. This paper provides evidence using longitudinal data that alcohol use is a risk factor for IPV perpetration among men with HIV and hazardous alcohol use.

### 8.1.2. Paper 2

In the second paper corresponding to the results of Aim 2 (Chapter 6), I tested whether depressive symptoms influenced the alcohol-IPV relationship. Unexpectedly, reporting depressive symptoms weakened the alcohol-IPV relationship for measures of proportion of days alcohol abstinent and heavy drinking. Depressive symptoms did not moderate the positive relationship between AUD and IPV perpetration, although a strong main effect of depressive symptoms on IPV perpetration was observed. This paper contributes novel findings that suggest depressive symptoms and alcohol use do not interact to exacerbate IPV perpetration in this population and setting. It also demonstrates that depressive symptoms may be an important risk factor for IPV perpetration to consider when developing IPV prevention interventions for men living with HIV.

### 8.1.3. Paper 3

In the third and final paper corresponding to the results of Aim 3 (Chapter 7), I evaluated the effects of two alcohol reduction interventions (two-session Brief Intervention [BI] and six-session Combined Intervention [CoI]) compared to the standard of care on IPV perpetration and tested alcohol use as a mediator. Results showed that the BI and CoI reduced IPV perpetration at three months compared to the standard of care group, although results were only significant in the BI group. Further, alcohol use did not significantly explain the intervention effects on IPV perpetration, suggesting an alternative pathway for effectiveness. This paper provides promising evidence that alcohol reduction interventions may be an efficient and effective strategy for IPV

prevention in this population and setting. However, as long-term effects were not observed, adapted interventions that incorporate IPV activities should be tested to improve and sustain intervention effects on IPV perpetration.

### 8.2. Key Discussion Points

### 8.2.1. Call for application of a syndemic framework in future work

The findings from this dissertation highlight the importance of applying a syndemic framework in future public health research and practice. Syndemic theory was first conceptualized by Merrill Singer in 1996 in response to the overlapping epidemics of substance abuse, violence, and HIV/AIDS found in urban areas across the United States [81]. Syndemic theory challenges the traditional biomedical approach to disease, in which diseases are considered in isolation, and instead promotes the importance of considering disease interactions [81, 82]. The theory suggests that co-occurring epidemics, such as substance abuse, mental health, violence, and HIV risk, are tied to each other and jointly exacerbate other negative health outcomes [81, 82]. Importantly, syndemic theory posits that epidemics are produced by both disease and social conditions, emphasizing how structural and cultural factors, such as poverty, increase vulnerability to adverse health outcomes [81, 82, 198].

A syndemic framework has typically been applied in research with women and sexual minority populations and focused on IPV victimization and HIV risk [198-201]. However, recent studies with heterosexual men in Uganda and South Africa have utilized a syndemic approach [57, 70, 195, 196]. These studies have found that the relationships between IPV perpetration, alcohol misuse, and HIV risk behavior among heterosexual men are consistent with a syndemic model [57, 70, 195]. Authors termed this a 'gendered syndemic' and posited that gender inequality explains the syndemic model [57, 195], as gender norms dictate that men should demonstrate their masculinity by controlling women and being sexually promiscuous [52, 58-60].

This dissertation research contributes understanding on how to use the syndemic framework to improve IPV efforts among heterosexual men. Based on this dissertation

research, there are three key opportunities for application of a syndemic framework in future IPV research and practice: (1) identifying high-risk populations for IPV research and intervention; (2) understanding the complexity of the alcohol-IPV relationship; and (3) developing and evaluating syndemic-informed interventions.

In this dissertation research, syndemic research was used to form the rationale for investigating the alcohol-IPV relationship among the study population of men with HIV and hazardous alcohol use. As discussed, syndemic research with heterosexual men shows that hazardous alcohol use, depression, IPV perpetration, and sexual risk behaviors co-occur, thereby increasing HIV risk [57, 70, 195, 196]. Further, studies among people living with HIV demonstrate that overlapping syndemic conditions increase risk of poor antiretroviral treatment (ART) adherence, condomless sex, and unsuppressed HIV viral load, all of which are HIV transmission risk factors [67-69]. Taken together, syndemic research demonstrates the importance of framing alcohol and IPV perpetration research among men living with HIV as informing both IPV and HIV prevention efforts. Future IPV research and intervention work should use the syndemic framework to identify groups at high risk for IPV perpetration by considering those who are most vulnerable to the syndemic of substance abuse, HIV risk, mental health, and violence.

Dissertation findings on the relationship between alcohol use, depressive symptoms, and IPV perpetration also point to the importance of applying a syndemic framework in future research and practice. Results demonstrated that depressive symptoms had an influence on the direction of the alcohol-IPV relationship, highlighting the need to consider how syndemic factors, such as hazardous alcohol use and depression, interact to influence negative health outcomes. As other meta-analytic research has demonstrated that the strength of the alcohol-IPV relationship varies across studies [12-14, 78], the syndemic framework offers a strategy for explaining this heterogeneity in effect sizes and understanding the complexity of the alcohol-IPV relationship. In addition to examining the intersection of psychosocial and behavioral factors,

future research should assess how social conditions, such as gender inequality and HIV stigma, influence the syndemic of alcohol use, IPV, mental health, and HIV risk among men living with HIV [57, 81, 82, 198].

Finally, the syndemic framework supports intervention development and evaluation that considers the importance of intervening on the intersection of syndemic factors, rather than on one factor in isolation [57]. Due to the need for efficient and effective interventions in lowresource settings, the syndemic framework offers a promising strategy to intervene on multiple co-occurring public health issues at the same time [57, 81, 82, 198]. In this dissertation research, both alcohol reduction interventions were developed using mental health methodologies (cognitive behavioral therapy/motivational enhancement therapy) and had promising short-term effects on IPV perpetration [138-141]. Although the intervention content did not directly address IPV, both interventions were found to reduce IPV perpetration immediately post-intervention (although only one of the interventions had a significant effect on IPV perpetration). However, as intervention effects were not sustained over time, intervention strategies may need to be adapted to better integrate content targeting each syndemic factor. For example, incorporating IPV-related content, such as behavioral couples therapy, into alcohol reduction interventions may improve and sustain effects on IPV perpetration over time [28, 39, 94]. Future intervention evaluation research should investigate how to integrate mental health and IPV-focused components to optimize alcohol reduction intervention effects on multiple syndemic factors.

# 8.2.2. Intervention implications: feasibility, scalability, and the need for implementation science research

Findings from this dissertation informed the following key recommendations: (1) to screen ART patients for hazardous alcohol use and depressive symptoms; and (2) to link at-risk ART patients to alcohol, IPV, and mental health services in Vietnam. It is critical to understand the feasibility of implementing these recommendations by considering the structural and

individual-level barriers for implementation, potential opportunities for success and scale-up, and guidelines for future research.

Despite promising evidence supporting screening and linking at-risk ART patients to alcohol, IPV, and mental health services [28-31, 39, 92-94, 193, 202], there are numerous barriers to implementation in Vietnam. First, the Vietnamese health system is currently experiencing a structural shift due to reduced funding for HIV services [79, 203, 204]. Previously, the majority of HIV services were funded by external aid from large HIV/AIDS donors, such as the U.S. President's Emergency Plan for AIDS Relief and The Global Fund to Fight AIDS, Tuberculosis and Malaria [79, 203, 204]. Due to the vertical structure of the HIV/AIDS funding streams, HIV services were delivered through a system of free outpatient clinics [204]. However, due to Vietnam's emergence as a middle-income country in 2010, major donors have scaled down aid substantially [79, 203, 204]. To respond, the Vietnamese government has moved to integrate HIV services into public health clinics and commune health stations and to finance HIV services through the social health insurance program [99, 100, 204]. Currently in the midst of this major structural shift, new patient-level barriers to HIV care and treatment, such as cost, stigma, and access, are arising due to inconsistent policies and lack of trained personnel [204]. Adding more complexity, capacity, and resources by incorporating linkages to alcohol, IPV, and mental health services at this time may be burdensome.

In addition to these structural issues, another barrier to implementation is the low availability of alcohol, IPV, and mental health services currently in Vietnam [18, 205, 206]. These services are not prioritized by the government or community, due often to sociocultural norms and limited resources [18, 205, 206]. For hazardous alcohol use, there are limited intervention responses due largely to sociocultural factors [18]. As drinking is highly normative for men in Vietnam [108], there is low awareness around alcohol addiction and alcohol treatment options among men living with HIV, despite the fairly high awareness of and acceptability for opioid addiction treatment (i.e., Methadone Maintenance Treatment [MMT])

[18]. IPV prevention interventions are also sparse in Vietnam due to high acceptability for IPV [206, 207]. Instances of IPV are thought to be private family matters that should only be intervened upon by community-based policing groups when they become severe [38, 111, 208]. Thus, integrating IPV prevention services into HIV care and treatment may be seen as unacceptable by community members. Finally, the dearth of mental health services in Vietnam is driven largely by limited capacity and resources in the health system, as well as stigma and low mental health awareness [205, 209, 210]. Taken together, alcohol, IPV, and mental health services would all be largely novel intervention strategies to introduce in Vietnam. As such, implementing these intervention approaches would likely be challenging due to limited resources and capacity, as well as stigma and low acceptability and awareness.

Despite these numerous barriers, there are potential opportunities for success and scalability of alcohol, IPV, and mental health services in Vietnam. First, the parent study for this dissertation successfully implemented two novel alcohol reduction interventions (BI and CoI) in HIV clinics in Thai Nguyen province. Both the BI and CoI had high intervention uptake, suggesting high acceptability for the alcohol reduction interventions among participants [127]. Importantly, both interventions effectively reduced alcohol use in the short- and long-term and the BI increased viral suppression and was found to be cost-effective [96, 127]. Before implementing both alcohol reduction interventions, extensive efforts were made to tailor evidence-based intervention content for the Vietnamese context [136]. Future IPV and mental health interventions developed for implementation in Vietnam should also focus efforts on cultural adaptation to address potential barriers of stigma and low acceptability and awareness.

Although there are major barriers to delivering psychosocial services and linking to HIV care and treatment, the Vietnamese health system has a proven track record for rapidly implementing and scaling combination HIV prevention interventions [203]. In the last decade, the health system has made major progress in expanding harm reduction interventions, such as MMT and needle and syringe programs [119]. This shift is particularly promising as stigma

towards injection drug use has historically been - and remains - a major issue in Vietnam [79, 80]. Vietnam's ability to shift from punitive drug control policies to funding rights-based approaches suggests that they could make a similarly drastic policy shift regarding the availability of alcohol, IPV, and mental health services [211]. The Vietnamese government has recently expressed increased interest in expanding community-based care for mental health, demonstrating a movement towards more investment in psychosocial services [205].

Finally, implementation science research is needed to evaluate the feasibility, effectiveness, and scalability of implementing alcohol, IPV, and mental health services and linking to HIV care and treatment [79]. In particular, it is important to understand how the new health insurance requirements and integration of HIV services into primary care affect HIV patients, primary care providers, and other relevant stakeholders [79]. This research is critical as it will highlight barriers to engagement in care as well as potential strategies for implementation of psychosocial services and improvements in integrated care. While the restructure of the Vietnamese health system brings implementation challenges, it may also provide unique opportunities for horizontal health systems strengthening [79, 212].

### **APPENDIX A: ADDITIONAL ANALYSES FOR CHAPTER 5**

# LONGITUDINAL ANALYSIS OF ALCOHOL USE AND INTIMATE PARTNER VIOLENCE PERPETRATION AMONG MEN LIVING WITH HIV IN VIETNAM

In Appendix A, results for the following additional analyses are presented:

- 1. Analyses conducted with psychological IPV perpetration as the outcome
- 2. Analyses conducted with physical IPV perpetration as the outcome
- 3. Analyses conducted with sexual IPV perpetration as the outcome
- 4. Analyses conducted with physical/sexual IPV perpetration as the outcome
- 5. Analyses conducted among participants in the standard of care group

The analyses presented in A.1-A.4 examine the effects of alcohol use on different forms of IPV perpetration. These outcome variables were created using the six-item shortened Conflict Tactics Scale 2 (CTS2) [143]. A binary outcome variable was created for each form of IPV (recent psychological IPV perpetration, recent physical IPV perpetration, recent sexual IPV perpetration, and recent physical and/or sexual IPV perpetration). The analyses examining the longitudinal effects of alcohol use on each type of IPV perpetration (A.1-A.4) showed that most significant results observed in the main analyses became non-significant. The models with psychological IPV perpetration as the outcome were the most similar to the main analyses (A.1); this is likely because psychological IPV was the most prevalent form of recent IPV perpetration reported throughout the study. When analyses were restricted to participants in the standard of care group (who reported being married/living with a partner at baseline), results were largely similar to the main analyses (A.5). However, the time-varying and proximal effects of proportion of days alcohol abstinent on IPV perpetration became non-significant. The differences in results between the participants in the standard of care group and the main analyses may be due to a reduced sample size for the standard of care group (N=104) and/or due to alcohol reduction intervention effects influencing results in the main analyses.

## A.1. Analyses conducted with psychological IPV perpetration as the outcome

**Table A.1.** Unconditional growth model (N=313)

|                   | 0 4" - 1 1 (0.5) 0.5                     | (050/ 01) |  |  |  |  |
|-------------------|--|-----------|--|--|--|--|
|                   | Coefficient (SE) or OR (95% CI), p-value |           |  |  |  |  |
| Fixed Effects     |  |           |  |  |  |  |
| Intercept         | -1.37 (0.17)                             | <0.0001   |  |  |  |  |
| Time              | 0.41 (0.30, 0.57)                        | <0.0001   |  |  |  |  |
| Random Effects    |  |           |  |  |  |  |
| Variance Componen | ts                                       |           |  |  |  |  |
| Covariance        | -0.041 (0.32)                            |           |  |  |  |  |
| Variance          | 0.97 (0                                  | .75)      |  |  |  |  |
| Model Fit         |  |           |  |  |  |  |
| AIC               | 1000.                                    | 34        |  |  |  |  |
| BIC               | 1015.33                                  |           |  |  |  |  |
| Neg 2 LL          | 992.3                                    | 34        |  |  |  |  |

Note: SE=Standard error; OR=Odds ratio; CI=Confidence Interval; AIC=Akaike information criterion; BIC=Bayesian information criterion; Neg 2 LL=Negative 2 log likelihood

**Table A.2.** Adjusted conditional growth models<sup>a</sup> (N=313)

|                     | Proportion of days alcohol abstinent      |         | Heavy drin        | Heavy drinking |                    | der (AUD) <sup>b</sup> |  |  |
|---------------------|---|---------|-------------------|----------------|--------------------|------------------------|--|--|
|                     | Coefficient (SE) or aOR (95% CI), p-value |         |                   |                |                    |                        |  |  |
| Fixed Effects       |   |         |                   |                |                    |                        |  |  |
| Intercept           | -1.29 (0.45)                              | 0.004   | -1.29 (0.45)      | 0.004          | -1.62 (0.48)       | 0.001                  |  |  |
| Time                | 0.50 (0.36, 0.67)                         | <0.0001 | 0.46 (0.34, 0.63) | <0.0001        | 0.49 (0.36, 0.67)  | <0.0001                |  |  |
| Alcohol use         |   |         |                   |                |                    |                        |  |  |
| Time-varying        | 0.46 (0.19, 1.09)                         | 0.08    | 0.99 (0.96, 1.02) | 0.49           | 3.39 (1.68, 6.84)  | 0.001                  |  |  |
| Proximal            | 0.39 (0.17, 0.88)                         | 0.02    | 1.04 (1.00, 1.07) | 0.02           | 4.57 (2.08, 10.06) | 0.0002                 |  |  |
| Random Effects      |   |         |                   |                |                    |                        |  |  |
| Variance Components |   |         |                   |                |                    |                        |  |  |
| Covariance          | -0.11 (0.3                                | 31)     | -0.18 (0.3        | -0.18 (0.32)   |                    | :5)                    |  |  |
| Variance            | 0.85 (0.6                                 | 9)      | 0.99 (0.7         | 0.99 (0.73)    |                    | 7)                     |  |  |
| Model Fit           |   |         |                   |                |                    |                        |  |  |
| AIC                 | 971.01                                    |         | 973.25            |                | 947.10             |                        |  |  |
| BIC                 | 1030.95                                   |         | 1033.19           |                | 1007.04            |                        |  |  |
| Neg 2 LL            | 939.01                                    |         | 941.25            |                | 915.10             |                        |  |  |

<sup>&</sup>lt;sup>a</sup>Controlling for education, grand-mean centered age, exposure to violence as a child, employment status, injection drug use, involvement in community violence ever, and intervention arm.

<sup>&</sup>lt;sup>b</sup>A random slope for person-mean cenetered AUD was added to this model as model fit improved when it was included. *Note:* SE=Standard error; aOR=Adjusted odds ratio; CI=Confidence interval; AIC=Akaike information criterion; BIC=Bayesian information criterion; Neg 2 LL=Negative 2 log likelihood

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### A.2. Analyses conducted with physical IPV perpetration as the outcome

**Table A.3.** Unconditional growth model (N=313)

|                  | Coefficient (SE) or OR (95% CI), p-value |         |  |  |  |  |
|------------------|--|---------|--|--|--|--|
| Fixed Effects    |  |         |  |  |  |  |
| Intercept        | -8.24 (0.99)                             | <0.0001 |  |  |  |  |
| Time             | 0.23 (0.067, 0.82)                       | 0.02    |  |  |  |  |
| Random Effects   |  |         |  |  |  |  |
| Variance Compone | nts                                      |         |  |  |  |  |
| Covariance       | -54.53 (34.16)                           |         |  |  |  |  |
| Variance         | 132.43 (7                                | 4.11)   |  |  |  |  |
| Model Fit        |  |         |  |  |  |  |
| AIC              | 233.2                                    | 5       |  |  |  |  |
| BIC              | 248.23                                   |         |  |  |  |  |
| Neg 2 LL         | 225.2                                    | 5       |  |  |  |  |

Note: SE=Standard error; OR=Odds ratio; CI=Confidence interval; AIC=Akaike information criterion; BIC=Bayesian information criterion; Neg 2 LL=Negative 2 log likelihood

**Table A.4.** Adjusted conditional growth models<sup>a,b</sup> (N=313)

|                         | Proportion of days alcohol abstinent |        | Heavy drin              | Heavy drinking |                    | rder (AUD) |  |
|-------------------------|--------------------------------------|--------|-------------------------|----------------|--------------------|------------|--|
|                         |                                      | Co     | efficient (SE) or aOR ( | 95% CI), p-v   | alue               |            |  |
| Fixed Effects           |                                      |        |                         |                |                    |            |  |
| Intercept               | -8.55 (2.30)                         | 0.0002 | -8.35 (2.27)            | 0.0003         | -8.27 (2.29)       | 0.0004     |  |
| Time                    | 0.63 (0.39, 1.02)                    | 0.06   | 0.60 (0.38, 0.95)       | 0.03           | 0.60 (0.38, 0.95)  | 0.03       |  |
| Alcohol use             |                                      |        |                         |                |                    |            |  |
| Time-varying            | 0.27 (0.029, 2.42)                   | 0.24   | 1.03 (0.95, 1.12)       | 0.52           | 1.66 (0.45, 6.10)  | 0.45       |  |
| Proximal                | 0.40 (0.021, 7.57)                   | 0.54   | 1.04 (0.94, 1.14)       | 0.48           | 4.65 (0.45, 48.05) | 0.20       |  |
| Random Effects          |                                      |        |                         |                |                    |            |  |
| Variance Components     |                                      |        |                         |                |                    |            |  |
| Covariance <sup>c</sup> | 8.40                                 |        | 7.58                    |                | 6.29               |            |  |
| Variance                | 14.76 (14.9                          | 90)    | 13.79 (14.              | 13.79 (14.72)  |                    | 70)        |  |
| Model Fit               |                                      |        |                         |                |                    |            |  |
| AIC                     | 248.45                               |        | 271.76                  |                | 270.51             |            |  |
| BIC                     | 308.39                               |        | 331.70                  |                | 330.45             |            |  |
| Neg 2 LL                | 216.45                               | 216.45 |                         | 239.76         |                    | 238.51     |  |

<sup>&</sup>lt;sup>a</sup>Controlling for education, grand-mean centered age, exposure to violence as a child, employment status, injection drug use, involvement in community violence ever, and intervention arm.

Note: SE=Standard error; aOR=Adjusted odds ratio; CI=Confidence interval; AIC=Akaike information criterion; BIC=Bayesian information criterion; Neg 2 LL=Negative 2 log likelihood

<sup>&</sup>lt;sup>b</sup>No random slope for time was included to improve model fit.

<sup>&</sup>lt;sup>c</sup>The standard errors for covariance were not generated although models converged.

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### A.3. Analyses conducted with sexual IPV perpetration as the outcome

**Table A.5.** Unconditional growth model (N=313)

|                  | Coefficient (SE) or OR (95% CI), p-value |         |  |  |  |  |
|------------------|--|---------|--|--|--|--|
| Fixed Effects    |  |         |  |  |  |  |
| Intercept        | -8.90 (0.97)                             | <0.0001 |  |  |  |  |
| Time             | 0.23 (0.058, 0.89)                       | 0.03    |  |  |  |  |
| Random Effects   |  |         |  |  |  |  |
| Variance Compone | nts                                      |         |  |  |  |  |
| Covariance       | -90.62 (5                                | 1.66)   |  |  |  |  |
| Variance         | 222.59 (11                               | 12.40)  |  |  |  |  |
| Model Fit        |  |         |  |  |  |  |
| AIC              | 281.1                                    | 0       |  |  |  |  |
| BIC              | 296.08                                   |         |  |  |  |  |
| Neg 2 LL         | 273.1                                    | 0       |  |  |  |  |

Note: SE=Standard error; OR=Odds ratio; CI=Confidence interval; AIC=Akaike information criterion; BIC=Bayesian information criterion; Neg 2 LL=Negative 2 log likelihood

**Table A.6.** Adjusted conditional growth models<sup>a,b</sup> (N=313)

|                     | Proportion of days alcohol abstinent      |         | Heavy drin        | Heavy drinking |                    | Alcohol use disorder (AUD) |  |  |
|---------------------|---|---------|-------------------|----------------|--------------------|----------------------------|--|--|
|                     | Coefficient (SE) or aOR (95% CI), p-value |         |                   |                |                    |                            |  |  |
| Fixed Effects       |   |         |                   |                |                    |                            |  |  |
| Intercept           | -7.00 (1.64)                              | <0.0001 | -6.29 (1.55)      | <0.0001        | -6.69 (1.59)       | <0.0001                    |  |  |
| Time                | 0.60 (0.40, 0.92)                         | 0.02    | 0.56 (0.38, 0.85) | 0.006          | 0.55 (0.36, 0.83)  | 0.004                      |  |  |
| Alcohol use         |   |         |                   |                |                    |                            |  |  |
| Time-varying        | 0.045 (0.0045,<br>0.44)                   | 0.01    | 1.05 (0.98, 1.13) | 0.16           | 2.80 (0.83, 9.36)  | 0.10                       |  |  |
| Proximal            | 0.26 (0.010, 6.46)                        | 0.41    | 1.06 (0.96, 1.17) | 0.28           | 2.62 (0.18, 37.59) | 0.48                       |  |  |
| Random Effects      |   |         |                   |                |                    |                            |  |  |
| Variance Components |   |         |                   |                |                    |                            |  |  |
| Covariance          | 16.78                                     |         | 13.53             | 13.53          |                    |                            |  |  |
| Variance            | 23.92 (17.8                               | 88)     | 19.78 (15.        | 19.78 (15.22)  |                    | 21.81 (16.50)              |  |  |
| Model Fit           |   |         |                   |                |                    |                            |  |  |
| AIC                 | 327.56                                    |         | 332.63            |                | 332.45             |                            |  |  |
| BIC                 | 387.49                                    |         | 392.57            |                | 392.39             |                            |  |  |
| Neg 2 LL            | 295.56                                    | 295.56  |                   | 300.63         |                    | 300.45                     |  |  |

<sup>&</sup>lt;sup>a</sup>Controlling for education, grand-mean centered age, exposure to violence as a child, employment status, injection drug use, involvement in community violence ever, and intervention arm.

*Note:* SE=Standard error; aOR=Adjusted odds ratio; CI=Confidence interval; AIC=Akaike information criterion; BIC=Bayesian information criterion; Neg 2 LL=Negative 2 log likelihood

<sup>&</sup>lt;sup>b</sup>No random slope for time was included because models did not converge.

<sup>&</sup>lt;sup>c</sup>The standard errors for covariance were not generated although models converged.

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### A.4. Analyses conducted with physical/sexual IPV perpetration as the outcome

**Table A.7.** Unconditional growth model (N=313)

| Coefficient (SE) or OR (95% CI), p-value |  |  |  |  |  |
|--|--|--|--|--|--|
|  |  |  |  |  |  |
| -7.44 (0.78)                             | <0.0001  |  |  |  |  |
| 0.21 (0.077, 0.56)                       | 0.002  |  |  |  |  |
|  |  |  |  |  |  |
| nts                                      |  |  |  |  |  |
| -50.91 (2                                | 5.40)  |  |  |  |  |
| 128.47 (5                                | 6.47)  |  |  |  |  |
|  |  |  |  |  |  |
| 448.5                                    | 3  |  |  |  |  |
| 463.51                                   |  |  |  |  |  |
| 440.53                                   |  |  |  |  |  |
|  | -7.44 (0.78) 0.21 (0.077, 0.56)  nts  -50.91 (29) 128.47 (5) 448.5 463.5 |  |  |  |  |

Note: SE=Standard error; OR=Odds ratio; CI=Confidence interval; AIC=Akaike information criterion; BIC=Bayesian information criterion; Neg 2 LL=Negative 2 log likelihood

**Table A.8.** Adjusted conditional growth models<sup>a,b,c</sup> (N=313)

|                     | Proportion of days alcohol abstinent      |       | Heavy drinking    |       | Alcohol use disorder (AUD) <sup>b</sup> |        |  |
|---------------------|---|-------|-------------------|-------|---|--------|--|
|                     | Coefficient (SE) or aOR (95% CI), p-value |       |                   |       |   |        |  |
| Fixed Effects       |   |       |                   |       |   |        |  |
| Intercept           | -4.62 (1.42)                              | 0.001 | -3.96 (1.15)      | 0.001 | -4.01 (1.09)                            | 0.0003 |  |
| Time                | 0.70 (0.51, 0.94)                         | 0.02  | 0.65 (0.49, 0.87) | 0.004 | 0.66 (0.49, 0.88)                       | 0.004  |  |
| Alcohol use         |   |       |                   |       |   |        |  |
| Time-varying        | 0.16 (0.035, 0.70)                        | 0.02  | 1.03 (0.98, 1.08) | 0.20  | 1.69 (0.75, 3.82)                       | 0.21   |  |
| Proximal            | 0.34 (0.054, 2.13)                        | 0.25  | 1.06 (1.00, 1.12) | 0.06  | 5.10 (1.24, 20.91)                      | 0.02   |  |
| Random Effects      |   |       |                   |       |   |        |  |
| Variance Components |   |       |                   |       |   |        |  |
| Covariance          | 2.29                                      |       | 1.00              |       | 0.64                                    |        |  |
| Variance            | 7.06 (6.53)                               |       | 5.39 (3.84)       |       | 5.21 (3.30)                             |        |  |
| Model Fit           |   |       |                   |       |   |        |  |
| AIC                 | 489.62                                    |       | 491.91            |       | 490.69                                  |        |  |
| BIC                 | 549.56                                    |       | 551.84            |       | 550.63                                  |        |  |
| Neg 2 LL            | 457.62                                    |       | 459.91            |       | 458.69                                  |        |  |

<sup>&</sup>lt;sup>a</sup>Controlling for education, grand-mean centered age, exposure to violence as a child, employment status, injection drug use, involvement in community violence ever, and intervention arm.

Note: SE=Standard error; aOR=Adjusted odds ratio; CI=Confidence interval; AIC=Akaike information criterion; BIC=Bayesian information criterion; Neg 2 LL=Negative 2 log likelihood

<sup>&</sup>lt;sup>b</sup>No random slope for time was included to improve model fit.

<sup>&</sup>lt;sup>c</sup>The standard errors for variance were not generated although models converged.

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## A.5. Analyses conducted with participants in the standard of care group

**Table A.9.** Unconditional growth model (N=104)

|                     | Coefficient (SE) or OR (95% CI), p-value |         |  |  |  |
|---------------------|--|---------|--|--|--|
| Fixed Effects       |  |         |  |  |  |
| Intercept           | -0.96 (0.23)                             | <0.0001 |  |  |  |
| Time                | 0.48 (0.31, 0.76)                        | 0.002   |  |  |  |
| Random Effects      |  |         |  |  |  |
| Variance Components |  |         |  |  |  |
| Covariance          | 0.22 (0.35)                              |         |  |  |  |
| Variance            | 0.34 (0.78)                              |         |  |  |  |
| Model Fit           |  |         |  |  |  |
| AIC                 | 385.34                                   |         |  |  |  |
| BIC                 | 395.92                                   |         |  |  |  |
| Neg 2 LL            | 377.3                                    | 34      |  |  |  |

Note: SE=Standard error; OR=Odds ratio; CI=Confidence interval; AIC=Akaike information criterion; BIC=Bayesian information criterion; Neg 2 LL=Negative 2 log likelihood

**Table A.10.** Adjusted conditional growth models<sup>a</sup> (N=104)

|                     | Proportion of day abstinent |   | Heavy drink       | king  | Alcohol use disord | der (AUD) |  |
|---------------------|-----------------------------|---|-------------------|-------|--------------------|-----------|--|
|                     |                             | Coefficient (SE) or aOR (95% CI), p-value |                   |       |                    |           |  |
| Fixed Effects       |                             |   |                   |       |                    |           |  |
| Intercept           | -1.26 (0.72)                | 0.08                                      | -1.19 (0.67)      | 0.08  | -1.10 (0.65)       | 0.09      |  |
| Time                | 0.43 (0.26, 0.74)           | 0.002                                     | 0.50 (0.31, 0.80) | 0.004 | 0.55 (0.35, 0.89)  | 0.01      |  |
| Alcohol use         |                             |   |                   |       |                    |           |  |
| Time-varying        | 3.15 (0.51, 19.28)          | 0.21                                      | 0.97 (0.91, 1.03) | 0.33  | 3.12 (1.28, 7.59)  | 0.01      |  |
| Proximal            | 0.55 (0.12, 2.66)           | 0.46                                      | 1.06 (1.01, 1.11) | 0.01  | 5.75 (1.69, 19.57) | 0.01      |  |
| Random Effects      |                             |   |                   |       |                    |           |  |
| Variance Components |                             |   |                   |       |                    |           |  |
| Covariance          | -0.32 (0.66                 | 6)  | -0.18 (0.5        | 2)    | -0.14 (0.50        | ))        |  |
| Variance            | 1.47 (1.44                  | l)  | 0.99 (1.13        | 3)    | 0.81 (1.10         | )         |  |
| Model Fit           |                             |   |                   |       |                    |           |  |
| AIC                 | 376.69                      |   | 371.71            |       | 363.92             |           |  |
| BIC                 | 413.72                      |   | 408.73            |       | 400.95             |           |  |
| Neg 2 LL            | 348.69                      |   | 343.71            |       | 335.92             |           |  |

<sup>&</sup>lt;sup>a</sup>Controlling for education, grand-mean centered age, exposure to violence as a child, employment status, injection drug use, involvement in community violence ever, and intervention arm.

<sup>&</sup>lt;sup>b</sup>A random slope for person-mean centered alcohol use was added to this model as model fit improved when it was included. *Note:* SE=Standard error; aOR=Adjusted odds ratio; CI=Confidence interval; AIC=Akaike information criterion; BIC=Bayesian information criterion; Neg 2 LL=Negative 2 log likelihood

#### **APPENDIX B: ADDITIONAL ANALYSES FOR CHAPTER 6**

# ALCOHOL USE, DEPRESSIVE SYMPTOMS, AND INTIMATE PARTNER VIOLENCE PERPETRATION: A LONGITUDINAL ANALYSIS AMONG MEN LIVING WITH HIV IN NORTHERN VIETNAM

In Appendix B, results for the following additional analyses are presented:

- Analyses conducted with a categorical depressive symptoms variable (none, minor symptoms, severe symptoms)
- 2. Analyses conducted with psychological IPV perpetration as the outcome
- 3. Analyses conducted with physical IPV perpetration as the outcome
- 4. Analyses conducted with sexual IPV perpetration as the outcome
- 5. Analyses conducted with physical/sexual IPV perpetration as the outcome
- 6. Analyses conducted among participants in the standard of care group

The analyses presented in B.1 replace the binary variable for depressive symptoms (symptoms are not present versus present) with a categorical variable (no symptoms, minor symptoms, severe symptoms). The categorical variable was derived from the same measure used to create the binary one, the Patient Health Questionnaire-9 (PHQ-9) [150, 151, 213]. Based on standard scoring guidelines, those who scored zero to four on the PHQ-9 were categorized as having no symptoms, those who scored five to nine were categorized as having minor symptoms, and those who scored 10 or above were categorized as having severe symptoms [150, 151, 213]. In analyses, the categorical depressive symptoms variable only significantly moderated the relationship between proportion of days alcohol abstinent and IPV perpetration (B.1). The moderation graph demonstrated a similar pattern of moderation as shown in the main analyses, with greater depressive symptoms weakening the alcohol-IPV relationship. However, only the associations between proportion of days alcohol abstinent and IPV perpetration for those reporting not having depressive symptoms and those reporting having severe depressive

symptoms were significant. In the models with heavy drinking and alcohol use disorder (AUD), no moderation was observed; however, at times when participants reported severe or moderate depressive symptoms, the odds of IPV perpetration increased when compared to those who reported no depressive symptoms (B.1). The analyses presented in B.2-B.5 examine whether alcohol use and depressive symptoms interact to influence different forms of IPV perpetration. These outcome variables were created using the six-item shortened Conflict Tactics Scale 2 (CTS2) [143]. The analyses investigating whether depressive symptoms influence the effects of alcohol use on each type of IPV perpetration (B.2-B.5) and the analyses restricted to those in the standard of care group (B.6) showed that most significant results observed in the main analyses became non-significant. However, depressive symptoms remained a significant risk factor for IPV perpetration across all models. Analyses with psychological IPV perpetration as the outcome were the most similar to the main analyses; moderation was only observed in the analyses with psychological IPV perpetration as the outcome (B.2). This is likely because psychological IPV was the most prevalent form of recent IPV perpetration reported across the study.

#### B.1. Analyses conducted with categorical depressive symptoms variable (none, minor symptoms, severe symptoms)

**Table A.11.** Adjusted conditional growth models by alcohol measure<sup>a</sup> (N=313)

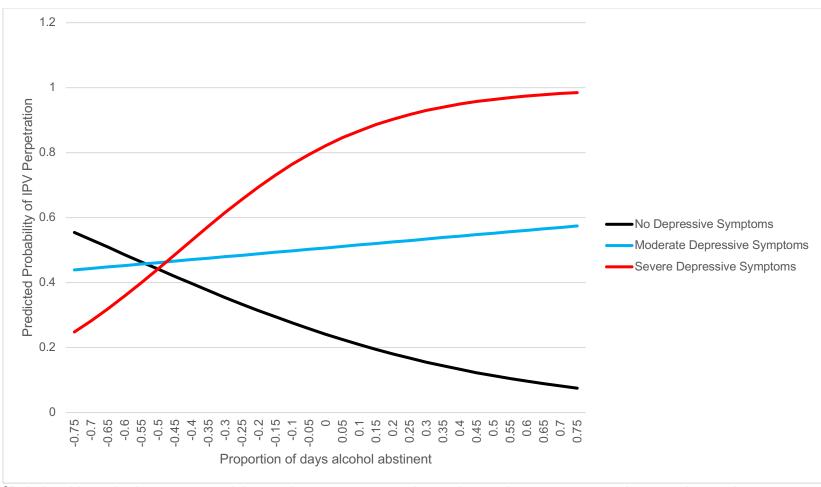
|                                 | Proportion of days abstinent | alcohol | Heavy drin             | king         | Alcohol use disor      | der (AUD) |
|---------------------------------|------------------------------|---------|------------------------|--------------|------------------------|-----------|
|                                 |                              | Coeffi  | icient (SE) or aOR (9  | 5% CI), p-va | lue                    |           |
| Fixed Effects                   |                              |         |                        |              |                        |           |
| Intercept                       | -1.15 (0.44)                 | 0.01    | -0.97 (0.42)           | 0.02         | -1.07 (0.44)           | 0.01      |
| Time                            | 0.54 (0.40, 0.72)            | <0.0001 | 0.50 (0.37, 0.67)      | <0.0001      | 0.52 (0.38, 0.69)      | <0.0001   |
| Alcohol use                     | 0.16 (0.061, 0.43)           | 0.0003  | 1.00 (0.97, 1.03)      | 0.91         | 2.58 (1.50, 4.44)      | 0.0006    |
| Depressive symptoms             |                              |         |                        |              |                        |           |
| Moderate vs none                | 3.24 (1.72, 6.08)            | 0.0003  | 3.02 (1.62, 5.62)      | 0.0005       | 2.77 (1.48, 5.21)      | 0.002     |
| Severe vs none                  | 14.49 (4.12, 50.95)          | <0.0001 | 12.59 (3.74,<br>42.33) | <0.0001      | 10.38 (3.02,<br>35.74) | 0.0002    |
| Alcohol use*Depressive symptoms |                              |         | ,                      |              | ,                      |           |
| Moderate vs none                | 8.88 (0.89, 88.48)           | 0.06    |                        |              |                        |           |
| Severe vs none                  | 207.47 (6.86,<br>6276.78)    | 0.002   |                        |              |                        |           |
| Random Effects                  | ,                            |         |                        |              |                        |           |
| Variance Components             |                              |         |                        |              |                        |           |
| Covariance                      | -0.014 (0.27)                | )       | -0.065 (0.2            | 28)          | -0.058 (0.2            | 9)        |
| Variance                        | 0.62 (0.61)                  |         | 0.70 (0.63             | 3)           | 0.73 (0.66             | i)        |
| Model Fit                       |                              |         |                        |              |                        |           |
| AIC                             | 1013.35                      |         | 1028.29                |              | 1015.96                |           |
| BIC                             | 1092.02                      |         | 1099.47                | ,            | 1087.13                |           |
| Neg 2 LL                        | 971.35                       |         | 990.29                 |              | 977.96                 |           |

<sup>&</sup>lt;sup>a</sup>Controlling for education, grand-mean centered age, exposure to violence as a child, employment status, injection drug use, involvement in community violence ever, and intervention arm.

**Table A.12.** Simple intercepts and slopes, proportion of days alcohol abstinent\*depressive symptoms

|                              | Intercept    |         | Slope        |         |  |
|------------------------------|--------------|---------|--------------|---------|--|
|                              | b (SE)       | p-value | b (SE)       | p-value |  |
| No depressive symptoms       | -1.15 (0.44) | 0.01    | -1.82 (0.50) | 0.0003  |  |
| Moderate depressive symptoms | 0.027 (0.53) | 0.96    | 0.36 (1.06)  | 0.73    |  |
| Severe depressive symptoms   | 1.53 (0.77)  | 0.05    | 3.51 (1.64)  | 0.03    |  |

Note: SE=Standard error



<sup>a</sup>Relationship probed at no reported depressive symptoms, moderate depressive symptoms, and severe depressive symptoms.

**Figure A.1.** Estimated IPV perpetration as a function of depressive symptoms and proportion of days alcohol abstinent at times when participants reported no, moderate, and severe depressive symptoms

# B.2. Analyses with psychological IPV perpetration as the outcome variable

**Table A.13.** Adjusted conditional growth model by alcohol measure<sup>a</sup> (N=313)

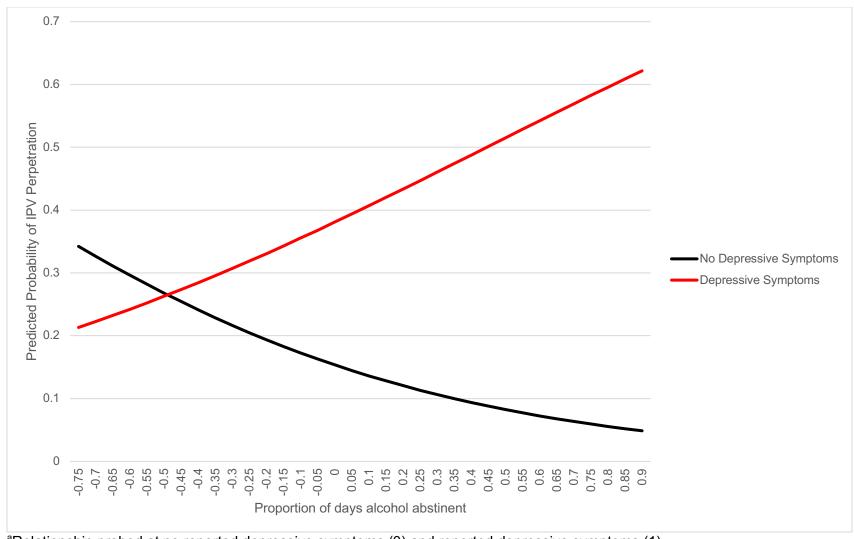
|                                 | Proportion of day abstinent |         | Heavy drink            | king        | Alcohol use diso  | rder (AUD) |
|---------------------------------|-----------------------------|---------|------------------------|-------------|-------------------|------------|
|                                 |                             | C       | Coefficient (SE) or aO | R (95% CI), | p-value           |            |
| Fixed Effects                   |                             |         |                        |             |                   |            |
| Intercept                       | -1.71 (0.46)                | 0.0002  | -1.57 (0.45)           | 0.001       | -1.67 (0.46)      | 0.0003     |
| Time                            | 0.52 (0.38, 0.71)           | <0.0001 | 0.48 (0.35, 0.66)      | <0.0001     | 0.51 (0.37, 0.70) | <0.0001    |
| Alcohol use                     | 0.25 (0.089, 0.68)          | 0.007   | 0.99 (0.96, 1.03)      | 0.74        | 2.58 (1.47, 4.52) | 0.001      |
| Depressive symptoms             | 3.39 (1.83, 6.29)           | 0.0001  | 3.19 (1.72, 5.92)      | 0.0002      | 2.87 (1.54, 5.36) | 0.001      |
| Alcohol use*Depressive symptoms | 12.15 (1.62,<br>91.19)      | 0.02    |                        |             |                   |            |
| Random Effects                  | ,                           |         |                        |             |                   |            |
| Variance Components             |                             |         |                        |             |                   |            |
| Covariance                      | -0.037 (0.3                 | 0)      | -0.12 (0.32            | 2)          | -0.075 (0.        | 32)        |
| Variance                        | 0.67 (0.69                  | )       | 0.83 (0.73             | 3)          | 0.78 (0.7         | (3)        |
| Model Fit                       |                             |         |                        |             |                   |            |
| AIC                             | 949.42                      |         | 956.38                 |             | 945.07            | ,          |
| BIC                             | 1016.86                     |         | 1020.07                |             | 1008.7            | 5          |
| Neg 2 LL                        | 913.42                      |         | 922.38                 |             | 911.07            | •          |

<sup>&</sup>lt;sup>a</sup>Controlling for education, grand-mean centered age, exposure to violence as a child, employment status, injection drug use, involvement in community violence ever, and intervention arm.

**Table A.14.** Simple intercepts and slopes, proportion of days alcohol abstinent\*depressive symptoms

|                        | Interc       | Intercept |              | pe      |  |  |
|------------------------|--------------|-----------|--------------|---------|--|--|
|                        | b (SE)       | p-value   | b (SE)       | p-value |  |  |
| No Depressive Symptoms | -1.71 (0.46) | 0.0002    | -1.40 (0.52) | 0.007   |  |  |
| Depressive Symptoms    | -0.49 (0.54) | 0.37      | 1.09 (0.88)  | 0.22    |  |  |

Note: SE=Standard error



<sup>&</sup>lt;sup>a</sup>Relationship probed at no reported depressive symptoms (0) and reported depressive symptoms (1).

**Figure A.2.** Estimated IPV perpetration as a function of depressive symptoms and proportion of days alcohol abstinent at times participants screened positive or negative for depressive symptoms<sup>a</sup>

### B.3. Analyses with physical IPV perpetration as the outcome variable

**Table A.15.** Adjusted conditional growth model by alcohol measure<sup>a-c</sup> (N=313)

|                                 | Proportion of days abstinent | alcohol | Heavy drinkir              | ng           | Alcohol use disord   | er (AUD) |
|---------------------------------|------------------------------|---------|----------------------------|--------------|----------------------|----------|
|                                 |                              | С       | pefficient (SE) or aOR (95 | 5% CI), p-va | alue                 |          |
| Fixed Effects                   |                              |         |                            |              |                      |          |
| Intercept                       | -11.29 (3.00)                | 0.0002  | -11.80 (3.15)              | 0.0002       | -10.86 (2.83)        | 0.0002   |
| Time                            | 0.44 (0.23, 0.83)            | 0.01    | 0.41 (0.22, 0.78)          | 0.007        | 0.39 (0.21, 0.74)    | 0.004    |
| Alcohol use                     | 0.12 (0.0084, 1.75)          | 0.12    | 1.10 (0.99, 1.23)          | 0.07         | 1.07 (0.22, 5.21)    | 0.94     |
| Depressive symptoms             | 61.60 (8.60, 441.06)         | <0.0001 | 90.58 (10.30, 796.88)      | <0.0001      | 50.52 (7.41, 344.47) | <0.0001  |
| Alcohol use*Depressive symptoms |                              |         |                            |              |                      |          |
| Random Effects                  |                              |         |                            |              |                      |          |
| Variance Components             |                              |         |                            |              |                      |          |
| Covariance                      | 21.61                        |         | 24.51                      |              | 18.76                |          |
| Variance                        | 27.69 (27.50                 | )       | 30.62 (30.97)              | )            | 24.83 (24.79         | ))       |
| Model Fit                       |                              |         |                            |              |                      |          |
| AIC                             | 245.77                       |         | 244.80                     |              | 248.28               |          |
| BIC                             | 309.46                       |         | 308.48                     |              | 311.96               |          |
| Neg 2 LL                        | 211.77                       |         | 210.80                     |              | 214.28               |          |

<sup>&</sup>lt;sup>a</sup>Controlling for education, grand-mean centered age, exposure to violence as a child, employment status, injection drug use, involvement in community violence ever, and intervention arm. <sup>b</sup>No random slope for time was included because models did not converge. <sup>c</sup>The standard errors for variance were not generated although models converged.

#### B.4. Analyses with sexual IPV perpetration as the outcome variable

**Table A.16.** Adjusted conditional growth model by alcohol measure<sup>a-c</sup> (N=313)

|                                 | Proportion of days abstinent | alcohol | Heavy drink               | ing           | Alcohol use disord | der (AUD) |
|---------------------------------|------------------------------|---------|---------------------------|---------------|--------------------|-----------|
|                                 |                              | Сс      | pefficient (SE) or aOR (9 | 95% CI), p-va | alue               |           |
| Fixed Effects                   |                              |         |                           |               |                    |           |
| Intercept                       | -7.79 (1.77)                 | <0.0001 | -7.17 (1.68)              | <0.0001       | -7.17 (1.67)       | <0.0001   |
| Time                            | 0.59 (0.38, 0.92)            | 0.02    | 0.55 (0.36, 0.85)         | 0.007         | 0.53 (0.34, 0.82)  | 0.004     |
| Alcohol use                     | 0.033 (0.0028, 0.38)         | 0.006   | 1.07 (0.99, 1.16)         | 0.07          | 2.02 (0.57, 7.12)  | 0.27      |
| Depressive symptoms             | 12.58 (2.97, 53.30)          | 0.0006  | 11.96 (2.96, 48.36)       | 0.0005        | 9.17 (2.30, 36.57) | 0.002     |
| Alcohol use*Depressive symptoms |                              |         |                           |               |                    |           |
| Random Effects                  |                              |         |                           |               |                    |           |
| Variance Components             |                              |         |                           |               |                    |           |
| Covariance                      | 21.37                        |         | 17.42                     |               | 17.16              |           |
| Variance                        | 28.55 (21.52                 | )       | 24.51 (18.10              | 6)            | 24.22 (18.0        | 4)        |
| Model Fit                       |                              |         |                           |               |                    |           |
| AIC                             | 316.20                       |         | 321.40                    |               | 323.64             |           |
| BIC                             | 379.88                       |         | 385.08                    |               | 387.33             |           |
| Neg 2 LL                        | 282.20                       |         | 287.40                    |               | 289.64             |           |

<sup>&</sup>lt;sup>a</sup>Controlling for education, grand-mean centered age, exposure to violence as a child, employment status, injection drug use, involvement in community violence ever, and intervention arm. <sup>b</sup>No random slope for time was included because models did not converge. <sup>c</sup>The standard errors for variance were not generated although models converged.

### B.5. Analyses with physical/sexual IPV perpetration as the outcome variable

**Table A.17.** Adjusted conditional growth model by alcohol measure<sup>a-c</sup> (N=313)

|                                 | Proportion of days abstinent | alcohol | Heavy drinki             | ng            | Alcohol use disord  | er (AUD) |
|---------------------------------|------------------------------|---------|--------------------------|---------------|---------------------|----------|
|                                 |                              | Сс      | efficient (SE) or aOR (9 | 95% CI), p-va | alue                |          |
| Fixed Effects                   |                              |         |                          |               |                     |          |
| Intercept                       | -6.61 (1.67)                 | <0.0001 | -6.17 (1.71)             | 0.0004        | -5.79 (1.70)        | 0.001    |
| Time                            | 0.62 (0.43, 0.89)            | 0.01    | 0.58 (0.40, 0.82)        | 0.002         | 0.56 (0.39, 0.81)   | 0.002    |
| Alcohol use                     | 0.084 (0.014, 0.49)          | 0.006   | 1.07 (1.00, 1.14)        | 0.04          | 1.29 (0.49, 3.39)   | 0.61     |
| Depressive symptoms             | 20.87 (5.77, 75.46)          | <0.0001 | 19.52 (5.41, 70.44)      | <0.0001       | 14.67 (4.24, 50.79) | <0.0001  |
| Alcohol use*Depressive symptoms |                              |         |                          |               |                     |          |
| Random Effects                  |                              |         |                          |               |                     |          |
| Variance Components             |                              |         |                          |               |                     |          |
| Covariance                      | 8.09                         |         | 6.50                     |               | 5.01                |          |
| Variance                        | 13.01 (12.70                 | )       | 11.37 (11.82             | 2)            | 9.83 (10.73         | )        |
| Model Fit                       |                              |         |                          |               |                     |          |
| AIC                             | 456.48                       |         | 460.21                   |               | 464.66              |          |
| BIC                             | 520.17                       |         | 523.89                   |               | 528.34              |          |
| Neg 2 LL                        | 422.48                       |         | 426.21                   |               | 430.66              |          |

<sup>&</sup>lt;sup>a</sup>Controlling for education, grand-mean centered age, exposure to violence as a child, employment status, injection drug use, involvement in community violence ever, and intervention arm. <sup>b</sup>No random slope for time was included because models did not converge. <sup>c</sup>The standard errors for variance were not generated although models converged.

## B.6. Analyses conducted with participants in the standard of care group

**Table A.18.** Adjusted conditional growth model by alcohol measure<sup>a,b</sup> (N=104)

|                                 | Proportion of days abstinent | alcohol | Heavy drinki              | ng          | Alcohol use disord | er (AUD) |
|---------------------------------|------------------------------|---------|---------------------------|-------------|--------------------|----------|
|                                 |                              | Сс      | pefficient (SE) or aOR (9 | 5% CI), p-v | alue               |          |
| Fixed Effects                   |                              |         |                           |             |                    |          |
| Intercept                       | -1.73 (0.70)                 | 0.02    | -1.76 (0.70)              | 0.01        | -1.79 (0.71)       | 0.01     |
| Time                            | 0.50 (0.30, 0.83)            | 0.008   | 0.52 (0.31, 0.86)         | 0.01        | 0.54 (0.32, 0.90)  | 0.02     |
| Alcohol use                     | 2.40 (0.50, 11.45)           | 0.27    | 0.98 (0.91, 1.05)         | 0.50        | 3.08 (1.19, 7.99)  | 0.02     |
| Depressive symptoms             | 4.92 (1.67, 14.50)           | 0.004   | 4.77 (1.62, 14.10)        | 0.005       | 4.75 (1.57, 14.41) | 0.006    |
| Alcohol use*Depressive symptoms |                              |         |                           |             |                    |          |
| Random Effects                  |                              |         |                           |             |                    |          |
| Variance Components             |                              |         |                           |             |                    |          |
| Covariance                      | -0.21 (0.54)                 |         | -0.23 (0.55)              |             | -0.18 (0.57)       |          |
| Variance                        | 0.99 (1.21)                  |         | 1.03 (1.25)               |             | 0.97 (1.30)        |          |
| Model Fit                       |                              |         |                           |             |                    |          |
| AIC                             | 359.34                       |         | 360.13                    |             | 354.80             |          |
| BIC                             | 399.01                       |         | 399.79                    |             | 394.47             |          |
| Neg 2 LL                        | 329.34                       |         | 330.13                    |             | 324.80             |          |

<sup>&</sup>lt;sup>a</sup>Controlling for education, grand-mean centered age, exposure to violence as a child, employment status, injection drug use, involvement in community violence ever, and intervention arm. <sup>b</sup>Interaction terms were non-significant in the model, leaving the adjusted conditional growth model as the final model.

#### **APPENDIX C: ADDITIONAL ANALYSES FOR CHAPTER 7**

EVALUATING THE EFFECTS OF TWO ALCOHOL REDUCTION INTERVENTIONS ON INTIMATE PARTNER VIOLENCE PERPETRATION AND TESTING ALCOHOL USE AS A PATHWAY FOR EFFECTIVENESS: RESULTS FROM A THREE-ARM RANDOMIZED CONTROLLED TRIAL AMONG MEN LIVING WITH HIV IN NORTHERN VIETNAM

In Appendix C, results for the following additional analyses are presented:

- Analyses examining intervention effects conducted with complete cases across all study visits
- 2. Analyses conducted with those who reported being married/living with a partner at baseline
- Analyses conducted with 20 imputed datasets excluding those who were incarcerated and/or died during the study (N=27/426)

The analyses with complete cases across all study visits (C.1) and the analyses conducted with those who reported being married/living with a partner at baseline (C.2) had no substantive differences from the main analyses. However, the indirect effect for the CoI on IPV perpetration through AUD was significant when the sample was restricted to men who were married/living with a partner at baseline. This suggests that the CoI effectively reduced IPV perpetration among partnered men by intervening on a pattern of severe hazardous drinking. The analyses with imputed datasets were also similar to the main analyses, demonstrating that missingness did not substantially influence results (C.3). In the analyses with imputed datasets, the total effects of the CoI on IPV perpetration calculated as part of the mediation analyses were significant across all models. Further, the natural direct effect of CoI on IPV perpetration while blocking the pathway for heavy drinking was significant. These analyses with imputed datasets suggest that there were significant effects of the CoI on IPV perpetration at three months, although they confirm that alcohol use was not a significant mediator. Notably, bootstrapping

could not be utilized due to software limitations, reducing the accuracy of the mediation results using imputed data.

#### C.1. Analyses examining intervention effects conducted with complete cases across all study visits

**Table A.19.** Complete case analysis, Intervention effects on intimate partner violence (IPV) perpetration<sup>a</sup> (N=346)

|                                     | 3 m               | onths             | 6 mc              | onths             | 12 m              | onths             |  |
|-------------------------------------|-------------------|-------------------|-------------------|-------------------|-------------------|-------------------|--|
|                                     | Brief             | Combined          | Brief             | Combined          | Brief Combined    |                   |  |
|                                     |                   |                   | aOR (9            | 95% CI)           |                   |                   |  |
| Any form of recent IPV perpetration | 0.34 (0.14, 0.86) | 0.48 (0.20, 1.14) | 0.81 (0.37, 1.79) | 0.87 (0.40, 1.86) | 0.92 (0.40, 2.09) | 0.87 (0.38, 1.97) |  |

<sup>&</sup>lt;sup>a</sup>Controlling for age, education, IPV perpetration (at previous time point), partnership status (at previous time point), employment status (at previous time point), injection drug use (at previous time point), exposure to violence as a child, and involvement in community violence ever (at previous time point).

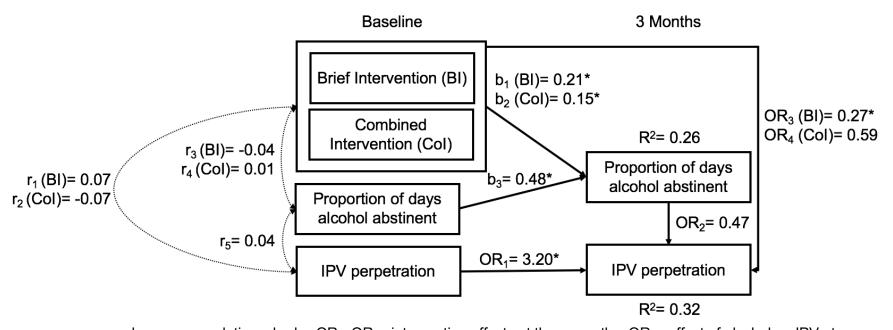
Note: Estimates in bold are significant at p<0.05. aOR=Adjusted odds ratio; CI=Confidence Interval; IPV=Intimate partner violence

#### C.2. Analyses conducted with participants who reported being married/living with a partner at baseline

Table A.20. Intervention effects on intimate partner violence (IPV) perpetration

|                                     | 3 months          | s (N=286)         | 6 months          | s (N=274)         | 12 month          | s (N=275)         |
|-------------------------------------|-------------------|-------------------|-------------------|-------------------|-------------------|-------------------|
|                                     | Brief             | Combined          | Brief             | Combined          | Brief             | Combined          |
|                                     |                   |                   | aOR (9            | 95% CI)           |                   |                   |
| Any form of recent IPV perpetration | 0.24 (0.09, 0.62) | 0.52 (0.23, 1.20) | 1.11 (0.48, 2.57) | 1.11 (0.49, 2.52) | 0.80 (0.33, 1.97) | 0.91 (0.39, 2.10) |

<sup>&</sup>lt;sup>a</sup>Controlling for age, education, IPV perpetration (at previous time point), employment status (at previous time point), injection drug use (at previous time point), exposure to violence as a child, and involvement in community violence ever (at previous time point). *Note:* Estimates in bold are significant at p<0.05. aOR=Adjusted odds ratio; CI=Confidence interval; IPV=Intimate partner violence

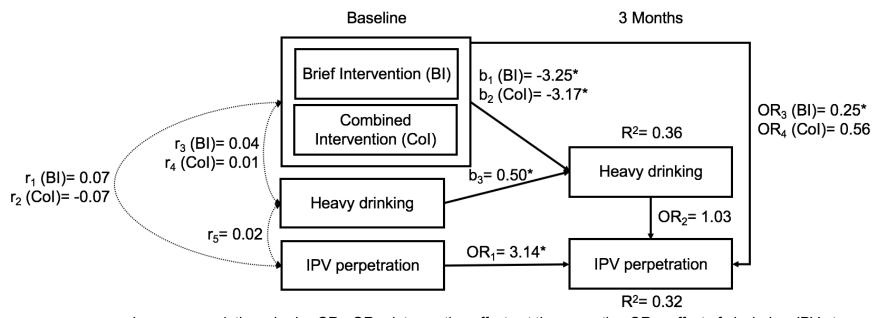


 $r_1$ ,  $r_2$ ,  $r_3$ ,  $r_4$ ,  $r_5$ = synchronous correlations;  $b_1$ ,  $b_2$ ,  $OR_3$ ,  $OR_4$ = intervention effects at three months;  $OR_2$ = effect of alcohol on IPV at three months;  $b_3$ ,  $OR_1$ = autoregressive (AR) pathways;  $R^2$ = variance explained. \*p<0.05

<sup>a</sup>Controlling for age, education, baseline employment status, baseline injection drug use, exposure to violence as a child, and baseline involvement in community violence ever.

Note: IPV=Intimate partner violence; OR=Odds ratio

Figure A.3. Path analysis, proportion of days alcohol abstinent<sup>a</sup> (N=286)

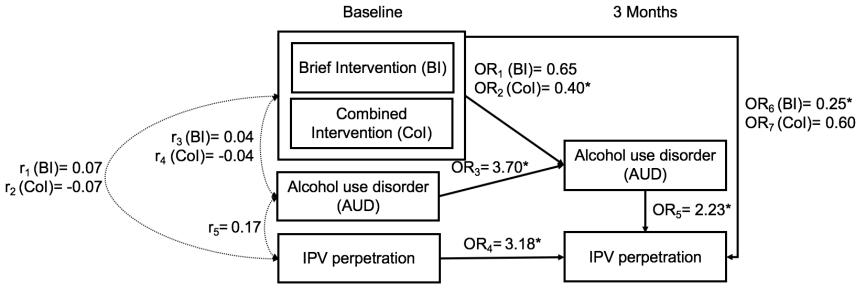


 $r_1$ ,  $r_2$ ,  $r_3$ ,  $r_4$ ,  $r_5$ = synchronous correlations;  $b_1$ ,  $b_2$ ,  $OR_3$ ,  $OR_4$ = intervention effects at three months;  $OR_2$ = effect of alcohol on IPV at three months;  $b_3$ ,  $OR_1$ = autoregressive (AR) pathways;  $R^2$ = variance explained. \*p<0.05; \*\*p<0.01

<sup>a</sup>Controlling for age, education, baseline employment status, baseline injection drug use, exposure to violence as a child, and baseline involvement in community violence ever.

*Note:* IPV= Intimate partner violence; OR= Odds Ratio.

Figure A.4. Path analysis, number of heavy drinking days<sup>a</sup> (N=286)



 $r_1$ ,  $r_2$ ,  $r_3$ ,  $r_4$ ,  $r_5$ = synchronous correlations; OR<sub>1</sub>, OR<sub>2</sub>, OR<sub>6</sub>, OR<sub>7</sub>= intervention effects at three months; OR<sub>5</sub>= effect of alcohol on IPV at three months; OR<sub>3</sub>, OR<sub>4</sub>= autoregressive (AR) pathways. \*p<0.05

<sup>a</sup>Controlling for age, education, baseline employment status, baseline injection drug use, exposure to violence as a child, and baseline involvement in community violence ever.

Note: R<sup>2</sup> could not be calculated by MPlus8 due to the binary mediator. IPV= Intimate partner violence; OR= Odds Ratio

Figure A.5. Path analysis, alcohol use disorder (AUD)<sup>a</sup> (N=286)

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**Table A.21.** Mediation results by alcohol measure<sup>a,b</sup> (N=286)

|  | Proportion of days alcohol abstinent |      | Heavy drinking    |      | Alcohol use disorder (AUD |      |
|--|--------------------------------------|------|-------------------|------|---------------------------|------|
|  |                                      |      | OR (95% CI), S    | SE . |                           |      |
| Effect of BI on IPV through alcohol use  |                                      |      |                   |      |                           |      |
| Natural indirect effect                  | 0.86 (0.65, 1.08)                    | 0.11 | 0.91 (0.78, 1.05) | 0.07 | 0.94 (0.79, 1.03)         | 0.06 |
| Natural direct effect                    | 0.28 (0.07, 0.73)                    | 0.18 | 0.26 (0.07, 0.66) | 0.16 | 0.26 (0.07, 0.64)         | 0.16 |
| Total effect                             | 0.24 (0.06, 0.61)                    | 0.15 | 0.24 (0.06, 0.59) | 0.14 | 0.24 (0.07, 0.60)         | 0.15 |
| Effect of Col on IPV through alcohol use |                                      |      |                   |      |                           |      |
| Natural indirect effect                  | 0.89 (0.71, 1.06)                    | 0.09 | 0.91 (0.77, 1.05) | 0.07 | 0.90 (0.72, 0.99)         | 0.07 |
| Natural direct effect                    | 0.59 (0.21, 1.51)                    | 0.34 | 0.56 (0.21, 1.42) | 0.33 | 0.61 (0.22, 1.51)         | 0.35 |
| Total effect                             | 0.53 (0.20, 1.27)                    | 0.29 | 0.51 (0.19, 1.26) | 0.28 | 0.54 (0.20, 1.33)         | 0.30 |

<sup>&</sup>lt;sup>a</sup>As causally-defined mediation effects are computed differently than effect estimates in the path analyses, total effect and natural direct effect estimates may differ slightly than those shown in the path analyses.

*Note:* Estimates in bold are significant at p<0.05. OR=Odds ratio; CI=Confidence interval; SE=Standard error; BI=Brief Intervention; CoI=Combined Intervention; IPV=Intimate partner violence

<sup>&</sup>lt;sup>b</sup>Controlling for age, education, baseline IPV perpetration, baseline employment status, baseline injection drug use, exposure to violence as a child, and baseline involvement in community violence ever.

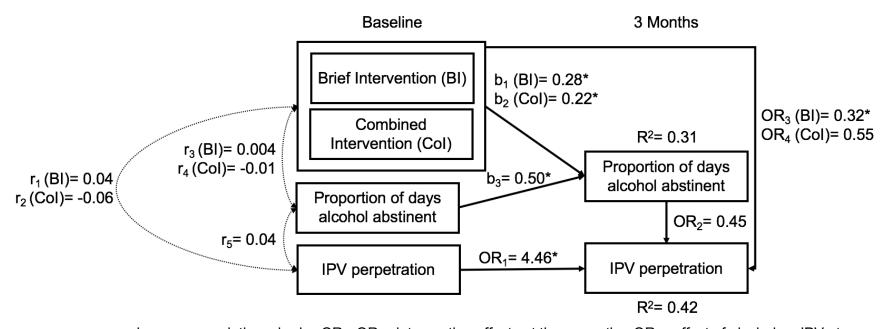
# C.3. Analyses conducted with 20 imputed datasets excluding those who were incarcerated and/or died during the study (N=27/426)

**Table A.22.** Intervention effects on intimate partner violence (IPV) perpetration (N=399)

| -                                   | 3 months          |                   | 6 months          |                   | 12 months         |                   |  |  |  |  |
|-------------------------------------|-------------------|-------------------|-------------------|-------------------|-------------------|-------------------|--|--|--|--|
|                                     | Brief             | Combined          | Brief             | Combined          | Brief             | Combined          |  |  |  |  |
|                                     | aOR (95% CI)      |                   |                   |                   |                   |                   |  |  |  |  |
| Any form of recent IPV perpetration | 0.29 (0.12, 0.71) | 0.49 (0.22, 1.12) | 0.94 (0.44, 2.03) | 0.95 (0.45, 2.01) | 0.99 (0.44, 2.22) | 0.85 (0.38, 1.91) |  |  |  |  |

<sup>&</sup>lt;sup>a</sup>Controlling for age, education, IPV perpetration (at previous time point) partnership status (at previous time point), employment status (at previous time point), injection drug use (at previous time point), exposure to violence as a child, and involvement in community violence ever (at previous time point).

Note: Estimates in bold are significant at p<0.05. aOR=Adjusted odds ratio; CI=Confidence Interval; IPV=Intimate partner violence

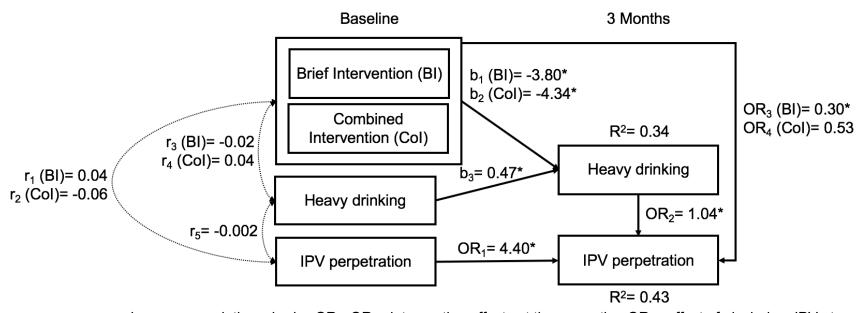


 $r_1$ ,  $r_2$ ,  $r_3$ ,  $r_4$ ,  $r_5$ = synchronous correlations;  $b_1$ ,  $b_2$ ,  $OR_3$ ,  $OR_4$ = intervention effects at three months;  $OR_2$ = effect of alcohol on IPV at three months;  $b_3$ ,  $OR_1$ = autoregressive (AR) pathways;  $R^2$ = variance explained. \*p<0.05

<sup>a</sup>Controlling for age, education, baseline partnership status, baseline employment status, baseline injection drug use, exposure to violence as a child, and baseline involvement in community violence ever.

Note: IPV= Intimate partner violence; OR= Odds ratio

Figure A.6. Path analysis, proportion of days alcohol abstinent<sup>a</sup> (N=399)

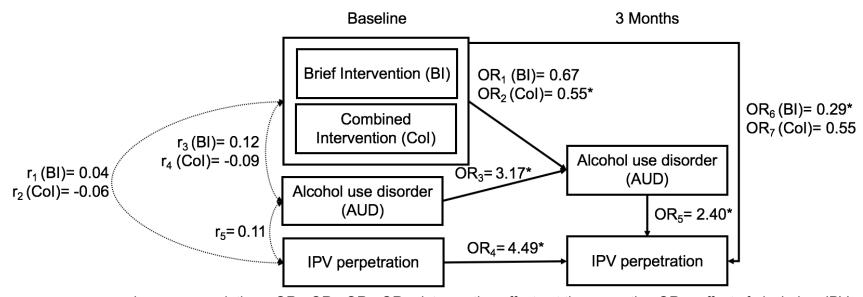


 $r_1$ ,  $r_2$ ,  $r_3$ ,  $r_4$ ,  $r_5$ = synchronous correlations;  $b_1$ ,  $b_2$ ,  $OR_3$ ,  $OR_4$ = intervention effects at three months;  $OR_2$ = effect of alcohol on IPV at three months;  $b_3$ ,  $OR_1$ = autoregressive (AR) pathways;  $R^2$ = variance explained. \*p<0.05; \*\*p<0.01

<sup>a</sup>Controlling for age, education, baseline partnership status, baseline employment status, baseline injection drug use, exposure to violence as a child, and baseline involvement in community violence ever.

Note: IPV= Intimate partner violence; OR= Odds ratio

**Figure A.7.** Path analysis, number of heavy drinking days<sup>a</sup> (N=399)



 $r_1$ ,  $r_2$ ,  $r_3$ ,  $r_4$ ,  $r_5$ = synchronous correlations; OR<sub>1</sub>, OR<sub>2</sub>, OR<sub>6</sub>, OR<sub>7</sub>= intervention effects at three months; OR<sub>5</sub>= effect of alcohol on IPV at three months; OR<sub>3</sub>, OR<sub>4</sub>= autoregressive (AR) pathways. \*p<0.05

<sup>a</sup>Controlling for age, education, baseline partnership status, baseline employment status, baseline injection drug use, exposure to violence as a child, and baseline involvement in community violence ever.

Note: R<sup>2</sup> could not be calculated by MPlus8 due to the binary mediator. IPV= Intimate partner violence; OR= Odds ratio

Figure A.8. Path analysis, alcohol use disorder (AUD)<sup>a</sup> (N=399)

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**Table A.23.** Mediation results by alcohol measure<sup>a,b,c</sup> (N=399)

|  | Proportion of days alcohol abstinent |      | Heavy drinking    |      | Alcohol use disorder (AUD) |      |  |  |  |  |  |  |
|--|--------------------------------------|------|-------------------|------|----------------------------|------|--|--|--|--|--|--|
|  |                                      |      |                   |      |                            |      |  |  |  |  |  |  |
| Effect of BI on IPV through alcohol use  |                                      |      |                   |      |                            |      |  |  |  |  |  |  |
| Natural indirect effect                  | 0.84 (0.64, 1.04)                    | 0.10 | 0.88 (0.75, 1.01) | 0.07 | 0.93 (0.82, 1.04)          | 0.06 |  |  |  |  |  |  |
| Natural direct effect                    | 0.33 (0.03, 0.63)                    | 0.15 | 0.31 (0.03, 0.58) | 0.14 | 0.29 (0.03, 0.55)          | 0.13 |  |  |  |  |  |  |
| Total effect                             | 0.27 (0.02, 0.52)                    | 0.13 | 0.27 (0.02, 0.51) | 0.13 | 0.27 (0.02, 0.52)          | 0.13 |  |  |  |  |  |  |
| Effect of Col on IPV through alcohol use |                                      |      |                   |      |                            |      |  |  |  |  |  |  |
| Natural indirect effect                  | 0.87 (0.71, 1.04)                    | 0.08 | 0.86 (0.72, 1.00) | 0.07 | 0.91 (0.78, 1.03)          | 0.06 |  |  |  |  |  |  |
| Natural direct effect                    | 0.56 (0.09, 1.02)                    | 0.24 | 0.54 (0.09, 0.98) | 0.23 | 0.56 (0.09, 1.03)          | 0.24 |  |  |  |  |  |  |
| Total effect                             | 0.48 (0.08, 0.88)                    | 0.20 | 0.46 (0.08, 0.84) | 0.20 | 0.51 (0.08, 0.93)          | 0.22 |  |  |  |  |  |  |

<sup>&</sup>lt;sup>a</sup>Due to software limitations, bootstrapping could not be performed with imputed datasets.

*Note:* Estimates in bold are significant at p<0.05. OR=Odds ratio; CI=Confidence interval; SE=Standard error; BI=Brief Intervention; CoI=Combined Intervention; IPV=Intimate partner violence

<sup>&</sup>lt;sup>b</sup>As causally-defined mediation effects are computed differently than effect estimates in the path analyses, total effect and natural direct effect estimates may differ slightly than those shown in the path analyses.

<sup>&</sup>lt;sup>c</sup>Controlling for age, education, baseline IPV perpetration, baseline partnership status, baseline employment status, baseline injection drug use, exposure to violence as a child, and baseline involvement in community violence ever.

#### REFERENCES

- 1. Preventing intimate partner and sexual violence against women: Taking action and generating evidence. Geneva, Switzerland: World Health Organization 2010.
- 2. Global and regional estimates of violence against women: prevalence and health effects of intimate partner violence and non-partner sexual violence. Geneva, Switzerland: World Health Organization, 2013.
- 3. Mitchell J, Wight M, Heerden AV, Rochat TJ. Intimate partner violence, HIV, and mental health: a triple epidemic of global proportions. International Review of Psychiatry. 2016;28(5):452-63.
- 4. Li Y, Marshall CM, Rees HC, Nunez A, Ezeanolue E, Ehiri J. Intimate partner violence and HIV infection among women: A systematic review and meta-analysis. Journal of the International AIDS Society. 2014(17):18845.
- 5. Maman S, Campbell J, Sweat MD, Gielen AC. The intersections of HIV and violence: directions for future research and interventions. Soc Sci Med. 2000;50(4):459-78. PMID: 10641800.
- 6. Jansen HA, Nguyen TV, Hoang TA. Exploring risk factors associated with intimate partner violence in Vietnam: results from a cross-sectional national survey. Int J Public Health. 2016;61(8):923-34.
- 7. Fulu E, Jewkes R, Roselli T, Garcia-Moreno C, Team UNM-CC-sSoMVR. Prevalence of and factors associated with male perpetration of intimate partner violence: findings from the UN Multi-country Cross-sectional Study on Men and Violence in Asia and the Pacific. Lancet Glob Health. 2013;1(4):e187-207.
- 8. Phil RO, Hoaken PNS. Biological bases of addiction and aggression in close relationships. Wall CWAM, editor. New York: Brunner-Routledge; 2002.
- 9. Fischer JL, Fitzpatrick J, Cleveland B, Lee JM, McKnight A, Miller B. Binge drinking in the context of romantic relationships. Addict Behav. 2005;30(8):1496-516.
- 10. White HR, Chen PH. Problem drinking and intimate partner violence. J Stud Alcohol. 2002;63(2):205-14.

- 11. Tschann JM, Flores E, Pasch LA, Marin BV. Emotional distress, alcohol use, and peer violence among Mexican-American and European-American adolescents. J Adolesc Health. 2005;37(1):11-8.
- 12. Foran HM, O'Leary KD. Alcohol and intimate partner violence: a meta-analytic review. Clin Psychol Rev. 2008;28(7):1222-34.
- 13. Stith SM, Smith DB, Penn CE, Ward DB, Tritt D. Intimate partner physical abuse perpetration and victimization risk factors: A meta-analytic review. Aggression and Violent Behavior. 2004;10(1):65-98.
- 14. Crane CA, Godleski SA, Przybyla SM, Schlauch RC, Testa M. The Proximal Effects of Acute Alcohol Consumption on Male-to-Female Aggression: A Meta-Analytic Review of the Experimental Literature. Trauma Violence Abuse. 2016;17(5):520-31. PMC4798910.
- 15. Reyes HL, Foshee VA, Bauer DJ, Ennett ST. Developmental Associations Between Adolescent Alcohol Use and Dating Aggression. J Res Adolesc. 2012;22(3):526-41. PMC3625033.
- 16. Quigley BM, Leonard KE. Alcohol and the continuation of early marital aggression. Alcohol Clin Exp Res. 2000;24(7):1003-10.
- 17. Tran BX, Nguyen N, Ohinmaa A, Duong AT, Nguyen LT, Van Hoang M, al. e. Prevalence and correlates of alcohol use disorders during antiretroviral treatment in injection-driven HIV epidemics in Vietnam. Drug and Alcohol Dependence. 2013;127(1-3):39-44.
- 18. Hershow RB, Zuskov DS, Vu Tuyet Mai N, Chander G, Hutton HE, Latkin C, Vuong ND, Sripaipan T, Lancaster KE, Ha TV, Go VF. "[Drinking is] Like a Rule That You Can't Break": Perceived Barriers and Facilitators to Reduce Alcohol Use and Improve Antiretroviral Treatment Adherence among People Living with HIV and Alcohol Use Disorder in Vietnam. Subst Use Misuse. 2018;53(7):1084-92. PMID: 29537932.
- 19. Chander G, Lau B, Moore RD. Hazardous alcohol use: a risk factor for non-adherence and lack of suppression in HIV infection. J Acquir Immune Defic Syndr. 2006;43(4):411-7. PMC2704473.
- 20. Hendershot CS, Stoner SA, Pantalone DW, Simoni JM. Alcohol use and antiretroviral adherence: Review and meta-analysis. Journal of Acquired Immune Deficiency Syndromes. 2009;52(2):180-202.

- 21. Tran BX, Nguyen LT, Do CD, Nguyen QL, Maher RM. Associations between alcohol use disorders and adherence to antiretroviral treatment and quality of life amongst people living with HIV/AIDS. BMC Public Health. 2014;14:27. PMC3893525.
- 22. Field CA, Caetano R, Nelson S. Alcohol and violence related cognitive risk factors associated with the perpetration of intimate partner violence. Journal of Family Violence. 2004;19(4):249-53.
- 23. Johnson H. Contrasting views of the Role of Alcohol in Cases of Wife Assault. Journal of Interpersonal Violence. 2001;16(1):54-72.
- 24. Lai HM, Cleary M, Sitharthan T, Hunt GE. Prevalence of comorbid substance use, anxiety and mood disorders in epidemiological surveys, 1990-2014: A systematic review and meta-analysis. Drug and Alcohol Dependence. 2015(154):1-13.
- 25. Satyanarayana VA, Hebbani S, Hegde S, Krishnan S, Srinivasan K. Two sides of a coin: Perpetrators and survivors perspectives on the triad of alcohol, intimate partner violence and mental health in South India. Asian J Psychiatr. 2015;15:38-43.
- 26. Elbogen EB, Johnson SC. The intricate link between violence and mental disorder: results from the National Epidemiologic Survey on Alcohol and Related Conditions. Arch Gen Psychiatry. 2009;66(2):152-61.
- 27. Quigley BM, Houston RJ, Antonius D, Testa M, Leonard KE. Alcohol use moderates the relationship between symptoms of mental illness and aggression. Psychology of Addictive Behaviors. 2018;32(7):770-8.
- 28. Easton CJ, Mandel DL, Hunkele KA, Nich C, Rounsaville BJ, Carroll KM. A cognitive behavioral therapy for alcohol-dependent domestic violence offenders: an integrated substance abuse-domestic violence treatment approach (SADV). Am J Addict. 2007;16(1):24-31.
- 29. Kraanen FL, Vedel E, Scholing A, Emmelkamp PM. The comparative effectiveness of Integrated treatment for Substance abuse and Partner violence (I-StoP) and substance abuse treatment alone: a randomized controlled trial. BMC Psychiatry. 2013;13:189. PMC3716952.
- 30. Satyanarayana VA, Nattala P, Selvam S, Pradeep J, Hebbani S, Hegde S, Srinivasan K. Integrated Cognitive Behavioral Intervention Reduces Intimate Partner Violence Among Alcohol Dependent Men, and Improves Mental Health Outcomes in their Spouses: A Clinic Based Randomized Controlled Trial from South India. J Subst Abuse Treat. 2016;64:29-34.

- 31. Stuart GL, Ramsey SE, Moore TM, Kahler CW, Farrell LE, Recupero PR, Brown RA. Reductions in marital violence following treatment for alcohol dependence. J Interpers Violence. 2003;18(10):1113-31.
- 32. Michau L, Horn J, Bank A, Dutt M, Zimmerman C. Prevention of violence against women and girls: lessons from practice. Lancet. 2015;385(9978):1672-84.
- 33. Barker G, Contreras M, Heilman B, Singh A, Verma R, Nascimento M. Evolving men: initial results from the International Men and Gender Equality Survey. Washington, DC: International Centre for Research on Women, 2011.
- 34. Jewkes R, Sikweyiya Y, Morrell R, Dunkle K. Gender inequitable masculinity and sexual entitlement in rape perpetration South Africa: findings of a cross-sectional study. PLoS One. 2011;6(12):e29590. PMC3247272.
- 35. Gilchrist G, Radcliffe P, Noto AR, d'Oliveira AF. The prevalence and factors associated with ever perpetrating intimate partner violence by men receiving substance use treatment in Brazil and England: A cross-cultural comparison. Drug Alcohol Rev. 2017;36(1):34-51.
- 36. Yount KM, Higgins EM, Vanderende KE, Krause KH, Minh TH, Schuler SR, Anh HT. Men's Perpetration of Intimate Partner Violence in Vietnam: Gendered Social Learning and the Challenges of Masculinity. Men and Masculinities. 2015;19(1):64-84.
- 37. Afifi TO, Henriksen CA, Asmundson GJ, Sareen J. Victimization and perpetration of intimate partner violence and substance use disorders in a nationally representative sample. J Nerv Ment Dis. 2012;200(8):684-91.
- 38. Hershow RB, Bhadra M, Mai NVT, Sripaipan T, Ha TV, Go VF. A Qualitative Study With Women Living With HIV on Perceived Gender Norms and Experiences of Intimate Partner Violence in Northern Vietnam. J Interpers Violence. 2017:886260517724834. PMID29294869.
- 39. Fals-Stewart W, Kashdan TB, O'Farrell TJ, Birchler GR. Behavioral couples therapy for drug-abusing patients: effects on partner violence. J Subst Abuse Treat. 2002;22(2):87-96.
- 40. Gilbert L, El-Bassel N, Wu E, Chang M. Intimate partner violence and HIV risks: a longitudinal study of men on methadone. J Urban Health. 2007;84(5):667-80. PMC2231853.

- 41. Hoang T, Quach TT, Tran TT. 'Because I am a man, I should be gentle to my wife and my children': positive masculinity to stop gender-based violence in a coastal district in Vietnam. Gender & Development. 2013;21(1):81-96.
- 42. Yount KM, Pham HT, Minh TH, Krause KH, Schuler SR, Anh HT, VanderEnde K, Kramer MR. Violence in childhood, attitudes about partner violence, and partner violence perpetration among men in Vietnam. Ann Epidemiol. 2014;24(5):333-9. PMC4058324.
- 43. Ellsberg M, Arango DJ, Morton M, Gennari F, Kiplesund S, Contreras M, Watts C. Prevention of violence against women and girls: what does the evidence say? Lancet. 2015;385(9977):1555-66.
- 44. Jewkes R, Flood M, Lang J. From work with men and boys to changes of social norms and reduction of inequities in gender relations: a conceptual shift in prevention of violence against women and girls. Lancet. 2015;385(9977):1580-9.
- 45. Kouyoumdjian FG, Findlay N, Schwandt M, Calzavara LM. A systematic review of the relationships between intimate partner violence and HIV/AIDS. PLoS One. 2013;8(11):e81044. PMC3840028.
- 46. Campbell JC, Baty ML, Ghandour RM, Stockman JK, Francisco L, Wagman J. The intersection of intimate partner violence against women and HIV/AIDS: a review. Int J Inj Contr Saf Promot. 2008;15(4):221-31. PMC3274697.
- 47. Koenig LJ, Moore J. Women, violence, and HIV: a critical evaluation with implications for HIV services. Matern Child Health J. 2000;4(2):103-9.
- 48. Medley A, Garcia-Moreno C, McGill S, Maman S. Rates, barriers and outcomes of HIV serostatus disclosure among women in developing countries: implications for prevention of mother-to-child transmission programmes. Bull World Health Organ. 2004;82(4):299-307. PMC2585956.
- 49. Marshall KJ, Fowler DN, Walters ML, Doreson AB. Interventions that Address Intimate Partner Violence and HIV Among Women: A Systematic Review. AIDS Behav. 2018;22(10):3244-63. PMC6035885.
- 50. Jewkes R, Sikweyiya Y, Morrell R, Dunkle K. The relationship between intimate partner violence, rape and HIV amongst South African men: a cross-sectional study. PLoS One. 2011;6(9):e24256. PMC3173408.

- 51. Raj A, Reed E, Welles SL, Santana MC, Silverman JG. Intimate partner violence perpetration, risky sexual behavior, and STI/HIV diagnosis among heterosexual African American men. Am J Mens Health. 2008;2(3):291-5.
- 52. Decker MR, Seage GR, Hemenway D, Gupta J, Raj A, Silverman JG. Intimate partner violence perpetration, standard and gendered STI/HIV risk behaviour, and STI/HIV diagnosis among a clinic-based sample of men. Sex Transm Infect. 2009;85(7):555-60. PMC3623286.
- 53. Go VF, Srikrishnan AK, Salter ML, Mehta S, Johnson SC, Sivaram S, Davis W, Solomon S, Celentano DD. Factors associated with the perpetration of sexual violence among wine-shop patrons in Chennai, India. Soc Sci Med. 2010;71(7):1277-84. PMC2975379.
- 54. Sambisa W, Angeles G, Lance PM, Naved RT, Curtis SL. Physical and sexual abuse of wives in urban Bangladesh: husbands' reports. Stud Fam Plann. 2010;41(3):165-78.
- 55. Hembling J, Andrinopoulos K. Evidence of increased STI/HIV-related risk behavior among male perpetrators of intimate partner violence in Guatemala: results from a national survey. AIDS Care. 2014;26(11):1411-8.
- 56. Raj A, Santana MC, La Marche A, Amaro H, Cranston K, Silverman JG. Perpetration of intimate partner violence associated with sexual risk behaviors among young adult men. Am J Public Health. 2006;96(10):1873-8. PMC1586132.
- 57. Hatcher AM, Gibbs A, McBride RS, Rebombo D, Khumalo M, Christofides NJ. Gendered syndemic of intimate partner violence, alcohol misuse, and HIV risk among peri-urban, heterosexual men in South Africa. Soc Sci Med. 2019:112637.
- 58. Connell RW. Masculinities. Berkeley, CA: University of California Press; 1995.
- 59. Connell RW, Messerschmidt JW. Hegemonic Masculinity: Rethinking the Concept. Gender & Society. 2005;19(6):829-59.
- 60. Dasgupta A, Silverman, J., Saggurti, N., Ghule, M., Donta, B., Battala, M., Nair, S., Gajanan, V., Raj, A. . Understanding Men's Elevated Alcohol Use, Gender Equity Ideologies, and Intimate Partner Violence Among Married Couples in Rural India. American Journal of Men's Health. 2018;12(4):1084-93.
- 61. Abramsky T, Devries K, Kiss L, Nakuti J, Kyegombe N, Starmann E, Cundill B, Francisco L, Kaye D, Musuya T, Michau L, Watts C. Findings from the SASA! Study: a cluster randomized controlled trial to assess the impact of a community mobilization

- intervention to prevent violence against women and reduce HIV risk in Kampala, Uganda. BMC Med. 2014;12:122. PMC4243194.
- 62. Pettifor A, Lippman SA, Gottert A, Suchindran CM, Selin A, Peacock D, Maman S, Rebombo D, Twine R, Gomez-Olive FX, Tollman S, Kahn K, MacPhail C. Community mobilization to modify harmful gender norms and reduce HIV risk: results from a community cluster randomized trial in South Africa. J Int AIDS Soc. 2018;21(7):e25134. PMC6058206.
- 63. Pronyk PM, Hargreaves JR, Kim JC, Morison LA, Phetla G, Watts C, Busza J, Porter JD. Effect of a structural intervention for the prevention of intimate-partner violence and HIV in rural South Africa: a cluster randomised trial. Lancet. 2006;368(9551):1973-83.
- 64. Minnis AM, Doherty IA, Kline TL, Zule WA, Myers B, Carney T, Wechsberg WM. Relationship power, communication, and violence among couples: results of a cluster-randomized HIV prevention study in a South African township. Int J Womens Health. 2015;7:517-25. PMC4435250.
- 65. Wechsberg WM, Zule WA, Luseno WK, Kline TL, Browne FA, Novak SP, Ellerson RM. Effectiveness of an Adapted Evidence-Based Woman-Focused Intervention for Sex Workers and Non-Sex Workers: The Women's Health CoOp in South Africa. Journal of Drug Issues. 2011;41(2):233-52.
- 66. Gibbs A. Tackling gender inequalities and intimate partner violence in the response to HIV: moving towards effective interventions in Southern and Eastern Africa. Afr J AIDS Res. 2016;15(2):141-8.
- 67. Blashill AJ, Bedoya CA, Mayer KH, O'Cleirigh C, Pinkston MM, Remmert JE, Mimiaga MJ, Safren SA. Psychosocial Syndemics are Additively Associated with Worse ART Adherence in HIV-Infected Individuals. AIDS Behav. 2015;19(6):981-6. PMC4405426.
- 68. Glynn TR, Safren SA, Carrico AW, Mendez NA, Duthely LM, Dale SK, Jones DL, Feaster DJ, Rodriguez AE. High Levels of Syndemics and Their Association with Adherence, Viral Non-suppression, and Biobehavioral Transmission Risk in Miami, a U.S. City with an HIV/AIDS Epidemic. AIDS Behav. 2019;23(11):2956-65. PMC6803095.
- 69. McMahon JM, Braksmajer A, Zhang C, Leblanc N, Chen M, Aidala A, Simmons J. Syndemic factors associated with adherence to antiretroviral therapy among HIV-positive adult heterosexual men. AIDS Res Ther. 2019;16(1):32. PMC6842154.
- 70. Okafor CN, Christodoulou J, Bantjes J, Qondela T, Stewart J, Shoptaw S, Tomlinson M, Rotherman-Borus MJ. Understanding HIV Risk Behaviors Among Young Men in South Africa: A Syndemic Approach. AIDS Behav. 2018;22(12):3962-70. PMC6330132.

- 71. Global status report on alcohol and health. Geneva: World Health Organization, 2014.
- 72. Bryant KJ, Nelson S, Braithwaite RS, Roach D. Integrating HIV/AIDS and alcohol research. Alcohol Res Health. 2010;33(3):167-78. PMC3860513.
- 73. Braithwaite RS, Bryant KJ. Influence of alcohol consumption on adherence to and toxicity of antiretroviral therapy and survival. Alcohol Res Health. 2010;33(3):280-7. PMC3860503.
- 74. Do HM, Dunne MP, Kato M, Pham CV, Nguyen KV. Factors associated with suboptimal adherence to antiretroviral therapy in Viet Nam: a cross-sectional study using audio computer-assisted self-interview (ACASI). BMC Infect Dis. 2013;13:154. PMC3614484.
- 75. Ghebremichael M, Paintsil E, Ickovics JR, Vlahov D, Schuman P, Boland R, Schoenbaum E, Moore J, Zhang H. Longitudinal association of alcohol use with HIV disease progression and psychological health of women with HIV. AIDS Care. 2009;21(7):834-41. PMC3292857.
- 76. Sullivan LE, Goulet JL, Justice AC, Fiellin DA. Alcohol consumption and depressive symptoms over time: a longitudinal study of patients with and without HIV infection. Drug Alcohol Depend. 2011;117(2-3):158-63. PMC3113463.
- 77. Capaldi DM, Knoble NB, Shortt JW, Kim HK. A Systematic Review of Risk Factors for Intimate Partner Violence. Partner Abuse. 2012;3(2):231-80. PMC3384540.
- 78. Ferrer VB, E.; Garcia, E.; Manassero, M. A.; Gili, M. . Meta-analytic study of differential characteristics between batterers and non-batterers: The case of psychopathology and consumption of alcohol and drugs. Psykhe. 2004;13(1):141-56.
- 79. Dao A, Hirsch JS, Giang le M, Parker RG. Social science research on HIV in Vietnam: a critical review and future directions. Glob Public Health. 2013;8 Suppl 1:S7-29. PMC3809010.
- 80. Hong K, Anh N, Ogden J. Understanding HIV and AIDS-related stigma and discrimination in Vietnam. 2004.
- 81. Singer M. A dose of drugs, a touch of violence, a case of AIDS: conceptualizing the SAVA syndemic. Free Inquiry in Creative Sociology. 1996;24(2):99-110.

- 82. Singer MC, Erickson PI, Badiane L, Diaz R, Ortiz D, Abraham T, Nicolaysen AM. Syndemics, sex and the city: understanding sexually transmitted diseases in social and cultural context. Soc Sci Med. 2006;63(8):2010-21.
- 83. Chan BT, Pradeep A, Prasad L, Murugesan V, Chandrasekaran E, Kumarasamy N, Mayer KH. Prevalence and correlates of psychosocial conditions among people living with HIV in southern India. AIDS Care. 2017;29(6):746-50. PMC5552362.
- 84. Levintow SN, Pence BW, Ha TV, Minh NL, Sripaipan T, Latkin CA, Vu PT, Quan VM, Frangakis C, Go VF. Prevalence and predictors of depressive symptoms among HIV-positive men who inject drugs in Vietnam. PLoS One. 2018;13(1):e0191548. PMC5783407.
- 85. Hershow RB, Ha TV, Sripaipan T, Latkin C, Hutton HE, Chander G, Bui Q, Nguyen VQ, Frangakis C, Go VF. Perpetration of intimate partner violence among men living with HIV in northern Vietnam. AIDS and Behavior. 2020.
- 86. Fals-Stewart W, Kennedy C. Addressing intimate partner violence in substance-abuse treatment. J Subst Abuse Treat. 2005;29(1):5-17.
- 87. Giusto A, Puffer E. A systematic review of interventions targeting men's alcohol use and family relationships in low- and middle-income countries. Glob Ment Health (Camb). 2018;5:e10. PMC5885490.
- 88. Klostermann KC. Substance abuse and intimate partner violence: treatment considerations. Subst Abuse Treat Prev Policy. 2006;1:24. PMC1564385.
- 89. Stuart GL, O'Farrell TJ, Temple JR. Review of the association between treatment for substance misuse and reductions in intimate partner violence. Subst Use Misuse. 2009;44(9-10):1298-317. PMC2786069.
- 90. Tarzia L, Forsdike K, Feder G, Hegarty K. Interventions in Health Settings for Male Perpetrators or Victims of Intimate Partner Violence. Trauma Violence Abuse. 2017:1524838017744772.
- 91. Wilson IM, Graham K, Taft A. Alcohol interventions, alcohol policy and intimate partner violence: a systematic review. BMC Public Health. 2014;14:881. PMC4159554.
- 92. O'Farrell TJ, Fals-Stewart W, Murphy M, Murphy CM. Partner violence before and after individually based alcoholism treatment for male alcoholic patients. J Consult Clin Psychol. 2003;71(1):92-102.

- 93. Stuart GL, Shorey RC, Moore TM, Ramsey SE, Kahler CW, O'Farrell TJ, Strong DR, Temple JR, Monti PM. Randomized clinical trial examining the incremental efficacy of a 90-minute motivational alcohol intervention as an adjunct to standard batterer intervention for men. Addiction. 2013;108(8):1376-84. PMC3681834.
- 94. O'Farrell TJ, Murphy CM, Stephan SH, Fals-Stewart W, Murphy M. Partner violence before and after couples-based alcoholism treatment for male alcoholic patients: the role of treatment involvement and abstinence. J Consult Clin Psychol. 2004;72(2):202-17.
- 95. Hartmann M, Datta S, Browne EN, Appiah P, Banay R, Caetano V, Floreak R, Spring H, Sreevasthsa A, Thomas S, Selvam S, Srinivasan K. A Combined Behavioral Economics and Cognitive Behavioral Therapy Intervention to Reduce Alcohol Use and Intimate Partner Violence Among Couples in Bengaluru, India: Results of a Pilot Study. J Interpers Violence. 2020:886260519898431.
- 96. Blackburn N. An Economic Evaluation of an Alcohol Reduction Intervention in the HIV Clinic Setting in Vietnam [Dissertation]: University of North Carolina Gillings School of Global Public Health; 2020.
- 97. Vietnam: Encyclopedia Britannica. Available from: <a href="https://www.britannica.com/place/Vietnam">https://www.britannica.com/place/Vietnam</a>.
- 98. The World Bank In Vietnam: The World Bank; 2018. Available from: <a href="https://www.worldbank.org/en/country/vietnam/overview">https://www.worldbank.org/en/country/vietnam/overview</a>.
- 99. Bi-annual report of the provincial HIV/AIDS Control Center, Thai Nguyen, Vietnam. Thai Nguyen, Vietnam: Provincial HIV/AIDS Control Center, 2014.
- 100. Todini N, Hammett TM, Fryatt R. Integrating HIV/AIDS in Vietnam's Social Health Insurance Scheme: Experience and Lessons from the Health Finance and Governance Project, 2014–2017. Health Systems & Reform. 2018;4(2):114-24.
- 101. Horton P, Rydstrom H. Heterosexual Masculinity in Contemporary Vietnam: Privileges, Pleasures, and Protests. Men and Masculinities. 2011;14(5):542-64.
- 102. Rydstrom H. Encountering "Hot" Anger: Domestic Violence in Contemporary Vietnam. Violence Against Women. 2003;9(6):676-97.
- 103. Schuler SR, Hoang TA, Vu SH, Tan HM, Bui TT, Pham VT. Constructions of gender in Vietnam: in pursuit of the 'Three Criteria'. Cult Health Sex. 2006;8(5):383-94.

- 104. Zhang HX, Locke C. Contextualising Reproductive Rights Challenges: The Vietnam Situation. Women's Studies International Forum. 2002;25(4):443-53.
- 105. Penz E, Kirchler E. Sex-Role Specialization in a Transforming Market: Empirical Evidence from Vietnamese Middle-Class Households. Journal of Macromarketing. 2011;32:61-73.
- 106. Luu BN, Nguyen TT, Newman IM. Traditional alcohol production and use in three provinces in Vietnam: an ethnographic exploration of health benefits and risks. BMC Public Health. 2014;14:731. PMC4223524.
- 107. Tran BX, Nguyen N, Ohinmaa A, Duong AT, Nguyen LT, Van Hoang M, Vu PX, Veugelers PJ. Prevalence and correlates of alcohol use disorders during antiretroviral treatment in injection-driven HIV epidemics in Vietnam. Drug Alcohol Depend. 2013;127(1-3):39-44.
- 108. Lincoln M. Alcohol and drinking cultures in Vietnam: A review. Drug Alcohol Depend. 2016;159:1-8. PMC4725306.
- 109. Recorded alcohol per capita (15 years) consumption in litres of pure alcohol, from 1990: World Health Organization; 2016. Available from: http://www.who.int/gho/alcohol/consumption\_levels/adult\_recorded\_percapita/en/.
- 110. Rasanathan JJK, Bhushan A. Gender-based violence in Viet Nam: Strengthening the response by measuring and acting on the social determinants of health. World Health Organization Regional Office for the Western Pacific, 2011.
- 111. "Keeping silent is dying": Results from the National Study on Domestic Violence Against Women in Vietnam. Hanoi, Vietnam: General Statistics Office of Vietnam, 2010.
- 112. Vung ND, Krantz G. Childhood experiences of interparental violence as a risk factor for intimate partner violence: a population-based study from northern Vietnam. J Epidemiol Community Health. 2009;63(9):708-14.
- 113. James-Hawkins L, Salazar K, Hennink MM, Ha VS, Yount KM. Norms of Masculinity and the Cultural Narrative of Intimate Partner Violence Among Men in Vietnam. J Interpers Violence. 2016;886260516674941.
- 114. Law on marriage and family 1986. In: Studies VWsUaCfWs, editor. Vietnamese women in the eighties. Hanoi, Vietnam: Foreign Languages Publishing House; 1989.

- 115. Penal Code. In: Vietnam TSRo, editor. 1989.
- 116. Law on Gender Equality, Law No.73/2006/QH11. 2006.
- 117. Law on Domestic Violence Prevention and Control, Law No.: 02/2007/QH12 C.F.R. . 2007.
- 118. Le TM, Morley C, Hill PS, Bui QT, Dunne MP. The evolution of domestic violence prevention and control in Vietnam from 2003 to 2018: a case study of policy development and implementation within the health system. Int J Ment Health Syst. 2019;13:41. PMC6555957.
- 119. Vietnam AIDS Response Progress Report 2014: Following up the 2011 Political Declaration on HIV/AIDS. Hanoi: National Committee for AIDS, Drugs and Prostitution Prevention and Control, 2014.
- 120. Optimizing Viet Nam's HIV Response: An Investment Case. Ministry of Health of Viet Nam, 2014.
- 121. Alcohol Use Disorder: National Institute on Alcohol Abuse and Alcoholism. Available from: <a href="https://www.niaaa.nih.gov/alcohol-health/overview-alcohol-consumption/alcohol-use-disorders">https://www.niaaa.nih.gov/alcohol-health/overview-alcohol-consumption/alcohol-use-disorders</a>.
- 122. Giang KB, Allebeck P, Spak F, Van Minh H, Dzung TV. Alcohol use and alcohol consumption-related problems in rural Vietnam: an epidemiological survey using AUDIT. Subst Use Misuse. 2008;43(3-4):481-95.
- 123. Go VF, Minh NL, Frangakis C, Ha TV, Latkin CA, Sripaipan T, Davis W, Zelaya C, Ngoc NP, Quan VM. Decreased injecting is associated with increased alcohol consumption among injecting drug users in northern Vietnam. Int J Drug Policy. 2013;24(4):304-11. PMC4774043.
- 124. Clements K, Schumacher JA. Perceptual biases in social cognition as potential moderators of the relationship between alcohol and intimate partner violence. Aggression and Violent Behavior. 2010;15(5):357-68.
- 125. Ito TA, Miller N, Pollock VE. Alcohol and aggression: a meta-analysis on the moderating effects of inhibitory cues, triggering events, and self-focused attention. Psychol Bull. 1996:120(1):60-82.

- 126. Steele CM, Josephs RA. Alcohol Myopia: Its Prized and Dangerous Effects. The American Psychologist. 1990;45(8):921-33.
- 127. Go VF, Hutton HE, Ha TV, Chander G, Latkin CA, Mai NVT, Quynh BX, Vu PT, Sripaipan T, Lancaster KE, Blackburn N, Hershow RB, Dowdy DW, Frangakis C. Two integrated counseling interventions to reduce alcohol use and increase viral suppression among ART clients in Vietnam: a three-arm randomized controlled comparative effectiveness trial. Manuscript prepared for publication. 2020.
- 128. Richard L, Gauvin L, Raine K. Ecological models revisited: their uses and evolution in health promotion over two decades. Annu Rev Public Health. 2011;32:307-26.
- 129. Heise LL. Violence against women: an integrated, ecological framework. Violence Against Women. 1998;4(3):262-90.
- 130. Wilson IM, Graham K, Taft A. Living the cycle of drinking and violence: A qualitative study of women's experience of alcohol-related intimate partner violence. Drug Alcohol Rev. 2017;36(1):115-24.
- 131. Fals-Stewart W, Leonard KE, Birchler GR. The occurrence of male-to-female intimate partner violence on days of men's drinking: the moderating effects of antisocial personality disorder. J Consult Clin Psychol. 2005;73(2):239-48.
- 132. McNaughton Reyes HL, Foshee VA, Bauer DJ, Ennett ST. Heavy alcohol use and dating violence perpetration during adolescence: family, peer and neighborhood violence as moderators. Prev Sci. 2012;13(4):340-9. PMC3906046.
- 133. Parker RN, Rebhun L. Alcohol and Homicide: A Deadly Combination of Two American Traditions. Albany: State University of New York Press; 1995.
- 134. Birkley E, Eckhardt CI. Anger, hostility, internalizing negative emotions, and intimate partner violence perpetration: A meta-analytic review. Clin Psychol Rev. 2015;37:40-56.
- 135. Maiuro RD. Anger, hostility, and depression in domestically violent versus generally assaultive men and nonviolent control subjects. Journal of Consulting and Clinical Psychology. 1988;56(1):17-23.
- 136. Hutton HEL, K. E.; Zuskov, D.; Mai, N. V. T.; Quynh, B. X.; Chander, G.; Latkin, C. A.; Vu, P. T.; Sripaipan, T.; Ha, T. V.; Go, V. F. . Cultural adaptation of two evidence-based alcohol interventions for antiretroviral treatment clinic paitents in Vietnam. Manuscript submitted for publication. 2018.

- 137. Carroll KM, Nich C, Sifry RL, Nuro KF, Frankforter TL, Ball SA, Fenton L, Rounsaville BJ. A general system for evaluating therapist adherence and competence in psychotherapy research in the addictions. Drug Alcohol Depend. 2000;57(3):225-38.
- 138. Miller W, Rollnick S. Motivational interviewing: Helping people change New York: Guilford Press; 2012.
- 139. Parsons JT, Rosof E, Punzalan JC, Di Maria L. Integration of motivational interviewing and cognitive behavioral therapy to improve HIV medication adherence and reduce substance use among HIV-positive men and women: results of a pilot project. AIDS Patient Care STDS. 2005;19(1):31-9.
- Smedslund G, Berg RC, Hammerstrom KT, Steiro A, Leiknes KA, Dahl HM, Karlsen K. Motivational interviewing for substance abuse. Cochrane Database Syst Rev. 2011(5):CD008063.
- 141. Magill M, Ray LA. Cognitive-behavioral treatment with adult alcohol and illicit drug users: a meta-analysis of randomized controlled trials. J Stud Alcohol Drugs. 2009;70(4):516-27. PMC2696292.
- 142. Babor TF, Higgins-Biddle JC, Saunders JB, Monteiro MG. The Alcohol Use Disorders Identification Test Guidelines for Use in Primary Care. 2001.
- 143. Straus MA, Douglas EM. A short form of the Revised Conflict Tactics Scales, and typologies for severity and mutuality. Violence Vict. 2004;19(5):507-20.
- 144. Murshid NS, Murshid N. Intergenerational Transmission of Marital Violence: Results From a Nationally Representative Sample of Men. J Interpers Violence. 2018;33(2):211-27.
- 145. Nguyen TD. Prevalence of male intimate partner abuse in Vietnam. Violence Against Women. 2006;12(8):732-9.
- 146. Fiellin DA, McGinnis KA, Maisto SA, Justice AC, Bryant K. Measuring alcohol consumption using Timeline Followback in non-treatment-seeking medical clinic patients with and without HIV infection: 7-, 14-, or 30-day recall. J Stud Alcohol Drugs. 2013;74(3):500-4. PMC3602364.
- 147. Vakili S, Sobell LC, Sobell MB, Simco ER, Agrawal S. Using the Timeline Followback to determine time windows representative of annual alcohol consumption with problem drinkers. Addict Behav. 2008;33(9):1123-30.

- 148. What's "at-risk" or "heavy" drinking?: NIAAA. Available from: <a href="https://www.rethinkingdrinking.niaaa.nih.gov/How-much-is-too-much/Is-your-drinking-pattern-risky/Whats-At-Risk-Or-Heavy-Drinking.aspx">https://www.rethinkingdrinking.niaaa.nih.gov/How-much-is-too-much/Is-your-drinking-pattern-risky/Whats-At-Risk-Or-Heavy-Drinking.aspx</a>.
- 149. Francis JM, Helander A, Kapiga SH, Weiss HA, Grosskurth H. Validation of the MINI (DSM IV) Tool for the Assessment of Alcohol Dependence among Young People in Northern Tanzania Using the Alcohol Biomarker Phosphatidylethanol (PEth). Int J Environ Res Public Health. 2015;12(11):14021-33. PMC4661629.
- 150. Pence BW, Gaynes BN, Atashili J, O'Donnell JK, Tayong G, Kats D, Whetten R, Whetten K, Njamnshi AK, Ndumbe PM. Validity of an interviewer-administered patient health questionnaire-9 to screen for depression in HIV-infected patients in Cameroon. J Affect Disord. 2012;143(1-3):208-13. PMC3500577.
- 151. Kroenke K, Spitzer RL, Williams JB. The PHQ-9: validity of a brief depression severity measure. J Gen Intern Med. 2001;16(9):606-13. PMC1495268.
- 152. Machisa MT, Christofides N, Jewkes R. Structural Pathways between Child Abuse, Poor Mental Health Outcomes and Male-Perpetrated Intimate Partner Violence (IPV). PLoS One. 2016;11(3):e0150986. PMC4795913.
- 153. Cunradi CB, Mair C, Todd M. Alcohol outlet density, drinking contexts and intimate partner violence: a review of environmental risk factors. J Drug Educ. 2014;44(1-2):19-33. PMC4422380.
- 154. Dahlberg LL, Toal SB, Behrens CB. Measuring violence-related attitudes, beliefs, and behaviors among youths: A compendium of assessment tools. Atlanta, GA: Division of Violence Prevention, National Center for Injury Prevention and Control, Centers for Disease Control and Prevention, 1998.
- 155. Raudenbush SW, et al. . Optimal Design Software for Multi-level and Longitudinal Research (Version 3.01). 2011.
- 156. Breslow NE, Clayton DG. Approximate Inference in Generalized Linear Mixed Models. Journal of the American Statistical Association. 1993;88(421):9-25.
- 157. Enders CK, Mistler SA, Keller BT. Multilevel multiple imputation: A review and evaluation of joint modeling and chained equations imputation. Psychol Methods. 2016;21(2):222-40.
- 158. Hoffman L. Longitudinal Analysis: Modeling Within-Person Fluctuation and Change. New York, NY: Routledge; 2015.

- 159. Preacher KJ, Curran JP, Bauer DJ. Computational Tools for Probing Interactions in Multiple Linear Regression, Multilevel Modeling, and Latent Curve Analysis. Journal of Educational and Behavioral Statistics. 2006;31(4):437-48.
- 160. Cole DA, Maxwell SE. Testing mediational models with longitudinal data: questions and tips in the use of structural equation modeling. J Abnorm Psychol. 2003;112(4):558-77.
- 161. MacKinnon DP. Introduction to statistical mediation analysis. Mahwah, N.J.: Erlbaum; 2008.
- 162. Selig JP, Preacher KJ. Mediation Models for Longitudinal Data in Developmental Research2009;6(2-3):144-64.
- 163. Nguyen TQ, Webb-Vargas Y, Koning IM, Stuart EA. Causal mediation analysis with a binary outcome and multiple continuous or ordinal mediators: Simulations and application to an alcohol intervention. Struct Equ Modeling. 2016;23(3):368-83. PMC4855301.
- 164. Vanderweele TJ, Vansteelandt S. Odds ratios for mediation analysis for a dichotomous outcome. Am J Epidemiol. 2010;172(12):1339-48. PMC2998205.
- 165. Mackinnon DP, Lockwood CM, Williams J. Confidence Limits for the Indirect Effect: Distribution of the Product and Resampling Methods. Multivariate Behav Res. 2004;39(1):99. PMC2821115.
- 166. Preacher KJ, Hayes AF. Asymptotic and resampling strategies for assessing and comparing indirect effects in multiple mediator models. Behav Res Methods. 2008;40(3):879-91.
- 167. Zhao X, Lynch JG, Chen Q. Reconsidering Baron and Kenny: Myths and Truths about Mediation Analysis. Journal of Consumer Research. 2010;37.
- 168. Crane HM, McCaul ME, Chander G, Hutton H, Nance RM, Delaney JAC, Merrill JO, Lau B, Mayer KH, Mugavero MJ, Mimiaga M, Willig JH, Burkholder GA, Drozd DR, Fredericksen RJ, Cropsey K, Moore RD, Simoni JM, Christopher Mathews W, Eron JJ, Napravnik S, Christopoulos K, Geng E, Saag MS, Kitahata MM. Prevalence and Factors Associated with Hazardous Alcohol Use Among Persons Living with HIV Across the US in the Current Era of Antiretroviral Treatment. AIDS Behav. 2017;21(7):1914-25. PMC5628735.
- 169. Whetten K, Shirey K, Pence BW, Yao J, Thielman N, Whetten R, Adams J, Agala B, Ostermann J, O'Donnell K, Hobbie A, Maro V, Itemba D, Reddy E, Team CR. Trauma

- history and depression predict incomplete adherence to antiretroviral therapies in a low income country. PLoS One. 2013;8(10):e74771. PMC3790775.
- 170. Brickman C, Propert KJ, Voytek C, Metzger D, Gross R. Association Between Depression and Condom Use Differs by Sexual Behavior Group in Patients with HIV. AIDS Behav. 2017;21(6):1676-83. PMC5423822.
- 171. Sobell LC, Brown J, Leo GI, Sobell MB. The reliability of the Alcohol Timeline Followback when administered by telephone and by computer. Drug Alcohol Depend. 1996;42(1):49-54.
- 172. Benebo FO, Schumann B, Vaezghasemi M. Intimate partner violence against women in Nigeria: a multilevel study investigating the effect of women's status and community norms. BMC Womens Health. 2018;18(1):136. PMC6085661.
- 173. Beyer K, Wallis AB, Hamberger LK. Neighborhood environment and intimate partner violence: a systematic review. Trauma Violence Abuse. 2015;16(1):16-47. PMC4476540.
- 174. Reed E, Silverman JG, Welles SL, Santana MC, Missmer SA, Raj A. Associations between perceptions and involvement in neighborhood violence and intimate partner violence perpetration among urban, African American men. J Community Health. 2009;34(4):328-35.
- 175. Enders CK, Tofighi D. Centering predictor variables in cross-sectional multilevel models: a new look at an old issue. Psychol Methods. 2007;12(2):121-38.
- 176. Kachadourian LK, Quigley BM, Leonard KE. Alcohol expectancies and evaluations of aggression in alcohol-related intimate-partner verbal and physical aggression. J Stud Alcohol Drugs. 2014;75(5):744-52. PMC4161694.
- 177. Quigley BM, Leonard KE. Alcohol expectancies and intoxicated aggression. Aggression and Violent Behavior. 2006;11(5):484-96.
- 178. Osgood WD, Johnston LD, O'Malley PM, Bachman JG. The Generality of Deviance in Late Adolescence and Early Adulthood. American Sociology Review. 1988;53(1):81-93.
- 179. Pan HS, Neidig PH, O'Leary KD. Predicting mild and severe husband-to-wife physical aggression. J Consult Clin Psychol. 1994;62(5):975-81.

- 180. Leonard KE, Senchak M. Alcohol and premarital aggression among newlywed couples. J Stud Alcohol Suppl. 1993;11:96-108.
- 181. Zhao M, Liu B, Zheng T, Xu J, Hao Y, Wang J, Zhang X, Nie W, Wang C, Wang F, Jiao M, Wu Q, Liang L. Factors associated with hostility among people living with HIV/AIDS in Northeast China: a cross-sectional study. BMC Public Health. 2019;19(1):1189. PMC6716866.
- 182. McIntosh RC, Hurwitz BE, Antoni M, Gonzalez A, Seay J, Schneiderman N. The ABCs of Trait Anger, Psychological Distress, and Disease Severity in HIV. Ann Behav Med. 2015;49(3):420-33. PMC4623323.
- 183. Graham K. The yin and yang of alcohol intoxication: implications for research on the social consequences of drinking. Addiction. 2003;98(8):1021-3.
- 184. Perinelli E, Gremigni P. Use of Social Desirability Scales in Clinical Psychology: A Systematic Review. J Clin Psychol. 2016;72(6):534-51.
- 185. Sugarman DB, Hotaling GT. Intimate Violence and Social Desirability: A Meta-Analytic Review. Journal of Interpersonal Violence. 1997;12(2):275-90.
- 186. Hahn JA, Dobkin LM, Mayanja B, Emenyonu NI, Kigozi IM, Shiboski S, Bangsberg DR, Gnann H, Weinmann W, Wurst FM. Phosphatidylethanol (PEth) as a biomarker of alcohol consumption in HIV-positive patients in sub-Saharan Africa. Alcohol Clin Exp Res. 2012;36(5):854-62. PMC3310261.
- 187. Arseniou S, Arvaniti A, Samakouri M. HIV infection and depression. Psychiatry Clin Neurosci. 2014;68(2):96-109.
- 188. Ge L, Yap CW, Ong R, Heng BH. Social isolation, loneliness and their relationships with depressive symptoms: A population-based study. PLoS One. 2017;12(8):e0182145. PMC5568112.
- 189. Breet E, Seedat S, Kagee A. Posttraumatic Stress Disorder and Depression in Men and Women Who Perpetrate Intimate Partner Violence. J Interpers Violence. 2019;34(10):2181-98.
- 190. Machisa M, Shamu S. Mental ill health and factors associated with men's use of intimate partner violence in Zimbabwe. BMC Public Health. 2018;18(1):376. PMC5859758.

- 191. Devries KM, Mak JY, Bacchus LJ, Child JC, Falder G, Petzold M, Astbury J, Watts CH. Intimate partner violence and incident depressive symptoms and suicide attempts: a systematic review of longitudinal studies. PLoS Med. 2013;10(5):e1001439. PMC3646718.
- 192. Nanni MG, Caruso R, Mitchell AJ, Meggiolaro E, Grassi L. Depression in HIV infected patients: a review. Curr Psychiatry Rep. 2015;17(1):530.
- 193. Tol WA, Murray SM, Lund C, Bolton P, Murray LK, Davies T, Haushofer J, Orkin K, Witte M, Salama L, Patel V, Thornicroft G, Bass JK. Can mental health treatments help prevent or reduce intimate partner violence in low- and middle-income countries? A systematic review. BMC Womens Health. 2019;19(1):34. PMC6376658.
- 194. Murphy CM, Ting L. The effects of treatment for substance use problems on intimate partner violence: A review of empirical data. Aggression and Violent Behavior. 2010;15(5):325-33.
- 195. Kibicho J, Campbell JK. Community perspectives of second-generation alcohol misuse and HIV risk in rural Kenya: A gendered syndemic lens. Glob Public Health. 2019;14(12):1733-43.
- 196. Kiene SM, Lule H, Sileo KM, Silmi KP, Wanyenze RK. Depression, alcohol use, and intimate partner violence among outpatients in rural Uganda: vulnerabilities for HIV, STIs and high risk sexual behavior. BMC Infect Dis. 2017;17(1):88. PMC5248514.
- 197. Hesser H, Axelsson S, Backe V, Engstrand J, Gustafsson T, Holmgren E, Jeppsson U, Pollack M, Norden K, Rosenqvist D, Andersson G. Preventing intimate partner violence via the Internet: A randomized controlled trial of emotion-regulation and conflict-management training for individuals with aggression problems. Clin Psychol Psychother. 2017;24(5):1163-77.
- 198. Wilson PA, Nanin J, Amesty S, Wallace S, Cherenack EM, Fullilove R. Using syndemic theory to understand vulnerability to HIV infection among Black and Latino men in New York City. J Urban Health. 2014;91(5):983-98. PMC4199444.
- 199. Gilbert L, Raj A, Hien D, Stockman J, Terlikbayeva A, Wyatt G. Targeting the SAVA (Substance Abuse, Violence, and AIDS) Syndemic Among Women and Girls: A Global Review of Epidemiology and Integrated Interventions. J Acquir Immune Defic Syndr. 2015;69 Suppl 2:S118-27. PMC4751344.
- 200. Halkitis PN, Wolitski RJ, Millett GA. A holistic approach to addressing HIV infection disparities in gay, bisexual, and other men who have sex with men. Am Psychol. 2013;68(4):261-73.

- 201. Poteat T, Scheim A, Xavier J, Reisner S, Baral S. Global Epidemiology of HIV Infection and Related Syndemics Affecting Transgender People. J Acquir Immune Defic Syndr. 2016;72 Suppl 3:S210-9. PMC4969059.
- 202. Easton CJ, Crane CA, Mandel D. A Randomized Controlled Trial Assessing the Efficacy of Cognitive Behavioral Therapy for Substance-Dependent Domestic Violence Offenders: An Integrated Substance Abuse-Domestic Violence Treatment Approach (SADV). J Marital Fam Ther. 2018;44(3):483-98.
- 203. Kato M, Long NH, Duong BD, Nhan do T, Nguyen TT, Hai NH, Giang le M, Hoa do M, Van NT, Suthar AB, Fontaine C, Nadol P, Lo YR, McConnell MS. Enhancing the benefits of antiretroviral therapy in Vietnam: towards ending AIDS. Curr HIV/AIDS Rep. 2014;11(4):487-95. PMC4264957.
- 204. Hammett TM, Trang NT, Oanh KTH, Huong NT, Giang LM, Huong DT, Nagot N, Des Jarlais DC. The relationship between health policy and public health interventions: a case study of the DRIVE project to "end" the HIV epidemic among people who inject drugs in Haiphong, Vietnam. J Public Health Policy. 2018;39(2):217-30. PMC5970070.
- 205. Murphy J, Corbett KK, Linh DT, Oanh PT, Nguyen VC. Barriers and facilitators to the integration of depression services in primary care in Vietnam: a mixed methods study. BMC Health Serv Res. 2018;18(1):641. PMC6097413.
- 206. Pells K, Wilson E, Thi Thu Hang N. Negotiating agency in cases of intimate partner violence in Vietnam. Glob Public Health. 2016;11(1-2):34-47.
- 207. Krause KH, Gordon-Roberts R, VanderEnde K, Schuler SR, Yount KM. Why Do Women Justify Violence Against Wives More Often Than Do Men in Vietnam? J Interpers Violence. 2015. PMC4636478.
- 208. Yount KM, Krause KH. Gendered Social Learning, Nonfamily Institutions, and Attitudes About Recourse After Partner Violence. Psychol Violence. 2017;7(1):128-39. PMC5382792.
- 209. Murphy J, Goldner E, Corbett KK, Morrow M, Nguyen VC, Linh DT, Oanh PT. Conceptualizing depression in Vietnam: Primary health care providers' explanatory models of depression. Transcult Psychiatry. 2018;55(2):219-41.
- 210. Bhadra NM, Hershow RB, Ha TV, Pence BW, Mai NVT, Go VF. Depression and coping strategies among Vietnamese women living with HIV: a qualitative study. Global Public Health. 2020;1-12.

- 211. Nguyen Ha P, Pharris A, Huong NT, Chuc NT, Brugha R, Thorson A. The evolution of HIV policy in Vietnam: from punitive control measures to a more rights-based approach. Glob Health Action. 2010;3. PMC2932461.
- 212. Taye A, Rosensweig F, Meline M. Building Vietnam's Capacity to Assess Its Health Systems. Bethesda, MD: Health Systems 20/20, 2012.
- 213. Gelaye B, Williams MA, Lemma S, Deyessa N, Bahretibeb Y, Shibre T, Wondimagegn D, Lemenhe A, Fann JR, Vander Stoep A, Andrew Zhou XH. Validity of the Patient Health Questionnaire-9 for depression screening and diagnosis in East Africa. Psychiatry Res. 2013;210(2):653-61. PMC3818385.