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The Moderating Effects Of Protective And Risk Factors On Outcomes For Behavioral Smoking Cessation Treatment

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THE MODERATING EFFECTS OF PROTECTIVE AND RISK FACTORS ON OUTCOMES FOR BEHAVIORAL SMOKING CESSATION TREATMENT

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CHAPTER 1: INTRODUCTION

Cigarette smoking is the chief cause of preventable death in the United States, accounting for one out of five deaths annually (CDC, 2014). Although 70 percent of smokers attempt to quit at least once in their lifetime, and 40 percent of smokers attempt to quit yearly, most smokers relapse to smoking within days of a quit attempt (CDC, 2014). People living with HIV/AIDS (PLWHA) are among a "special population" of smokers, identified as those with greater smoking prevalence, disproportionate tobacco-related health disparities, and less access to treatment relative to the general population (Borelli, 2010). This high-risk group is also characterized by a deficit of population-specific longitudinal treatment trials. PLWHA report significantly higher rates of cigarette smoking, face increased negative physical health outcomes associated with smoking, and experience more cessation treatment barriers than do those without HIV/AIDS (Burkhalter, Springer, Chhabra, Ostroff, & Rapkin, 2005; Crothers et al., 2009). The current study is part of the first randomized clinical trial of contingency management (CM) for smoking cessation among PLWHA.

Cigarette Smoking and HIV/AIDS. Up to 70% of PLWHA report being daily cigarette smokers (Burkhalter et al., 2005). Smokers who are HIV positive have significantly increased mortality rates in addition to reduced CD4 cell counts, higher viral load levels (i.e., HIV RNA), less responsiveness to antiretroviral treatment, and more rapid progression to AIDS (Furber, Maheswaran, Newell, & Carroll, 2007). Relative to non-infected smokers, HIV-positive smokers have increased rates of cancer, respiratory, pulmonary, and bacterial illnesses, and lower life quality (Crothers et al., 2009; Feldman et al., 2006). Importantly, quitting as well as increasing one's duration of

smoking abstinence significantly reduces HIV symptom burden (Vidrine, Arduino, & Gritz, 2007).

There is a strong desire among PLWHA to quit smoking. One study showed that 63% of HIV-positive smokers want to stop smoking, and 72% have unsuccessfully attempted to abstain from smoking (Mamary, Bahrs, & Martinez, 2002). Almost 70% of these individuals expressed interest in a group intervention for smoking cessation, and 82% of these smokers were willing to participate in nicotine replacement therapy for cessation. Despite this desire and motivation to discontinue tobacco use, there has been a lack of consistent literature confirming the efficacy of varying cessation treatments for HIV-positive individuals. Although pharmacological treatment trials centered on nicotine replacement have sometimes demonstrated higher abstinence rates relative to a no-treatment control (Elzi et al., 2006), others have shown low medication adherence (Ingersoll, Cropsey, & Heckmann, 2009) or low rates of smoking abstinence (Lloyd-Richardson et al., 2009). Behaviorally-based studies that aim to tailor to PLWHA by addressing physical treatment barriers have demonstrated some short-term cessation effects (e.g., Vidrine, Arduino, Lazev, & Gritz, 2006); however, more longitudinal information is needed (Vidrine, Arduino, Lazev, & Gritz, 2012; Fjeldsoe, Marshall, & Miller, 2009). There is a need to continue testing randomized longitudinal treatment methods tailored to HIV-infected smokers. The larger randomized trial that houses the current study is adapted to PLWHA not only through its use of bupropion as a smoking cessation medication that does not interfere with antiretroviral treatment, but also by its modification of ongoing treatment based on the individual responses of participants, enhancing time and cost effectiveness.

Cigarette Smoking and Violence Exposure. Two prominent types of violence exposure commonly linked to psychological distress and functioning are community violence and intimate partner violence. Violence is commonly defined as the use of threatened or actual power or force against an individual or group that may result in injury, death, psychological harm, abnormal development, or deprivation (Dahlberg & Krug, 2002). Community violence exposure can be further characterized by witnessing or personally experiencing stabbings, muggings, shootings, murders or other assaults (Gorman-Smith, Henry, & Tolan, 2004). Intimate partner trauma refers to physical, sexual, and psychological violence enacted by previous or current spouses and dating partners, often occurring in the form of rape, physical assault, and stalking (CDC, 2011). Although more women than men report partner violence exposure, almost 30% of men report experiencing intimate partner violence in their lifetimes (Reid et al., 2008).

Interpersonal trauma exposure accounts for over 500,000 deaths annually and is associated with significant physical, psychological, emotional, and financial burden (CDC, 2003). Similarly, community violence exposure is consistently linked to mortality in addition to social, psychological and physical affliction (World Health Organization [WHO], 2002). Though violence exposure does not necessarily need to be recognized by the victim as traumatic, individuals who undergo exposure in these forms often experience posttraumatic stress symptoms or develop Posttraumatic Stress Disorder (PTSD). Congruently, PTSD diagnoses require exposure to an event that involves actual or threatened death or injury (American Psychiatric Association, 2013).

Substance use is one of the clearest correlates of these forms of violence victimization (Beckham et al., 2005; Calhoun, Denis, & Beckham, 2007; Feldner et al.,

2007; Koenen et al., 2005; Morissette et al. 2007), with 90% of adults with substance use disorders reporting history of psychological trauma exposure (CDC, 2003). Studies specifically show a strong relationship between violence exposure and increased rates of smoking and nicotine dependence across genders and ethnicities (Buckley et al., 2004; Dobie et al., 2004; Feldner et al., 2007; Hapke et al., 2005; Lassar et al., 2000). These associations are observed independent of whether the smoker meets clinical criteria for PTSD (Al Mamun et al., 2007; Hapke et al., 2005) and are sometimes stronger than the correlation between prior trauma and alcohol consumption (Breslau et al., 2003; Op Den Velde et al., 2002). Women who experience psychological (e.g., verbal) partner violence show increased risk of cigarette smoking, with even higher risk of use when physical or sexual abuse enacted by intimate partners is reported (Jun et al., 2008). Global studies additionally find that domestic violence is strongly associated with tobacco use cross-culturally, particularly in areas where violence against women is more prevalent (Ackerson, Kawachi, Barbeau, & Subramanian, 2007). Additionally, not only does experiencing violence increase risk of initiating cigarette use, substance use also reciprocally increases risk of being a victim or perpetrator of violence (Atkinson et al., 2009; Krug et al., 2002). There is a strong need to examine the effects of different modes of violence exposure on treatment outcomes and smoking abstinence.

There are a number of conceptual models that aim to explain linkages between trauma and cigarette use. It is often suggested that nicotine is used to reduce the emotional and sometimes physical discomfort associated with trauma (e.g., Logan et al., 2002). Stress and coping models also explain smoking as a method of stress reduction, associating stress with increased urges for cigarette smoking and cyclically

less success in quitting smoking (Cohen & Lichtenstein, 1990; Jun, Rich-Edwards, Boynton-Jarrett, & Wright, 2008). Severity of nicotine dependence has been positively associated with avoidance, hyperarousal, and other PTSD symptoms (Thorndike, Wernicke, Pearlman, & Haaga, 2006), suggesting that current tobacco use may be related to peri-traumatic modes of tension reduction. Unsurprisingly, risk factors specific to quitting smoking include anxiety, which is focal to post-traumatic stress.

Trauma exposure has also been implicated as a negative causal force on smoking cessation treatment outcomes (Lasser et al., 2000), likely related to the challenging cognitions and mood states that characteristically accompany trauma. This post-trauma experience may include persistent negative beliefs (e.g., “no one can be trusted”), distorted thought processes about the cause or outcomes of violence (e.g., self-blame), negative emotional states (e.g., fear, anger), difficulty experiencing positive emotional states, and feelings of detachment from others (APA, 2013). Survivors of violence are often challenged by building trust and finding purpose in life events, as well as by feelings of guilt, shame, lack of power, and uncertainty (Feldner, Babson, & Zvolensky, 2007). One study found that the severity of symptoms associated with witnessing violent assaults and history of emotional abuse predicted poorer retention and abstinence outcomes in a CM-based intervention for substance use (Ford et al., 2007). Studies also show correlations between sexual trauma and treatment indicators such as lack of trust, expression of feelings, and thought processing (Rosen et al., 2002; Sikkema, 2007).

Violence Exposure and HIV/AIDS. Both intimate partner violence and community violence exposure are common among individuals living with HIV/AIDS.

Compared to 8.7% of the general population who report traumatic experiences, 62% of HIV-positive individuals have experienced trauma exposure (Matchinger, Wilson, Haberer, & Weiss, 2012). The experience of sexual abuse is strongly associated with both HIV status and broader engagement in risky sexual behaviors (Sikkema et al., 2007), with individuals who were abused being more likely to participate in high-risk behaviors that increase exposure to HIV (e.g., Carballo-Dieguez & Dolezal, 1995). Violence against women has also specifically been linked to sexually transmitted infections including HIV infection (Gore-Felton, DiMarco, & Anderson, 2007). For example, women who are in violent and abusive relationships are more likely to experience abuse as a result of requesting the use of condoms, thus being less likely to use sexual protection (Ajuwon et al., 2001; Kalichman et al. 1998; WHO, 2014; Wingood & DiClemente, 1997).

Even among healthy individuals, violence exposure is associated with increased engagement in risky behaviors, more somatic symptoms and fatigue, and poorer immune and general health function (Baroso et al., 2010). The physiological effects of violence exposure and post-trauma symptoms are even more highly related to health outcomes among PLWHA. Physical and psychological stress associated with violent experiences can contribute to hypothalamic-pituitary-adrenal (HPA) dysregularities that negatively impact disease progression, HIV processes and presentation, and general immunologic functioning (Biglino et al., 1995; Cole & Kemeny 1997). For example, HIV positive individuals are more likely to have chronic cortisol elevations (i.e., hypercortisolemia), which may increase viral replication (Swanson et al., 1998).

Violence is also linked to negative HIV-specific treatment outcomes. Interestingly, one study found that trauma exposure was related to poorer treatment adherence in black men as mediated by perceived discrimination (Wagner et al., 2012). The literature additionally shows linkages among past violence exposure (e.g., combat, partner abuse), development of PTSD from the HIV diagnosis itself, and post-diagnostic psychiatric distress (Kelly et al., 1998), again highlighting a need to clinically attend to previous trauma exposure. It has thus been suggested that HIV treatment incorporate trauma and domestic violence screening as a mode of reducing symptoms that may interfere with HIV treatment and health (Humphrey, 2014).

Protective Factors in the Treatment of Cigarette Smoking. Though protective factors such as social support are related to smoking behaviors as well as health outcomes for PLWHA, few studies have clearly examined the role of these buffers in smoking treatment among this population. Further, no studies have examined psychosocial protective and risk factors in a CM smoking cessation treatment design. Social support, psychological distress, and life quality will be discussed here.

Social Support. Several studies show that social support not only increases mobilization of psychological resources, but also provides tangible support such as money, skills, and guidance (Bao, Whitbeck, & Hoyt, 2000; Cohen & Wills, 1985; Maulik, Eaton, & Bradshaw, 2010). By the stress buffering model, social support positively influences appraisals of threat following stressful events, bolstering perceived ability to adequately cope with stressors. Alternatively, support can intervene after a stress appraisal has been made, protecting against negative outcomes by increasing positive reappraisals of the stressor, reducing perceived importance of the problem, or

providing solutions to the problem (Cohen et al., 1985). Importantly, lack of social support is associated with elevated smoking rates and reduced cessation (May & West, 2000), and has been shown to be related to maladaptive, avoidant coping strategies among HIV-positive men (Leserman et al., 1992; Tate et al., 2006). Social support has congruently been found to be effective in smoking cessation treatment in the general nicotine-dependent population (Fiore et al., 2008). Perceived social support is also related to experiences of violence victimization, often buffering against negative effects of traumatic events among adults (Nordentoft, 2010).

There remains a need to research the effects of social support on health and substance cessation among PLWHA. The limited literature on social support as a protective factor in the mental and physical health of HIV-positive adults is mixed (Ironson & Hayward, 2008). Some studies show a positive effect of social support on physical and mental health (Ashton et al., 2005; Leserman et al., 2002), while others find weak associations (e.g., Ironson et al., 2005), or suggest that support is only predictive of better health when individuals are in advanced stages of disease infection (Patterson et al., 1996).

It is possible that there are social components specific to HIV/AIDS that separate the disease from other chronic medical illnesses (e.g., cancer, cardiovascular disease) for which social support is consistently linked to increased health. One study found that PLWHA who report increased social support also report more engagement in risky sexual behaviors (Holmes & Pace, 2002; Miller & Cole, 1998). It is also likely that certain groups more prevalently sampled in earlier studies (e.g., HIV-positive men who sleep with men) are operationally different than other HIV-positive subgroups in social

and social support characteristics. Additionally, social support for PLWHA may differ as a product of the social stigma associated with HIV status (Gostin & Webber, 1998; Vanable et al. 2006). In light of the social and medical complexity of HIV/AIDS, it is important to continue investigating the multifaceted impact of social support on PLWHA.

Psychological Distress and Life Quality. Depression is associated with more than twice the risk of HIV progression to AIDS (Golub et al., 2003). Examining change in depressive symptoms longitudinally, the Coping in Health and Illness Project found that for every cumulative increase in depressive symptoms, AIDS risk doubled at 5.5 years (Leserman et al., 2008, 2002, 1999). Another study among women showed that depression measured longitudinally was associated with 61% increased risk of clinical progression and more than double the risk of fatality (Antelman et al., 2007). Distress specifically related to trauma is also instrumental in the medical health of HIV-positive individuals, with post-violence avoidance and intrusion being linked to more depression and lower CD4+ percentages (Lutgendorf et al., 1997). Depression is also heavily linked to nicotine dependence, withdrawal, and ability to quit smoking in the general population (Glassman et al., 2001; O'Brien et al., 2004). As rates of current depression are three times higher in HIV-positive individuals than in the general population (Ferrando & Freyberg, 2008; Reynolds, 2009), there is a critical need to address the role of depression in substance treatment for PLWHA.

Stressful life events are also strongly related to rapid HIV disease progression. One early study found that for each stressful life event in a six-month period, the risk of early HIV disease progression doubled (Evans et al., 1997). Meta-analyses have shown that, along with distress and prior trauma, stressful events negatively impact HIV

disease progression, acting to lower CD4 T lymphocytes, and increase viral load, clinical decline, and mortality (Leserman, 2008). Life quality is also often linked to cigarette smoking, with increased smoking being related to lower life quality, and abstinence promoting higher life quality (Hays, Croghan, Baker, Cappelleri, & Bushmakin, 2012). Both depression and life quality negatively impact smoking cessation treatment in the general population (Cinciripini et al., 2003; Freedland et al., 2005), though it is unclear the specific effect it has on smokers living with HIV/AIDS, who report significantly higher rates of psychiatric illness.

Contingency Management. Among a growing number of interventions focused on reducing smoking behaviors, contingency management (CM) has demonstrated efficacy in improving substance cessation outcomes across several patient populations (e.g., Alessi et al., 2004; Dutra et al., 2008; Ledgerwood et al., 2008; Petry & Alessi, 2010). CM is founded on the concept that voluntary behavior exists in the context of environmental contingencies, wherein frequency of behavior occurs in correlation with the desire to obtain or avoid positive or negative consequences (Skinner, 1953).

By this theoretical approach, cigarette smoking is reinforced and maintained via an operant conditioning process involving the biochemical effects of nicotine as well as environmental reinforcers (e.g., increased social interaction; Higgins & Petry, 1999). Providers select a target behavior that indicates smoking abstinence (e.g., low cotinine levels), and offer incentives which are likely to be rewarding to the smoker. Through this process, behavioral principles of reinforcement operate to counteract the reinforcing mechanisms of chronic smoking. CM has previously been used to reduce HIV-related risk behaviors, such as antiretroviral medication adherence (Haug & Sorensen, 2006).

The present study uses prize-based CM, which was developed as an alternative to more costly monetary and voucher CM treatment systems (Petry et al., 2000). Rather than offering money or vouchers, which typically exceed maximum reinforcement amounts of 1,000 dollars (Higgins et al., 2004; Silverman et al., 1998), prize-based CM offers the opportunity to win prizes of varying magnitudes at a particular reinforcement schedule. Prize-based CM is an efficacious approach to the treatment of a range of substances (Petry et al., 2005; Pierce et al., 2006) including nicotine (Ledgerwood et al., 2014). The larger clinical trial that houses the present study is the first to assess the effects of prize-based CM among PLWHA.

Current Study

The present study examined how risk and protective factors impact cessation efforts among PLWHA undergoing contingency-based treatment for cigarette smoking. Specifically, this study aimed to assess (1) how community and interpersonal partner violence exposure affect smoking cessation among PLWHA, (2) how high depression and low life satisfaction affect smoking cessation among PLWHA, (3) how social support affects smoking cessation among PLWHA, and (4) whether social support moderates relationships between predictors and smoking cessation success among PLWHA.

Smoking Cessation. Smoking cessation success was measured by six smoking indicators: (1) urinary cotinine, (2) longest duration of continuous abstinence, (3) self-reported cigarette use, (4) self-reported change in withdrawal symptoms, (5) self-reported change in smoking urges, and (6) self-reported change in motivations for quitting smoking. To better understand change in smoking cessation from pre to post-

treatment, change scores (*X*-*Y*) were assessed from intake (*X*) to post-Phase 1 (*Y*) and 2 (*Z*). Smoking cessation scores assessed following the first and second treatment phases were used for the current analyses. These cessation indicators are described in more detail in Chapter 2 (Method).

Community and Partner Violence Exposure. Self-reported violence exposure scores reported at the intake assessment were used to predict cigarette use outcomes measured after the first and second phases of treatment. I hypothesized that both increased community violence and intimate partner violence exposure would be inversely related to smoking cessation indicators. Specifically, increased violence exposure will predict lower levels of abstinence, fewer days of continuous abstinence, greater cigarette use, greater withdrawal symptoms, more urges to smoke, and fewer reasons for quitting smoking.

High Depression and Low Life Satisfaction. Depression and life satisfaction assessed during the intake session were used to predict post-Phase 1 and post-Phase 2 smoking outcomes. I hypothesized that higher depression and lower life satisfaction would be inversely related to smoking cessation indicators. Specifically, high depression and low quality of life will predict lower levels of abstinence, fewer continuous days of abstinence, greater use frequency, greater withdrawal symptoms, more urges to smoke, and fewer reasons for quitting smoking.

Social Support. Social support assesses appraisal, belonging, available help, and self-esteem support. A moderation model examined the effect of perceived social support, as self-reported in the intake assessment, on the relationship between the above noted predictor variables (community violence, interpersonal violence,

depression, and life satisfaction), and the above noted outcome variables (changes in cotinine, changes in use frequency, changes in withdrawal, changes in urges, changes in motivations for quitting smoking, and LDA). I hypothesized that social support would be positively related to smoking cessation indicators. Additionally, increased social support will moderate the relationship between increased violence exposure and smoking, higher depression and smoking, and lower life satisfaction and smoking, such that increased social support will be related to reduced smoking indicators.

Treatment Condition. Treatment condition assignment in Phase 1 of the study was additionally assessed and controlled for. Treatment condition was randomly assigned. Phase 1 treatments are described in detail at a later point.

Thus, this study used longitudinal data and hierarchical regressions to test four hypotheses: (1) increased violence exposure is related to poorer smoking cessation outcomes, (2) higher depression and lower life satisfaction is related to poorer smoking cessation outcomes, (3) social support is positively related to smoking cessation outcomes, and (4) social support moderates the relationship between violence history and smoking, depression and smoking, and life satisfaction and smoking.

CHAPTER 2: METHOD

Participants

Participants are those who are enrolled in a larger randomized clinical trial of contingency management for smoking cessation among PLWHA (NIH grant R01 DA034537-01A1; clinical trial identifier: NCT01965405). Participants are 40 daily cigarette smokers from the Wayne State University Physician's Group (WSUPG) adult HIV/AIDS clinic located in Detroit. These individuals represent a portion of the participants in the larger clinical trial study. The number of participants included in the current study was dependent on the rate of participant recruitment and eligibility. Inclusion criteria are as follows: at least 18 years of age, ability to read and understand English, use of at least 10 cigarettes daily, and patient attendance at the WSUPG HIV/AIDS clinic. Exclusion criteria are as follows: active suicidality, uncontrolled manic or psychotic symptoms, being in recovery for pathological gambling, having contraindications for bupropion treatment (e.g., epilepsy, use of MAO inhibitors/other antidepressants, presence of eating disorders/low body mass), or participation in other smoking cessation interventions.

A sequential Multiple Assignment Randomized Trial (SMART) design is used in the larger study. SMART utilizes a stepped-care method that tailors treatment to initial treatment response. Participants in Phase 1 who respond to initial brief treatment (i.e., reduce their smoking) received a different treatment assignment in Phase 2 than participants in Phase 1 who do not respond to initial treatment (i.e., do not reduce their smoking).

Participants in Phase 1 were randomized to one of two brief interventions: (1) counseling care and bupropion pharmacotherapy (i.e., standard of care), or (2) Standard of care in addition to high magnitude prize contingency management (CM). Treatments are described in more detail below. Upon completion of Phase 1, participants were classified as Responders or Non-Responders based on their smoking reduction or abstinence.

Non-responders who enter Phase 2a were randomly assigned to one of two conditions: (1) continued counseling and monitoring support, or (2) monitoring support and prize CM. Phase 1 responders who enter Phase 2b were randomly assigned to one of two conditions: (1) no additional treatment, or (2) continued monitoring and low intensity prize CM. Treatments are described in more detail below.

Random assignment to treatment conditions was balanced by gender, and average daily number of cigarettes smoked (< 1 pack/day, or $\geq$ 1 pack/day). An urn randomization procedure was used to equate groups across these parameters. Random assignment to Phase 1 treatment conditions occurred during the initial intake (day 1 of treatment), and random assignment to Phase 2a and 2b treatment conditions occurred at the start of Phase 2, balanced in the same way as Phase 1. The present study was conducted in accordance with Declaration of Helsinki and was approved by the Wayne State University Institutional Review Board.

The study's stepped care design addresses criticisms of a CM-based approach to cessation treatment in that it aimed to be less time-intensive and more cost-effective. The design minimized time requirements and prize-based treatment for individuals who responded to brief treatment and do not require continuing CM, implementing a more

intensive CM schedule only for those with matching clinical need. The study additionally used prize-based CM in place of more costly cash or voucher-based alternatives (Higgins et al. 2004), reducing costs by rewarding opportunities to earn prizes of varying magnitude rather than using money or money-based vouchers (Petry et al., 2000, 2005).

Treatments

The larger clinical trial involves two treatment phases (4 weeks, and 8 weeks long respectively), followed by 6 and 12-month post-treatment follow-ups. The present study used data from the baseline assessment, the post-Phase 1 (weeks 1-4) treatment assessment, and the post-Phase 2 (weeks 5-12) treatment assessment.

Phase 1. Phase 1 spanned four weeks, and included participant randomization into either Standard of Care, or Standard of Care plus High-Magnitude Prize Contingency Management.

Standard of Care. Standard care involved (1) monitoring of smoking cessation using biological indicators (urinary cotinine) and medication compliance, (2) brief counseling based on clinical practice guidelines (Fiore et al., 2008), and (3) bupropion pharmacotherapy. Participants met with the research therapist weekly for four weeks to provide samples and receive 15-minute counseling geared toward smoking cessation. Counseling was based on the 5As/Rs model.

The 5As/Rs approach involved the 5 As: (1) Ask about tobacco use, (2) Advise the participant to quit smoking, (3) Assess willingness to quit, (4) Assist in the quit attempt through supportive counseling, and (5) Arrange follow-up assessments (U.S. Public Health Service [USPHS]). The 5 Rs were used to address ambivalence about

quitting smoking, and are based on motivational interviewing principles. The 5 Rs are: (1) encourage the participant to indicate personal Relevance of quitting smoking, (2) have the participant identify Risks of continued tobacco use, (3) together identify potential Rewards of quitting smoking, (4) identify Roadblocks or barriers to quitting, and show how counseling may address these barriers, and (5) Repeat motivational interviewing at each subsequent visit. The therapist also reviewed a smoking cessation self-help quit guide with the participant titled *You Can Quit Smoking*, which emphasizes the importance of motivation, social support, and behavioral skills in reducing smoking behaviors (U.S. Department of Health and Human Services). Specifically, the guide addresses reasons for quitting smoking, preparing to quit, gaining social support specific to cessation, skills building, use of medications, and relapse prevention and preparation.

All participants, regardless of condition, were prescribed sustained release bupropion, which has demonstrated efficacy in smoking cessation (Hughes, Stead, Hartmann-Boyce, Cahill, & Lancaster, 2014), is in-line with current Standard of Care, and does not interfere with assessment of urinary cotinine levels. Participants received a flexible dosing procedure of 150 mg/day for the first three days followed by a potential dose increase to 300 mg/day depending on medication tolerability. The recommended maximum dose of bupropion for smoking is 300 mg/day, or 150 mg twice/day at 8-hour intervals (Aubin, 2002). The study physician prescribed bupropion and adjusted dosages based on clinical observation and monitoring of participant responses using the Bupropion Adverse Effects Checklist. Bupropion treatment continued for 12 weeks (Phase 1, and Phase 2a/b) with a two-week taper to 150 mg in week 11, 75 mg in week

12, and 0 mg at the end of week 12. Studies show that bupropion is efficacious and safe for treating PLWHA and those taking antiretroviral medications (Currier, Molina, & Kato, 2003; Pedrol-Clotet et al., 2006). Bupropion administration compliance was closely monitored to assess differential impact on treatment outcomes among treatment conditions. Research assistants conducted pill counts and reviewed participant self-reports of medication compliance.

Standard of Care Plus High Magnitude Prize Contingency Management. Participants in the High Magnitude condition received the same cotinine test result monitoring and brief counseling procedures described above (see Standard of Care section, p. 16), and provided CO and cotinine samples on the same schedule. Participants additionally earned chances to win prizes if they met criteria for early smoking reduction or abstinence (Petry, 2000; Petry et al., 2004). Criteria included cotinine levels lower than the most recent level provided, or below the abstinence cut off of $\leq$ 2. If the participant reduced their cotinine score, but then lapsed, the participant had to reduce subsequent cotinine levels from the point of the lapse to regain a chance for reinforcement. Presenting with a cotinine level above the cutoff, refusing to provide a sample, or being absent without excuse (e.g., illness, family emergency) resulted in draws being reset to one draw for the next provided negative sample. Participants earned draws from the prize-bowl during each weekly session in which they met the reduction/abstinence criteria. In each of weeks 2-4 of treatment, when participants reduce scores from the previous week, they earn one additional draw. That is, reducing smoking at the week 2 session earns the participant two draws; reducing smoking at

week 3 earns the participant three draws, et cetera. By the end of the fourth treatment week, participants may earn up to a total of 10 draws.

The prize urn for Phase 1 of treatment included 50 slips of paper with the following breakdown: 60% (30 slips) result in a large prize ($20); 30% (15 slips) result in a super prize ($50); 10% (5) result in a jumbo prize ($100). Participants may earn an average maximum of $370 in reinforcement if they abstain from smoking throughout this phase.

Phase 2. Phase 2a assigned treatment non-responders from Phase 1 to one of two conditions: (1) continued counseling and monitoring, or (2) continued counseling plus 8-week prize CM. Phase 2b assigned treatment responders from Phase 1 to one of two conditions: (1) no additional intervention, consistent with standard of care, or (2) continued counseling and monitoring, and 8-week low-magnitude prize CM. Phase 2a and 2b treatments are described in more detail below.

Treatment non-responders were classified as those who continued to smoke at levels similar to baseline at the conclusion of Phase 1, determined by objective indicators of smoking: (1) urinary cotinine levels that are not reduced from baseline (i.e., < 2 points pre to post change), or (2) expired CO levels that are not significantly reduced (i.e., > 3 ppm on the final day of testing). Treatment responders were classified as those who significantly reduced smoking as defined by cotinine levels substantially lower than baseline scores ($\geq$ 2 point reduction), or abstinence based on expired CO at the final visit ($\leq$ 3 ppm). The cut-off of $\leq$ 3 ppm has demonstrated high sensitivity and specificity for abstinence (e.g., Javors, Hatch, & Lamb, 2004). As cotinine has a longer half-life relative to that of CO (20 hours versus four hours for CO; Benowitz & Jacob,

1997), a reduction score of $\geq$ 2 points was chosen as an indicator of recent treatment responsiveness.

Phase 2a. All non-responders continued to receive medical monitoring of bupropion for 8 additional treatment weeks, in addition to the randomly assigned treatment.

Counseling and Monitoring of Smoking (MS). Participants met with the study therapists twice weekly to review smoking cessation progress, provide breath samples for CO testing, and provide urine samples for weekly cotinine testing. Therapists provided support and encouragement as well as results of weekly testing.

MS Plus Contingency Management (MS+CM). Participants received the treatment as MS treatment participants, in addition to prize CM for abstinence, weekly for 8 weeks. The method for prize drawing was similar to that of Phase 1; thus, participants received prize draws during weekly therapist meetings if cotinine levels were lower than the most recent level previously provided, or levels were below or at the absolute cutoff of 2. Participants received one draw on the first day of treatment, with escalating draws at each consecutive, negative cotinine test up to a total of 10 draws on a given day. Once the participant earned the maximum number of draws, the participant received that number of draws at each subsequent negative cotinine reading. The same reset contingencies as described in Phase 1 were used; however, resets were reversed once the participant reached three consecutive, negative samples (i.e., the number of prizes will be restored to the highest achieved level pre-reset). Additionally, every third consecutive test that met reinforcement criteria earned the participant five bonus draws. Phase 2 reset contingencies are dissimilar to the Phase 1

reset protocol solely because there were not enough testing sessions in Phase 1 to restore resets.

The prize urn for Phase 2 of treatment included 500 slips of paper with the following breakdown: 50% (250 slips) result in no prize (e.g., “Good job!”); 42.6% (213 slips) result in a small prize ($2); 7% (35 slips) result in a large prize ($20); .4% (2 slips) result in a jumbo prize ($100). Participants may earn up to 115 draws plus 25 bonus draws over the eight-week treatment period, with an average maximum value of $371 in prize reinforcement.

Phase 2b. Similar to Phase 2a (non-responder) treatments, all responders continued to receive medical monitoring of bupropion treatment for 8 treatment weeks, in addition to randomly assigned Phase 2 conditions.

No Additional Treatment (NAT). Participants received no additional treatment of smoking monitoring following Phase 1, consistent with SoC methods that followed brief treatment and medication administration. NAT participants were contacted for follow-up assessments only.

Counseling and Monitoring of Smoking Plus Low Intensity Prize CM. Participants received the same counseling and monitoring as described in 2a treatments, with less frequency. Participants met with research therapists weekly for four weeks, and biweekly for four weeks (i.e., 6 total meetings), to provide cotinine and CO samples. Participants received prize draws during therapy meetings if their cotinine levels were less than either the most recent level, or level 2. Participants used the same prize urn specified in the MS+CM condition, but began with 5 draws, which escalated to a maximum of 10 draws for subsequent negative cotinine tests. Reset contingencies were

in-place for positive tests or missed sessions. The prize urn was similar to that described above for Phase 2a and participants had the opportunity to earn up to 45 draws plus 5 bonus draws over the 8-week treatment period, with an average maximum dollar value of $133 in prizes.

Measures

Demographics and Inclusion/Exclusion. Gender, age, marital status, education and annual income were collected at intake. To assess exclusion criteria, a brief screen of suicidality, psychosis, and substance use symptoms were used, adapted using scales from the Structured Clinical Interview for the DSM-IV-TR (First, Spitzer, Gibbon, & Williams, 2002).

Smoking History. At the time of the intake interview, participants were asked about age of first smoking, age of first daily smoking, current number of cigarettes typically smoked daily, past quit attempts, and periods of abstinence. The Fagerström questionnaire was a brief measure of physical dependence to nicotine (Fagerström, 1978; Heatherton et al., 1991) and was used as a measure of nicotine dependence.

Things I Have Seen and Heard (TSH). A modified version of the TSH (Richters & Martinez, 1990) was used to assess exposure to community violence. The TSH includes 20 items that assessed exposure to neighborhood violence, specifically measuring witnessing or being victimized by violence in the community. Respondents self-reported their lifetime exposure to specific violent events on a 5-point scale ranging from “never” to “always.” The TSH demonstrates good psychometric properties, including internal consistency (Cronbach’s alpha = .76-.80; Richters & Martinez). The TSH was administered at intake.

Revised Conflict Tactics Scale—Short Form (CTS2S). The CTS2S is a brief form of the Revised Conflict Tactics Scale (Straus & Douglas, 2004). It includes 20 items that measure exposure to interpersonal violence among intimate partners, assessing psychological and physical attacks experienced and perpetrated in the past year. The CTS2S demonstrates good internal consistency, test-retest reliability, and validity (Straus, Hamby, Boney-McCoy, & Sugarman, 1996). The CTS2S was administered at intake.

Beck Depression Inventory, Second Edition (BDI-II). The Beck Depression Inventory, Second Edition (BDI-II) is a 21-item self-report measure of past 14-day depression symptoms (Beck, Steer, & Garbin, 1988). Scores range from 0-63, with scores of 0-13 categorized as Minimal Depression, scores of 14-19 categorized as Mild Depression, scores of 20-28 categorized as Moderate Depression, and scores of 29-63 categorized as Severe Depression. The BDI-II is a widely used, valid, and reliable measure of recent depression symptoms. The BDI-II was administered at intake.

Quality of Life Inventory (QOL). The QOL assesses satisfaction in 17 life areas, including work, health, recreation, and goals (Frisch, 1994). This measure was used to assess life satisfaction. The QOL has test-retest coefficients ranging from .80-.91 and correlates with other measures of well-being (Frisch et al., 2005). The QOL was administered at intake.

Interpersonal Support Evaluation List (ISEL). The ISEL (Cohen & Hoberman, 1983) is a 40-item scale that provides an index of overall perceived social support. The ISEL assesses appraisals, belongingness, available help, and self-esteem support. Response options range from definitely false (1) to definitely true (4), with high scores

indicating greater overall social support. The ISEL total score demonstrates good reliability in a variety of samples (Cronbach's alpha = .88-.90; Cohen & Hoberman, 1983). The ISEL was administered at intake.

Cotinine. Cotinine is nicotine metabolite and a sensitive measure of smoking that was assessed by urinalysis using the Accutest NicAlert test-strip system (JANT Pharmacal Corporation). Through this test-strip system, cotinine was reported semi-quantitatively, with ordinal scores of 0 through 6 being assigned based on cotinine concentrations (ng/mL). Level 0 indicates 1-10 ng/mL; level 1 indicates 11-30 ng/mL; level 2 indicates 31-100 ng/mL; level 3 indicates 101-200 ng/mL; level 4 indicates 201-500 ng/mL; level 5 indicates 501-1000 ng/mL; level 6 indicates > 1000 ng/mL. Cotinine levels less than or equivalent to 100 ng/mL, or $\leq$ level 2, were considered negative for cigarette smoking (Benowitz, Hukkanen, & Jacob, 2009).

Due to the variance within each ordinally assigned level and the subsequent inability to analyze precise cotinine concentrations, a change score (X-Y) was used to assess whether cotinine level was reduced from intake (X) to post-treatment (Y). This was done to better detect change in smoking abstinence. Use of change scores as dependent variables is effectively used in regression analyses (Allison, 1990). Change scores may be limited by the fact that they are associated with both baseline and time-point (post-Phase 1 and 2) scores. However, use of change scores in this study preserves power of analyses relative to alternative methods of change measurement (e.g., predicting raw time-point scores while additionally controlling for baseline scores) by reducing the number of variables analyzed. Though cotinine was assessed at

various time points, cotinine levels assessed following the first and second treatment phases were used for the current analyses.

Timeline Follow-back – Cigarette (TLFB-C). The TLFB-C is a self-report, calendar-based measure of the quantity and frequency of cigarette smoking (Sobell et al., 1979). Average number of cigarettes smoked in the past month (30 days) was used to assess cigarette use. This particular measure of cigarette use with the TLFB-C is valid and reliable (Brown et al., 1998; Robinson, Sobell, Sobell, & Leo, 2012). To better understand change in cigarette use from pre to post-treatment, a change score (*X*-*Y*) was assessed from intake (*X*) to post-Phase 1 (*Y*) and 2 (*Z*) (Allison, 1990). Though the TLFB-C will be administered at various time points, TLFB-C scores assessed following completion of the first and second treatment phases were used for the current analyses.

Longest Duration of Continuous Abstinence (LDA). Duration of continuous abstinence from smoking was measured in days, and is defined as the number of the longest string of consecutive days of smoking abstinence (Ledgerwood, Arfken, Petry, & Alessi, 2014). LDA was constructed using the TLFB-C in conjunction with (i.e., confirmed by) reported cotinine levels, and used data from intake to the conclusion of treatment (post-Phase 2). LDA is a robust predictor of future abstinence (Ferguson et al., 2003; Petry, Alessi, & Ledgerwood, 2012).

Minnesota Nicotine Withdrawal Scale (MNWS). The MNWS is a 15-item self-report of nicotine withdrawal symptoms such as anxiety, hunger, and irritability (Cappelleri et al., 2005; Hughes & Hatsukami, 1986). Each symptom is rated on a 5-point scale ranging from “no” to “severe” withdrawal symptoms. To better understand change in withdrawal from pre to post-treatment, a change score (*X*-*Y*) was assessed

from intake (*X*) to post-Phase 1 (*Y*) and 2 (*Z*) (Allison, 1990). Though the MNWS was administered at various time points, MNWS scores assessed following the first and second treatment phases were used for the current analyses.

Questionnaire of Smoking Urges (QSU-Brief). The brief form of the QSU is a 10-item self-report measure that assesses craving to smoke, including anticipated positive effects of smoking, and intention to smoke (Davies, Willner, & Morgan, 2000; Willner, Hardman, & Eaton, 1995; Tiffany & Drobes, 1991). To better understand change in urges from pre to post-treatment, a change score (*X*-*Y*) was assessed from intake (*X*) to post-Phase 1 (*Y*) and 2 (*Z*) (Allison, 1990). Though the QSU was administered at various time points, QSU scores assessed following the first and second treatment phases was used for the current analyses.

Reasons for Quitting (RFQ). The RFQ is a 20-item self-report measure of motivation to quit smoking (Curry et al., 1991). This measure includes four scales, with two reflecting intrinsic motivation (health concerns and desire for self-control), and two reflecting extrinsic motivation (immediate reinforcement and social influence). The present study uses the RFQ total score in order to understand overall motivation to quit smoking. This scale has been validated in studies of smokers (Curry, et al., 1991). To better understand change in motivation to quit smoking from pre to post-treatment, a change score (*X*-*Y*) was assessed from intake (*X*) to post-Phase 1 (*Y*) and 2 (*Z*) (Allison, 1990). Though the RFQ was administered at various time points, RFQ scores assessed following completion of the first and second treatment phases were used for the current analyses.

Analysis of Aims

Four hypotheses were tested using hierarchical multiple regressions. (1) The relationship between increased violence exposure (community violence, intimate partner violence) and smoking cessation outcomes (change in cotinine scores, cigarette use, withdrawal, urges, motivations for quitting smoking, and LDA for the total treatment period) was tested while controlling for differences explained by sociodemographic and initial treatment condition variables (age, gender, education, nicotine dependence, initial treatment condition assignment). This regression included community violence and intimate partner violence scores as predictor variables in the first hierarchical regression block, and sociodemographic and treatment variables in the second regression block. Primary study variables were entered in the first blocks to gain a better understanding of both the model fit of the primary study variables, and the model fit of the primary variables while secondarily controlling for sociodemographic variables and initial treatment condition (e.g., Newman & Thompson, 2003). Outcome variables measured at the completion of Phase 1 (Post-Phase 1 change in cotinine, cigarette use, withdrawal, urges, motivations for quitting smoking) and outcome variables measured at the completion of Phase 2 (Post-Phase 2 change in cotinine, cigarette use, withdrawal, urges, motivations for quitting smoking, and LDA) were assessed in separate regression sets.

(2) The relationship between higher depressive symptoms scores and lower life satisfaction scores and smoking cessation outcomes (change in cotinine scores, cigarette use, withdrawal, urges, motivations for quitting smoking, and LDA for the total treatment period) was tested while controlling for differences explained by sociodemographic and initial treatment condition variables (age, gender, education,

nicotine dependence, initial treatment condition assignment). This regression included depressive symptoms and life satisfaction scores as predictor variables in the first hierarchical regression block, and sociodemographic and treatment variables in the second regression block (Newman & Thompson, 2003). Outcome variables measured at the completion of Phase 1 (Post-Phase 1 change in cotinine, cigarette use, withdrawal, urges, motivations for quitting smoking) and outcome variables measured at the completion of Phase 2 (Post-Phase 2 change in cotinine, cigarette use, withdrawal, urges, motivations for quitting smoking, and LDA) were assessed in separate regression sets.

(3) The relationship between social support and smoking cessation outcomes (change in cotinine scores, cigarette use, withdrawal, urges, motivations for quitting smoking, and LDA for the total treatment period) and (4) the moderation of social support on the relationships between violence exposure and smoking, depressive symptoms and life satisfaction and smoking was tested while controlling for differences explained by sociodemographic and initial treatment condition variables (age, gender, education, nicotine dependence, initial treatment condition assignment). Regressions included social support and primary predictor variables (community violence and intimate partner violence exposure; depressive symptoms and life satisfaction scores) in the first blocks, and sociodemographic and treatment variables in the second blocks (Newman & Thompson, 2003). Third blocks included two-way interaction terms between social support and predictor variables (social support by exposure to community violence, and social support by intimate partner violence; social support by depressive symptoms scores, and social support by life satisfaction scores). Regressions were run

separately for each primary predictor variable to reduce collinearity and increase the power of analyses. Outcome variables measured at the completion of Phase 1 (Post-Phase 1 change in cotinine, cigarette use, withdrawal, urges, motivations for quitting smoking) and outcome variables measured at the completion of Phase 2 (Post-Phase 2 change in cotinine, cigarette use, withdrawal, urges, motivations for quitting smoking, and LDA) were assessed in separate regression sets.

Analyses did not involve correction procedures for the study's 22 multiple regressions and 24 moderation regressions. This decision was made because the consequent reduction in Type I statistical error (i.e., false discoveries) would subsequently increase the probability of Type II statistical error (i.e., false rejections), particularly with the modest sample size of the current study (Gelman, Hill, & Yajima, 2012).

CHAPTER 3: RESULTS

Preliminary Analyses

Statistical Package for the Social Sciences (SPSS) Version 22 was used for all analyses. Adequate power existed for all analyses given these statistical tests and the present study's sample size. This determination was based on literature which reports a standard of five observations needed per independent variable in multiple linear regression to maintain adequate power (Bartlett, Kotrlik, & Higgins, 2001; Hair, Anderson, Tatham, & Black, 1995). In addition, a power analysis was conducted using Gpower 3.1.9.2 statistical analysis software (Faul, Erdfelder, Lang, & Buchner, 2007). This power analysis of the current study parameters reported a power statistic of .81 at a .05 significance criterion, indicating adequate statistical power for the current analyses (Cohen, 1998; Ellis, 2010).

Data were screened for accuracy of input, univariate and multivariate outliers, and amount and distribution of missing data (see Table 1 for full descriptive statistics). No univariate or multivariate outliers were detected. Six participants failed to complete the intended 12-week treatment. Missing data for these 6 participants were identified as data not-missing-at-random. Missing data for these participants were imputed by method of last observation carried forward (LOCF). LOCF is a frequently used, conservative (i.e., underestimates, rather than falsely overestimates, true treatment effects) method of data imputation in longitudinal studies (Gelman & Hill, 2006), particularly when data are missing not-at-random (Higgins & Green, 2011). LOCF additionally avoids distorting both the distribution of data, and multivariate relationships,

as commonly occurs when missing data are replaced with the mean of observed values (i.e., mean imputation; Gelman & Hill).

Normality and linearity among variables were examined using skew and kurtosis statistics, histograms of standardized residuals, and P-Plots of regression standardized residuals. Calculations of skew and kurtosis identified skew among the outcome variables of Post-Phase 1 and Post-Phase 2 cotinine change scores, and LDA (using a skew and kurtosis cut-off level of $\pm$1.96). Variables were transformed for normality using square root and log transformations; however, variables remained above the cut-off level indicating skew and kurtosis. Skew of these variables was interpreted as related to the smoking severity of the current sample, particularly with use of the ordinal-level, range-restricted variable of cotinine. Residual plots of standardized residuals appeared linear and homogenous, and thus indicated appropriate fit of study variables to parametric analyses (Stevens, 2009). Research further indicates that multiple regression analysis is robust to assumptions of normality (Frost, 2014; Gelman & Hill, 2007; van Belle, 2008). Variables were additionally screened for multicollinearity and singularity by examination of bivariate correlations, variance inflation factors (VIF), and tolerance statistics. There was no evidence of multicollinearity or singularity. Bivariate correlations were calculated for all study variables (see Table 2).

Sociodemographic Variables. Descriptive statistics for all study variables are presented in Table 1. Participants (N = 40) were 25-63 years old (M = 46.95, SD = 9.81). Participants included more men (N = 25; 62.5%) than women (N = 15; 37.5%). The majority of participants were African-American (N = 38; 95%), with the remainder identifying as Caucasian (n = 1; 2.5%) and Hispanic (N = 1; 2.5%). Sixty-eight percent

of the sample (*N* = 27) completed at least 12 years of education. The mean Fagerström Test for Nicotine Dependence score was 5.5 (*SD* = 1.97) indicating an overall Moderate level of nicotine dependence. Fifty-five percent of the sample (*N* = 22) was initially randomized to the standard of care treatment condition; 45% (*N* = 18) were initially randomized to the contingency management treatment condition.

Predictor Variables. Participants reported a mean BDI-II depression score of 15.1 (*SD* = 12.41) indicating an overall Mild level of depression. Measures of community violence (The Things I Have Seen and Heard), intimate partner violence (Revised Conflict Tactics Scale - Short Form), social support (Interpersonal Support Evaluation List), and life satisfaction (Quality of Life Inventory) do not indicate clinical significance thresholds and are described below.

Total possible TSH community violence scores range from 0-54, with greater scores indicating more exposure to community violence. Participants reported a mean community violence score of 28.25 (*SD* = 13.11). All participants reported exposure to at least one lifetime community violence event. Specific violence items endorsed are presented in Table 11.

Total possible CTS2S intimate partner violence scores range from 0-120, with greater scores indicating more exposure to intimate partner violence. Participants reported a mean partner violence score of 15.60 (*SD* = 14.30). Sixty percent of participants reported at least one partner violence victimization event in the past year. Specific violence victimization items endorsed are presented in Table 12. Sixty-eight percent of participants reported at least one partner violence perpetration event in the past year. Specific violence items endorsed are presented in Table 13.

Total possible ISEL social support scores range from 0-160, with higher scores indicating greater perceived social support. Total social support scores reported in this sample ranged from 61 to 147. Participants reported a mean total social support score of 121.20 (*SD* = 22.00). ISEL social support subscales include Appraisal (perceived availability of others to trust and confide in), Tangible support (perceived availability of instrumental help), Self-Esteem support (perceived positive comparisons in interpersonal relationships), and Belongingness support (perceived sense of social belongingness). Possible subscale scores range from 0-40. Participants reported a mean Appraisal subscale score of 30.93 (*SD* = 6.16). The mean Tangible support subscale score was 29.73 (*SD* = 6.10). The mean Self-Esteem support subscale score was 29.58 (*SD* = 5.35). The mean Belongingness support subscale score was 30.98 (*SD* = 6.44). All four ISEL social support subscale scores were comparable, with means ranging from 29.58 to 30.98. The ISEL total perceived social support score was used in the current analyses.

Total possible life satisfaction scores on the Quality of Life Inventory range from -51 to 51 (with individual items endorsed on a scale from -3, or very dissatisfied, to 3, very satisfied). Higher scores indicate greater life satisfaction. Total life satisfaction scores in the current sample ranged from -33 to 51. Participants reported a mean total life satisfaction score of 13.75 (*SD* = 21.61). Mean life satisfaction scores reported in each of the 17 areas of functioning are presented in Table 14. As seen in Table 14, participants reported the lowest life satisfaction scores in the following three areas: Standard of Living (income, possessions such as cars or furniture, and expectations for having financial needs met), Work (pay, surroundings, security, relationships with co-

workers, and availability of needed equipment and supervision), and Health (being physically fit and free from sickness, pain, or disability). The highest life satisfaction scores were reported in the following three areas: Relationships with Children (getting along with, helping, teaching, and caring for child/children), Friendships (number and quality of close friends with mutual companionship, acceptance, trust, and support), and Social Service (helping, encouraging, and promoting the welfare of others such as through church, clubs, or volunteer groups).

Outcome Variables. Change scores (*X*-*Y*) were assessed for all outcome variables with the exception of longest duration of abstinence (LDA). As previously noted, cotinine scores are reported semi-quantitatively, with ordinal scores of 0-6 being assigned based on cotinine concentrations. Of note, there is unequal variance within each ordinally assigned level (e.g., a score of 0 indicates cotinine levels of 1-10 ng/mL, while a score of 6 indicates cotinine levels of >1000 ng/mL). The mean baseline cotinine score was 5.63 (*SD* = .93) indicating a high level of cigarette use at baseline. The mean change in cotinine scores from baseline to Time 1 was .50 (*SD* = .18), and mean change in cotinine scores from baseline to Time 2 was .55 (*SD* = .20). Higher change scores indicate decreased cotinine levels from baseline to time point.

The mean past-month number of cigarettes smoked at baseline was 13.97 (*SD* = 1.09). Mean change in cigarette use from baseline to Time 1 was 8.29 (*SD* = .91), and mean change in cigarette use from baseline to Time 2 was 9.12 (*SD* = 1.12). Higher change scores indicate decreased cigarette use from baseline to time point. Participants reported a mean LDA (longest string of consecutive days of smoking abstinence) score of 10.43 (*SD* = 16.56) throughout the duration of treatment.

Total Minnesota Nicotine Withdrawal Scale (MNWS) scores range from 0-60 with higher scores indicating greater withdrawal. Mean change in MNWS withdrawal scores at baseline was 20.33 (*SD* = 15.88). Mean change in withdrawal scores from baseline to Time 1 was 4.0 (*SD* = 1.92), and mean change in withdrawal from baseline to Time 2 was 3.98 (*SD* = 2.06). Higher change scores indicate decreased withdrawal symptoms scores from baseline to time point.

Total Questionnaire of Smoking Urges (QSU) scores range from 0-70 with higher scores indicating more smoking urges. Mean QSU smoking urges scores at baseline was 45.95 (*SD* = 15.37). Mean change in smoking urges from baseline to Time 1 was 21.33 (*SD* = 2.66), and mean change in smoking urges from baseline to Time 2 was 22.35 (*SD* = 2.66). Higher change scores indicate decreased smoking urges scores from baseline to time point.

Total Reasons for Quitting Smoking (RFQ) scores range from 0-80 with higher scores indicating greater motivations to quit smoking. The mean RFQ quitting motivations score was 42.90 (*SD* = 15.72). Mean change in quitting smoking motivations from baseline to Time 1 was -1.65 (*SD* = 1.92), and mean change in quitting smoking motivations from baseline to Time 2 was .43 (*SD* = 2.64). Lower change scores indicate increased motivations to quit smoking from baseline to time point.

Differences by Sociodemographic Variables. Differences on exposure to community violence, exposure to interpersonal violence, depression scores, life satisfaction, and perceived social support, as well as differences on the outcome variables of cotinine, LDA, cigarette use, withdrawal, smoking urges, and motivations for quitting smoking, were examined by socio-demographic variables (age, gender,

ethnicity, education, and nicotine dependence), and initial treatment condition. These tests were conducted to determine if there were significant group differences based on baseline demographic and study characteristics. Comparisons were conducted using analysis of variance (ANOVA). There were no significant differences in the predictor or outcome variables based on age, ethnicity, education, nicotine dependence, or initial treatment condition. Results showed a significant difference in community violence exposure by gender ($F(1, 38) = 4.53$, $p < .05$), with men reporting greater community violence exposure.

Primary Analyses

Effect of Violence, Depressive Symptoms, and Life Satisfaction on Smoking Cessation. Hierarchical multiple regressions were used to assess the unique effect of each predictor variable on each outcome variable while controlling for differences explained by sociodemographic variables and initial treatment condition. Variables were entered in two hierarchical blocks. For all regressions, the first block contained primary predictor variables (community violence, and intimate partner violence; depressive symptoms, and life satisfaction). In the second blocks, sociodemographic and initial treatment condition variables (age, gender, education, initial treatment condition assignment, and Fagerström nicotine dependence scores) were entered. As 95% of the sample was African-American, ethnicity was not associated with any predictor or outcome variables; thus, the sociodemographic variable of ethnicity was excluded from all future regression analyses. Primary study variables were entered in the first block to gain a better understanding of both the model fit of the primary study variables, and the model fit of the primary variables while

secondarily controlling for sociodemographic variables and initial treatment condition (e.g., Newman & Thompson, 2003).

Regressions were run separately for violence variables (community violence, and intimate partner violence), and mood/protective variables (depressive symptoms, and life satisfaction). Five regressions were run with community violence and intimate partner violence predicting each post-Phase 1 (initial smoking response) outcome variable (change in cotinine, cigarette use, withdrawal, urges, and motivations for quitting). Six regressions were run with community violence and intimate partner violence predicting each post-Phase 2 (total cessation treatment response) outcome variable (change in cotinine, cigarette use, withdrawal, urges, motivations for quitting, and LDA). Five regressions were run with depressive symptoms and life satisfaction predicting each post-Phase 1 outcome variable separately. Finally, six regressions were run with depressive symptoms and life satisfaction predicting each post-Phase 2 outcome variable separately. In total, 22 regressions were run.

Initial smoking cessation response was assessed by analyzing intake predictor variables and post-Phase 1 outcome variables, excluding the outcome of longest duration of abstinence (LDA), which measured continuous abstinence throughout the total 12-week treatment period (Phase 1 and Phase 2). Total cessation treatment response was assessed by analyzing intake predictor variables and post-Phase 2 outcome variables. For both post-Phase 1 and post-Phase 2 outcome variables, change scores (*X*-*Y*) were assessed from intake (*X*) to post-Phase 1 (*Y*) and 2 (*Z*) for the variables of cotinine, withdrawal, urges, and motivations for quitting smoking.

Treatment condition in Phase 1 was assessed as a predictor variable in both post-Phase 1 and post-Phase-2 regressions. Though it would be ideal to analyze Phase 2 treatment condition in the post-Phase 2 analysis, Phase 2 treatment condition was confounded by the role of initial treatment response in Phase 2 treatment assignments; thus, initial (Phase 1) treatment condition alone was controlled for in analyses of both post-Phase 1 and post-Phase 2 outcomes.

Effect of Violence on Initial Smoking Cessation. Results are presented in Table 3. There were no significant effects of predictors on Post-Phase 1 (initial) change in cotinine, withdrawal, or smoking urges (all *p*s > .05). There were significant effects of predictors on initial change in motivations to quit smoking and cigarette use (described below).

There was a significant effect of Block 1 (community violence and intimate partner violence exposure) on change in cigarette use ($F(2, 37) = 6.07$, $p < .05$). Block 1 of this model explained 13.8% (R^2) of the total variance in cigarette use. In Block 1, intimate partner violence exposure significantly predicted initial cigarette use ($\beta = -.37$, $p < .05$), with greater intimate partner violence exposure being related to increased cigarette use relative to intake. Block 2, which contained sociodemographic and initial treatment variables, was also significant ($F(2, 37) = 2.92$, $p < .05$). Block 2 did not significantly explain increased variance in cigarette use from Block 1 ($\Delta F = 2.11$, $p = .09$). In Block 2, partner violence remained significant ($\beta = -.10$, $p < .05$) and was related to increased cigarette use. Gender was also significant ($\beta = -.33$, $p = .05$), with male gender being related to reduced cigarette use.

There was a significant effect of Block 1 (community violence and intimate partner violence exposure) on initial change in motivations for quitting smoking ($F(2, 37) = 3.34$, $p < .05$). Block 1 of this model explained 15.3% (R^2) of the total variance in motivations for quitting smoking. In Block 1, community violence exposure significantly predicted initial motivations for quitting smoking ($\beta = 2.49$, $p < .05$), with greater community violence exposure being related to decreased motivations to quit smoking relative to intake. Block 2, which contained sociodemographic and initial treatment variables, was not significant ($p > .05$).

Effect of Violence on Total Smoking Cessation. Results are presented in Table 4. There were no significant effects of predictors on Post-Phase 2 (total treatment) change in cotinine, cigarette use, withdrawal, urges, motivations for quitting smoking, or LDA (all *p*s > .05).

Effect of Depressive Symptoms and Life Quality on Initial Smoking Cessation. Results are presented in Table 5. There were no significant effects of predictors on initial change in cotinine, urges, or motivations for quitting smoking (all *p*s > .05). There were significant effects of predictors on initial change in withdrawal and cigarette use (described below).

There was a significant effect of Block 2 (sociodemographic and initial treatment condition variables) on initial change in cigarette use ($F(2, 37) = 2.58$, $p < .05$). Block 2 of this model explained 36% (R^2) of the variance in initial treatment cigarette use, which represented a significant increase in variance explained from Block 1 of the model ($\Delta F = 3.50$, $p < .05$). In Block 2 of this model, gender significantly predicted initial cigarette use ($\beta = -.33$, $p < .05$), with male gender being related to reduced cigarette use relative to

intake. Block 1 (depressive symptoms scores and life quality) was not significant (p > .05).

There was a significant effect of depressive symptoms and life satisfaction scores (Block 1) on initial change in withdrawal symptoms ($F(2, 37) = 3.27$, $p < .05$). Block 1 (depressive symptoms and life satisfaction) of this model explained 15% (R^2) of the total variance in withdrawal from smoking. In Block 1, depressive symptoms scores significantly predicted smoking withdrawal ($\beta = 2.33$, $p < .05$), with greater depressive symptoms being related to reduced experience of initial withdrawal symptoms. Block 2, which contained sociodemographic and initial treatment variables, was not significant (p > .05).

Effect of Depressive Symptoms and Life Quality on Total Smoking Cessation. Results are presented in Table 6. There were no significant effects of predictors on total treatment change in cotinine, urges, motivations for quitting smoking, or LDA (all ps > .05). There was a significant effect of predictors on total treatment change in withdrawal (described below).

There was a significant effect of depressive symptoms and life satisfaction scores (Block 1) on total treatment change in withdrawal ($F(2, 37) = 3.62$, $p < .05$). Block 1 of this model explained 16.4% (R^2) of the total variance in total treatment smoking withdrawal. In Block 1, depressive symptoms scores significantly predicted smoking withdrawal ($\beta = 2.64$, $p < .05$), with greater depressive symptoms being related to reduced post-Phase 2 smoking withdrawal relative to intake. Block 2, which contained sociodemographic and initial treatment variables, was not significant (p > .05).

Moderating Effect of Social Support. Hierarchical regressions were used to assess the moderating effects of social support, as measured by the ISEL. This analysis allowed for evaluation of both the independent and interactive effects of social support on all outcome variables while controlling for differences explained by sociodemographic and initial treatment variables (Aiken & West, 1991).

Variables were entered into the regression equation by blocks. In the first block, social support and primary predictor variables (community violence and intimate partner violence; depressive symptoms and life quality) were entered. In the second block, sociodemographic and initial treatment variables (age, gender, education, initial treatment condition, and nicotine dependence scores) were added. The third step added two-way interaction terms between social support and predictor variables (social support by exposure to community violence, and social support by intimate partner violence; social support by depressive symptoms, and social support by life satisfaction). These regressions were run separately for each primary predictor variable. This was done to reduce collinearity and increase power of analyses. Regressions were run separately for each outcome variable. In total, 24 moderation regressions were run.

To determine if the interactions entered in the third block significantly added to the variance in the given outcome variable, significant change in the F statistic was examined (West & Aiken, 1997, 1991). If the third model yielded significant change, social support and the given predictor significantly interacted to predict the outcome variable. Squared semi-partial correlations were also examined to determine the unique effects of each predictor and interaction.

Moderating Effect of Social Support on Violence in Predicting Initial Smoking Cessation. There were no independent or moderated effects on initial change in cotinine, withdrawal, urges, or motivations for quitting smoking (all *p*s > .05). There were significant model effects for initial change in cigarette use only (described below and presented in Table 7). Results showed no significant moderation effects for any outcome variables.

There was a significant effect of the model containing community violence, social support, and sociodemographic and treatment variables, on initial treatment change in cigarette use (Table 7). Block 1 (community violence and social support) was not significant ($p > .05$). There was a significant effect of Block 2 (sociodemographic and initial treatment variables added) on initial treatment cigarette use ($F(7, 32) = 2.40$, $p < .05$). Block 2 of this model explained 34% (R^2) of variance in initial change in cigarette use, and significantly increased variance explained from Block 1 ($\Delta F = 3.35$, $p < .05$). In Block 2 of this model, gender significantly predicted initial change in cigarette use ($\beta = -.34$, $p < .05$), with male gender being related to decreased cigarette use (measured relative to intake). Block 3 (community violence by social support interaction term added) was significant ($F(8, 31) = 2.23$, $p < .05$). Block 3 of this model explained 37% (R^2) of variance in initial change in cigarette use, but did not significantly increase variance explained from Block 1 ($\Delta F = 1.03$, $p > .05$). In Block 3 of this model, gender alone significantly predicted initial change in cigarette use ($\beta = -.37$, $p < .05$), with male gender being related to decreased cigarette use from intake.

There was a significant effect of the model containing intimate partner violence, social support, and sociodemographic and treatment variables, on initial change in

cigarette use (Table 7). Block 1 (intimate partner violence and social support) was not significant ($p > .05$). Block 2 of this model added sociodemographic and initial treatment variables, and showed a significant effect on initial change in cigarette use ($F(7, 32) = 2.43$, $p < .05$). Block 2 of this model explained 35% (R^2) of variance in total treatment cigarette use, but did not significantly increase variance explained from Block 1 ($\Delta F = 2.03$, $p > .05$). In Block 2 of this model, gender significantly predicted initial change in cigarette use ($\beta = -.31$, $p < .05$), with male gender being related to decreased cigarette use relative to intake. Block 3 (intimate partner violence by social support interaction term added) was significant ($F(8, 31) = 2.32$, $p < .05$), and explained 37% (R^2) of variance in total treatment cigarette use, but did not significantly increase variance explained from Block 2 ($\Delta F = 1.36$, $p > .05$). In Block 3 of this model, gender alone significantly predicted initial change in cigarette use ($\beta = -.32$, $p < .05$), with male gender being related to decreased cigarette use from intake.

Moderating Effect of Social Support on Violence in Predicting Total Smoking Cessation. There were no independent or moderated effects on total treatment change in cotinine, cigarette use, withdrawal, urges, motivations for quitting smoking, or LDA (all *p*s > .05). Results showed no significant moderation effects for any outcome variables.

Moderating Effect of Social Support on Depressive Symptoms and Life Quality in Predicting Initial Smoking Cessation. There were no independent or moderated effects on initial change in cotinine, urges, or motivations for quitting smoking (all *p*s > .05). There were significant model effects for initial change in withdrawal and cigarette use (described below and presented in Tables 8-9). Results showed no significant moderation effects for any outcome variables.

There was a significant effect of the model containing depressive symptoms, social support, and sociodemographic and treatment variables, on initial change in withdrawal symptoms (Table 8). Block 1 (depressive symptoms and social support) significantly predicted initial change in withdrawal ($F(2, 37) = 3.28$, $p < .05$), and explained 15% (R^2) of the variance in initial smoking withdrawal. In Block 1 of this model, depressive symptoms scores significantly predicted initial change in withdrawal symptoms ($\beta = 2.10$, $p < .05$), with higher depression scores being related to reduced withdrawal relative to intake. Block 2 of this model added sociodemographic and initial treatment variables, and was not significant ($p > .05$); however, depression scores remained a significant predictor in this block ($\beta = 2.14$, $p < .05$). Block 3 of this model added the interaction of social support and depressive symptoms, and was not significant ($p > .05$).

There was a significant effect of the model containing depressive symptoms, social support, and sociodemographic and treatment variables, on initial change in cigarette use (Table 9). Block 1 (depressive symptoms and social support) was not significant. There was a significant effect of Block 2 (sociodemographic and initial treatment variables added) on initial treatment change in cigarette use ($F(7, 32) = 2.57$, $p < .05$). Block 2 of this model explained 36% (R^2) of variance in initial cigarette use, and significantly increased variance explained from Block 1 ($\Delta F = 3.57$, $p < .05$). In Block 2 of this model, gender ($\beta = -.33$, $p < .05$) and education ($\beta = -.33$, $p < .05$) significantly predicted initial cigarette use, with male gender and higher level of education being related to decreased cigarette use relative to intake. There was a significant effect of Block 3 on initial change in cigarette use ($F(8, 31) = 2.31$, $p < .05$).

Block 3 of this model explained 37% (R^2) of variance in initial change in cigarette use, but did not significantly increase variance explained from Block 2 (ΔF = .64, p > .05). In Block 3, gender (β = -.38, p < .05) and education (β = -.34, p < .05) significantly predicted initial change in cigarette use. Male gender and higher education level related to decreased cigarette use relative to intake.

There was a significant effect of the model containing life satisfaction, social support, and sociodemographic and treatment variables, on initial change in cigarette use (Table 9). Block 1 (life satisfaction and social support) was not significant (p > .05). Block 2 of this model added sociodemographic and initial treatment variables. There was a significant effect of Block 2 on initial change in cigarette use (F(7, 32) = 2.57, p < .05). Block 2 of this model explained 36% (R^2) of the variance in initial change in cigarette use and significantly increased variance explained (ΔF = 3.48, p < .01). In Block 2 of this model, gender (β = -.34, p < .05) and education (β = -.33, p < .05) significantly predicted initial cigarette use, with male gender and higher level of education being related to decreased initial cigarette use relative to intake. Block 3 added the interaction term between social support and depressive symptoms. There was a significant effect of Block 3 on total treatment cigarette use (F(8, 31) = 2.36, p < .05). Block 3 of this model explained 38% (R^2) of variance in initial cigarette use, but did not significantly increase explained variance (ΔF = .90, p > .05). In Block 3, gender (β = -.38, p < .05) and education (β = -.35, p < .05) remained significant predictors, with male gender and higher level of education being related to decreased cigarette use relative to intake.

Moderating Effect of Social Support on Depressive Symptoms and Life Quality in Predicting Total Smoking Cessation. There were no independent or moderated effects on total treatment change in cotinine, cigarette smoking, urges, motivations for quitting smoking, or LDA (all *p*s > .05). There were significant model effects for total treatment change in withdrawal only (described below and presented in Table 10). Results showed no significant moderation effects for any outcome variables.

There was a significant effect of the model containing depressive symptoms, social support, and sociodemographic and treatment variables, on total treatment change in withdrawal symptoms (Table 10). Block 1 (depressive symptoms and social support) significantly predicted initial change in withdrawal ($F(2, 37) = 3.32$, $p < .05$), and explained 15.2% (R^2) of variance in total smoking withdrawal. In Block 1 of this model, depressive symptoms scores significantly predicted total treatment change in withdrawal symptoms ($\beta = .34$, $p < .05$), with greater depression being related to reduced post-treatment withdrawal relative to intake. Block 2 of this model added sociodemographic and initial treatment variables, and was not significant ($p > .05$). Block 3 of this model added the interaction of social support and depressive symptoms, and was not significant ($p > .05$).

CHAPTER 4: DISCUSSION

The purpose of this study was to investigate the impact of protective and risk factors on smoking cessation among PLWHA in smoking cessation treatment. Specifically, this study examined the roles of two prevalent components of violence among this population, community violence and intimate partner violence, as well as two variables related to well-being, depression and life satisfaction. This study also assessed the influence of the protective factor of social support on smoking cessation, including moderating effects of social support on violence and well-being variables in predicting smoking cessation outcomes. In addition to primary predictor variables, this study accounted for sociodemographic factors that are often associated with smoking cessation treatment outcomes. These factors included age, gender, education, baseline nicotine dependence scores, and initial treatment condition assignment in the larger clinical trial.

Outcome variables were a range of smoking indicators, including withdrawal symptoms, urges to smoke, motivations for quitting smoking, cotinine scores, self-reported cigarette use, and longest duration of continuous abstinence (longest consecutive number of days not smoking). The purpose of including this range of smoking cessation variables was exploratory. These particular smoking indicators were chosen because they are central to both the experience (withdrawal, urges, motivations for quitting) and the assessment (cotinine, cigarette use, LDA) of quitting smoking, particularly among clinically high-risk groups (Leventhal, Ameringer, Osborn, Zvolensky, and Langdon, 2013; Leventhal et al., 2013; Reid & Ledgerwood, 2015; Weinberger, McKee, & George, 2012; Ziedonis et al., 2008).

Significant Findings

This study found that increased community violence predicted reduced motivations for quitting smoking (relative to intake) after the first phase of treatment. Increased intimate partner violence was related to increased cigarette use (relative to intake) after the first phase of treatment. These findings suggested that discrete components of violence exposure are differentially associated with smoking outcomes, particularly in the beginning of smoking cessation treatment. Increased experience of community violence appears to be related to a proxy of cigarette use (i.e., motivations for quitting smoking), while interpersonal violence is related to actual increase in smoking behaviors (i.e., number of cigarettes smoked). It is possible that interpersonal violence is experienced as being localized to the relationship, and thus predicts a more acute coping need, achieved through smoking. This is in-line with cross-cultural research showing that domestic violence is associated with tobacco use, particularly in areas where greater rates of violence are reported (Ackerson, Kawachi, Barbeau, & Subramanian, 2007).

In contrast, community violence may be experienced more systemically, which could impact distal smoking behaviors. Systemic stress related to chronic experiences of community violence across multiple domains (e.g., neighborhood, home, school/work) may increase hopelessness, which may then mediate between community violence and motivations to quit smoking. Challenges related to these varying forms of violence are of particular importance as 100% of the current sample reported exposure to at least one lifetime community violence event, and 60% of the current sample reported experiencing partner violence victimization in the last year. Specific item

endorsements are illustrated in Table 11 (community violence) and Table 12 (intimate partner violence).

Relationships between partner violence and increased cigarette use, and community violence and reduced quitting motivations, were found after the first phase of treatment only. Phase 1 (initial) treatment condition alone was controlled for in the present analyses, as Phase 2 treatment condition was confounded with the role of initial treatment response in Phase 2 treatment assignments. It is likely that because individuals are assigned to different treatment conditions in Phase 2 based on their response to treatment in Phase 1, Phase 2 treatment assignment was predictive of total treatment smoking cessation success.

Participants who reported relatively greater depressive symptoms at baseline were more likely to demonstrate reductions in smoking withdrawal after both the first and second phases of treatment (relative to intake). This finding was congruent with previous literature showing that smokers with greater depression scores at baseline report significantly different withdrawal experience than smokers in the same treatment with lower depressive scores at baseline (Reid & Ledgerwood, 2015). Though it could be argued that withdrawal is experienced as a result of achieving abstinence from smoking, the previous study showed differences in withdrawal experience despite non-differential levels of tobacco use throughout treatment (assessed through expired carbon monoxide).

In order to further understand these differences in withdrawal experience as a function of depression scores, means and group differences for withdrawal were evaluated by depression score categorization. In the present study, greater depressive

symptoms scores at intake were associated with greater withdrawal sensitivity at intake (r = .76, p < .01), post-Phase 1 (r = .57, p < .01), and post-Phase 2 (r = .50, p < .01). To better evaluate differences in withdrawal experience in a clinically interpretable manner, depressive symptoms were dichotomized into low (scores $\leq$ 13; n = 22) versus high scores (scores > 13; n = 18) based on BDI-II clinical significance cut-offs (with scores of 0 to 13 representing Minimal depression, and 14 and above representing Moderate to Severe depression). Mean scores and group differences demonstrated that mean withdrawal scores were significantly higher among individuals reporting greater depressive symptoms, but that withdrawal decreased more for high-depression scorers (compared to low-depression scorers) between time points (see Figure 1).

It is possible that smokers with higher depression scores demonstrated greater reductions in withdrawal throughout treatment by virtue of reporting significantly higher withdrawal scores at baseline; those with greater baseline depression scores reported almost four-fold higher withdrawal scores than those with low baseline depression scores. Additionally, unmeasured components of treatment, in particular, treatment of all participants with bupropion (commonly prescribed as an antidepressant), may have secondarily aided in reducing depressive symptoms. Buproprion treatment, though not prescribed in the present study for depression symptoms, may be associated with greater changes in withdrawal experience specifically for smokers with greater baseline depressive symptoms, and relatedly greater baseline withdrawal sensitivity.

Increased initial sensitivity to withdrawal among individuals who report mood instability is potentially associated with decreased distress tolerance, or the ability to experience and manage negative internal states, as well as discomfort intolerance, or

the ability to withstand uncomfortable physical sensations (Ellis, Vanderling, & Beevers, 2012; Schmidt & Lerew, 1998; Williams, Thompson, & Andrews, 2013). It is thus possible that individuals who report high depression scores have a more aversive initial experience of withdrawal from tobacco. PLWHA who have co-occurring tobacco use disorder and depression might then be increasingly vulnerable to greater dysphoria during initial withdrawal, particularly when withdrawal is experienced in combination with disease-related physical discomfort.

The sociodemographic variable of male gender was related to greater reductions in cigarette use after the first phase of treatment (in models testing both mood and life satisfaction, and violence). This is congruent with men reporting more prevalence and severity of tobacco use, particularly among PLWHA (CDC, 2015; Mdodo et al., 2015; WHO, 2007), but demonstrating greater abstinence success across a variety of cessation treatments (Carlson et al., 2002; McKee et al., 2005; Scharf & Shiffman, 2004; Wetter et al., 1999). One smoking cessation study in particular showed prominent gender differences in a treatment that incorporated bupropion and behavioral counseling (Collins et al., 2004). Collins and colleagues (2004) not only found that men were more likely to quit and remain abstinent after treatment and at follow-up, but also identified a gender interaction whereby women benefitted more from bupropion treatment if they were light smokers, and men benefitted more from bupropion if they were heavy smokers.

It is then possible that women who reduced smoking as a result of their engagement in smoking treatment were increasingly benefitted by the bupropion component of treatment as treatment continued (i.e., equally reduced cigarette use in

the second phase of treatment). As noted above, tailored treatment assignment in the second phase of treatment may have additionally been related to increased smoking success across participant groups in the second phase of treatment.

There were no independent or moderating effects of social support on smoking outcome variables. This could be related to the socially complex nature of HIV/AIDS. As a result of social stigma associated with HIV status, PLWHA are less likely to disclose their status to friends and family (Sayles et al., 2007). This may indicate less access to instrumental and emotional support for challenges PLWHA uniquely face, even if reports of broad social support are high among this group. That is to say, PLWHA may experience lower-quality social support even when support is present. Findings are congruent with research showing weak associations between social support and health among PLWHA (Ironsen et al., 2005; Ironson & Hayward, 2008). However, rather than indicating reduced need for social support among smokers living with HIV/AIDS, findings support the notion that different forms of support, for instance peer support, may be particularly important among this group. Literature has shown effective peer-based interventions for depressive symptoms among PLWHA with high levels of substance use (Simoni, Pantalone, Plummer, & Huang, 2007).

Implications

Based on the relationship between community violence and quitting motivations, and intimate partner violence and cigarette use, it is important to assess interpersonal and community violence exposure among smokers prior to cessation treatment. This is specifically important among smokers with HIV/AIDS, as base rates of violence and trauma are greater among this population (Matchinger, Wilson, Haberer, & Weiss,

2012). Every individual in the current sample reported community violence exposure, with the most prevalently endorsed experiences including witnessing police arrests, hearing gunfire, witnessing drug use and drug deals in the neighborhood, and witnessing and personally experiencing physical assault (Table 11). The least prevalently experienced community violence events (e.g., experiencing threats of being stabbed, or killed, and witnessing someone get shot or stabbed in the home) were still endorsed by 15-30% of the sample.

Although fewer participants endorsed partner violence exposure (potentially partially due to the necessity of having had a recent romantic partnership), the majority of the sample (60%) still reported violence victimization enacted by intimate partners in the past year (Table 12). The most prevalently reported items included experiences of being verbally (i.e., insulted, swore, or shouted at) assaulted, and physically injured (e.g., experiencing a sprain, bruise, small cut, or physical pain after an altercation). Relatively "severe" partner violence experiences were endorsed by 10% of the sample, and included needing medical attention after an altercation, being forced (through hitting, holding down, or use of a weapon) to have sex, and being forced to have sex without a condom (without physical force). Severe sexually violent experiences may be directly related to HIV disease status, and thus potentially traumatizing on several levels.

Sixty-eight percent of the sample reported perpetrating violence on intimate partners in the past year (Table 13). Prevalence rates of specific perpetration events were comparable to the prevalence of victimization events. The most prevalently reported perpetration event was insulting, swearing, shouting, or yelling at an intimate

partner (65%). Relatively severe violence perpetration experiences were endorsed by 10-12.5% of the sample.

For smokers reporting high levels of intimate partner violence exposure, therapeutic attention should be given to adaptive coping (Brady et al., 2009; Crane, Hawes, & Weinberger, 2013). Two forms of adaptive coping in particular, behavioral coping (e.g., problem solving), and cognitive coping (e.g., positive reappraisals), have been found to mitigate the influence of violence victimization on substance use (Brady et al.). For individuals with significant community violence experience, intrinsic motivations to reduce or abstain from smoking should be evaluated. Motivational interviewing is one evidence-based approach that would address conscious and unconscious motivations to fully engage in treatment to reduce smoking behaviors (Lai, Cahill, Qin, Tang, 2010). The 5As/Rs counseling model used in the clinical trial that houses the present study emphasizes motivation and behavioral skills in reducing smoking (USPHS), and could additionally be used in a targeted fashion when individuals report interpersonal or community violence experiences.

Based on findings related to different patterns of withdrawal among HIV-positive smokers who report greater versus lower depression scores, it is important for clinicians to assess psychological distress, particularly depression, prior to engaging smokers in smoking cessation treatment. It may also be useful to tailor components of treatment to the aversive withdrawal experience PLWHA may uniquely face, particularly at the outset of smoking treatment. Interventions that emphasize acceptance and tolerance are empirically based approaches to the treatment of substance use among individuals with mood symptoms (Brewer, Bowen, Smith, Marlatt, & Potenza, 2010; Dimeff & Linehan,

2008; Linehan et al., 2002). This is particularly relevant given increased reports of mood disorders among PLWHA (Ferrando & Freyberg, 2008; Reynolds, 2009).

Limitations

Findings should be considered in the context of study limitations. The study's sample size (*n* = 40) was small, limiting the detection of the full strength of relationships among violence, mood, life satisfaction, social support, and smoking cessation indicators. The sample was also unevenly distributed across gender and ethnic groups, with more men (*n* = 25) than women, and most participants identifying as African-American (*n* = 38). In addition, this sample included no individuals that identified as a gender other than man or woman. These distributions are particularly important when assessing experiences that are known to vary by gender and ethnicity, including exposure to community violence and intimate partner violence (Foster, Kuperminc, & Price, 2004; Saewyc et al., 2009). It will be important to test the present hypotheses with a larger, more equally distributed sample in order to provide firmer basis for the present study's clinical implications. A larger, evenly distributed sample would also allow for a deeper analysis of the specific subcomponents of community violence, intimate partner violence, and social support that tobacco-using PLWHA may uniquely report.

An additional limitation as previously specified was the inability to control for Phase 2 treatment condition assignment in analyses of Phase 2 treatment outcomes. This was due to the confounding nature of Phase 2 assignment with Phase 1 treatment response. Finally, this study relies on self-report to measure constructs that may be particularly influenced by social desirability (e.g., partner violence, community violence,

social support). Of note, self-reported cigarette use behaviors (through TLFB-C) were compared to, and congruent with, urinary cotinine levels.

Directions for Future Research

Future research should assess mechanisms underlying relationships between community and interpersonal violence and smoking indicators. Specifically, research should explore mediational relationships between cognitive and emotional helplessness and reduced motivations for quitting smoking. In a larger sample, it would also be relevant to examine subcomponents of each form of violence. It is possible that physical versus emotional partner violence indicates different outcomes for smoking, particularly given that HIV status may be more related to a particular form of partner violence (Burke, Thieman, Gielen, O'Campo, & McDonnell, 2005; Maman et al., 2002). The Conflict Tactics Scale used in the present analyses has the capacity in a large, distributed sample to assess physical versus emotional conflict, frequency, severity, and perpetrator experience. Future research could additionally stratify a large sample by gender and sexual orientation to examine whether differential patterns of violence exposure hold among subsamples of gender and orientation.

Conclusions

This dissertation is an important exploration of the impact of violence, mood, and protective factors on smoking cessation experience in a modest sample of HIV-positive smokers undergoing smoking cessation treatment. This study's findings revealed important linkages between community violence exposure and motivations for quitting smoking, as well as between increased depressive symptoms and patterns of smoking withdrawal among HIV-positive smokers. Findings also provided prevalence data on

various violence events experienced in the community and within intimate partnerships. Continued investigations into risk and protective factors would deepen clinical insight and knowledge about therapeutic approaches for a population of smokers with heightened clinical and medical risk.

Table 1

Descriptive Statistics

Study Variables	Mean (SD)	Percentage	Sample Size
Socio-demographic Variables			
Age	46.95 (9.81)		40
Gender			40
Male		62.5	25
Female		37.5	15
Ethnicity			
African-American		95	38
Caucasian		2.5	1
Hispanic		2.5	1
Education (years)	12.15 (1.42)	67.5	40
Nicotine Dependence	5.50 (1.97)		40
Treatment Assignment			39
Standard		55.0	22
Contingency Management		45.0	18
Predictor Variables			
Community Violence	28.25 (13.11)		40
Intimate Partner Violence	15.60 (14.30)		40
BDI	15.10 (12.41)		40
Quality of Life	13.75 (21.61)		40
Social Support	121.20 (22.00)		40
Outcome Variables			
Cotinine Intake	5.63 (.93)		40
Cotinine Change Time 1	.50 (.18)		40
Cotinine Change Time 2	.55 (.20)		40
Cigarette Use Intake	13.97 (1.09)		40
Cigarette Use Change Time 1	8.29 (.91)		40
Cigarette Use Change Time 2	9.12 (1.12)		40
MNWS Intake	20.33 (15.88)		40
MNWS Change Time 1	4.00 (1.92)		40
MNWS Change Time 2	3.98 (2.06)		40
QSU Intake	45.95 (15.37)		40
QSU Change Time 1	21.33 (2.66)		40
QSU Change Time 2	22.35 (2.66)		40
RFQ Intake	42.90 (15.72)		40
RFQ Change Time 1	-1.65 (1.92)		40
RFQ Change Time 2	.43 (2.64)		40
LDA	10.43 (16.56)		40

Table 2

Bivariate Correlations

Primary Variables	1	2	3	4	5	6	7	8	9	10	11	12	13	14	15	16
1 Comm.Violence		.03	.40*	-.16	-.24	-.10	-.10	-.01	-.01	.18	.12	-.10	-.02	.38*	.22	-.07
2 Partn. Violence			.10	-.21	-.03	.09	.06	-.37*	-.18	.11	.05	-.13	-.18	.10	-.17	-.14
3 BDI				-.50**	-.47**	-.16	-.09	.06	.04	.39*	.38*	-.04	-.09	.23	.08	-.04
4 Quality of Life					.55***	.12	.03	-.11	-.14	-.16	-.08	.23	.24	.01	-.09	-.10
5 Social Support						.17	-.02	-.03	-.15	-.22	-.25	.17	.09	-.08	-.22	.12
6 Cot. Change T1							.68***	.37*	.31	.11	.00	.27	.25	-.02	.07	.49**
7 Cot. Change T2								.08	.09	.03	-.03	.16	.14	-.08	.10	.46**
8 Cig. Use Ch T1									.83***	.25	.15	.35*	.20	.02	.03	.46**
9 Cig. Use Ch T2										.23	.13	.33*	.16	-.22	-.14	.47**
10 MNWS Ch T1											.89***	.00	.00	.10	.02	.18
11 MNWS Ch T2												-.06	.05	.08	.04	.04
12 QSU Ch T1													.75***	-.20	-.29	.23
13 QSU Ch T2														-.33*	-.26	.15
14 RFQ Ch T1															.58**	-.34*
15 RFQ Ch T2																-.23
16 LDA																

Note. *p < .05; **p < .01; ***p < .001. All outcome scores reported are change scores calculated from Intake scores.

Table 3

Multivariate Regression Effects of Violence on Time 1 Smoking Cessation Change Scores

Predictor Variable	Beta	SE	*t*	*p*	R^2 (*F*)
Explaining cotinine scores					
Block 1					
Community violence	-.10	.01	-.62	.54	
Partner violence	.09	.01	.55	.58	
Block 2					
Community violence	-.06	.02	-.30	.77	
Partner violence	.21	.02	1.01	.32	
Age	-.34	.02	-1.78	.08	
Gender	-.13	.44	-.68	.50	
Education	-.14	.16	-.70	.49	
Initial treatment	.19	.41	1.08	.29	
Nicotine dependence	.23	.11	1.18	.25	
Explaining cigarette use					
Block 1					
Community violence	.02	.07	.02	.98	
Partner violence	-.37	.06	-2.46	.02	
Block 2					
Community violence	-.05	.07	-.33	.74	
Partner violence	-.10	.07	-2.32	.04	
Age	.11	.10	.63	.53	
Gender	-.33	1.90	-2.03	.05	
Education	-.26	.69	-1.54	.13	
Initial treatment	.20	1.79	1.26	.22	
Nicotine dependence	.19	.49	1.10	.28	
					.14 (6.07)*
Explaining withdrawal					
Block 1					
Community violence	.18	.15	1.11	.27	
Partner violence	.10	.14	.63	.54	
Block 2					
Community violence	.25	.18	1.32	.20	
Partner violence	.22	.18	1.03	.31	
Age	.02	.25	.09	.93	
Gender	.03	4.74	.06	.95	
Education	-.24	1.73	-1.17	.25	
Initial treatment	.01	4.47	.06	.95	
Nicotine dependence	.02	1.23	.11	.91	
Explaining urges					

Block 1					
Community violence	-.09	.21	-.53	.60	
Partner violence	-.13	.19	-.79	.44	
Block 2					
Community violence	-.11	.24	-.58	.57	
Partner violence	-.04	.25	-.17	.87	
Age	.15	.34	.76	.45	
Gender	-.13	6.54	-.67	.51	
Education	-.17	2.39	-.83	.41	
Initial treatment	-.07	6.18	-.39	.87	
Nicotine dependence	-.08	1.70	-.40	.69	
Explaining motivations for quitting smoking					
Block 1					
Community violence	.38	.14	2.49	.02	
Partner violence	.09	.13	.61	.55	
Block 2					
Community violence	.29	.16	1.92	.04	
Partner violence	.08	.17	.38	.70	
Age	.02	.23	.12	.90	
Gender	-.20	4.44	-1.09	.28	
Education	.11	1.62	.60	.55	
Initial treatment	-.03	4.19	-.16	.87	
Nicotine dependence	.06	1.16	.33	.74	
					.15 (3.34)*

Note. $*p < .05$; $**p < .01$; $***p < .001$. All outcome scores reported are change scores from intake to Time 1. Standardized beta (β) coefficients and unstandardized standard error (SE) coefficient values are reported. Final R^2 and F statistics are reported for significant models.

Table 4

Multivariate Regression Effects of Violence on Time 2 Smoking Cessation Change Scores

Predictor Variable	Beta	SE	*t*	*p*	R^2 (*F*)
Explaining cotinine scores					
Block 1					
Community violence	-.11	.02	-.64	.52	
Partner violence	.06	.02	.38	.71	
Block 2					
Community violence	-.08	.02	-.40	.69	
Partner violence	.19	.02	.88	.39	
Age	-.11	.03	-.54	.59	
Gender	-.01	.50	-.01	.10	
Education	-.14	.18	-.68	.50	
Initial treatment	.25	.47	1.33	.19	
Nicotine dependence	-.01	.13	-.04	.97	
Explaining cigarette use					
Block 1					
Community violence	.01	.09	.08	.94	
Partner violence	-.18	.08	-1.11	.27	
Block 2					
Community violence	-.03	.09	-.18	.86	
Partner violence	.14	.09	.79	.44	
Age	.20	.12	1.19	.24	
Gender	-.33	2.39	-1.99	.06	
Education	-.34	.87	-1.97	.06	
Initial treatment	.13	2.25	.83	.41	
Nicotine dependence	.22	.62	1.26	.22	
Explaining withdrawal					
Block 1					
Community violence	.12	.16	.73	.47	
Partner violence	.05	.15	.30	.77	
Block 2					
Community violence	.22	.19	1.19	.24	
Partner violence	.08	.19	.38	.71	
Age	.01	.26	.07	.94	
Gender	.11	5.06	.59	.56	
Education	-.17	1.84	-.85	.40	
Initial treatment	-.16	4.78	-.89	.38	
Nicotine dependence	.15	1.32	.75	.46	
Explaining urges					
Block 1					

Community violence	-.09	.21	-.11	.91
Partner violence	-.18	.19	-1.12	.27
Block 2				
Community violence	.05	.24	.25	.81
Partner violence	-.18	.25	-.86	.39
Age	-.17	.35	-.85	.40
Gender	.07	6.62	.35	.73
Education	-.07	2.41	-.37	.72
Initial treatment	.01	6.23	.05	.96
Nicotine dependence	.02	1.72	.09	.93
Explaining motivations for quitting smoking				
Block 1				
Community violence	.23	.20	1.46	.15
Partner violence	-.18	.18	-1.12	.27
Block 2				
Community violence	.30	.23	1.65	.11
Partner violence	-.18	.24	-.88	.39
Age	-.06	.33	-.32	.75
Gender	.12	6.28	.66	.51
Education	-.01	2.29	-.01	.99
Initial treatment	.01	5.93	.01	.10
Nicotine dependence	.23	1.63	1.18	.25
Explaining LDA				
Block 1				
Community violence	-.06	.21	-.38	.71
Partner violence	-.13	.19	-.82	.42
Block 2				
Community violence	.01	.22	.08	.94
Partner violence	.14	.22	.73	.47
Age	-.03	.31	-.14	.89
Gender	.06	5.88	.31	.76
Education	-.37	2.14	-1.99	.06
Initial treatment	.40	5.55	2.38	.02
Nicotine dependence	-.11	1.53	-.58	.57

Note. *$p < .05$; **$p < .01$; ***$p < .001$. All outcome scores reported are change scores from intake to Time 2. Standardized beta (β) coefficients and unstandardized standard error (SE) coefficient values are reported. Final R^2 and F statistics are reported for significant models.

Table 5

Multivariate Regression Effects of Mood and Life Quality on Time 1 Smoking Cessation Change Scores

Predictor Variable	Beta	SE	*t*	*p*	R^2 (*F*)
Explaining cotinine					
Block 1					
Depressive symptoms	-.14	.02	-.72	.48	
Quality of life	.06	.01	.30	.77	
Block 2					
Depressive symptoms	-.05	.02	-.23	.82	
Quality of life	.04	.01	.21	.84	
Age	-.34	.02	-1.69	.10	
Gender	-.07	.42	-.42	.70	
Education	-.05	.14	-.32	.75	
Initial treatment	.12	.40	.69	.49	
Nicotine dependence	.21	.12	1.02	.32	
Explaining cigarette use					
Block 1					
Depressive symptoms	.01	.09	.03	.98	
Quality of life	-.10	.05	-.54	.59	
Block 2					
Depressive symptoms	.09	.08	.51	.61	
Quality of life	-.08	.04	-.45	.65	
Age	.07	.10	.38	.71	
Gender	-.33	1.76	-2.18	.04	
Education	-.33	.58	-2.28	.03	
Initial treatment	.24	1.71	1.60	.12	
Nicotine dependence	.22	.51	1.29	.21	
					.36 (2.58)*
Explaining withdrawal					
Block 1					
Depressive symptoms	.41	.17	2.33	.03	
Quality of life	.05	.10	.27	.79	
Block 2					
Depressive symptoms	.46	.19	2.35	.03	
Quality of life	.06	.11	.29	.77	
Age	-.08	.24	-.43	.67	
Gender	.03	4.22	.02	.99	
Education	-.10	1.39	-.60	.56	
Initial treatment	.05	4.10	.27	.79	
Nicotine dependence	.12	1.21	.62	.54	
					.15 (3.27)*
Explaining urges					
Block 1					

Depressive symptoms	.10	.25	.52	.61
Quality of life	.28	.14	1.50	.14
Block 2				
Depressive symptoms	.06	.27	.28	.79
Quality of life	.28	.15	1.45	.16
Age	.19	.34	.93	.36
Gender	-.09	5.10	-.54	.59
Education	-.20	1.96	-1.21	.23
Initial treatment	-.09	5.79	-.53	.59
Nicotine dependence	-.07	1.71	-.34	.73
Explaining motivations for quitting smoking				
Block 1				
Depressive symptoms	.31	.18	1.70	.10
Quality of life	.17	.10	.90	.37
Block 2				
Depressive symptoms	.29	.19	1.49	.15
Quality of life	.18	.11	.94	.36
Age	.01	.24	.04	.97
Gender	-.26	4.20	-1.54	.13
Education	.22	1.38	1.34	.19
Initial treatment	.00	4.08	.03	.98
Nicotine dependence	.11	1.20	.58	.57

Note. $*p < .05$; $**p < .01$; $***p < .001$. All outcome scores reported are change scores from intake to Time 1. Standardized beta (β) coefficients and unstandardized standard error (SE) coefficient values are reported. Final R^2 and F statistics are reported for significant models.

Table 6

Multivariate Regression Effects of Mood and Life Quality on Time 2 Smoking Cessation Change Scores

Predictor Variable	Beta	SE	*t*	*p*	R^2 (*F*)
Explaining cotinine					
Block 1					
Depressive symptoms	-.10	.20	-.52	.61	
Quality of life	-.03	.01	-.13	.90	
Block 2					
Depressive symptoms	-.06	.02	-.26	.79	
Quality of life	-.04	.01	-.20	.84	
Age	-.12	.03	-.58	.57	
Gender	.05	.48	.29	.78	
Education	-.07	.16	-.41	.68	
Initial treatment	.19	.46	1.03	.31	
Nicotine dependence	-.03	.14	-.12	.90	
Explaining cigarette use					
Block 1					
Depressive symptoms	-.04	.11	-.22	.83	
Quality of life	.16	.06	.85	.40	
Block 2					
Depressive symptoms	.02	.10	.10	.92	
Quality of life	-.12	.06	-.71	.48	
Age	.16	.13	.92	.36	
Gender	-.30	2.24	-1.93	.06	
Education	-.30	.73	-1.99	.06	
Initial treatment	.11	2.17	.71	.49	
Nicotine dependence	.22	.64	1.25	.22	
Explaining withdrawal					
Block 1					
Depressive symptoms	.46	.18	2.64	.01	
Quality of life	.16	.11	.89	.38	
Block 2					
Depressive symptoms	.54	.20	2.89	.01	
Quality of life	.19	.11	1.06	.30	
Age	-.08	.25	-.41	.68	
Gender	.07	4.32	.43	.67	
Education	-.10	1.42	-.64	.53	
Initial treatment	-.10	4.20	-.59	.56	
Nicotine dependence	.28	1.24	1.47	.15	
					.16 (3.62)*
Explaining urges					
Block 1					
Depressive symptoms	.04	.25	.23	.82	

Quality of life	.26	.14	1.39	.17
Block 2				
Depressive symptoms	.08	.28	.39	.70
Quality of life	.25	.15	1.24	.23
Age	-.13	.35	.27	.79
Gender	.03	6.14	.16	.88
Education	-.14	2.01	-.81	.43
Initial treatment	.05	5.97	.27	.79
Nicotine dependence	.04	1.76	.18	.86
Explaining motivations for quitting smoking				
Block 1				
Depressive symptoms	.04	.26	.21	.84
Quality of life	-.07	.15	-.36	.72
Block 2				
Depressive symptoms	.14	.28	.65	.52
Quality of life	-.03	.15	-.16	.87
Age	-.06	.35	-.31	.76
Gender	.00	6.14	.02	.99
Education	-.01	2.01	-.08	.94
Initial treatment	.10	5.97	.54	.59
Nicotine dependence	.26	1.76	1.23	.23
Explaining LDA				
Block 1				
Depressive symptoms	-.12	.25	-.65	.52
Quality of life	-.16	.14	-.86	.40
Block 2				
Depressive symptoms	-.06	.25	-.29	.77
Quality of life	-.19	.14	-1.03	.31
Age	-.06	.31	-.30	.77
Gender	.07	5.47	.43	.67
Education	-.31	1.79	-1.98	.06
Initial treatment	.38	5.3	2.33	.03
Nicotine dependence	-.12	1.57	-.65	.52

Note. $*p < .05$; $**p < .01$; $***p < .001$. All outcome scores reported are change scores from intake to Time 2. Standardized beta (β) coefficients and unstandardized standard error (SE) coefficient values are reported. Final R^2 and F statistics are reported for significant models.

Table 7

Moderating Effects of Social Support on the Relationship between Violence and Time 1 Change in Cigarette Use

Predictor Variable	Beta	SE	*t*	*p*	R^2 (*F*)	Δ*F*
Community violence						
Block 1						
Social support	-.02	.07	-.11	.91		
Community violence	-.04	.04	-.22	.83		
					.01	.03
Block 2						
Social support	-.02	.04	-.13	.90		
Community violence	-.06	.07	-.34	.73		
Age	.11	.10	.66	.52		
Gender	-.34	1.87	-2.15	.04		
Education	-.30	.61	-1.99	.06		
Initial treatment	.22	1.75	1.46	.15		
Nicotine dependence	.19	.50	1.10	.28		
					.34 (2.40)*	3.35*
Block 3						
Social support	-.48	.13	-1.00	.32		
Community violence	-1.02	.42	-1.06	.30		
Age	.08	.10	.46	.65		
Gender	-.37	1.89	-2.27	.03		
Education	-.30	.61	-1.98	.06		
Initial treatment	.25	1.77	1.60	.12		
Nicotine dependence	.18	.50	1.04	.31		
Soc. support by Comm. violence	.96	.00	1.01	.32		
					.37 (2.23)*	1.03
Intimate partner violence						
Block 1						
Social support	-.04	.04	-.27	.79		
Intimate partner violence	-.37	.06	-2.44	.02		
					.14 (2.10)	2.10
Block 2						
Social support	-.01	.04	-.05	.10		
Intimate partner violence	-.09	.07	-.49	.62		
Age	.10	.10	.60	.56		
Gender	-.31	1.83	-1.10	.04		
Education	-.28	.67	-1.67	.11		
Initial treatment	.19	1.81	1.23	.23		
Nicotine dependence	.19	.50	1.11	.28		
					.35 (2.43)*	2.03
Block 3						
Social support	-.23	.06	-.95	.35		

Intimate partner violence	-1.16	.38	-1.24	.22		
Age	.11	.10	.64	.53		
Gender	-.32	1.82	-2.08	.04		
Education	-.27	.67	-1.63	.11		
Initial treatment	.20	1.81	1.28	.21		
Nicotine dependence	.17	.50	1.01	.32		
Soc. support by Part. violence	1.10	.00	1.17	.25		
					.37 (2.32)*	1.36

Note. *p < .05; **p < .01; ***p < .001. All outcome scores reported are change scores from intake to Time 1. Standardized beta (β) coefficients and unstandardized standard error (SE) coefficient values are reported. R^2, F, and F change statistics are reported.

Table 8

Moderating Effects of Social Support on the Relationship between Depressive Symptoms and Time 1 Change in Withdrawal

Predictor Variable	Beta	SE	*t*	*p*	R^2 (*F*)	Δ*F*
Depressive Symptoms						
Block 1						
Social support	-.05	.10	-.30	.77		
Depressive symptoms	.36	.17	2.10	.04		
					.15 (3.28)*	3.28*
Block 2						
Social support	-.02	.11	-.11	.92		
Depressive symptoms	.43	.19	2.14	.04		
Age	-.09	.24	-.44	.66		
Gender	.01	4.24	.02	.99		
Education	-.10	1.39	-.59	.56		
Initial treatment	.05	4.13	.29	.78		
Nicotine dependence	.11	1.25	.55	.59		
					.17 (.95)	.17
Block 3						
Social support	-.32	.21	-.81	.42		
Depressive symptoms	-.42	.96	-.42	.68		
Age	-.13	.25	-.64	.53		
Gender	-.06	4.58	-.30	.76		
Education	-.11	1.40	-.68	.50		
Initial treatment	.05	4.15	.27	.79		
Nicotine dependence	.14	1.27	.68	.51		
Soc. support by Dep. Symptoms	.77	.01	.88	.39		
					.19 (.93)	.77

Note. *p < .05; **p < .01; ***p < .001. All outcome scores reported are change scores from intake to Time 1. Standardized beta (β) coefficients and unstandardized standard error (SE) coefficient values are reported. R^2, F, and F change statistics are reported.

Table 9

Moderating Effects of Social Support on the Relationship between Depressive Symptoms/Life Satisfaction and Time 1 Change in Cigarette Use

Predictor Variable	Beta	SE	*t*	*p*	R^2 (*F*)	Δ*F*
Depressive Symptoms						
Block 1						
Social support	-.01	.05	-.04	.97		
Depressive symptoms	.05	.09	.29	.78		
					.01 (.06)	.06
Block 2						
Social support	.08	.04	.45	.66		
Depressive symptoms	.17	.08	.95	.35		
Age	.06	.10	.36	.72		
Gender	-.33	1.77	-2.20	.04		
Education	-.33	.58	-2.30	.03		
Initial treatment	.23	1.72	1.53	.14		
Nicotine dependence	.25	.52	1.41	.17		
					.36 (2.57)*	3.57*
Block 3						
Social support	-.16	.09	-.48	.64		
Depressive symptoms	-.51	.40	-.59	.56		
Age	.03	.11	.15	.88		
Gender	-.38	1.91	-2.33	.03		
Education	-.34	.59	-2.35	.03		
Initial treatment	.23	1.74	1.50	.14		
Nicotine dependence	.28	.53	1.51	.14		
Soc. support by Dep. Symptoms						
					.37 (2.31)*	.64
Life satisfaction						
Block 1						
Social support	.04	.05	.19	.85		
Life satisfaction	-.13	.05	-.64	.53		
					.01 (.22)	.22
Block 2						
Social support	.09	.05	.50	.62		
Life satisfaction	-.17	.05	-.95	.35		
Age	.07	.10	.44	.66		
Gender	-.34	1.78	-2.24	.03		
Education	-.33	.58	-2.29	.03		
Initial treatment	.22	1.72	1.45	.16		
Nicotine dependence	.21	.50	1.26	.22		
					.36 (2.57)*	3.48*
Block 3						
Social support	.08	.05	.47	.64		

Life satisfaction	.66	.24	.75	.46		
Age	.04	.10	.24	.81		
Gender	-.38	1.84	-2.40	.02		
Education	-.35	.59	-2.43	.02		
Initial treatment	.22	1.72	1.46	.16		
Nicotine dependence	.24	.50	1.40	.17		
Soc. support by Life satisfaction	-.85	.01	-.95	.35		
					.38 (2.36)*	.90

Note. *$p < .05$; **$p < .01$; ***$p < .001$. All outcome scores reported are change scores from intake to Time 1. Standardized beta (β) coefficients and unstandardized standard error (SE) coefficient values are reported. Final R^2, F, and F change statistics are reported.

Table 10

Moderating Effects of Social Support on the Relationship between Depressive Symptoms and Time 2 Change in Withdrawal

Predictor Variable	Beta	SE	*t*	*p*	R^2 (*F*)	Δ*F*
Depressive Symptoms						
Block 1						
Social support	-.09	.10	-.54	.60		
Depressive symptoms	.34	.18	1.97	.04		
					.15 (3.32)*	3.32*
Block 2						
Social support	-.01	.11	-.07	.94		
Depressive symptoms	.44	.20	2.28	.03		
Age	-.09	.25	-.47	.65		
Gender	.07	4.41	.40	.69		
Education	-.10	1.44	-.65	.52		
Initial treatment	-.09	4.29	-.55	.59		
Nicotine dependence	.25	1.30	1.28	.21		
					.22 (1.32)	.60
Block 3						
Social support	-.36	.22	-.97	.34		
Depressive symptoms	-.55	.99	-.58	.57		
Age	-.14	.26	-.72	.48		
Gender	-.01	4.73	-.02	.98		
Education	-.12	1.45	-.76	.46		
Initial treatment	-.10	4.29	-.57	.57		
Nicotine dependence	.29	1.31	1.44	.16		
Soc. support by Dep. Symptoms	.91	.01	1.07	.29		
					.25 (1.31)	1.14

Note. *$p < .05$; **$p < .01$; ***$p < .001$. All outcome scores reported are change scores from intake to Time 1. Standardized beta (β) coefficients and unstandardized standard error (SE) coefficient values are reported. Final R^2, F, and F change statistics are reported.

Table 11

Selected Item Endorsements of Community Violence Exposure on Things I Have Seen and Heard

	Prevalence (%)	
Item	**At least once**	**Never**
Heard guns being shot	87.5	12.5
Seen somebody arrested	90	10
Seen drug deals	82.5	17.5
Seen somebody being beat up	82.5	17.5
Been beaten up	62.5	37.5
Seen somebody get stabbed	42.5	57.5
Seen somebody get shot	55	45
Seen a gun in my home (not my own gun)	35	65
Seen drugs/drug paraphernalia (items used to administer drugs) in my neighborhood	85	15
Somebody threatened to kill me	30	70
Seen a dead body outside	45	55
Somebody threatened to shoot me	35	65
Somebody threatened to stab me	30	70
Seen somebody in my home get shot or stabbed	15	85

Note. One-hundred percent of participants reported at least one community violence event. Above items represent selected items from the Things I Have Seen and Heard. Reverse-scored items are not presented.

Table 12

Selected Item Endorsements of Intimate Partner Violence Victimization on Revised Conflict Tactics Scale

	Past-year Prevalence (%)	
Item	**At least once**	**Never**
My partner insulted or swore or shouted or yelled at me	57.5	42.5
Had a sprain, bruise, or small cut, or felt pain the next day because of a fight with my partner	20	80
My partner pushed, shoved, or slapped me	17.5	82.5
My partner punched or kicked or beat me up	15	85
My partner destroyed something belonging to me or threatened to hit me	12.5	87.5
Went see a doctor (M.D.) or needed to see a doctor because of a fight with my partner	10	90
My partner used force (like hitting, holding down, or using a weapon) to make me have sex	10	90
My partner insisted on sex when I did not want to or insisted on sex without a condom (but did not use physical force)	10	90

Note. Sixty percent of participants reported at least one intimate partner violence victimization event. Above items are violence victimization items from Revised Conflict Tactics Scale (Short Form) presented in order of item severity.

Table 13

Selected Item Endorsements of Intimate Partner Violence Perpetration on Revised Conflict Tactics Scale

	Past-year Prevalence (%)	
Item	**At least once**	**Never**
I insulted or swore or shouted or yelled at my partner	65	35
My partner had a sprain, bruise, or small cut, or felt pain the next day because of a fight with me	17.5	82.5
I pushed, shoved, or slapped my partner	20	80
I punched or kicked or beat up my partner	10	90
I destroyed something belonging to my partner or threatened to hit my partner	20	80
My partner went see a doctor (M.D.) or needed to see a doctor because of a fight with me	10	90
I used force (like hitting, holding down, or using a weapon) to make my partner have sex	12.5	87.5
I insisted on sex when my partner did not want to, or insisted on sex without a condom (but did not use physical force)	10	90

Note. Sixty-eight percent of participants reported at least one intimate partner violence perpetration event. Above items are violence perpetration items from Revised Conflict Tactics Scale (Short Form) presented in order of item severity.

Table 14

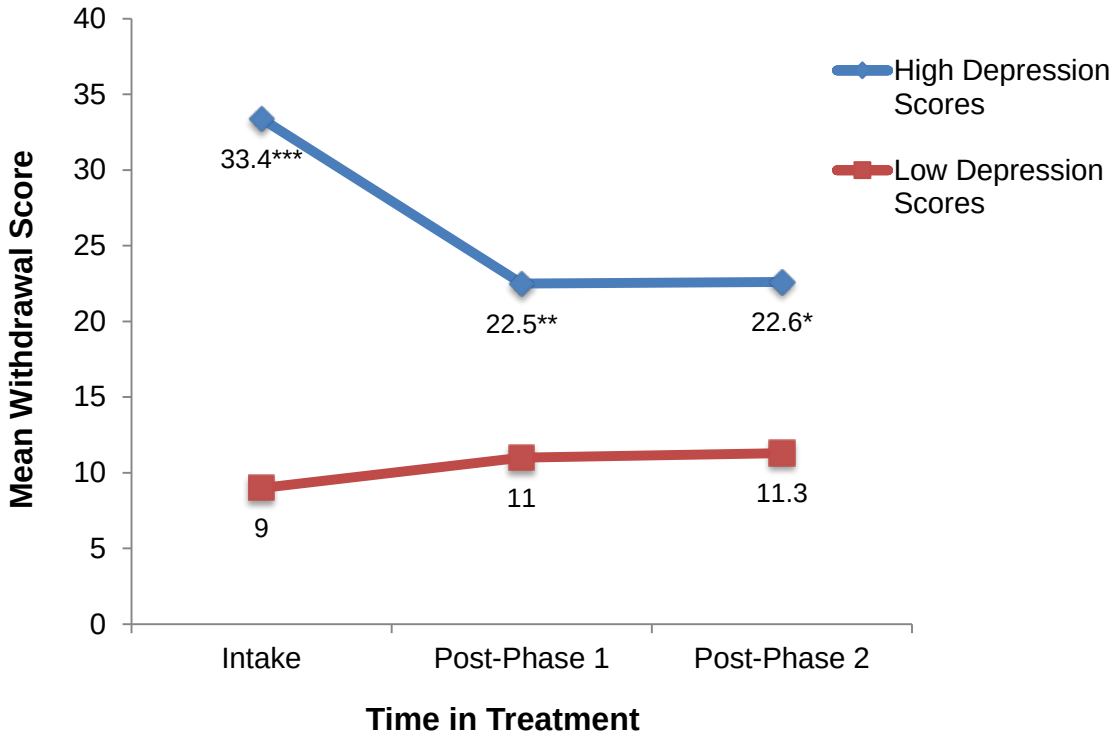
Mean Reports of Life Satisfaction in 17 Areas of Life Functioning

Life Area	Mean	SD
Standard of Living	0.05	1.10
Work	0.08	1.99
Health	0.20	1.76
Neighborhood	0.33	1.85
Civic Action	0.45	1.95
Community	0.58	1.77
Recreation	0.67	1.88
Creativity	0.83	1.85
Love Relationships	0.85	2.06
Home	0.90	1.85
Self-regard	1.03	1.69
Learning	1.13	1.61
Philosophy of Life	1.18	1.52
Relationships with Relatives	1.23	1.75
Social Service	1.25	1.66
Friendships	1.48	1.72
Relationships with Children	1.60	1.50
Total score	*13.75*	*21.61*

Note. Items are from the Quality of Life Inventory, presented in ascending order of satisfaction (i.e., lowest mean satisfaction scores to highest). Items are scored on a scale from -3 to 3, with higher scores indicate greater life satisfaction.

Figure 1

Reductions in Nicotine Withdrawal by High/Low Depression Scores



Note. Group means differed significantly at each time point at the following levels: *$p < .05$; **$p < .01$; ***$p < .001$. Post-Phase 1 and Post-Phase 2 values represent raw values (not change-scores) at each time point. Change score values at each time point also differed significantly between high and low depression scorers.

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ABSTRACT

THE MODERATING EFFECTS OF PROTECTIVE AND RISK FACTORS ON OUTCOMES FOR BEHAVIORAL SMOKING CESSATION TREATMENT

by

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Major: Clinical Psychology

Degree: Doctor of Philosophy

The rate of cigarette smoking is three-fold higher among adults living with Human Immunodeficiency Virus (HIV)/Autoimmune Deficiency Syndrome (AIDS) than in the general population (Centers for Disease Control and Prevention, 2015). Relative to non-smoking HIV-positive adults, HIV-positive cigarette smokers have even higher mortality rates, more physical health problems, greater tobacco-related health disparities, lower quality of life, and more barriers to treatment. These barriers are often interrelated with the significantly higher rate of trauma and violence exposure reported in both cigarette smokers and persons living with HIV/AIDS (PLWHA; CDC). Violence exposure not only predicts emotional distress and substance use, but also mediates between victimization and cigarette use (e.g., Feldner, Babson, & Zvolensky, 2007). Psychological and physical trauma additionally impact both short and long-term substance use treatment outcomes, independent of treatment modality (e.g., Ford et al., 2007). Though social support is consistently linked to both smoking behaviors, and mental and behavioral outcomes for PLWHA, few studies have examined the role of social support in smoking treatment among this population. Additionally, no studies have explored social and

individual-level protective and risk factors in the context of a contingency management smoking cessation treatment design. The aim of this study was to examine how different risk and protective factors affect cessation outcomes among PLWHA undergoing contingency-based treatment for cigarette smoking. Specifically, this study explored how different components of violence (community and interpersonal partner violence), distress, life satisfaction, and social support affect cigarette use and cessation success. Study findings showed that intimate partner violence predicted increased initial change in cigarette use, while community violence predicted reduced initial motivations for quitting smoking. Increased depression scores predicted both initial and total treatment change in withdrawal experience. Findings have implications for the assessment of and therapeutic approaches to smoking cessation for smokers living with HIV/AIDS.

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EDUCATION AND TRAINING

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M.A. Clinical Psychology **2012**
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