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Informing integrated interventions for trauma and smoking:
The role of gender from childhood to adulthood

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Abstract

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Integrated interventions for trauma and substance use often overlook smoking despite evidence that interpersonal traumas and smoking are related. When integrated treatments are specifically designed to address smoking and trauma they are based upon primarily male samples with combat related stressors and it remains unclear if these interventions will be as effective with women who experience differing forms of trauma and have different risk factors for smoking.

This dissertation examined the life course and potentially, gendered relationship between child maltreatment, partner violence victimization and smoking behavior across four developmental periods (childhood, adolescence, young adulthood and adulthood) by capitalizing on existing longitudinal data from the Seattle Social Development Project, a 30-year prospective cohort study. Chapter 1 investigated whether a commonly used measure of partner violence, the Conflict Tactics Scale, is consistent in its measurement of victimization across gender and time. Chapter 2 explored gender similarities and differences in the relationship between partner violence and smoking behavior in young adulthood and adulthood (ages 24 to 33). Chapter 3 examined the influence of child maltreatment on smoking onset, adolescent smoking duration

and becoming a high-risk smoker in young adulthood in the context of other risk factors among those people who have ever smoked and whether this differs for men and women.

Findings suggest that interpersonal traumas and smoking are consistently related from childhood to adulthood, for both men and women, and remain significant even after controlling for important risk factors. Directional findings support a bidirectional relationship where interpersonal traumas influence a wide array of smoking behaviors (onset, adolescent duration, smoking type and high risk smoking in young adulthood and adulthood) and smoking in young adulthood uniquely predicts increased risk for later interpersonal victimization in adulthood. The strong connection between interpersonal traumas, a prominent theme in the lives of women, and the identification of gender-specific risk factors for smoking underscore the need for considering gender in future integrated intervention development. Collectively, findings provide information of use to those designing gender-informed primary and secondary prevention efforts which ultimately will contribute to reducing the large public health burdens of both smoking and interpersonal violence.

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DEDICATION

This work is dedicated to my family who is the only constant in life's ever-changing adventure. Mom and Dad, this dissertation would not have been possible without your love, sacrifice and unconditional belief in me. John, you are my best friend and the definition of partner, with you by my side anything is possible. And to Ender, you are too young to read this but know that you are in every word and every line of this dissertation, just as surely as you are in every beat of my heart.

INTRODUCTION

Smoking is a serious public health burden. Tobacco use kills more people than the use of any other drug and is the leading cause of morbidity and mortality among adults in the United States (Mokdad, Marks, Stroup, & Gerberding, 2004; Campaign for Tobacco-Free Kids, 2012; Chassin, Presson, Rose, Sherman, & Prost, 2002). National data indicate that use of tobacco, like other drugs, begins in adolescence and peaks in young adulthood (age 21 to 24), but, unlike other drugs, levels of tobacco use do not decrease noticeably throughout adulthood (Delucchi & Weisner, 2010; Dodgen, 2005; Substance Abuse and Mental Health Services Administration, 2004). Despite a significant decline in smoking prevalence during the '80s, this decline has stalled. In particular, women's smoking rates have declined more slowly than men's and women's smoking-related mortality continues to rise (U.S. Department of Health and Human Services, 2014), which may be a sign that women have been less receptive to the smoking prevention and cessation efforts to date (Warner & Mendez, 2010). New insights into the etiology of smoking are needed.

One factor that may influence these gender differences in the etiology of smoking is the experience of interpersonal trauma. Interpersonal trauma itself is a significant public health issue (Black et al., 2011; White, 2009) and is inherently gendered, with differences in the prevalence rates, types of violence, and consequences. Men are more likely to report combat-related violence, whereas women are more likely to report experiencing interpersonal trauma, such as childhood sexual abuse, and are also more likely to receive a trauma-related diagnosis, such as Post Traumatic Stress Disorder (PTSD) (Hien, Cohen, & Campbell, 2005; Kessler, Sonnega, Bromet, Hughes, & Nelson, 1995). Empirical studies have consistently shown that violent

victimization, the resulting trauma, and substance use are related, and the experience of interpersonal trauma has been suggested as a reason for gender disparities in substance use outcomes (Widom, Ireland, & Glynn, 1995). Interpersonal trauma may be specifically relevant for smoking behavior; however, much less is known about *the role of gender in the link between interpersonal traumas and smoking* as compared with other substances (Hien et al., 2005; Lansford, Dodge, Pettit, & Bates, 2010; Widom, Marmorstein, & White, 2006; Kristman-Valente & Wells, 2013; Spratt et al., 2009; Crane, Hawes, & Weinberger, 2013).

Studies have connected stressful life events such as interpersonal trauma, and trauma-related disorders with increased smoking behavior, resistance to quitting, and failure to quit (McFall et al., 2010; Pomerlau & Pomerlau, 1991, Rheingold, Acierno, & Resnick, 2004). Strong empirical support exists for a connection between interpersonal traumas and alcohol (O'Leary & Schumacher, 2003) and drugs (Kilpatrick, Acierno, Resnick, Saunders, & Best, 1997; Testa, Livingston, & Leonard, 2003); though these studies have largely excluded smoking behavior from analysis. Since adolescent smoking is highly comorbid with other substance use at any age and smoking is predictive of engaging in harder drug use later in life (U.S. Department of Health and Human Services, 2012), it is important to better understand the unique contribution of interpersonal traumas and smoking and if this differs for women and men, in order to better target prevention and cessation efforts for these two critical social issues. Understanding the role of interpersonal traumas, in conjunction with other risk factors for smoking, is particularly important among women who are more likely to receive a diagnosis of Post-Traumatic Stress Disorder (PTSD) based on interpersonal traumas compared to men (Hien et al., 2005) and since it remains unclear which smoking interventions are most effective for women (Singleton, Levin, Feldman, & Truglio-Londrigan, 2005; Warner & Mendez, 2010). However, few studies have

investigated gender differences in the relationship between interpersonal violence victimization experience and smoking.

One complication to investigating the relationship between interpersonal trauma and other factors including smoking is the difficulty in assessing the experience of victimization particularly within an intimate partner context. Problems measuring partner violence victimization may impede the development of effective interventions that target risk factors contributing to intimate partner violence. As a result a call for better, more reliable, and valid assessment of partner violence victimization has been made in the violence literature (Ehrensaft, 2008; Follingstad & Rogers, 2013) with particular attention placed on whether the construct of partner violence victimization is measured the same across men and women and means the same thing over time.

One of the most commonly used assessment tools for evaluating the perpetration and victimization experience of partner violence is the Conflict Tactics Scale (CTS) (Straus, 1979, 1990). Despite a great deal of measurement work on the CTS, the majority of psychometric evaluation has been focused on the underlying factor structure of violence as assessed by the CTS, and the reliability and validity of items. Few studies investigate if the CTS is measuring the same construct in men and in women and if this construct is consistent over time. This is problematic because without establishing measurement invariance across gender and over time researchers who investigate gender differences or the changing risk for partner violence over time cannot have confidence that findings are not due to measurement error. Thus, establishing multi-group measurement and temporal invariance in the CTS, the most widely used measure of partner violence, will add to existing psychometric work, increase confidence in research findings, and ultimately promote the prevention and intervention of partner violence by ensuring

that intervention efforts are targeting those risk factors that are universal or specific for men and women and over time.

Partner violence victimization is a public health issue in its own right independent of smoking, though the combination of the two may exacerbate the other. Partner violence victimization has been associated with smoking behavior using cross-sectional studies (El-Mohandes, El-Khorazaty, Kiely, & Gantz, 2011; Dichter, Cerulli, & Bossarte, 2011; Vest, Catlin, Chen, & Brownson, 2002; Crane et al., 2013), but little is known about how the two issues influence each other across time or if the relationship differs for men and women. Research that has examined the relationship between interpersonal traumas, and substance use in general (including, but not limited to smoking) suggests that the direction of the relationship between interpersonal trauma and substance use may differ for women and men. There is evidence that substance use leads to perpetration of violence, including partner conflict and sexual and physical assault among men (Kantor & Asdigian, 1997) while *substance use as a consequence of interpersonal victimization* has been better supported for women (Testa, 2004). Further, a longitudinal study by Kilpatrick and colleagues (1997) found that women's drug use (but not alcohol use) led to later sexual and physical victimization, which in turn led to increased substance use indicating a reciprocal influence between the two. Research exploring the link between interpersonal trauma and substance use has also indicated that the relationship may be more robust for women over time, and that the substance-use-related consequences of interpersonal trauma continue across more developmental periods for women than for men (Lansford, Dodge, Pettit, & Bates, 2010; Widom, Marmorstein, & White, 2006; Widom et al., 1995; Widom & White, 1997; Widom, White, Czaja, & Marmorstein, 2007). Thus, especially for women, the relationship between substance use and interpersonal trauma, including partner

violence victimization, may be bi-directional and substance specific but it is not known if this extends to smoking. A better understanding of the role that gender plays in the etiology of smoking and partner violence victimization and how these two behaviors influence each other across time is needed in order to inform new intervention efforts.

It will also be important to better understand how early interpersonal victimization influences proximal smoking behavior (i.e., onset and duration) and more distal high-risk smoking behavior and if this differs for men and women since reaching age 18 without having used tobacco is associated with reduced likelihood of ever smoking, fewer health consequences, and a lower likelihood of becoming addicted to nicotine (U.S. Department of Health and Human Services, 2012). A limited subset of researchers have studied gender differences in the development of smoking behavior or the role that child maltreatment plays in the onset and escalation of smoking among those people who have smoked. In general, research shows that girls become regular smokers later than boys do, and this delay has been attributed in part to more restrictive social norms and fewer risk factors for smoking among young girls compared to boys (Robinson & Klesges, 1997). As boys and girls age, risk factors and social norms surrounding them change, resulting in similar smoking rates between women and men by young adulthood. However, one study found that among women, the experience of early child physical and sexual abuse was associated with early smoking onset (by age 14), suggesting that early interpersonal victimization experience negates some of the protective factors that might otherwise delay smoking in young women's lives (Jun, Rich-Edwards, Boynton-Jarrett, Austin, Frazier, & Wright, 2008). This finding may also point to child maltreatment acting as a pivotal risk factor for smoking-susceptible adolescents, that is, those who are already at increased risk for smoking initiation and experimentation (Unger et al., 1997) and uniquely contribute to the

development of high-risk smoking behavior. More research is needed to identify early risk factors that are malleable to change among high-risk smokers and if these risks are salient for both genders.

This dissertation research seeks to better understand the role of gender in the relationship between multiple forms of interpersonal trauma and smoking. Specifically, the dissertation examines how the link between interpersonal trauma experiences (including child maltreatment and partner violence victimization) and smoking behavior (onset, duration and high-risk smoking) varies in different developmental periods (childhood-adolescence, young adulthood, adulthood) for women and men, and the extent to which interpersonal violence victimization is a unique risk factor for smoking behavior across time in the context of other risk factors (i.e., socio-economic status and other drug use). To accomplish this, Chapter 1 presents an investigation of the measurement invariance of a commonly used measure of partner violence victimization to determine adequacy of assessment between genders and over time. In Chapter 2, the relationship between partner violence victimization and smoking behavior in young adulthood and adulthood between ages 24 and 33, and how this is different or similar for men and women is examined. Lastly, Chapter 3 explores the influence of early victimization in childhood predicts onset and adolescent smoking duration and becoming a high-risk smoker in young adulthood among those people who have ever smoked and if this is different for men and women.

Gender differences and similarities in the link between interpersonal traumas and smoking behavior will be examined using existing data from the Seattle Social Development Project (SSDP), a sample of 412 men and 396 women who have been followed from childhood (age 10) to adulthood (age 33). The SSDP data is ideal for studying the role of gender in the

victimization-smoking behavior link because it includes comprehensive longitudinal measures of smoking onset, frequency, and patterns of use, multiple measures of victimization, including childhood maltreatment and partner conflict and is gender-balanced which provides the necessary statistical power to test the proposed hypotheses.

This study seeks to enhance current understanding of the role gender plays in the relationship between various forms of interpersonal traumas and smoking from childhood to adulthood and will contribute to the primary prevention of smoking uptake and secondary smoking cessation efforts for those individuals with comorbid smoking and interpersonal trauma experience and trauma-related diagnosis, a sub-population of smokers at higher risk for smoking onset and escalation and who are increasingly resistant to smoking cessation (Hapke et al., 2005). Finally, this study seeks to provide insight into gender-based constructs and risk factors by examining the extent to which the relationship between interpersonal traumas and smoking differ for women and men, thus contributing to the design of better gender-responsive strategies for reducing the tobacco burden.

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CHAPTER 1. MEASURING PARTNER VIOLENCE VICTIMIZATION ACROSS TIME AND BETWEEN GENDERS

1. Introduction

Partner violence victimization remains a significant public health problem in the United States (White, 2009), where approximately 36% of women and 29% of men report experiencing some form of violence at the hands of an intimate partner in their lifetime. These percentages are largely considered to be an underestimate of the phenomenon and are plagued by measurement issues (Black et al., 2011; Emery, 2010). Being a victim of partner violence has been associated with several negative outcomes both proximally and distally (Breiding, Black, & Ryan, 2008; Coker et al., 2002; Black et al., 2011). For example, people reporting partner violence victimization have long-term negative health effects, including being more likely to develop a chronic disease and mental illness (Coker et al., 2002). Further, people reporting partner violence victimization are more likely to engage concurrently in HIV risk behaviors and substance use, and report impaired physical health and increased rates of depression (Breiding, Black, & Ryan, 2008, Bonomi et al., 2006; Coker et al., 2002). There is evidence that the negative outcomes resulting from partner violence victimization differ for men and women. Research has found that women sustain worse outcomes from domestic violence victimization compared to men (Romito & Grassi, 2007; Black et al., 2011).

Even more troubling than the prevalence and outcome of partner violence victimization is the relative ineffectiveness of current interventions and prevention efforts for partner violence (Whitaker et al., 2006; Babcock, Green, & Robie, 2004). New insights into partner violence victimization are needed in order to inform the development of new prevention and intervention efforts in order to reduce the impact of this public health problem.

Despite the importance of this problem and the need for new treatment approaches, partner violence victimization remains a topic that is rarely spoken about in public, and as a result it is difficult to assess. Yet the assessment of partner violence victimization in marital, dating, or cohabiting relationships plays an important role in the development of effective intervention and in furthering our understanding of the precursors and correlates of the behavior. Valid and reliable assessment tools are important clinically to evaluate the needs and resources of clients and epidemiologically to aid in determining the prevalence and experience of partner violence victimization, which is a key indicator of social wellbeing. A better understanding of the applicability, stability and limitations of current measures of partner violence victimization are necessary for research that investigates the relationship between partner violence victimization and other factors particularly for research targeting the development of interventions (Pentz & Chou, 1994). Specifically, it has been noted that more research is needed to establish if existing measures that evaluate partner violence victimization is measuring the same construct across men and women and over time (Ehrensaft, 2008; Follingstad & Rogers, 2013).

1.1 Measuring Intimate Partner Violence

One of the most commonly used assessment tools for evaluating the perpetration and victimization experience of partner violence is the Conflict Tactics Scale (CTS) (Straus, 1979, 1990). The primary goal of the CTS is to evaluate a person's subjective experience of violence in his or her life in two primary forms: perpetration (the act of committing the violence against another) or victimization (being the recipient of acts of violence by another). Originally, the CTS was developed to assess partner conflict across multiple familial relationships, from husband-wife dyadic exchanges to parent-child interactions. The CTS has since been used to assess partner violence in adolescent dating scenarios, same-sex couples, college samples, incarcerated

women, and in studies focused specifically on African American and Latino communities (Straus, Hamby, Boney-McCoy, & Sugarman, 1996; Connelly, Newton, & Aarons, 2005). The CTS has been administered in over 20 countries, and it is utilized across multiple fields such as criminology, social work, psychology, and human services (Dwyer, 1999; Straus et al., 1996). Though it has been used in multiple capacities, the primary use remains the assessment of heterosexual husband-wife conflict assessment (Straus, 2004).

The original form of the CTS measured conflict negotiation tactics employed during partner conflict and subjective experiences of verbal and physical conflict (Straus, 1990). Later revisions of the instrument included subscales of sexual violence and injury resulting from conflict. The revised CTS, referred to as the CTS2, includes the following scales: 1. Psychological-Verbal Aggression Scale, 2. Physical Assault Scale, 3. Injury Scale, and 4. Sexual Coercion Scale (Straus et al., 1996). Questions are used to assess the degree to which the individual has experienced or perpetrated differing forms of violence and the frequency at which the violence occurs. Both the CTS and the CTS2 have good internal reliability, ranging from Cronbach's alphas of .62 (verbal aggression) to .88 (physical aggression) in the original CTS (Straus, 1990) and .79 (verbal aggression) to .86 (physical assault) for the CTS2 (Straus et al., 1996; Straus, 2004). The CTS has reported concurrent, construct, and internal validity (Straus, 1990, Straus et al., 1996, Straus, 2004).

The CTS has been used to assess differences of partner violence experience across multiple subgroups, including military men and women (Pan, Neidig, & O'Leary, 1994), cross-cultural differences in partner violence (Nocentini, Menesini, Pastorelli, Connolly, Pepler & Craig, 2011; Straus, 2004), and racial and ethnic group differences in the experience of IPV (Connelly et al., 2005). The widespread application of the CTS to compare groups operates under the assumption

that the assessment works equally well in assessing the construct of partner violence across subpopulations.

Establishing measurement invariance for the CTS has been limited in application despite its widespread use in comparing groups and investigating correlates of IPV. When psychometric work is done, most studies to date exploring group-based differences, including gender, in the CTS/CTS2 have focused primarily on varying factor structures of the violence construct across groups (Nocentini et al., 2011; Viejo, Sanchez, & Ortega-Ruiz, 2014; Schafer, 1996; Connelly et al., 2005), but have not formally tested other aspects of measurement invariance, such as metric, scalar or temporal invariance, necessary to establish psychometric equivalence.

The physical violence subscale, the most widely used scale from the CTS/CTS2 (Straus et al., 1996), has demonstrated the most inconsistency in measuring physical partner conflict between groups. For example, in one study while all other subscales of the CTS2 were similar, the physical violence subscale was found to have different factor structures across language groups when tested among Spanish- vs. English-speaking Latina women (Connelly et al., 2005). Similarly, using a sample of college age students in the U.S. Schafer (1996) found in a confirmatory factor analysis that a one-dimensional model fit well for women but not for men, indicating that the structure of the latent factor of physical partner violence perpetration differed by gender when violence items were restricted to perpetration responses, self-to-spouse directionality. The authors concluded that the differing factor structure of physical partner violence perpetration may suggest underlying gender-based differential item responses to items from the original CTS, which was tested using a multiple group analysis and found to be significantly different. It is not known if this finding of gender difference in response to items on

the original CTS form, focused on perpetration, will be found in different samples, when focused on victimization, or when the revised CTS2 is used.

Establishing construct and measurement equivalency between groups and across time is an important step for quantitative violence research that is rarely done but represents progress for the field on multiple levels. On a broad level, investigation into measurement invariance acknowledges the diversity of individuals exposed to violence, honors the potential that there may exist differences in how measured items are interpreted and responded to, and explores the complexity of the violence construct over time. On a direct service level, the examination of measurement invariance enhances confidence in research findings. A primary focus of violence research is to understand the risk for and pathways to violence exposure so that prevention and intervention efforts can be implemented in order to reduce experiences of violence. Yet, what if items from a violence measure elicit differing responses from different groups? What if the construct of violence looks different across distinct periods in a person's life? When researchers neglect to assess if the violence measure is equivalent across groups or if the construct is the same across time, findings from the research and subsequent conclusions may be missing group or temporal differences. Thus, identifying differences, even on the measurement or construct level, could inform tailored prevention and intervention efforts (Catalano et al., 1993; Harachi, Choi, Abbott, Catalano, & Bleisner, 2006).

1.2 The Potential Role of Gender

One issue of particular interest and controversy in the intimate partner violence literature is whether there are gender differences in the experience of partner violence victimization. Critics of the CTS have argued that the CTS structure is the reason for findings of gender symmetry in violence victimization and perpetration (Chan, 2011; Giles, 2004, Kimmel, 2002). This criticism

involves an underlying belief that the CTS is not capturing the same construct of partner violence for men and women. It has also been suggested that the use of the CTS in specific samples influences the identification of gender differences in partner violence. Meta-analytic studies have found that studies using the CTS with community samples tend to find gender symmetry, whereas studies using the CTS with clinical samples have more often found differences in partner violence, including severity level differences and injury consequence, between men and women (Chan, 2011). However, studies of clinical samples, such as that of Tetan, Sherman, and Han (2009), have found gender symmetry, and studies of community samples, such as the one by Tjadens and Thoennes (2000), have found gender differences in partner violence, leaving us unclear on the role of measurement in identifying gender differences in partner violence.

1.3 Developmental Change in Partner Conflict

Another measurement consideration for assessing partner violence in men and women is the potentially changing meaning of partner violence over time. Existing literature suggests that the prevalence and typology of partner violence changes developmentally (Mezey, Post, & Maxwell, 2002; Capaldi, Shortt, & Kim, 2005), with domestic violence incidences peaking in late adolescence to early adulthood (~25) and declining thereafter (Kim, Laurent, Capaldi, & Feingold, 2008; Nocentini, Menesini, & Pastorelli, 2010; Capaldi et al., 2005; O'Leary, 1999), and that this may differ for men and women with men's violent aggression towards a partner declining slower than women's (Browne & Williams, 1993). Young adults compared to older adults report engaging in more severe forms of partner violence (Kim et al., 2008), indicating the behavioral indicators of the violence construct may change developmentally. Despite the widespread use of the CTS, few studies utilize longitudinal data and even fewer test for temporal invariance of the CTS or CTS2. In one study by Nocentini and colleagues (2010), longitudinal

invariance of a modified CTS was established in late adolescence when the CTS was administered at ages 16, 17, and then at age 18 in a sample of Italian adolescents. To my knowledge this is the only study to date that has tested for temporal invariance while employing the CTS over time. Not utilizing a developmental framework or longitudinal designs has been hypothesized as another reason for the ineffectiveness of current DV interventions (Ehrensaft, 2008; Capaldi et al., 2005; Chan, 2011). The lack of information about the temporal invariance of a common assessment tool for partner violence juxtaposed against the increasing importance of using longitudinal methodology point to the need for assuring that the CTS/CTS2 measures the same thing over time.

1.4 Aims of the Current Study

Recent literature has prioritized the need to assess commonly used measurement tools for invariance between groups and across time in order to ensure that the same theoretical construct is being measured, accurate comparisons between groups are being upheld, and valid conclusions are being derived from the research (Harachi et al., 2006; Widaman, Ferrer, & Conger, 2010; Dimitrov, 2010; Capaldi & Langhinrichsen-Rohling, 2012). Although many papers have been published regarding the reliability, validity, and factor structure of the CTS, the majority of analyses rely upon correlational and factor-analytic methods (Schaefer, 1996). Exploratory factor analysis is an important tool for establishing the best indicators of a construct; however, it does little to establish that the construct is valid across groups or over time (Follingstad & Rogers, 2013). More advanced methodologies such as Multiple Group Confirmatory Factor Analysis (MGCFA) and Structural Equation Modeling (SEM) might be useful for evaluating the measurement validity and reliability of commonly used assessment tools, such as the CTS, to

establish measurement invariance between groups and over time (Pentz & Chou, 1994; Widaman et al., 2010; Dimitrov, 2010; Schaefer, 1996).

The current study used a MGCFA and SEM strategy to examine configural, multi-group measurement, and temporal invariance of participant self-reports of physical-verbal partner violence victimization ascertained from items from the CTS2 between gender and across two developmental periods, young adulthood (age 24) and adulthood (ages 27, 30, and 33).

2. Methods

This study draws on existing longitudinal data between 1999 and 2008 from the Seattle Social Development Project (SSDP). SSDP is a theory-driven developmental study of childhood and adolescent risk and protective factors for substance abuse and related health and behavior problems. SSDP is a longitudinal panel study of children recruited in 1985 when children were approximately 10 years of age from 18 Seattle public elementary schools to participate in a multicomponent intervention study focused on protective and risk factors including violence. Schools selected for the study served high crime neighborhoods though due to mandatory bussing at that time, students from other areas of the city were also enrolled. Assessment for partner violence began in 1996 when the participants were on average 21 years of age using five items from the original CTS (Straus, 1990). Additional questions from the CTS2 were included beginning in 1999 when participants were 24 years old and asked every 3 years thereafter; information from the CTS2 are used in the current study. All participants provided informed consent. Additional information on the original intervention study can be found in Hawkins, Catalano, Morrison, O'Donnell, Abbott, and Day (1992).

2.1 Study Sample

The SSDP sample was recruited in the fall of 1985 from a population of 1053 fifth grade students in 18 elementary schools serving high crime neighborhoods in Seattle. From this population, 808 students (76.7% of the population) consented to participate in the longitudinal study and constitute the SSDP sample. That consent rate is comparable to other studies attempting to recruit children or adolescents (Ellickson & Bell, 1990; Elliot, Knowles, & Cantor, 1981; Thornberry, Lizotte, Krohn, & Farnsworth, 1990). Retention of participants in the SSDP study has been high: 92% at age 33. Thirteen waves of data have been collected. The study is gender balanced; of the 808 participants, 49% (n=396) are women and 51% (n=412) are men. In young adulthood (24) and adulthood (27-33) between 62-64% of participants reported being in a current relationship (married, cohabitating, engaged, or exclusively dating) at any given wave, and an additional 10-17% reported being in a relationship during the prior year. At all ages with the exception of age 33, women were more likely than men to report being in a current relationship. The number of participants who were eligible to complete CTS2 questions in a given year varied: 644 (79.7%) at age 24, 510 (63.1%) at age 27, 561 (71.8%) at age 30 and 589 (75.0%) at age 33. Across all four assessment periods 735 (91%) (365 males and 370 females) of the original sample had reported being in a relationship during at least one time period between ages 24 and 33 and provided information on the CTS2 items; these participants comprise the sample for the current analysis.

2.2 Measures

2.2.1. Conflict Tactics Scale

Questions from the CTS2 were collected beginning in 1999 when participants were 24 years old. The SSDP survey used a subset of questions from the CTS2 to assess the experience of

partner violence victimization and perpetration during young adulthood and adulthood. In SSDP both victimization and perpetration questions were asked of respondents; however, only those responses regarding victimization experience were used in the present analysis. Participants were asked the following questions as gateways to CTS2 items. “*Are you currently in a relationship with another person?*” If the participant answered no to current relationship then they were asked, “*In the past year have you been in a relationship with another person?*” At ages 24, 30 and 33 if a respondent answered “yes” to either question they were directed to answer the CTS2 items. At age 27 the gate questions differed in that participants were only asked if they were in a current relationship, thus excluding the 123 (15.2%) participants who reported being in a relationship in the prior year but who were not in a current relationship and who would have been asked to answer the CTS2 items in the other assessment periods. This explains the higher proportion of missing data at age 27. The CTS2 items used in SSDP are shown in Table 1.1 and the rate of missingness over time and by gender is shown in Table 1.2.

Table 1.1
Conflict Tactic Scale Items Used Across Time

CTS Psychological/Verbal Aggression Subscale	
CTS Item — Ages 24 & 27	CTS Item — Ages 30 & 33
1. My partner insulted, swore, or yelled at me.	1a. My partner insulted or swore at me. 1b. My partner shouted or yelled at me.
2. My partner threatened to hit or throw something at me	2. My partner threatened to hit or throw something at me.
3. My partner threatened me with a knife or gun. ***	

Table 1.1 Continued.

Physical Assault Subscale	
1. My partner pushed, grabbed, slapped, shoved me or threw something at me that could hurt.	1a. My partner pushed or shoved me.
	1b. My partner slapped me.
2. My partner kicked, bit, or punched me.	2a. My partner kicked me.
	2b. My partner punched or hit me with something that could hurt.
	3. My partner used a knife or gun on me. ***
Injury Subscale	
1. I saw or needed to see a doctor because of a fight with my partner.	1. I went to a doctor because of a fight with my partner.
2. I had a sprain, bruise or small cut because of a fight with my partner.	2. I had a sprain, bruise or small cut because of a fight with my partner.
Response Scale Ages 24 & 27: Very Often(1), Often (2), Sometimes(3), Rarely (4), Never (5), or Not in past year but did happen before (6).	Response Scale Ages 30 & 33: Never (0), Once (1), Twice(2), 3-5 (3), 6-10 (4), 11-20 (5), 20+ (6), Not in past year but has happened before (7).

The use of only some questions from the CTS has been found to yield similar findings to those found with use of the full scale (Straus, 1990). Seven victimization questions from the CTS2 were asked at ages 24 and 27. The sexual coercion and reasoning scale questions of the CTS2 were not asked in the SSDP survey. At ages 30 and 33, ten questions regarding victimization were used. These questions mirrored the seven previous questions asked at ages 24 and 27 but separated out forms of violence. As shown in Table 1.1 the single item at age 24 and 27, being shouted at and yelled at, was asked as two distinct questions at ages 30 and 33.

In the CTS2 respondents are asked to rate the presence of each item with the frequency at which it occurs. Ages 24 and 27 used a 6-point Likert scale of frequency of the event in the prior year: Very Often (1), Often (2), Sometimes (3), Rarely (4), Never (5), or Not in past year but did happen before (6) which was reverse coded to (0 "Never happened," 1 "Rarely," 2 "Some of the

time,” 3 “Often,” 4 “Very Often” There was also the option to select 5, which indicated that the event had happened in the past but not in the last year. At ages 30 and 33 participants were asked to rate the frequency of items, using a 7-point Likert scale, Never (0), Once (1), Twice (2), 3-5 (3), 6-10 (4), 11-20 (5), 20+ (6), Not in past year but has happened before (7).

For analysis, items were dichotomized to reflect the occurrence of the event. Dichotomizing the variable addresses non-normality of the distribution of a relatively low frequency behavior, minimizes measurement error due to item responses being collected in two formats across time, and meaningfully delineates findings to experiencing any partner violence victimization (Farrington & Loeber, 2000). This method of dichotomization has been done in prior psychometric work on the CTS2 as seen in Connolly, Nocentini, Menesini, Pepler, Craig & Williams (2010). Following Connolly’s dichotomization strategy the present study dichotomized Verbal aggression indicators (i.e., swearing/yelling) where endorsements of rarely (1-2 a year), never in the past year, or in the past but not in the last year were given a “0” and counted as not experiencing the event. Reports of sometimes (3-5 times per year), often (6-10/year), and very often (11 or more times/year) were indicative of experiencing verbal abuse and given a 1. To adjust for increased level of violence and potential for injury, items from the Physical Assault and Injury Scales were dichotomized in a similar manner as above but included reports of rarely (1-2/year) as an indicator of experiencing that abuse. This decision to account for the higher level of severity that threats of violence and physical assault represented was based on recommendations by Straus (1990).

Once items at all four time periods were dichotomized, items from ages 30 and 33 were recombined to maintain consistency with the seven items measuring partner conflict at ages 24 and 27. Further, due to sparsity and high correlation, the two questions from the Injury Scale

regarding violence resulting in injury were collapsed to create a single dichotomous item reflecting abuse that caused injury and/or required the attention of a doctor. Items regarding threats of physical violence (e.g., “my partner threatened to hit me” or “my partner threatened me with a knife or gun”) were combined to create a single indicator that constituted the threat of either physical violence and/or violence with a weapon. In total, five indicators of violence were created: 1. Verbal abuse (i.e., being sworn at, insulted, or yelled at); 2. Threat of violence (physical or by weapon); 3. Minor violence (slapped, pushed or shoved); 4. Severe violence (kicked or hit); and 5. Injury (breaks, sprains, or having to see a doctor due to violence). These indicators are consistent with prior factor work on the CTS and CTS2 (Straus, 1990).

The number of respondents who were interviewed varied by age, as did respondents reports of being in a current relationship or having been in a relationship in the prior year. These respondents did not provide information on CTS2 items and for the purpose of this analysis were coded as missing. At age 30 and 33 participants were given the option to take a shortened version of the full interview which did not have CTS2 items; respondents who completed the short form were also coded as missing. Men were more likely to not be interviewed at ages 27 and 33 and to complete the short form at ages 30 and 33 compared to women. Among people who were eligible to complete the CTS2 items missingness was only related to gender at age 24, where men were more likely to have missing data. A description of the patterns of missingness and response by age and gender are provided in Figures 1.1 & 1.2.

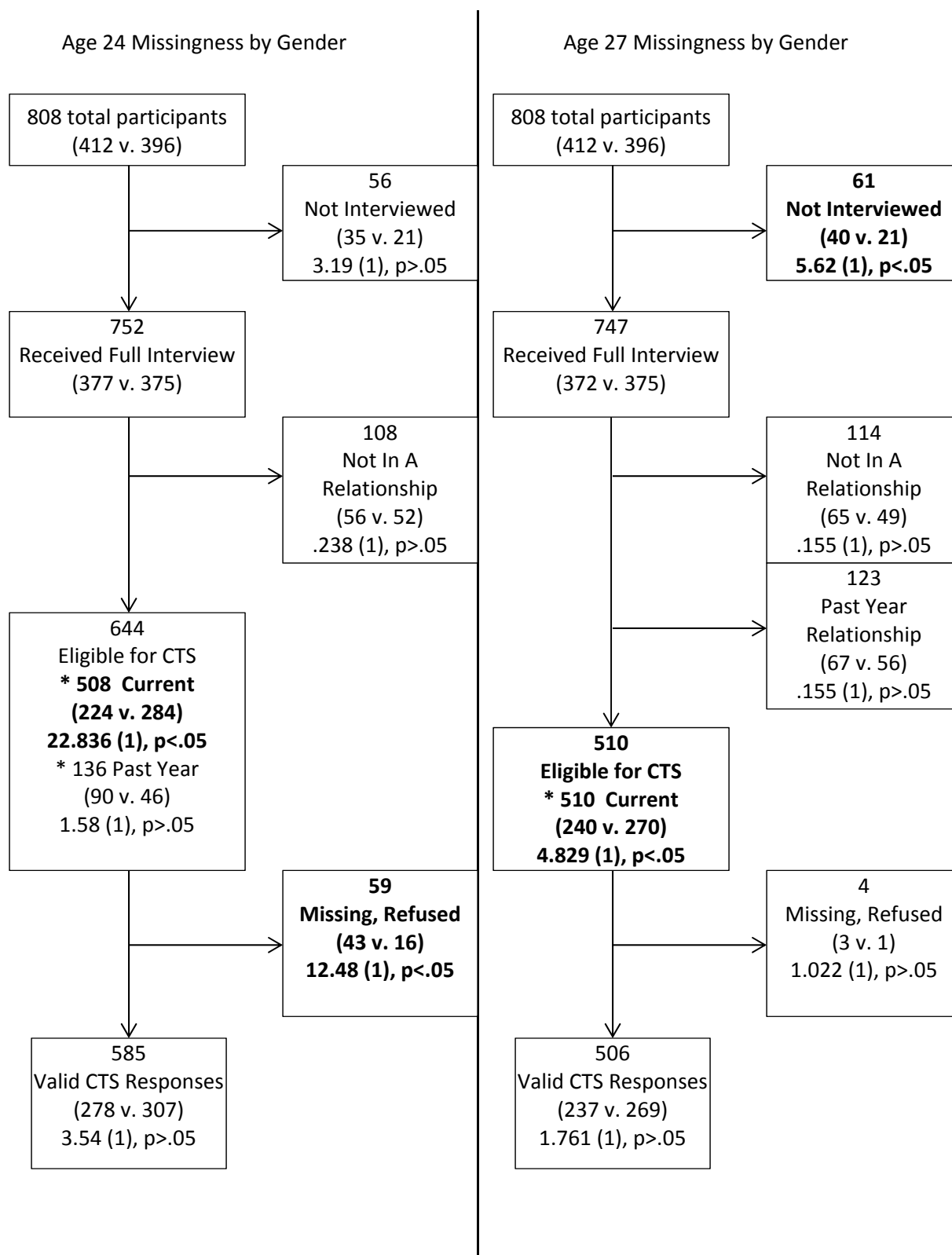


Figure 1.1. Response and missingness patterns of the CTS age 24 & 27 by gender. (Males v. Females) **Bold:** Significant difference at $p < .05$

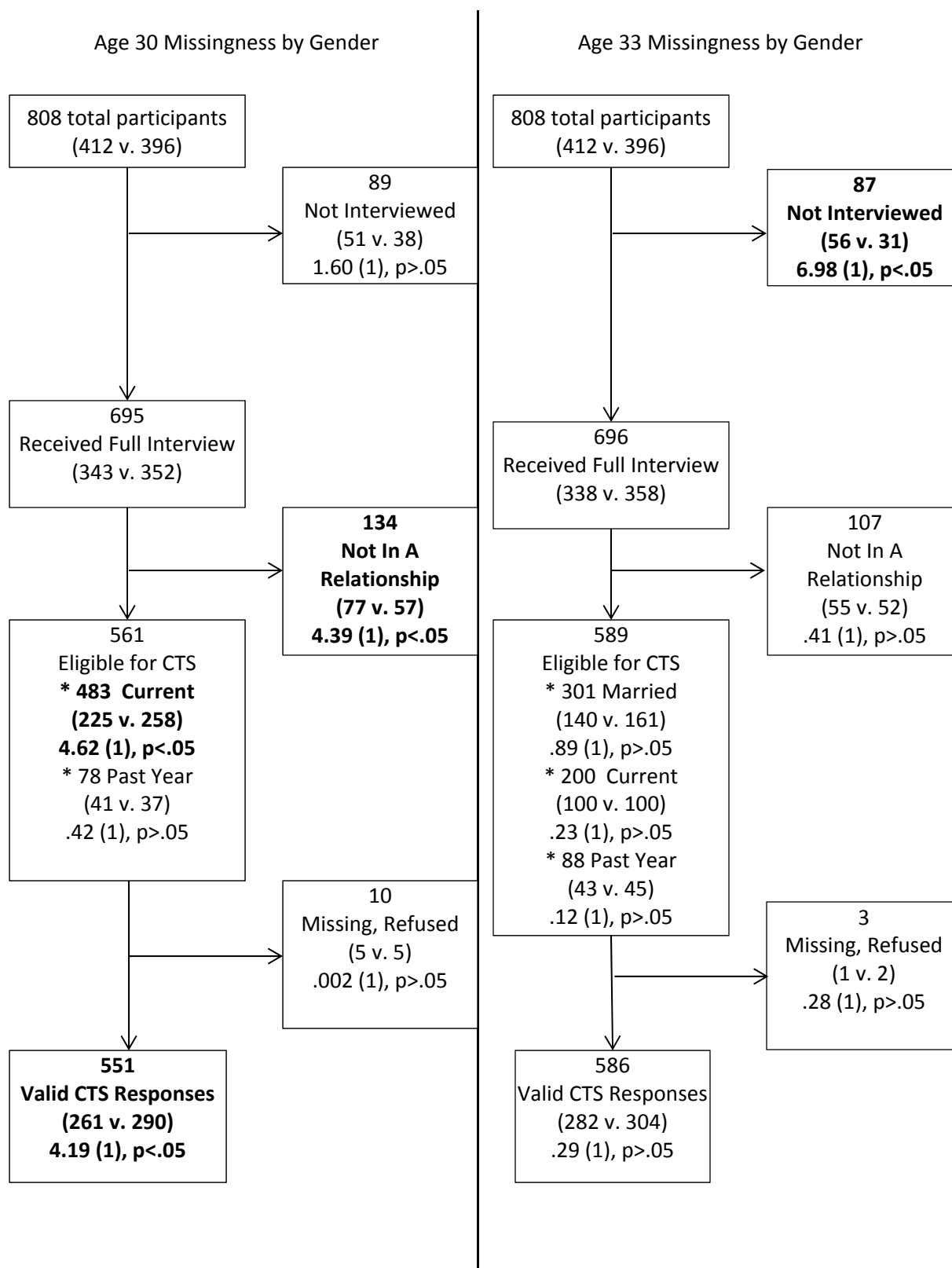


Figure 1.2. Response and missingness patterns of the CTS age 30 & 33 by gender (Males v. Females) **Bold**: Significant difference between men and women at $p < .05$

2.3. Statistical Analysis

All analyses were conducted using Mplus (Version 7.11) software (Muthen & Muthen, 1998-2012). Weighted least squares parameters estimation (WLSMV) was used in all analyses, because this estimator has been identified as performing well for categorical and binary variables and is robust to small sample sizes (Brown, 2006). Missingness was addressed using Full Information Maximum Likelihood. There was minimal missingness among those participants who gated into the CTS2 items at each time period, lowering the threat of substantial bias in estimates, as described in Arbuckle (1996). Gender was not related to missingness except for at age 24 when men were more likely to have missing data on CTS2 items compared to women.

MGCFA and SEM were used to investigate configural, multi-group measurement and temporal invariance, as suggested by Curran, Edwards, Wirth, Hussong, & Chassin, 2007, Pentz and Chou, 1994, and Dimitrov, 2010. CFAs involve a theory-driven approach to investigating factorial structures of latent constructs and item response, and they take into account the differential weighting of each indicator of a larger latent construct, thus not assuming equal contributions among violence experiences as would proportion scoring models (Curran et al., 2007; Dimitrov, 2010).

2.3.1. Model Fit

No single fit index is recommended to assess model fit. Rather, several fit indices should be looked at holistically to determine the goodness of fit of a model (Hu & Bentler, 1999).

Suggested fit indices include the Comparative Fit Index (CFI) (Bentler, 1990; McDonald & Marsh, 1990), Tucker Lewis Index (TLI) (Tucker & Lewis, 1973), and the Root Mean Square Error of Approximation (RMSEA) (Steiger, 1990; Browne & Cudeck, 1993). Recommendations for cut-offs on goodness of fit include the following: $RMSEA \leq .06$, $CFI \geq .95$, and the TLI

$\geq .95$. Vandenberg and Lance (2000) recommend using a change in the CFI fit statistic as a measurement of invariance, where a change in CFI value more than .02 indicates non-invariance. The CFI, TLI, and RMSEA have all been found to be appropriate for use with categorical variables (Muthen & Muthen, 2012).

2.3.2. Measurement Invariance by Gender Within Time

The first goal of this study was to assess the consistency of the partner violence victimization latent construct for men and women within a given time period. Measurement invariance between groups refers to the consistency of the underlying latent construct across groups as opposed to the consistency of observed scores on the indicators of the construct between men and women. Testing for factorial invariance between groups targets the assessment of generalizable and structural validity as it explores the viability of a construct across differing groups (Messick, 1995; Dimitrov, 2010). Factorial invariance answers the question, “Does this construct, as measured by this assessment, have the same meaning for different groups?” (Dimitrov, 2010). If weak or no support for measurement invariance is found, then items that determine the construct of partner violence victimization are likely not the same for men and women, making subsequent interpretation of associations with other variables difficult. However, if measurement invariance is supported, the same indicators can be used to determine the construct of partner violence victimization for both males and females.

Ferrer and colleagues (2008) suggested that multi-group invariance has four levels of invariance by strength. *Configural* invariance shows that for differing groups the same items are used in the latent construct. *Metric* invariance, the next level of rigor, refers to equivalent factor loadings across groups. *Scalar* invariance refers to equality in the item thresholds, or probability a respondent would report the violence indicator, for categorical data across groups. Finally, the

highest level of invariance, *error* invariance, refers to equity between groups regarding the item error. The designation of weak measurement invariance purely assesses for metric invariance and allows the researcher to make comparisons between groups about the relationship between the latent construct and indicator variables. Strong measurement invariance is determined when metric, scalar, and error invariance is found across groups. Strong measurement invariance allows the researcher to conclude that differences between groups are due exclusively to group differences and cannot be attributed to measurement issues. However, Dimitrov (2010) notes that constraining item error variance/covariance is potentially overly restrictive and offers little practical interest in comparing the groups. The current analysis does not assess error variance.

2.3.3 Temporal Invariance

The second goal of this study was to determine if the construct of partner violence victimization was the same when measured across young adulthood and adulthood. Difference in a factor structure across groups is termed factorial or multi-group invariance, whereas testing for structural consistency of the CFA model across time is termed configural, structural, or temporal invariance (Meredith, 1993; Meredith & Teresi, 2006). Temporal invariance investigates the temporal validity of a measure and assesses if the relationship between a latent variable and its indicators is stable across time periods (Widaman et al, 2010). Temporal invariance answers the question, “Does the construct, as measured by this assessment, maintain the same meaning and importance over time?” (Vandenberg & Lance, 2000).

Widaman and Reise (1997) established four basic levels of longitudinal invariance: 1. Configural invariance, where the items used to identify the construct are the same across time; 2. Weak factorial invariance, where the factor loadings on the latent construct do not differ across time; 3. Strong factorial invariance, where both the factor loadings and the item thresholds do not

significantly vary across time; and, 4. Strict factorial invariance, where the factor loadings, thresholds, and error terms for the indicators do not differ across time period. The current analysis does not assess the stability of error terms across time.

2.3.4. Partial Invariance

In longitudinal research partial invariance is often expected (Widaman et al., 2010) though few guidelines have been established for comparing partial measurement invariance. Partial invariance would allow some parameters, such as factor loadings or thresholds, to vary across time points or between groups (Byrne, Shavelson, & Muthen, 1989; McArdle & Cattell, 1994). To determine partial invariance some parameters of the model are allowed to be freely estimated across the comparison groups while other parameters are forced to be the same value across groups. To determine which parameters should be allowed to vary between groups, Dimitrov (2010) suggests following the recommendations of the modification indices provided by the statistical software used in the analyses. Further, he recommends that the parameters should be freed sequentially, one at a time, to best determine the most parsimonious fit of the model. There is no strict rule for determining what portion of the model can be freed for partial invariance and still be considered consistent in measurement across time. Developmental theory would suggest that all behaviors change over time and thus it is expected in social science that a certain degree of partial invariance is acceptable. A general rule is that <20% of parameters can be freed, though the primary suggestion to date is that the “observed degree of invariance” is reported with the results (Byrne et al., 1989; Levine, Kaplan, Kripke, Bowen, Naughton, & Shumaker, 2003).

3. Results

3.1. Descriptive

Overall, 28.1% of study participants (n=227) reported being a victim of partner violence at least once between ages 24 and 33, with 157 (69.2%) of those respondents reporting two or more periods of partner violence victimization. Gender differences in reported exposure to any partner violence from age 24 through 33 were minimal. Women were more likely to report an injury or having to see a doctor at age 27 compared to men ($\chi^2 4.127 (1), p = .056$) and men were more likely to report threats of violence at age 33 compared to women ($\chi^2 6.198 (1), p = .02$). No other differences were found (Table 1.2). All indicators were correlated significantly with each other within time for males and females (Table 1.3).

Table 1.2

Prevalence of Reported Partner Violence Victimization Exposure by Age and Gender

Age 24	Violence Indicator	Full Sample 644		Males 314		Females 330		χ^2
		n	%	n	%	n	%	
	Verbal Violence	147	22.8	60	19.1	87	26.4	3.54(1), p=.07
	Threat of Violence	132	20.8	63	20.1	69	20.9	.06(1), p =.84
	Minor Violence	140	21.7	67	21.3	73	22.1	.01(1), p =.92
	Severe Violence	85	13.2	45	14.3	40	12.1	.71(1), p =.42
	Injury/Medical Attention	75	11.6	33	10.5	42	12.7	.73(1), p=.46
	Any Reported Partner Violence Victimization	201	31.2	88	28.0	113	34.2	1.7(1), p =.22
	Two or more forms of Partner Violence	116	18.0	52	16.6	64	19.4	1.8(1), p =.40
Age 27	Violence Indicator	Full Sample 510		Males 240		Females 270		χ^2
		n	%	n	%	n	%	
	Verbal Violence	123	24.1	64	26.7	59	21.9	1.8(1), p=.21
	Threat of Violence	79	15.5	40	16.7	39	14.4	.54(1), p=.47

Table 1.2 Continued

Age 27	Violence Indicator	Full Sample 510		Males 240		Females 270		χ^2
	Minor Violence	105	20.6	56	23.3	49	18.1	2.3(1), p=.15
	Severe Violence	47	9.2	22	9.2	25	9.3	.00(1), p =1.0
	Injury/Medical Attention	36	7.0	11	4.6	25	9.3	4.1(1), p=.06
	Any Reported Partner Violence Victimization	167	32.7	84	35.0	83	30.7	1.2(1), p=.30
	Two or more forms of Partner Violence	68	13.3	34	14.2	34	12.6	1.2(2), p=.55
Age 30	Violence Indicator	Full Sample 580		Males 282		Females 298		χ^2
		n	%	n	%	n	%	
	Verbal Violence	275	47.4	133	47.2	142	47.7	.22(1), p = .67
	Threat of Violence	72	12.4	38	13.5	34	11.4	1.0(1), p =.32
	Minor Violence	108	18.6	55	19.5	53	17.8	.71(1), p=.45
	Severe Violence	39	6.7	23	8.2	16	5.4	2.3(1), p = .14
	Injury/Medical Attention	33	5.7	11	3.9	22	7.4	2.8(1), p = .11
	Any Reported Partner Violence Victimization	288	49.7	139	49.3	149	50.0	.24(1), p=.67
	Two or more forms of Partner Violence	80	14.0	40	14.2	40	13.4	.38(2), p =.83
Age 33	Violence Indicator	Full Sample 606		Males 295		Females 311		χ^2
		n	%	n	%	n	%	
	Verbal Violence	273	45.1	125	42.4	148	47.6	1.2(1), p=.28
	Threat of Violence	71	11.7	44	14.9	27	8.7	6.2(1), p=.02
	Minor Violence	118	19.5	62	21.0	56	18.0	1.1(1), p=.31
	Severe Violence	41	6.8	25	8.5	16	5.1	2.9(1), p=.11
	Injury/Medical Attention	42	6.9	19	6.4	23	7.4	.16(1), p=.75
	Any Reported Partner Violence Victimization	285	47.0	134	45.4	151	48.6	.27(1), p=.62
	Two or more forms of Partner Violence	81	13.4	45	15.3	36	11.6	3.6(2), p=.17

Table 1.3
Bivariate Correlations of Partner Violence Victimization Indicators by Age and Gender

Age 24						Age 27					
	1.	2.	3.	4.	5.		1.	2.	3.	4.	5.
1. Verbal 24	1					1. Verbal 27	1				
2. Threat 24	.67 .73/.61	1				2. Threat 27	.61 .63/.58	1			
3. Minor 24	.67 .65/.70	.91 .86/.94	1			3. Minor 27	.61 .65/.55	.90 .89/.91	1		
4. Severe 24	.55 .60/.51	.89 .90/.89	.87 .85/.89	1		4. Severe 27	.54 .52/.56	.78 .65/.87	.85 .72/.94	1	
5. Injury 24	.50 .59/.43	.83 .77/.88	.83 .78/.87	.86 .84/.89	1	5. Injury 27	.47 .54/.46	.73 .60/.81	.83 .68/.91	.88 .88/.90	1
Age 30						Age 33					
	1.	2.	3.	4.	5.		1.	2.	3.	4.	5.
1. Verbal 30	1					1. Verbal 33	1				
2. Threat 30	.57 .59/.55	1				2. Threat 33	.60 .63/.59	1			
3. Minor 30	.54 .49/.58	.86 .76/.94	1			3. Minor 33	.65 .59/.73	.87 .86/.91	1		
4. Severe 30	.52 .53/.51	.77 .79/.74	.90 .92/.88	1		4. Severe 33	.56 .50/.70	.87 .90/.82	.87 .91/.81	1	
5. Injury 30	.46 .36/.52	.66 .59/.73	.84 .84/.85	.83 .92/.84	1	5. Injury 33	.61 .45/.76	.76 .76/.78	.85 .85/.85	.84 .92/.76	1

All standardized coefficients were significant at $p < .001$; Male/Female correlation

3.2. Multiple-Group Confirmatory Factor Model

3.2.1. Measurement Invariance across Groups

A latent variable framework approach was implemented to test for invariance of the latent construct of partner violence victimization between men and women as outlined by Pentz and Chou (1994). A latent variable for partner violence victimization was created using the following categorical indicators: 1. Verbal abuse (i.e., being sworn at, insulted, or yelled at); 2. Threat of violence (physical or by weapon); 3. Severe violence (kicked or hit); and 4. Injury (breaks, sprains or having to see a doctor due to violence) (Figure 1.3). Binary scores were used in the present analysis. The indicator “minor violence” (slapped, pushed, shoved) was not used in the present analysis due co-linearity with the indicator of Severe Violence (r ranged from .85 to .90). Sensitivity analysis revealed no significant differences in the results when the minor violence indicator was exchanged with the severe violence indicator in the model. The CFA model was tested on the full sample in each assessment year separately to determine if there was an underlying construct of victimization in the data at each time point (Table 1.4).

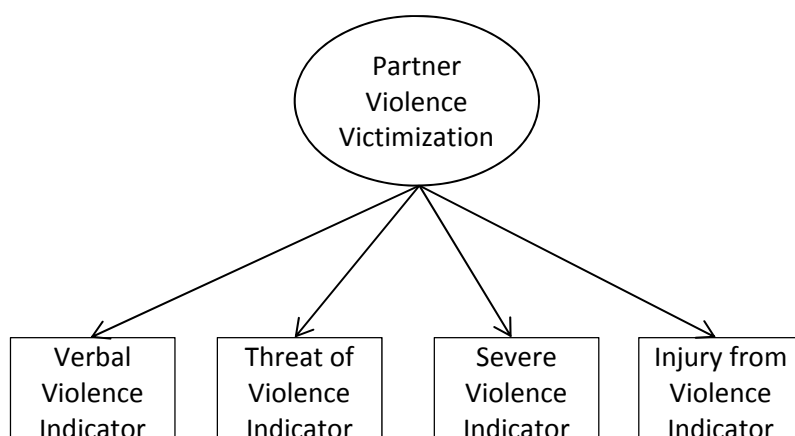


Figure 1.3. Conceptual model of Partner Violence Victimization tested at ages 24, 27, 30 & 33.

Table 1.4
Confirmatory Factor Analysis of the Partner Violence Victimization Construct by Assessment Time/Age

	χ^2	df	p-value	RMSEA	CFI	TLI
Age 24						
Full Sample	9.735	6	.1363	.045	.998	.997
Females	4.906	2	.086	.068	.998	.993
Males	3.781	2	.151	.054	.998	.994
Age 27						
Full Sample	9.153	6	.1652	.046	.997	.994
Females	2.295	2	.3174	.023	1.00	.999
Males	4.796	2	.0909	.077	.989	.967
Age 30						
Full Sample	9.962	6	.1263	.049	.994	.988
Females	.744	2	.6895	.000	1.00	1.01
Males	5.813	3	.1211	.059	.992	.985
Age 33						
Full Sample	12.346	6	.0547	.060	.994	.989
Females	2.099	2	.3502	.013	1.00	.999
Males	6.217	3	.1015	.061	.996	.992

Note: All time periods are composed of the same four categorical indicators.
 At ages 30 and 33 the indicator being kicked was constrained to 1 for men.

Second, to evaluate if the data supported the underlying construct for both men and women (configural invariance), the CFAs were run stratified by gender. All models at all ages and for both genders fit the data well as shown in Table 1.5.

Third, to determine if the construct of partner violence victimization was invariant for men and women within time, a multi-step process to determine metric and scalar invariance was engaged as suggested by Dimitrov (2010). For each age the model was tested for metric and scalar invariance using a chi square test of difference between a freely estimated model where all parameter estimates could vary across gender, and a constrained model where all factor loadings and threshold estimates were forced to be equal. At each age (24, 27, 30, 33), the factor loadings of the partner violence victimization indicators were statistically equivalent, resulting in metric invariance for men and women. Similarly, the thresholds estimates of the four indicators were all

statistically equivalent across gender at each age, resulting in scalar invariance. Based on these tests the construct of partner violence victimization, measured by items from the CTS2, has strong measurement invariance for men and women at each time point. Table 1.5 depicts the comparison of the freely estimated model and fully constrained model with their tests for difference.

Table 1.5
Tests of Factor Loading and Threshold Measurement Invariance by Gender

Year	Model	χ^2	df	p-value	RMSEA	CFI	Δ CFI	TLI
Age 24	M0	8.56	4	.073	.061	.998	---	.994
	M1	8.22	8	.413	.009	1.00	+0.002	1.00
χ^2 Test of Difference		3.06	4	.549			Invariant	
Age 27	M0	7.79	4	.100	.061	.996	---	.988
	M1	10.57	8	.023	.036	.997	+0.001	.996
χ^2 Test of Difference		4.81	4	.307			Invariant	
Age 30	M0	6.46	5	.264	.032	.998	---	.995
	M1	10.80	10	.373	.017	.999	+0.001	.999
χ^2 Test of Difference		5.28	5	.382			Invariant	
Age 33	M0	7.85	5	.165	.044	.997	---	.994
	M1	17.61	9	.040	.057	.992	-0.005	.990
χ^2 Test of Difference		9.29	4	.054			Invariant	

Notes: M0 represents the baseline model where all parameters are allowed to estimate freely. M1 is the comparison model in which all parameters are constrained to equality between men and women.

At ages 30 and 33 the indicator being kicked was constrained to 1 for men thus increasing the df.

3.2.2. Measurement Invariance across Time

A second goal of this study was to assess whether the construct of partner violence victimization changed in meaning or importance between the ages of 24 and 33. To establish temporal invariance, the structural consistency of the factor was tested across all four time periods. Following steps outlined by Widaman et al., (2010), using the full sample, since

invariance of the construct within time had been established for men and women, I constrained all factor loadings and thresholds of each like indicator to equivalence across the four time points. For example, factor loadings from the indicator “verbal abuse: being yelled, screamed or sworn at” were held equal across periods: $\lambda_1=\lambda_2=\lambda_3=\lambda_4$. A freely estimated model where factor loadings and thresholds were estimated freely across time, fit the data well ($\chi^2=268.63(142)$, $p<.000$, RMSEA = .035, CFI = .97, TLI = .96). A fully constrained model in which like item factor loadings and thresholds were constrained to equality across time fit the data well ($\chi^2=443.82(170)$, $p<.000$, RMSEA = .047, CFI = .93, TLI = .93), though there was a significant detriment in model fit when the constraints were applied ($\chi^2=167.66(28)$, $p<.000$) indicating differences in the construct over time.

Constraints were systematically removed from the thresholds and factor loadings one at a time to determine where the differences in the latent constructs were across time. A partially invariant model demonstrated an increase in model fit from the base line model ($\chi^2=268.09(16)$, $p<.000$, RMSEA = .030, CFI = .97, TLI = .97) supported by a non-significant chi-square test of difference of the latent construct partner conflict across the four age points ($\chi^2=16.72(18)$, $p<.542$). In the final partially invariant model all factor loadings remained constrained suggesting that the same like items over time could be used to define the latent construct and that similar items had the same strength relative to the latent construct; determining configural and metric temporal invariance. Thresholds for the verbal violence and threat of violence indicators were relaxed across all ages whereas thresholds for the severe and injury indicators remained constrained at ages 27-33. Thresholds can be transformed into probabilities by exponentiating the threshold value and calculating the probability that the response of the indicator ($y=1$) when $\theta = 0$ following the formula: $1 - [(\exp(\text{threshold}(a)) / 1 + \exp(\text{threshold}(a)))]$. When this was

done for the identified relaxed thresholds of the four indicators of partner violence across ages 24-33 it was found that the probability of an individual reporting verbal violence differed from ages 24 and 27 to ages 30 and 33 such that at earlier ages there was 33% chance that a person would report verbal violence compared to 50% at age 30 and 52% at age 33. The probability that a person would report threats of violence decreased across time. At age 24 there was 31% probability that a participant would report threats of violence, 27% at age 27, 24% at age 30 and 24% at age 33. Young adulthood, whose thresholds on all indicators were freed, had higher probabilities on all indicators compared to later ages indicating that the probability of reporting all violence types was higher during young adulthood compared to adulthood. Severe violence and injury indicators did not differ significantly in adulthood (27-33). This suggests that the likelihood of reporting certain forms of partner violence victimization may differ over time and that young adulthood (age 24) may be unique compared to later time periods; see Figure 1.4.

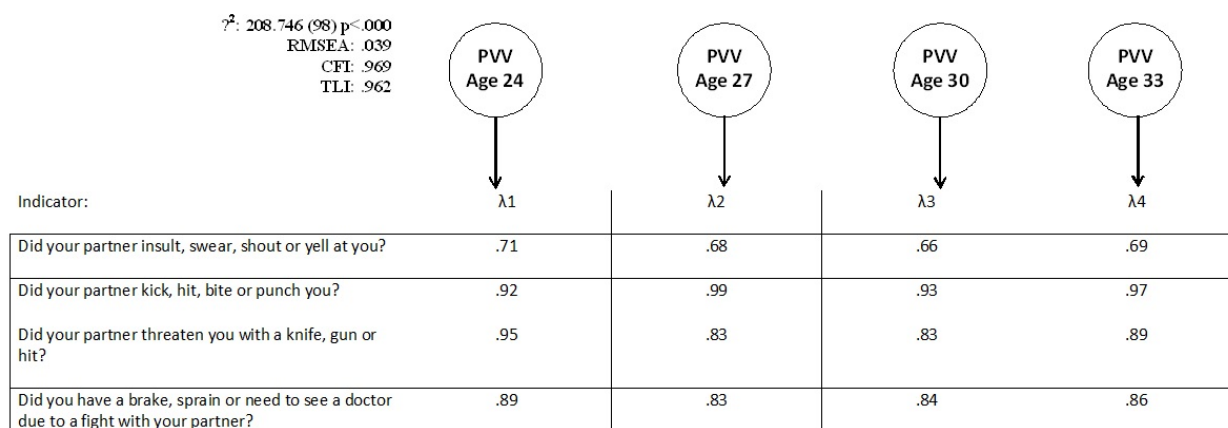


Figure 1.4. Temporal invariance of partner violence victimization factor loadings across ages 24-33 (full sample). Though the strength of like type indicators varied across time they did not differ significantly.

Lastly, to determine if the correlations between the partner violence victimization construct differed by gender all four time points were correlated with each other and tested in a multiple group SEM model. When run separately, for both men ($\chi^2=136.68(98)$, $p=.006$, RMSEA = .033, CFI = .98, TLI = .97) and women ($\chi^2=164.49(99)$, $p<.000$, RMSEA = .042, CFI = .97, TLI = .96), the model fit the data well. For women, all loadings were significant at $p<.000$ and greater than .63; for men, all loadings were significant at $p<.000$ and greater than .66 across ages 24, 27, 30, and 33. When correlations between time points were constrained to equality across men and women a chi-square test of difference ($\chi^2=16.405(7)$, $p=.02$) that imposing equality on the correlations between partner violence victimization across men and women significantly reduced the model fit indicating that there were differences in the correlations of partner violence victimization factors for men and women. Each correlation parameter was released one at a time and tested against a fully constrained model until the chi square test of difference resulted in an invariant test $p>.05$ ($\chi^2=5.679(6)$, $p=.46$) and the most parsimonious model. A difference between men and women was detected in the correlation between partner violence victimization at age 30 and 33, where women had a significantly stronger relationship compared to men (.86 vs. .47) though both were significant. This parameter was allowed to vary by gender, which resulted in invariance across men's and women's measured partner violence victimization across four time periods. The final model is shown in Figure 1.5 and includes parameter estimates for both men and women, though these estimates are statistically equivalent with the exception of the correlation between partner violence at age 30 and 33.

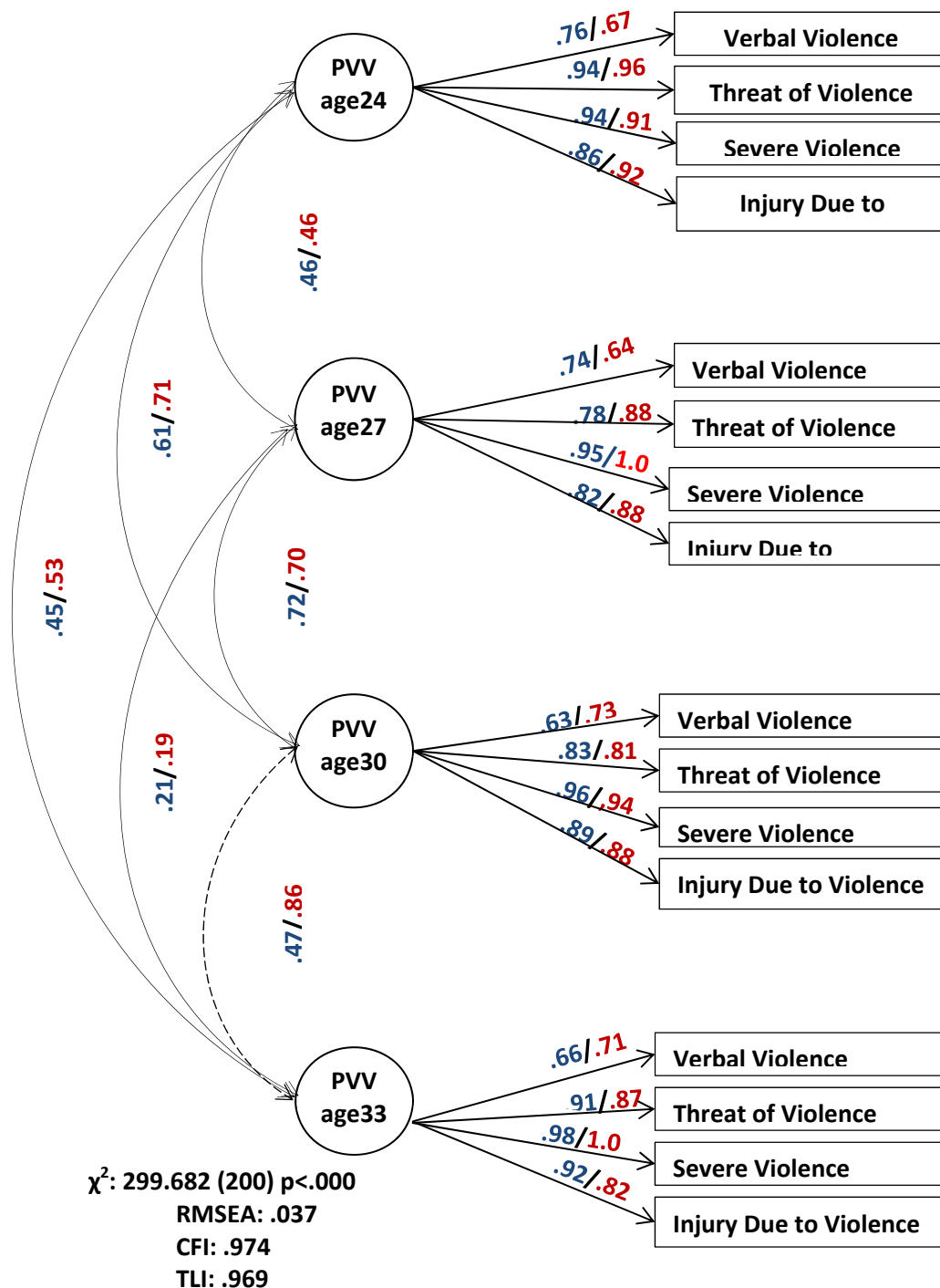


Figure 1.5. Model of the factorial structure of partner violence victimization invariance by male and female respondents ages 24-33.
 Note: Standardized coefficients, male/female.

4. Discussion

In the present data, support was found for configural, metric, and scalar invariance across gender and configural, metric and partial scalar invariance of a latent construct of partner violence victimization derived from items from the CTS2. Configural invariance, or the same indicators of partner violence victimization, was found between men and women and across time. Strong multi-group measurement invariance where metric and scalar parameters were equivalent between men and women within time was found at ages 24, 27, 30, and 33. This finding differs from that of Schaeffer (1996), the only other study that investigated gender differences in measurement invariance of the CTS. The current study used items from the CTS2 which were similar to but not the same as those used by in Schaefer who used items from the original CTS. For example the indicator used in the current analysis “My partner pushed, grabbed, slapped, shoved me, or threw something at me that could hurt” roughly corresponds to Schaefer’s indicator “Pushed, grabbed, or shoved the other one.” It is not clear by the description of Schaefer’s indicators if the violence measured is reciprocal or purely victimization whereas in the current study victimization and perpetration responses are clearly delineated in the assessment. In addition, Schafer recoded each indicator as 0, 1, 2 (two or more), and it is not clear if this refers to the number of listed events or two or more of the described behaviors in the indicator. Unlike Schafer’s, the present study clearly identifies that exposure is based upon the dichotomized frequencies of the item in the prior year representing any exposure to that violence type. Potentially, differences in the operationalization and treatment of the violence items could have contributed to the differing findings.

This study’s use of a community-based sample may have biased the findings towards more gender symmetry, as seen in a previous review of the literature (Chan, 2011). Johnson (2011) attributes finding gender symmetry in community samples to less severe type of violence that

measures are collecting in general community samples as compared with clinical samples.

Johnson reports that community samples are more likely to report situational type violence that forms a progressive pattern of conflict, starting with arguing and escalating into a violent encounter. Situational violence is more common, more variable in degree, and more likely to be perpetrated in more equivalency for men and women compared to what he terms “intimate terrorism” (Johnson, 2011, p. 290). Intimate terrorism is described as acts between partners that entail physical/sexual violence or non-violent extreme coercion, is more likely to escalate to the attention of law enforcement and the medical community, and has been found to be perpetrated predominantly by men against women (Johnson, 2011; Archer, 2000). In the present data, the item that approaches capturing intimate terrorism is the Injury Scale item regarding needing to see a doctor or sustaining an injury due to a conflict with your partner. Women were in fact more likely to report this item at all four time periods though this difference was only significant at age 27. Factor loadings for this indicator on the latent factor did not differ for men and women. It may be that the use of a community sample created a latent construct of situational violence and did not fully tap into this construct of intimate terrorism which could have contributed to gender differences not being identified.

This study also found evidence of configural, metric and partial scalar invariance across time. While factor loadings were equivalent across time, some thresholds needed to be freely estimated. This finding differed from the one other study that investigated temporal invariance of the CTS by Nocentini and colleagues (2010), who reported full configural, metric and scalar temporal invariance between the ages of 16 and 18. It is important to note that Nocentini et al.’s measurement period did not extend beyond one developmental period (adolescence), whereas the present study tested for temporal invariance across two developmental periods, young adulthood

(age 24) and adulthood (ages 27, 30, 33). The partial invariance in this study appears to be dependent upon the timing and the type of indicator. For instance, the thresholds for the verbal abuse items needed to be freely estimated across time, unlike the injury items, which held equivalency in adulthood from age 27 through age 33. Earlier developmental time periods (age 24) had more relaxed thresholds compared to later time points (i.e., ages 27-33) supporting previous findings that partner violence victimization changes developmentally. When thresholds were exponentiated the probability of reporting threats of violence decreased over time suggesting that exposure to this form of violence decreased or the likelihood of reporting this form of violence was reduced. This could be due to the changing/developmental role of relationships and the violence associated with those forms of intimate partnerships or could suggest that the likelihood of reporting violence within a partnership changes over time. Younger developmental periods may involve short term, more volatile, partnerships compared to relationships as one gets older which tend to be more stable.

4.1. Limitations

A limitation of the current study is only having a subset of questions from the CTS2 and only from limited subscales. The exclusion of the sexual violence subscale is particularly noteworthy since it may capture more gendered nuances of violence within the intimate partner context (Black et al., 2011; Thompson et al., 2006; Plichta, 2004). That said, recent literature has pointed out the need to identify differences and similarities in the assessment of differing forms of physical conflict among subcultures in the U.S. (Follingstad & Rogers, 2013). With the CTS/CTS2 physical violence subscale being the most widely used assessment of physical conflict, the findings of this study add to the psychometric rigor of the violence field.

The decision to dichotomize violence items to capture exposure verses frequency of exposure may be viewed as a limitation. However, there is precedence in the literature for dichotomization of CTS items (Connolly et al., 2010). Further, dichotomizing items of rare behaviors have not been found to significantly reduce explanatory power of the variable and dichotomization of a variable has been noted as increasing interpretability of findings (Farrington & Loeber, 2000).

Lastly, as pointed out by Widaman et al., (2011) it is important to contextualize the findings of any measurement and temporal invariance work within the conceptual limitation of the items and latent construct. In the current study the latent partner violence victimization construct used items reflecting any exposure to a violent behavior from a partner. Thus, the findings of invariance indicate consistency and equivalence of measured partner violence exposure, as captured by the CTS2 items, in relation to the latent physical violence factor for men and women and across time. The current study does not model the psychological reaction to violence, the context of violence, or perceived danger of the experience – all essential components necessary in understanding the full experience of partner violence victimization, but it does begin to lay a foundation of confidence in the quantitative measurement of victimization exposure between genders and across time.

4.2. Implications for Practice

Domestic violence in the United States is a significant public health issue (Black et al., 2011). Increased rigor in domestic violence research is needed to correctly identify and disentangle risk factors that will inform the next wave of intervention and prevention efforts. This may be particularly important for marginalized subpopulations, including women, individuals with low socioeconomic status, and racial and ethnic minorities who

disproportionately carry the burden of partner violence victimization and the resulting negative long-term effects (National Center for Injury Prevention and Control, 2003; Taft, Bryant-Davis, Woodward, Tillman, & Torres, 2009; Plichta, 2004). With health disparities among these subgroups growing in the U.S., the need to understand whether similar or disparate factors contribute to behavior across groups and over time is becoming a necessity. Identifying common predictors contributes to the development of universal programming, whereas identifying unique predictors in subpopulations and at sensitive time periods aids the development of tailored group or time-responsive interventions (Harachi et al., 2006), which may increase the efficacy of the intervention and ultimately lower the public health burden of partner violence victimization in the United States.

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CHAPTER 2.
THE ROLE OF GENDER IN THE RELATIONSHIP BETWEEN
SMOKING BEHAVIOR AND PARTNER VIOLENCE VICTIMIZATION
ACROSS TIME

1. Introduction

Smoking and partner violence victimization independently cost the United States billions of dollars in healthcare and place a heavy personal toll on millions of individuals and their families (Thun et al., 2013; Plichta, 2004). Yet little is known about how these two phenomena influence each other, namely if partner violence victimization causes smoking, if smoking leads to partner violence victimization, or if the two phenomena reciprocally influence each other over a person's life course. A better understanding of how smoking and partner violence victimization are related could inform future public health efforts, ultimately reducing the large public health burden these two issues have placed on society, and could better the lives of individuals who smoke and are faced with violence on a daily basis.

1.1. Intimate Partner Violence and Smoking

The relationship between partner violence victimization and substance use is complex. Victimization by a partner has consistently been found, in both cross-sectional and longitudinal studies, to increase the risk for alcohol use (for a review, please see Foran and O'Leary, 2008) and use of illicit substances such as marijuana (for a review, please see Moore et al., 2008). Alcohol and drug use, by one or both partners, have been found to increase the risk for reported partner violence victimization (Foshee, Benefield, Ennett, Bauman, & Suchindran, 2004; Huizinga, Weiher, Espirtu, & Esbesnen, 2003). The association between the two issues is prominent in clinical samples, where approximately half of all women and one out of ten men currently engaged in substance use treatment report being victims of partner violence (Chase, O'Farrell, Murphy, Fals-Stewart, & Murphy, 2003; Chermack, Fuller, & Blow, 2000; Drapkin,

McCrary, Swingle, & Epstein, 2005; Najavits, Sonn, Walsh, & Weiss, 2004; Chermack, Walton, Fuller, & Blow, 2001; Schneider, Burnette, Ilgen & Timko, 2009), a disturbing statistic given the fact that experiencing partner violence has been linked to lowered substance use treatment success, higher treatment drop out, and increased likelihood of treatment re-entry (Schneider et al., 2009; Lipsky et al., 2010).

Although a strong connection between alcohol and illicit drug use and partner violence victimization has been established, information on the link between partner violence victimization and smoking is limited, despite the fact that smoking remains one of the most available and widely used substances in the United States (U.S. Department of Health and Human Services, 2014) and is highly comorbid with other substances, particularly alcohol (Falk, Yi, & Hiller-Sturmhofel, 2006). In studies investigating the linkage between partner violence victimization and substance use, smoking is often excluded, rolled into generalized measures of substance use, or ignored entirely, leaving us uncertain of the unique relationship smoking may have with partner violence victimization. It could be that findings linking partner violence victimization and alcohol or drug use are due to smoking behavior or a combination of smoking with other substances, but this relationship remains entangled and unclear.

What is known of the connection between smoking and partner violence victimization is hampered by the methodological limitations of existing literature, including an over-reliance on cross-sectional data, use of non-random convenience samples, exclusive focus on female samples, and lack of control for factors that may explain the occurrence of both partner violence victimization and smoking (Crane, Hawes, & Weinberger, 2013; Fletcher, 2010). Additionally, most studies to date have used current smoking as the outcome measure, which limits the scope of the investigation by not accounting for the variability and risk associated with other smoking

behaviors, and potentially missing the nuances of different smoking groups in relation to partner violence victimization (Crane et al., 2013; Okoli, Torchalla, Ratner, & Johnson, 2011). These limitations prevent a clear understanding of how smoking and partner violence victimization influence each other and add to the uncertainty of whether smoking and partner violence victimization have a unique relationship beyond that of other substances.

Cross-sectional studies have demonstrated that partner violence victimization is associated with smoking (El-Mohandes, El-Khorazaty, Kiely, & Gantz, 2011; Dichter, Cerulli, & Bossarte, 2011; Vest, Catlin, Chen, & Brownson, 2002; Crane et al., 2013). In a recent meta-analysis of available literature the strength of the association smoking and partner violence victimization had an estimated effect size small-to-medium in magnitude ($d=.41$) (Crane et al., 2013). In cross-sectional, single gender, studies comparing women who had been victimized with those who had never been victimized, those experiencing partner violence who had ever smoked were more likely to be a current smoker (Black & Brieding, 2008; Jun, Rich-Edwards, Boynton-Jarrett, & Wright, 2008; Stene, Jacobsen, Dyb, Tverdal, & Schei, 2013; Ackerson et al., 2007; Vest et al., 2002; Scott-Storey, Wuest, & Ford-Gilboe, 2009) and more likely to have begun smoking at an earlier age (Yoshiham, Horrocks, & Bybee, 2010). The association between partner violence victimization and smoking among men is under-explored due to the literature's bias of focusing on the substance use-victimization link among women and the substance use-perpetration link among men (for example, Easton, Weinberger, & George, 2007). When the relationship between smoking and partner violence victimization is explored among men, one study using BRFSS, a large population-based study of health, found that men reporting partner violence victimization, similar to their female counterparts, were at higher risk of being current smokers compared to men with no reported partner violence experiences (Black & Breiding, 2008). The link between

partner violence victimization and smoking, for women, has been replicated in several countries, including the United States (Black & Brieding, 2008; Jun, Rich-Edwards, Boynton-Jarrett & Wright, 2008), Japan (Yoshiham et al., 2010), Norway (Stene et al., 2013), and India (Ackerson et al., 2007).

Because of the dearth of longitudinal studies, whether smoking behavior and partner violence victimization have a relationship that extends beyond within-time associations remains unknown. Based on research linking other substances with partner violence victimization, I postulate that the relationship between the two phenomena may exist in the four ways described below.

1.1.1. Common-Cause Hypothesis

The within-time correlation found in the existing literature may be explained by a common-cause model where engagement in the parallel processes of smoking and partner violence victimization across time are compelled by similar risk factors. There are several common influences that have been identified as risk factors for both smoking and partner violence victimization. For instance, lower socio-economic status has been associated with higher risks of reporting partner violence victimization (Campbell, 2002) and is a significant risk factor for smoking behavior (Gilman, Abrams, & Buka, 2003). Similarly, experiencing physical abuse as a child increases a person's risk of being a victim of partner violence in adolescence, young adulthood, and adulthood (Fang & Corso, 2007; Coid et al., 2001; Smith, White, & Holland, 2003; Sunday et al., 2011; Desai, Arias, Thompson, & Basile, 2002) and of ever using tobacco and daily cigarette smoking in adulthood (Mersky & Topitzes, 2010; Topitzes, Mersky, & Reynolds, 2010). Depression is also a common risk factor for both partner violence victimization (Howard & Wang, 2003; Lehrer, Buka, Gortmaker, & Shrier, 2006) and daily smoking and

nicotine dependence (Breslau, Peterson, Schultz, Chilcoat, & Andreski, 1998; Fergusson, Goodwin, & Horwood, 2003). It could be that smoking and partner violence victimization are correlated but not causally associated and that the within-time relationship reflects a common cause risk profile. Figure 2.1, Panel A, depicts the conceptual common cause model of smoking and partner violence.

1.1.2. Self-Medication Hypothesis

The most common theoretical explanation of the link between smoking and partner violence victimization is grounded in a stress and coping framework, where partner violence victimization constitutes the stress and smoking the coping mechanism. Self-medication theory, a subset of stress-coping, posits that experiences of trauma can lead to higher levels of substance use in an attempt to cope or self-medicate (Khantzian, 1985; Khantzian, 1997). Empirical studies have shown that people use alcohol (O’Leary & Schumacher, 2003) and illicit drugs (Kilpatrick, Acierno, Resnick, Saunders, & Best, 1997; Testa, Livingston, & Leonard, 2003) to self-medicate. It has been suggested in the literature that smoking is also used as a self-medication behavior, where the act of smoking becomes a maladaptive coping mechanism for the individual exposed to stressors such as partner conflict and relationship violence (Baker, Piper, McCarthy, Majeskie, & Fiore, 2004; Khantzian, 1985; Scott-Storey et al., 2009). Nicotine has in fact been found to reduce stress, depression and anxiety, albeit temporarily (Baker et al., 2004; Picciotto, Brunzell, & Caldarone, 2002).

Focusing on smoking may be particularly important in understanding, and potentially disrupting, the relationship between victimization and substance use in general because compared to other drugs smoking is legal, widely available, and to some extent remains socially acceptable (U.S. Department of Health and Human Services, 2014), is believed by the general

population to reduce stress (Warburton, 1992), and is one of the first substances most commonly used by youth (Johnston, O'Malley, & Bachman, 2003). All these factors may promote the selection of smoking as the substance of choice to self-medicate with among individuals with stressful life events including partner violence victimization. Figure 2.1, Panel B, depicts the self-medication pathways where partner violence victimization predicts future smoking.

1.1.3. Lifestyle Hypothesis

An alternative directional pathway between smoking and partner violence victimization is the Lifestyle Theory (Hindelang, Gottfredson, & Garofalo, 1978; Riley, 1987), which hypothesizes that engagement in risky social behaviors, such as substance use, increases the risk for victimization because of heightened exposure to delinquent others, impaired judgment, and lowered inhibitions (Brooks-Russell, Foshee, & Ennett, 2013). Empirical support has been found for the Lifestyle Theory. An individual's alcohol and illicit drug use has been found to increase the risk for partner violence victimization in longitudinal studies (Foshee et al., 2004; Huizinga et al., 2003; Vezina et al., 2011), most likely operating through the mechanisms of lowered inhibition and impaired judgment. Vezina et al. (2011) also found that higher associations with deviant peers defined by factors such as crime involvement and smoking, predicted increased dating violence victimization and that this link was only partially mediated by the individual's engagement in a risky lifestyle, thus supporting the Lifestyle Theory's mechanism of heightened exposure to delinquent others for increasing the risk of victimization.

It is plausible that smoking uniquely increases an individual's risk for victimization as well and that the primary mechanism this operates through is increased exposure to individuals who engage in risky behavior. People who smoke are more likely to partner with other smokers (Banks, Kelley, & Smith, 2013) who via their own smoking behavior are also likely to use other

substances and be engaged in delinquent behaviors in adulthood. Having a partner who uses alcohol and illicit drugs has been found to increase violence perpetration and victimization (Stuart et al., 2006; Stuart et al., 2008). Further, Carney and colleagues (2013) found that adolescents who reported smoking as well as participating in delinquent behaviors was significantly more likely to persist in their delinquency at later time points compared to delinquent adolescents who did not smoke. This interaction was not found for alcohol or other drugs indicating that smoking plays a unique role, beyond that of other drugs, in predicting engagement in chronic delinquent behaviors and risky activities that have been identified as significant risk factors for victimization (Vezina et al., 2011). Thus, smoking, beyond other substances, may uniquely place people at increased risk for victimization. However, to date this model has not been tested with smoking specifically. Figure 2.1, Panel C, depicts the conceptual pathways of Lifestyle Theory for smoking and partner violence victimization.

1.1.4. Reciprocal/Bidirectional Hypothesis

Lastly, it may be that these processes – the common-cause model, Self-Medication Model, and Lifestyle Theory – work in tandem. There is evidence in the substance use literature that a reciprocal process occurs. One study found that delinquent behavior, including substance use, increased the risk for victimization and victimization increased the risk of delinquent behavior and substance use (Begle et al., 2011) creating a vicious cycle between the two phenomena. This model has not been tested specifically looking at the smoking-partner violence victimization link. In the present study reciprocal influences will be assessed across four time periods; this is depicted in Figure 2.1, Panel D.

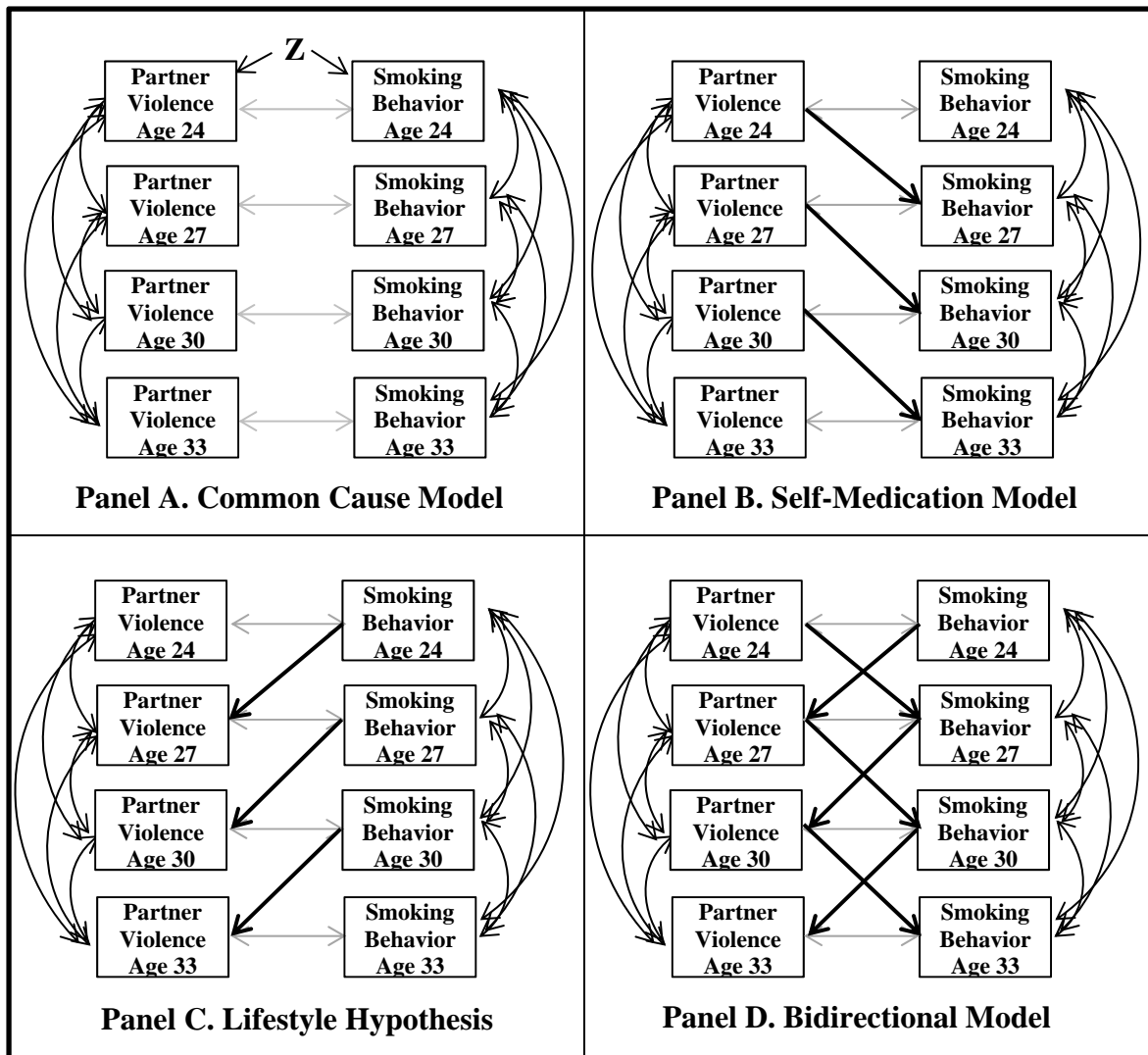


Figure 2.1. The models of reciprocal influence of smoking behavior and partner violence victimization. Panel A. Common cause model assuming within-time relationships between smoking and partner conflict. Panel B. Self-medication model where partner conflict precedes smoking. Panel C. Lifestyle hypothesis where smoking predicts engagement in partner conflict at later time points. Panel D. Bidirectional model where smoking and partner violence influence mutually unfolds over time.

1.2. Potential Role of Gender

Gender may play an important role in the link between smoking and partner violence victimization (Crane et al., 2013). Differences in risk factors for alcohol use and victimization have been noted between genders (Nolen-Hoeksema, 2004; Nolen-Hoeksema & Hilt, 2006;

Schulte, Ramo, & Brown, 2009; Saner & Ellickson, 1996) suggesting that the common cause model may look different for men and women and be substance specific. Gender differences have also been noted in the strength of the connection between alcohol and illicit drugs and being a victim of partner violence. For example, several studies have found that the strength of association between alcohol use and partner violence victimization is more robust for women than for men (Renner & Whitney, 2012; Temple, Weston, Stuart, & Marshall, 2008; Foran & O'Leary, 2008). There is also evidence that the direction of influence differs for men and women. Specifically, Self-Medication theory, in which victimization precipitates substance use has been better supported in relationship with alcohol (Testa et al., 2003; Testa, 2004) and drug use (Salomon, Bassuk, & Huntington, 2002) primarily for women whereas the Lifestyle Hypothesis, where engagement in alcohol use and delinquent behaviors, predicted later victimization for men but not for women (Foshee et al., 2004). Lastly, longitudinal research has found that women are more likely than men to use alcohol or meet criteria for an alcohol related diagnosis as a direct consequence of child victimization and that this influence extends across more developmental periods (Lansford, Dodge, Pettit, & Bates, 2010; Widom, Marmorstein, & White, 2006).

Based on the findings from the substance use literature focused on alcohol and other drugs, gender differences may also exist in the smoking-partner violence victimization link. Risk factors for smoking onset and use appear to vary by gender (Vezina et al., 2011; Crane et al., 2013). For example, childhood physical abuse predicted adolescent smoking frequency for males but not females (Kristman-Valente, Brown, & Herrenkohl, 2013). Further, the underlying mechanisms that support the emergence of gender difference in the directional influence between alcohol and partner violence victimization are likely similar for smoking. For instance, males and

females choose different coping mechanisms with women more likely than men to choose internalizing behaviors including substance use (Diehl, Coyle, & Labouvie-Vief, 1996). Studies have found that females, compared to males, are more likely to begin smoking as a result of stress (Byrne & Mazanov, 2003), indicating that females are more likely to select smoking as a coping mechanism. Unfortunately, to date very few studies that investigated the relationship between smoking and partner violence victimization looked at gender differences (Crane et al., 2013). A better understanding of the role that gender plays in the relationship between smoking and partner violence victimization is potentially important because smoking cessation efforts to date have been less successful with women than with men (Dodgen, 2005), and new targeted cessation efforts are needed in order to further reduce the public health burden of smoking.

1.3. Aims of the Current Study

Longitudinal studies of health and behavioral outcomes can help disentangle the role of smoking from other contextual factors as well as improve the understanding of the developmental etiology of the relationship between smoking and partner violence. To date most longitudinal work that looks at victimization and its relationship with health outcomes has been relegated to one developmental period (violence within adolescence, for example Foshee et al., 2007), has used samples with only a single gender (Jun, Rich-Edwards, Boynton-Jarrett & Wright, 2008), or has not employed advanced statistical analyses that can account for multiple competing confounds (Plichta, 2004).

The present study contributes to the growing body of literature examining the relationship between smoking and partner violence victimization by using a longitudinal, dual-gendered, community sample, spanning multiple developmental periods, while accounting for other potential confounding factors in order to better understand the unique relationship between

partner violence victimization and smoking.

The current study used data from the Seattle Social Development Project (SSDP). SSDP is a theory-driven developmental study of childhood and adolescent risk and protective factors for substance abuse and related health and behavior problems including partner violence.

This study will explore 1) if smoking behavior is related to partner violence victimization within time across four assessment points, 2) if partner violence victimization is predictive of smoking or if smoking is predictive of partner violence victimization, beyond within-time associations, 3) if partner violence victimization has a unique influence on smoking behavior after accounting for other explanations (e.g., poverty, education, ethnicity, mental health diagnosis, and other substance use), and, 4) whether the strength, directionality, and longitudinal persistence of this relationship differs for women and men.

2. Methods

2.1. Participants

The SSDP sample was recruited in 1985, when students were in the fifth grade, from 18 elementary schools in high crime neighborhoods in Seattle, WA, to participate in a multicomponent intervention study focused on protective and risk factors. From a population of 1,053 students, 808 students (76.7% of the population) consented to participate in the longitudinal study, and they constitute the ongoing SSDP sample, the sample used in the current analysis. Retention of participants has been high—92% at age 33. The study is gender balanced: 49% (396) are females and 51% (412) are males. For more information on the original study please see Hawkins, Catalano, Morrison, O'Donnell, Abbott, and Day (1992).

Nonparticipation at each assessment wave was not related to gender; lifetime use of tobacco, alcohol, or participation in delinquency by age 10; nor consistently related to ethnicity. These

retention rates exceed Hansen, Tobler, and Graham's (1990) estimated target retention rate of 87% for studies of 3 or more years' duration required to minimize threats to internal and external validity.

2.2. Measures

2.2.1. Smoking Behavior

The majority of studies that have explored the relationship between smoking and partner violence have been criticized for their limited measurement of smoking. The current study, following suggestions by Okoli et al. (2011), combined multiple items regarding smoking behavior to group smokers by similar behavior and risk at each age during young adulthood and adulthood (24-33). Using prospective self-reports of having ever smoked, frequency of use in the past month, and nicotine dependence in the past year based on the Diagnostic Interview Schedule (DIS) (Robins, Cottler, Bucholz, & Compton, 1981), participants were categorized by increasing intensity and risk related to smoking, where 0 = non-smokers, 1 = past user but not current, 2 = casual smoker, 3 = daily smoker, and 4 = smoker who meets diagnostic criteria for nicotine dependence. Missingness in response to smoking items was low (<7%) across time. When needed, forward or backward procedures were used to fill in missing data. For example, if at age 27 the response to "Have you ever smoked?" was missing but the respondent had reported smoking at a prior assessment between ages 10-21 they were coded at age 24 as having smoked. This resulted in 56 cases where no information on smoking was available and were excluded from the analysis.

2.2.2. Intimate Partner Violence

As suggested by Cho (2012), latent variable modeling was utilized to develop a comprehensive model of partner violence victimization using quantitative data. Questions from

the Revised Conflict Tactic Scale (CTS2) were used to assess *Partner Violence Victimization* in the SSDP sample at ages 24-33. Items representing three subscales of the CTS2—Verbal conflict, Physical conflict, and Injury—were used. An example question is, “Has your partner ever pushed, or shoved you?” Four items were used to identify a latent variable of physical partner violence victimization at each age. Latent constructs were tested for measurement invariance across gender and time. Strong invariance (configural, metric and scalar) was determined across gender within time was determined in each year, and configural, metric and partial-scalar temporal invariance was found across all age points. For a more in-depth description of the partner violence victimization variable construction, see Chapter 1. Model-predicted factor scores were used in the present analysis to facilitate estimation of the models.

2.2.3. Covariates

Confounders suggested by Crane et al. (2013) and Fletcher (2010) were used as covariates in the model, including socioeconomic status, race/ethnicity, major depression, and alcohol abuse/dependence, and were included in the final analyses to best determine the unique and developmental relationship between partner violence and smoking.

Ethnicity was dummy coded with white as the referent category. Three variables were created using the dummy code: African American, Native American, and Asian American.

Socio-economic Status was a dichotomous variable, based on parent report of their children’s eligibility for free and reduced-price school lunch when they were enrolled in grades 5, 6, or 7.

Childhood Physical Abuse (CPA) (before age of 10) was measured by items from the Childhood Trauma Questionnaire short form (CTQ-SF) (Bernstein et al., 2003), which was administered in the SSDP study at age 24 to retrospectively assess experiences of child

maltreatment. For the present study, if a participant endorsed a physical abuse item from the CTQ and reported that this occurred prior to the age of 10, the participant was assigned a “1” indicating the experience of CPA and a “0” indicating no CPA experience prior to the age of 10.

Alcohol abuse or dependence diagnosis at age 21. In the SSDP study, DSM-IV diagnosis and criterion counts of alcohol abuse and dependence were assessed at age 21 using the Diagnostic Interview Schedule (DIS) (Robins et al., 1995). A dichotomous item was created indicating whether the participant met diagnostic criteria for alcohol abuse or dependence in the past year.

Major Depression diagnosis at age 21. The DIS was also used to create past-year diagnostic measures for major depression disorder based on the American Psychiatric Association’s DSM-IV diagnostic criteria (Herrenkohl, Lee, Kosterman, & Hawkins, 2012; Lee, Kosterman, McCarty, Hill, & Hawkins, 2012). A dichotomous item was created at age 21 where 0- indicated not meeting criteria for major depression in the past year and 1 indicated meeting criteria for major depression.

2.2.4. Consideration of Intervention

During the elementary grades, a portion of the sample participated in a multicomponent social development intervention promoting positive youth development. Intervention effects have been found in level differences in predictors and outcomes (e.g., Hawkins, Catalano, Kosterman, Abbott, & Hill, 1999; Hawkins, Kosterman, Catalano, Hill, & Abbott, 2008). To address possible threats to validity and confounding effects of the intervention within etiological studies, extra caution has been used, including testing the etiological model for level differences in the strength of the relationship between study variables based on the *intervention assignment*. No etiological study to date in SSDP has found evidence of differences in associations among

variables across study conditions (e.g., Catalano & Hawkins, 1996; Huang, Kosterman, Catalano, Hawkins, & Abbott, 2001; Bailey, Hill, Oesterle, & Hawkins, 2006; Hill, Hawkins, Catalano, Abbott, & Guo, 2005). The current study examined multiple group analysis based on intervention assignment to determine if the etiological pathways between smoking and partner violence victimization differed between those participants who received the intervention and those that did not.

2.3. Data Analysis

First, to determine if there were level differences in the relationships between study variables based on intervention status, a multiple group model was conducted on the full model covariance matrix, including risk factors, covariates, smoking behavior, and partner violence victimization across all time points. Second, to address the primary aims of the study correlations were estimated within and across time to determine bivariate relationships between smoking and partner violence victimization. Third, competing models were compared to determine the best model fit. Fourth, a cross-lag model was selected to investigate the directionality, strength, continuity, and discontinuity of the relationship between partner violence victimization and smoking over time. Fifth, covariates (e.g., poverty, education, ethnicity, major depression diagnosis and alcohol abuse/dependence diagnosis) were added to the model to determine if partner violence victimization and smoking behavior have a unique relationship after accounting for other confounds.

Finally, to determine gender differences, all relationships were initially examined separately for men and women and formally tested for gender differences using multiple-group analyses (MGA). MGAs compare the fit of an unconstrained model, where all parameter estimates are allowed to vary for males and females, to a constrained model, where these parameters are

forced to be estimated with equal parameter estimates for males and females. A significant gender difference is indicated if the model with the unconstrained coefficient fits the data better than a model that constrains the coefficient to be equal for women and men, as indicated by a chi-square difference test. Model fit was assessed using the following criteria: the Comparative Fit Index (CFI) (Bentler, 1990; McDonald & Marsh, 1990), Tucker Lewis Index (TLI) (Tucker & Lewis, 1973), and the Root Mean Square Error of Approximation (RMSEA) (Steiger, 1990; Browne & Cudeck, 1993). Recommendations for cut-offs on goodness of fit include the following: $RMSEA \leq .06$, $CFI \geq .95$, and the $TLI \geq .95$. The CFI, TLI, and RMSEA have been found to be appropriate for use with categorical variables (Muthen & Muthen, 2012).

All analyses were conducted using Mplus version 6.1 and Full Information Maximum Likelihood (FIML) estimation to address missing data and to obtain optimally unbiased estimates of model parameters and their standard errors as suggested by Schafer & Graham (2002). In the full model the number of cases with no information on the dependent variables ($n=109$, 14%) was less than 20% of the total sample, which indicates a low threat of substantial bias in estimates, as described in Arbuckle (1996). These cases were not included in the present analysis resulting in a final sample size of $n=699$.

3. Results

3.1. Intervention Effects

A multiple group model analysis revealed that a constrained model, where all pathways were forced to equality between those that received the intervention and those that did not, fit the data well ($\chi^2=140.774$ (120), $p=.095$, RMSEA: .021, CFI: .999, TLI: .998). When tested against a model that allowed the pathways to estimate freely between intervention and control the chi-square test of difference was non-significant ($\chi^2=141.828$ (120) $p=.085$) indicating that allowing

pathways to be freely estimated did not significantly improve the fit and that the strengths of association between smoking behavior, partner violence victimization, and other study variables were not significantly different based on receipt of the intervention. As a result of these findings, all further analyses were conducted using the full sample undifferentiated by intervention.

3.2. Descriptive Statistics

Demographic information on study variables for the full sample and by gender is provided in Table 2.1 Partner violence victimization was commonly experienced at all ages. The prevalence of partner violence victimization in this sample, ranging from 21-36% between the ages of 24 and 33, is similar to findings from other samples (Halpern, Spriggs, Martin, & Kupper, 2001; Renner & Whitney, 2012). Factor scores of partner violence victimization did not significantly differ for men and women at any age in the current analysis. In the present study, smoking was prevalent, with 77% of participants having reported smoking at least once in their lifetime by age 33. Overall, men and women reported similar smoking behaviors, with a few exceptions. At age 24, men were more likely to report daily smoking at age 27 (χ^2_{diff} : 12.558 (4) $p=.014$) and at age 30 (χ^2_{diff} : 13.062 (4) $p=.011$). At age 21, men were more likely than women to have met criteria for an alcohol abuse or dependence diagnosis (χ^2_{diff} : 31.450 (1) $p=.000$).

Table 2.1
Demographic Information of Partner Violence Victimization, Smoking, and Covariates by Age/Gender

Variable	Age	Full Sample n=808	Males n=412	Females n=396
Any Partner Violence Victimization				
	24	201 (25%)	88 (21%)	113 (29%)
	27	167 (21%)	84 (20%)	83 (21%)
	30	288 (36%)	139 (34%)	149 (38%)
	33	285 (35%)	134 (33%)	151 (38%)
Smoking Behavior				
		Nonsmoker	74 (18%)	85 (22%)
		Past, not current	173 (42%)	191 (48%)
	24	Casual	26 (6%)	20 (6%)
		Daily	91 (22%)	73 (18%)
		Nicotine Dx.	34 (8%)	19 (5%)
		Nonsmoker	60 (15%)	80 (20%)
		Past, not current	195 (47%)	194 (49%)
	27	Casual	18 (4%)	27 (7%)
		Daily	60 (15%)	35 (9%)
		Nicotine Dx.	65 (16%)	52 (13%)
		Nonsmoker	53 (13%)	74 (19%)
		Past, not current	204 (50%)	200 (51%)
	30	Casual	22 (5%)	16 (4%)
		Daily	65 (16%)	51 (13%)
		Nicotine Dx.	51 (12%)	41 (10%)
		Nonsmoker	52 (13%)	75 (19%)
		Past, not current	216 (52%)	222 (56%)
	33	Casual	16 (4%)	11 (3%)
		Daily	42 (10%)	24 (6%)
		Nicotine Dx.	72 (18%)	52 (13%)
Major Depression	21	150 (19%)	66 (16%)	84 (21%)
Alcohol Abuse or Dx.	21	203 (25%)	136 (33%)	67 (17%)
Physical Abuse	≤ 10	138 (17%)	80 (19%)	58 (15%)

Notes: Significant gender differences are in **bold**.

Bivariate correlations among partner violence factors across the four time periods ranged from .40 to .86, and among smoking factors ranged from .81 to .90, indicating strong continuity in both smoking and partner violence victimization across time. All partner violence

victimization factors were significantly correlated with smoking behaviors at each time point at $p < .000$ and ranged from .15 to .23 in strength (Table 2.2).

Table 2.2
*Bivariate Correlation between Partner Violence Victimization and Smoking Behavior
Ages 24-33
Full Sample and by Gender*

	1.			2.			3.			4.			5.			6.			7.			8.
	FS	M	F	FS	M	F	FS	M	F	FS	M	F	FS	M	F	FS	M	F	FS	M	F	
1. IPV24			1																			
2. IPV27	.62	.65	.60			1																
3. IPV30	.40	.41	.38	.86	.87	.84			1													
4. IPV33	.65	.66	.64	.86	.86	.86	.86	.84	.88			1										
5. SMK24	.17	.19	.16	.17	.16	.17	.15	.16	.13	.15	.16	.13			1							
6. SMK27	.20	.24	.17	.21	.23	.20	.19	.23	.16	.20	.24	.15	.88	.85	.91			1				
7. SMK30	.23	.29	.17	.22	.22	.21	.19	.19	.18	.21	.24	.17	.81	.74	.88	.89	.84	.93			1	
8. SMK33	.20	.24	.18	.20	.19	.21	.18	.17	.18	.20	.21	.19	.84	.78	.90	.89	.85	.94	.90	.86	.94	1

Notes: All Standardized Coefficients were * $p < .05$

FS – Full Sample Correlation distinguished in **bold**; M – Males Bivariate Correlation Standardized Coefficient; F- Female Bivariate Standardized Coefficient; IPV – Intimate Partner Violence (Age); SMK – Smoking Behavior (Age)

3.3. Model Selection

Based on the strong continuity within construct suggested in the bivariate correlations shown in Table 2.2, a continuity model was used as a baseline model to explore the relationship between partner violence victimization and smoking across time. The baseline model was tested against models representing the addition of Self-Medication pathways, Lifestyle Theory pathways and reciprocal pathways to determine if the addition of these pathways helped explain the relationship better than the baseline model alone (Table 2.3). The addition of the Self-Medication pathways did not significantly increase model fit (χ^2_{diff} : 5.282 (3) $p = .1523$), nor did the addition of the Lifestyle pathways (χ^2_{diff} : 2.259 (3) $p = .5204$).

Table 2.3
Fit statistics for Models of Reciprocal Influence between Partner Violence Victimization and Smoking Behavior Ages 24-33

Model	χ^2	Df	p	RMSEA	CFI	TLI
Continuity	228.249	66	<.000	.059	.990	.985
Self-Medication	232.615	63	<.000	.062	.989	.984
			Self-Medication vs Continuity		χ^2 diff: 5.282 (3)	p=.1523
Lifestyle Hypothesis	235.386	63	<.000	.063	.989	.984
			Lifestyle vs Continuity		χ^2 diff: 2.259 (3)	p=.5204
Bidirectional	220.623	60	<.000	.062	.990	.984
			Bidirectional vs Continuity		χ^2 diff: 11.196 (6)	p=.0825

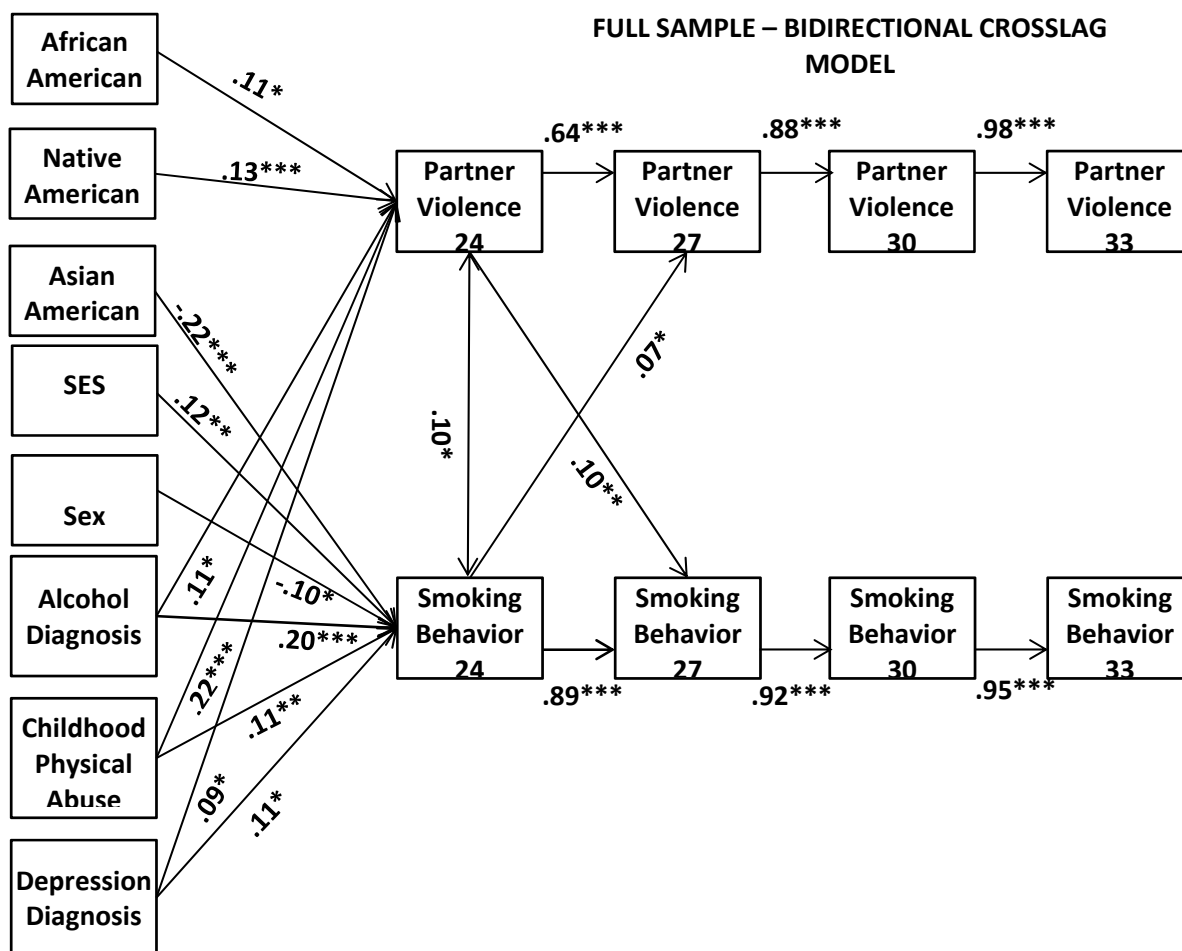
Note: RMSEA: Root mean square error of approximation; CFI: Comparative Fit index; TLI: Tucker-Lewis Index

However, the addition of the bidirectional pathways marginally improved the model fit, suggesting that the inclusion of bidirectional lagged pathways from smoking to partner violence victimization and partner violence victimization to smoking helped explain the relationship between the two phenomena when compared to the continuity model alone (χ^2 diff: 11.196 (6) p=.0825). When the bidirectional model was tested against the Self-Medication and Lifestyle models, the bidirectional model fit the data significantly better (χ^2 diff: 14.619 (3) p=.002) than the Lifestyle model and marginally better than the Self-Medication model (χ^2 diff: 6.795 (3) p=.0787). To answer the remaining study questions about the unique role that partner violence plays in smoking behavior, above other risk factors, the bidirectional model (Figure 2.1, Panel D) was selected.

3.3.2. Contextual Cross-Lag Analysis

To determine if smoking behavior was uniquely related to partner violence victimization, the bi-directional model was tested, including risk factors and covariates. The final bidirectional model, using the full sample, including all covariates listed above, fit the data well (χ^2 220.623

(60), $p < .000$, CFI=.99, TLI: .98 RMSEA=.062). There was strong continuity for both smoking and partner violence victimization (β range: .64 to .98, partner conflict; .89 to .95, smoking), within-time association at age 24, and reciprocal bidirectional relationship between partner conflict and smoking from age 24 to age 27 (as shown in Figure 2.2) suggesting that experiencing partner violence victimization at age 24 uniquely predicted increased smoking behavior at age 27 and that smoking behavior at age 24 uniquely predicted heightened exposure to partner violence victimization at age 27 even after controlling for covariates and accounting for the high within construct continuity.



NOTES: **SES:** Socio-economic status defined by eligibility for free and reduced lunch during grades 5, 6 and 7. **SEX:** 1-Male, 2-Female. **Physical Abuse:** Childhood physical abuse before age 10. **Alcohol Diagnosis:** Meeting criteria for alcohol abuse or dependence diagnosis at age 21. **Depression Diagnosis:** Meeting criteria for Major Depression at age 21.

*** $p < .001$; ** $p < .01$; * $p < .05$

Figure 2.2. Cross-Lag model of Partner Violence Victimization and Smoking Behavior from ages 24-33.

3.2. Gender Differences

To assess whether the model differed for men and women, multiple group analysis was conducted on the final model depicted above (Figure 2.2). A freely estimated model where pathways were allowed to differ for men and women, fit the data well (χ^2 245.326 (108) $p < .000$,

RMSEA: .06; CFI: .992, TLI: .987). This model was tested against a fully constrained model, where pathways were forced to be equal for men and women (χ^2 220.564 (137) $p < .000$, RMSEA: .04; CFI: .995; TLI: .994). A chi-square test of difference indicated that the constrained model fit the data significantly less well (χ^2 diff: 46.786 (29) $p = .0196$) and that further investigation was needed into which pathways were significantly different for men and women. Pathways were systematically constrained one at a time and tested against a fully free model to determine which pathways significantly differed for men and women.

3.2.1. Gender Differences in the Smoking – Partner Violence Victimization Link

No cross-lagged pathways between smoking behavior and partner violence victimization were found to differ significantly for men and women. The within-time association between Smoking at age 24 and Partner Violence Victimization at age 24 was significant for men ($\beta = .14$, $p < .05$) but not for women ($\beta = .07$, $p > .05$), though this was not statistically different. Both the male and female final models suggested a Self-Medication pathway where partner violence victimization at age 24 increases smoking behavior at age 27, but there was less support for the Lifestyle pathway where smoking at age 24 increases the likelihood of later partner violence victimization at age 27, as in the full model. Compared to the model estimated in the full sample (men and women combined), smoking at age 24 did not significantly predict higher partner violence victimization at age 27 for men ($\beta = .05$, $SE = .049$, $p > .05$) and only marginally predicted partner violence victimization for women ($\beta = .09$, $SE = .045$, $p = .07$); these coefficients did not differ significantly when tested using a chi-square test of difference. However, there may not have been sufficient power to detect the effect of smoking on predicting partner violence victimization in the multiple group models (Figure 2.3).

3.2.2. Gender Differences in Risk for Smoking and Partner Violence Victimization

There were several differing pathways in the model when run separately for men and women. The following pathways were identified as being significantly different for men and women in the model: Asian women compared to Asian men were significantly less likely to smoke (χ^2_{diff} : 5.495 (1) $p=.0191$); a diagnosis of alcohol abuse or dependence at age 21 was a stronger predictor of smoking behavior at age 24 for women than for men (χ^2_{diff} : 5.614 (1) $p=.0178$); and Smoking behavior at age 24 was a stronger predictor of smoking behavior at age 27 for women than for men (χ^2_{diff} : 6.672 (1) $p=.0098$). Other pathways that appeared to differ in the stratified model, such as lower socio-economic status increasing smoking behavior at age 24 for women but not for men, did not differ significantly when tested using a chi-square test for difference. The final model, allowing pathways linking Asian ethnicity and alcohol use diagnosis to Smoking Behavior at age 24 and Smoking Behavior at age 24 to Smoking Behavior at age 27 to be estimated freely across males and females while all other pathways were constrained to equality, fit the data well (χ^2 171.446 (134) $p=.0160$, RMSEA: .03; CFI: .998, TLI: .997).

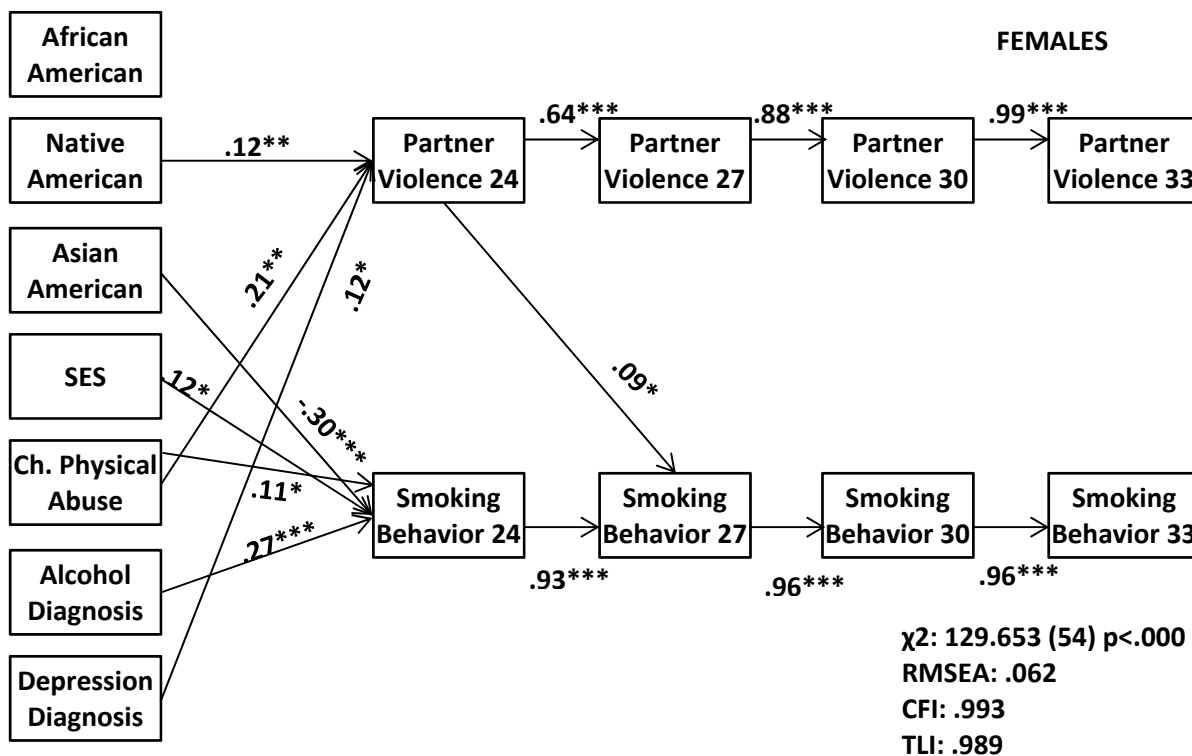
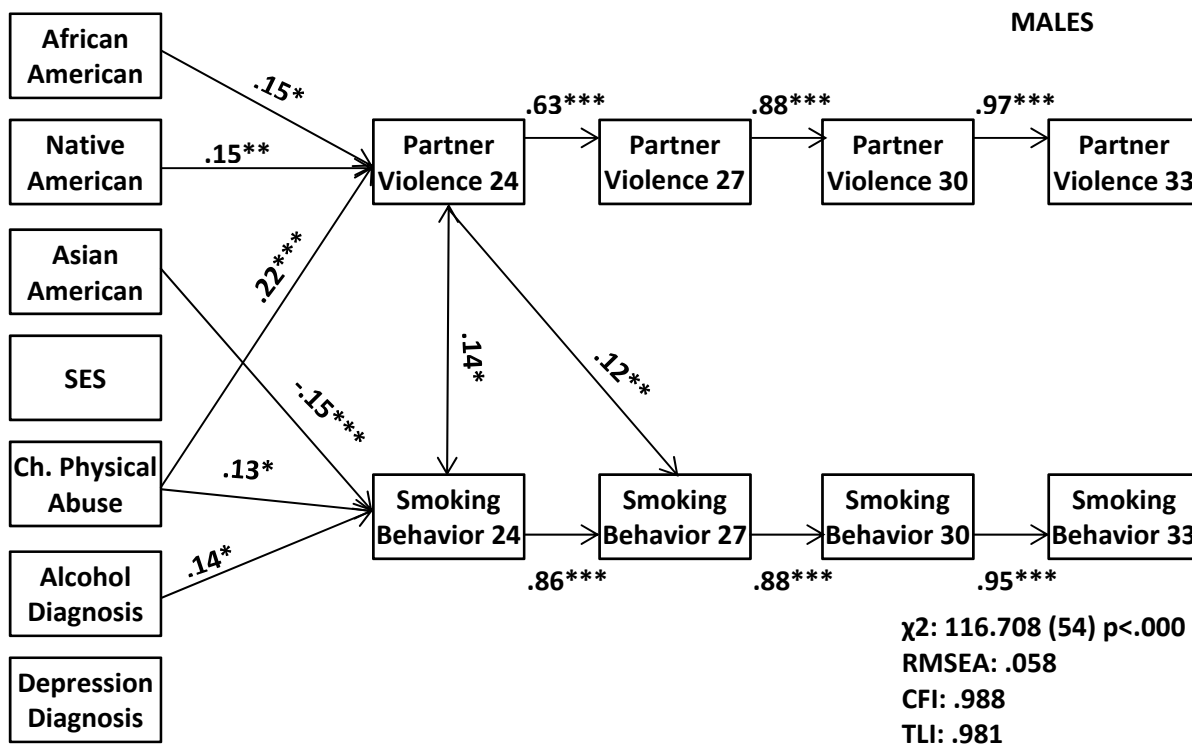


Figure 2.3 Cross-Lag model of Partner Violence Victimization and Smoking Behavior from ages 24-33 by gender. *** $p < .001$; ** $p < .01$; * $p < .05$

4. Discussion

This study attempted to further the understanding of the longitudinal relationship between partner violence victimization and smoking in young adulthood and adulthood from age 24 to age 33 by disentangling the unique contribution adjusting for earlier risk and confounding factors; and to determine if these processes differed or were similar for men and women.

Bivariate within-time associations between partner violence victimization and smoking behavior were found at each age (24, 27, 30, 33) supporting prior cross-sectional findings. However, when contextual factors were added to the model only the within-time association at age 24 remained significant. This is most likely due to the high within-construct continuity across time for both smoking behavior and partner violence victimization. Developmental psychopathology posits that early behavior and experience are among the strongest influences on behaviors and experiences in later developmental periods (Rutter & Sroufe, 2000). The present findings corroborate those of prior studies that found that early smoking increases the risk for later smoking and early victimization increases the risk for re-victimization later in life (U.S. Department of Health and Human Services, 2012; Barnes, Noll, Putnam, & Trinkett, 2009; Classen, Palesh, & Aggarwal, 2005), indicating that once an individual is situated on a pathway of partner violence victimization or smoking, there is a high likelihood he or she will continue on it.

While the continuity of partner violence victimization and smoking within the common cause model (Figure 2.1, Panel A) was the strongest predictor of each behavior in the future, the addition of bidirectional-reciprocal pathways to the model marginally increased model fit suggesting that those pathways helped to explain the relationship between smoking and partner

violence victimization from age 24 to 33. This suggests that the stress of partner violence victimization may increase smoking behavior in a Self-Medication process, but also that smoking may influence further engagement in conflictual relationships, thereby increasing the risk for victimization via the Lifestyle Theory process. The bi-directional relationship between smoking and partner violence victimization was only found from young adulthood (age 24) to adulthood (age 27) suggesting there may be unique developmental risk factors or life events that occur in this transition that strengthen the bidirectional link between partner violence victimization and smoking behavior. It could also be that there was no further variance left to be explained at later time periods due to the strong stability of these behaviors/experiences. One mechanism may be the nature of the individual's partnership. Crane et al. (2013) suggested that relationship status (i.e., dating vs. marriage) may explain developmental differences in the link between smoking and partner violence victimization experienced in younger periods versus older periods though their meta-analysis did not find significant differences when they looked at the association between smoking and partner violence victimization across dating and married partners across studies. It is worth noting that the studies in Crane's meta-analysis are primarily cross-sectional, mostly single-gendered (female) samples, and the vast majority of studies used current smoking as the outcome. It may be that these limitations obscured the detection of differences based on partnership status. Future research should explore the role of relationship stability and volatility as influencing smoking adoption as a coping strategy for partner conflict, and smoking as influencing selection of risky partners, which may increase the risk for partner violence victimization.

The findings herein suggest that even after controlling for potential confounders, a link exists between partner violence and smoking behavior that is particularly salient in the

developmental period of young adulthood. The relationship between partner violence victimization and smoking behavior persisted even after the inclusion of other risks such as alcohol abuse and dependence at age 21, major depression at age 21, and childhood physical abuse before age 10, and confounding factors such as SES and ethnicity. This finding highlights that the omission of smoking from investigations linking substance use and partner violence victimization may lead to missing a critical factor that could be targeted for intervention.

In general, gender differences were not detected in the relationship between smoking and partner violence victimization. The within-time association between smoking behavior and partner violence victimization at age 24 was significant for men but not for women; however, when tested formally the strength of the coefficient did not differ significantly for men and women. The analysis of gender differences showed that the predictive influence of smoking at age 24 to partner violence victimization at age 27 was non-significant for men, whereas it was marginally significant for women, but the difference between the two genders was again not statistically significant. The non-significance of these pathways in the model separated by gender should be interpreted with caution. It could be that these relationships indeed exist, and that by reducing the sample size in the MGA, the power to detect these relationships was lost. Insufficient power has been noted as particularly problematic in substance use research, and it has been estimated that most psychological research has only a 50% chance of accurately detecting an actual effect (MacKinnon & Lockwood, 2003). A power analysis using G*Power version 3.0.10 (Faul, Erdfelder, Lang, & Buchner, 2007) was used to test the effect size needed to detect group differences given the current model parameters. Within the model parameters, an effect size of .21 would have been needed in order to detect it with .80 power. Effect sizes in social science research are generally small with interaction effects estimated to explain only 1-

3% of outcomes (Aiken & West, 1991; Fairchild & MacKinnon, 2009). In the current study, the effect size was likely too small to detect at a statistically significant level. However, it is important to note that even small effects may be important to consider in prevention and intervention development (MacKinnon & Lockwood, 2003). Replication that investigates the bidirectional relationship between smoking and partner violence victimization is needed.

Gender differences were found in the risk factors for engaging in the smoking behavior pathway. A diagnosis of alcohol abuse or dependence at age 21 was a stronger predictor of future smoking behavior for women than for men. Alcohol diagnosis or heavy drinking has been associated with more severe smoking behavior such as higher frequency smoking and nicotine dependence (Pomerleau, Aubin, & Pomerleau, 1997). Gender differences in this relationship may also be driven by an underlying mechanism, such as Post-Traumatic Stress Disorder (PTSD), which is more prevalent in women (Breslau, 2001). The consequences of reaching clinically problematic alcohol use are more negative for women than for men (Nolen-Hoeksema, 2004), which may become an additional stressor in and of itself that potentially exacerbates the adoption of smoking behavior among women. The stronger relationship found for women could therefore reflect the higher rates of PTSD, more negative consequences of their diagnosis and the higher likelihood of adopting substance-use-related coping mechanisms. Future analyses are planned investigating the role of PTSD in the model.

Asian women were also less likely than Asian men to engage in smoking behavior at age 24 compared to the white referent group. This finding is consistent with other research that has found that compared to Asian men Asian women have significantly lower lifetime, past month, and daily smoking behaviors in adolescence (Wallace et al., 2003). In addition, the current data extend the developmental period of this difference through young adulthood.

4.1. Limitations

This secondary analysis study is not without limitations. The measure of partner violence victimization used in this study did not include measures of sexual violence in intimate partnerships, which may impact findings. Studies have found that the type of partner violence experienced may vary by developmental period and gender. Young adulthood has been characterized by a higher likelihood of sexual assault, whereas adulthood has been identified as a vulnerable period for domestic violence (Flitcraft, 1995). Further, males and females differ in the forms of partner violence they experience, with females being subjected to more severe forms of abuse, resulting in more injury (Archer, 2000). Exposure to multiple forms or more severe forms of violence has been found to significantly increase the risk of smoking among women (Jun, Rich-Edwards, Boynton-Jarrett & Wright, 2008; Weaver & Etzel, 2003). Especially for women who experience a high prevalence of sexual assault, not including sexual violence in the measure of partner conflict may underestimate the prevalence of partner violence victimization, particularly at younger time points, making the findings more conservative and less likely to demonstrate gender differences in the relationship with smoking behavior. The inclusion of the sexual coercion scale of the CTS2 has been noted as an area of importance for the identification of gender difference in the experience of partner violence (Hamby, 2009). Future research using longitudinal studies should investigate the reciprocal relationship of partner violence that includes sexual coercion and assault in its measure of the relationship between partner violence victimization and smoking behavior.

Lastly, the first reference point for partner violence victimization in this study was at age 24. Given the high continuity, it will be important to include earlier experiences of partner violence victimization in future work. This is particularly true in light of recent literature pointing to the significance and growth of dating violence among teens in the U.S., the relationship dating

violence has with smoking, and the noted gender differences in tobacco, alcohol, marijuana use, and other health indicators (Haynie et al., 2013). The addition of earlier violence exposure could aid in identifying a critical age for partner violence prevention efforts, potentially having long-term impacts on reducing smoking and future engagement with violent others.

4.2. Implications for Practice

Findings from this study can contribute to smoking cessation and relapse (secondary) prevention efforts for those individuals with comorbid smoking and partner violence victimization, a sub-population of smokers who are increasingly resistant to smoking cessation (Cohen & Lichstein, 1990; McFall et al., 2010). Findings here also point to the need for early primary and secondary prevention efforts delivered by clinicians working with men and women in substance use or domestic violence treatment settings, in order to reduce both partner conflict and smoking across the life-course. The presence of the directional link where smoking at age 24 predicts partner violence victimization at age 27 in the full sample and the marginal significance among women is a new and potentially important pathway between these two phenomena. Similar findings where smoking or nicotine dependence precedes trauma exposure have been identified (Perkonigg et al., 2000) who found that nicotine dependence increased the likelihood of being exposed to a traumatic event among adolescent and young adult participants from Munich. This study was cross-sectional and the current findings build on the directionality that smoking uniquely places an individual at risk for victimization even after considering other factors. The presence of this link may suggest that for some, engaging in smoking behavior, beyond other risk behaviors during young adulthood, may place them at heightened risk of experiencing partner violence victimization in adulthood and that this may particularly

meaningful for women. This again points to the need for coordinated primary prevention of smoking and partner violence.

Prevention efforts targeting these two phenomena have historically been conducted separately; however, findings from previous work and the current study support integration of violence prevention into substance treatment and integration of substance use intervention into violence programming (Vest et al., 2002; Reingle, Staras, Jennings, Branchini, & Maldonado-Molina, 2011). Successful interventions have been developed for the treatment of smoking among patients with Post Traumatic Stress Disorder (PTSD) the diagnostic manifestation of trauma commonly related to partner violence victimization. An example of this is the *VA Cooperative Studies Program: Integrating Tobacco Cessation into PTSD Care*, a brief intervention that has led to increased rates of cessation among veterans with PTSD (McFall et al., 2010). While promising, the existing smoking interventions for people with trauma are largely based upon samples composed primarily of males with combat-related PTSD, and it is not known if the success of these interventions will translate to a female population (Fu et al., 2007) or populations with different trauma types, such as partner violence. Continued investigation into the role that gender plays in the relationship between smoking and victimization is warranted to achieve greater insight into methods for developing new cessation efforts and a better understanding of the potential for decreasing the public health burden of smoking.

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CHAPTER 3.
**BECOMING A HIGH-RISK SMOKER IN YOUNG ADULTHOOD: A TEST OF
GENDER DIFFERENCES AND FOUR THEORETICAL PATHWAYS**

1. Introduction

Any amount of smoking is bad for an individual's health, but those who smoke daily or have a nicotine dependence diagnosis carry the highest risk of smoking-related disease and mortality, and struggle the most with quitting (Baumert et al., 2010). These smokers have been termed "high-risk smokers" (Mohiuddin et al., 2007) because they are markedly resistant to the smoking cessation efforts employed to date and bare the highest burden of tobacco-related health consequences (Warner & Mendez, 2010). What leads a person to become a high-risk smoker is still unclear, but future research is warranted to reduce the impact of smoking behavior in this high risk group.

In the 50 years since the release of the first *Surgeon General's Report on Smoking*, almost 21 million premature deaths have been caused by smoking and second-hand smoke (U.S. Department of Health and Human Services, 2014), and it is estimated that if nothing changes 5.6 million American youth will suffer the same fate. Indeed, the decline in smoking that we have seen in the past decades has all but stopped (Holford et al., 2014), due in part to ineffective prevention and cessation efforts targeting high-risk smokers (Warner & Mendez, 2010). If public health programs are going to continue to affect and lower the rates of smoking in the U.S., then research must focus on smoking issues among the highest risk groups. A better understanding of the differing mechanisms that lead to becoming a high-risk smoker is needed to develop more effective prevention and cessation interventions for, and thereby decrease smoking in, this highly resistant group, ultimately lowering the public health burden of smoking.

Over the past few decades, women have closed the gap in smoking prevalence, and, despite the successes of public health efforts in the general population, smoking-related deaths among women in the U.S. continue to rise (U.S. Department of Health and Human Services, 2014). However, little is known about how gender contributes to becoming a high-risk smoker (Baumert et al., 2010). Studies have suggested that escalation in smoking behavior to high-risk smoking may differ by gender, but findings are mixed. For instance, some studies have found that girls transition faster from being occasional smokers to daily smokers (Ariza-Cardenal & Nebot-Adell, 2002), while other studies point to boys' smoking severity progressing faster than girls' (Wetter et al., 2004). Considering the role of gender in how and why a person goes on to become a high-risk smoker is important in developing targeted intervention efforts where gender specificity may play an important role in maximizing prevention and cessation effectiveness among high-risk smokers.

1.1. Aims of the Current Study

This study is guided by three aims: first, the present study investigates five theoretical pathways to becoming a high-risk smoker in young adulthood, defined here as those people who smoke daily or who meet criteria for nicotine dependence by age 24, among a sample of people who have smoked at some point in their life; theoretical pathways include — *developmental psychopathology*, *stress-coping*, *self-medication* (a subset of stress-coping), *reverse-gateway*, and *social-developmental theory*.

Second, the study considers that these theoretical pathways in addition to directly influencing high-risk smoking in young adulthood may operate indirectly through earlier smoking behaviors. The current study examines the mediating effect of adolescent smoking behavior, age-of-smoking onset and duration of adolescent years of smoking, within each model.

And third, the study explores whether the theoretical models work equally well for men and women and whether the specific pathways from the proposed theoretical mechanisms (e.g., child maltreatment → high-risk smoking) are uniquely similar or dissimilar across gender.

1.2. Theoretical Pathways

1.2.1. Developmental Psychopathology: High-Risk Smoking Continuity

Becoming a high-risk smoker in young adulthood is likely influenced by being a high-risk smoker in an earlier time period. Developmental psychopathology posits that early behaviors are the strongest predictors of similar behaviors in later developmental periods and that this behavioral continuity is robust across time (Rutter & Sroufe, 2000). Thus, we would expect, and in fact find, that early smoking behavior increases the risk for later smoking (U.S. Department of Health and Human Services, 2012). Several studies have found a graded, dose-response relationship between adolescent smoking trajectories and adult regular smoking and nicotine dependence, where more severe smoking in adolescence, defined by regularity of use, leads to higher risk smoking in adulthood (Riggs, Chou, Li, & Pentz, 2007; Chassin, Presson, Sherman, & Edwards, 1990). When other proposed pathways to increased tobacco use are investigated in tandem with the continuity model, the alternate model prediction of later smoking is usually secondary to the continuity model's prediction of cigarette use. For instance one study found a significant pathway from adolescent marijuana use to increased smoking behavior but also noted that this pathway was not as strong as prior tobacco use behavior which was a stronger predictor of later smoking behavior (Patton, Coffey, Carlin, Sawyer, & Lynskey, 2005).

Two mechanisms of early tobacco use are decidedly important in the continuity model: age of onset and the extent of the involvement, including duration of use (Kandel & Faust, 1975; Kandel, Yamaguchi, & Chen, 1992). The most commonly investigated adolescent smoking risk

behavior for becoming a high-risk smoker in adulthood is early smoking onset. Most smoking onset occurs prior to age 18 (~87%), and almost all people who smoke as adults begin smoking before age 26 (98%) (U.S. Department of Health and Human Services, 2014). Early onset has in fact been found to be associated with becoming a regular smoker in young adulthood (Chassin et al., 1990; Buchmann et al., 2013), a higher frequency of smoking (Everett et al., 1999; Buchmann et al., 2013), daily smoking (Everett et al., 1999), and a greater likelihood of developing nicotine dependence (Buchmann et al., 2013). Studies comparing men's and women's age of smoking initiation to the likelihood of becoming a high-risk smoker have found that men who had initiated smoking at younger ages were 2.2-2.4 times as likely to be heavy smokers, while women were 4.5-5.4 times as likely to be heavy smokers when reporting a younger age of onset (Fernandez et al., 1999; D'Avanzo, La Vecchia, & Negri, 1994). The link between early onset and later problem substance use behavior has been found for substance use in general (Hawkins, Catalano, & Miller, 1992) but may be particularly true for smoking. Nicotine is highly addictive and the addiction process of smoking is thought to begin in adolescence (DiFranza et al., 2007). One study by DiFranza and colleagues (2000) found that for some adolescents, signs of addiction to nicotine occur at the first cigarette, largely attributed to the biochemical impact of nicotine on the brain (DiFranza et al., 2002; DiFranza et al., 2007).

An equally likely, though far less studied adolescent smoking risk behavior is the duration, or number of years, an adolescent smokes, which may also increase a person's risk for becoming a high-risk smoker. Using substances for longer periods of time has been indicated as a mechanism for becoming more problematic substance users later (Anthony & Petronis, 1995) most likely due to the repetition of use leading to habitual behavior (Lally, Van Jaarsveld, Potts, & Wardle, 2010). We would expect, and find, that adolescents who smoke for more years in

adolescence are more likely to become nicotine dependent in young adulthood (Van De Ven, Greenwood, Engles, Olson, & Patton, 2010; Timberlake et al., 2007). For example Van De Ven and colleagues (2010), using longitudinal data from Australian adolescents who smoked, found that a longer reported duration of ever smoking in adolescence, or more years reported smoking, increased the odds of developing nicotine dependence in young adulthood and that this relationship became stronger as the duration and severity of smoking behavior increased. Those participants who reported daily smoking between 3-6 assessment periods in adolescence were 5.2 times as likely to develop nicotine dependence compared to smokers who didn't smoke daily. Findings from this study point to the strong role of high-risk smoking continuity that begins in adolescence and extends through young adulthood.

Very little is known regarding gender differences in the continuity of high-risk smoking. Prevalence rates for age of onset, current, and regular use of cigarettes do not vary significantly by gender (Substance Abuse and Mental Health Services, 2011; Buchmann et al., 2013; U.S. Department of Health and Human Services, 2012). The equality of smoking prevalence across males and females is a newer phenomenon that has been attributed to changing gender norms around substance use behavior in recent decades (Waldron, 1991; Robinson & Klesges, 1997). Despite the parity in smoking prevalence it has been suggested that men and women begin and continue to smoke for different reasons (World Health Organization, 2003; Waldron, 1991) and the genetic and environmental determinants of smoking differ by gender (Hamilton et al., 2006). For example the risk factors predicting certain behaviors, like early onset, appear to differ for men and women (Simantov, Schoen, & Kein, 2003; Waldron, Lye & Brandon, 1991; Flay et al., 1994; Acierno et al., 2000). Simantov (2003) found that girls, when compared to boys, were more likely to identify stress relief and weight control as reasons for beginning to smoke,

whereas boys were more likely to report beginning to smoking to “be cool” with peers. Having a family member who has an alcohol or drug problem has been found to increase the risk of cigarette use among boys but not girls (Acierno et al., 2000). In general, girls become regular smokers later than boys (Robinson & Klesges, 1997). One study found that for girls, but not boys, family conflict was a significant risk factor for regular smoking in adolescence (Flay et al., 1998). Little is known about gender differences in the trajectories (i.e., onset, escalation and persistence) of smoking behavior. The little available evidence is mixed. Ariza-Cardenal and Nebot-Adell (2002) found that girls transition faster from being occasional smokers to daily smokers compared to boys, while Wetter et al. (2004) noted that boys’ smoking severity progressed faster than girls’. One study by Wills, McNamara, Vaccarro, and Hirky (1996) found that for women, but not men, earlier onset of smoking was predictive of reporting heavy smoking behavior in adulthood.

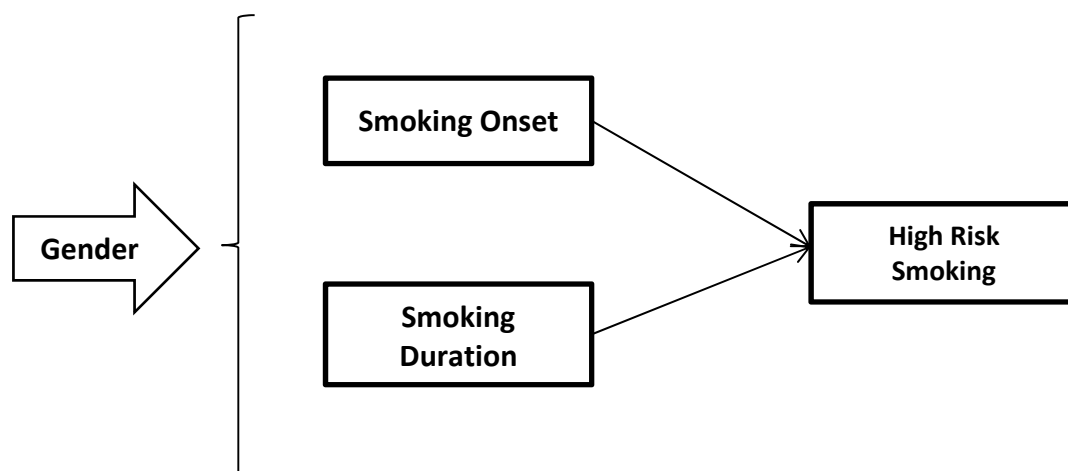


Figure 3.1. Proposed conceptual model of Developmental Psychopathology Model: High Risk Smoking Continuity

Given the critical role that adolescent smoking behavior plays in future smoking, it will be important to better understand whether this influence is similar or disparate across men and

women. The present study will examine whether age of smoking onset or duration of smoking years in adolescence influences becoming a high-risk smoker in young adulthood in two ways. First, the study will explore the direct influence of adolescent smoking behavior on becoming a high risk smoker and second, given the strong continuity suggested in the available literature, the present study will examine if the adolescent smoking behavior mediates the other four proposed pathways to becoming a high risk smoker in young adulthood. Lastly, the present study will see if this relationship differs for men and women (Figure 3.1).

1.2.2. Social Developmental Theory: Smoking Environment & High Risk Smoking

An alternative theory that may explain how people transition from normative experimental smoking into high risk smokers is the Social Development Model which posits that substance use results from exposure to pro-substance using influential others, norms and opportunities within their home environment, among other contexts (Catalano & Hawkins, 1996). Exposure to engagement in substance use behavior, pro-substance use attitudes and norms, and heightened involvement with substance use through substance use related opportunities, either enhances or reduces the likelihood that the adolescent will engage in using substance. This theoretical model of increased substance use has been empirically tested and supported for adolescent and young adult substance use behavior (Lonczak, Abbott, Hawkins, Kosterman, & Catalano, 2002; Hill, Hawkins, Catalano, Abbott, & Guo, 2005). Using the Social Development Model as a guiding theory of daily smoking initiation, Hill and colleagues (2005) found that the family smoking environment (behaviors, opportunities and norms) played a critical role in daily smoking initiation, with parent and sibling smoking behaviors in particular predicting the child's own daily smoking even after controlling for a host of confounding variables.

Other studies have also found that children and adolescents who live with parents and siblings who smoke have a higher likelihood of ever smoking themselves (Chassin, Presson, Rose, Sherman, & Prost, 2002; Kandel & Wu, 1995; Andrews, Hops, & Duncan, 1997; Foshee & Baumann, 1992; Rajan et al., 2003; Flay et al., 1994; Chassin et al., 1990), report smoking more cigarettes per day and for more years total (Chassin et al., 1990), are more likely to become daily smokers (Bricker et al., 2006), and have higher rates of nicotine dependence (Timberlake et al., 2007) or perceived nicotine addiction (Chassin, et al., 1990) compared to those smokers who did not have familial smoking environments. Using a prospective longitudinal study, Bricker et al. (2006) found that both parent and sibling smoking had a significant influence on children's ever smoking and transitioning to higher levels of smoking behavior (i.e., daily smoking). Having one parent who smoked increased the probability of the child trying smoking by 32%, which increased to 53% when both parents smoked. Having even a single parent who smoked predicted a 15% increase in the likelihood that a child would move from trying a cigarette to monthly smoking and 28% increased risk that a monthly smoker would escalate to daily smoking. The study found similar results for sibling influence, where having an older sibling who smoked was predictive of a 29% increase in the likelihood of ever smoking and 20% increased likelihood of transitioning from a monthly to daily smoker. The combination of having a smoking parent and sibling was more influential in predicting smoking transitions compared to their separate influences, indicating that the larger smoking environment was the strongest risk for smoking behavior in adolescence. Expanding the tobacco environment to include family norms and tobacco socialization as theorized by the Social Development Model, and found in Hill (2005), may suggest to better explain the development of smoking behavior in adolescence, yet few studies conceptualize familial smoking in this way (Avenevoli & Merikangas, 2003).

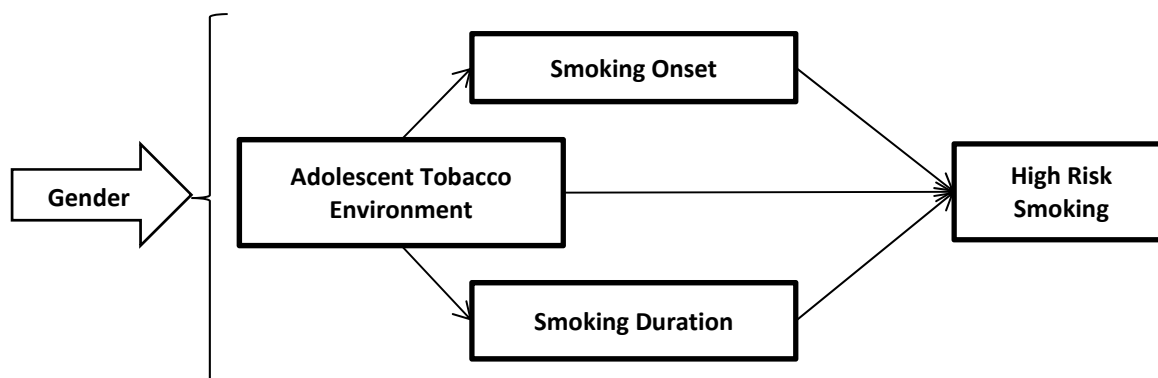


Figure 3.2. Proposed conceptual model of Social Development Model: Tobacco Environment & High Risk Smoking

The relationship between family tobacco environment and child smoking behavior may be gendered. Gender differences in the relationship between familial smoking and child smoking have been detected, where girls appear to be more susceptible to the social tobacco environment such as parental smoking behavior (Amos, 1996; Hill et al., 2005) and female sibling smoking (Avenevoli & Merikangas, 2003). Tobacco environment (peers and parental smoking norms and behaviors) have been identified as risk factors for smoking onset more consistently for women than men (Waldron et al., 1991; Flay et al., 1994). This gender difference has been attributed to girls being socialized to develop their sense of identity within the context of social environments more so than boys who are encouraged to develop a sense of self with more autonomy (Markus & Oyserman, 1989). However, the underlying mechanisms of intergenerational smoking transmission, and the extent to which they are moderated by child gender, are not well understood. In their concluding remarks, Avenevoli & Merikangas (2003) noted that gender may be an important moderator in this relationship, but few studies to date have investigated potential sex differences (Figure 3.2).

1.2.3 Stress-Coping Hypothesis: Child Maltreatment & High Risk Smoking

Another theoretical model that may help explain the progression to becoming a high risk smoker is the Stress-coping hypothesis in which a social stressor, such as an adverse life event, results in the escalation and adoption of high risk, non-normative substance use behavior in order to deal with the stress resulting from the experience of the social stressor (Wills, 1985). That is, the extreme use of substances functions as a maladaptive coping strategy to deal with the negative emotions and affect resulting from the stressful life event (Weaver & Etzel, 2003). Nicotine has in fact been found to reduce stress, suggesting a physiological effect, and is believed by the many to reduce stress, supporting a psychological perception of nicotine as a stress reducer (Baker, Brandon, & Chassin, 2004; Picciotto, Brunzell, & Caldarone, 2002; Warburton, Revell, & Walters, 1988). The linkage between stress and smoking is evidenced by the high number of smokers who report smoking to reduce their stress level (McEwan, West, & McRobbie, 2008).

One stressor that has been proposed as a specific mechanism in the development of smoking behavior is the experience of childhood maltreatment. Cross-sectional associations have been found supporting this relationship including a linkage between early childhood maltreatment and younger age of smoking onset (Anda et al., 1999; Jun, Rich-Edwards, Boynton-Jarrett, Austin, Frazier & Wright, 2008; Yoshihama, Horrocks, & Bybee, 2010) and tobacco use in adolescence (Moran, Vuchinich, & Hall, 2004). Longitudinal studies investigating this connection are rare (Kristman-Valente & Wells, 2013). The few longitudinal studies that investigate this connection have also found support for child maltreatment increasing the risk of smoking behavior including any tobacco use in adulthood (Mersky & Topitzes, 2010), higher smoking frequency in adolescence and adulthood (Kristman-Valente, Brown & Herrenkohl, 2013), daily smoking (Topitzes, Mersky, & Reynolds, 2010), and nicotine dependence (Nelson et al., 2002).

Gender differences in the stress-coping model may emerge for several reasons including the selection of differing coping mechanisms and differences in the stressors. Men and women select differing coping mechanisms to deal with stress, with women more likely to select internalizing behaviors and males more likely to exhibit externalizing behaviors (Diehl, Coyle, Labouvie-Viet, 1996) in response to stressful experiences. There are mixed findings about the use of smoking among men and women to reduce stress with some studies finding that men and women turn to smoking to reduce stress equally (Dugan, Lloyd, & Lucas, 1999) while other studies find women more likely to report that they smoked to relax or calm themselves compared to men (U.S. Department of Health and Human Services, 2001). While men and women may equally use smoking to cope with stressful experiences, the question remains whether the selection of smoking as a coping mechanism, in response to a stressor, is associated with progression to high risk smoking.

The type of stressor that precedes smoking uptake may impact gender differences in the progression from stress to high risk smoking. Men are more likely to report combat-related trauma while women are more likely to report interpersonal trauma such as childhood maltreatment and are also more likely to receive a trauma-related diagnosis including Post-traumatic Stress Disorder (PTSD) (Hien, Cohen, & Campbell, 2005; Kessler, Sonnega, Bromet, Hughes, & Nelson, 1995). Child abuse is itself gendered with women more likely to experience sexual abuse compared to men (Finkelhor, 1994; Finkelhor, Hotaling, Lewis, & Smith, 1990; Molnar, Buka, & Kessler, 2001). The experience of sexual abuse in childhood has been found to predict higher frequency of smoking in adolescence and adulthood for women but not men (Kristman-Valente et al., 2013). Child maltreatment has also been linked with earlier smoking onset for women (Jun, Rich-Edwards, Boynton-Jarrett, Austin, Frazier, & Wright, 2008) in an

all-female sample. However, few studies investigate the role of gender in the linkage between child maltreatment and smoking behavior and when they do findings are mixed (Topitzes et al., 2010; Strine et al., 2012; Kristman-Valente et al., 2013; Kristman-Valente & Wells, 2013). It may be that the chronic nature of interpersonal violence such as child maltreatment reinforces the need for coping behaviors, such as smoking, leading to exacerbated smoking behavior and that this differs by gender due to differential exposure to violence types. It may also be that men react differently to child maltreatment through the selection of different coping mechanisms. These later findings point to the potential role that gender may play in the link between early childhood maltreatment and becoming a high risk smoker.

Since delaying onset to age 18 is associated with reduced likelihood of ever smoking, reduced frequency of use, and a lower likelihood of becoming addicted to nicotine (U.S. Department of Health and Human Services, 2012), it will be important to better understand the specific mechanisms of early life stress, such as childhood maltreatment, that operate to influence high-risk smoking behavior across time and whether this differs for men and women (Figure 3.3).

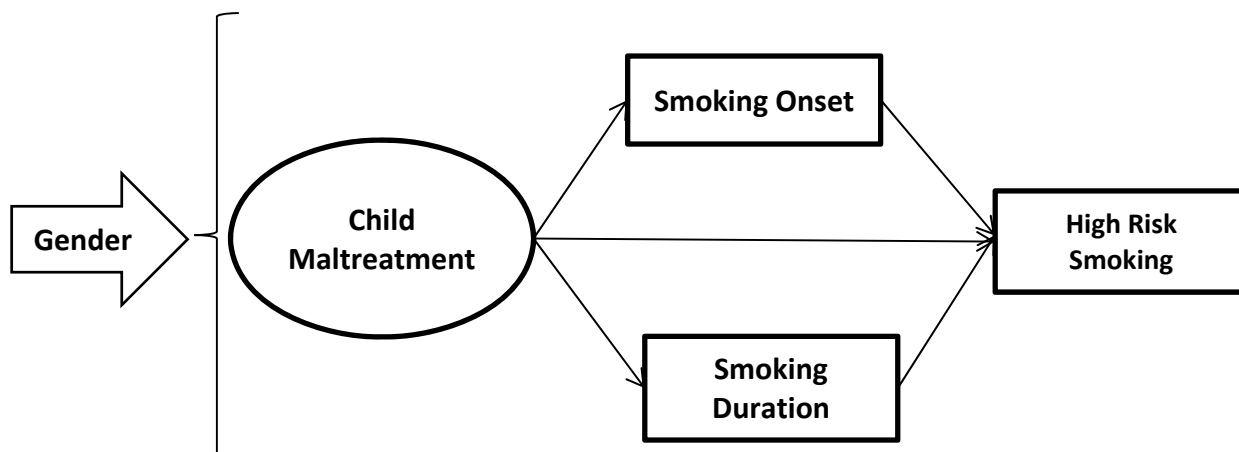


Figure 3.3. Proposed Conceptual Model of Stress-Coping Hypothesis: Child Maltreatment & High-Risk Smoking

1.2.4. Self-Medication Hypothesis: Attention Problems & High Risk Smoking

In addition to exposure to social stressors, the stress-coping hypothesis can operate through a biological, trait based, mechanism that reflects an underlying psycho-physiological issue whose symptoms are treated by the use of substances as proposed by the Self-Medication theory (Khantzian, 1985). Self-medication theory, posits that individuals with mental illness use substances at higher levels in an attempt to treat the underlying symptoms of their mental illness (Khantzian, 1985; Khantzian, 1997). Supporting this theory is the high prevalence of smoking observed among the mentally ill (Lasser et al., 2000; U.S. Department of Health and Human Services, 2014). Applied to smoking, it may be that among people with mental illness escalated smoking behavior, above and beyond normative experimentation, is used to remedy symptoms they experience due to their mental health.

Attention related problems and disorders such as Attention Deficit Hyperactivity (ADHD) are mental health issues that are believed to lead to smoking. Due to the onset of attention problems issues in childhood and the significance of the problem in adolescence (Milberger,

Biederman, Faraone, Chen, & Jones, 1997), a time of normative experimentation and onset of substance use (Galera, Fombonne, Chastang, & Bouvard, 2004), investigating attention problems as precursors to later smoking behavior is an important area of study and potential point of prevention. Further, due to the timing of onset as attention problems generally preceding smoking onset it has been recommended that more attention be placed on the ADHD-smoking link for potential prevention efforts (Hall et al., 2009). Nicotine does appear to impact the underlying physiological systems of attention. When exposed to nicotine, individuals with Attention Deficit Hyperactive Disorder (ADHD), demonstrate elevated dopamine responses, which are associated with attention (Volkow et al., 2007) and report smoking as more rewarding (Levin, McClernon, & Rezvani, 2006). Thus, the adoption of smoking behavior may be a primary substance that is selected to self-medicate with among individuals with attention problems.

People with the psychiatric disorder ADHD have significantly higher rates of smoking compared to the general population (Matthies et al., 2013; Lasser et al., 2000), report earlier onset and faster escalation to regular use and daily smoking, and a graded relationship between the ADHD symptoms and the use of nicotine has been found, suggesting that the more attention problems a person has, the more a person utilizes increasing amounts of nicotine (Kollins, McClernon, & Fuemmeler, 2005; Milberger et al 1997; Van Voorhees, Mitchell, McClernon, Beckham, & Kollins, 2012; Fuemmeler, Collins, & McClernon, 2007). However, the existing literature examining the relationship between ADHD and smoking behavior has several limitations: relying too heavily on high risk or clinical samples (Kollins et al., 2005), providing little insight into mechanisms linking the two (McClernon & Kollins, 2008), and often limit their investigations to utilizing earlier smoking behaviors such as onset as outcomes rather than fully

explore the distal impact of attention problems on later smoking trajectories (example: Kollins et al., 2005). Because of these limitations, there is uncertainty about the sequence and the extent of the relationship between attention problems and smoking.

It has been noted that the self-medication strategy is more likely to be used by women than men (Testa, 2004). Gender differences in the attention problem-smoking link, while not commonly investigated, have also been found. Van Voorhees and colleagues (2012), after reviewing available literature on sex differences in the ADHD-smoking link and synthesizing preliminary evidence from several of their own studies, have pointed to gender differences in the relationship. The Van Voorhees group suggest that, compared with men, women with ADHD are more at risk for developing smoking behaviors and negative smoking outcomes such as more perceived difficulty with abstinence, more cravings, and faster relapse rates. Increased risk of smoking behavior for women with attention problems have been found in studies such as Galera et al. (2005), who found that for girls but not boys, ADHD symptomology independently predicted daily smoking, and Wilens et al. (2008), who found that women with ADHD were more likely to have more severe nicotine dependence as assessed by the Fagerstrom test for nicotine dependence compared to men with ADHD (though the difference was marginal [$p=.09$]). Most likely this increased risk for smoking among women with ADHD is related to the unique interaction of ovarian hormones that have been linked with both the expression of ADHD and the exacerbation of smoking behavior through the dopamine reward system (Van Voorhees et al., 2012). More research is needed to explore the role of gender in the link between ADHD and smoking (McClernon & Kollins, 2008) in order to develop targeted gender sensitive intervention that can attend to the high comorbidity of ADHD and smoking in adolescence which could potentially disrupt later smoking among a high risk group of smokers.

The current study uses a community based, dual-gendered sample, test the applicability of the whether early childhood attention problems predict becoming a high risk smoker directly or if more proximal attention problems in adolescence predicts high risk smoking at age 24 and will consider the role of early onset and adolescent smoking duration as potential mediators of the link between attention problems in childhood and adolescence and the likelihood of becoming a high risk smoker in young adulthood. Lastly, the study will determine if the self-medication hypothesis is supported for both men and women, and if the specific mechanism of attention problems is a distinct risk factor for males and females (Figure 3.4).

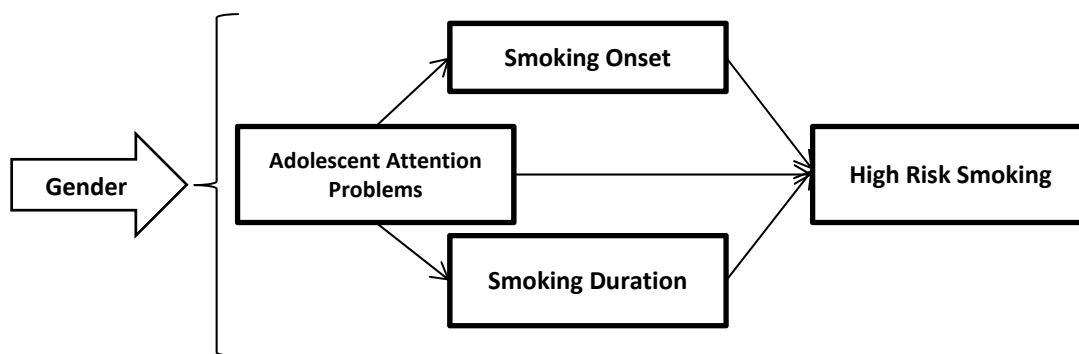


Figure 3.4. Proposed conceptual model of Self-Medication Hypothesis: Attention Problems & High Risk Smoking

1.2.5 Gateway/Reverse Gateway Hypothesis: Substance Use & High Risk Smoking

An alternative pathway to becoming a high risk smoker is the use of other substances. It has been found that escalation of smoking behavior (moving from regular use to dependence) is significantly impacted by the use of other substances. One theoretical model that may explain the relationship between other substance use and risky smoking behavior is the reverse-gateway hypothesis. The original gateway theory, or sequential stages of substance use, posits that lower risk substance use such as alcohol and tobacco use precede engagement with marijuana use,

which in turn facilitates engagement with more severe illicit substances (Kandel & Faust, 1975; Kandel et al., 1992). Adolescents who have not tried alcohol or cigarettes have very low risk of experimenting with marijuana (Kandel et al., 1992), but among those who do progress to using other substances, alcohol is most likely the first substance tried in childhood (Hawkins, Hill, Guo, & Battin-Pearson, 2002). Prior work with the current study sample has found that, in line with the gateway-hypothesis, early initiation of alcohol use increased the chance of tobacco initiation and marijuana use (Hawkins et al., 2002), which is consistent with findings in other samples (O'Loughlin et al., 2014). The reverse-gateway hypothesis, however, suggests that higher risk drugs like marijuana predict tobacco onset and escalation (Patton et al., 2005).

In the development of high-risk smoking behavior, the reverse-gateway hypothesis has in fact found support. Most studies target adolescent marijuana as the explanatory mechanism of high risk smoking, since other than tobacco and alcohol, marijuana remains the most frequently used substance in adolescence and often co-occurs with both substances (Johnston, O'Malley, Bachman, & Schulenberg, 2003; Johnston, O'Malley, Bachman, & Schulenberg, 2005; Agrawal, Madden, Bucholz, Heath, & Lynskey, 2008; Duhig, Cavallo, McKee, George, & Krishnan-Sarin, 2005). Marijuana use appears to be a significant risk factor for smoking onset and nicotine dependence (Patton et al., 2005; Timberlake et al., 2007). For example, Patton and colleagues (2005) found that people who had never used tobacco but frequently used marijuana were 8 times more likely to begin tobacco use compared to non-smokers with no marijuana use. The study also found in later time periods that marijuana use, above and beyond other smoking behavior, directly predicted transitioning to nicotine dependence, indicating that marijuana use may be a unique risk factor for both onset and escalation of smoking behavior over time. Similarly, Timberlake (2007) found that older adolescents, ages 17-21, who used marijuana were

significantly more likely to report becoming daily smokers by age 24. They also reported earlier initiation into daily smoking and a higher likelihood of being nicotine dependent in young adulthood; this association was not found for the younger adolescent group (12-16).

It is unknown whether the reverse-gateway hypothesis is applicable for both men and women, due to the dearth of literature using this model that examines potential moderation. Using the traditional gateway hypothesis, Kandel (1992) found that the use of either alcohol or tobacco preceded marijuana use and that this differed for men and women. For men the use of alcohol was more important in substance use stage advancement, while for women tobacco use played a more significant role, indicating that gender differences in a stage hypothesis of substance use are likely. Using the reverse-gateway model in an all-female twin study, Agrawal (2008) investigated the impact of marijuana use on smoking escalation. Findings demonstrated that lifetime marijuana use was related to transitioning from experimentation to regular use and from regular use to nicotine dependence, even after controlling for potential confounders including attention issues, child abuse and alcohol use. Further analyses revealed that more severe measures of marijuana use, like frequency of use and dependence symptoms, were also associated with smoking escalation. This study provides evidence for the reverse-gateway hypothesis for women but due to an all-female sample it is not known if this process works for men.

Agrawal (2008) concluded that their findings linking marijuana use to increased risk of smoking intensity reveal a significant public health problem. Given the recent policy changes regarding marijuana legality in the U.S. and the growing perception among adolescents that marijuana use does not have negative consequences, (National Institute on Drug Abuse, 2013) the potential connection with smoking escalation is particularly alarming. The current study

investigates the role of adolescent marijuana use frequency, controlling for earlier involvement with alcohol use as a child, on becoming a high risk smoker in young adulthood. The proposed analysis also tests for the indirect role of adolescent marijuana use, after including age of smoking onset and adolescent smoking duration in the model. Lastly, the current study, evaluates whether there are differences in the applicability of the reverse-gateway hypothesis for men and women, which is the first evaluation of its kind, and specifically, whether gender differences in the proposed mechanism of marijuana use in adolescence lead to high risk smoking in young adulthood (Figure 3.5).

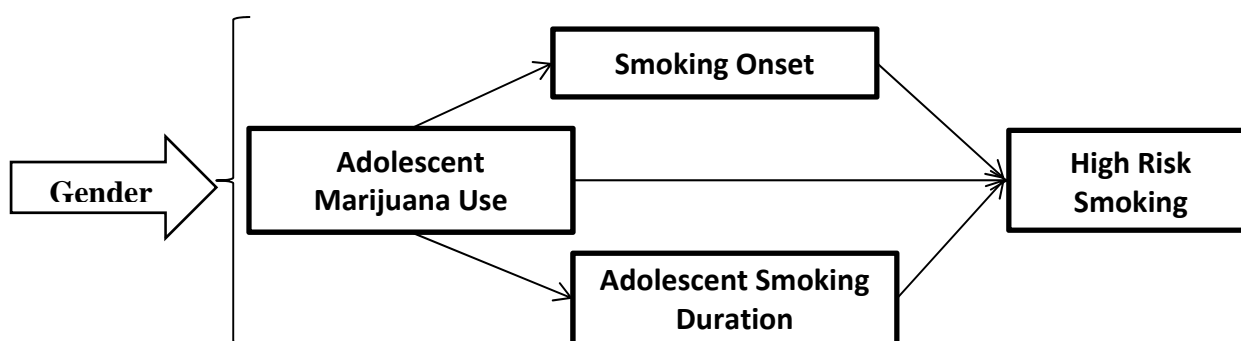


Figure 3.5. Proposed conceptual model of Reverse-Gateway Hypothesis: Marijuana Use & High Risk Smoking

1.2.6 Comprehensive Contextual Model

Based on the evidence reviewed above, it is possible, and highly likely, that no single model fully explains high risk smoking in young adulthood. A behavior as complex as smoking may be better captured using a multi-contextual lens. Cook (2003) recommended that studies investigating behaviors such as smoking use a multi-contextual approach to account for the contributions and potential confounds of multiple plausible predictors on smoking. The inclusion of multiple contexts allows researchers to account for the intra-context correlations of predictors, provides better comparisons of effect sizes, allows for the estimation of joint effects, and

uncovers potential mediating pathways between contexts. Despite the benefits of multi-contextual models, smoking research to date rarely accounts for or is unable to include other confounding factors or pathways that may work in tandem (Cook, 2003). Cook (2003) recommended that more complex models of social behaviors like smoking need to be developed using multiple theory driven pathways in order to more closely approximate the complexity of the human experience. In particular, becoming a high risk smoker is likely a complicated and multi-factorial process. In the current study a final model, incorporating all proposed pathways, will be examined to determine whether the relationship between the proposed mechanisms and becoming a high risk smoker changes when accounting for other possible causes, and whether this differs for men and women.

2. Methods

2.1. Sample

This secondary analysis was conducted using data collected between 1985-1999 from the Seattle Social Development Project (SSDP). SSDP is a longitudinal panel study of 808 participants recruited during 5th grade to participate in a multicomponent intervention study focused on protective and risk factors. Participants were recruited from 18 Seattle public elementary schools that served higher crime neighborhoods. Due to mandatory bussing at that time, students from other areas of the city were also enrolled. In total, 1,053 students were approached and 808 students consented to participate (76.7% consent rate). Assessments were conducted yearly beginning in 5th grade through 10th grade, again in the 12th grade, and every 3 years thereafter through age 33. Interviews were conducted using multi-modal survey formats, including face to face interviews, computer-assisted interviews, and self-administered questionnaires. All participants provided informed consent during adulthood (age 18 and over),

and youth assent and parental consent were obtained when the participant was a child. Youth interviews were conducted in English, some parent interviews were conducted in the parent's preferred language (Vietnamese, Spanish, Cantonese, Tagalog, etc.). Early in the study, youths received a small incentive (e.g., an audiocassette tape) for their participation and later they received monetary compensation. Respondents were tracked and interviewed wherever they moved. Retention has been high across all waves of the ongoing longitudinal study (92% at age 33).

The present analysis focuses on the possibility of a smoker becoming a high risk smoker by young adulthood (age 24). Age 24 was selected because earlier analyses on this sample has found that risky smoking behavior at age 24 strongly predicted future smoking behavior, indicating that when engaged in risky smoking in young adulthood participants were likely to continue along this pathway into adulthood (for additional description of the risky smoking continuity please see Chapter 2). Transitions in tobacco use generally progress as follows: trying a cigarette (initiation and experimentation), using regularly (continuation and persistence), and using heavily (dependence). An individual who has not engaged in even trying a cigarette does not have the opportunity to progress through the stages of smoking (Agrawal et al., 2008). For this reason we excluded those participants who had reported never trying a cigarette by age 24 (n=183), resulting in a final sample of 625 individuals who at some point had tried smoking prior to the age of 24. In the original cohort of 808 participants, 50% had tried cigarettes by the 12th grade which is higher than the estimated 38% prevalence of ever trying cigarettes by 12th grade found currently in national samples (U.S. Department of Health and Human Services, 2014) but was collected during a decade when smoking prevalence was higher overall. By age 24, 77% had reported trying a cigarette, which is comparable to the prevalence of “ever having smoked”

found in community studies (e.g., Patton et al., 2005). This exclusion process has been used in similar studies and sets a precedent for the exclusion of non-smokers (Agrawal et al., 2008; Timberlake et al., 2007). There were no gender differences in the rate of ever smoking by age 24 in SSDP.

2.2. Measures

2.2.1. High-Risk Smoking Outcome

High-Risk Smoking: There is often a high overlap between nicotine dependence and daily smoking. Daily smoking describes the frequency of cigarette consumption whereas nicotine dependence is defined by the impact of smoking on lifestyle and behavior, such as choosing to continue smoking despite health concerns. The current study conceptualized high risk smokers as those participants who reported daily smoking in the past month at age 24 or who met criteria for nicotine dependence. Daily smoking is based on self-reported smoking frequency in the prior month at age 24. Nicotine dependence is based on criteria from the Diagnostic Interview Schedule (DIS) (Robins, Cottler, Bucholz, & Compton, 1995) for smoking behavior in the past year. Participants were considered a high-risk smoker and coded a “1” if they met criteria for nicotine dependence or reported daily smoking; 0s represented those smokers who had ever tried smoking, were intermittent, or were light smokers.

2.2.2. Mediating Smoking Behaviors

Smoking Onset: The age that a person first tried smoking was a prospective measure of the age at which the participant first reported ever smoking. The age of the respondent was calculated based on participant birthday and date of assessment at each time period. Beginning in 5th grade, participants were asked if they had “Ever smoked cigarettes.” This question remained constant through age 24, the last time point used in the current analysis.

Adolescent smoking duration: Self-reports of smoking in the past month were dichotomized at each of the six assessment points in adolescence and added together to create a score, ranging from 0-6, reflecting the number of years that a participant reported current smoking in the past month between 6th and 12th grade.

2.2.3. Stress-Coping: Child Maltreatment & High Risk Smoking Measures

Child Maltreatment: The Childhood Trauma Questionnaire Short Form (CTQ-SF) was administered in the SSDP study at age 24 to retrospectively assess child and adolescent experiences of maltreatment (Bernstein et al., 2003). The CTQ-SF is a 28-item self-report, inventory of child maltreatment histories that assesses experiences of physical abuse, sexual abuse, emotional abuse, and neglect. Each domain is assessed with five-point Likert-type scale: “never true,” “rarely true,” “sometimes true,” “often true” to “very often true.” Further, the CTQ first assesses whether the event occurred prior to the age of 18. If respondents indicated they had had this experience, they were then asked if these event (s) occurred prior to the age of 10, creating developmentally distinct time frames of childhood and adolescence (ages <10 vs. 10-18 respectively). The present analysis only used responses reports of child maltreatment that occurred prior to the age of 10 in order to preserve time ordering. The CTQ-SF scales demonstrate good internal consistency across samples (Bernstein et al., 2003).

2.2.4. Self-Medication Hypothesis: Attention Problems & High Risk Smoking

Childhood Attention Problems: ADHD symptoms of attention problems were assessed using the Child Behavior Checklist (CBCL) Teacher form (Achenbach & Edlebrock, 1983) in grades 5-8. A scale, following DSM guidelines, was created following recommendations of Achenbach, Dumenci, and Rescorla (2001) and included both attention and hyperactivity problems. Examples of items within the scale include “Child can’t concentrate or pay attention for long”

and “Child has difficulty following direction.” Response options included 0 – Never true, 1- somewhat or sometimes true, and 2 – very true. In the fifth grade, the attention scale, with 13 items, had an alpha level of .93. *Adolescent Attention Problems*: A measure for grades 6-8 was constructed to capture adolescent attention problems using the same 13 items mentioned above. A mean attention score was calculated and standardized across grades 6, 7, and 8 in the present analysis for adolescent attention problems.

2.2.5. Reverse-Gateway Hypothesis: Adolescent Marijuana Use & High Risk Smoking Measures

Childhood Alcohol Use: Alcohol use at age 10 was included in the model based on a single dichotomous item of any alcohol use in the past month during 5th grade (0 – no, 1- yes) (participant report). *Adolescent Marijuana Use Duration*: Past-month reports of marijuana use frequency were dichotomized to reflect current versus no use at each year from grades 6-12. A sum score of the number of years in which a respondent reported marijuana use in the past month was calculated to reflect the duration of current marijuana use in adolescence

2.2.6. Social Learning: Tobacco Environment & High Risk Smoking Measures

Childhood Tobacco Environment: A composite measure of childhood tobacco environment in grade 5 was constructed using: *sibling smoking, parent smoking and family smoking norms*. To assess sibling smoking, participants were asked “If you have any brothers or sisters, do any of them smoke cigarettes?” Participants with no siblings were coded as missing. Parent smoking behavior was captured for both the study participatory parent and spouse/partner via parent report. In the grade-five survey, parents were asked how often they smoked and whether they smoked in front of their child. These questions were also asked regarding smoking behavior of the parent’s partner. Finally, family norms was based on parent response to two questions: “How

much do you think people risk harming themselves (physically or in other ways) if they smoke cigarettes occasionally?” and “How would you feel about your child using cigarettes before graduating from high school?” A composite score was calculated by first standardizing all individual items and calculating a subscale mean and second averaging across subscales of tobacco environment. *Adolescent Tobacco Environment*: The same items were measured for grades 6-10 for the *sibling smoking*, *parent smoking*, *family smoking norms* subscales. An additional question regarding *participant involvement in family smoking* based upon parent report of a single question, “How often does your child get or light cigarettes for a family member?” assessed in grades 6-8 was included in the calculation in the composite score of adolescent tobacco environment as explained above.

2.2.7. Confounding Factors

Other important explanations for high risk smoking need to be considered and controlled for in the model to more confidently attribute our findings to the proposed variables. Potential confounds include socioeconomic status during childhood defined as those participants who were eligible for free and reduced-price lunch in childhood when they were enrolled in grades 5, 6 or 7 (parent-report) and ethnicity (participant self-report), which was dummy coded into four categories: Caucasian, African American, Native American and Asian American. Gender was based upon participant self-report.

During the elementary grades, a portion of the sample participated in a multicomponent social development intervention promoting positive youth development. Intervention effects have been found in level differences in predictors and outcomes (e.g., Hawkins, Catalano, Kosterman, Abbott, & Hill, 1999; Hawkins, Kosterman, Catalano, Hill, & Abbott, 2008). To address possible threats to validity and confounding effects of the intervention within etiological

studies, extra caution has been used, including testing the etiological model for level differences based on the *intervention assignment* (e.g., Hill et al., 2005). No etiological study to date in SSDP has found evidence of differences in associations among variables across study conditions (e.g., Catalano, Kosterman, Hawkins, Newcomb, & Abbott, 1996; Huang, White, Kosterman, Catalano, & Hawkins, 2001, Bailey, Hill, Oesterle, & Hawkins, 2006, Hill et al., 2005). The current study conducted a preliminary multiple group analysis of the covariance matrix of study variables for the proposed models to determine if the relationships among variables differed by intervention assignment in order to detect intervention effects.

2.3. Human Subjects Protection

The proposed study is a secondary analysis of existing data. No new data were collected. All data used in the current analysis were collected under protocols approved by the University of Washington Human Subjects Review Committee, including provisions for active consent from parents and annual consent from children. Consent/assent forms had full approval and met all guidelines at the time of data collection.

2.4. Data Analysis Plan

2.4.1. Aim 1 – SEM & Path Analysis

To answer the first guiding aim—exploring five theoretical pathways to becoming a high risk smoker—analyses utilized the entire sample, consisting of both men and women, and included SEM and path analyses with covariates and the proposed mechanisms of the theoretical model for each of the proposed mechanisms separately. Model fit was assessed using the following criteria: the Comparative Fit Index (CFI) (Bentler, 1990; McDonald & Marsh, 1990), Tucker Lewis Index (TLI) (Tucker & Lewis, 1973), and the Root Mean Square Error of Approximation (RMSEA) (Steiger, 1990; Browne & Cudeck, 1993). Recommendations for cut-

offs on goodness of fit include the following: $RMSEA \leq .06$, $CFI \geq .95$, and the $TLI \geq .95$. The CFI, TLI, and RMSEA have been found to be appropriate for use with categorical variables (Muthen & Muthen, 2012). This model establishes the direct effect of the proposed theoretical mechanisms becoming a high risk smoker in young adulthood.

2.4.2. Mediating Role of Age of Onset and Adolescent Smoking Duration

To address the second aim regarding the potential mediating role of adolescent smoking behavior-- onset and duration-- adolescent smoking behaviors were added to each model to determine if their inclusion partially or fully mediated the proposed theoretical pathway. Mediation analyses utilized the following steps as outlined by MacKinnon (2008) and Baron and Kenny (1986). 1. Establish that the independent variable (proposed theoretical mechanisms) is significantly correlated with the dependent variable (high-risk smoking). 2. Show that the independent variable, is significantly correlated with the proposed mediators, in this case age of smoking onset and duration of smoking in adolescence. 3. Show that the mediators, onset, and duration, predict the outcome, i.e., high-risk smoking, while controlling for the independent variable. 4. Determine if the relationship between the independent variable and dependent variable is partially or fully mediated. A fully mediated model would result from the coefficient between the independent variable and the outcome equating to 0 after the inclusion of the proposed mediators in the model. A coefficient which decreases but remains significant is considered partially-mediated. A Sobel test was used to test if the indirect effect of the independent variable, through the mediating variables to the dependent variable was significant.

2.4.3. Potential Gender Effects

To address the third and final aim, investigating gender differences in the proposed pathways, Multiple Group Structural Equation Models (MGSEM) were used to compare the fit

of a fully unconstrained model where all parameters for both genders are freely estimated to a constrained model, where all parameter factor loadings and thresholds are constrained to be equal for women and men following recommendations of Dimitrov (2010). Relative fit between constrained and unconstrained models was assessed using the difference in chi-square. A significant difference in the chi-square value ($p < .05$) indicates that imposing equality between men and women significantly worsens the fit of the model and the model differs across gender. When a chi-square indicates that the model is different for men and women each parameter was assessed for gender differences one at a time to determine which pathway within the model differed. A specific path test, where the fully free model was compared against a model in which only the pathway of interest, was also used to test for gender differences.

All analyses were conducted using Mplus version 6.1 and used Full Information Maximum Likelihood (FIML) estimation to address missing data and to obtain optimally unbiased estimates of model parameters and their standard errors, as suggested by Schafer and Graham (2002). On highly sensitive questions, such as those asking about sexual abuse, the amount of missingness was approximately 11-12%, which was less than 20% of the total sample, decreasing the threat of substantial bias in estimates as described in Arbuckle (1996). In the full contextual model there were no cases that had no information across study variables resulting in a final sample size of $n=625$ used in the present analysis.

3. Results

3.1. Demographic information

By age 24, 625 (77%) of the original 808 SSDP study participants had ever tried smoking (40% of men vs. 37% of women). Of the 625 people at age 24 who had reported ever smoking at some point, approximately 216 (35%) were high-risk smokers at age 24. Of those people who were

high-risk smokers 163 participants (76%) reported daily smoking in the past month but did not meet nicotine dependence criteria (90 males vs. 73 females), 46 (21%) met criteria for nicotine dependence and were daily smokers (29 males vs. 17 females), and 7 (3%) participants met nicotine dependence diagnosis but were not daily smokers (5 males vs. 2 females). Men were more likely than women to be high risk smokers: 124 (males) vs. 92 (females) (χ^2 4.332 [1], $p < .05$). There were no differences in onset age by gender or in the duration of smoking years in adolescence by men and women (Table 3.1).

Table 3.1
Prevalence and mean differences between males and females among study variables.

	Total		Males	Females	χ^2 diff test
	<i>Range</i>	<i>N (%)</i>	<i>N (%)</i>	<i>N (%)</i>	
High-Risk Smoker	0-1	216 (35%)	124 (20%)	92 (15%)	4.34(1), p<.05
	<i>Range</i>	<i>M (SD)</i>	<i>M (SD)</i>	<i>M (SD)</i>	
Age of Smoking Onset	10-19	13.9 (2.5)	13.9 (2.5)	14.0 (2.4)	1.51(1), p>.05
Duration of Adolescent Smoking	0-6	1.2 (1.4)	1.2 (1.4)	1.1 (1.4)	.36 (1), p>.05
	<i>Range</i>	<i>N (%)</i>	<i>N (%)</i>	<i>N (%)</i>	
Age Onset <10	0-1	30 (5%)	12 (2%)	18 (3%)	1.37(1), p>.05
Age Onset 11-14	0-1	299 (48%)	157 (25%)	142 (23%)	1.23(1), p>.05
Age Onset 15-18	0-1	192 (31%)	92 (15%)	100 (16%)	.72(1), p>.05
Age Onset 19	0-1	19 (3%)	11 (2%)	8 (1%)	.45(1), p>.05
Table 3.1 Continued	Total		Males	Females	χ^2 diff test
	<i>Range</i>	<i>M (SD)</i>	<i>M (SD)</i>	<i>M (SD)</i>	
Childhood Tobacco Environment	-1.3 – 2.85	.05 (.79)	.06 (.80)	.04 (.79)	.053 (1), p>.05
Adolescent Tobacco Environment	-1.2 – 3.37	.07 (.70)	.04 (.64)	.10 (.76)	.512 (1), p>.05
	<i>Range</i>	<i>N (%)</i>	<i>N (%)</i>	<i>N (%)</i>	
Sexual Abuse	0-1	88 (14%)	23 (4%)	65 (10%)	24.658 (1), p<.000
Physical Abuse	0-1	111	66	45 (7%)	4.784 (1),

Emotional Abuse	0-1	(18%) 178 (29%)	(11%) 85 (14%)	93 (15%)	p<.05 .659 (1), p>.05
Neglect	0-1	64 (10%)	35 (6%)	29 (4%)	.542 (1), P>.05
# of Reported Maltreatment Types	<i>Range</i> 0-4	<i>M (SD)</i> .77 (1.0)	<i>M (SD)</i> .73 (.99)	<i>M (SD)</i> .80 (1.1)	.59 (1), p>.05
Childhood Attention Problems	-.70 – 2.64	.05 (.74)	.19 (.78)	-.10 (.65)	11.39 (1), p<.000
Adolescent Attention Problems	-1.19 – 3.37	.08 (.69)	.27 (.76)	-.12 (.53)	22.54 (1), p<.000
Alcohol Use at Age 10	<i>Range</i> 0-1	<i>N (%)</i> 152 (24%)	<i>N (%)</i> 87 (14%)	<i>N (%)</i> 65 (10%)	2.85 (1), p>.05
Duration of Adolescent Marijuana Use	<i>Range</i> 0-6	<i>M (SD)</i> .64 (.98)	<i>M (SD)</i> .66 (.95)	<i>M (SD)</i> .61 (1.0)	.376 (1), p>.05

Notes: Significant mean and proportion differences between males and females are in **bold**.
M(SD) = Mean and (St. Deviation); N (%) = Number of participants and (prevalence).

In the current sample there was a high rate of retrospectively reported childhood maltreatment (39.5%). Of those people reporting maltreatment, 17% reported experiencing two or more forms of child abuse (there were no gender differences in the mean number of reported CM experiences (.73 vs .80, males to females respectively). Sexual abuse was experienced by 14% of the sample, and females were significantly more likely to report this form of abuse as seen in other studies such as that of Whitfield, Anda, Dube, and Felitti (2003). Physical abuse, which was significantly more likely to be reported by male participants, was reported by 18% of the sample. The most commonly reported CM experience was emotional abuse at 29%, and the least reported was childhood neglect (10%). There were no gender differences in reporting either emotional abuse or neglect. Twenty-four percent of participants had reported using alcohol in the fifth grade. There was no significant difference in reported childhood alcohol use between men

and women. As expected, boys in grade five were more likely to have higher standardized mean scores of attention problems in childhood, which corresponds to distributed prevalence in the general population for that age (Van Voorhees et al., 2012). The majority of participants began smoking between 11-14 years of age (48%), followed by 15-18 (31%). Only 5% of the sample reported ever smoking at age 10, and 3% reported new onset at age 19. Duration of smoking analysis revealed that 32% of the sample reported smoking in the past month at least twice between grades 6-12. Descriptive statistics of the study variables are provided in Table 3.1.

Table 3.2
Bivariate Correlations of Study Variables for Combined Gender Sample and by Gender Separately

	1.	2.	3.	4.	5.	6.	7.	8.	9.	10.	11.	12.	13.	14.	15.
1. Sex	1														
2. Black	.03	1													
3. Native	.16	-.59** [-.46*] {-.57**}	1												
4. Asian	-.19**	-.77** [-.76] {-.68**}	-.52* [-.43*] {-.41†}	1											
5. SES	.06	.48 [.40] {.57}	.22* [.21] {.23†}	.22** [.34] {.05}	1										
6. Child Maltx	.08	.33 [.28**] {.37}	.12 [-.02] {.20}	-.14† [-.09] {-.17}	.32 [.32] {.31}	1									
7. Fam Tob Env - 5 th grade	-.02	.10† [.07] {.13}	.13* [.19†] {.07}	-.14** [-.23] {-.03}	.12* [.05] {.20**}	.02 [-.05] {.08}	1								
8. Attent. Prob. - 5 th grade	-.25	.33 [.38] {.31}	.07 [.10] {.10}	-.35 [-.40] {-.44}	.18 [.11] {.32}	.24 [.24**] {.26}	.13** [.18**] {.08}	1							

Table 3.2. Continued

	1.	2.	3.	4.	5.	6.	7.	8.	9.	10.	11.	12.	13.	14.	15.
9. Alcohol Use - 5 th grade	-.12 [†]	-.16* [-.42] {.11}	.06 [-.02] {.04}	-.22** [-.24*] {-.22 [†] }	.00 [-.17 [†]] {.23*}	-.03 [-.07] {.02}	.10 [†] [.07] {.12}	-.01 [-.07] {.03}	1						
10. Smoking Onset	.03	.00 [.06] {-.06}	-.16 [†] [-.40*] {-.05}	.18** [.20**] {.16}	-.04 [-.05] {-.02}	-.19** [-.13] {-.26**}	-.12** [-.19**] {-.05}	-.13** [-.14*] {-.11}	-.30 [-.29] {-.32}	1					
11. Smok. Duration- 6-12 th	-.02	-.13* [-.21*] {-.06}	.31 [.31] {.31}	-.26 [-.15] {-.49}	.16 [.11] {.22**}	.19 [.23**] {.15 [†] }	.08* [.07] {.10 [†] }	.21 [.25] {.16**}	.16** [.14 [†]] {.18*}	-.28 [-.22] {-.29}	1				
12. Adol. Attent Prob	-.34	.36 [.37] {.41}	.08 [.14] {.11}	-.41 [-.50] {-.41}	.20 [.19**] {.26}	.21 [.23**] {.26}	.11** [.15**] {.05}	.53 [.54] {.44}	.03 [-.00] {.02}	-.13** [-.14*] {-.13 [†] }	.26 [.26] {.28}	1			
13. Adol. Family Tob. Env	.05	-.01 [.06] {-.07}	.26 [.16] {.32}	-.36 [-.40] {-.30**}	.17 [.13 [†]] {.21**}	.11* [.11] {.11}	.41 [.43] {.40}	.20 [.32] {.11 [†] }	.03 [.05] {.03}	-.13** [-.18*] {-.08}	.35 [.36] {.35}	.17 [.22] {.17}	1		
14. Adol POT	-.03	.18 [.12 [†]] {.24**}	.21 [.17 [†]] {.24**}	-.36 [-.36] {-.41}	.11* [-.01] {.24**}	.22 [.27] {.18*}	.11** [.04] {.18}	.19 [.18] {.21}	.16** [.05] {.26}	-.16** [-.10] {-.22}	.51 [.43] {.59}	.29 [.32] {.29}	.22 [.17**] {.27}	1	
15. High Risk Smoker (24)	-.14*	-.07 [-.12] {.01}	.27** [.34*] {.24 [†] }	-.09 [-.04] {-.21 [†] }	.10 [.02] {.20*}	.23 [.28**] {.20*}	.03 [-.02] {.09}	.20 [.21**] {.15*}	-.17* [-.22*] {-.13}	.07 [.06] {.08}	.41 [.38] {.43}	.19 [.18**] {.15*}	.23 [.12 [†]] {.34}	.21 [.16*] {.25}	1

Notes: All standardized coefficients. Gender Combined Sample – [Male Coefficients] – {Female Coefficients}

p<.001; ** p<.01; * p<.05; † p<.1

3.2. Structural Equation model in the full sample

3.2.1. Intervention Effects

All study variables were correlated with each other in a covariance matrix (Table 1.2). A multiple group model analysis revealed that a constrained model of the covariance matrix of study variables, where all associations were forced to equality between those that received the intervention and those that did not, fit the data well ($\chi^2=236.075$ (195), $p=.024$, RMSEA: .029, CFI: .956, TLI:.931). When tested against a model that allowed the associations to estimate freely between intervention and control the chi-square test of difference was non-significant ($\chi^2=108.755$ (105) $p=.381$) indicating that allowing pathways to be freely estimated across treatment assignment did not significantly improve the fit and that the relationship among study variables did not significantly differ based on receipt of the intervention. As a result of these findings, all analyses were conducted using the full sample undifferentiated by intervention.

3.2.2. Child Maltreatment Measurement Model

Child maltreatment was conceptualized as a latent variable, including four dichotomous indicators of experiencing sexual, physical, emotional abuse and neglect. The data fit the model well χ^2 1.764 (2) $p=.4139$, RMSEA : .000; CFI:1.00 TLI: 1.00. Using the sample with both men and women combined, the loadings of each item were significant at ($p<.000$) and ranged from .49 (neglect) to .91 (physical abuse). Because it is important to establish that latent constructs work in the same way across differing groups (Harachi et al, 2006) a multiple group model was used to test for measurement invariance of the latent construct of child maltreatment across gender. The model of child maltreatment was first tested separately for men and women. For men, the data fit the model well χ^2 2.873 (2) $p=.2378$, RMSEA : .039; CFI: .993 TLI: .978, and for women the fit adequately χ^2 10.700 (3) $p=.014$, RMSEA : .09; CFI: .957 TLI: .914. For

both men and women, all indicators loaded significantly onto the latent construct at $p < .000$; for men the standardized loading ranged from .47 (neglect) to .89 (physical abuse), while for women loadings ranged from .51 (neglect) to .82 (physical abuse).

To formally test for gender differences in the latent construct of child maltreatment, a freely estimated multiple group model ($\chi^2 3.280 (5) p = .657$, RMSEA : .000; CFI: 1.00 TLI:1.00) was compared to a fully constrained model where all thresholds and factor loading were set to be equal for males and females ($\chi^2 34.143 (12) p = .0006$, RMSEA : .081; CFI: .926 TLI: .926). A chi-square test of difference ($\chi^2 26.454 (7) p = .004$) was significant, indicating that constraining all factor loadings to be equal for men and women significantly worsens the fit of the model and the latent construct of child maltreatment differed across gender. Thresholds and loadings of the model were systematically allowed to be freely estimated for males and females, one parameter at a time, to determine where the difference was in the model as suggested by Dimitrov (2010). The threshold for sexual abuse, or probability that the respondent received a 1 on that indicator, differed for males and females, which is congruent with the significantly higher prevalence in reported sexual abuse for women shown above. A partially invariant model, in which the threshold for sexual abuse was allowed to be estimated freely between men and women while all other factor loadings and thresholds remained constrained, resulted in a chi-square test of difference ($\chi^2 10.229 (11) p = .510$), demonstrating full configural, metric, and partial scalar invariance in the latent construct of child maltreatment between men and women (Table 3.3).

Table 3.3
Standardized Factor Loadings for Child Maltreatment Latent Factor

Model: Child Maltreatment Latent Construct			
Standardized Factor Loadings:	FS	Males	Females
Sexual Abuse	.55	.53	.65
Physical Abuse	.91	.89	1.0
Emotional Abuse	.73	.74	.69
Neglect	.49	.47	.51
Free Model	χ^2 3.280 (5) p=.6570		
	RMSEA: .000		
	CFI: 1.0		
	TLI: 1.0		
Constrained Model	χ^2 34.143 (12) p=.0006	χ^2 diff test	NON-INVARIANT
	RMSEA: .081	26.454 (7), p=.0004	
	CFI: .926		
	TLI: .926		
Partially Constrained	*Threshold for SA freed	χ^2 diff test	INVARIANT
	χ^2 10.229 (11) p=.51 RMSEA: .000	6.238 (6)	
	CFI: 1.0	p=.3970	
	TLI: 1.0		

Notes: FS: Sample with males and females combined.

3.3. Aim 1 – Direct Effects of Proposed Theoretical Mechanisms on High Risk Smoking

3.3.1. Developmental Psychopathology: High Risk Smoking Continuity

In a path model both age of smoking onset ($\beta=.22$, $p<.000$) and duration of adolescent smoking ($\beta=.48$, $p<.000$) significantly predicted becoming a high risk smoker. After controlling for potential confounds, duration of adolescent smoking ($\beta=.39$, $p<.000$) continued to

significantly predict becoming a high risk smoker by age 24 but age of onset did not significantly predict high risk smoking ($\beta=.09$, $p=.11$) after accounting for sex, SES, and ethnicity. The model with covariates explained 19.0% of the variance in becoming a high risk smoker by young adulthood (Figure 3.6. Model A).

3.3.2. Social Developmental Theory: Smoking Environment & High Risk Smoking

To test a social developmental hypothesis, we investigated the effect of living in a tobacco environment in adolescence on becoming a high risk smoker in young adulthood. Path analysis found that tobacco environment in adolescence positively predicted being a high risk smoker in young adulthood ($\beta=.23$, $p<.000$). After controlling for tobacco environment in childhood, SES, sex, and ethnicity, the beta coefficient of the path between adolescent tobacco environment and high risk smoking remained significant ($\beta.27$, $p<.000$). The model with covariates explains 10.7% of the variance of becoming a high risk smoker (Figure 3.6 Model B).

3.3.3. Stress-Coping Hypothesis: Child Maltreatment & High Risk Smoking

To test the stress-coping hypothesis, we investigated the impact of childhood maltreatment before the age of 10 on becoming a high risk smoker in young adulthood (age 24). In a path analysis childhood maltreatment significantly predicted becoming a high risk smoker ($\beta = .22$, $p<.01$). After controlling for demographic characteristics (SES, sex, ethnicity), this relationship remained significant ($\beta=.21$, $p<.01$). The child maltreatment stress-coping model fit the data adequately: $\chi^2 87.764 (31)$ $p<.000$, RMSEA : .054; CFI: .884 TLI: .791. The model explained 8.9% of the variance in high risk smoking (Figure 3.6 Model C).

3.3.4. Self-Medication Hypothesis: Attention Problems & High Risk Smoking

To test a self-medication hypothesis, this study investigated pathways from attention problems in adolescence to becoming a high risk smoker in young adulthood. Path analysis

revealed a significant, positive relationship between attention problems in adolescence, grades 6-8, and high risk smoking at age 24 ($\beta=.19$, $p<.000$). After controlling for demographic characteristics, sex, ethnicity, SES, and attention problems in 5th grade, the standardized coefficient from adolescent attention problems to high risk smoking became marginally significant at ($\beta=.12$, $p=.07$). The self-medication model of adolescent attention problems and high risk smoking explained 7.9% of the variance in high risk smoking at age 24 (Figure 3.6 Model D).

3.3.5. Gateway/Reverse Gateway Hypothesis: Substance Use & High Risk Smoking

To test a reverse-gateway hypothesis, we investigated the duration of adolescent marijuana use on the likelihood of becoming a high risk smoker in young adulthood. Path analysis demonstrated that the duration of adolescent marijuana use was positively related to becoming a high risk smoker ($\beta=.17$, $p<.000$). After controlling for alcohol use in 5th grade, sex, SES, and ethnicity, adolescent substance use was still significantly related to high risk smoking ($\beta=.17$, $p<.000$). This model with covariates explained 6.9% of the variance in becoming a high risk smoker in young adulthood (Figure 3.6 Model E).

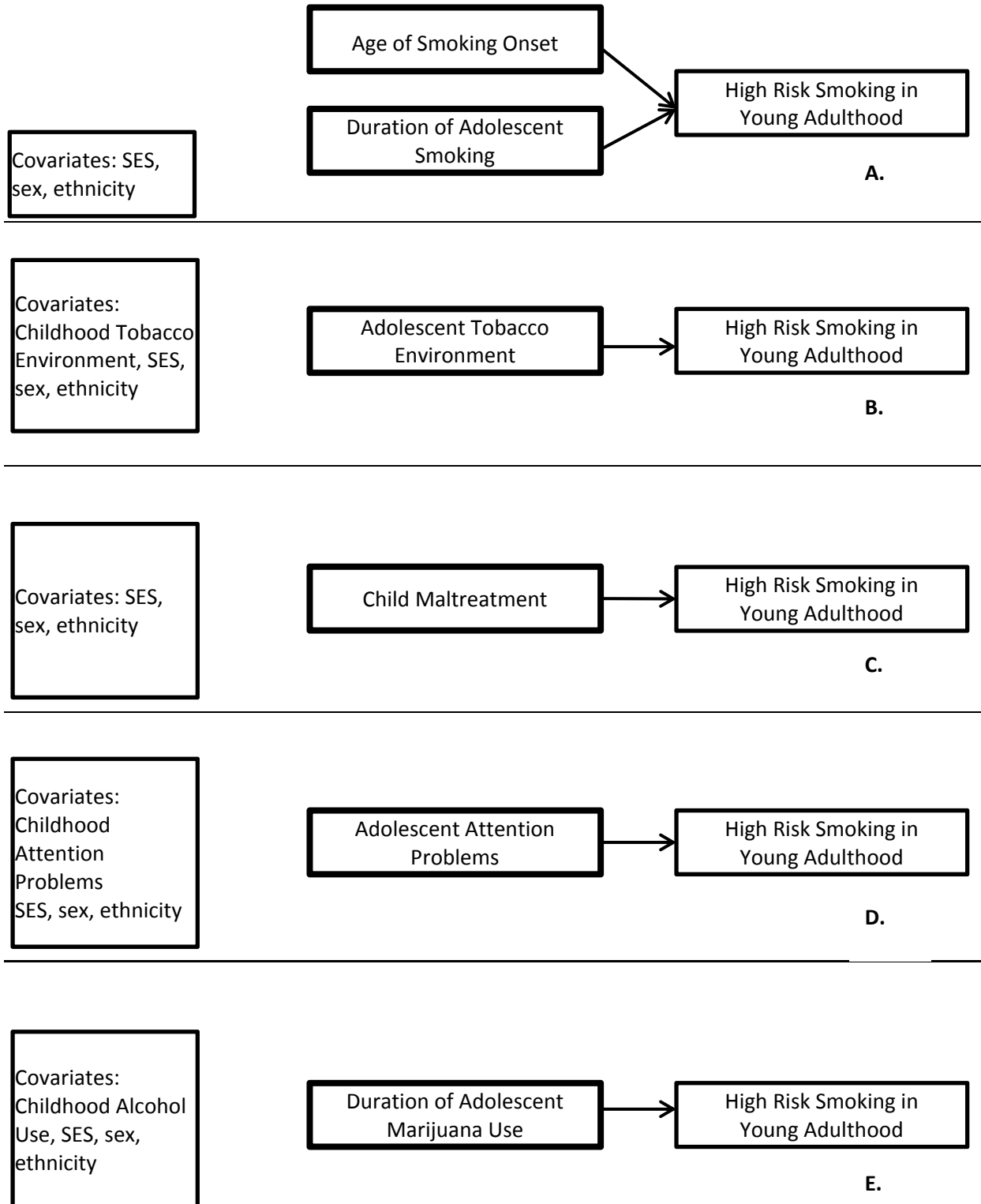


Figure 3.6. Pathways to High Risk Smoking in Young Adulthood.

A: Developmental Psychopathology – Smoking Continuity; B. Social Developmental Theory – Adolescent Tobacco Environment; C. Stress-Coping Theory – Child Maltreatment; D. D. Self-Medication Theory – Adolescent Attention Problems; E. Reverse Gateway Hypothesis – Adolescent Marijuana Use.

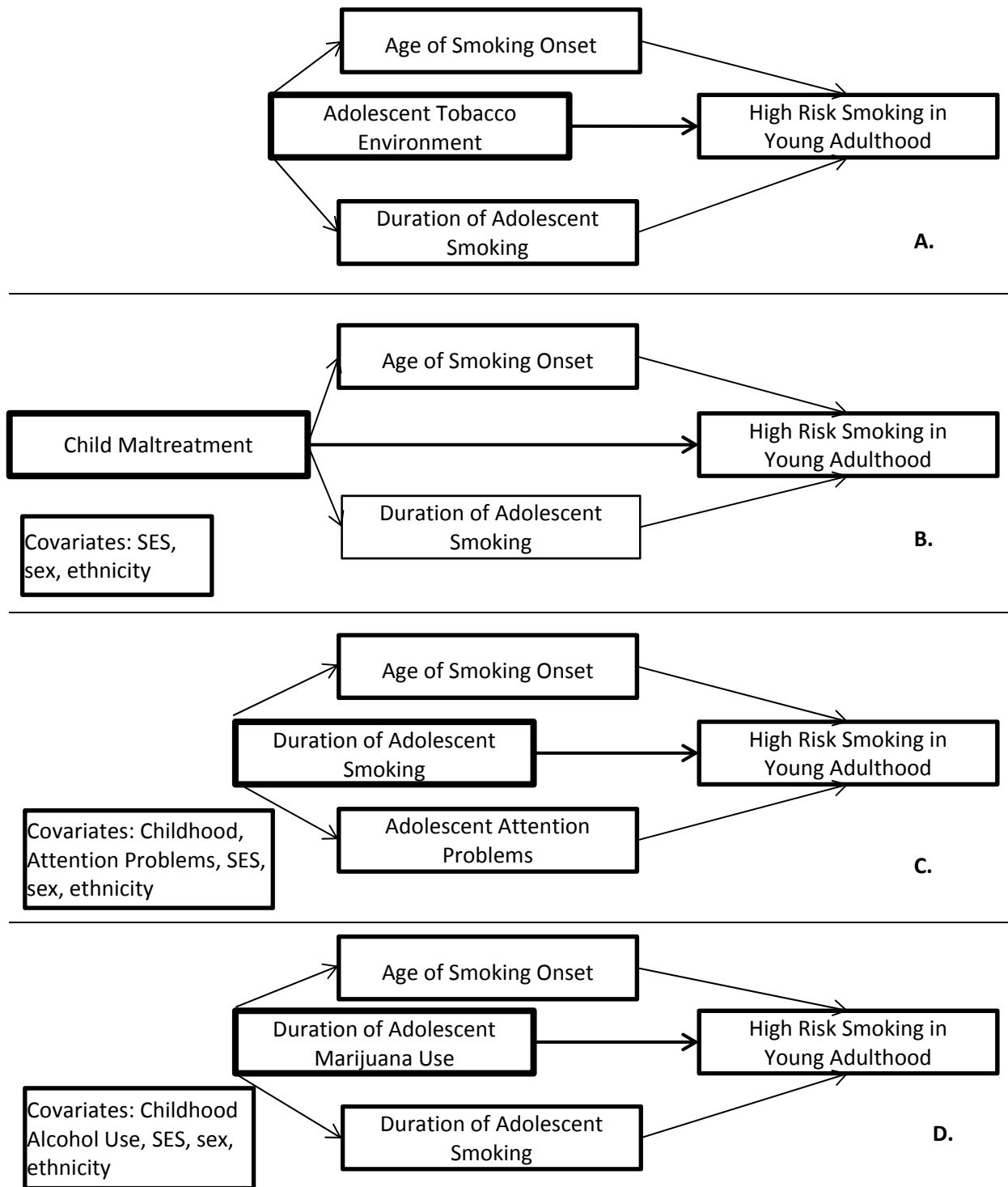


Figure 3.7. Mediation of Pathways to High Risk Smoking in Young Adulthood through Adolescent Onset and Smoking Duration.

A: Social Developmental Theory – Adolescent Tobacco Environment; B: Stress-Coping Theory – Child Maltreatment; C: Self-Medication Theory – Adolescent Attention Problems; D: Reverse Gateway Hypothesis – Adolescent Marijuana Use.

3.4. Indirect Effects of Proposed Mechanisms through Adolescence Smoking Behavior

Age of smoking onset and adolescent smoking duration significantly mediated, either fully or partially, pathways between the proposed mechanisms in each model and high risk smoking in young adulthood. Partially mediated pathways included the social developmental pathway from adolescent tobacco environment to high-risk smoking which was reduced from (.27 to .16, $p=.004$) (Figure 3.7 Model A) and the stress-coping pathway from child maltreatment to high risk smoking which was reduced from (.21 to .19, $p = .006$) (Figure 3.7 Model B). The self-medication pathway from adolescent attention problems to high-risk smoking (Figure 3.7 Model C) and the reverse-gateway pathway from adolescent marijuana use to high-risk smoking (Figure 3.7 Model D) both approached being fully mediated through age of smoking onset and adolescent smoking duration. All indirect effects were significant based upon Sobel tests. Across mediated models smoking duration was a stronger mediator compared to age of onset. The attributed indirect effect of proposed theoretical mechanisms through adolescent smoking behavior to high-risk smoking in young adulthood in the four proposed theoretical pathways ranged from 9.5% in the stress-coping model to 88.2% in the reverse-gateway model. Table 3.4 shows the influence of proposed smoking continuity mediators on the four alternative theoretical pathways to high risk smoking.

Table 3.4.
*Estimated Standardized Effects of Background and Mediating Factors
 on Being a High-Risk Smoker*

	Model	Social Dev. Model		Stress-Coping Model		Self-Med. Model		Reverse- Gateway Model	
Effect of Proposed Mechanism on High-Risk Smoking:	Adolescent Tobacco Env.	Adolescent Tobacco Env.		Child Maltreatment		Adolescent Attention Problems		Adolescent Marijuana Use	
	st. β	p	st. β	p	st. β	p	st. β	p	
Unadjusted β	.23	.000	.22	.004	.19	.000	.17	.000	
Adjusted β	.27	.000	.21	.004	.12	.066	.17	.000	
Inclusion of Mediators	.16	.004	.19	.006	.04	.508	.02	.789	
Sobel Test of Significance									
Onset	-2.1	.036	-2.3	.019	-2.1	.035	-2.7	.007	
Duration	5.9	.000	2.7	.008	5.4	.000	7.7	.000	
% of Direct Relationship Mediated									
by Age of Smoking Onset & Duration of Adolescent Smoking	40.7%		9.5%		66.7%		88.2%		

Notes: All coefficients presented are standardized.

3.5. Gender differences in proposed theoretical pathways

Gender differences in the etiology of high risk smoking across five theoretical pathways were tested. All models, containing covariates (e.g. SES, ethnicity, and early risk) and mediators, were run stratified by gender with the exception of the Developmental Psychopathology model which did not have proposed mediators. In the stratified models several gender differences appeared in pathways to high risk smoking. For women, but not men, adolescent tobacco environment significantly predicted high risk smoking in young adulthood (.25, $p < .001$ vs. .02, $p = .788$, women to men respectively). For men, but not women, adolescent attention problems moderately predicted high risk smoking (.14, $p = .089$). Child maltreatment was a significant predictor of high risk smoking for men (.23, $p = .023$) but was only moderately significant for women (.16, $p = .069$). For both men and women age of smoking onset and adolescent smoking duration were significant predictors of high risk smoking while marijuana use in adolescence did not predict high risk smoking in either the male or female reverse-gateway model. Variance

explained of high risk smoking behavior differed the most extensively in the social developmental model with adolescent tobacco environment explaining 31.0% for women and 18.4% for men. An example of stratified model comparison is depicted in Figure 3.8.

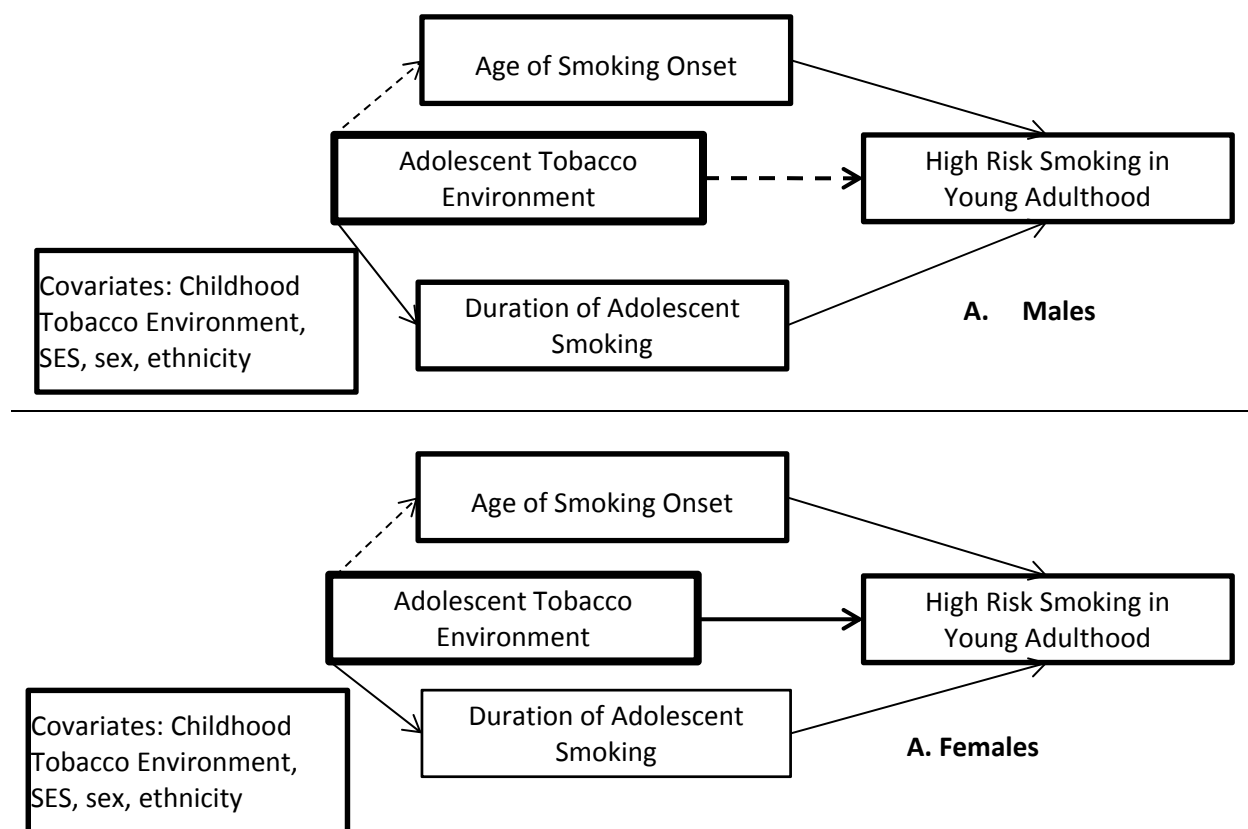


Figure 3.8. Gender Moderation of Social Developmental Pathway to High Risk Smoking in Young Adulthood through Adolescent Onset and Smoking Duration.

To formally test these differences a Multiple Group Analysis was performed for each model where a freely estimated model where parameters were allowed to vary across gender, was compared to a fully constrained model in which all factor loadings were forced to be equal for men and women. This MGA investigates gender differences in the overall model fit. Across all five proposed models there were no noted differences for men and women in the overall model, demonstrated by non-significant chi-square tests of difference, indicating that the proposed overall models fit the data equally well for men and women. To determine if the

pathway of interest in each of the proposed models was significantly different for men and women, a partially constrained model where only the pathway from the proposed model mechanism to high risk smoking was forced to be equal across gender. The partial constraint model was tested against the freely estimated model to determine if constraining the pathway resulted in a decrement of fit. All path-specific tests of difference, with the exception of the adolescent tobacco environment – high risk smoking pathway, were found to be invariant, or not different for men and women. The pathway from adolescent tobacco environment to high risk smoking was significantly different for men and women (4.001(1), $p=.036$) leading to the conclusion that for women, but not men adolescent tobacco environment significantly predicted high risk smoking in young adulthood. Full tests of gender effects across the five proposed models are reported in Table 3.5.

Table 3.5
Tests of Model Moderation and Path Specific Moderation

Model	Developmental Psychopathology		Social Developmental		Stress-Coping		Self-Medication		Reverse-Gateway	
Mechanism	Adolescent Smoking Onset&Duration		Adolescent Tobacco Environment		Childhood Maltreatment		Adolescent Attention Problems		Adolescent Marijuana Use	
Path Coefficient	St. β	p	St. β	p	St. β	p	St. β	p	St. β	p
M	O: .18	.009	.02	.756	.23	.023	.14	.089	.01	.923
	D: .41	.000								
F	O: .22	.006	.25	.001	.16	.069	.01	.884	.01	.934
	D: .47	.000								
% Variance Explained										
M	18.8%		18.4%		23.7%		21.1%		22.4%	
F	24.3%		31.0%		26.7%		22.7%		26.9%	
Free Model	Saturated		Saturated		χ^2 93.85 (57) p=.002 RMSEA: .045 CFI: .92 TLI: .86		Saturated		Saturated	
Constrained Model	χ^2 10.446 (15) p=.791		χ^2 24.07 (27) p=.627		χ^2 106.1 (74) p=.009 RMSEA: .037 CFI: .93 TLI: .91		χ^2 22.85 (26) p=.641		χ^2 23.83 (26) p=.584	

Table 3.5 Continued

Test of Difference for Overall Model	χ^2 10.727 (15) p=.773	χ^2 23.23 (25) p=.564	χ^2 14.24 (17) p=.650	χ^2 20.37 (24) p=.676	χ^2 21.52 (25) p=.660
Overall Model Invariance	Invariant	Invariant	Invariant	Invariant	Invariant
Path Specific Diff Test	χ^2 .140 (1) p=.409	χ^2 4.001 (1) p=.036	χ^2 .173 (1) p=.677	χ^2 1.674 (1) p=.196	χ^2 .000 (1) p=.997
Path Specific Invariance	Invariant	Non-Invariant	Invariant	Invariant	Invariant

3.6. Contextual Full Model

To evaluate the contribution of each hypothesized path on becoming a high risk smoker, in the context of other plausible explanations, a model with all four pathways (childhood maltreatment, adolescent attention problems, adolescent marijuana frequency and adolescent tobacco environment) was tested to determine which mechanisms persisted in explaining high-risk smoking. The final model, controlling for alcohol use in 5th grade, attention problems at age 10, tobacco environment in childhood, SES, sex, and ethnicity fit the data well (χ^2 78.276(66), p=.1433; RMSEA: .017; CFI: .974; TLI: .940). A chi-square test of difference found that the overall model fit equally as well for men and women (χ^2 38.674 (47) p=.801), but path-specific comparison found that the path from adolescent tobacco environment to high risk smoking was significantly different for men and women (χ^2 4.806 (1), p<.05). For males the path between adolescent tobacco environment was not significant (β =-.03, p=.785), but for women the path was significant (β =.191, p=.007). The full model explains 28% of the variance of becoming a high risk smoker (Figure 3.9).

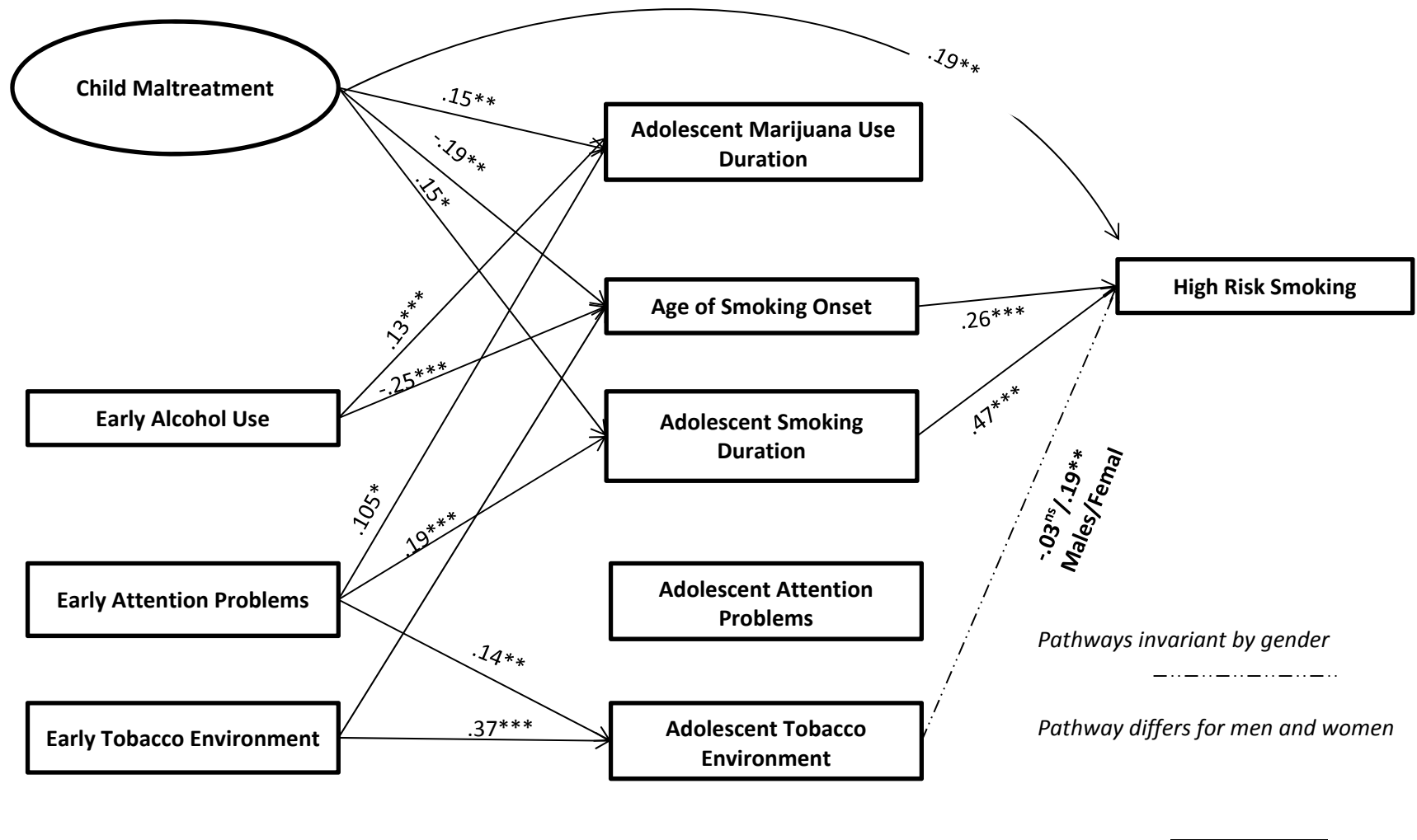


Figure 3.9. Contextual Full Model of Pathways to High Risk Smoking
 Covariates controlled for but not depicted in the model: SES and Ethnicity

4. Discussion

The current study investigated five potential theoretical pathways to becoming a high risk smoker among those people who had ever smoked by young adulthood. As expected the strongest explanation for being a high risk smoker in young adulthood was riskier smoking behavior in adolescence, defined in the current study as age of smoking onset and duration of years that adolescents smoked. The greater number of years that an adolescent reported smoking in the past month the more likely they were to become a high risk smoker in young adulthood. While smoking frequently, defined as either greater quantities of cigarettes or more cigarettes within a given time period, has consistently been linked to daily smoking and nicotine dependence this study points to the important role that adolescent smoking chronicity plays in becoming a high risk smoker. Delaying age of onset or decreasing the number of years of smoking in adolescence may effectively reduce the likelihood that a person goes on to high risk smoking behavior.

Interestingly the current study found that the later an adolescent began to smoke the more likely they were to become a high risk smoker. This is contrary to findings that link early onset to higher risk smoking behavior. The bivariate association of these two variables was $r=.07$, $p>.05$ and thus the significant relationship found in the multivariate model should be interpreted with caution. This finding may be due in part to our measure of age of onset which considered onset as the first time a person ever smoked. Onset of regular use or daily smoking earlier may lead to higher risk smoking as found in other studies. The current findings may also suggest that adolescents who wait to experiment with smoking begin to smoke in riskier situations, with more delinquent peers or have more access to smoking compared to younger ages. Prior research has found that people with diagnosis of PTSD report later smoking onset compared to those who did

not have a PTSD diagnosis (Breslau et al., 2004). It may be that participants in our model who had experienced traumatic events such as child maltreatment, and who went on to develop PTSD, onsetted smoking later and were more likely to become high risk smokers. PTSD as a mediating mechanism will be important to investigate in future studies. This finding points to the fact that smoking prevention efforts that are implemented in childhood may want to consider a “booster” session later in adolescence to reduce the likelihood of experimentation at older ages which may lead to higher risk smoking.

Beyond, smoking continuity, two other models, the social developmental model and stress-coping model, predicted higher risk smoking in young adulthood and were only partially mediated by adolescent smoking behavior. This study is the first to investigate the role that child maltreatment plays in high risk smoking among people who have engaged in the smoking process and are therefore at higher risk, compared to those who have never smoked, to continue on to more detrimental smoking patterns. The pathway from child maltreatment before the age of ten to high risk smoking at age twenty-four showed the least attenuation when adolescent smoking behaviors were entered into the model, pointing to the potential importance of prevention and intervention efforts with maltreated youth as a way to reduce high-risk smoking behavior in young adulthood. Given that the latest Surgeon General’s Report on Smoking prioritizes targeted intervention for people who continue to smoke despite policy and cessation efforts to date, the identification of child maltreatment as a risk factor for smoking behavior in this group is noteworthy.

The social developmental model, investigated via the mechanism of tobacco environment in adolescence, also predicted high risk smoking – but only for women. Women’s vulnerability to smoking behavior through social influences has been identified in previous studies (Amos, 1996;

Hill et al., 2005; Avenevoli & Merikangas, 2003) and replication of this finding in the current study supports that family tobacco environment is an important risk factor for women that should inform gender-responsive programming. While adolescent tobacco environment did not significantly predict being a high risk smoker among men, childhood tobacco environment predicted earlier age of onset among both men and women. Since earlier onset has been linked with continued smoking behaviors it will be important to continue targeting the family system as a point of prevention. These findings suggest that family interventions aimed at reducing smoking behavior that are implemented during childhood could be universally developed whereas targeted family interventions for girls should be developed in adolescence.

Pathways to becoming a high risk smoker that were less supported in this study were the reverse-gateway hypothesis and the self-medication theory. These pathways, through adolescent marijuana use duration and adolescent attention problems respectively, were significantly attenuated once adolescent smoking behavior was considered in the model. An indirect effect of adolescent attention problems through increased duration of adolescent smoking to high risk smoking in young adulthood was significant. McClernon and Kollins (2008) in their review of the literature noted that the mechanisms connecting ADHD and smoking behavior are poorly understood and it could be that studies finding a significant association between early smoking onset and ADHD have not included other potential smoking mechanisms, such as adolescent smoking duration, which could be the actual mechanism that early onset works through when predicting later smoking outcomes.

Adolescent marijuana use did not significant predict high-risk smoking in young adulthood in the reverse gateway hypothesis. It may be that that there were alternative drug sequences that the reverse gateway hypothesis operated through. Unfortunately, the current analyses was not

able to test alternate drug sequence due to smoking behavior being highly co-occurring with alcohol and marijuana and low prevalence of other drugs in the current sample which decreased the available variance needed to explore alternate pathways discretely.

Becoming a high risk smoker among those who have ever smoked is a complex process as evidenced by the integration of all pathways into the final contextual model. The integrated model supported individual model findings of mediation and moderation, improved the overall model fit, and increased the explained variance in becoming a high risk smoker to 28%. The moderate level of variance explained by the contextual model indicates that other mechanisms have yet to be explored and that more research is needed in order to identify other points of intervention and prevention of high risk smoking.

4.1. Limitations

An alternative to several of the proposed theories is that underlying genetic contributors to smoking behavior account for the escalation to high risk smoking. About 50% of tobacco use behavior can be attributed to genetic influence, whereas 25% can be attributed to social-environmental influences (Avenevoli & Merikangas, 2003). It has been suggested that genetics plays more of a role in nicotine dependent behaviors compared to initiating smoking. Future analyses looking at the genetic contributions to becoming a high risk smoker are planned, and other studies with the ability to investigate the role of genetics in addition to environmental influences on high risk smoking should also explore these potentially complementary pathways.

4.2. Implications for Practice

Given that little success has been documented in the majority of current adolescent smoking prevention efforts that attempt to stop smoking all together or delay the onset of smoking, (Peterson, Kealey, Mann, Marek, & Sarason, 2000), findings from the current study can provide

several avenues of guidance for clinicians, clinical practice and future research endeavors.

Interventions with children exposed to childhood maltreatment should consider incorporating smoking prevention curricula as a way to reduce engagement in smoking to begin with which ultimately may lower the public health burden of smoking in the U.S. Further, interventions that disrupt the intergenerational influence of smoking are an area of potential importance for young girls, who appear to be more susceptible to familial smoking influences. Cessation efforts with adults who have children, especially daughters, could incorporate a family component to help break the cycle of smoking.

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CONCLUSION

Smoking and interpersonal trauma are two social issues that individually have significant health and social repercussions (U.S. Department of Health and Human Services, 2014; Black et al., 2011). Findings from the current study suggest that these two issues are consistently related from childhood to adulthood for both men and women and remain significant even after controlling for socioeconomic status, race, alcohol abuse/dependence, major depression and other alternative risk pathways to smoking behavior such as adolescent attention problems, family tobacco environment and marijuana use in adolescence, suggesting that the relationship between smoking and interpersonal trauma is robust and unique across the life course. Results support prior work that has found within-time associations between smoking and trauma (El-Mohandes, El-Khorazaty, Kiely, & Gantz, 2011; Dichter, Cerulli, & Bossarte, 2011; Vest, Catlin, Chen, & Brownson, 2002; Crane et al., 2013; Anda et al., 1999) and extend these findings by identifying a reciprocal cross-developmental influence between the two issues. Directional findings point to interpersonal traumas (both child maltreatment and partner violence) influencing a wide array of smoking behaviors including onset, adolescent duration, smoking type in young adulthood and adulthood and high-risk smoking (daily or nicotine dependence). In addition, it was found that smoking behavior in young adulthood uniquely increased the risk for later interpersonal victimization in adulthood. It is important to consider that the shared relationship between these two significant social issues may worsen or complicate the consequences of both issues and because of this more research is warranted and further development of treatments that target co-occurring trauma and smoking are needed.

Integrated treatments that address substance use and comorbid trauma, and trauma-related diagnosis, have previously been identified as a preferred treatment method in the addiction field

(Torchalla, Nosen, Rostam, & Allen, 2012; Finkelstein et al., 2004) and calls for the development of new innovative integrated treatments has become a national priority (Finkelstein et al., 2004). While the past decade has provided the clinical field with several promising integrated treatments for trauma and addiction (Covington, 2008, Hien et al., 2004; Frisman et al., 2008) these interventions have rarely been used for smoking cessation or nicotine dependence (Torchalla et al., 2012; Moses et al., 2003) and to date only one treatment has focused specifically on the link between smoking and trauma (McFall et al., 2005; McFall et al., 2010). The integrated tobacco cessation intervention led to increased rates of smoking cessation among primarily male, military veterans with combat related PTSD diagnosis.

It is not yet known if the success of this intervention will translate to a female population (Fu et al., 2007). In general, smoking cessation strategies such as nicotine replacement therapy (NRT), which was a primary component of the McFall intervention, have not been as effective with women (Wetter et al., 1999; Cepeda-Benito, Reynoso, & Erath, 2004; Reynoso, Susabda, & Cepeda-Benito, 2005). Further, women are more likely to receive a PTSD diagnosis due to interpersonal trauma experience (Kessler et al., 1995; Herman, 1992) compared to combat related traumas. Research on treatment for women who have substance use and addiction suggest that it is essential, when working with women, that effective treatments consider incorporating gender-responsive components such as incorporating common themes in women's lives, such as interpersonal trauma, to targeting risk factors that are salient for women's substance use (Covington, 2008).

This dissertation is the first prospective, gender-moderated, examination of the relationship between multiple forms of interpersonal traumas and smoking behavior from childhood to adulthood. The current dissertation yielded several findings with relevance for the clinical

treatment of comorbid smoking and trauma and provides information of use to individuals designing future interventions for smoking and trauma that are universal and gender-responsive.

- *Smoking matters.* There is considerable evidence connecting alcohol and other drugs (AOD) to interpersonal trauma, and most integrated treatments focus on or are applied to the AOD-trauma link (Torchalla et al., 2012). The findings of this dissertation underscore that even after controlling for socioeconomic status, ethnicity, alcohol abuse and dependence, major depression, childhood alcohol use, adolescent marijuana use, attention problems and family smoking environment a person's smoking behavior remained significantly related to interpersonal trauma exposure and this was consistent across time and between genders. This finding is in stark contrast to the paucity of available integrated treatments for smoking-trauma (Finkelstein et al., 2004; Torchalla et al., 2012) and the low rate of smoking cessation programs in clinical treatment agencies whose clients have high rates of co-occurring trauma exposure and smoking prevalence (Baca & Yahne, 2009). In particular, the finding that smoking in young adulthood uniquely predicts partner violence victimization in adulthood suggests that agencies who use integrated treatments to address co-occurring AOD problems and trauma, but who fail to treat smoking or nicotine dependence, may be missing a critical point of intervention to disrupt exposure to future violence for both men and women. Theoretically existing integrated interventions for substance use and trauma may be translatable to smoking and nicotine addiction but this has not been tested (Finkelstein et al., 2004). It is also possible that integrated treatment that are effective with AOD will not be as effective with smoking since smoking is distinct from other substances in several ways

including the etiology and course of smoking, prevalence in co-occurrence, legality and general ambivalence of treatment centers to address this dependence. New integrated interventions targeting smoking-trauma are needed and further research is needed to determine if the available integrated treatments will extend to smoking related behavior in order to impact the enduring and intertwined relationship of these two issues.

- *Gender responsiveness starts here.* Interpersonal traumas are a common theme in the lives of women and have been identified in prior research as key experiences that hinder the treatment success of substance use treatment for women (Covington, 2008; Grella & Joshi, 1999). The robust and consistent finding that interpersonal traumas are related to smoking behavior at all points in the life course suggests that applying a gender-responsive lens to intervention development is a significant first step towards informing integrated smoking specific services. To date the primary focal group for research informing integrated smoking cessation and trauma treatments is veterans with combat related trauma (McFall et al., 2010) however, there is evidence that the types of stressors experienced may illicit different responses for men and women (Stroud, Salovey, & Epel, 2002) and that the association between interpersonal violence types (rape, sexual abuse) and smoking behavior (ever or current or nicotine dependence) is stronger compared to combat related stressors (Hapke et al., 2005). This suggests that people with interpersonal trauma exposure are particularly in need of integrated treatments for smoking and that women, who report higher rates of interpersonal trauma in their lifetime, may benefit by the development of integrated

interventions that account for interpersonal types of trauma – a common theme in their life.

In addition, the current study identified gender-specific risk factors for smoking that should be considered in the construction of new interventions. The identification of adolescent smoking environments as a significant predictor of high-risk smoking behavior in young adulthood for women, but not for men, is particularly noteworthy given that women have not responded as well to current treatments for smoking in general and that people who continue to smoke at high rates or who reach levels of clinical dependence on nicotine pose a large public health issue due to their resistance to available cessation efforts. The American Psychiatric Association and the National Institute of Health have both prioritized the development of targeted interventions for high-risk subgroups in the substance use treatment population (Hall & Prochaska, 2009). Researchers and clinicians who respond to that call should consider that for women including a family smoking environment component into cessation treatment may increase efficacy, reduce smoking behavior, and potentially lower a woman's risk for trauma exposure in the process.

- *Intervene Early.* When considering when to intervene the current study identified several points from childhood to adulthood that may be particularly impactful for disrupting the smoking-trauma link. First, Chapter 2 found a reciprocal relationship between smoking behavior and partner violence victimization in the transition from young adulthood to adulthood, ages 24 to 27, indicating that this developmental period is well positioned for integrated intervention focused on the trauma-smoking link. Second, when considering primary prevention in the early course of smoking

onset and escalation, the current study provided results suggesting that childhood maltreatment had a significant direct influence on smoking onset, adolescent smoking duration and becoming a high risk smoker in young adulthood, even after controlling for competing explanatory pathways. The strength of the relationship between child maltreatment and high risk smoking 14 years later was only slightly attenuated by considering these other factors suggesting a robust and meaningful impact of early interpersonal trauma on shaping an individual's smoking use trajectory. These findings point to early indicated treatment, such that children who have been exposed to maltreatment may benefit from integrated treatment for smoking-trauma which could potentially disrupt the smoking cycle among a group of youth at risk for the most risky forms of smoking behavior: daily smoking and nicotine dependence.

Information on the relationship between smoking and interpersonal forms of trauma across time and between genders is an important step towards informing the next wave of integrated interventions by pinpointing critical time periods and gender-responsive points for treatment. As new integrated treatments for substance use and trauma are being developed there is a need for novel interventions that address the smoking-trauma link and that incorporate gender-responsive components into future interventions in order to maximize effectiveness and applicability to both men and women (Covington, 2008). Collectively, findings from this study serve to fill the gap in our knowledge of the role of gender in the relationship between smoking behavior and interpersonal trauma from childhood to adulthood and how this differs or is similar for men and women and should inform intervention development and ultimately help decrease the public health burden of both smoking and interpersonal violence.

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Curriculum Vitae

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Curriculum Vitae

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EDUCATION

PhD	University of Washington	June 2014
MSW	University of Washington	June 2003
BS	California Polytechnic State University San Luis Obispo	December 2000

AWARDS, HONORS, GRANTS, & FELLOWSHIPS

University of Washington Press Release http://www.washington.edu/news/2013/08/05/abused-children-found-to-smoke-more-as-teens-and-adults/	08/2013
Drug Abuse Dissertation Research- Epidemiology, Prevention, Treatment, Services and/or Women and Sex/Gender Differences (R36): “ <i>Gender differences in the link between trauma and smoking in two generations</i> ”	Submitted: 05/2013
Certificate for Advanced Statistics in the Social Sciences – University of Washington Center for Statistics in the Social Sciences	Completed: 06/2013
General Exam: “The role of gender in the association between child maltreatment and substance use behavior: A systematic review of current longitudinal research 1995-2011”	Passed: 03/2012
Multidisciplinary Predoctoral Clinical Research Trainee National Institute of Health (NIH)/ National Center for Research Resources (NCRR) funded Institute of Translational Health Sciences (ITHS) (TL1TR000422)	09/2012-09/2013
National Institute of Mental Health (NIMH) Prevention Research Fellowship (T32MH20013)	09/2008-09/2009 & 09/2010- 09/2012
University of Washington Tobacco Scholars Career Development Award	10/2011-09/2012
University of Washington Boeing Fellowship for the Social Sciences	10/2009-06/2010
Substance Abuse and Mental Health Services Administration Science to Service Award	Awarded: 2008

RESEARCH INTERESTS & EXPERIENCE

Interests

Gender Differences in Substance Use Behavior
 Substance Use Etiology
 Co-Occurring Disorders
 Implementation of Evidence Based Interventions
 Advanced Quantitative Methods

Experience

Pre-Doctoral Research Analyst at the Social Development Research Group (SDRG) 09/2010-current

Examine etiological pathways of substance use among vulnerable youth participating in the Seattle Social Development Project. Study quantitative strategies used to analyze complex longitudinal data. Participate in interdisciplinary research team.

Pre-Doctoral Research Analyst for the Lehigh Longitudinal Study 07/2009-current

Examine pathways of substance use etiology among maltreated youth. Develop secondary analysis skills within a complex longitudinal dataset. Investigate gender differences in pathways to alcohol and drug use using statistical techniques such as Structural Equation Modeling.

NIMH Prevention Fellow with Elizabeth Wells, PhD - 09/2008-2012

Collaborate with the National Institute on Drug Abuse (NIDA) Clinical Trials Network Women and Trauma Project conducting secondary analysis investigating the role of interpersonal violence on interpersonal conflict among chemically dependent women.

PUBLICATIONS & PRESENTATIONS

Peer-Reviewed Publications

Kristman-Valente, A., Brown, E. C., & Herrenkohl, T. (2013). Child physical and sexual abuse and cigarette smoking behavior in adolescence and adulthood. *Journal of Adolescent Health, 53*, 4 533-538.

Kristman-Valente, A. N., & Wells, E. A. (2013). The role of gender in the association between child maltreatment and substance use behavior: A systematic review of longitudinal research from 1995 to 2011. *Substance Use and Misuse, 48*, 645-660.

Wells, E. A., **Kristman-Valente, A. N.,** Peavy, K. M., & Jackson, T. R. (2013). Social workers and delivery of evidence-based psychosocial treatments for substance use disorders. *Social Work in Public Health, 28*(3-4), 279-301. NIHMSID # 464086.

Hien, D. A., Wells, E. A., Huiping, J., Suarez-Morales, L., Campbell, A. N. C., Cohen, L. R., Miele, G. M., Killeen, T., Brigham, G. S., Zhang, Y., Hansen, C., Hodgkins, C., Hatch-Maillette, M., Brown, C., Kulaga, A., **Kristman-Valente, A.**, Chu, M., Sage, R., Robinson, J. A., Liu, D., & Nunes, E. V. (2009). Multisite randomized trial of behavioral interventions for women with co-occurring PTSD and substance use disorders. *Journal of Consulting and Clinical Psychology, 77*(4), 607-619.

Chambers, S., & **Kristman-Valente, A.** (2008). Integrating trauma care into substance use treatment, *Behavioral Health, 28*(5), 34-35.

Killeen, T., Hien, D., Campbell, A., Brown, C., Hansen, C., Jaing, H., **Kristman-Valente, A.**, Neuenfeldt, C., Rocz, N., Sampson, R., Suarez, L., Wells, E., & Nunes, E. (2008). Adverse events in an integrated trauma-focused intervention for women in community substance use treatment. *Journal of Substance Abuse Treatment, 35*(3), 304-311.

Manuscripts Under Review or In Process

Wells, E., Masters, T., Morrison, D.M., Beadnell, B., Hoppe, M.J., **Kristman-Valente, A.**, & Casey, E. "Alcohol is the eraser": A qualitative analysis of drinking's role in courtship and sex among young, heterosexually active women and men. Targeted Journal: *Journal of Contemporary Drug Problems* (Under Review).

Kristman-Valente, A., Hong, S., Herrenkohl, T. I. The mediating role of peer and individual's beliefs on binge drinking among maltreated youth. Target Journal: *Journal of Studies on Alcohol & Drugs*. (Analysis complete/Partially drafted).

Flaherty, B., & **Kristman-Valente, A.** Cigarette smoking profiles among a US national sample of African American tobacco users. Targeted Journal: *The Journal of Health Disparities Research & Practice* (Analysis Complete/In Process).

Selected Presentations

Starks, S. H., Goodley, T.B., & **Kristman-Valente, A.** (2013, October). *Is there still room for women in social work's global era?*, Partnership panel presentation of the NASW National Council on Women's Issues (NCOWI) in collaboration with the CSWE Council on the Role and Status of Women. Council on Social Work Education Annual Program Meeting, Dallas, TX.

Kristman-Valente, A. (2013, May). *Re-Conceptualizing Trauma Measurement: A Test of Structural Consistency and Continuity in Three Developmental periods*. Poster presentation, University of Washington Institute of Translational Health Sciences, Seattle, WA.

Kristman-Valente, A. (2013, May). *Child physical and sexual abuse and cigarette smoking behavior in adolescence and adulthood*. Poster presentation, Society for Prevention Research, San Francisco, CA.

- Kristman-Valente, A.** (2013, June). *The role of gender in the association between child maltreatment and substance use behavior: A systematic review of current longitudinal research 1995-2011*. Poster presentation, College on Problems of Drug Dependence, San Diego, CA.
- Kristman-Valente, A.,** Hien, D., Campbell, A., & Wells, E.A. (2012, January). *Behavioral Treatment for Women with Co-Occurring Posttraumatic Stress and Substance Use Disorders: The NIDA CTN Women & Trauma Study*. Panel, Society for Social Work Research, Washington, D.C.
- Herrenkohl, T.I., **Kristman-Valente, A.,** Hong, S., Prince, D., & Klika, J.B. (2011, January). *Adulthood Consequences of Child Maltreatment and Longitudinal Pathways to Resilience*. Panel, Society of Social Work Research, Tampa, FL.
- Kristman-Valente, A.** (2011, March). *Trauma Types, Timing and Interpersonal Functioning in Substance Using Women from a National Trial*. Panel presentation, 19th Annual Congress on Women's Health, Washington, D.C.
- *Hong, S., Herrenkohl, T.I., Klika, J.B., & Sousa, C. (2010, March). *Findings of Longitudinal Research on Child Maltreatment and Resilience*. Panel, Society for Research on Adolescence, Philadelphia, PA.
- Kristman-Valente, A.,** & Chambers, S. (2009, March). Methods for Disseminating Evidence-Based Treatments from the Frontlines of Community Treatment Programs. Special Presentation to the NIH Clinical Trials Network, Bethesda, MD.
- Kristman-Valente, A.** (2007, October). *The Chemically Dependent Older Adult: Treatment needs and research perspectives*. First Annual Providers Conference, Seattle, WA.
- Kristman-Valente, A.** (2007, August). Treating women with co-occurring trauma and chemical dependency: Integrating a trauma specific curriculum into a chemical dependency program. Merrill Scott Symposium, Seattle, WA.
* Original work presented by panel due to medical restriction prohibiting travel.
- Kristman-Valente, A.** (2007, October). *Treating the Chemically Dependent Older Adult*. Women's Coalition of Washington Annual Conference, Leavenworth, WA.
- Kristman-Valente, A.** (2005, March). *Research Collaboration: Treatment of Trauma and Substance Use in Women*. 7th Annual Fundamentals of Addiction Medicine, Seattle, WA.
- Kristman-Valente, A.** (2004, October). *Experience of Community Treatment programs in the CTN*. Substance Abuse Research Forum, Seattle, WA.
- Kristman-Valente, A.** (2003, June). *Intimate Partner Violence, Maternal Parenting Beliefs, and Child Outcomes*. NASW Conference – Washington State Chapter, Seattle, WA.

TEACHING INTERESTS & EXPERIENCE

Interests

Addiction and Mental Health
Statistics in Social Sciences

Research Methods
Women Specific Needs & Services

Curriculum Development

*“Women and Chemical Dependency:
An interdisciplinary perspective for the
social & behavioral sciences”*

This course discusses the epidemiology, etiology, treatment and outcomes of women with chemical dependency. The course will address how substance using women are a prevalent population in multiple social service areas such as the criminal justice, mental health field and child welfare services. This course will provide students interested in pursuing social service occupations with a gender specific look at the impact of addiction as well as treatment options.

Experience

“Foundations of Social Welfare Research” Invited Lecturer 12/06/2013

Required course in the University of Washington (UW)
2-year MSW program.

Talk: “Evidence Based Practice and Practice Based Research.”

“Interdisciplinary Research Methods” Invited Lecturer 11/07/2013

Required course in the University of Washington Bothell Interdisciplinary
Arts and Sciences program.

*Talk: “Understanding the relationship between concepts and variables:
Examples from research on gender, substance use and trauma.”*

“Social Welfare Research” Invited Lecturer 09/27/2013

Required course in the University of Washington BASW program.

Talk: “Introduction to data analysis and SPSS.”

“Understanding Addiction, Pharmacology of Drugs, and Treatment,” Co-Instructor	01/2012-03/2012
Clinical course in the University of Washington; 2-year MSW program.	
“Foundations of Social Work Research” Co-Instructor	01/2011-03/2011
Required course in the UW; 2-year MSW program	
“Social Work Practice with Chemically Dependent Adults: Understanding Assessment, Evaluation, and Counseling” Invited Lecturer	04/2012
Talk: “ <i>Women and Addiction: Understanding women’s unique experience with substance use.</i> ”	
“Statistics for Social Workers” Invited Lecturer	09/2010
University of Washington MSW Program	
Talk: “ <i>Application of chi-squares and t-tests within clinical practice.</i> ”	
“Social Welfare Research and Evaluation” Invited Lecturer	08/2009
Required research course in the University of Washington Advanced Standing Program	
Talk: “ <i>Using research skills within community clinical practice</i> ”	

ADDITIONAL PROFESSIONAL EXPERIENCE

Residence XII (Substance Use Treatment Program for Women)

Macro Practice – Research Coordinator Residence XII 09/2003-08/2008
Implemented a national trial (Women & Trauma Project) within a community agency. Conducted research protocol and managed research team. Led the adoption of evidence based practice and tested dissemination effectiveness. Program evaluation.

Direct Practice – Residence XII Clinical Responsibilities 09/2003-08/2008
Conducted co-occurring eating disorder groups. Facilitated psycho-educational groups on women’s health and sexuality. Attended and participated in clinical case consultations.

Seattle Children’s Hospital

Direct Practice – Social Worker 06/2002-06/2003
Conduct psycho-social assessments and evaluate needs and risk of client; perform necessary crisis intervention with families being treated within the Emergency Department and Diabetes Clinic.

Child Protective Services

Direct Practice – Social Worker

12/2001-06/2002

Provided case management, crisis intervention and group counseling for families involved in the child welfare system.

Atascadero State Hospital Conditional Release Program

06/1998-12/2000

Direct Practice – Clinician

Provided case management, crisis intervention and group counseling to conditionally released criminal offenders with co-occurring mental health issues from Atascadero State Hospital.

 PROFESSIONAL AFFILIATIONS & SERVICE

Academic Service

Social Work Research, Consulting Editor

01/2012 – Current

Journal of Interpersonal Violence, Consulting Editor

10/2012 – Current

Journal of Studies on Alcohol & Drugs, Consulting Editor

05/2013 – Current

Journal of Substance Abuse and Alcoholism, Consulting Editor

09/2013 - Current

Professional Service

Appointed Member of the NASW

11/2012 – 06/2015

National Committee of Women's Issues

Membership in Professional Associations

Society for Social Work Research

12/2008 – Current

National Association of Social Workers

01/2012 – Current

American Psychological Association

11/2011 – Current