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Effects of Psychological and Structural-Level Factors on Sexual Risk and STI Incidence in High-Risk Men Who Have Sex with Men

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UNIVERSITY OF MIAMI

EFFECTS OF PSYCHOLOGICAL AND STRUCTURAL-LEVEL FACTORS ON SEXUAL RISK AND STI INCIDENCE IN HIGH-RISK MEN WHO HAVE SEX WITH MEN

By

Calvin J. Fitch

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Men who have sex with men (MSM) with histories of childhood sexual abuse (CSA) are at disproportionate risk for sexually transmitted infections (STIs) and HIV. Psychological variables explain part of this; however, standing alone, they fail to account for all of the increased risk. This secondary data analysis investigates the relationship between psychological factors (PTSD and substance use), structural factors (government benefits, unstable housing and neighborhood crime), unprotected sex and STI incidence and also investigates possible moderation effects of race/ethnicity. MSM from Boston and Miami (N= 296) were recruited for a larger RCT testing the effect of Cognitive Processing Therapy (CPT) adapted for sexual risk on improving PTSD symptoms and decreasing unprotected sex. Participants completed psychosocial questionnaires and clinician-administered assessments and were followed for one year. A subset of 108 participants provided information on structural barriers. Cross-sectionally, higher PTSD symptoms, crystal meth use, unstable housing, and use of government benefits at baseline were associated with a higher number of unprotected sex encounters. Higher PTSD symptomology was also associated with lower odds of incident STI for Black (OR = 0.957, p = .007, 95%CI = 0.928 – 0.988) and Latino (OR = 0.965, p = .014, 95%CI =

0.937 – 0.993) MSM only. No other evidence of racial moderation was found. Longitudinally, cocaine use at baseline predicted decreased condom use and use of government benefits predicted increased condom use. Though underpowered in some structural variable tests, the study provides plausible intervention targets for a population at high risk for HIV infection whose risk reduction needs have been challenging to address. Future work should continue to address mechanisms for the effects found here, particularly those which are structural in nature. This work might also investigate other theoretically-guided variables and their relation to structural barriers and unprotected sex.

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Chapter 1: Introduction

Sexually transmitted infections (STIs) are a major public health concern in the US. Nearly 20 million new cases occur each year accounting for over $16 billion in health care costs. Data from the Centers for Disease Control and Prevention (CDC; CDC, 2015) indicate that, in 2014, incidence of chlamydia, syphilis (primary and secondary) and gonorrhea had increased from previous years. Compared to the rates in 2013, rates of chlamydia in 2014 rose by 2.8%, gonorrhea rose by 5.1%, and primary and secondary syphilis rose by 15.1%. In fact, the over 1.4 million cases of chlamydia reported in 2014 represent the highest number of cases for any condition ever reported to the CDC (CDC, 2015). In addition to disproportionately impacting young people (age 15-24), men who have sex with men (MSM) have also been disproportionately affected. MSM have and continue to be heavily and disproportionately affected by sexually transmitted infection (STI) incidence as rates have been on a steady increase for the past decade (CDC, 2015). The U.S. has seen a concerning rise in syphilis infections among this group. In 2014, men represented over 90% of all primary and secondary syphilis cases and MSM represented 83% of this subset of cases. Recent reports from the CDC suggest that not only do MSM account for 3 out of 4 syphilis infections, but they are more likely to be infected with drug-resistant strains of gonorrhea (Goldstein et al., 2012; Kirckaldy et al., 2013). Additionally, Black and Hispanic men are thought to be increasingly affected by these STIs relative to their non-ethnic minority counterparts (Millett et al., 2007). Although much of the work in sexual health, particularly in MSM has focused on HIV outcomes, STIs are an important outcome as well as presence of an STI can cause inflammation or open sores which may facilitate HIV transmission (Fleming &

Wasserheit, 1999). Indeed, half of MSM infected with syphilis also have HIV, on average (CDC, 2015). Consequently, it is no surprise that gay and bisexual men accounted for 67% all new HIV infections in 2014. The CDC estimates that, if current rates continue, 1 in 6 gay/bisexual men will be infected with HIV in their lifetime (CDC, 2016). Thus, STI prevention may represent a key pathway to HIV prevention. Because of this disproportionate rise, which has occurred for at least a decade now, research has tried to identify psychosocial risk behaviors and social/cultural factors pertinent to MSM that might contribute to the disparities in STI incidence we see among this group.

In the following literature review, we begin by providing justification for studying this vulnerable population by reviewing evidence substantiating the association of CSA with risky sexual behavior and higher HIV/STI risk relative to MSM without CSA. We continue by examining the association between CSA and psychological factors known to influence risky sexual behavior, namely PTSD symptoms and substance use. In recognition of the role of structural disadvantage in predicting risk above and beyond psychological factors, we will then examine evidence of the role of structural factors in sexual risk behaviors. Later, we will review evidence for ethnic disparities in CSA, psychological, and structural factors. Lastly, we will present Social Action Theory and Intersectionality Theory to provide justification for examining the role of psychological and contextual factors as well as racial moderation effects.

CSA, Sexual Behavior, and HIV/STI risk

In exploration of some of these unique factors that might influence STI risk disparities among MSM, research has uncovered that, compared to heterosexual men, MSM are much more likely to report a history of childhood sexual abuse (CSA;

Lenderking et al., 1997; Lloyd & Operario, 2012; Sweet & Welles, 2012)). Although definitions and conceptualizations of CSA have differed across studies, many have adopted the definition put forth by Finkelhor (1979) where sexual abuse is defined as "a sexual experience with a person at least 5 years older if the child was 12 or under, or 10 years older if the child is between 13 and 16 inclusive, with or without physical contact and whether or not sex was wanted by the child." Prevalence of CSA among MSM across studies through 2012 has ranged from 15% (Kalichman, Gore-Felton, Benotsch, Cage, & Rompa, 2004; O'Leary, Purcell, Remien, & Gomez, 2003) to 50% in a convenience sample of Latino MSM (Diaz, Morales, Bein, Dilán, & Rodriguez, 1999). Overall, across these studies the prevalence of CSA has been estimated at 27.3%. Among those studies using probabilistic sampling, the prevalence of CSA among MSM was 21.8% (Lloyd & Operario, 2012). Data from the National Epidemiologic Survey on Alcohol and Related Conditions suggests that, compared to their heterosexual counterparts, gay and bisexual men have 9.5 and 12.8 times the odds, respectively, of reporting CSA (Sweet & Welles, 2012). These data represent the most current estimates of CSA among a nationally representative sample using a heterosexual referent group. This analysis also showed that those who reported occasional or frequent abuse were at increased odds for HIV/STI incidence. Furthermore, this effect was moderated by sexual minority status where, among those that had experienced occasional or frequent abuse, sexual minority men had 4.2 times the odds of HIV/STI incidence and heterosexual men only had 1.5 times the odds relative to those who had not experienced abuse. A similar moderation pattern held for sexual minority women versus heterosexual women. This disparity mirrors the disproportionate prevalence of CSA that has been found in other convenience samples

(Lenderking et al., 1997; Mimiaga et al., 2009) as well as meta-analyses (Friedman et al., 2011; Lloyd & Operario, 2012). Taken together, these data indicate that sexual minorities are more likely to have experienced trauma precipitated by sexual abuse during childhood when compared to heterosexuals and this may have important implications for sexual health.

CSA has been shown to be a pervasive condition influencing many different syndemic behavioral risk factors for STI transmission. In a meta-analysis of 12 studies comparing MSM with CSA histories to those without, those with CSA histories were more likely to engage in recent condomless anal intercourse and have sex while under the influence of drugs or alcohol (Lloyd & Operario, 2012). Additionally, those with CSA histories are more likely to engage in transactional sex (i.e., sex in exchange for money or drugs; Haley, Roy, Leclerc, Boudreau, & Boivin, 2004; Kalichman et al., 2004). Furthermore, some striking evidence suggests that those with CSA histories do not benefit from traditional sexual risk reduction counseling (Mimiaga et al., 2009) and are more likely to be diagnosed with an STI (Brennan, Hellerstedt, Ross, & Welles, 2007).

CSA and Psychological Factors

However, CSA does not occur in a vacuum and can operate through a variety of conditions to influence risky sexual behavior and STI. One program of research has looked at psychosocial mechanisms. Interestingly, a couple of studies have used cotwin control designs and have shown that male and female co-twins exposed to CSA are at increased odds of developing psychopathology than co-twins not exposed to CSA (Dinwiddie et al., 2000; Kendler et al., 2000). One of the more established models of how CSA might influence psychosocial functioning is the Four-Factor Traumagenics

Model (Finkelhor & Browne, 1985). This model suggests that CSA alters a child's cognitive and emotional orientation to the world and distorts their self-concept, worldview, and affective processes. The model further argues that four traumagenic factors are responsible for this distortion – *traumatic sexualization, betrayal, powerlessness*, and *stigmatization*.

Traumatic sexualization refers to the process in which a child's sexual expression (including attitudes about sex) is shaped in a maladaptive fashion by the sexual abuse experience. For example, exchange of gifts or affection during a CSA experience may lead a child to learn to use sexual behavior as a way of obtaining these things in future interpersonal interactions.

Betrayal is the process by which a child comes to learn that someone they were dependent on or someone who was supposed to protect them caused them harm. A child may learn that they were tricked into a sexual experience through misrepresentation or coercion. On another note, a child may come to resent a person to whom they disclosed about the abuse who was subsequently unwilling or unable to protect them.

Powerlessness may come into play because of the power dynamics of the abuse experience wherein a child's personal space is repeatedly invaded and their self-efficacy related to protecting themselves is diminished slowly by failed attempts to stop the abuse. This might be exemplified in an abuse situation wherein authoritarian abusers use threat or coercion to make a child feel powerless.

Stigmatization encompasses the negative connotations that surround the abuse experience that are subsequently internalized by the child. For instance, some may

internalize the cognition that they are “damaged goods” or internalize self-blame for the experience. The four-factor traumagenics model informs possible post-traumatic cognitive changes that might then lead to negative effects on mental and sexual health.

Although most children experience a normal recovery following one or more CSA experiences, anywhere from one-third to one-half of victimized children will show clinically significant post-traumatic stress symptoms (Collin-Vézina, Daigneault, & Hébert, 2013). These pathologies are likely to extend into adulthood. Indeed, a large prospective cohort case-control study which followed sexually abused children for 43 years found that, compared to controls, those with a CSA history were 3.65 times more likely to have a record of mental health service utilization. Furthermore, these individuals were more likely to be diagnosed with psychotic, affective, anxiety, substance use, and personality disorders (Cutajar et al., 2010). Additionally, a review of 7 meta-analyses showed CSA to be associated with psychopathology in adulthood with effect sizes ranging from small to moderate (Hillberg, Hamilton-Giachritsis & Dixon, 2011). Data from a nationally representative sample of 13,274 U.S. men suggested that post-traumatic stress disorder (PTSD) might be an important condition to consider in examining how CSA might contribute to these risky profiles (Reisner, Falb & Mimiaga, 2011).

How might PTSD related to a CSA experience in childhood play out in a sexual situation? As noted in O’Cleirigh, Safren and Mayer (2012), closer examination of the three clusters of PTSD (i.e., intrusive and distressing thoughts related to the traumatic event, avoidance of stimuli related to the traumatic event, and hyperarousal) provides some logical explanations for how PTSD might foster risky sexual behavior. Intrusive thoughts are often centered around self-blame and self-loathing can lead to maladaptive

cognitions about power and control in sexual relationships. This might then lead to a reduced ability to negotiate condom use. To deal with the intrusive thoughts and accompanying negative emotion, individuals will often use avoidant coping strategies (e.g., substance use or dissociation). These avoidance strategies can impede ability to address sexual risk by hindering the ability to negotiate safer sex (e.g., negotiating condom use) or execute safety strategies (e.g., obtaining and using a condom). The hyperarousal symptoms may lead a person to be consumed with anxiety to the extent that it interferes with the ability to make accurate sexual risk appraisals. Thus, a person with these symptoms will have trouble distinguishing safe situations from unsafe ones. This may raise doubts in one's minds as to their ability to either detect risky situations or confront them in an adaptive manner.

As mentioned previously, a common theme among those with traumatic stress symptoms resulting from CSA is avoidance and substance use is a common way in which this is achieved. PTSD often results in negative cognitions that serve to maintain the disorder (Foa, Rothbaum, Riggs, & Murdock, 1991; Ehlers & Clark, 2000). These cognitions often present the world as entirely dangerous and/or the self as incompetent and defenseless from threat. These cognitions thus maintain a sense of current threat. These cognitions are met with maladaptive affective responses such as shame, guilt, or anger. The affective responses are then met with dysfunctional behaviors (e.g., substance use or behavioral disengagement), which serve to temporarily ameliorate emotional distress; however, these behaviors also inhibit the integration of new, less threatening, and potentially more accurate information. One line of thinking suggests that this substance use may serve to self-medicate and attenuate some of these intrusion and

hyperarousal symptoms (Khantzian, 1997). Additional research studies have supported this hypothesis (Allwood, Esposito-Smythers, Swenson, & Spirito, 2014; Jayawickreme, Yaskinki, Williams, & Foa, 2012). Another line of thinking suggests that, while this theory may be true, it fails to explain why all individuals with PTSD do not develop substance use disorders. More recent evidence has thus pointed to the effect of moderating role of stress/vulnerability factors such as being male, expectancies, and previous patterns of maladaptive coping to explain substance use patterns (Hruska & Delahanty, 2012).

Accordingly, mounting evidence suggests that those with CSA are more likely to have concomitant substance use disorders than those without CSA (Hughes, McCabe, Wilsnack, West, & Boyd, 2010; Simpson & Miller, 2002). Data from the National Comorbidity Survey suggest that both men and women with a history of CSA are at increased odds for alcohol and drug dependence (Molnar, Buka, & Kessler, 2001). Additionally, research suggests that sexual minorities are more likely to report substance use disorders than heterosexuals (McCabe, Hughes, Bostwick, West, & Boyd, 2009). In an analysis of data from a U.S. population-based survey, McCabe et al. (2009) found that gay and bisexual men had 2.9 and 4.2 times the odds, respectively, of meeting criteria for alcohol dependence based on the Diagnostic and Statistical Manual for Mental Disorders (4^{th} edition; DSM-IV) compared to heterosexuals. Likewise, gay and bisexual men had 4.2 and 6.3 times the odds, respectively, of meeting criteria for illicit drug dependence. These statistics suggest that both sexual minority identity and CSA are risk factors for development of substance use disorders.

Among sexual minority men, there seems to be a synergistic relationship between CSA and substance use, which may lead to worsened sexual health (Senn, Carey, & Vanable, 2010). Patients attending STD clinics tend to report high rates of substance abuse (Cook et al., 2006; Hutton, McCaul, Santora, & Erbelding, 2008; Senn et al., 2010) and sexual abuse (Petrak, Byrne & Baker, 2000; Senn et al., 2006). These individuals have been shown to be more likely to engage in condomless sex, sex for money or drugs, and to be diagnosed with a STD (Senn, Carey, Vanable, Coury-Doniger, & Urban, 2006). However, the studies assessing the effect of CSA and substance use on sexual health outcomes is scarce. It remains unknown what the additive synergistic effect of these two factors might be relative to either one of them alone.

Stall and colleagues (2001) conducted a study, which tested whether the quantity of these overlapping synergistic or syndemic factors influenced sexual health and found that the accumulation of these factors influenced HIV risk in a dose-response fashion. However, it was unclear which specific factors were at play as only the number of syndemic factors was measured. Mimiaga et al. (2015) expanded upon this work by testing different permutations of CSA and substance abuse risk factors (e.g., CSA and stimulant use vs. CSA only or Stimulant use only). Those with CSA and stimulant use were more likely to test positive for HIV over a period of four years when compared to those with either one of these risk factors alone. Additionally, the analyses showed a trend for the combination of CSA, stimulant use, and polydrug use. CSA appears to interact with substance use in a synergistic fashion to influence sexual risk and STI transmission outcomes.

Regardless of whether an individual has experienced CSA, substance use remains a risk factor for sexual behaviors that may put MSM at risk for STIs. Binge alcohol use ("excessive" alcohol use or 4 or more drinks on one occasion) and methamphetamine use have consistently been linked to riskier sexual behavior even at the event level (Vosburgh, Mansergh, Sullivan, & Purcell, 2012). In terms of alcohol, it seems to be that binge drinking is linked strongly to sexual risk behaviors relative to general alcohol consumption. Interestingly, emerging evidence even suggests that the recently popular combination of alcohol and energy drinks may be particularly dangerous as it promotes longer episodes of binge drinking and greater likelihood of engaging in condomless sex (Rehm, Shield, Joharchi, & Shuper, 2012; Snipes & Benotsch, 2013, Thombs et al., 2010). In a diverse sample of MSM, Mansergh et al. (2008) found that those who reported drinking or using drugs during their last sexual experience were more likely to report having tested positive for an STI in the past 12 months. A number have studies have linked heavy alcohol use with sexual risk behavior (Koblin et al., 2003; Mustanski, 2008; Woolf & Maisto, 2009; Vanable et al., 2004). In an event-level study of alcohol use and sexual behavior, Mustanski (2008) found that each drink raised the probability of having a sexual partner by 18% and each drink was associated with increased likelihood of engaging in more condomless sex. Notably, research suggests that it may not simply be the use of these substances that contributes to risk but more so the use of these substances in the context of sexual situations that contributes to STI risk (Mayer, 2011; Wilson, Cook, McGaskey, Rowe, & Dennis, 2008).

Use of illicit drugs such as methamphetamine, cocaine, gamma hydroxybutyrate (GHB), and ecstasy have reported to be more prevalent in MSM compared to the general

population and are likely to be used in sexual situations. Research suggests that use of these illicit substances may operate in a dose-response fashion in predicting sexual risk behaviors (Santos et al., 2013). Of the illicit drugs, the clearest evidence exists for methamphetamine. Methamphetamine use has been a consistent predictor of risky sexual activity among MSM (Vosburgh et al., 2012). Considering its effects (i.e., heightened senses and stamina, increased libido, and decreased inhibition) it is no wonder why this drug is often used in sexual situations. Evidence suggests that this drug may often be used to enhance sexual pleasure and dull negative feelings including pain associated with sex (Prestage, Grierson, Bradley, Hurley, & Hudson, 2009; Lee, Nakama, Goebert, & Alicata, 2015). Studies have demonstrated a clear link between these substances and condomless sex (Buchbinder et al., 2005; Colfax et al., 2004; Colfax et al., 2005; Stall & Purcell, 2000; Vosburgh et al., 2012).

CSA likely leads to profound and pervasive changes in a person's life that may then affect more proximal determinants of STI risk. Sexual minority men appear to be particularly vulnerable to these changes by way of their increased likelihood of experiencing CSA. PTSD in those who have experienced CSA can impair condom use self-efficacy and may lead to other distressing symptoms. Substance use is a common way in which these symptoms are temporarily alleviated. However, this momentary distress relief comes at a price- sexual safety. In order to truly understand how these psychological risk factors operate, we must consider the context in which they occur.

Structural Factors

While these psychological level factors have been shown to influence STI transmission risk, research shows that these factors alone do not account for all the

variance in risk. An emerging field of literature has focused on structural factors. Structural factors refer to economic, social, policy, or organizational aspects of the environment that serve as barriers to or facilitators of an individual's HIV prevention behaviors (Sumartojo, 2000). From this perspective, another program of research has focused on disadvantaged social contexts, the risk factors associated with those environments (e.g., poverty, unstable housing, and crime) and the degree to which these factors impact sexual risk behaviors. However, there is much work to be done in terms of assessing the impact of these factors particularly for high-risk MSM.

One commonly examined structural risk factor is low income. Lower income and socioeconomic status (SES) has been shown to be a risk factor for risky sexual behavior and STI acquisition at both the individual and community levels (Hogben & Leichliter, 2008). At the individual level, a few studies have asked about personal or household income level and have found negative associations between income and sexual risk/STI-related outcomes (Datta et al., 2007; Harling, Subramanian, Barnighausen, & Kawachi, 2013; Newbern, Miller, Schoenbach, Kaufman, 2004; Sionean et al., 2001). For example, analysis of National Health and Nutrition Examination Survey (NHANES) data suggested that individuals living below the poverty line were at increased risk of having an STI (Datta et al., 2007). At the community-level, ecological studies have typically assessed poverty within census tracts and have shown that areas with high rates of poverty and socioeconomic disadvantage have been associated with higher rates of STI and HIV infection (Dolan & Delcher, 2008; Springer, Samuel, & Bolan, 2010). Much of this data has been used to explore reasons for the HIV/STI disparity among races (i.e. Blacks vs. Whites) given the high prevalence of these sexual risk outcomes among

Blacks. However, virtually none of this research to our knowledge has been done exclusively with MSM (a group that we know to be heavily and disproportionately affected by STIs and HIV) with socioeconomic status as a primary focus of the analysis. This is problematic as it puts limitations on our understanding of socioeconomic disadvantage, especially for Black MSM.

Socioeconomic disadvantage or low income may lead to sexual risk and STI transmission in a couple of ways. Lack of access or use of healthcare may be one possible way. This could be a dangerous route because it could lead to delayed testing behaviors and untreated mental health concerns which may in turn affect sexual risk behaviors. Network characteristics of those living in poverty may also be at play. Those who are living in poverty where STI and HIV rates are disproportionately high often have sexual networks containing members who are also living in poverty. This may keep STI and HIV epidemics concentrated within these impoverished locales. Lower rates of education within these communities may also play a role. Additionally, there may be higher rates of risky sexual activity for the purposes of survival (e.g., survival sex/transactional sex).

Along the same lines as poverty, extant literature suggests that unstable housing is also connected to risk behaviors as unstably-housed individuals are three to nine times more likely to be HIV-infected than those who are stably housed (Kidder et al., 2007). This higher degree of risk has been associated with a range of risk behaviors which are more commonly seen among homeless or unstably housed individuals such as condomless sex with casual partners, sex trading, multiple sex partners, and substance use (Aidala, Cross, Stall, Harre, & Sumartojo, 2005; O'Toole et al., 2004). For example, homeless and unstably housed individuals in one cohort were found to have two to three

times the odds of reporting sex trading within the past six months and three to four times more likely to report hard drug use in the past six months (Aidala et al., 2005). Furthermore, those individuals who changed their housing status to more stable housing reduced these outcomes by 50 percent and were also less likely to engage in condomless sex. This study also provides evidence that homelessness and unstable housing may affect risk behaviors in a dose-response fashion where homelessness is associated with a more severe risk profile than unstable housing. Homeless and unstably housed individuals are also less likely to reduce their risk (Elifson, Sterk & Theall, 2007) and less likely to benefit from sexual risk reduction programs. Perhaps more relevant to the sample in the current proposal, evidence suggests that homelessness and unstable housing might be more common among younger sexual minorities (Clatts, Goldsamt, Yi, & Gwadz, 2005; Corliss, Goodenow, Nichols, & Austin, 2011) – a group disproportionately affected by STI and HIV incidence. History of sexual abuse has been identified as a risk factor for unstable housing however; no studies have examined the impacts of unstable housing in a cohort of MSM with histories of CSA. Together, these studies suggest that unstable housing might be an important structural barrier to consider when looking at sexual risk behavior.

Many theories have been offered as to why unstable housing might lead to increased sexual risk behaviors. Some have suggested that it may be a more potent stressor where the need to address the most basic subsistence concerns supersedes that of altering risky behaviors (Wong & Piliavin, 2001; Mizuno, Purcell, Borkowski, Knight, & SUDIS Team, 2003). Thus, exchanging sex for money or food may limit the degree to which a person is able to negotiate condom use during sexual encounters. Others have

pointed to the fact that being homeless or unstably housed often comes with neighborhood characteristics that may influence risk. These researchers have noted that homeless and unstably housed individuals are often placed in locations that come with additional structural barriers such as low economic/employment resources, crime/violence, and limited access to services which could provide risk reduction resources (Nwakeze, Magura, Rosenblum, & Joseph, 2003; Saegert & Evans, 2003). The stress of some of these things might lead some to use substances to self-medicate (Khantzian, 1997). Dependence on these substances could then affect likelihood of trading sex in to obtain them (Newman, Rhodes, & Weiss, 2004). Additionally, given the low resources and barriers to healthcare access, for example, the individuals within a person's sexual network might be more likely to have undiagnosed HIV/STIs. Each of these theories provides a plausible pathway by which unstable housing might influence sexual risk and STI transmission.

Among structural factors, the effect of neighborhood-level characteristics has received an increasing amount of attention. One neighborhood characteristic that has been under studied is neighborhood crime. Research does suggest that exposure to violence in one’s immediate environment is associated with sexual risk (e.g., physical abuse or intimate partner violence). The few studies that have examined the relationship between exposure to violence in the community and sexual risk behaviors have found that, indeed, exposure to this violence is associated with increased sexual risk behaviors including inconsistency in condom use (Senn, Walsh, & Carey, 2016). For example, in a recent study conducted by Senn et al. (2016), community violence was associated with more sexual partners and more episodes of condomless sex controlling for SES. This

suggests that the effects of violence exposure within a community affect sexual risk above and beyond some of the socioeconomic correlates of living in a violent neighborhood. Additionally, these researchers found that this association was mediated by both substance use and mental health – two variables that, in and of them, affect sexual risk as discussed earlier. However, the literature linking neighborhood crime to sexual risk and STI is not without its methodological limitations. One such limitation is that much of this research has been conducted in adolescents and in females. This is problematic as the relationship between exposure to community violence and sexual risk behaviors can different by gender (Voisin, 2007). Additionally, virtually none of this research has been conducted in MSM and if neighborhood crime truly influences STI infection, it may be particularly potent among a group with previous exposure to trauma (i.e. MSM with CSA). Research on neighborhood crime and sexual risk shows preliminary links; however, future work should include adult men, particularly from already vulnerable populations.

Neighborhood crime could potentially influence sexual risk behaviors in a few different ways. As reported by Senn et al., (2016), one possible way that this may occur is by way of mental health pathways. Exposure to crime may influence psychological distress that may manifest as depressive, anxious or PTSD symptoms (Curry, Latkin & Davey-Rothwell, 2008; Wilson & Rosenthal, 2003). These mental health symptoms may serve as risk factors for risky sexual behaviors. In fact, in one study among adolescent males, increases in PTSD mediated the relationship between exposure to community violence and an earlier sexual debut (Voisin, Hotton, & Neilands, 2014). Substance use is another potential pathway. Neighborhood crime has been associated with increase

substance use and this increased substance use may then lead to risky sexual behavior. Another factor discussed in the literature is change in sexual behavior norms. The norms around sexual behaviors in communities with high crime might be less strict and thus the rules of sexual safety might be more lenient. Voisin et al., (2014) found that the link to exposure to community violence was also partially mediated by negative attitudes about sexual safety. Although this evidence has not yet been linked specifically to MSM, preliminary direct and indirect links between neighborhood crime and sexual risk behaviors are evident.

The collection of these studies highlights the fact that structural factors play a role in predicting sexual risk behaviors and STI transmission. Low income, unstable housing and neighborhood crime are all structural risk factors for STI transmission. The pathways by which these structural variables might influence health outcomes are varied and complex. Structural factors may exert their influence through mental health, health behaviors, or by way of influencing other structural variables. Despite MSM being disproportionately affected by STI/HIV prevalence, there is a paucity of research that examining the effects of structural variables in this population. None of this research has been done in a cohort of high-risk MSM with a history of abuse. This may limit our understanding of how these variables function in this population, particularly among ethnic minority MSM.

Ethnic and Racial Disparities

Existing data show that HIV and STIs disproportionately affect Black and Latino MSM and STIs compared to non-Hispanic whites. Black MSM currently account for the highest number (38%) of HIV infections among MSM. Despite representing only 17% of

the U.S. population, Hispanic/Latinos account for almost a quarter of new HIV infections. Around three quarters of these new diagnoses occur in gay and bisexual men. Furthermore, the data suggest that rates of chlamydia, gonorrhea, and syphilis are two to four times higher in Latinos than in whites (CDC, 2017). Black MSM have eight times the odds of having a current STI diagnosis than non-Black MSM. The CDC recently estimated that, should rates continue as they are 1 in 2 black MSM and 1 in 4 Latino MSM would have a lifetime HIV-infection compared to 1 in 11 white MSM.

This disproportionate prevalence of STIs and HIV among Black and Latino MSM, particularly for black MSM, is likely not due to heightened sexual risk behaviors among these ethnic groups (Millett, Flores, Peterson, & Bakeman, 2007; Millett et al., 2012). Despite similar or less risky profiles among Black MSM, the STI and HIV disparities persist; thus, researchers have turned to more distal factors that might influence these rates. For example, Latino MSM in a large probability sample were two times more likely to report a history of CSA compared to non-Latino MSM (Arreola, Neilands, Pollack, Paul, & Catania, 2005). In a separate analysis, black MSM were almost two times more likely to report a history of childhood sexual abuse relative to other MSM (Millett et al., 2012; Phillips et al., 2014). Furthermore, Black and Latino men are likely to report similar severity in terms of CSA experiences (Moisan, Sanders-Philips, & Moisan, 1997). Only one study has tested race differences in the effects of CSA on sexual risk and this study found that the effects did not differ by race (Phillips et al., 2014). However, some methodological concerns existed related to the assessment CSA. Namely, that a single question was used which directly asked about sexual abuse. This may have resulted in under-reporting of CSA particularly by ethnic minority MSM

due to the cultural stigma of reporting such abuse (Phillips et al., 2014). Evidence from the broader mental health literature suggests that PTSD following a traumatic event is more likely among Blacks compared to whites (Roberts, Gilman, Breslau, Breslau, & Koenen, 2011). PTSD has a greater likelihood of being untreated across all ethnic minorities (Roberts et al., 2011). Given previously reviewed literature on the association of CSA with PTSD and PTSD with other risk behaviors (e.g. substance use), and the disproportionate rates of trauma among Black and Latino MSM, it is crucial to understand how psychological sequelae of trauma might differentially impact Black and Latino MSM.

Differences in structural risk factors among Black and Latino MSM versus others may be another factor that may account for disproportionate STI incidence. Blacks and Latinos are two to three times more likely to be living in poverty compared to whites (Williams & Sternthal, 2010). Compared to white MSM, black MSM are more likely to live in areas characterized by poverty, unemployment, and lower median annual income (Sullivan et al., 2014). This puts Black and Latino MSM at increased likelihood of being exposed to neighborhood crime and community violence (Margolin & Gordis, 2000). Research also indicates that factors such as unstable housing as well a limited access to affordable housing disproportionately affects ethnic minorities compared to the White majority (DeNavas-Walt, Proctor, & Smith, 2011). Further, these social factors may interact with each other or with psychological factors in order to influence risk (Widman, Noar, Golin, Willoughby, & Crosby, 2014).

The way in which ethnic minorities might be exposed to disproportionate STI risk may be by way of psychological or structural-level risk factors. Indeed, Black

and Latino MSM seem to be disparately affected by potent risk psychological factors known to influence sexual risk (e.g., CSA and substance use). Additionally, the social context for many Black and Latino MSM likely exacerbates the level of risk. These social risk factors such as poverty, unstable housing, and neighborhood crime likely have direct and indirect influence on risk behavior. However, what remains unknown is how these variables might differentially impact sexual risk and STI for ethnic minorities versus the ethnic majority. Furthermore, this has not been tested in a high-risk population with exposure to prior trauma. Examining the moderating role of ethnic minority status may help us to better understand the function of psychological and structural risk factors.

STIs have heavily and disparately affected MSM and particularly Black and Latino MSM. Research has indicated that CSA is a pervasive issue for sexual minorities and is more likely to be experienced among these risk groups. We know that the effect of CSA on certain psychological factors may be similar across sexual minorities; however, we do not know the degree to which race might moderate the effect of these factors on sexual risk and STI incidence. Considering the context in which these psychological factors occur, it appears that in some cases these men may be more likely to be in a context that fosters sexual risk and STI. However, the degree to which these factors differentially impact Black/Latino MSM compared to White MSM remains unstudied, especially in high-risk traumatized populations such as that in the current proposal. Thus, a secondary aim of this proposal is to address these gaps in the literature by evaluating pathways by which psychological and structural level factors influence sexual risk and STI for ethnic minority and ethnic majority MSM.

Theoretical Underpinnings

Most work that has sought to assess and intervene on psychological and structural influences on HIV risk behaviors has used Social Cognitive Theory as a guiding framework. Conceptualized by Bandura (1986), this theory highlights how an individual's self-efficacy might influence their ability to change their behavior. Thus, HIV risk interventions have often sought to address psychosocial barriers to people's self–efficacy to engage in safe sex (Meader et al., 2013). However, as alluded to earlier, structural/contextual factors have a strong influence in explaining risk behavior. Social Action Theory is a useful extension of social cognitive theory, which emphasizes different social, motivational, and contextual influences that shape behavior (Ewart, 1991; Reynolds et al., 2010). Using this theory, the outcomes in our study (i.e., unprotected sex and STI incidence) are conceptualized as being influenced by resources and social power that are afforded by an individual's environmental context as well as psychopathology, attitudes and knowledge, and self-regulatory processes. Using Social Action Theory as a guiding framework, the hypothesized explanatory variables in this study will focus on psychopathology and affect (post-traumatic stress symptoms and substance use) as well as the contextual environment (government benefits as a proxy for low income, unstable housing, and neighborhood crime).

We also know from the literature that these variables do not often affect people in the same way and that the prevalence of these variables and their impact on risk behaviors can differ as a function of race. This highlights the relevance of Intersectionality Theory. This term was originally termed by Kimberlé Crenshaw to describe the interaction between racism and sexism in promoting violence against women

of color (Crenshaw, 1991). She also describes the concept of structural intersectionality, which highlights the idea that there are multilayered structural contexts which must be addressed in the context of intervention. Thus, in examining moderating effects of both race and structural disadvantage, we will be using the theory of intersectionality as a guiding framework.

This study is comprised of both a cross-sectional and longitudinal investigation of the effects of psychological/social determinants on sexual risk and STI incidence. This study is an ancillary study coming from an existing NIMH-funded, multisite clinical trial examining the efficacy of a cognitive-behavioral HIV prevention intervention in reducing post-traumatic symptom severity and subsequent sexual risk in MSM with histories of CSA.

Aims

Specific Aim #1: To examine baseline cross-sectional interrelationships between psychological (PTSD and substance use) and structural factors (income, unstable housing, and neighborhood crime) and number of unprotected sex encounters and evaluate the model among racial/ethnic minority MSM.

Hypothesis #1: Increased PTSD symptomology and substance use will be associated with a higher number of unprotected sex encounters.

***Hypothesis #2*:** Higher structural disadvantage (Lower income and more unstable housing and neighborhood crime) will be associated with a higher number of unprotected sex encounters.

Hypothesis #3: Racial or ethnic minority status will moderate the relationship between

psychological/structural determinants and unprotected sex such that this link will be stronger for ethnic minority men compared to their non-ethnic minority counterparts.

Specific Aim #2: To examine baseline cross-sectional interrelationships between psychological factors (PTSD and substance use) and structural factors (income, unstable housing, and neighborhood crime) to STI incidence and evaluate the model among racial/ethnic minority MSM.

Hypothesis #1: Increased PTSD symptomology and substance use will be associated with self-reported STI infection in the past 12 months.

***Hypothesis #2*:** Higher structural disadvantage (Lower income and more unstable housing and neighborhood crime) will be associated with self-reported STI infection in the past 12 months

***Hypothesis #3*:** Racial or ethnic minority status will moderate the relationship between psychological/structural determinants and self-reported STI infection such that the link will be stronger for ethnic minority men relative to their non-ethnic minority counterparts

Specific Aim #3: To longitudinally model the impact of psychological/social determinants on unprotected sex encounters and examine impact of structural factors on treatment response.

Hypothesis #1: Increased PTSD symptomology and substance use will predict steeper increases in unprotected sex encounters over a period of 12 months.

***Hypothesis #2*:** Higher structural disadvantage (Lower income and more unstable housing and neighborhood crime) will predict steeper increases in unprotected sex encounters over a period of 12 months.

***Hypothesis #3*:** There will be significant structural factor x group interactions where

those in the intervention will experience a weaker relationship between structural level factors and sexual risk than those in the control group.

Chapter 2: Method

Study Overview

At baseline, participants completed a battery of self-report psychosocial measures, a clinician-administered diagnostic PTSD assessment, a computer-assisted (ACASI) a thorough assessment of substance use, sexual risk and STI within the past 12 months, and self-reported STI history in the past year. Assessments and questionnaires were repeated every 3 months for one year. An independent assessor completed all follow-up assessments of PTSD. All procedures were approved by the institutional review boards (IRB) at the respective sites at which the study took place (i.e., Massachusetts General Hospital IRB, Fenway Community Health IRB, and University of Miami IRB).

Recruitment/Participants

Participants were MSM reporting a history of CSA, recent sexual risk (>1 act of condomless sexual intercourse in the past three months), and current HIV-negative status. Major methods of recruitment included venue outreach (Clubs, bars, cruising areas), community outreach (HIV prevention organizations, HIV testing centers, Department of Health), and advertising (Craigslist – men seeking men and volunteer sections, mobile social networking applications, and MSM-oriented magazines). Participants were excluded if they required immediate treatment for severe mental illness (e.g. untreated bipolar disorder, active psychotic episodes). Due to substance use being a common correlate of CSA and its effects on sexual risk and STI acquisition, individuals currently managing substance addiction or dependence were included and referred for concurrent substance abuse treatment as appropriate.

Procedure

Participants had five major assessment points: baseline, acute post-treatment (3 months), 6 months, 9 months, and 12 months. At baseline, participants completed the informed consent process with a clinician and underwent psychodiagnostic assessment for PTSD via the Structured Clinical Interview for the DSM-IV Axis I Disorders (SCID-I; First, 2005) and the Davidson PTSD Scale (Davidson, Malik, & Travers, 1997). Participants also completed a self-report battery, which assessed demographics, recent substance use, structural barriers, sexual activity, and STI history. Computer-assisted data collection (ACASI) was used to facilitate likelihood of disclosure of sensitive information of psychosocial questionnaires (e.g. risky sexual activity, STI history, and substance use; Islam et al., 2012; Morrison-Beedy, Carey & Tu, 2006). At the next four major study time points (acute post-treatment, 6, 9, and 12- month follow-ups) an independent assessor (who was blind to study condition) re-administered the diagnostic assessments for PTSD.

Baseline time point (pretreatment). At baseline, participants provided informed consent. Participants also completed a psychosocial questionnaire battery, which included measures of recent substance use, sexual risk, and STI history. ACASI was used to facilitate accurate reporting of substance use, sexual risk, and STI history. This computer-administered questionnaire also gathered information about income/government benefits. A clinician then conducted a CSA interview to ensure eligibility requirements were met and obtained additional information on PTSD symptomology.

Post Treatment and Follow-up Time points: At post-treatment and subsequent follow-ups, the same procedure as the baseline assessment was followed; however, the

demographics questionnaire was not administered (thus, we did not have repeated measures of government benefits), and the CSA interview was not repeated. As with the baseline assessment, the Davidson PTSD scale was administered; however, this was done by an independent assessor blind to study condition rather than the treating therapist. Those who had not completed the interview for structural barriers completed it during the follow-up and this was used as a proxy for baseline.

Post-completion interview. A random subset of participants who had completed the study returned after the 12-month follow-up and completed additional questionnaires related to structural barriers (i.e., unstable housing and neighborhood crime) and were reimbursed $25. These measures were used as a proxy for baseline measures.

Measures

Demographics. Information on age, race, ethnicity, gender, income, relationship/marital status, housing status, occupational, educational, and employment status was collected from each participant.

Government benefits. As part of the demographics questionnaire, participants self-reported whether they received all or part of their annual income from government-sponsored programs (i.e., welfare, social security, or disability). Those who reported receiving all or part of their income from these programs were coded 1 and this was conceptualized as a structural barrier.

Unstable housing. As part of this ancillary study, a measure was added to the original study protocol in which participants were asked three questions as part of a self-report questionnaire. (1) In the last six months have you been homeless? By homeless we

mean sleeping in a car, public place not intended for sleeping, homeless shelter, single room occupancy (SRO) or welfare hotel or motel. (2) In the last six months have you been in temporary or transitional housing (by temporary or transitional we mean temporarily doubled up with family or friends, temporarily in someone else's home, in halfway house with no other address, in drug treatment, or in jail. (3) For the past six months have you had secure, permanent housing, in an apartment, house, or group quarters? Based on their answers to these three questions, participants were categorized into three categories: Stably housed, unstably housed, or homeless. Those who reported being unstably housed or homeless were considered as having unstable housing. This was adapted from previous work done with housing status and HIV risk (Aidala et al., 2005).

Neighborhood crime. The crime subscale of the Neighborhood Environment Walkability Scale (Saelens, Sallis, Black, & Chen, 2003) contains four questions used to assess perceptions of neighborhood crime and safety. Participants self-reported, on a 4-point Likert scale, the degree to which they agreed with the following statements: "There is a high crime rate in my neighborhood", "The crime rate in my neighborhood makes it unsafe to go on walks during the day", "The crime rate in my neighborhood makes it unsafe to go on walks at night", and "My neighborhood is safe enough so that I would let a 10-year-old boy walk around my block alone in the daytime (reverse coded item)." This scale has demonstrated good validity and reliability (Saelens et al., 2003).

Structural disadvantage. The structural disadvantage variable was created by summing up the number of theoretical structural barriers endorsed. A participant received a score of one for each structural barrier endorsed i.e., being unstably housed or homeless, receiving government benefits as a source of income, or having a score above

the median on the neighborhood crime scale. The number of endorsements was tallied and participants could receive a score ranging from 0 to 3.

Substance use. Substance use was assessed using the Addiction Severity Index – Lite Version (ASI-Lite; McLellan, Cacciola & Zanis, 1997). The ASI-Lite is a semi-structured clinician-administered instrument that obtains lifetime information about problem drug and alcohol use as well as the frequency of use within the past 30 days. This questionnaire inquired about a variety of substances: alcohol use, alcohol use to intoxication, heroin, methadone, opiates, barbiturates, sedative/hypnotics, cocaine, amphetamines, cannabis, hallucinogens, inhalants, and poly substance use. It also inquired about whether each substance has been a problem for the participant in their lifetime. We focused specifically on stimulant drug use (cocaine, crack, amphetamines (e.g., Ritalin®, Vyvanse®), crystal methamphetamine and use of poppers. Substance use in the past 30 days was dichotomized into a yes or no variable. This abbreviated scale demonstrates acceptable validity and reliability and is psychometrically equivalent to the full version (Cacciola, Alterman, McLellan, Lin, & Lynch, 2007).

PTSD symptomology. The Davidson PTSD scale was used to assess PTSD symptomology. The Davidson is a 17-item self-report measure, based on the PTSD symptom clusters as defined by DSM-IV (Davidson et al., 1997). The frequency and severity of each symptom during the past week is rated on a Likert scale ranging from 0 to 4. Increasing numbers indicate higher frequency and severity. Items were summed to yield a total score. The scale has good test-retest reliability (r = .86) and internal consistency (r = .99) and has been validated for use in those with CSA (Zlotnick, Davidson, Shea, & Pearlstein, 1996).

Structured CSA interview. The CSA Interview contains standardized questions that assess sexual abuse history. The interview was developed and used in medical populations including populations of people living with HIV (Leserman, Li, Drossman, & Hu, 1998; Leserman, Ironson, O'Cleirigh, Fordiani, & Balbin, 2008). Though sexual abuse typically implies the threat of force or harm, in children under 13 this is thought to be implied by a five-year difference in age between the perpetrator and the victim.

Sexually transmitted infections. STI incidence was measured via the ACASI self-report where participants will indicate whether they have been diagnosed or treated for an STI in the past year (at baseline) or since their last study visit (at follow-up visits).

Unprotected sex. Sexual risk was assessed at each of the major time points (Baseline, 3-months, 6-months, 9-months and 12-months post-baseline) and participants were asked standard sexual risk questions via ACASI. The measure asked about receptive and insertive anal and vaginal intercourse, with and without condoms, with male and female partners separately. Partners were further broken down into those who are seropositive, seronegative, and those of unknown HIV status. Each reported condomless anal or vaginal intercourse with casual partners (versus monogamous partners) was counted as a risk episode. Each condomless anal or vaginal intercourse with a partner of known seropositive status (regardless of whether the partner is a casual or monogamous partner) was also counted as a risk episode.

Notably, 12 participants reported being on pre-exposure prophylaxis (PrEP) during the study and those participants who reported 80% adherence or more were coded zero for sexual risk. Participants who reported being on PrEP, but were missing adherence values were imputed with the average adherence values reported for the

respective timepoint. Thus, in terms of our sexual risk outcome, we are primarily interested in anal or vaginal intercourse with a casual partner or a partner of known seropositive status. *Unprotected* in the context of this study refers to those intercourse episodes in which a condom was not used and a person was either not on PrEP or was less than 80% adherent to PrEP.

Statistical Analysis

A conceptual model is depicted in Figure 1. The primary outcomes in the proposal were sexual risk, analyzed as a continuous outcome (number of sexual risk episodes) and sexually transmitted infection (yes/no) analyzed as a dichotomous categorical outcome. Additionally, in aims #1 and #2 ethnic minority status was analyzed as a moderator to the effects of psychological and structural-level factors on sexual risk and STI incidence. In the longitudinal aim, to reduce overlap between this proposal and the main study hypotheses of the parent grant, all longitudinal analyses will controlled for randomization.

Missing data. In cross-sectional analyses, missing data was handled using multiple imputation methods. Sensitivity analyses were run and the results and compared. If the two sets of results are substantially different, both will be reported. In longitudinal analysis, missing data were managed using the Maximum Likelihood (ML) method under the assumption of missingness at random (MAR). ML uses all the available data from a case to produce unbiased and efficient parameter estimates (Enders & Bandalos, 2001; Wothke, 2000). Additionally, precipitants of attrition were examined by analyzing whether systematic differences exist between participants who complete the study and those who drop out. Differences were also assessed among those participants who

completed additional questionnaires relating to structural barriers and those who did not. If systematic differences did exist, these variables were added as covariates to the models.

Covariates. All analyses controlled for geographical location (Boston vs. Miami) and age at enrollment. Race/ethnicity and intervention arm were only controlled in models where it was not of direct interest. Thus, race and ethnicity were only covariates in the longitudinal analyses (i.e., Aim #3). Intervention arm was also only a covariate in the longitudinal aim (Aim #3). Education was added as a covariate in all hypotheses testing psychological factors. Due to potential multicollinearity with government benefits/income, it was not added as a covariate in models testing effects of structural factors.

Specific aim 1: cross-sectional interrelationships between psychological/structural-level factors and sexual risk. The primary analysis for this aim examined the strength of relationships between psychological/structural-level factors and the number of unprotected sex encounters using OLS regression. Davidson total score, substance use, government benefits, neighborhood crime, and housing status were entered separately as predictors into the model. Each of the predictors was continuous variables except for substance use, housing status, and government benefits. These three variables were dichotomous predictors and were dummy-coded into 0 and 1. ‘0’ indicated "not endorsed" and ‘1’ indicated "endorsed." For example, a score of "1" on the government benefits variable indicated that the participant endorsed using government benefits to supplement their income. In each analysis, the ‘not endorsed’ group served as the reference group. The secondary analysis examined Black/Latino minority status as a

moderator of these relationships. Dummy interaction terms were created and added to the model to examine the interaction between racial/ethnic minority status and psychological/structural-level factors. P-values less than .05 were considered significant. Following recommendations of Cohen (1992), our a priori power analyses revealed that, with 4 predictor variables 84 subjects would be needed to detect medium effects with power at .8 and the alpha level set at .05.

Specific aim 2: cross-sectional interrelationships between psychological/structural factors and STI incidence. The primary analysis for this aim examined the effect of psychological and structural predictors on the odds of having tested positive for an STI within the past 12 months. Since the outcome was a dichotomized categorical variable, logistic regression was used as the analytic method[24]. Davidson total score, substance use, government benefits, neighborhood crime, and housing status were entered separately as predictors into the model. Each of the predictors was continuous except for substance use, housing status, and government benefits. These three variables were dichotomous predictors and were dummy-coded into 0 and 1. ‘0’ indicated "not endorsed" and ‘1’ indicated "endorsed." The secondary analysis examined racial/ethnic minority status as a moderator of these relationships. Interaction terms were created and added to the model to examine their effects on STI in the past 12 months. Our a priori power analysis indicated that, to detect a minimum odds ratio of 0.1 at 0.8 power, With Pr(y=1) = .175, a sample size of 147 subjects would be needed (Hsieh, 1989).

Specific aim 3: longitudinal effect of psychological/structural-level factors on sexual risk. To accomplish this aim, we used hierarchical linear modeling (HLM;

Raudenbush & Bryk, 2002) to investigate the impact of psychological/structural-level factors on sexual risk, as measured by number of unprotected sex encounters over the course of one year. The model included two levels (Equations are given in Table 1). Level 1 included time since baseline (measured in months), which corresponded to the time at which each of the repeated measures of sexual risk was done (including the baseline measure).

The level 2 dataset focused on individual differences in change in the level 1 data set controlling for a priori covariates relevant to unprotected sex encounters. Number of unprotected sex encounters at baseline was also be added as a covariate to control for the possibility that initial risk may influence subsequent risk throughout the study. All continuous variables were centered and categorical variables were dummy-coded in the same way as previous aims. Separate models were run for each psychological and structural factor. With the anticipated sample size of 200, for the continuous measure of change in number of sexual risk episodes, with a two-tailed p-value of 0.05, assuming a 15% attrition over the course of the study, and estimating correlations between repeated measures in the outcome of .8, with 5 repeated measures, there is 91% power to detect a small to medium effect sizes (Pan & McBee, 2014). The power for estimating the moderated effect of race/ethnicity is approximate and we estimate power, based upon similar assumptions to be 80%.

Chapter 3: Results

Table 2 contains demographic information for the total sample and the reduced structural sample. No significant differences existed in terms of demographics between the two samples. Missingness on all variables was less than 3%. Table 3 shows descriptive information for the variables of interest in the study for both the full and reduced sample. No significant differences existed on these variables between the full and reduced samples. Table 4 shows interrelationships between variables of interest in the current analysis. In terms of covariates, educational attainment was significantly associated with proportion of sexual risk activity $F(1, 279) = 11.065$, $p = .001$), where those with lower educational attainment reported a higher proportion of unprotected sex episodes relative to those with higher educational attainment ($b = -0.314$, $SE = 0.094$). Educational attainment was not significantly related to STI in the past year. Age was not significantly related to number of unprotected sex encounters, $F(1, 280) = 0.366$, $p = .545$, b=.005; however, it was significantly related to STI in the past year (OR = 0.930, $p < .001$). Site was not significantly related to unprotected sex encounters, nor was it related to STI in the past year. Thus, for AIM #1, analyses examining the relationship between psychological/structural factors and number of unprotected sex encounters remained unadjusted. For Aim #2, all analyses adjusted for age.

AIM #1 Analyses

Table 5 shows the unadjusted unstandardized coefficients and corresponding p-values for OLS regressions examining the relationship between each psychological and structural level factor and unprotected sex encounters. PTSD symptomology was associated with number of unprotected sex episodes, $F(1, 278) = 7.163$, $p = .008$, $R^2 =$

.025) such that more frequent and severe PTSD symptoms were predictive of a higher number of unprotected sex episodes (b.010, SE = .004). Crystal meth use in the preceding thirty days was also associated with proportion of unprotected sex episodes, ($F(1, 285) = 6.977$, $p = .009$, $R^2 = .024$) such that those who used crystal meth in the past month endorsed a higher number of unprotected sex episodes than those who did not (b = 0.863, SE = 0.327). Cocaine, prescription amphetamine, and poppers/inhalant use were not significantly associated with proportion of unprotected sex episodes.

Among the structural level factors, income was significantly associated with number of unprotected sex encounters, $F(1, 280) = 4.574$, $p = .033$, $R^2 = .016$) where higher income predicted a lower number of unprotected sex episodes ($b = -0.168$, $SE = 0.078$). Additionally, use of government-sponsored benefits was associated with a higher number of unprotected sex encounters, $F(1, 286) = 13.346$, $p < .001$, $R^2 = .045$) wherein use of government benefits was associated with more unprotected sex encounters ($b = 0.715$, $SE = 0.196$). Those using government benefits at baseline had an average of 0.72 more unprotected sex encounters than those not using government benefits. Unstable housing was also significantly associated with unprotected sex encounters ($F(1, 286) = 3.983$, $p = .047$, $R^2 = .014$) where those who reported being homeless or unstably housed in the past six months reported a higher number of unprotected sex episodes than those who reported having stable housing for the past six months ($b = 0.680$, $SE = 0.341$). Thus, those who had been unstably housed in the previous six months had about 0.68 more unprotected sex encounters, on average, than those who were stably housed during this time. Neighborhood crime was not significantly associated with number of unprotected sex episodes.

Aim #1 adjusted analyses. Results of adjusted analyses examining the association of psychological and structural factors with number of unprotected sex episodes are shown in Table 6. Among the psychological factors, PTSD symptoms continued to be significantly associated with number of unprotected sex episodes. Over and above educational attainment, those with higher PTSD scores reported having more unprotected sex episodes (b = 0.008, SE = 0.004, p = .027). Crystal Meth use in the preceding month also remained significantly associated with unprotected sex episodes over and above educational attainment. Those endorsing crystal meth use reported having a higher number of unprotected sex episodes relative to those reporting no use in the preceding month (b = .890, SE = .328, p = .007). Use of cocaine, crack, amphetamine, or poppers was not significantly associated with number of unprotected sex encounters. Structural factors remained unadjusted to avoid multicollinearity with education as a covariate.

In order to test whether any of these relationships were moderated by race, interaction terms were created, after centering the variables where appropriate, and tested above and beyond main effects. However, we did not find that race moderated any of the relationships between psychological/structural factors and unprotected sex episodes.

AIM #2 Analyses

Table 7 shows the unadjusted unstandardized coefficients, odds ratios, and corresponding p-values for binary logistic regressions examining the relationship between each psychological/structural factor and STI infection in the past year. PTSD symptomology was the only psychological factor associated with odds of having an STI in the past year such that greater frequency and severity of PTSD symptoms was associated with lower odds of testing positive for an STI during the past year (OR =

0.986, p = .040, 95%CI = 0.973 – 0.999). Cocaine, prescription amphetamine, crystal meth, and poppers/inhalant use were not significantly associated with odds of STI in the past year. Additionally, none of the structural predictors (income, neighborhood crime, or unstable housing) were shown to be associated with odds of STI infection in the past year.

To test whether any of these relationships were moderated by race, interaction terms were created, after centering the variables where appropriate, and tested above and beyond main effects. There was a significant race x PTSD interaction (p = .026) where higher PTSD symptoms predicted lower odds of STI infection for blacks (OR = 0.957, p = .007, 95%CI = 0.928 – 0.988) and Latinos, but not for others (OR = 0.996, p = .638, 95%CI = 0.981 – 1.012). (OR = 0.965, p = .014, 95%CI = 0.937 – 0.993)

Aim #2 adjusted analyses. Table 8 shows the adjusted unstandardized coefficients, odds ratios, and corresponding p-values for binary logistic regressions examining the relationship between psychological/structural level factors and STI infection in the past year. When adjusting for age, the main effect of PTSD symptoms on STI was no longer significant (p = .159). However, the PTSD x race/ethnicity was marginally significant for Black (p = .051) and Latino MSM (p = .090) wherein PTSD continued to predict lower odds of incident STI for these racial minority men and PTSD had no association with incident STI for others. Cocaine, prescription amphetamine, crystal meth, and poppers/inhalant use as well as our structural predictors were not significantly associated with odds of STI infection in the past year. Additionally, we did not find any interactions between race and our psychological/structural level factors that

were significantly related to STI in the past year as the previous PTSD x race interaction in the unadjusted model was no longer significant.

AIM #3 Analyses

The basic model for predicted unprotected sex episodes over the course of the study period adjusting for age, race/ethnicity, education, randomization, and number of unprotected sex encounters at baseline. There was a significant linear decrease in unprotected sex encounters controlling for covariates. The model indicates that, on average, people began the study with 2.48 unprotected sex encounters in the preceding 3 months, and this decreased at a rate of 0.128 unprotected sex encounters per month (about 1.5 unprotected sex encounters per year) above and beyond the effects of white race, average age, and a HS education or below. There was also significant individual variation in change in number of unprotected sex encounters over time $\chi^2(161) = 200.751$, $p < .001$.

Psychological predictors. Table 9 contains the results and significance tests for HLM models testing the effects of PTSD and substance use at baseline on changes in unprotected sex encounters. Cocaine use at baseline predicted increases in unprotected sex encounters at a rate of 0.051 encounters per month (about 0.6 unprotected sex encounters per year), $\beta_{16} = 0.051$, $t(177) = 2.354$, $p = .020$. Use of crystal meth, poppers, amphetamines, or crack was not associated with changes in number of unprotected sex encounters over time. PTSD at baseline also did not predict unprotected sex encounters over time.

Structural predictors. Table 9 also contains the results and significance tests for HLM models testing the effects of structural barriers endorsed at baseline on changes in unprotected sex encounters. Use of government benefits (i.e., SSI, Welfare, Disability) predicted decreases in unprotected sex encounters at a rate of 0.059 episodes per month (about 0.7 unprotected sex encounters per year) $\beta_{16} = 0.051$, $t(177) = 2.354$, $p = .020$. Unstable housing and neighborhood crime did not predict changes in unprotected sex encounters. Our structural disadvantage variable (i.e., number of structural barriers endorsed) also did not predict changes in number of unprotected sex encounters.

Chapter 4: Discussion

This analysis sought to examine the relationship between psychological and structural factors and how they relate to engagement in unprotected sex and past-year incidence of STI in a cohort of MSM endorsing histories of childhood sexual abuse. For our cross-sectional hypothesis related to unprotected sex encounters (Aim #1), we found support for a relationship between psychological factors (PTSD and substance use in the form of crystal meth) and number of unprotected sex encounters. We also found support for an association of structural barriers with increased unprotected sex encounters where those receiving government benefits and those endorsing unstable housing also reported a higher number of unprotected sex episodes. For our cross-sectional hypothesis related to incidence of STI (Aim #2), we found surprisingly, that PTSD was significantly associated lower odds of STI in the past year and this result was primarily driven by Black MSM. In our analysis of our longitudinal hypothesis related to unprotected sex encounters over twelve months, cocaine use at baseline predicted more unprotected sex encounters, while use of government benefits predicted less unprotected sex encounters (which is the opposite of what was found cross-sectionally for government benefits). Overall, our results suggest that higher PTSD symptomology is cross-sectionally related to more unprotected sex encounters, and substance use and structural factors are both cross-sectionally and longitudinally related to more unprotected sex encounters; however, we were not able to show that these factors necessarily translate into STI risk.

Many of our findings were consistent with the extant literature. In terms of our psychological variables, we found cross-sectional support for the link between higher frequency and severity of PTSD symptoms and increased number of unprotected sex

encounters. In terms of substance use, we found that crystal methamphetamine was cross-sectionally linked with more episodes of unprotected sex and cocaine use predicted increased unprotected sex encounters over a period of 12 months. As alluded to previously in the introduction, avoidance is a central symptom and critical maintaining factor in PTSD (Foa et al., 1991; Ehlers & Clark, 2000). One of the common ways avoidance is accomplished is through use of substances. In our sample of traumatized sexual minority men, it may be that use of cocaine or crystal methamphetamine represents avoidance-driven substance use behaviors that are key pathways to unprotected sex. In terms of structural barriers, the data showed evidence for a cross-sectional structural syndemic, wherein structural variables were all associated each other and, with the exception of neighborhood crime, were also associated with higher number of unprotected sex encounters.

Other findings were divergent from what has been found in the literature and different from our original hypotheses. While we found a cross-sectional link between higher frequency and severity of PTSD and unprotected sex encounters, higher frequency and severity of PTSD symptoms seemed to be linked to lower odds of being diagnosed with an STI in the preceding 12 months before baseline. Closer examination of this finding revealed that it was primarily driven by the Black MSM in our sample. That is, when we examined the racial interaction, the inverse relationship between PTSD and STI incidence was significant for Black and Latino men, but there was no relationship for non-Black/Latino MSM. Notably, our data showed that Black and Latino participants had similar levels of risk to other racial/ethnic groups (see Table 3). Previous meta-analytic work has also shown similar findings wherein despite Black MSM having similar

amounts of unprotected sex, they are more likely to have undiagnosed STIs (Millett et al., 2012). Latino MSM are also less likely to be tested for HIV and are more likely than White MSM to cite fear of a positive test result as a barrier to testing (Mackellar et al., 2011). Thus, it may be that the Black and Latino MSM in our sample, who are particularly sensitive to fear due to their trauma histories, may be more likely to have an undiagnosed STI and/or less likely to be tested in the preceding 12 months before study initiation. This finding may also be an artifact of the number of tests done within this scope of work. Further analysis will be done to determine whether this is the case. We also found a stark contrast from the literature in terms of structural barriers, wherein those receiving government-assisted income (i.e., welfare, SSI, or disability) had decreased number of unprotected sex encounters compared to those not receiving government benefits, despite an initial cross-sectional association with a higher number of unprotected sex encounters. In exploring this finding further, we found that those receiving government benefits tended to be older (see Table 3) and had more regular engagement with the healthcare system ($r = .211$, $p < .001$) through a higher number of preventative care visits (r = .174, $p = .020$), ER visits ($r = .243$, $p < .001$) and visits with a mental health professional ($r = .111$, $p = .059$). Notably, there was no association between receiving government benefits and number of medical visits for clinical concerns of an STI. Thus, it may be that participants receiving government benefits have more chronic health conditions which may result in less engagement in sex in general over time. It may also be that something about being regularly engaged with the healthcare system leads to less unprotected sex encounters (e.g., individuals could be getting tested more regularly and/or receiving sexual risk reduction counseling).

Therefore, government benefits served as a good proxy for low-income as a structural barrier, cross-sectionally; however, when examined as a longitudinally, it may represent an intervention in and of itself helping people with more chronic health conditions access medical care.

Although it makes important contributions, the study is not without its own limitations. Firstly, due to sample size constraints, we were a bit underpowered to detect effects, specifically in tests of structural variables. Consequently, while some of our cross-sectional hypotheses initially showed relationships between structural barriers and unprotected sex encounters, these relationships did not always pan out longitudinally. Particular areas of concern for low power were in Aim #2 specifically in tests of structural factors as we only had 108 participants with structural data and to be sufficiently powered we needed 147. Additionally, in Aim#3 tests of structural factors, there were only 87 degrees of freedom and the a priori power analysis had indicated 91% power with 200 subjects. Thus, it is very likely that these analyses were underpowered. Measures of structural barriers were added after the parent study had been underway for about two years. Thus, a significant proportion of participants needed to be called back after study completion and, although we obtained a random sample of individuals (with respect to the variables in this study); we were not able to get enough participants to be sufficiently powered in some of our more rigorous statistical tests. Secondly, PrEP received FDA approval in summer of 2012 when study recruitment had already been underway for about one year. Thus, only 12 people reported taking PrEP; however, we attempted to account for PrEP with the limited adherence data we had. While PrEP is an effective intervention to prevent HIV, it does not offer protection from STIs. While it is

possible that results may not generalize directly to HIV risk with more people being on PrEP, it is likely that the psychological and structural variables studied here would still contribute indirectly to HIV risk by interfering with adherence (Milloy et al., 2012; Vranceanu et al., 2008; White, Gordon & Mimiaga, 2014), which is critical to ensure efficacy of PrEP. Third, we relied on self-reported STI status, which is vulnerable to self-report bias. We did, however, analyze the link between social desirability and self-reported STI in the past twelve months and did not find a significant relationship. Additionally, we used presence of any STI which introduces the possibility that some STIs may have been transmitted by unprotected oral sex rather than unprotected anal or vaginal sex. *Lastly*, some important factors were not included in the analysis simply because they were not measured in the parent study. For example, we did not measure use of erectile dysfunction drugs which can also be used in the context of stimulant drugs to help men sustain erections. In terms of structural barriers food insecurity and incarceration history are additional factors not measured in this study which may potentially influence HIV risk (Jones et al., 2008; Weiser et al., 2011). Though unmeasured in this study, these represent additional structural barriers that have been shown to affect HIV risk.

Future studies might expand on some of the questions asked here using more sufficiently powered designs. The finding about government benefits leading to lower risk over time despite an initial cross-sectional association indicating higher risk is an interesting one. We did not systematically collect information on medical comorbidities, which could have been useful here. It would be interesting to know whether accessing government benefits is a potential sexual risk reduction strategy for patients with medical

comorbidities who might otherwise have trouble accessing medical care. Particularly as LGBT aging becomes a better studied topic especially in the context of sexual risk, future studies might consider collecting information on medical comorbidities. The Social Action Theoretical (SAT; Ewart, 1991) framework provides a well-timed conceptualization for understanding how individual and structural factors might explain HIV risk behaviors and future studies might expand on the scope of work of the current study using this same framework. One way to do this might be to explore additional variables within psychological and contextual constructs. As mentioned previously, use of erectile dysfunction drugs and the role of incarceration might be particularly useful, the latter being particularly important for the population in this study since those with trauma are at increased risk for issues with the legal system. Another way to incorporate the SAT framework is to investigate other factors besides psychological and contextual ones such as self-regulatory processes. This work could not only look at the contribution of self-regulatory processes (e.g., affect regulation; Carrico et al., 2015; Carrico et al., 2016) on sexual risk behaviors and psychological factors, it might also look at the relationship between structural barriers and self-regulatory processes. Some research suggests that poverty may negatively affect brain function by consuming mental resources and reducing cognitive capacity, leaving less mental faculties to be used on other tasks (Mani, Mullainathan, Shafir, & Zhao, 2013). In terms of intervention, this study and others indicate that structural barriers are related to sexual risk behaviors; however, given the mixed results of addressing structural factors alone in decreasing unprotected sex, more interventions are needed that integrate intervention for structural barriers in addition to psychological ones.

Clinical Implications

Our findings generally confirm that both psychological and structural factors are associated with unprotected sex and, importantly, extend these findings to a particularly vulnerable and understudied population. Results are suggestive of potential points for intervention and, fortunately, some groundwork has been laid for interventions to address these factors, albeit with various levels of efficacy. For example, psychological treatments such as Cognitive Processing Therapy (CPT) and Prolonged Exposure (PE) are considered highly effective treatments to address PTSD symptoms (Cusack et al., 2016). Although more research is needed in this area, promising evidence of efficacy also exists in the realm of pharmacotherapy for selective serotonin reuptake inhibitors (SSRIs; Hoskins et al., 2015). The degree to which these psychological and pharmacological treatments for PTSD translate to improve sexual risk reduction remains unknown. However, data analysis from the parent grant of the current study is under way to determine whether psychological PTSD treatment adapted for sexual risk might be effective in treating PTSD and improving sexual risk reduction. In terms of substance use, behavioral interventions (e.g., motivational interviewing and cognitive-behavioral interventions) have been shown to be effective in reducing unprotected anal sex although the degree to which this is attributable to reductions in substance use is inconclusive (Carrico, Zepf, Meanley, Batchelder, & Stall, 2016). Additionally, emerging preliminary evidence has shown positive effects of pharmacotherapy agents (e.g., naltrexone) on reducing unprotected sex in substance using MSM (Santos et al., 2016). However, as the current study demonstrates, structural syndemics are also associated with unprotected sex. There is a paucity of well-controlled RCTs examining the structural barriers studied

in the current analysis and most of the work has focused on unstable housing/homelessness. Although the association of unstable housing/homelessness with HIV risk behaviors has been well-studied, the degree to which improvements in housing status confer risk reduction benefits is unclear. A couple of studies have addressed the question of whether improved housing translates into sexual risk reduction (Aidala et al., 2005, Wolitski et al., 2010). One study (Aidala et al., 2005) showed that those participants whose housing improved were less likely to report unprotected sex at last sex encounter and the other (Wolitski et al., 2010) did not show improved sexual risk reduction benefit between intervention and control groups. These types of studies are often difficult to conduct given the methodological, operational, and ethical challenges of conducting a housing RCT. However, notably these two studies did show decreased substance use, improved mental health and increased health utilization which are variables we know to be associated with unprotected sex. This emphasizes the need for collaborative care interventions which address multiple psychological and structural HIV syndemics.

Conclusion

Our study demonstrates generally that both psychological and structural factors are at play in terms of influencing sexual risk behavior. Specifically, it provides evidence of cross-sectional associations of crystal methamphetamine use, unstable housing, and government benefits with a higher number of unprotected sex encounters and higher PTSD symptomology with both higher number of unprotected sex encounters and lower odds of incident STI for Black MSM. It also provides longitudinal evidence of the effect of cocaine use on increased unprotected sex encounters and the effect of government

benefits on decreased unprotected sex encounters over a period of 12 months. Importantly, this study examines effects of these psychological and structural factors in a population at high-risk for HIV (MSM with histories of CSA) that is less likely to derive benefit from sexual risk reduction programs. Thus, the study makes important contributions to the literature in terms of providing plausible psychological and contextual points of intervention that might need to be given attention when designing interventions for this population. Future studies should continue to examine the role of structural factors (e.g., government benefits) as both a risk factor and an intervention for risk behavior in populations for high-risk of HIV and STIs and continue to look at other Social Action Theory-related constructs in relation to HIV/STI risk.

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Figure 1: Conceptual model for cross-sectional and longitudinal associations of psychological and structural-level factors on unprotected sex and STI and the moderating role of race/ethnicity and structural disadvantage.

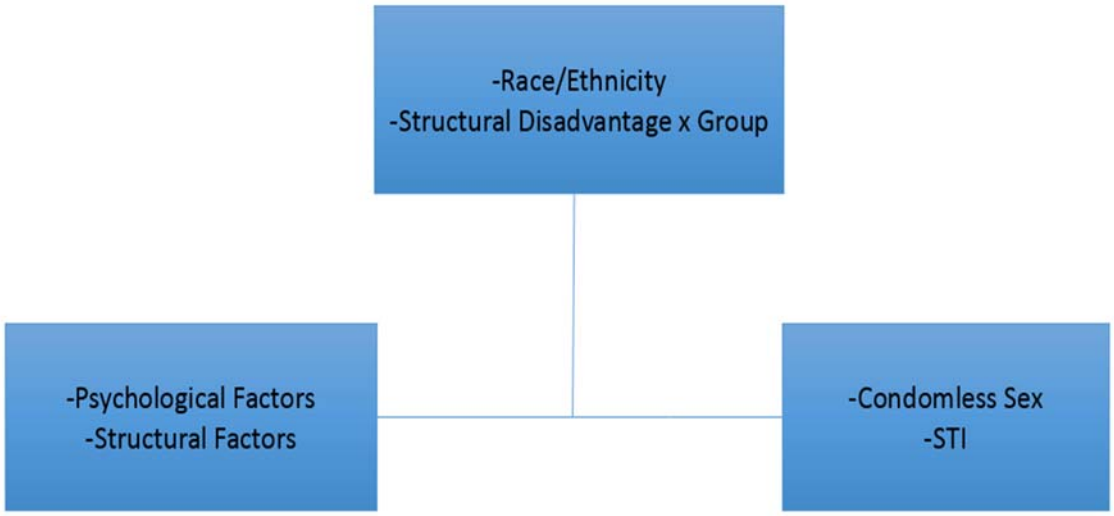

Table 1*: HLM Equations Pertaining to Aim #3 (Testing Longitudinal Effects of Psychological/Structural Factors on Sexual Risk)*

Equations Testing Effects of Individual-Level Factors:

Level 1 (repeated measures):

$Y_{ti} = \beta_{0i} + \beta_{1i}\,(\text{time})_t + e_{ti}$

Level 2 (individuals):

$\beta_{01}\,(\text{intercept}) = \gamma_{00} + u_0$

$\beta_{1i}\,(\text{slope}) = \gamma_{10} + \gamma_{11}\,(\text{Race/Ethnicity}) + \gamma_{12}\,(\text{Education})_i + \gamma_{13}\,(\text{Age})_i + \gamma_{14}\,(\text{BL sex risk})_i + \gamma_{15}\,(\text{Group})_i + \gamma_{16}(\text{Psychological Factor})_i + u_{1i}$

Equations Testing Effects of Structural-Level Factors and Structural Factor x Group Interactions:

Level 1 (repeated measures):

$Y_{ti} = \beta_{0i} + \beta_{1i}\,(\text{time})_{ti} + e_{ti}$

Level 2 (individuals):

$\beta_{01}\,(\text{intercept}) = \gamma_{00} + u_0$

$\beta_{1i}\,(\text{slope}) = \gamma_{10} + \gamma_{11}\,(\text{Race/Ethnicity}) + \gamma_{12}\,(\text{Age})_i + \gamma_{13}\,(\text{Group})_i + \gamma_{154}(\text{BL Risk})_i + \gamma_{15}\,(\text{Structural Factor})_I + \gamma_{15}\,(\text{Structural x group}) + u_{1i}$

Table 2: *Demographics for total and reduced sample of MSM with histories of childhood sexual abuse (CSA).*

	Total Sample (N = 299)	*Structural Sample (n = 108)*
Age	*M* = 37.95, *SD* = 11.68	*M* = 37.73, *SD* = 11.67
Education		
HS graduate/GED or below	25.3%	21.4%
Some college	36.7%	34.0%
College Graduate	24.6%	25.2%
Graduate Degree	13.5%	19.4%
Race/Ethnicity		
Caucasian	68.6%	67.6%
Black	22.1%	24.8%
Asian/Pacific Islander	3.7%	2.9%
Other	5.6%	7.6%
Hispanic*	29.9%	29.8%
Site		
Boston	61.5%	63.0%
Miami	38.5%	37.0%
Annual Income		
Below $10k	30.3%	27.9%
$10k-$20k	22.9%	23.1%
$20k-$40k	18.5%	16.3%
$40k and above	28.3%	32.7%

*Hispanic is collapsed across all races

Table 3: *Descriptive statistics for variables of interest separated by total and reduced samples of MSM reporting histories of CSA*

	Total Sample *N* = 299	*Structural (Reduced) Sample (N=108)*
Unprotected Sex Encounters	*M* = 6.75, *SD* = 10.54	*M* = 7.03, *SD* = 12.97
0 encounters	8.0%	4.8%
1 encounter	14.6%	17.1%
2 encounters	17.7%	21.0%
3-4 encounters	19.4%	23.8%
5-9 encounters	20.1%	18.1%
10+ encounters	20.1%	15.2%
STI in past 12 months		
Yes	29.5%	26.3%
No	70.5%	73.7%
Syphilis	22.4%	33.3%
Gonorrhea	30.6%	33.3%
Chlamydia	26.5%	20.0%
Unknown/Don't Remember	20.5%	13.4%
Davidson PTSD Total Score	*M* = 34.54, *SD* = 26.22	*M* = 32.30, *SD* = 24.58
Cocaine Use		
Yes	81.4%	81.4%
No	18.6%	18.6%
Crack use		
Yes	6.1%	4.8%
No	93.9%	95.2%
Crystal Meth use		
Yes	8.8%	6.7%
No	91.2%	93.3%
Amphetamine use		
Yes	9.8%	9.5%
No	90.2%	90.5%
Poppers use		
Yes	25.3%	25.2%
No	74.7%	74.8%
Government Benefits		
Yes	29.6%	31.4%
No	70.4%	68.6%
Unstable Housing	-	
Yes	-	21.5%
No	-	78.5%
Neighborhood Crime	-	*M* = 7.61, *SD* = 3.10
No. of Structural Factors		
Zero	-	38.0%
One	-	37.0%
Two	-	14.8%
Three	-	10.2%

Table 4: Correlation Table examining interrelationships between dependent, independent, and covariate variables of interest among N = 299 MSM with histories of CSA

	1	2	3	4	5	6	7	8	9	10	11	12	13	14	15	16
1. Sexual Risk	1	-.098	.036	-.195**	.067	.158**	-.018	.155**	.015	-.053	-.127*	.144	.117*	.211**	.320**	-.015
2. STI	-.098	1	-.350**	.102	.101	-.163*	-.003	.002	.028	-.071	-.028	.005	-.090	-.099	-.128	.081
3. Age	.036	-.350**	1	.048	-.047	.053	-.062	.095	-.079	.092	.185**	-.147	-.036	.177**	-.007	-.152*
4. Education	-.195**	.102	.048	1	-.165**	-.166**	-.047	-.007	.051	.232**	.448**	-.288**	-.144*	-.306**	-.450**	.057
5. Black v. Other	.067	.101	-.047	-.165**	1	.024	-.027	-.023	-.095	-.183**	-.171**	-.043	.087	.105	.045	-.309**
6. Davidson Total	.158**	-.163*	.053	-.166**	.024	1	-.027	-.023	-.095	-.183**	-.171**	-.043	.087	.105	.045	-.032
7. Cocaine	-.018	-.003	-.062	-.047	-.027	.033	1	.250**	.251**	.189**	.062	.114	-.042	.084	.070	.044
8. Crystal Meth	.155**	.002	.095	-.007	-.023	.115	.250**	1	.138*	.067	-.066	.068	.043	.121*	.167	-.020
9. Amphetamines	.015	.028	-.079	.051	-.095	.033	.251**	.138*	1	.122*	.104	.138	-.054	.043	.046	-.127*
10. Poppers	-.053	-.071	.092	.232**	-.183**	-.105	.189**	.067	.122*	1	.227	-.206*	.063	-128*	-.051	-.024
11. Income	-.127*	-.028	.185**	.448**	-.171**	-.151*	.062	-.066	.104	.227**	1	-.234*	-.154**	-.361**	-.397**	.092
12. N. Crime	.144	.005	-.147	-.288**	-.043	.167	.144	.068	.138	-.206*	-.234*	1	.169	.286**	.637**	-.061
13. Unstable Housing	.117*	-.090	-.036	-.144*	.087	.059	-.042	.043	-.054	.063	-.154**	.169	1	.186**	.697**	-.027
14. Gov. Benefits	.211**	-.099	.177**	-.306**	.105	.149*	.084	.121*	.043	-.128*	-.361**	.286**	.186**	1	.732**	-.189**
15. # Struct. Factors	.320**	-.128	-.007	-.450**	.045	.268**	.070	.167	.046	-.051	-.397**	.637**	.697**	.732**	1	-.131
16. Hispanic v. Other	-.015	.081	-.152*	.057	-.309**	-.032	.044	-.020	-.127*	-.024	.092	-.061	-.027	-.189**	-.131	1

*$p < .05$, **$p < .01$

Table 5: *Unstandardized and unadjusted OLS regression coefficients for the relationship between each psychological and structural-level factor and number of sexual risk episodes*

	B	*SE*	*t*	*p*
Psychological Factors				
Davidson Total	.008	.004	2.229	.027*
Cocaine	-.041	.238	-.172	.863
Crystal Meth	.890	.328	2.713	.007**
Amphetamines	.140	.310	.450	.653
Poppers	-.033	.221	-.150	.881
Structural Factors				
Neighborhood Crime	.065	.051	1.273	.206
Unstable Housing	.680	.341	1.996	.047*
Gov. Benefits	.583	.205	2.848	.005**
Structural Disadvantage	.507	.154	3.285	.001**

*p <.05, **p <.01, ***p <.001

Table 6: *Unstandardized OLS regression coefficients examining the relationship between each psychological/structural level factor and number of unprotected sex encounters adjusting for education (SES).*

	B	*SE*	*t*	*p*
Psychological Factors				
Davidson Total	.010	.004	2.676	.008**
Cocaine	-.072	.239	-.303	.762
Crystal Meth	.863	.327	2.641	.009**
Amphetamines	.080`	.314	.255	.799
Poppers	-.193	.215	-.899	.369
Structural Factors	-.168	.078	-2.139	.033*
Neighborhood Crime	.070	.047	1.476	.143
Unstable Housing	.531	.341	1.558	.120
Gov. Benefits	.715	.196	3.653	<.001***
No. of Structural Factors	.472	.138	3.429	.001**

*p <.05, **p <.01,

Table 7: *Unstandardized and unadjusted logistic regression coefficients for the relationship between each psychological and structural factor and STI infection in past 12 months prior to baseline visit*

	B	*SE*	*Wald*	*df*	*p*	*Exp(B)*
Psychological Factors						
Davidson Total	-.014	.007	4.228	1	.040	.986*
Cocaine	-.017	.422	.002	1	.967	.983
Crystal Meth	.016	.521	.001	1	.975	1.017
Amphetamines	.190	.532	.128	1	.721	1.209
Poppers	-.353	.389	.825	1	.364	.703
Structural Factors						
Neighborhood Crime	.004	.109	.002	1	.968	1.004
Unstable Housing	-.891	.788	1.278	1	.258	.410
Gov. Benefits	-.476	.376	1.600	1	.206	.621
Structural Disadvantage	-.313	.327	.917	1	.338	.731

*$p < .05$

Table 8: *Unstandardized logistic regression coefficients, odds ratios, and significance values for the relationship between individual and structural level factors and STI infection in past 12 months adjusting for age*

	B	*SE*	*Wald*	*df*	*p*	*Exp(B)*
Psychological Factors						
Davidson Total	-.011	.007	2.367	1	.124	.989
Cocaine	.074	.429	.030	1	.863	1.077
Crystal Meth	.128	.529	.059	1	.808	1.137
Amphetamines	.145	.539	.073	1	.787	1.156
Poppers	-.599	.416	2.075	1	.150	.550
Structural Factors						
Neighborhood Crime	.058	.118	.239	1	.625	1.059
Unstable Housing	-.791	.793	.994	1	.319	.453
Gov. Benefits	-.328	.398	.679	1	.410	.720
Structural Disadvantage	-.185	.357	.268	1	.605	.831

Table 9: *Hierarchical Linear Modeling results for prediction to number of unprotected sex encounters from baseline psychological and structural factors adjusting for age, randomization, race/ethnicity, education[a], and baseline number/ proportion of unprotected sex episodes.*

Predictor	*γ coefficient*	*t-ratio*	*df*	*p*
Psychological Factors				
PTSD Symptoms	0.0002	0.448	177	.655
Cocaine use	0.049	2.312	177	.022*
Crack Use	-0.014	-0.339	177	.735
Crystal Meth use	0.017	0.585	177	.559
Amphetamine use	0.001	0.045	177	.964
Poppers use	-0.010	-0.514	177	.608
Structural Factors				
Government Benefits	-0.059	-2.402	87	.018*
Unstable Housing	-0.027	-0.988	87	.326
Neighborhood Crime	-0.0004	-0.112	87	.911
Structural Disadvantage	-0.018	-1.508	87	.135

[a]Education was only added as a covariate in tests of psychological factors
*$p < .05$