ABSTRACT

Heterogeneity in Etiological Factors for Substance Use Among Low-Income African American Adolescents

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Impoverished African American adolescents comprise a subgroup of individuals who are at a uniquely increased risk for substance use, making early identification of African American adolescents who are most likely to engage in substance use a relevant public health issue. While much research has explored a variety of risk and protective factors for substance use, complementary approaches may uncover distinct subgroups of individuals with homogeneous patterns of risk and/or protective factors which may discriminate among those who are more or less likely to use substances across adolescence. The present study draws on the Social Development Model and holistic-interactionism to identify unique prototypical patterns of risk and protective factors and assess the extent to which these patterns are predictive of alcohol, cigarette, and marijuana use across adolescence. Using data from the Mobile Youth Survey, (n = 1,576, 100% African American, 45.5% female), we identified three discrete patterns of protective factors, characterized by high, average, and low levels of parental knowledge; six patterns of protective factors characterized by sequentially increasing levels of delinquency, exposure to violence, and peer pressure; and six patterns of etiological factors characterized by sequentially increasing levels of delinquency, exposure to violence, accompanied by concurrently decreasing levels consisting of parental knowledge and self-worth. While there was no evidence of

heterogeneity in cigarette or marijuana use trajectories, we identified *high use* and *average use* alcohol trajectories. Patterns characterized by high levels of delinquency, exposure to violence, and peer pressure as well as either low or average levels of parental knowledge were associated with increased odds of demonstrating either the *high use* alcohol trajectory or higher initial levels of cigarette or marijuana use. The results indicate that risk and protection are two discrete, yet interactive, dimensions, wherein greater predictive specificity may be attained through examining risk and protective factors separately. While no factors emerged as strong protective factors, protection may have a discrete and specific function in the context of certain patterns of risk factors; which factors provide the most protection against substance use among members of this population should be investigated in future research.

Heterogeneity in Etiological Factors for Substance Use Among Low-Income African American Adolescents

by

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DEDICATION

To Greg, Mom, Dad, Grace, and Marie

CHAPTER ONE

Background and Literature Review

Overview

Adolescent substance use is a major public health concern, given that individuals who initiate into substance use early in adolescence are at an increased risk for substance use disorders (SUDs) later in life (Grant & Dawson, 1998). Further, those who engage in increasing or uniformly high levels of substance use throughout adolescence are at risk for other maladaptive outcomes, such as criminal behavior, both in adolescence and into young adulthood (Lynne-Landsman, Bradshaw, & Ialongo, 2010; Schulenberg, Patrick, Maslowsky, & Maggs, 2014). These concerns are especially pronounced with respect to African Americans, who are less likely to receive treatment for substance use problems and are more likely to experience consequences associated with substance use compared to their white peers (Copeland-Linder, Lambert, Chen, & Ialongo, 2011; Cummings, Wen, & Druss, 2011; Schmidt, Greenfield, & Mulia, 2006; Zapolski, Pedersen, McCarthy, & Smith, 2014). As such, attention should be given to identifying factors that predict substance use among African American adolescents.

The present study aims to identify prototypical patterns of risk and protective factors and assess the extent to which these patterns predict trajectories of alcohol, cigarette, and marijuana use. Chapter One presents empirical research on risk and protective factors among African American adolescents. Chapter Two combines the Social Development Model and holistic-interactionism to present a theoretical framework for identifying unique prototypical patterns of risk and protective factors. Chapter Three outlines the methodology of utilizing data from the Mobile Youth Survey to identify patterns of risk, protective, and etiological factors using latent profile analysis, and trajectories of alcohol, cigarette, and marijuana use using growth mixture modeling. Chapter Four provides the results of

the analysis, exploring the extent to which the conditional associations between the risk and protective factor patterns and the etiological factor patterns predict the substance use trajectories. Chapter Five discusses the results and provides implications for theoretical considerations of risk and protective factors as separate entities.

This chapter discusses empirical research identifying various risk and protective factors for substance use among low-income African American adolescents. We begin by discussing the societal problem of substance use and explore why specific attention should be given to substance use among African American adolescents. We then turn our attention to previous research on discrete etiological factors for substance use, exploring how these factors individually and collectively predict later substance use.

Adolescent Substance Use

Adolescence is a critical period for the development of substance use. Data from the Youth Risk Behavior Surveillance Survey indicate that nearly 30% of adolescents drank alcohol, nearly 20% had used marijuana, and nearly 9% had smoked a cigarette in the past 30 days (Kann et al., 2018). The prevention literature encourages focus on adolescence as a critical period of identifying risk factors for and delaying the initiation of substance use, particularly because early substance use has been associated with a number of adverse consequences later in life (Brook, Lee, Finch, Brown, & Brook, 2013; Grant & Dawson, 1998; Schulenberg et al., 2014). Prior research suggests that those who begin to use drugs earlier in life are at increased risk for SUDs, evidenced by data from a national sample showing that for each additional year substance use initiation was delayed, odds of substance abuse and dependence were reduced by 5% and 4%, respectively (Grant & Dawson, 1998).

Emphasis is also placed on adolescent substance use because, once initiated, substance use typically increases across this developmental period (K. A. Bolland et al., 2016; Chen & Jacobson, 2012). One study examining an urban community sample of African American and white females found that average trajectories of alcohol use indicated steady

increases in alcohol use across ages 11-15 in both racial/ethnic groups (Loeber, Stepp, Chung, Hipwell, & White, 2010). Another study examined average change in alcohol and cigarette use across ages 13-19 in an urban, mixed-race sample of both males and females, similarly finding that both alcohol and cigarette use increased across adolescence regardless of gender or racial/ethnic group, with particularly sharp increases across ages 15-19 (Gutman, Eccles, Peck, & Malanchuk, 2011). Similar results have been found for marijuana, as another study found that average trajectories of marijuana use among a sample of urban white and African American adolescents increased from grade 6 through high school graduation (T. L. Brown, Flory, Lynam, Leukefeld, & Clayton, 2004).

The observed normative escalation in substance use across adolescence is frequently attributed to the unique developmental transitions that occur during this time period, namely rapidly changing social contexts and individual physiological and identity changes (Schulenberg et al., 2014). Adolescents begin to spend more time away from their parents as they take on more independent activities and responsibilities at school and in their social contexts, resulting in the decreasing influence of parental knowledge and parental monitoring and the increasing influences of peer influence on behavior (Wood, Read, Mitchell, & Brand, 2004). Adolescents' increasing amounts of time spent with friends may increase likelihood for substance use through direct peer influence or through changing adolescents' attention to socially rewarding situations (O'Brien, Albert, Chein, & Steinberg, 2011). Working a part-time job, a rite of passage for many adolescents, has also been associated with an increased risk of substance use (Monahan, Lee, & Steinberg, 2011). Puberty, the most characteristic process of the adolescent experience, has also been thought to indirectly influence the initiation and escalation of substance use across adolescence, as early maturing peers are likely to befriend slightly older adolescents, leading to greater exposure and opportunities to use substances (Downing & Bellis, 2009; Susman & Dorn, 2009). Further, brain structures associated with emotion regulation, decision-making, and planning develop across adolescence, but do not fully mature until the young adult years; this underdeveloped

nature of the adolescent brain may contribute to risky decision making and substance use escalation across adolescence (Sturman & Moghaddam, 2011). While the above does not include a comprehensive review of the multitude of factors that are thought to contribute to the escalation of substance use across adolescence, it is important to note that adolescence constitutes a unique developmental context that provides numerous opportunities for initiation into and escalation of substance use.

Although substance use increases on average across adolescence as part of a developmentally normative progression, additional research has suggested that multiple trajectories (comprised of initial levels of use and rates of change across adolescence) may best represent a spectrum of adolescent substance use behaviors across adolescence (White et al., 2006); that is, although substance use increases on average across adolescence, not all adolescents demonstrate uniform initial levels and rates of change in their substance use. Multiple discrete patterns of alcohol, cigarette, and marijuana use have been found across adolescence. For example, one study utilizing a nationally representative sample found five discrete trajectories of marijuana use consisting of (1) non-users, (2) early users who subsequently remit, (3) gradually increasing users, (4) early increasing users who remit in early adulthood, and (5) early increasing, persistent users; three trajectories of alcohol use consisting of (1) abstainers, (2) early excessive drinkers who subsequently remit, and (3) persistently increasing heavy drinkers; and four trajectories of cigarette smoking consisting of (1) abstainers, (2) early increasers who subsequently remit, (3) moderate stable smokers, and (4) persistently increasing smokers (Park, McCoy, Erausquin, & Bartlett, 2018). A second study identified five trajectories of alcohol use among low-income urban youths, consisting of (1) no use, (2) consistently infrequent use starting at age 14, (3) monthly use starting at age 14, (4) monthly use at age 13 to heavy use by age 14, and (5) heavy use onset at age 12 (Komro, Tobler, Maldonado-Molina, & Perry, 2010).

Research indicating that there are multiple trajectories that may best describe longitudinal patterns of adolescent substance use is important for two primary reasons. First,

trajectories are differentially associated with outcomes later in life such that, in general, trajectories indicating predominately high or increasing levels of substance use are predictive of poorer young adult adjustment. Among a sample of African American adolescents, individuals who were likely to be members of trajectories characterized by increasing cigarette use were less likely to graduate on time and were more likely to engage in illicit drug use, have committed both violent and nonviolent juvenile crimes, and have a criminal record in early adolescence (Lynne-Landsman et al., 2010). The same study found that those most likely to demonstrate marijuana trajectories characterized by consistently high use across adolescence and initially high followed by decreasing use across adolescence were differentially associated with later levels of violent and nonviolent offenses. As such, trajectories characterized by high substance use may be part of a larger developmental cascade characterized by further problem behavior.

Second, trajectories may be differentially predicted by unique etiological factors, which are contextual factors or characteristics that indicate differential likelihood of exhibiting substance use; that is, we may be able to identify specific factors that predict problematic trajectories of substance use, in turn allowing for identification of adolescents who are at greatest risk for problematic trajectories. For instance, one study found that higher levels of delinquency and violent behavior were associated with increased odds of demonstrating trajectories of infrequent or heavy alcohol use relative to the odds of no alcohol use across adolescence (Komro et al., 2010). Other research suggests that, especially among females, early initiation into substance use is associated with increased odds of demonstrating trajectories of increasing alcohol use across adolescence (K. A. Bolland et al., 2016). Identifying factors that are associated with increased likelihood of demonstrating trajectories of problematic substance use across adolescence may provide insight into the unique etiology underlying various types of substance use and also allow for preemptive monitoring of young adolescents who demonstrate those characteristics. Given the heterogeneity in trajectories of substance use across adolescence, and the extent to which other factors may

indicate increased likelihood of membership in one or more trajectories, it is of interest to continue to identify what factors, or combination of factors, indicate increased likelihood of membership in substance use trajectories.

Substance Use Among Low-Income African American Adolescents

African American adolescents are engaging in substance use at similarly concerning rates. In 2017, 25% of African American adolescents reported using marijuana in the past 30 days, 21% reported drinking alcohol, and 4% reported smoking cigarettes (Kann et al., 2018). Adolescent-initiated substance use among African Americans specifically has been associated with a multitude of adverse health and social consequences during both adolescence and adulthood, including increased prevalence of psychiatric disorders (Roberts, Roberts, & Xing, 2007), engagement in risky sexual behavior (Elkington, Bauermeister, & Zimmerman, 2010; Ritchwood, Ford, DeCoster, Sutton, & Lochman, 2015), altered brain development (Squeglia, Jacobus, & Tapert, 2009; Volkow, Baler, Compton, & Weiss, 2014), risk of incarceration (Slade et al., 2008), and suicidal ideation (Joe, Baser, Neighbors, Caldwell, & Jackson, 2009). In addition to facilitating the prevention of these consequences, attention should be given to substance use among African American adolescents living in impoverished neighborhoods for a number of reasons. First, the history of systematic oppression and discrimination of African Americans is well-documented, and has been associated with an increased risk of substance use and other psychosocial consequences. Second, African American adolescents are more likely to live in disadvantaged neighborhoods, which may significantly increase their exposure to substance use, deviant behaviors, and related risk factors. Relatedly, African American adolescents have limited access to treatment resources, making prevention of substance use an integral component in fostering healthy development. Lastly, African Americans experience a disproportionate number of consequences related to substance use relative to white Americans at similar levels of consumption. We briefly outline each of these points below.

Systematic Oppression and Substance Use

It is well known that African Americans have consistently been the victims of discrimination in the United States since its founding (Banton, 1998). Although societal-level, systematic discrimination is generally outlawed, the residual effects of formal discrimination are still present in African Americans' experiences of interpersonal discrimination (Hebl, Foster, Mannix, & Dovidio, 2002), de facto segregation (Rothstein, 2015), and limited access to life reinforcers such as financial stability (Zapolski et al., 2014). The lingering effects of formal discrimination and consistent experiences of interpersonal discrimination are thought to account for a wide range of health disparities between white and African Americans (Williams, Yu, Jackson, & Anderson, 1997).

Indeed, experiencing discrimination has been linked to increased risk for a number of psychiatric, health, and mental health-related disorders (Pascoe & Smart Richman, 2009; Stock, Peterson, Molloy, & Lambert, 2017; Williams, Neighbors, & Jackson, 2003). Specifically, experiencing discrimination has been linked to increased alcohol use among African Americans (Gibbons, Gerrard, Cleveland, Wills, & Brody, 2004; Parenteau, Waters, Cox, Patterson, & Carr, 2017; Terrell, Miller, Foster, & Watkins, 2006; Yen, Ragland, Greiner, & Fisher, 1999). African Americans who reported experiencing racial discrimination had 51% higher odds of using alcohol compared to African Americans who did not report experiencing racial discrimination (Borrell et al., 2010). Further, a separate study found that every additional experience of discrimination reported among a sample of both African American and Caribbean Blacks was associated with 10% increased odds of AUD (Hunte & Barry, 2012). Less research has focused on relationships between experiences of discrimination and cigarette or marijuana use, but those studies that do exist point towards significant associations between perceived discrimination and cigarette/marijuana use, but with small effect sizes (e.g., a .02% increase in likelihood of cigarette use as perception of discrimination increases; Horton & Loukas, 2013; Kendzor et al., 2014; Purnell et al., 2012).

Additional research has identified an increased risk for substance use among individuals who experience one or more forms of minority stress including, but not limited to, other racial identification, sexual orientation, gender identity, and indigenous heritage (Cheadle & Whitbeck, 2011; Flores, Tschann, Dimas, Pasch, & de Groat, 2010; Lehavot & Simoni, 2011; Mereish & Bradford, 2014; Yoo, Gee, Lowthrop, & Robertson, 2010). However, we elected to focus the present study on an African American population for two reasons. First, the history of systematic oppression against African Americans is particularly well-documented over four centuries (Feagin, 2013). Second, a basic theoretical framework, discussed previously, has been proposed to account for higher levels of alcohol use specifically among African Americans (Zapolski et al., 2014). Given the exploratory nature of the current study, it was important that we focus specifically on a population for whom theory has identified particular motivating and mitigating factors that may influence alcohol, cigarette, and marijuana use. While we specifically chose to focus on African Americans for this particular study, we anticipate that a similar examination of etiological factors for substance use could, and should, be conducted among members of other minority populations who may experience similar health-related consequences, including, but not limited to, mental health and suicide risk, substance use, and sexual health outcomes (e.g., HIV) as a result of systematic oppression.

Effects of Neighborhood Disadvantage

African American adolescents living in impoverished neighborhoods may be at a unique risk for substance use as a result of ecological and contextual factors common in these neighborhoods that may increase the prevalence of opportunities for drug use and crime. Indeed, low socioeconomic status and poverty may increase neighborhood disadvantage which in turn may place individuals at a higher risk for substance use (Unger, 2012). Neighborhood disadvantage indicates the presence of a number of contextual factors (e.g., living below the poverty line, unemployment rate, percentage of female-headed

households, and receipt of public assistance) that are associated with limited opportunities for treatment resources (as detailed below), discrimination, and exposure to stressful experiences, all of which are associated with an increased likelihood of substance use (Boardman, Finch, Ellison, Williams, & Jackson, 2001; Unger, 2012). Empirical findings suggest that neighborhood disadvantage is associated with an increased likelihood of adolescent health risk behaviors (Leventhal & Brooks-Gunn, 2000) and both directly and indirectly related to increased numbers of alcohol and marijuana dependence symptoms among adolescents (Handley, Rogosch, Guild, & Cicchetti, 2015). Given that a disproportionately large number of African Americans live in disadvantaged neighborhoods in the United States (Acevedo-Garcia, Osypuk, McArdle, & Williams, 2008), African Americans residing in these neighborhoods are at a unique risk for substance use.

There are at least two relevant theoretical perspectives that may inform why African American adolescents living in low-income neighborhoods are at risk for substance use. The Social Stress Model suggests that adolescents who engage in substance use do so in order to cope with stressors they feel across various domains of life (Rhodes & Jason, 1990). The model suggests that adolescents are more likely to use substances if their development of social competencies (i.e., general coping skills, self-efficacy, etc.) have been interrupted by poor interpersonal relationships with parents, teachers, and other figures such that (1) the stressors in their environment outweigh their available coping resources, and (2) they do not have another model for successful development and coping (Rhodes & Jason, 1990). On the other hand, successful development also occurs through a transactional process wherein parents who support their children foster the development of good social competence, who then feel that they have the personal resources available to confront challenges and adversity; as a result, these children are more able to effectively cope with stressful experiences and are less likely to resort to substance use (Rhodes & Jason, 1990). Living in disadvantaged neighborhoods has been associated with increased levels of

psychosocial stress (T. D. Hill, Ross, & Angel, 2005; Latkin & Curry, 2003); as a result, individuals who live in disadvantaged neighborhoods may experience higher levels of stress, and engage in substance use as a means of coping with contextual stressors (Boardman et al., 2001; Copeland-Linder et al., 2011).

Social Disorganization Theory provides another potential explanation for why low-income African American adolescents are at a unique risk for substance use (Sampson & Groves, 1989; Shaw & McKay, 1942). According to the theory, neighborhood characteristics such as low socioeconomic status and high residential mobility lead to increased prevalence of substance use and delinquency as a result of low neighborhood cohesion (Handley et al., 2015; Sampson & Groves, 1989; Shaw & McKay, 1942). Neighborhoods with low socioeconomic status and high residential mobility are more likely to exhibit social disorganization, or the inability of a community to collectively recognize common values and support effective social controls (Bursik, 1984; Kornhauser, 1978; Sampson & Groves, 1989, p.777). Neighborhoods with high levels of social disorganization are unable to facilitate strong social networks in a community by reducing the ability of the community to provide control over public congregations of adolescents, providing few opportunities to develop strong peer relationships, and failing to facilitate local participation in formal and voluntary organizations; together, these effects reduce the ability of the neighborhood to solve common problems, resulting in higher levels of crime, victimization, and other negative outcomes (Sampson & Groves, 1989). As a result, social disorganization is thought to mediate the relationship between poverty and residential mobility and various outcomes, such as substance use and delinquency (Sampson & Groves, 1989). Because impoverished African American adolescents live in areas with high levels of neighborhood disadvantage and social disorganization, they are at a unique risk for substance use due to an increased prevalence of substance use and delinquency in their communities.

As a result of living in disadvantaged neighborhoods, African Americans may have a disproportionate exposure to risk factors for substance use (Unger, 2012). Disadvantaged

neighborhoods have higher levels of poverty, discrimination, and neighborhood violence, all of which are risk factors for substance use and are more prevalent in African American neighborhoods (Mulia, Ye, Zemore, & Greenfield, 2008). Further, one study examined the relative prevalence of 55 predictors of substance use in African American and white adolescents (Hawkins, Catalano, & Miller, 1992; J. M. Wallace & Muroff, 2002). Their results suggested that there were significant differences between African American and white adolescents on over half of the studied risk factors, indicating that African American adolescents may be exposed to more risk factors for substance use than white adolescents (J. M. Wallace & Muroff, 2002). It is likely that the same risk factors are associated with later substance use among both white and African American adolescents, but because African Americans have exposure to a greater number of risk factors they are at a unique risk for substance use (Unger, 2012). Because African American adolescents may experience significantly more risk factors overall, it is of interest to examine how risk and protective factors are distributed and related to substance use specifically within a low-income African American sample.

Limited Access to Treatment Resources

In addition to an increased likelihood of substance use in one's community and differential exposure to risk factors, attention should be given to substance use among African American adolescents because African Americans are significantly less likely than white Americans to receive psychiatric or substance use treatment services (Angold et al., 2002; Merikangas et al., 2011; Schmidt et al., 2006). For example, in a study by Cummings et al. (2011) only 6.9% of African American adolescents received clinician-provided substance use treatment compared to 10.7% of white adolescents. Further, among communities where over 50% of adolescents live in poverty (most of which are predominately African American communities), there are on average zero child/adolescent psychiatrists per 100,000 adolescents available to provide care (Thomas & Holzer, 2006). The few services that are

available to low-income adolescents (many of which live in primarily African American neighborhoods) are typically of suboptimal quality (e.g., they do not provide medication or therapies that are up-to-date with current literature) compared to those programs that are available to higher-income adolescents (most of which live in primarily white neighborhoods; Garner, 2009).

These findings demonstrate that there are currently few resources available for adolescents in low-income communities; unfortunately, the number of available resources is anticipated to decrease, as federal spending on substance use and mental health treatment programs is expected to decline through 2020 (Mark, Levit, Yee, & Chow, 2014). This projected decrease in spending on care for substance use among African Americans suggests that relying on retroactive treatment to resolve adolescent substance use in low-income communities will likely fail; alternatively, efforts should be directed toward monitoring those most likely to engage in substance use.

Racial/Ethnic Disparities in Substance Use-Related Consequences

An additional reason to explore substance use among low-income African Americans is that members of this racial/ethnic group experience more consequences associated with substance use than do white Americans at the same level of consumption; a majority of research has focused on discrepancies in consequences specifically related to alcohol (Keyes et al., 2015; Mulia, Ye, Greenfield, & Zemore, 2009; Zapolski et al., 2014). Some consequences reflect health disparities; for instance, African Americans have a 10% higher risk of mortality due to liver cirrhosis (a common consequence of alcohol abuse) than any other ethnic group (Kochanek, Murphy, Anderson, & Scott, 2004). However, other consequences reflect difficulties in social functioning. Mulia et al. (2009) found that even among those who consumed relatively small amounts of alcohol, African Americans were three times more likely than white Americans to experience social consequences related to alcohol use, and were five times more likely to experience symptoms of dependence. Further,

race has been shown to moderate the inverse relationship between income and alcohol-related problems, such that these associations are stronger among non-white ethnic groups in the United States (Brenner, Diez Roux, Barrientos-Gutierrez, & Borrell, 2015).

Zapolski et al. (2014) provide an extensive review that postulates three potential reasons why African Americans may experience more alcohol-related consequences than do white Americans. First, African Americans may contain a genetic or biological vulnerability that increases their sensitivity to the effects of alcohol (Ehlers, Carr, Betancourt, & Montane-Jaime, 2003). If African Americans are more sensitive to the effects of alcohol or drugs, they may experience symptoms of intoxication and impairment at lower levels of consumption. The rapid experience of intoxication may lead to behavioral dysregulation that may result in social disapproval and/or consequences. Second, African American culture may have a lower tolerance for exhibition of intoxication, leading to social sanctions from other in-group members (Herd, 1994). Social tensions among friends and family members may decrease quality of life and inadvertently promote substance use as a means of self-medicating interpersonal conflict. Lastly, African Americans may be more likely to experience legal consequences related to alcohol use either as a result of increased surveillance over African American neighborhoods by majority ethnic groups, or because of racial discrimination (Conley, 1994; Mastrofski, Parks, Reiss, & Worden, 1999; Mulia et al., 2009; Zapolski et al., 2014). Regardless of the potential reasons for increased rates of alcohol-related consequences among African Americans, disparities in these rates are evidence that special attention should be given to drug and alcohol use within this population. While most of these consequences occur or are most prevalent in adulthood, further research should continue to examine potential avenues for preventing substance use among African American adolescents in order to prevent the onset or experience of these consequences, given the strong association between adolescent use and later consequences (Brook, Lee, Finch, et al., 2013; Grant & Dawson, 1998; Schulenberg et al., 2014).

Summary

African American adolescents living in low-income neighborhoods are at increased risk for problems related to substance use following early initiation (Brook, Lee, Rubenstone, et al., 2013; Grant & Dawson, 1998) and have limited access to substance abuse treatment resources (Costello, He, Sampson, Kessler, & Merikangas, 2014; Cummings et al., 2011; Mulia et al., 2009). Given that African American adults are more likely to live in disadvantaged neighborhoods (Acevedo-Garcia, 2008), have a greater exposure to risk factors (Unger, 2012; J. M. Wallace & Muroff, 2002), and experience alcohol-related consequences at the same, even low, levels of use compared to white Americans (Mulia et al., 2009; Zapolski et al., 2014), it is of interest to assess substance use etiology during adolescence among a sample of low-income African American adolescents. If substance use is preemptively addressed among African Americans during adolescence, youths may delay initiation, avoid an increase in substance use across adolescence, and limit the experience of adverse consequences associated with drug and alcohol use in adolescence and beyond. These potential benefits to the prevention of substance use among African American adolescents demonstrates the importance of research focused on early identification of those who are at greatest risk for substance use.

Prior Research on Substance Use Etiology

In order to identify adolescents who are most likely to engage in substance use throughout adolescence, the prevention literature has encouraged identifying etiological factors that are associated with increased likelihood for substance use, in addition to etiological factors that may buffer or protect against the effects of these risk factors. Risk factors generally refer to behaviors or traits present in an individual prior to drug use and are thought to be associated with using drugs, while protective factors are environmental contexts or personal traits which mitigate the effects of risk on substance use (Hawkins et

al., 1992). The following subsections review the empirical literature on the role of risk factors and protective factors separately in predicting substance use and provide evidence for a number of traits or contexts to be considered either risk factors or protective factors. The exploration of evidence supporting definitions of certain factors as risk factors or protective factors will be followed by a discussion of the theoretical implications of interactions across multiple risk and protective factors in predicting substance use.

Risk Factors

Broadly, risk for substance use has been conceptualized as an increased probability of having a negative outcome, namely, engaging in substance use (Sloboda, Glantz, & Tarter, 2012). Following this definition, risk factors are specific traits, attitudes, experiences, or contexts belonging to either the individual or interpersonal exchanges that occur before drug use and are associated with an increased likelihood of drug use (Hawkins et al., 1992). A similar conceptualization of risk factors reflects contexts or processes that are associated with poor outcomes relating to health or quality of life (Jessor, 1991), or that are associated with failures to reach normative developmental milestones (Nash & Bowen, 2002).

Much prior research has attempted to isolate the effects of specific risk factors on substance use. Below we briefly summarize the empirical literature on associations between substance use and risk factors that will be examined in the present investigation.

Delinquency. Engaging in delinquency has consistently been linked to increased likelihood of substance use, and these relationships appear to be robust across adolescence (W. A. Mason, Hitchings, McMahon, & Spoth, 2007). Delinquency refers to a number of behaviors including, but not necessarily limited to, gang activity, vandalism, and shoplifting or burglary. Regardless of whether research studies the effects of single instances of delinquency or composites of the number of delinquent acts engaged in over the past year, delinquency appears to be associated with later substance use (D'Amico, Edelen, Miles, & Morral, 2008).

In addition, delinquency may be another manifestation of the single underlying factor of problem behavior or unconventionality as proposed by Problem Behavior Theory (PBT; Donovan & Jessor, 1985; Jessor, 1987; Jessor & Jessor, 1977). PBT defines problem behavior as "behavior that is socially defined as a problem, a source of concern, or as undesirable by the conventional norms of society" (Jessor & Jessor, 1977, p.33), which covers a number of behaviors such as marijuana use, alcohol use, early sexual behavior, delinquency, and violent behavior. Rather than proposing that certain risk factors are more likely to predispose one to develop substance use or other problematic behavior, PBT suggests that substance use and other deviant behaviors are various expressions of a single underlying trait; that is, substance use and related risk factors are two observed manifestations of the same trait for deviance or unconventionality (Jessor, 1987). Because of its rather broad conceptualization of problem behavior, PBT may provide a theoretical background for the relationship between delinquency (in addition to other risk factors) and substance use.

Evidence from longitudinal studies suggests that the relationships between delinquency and substance use across adolescence appears to be causal such that delinquency predicted later substance use, but that substance use did not predict later delinquency (Hoyland, Rowatt, & Latendresse, 2017; Hunter, Miles, Pedersen, Ewing, & D'Amico, 2014). However, some evidence for bidirectional associations between delinquency and substance use have been found, suggesting that young adolescents who engage in delinquency may be more likely to use substances across adolescence, but that such substance use may then lead to participation in delinquent behaviors (W. A. Mason & Windle, 2002). Further, the association between delinquency and substance use may be dose-dependent, as longitudinal studies examining trajectories of both delinquency and substance use across adolescence have suggested that trajectories of increasing delinquency are predictive of trajectories of increasing substance use (Brook, Lee, Finch, et al., 2013; Lynne-Landsman, Graber, Nichols, & Botvin, 2011).

However, there may be some caveats regarding the extent to which delinquency is associated with substance use. For instance, there may be gender differences in the relationship between delinquency and later substance use, as some studies have suggested that delinquency is associated with substance use for boys but not for girls (W. A. Mason, Hitchings, & Spoth, 2007; W. A. Mason & Windle, 2002). The discrepancies in these findings may reflect socialized gender differences where boys are taught to engage in externalizing behaviors while girls are taught to primarily internalize their emotions. Lastly, while there is robust evidence for associations of delinquency with general substance use composites (Nebbitt, Lombe, Yu, Vaughn, & Stokes, 2012) and alcohol use (Hoyland et al., 2017; Hunter et al., 2014), results are mixed for marijuana use, with at least one study suggesting that delinquency was not associated with marijuana use (Hunter et al., 2014) while another found evidence for a relationship (Brook, Lee, Finch, et al., 2013). The suggestion that delinquency may be a substance-specific risk factor is inconsistent with PBT; as such, additional research is needed to examine the extent to which the association between delinquency and substance use is conditioned on the presence of other risk and/or protective factors.

Exposure to violence. Exposure to violence has been associated with substance use, violence, and an increased variety in the number of substances used (Fagan, Wright, & Pinchevsky, 2014). Exposure to violence is sometimes segmented into *indirect exposure* (also referred to as *vicarious victimization*), reflecting the individual witnessing someone else being the victim of a violent act (e.g., being shot at, chased, attacked with a weapon, assaulted, mugged), and *direct exposure* to violence, encompassing violent acts committed towards the adolescent's self. Both direct and indirect exposure to violence have been associated with increased substance use (Fagan, Wright, & Pinchevsky, 2015; Pinchevsky, Fagan, & Wright, 2014; Pinchevsky, Wright, & Fagan, 2013). Moreover, it appears that

witnessing violence (i.e., indirect exposure to violence) is associated with increased substance use even after direct trauma exposure has been accounted for (Zinzow et al., 2009); thus, these discrete forms of exposure to violence may account for both shared and unique proportions of the variability in substance use.

General Strain Theory (GST) suggests that exposure to violence may act as a stressor which leads adolescents to engage in maladaptive behaviors (such as substance use) as a means of coping (Agnew, 1992, 2001, 2006). GST proposes that individuals may develop anger or other negative emotions as a result of negative experiences with others in which the individual feels his or her goal seeking has been blocked, positive stimuli has been removed, or negative stimuli has been presented; in turn, this anger may be expressed as delinquency or substance use in an attempt to resolve or mitigate such negative emotions (Agnew, 1992). Although GST has roots as a theory of criminology, GST is frequently invoked in research on low-income African Americans given that African Americans are routinely subject to unequal treatment and live in poverty, both of which are contextual stressors that may be associated with deviant behavior (Copeland-Linder et al., 2011). A study of low-income African American adolescents in Flint, Michigan who were followed to early adulthood found that although exposure to violence was not associated with initial levels of substance use, violence exposure was associated with faster rates of change in substance use frequency across time (Roehler, Heinze, Stoddard, Bauermeister, & Zimmerman, 2017). The same study found that only two instances of exposure to violence were needed to predict an increase in rates of substance use accumulation, suggesting that individuals who have been exposed to violence may use drugs and alcohol more frequently as they encounter more instances of violence in order to cope with the stress related to this exposure (Roehler et al., 2017). These effects may be more salient for African Americans because of additional contextual factors that add increased stress to daily life (e.g., racial discrimination, poverty, etc.; Copeland-Linder et al., 2011).

Indeed, empirical research suggests that individuals are likely to use substances in response to exposure to violence, although the effects of exposure to violence on substance use appear to differ across substances. Indirect exposure to violence was associated with increased alcohol use within the first year following the experience, but the effects were no longer significant following the first year (Miller, Fagan, & Wright, 2014; Pinchevsky et al., 2014). Other studies have found relationships between indirect exposure to violence and alcohol use (R. Lee, 2012; K. W. Taylor & Kliewer, 2006). Prior research also suggests that marijuana use increases following both indirect and direct exposure to violence, with those experiencing multiple forms and multiple instances of violence exposure having the highest levels of marijuana use (Fagan et al., 2015; Pinchevsky et al., 2013; E. M. Wright, Fagan, & Pinchevsky, 2013). Similar findings have been suggested from research using broad indicators of substance use, frequently using composite measures of alcohol, tobacco, and marijuana use (Copeland-Linder et al., 2011; Fagan et al., 2014; Pinchevsky et al., 2014; Roehler et al., 2017; Zinzow et al., 2009).

Peer pressure. Peer influence on substance use may become an important risk factor for the development of substance use during adolescence, specifically because adolescents begin to spend more time with their peers and less time with their parents during adolescence (S. E. Goldstein, Davis-Kean, & Eccles, 2005; Simons-Morton, Haynie, Crump, Eitel, & Saylor, 2001). Numerous studies have suggested that peer pressure is a risk factor for substance use (Geyer, Roux, & Hall, 2015; Iwamoto & Smiler, 2013; Studer et al., 2016). Indeed, those whose peers use drugs, cigarettes, or alcohol, or those whose peers approve of alcohol or drug use have greater intentions to smoke and drink alcohol (Trucco, Colder, Bowker, & Wieczorek, 2011). Further, those who report peer pressure to use substances also report increased intentions to use alcohol or tobacco in the future (Scull, Kupersmidt, Parker, Elmore, & Benson, 2010). The increased intentions to use substances

stemming from peer pressure may be indicative of a cognitive shift regarding substance use after experiencing pressure from peers.

Research has also investigated the way that peers exert influence on substance use; that is, some peers may present direct pressure to use substances (e.g., offering an alcoholic beverage or cigarette, explicitly encouraging others to use substances), while others may indirectly encourage the use of substances (e.g., use of drugs in peers' presence). As would be expected, direct peer pressure was associated with increased likelihood of smoking and drinking alcohol (Simons-Morton et al., 2001). Interestingly, however, adolescents whose peers appeared indifferent to substance use were more likely to use substances, while only those whose peers actively disapproved of substance use were less likely to use substances (M. J. Mason, Mennis, Linker, Bares, & Zaharakis, 2014). As such, an adolescents' likelihood for substance use may be sensitive to their peers' encouragement of substance use.

The literature on peer pressure as a predictor for substance use is complicated because of the extent to which peer pressure for substance use may be confounded with other factors. Affiliation with deviant peers is frequently associated with substance use and experiencing peer pressure to use drugs or alcohol may be another mechanism through which deviant peers are associated with substance use outcomes (T. T. Clark, Belgrave, & Nasim, 2008; Patrick & Schulenberg, 2013; D. R. Wright & Fitzpatrick, 2004). Further, risky peer norms are associated with increased likelihood of substance use (Marotta & Voisin, 2017), which may manifest in perceived peer pressure to use drugs or alcohol. Broad contextual influences appear to play a minimal role in the extent to which peer pressure is associated with substance use, as there appears to be no difference between white and African American youths in how peer pressure is associated with substance use (Abbey, Jacques, Hayman, & Sobeck, 2006). Research that focuses specifically on peer pressure, and the specific aspects of peer pressure itself that are most related to substance use, may provide clarity on how peer pressure directly influences one's likelihood of substance use.

In an attempt to understand the complex relationship between peer pressure and substance use, some researchers have disaggregated peer pressure into a number of component parts to examine the effects of various types of peer pressure on likelihood of using alcohol or drugs. Clasen and Brown (1985) theorized five discrete dimensions of peer pressure, including peer involvement, peer conformity, involvement in school, involvement with family, and misconduct. While it is possible that some dimensions of peer pressure may yield protective effects (i.e., involvement in school or involvement with family both capture an individuals' engagement with prosocial institutions that may insulate against deviant behavior), other dimensions are more likely to promote maladaptive behaviors. For example, peer pressure for misconduct specifically refers to pressure to engage in minor delinquency, substance use, and risky sexual behavior, among other behaviors deemed unconventional during adolescence (Clasen & Brown, 1985). Studies that have examined the effects of peer pressure on misconduct specifically have identified a positive relationship with alcohol use, risky drinking, and smoking (Studer et al., 2014, 2016). The finding that peer pressure to engage in any type of unconventional behavior is predictive of substance use provides evidence that future studies should include indicators of peer pressure for multiple kinds of maladaptive and/or socially unacceptable behaviors when examining the effects of peer pressure on substance use, as we do in the current study.

Traumatic stress. A wide literature exists to suggest that those who experience more stressors have an increased likelihood of drinking alcohol and/or developing alcohol-related problems (Casement, Shaw, Sitnick, Musselman, & Forbes, 2015), but stressors may take on a wide range of experiences, from daily hassles to traumatic events. While many kinds of stress have been associated with increased substance use, traumatic stress specifically (as opposed to general life stress) was associated with increased alcohol use among a college student sample (Broman, 2005). Traumatic stress reflects a number of emotions that may arise after experiences of adversity, maltreatment, or violence occurring

in an individuals' psychosocial context (Hooper et al., 2015, p.356). Following the experience of a traumatic event such as death of a parent, witnessing violence, or experiencing childhood sexual abuse, some individuals may experience symptoms of posttraumatic stress disorder (PTSD), but do not necessarily meet the critical threshold for receiving a diagnosis of PTSD or acute stress disorder, the temporal precursor to PTSD (American Psychiatric Association, 2013). These distressing experiences are classified as traumatic stress, which refers to disturbances in emotional or behavioral functioning following a traumatic experience; common symptoms of traumatic stress include experiencing severe emotional distress (e.g., heightened anxiety, dissociation, detachment), re-experiencing the event, emotional numbing, and avoidance of the traumatic experience (Gerson & Rappaport, 2013, p.138). Given the overlap between traumatic stress and PTSD, the following discussion encompasses studies that examine both traumatic stress and symptoms (but not diagnoses) of PTSD.

Empirical evidence suggests that those with elevated levels of traumatic stress or increasing number of traumatic stress symptoms may be more likely to engage in substance use. Prior research has associated symptoms of PTSD with increased likelihood of using drugs (L. Khoury, Tang, Bradley, Cubells, & Ressler, 2010). A study of adolescents in the child welfare system suggested that anger and dissociation, two specific symptoms of traumatic stress, were associated with increased likelihood of substance use and substance-related problems (A. L. Goldstein et al., 2011). As such, individuals may have an increased likelihood for substance use after exhibiting traumatic stress symptoms, even in the absence of a PTSD diagnosis. These correlational results suggest that substance use may be common among those suffering with symptoms of traumatic stress, but do not give insight into a mechanism through which these relationships may operate.

A frequent explanation for the co-occurrence of traumatic stress symptoms and substance use has been the self-medication hypothesis, wherein individuals are likely to use alcohol or other drugs to alleviate or cope with negative emotionality (Khantzian, 1997).

In line with the self-medication hypothesis, a nationally representative sample of Americans indicated that they are aware of their use of alcohol as a technique to self-medicate against PTSD-like symptoms (Leeies, Pagura, Sareen, & Bolton, 2010). Symptoms of traumatic stress appear to be specifically associated with self-medication motives and not with other motives, as demonstrated by a study finding that an increased number of post-traumatic stress symptoms over the prior two weeks was associated with increased self-medication motives for marijuana use, but were unrelated to social, enhancement, or conformity motives (Bujarski et al., 2012). Further, specific traumatic stress symptoms such as re-experiencing the traumatic event and hyperarousal were specifically associated with drinking to cope with negative emotions to a greater extent than these symptoms were associated with any other drinking motives (Dixon, Leen-Feldner, Ham, Feldner, & Lewis, 2009). Additionally, knowingly self-medicating against traumatic stress symptoms was associated with increased risk of a number of other adverse outcomes, including dysthymia, lower quality of life, and suicide attempts, indicating that the effects of traumatic stress are far-reaching (Leeies et al., 2010). As such, the experience of traumatic stress may place individuals at an increased risk for substance use as alcohol or drug use may be an opportunity to mitigate traumatic stress symptoms.

Protective Factors

As described previously, protective factors refer to experiences, traits, or contexts that mitigate the effects of risk factors on likelihood of engaging in substance use (Hawkins et al., 1992; Jessor, 1991; Nash & Bowen, 2002). Prevention scientists have placed special emphasis on identifying factors that diminish one's likelihood for substance use. Although risk factors are often difficult, if not impossible to modify (e.g., genetic risk), protective factors are often subject to modification under the appropriate environmental circumstances (Glynn, 1981). Thus, modifiable protective factors may be especially important in the prevention of adolescent substance use.

Similar to research on risk factors for substance use, much research has focused on isolating factors associated with decreased substance use. Below we describe prior research on the influence of specific protective factors that are of interest in the proposed project.

Neighborhood connectedness. Neighborhood connectedness, also referred to as *sense of community*, *neighborhood attachment*, *community cohesion*, and *neighborhood belonging* refers to a set of behaviors and attitudes that reflect one's perception of one's community and one's role within that community (Glynn, 1981). Neighborhood connectedness may encompass multiple aspects of community life, including perception of social support in the community, neighborhood traditions or culture, perceived safety of the community, conflict within the neighborhood, and the ability of the neighborhood community to cope with adversity (Glynn, 1981). Prior research on neighborhood impact on substance use has suggested that contextual factors (e.g., neighborhood connectedness, school connectedness, association with drug-using peers) have both direct and indirect influences on substance use; specifically, those who are strongly connected to their communities are less likely to use drugs or alcohol than are adolescents who are not as attached to their communities (Su & Supple, 2014).

Other research examining the effects of neighborhood connectedness on substance use has identified inverse relationships between increased levels of neighborhood connectedness and lower levels of substance use (Mayberry, Espelage, & Koenig, 2009). Specifically, one study found that feeling a sense of community (relative to not) was associated with a 55% decrease in the odds of drug use one year later among a nationally representative sample of adolescents (Yan, 2013). Another study found somewhat attenuated effects, reporting that increases in neighborhood cohesion (on a five-point scale) were associated with 25% decreased odds of cigarette use, 16% lower odds of marijuana use, and 17%

lower odds of e-cigarette use, and no association with alcohol use (Shih et al., 2017). Overall, there appears to be an inverse relationship between neighborhood connectedness and substance use.

Some research has proposed mechanisms through which neighborhood connectedness may reduce the likelihood of substance use, although there is no consensus on how this protective effect may function. It is possible that neighborhood cohesion mediates the effect of neighborhood poverty and drug availability on adolescent substance use by decreasing perceptions of neighborhood problems with drugs and alcohol (Duncan, Duncan, & Strycker, 2002). Another proposed mechanism is that neighborhood belongingness increases the availability of adult social support, thereby decreasing one's risk of substance use because adolescents feel safe as they begin spending time in their neighborhood public spaces without parental supervision (Brooks, Magnusson, Spencer, & Morgan, 2012). Given the lack of consensus on how neighborhood connectedness may influence substance use, it is of interest to investigate how a sense of community co-occurs with other protective or etiological factors to reduce substance use.

However, not all research suggests that neighborhood connectedness is necessarily protective against the development of substance use. For instance, one study found that neighborhood attachment was not associated with either alcohol or marijuana use among African American adolescents (T. T. Clark et al., 2008). This may indicate that there are certain subgroups of African American adolescents for whom feeling attached to one's neighborhood is not protective against substance use, and the extent to which this is true may depend on other aspects of their psychosocial context such as urban/rural community affiliation (T. T. Clark, Nguyen, & Belgrave, 2011). Additionally, some research has conceptualized lack of neighborhood connectedness as a risk factor for psychopathology, including alcohol use (Monahan, Oesterle, Rhew, & Hawkins, 2014). Given that a lack of neighborhood connectedness may function as a risk factor for increased substance use,

and that the extent to which neighborhood connectedness protects against the onset of substance use may be dependent on other aspects of one's psychosocial context, neighborhood connectedness is important to examine as a contextual factor that may interact with other risk and protective factors to predict unique substance use outcomes.

Parental knowledge. Parental knowledge refers to information that parents obtain about their children's whereabouts, who they are with and what they are doing, usually as a result of parental monitoring (Racz & McMahon, 2011; Stattin & Kerr, 2000).[1] Prior research has found that higher levels of parental knowledge is associated with decreased risk for substance use among adolescents (Piko & Kovács, 2010). Given that African American families tend to exhibit more stringent monitoring practices that may result in increased parental knowledge, this is an important protective factor to examine among low-income African American youths (Jarrett, 1995). While some have conceptualized little or no parental knowledge as a risk factor for substance use (Blustein et al., 2015; Latendresse, Ye, Chung, Hipwell, & Sartor, 2017), we choose to conceptualize the presence of parental knowledge as a protective factor because of its previously mentioned cultural significance in African American families (Jarrett, 1995).

Indeed, parental knowledge has been associated with decreased substance use among African American adolescents (Tebes et al., 2011; Tobler & Komro, 2010; Udell, Hotton, Emerson, & Donenberg, 2017). Further, longitudinal increases in parental knowledge were associated with longitudinal decreases in substance use behaviors (Tebes et al., 2011). One way in which parental knowledge may prevent the escalation of substance use across adolescence may be by preventing substance use initiation among adolescents in the first place;

[1] The term *parental monitoring* is used more frequently in the literature but is frequently mistaken for *parental knowledge*. *Parental monitoring* refers to the process and activities through which parents obtain knowledge about their children, while *parental knowledge* measures the end product, or the extent to which parents know about their child's activities and whereabouts. Much of the cited literature uses the term *parental monitoring* but utilize items that actually refer to *parental knowledge*. As such, we use the term *parental knowledge* to retain the validity of the construct represented in this study, but reference literature using both terms.

adolescents who reported higher levels of parental knowledge at age 11 were less likely to have initiated cannabis use when re-assessed at age 17 (Bohnert, Anthony, & Breslau, 2012). Regardless of mechanism, much research has suggested an association between increased levels of parental knowledge and decreased likelihood of substance use among adolescents.

While the association between parental knowledge and substance use appears to be robust when examining composite indicators of substance use, some results are mixed with respect to the effect of parental knowledge on individual substances. For instance, while parental knowledge was associated with increased risk of alcohol dependence one year later, parental knowledge was unrelated to risk of cannabis dependence across the same time frame (Kaynak et al., 2013). In contrast, a meta-analysis suggested that parental knowledge is consistently associated with decreases in marijuana use (but not necessarily marijuana dependence), finding an average association of -.21 between parental knowledge and marijuana use (Lac & Crano, 2009). An additional study found that although high levels of parental knowledge were associated with the decreased use of a number of illegal substances (e.g., cocaine, LSD, etc.) and alcohol across a one-year period, similarly high levels of parental knowledge were not associated with cigarette use across the same time frame (H. K. Clark, Shamblen, Ringwalt, & Hanley, 2012). While some research suggests that parental knowledge is associated with decreased frequency of alcohol consumption (Kelly, Becker, & Spirito, 2017), other research finds that parental knowledge is associated with decreased frequency of most substance use with the exception of alcohol use, suggesting that parental knowledge is not effective in either preventing nor decreasing alcohol use (Tebes et al., 2011). These discrepancies provide evidence that future research should continue to examine the exact conditions under, and the specific substances for which parental knowledge functions as a protective factor.

Religiosity. Much prior research has suggested that religiosity is a protective factor against substance use (Edlund et al., 2010; Kendler et al., 2003; Steinman & Zimmerman, 2004). Religiosity may be operationalized in many ways, including attendance at religious services (Vidourek & King, 2010), importance of religion (Edlund et al., 2010), obtaining guidance from a religious or spiritual leader (Ellison, Musick, & Henderson, 2008), a composite indicator comprising multiple dimensions of religiosity (Patrick & Schulenberg, 2013), or a categorical variable wherein religiosity represents a pattern of multiple indicators of religiosity (Hodge, Andereck, & Montoya, 2007; Hoyland et al., 2017; Salas-Wright, Vaughn, Hodge, & Perron, 2012). Regardless of the operationalization of religiosity, most prior research has found inverse relationships between religiosity and substance use (Nonnemaker, McNeely, & Blum, 2003).

There are a number of mechanisms through which religiosity may be associated with decreased substance use. First, religiosity has been linked to increased (or an ability to quickly replenish depleted) self-control, indicating that religiosity may increase one's ability to restrain impulses or to regulate behavior such that it is in line with a predetermined standard; this self-control may afford resources to help avoid alcohol and other drug use (Desmond, Ulmer, & Bader, 2013; DeWall et al., 2014; Pirutinsky, 2014). Further, being involved with a religious group may provide social alternatives to drug use. For instance, spending time with prosocial peers through religiously-affiliated activities may take away from time to be spent engaging with deviant peers who may be more likely to use substances; this may decrease likelihood for substance use as affiliation with deviant peers is associated with increased likelihood for substance use (Fowler, Ahmed, Tompsett, Jozefowicz-Simbeni, & Toro, 2008). Religiosity may have influences on adolescents both internally (i.e., by increasing their own self-control) and externally (i.e., by modifying their social context) such that religiosity may buffer against the effects of a number of substance use risk factors.

The effects of religiosity may extend beyond the individual and may be heavily influenced by the religious milieu within one's family. Adolescents' religious service attendance is strongly influenced by their family members' attendance, as increased parental attendance at religious services has been shown to be associated with decreased levels of drug and alcohol use among offspring (Farmer & Brown, 2013). Familial religiosity may also be associated with decreased substance use, at least in part by influencing the extent to which parents are aware of their children's activities and whereabouts (Kim-Spoon, Farley, Holmes, Longo, & McCullough, 2013).

Religiosity is especially important to consider when examining substance use among African American adolescents given the familial and historical context of religiosity in the lives of African Americans (J. M. Wallace, Brown, Bachman, & LaVeist, 2003). Historically, African American families have been, on average, more religious than white families in the United States (R. J. Taylor, Chatters, Jayakody, & Levin, 1996), and this has been supported by more recent empirical work suggesting that African American adolescents and emerging adults report higher levels of religiosity than do their white peers (Fowler et al., 2008). Increased religiosity among African Americans has been posited to account, in part, for discrepancies in substance use between African American and white adolescents (J. M. Wallace et al., 2003; Watt & Rogers, 2007). Religiosity in general, and specifically attendance at religious services and obtaining guidance from religious figures, has also been shown to buffer the effects of racial discrimination on alcohol use among African American adolescents (Bierman, 2006; Ellison et al., 2008; Parenteau et al., 2017). Given that religiosity appears to buffer the effects of discrimination, a pervasive social force influencing the lives of all African American adolescents, it is warranted for exploration as a protective factor against a number of other risk factors among African Americans.

Self-worth. Self-worth, also referred to as *self-esteem*, has been shown to be correlated with decreased levels of drug and alcohol use among adolescents (Copeland-Linder et

al., 2011; Patrick & Schulenberg, 2013). Self-worth initially protects against the initiation into substance use, as one study found that adolescents who had higher levels of self-worth had reduced odds of initiating into alcohol, marijuana, and tobacco use (Richardson, Kwon, & Ratner, 2013). Even after initiating into substance use, self-worth has been shown to have differential effects on various substances. For example, although self-worth was inversely associated with cigarette and alcohol use over the past month, it was not associated with past month marijuana use (Schwinn, Schinke, Hopkins, & Thom, 2016). However, another study found that self-worth was predictive of binge drinking, marijuana use, and cocaine use among a nationally representative sample of 15-year old adolescents (C. G. Lee, Seo, Torabi, Lohrmann, & Song, 2018). As such, research should continue to examine the extent to which self-worth is uniquely predictive of various substances.

Self-worth may be a context-dependent protective factor; that is, the extent to which self-worth is protective against substance use may depend on other characteristics of the individual and aspects of his or her environment (Zeigler-Hill, Dahlen, & Madson, 2017). For instance, self-worth is differentially predictive of substance use for males and females, with confusing results; interestingly, research has shown that self-worth protects against the development of substance use in girls, but not in boys (Wheeler, 2010), and conversely that increased levels of self-worth are associated with decreased alcohol use among boys, but not among girls (Huurre et al., 2010). Further, self-worth may be multidimensional and unique to a particular situation or context, and the extent to which an adolescent has self-worth in a particular domain may be associated with a differential likelihood of using substances depending on whether one's self-worth (or lack thereof) in a particular domain has been made salient (Lockhart et al., 2017). In order to understand the extent to which self-worth influences later substance use, it is important to examine self-worth in the context of other demographic characteristics, risk factors, and protective factors.

Stemming from research highlighting the contextual nature of self-esteem, other research has focused on the reasons self-worth may be associated with decreased substance

use. It may be that individuals with high levels of global self-worth are able to develop healthy coping strategies to accompany feelings of negative affect or traumatic experiences, while those with low levels of self-worth instead adopt maladaptive coping strategies that may include use of alcohol or other drugs (Tomaka, Morales-Monks, & Shamaley, 2013). In general, individuals with high levels of self-esteem are better able to cope with receiving negative feedback; that is, those who have high levels of self-worth are less likely to experience negative affect after receiving criticism or experiencing an event that may induce negative emotions, indicating resilience in the face of negative feedback (J. D. Brown, 2010). This may suggest that without high levels of self-worth, individuals may be more likely to engage in substance use in order to help reduce feelings of negative affect (e.g., self-medicate; Khantzian, 1997; Zeigler-Hill et al., 2017). As such, while self-worth may only have a small direct influence on substance use, it may provide its greatest contribution by acting as a buffer against the effects of many other risk factors to prevent substance use (Boden, Fergusson, & Horwood, 2008). The collective results of these studies demonstrate that the effects of self-esteem on substance use should be considered within the context of other risk and protective factors unique to an individual.

Interactions Across Risk and Protective Factors

Risk and protective factors as interacting processes. Much prior research has been devoted to classifying and describing the extent to which various risk and protective factors are indicative of later substance use. These studies are useful in specifying important indicators that may signal an increased likelihood of later substance use. However, identifying individual factors that are associated with increased (or decreased) likelihood of substance use is not sufficient to distinguish adolescents who are most likely to use drugs. Research that analyzes the effects of a single (or multiple) risk or protective factor(s) in predicting substance use makes an implicit assumption that an increase (or decrease) in some factor will be associated with a certain amount of substance use *even after controlling for*,

or *holding constant*, other factors across individuals. However, holding all possible confounding variables constant is impossible with respect to any one individuals' environment because the constructs of risk and protection are not independent (Bogat, Eye, & Bergman, 2016). For example, if one's neighborhood attachment changes such that they are spending additional time in public spaces, we may see simultaneous increases in peer influence and decreases in parental influence; that is, we could not practically examine the effects of a change in one variable, because this change would inherently lead to changes in other aspects of one's environment. Rather, we should focus on conceptualizing the broad, unique, holistic effects of one's entire psychosocial context in predicting outcomes.

Research studying the effects of numerous risk and protective factors on a wide range of problem behaviors in adolescence (including substance use) has found that the unique interplay among multiple risk and/or protective factors, not necessarily cumulative levels of exposure to risk and/or protective factors, was most influential in predicting likelihood of engaging in problem behavior (Jessor, Van Den Bos, Vanderryn, Costa, & Turbin, 1995). From this, we can infer that risk and protective factors are not only discrete traits or contexts that are predictive of some outcome, but they are products of an individuals' entire psychosocial context. This is important because certain factors may not be directly associated with increased or decreased likelihood of substance use, but those factors may be indicators of more generalized risk that is only identified when examined in the context of other potential risk and protective factors (Lloyd, 1998). In essence, risk and protective factors are not discrete factors that operate in isolation to predict substance use, but rather they function dynamically within conditional *processes* where a trait may be associated with increased substance use in one context but may be protective against substance use in another (Rutter, 1987). It is on this premise that we base our theoretical conceptualization of risk and protective factors.

A keynote address by Rutter (1987) discusses the genesis of theoretical conceptualizations of risk and protective factors as interacting processes. Research had already begun

to focus on the extent to which protective factors may insulate individuals from experiencing the negative consequences associated with risk factors, given that buffering or mitigating (i.e., protective) contexts are frequently more malleable than are risk factors. In identifying the extent to which one is vulnerable to negative outcomes (i.e., the extent to which one is at risk), Rutter (1987) notes that some individuals respond more positively to a stressor than do other individuals. Thus, there must be other factors that differentiate individuals who will have either positive or negative outcomes as a result of exposure to any given risk.

However, the same individuals may not be resilient to all sources of vulnerability. That is, one who had a normative outcome as a result of exposure to one risk factor may not have an equally normative outcome as a result of exposure to another; if the source of vulnerability or risk would shift, one's exposure or access to potential protective factors may also shift (Luthar, Doernberger, & Zigler, 1993). This suggests that protection or resilience is not due to a single insulating factor that some individuals have and others lack, but rather to a dynamic relationship between exposure to multiple sources of vulnerability and protection (Lloyd, 1998). Viewed from this perspective, "a risk or protective factor is a 'contributive component in an interactive system that leads to emergent factors that in turn interact and evolve' " (Glantz, 1992; Lloyd, 1998, p. 227). Essentially, "the search is not for broadly defined protective factors but, rather, for the developmental situational mechanisms involved in protective processes" (Rutter, 1987, p. 317).

To address the contextual and interactive nature of risk and protective processes, additional research has emphasized multiple interacting levels on which these processes operate. All behavior occurs in a specific time and place, and all developmental processes are influenced by the culture, norms, and standards of behavior expected in that time and place. These contextual factors may directly influence the extent to which one is exposed to risk factors for substance use by determining legislation surrounding substance use and the availability of drugs or alcohol (Hawkins et al., 1992). Similarly, those who grow up

in impoverished or disorganized neighborhoods may be exposed to an increased number of risk factors, demonstrating the effects of neighborhood context on generalized vulnerability to substance use (Hawkins et al., 1992; Sloboda et al., 2012; S. A. Wallace, Neilands, & Sanders Phillips, 2017). Contextual factors may be further decomposed into various types of environments such as the school environment, family environment, and neighborhood environment, each of which exert individual and interactive influences that are associated with increases in either vulnerability for or protection against negative outcomes (Sloboda et al., 2012; E. M. Wright et al., 2013). Finally, personality systems including temperament, and more recently, genetics and neurobiology independently and interactively influence one's exposure to potential risk factors (Hawkins et al., 1992; Jessor et al., 1995; Sloboda et al., 2012).

Risk and protective factors at certain levels of an individuals' environment may be more strongly associated with substance use among individuals who demonstrate other contextual factors. For example, individual (e.g., personal norms) and peer (e.g., peer truancy) risk and protective factors were more strongly associated with past 30-day alcohol and marijuana use among urban African American adolescents, while family (e.g., family substance use) and community (e.g., availability of drugs) risk factors were more strongly associated with past 30-day alcohol and marijuana use for rural African American adolescents (T. T. Clark et al., 2011). Other research has found that low connectedness to one's family was associated with increased risk for substance use among those with low levels of school connectedness, but this relationship was not present among those who had comparatively high levels of school connectedness (Brooks et al., 2012). The numerous interacting levels of an individuals' biopsychosocial context suggests that an individual's development is inextricably dependent upon multiple sources, and any change in one aspect of the environment may have cascading effects that may influence other developmental processes in turn (Masten & Cicchetti, 2010).

Conceptualizing risk and protective factors as etiological factors. The discussion of risk and protective factors has made an implicit assumption that risk and protective factors are bipolar dimensions wherein the presence of one factor may imply protection against some negative outcome while the absence of the same factor implies increased likelihood of encountering that negative outcome. Given the above discussion about the extent to which risk and protective factors should be considered interacting processes, this conceptualization is misleading. Rutter (1987) provides an important distinction: While there are two discrete dimensions of risk and protection, a decrease on the continuum of one does not necessarily equate to an increase on the other. For instance, if an individual has a reduction in exposure to risk (e.g., moving out of a poor neighborhood), it does not mean that individual has been exposed to new protective factors. Rather, we are just removing the vulnerability of the exposure to the risk factor, which may, but *does not necessarily*, provide an increase in exposure to protective factors. The most important reason to highlight a particular risk or protective process is to highlight the portion of functioning that we infer is the mechanism driving that specific developmental process (Rutter, 1987). As such, it is important to retain distinct conceptualizations of risk and protective processes, but what is considered risk as opposed to protection should reflect the overarching theory.

This distinction of risk and protective factors as discrete processes permits a novel conceptualization of the entire combination of risk and protective factors. While risk and protective factors inhabit two separate dimensions, these dimensions are strongly related to one another. Empirical investigations of the extent to which risk and protective factors are indicative of later substance use have identified numerous instances where specific combinations of risk and protective factors may indicate differing levels of risk depending on the individual composition of risk and protective factors. For instance, although self-esteem is protective against substance use for most adolescents, self-esteem is not necessarily protective against substance use among adolescents who are either members of a gang or are involved with a gang (Yoder, Whitbeck, & Hoyt, 2003).

In order to determine the extent to which one is at risk for substance use, we must consider the individual's entire psychosocial context, including combinations of risk and protective factors that are unique to an individual's self and/or environment, the interplay of which may be indicative of one's likelihood of engaging in substance use. As such, we have used the term *etiological factors* to refer to unique combinations of risk and protective factors present across an individual's psychosocial context that may indicate increased or decreased likelihood of later substance use. When discussing certain variables as *risk factors* or as *protective factors*, we make an implicit assumption that the particular variable will exert some type of negative or positive influence on a later outcome. Alternatively, when discussing certain variables as *etiological factors*, we make a different assumption, namely that the variable is part of an individuals' psychosocial context but may exert either positive or negative effects depending on the composition of other variables in an individuals' environment.

Empirical evidence for interactions across etiological factors. The empirical evidence referenced above discusses the extent to which each risk and protective factor individually is predictive of increased and decreased substance use, respectively. However, in line with our objective of conceptualizing risk and protective factors as interacting processes associated with later substance use, it is of interest to examine moderating and interactive influences among combinations of etiological factors. As such, the following paragraphs provide examples of prior literature that has examined interactions among some of these etiological factors.

While we were unable to locate research that provided examples of every combination of etiological factors addressed in our earlier literature review, we hope that this limited summary of research examining associations between at least two risk and protective factors demonstrates the importance of investigating risk and protective processes

together as etiological factors. We also note that these examples describe statistical interactions between two or more etiological factors which are unique from dynamic interactions (Bergman, Magnusson, & Khouri, 2003). However, we provide these examples to support our argument that the extent to which certain etiological factors influence behavior may be conditioned on the presence of other risk and protective factors within the individuals' psychosocial context.

Parental knowledge, although consistently demonstrating a negative association with substance use when examined independently, has also been shown to buffer the impact of certain risk factors on later adolescent substance use. For instance, parental knowledge has been shown to mitigate the effects of adolescent depressed mood on substance use (Kelly et al., 2017), as well as the effects of affiliating with deviant peers on using drugs in the presence of friends, and on individual substance use (Kiesner, Poulin, & Dishion, 2010; Simons-Morton et al., 2001). However, other research has shown no moderating effects of parental knowledge on the influence of exposure to violence on substance use (R. Lee, 2012; K. W. Taylor & Kliewer, 2006). Interestingly, urban youths who experienced indirect victimization (i.e., were exposed to community violence) were more likely to drink alcohol immediately following the victimization experience, but those who also had high levels of prior parental support were even more likely to drink alcohol compared to those with lower levels of parental support (Miller et al., 2014). It may be that individuals who are exposed to protective factors (e.g., parental knowledge) before a severe event occurs experience those severe events (such as victimization) more strongly. Because the initial presence of one etiological factor may modify exposure to later etiological factors, the initial presence of factors that may demonstrate a protective influence should be accounted for in studies of risk factors to gain a truly holistic account of an individual's unique substance use etiology. The mixed findings on the effectiveness of parental knowledge as a protective

factor evidences why we should aim to examine etiological factors that function as a protective factor in one context, but as a risk factor or a neutral factor (i.e., unassociated with the outcome) in another.

African American adolescents living in inner-city neighborhoods are at a high risk of being exposed to violence in their neighborhoods. Almost half of adolescents living in a low-income inner-city neighborhood reported watching someone get shot or stabbed, and nearly a quarter reported watching someone get killed; the same study found that the rate of violence exposure may be as high as 81% among African American adolescents from inner-city neighborhoods (Schubiner, Scott, & Tzelepis, 1993). Given that exposure to violence is associated with increased risk for substance use (Fagan et al., 2015; Pinchevsky et al., 2014, 2013; Zinzow et al., 2009), research has focused on identifying factors that may mitigate the deleterious effects of exposure to violence on substance use. Measures of contextual stress, including exposure to violence, were predictive of substance use and aggressive behavior two years later among African American youths in the Baltimore metropolitan area, but high levels of self-worth buffered against these effects (Copeland-Linder et al., 2011). Additionally, religiosity may provide some protection against the effects of community violence exposure, as African Americans who experienced high levels of community violence, but also reported high levels of public religiousness (e.g., church attendance), reported lower levels of substance use compared to African Americans who reported high levels of community violence and low levels of public religiousness (Fowler et al., 2008). The extent to which these various protective factors are effective in buffering the effects of community violence on substance use highlight potential targets for modification via prevention and intervention programming.

Having a positive sense of community or connection to one's neighborhood appears to provide protection against certain risk factors for substance use. Neighborhood connectedness, generally conceptualized to be an overarching contextual factor, appears to

protect against the influence of some individual-level factors, as neighborhood connectedness buffered against the impact of low positive peer influence and affiliation with deviant peers on use of alcohol, cigarettes, and marijuana (Mayberry et al., 2009). Perceiving a strong sense of cohesion in one's neighborhood attenuated the effects of delinquency and witnessing community violence on substance use among African American adolescents living in public housing (Nebbitt et al., 2012). The relationship between exposure to violence and substance use was weaker among adolescents who also expressed high levels of neighborhood connectedness, relative to those who demonstrated low levels of neighborhood connectedness (Fagan et al., 2014). Additionally, protective factors may exert an additive influence on likelihood for substance use, as adolescents who had high levels of both self-worth and neighborhood connectedness were the least likely to use marijuana (Shih et al., 2017). It may be that having a sense of connection to one's neighborhood may increase community social support, providing social reinforcement for avoiding substance use (Brooks et al., 2012), or improve psychological outlook on life, encouraging goal setting and reducing the need for self-medication (S. A. Wallace et al., 2017). Overall, neighborhood connectedness may function as a protective factor against multiple risks for substance use.

Summary

We have outlined a rationale for assessing substance use etiology among a sample of low-income African American adolescents. Given that African American adults are more likely to experience alcohol-related consequences at similar levels of consumption to white Americans, members of this racial/ethnic group are at a unique risk for substance use (Zapolski et al., 2014). Although most research on the risks and consequences of substance use among African Americans has been conducted on adult samples, adolescence is considered a critical period in the development of substance use as those who initiate into substance use in adolescence are at an increased risk of later substance-related consequences (Grant

& Dawson, 1998; Marshall, 2014). As such, attending to potential risk and protective factors for substance use among African American adolescents may provide a contribution to the prevention literature about substance use risk particularly among a high-risk population (Kraemer et al., 1997; Pollard, Hawkins, & Arthur, 1999).

The above literature reflects a wide breadth of knowledge on the extent to which various risk and protective factors, as well as interactions among them, may influence adolescent substance use. Based on prior literature, delinquency, exposure to violence, traumatic stress, and peer pressure have all been associated with an increased likelihood of developing substance use, while parental knowledge, neighborhood connectedness, religiosity, and self-worth have been associated with decreased likelihood of substance use (Copeland-Linder et al., 2011; Fagan et al., 2014; L. Khoury et al., 2010; W. A. Mason, Hitchings, McMahon, & Spoth, 2007; Mayberry et al., 2009; Piko & Kovács, 2010; Steinman & Zimmerman, 2004; Studer et al., 2014, 2016). However, risk and protection are fluctuating constructs, as a single factor may exhibit risk in one context, but protection in another (Kraemer et al., 1997; Rutter, 1987). As such, we propose the term *etiological factor* to distinguish a variable that may be present in an individual's environment, but whose effects are dependent upon the unique combination of other factors present in an individual's environment.

In the following chapter we build upon the previously discussed empirical evidence by introducing a framework through which we intend to examine unique combinations of etiological factors and their associations to later trajectories of substance use. Following our discussion of an overarching framework to study etiological factors we present the specific aims of the current study. Overall, we aim to identify unique patterns of etiological factors for substance use and assess their relation to trajectories of alcohol, marijuana, and cigarette use across adolescence.

CHAPTER TWO

An Interactionist Approach to Substance Use Etiology

Overview

The previous chapter reviewed empirical literature that discussed the extent to which various etiological factors have been associated with an increased likelihood for substance use, and the importance of examining substance use etiology specifically among African American adolescents. As previously demonstrated, most research has examined the effects of individual predictors, or interactions between two or more predictors, on substance use among African American adolescents (T. T. Clark et al., 2008, 2011; S. A. Wallace et al., 2017). While such research has made important contributions to our understanding of substance use etiology, we may gain additional information about the extent to which multiple etiological factors co-act to collectively influence later substance use via the use of a person-centered approach to study unique patterns of etiological factors (Bogat et al., 2016; Magnusson, 1990, 1999, 2003). These discrete patterns of etiological factors may further help identify adolescents who have the greatest likelihood of later substance use (Lanza & Rhoades, 2013; Syvertsen, Cleveland, Gayles, Tibbits, & Faulk, 2010).

As part of a research agenda aimed at preventing the onset of problematic substance use, a number of theories have been proposed to provide a framework for the reasons that individuals, and especially those from impoverished communities, may engage in substance use. While there are many theoretical perspectives that may inform research on the development of substance use, there is no overarching theory that explicitly describes how these exact etiological factors may interactively contribute to adolescent substance use. As such, we approach the study of substance use etiology through a combination of two distinct perspectives. The first component of this perspective is the Social Development Model (Catalano & Hawkins, 1996; Catalano, Kosterman, Hawkins, Newcomb, & Abbott, 1996),

a general social developmental theory that focuses on interactions across risk and protective factors to predict later antisocial behavior. This theoretical framework is combined with a *holistic-interactionist* perspective (Magnusson, 1999) that highlights the importance of dynamic interplay across multiple interacting components of the psychosocial environment in shaping behavior and applies this systems perspective to the etiological factors described above. This *holistic-interactionist* approach to the study of development advocates the use of person-centered methods, which we will use in the current study to model etiology and behavior.

In this chapter, we complement the previous discussion of prior empirical work with a discussion of an overarching framework through which we may understand unique combinations of risk and protective factors. We begin by describing the theoretical framework for the current study, including the Social Development Model and holistic-interactionism. We then explore the person-centered approach wherein we conceptualize the individual as a unique whole and discuss the use of person-centered methods to examine the individual's holistic psychosocial context. We then outline the rationale for the current study and present the specific aims. The current study aims to characterize discrete prototypical patterns of etiological (i.e., risk and/or protective) factors for substance use among African American adolescents, and their associations with later substance use. Specifically, we aim to identify discrete patterns of the outlined etiological factors and examine the extent to which likelihood of membership in these discrete patterns may be predictive of substance use trajectories.

The Social Development Model

The Social Development Model (SDM) is a developmental model of antisocial and prosocial behavior across various epochs of development (Catalano & Hawkins, 1996; Catalano et al., 1996). Designed to incorporate a wide array of risk and protective processes throughout development, the model includes aspects of social learning theory (Bandura

& Walters, 1977), differential association theory (Sutherland, 1973), and control theory (Hirschi, 1969). Broadly, the model proposes that antisocial behavior develops under one of three conditions: (1) when there is little prosocial socialization, (2) when the individual believes there is something to be gained from antisocial behavior, or (3) when the bonded agents hold antisocial values (Catalano & Hawkins, 1996). A number of risk factors for antisocial behavior were explicitly identified by the model, including neighborhood disorganization and deprivation, poor family management or parental control, low socioeconomic status, problem behaviors, low educational achievement, and affiliation with deviant peers, among others (Catalano & Hawkins, 1996, pp. 152-153). Additionally, three broad categories of protective factors include (1) individual traits, (2) family warmth, cohesion, or bonding, and (3) external social supports that may provide a values system (Catalano & Hawkins, 1996, p. 153). Of particular interest to the current study, the model provides a theoretical framework through which risk and protective factors interact to predict later antisocial behavior, including both crime and drug use.

The model is founded on the assumption that adolescents learn antisocial or prosocial behavior patterns via multiple domains of influence within the psychosocial environment, positing that the effects of individual characteristics on later antisocial or prosocial behavior are mediated or moderated by family, school, peer, and neighborhood/community effects (Catalano & Hawkins, 1996; Catalano et al., 1996). Individuals develop attachments or bonds to various socializing agents in these aspects of the environment, and regulate their own behavior in light of norms or standards set by those bonded agents. Behaviors that are in line with the bonded agents' standards or norms are likely to be reinforced and repeated, where behaviors out of line with the bonded agents' norms or standards are likely to not be reinforced and repeated.

Socialization Processes in the Social Development Model

The SDM proposes that individuals engage in prosocial or antisocial behavior as a result of repeated interactions and involvement with socializing agents to whom they become bonded or attached, and are more likely to engage in behaviors that reflect norms or standards set by the bonded agents (Catalano & Hawkins, 1996; Catalano et al., 1996). Below, we outline the specific mechanism through which individuals become bonded to a socializing agent which then has direct implications for one's behavior. While the model has been described for antisocial and prosocial behavioral outcomes separately (Catalano & Hawkins, 1996; Catalano et al., 1996), we describe a general model that may be applied to the development of either antisocial or prosocial behavior. Individuals become bonded to a socializing agent through a multi-step socializing process as described in Catalano and Hawkins (1996) and Catalano et al. (1996).

First, individuals must perceive opportunities to engage in prosocial or antisocial behavior (Catalano & Hawkins, 1996; Catalano et al., 1996). The extent to which an individual perceives an opportunity to interact with other people (e.g., family members, peers, neighborhood members, etc.) or become involved with either prosocial or antisocial behavior directly influences the extent to which an individual actually engages with that potential bonded agent. Second, after perceiving an opportunity to interact with a potential socializing agent or become involved with a specific behavior, the extent to which an individual develops a bond to that agent depends on the quality of the interaction or involvement. As the behavioral change literature has shown, an individual's behavior is likely to change before they experience an attitude change (Bem, 1972; Festinger, 1964), so the theory assumes that the behavioral investment of relationship with the socializing agent is more likely to occur before individuals experience the attitude change of feeling connected to the socializing agent.

Third, interactions with the agent or behavior become reinforced (Catalano & Hawkins, 1996; Catalano et al., 1996). Simply interacting or becoming involved with a behavior or

agent will increase the likelihood that one will perceive interactions and involvements as rewarding, but the extent to which rewards are actually perceived will vary across individuals. That is, individuals with greater social and/or cognitive skills are more likely to perceive and adequately process rewards because they have higher levels of mental functioning that allow them to do so. As such, individuals with greater social and/or cognitive skills may be more likely to experience rewards through engagement in either antisocial or prosocial behavior. Further, the nature of these rewards may be either social (e.g., approval from potential bonding agents) or nonsocial (e.g., lack of reprimand from social or community structures). The extent to which a particular individual perceives and experiences a reward will be dependent upon what that particular individual finds rewarding; that is, different individuals will perceive certain rewards to be more or less rewarding than others. As such, there is an individual specificity to the nature of reinforced interactions with potential bonding agents or involvement with behavior.

Fourth, and finally, if the individual perceives these interactions and involvements to be rewarding, the individual develops an attachment or bond to those socializing agents and behaviors, and subsequently an internalization of the bonded agent's values (Catalano & Hawkins, 1996; Catalano et al., 1996). Behaviors that are congruent with norms and standards held by the bonded agent are more likely to be repeated (in line with the reinforcement given from the bonded agent), while those behaviors incongruent with these norms and standards are less likely to be repeated, leading to a general pattern of antisocial or prosocial behavior in line with the bonded agent's norms and standards. As the bonding process progresses, the individual will consider the bonding agent's norms and standards and will be less likely to engage in behaviors unaligned with those conventions. This suggests that socializing agents may foster either prosocial or antisocial behavior, although this process is more likely to occur if the bonding agent holds prosocial values. Through this internalization, individuals are more likely to engage in prosocial or antisocial behavior congruent with the bonding agent's norms and standards.

Additional Considerations in the Social Development Model

Aside from the broad socialization process, the SDM specifies a number of additional factors that may influence how this socialization process occurs. The model specifies three exogenous factors that influence all other socialization processes (Catalano & Hawkins, 1996). First, individuals' social status, conceptualized by indicators such as socioeconomic status, age, race/ethnicity, and gender, appear to be related to a variety of antisocial behaviors such as drug use and crime (Catalano et al., 1992; Elliott, Huizinga, & Menard, 1989; Gottfredson & Hirschi, 1990; G. D. Hill & Atkinson, 1988; Larzelere & Patterson, 1990; Singer & Levine, 1988). The SDM does not propose that these social status variables are directly related to antisocial outcomes, but rather function as an indirect process where social status influences perceived opportunities for prosocial or antisocial interaction and involvement, and also the extent to which various social institutions directly or indirectly influence this perception (e.g., community policing, discrimination, etc.). Second, individuals' unique personality or physiological traits (e.g., general level of arousal, cognitive ability, etc.) influence the extent to which individuals may perceive opportunities for involvement and interaction, in addition to determining what behaviors or interactions individuals find rewarding or reinforcing. This inclusion posits that there are emotional, cognitive, and behavioral skills that differ in presentation and quality across individuals that are necessary for observing and regulating potentially rewarding behaviors. For instance, an individual with low self-worth may not recognize an opportunity to engage with others in a way that may be rewarding. Finally, the application of external constraints may influence individuals' socialization processes. External social structures such as controls on behavior from police or other officials, monitoring or watchfulness of one's parents, or the risk of ostracism or disapproval from one's community or broader society may result in an increased likelihood of perceiving opportunities for interaction or involvement. The social structures that exert external constraints may differ across developmental periods, as will be discussed in the next section.

Further, the SDM outlines sub-models describing the most important factors influencing the socializing process during the preschool, elementary school, middle/junior high school, and high school years and their corresponding developmental periods (Catalano & Hawkins, 1996). Of particular interest to our study are the sub-models for the middle/junior high school and high school years. During the middle/junior high school years, peer influence begins to have a large effect on behavior. In addition to peer influence, school policy, classroom management, and family management practices or parental control continue to have an influence on behavior. Further, the legal system begins to influence choices to engage in various behaviors. Many of these influences continue to be important throughout the high school years, and the model acknowledges that many risk factors for antisocial behavior have already been established by adolescence. However, continuing family management or parental control practices, peer influences, poor educational experiences, legal system exposure, and early antisocial behaviors continue to play a role in exposure to and perception of socializing agents.

Additionally, the SDM was developed under the conditions of two primary assumptions (Catalano & Hawkins, 1996). The first assumption is that human behavior is motivated by a desire to gratify desires; that is, humans enjoy positive feelings and are motivated to engage in behavior that will maximize these positive feelings in either a long-term or short-term situation. The second assumption is that there exists a general social contract about how people should behave, but there are differences in how strongly these beliefs are held and the specific content of those beliefs. This assumption provides a foundation for general knowledge of what consists of prosocial or antisocial behavior, but also indicates that there may be individual variability in one's motivation to adhere to any one definition of prosocial or antisocial behavior.

Holistic-Interactionism and the Person-Centered Approach

The holistic-interactionist perspective provides a conceptual framework for the way in which we may examine constellations of etiological factors among low-income African American adolescents. Reviewed by Magnusson (1999), the holistic-interactionist perspective frames development as a process resulting from the integration of and reciprocal relations between all biopsychosocial aspects of the environment, and posits that an individuals' context can only be understood by examining multiple factors simultaneously. According to the holistic-interactionist perspective, no single factor operates in isolation; every factor operates simultaneously with every other factor that makes up the whole of the individual (Bergman et al., 2003). That is, an individual is conceptualized as a whole, with distinct factors representing component parts that simultaneously and dynamically interact to create the observed whole.

Most research on human behavior examines average effects across multiple individuals, assuming that the observed effects should generalize to all individuals in the population under study; however, it is an ecological fallacy to assume that aggregated information should adequately describe a single individual's behavior (Bogat et al., 2016). By examining the individual as a whole, and examining the influences on or outcomes of that whole, we obtain a unique perspective on individual development, as stated by (Magnusson, 1990, p.197), "the whole picture has an information value beyond what is contained in its separate parts." When we consider an individual as a whole, we are able to examine how a specific combination of factors unique to that individual are associated with other developmental processes, potentially giving insight as to which factors are most influential in that individual's specific context.

The previous discussion on the importance of examining every individual as a unique set of etiological factors may imply that risk for substance use is so individualized that generalizations cannot be made to broadly summarize how etiological factors may demonstrate increased likelihood of substance use across individuals. On the contrary, an increasingly

common way to identify combinations of etiological factors that may predict substance use is to empirically examine the presence of etiological factors across various subgroups of individuals using person-centered analyses. Person-centered analyses may be specifically useful in identifying distinct prototypical patterns of etiological factors that reflect uniqueness in individuals' functioning but also generalize to subpopulations of individuals. In our discussion of person-centered analyses, we use the following definition of *pattern*: "The operating factors are organized and function in terms of functional configurations [...] Important individual differences are to be found in differences in the patterning of operating factors in the system under investigation" (Bergman et al., 2003, p. 12).

Person-centered approaches, which refer to analytic approaches such as latent class analysis, latent profile analysis, finite mixture analysis, growth mixture modeling, latent class growth analysis, among others, statistically identify latent homogeneous subgroups of individuals within a population, wherein the individuals in each subgroup share characteristics with other members of their own subgroup and have qualitatively different characteristics from members of other subgroups (Magnusson, 2003). That is, these approaches allow for the examination of the individual (or groups of similar individuals) as a whole entity, made of multiple dynamic components. These analytic approaches allow for the unique interplay of multiple variables across various subgroups of individuals, without assuming that variables function in the same way across all individuals, consistent with the interactionist approach (Magnusson, 2003). In relation to the current investigation, these subgroups represent individuals who share similar characteristics of their psychosocial environment and may exhibit patterns of etiological factors that distinguish varying levels of likelihood for substance use. Of note, person-centered analyses uncover prototypical patterns of factors, which are patterns that are likely to exist in a population, thus describing the possible patterns of factors that may describe characteristics of subpopulations, allowing for summary of many different types of individuals who may exist within the larger

population. The empirical nature of these approaches will be described in detail in the method.

Importantly, combinations of etiological factors may provide clues as to the mechanistic process that lead certain individuals to use substances, prevent others from engaging in drug or alcohol use, or lead others still to use substances by an alternative pathway. For example, there are at least two distinct developmental pathways associated with increased likelihood of substance use in African American youths across adolescence, one marked by high cumulative exposure to many risk factors, and another marked by negative self-evaluation in early adolescence (Gil, Vega, & Turner, 2002). Giving appropriate attention to unique pathways to substance use may aid in identifying those who would experience the greatest benefits from preemptive interventions.

Utility of Discrete Typologies to Examine Substance Use Etiology

There are certain advantages to conceptualizing the individual as a dynamic whole and examining the extent to which various factors interact to predict later outcomes. Using person-centered methods, specifically empirically-derived discrete typologies of individuals, provides increased statistical power to identify the presence of complex interactions. We later describe eight etiological factors that we intend to examine as possible predictors of substance use; if we were interested in examining an eight-way interaction across all eight etiological factors, we would need an inconceivably large sample to have enough statistical power to adequately test for the presence of such an interaction. However, with typological person-centered methods, we are able to empirically derive patterns of etiological factors that may represent the extent to which higher-order interactions occur among the variables (Bergman et al., 2003; Bogat et al., 2016); while the person-centered approach does not necessarily capture the actual statistical interaction among all eight variables, it may simulate the presence of such interactions in a smaller sample than would be required for the eight-way interaction.

Further, there are an infinite number of ways that individuals may differ in their exposure to etiological factors for substance use. While we appreciate the importance of understanding individual uniqueness of substance use etiology, it is also important to develop some prototypical patterns that generalize across more than one individual, while preserving uniqueness across different types of people who are likely to share similar characteristics (Bogat et al., 2016). This is exactly what person-centered typologies accomplish, identifying unique trajectories of substance use across adolescence or discrete patterns of etiological factors that represent subgroups of individuals, but also allow for some generalizations across similar types of people.

Finally, person-centered typological approaches are also probabilistic in nature; that is, every individual has a probability of belonging to each derived pattern, but in well-defined solutions individuals have a high probability of membership in one pattern while low probabilities of membership in all others (Bergman et al., 2003). This makes the resulting patterns prototypical, in the sense that they are likely to exist in the population, but specific individuals may not exactly fit the prescribed patterns. This allows for flexibility in categorizing individuals, as an individual is not forced to fit into a category that may not adequately describe the individual's characteristics.

Person-Centered Investigations of Risk and Protective Factors

We have previously discussed how etiological factors may operate as dynamic processes, wherein the extent to which a single variable impacts likelihood for later substance use is dependent on the entire combination of etiological factors present in that individuals' unique psychosocial context. We now examine these dynamic interactions across etiological factors through the lens of holistic interactionism and explore how person-centered analyses allow for an investigation into unique patterns of etiological factors that are likely to be present among low-income African American youths.

Prior research has employed person-centered approaches to examine patterns of combined risk and protective factors for a number of behaviors and characteristics including vocabulary (Christensen, Taylor, & Zubrick, 2017), internet addiction (Li et al., 2017), school readiness (Abenavoli, Greenberg, & Bierman, 2017; Pratt, McClelland, Swanson, & Lipscomb, 2016), and adolescent psychopathology (Parra, DuBois, & Sher, 2006). However, few studies have used similar methodologies to identify subgroups of adolescents with homogeneous patterns of response in relation to risk or protective factors for substance use. In one notable exception, investigators seeking to explain differential responses to substance use treatment in a nationally representative sample of adolescents used LCA to identify five prototypical patterns of risk factors for substance use: (1) A *low-risk group*, (2) a *peer cigarette and alcohol use risk group*, (3) an *economic risk group*, (4) a *household and peer risk group*, and (5) a *multicontextual risk group* (Lanza & Rhoades, 2013). Adolescents most likely to be members of the *low-risk* and *economic risk groups* had the lowest levels of binge drinking overall. When examining treatment outcomes among individuals most likely to be members of each group, treatment was only effective in decreasing binge drinking among those most likely to be members of the *peer risk group* relative to those most likely to be members of the *low-risk group*, demonstrating differential treatment effects among the risk factor patterns.

Likewise, and again using a nationally representative sample of adolescents, investigators interested in fostering positive youth development identified prototypical response patterns across a set of factors previously shown to protect against substance use (Syvertsen et al., 2010). These analyses resulted in the following five subgroups: (1) an *adequate protection group*, (2) a *generally inadequate protection group*, (3) an *adequate etiological but low internal protection group*, (4) an *adequate protection with low adult communication group*, and (5) an *adequate protection with risky peer behavior group*. Alcohol use was associated with a small increase in the odds of membership in the adequate protection with low adult communication group and a large increase in odds of membership in the adequate

protection with peer risky behavior group, both relative to the odds of membership in the adequate protection group. Cigarette use was most strongly related to increased odds of membership in the inadequate protection group relative to the odds of membership in the adequate protection group (Syvertsen et al., 2010).

Given evidence that discrete patterns of risk factors and protective factors are differentially associated with alcohol use, cigarette smoking, and treatment outcomes within the broader population, a prudent strategy moving forward might be to identify patterns of theoretically-motivated etiological factors (both extant and novel) with even greater explanatory power and/or relevance among segments of the population with a heightened likelihood of substance use or abuse. However, the above studies examined only risk or protective factors separately. Given the interactive nature of etiological factors and the previously mentioned need to examine one's entire psychosocial context when assessing the extent to which combinations of risk and protective factors are predictive of substance use (Catalano et al., 1992; Hawkins et al., 1992; Jessor et al., 1995; Lloyd, 1998; Rutter, 1987), it is of interest to assess patterns of risk and protective factors as collective combinations of etiological factors, wherein certain factors may exhibit risk or protective effects in one context, but not in another. Not only will the examination of discrete etiological factor patterns contribute to the literature on substance use etiology, but the resulting patterns of etiological factors may serve as additional resources for clinicians or community workers involved with disadvantaged adolescents to characterize adolescents in terms of the identified prototypical patterns, providing a novel way of identifying adolescents who are at increased likelihood for substance use.

Rationale for Present Studies and Significance

This chapter has previously discussed the theoretical foundation of the SDM and the features of holistic-interactionism, including prior empirical work identifying unique patterns of substance use predictors among adolescents. Combining the SDM with a holistic-interactionist perspective provides a foundation for assessing unique patterns of etiological factors for substance use among a sample of low-income African American adolescents. The SDM posits that risk and protective factors at varying levels of influence (e.g., peer, family, community) collectively interact to influence one's likelihood of engaging in antisocial or prosocial behavior. Specifically, antisocial behavior develops as the result of a socialization process that bonds individuals to certain socializing agents, and that attachment to these bonded agents increase the likelihood of participating in behaviors reflective of the bonded agent's norms and standards, whether prosocial or antisocial (Catalano & Hawkins, 1996; Catalano et al., 1996). However, the model also posits that these socializing agents mediate or moderate the effects of individual-level factors (e.g., general cognitive ability, level of arousal, self-worth, etc.) on antisocial behavior outcomes. As such, there is likely to be individual variability in the socialization processes leading to antisocial behavioral outcomes depending on the specific combination of individual, risk, and protective factors unique to an individual.

The SDM provides a framework that theorizes the socialization mechanism through which factors interact to produce antisocial behavioral outcomes, including substance use, that can be applied to a wide range of risk and protective factors. In turn, we can view the previously described risk and protective factors within the SDM framework as representing individual, neighborhood, and family factors that may exert either risk for or protection against later substance use. These factors have been outlined in Table 2.1. We do acknowledge that many of these factors could be considered as part of multiple domains of influence; for instance, exposure to violence may occur in the neighborhood and in the family, while religiosity may develop in conjunction from both the family and the neighborhood.

We also note that, as discussed earlier, many of these factors may exhibit either risk or protection depending upon the unique combination of other factors present in an individual's environment; however, we place these factors into categories of risk and protection to align with concepts discussed in the SDM.

Table 2.1

Examined Etiological Factors by Social Development Model Domain of Influence

Domain of Influence	Risk	Protection
Individual	Traumatic stress	Self-worth
Family		Parental knowledge Religiosity
External social support	Peer pressure Exposure to violence Delinquency	Neighborhood connectedness

Note. External social support refers to the neighborhood, peer, and school domains.

Combining the SDM with a holistic-interactionist framework provides an extension of the theoretical mechanisms outlined in the SDM that suggests unique patterns of theoretical socialization processes across various domains of influence may result in different patterns of antisocial behavior, including substance use. From a holistic-interactionist perspective, we must examine the socialization processes across multiple domains of influence simultaneously to understand the extent to which each of these processes influence other concurrent processes. Combined with our earlier discussion of the variable nature of risk and protective processes, we may take this to mean that there are unique patterns of interaction across etiological factors in which factors provide risk and/or protection depending upon the specific combination of variables present in an individual's environment. As such, examining unique patterns of etiological factors and assessing the extent to which they predict distinct patterns of behavioral outcomes may allow us to identify discrete subpopulations of individuals who are influenced by different socialization processes leading to specific patterns of behavioral outcomes. This may allow for early identification of those

individuals who are at greatest risk for maladaptive outcomes, and may propose potential pathways through which these poor behavioral outcomes may be mitigated.

While the previously described empirical studies on unique patterns of risk and protective factors have contributed to the literature on patterns of substance use risk, there is still a need for research regarding heterogeneity in etiological factors associated with substance use. First, most prior research has exclusively examined constellations of *either* risk (Lanza & Rhoades, 2013) *or* protective (Syvertsen et al., 2010) factors for substance use. While both risk and protective factors are influential in the development of substance use, it is the holistic interaction of individual risk and protective factors (i.e., etiological factors), rather than cumulative effects of each, that is most predictive of substance use (Catalano & Hawkins, 1996; Hawkins et al., 1992; Jessor et al., 1995; Lloyd, 1998; Rutter, 1987). Person-centered approaches that identify combinations of *both* risk and protective factors are uniquely equipped to assess complex interactions among these factors that may be predictive of substance use.

Second, previous research has identified patterns of either risk (Lanza & Rhoades, 2013) or protective (Syvertsen et al., 2010) factors for substance use using nationally representative samples. Although this research is broadly informative with respect to the population at large, identifying patterns of etiological factors solely among low-income inner-city African American adolescents provides an even more focused approach to identifying those who should be most carefully monitored for substance use specifically within this population; this is in contrast to most prior literature that has instead focused on between-group differences across various racial/ethnic groups. Given their generalized risk for substance use and heightened likelihood of health and social consequences related to substance use, in addition to limited access to treatment resources (Cummings et al., 2011; Mulia et al., 2009; Zapolski et al., 2014), African American adolescents are an important population in which to identify those who are most likely to engage in later substance use.

Finally, prior research using typologies based on patterns of endorsement on risk or protective factors have only related profile group membership to substance use at a single time point. Adolescence is a critical developmental period for the initiation and escalation of substance use, and individuals' substance use increases on average across adolescence; adolescents with increasing substance use trajectories across the teenage years are at greater risk for other maladaptive outcomes in early adulthood (Lynne-Landsman et al., 2010; Schulenberg et al., 2014). However, there is heterogeneity in the extent to which adolescents engage in substance use, as multiple prototypical patterns of substance use development have been identified in prior research on substance use among low-income African American adolescents (Komro et al., 2010; Park et al., 2018). Assessing the extent to which patterns of etiological factors are associated with changes in substance use provides a more in-depth examination of the extent to which certain types of adolescents are at risk for the most maladaptive patterns of substance use. By examining the etiology of problematic substance use trajectories, we may develop insight into factors associated with broader developmental trajectories of problematic behaviors that extends beyond adolescence.

Given well-documented consequences of substance use, combined with limited availability of treatment resources for minority adolescents in low-income communities, it is imperative to distinguish among adolescents who have varying presentations of etiological factors that may indicate increased likelihood for substance use. Identifying subgroups of low-income African American adolescents who demonstrate discrete patterns of etiological factors would allow for improved allocation of available resources to adolescents who are most likely to misuse substances. The proposed study will use person-centered approaches to identify subgroups of impoverished African American adolescents who demonstrate discrete patterns of etiological factors, and then assess the extent to which these patterns are associated with substance use outcomes across adolescence. This will provide a more holistic (i.e., breaching multiple components of an individuals' environment) understanding of

what types of risk and protective factors tend to cluster together, and how these clusters are uniquely predictive of substance use patterns in African American adolescents.

Specific Aims

The research presented here is organized into four aims that are concerned with understanding the extent to which patterns of risk and protective factors are indicative of increased likelihood of manifesting discrete substance use trajectories among impoverished African American adolescents.

Aim 1.a. Identify discrete prototypical patterns of (1) risk factors and (2) protective factors in low-income African American adolescents. Previous research has identified a number of risk and protective factors for substance use and has suggested that discrete patterns of risk and protective factors are uniquely associated with use of alcohol and cigarettes (Lanza & Rhoades, 2013; Syvertsen et al., 2010). However, the majority of research in this area has focused on the use of nationally-representative samples. While the use of nationally-representative samples in studying the etiology of substance use is informative for the population at large, these large studies may not contain adequate oversampling of African Americans to assess the extent to which risk and protective factors are associated with substance use among this specific population. The use of a predominately low-income African American sample allows us to assess the presence of latent subgroups of risk and protective factors for substance use among a demographically homogeneous sample, providing detailed information about how risk and protective factors (separately and collectively) are likely to cluster specifically within this population.

The present study first assessed whether there are multiple prototypical patterns of risk factors and multiple prototypical patterns of protective factors (separately) among a sample of low-income African American adolescents at age 12, allowing us to describe homogenous subgroups of risk for and protection against substance use among this particular demographic. A theoretical model depicting these analyses are presented in Figure 2.1.

Given the exploratory nature of this aim, we did not make specific hypotheses about the number or nature of these risk and protective factor patterns. However, given prior research on patterns of risk and protective factors for substance use among nationally representative samples (Lanza & Rhoades, 2013; Syvertsen et al., 2010), we did anticipate the presence of multiple prototypical patterns of risk factors and multiple prototypical patterns of protective factors.

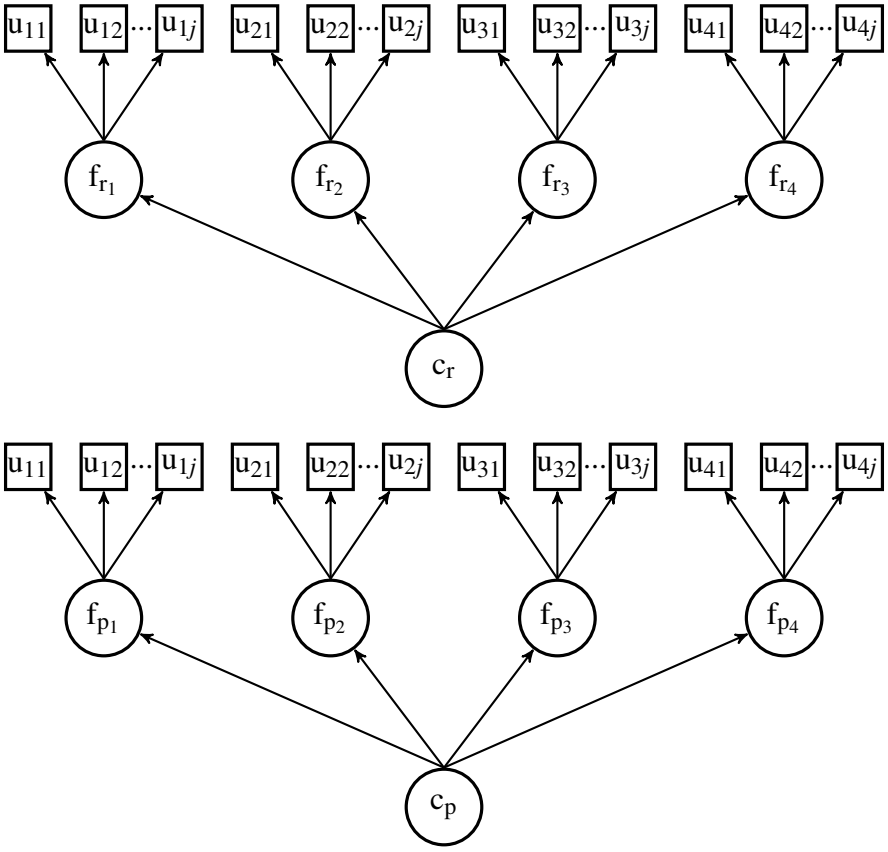

Figure 2.1. Theoretical model depicting analyses to be completed in *Aim 1.a*. *U*s indicate manifest indicators of etiological factors, *f*s indicate measurement models used to represent each individual etiological factor, and *c*s represent a mixture of etiological factors used to represent discrete patterns across etiological factors. Two latent categorical variables are used to represent discrete patterns across risk factors and protective factors separately.

Aim 1.b. Identify conditional associations among prototypical patterns of risk factors and prototypical patterns of protective factors. As described above, prior research has suggested that discrete patterns of risk factors and discrete patterns of protective factors

(separately) are uniquely associated with use of alcohol and cigarettes (Lanza & Rhoades, 2013; Syvertsen et al., 2010). However, prior research also suggests that risk and protective factors do not operate in isolation to predict substance use; that is, cumulative co-action of multiple risk and protective factors may indicate whether one has an increased likelihood of substance use (Hawkins et al., 1992; Jessor et al., 1995; Rutter, 1987). While separate prototypical patterns of risk factors and prototypical patterns of protective factors may differentiate among adolescents who are more or less likely to demonstrate problematic trajectories of substance use, understanding whether certain combinations of risk profiles and protective profiles are associated with lower likelihood of problematic substance use will provide a more robust understanding of the extent to which the interplay across risk and protective factors is predictive of likelihood of demonstrating adolescent substance use trajectories.

To supplement our understanding of risk and protective patterns in adolescence, we also identified conditional associations (i.e., conditional most likely latent risk and protective factor profile membership) among the previously identified risk factor patterns and protective factor patterns. To do so, we first established the most likely latent profile membership in risk factor and protective factor patterns separately, and then created a single manifest categorical indicator that represented conditional profile membership (e.g., risk factor pattern 1 and protective factor pattern 1, risk factor pattern 1 and protective factor pattern 2, etc.) for each individual in the sample. This is theoretically depicted in Figure 2.2. To our knowledge prior research has focused only on identifying prototypical patterns of risk factors and protective factors separately but has not identified conditional associations between risk factor and protective factor profile memberships; prior research has also not examined these conditional associations in a homogenous African American sample. Given our anticipation of multiple prototypical patterns of both risk factor patterns and protective

factor patterns, we expected that there would similarly be multiple conditional associations of risk factor and protective factor patterns identified in the sample.

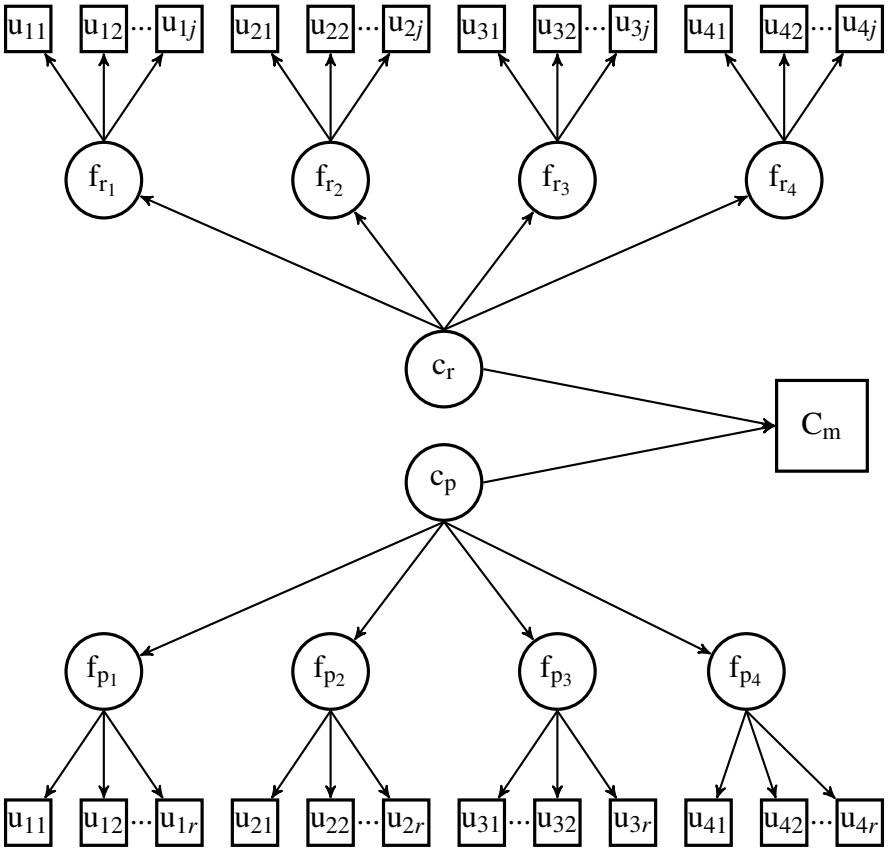

Figure 2.2. Theoretical figure depicting *Aim 1.b*. The derived patterns of risk and protective factors are used to create a single categorical variable indicating conditional profile membership.

Aim 2. Identify discrete prototypical patterns of combined etiological factors in low-income African American adolescents. Although prior research has identified discrete patterns of risk factors (Lanza & Rhoades, 2013) and discrete patterns of protective factors (Syvertsen et al., 2010), little research has investigated the presence and characteristics of prototypical patterns of etiological factors (i.e., risk and protective factors assessed together). Understanding the etiology of substance use requires attention to unique constellations of both risk and protective factors that may differentiate among adolescents at varying levels of risk for substance use, given that some factors may indicate risk for or protection

against substance use in the presence of certain etiological factors, but not others (Jessor et al., 1995). Further, little research has examined etiological factors for substance use within a homogenous African American sample.

The second aim of this study was to identify prototypical patterns of etiological factors for substance use (i.e., combined risk and protective factors) in the same sample of low-income African American adolescents at age 12. This analysis is theoretically depicted in Figure 2.3. To our knowledge, there is little previous research on discrete patterns of etiological factors for substance use; as such, we consider these analyses exploratory. While we made no specific hypotheses about the characteristics of individual etiological factor patterns, based on prior research identifying at least five patterns of risk (Lanza & Rhoades, 2013) and five patterns of protective (Syvertsen et al., 2010) factors separately, we hypothesized that multiple prototypical patterns of etiological factors would be present among impoverished African American adolescents.

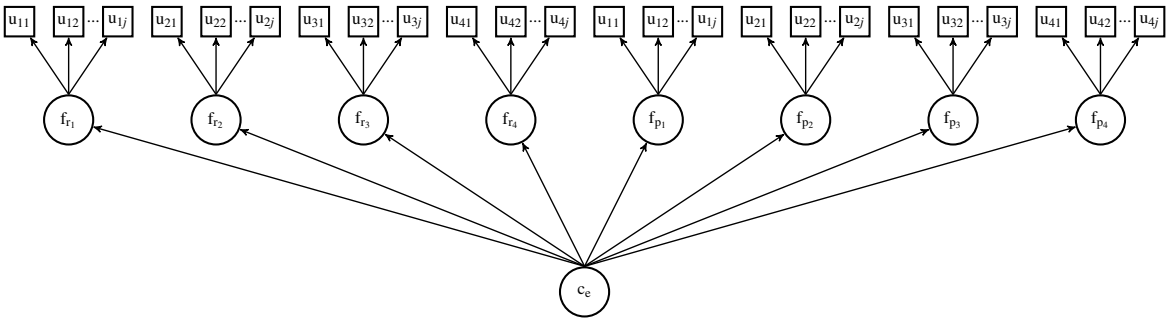

Figure 2.3. Theoretical model depicting analyses to be completed in *Aim 2*. *U*s indicate manifest indicators of etiological factors, *f*s indicate measurement models used to represent each individual etiological factor, and *c*s represent a mixture of etiological factors used to represent discrete patterns across etiological factors. A single latent categorical variable is used to represent patterns across all eight etiological factors.

Aim 3. Identify discrete trajectories of substance use across adolescence among low-income African American adolescents. Prior research has suggested that substance use on average increases across adolescence as part of a developmentally normative trajectory (Schulenberg et al., 2014). However, adolescents are not necessarily heterogeneous in the

extent to which they engage in substance use across the teen years, as prior research has demonstrated that discrete subgroups of adolescents are best characterized by unique trajectories of substance use (K. A. Bolland et al., 2016; Chen & Jacobson, 2012; White et al., 2006). These trajectories have been shown to predict behavioral outcomes in young adulthood; of interest, adolescents who demonstrate trajectories of increasing substance use are at increased risk for maladaptive outcomes such as criminal activity and substance use disorders later in adolescence and in young adulthood (Lynne-Landsman et al., 2010). As such, it is of interest to assess risk and protective factors associated with longitudinal patterns of substance use, particularly those trajectories characterized by increasing use over time.

We identified discrete patterns of alcohol, tobacco, and marijuana use in the same sample of low-income African American adolescents across ages 13-17, examining trajectories across the three separate substances independently. These analyses are theoretically depicted in Figure 2.4. While we did not make specific hypotheses regarding the exact number of trajectories of alcohol, cigarette, and marijuana use across adolescence, we did anticipate the presence of trajectories consisting of consistently abstaining, increasing across adolescence, or consistently high levels of substance use, as these trajectories have been identified in prior research (K. A. Bolland et al., 2016; Chen & Jacobson, 2012).

Aim 4.a. Assess the extent to which conditional associations among discrete prototypical patterns of risk factors and discrete prototypical patterns of protective factors are associated with substance use trajectories across adolescence. Although some prior research has explored discrete patterns of risk and protective factors in adolescence and has identified the extent to which alcohol and cigarette use correlate with likelihood of demonstrating those patterns (Lanza & Rhoades, 2013; Syvertsen et al., 2010), little research has extended this examination to how conditional associations between discrete risk and protective factor profiles are predictive of the longitudinal development of substance use. Understanding how typologies of risk and protective factors are associated with substance

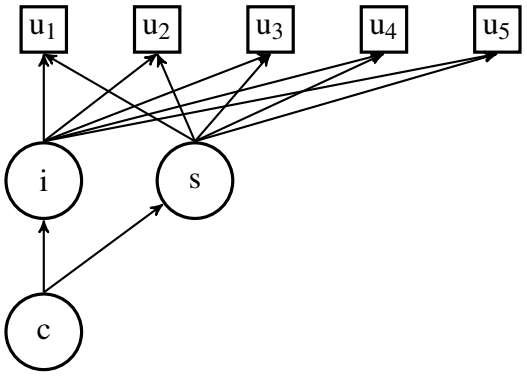

Figure 2.4. Theoretical figure depicting *Aim 3*. *U*s represent manifest substance use indicators, *i* represents the intercept or initial level of use, *s* represents the slope or rate of change, and *c* represents a mixture of intercepts and slopes indicating discrete patterns of trajectories.

use across adolescence may ultimately allow for more effective identification of at-risk adolescents.

In order to determine the extent to which the interaction of separate patterns of risk factors and protective factors provide a unique prediction of adolescents' likelihood of demonstrating discrete substance use trajectories across adolescence, we used adolescents' conditional membership in risk and protective factor patterns to predict their likelihood of membership in substance use trajectories. That is, we used the conditional associations between risk and protective factor patterns identified in *Aim 1.b.* to predict either the mean growth trajectory or likelihood of membership in substance use trajectories. We did so by regressing the latent categorical variable representing substance use trajectories on the dummy-coded indicator variable representing conditional profile membership (i.e., risk factor profile and protective factor profile combination); doing so allows us to examine the interaction between discrete patterns of risk factors and discrete patterns of protective factors in predicting substance use trajectories. If no heterogeneity in trajectories was identified for a particular substance, we regressed the intercept and slope of the mean growth trajectory on on the dummy-coded indicator variable representing conditional profile membership. A theoretical figure of this aim is presented in Figure 2.5. Should we identify more than one

discrete trajectory of substance use, we hypothesize that conditional associations of profile membership consisting of high endorsement of multiple risk factors and low endorsement of protective factors will be predictive of membership in higher substance use trajectories.

Aim 4.b. Assess the extent to which discrete prototypical patterns of etiological factors are associated with the likelihood of membership in discrete substance use trajectories across adolescence. Extending the rationale expressed above for *Aim 4.a.*, little research has examined the extent to which combinations of risk and protective factors (together) are associated with substance use trajectories across adolescence. However, given that the extent to which certain factors may represent risk and/or protection for or against substance use depends on other etiological factors present in one's psychosocial environment, it is of interest to assess how unique combinations of etiological factors that are not explicitly described as "risk factors" or "protective factors" are associated with substance use across adolescence.

In order to examine the extent to which patterns of etiological factors are predictive of substance use trajectories, we also used the patterns of etiological factors identified in *Aim 2* to predict likelihood of membership in substance use trajectories (see Figure 2.6). Should we identify more than one discrete trajectory of substance use, we hypothesized that likelihood of membership in various etiological factor patterns will be differentially associated with likelihood of membership in discrete substance use trajectories. By using the conditional associations among separate risk factor and protective factor patterns to predict substance use trajectories (as described in *Aim 4.a.*) in addition to using etiological factor patterns to predict substance use trajectories (as described in this *Aim*), we may gain insight into the extent to which conceptualizing various psychosocial variables as risk factors, protective factors, or etiological factors provide unique information for predicting likelihood of developing various substance use trajectories across adolescence.

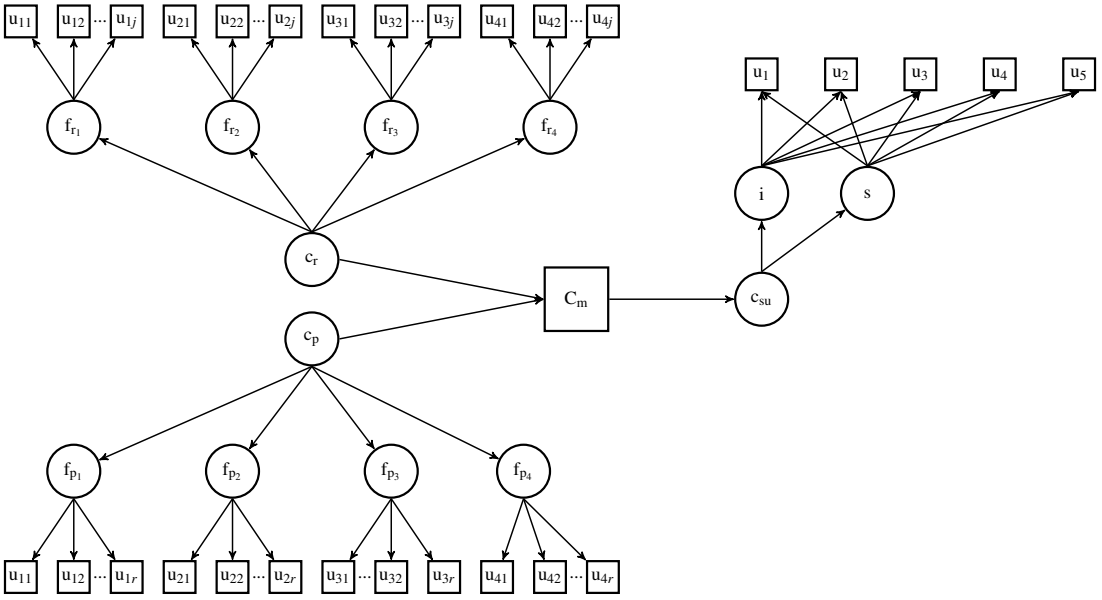

Figure 2.5. Theoretical figure depicting *Aim 4.a.*. The derived patterns of risk and protective factors are used to create a single categorical variable indicating conditional profile membership. This single categorical variable of conditional profile membership is used to predict trajectories of substance use.

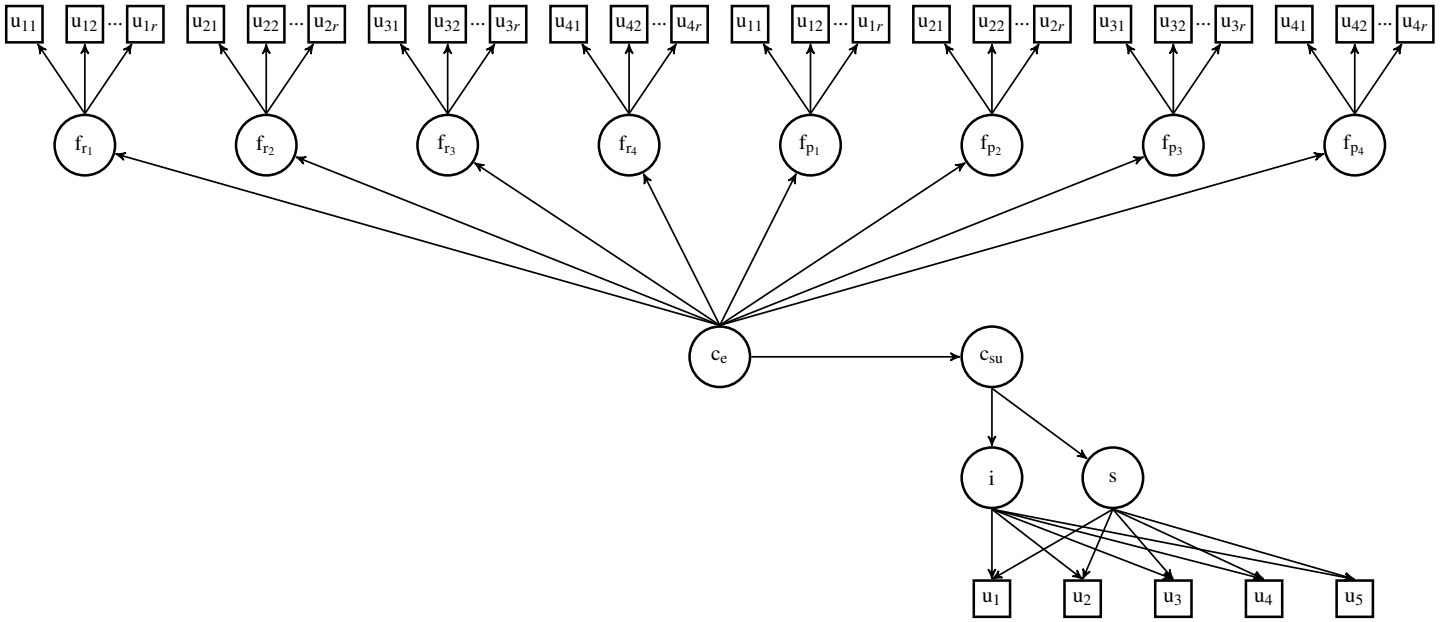

Figure 2.6. Theoretical figure depicting *Aim 4.b.*. Theoretical model depicting analyses in *Aim 4.b.* Discrete patterns of etiological factors will be used to predict discrete trajectories of substance use.

CHAPTER THREE

Research Design and Methods

Sample and Procedure

Data for this study was taken from the Mobile Youth Survey (MYS), a cohort longitudinal study of adolescents sampled from the poorest neighborhoods of Mobile, Alabama (J. M. Bolland, 2007; K. A. Bolland et al., 2016). All sampling and data collection procedures for the MYS were approved by the Institutional Review Board at the University of Alabama. Data collection began in 1998 by identifying the most impoverished neighborhoods in the Mobile metropolitan area using data from the 1990 census. The final sampling frame consisted of neighborhoods primarily in Mobile (population approx. 200,000) and Pritchard (population approx. 30,000), Alabama. Seven of the targeted neighborhoods consisted of primarily public housing, whereas the other six consisted of nonpublic housing. Over 73% of the individuals living in these neighborhoods lived below the poverty line, with a median household income of $5,000 as of the 1990 census (K. A. Bolland et al., 2016). Within the seven public housing neighborhoods, the researchers used housing authority records to identify homes with adolescents ages 10-18 and randomly selected half of these homes to comprise the active recruitment public housing sample. They also randomly selected half of the nonpublic houses and apartments to comprise the actively recruited nonpublic housing sample. The other halves of these neighborhoods were not actively recruited, but some adolescents were passively recruited by posting fliers in the neighborhood and by word of mouth.

Adults residing with adolescents targeted for study inclusion were contacted and asked to give consent for the adolescents to participate. After obtaining primary caregiver consent, MYS surveys were administered to adolescents in groups of 20-30 at community

centers. Adolescents were given a questionnaire packet and were asked to sign the adolescent assent provided on the first page and then turned into the survey administrator. The survey administrator read questions aloud to the adolescents, who then indicated their response by filling in the appropriate bubble on the questionnaire packet. Adolescents who had difficulty understanding an oral question or needed additional time to respond to items were given the survey in a separate room with a survey administrator. Adolescents were given $10 as compensation for completing the survey; this amount increased to $15 in 2006.

During the first recruitment year (1998), the MYS recruited 1,771 adolescents (ages 10-18) using both active and passive recruitment strategies (K. A. Bolland et al., 2016). In each subsequent year of the survey (through 2011), researchers attempted to re-contact previously surveyed adolescents, in addition to recruiting a new cohort of neighborhood adolescents (i.e., another 1,213 adolescents age 10-18 were recruited in 1999; 615 new adolescents age 10-18 were recruited in 2000, etc.). The MYS surveyed a total of 12,387 adolescents (36,164 assessments) from 1998 to 2011. Respondents were not interviewed past age 18.

Over the course of the survey, there was considerable attrition, primarily attributed to aging out of the study (i.e., not interviewed past age 18) or moving to a different neighborhood and lost to follow-up. However, over 80% of individuals surveyed during the first wave were surveyed during at least one additional wave (K. A. Bolland et al., 2016). Missing data due to attrition (i.e., "monotone" missing data) may be treated as missing at random if missing data are not dependent on observed variables collected prior to drop out (Little & Rubin, 2002, p. 5). Previous research on missing data in the MYS indicates that attrition does not appear to be associated with a number of risk factors, including substance use, suggesting that data may be treated as missing at random (A. C. Bolland, 2012).

We placed restrictions on the sample in order to (1) identify a demographically homogeneous population to make our conclusions as representative of the population of interest as possible, and (2) make as valid inferences about longitudinal trends as possible. We limited the sample to include adolescents who indicated African American ethnicity and who received free or reduced lunch, as incorporating these restrictions has been shown to not bias parameter estimates and provides a homogeneous sample of interest representative of the sampling population (A. C. Bolland, 2012). Over 98% of MYS participants are of African American ethnicity, and poverty rates in neighborhoods targeted by MYS range between 31.5% to 81.4% (J. M. Bolland, 2007). We also retained all cases that (1) responded to at least one of the etiological factor items at age 12 (listed below in *Measures*); and (2) responded to at least one substance use item (even if they do not initiate into substance use across these years). The final analytic sample consisted of 1,576 African American adolescents (45.5% female). The same analytic sample was used in all aims of this project. All measures and analytic procedures were determined as exempt research by the Institutional Review Board at Baylor University.

Measures

Risk and protective factors for substance use were measured with scales provided within the MYS questionnaire at age 12. Question wording for all items are available in Appendix A.

Risk Factors

Delinquency was measured by six items used in prior studies of delinquent behavior in the MYS (K. A. Bolland et al., 2016; Church et al., 2015, 2012; Jaggers et al., 2014). These items captured six delinquent behaviors: (1) ever carrying a gun, (2) ever carrying a knife or razor, (3) ever pulling a knife or gun on someone else, (4) ever cutting, shooting, or stabbing someone else, (5) ever being involved in a gang, and (6) ever being arrested. All items were dichotomous (0 = "No" and 1 = "Yes"). K. A. Bolland et al. (2016) reported that

that a single factor solution accounted for 49% of the variance in the latent construct with a single eigenvalue of 2.95 and other eigenvalues below 1, and that these items had acceptable internal consistency (α = .73). Church et al. (2015) similarly reported that a single factor accounted for 51% of the variance in the latent construct with a single eigenvalue of 3.08 and all other eigenvalues less than 1; these items had good internal consistency (α = .80). Jaggers et al. (2014) reported that a principle components analysis found that a single scale accounted for 47% of the variance, with a single eigenvalue of 2.79 and all others less than 1; internal consistency was also acceptable (α = .76). Finally, Church et al. (2012) found internal consistency to be .77. Internal consistency in our analytic sample was similarly good (α = .78). These items were used as indicators of a latent construct of *delinquency* as identified by confirmatory factor analysis (CFA; described in *Analytic Strategy*).

Exposure to violence was measured by five items that have been used in prior studies of exposure to violence using the MYS (Spano, Rivera, & Bolland, 2006, 2010, 2011; Spano, Rivera, Vazsonyi, & Bolland, 2008, 2012; Spano, Vazsonyi, & Bolland, 2009). Items measuring exposure to violence consisted of: (1) ever threatened with a knife or gun; (2) ever been cut bad enough to see a doctor; (3) ever been shot; (4) a family member or friend ever been shot or stabbed; and (5) ever witness someone being shot, stabbed, or cut. All items were dichotomous (0 = "No" and 1 = "Yes"). While internal consistency of the exposure to violence items reported in previous literature has been lower than is typically considered acceptable (α = .57-.67), these items have consistently demonstrated inverse associations with parental knowledge, providing evidence of discriminant validity of this exposure to violence measure (Spano et al., 2008, 2012, 2009). As expected, the internal consistency in our analytic sample was similar to other studies (α =.66). These five items were used as indicators of a single latent construct of *exposure to violence* identified by CFA (see *Analytic Strategy*).

Peer pressure was measured by six items created by the MYS asking how many of the respondent's friends think s/he is a "punk" for engaging in the following behaviors: (1)

not drinking alcohol, (2) not using drugs, (3) not carrying a weapon, (4) refusing to fight after being insulted, (5) doing well in school, and (6) not having sex. We chose a set of items that reflect peer pressure to engage in behaviors that are maladaptive for adolescents given prior research that peer pressure to misconduct is associated with increased substance use (Studer et al., 2014, 2016). Peer pressure items were responded to on a three-point scale with response options ranging from 0 = "Almost none of them" to 2 = "Most of them" (α = .72 to .90). Internal consistency in our analytic sample was excellent (α = .86). These six items were used as indicators of a single latent construct of *peer pressure* identified by CFA (see *Analytic Strategy*).

Traumatic stress was measured by seven items adapted from the Global Appraisal of Individual Needs (GAIN; Dennis, Titus, White, Unsicker, & Hodgkins, 1998; Titus, Dennis, Lennox, & Scott, 2008). The GAIN is an inventory of assessments designed to aid in clinical decision-making, including measures of substance use, physical health, mental health and treatment utilization (Dennis et al., 1998; Titus et al., 2008). The traumatic stress items used in this analysis capture adolescents' emotional state following the experience of traumatic events (Hooper et al., 2015). Items include "I have trouble sleeping at night when something bad happens to a family member or friend," or "I think about bad things that have happened to a family member or friend, even when I don't want to." These seven items were responded to on a scale from 0 = "Almost never" to 2 = "Very often." Prior research has demonstrated that these seven items measured in the MYS sample reflected a single unidimensional construct, and had a coefficient alpha of .77 (Hooper et al., 2015). The items used in our analysis similarly demonstrated good internal consistency (α = .76). These seven items were used as indicators of a single latent construct of *traumatic stress* identified by CFA (see *Analytic Strategy*).

Protective Factors

Neighborhood connectedness is typically assessed in the MYS utilizing 11 dichotomous items wherein adolescents reported whether they agreed or disagreed with statements about their neighborhood (Glynn, 1981; Perkins, Florin, Rich, Wandersman, & Chavis, 1990). Example items include "I feel I am an important part of my neighborhood" and "I have friends in my neighborhood I can depend on," responded to on a scale of 0 = "Disagree" and 1 = "Agree." However, half of these items were positively worded and half were negatively worded (e.g., "There are people in my neighborhood, other than my family, who really care about me" is positively worded, as opposed to, "Very few of my neighbors know me," which is negatively worded). We found that the positively worded and negatively worded items represented two distinct latent constructs (see Results); as such, we elected to retain only the six positively worded items to represent neighborhood connectedness (see Appendix A for the exact wording of the final items). These items demonstrated acceptable internal consistency (α = .76). These six items were used as indicators of a latent variable of *neighborhood connectedness* as defined by CFA (see *Analytic Strategy*).

Parental knowledge was assessed with six items reflecting the extent to which adolescents' parents are aware of adolescents' activities and whereabouts (e.g., "Does your mother or father know who you hang out with?" and "How much does your mother or father really know about where you go at night?"). Two items were responded to on dichotomous Likert-type scales (0 = "No" and 1 = "Yes"), three items were responded to on three-point Likert-type scales (0 = "They don't know" to 2 = "They know a lot"), and one item was responded to on a four-point Likert-type scale (0 = "I don't go out at night" to 3 = "They know a lot"; see Appendix A). Initial validation studies reported internal consistency between .56 and .65, and test-retest reliability of .62 (Lamborn, Mounts, Steinberg, & Dornbusch, 1991). Other research using these items in the MYS has found good internal consistency (α = .77; Jaggers et al., 2018). These items demonstrated acceptable internal

consistency in our analytic sample (α = .70). These six items were used as indicators of a latent construct of *parental knowledge* identified by CFA (see *Analytic Strategy*).

Religiosity was assessed by three items reflecting (1) the extent to which religiosity is important to the respondent, with three response options ranging from 0 = "Not important" to 2 = "Very important;" and the frequency with which the respondent (2) goes to church, worship services, or other religious activities, and (3) reads or studies a Holy Book, both with item response options ranging from 0 = "Never" to 4 = "Once a week or more." These items demonstrated low internal consistency (α = .43), but were retained given that these were the only indicators of religiosity available in the MYS. These three items were used as indicators of a latent variable of *religiosity* as identified by CFA (see *Analytic Strategy*).

Self-worth was measured with a subscale of The Perceived Competence Scale for Children (Harter, 1982). The self-worth subscale consists of nine dichotomous items, where adolescents are asked to select which of two statements is most reflective of how they think about themselves (e.g., "I am usually unhappy with myself" or "I am usually happy with myself"). The items demonstrated good internal consistency (α = .73 to .82) and test-retest reliability (r = .69 to .70; Harter, 1982) in the validation study. The internal consistency identified in our sample was .63, which is very similar to that obtained in other studies utilizing a primarily African-American sample (Copeland-Linder et al., 2011). These nine items were used as indicators of a latent construct of *self-worth* as identified by CFA (see *Analytic Strategy*).

Substance Use

Substance use was measured at five annual assessments subsequent to the age 12 assessment (i.e., substance use was measured annually across ages 13-17). We used a single indicator of frequency of substance (i.e., alcohol, cigarettes, and marijuana) use in the past 30 days to conduct three separate growth mixture models, one for each substance of interest. Participants responded to every item on a scale of 0 = *did not drink or use a drug*, 1 =

drank alcohol or used a drug once, or 2 = *drank alcohol or used a drug more than once* in the past 30 days. Previous research using the MYS utilized a different substance use item representing a composite indicator of recency and frequency of substance use (K. A. Bolland et al., 2016). We chose to use the current indicator in our analyses because we felt that this item had a more straightforward interpretation compared to a composite indicator of both recency and frequency of substance use.

Analytic Strategy

Data Reduction

We used CFA to establish the extent to which the observed items were indicators of a latent construct (i.e., a specific risk or protective factor). CFA is a measurement model that specifies relationships between observed indicators and a latent construct (T. A. Brown, 2014, p. 1). To measure a construct using CFA, the researcher operationalizes the latent construct by specifying certain indicators that have been shown (via theory and previous research) to be indicative of the unobserved variable of interest; that is, CFA is a theory-driven practice that requires the researcher to specify all aspects of the model prior to analysis. The extent to which an indicator is reflective of a latent construct (i.e., the extent to which the indicator *loads* onto the factor) is represented in a factor loading, which is a measure of the relationship between the latent variable and the indicator; similarly, item communalities provide measures of the proportion of variance in the observed item response that can be accounted for by the latent variable (T. A. Brown, 2014, p. 15). By comparing covariance structures implied by the prescribed model and the observed data, fit indices for CFA provide absolute or relative metrics of how well the outlined factor structure replicates that observed in the data (T. A. Brown, 2014).

In most prior research, the above etiological factors have been operationalized as sum scores of the indicators; for example, the items measuring delinquency have previously been summed to create an overall delinquency composite (K. A. Bolland et al., 2016;

Church et al., 2015; Jaggers et al., 2014). There are two related advantages to measuring the above risk and protective factors via CFA as opposed to sum scores: first, CFA accounts for measurement error in observed indicators (T. A. Brown, 2014). That is, CFA does not assume that all items are measured perfectly, and that a certain amount of variance in each indicator can be attributed to the latent variable, with the remainder of variance in the indicator being attributed to measurement effects or random error. This provides a more reliable representation of the construct of interest.

The second advantage of utilizing CFA to represent the above etiological factors is that indicators are not required to be equally representative of the latent construct (T. A. Brown, 2014). That is, because the latent variable accounts for a certain amount of variance in the observed response, the latent variable may account for different amounts of variance across indicators. Allowing factor loadings to vary across items means that not every indicator is required to be equally representative of the construct of interest, allowing for a more nuanced and reliable operationalization of each etiological factor. By conducting CFA on the items for each etiological factor, we will be able to identify loadings of each item onto its respective factor (i.e., the relationship between the latent construct and the observed indicator) to be utilized in subsequent analyses. Essentially, using CFA to measure the etiological factors allows us to have a more nuanced and reliable representation of the factors than would be possible with other methods (i.e., sum scores).

We specified and estimated eight measurement models using CFA, wherein each etiological factor was represented by a single factor and each indicator loaded on the construct it reflected. Conducting CFA with categorical indicators requires that alternative estimation procedures be used, as utilizing ordinary maximum likelihood may result in incorrect parameter estimates and significance tests, in addition to the risk of reduced estimates of the correlations between factors and indicators (i.e., factor loadings; T. A. Brown, 2014, p. 387). Mplus is equipped with a robust maximum likelihood estimator that recruits numerical integration to conduct CFA on categorical indicators without the risk of biased

parameter estimates (T. A. Brown, 2014; L. K. Muthén & Muthén, 1998-2018). Using robust maximum likelihood with numerical integration is favorable to alternative procedures that may be utilized in CFA with categorical indicators such as robust weighted least squares because robust maximum likelihood will utilize all available data in the analysis whereas robust weighted least squares will utilize pairwise deletion in conducting the analysis (Asparouhov & Muthén, 2010). In determining the acceptability of the model fit to the data, we consulted factor correlations and loadings, chi-square, standardized root mean square residual (SRMR), comparative fit index (CFI), Tucker-Lewis index (TLI), and root-mean squared error of approximation (RMSEA). After assessing model fit, we examined the factor loadings of each item onto the factor of interest to ensure that the factor loadings were adequately representative of the latent construct (i.e., factor loadings greater than .7 are considered adequate; T. A. Brown, 2014). We extracted factor scores based on the CFA models using maximum a posteriori estimation for use in later stages of the analysis (L. K. Muthén & Muthén, 1998-2018).

Aim 1.a.

We used latent profile analysis (LPA) to identify prototypical patterns of responding to a set of risk factor indicators and a set of protective factor indicators separately. LPA allows us to identify whether there are qualitatively different subgroups of adolescents who are differentiated by the extent to which they demonstrate various patterns of risk factors and various patterns of protective factors (as each risk and protective factor has been operationalized as a factor score extracted from CFA), allowing us to identify discrete patterns of risk factors and protective factors that adolescents in low-income neighborhoods are likely to demonstrate (McCutcheon, 1987).

We estimated two latent profile models, wherein the first model identified discrete patterns of risk factors, and the second model identified discrete patterns of protective factors. The model for the risk factors consisted of identifying homogeneous subgroups (latent

profiles) across four factor scores extracted from the CFAs of the individual risk factors. To determine whether the extraction of an additional profile results in a significant decrement in model fit in LPA, we compared a model with k classes to a model with $k-1$ classes, using the Vuong-Lo-Mendell-Rubin likelihood ratio test (VLMR-LRT; Lo, Mendell, & Rubin, 2001; Vuong, 1989) and the Lo-Mendell-Rubin adjusted likelihood ratio test (LMR-ALRT; Lo et al., 2001). We also consulted Bayesian Information Criteria (BIC; Schwartz, 1978) and sample-size adjusted Bayesian Information Criteria (SSA-BIC; Sclove, 1987) when comparing the relative fit of competing models, with lower BIC and SSA-BIC values indicating better model fit. After identifying the best-fitting number of profiles, we explored the characteristics of the profiles by examining the mean risk factor scores among individuals most likely to be members of each latent subgroup. Doing so aids in understanding the specific risk factors individuals most likely to be members of each subgroup are likely to demonstrate, and how those patterns of risk factors are different from patterns exhibited by individuals likely to be members of other subgroups.

Following the identification of the discrete patterns of risk factor items, we conducted the same procedure for the protective factors. We ran LPA on the four factor scores extracted from the CFAs representing each of the protective factors, comparing a model with k classes to a model with $k-1$ classes, and utilizing VLMR-LRT, LMR-ALRT, BIC, and SSA-BIC to determine whether the extraction of an additional class resulted in a significant decrement in model fit. Similarly, after the extraction of an additional class resulted in a significant decrement in model fit, signaling the most appropriate number of classes has been identified, we examined mean factor scores among individuals most likely to be members in each class to assess the characteristics of each prototypical pattern.

Aim 1.b.

We also examined conditional associations among likelihoods of membership in discrete patterns of (1) risk factors and (2) protective factors. To do so, we categorized individuals into their most likely latent profile membership based on their highest posterior

probability of membership in both the risk factor profiles and the protective factor profiles. We then examined individuals' most likely latent risk profile membership and most likely latent protective profile membership, and constructed a categorical variable to represent conditional most likely latent profile membership (e.g., most likely to demonstrate risk profile 1 and protective profile 1, most likely to demonstrate risk profile 1 and protective profile 2, most likely to demonstrate risk profile 2 and protective profile 2, etc.). We then created a series of dummy coded variables from the conditional profile membership patterns for use in future analyses, wherein we set the reference group for the conditional patterns to be the group with the lowest endorsement of risk factors and the highest endorsement of protective factors.

Aim 2

The analysis plan for the second aim was be similar to that listed above for *Aim 1.a.* in that we utilized LPA to identify discrete patterns of risk and protective factors for substance use. However, in this aim we conducted LPA on the entire set of etiological factors (i.e., conduct LPA on risk and protective factors simultaneously), whereas in *Aim 1.a.* we conducted LPA on the risk factors and on the protective factors separately. We assessed the presence of multiple prototypical patterns of the eight etiological factors (operationalized as factor scores extracted from CFA) via an iterative modeling procedure wherein a model with k profiles was compared to a model with $k-1$ profiles, increasing next to a model with $k+1$ profiles compared to a model with k profiles, and so on. We again used LMR-LRT, BLRT, and BIC to determine whether the extraction of an additional profile resulted in a significant decrement in model fit. After the most appropriate number of profiles was identified, we examined the characteristics of each profile by examining the mean item responses among individuals most likely to be members of each latent profile.

Aim 3

For the third aim, we used growth mixture modeling (GMM) to identify discrete trajectories of substance-specific use across five subsequent assessments following assessment at age 12, conducting GMM on the alcohol, cigarette, and marijuana use indicators separately. GMM is an extension of conventional growth modeling, which examines individual variation in how an observed outcome variable is associated with time (B. O. Muthén & Muthén, 2000), and assesses differences across individuals in how each individual changes across time (Curran, Obeidat, & Losardo, 2010). Essentially, conventional growth modeling estimates trajectories of a behavior across time and is comprised of two components: (1) a latent intercept that describes the initial level of the behavior of interest, and (2) a latent slope, describing the linear rate of change in the behavior across time. In some cases, a quadratic component may also be added, representing the rate of nonlinear change (i.e., acceleration, deceleration) across time. These parameters are referred to as *growth factors* (Curran et al., 2010). Both the intercept and slope(s) can be either a fixed or random effect across population members, meaning that the slope(s) and/or intercept must remain equal across all population members, or may vary across individuals (Curran et al., 2010). The means of the growth factors represents the average trajectory of behavior across time, while the variances of the growth factors refers to the amount of heterogeneity in growth factors among individuals (Colder, Campbell, Ruel, Richardson, & Flay, 2002). Of note, conventional growth modeling assumes that all individuals are members of a single population, meaning that a single set of parameters adequately represents all members of the population (Jung & Wickrama, 2008).

The difference between conventional growth modeling and GMM is that GMM does not assume all individuals are members of a single population. Rather, GMM identifies unobserved heterogeneity in developmental trajectories, meaning that it identifies unique prototypical patterns in the intercepts and slopes of a behavior of interest measured over

time (Ram & Grimm, 2009). In essence, GMM identifies subpopulations of growth trajectories across time, and estimates individual variation around the mean growth curve for each individual subpopulation (B. O. Muthén & Muthén, 2000). As a result, each discrete subpopulation has unique sets of growth factors that best describe the average growth trajectories demonstrated by members of the respective subpopulations (Jung & Wickrama, 2008).

Before running the GMM, we first examined mean linear growth trajectories separately for each substance. We assessed four parameterizations of the mean growth trajectory model including trajectories where (1) the trajectory variance was set to zero, (2) the trajectory variance was free to vary, (3) the trajectory variance was set to zero and a quadratic term was added, and (4) the trajectory variance was allowed to freely vary and a quadratic term was added.

In using GMM to identify substance use trajectories across adolescence we conceptualize substance use trajectories as a latent categorical variable, wherein categories represent discrete substance use trajectories. Following our assessment of the mean trajectories, we explored heterogeneous trajectories of substance use. The best-fitting number of trajectories in GMM is identified in a similar way to the best-fitting number of classes in LPA. We conducted an iterative modeling procedure, specifying sets of models comparing a model with k to a model with $k-1$ classes (followed by comparing a model with $k+1$ classes to a model with k classes, etc.) and used the LMR-LRT, BLRT, and BIC to assess the relative fit of each subsequent model (Jung & Wickrama, 2008). We followed this procedure for trajectories of alcohol, cigarette, and marijuana use separately, identifying solutions with between one and four classes for each.

For each class solution (i.e., one through four classes), we tested models in which 1) the variance within class was allowed to vary, 2) the variance within class was fixed to equality, 3) the between class-variance was allowed to vary, and 4) the between class-variance was fixed to equality; moreover, we tested all combinations of these constraints

(e.g., the within-class variance was fixed to equality and the between-class variance was fixed to equality, etc.). Further, we also tested models in which a quadratic component was added to the linear model to assess whether a nonlinear model fit the data better than a linear model. We used BIC and SSA-BIC to determine the overall comparative fit of the models, and also used Wald Tests of Parameter constraints to determine whether within-class or between-class variability could be set to zero.

In addition to model fit statistics, we also considered interpretability and representativeness (e.g., what proportion of the sample does each trajectory represent) of the trajectories and fit with prior research on substance use trajectories before determining which model to use in the final stages of the analysis. Once the best-fitting number of classes had been identified, we plotted the average trajectories in each class while also plotting the variability around each trajectory. We repeated this procedure for trajectories of alcohol, cigarette, and marijuana use.

Aim 4.a. - Aim 4.b.

In the final aims of this analysis, we combined the analyses completed in *Aims 1-3* to identify the extent to which (1) conditional patterns of risk and protective factors (identified in *Aim 1.b.*) and (2) discrete patterns of etiological factors (identified in *Aim 2*) were differentially predictive of substance use trajectories across adolescence (identified in *Aim 3*). This allowed us to determine whether these discrete patterns are indeed associated with later substance use but also provided insight into whether conditional but separately-identified patterns of risk and protective factors result in similar predictions of later substance use as do combined (i.e., simultaneously estimated) patterns of etiological factors. All models discussed in *Aim 4* controlled for sex.

Aim 4.a. We utilized the dummy-coded variable representing the conditional membership in risk and protective factor profiles to predict (via multinomial logistic regression) the latent categorical variable representing substance use trajectories; we conducted this procedure on the alcohol, cigarette, and marijuana use trajectories, separately. We used odds ratios to examine the extent to which conditional associations between risk factor and protective factor profiles predicted likelihood of membership in substance use trajectories. For substances where a single growth trajectory was identified, we regressed the intercept and slope of the growth model on the dummy-coded variable representing conditional membership in risk and protective factor profiles using linear regression. We parameterized the model such that the conditional membership in risk factor patterns and protective factor patterns consisting of the highest levels of protective and/or lowest levels of risk factors served as the reference group, so that all other conditional memberships could be evaluated as likelihood of membership in substance use trajectories for individuals most likely to be members of patterns demonstrating high levels of risk (and/or low levels of protection) relative to those most likely to be members of patterns demonstrating low levels of risk (and/or high levels of protection).

Aim 4.b. We assessed the impact of etiological factor patterns identified in *Aim 2* in predicting substance use trajectories by constructing three multinomial logistic regression models to regress likelihood of demonstrating discrete alcohol, cigarette, and marijuana use trajectories (separately) on likelihood of demonstrating etiological factor patterns. We used odds ratios to assess the extent to which likelihood of demonstrating etiological factor patterns predicted likelihood of demonstrating substance use trajectories relative to a reference substance use trajectory. For substances where a single growth trajectory was identified, we regressed the intercept and slope of the growth model on the dummy-coded variable representing membership in etiological factor profiles using linear regression. We parameterized the model such that the prototypical pattern with the lowest frequencies of

risk factors (and/or highest frequencies of protective factors) was the reference profile for predicting substance use trajectories. This allowed for examination of the likelihood of demonstrating substance use trajectories conditional upon likelihood of demonstrating a prototypical pattern of etiological factors that represents on average greater theoretical risk for substance use relative to a pattern that represents on average lower risk.

CHAPTER FOUR

Results

All analyses were conducted in Mplus version 8.2 (L. K. Muthén & Muthén, 1998-2018) and SPSS version 25 (Corp., 2017). Full syntax for the final analyses is provided in Appendix B. Syntax for data management and sample selection is available upon request.

Data Reduction

We first conducted confirmatory factor analysis (CFA) to establish the extent to which the observed items were indicative of a latent etiological factor. In each of the CFA models, we specified a single factor with each item loading onto only one factor, and conducted the analysis using a weighted least square mean and variance adjusted estimator (Asparouhov & Muthén, 2010). The results of the CFAs for each etiological factor are presented in Table 4.1. Tables 4.2-4.9 provide correlation matrices of indicators within each etiological factor, and Table 4.10 presents the loadings of each item onto its latent construct.

Table 4.1

Results of Confirmatory Factor Analyses for Etiological Factors

Factor	$\chi^2(df)$	RMSEA	CFI	TLI	SRMR
Delinquency	36.05(9)	0.044	0.995	0.991	0.031
Exposure to violence	79.14(5)	0.097	0.950	0.900	0.067
Peer pressure	87.39(9)	0.075	0.944	0.990	0.025
Traumatic stress	61.74(14)	0.047	0.985	0.978	0.023
Neighborhood connectedness	40.16(9)	0.047	0.982	0.970	0.038
Parental knowledge	89.82(9)	0.076	0.969	0.948	0.044
Religiosity	0.00(0)	0.000	1.000	1.000	0.000
Self-worth	233.13(27)	0.070	0.876	0.835	0.078

Note. Fit statistics for neighborhood connectedness reflect the revised items. The CFA for religiosity was just-identified with three indicators. All chi-square statistics were significant at $p < .001$.

Table 4.2

Correlations Among Delinquency Indicators

Item	Carry gun	Carry knife	Pull knife or gun	Cut or shot someone	Been in a gang	Been arrested
Carry gun	1.000					
Carry knife	.384	1.000				
Pull knife or gun	.491	.471	1.000			
Cut or shot someone	.581	.383	.630	1.000		
Been in a gang	.355	.290	.372	.373	1.000	
Been arrested	.346	.213	.295	.269	.265	1.000

Note. All correlations were significant at $p < .001$.

Table 4.3

Correlations Among Exposure to Violence Indicators

Item	Gun/knife pulled	Cut or stabbed	Shot at	Family/friend cut/shot	Witnessed cut/shot
Gun/knife pulled	1.000				
Cut or stabbed	.361	1.000			
Shot at	.380	.450	1.000		
Family/friend cut/shot	.196	.139	.150	1.000	
Witnessed cut/shot	.296	.196	.189	.320	1.000

Note. All correlations were significant at $p < .001$.

Table 4.4

Correlations Among Peer Pressure Indicators

Item	Drinking	Doing drugs	Carrying weapon	Fighting	Do poorly in school	Having sex
Drinking	1.000					
Doing drugs	.707	1.000				
Carrying weapon	.683	.686	1.000			
Fighting	.383	.400	.395	1.000		
Do poorly in school	.528	.554	.533	.372	1.000	
Having sex	.525	.514	.524	.465	.502	1.000

Note. All correlations were significant at $p < .001$.

Table 4.5

Correlations Among Traumatic Stress Indicators

Item	Bad dreams	Trouble sleeping	Feel better if talk	Bad things happen to friend	Can't stop ruminating	Feel uncomfortable	Worry about bad things
Bad dreams	1.000						
Trouble sleeping	.425	1.000					
Feel better if talk	.295	.315	1.000				
Bad things happen to friend	.304	.300	.249	1.000			
Can't stop ruminating	.311	.370	.323	.365	1.000		
Feel uncomfortable	.280	.285	.276	.299	.277	1.000	
Worry about bad things	.239	.285	.249	.243	.291	.276	1.000

Note. All correlations were significant at $p < .001$.

Table 4.6

Correlations Among Neighborhood Connectedness Indicators

Item	Important part	Sorry to leave	Friends depend	Have people	Have friends	People talk to
Important part	1.000					
Sorry to leave	.223	1.000				
Friends depend	.188	.261	1.000			
Have people	.182	.226	.386	1.000		
Have friends	.211	.206	.385	.338	1.000	
People talk to	.250	.207	.266	.247	.351	1.000

Note. All correlations were significant at $p < .001$.

Table 4.7

Correlations Among Religiosity Indicators

Item	Attend religious activites	Importance of religion	Read or study Bible
Attend religious activites	1.000		
Importance of religion	.182	1.000	
Read or study Bible	.320	.113	1.000

Note. All correlations were significant at $p < .001$.

Table 4.8

Correlations Among Self-Worth Indicators

Item	Happy with self	Do things I should	Like the way I behave	Like the person I am	Don't get into trouble	Make good decisions	Behave myself	Happy with how I do things	Like the way I live
Happy with self	1.000								
Do things I should	.001*	1.000							
Like the way I behave	.131	.136	1.000						
Like the person I am	.204	.041*	.185	1.000					
Don't get into trouble	.044*	.274	.182	.037	1.000				
Make good decisions	.113	.137	.086	.212	.134	1.000			
Behave myself	.113	.105	.101	.202	.101	.312	1.000		
Happy with how I do things	.171	.169	.301	.167	.215	.186	.151	1.000	
Like the way I live	.231	.151	.233	.247	.155	.189	.177	.271	1.000

Note. * indicates correlations where $p > .05$. All other correlations were significant at $p < .001$.

Table 4.9

Correlations Among Parental Knowledge Indicators

Item	Who hang out with	Where you are afternoon	What you do afternoon	Where you go at night	Try to know how spend time	Know how spend time
Who hang out with	1.000					
Where you are afternoon	.230	1.000				
What you do afternoon	.267	.320	1.000			
Where you go at night	.183	.262	.391	1.000		
Try to know how spend time	.157	.181	.248	.229	1.000	
Know how spend time	.244	.277	.433	.359	.454	1.000

Note. All correlations were significant at $p < .001$.

Table 4.10

Factor Loadings for Etiological Factors

Item	Delinquency	Exposure to violence	Peer pressure	Traumatic stress	Neighborhood connectedness	Parental knowledge	Religiosity	Self-worth
Item 1	.835	.771	.920	.649	.496	.542	.736	.457
Item 2	.736	.777	.927	.669	.532	.600	.341	.406
Item 3	.931	.807	.914	.575	.772	.729	.524	.546
Item 4	.912	.513	.656	.600	.710	.659		.557
Item 5	.672	.626	.801	.667	.766	.600		.461
Item 6	.582		.802	.565	.644	.815		.557
Item 7				.528				.665
Item 8								.678

We evaluated the fit of each model before assessing the pattern of item loadings. While all chi-square statistics were significant at $p < .001$, indicating a lack of model fit to the data, additional fit indices are always included due to important criticisms of chi-square as a sole indicator of model fit (see T. A. Brown, 2014, p. 81). We utilized criteria from Hu and Bentler (1999) in evaluating model fit, where SRMR less than .08, RMSEA less than .05, and CFI and TLI greater than .95 indicate good fit. Based on all three criteria, the factor models for delinquency and traumatic stress had good fit; the models for exposure to violence (CFI, SRMR), peer pressure (TLI, SRMR), and parental knowledge (CFI, SRMR) demonstrated good fit based on at least two indicators, always including at least one indicator of absolute fit (SRMR). The model for religiosity was just-identified given that this construct was only reflected in three items, but these three items reflect face validity of overall religiousness and are similar to items used in other studies of religiousness and drug use in large secondary datasets (Agrawal et al., 2017; Meyers, Brown, Grant, & Hasin, 2017).

All models were constructed such that factor variance was set to 1 so item loadings could be completely free to vary. Table 4.10 provides the factor loadings for all items onto their respective construct. Four of six delinquency items, three of five exposure to violence items, five of six peer pressure items, two of six parental knowledge items, and one of three religiosity items had loadings greater than .7. Item loadings greater than .7 are typically considered acceptable in factor analysis. While a number of items had factor loadings lower than this threshold, we retained these items primarily because model fit was still adequate with the retention of these items. Additionally, we wanted to retain all items when possible to remain consistent with other research conducted using these indicators from the MYS. While none of the traumatic stress indicators had loadings that met this threshold, three of the items had R^2 values greater than .4, internal consistency was acceptable, model fit was adequate, and the factor loadings did not suggest the presence of multiple distinct factors (i.e., factor loadings ranged from .528 to .669).

We initially conceptualized neighborhood connectedness as a composite of 11 items. However, an initial CFA found poor fit to the data when utilizing all 11 items ($\chi^2(44) = 1140.94, p < .001$, RMSEA = 0.126 (90% CI [0.120,0.133]), CFI = 0.550, TLI = 0.438, SRMR = .135). There were two items that had negative loadings and a number of items with very poor loadings onto the broad construct. We anticipated that this may be a method effect, as the indicators with poor and/or negative loadings were worded in a negative manner (e.g., "I do not like living in my neighborhood"). We then included only the six positively worded items and re-ran the model with similarly poor results ($\chi^2(10) = 419.57, p < .001$, RMSEA = 0.162 (90% CI [0.149,0.176], CFI = 0.761, TLI = 0.641, SRMR = 0.138). However, we noticed a convergence issue in this model where, although we set the factor variance to be 1, the model also attempted to set the first indicator loading to 1 as well. We provided a starting value of .6 for the loading of the first indicator and re-ran the model, obtaining a similar pattern of factor loadings across the indicators, but a loading of the first indicator that was not equal to 1. (see Table 4.10 for factor loadings) When we made this adjustment, the model fit was acceptable (as is presented in Table 4.1).

The self-worth factor did not have good fit to the data across multiple criteria as seen in Table 4.1. Examinations of the item correlations in Table 4.8 demonstrated moderate associations across items, and loadings in Table 4.10 also demonstrated moderate associations, even though factor loadings were somewhat small. Further, the included indicators had an internal consistency that is generally lower than considered acceptable (.65) but also was lower than provided in the validation study (.73-.82, depending on the sample; Harter, 1982). Despite an acceptable internal consistency reported in the validation study, the study did not specify the racial/ethnic breakdown of the sample participants (Harter, 1982). As mentioned in the previous chapter, other studies using the same measure in African American adolescent samples have also had low levels of internal consistency (e.g., Copeland-Linder et al., 2011). Given that this measure was available in the MYS dataset and that our

internal consistency was aligned with previous research, we chose to retain this measure for use in further analyses, despite the less-than-perfect model fit.

Aim 1.a.

Following identification of the factor structure for each etiological factor, we extracted factor scores using a maximum posteriori estimation (L. K. Muthén & Muthén, 1998-2018). We then conducted latent profile analysis (LPA) on the risk factors and protective factors separately. Model fit statistics for the protective factor models and risk factor models are provided in Table 4.11 and Table 4.12, respectively.

We selected the three-profile model when examining unique patterns of protective factors due to a nonsignificant likelihood ratio test when moving from three to four profiles (see Table 4.11). Entropy values greater than .7 are considered acceptable (Celeux & Soromenho, 1996), so we classified individuals into groups based on their highest posterior probability of membership and examined mean factor scores of the protective factor indicators among individuals most likely to be members of each pattern. Figure 4.1 provides a graphical depiction of these protective factor patterns. While we provide labels to describe and distinguish the factor score patterns, we emphasize (1) that the labels apply to the pattern of factor scores, *not* the individuals who are most likely to demonstrate these patterns, and (2) the probabilistic nature of these techniques (that is, every individual has a likelihood of demonstrating all factor score patterns, but are classified into their highest probability pattern).

The three protective factor patterns were primarily characterized by differences in levels of parental knowledge. Individuals most likely to demonstrate the *high parental knowledge* (n = 695, 44% of the sample) pattern on average exhibited levels of parental knowledge nearly three-fourths of a standard deviation above the full sample mean, with levels of neighborhood connectedness, religiosity, and self-worth near sample mean levels.

Table 4.11

Model Fit Statistics for Protective Factor Latent Profile Analysis

Profiles	BIC	SSA-BIC	Entropy	VLMR-LRT	*p*	LMR-ALRT	*p*
2	14045.239	14003.941	0.695	314.900	.0000	306.566	.0000
3	13971.507	13914.325	0.769	110.516	.0074	107.591	.0082
4	13901.515	13828.449	0.810	106.776	.4200	103.951	.4259

Note. BIC = Bayesian Information Criteria, SSA-BIC = Sample Size-Adjusted Bayesian Information Criteria, VLMR-LRT = Vuong-Lo-Mendell-Rubin Likelihood Ratio Test, LMR-ALRT = Lo-Mendell-Rubin Adjusted Likelihood Ratio Test.

Individuals most likely to demonstrate the *average protection* pattern ($n = 675$, 43%) on average were characterized by average levels of neighborhood connectedness, religiosity, and self-worth that were near sample mean levels, and with average parental knowledge levels nearly one-half standard deviation below the mean. Individuals most likely to demonstrate the *low protection* pattern ($n = 197$, 13%) on average showed levels of parental knowledge that were nearly one and a half standard deviations below the sample mean and also levels of self-worth that were nearly one-half standard deviations below the full sample mean.

We selected the six-profile model when examining prototypical patterns of risk factors (see Table 4.12). Although BIC, SSA-BIC, entropy, VLMR-LRT, and LMR-ALRT all continued to indicate a seven-profile model may fit the data, one of these seven profiles only best characterized 3.5% of the sample (i.e., only 3.5% of the sample had a highest probability of demonstrating this profile; see Appendix C). Convention suggests a pattern may not be adequately representative of a unique subgroup when that pattern characterizes less than 5% of a sample (Collins & Lanza, 2010), and, as such, we selected the six-profile model.

Broadly, the six risk factor patterns were distinguished by comparative levels of delinquency, exposure to violence, and peer pressure (see Figure 4.2). Individuals most likely to demonstrate the *low-risk* pattern ($n = 688$, 43%) had average levels of all risk factors at or below full sample mean levels; specifically, on average, delinquency levels

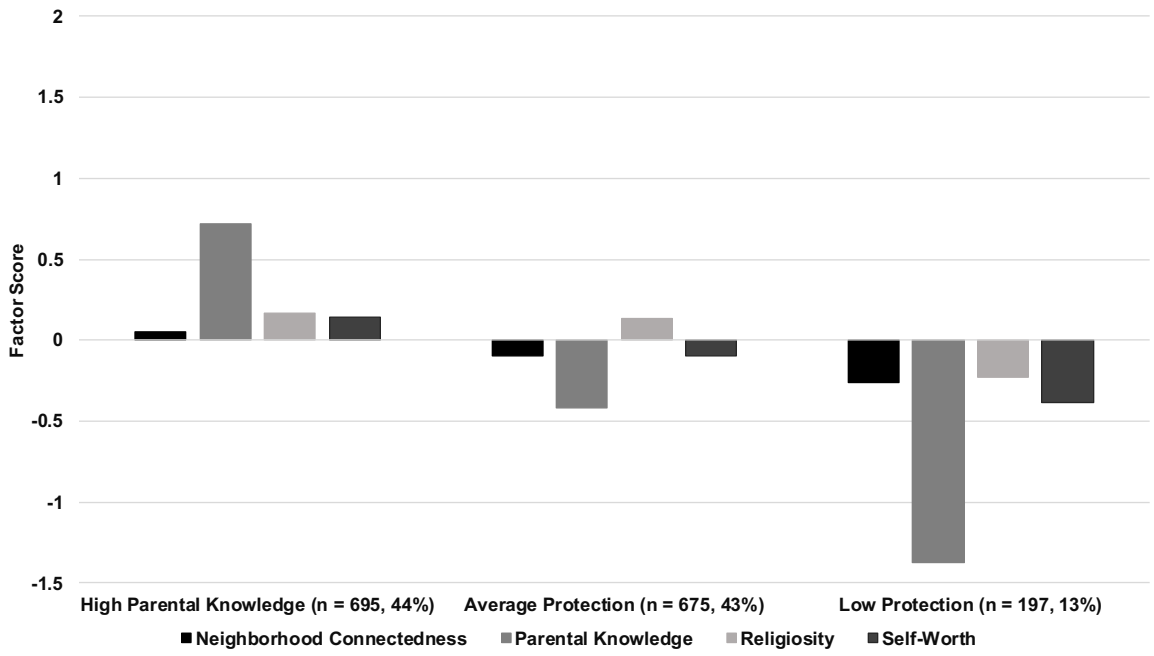

Figure 4.1. Mean factor scores for protective factors among individuals most likely to be members of each latent protective factor profile.

were just over one-half standard deviation below the full sample mean. Those most likely to demonstrate the *average risk* (n = 274, 17%) and *some delinquency* (n = 169, 11%) patterns were all characterized by average levels of all risk factors near mean levels. However, individuals most likely to demonstrate the *some delinquency* pattern were distinguished by average levels of delinquency nearly one-half standard deviation above the mean. Individuals most likely to demonstrate the *elevated delinquency* pattern (n = 167, 11%) were primarily characterized by average levels of delinquency over one-half standard deviations above the sample mean, and also by slightly elevated exposure to violence levels just under one-half standard deviation above the sample mean. Individuals most likely to demonstrate the *delinquency & violence-exposure* pattern (n = 146, 9%) were primarily characterized by average delinquency levels over one full standard deviation higher than the sample mean and average exposure to violence levels higher than one-half standard deviation above the full sample mean. Finally, the *multi-risk* pattern (n = 123, 8%) best characterized the smallest portion of the full sample, and demonstrated average levels of delinquency greater than

Table 4.12

Model Fit Statistics for Risk Factor Latent Profile Analysis

Profiles	BIC	SSA-BIC	Entropy	VLMR-LRT	*p*	LMR-ALRT	*p*
2	13371.55	13330.25	0.86	1177.75	.0000	1146.58	.0000
3	12974.81	12917.63	0.88	433.53	.0016	422.05	.0018
4	12371.03	12297.96	0.96	640.56	.0000	623.61	.0000
5	11865.69	11776.74	0.97	542.12	.0056	527.78	.0063
6	11548.04	11443.21	0.98	345.44	.0000	345.06	.0000
7	11142.97	11022.25	0.99	441.86	.0000	430.16	.0000

Note. BIC = Bayesian Information Criteria, SSA-BIC = Sample Size-Adjusted Bayesian Information Criteria, VLMR-LRT = Vuong-Lo-Mendell-Rubin Likelihood Ratio Test, LMR-ALRT = Lo-Mendell-Rubin Adjusted Likelihood Ratio Test.

one-and-a-half standard deviations above the full sample mean, average levels of exposure to violence greater than one standard deviation above the full sample mean, and average peer pressure levels greater than one-half standard deviation above the full sample mean. With the exception of traumatic stress, individuals most likely to demonstrate the *multi-risk* pattern exhibits levels of risk factors at levels one to one-half standard deviations higher than those most likely to be members of the delinquency & violence exposure pattern.

Aim 1.b.

Frequencies of the 18 conditional profile membership (i.e., overlap between highest posterior probability risk factor pattern and highest posterior probability protective factor pattern) are presented in Table 4.13. The most common conditional profile memberships consisted of those most likely to demonstrate the *high parental knowledge* and *low risk* patterns (n = 376, 24.0%) and those most likely to demonstrate the *average protection* and *low risk* patterns (n = 255, 16.3%), followed by those most likely to demonstrate the *high parental knowledge* and *average risk* patterns (n = 127, 8.1%), and those most likely to demonstrate the *average protection* and *average risk* patterns (n = 120, 7.7%). The least endorsed patterns, comprising less than 2% of the sample, included those most

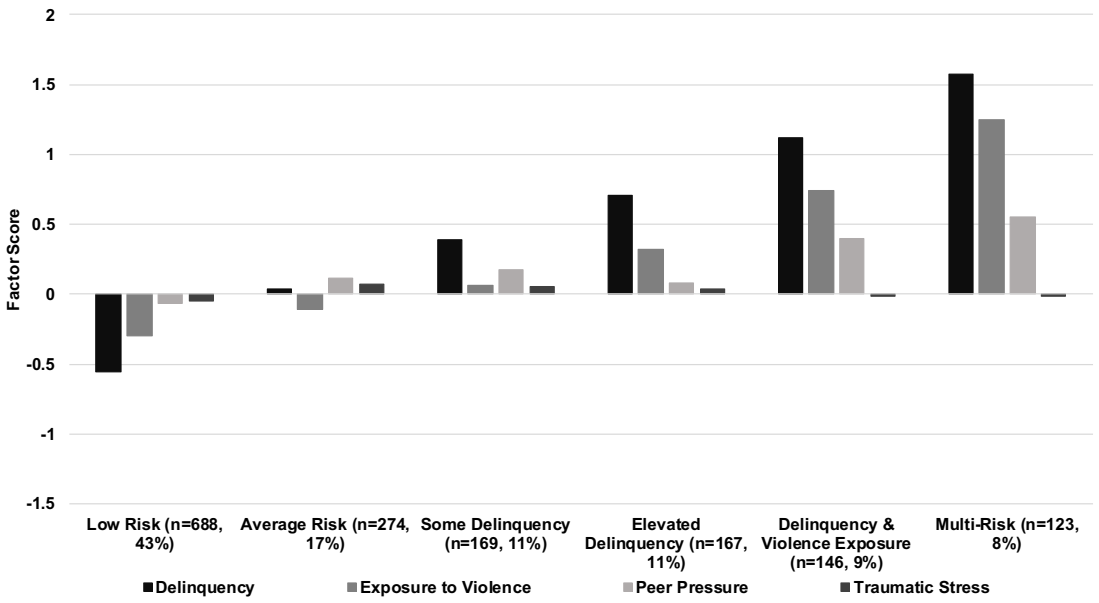

Figure 4.2. Mean factor scores for risk factors among individuals most likely to be members of each latent risk factor profile.

likely to demonstrate the *high parental knowledge* and *multi-risk patterns* (*n* = 22, 1.4%), those most likely to demonstrate the *low protection* and *some delinquency* patterns (*n* = 22, 1.4%) those most likely to demonstrate the *low protection* and *average risk patterns* (*n* = 27, 1.7%), those most likely to demonstrate the *low protection* and *delinquency & violence exposure* patterns (*n* = 27, 1.7%), and those most likely to demonstrate the *low protection* and *elevated delinquency* patterns (*n* = 29, 1.9%). The other nine conditional pattern combinations each comprised between 2.2%-5.2% of the sample.

Table 4.13

Conditional Profile Membership Based on Highest Posterior Probability of Membership in Protective Factor and Risk Factor Profiles

	Protective factor pofiles			
Risk factor profiles	High parental knowledge	Average protection	Low protection	Total
Low risk	376 (24.0%)	255 (16.3%)	57 (3.6%)	688 (43.9%)
Average risk	127 (8.1%)	120 (7.7%)	27 (1.7%)	274 (17.5%)
Some delinquency	71 (4.5%)	76 (4.9%)	22 (1.4%)	169 (10.8%)
Elevated delinquency	57 (3.6%)	81 (5.2%)	29 (1.9%)	167 (10.7%)
Delinquency & violence exposure	42 (2.7%)	77 (4.9%)	27 (1.7%)	146 (9.3%)
Multi-risk	22 (1.4%)	66 (4.2%)	35 (2.2%)	123 (7.8%)
Total	695 (44.4%)	675 (43.1%)	197 (12.6%)	1,567 (100%)

Aim 2

Results from the LPA on all eight etiological factors are available in Table 4.14. BIC and SSA-BIC continually decreased while entropy increased across the two through seven profile models, and the VLMR-LRT and LMR-ALRT also suggested that the extraction of each additional profile had better fit to the data compared to a model with one fewer profiles. However, we elected to retain the six-profile model because one of the profiles in the seven-profile model best characterized only 3% of the total sample, indicating that the profile may not characterize a meaningful portion of the sample (Collins & Lanza, 2010, see Appendix C).

Table 4.14

Model Fit Statistics for Combined Etiological Factor Latent Profile Analysis

Profiles	BIC	SSA-BIC	Entropy	VLMR-LRT	*p*	LMR-ALRT	*p*
2	27483.32	27403.90	0.86	1418.76	.0000	1397.65	.0000
3	27073.75	26965.74	0.88	475.78	.0002	468.70	.0002
4	26507.73	26371.13	0.96	632.24	.0000	622.83	.0000
5	26024.71	25859.52	0.97	549.23	.0421	541.061	.0436
6	25729.21	25535.43	0.98	361.71	.0000	356.33	.0000
7	25352.67	25130.29	0.99	442.76	.0000	436.17	.0000

Note. BIC = Bayesian Information Criteria, SSA-BIC = Sample Size-Adjusted Bayesian Information Criteria, VLMR-LRT = Vuong-Lo-Mendell-Rubin Likelihood Ratio Test, LMR-ALRT = Lo-Mendell-Rubin Adjusted Likelihood Ratio Test.

The etiological factor patterns are graphically depicted in Figure 4.3. The largest portion of the sample was characterized by the *low risk/average protection* pattern (n = 688, 43.9%). This pattern represented mean levels of delinquency less than one-half standard deviation below the full sample mean and mean levels of all other etiological factors within one-half standard deviation of the sample mean. The next largest portion of the sample was characterized by the *average risk and protection* pattern (n = 274, 17.5%), which demonstrated levels of all eight etiological factors that were near the full sample mean. The *some*

delinquency/average protection pattern (n = 169, 10.8%) was characterized by mean delinquency levels that were slightly high, but less than one-half standard deviation above the full sample mean, and mean levels of all other etiological factors that were near the full sample mean. The *elevated risk/decreased protection* pattern (n = 167, 10.7%) was characterized by mean levels of delinquency that were greater than one-half standard deviation above the full sample mean, mean levels of exposure to violence that were higher than those demonstrated by individuals most likely to be members of the *some delinquency/average protection* pattern, and mean levels of parental knowledge and self-worth that were slightly lower than those most likely to be members of the *some delinquency/average protection* pattern. The *moderate risk/decreased protection* pattern (n = 147, 9.4%) was characterized by mean delinquency levels greater than one full standard deviation above the full sample mean, mean exposure to violence levels greater than one-half standard deviation above the full sample mean, and mean peer pressure levels that were greater than those exhibited by individuals most likely to be members of the *elevated risk/decreased protection* profile. Further, individuals most likely to demonstrate the *moderate risk/decreased protection* profile were also likely to demonstrate mean levels of neighborhood connectedness, parental knowledge, and self-worth that were lower than individuals most likely to be members of the *elevated risk/decreased protection* profile. Finally, the *high risk/low protection* pattern (n = 122, 7.8%) characterized the smallest portion of the sample, and represented mean levels of delinquency that were one-and-a-half standard deviations above the full sample mean, mean exposure to violence levels that were greater than one full standard deviation above the sample mean, and mean levels of peer pressure that were at least one-half standard deviation above the full sample mean. Additionally, individuals most likely to be members of the *high risk/low protection* profile were likely to demonstrate mean levels of parental knowledge that were over one-half standard deviations below the sample mean, but mean levels of self-worth and neighborhood connectedness that were comparable to those

demonstrated by individuals most likely to be members of the *moderate risk/decreased protection* profile.

Aim 3

Frequencies of alcohol, cigarette, and marijuana use across the five time points of interest are available in Table 4.15. Alcohol, cigarette, and marijuana use generally increased across ages 13-17. Alcohol was more commonly used overall than cigarettes or marijuana, but marijuana was more commonly used relative to cigarettes as the sample aged. By age 17, nearly half of the overall sample had endorsed alcohol use, 30% endorsed cigarette use, and 43% endorsed marijuana use.

Table 4.15

Proportion of Sample Endorsing Alcohol, Cigarette, and Marijuana Use Across Ages 13-17

Age	Frequency of Use	Alcohol	Cigarette	Marijuana
13	No	64.8	79.2	81.4
	Yes, just once	22.3	14.7	10.3
	Yes, more than once	13.0	6.1	8.3
14	No	62.1	78.3	72.0
	Yes, just once	23.1	13.5	13.2
	Yes, more than once	14.8	8.2	14.8
15	No	54.5	78.0	66.9
	Yes, just once	27.7	12.3	13.9
	Yes, more than once	17.8	9.7	19.3
16	No	53.2	72.9	62.3
	Yes, just once	27.3	13.3	15.5
	Yes, more than once	19.5	13.8	22.2
17	No	50.7	70.4	57.0
	Yes, just once	25.0	12.3	14.7
	Yes, more than once	24.2	17.3	28.4

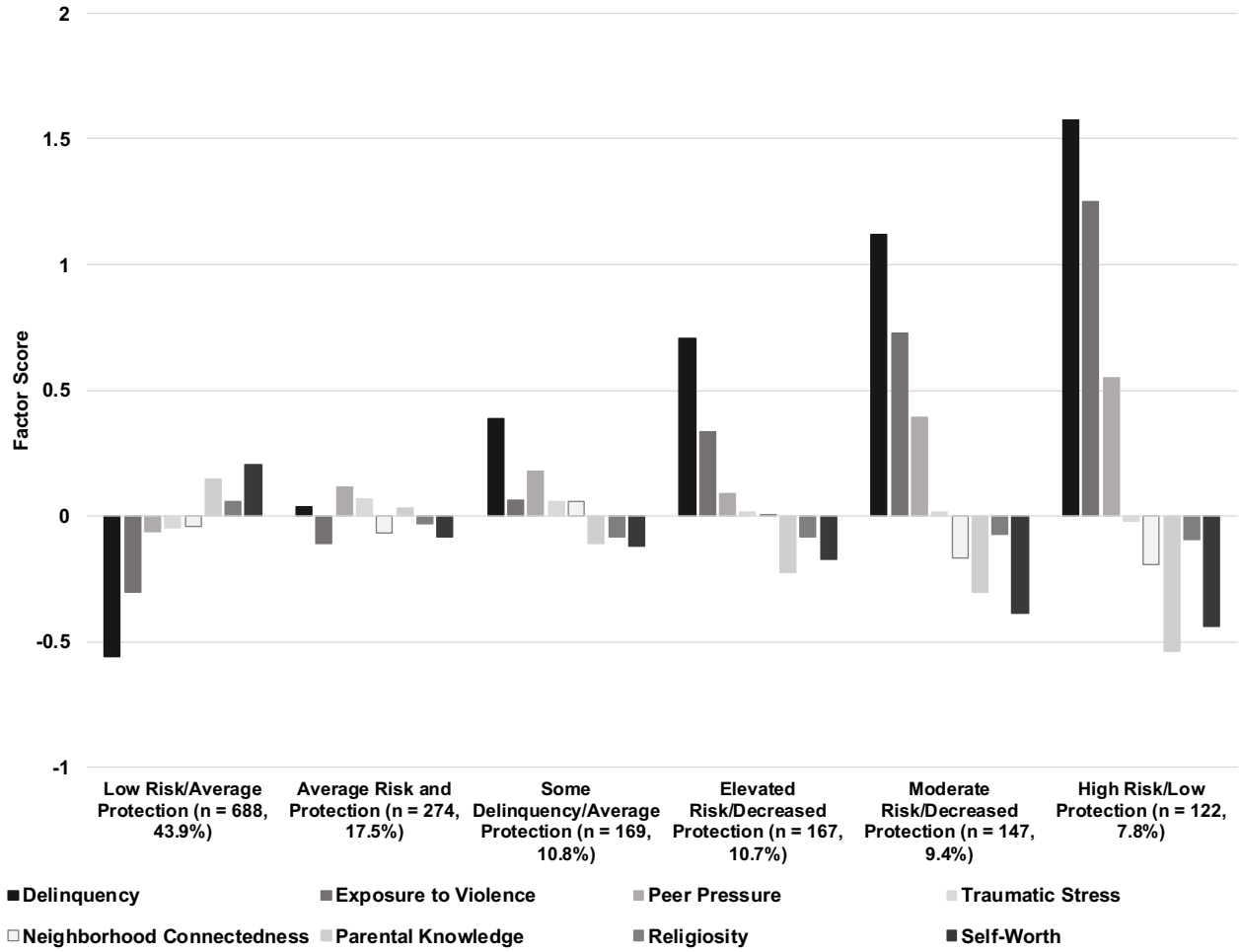

Figure 4.3. Mean factor scores for all etiological factors among individuals most likely to be members of each latent etiological factor profile.

Below we present the results of the growth model analyses conducted according to the procedure outlined in Ram and Grimm (2009), wherein we first identify the best-fitting mean growth trajectory and then add a mixture component to determine the presence of latent heterogeneity in trajectories of alcohol, cigarette, and marijuana use, separately. We did test alternative parameterizations of growth mixture models (GMMs) as outlined in the Analytic Strategy, including variations in freeing or fixing variance within- or between-classes. Results of all tested GMMs are available in Appendix C. Item thresholds for each substance were held equal across all time points (but different substances had different item thresholds) to hold measurement of a specific substance use invariant across time; this allowed us to assess how use of a specific substance changed over time using a consistent metric for use. In singular growth trajectories, both the intercept and slope were freely estimated while item thresholds were fixed to equality across time within the same substance.

We first examined mean trajectories of alcohol use, assessing (1) whether freely estimated variance (compared to variance fixed to equality) across individuals and (2) a nonlinear component (compared to a linear component) best fit the data. A linear model wherein variance was freely estimated across individuals had better fit to the data (BIC = 9205.64, SSA-BIC = 9186.58) than a linear model with fixed variance across individuals (BIC = 9293.80, SSA-BIC = 9584.27), a quadratic model with freely estimated variance (BIC = 9225.78, SSA-BIC = 9193.99), and a quadratic model with variance fixed to equality across individuals (BIC = 9300.71, SSA-BIC = 9288.00). We then examined whether multiple alcohol use trajectories were present with freely estimated variance. The results of the two- and three-class models are available in Table 4.16. We determined that a two-class linear trajectory model wherein variances within class (i.e., intercept variance and slope variance) were allowed to freely vary, but variances between classes could be fixed to equality, best fit the data. When compared to the three-class model, the two-class model demonstrated

lower BIC and SSA-BIC. While the *p*-values for the LMR-ALRT and VLMR-LRT suggested that a third class may be extracted, that third class comprised less than 5% of the total sample.

Table 4.16

Model Fit Statistics for Alcohol Use Growth Mixture Model

Profiles	BIC	SSA-BIC	Entropy	VLMR-LRT	*p*	LMR-ALRT	*p*
2	9168.52	9139.92	0.78	59.20	.0001	56.63	.0001
3	9182.23	9144.11	0.73	8.35	.0028	7.99	.0035

Note. BIC = Bayesian Information Criteria, SSA-BIC = Sample Size-Adjusted Bayesian Information Criteria, VLMR-LRT = Vuong-Lo-Mendell-Rubin Likelihood Ratio Test, LMR-ALRT = Lo-Mendell-Rubin Adjusted Likelihood Ratio Test.

We plotted the average trajectories of individuals most likely to be members of each latent trajectory to determine the average characteristics (see Figure 4.4; estimated growth parameters for each class are presented in Table 4.17). The first trajectory demonstrated an *average use* pattern across time and best characterized the majority of the sample (*n* = 1,490, 95.1%). The second trajectory demonstrated a *high use* pattern across ages 13-17, with over 90% of individuals most likely to be members of this trajectory endorsing alcohol use across ages 13-17. This trajectory best characterized a small, yet representative, proportion of the sample (*n* = 76, 4.9%).

We followed the same procedure when examining models for cigarette use, first examining the mean trajectory for cigarette use. A linear model wherein variance was freely estimated across individuals had better fit to the data (BIC = 6569.23, SSA-BIC = 6577.17) than a linear model with fixed variance across individuals (BIC = 6833.66, SSA-BIC = 6824.13), a quadratic model with freely estimated variance (BIC = 6618.86, SSA-BIC = 6587.09), and a quadratic model with variance fixed to equality across individuals (BIC = 6838.73, SSA-BIC = 6826.02). We then examined whether multiple cigarette use trajectories were present with freely estimated variances. A two-class model did not fit the data

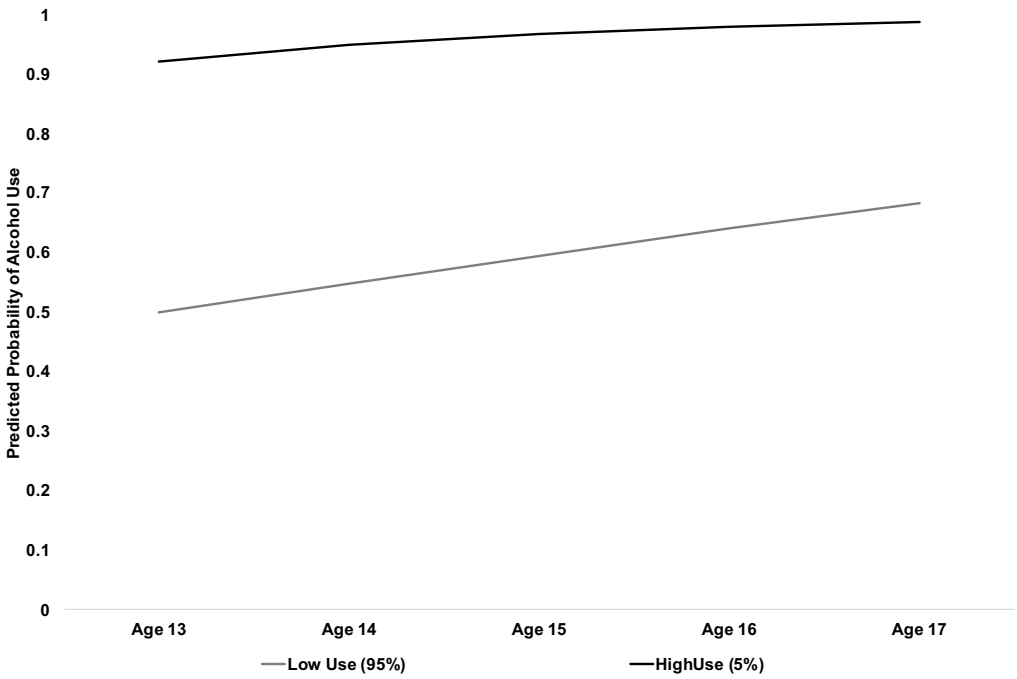

Figure 4.4. Predicted probabilities of engaging in any alcohol use among individuals most likely to demonstrate each latent trajectory.

better than a single, mean trajectory when variances were freely estimated within class but were fixed to equality between classes; relaxing the equality of variances between class restriction similarly did not indicate a better fit to the data with a two-class model compared to a single trajectory (see Table 4.18). As such, we retained a model consisting of a mean growth trajectory allowing for individual variation in the intercept and slope.

We once again followed an identical procedure when testing models for marijuana use. As with cigarette use, a linear model wherein variance was freely estimated across individuals had better fit to the data (BIC = 7630.97, SSA-BIC = 7611.91) than a linear model with fixed variance across individuals (BIC = 7857.23, SSA-BIC = 7847.70), a quadratic model with freely estimated variance (BIC = 7639.93, SSA-BIC = 7608.16), and a quadratic model with variance fixed to equality across individuals (BIC = 7858.95, SSA-BIC = 7846.25). We then examined whether multiple trajectories were present with freely estimated variances. While a two-class model with freely estimated variances both within- and between-classes did fit the data better than a one-class model (based on the

Table 4.17

Estimated Growth Parameters for Alcohol Use Trajectories

Parameter	Average Use Class	High Use Class
Intercept	0.00(0.00)	2.47(0.31)***
Intercept variance	0.84(0.30)**	0.84(0.30)**
Linear slope	0.19(0.03)***	0.47(0.27)
Linear slope variance	0.21(0.05)****	0.21(0.05)***

Note. $^{**}p < .01$, $^{***}p < .001$. Standard errors in parentheses. To aid in model identification, the intercept in the *average use* class was fixed at zero.

Table 4.18

Model Fit Statistics for Two-Class Cigarette Use Growth Mixture Model

BC Variance	BIC	SSA-BIC	Entropy	VLMR-LRT	*p*	LMR-ALRT	*p*
Fixed	6611.49	6582.90	0.87	6.80	.1082	6.51	.1187
Free	6622.34	6587.39	0.75	10.67	.5051	10.38	.5129

Note. BC Variance = Between Class Variance, BIC = Bayesian Information Criteria, SSA-BIC = Sample Size-Adjusted Bayesian Information Criteria, VLMR-LRT = Vuong-Lo-Mendell-Rubin Likelihood Ratio Test, LMR-ALRT = Lo-Mendell-Rubin Adjusted Likelihood Ratio Test.

VLMR-LRT and the LMR-ALRT; see Table 4.19), one of the classes best characterized less than 5% of the sample. As such, we retained a model consisting of a mean growth trajectory allowing for individual variation in the intercept and slope.

The growth parameters for the mean cigarette and marijuana trajectories are presented in Table 4.20.

Aim 4.a.

In the first part of the fourth aim we used conditional profile membership (based on individuals' specific combination of most likely latent profile membership in risk factor and protective factor profiles) to predict likelihood of demonstrating alcohol use trajectories and to predict growth parameters of the cigarette and marijuana use trajectories. The

Table 4.19

Model Fit Statistics for Two-Class Marijuana Use Growth Mixture Model

BC Variance	BIC	SSA-BIC	Entropy	VLMR-LRT	*p*	LMR-ALRT	*p*
Fixed	7642.50	7613.91	0.88	10.54	.0692	10.08	.0770
Free	7656.42	7621.47	0.86	11.33	.0212	11.03	.0238

Note. BC Variance = Between Class Variance, BIC = Bayesian Information Criteria, SSA-BIC = Sample Size-Adjusted Bayesian Information Criteria, VLMR-LRT = Vuong-Lo-Mendell-Rubin Likelihood Ratio Test, LMR-ALRT = Lo-Mendell-Rubin Adjusted Likelihood Ratio Test.

Table 4.20

Estimated Growth Parameters for Cigarette, and Marijuana Use Trajectories

Parameter	Cigarette	Marijuana
Intercept	-0.81(0.12)***	-1.15(0.11)***
Intercept variance	1.20(0.38)**	1.50(0.40)***
Linear slope	0.04(0.05)	0.36(0.04)***
Linear slope variance	0.32(0.08)***	0.28(0.07)***

Note. $^{**}p < .01$, $^{***}p < .001$. Standard errors in parentheses.

results of the analysis using conditional risk/protective factor profile membership to predict likelihood of membership in alcohol use trajectories is available in Table 4.21[1] . There were no significant sex differences in likelihood of demonstrating the *high use* alcohol pattern relative to the *average use* pattern ($b = -0.37, SE = 0.25, p = .134, OR = .69$). We used the *low risk/high parental knowledge* profile as the reference group for the conditional risk/protective factor profiles in predicting substance use trajectories. Generally, those who were most likely to be members of the *low risk* or *average risk* profiles were less likely to be members of the *high use* trajectory regardless of their most likely latent protective factor pattern; however, those who were most likely to be members of the *low risk/average protection* and *average risk/average protection* patterns were more likely to demonstrate

[1] Appendix C provides raw frequencies of substance use at ages 13-17 among individuals likely to demonstrate conditional factor patterns and among individuals likely to demonstrate etiological factor patterns.

Table 4.21

Most Likely Alcohol Use Trajectory Membership Predicted by Conditional Profile Membership

	Protective Factor Profiles					
	High Parental Knowledge		Average Protection		Low Protection	
Risk Factor Profile	*B(SE)*	*OR*	*B(SE)*	*OR*	*B(SE)*	*OR*
Low risk	Ref.	Ref.	1.00(0.49)*	2.71	1.07(0.71)	2.91
Average risk	1.02(0.57)	2.77	1.23(0.55)*	3.41	0.88(1.10)	2.42
Some delinquency	0.93(0.71)	2.53	1.19(0.66)	3.30	0.98(1.10)	2.66
Elevated delinquency	1.14(0.72)	3.12	0.39(0.82)	1.48	1.89(0.73)**	6.63
Delinquency & violence exposure	1.04(0.83)	2.48	1.91(0.55)***	6.74	0.81(1.10)	2.25
Multi-risk	2.30(0.75)**	10.00	2.13(0.56)***	8.37	1.34(0.85)	3.82

Note. $*p < .05$, $**p < .01$, $***p < .001$. The *low risk/high parental knowledge* profile served as the reference category for the predictors, while the *average use* trajectory served as the reference category for the outcome.

the *high use* alcohol trajectory relative to the *average use* trajectory. Four other profile combinations had significantly higher odds (relative to those most likely to demonstrate the *low risk/high parental knowledge* patterns) of demonstrating the *high use* alcohol trajectory relative to the *average use*, including the *elevated delinquency/low protection*, *delinquency & violence exposure/average protection*, *multi-risk/high parental knowledge*, and *multi-risk/average protection* patterns.

Results for the analysis using the conditional risk factor/protective factor profile membership in predicting trajectories of cigarette use while controlling for sex are available in Table 4.22. There were no significant sex differences in initial levels of cigarette use ($b = -0.21, SE = 0.14, p = .151$), but there were significant differences in the slope ($b = 0.38, SE = 0.07, p < .001$) with men having a greater increase in use across adolescence. Among those most likely to demonstrate the *average protection* pattern, individuals also most likely to demonstrate the *delinquency & violence exposure* or *multi-risk* patterns had higher initial levels of use relative to those most likely to demonstrate the *low risk/high parental knowledge* patterns. Among those most likely to demonstrate the *high parental knowledge* pattern, only those also most likely to demonstrate the *multi-risk* pattern had higher initial levels of use relative to those most likely to demonstrate the *low risk/high parental knowledge* patterns. Finally, among those most likely to demonstrate the *low protection* pattern, those who were also most likely to demonstrate the *elevated delinquency*, *delinquency & violence exposure*, or *multi-risk* patterns had higher levels of initial use relative to those most likely to demonstrate the *low risk/high parental knowledge* patterns. There were no significant differences in any conditional profile memberships relative to those most likely to demonstrate the *low risk/high parental knowledge* pattern in the rate of linear change in use across time.

Results for the analysis using the conditional risk factor/protective factor profile membership in predicting trajectories of marijuana use while controlling for sex are available in Table 4.23. Echoing the results seen for cigarette use, there were no significant

Table 4.22

Cigarette Use Growth Parameters Predicted by Conditional Profile Membership

	Protective Factor Profiles					
	High Parental Knowledge		Average Protection		Low Protection	
Risk Factor Profile	*i*	*s*	*i*	*s*	*i*	*s*
Low risk	Ref.	Ref.	0.04(0.23)	0.00(0.12)	0.57(0.37)	-0.20(0.19)
Average risk	0.17(0.29)	0.17(0.14)	0.29(0.28)	0.14(0.14)	0.84(0.64)	-0.11(0.31)
Some delinquency	0.35(0.36)	0.15(0.15)	0.56(0.33)	0.09(0.20)	-0.02(0.68)	0.39(0.34)
Elevated delinquency	0.55(0.35)	0.11(0.16)	0.47(0.32)	0.08(0.15)	1.55(0.53)**	-0.39(0.28)
Delinquency & violence exposure	0.66(0.43)	0.20(0.21)	0.98(0.33)**	0.04(0.16)	2.31(0.49)***	-0.30(0.24)
Multi-risk	1.11(0.54)*	-0.26(0.30)	1.04(0.33)**	-0.10(0.16)	1.10(0.42)**	-0.20(0.23)

Note. $*p < .05$, $**p < .01$, $***p < .001$. Standard errors are in parentheses. The *low risk/high parental knowledge* profile served as the reference category for the predictors.

Table 4.23

Marijuana Use Growth Parameters Predicted by Conditional Profile Membership

	Protective Factor Profiles					
	High Parental Knowledge		Average Protection		Low Protection	
Risk Factor Profile	*i*	*s*	*i*	*s*	*i*	*s*
Low risk	Ref.	Ref.	-0.09(0.23)	0.10(0.10)	-0.09(0.37)	0.09(0.17)
Average risk	0.18(0.30)	0.16(0.14)	0.66(0.28)*	-0.05(0.14)	-1.01(0.48)	-0.35(0.25)
Some delinquency	0.15(0.34)	0.27(0.15)	0.63(0.30)*	0.19(0.16)	-0.01(0.71)	0.30(0.27)
Elevated delinquency	0.77(0.40)	0.15(0.17)	0.25(0.33)	0.04(0.15)	1.35(0.52)**	-0.15(0.21)
Delinquency & violence exposure	1.37(0.44)**	-0.13(0.21)	1.43(0.31)**	-0.09(0.14)	1.11(0.68)	0.03(0.32)
Multi-risk	1.66(0.47)***	-0.34(0.20)	1.24(0.34)***	-0.10(0.15)	1.64(0.37)***	-0.04(0.19)*

Note. $*p < .05$, $**p < .01$, $***p < .001$. Standard errors are in parentheses. The *low risk/high parental knowledge* profile served as the reference category for the predictors.

differences in initial levels of marijuana use across sex ($b = 0.11, SE = 0.15, p = .470$), but men had greater increases in marijuana use across adolescence relative to women (i.e., there was a significant effect of sex on the linear slope; $b = 0.18, SE = 0.07, p = .007$). Among those most likely to demonstrate the *average protection* pattern, those also most likely to demonstrate the *delinquency & violence exposure*, *average risk*, *some delinquency*, or *multi-risk* patterns had higher initial levels of marijuana use relative to those most likely to demonstrate the *low risk/high parental knowledge* patterns. Among those most likely to demonstrate the *high parental knowledge* pattern, those also most likely to demonstrate the *delinquency & violence exposure* or *multi-risk* patterns had higher initial levels of marijuana use relative to those most likely to demonstrate the *low risk/high parental knowledge* patterns. Among those most likely to demonstrate the *low protection pattern*, those most likely to also demonstrate the *elevated delinquency* or *multi-risk* patterns had higher initial levels of marijuana use relative to those most likely to demonstrate the *low risk/high parental knowledge patterns*, while those most likely to demonstrate the *average risk/low protection* pattern had lower initial levels of marijuana use relative to those most likely to demonstrate the *low risk/high parental knowledge* pattern. Finally, those most likely to demonstrate the *multi-risk/low protection* patterns had declining marijuana use across time relative to those most likely to demonstrate the *low risk/high parental knowledge* patterns.

Aim 4.b.

We also examined the extent to which the patterns of etiological factors predicted the likelihood of demonstrating alcohol trajectories across adolescence (Table 4.24). Only two etiological factor patterns had significantly higher odds of demonstrating the *high use* alcohol pattern (relative to the *average use* pattern) than those likely to demonstrate the *low risk/average protection* pattern, which we used as the reference category for the predictors. Those who were most likely to demonstrate the *high risk/low protection* pattern had 278% higher odds of demonstrating the *high use* alcohol trajectory (relative to the *average use*

trajectory) and those who were most likely to demonstrate the *moderate risk/decreased protection* pattern had 189% higher odds of demonstrating the *high use* alcohol trajectory (relative to the *average use* trajectory), compared to those most likely to demonstrate the *low risk/average protection* etiological factor pattern.

Table 4.24

Most Likely Alcohol Use Trajectory Membership Predicted by Most Likely Etiological Factor Profile Membership

Etiological Factor Profile	*B(SE)*	*p*	*OR*
Average risk and protection	0.54(0.36)	.128	1.71
Some delinquency/average protection	0.49(0.43)	.255	1.62
Elevated risk/decreased protection	0.49(0.44)	.250	1.63
Moderate risk/decreased protection	1.06(0.38)	.005	2.89
High risk/low protection	1.33(0.40)	.001	3.78

Note. OR = odds ratio. Odds ratios represent odds of demonstrating the *high use* alcohol trajectory relative to the odds of demonstrating the *average use* trajectory. The *low risk/average protection* pattern served as the reference category for the predictors.

We also used the etiological factor patterns to predict trajectories of cigarette use (Table 4.25). There were no significant sex differences in initial levels of cigarette use ($b = -0.18, SE = 0.14, p = .211$), but men did have significantly greater increases in use across adolescence relative to women (i.e., there was a significant effect of sex on the linear slope; $b = 0.37, SE = 0.07, p < .001$). There were a number of differences across the profiles in initial levels of use, wherein individuals most likely to be members of any profile (except *some delinquency/average protection*) had significantly higher initial levels of cigarette use relative to those most likely to demonstrate the *low risk/average protection* reference group. However, only those most likely to demonstrate the *moderate risk/decreased protection* pattern had a significantly lower rate of change across time relative to those most likely to demonstrate the *low risk/average protection* reference group.

Results of etiological factor patterns predicting marijuana use trajectories are available in Table 4.26. There were once again no significant sex differences with respect to

Table 4.25

Cigarette Use Trajectories Predicted by Most Likely Etiological Factor Profile Membership

Etiological Factor Profile	*i*	*s*
Average risk and protection	0.22(0.20)	0.15(0.10)
Some delinquency/average protection	0.33(0.24)	0.17(0.12)
Elevated risk/decreased protection	0.64(0.23)**	0.02(0.11)
Moderate risk/decreased protection	1.06(0.25)***	0.06(0.12)
High risk/low protection	1.00(0.25)***	-0.13(0.13)

Note. $*p < .05$, $**p < .01$, $***p < .001$. Standard errors are in parentheses The *low risk/average protection* pattern served as the reference category for the predictors.

initial levels of marijuana use ($b = 0.12, SE = 0.14, p = .400$) but men exhibited a greater increase in use across adolescence relative to women (i.e., there was a significant effect of sex on the linear slope; $b = 0.17, SE = 0.07, p = .010$). Once again, individuals most likely to be members of any profile (except *some delinquency/average protection*) had significantly higher initial levels of marijuana use relative to those most likely to demonstrate the *low risk/average protection* reference group. However, those most likely to demonstrate the *high risk/low protection* pattern had significantly lower rates of change across adolescence relative to those most likely to demonstrate the *low risk/average protection* reference group.

Table 4.26

Marijuana Use Trajectories Predicted by Most Likely Etiological Factor Profile Membership

Etiological Factor Profile	*i*	*s*
Average risk and protection	0.53(0.20)**	-0.03(0.09)
Some delinquency/average protection	0.40(0.22)	0.19(0.10)
Elevated risk/decreased protection	0.71(0.24)**	-0.02(0.11)
Moderate risk/decreased protection	1.38(0.25)***	-0.11(0.12)
High risk/low protection	1.50(0.24)***	-0.28(0.11)*

Note. $*p < .05$, $**p < .01$, $***p < .001$. Standard errors are in parentheses The *low risk/average protection* pattern served as the reference category for the predictors.

CHAPTER FIVE

Discussion and Conclusions

Summary of Study Aims and Findings

Given the unique risk of substance use and limited access to treatment resources among low-income African American adolescents, the present study aimed to (1) independently identify discrete patterns of risk and protective factors at age 12 as well as conditional memberships among the patterns, (2) identify discrete patterns of etiological factors (i.e., combined risk and protective factors) at age 12, (3) assess heterogeneity in trajectories of alcohol, cigarette, and marijuana use across ages 13-17, and (4) use the conditional risk and protective factor patterns and the discrete patterns of etiological factors at age 12 to predict distinct trajectories of alcohol, cigarette, and marijuana use across ages 13-17. By doing so, we aimed to identify distinguishable subgroups of adolescents who may demonstrate increased likelihood for substance use. Investigating these subgroups may (1) provide distinct combinations of factors that predict an increased likelihood for substance use in unique subgroups of adolescents, thus distinguishing individuals who may benefit from preemptive intervention, and (2) provide evidence of the extent to which factors that have traditionally been conceptualized to present "risk" for or "protection" against substance use actually exhibit these risk or protective effects in combination with one another. We based our selection of risk and protective factors and rationale for the use of person-centered approaches to study etiology and substance use on prior empirical evidence (Hawkins et al., 1992; Jessor et al., 1995; Nash & Bowen, 2002; Sloboda et al., 2012), the theoretical model of socialization outlined in the Social Development Model (SDM; Catalano & Hawkins, 1996; Catalano et al., 1996), and a holistic-interactionist conceptual framework (Magnusson, 1999).

Aims 1-2: Risk, Protective, and Etiological Factors

Aim 1.a-1.b.: Patterns of separate risk factors and protective factors. When examining risk factors and protective factors for substance use separately, we identified three discrete patterns of protective factors and six discrete patterns of risk factors. While the discrete protective factor patterns were characterized by three unique combinations of neighborhood connectedness, parental knowledge, religiosity, and self-worth, parental knowledge appeared to play the largest part in distinguishing the three patterns. Prior research has suggested that African American families engage in stricter monitoring of their children and adolescents' behavior which may result in increased parental knowledge, so it is not surprising to see that parental knowledge is a primary distinguishing variable across subgroups of protective factors and that the largest proportion of the sample was most likely to be characterized by a pattern that demonstrates high levels of parental knowledge (Jarrett, 1995). Given that religiosity, self-worth, and neighborhood connectedness have all been shown to protect against the development of substance use among African American adolescents, it is interesting that there is less variation across the protective factor patterns with respect to these factors (Copeland-Linder et al., 2011; Mayberry et al., 2009; Steinman & Zimmerman, 2004). One potential explanation for the observed homogeneity in this sample may be measurement. For instance, religiosity was only measured by three indicators, making the resulting factor (from which we extracted the factor score) as just-identified. Despite these issues, the wide variation in mean levels of parental knowledge across the three groups suggest that this may be a particularly important protective factor for substance use among African American adolescents.

There were qualitative differences between the characteristics of the protective factor patterns identified in our study and those found in prior research also examining unique patterns of protective factors (Syvertsen et al., 2010). These discrepancies are likely due to the nature of the variables used to comprise the patterns. While the Syvertsen et al. (2010)

study also utilized a measure of parental monitoring and limit-setting, which may be analogous to the parental knowledge measure utilized in the current study, and a measure of life satisfaction, which may address some domains common to our self-worth measure, this study also included measures of planful competence (considering future aspirations and setting goals to maintain them), physical activity, positive school orientation, parent-child communication, low friends' drug use, and other adult communication. These factors comprised a wider range of the social environment than the factors that were included in our study. The Syvertsen et al. (2010) study also examined a nationally representative sample of high school students, including only 12% Black or African American students, suggesting that our results may be more likely to uncover heterogeneity specifically among African American adolescents that is masked when only small sub-samples of African American adolescents are included in larger samples. The discrepancies across these patterns of protective factors highlight the importance of identifying factors that may be most likely to indicate protection against substance use among specific subpopulations of individuals (e.g., samples of solely African American adolescents as opposed to nationally representative samples).

The six risk factor patterns appeared to be distinguished first by varying levels of delinquency, then by mean levels of exposure to violence, and then by mean levels of peer pressure. The prevalence of delinquency as a distinguishing factor across our six empirically-derived patterns emphasize prior research indicating that delinquency is one of the most salient risk factors for additional deviant behavior, including substance use (W. A. Mason, Hitchings, McMahon, & Spoth, 2007). It is of interest that mean levels of delinquency were usually slightly higher than mean levels of exposure to violence but increased in tandem across different subgroups of adolescents, as prior research has suggested that individuals may be more likely to engage in delinquent behavior following exposure to violence (Chen, Voisin, & Jacobson, 2016; Rubens, Fite, Cooley, & Canter, 2014). It

may be that exposure to violence is only one factor that predicts increased levels of delinquency, which in turn may lead to later substance use. Future research should consider factors that influence the development of these discrete patterns and unique combinations of delinquency and exposure to violence.

However, it is interesting that most of the patterns demonstrated low mean levels of responding to peer pressure items, as it would make sense that adolescents who feel peer pressure to engage in substance use also feel peer pressure to engage in delinquent behavior, a general factor referred to as peer pressure to misconduct (Clasen & Brown, 1985). This could be a function of social desirability as it relates to adolescents' developing need for autonomy or desire for peer acceptance. Perhaps adolescents are less likely to acknowledge peer influence over either their adaptive or maladaptive behaviors due to a knowledge that being susceptible to peer influence is undesirable (Fisher, 1993). It may be that simple affiliation with deviant peers, rather than explicit peer pressure to drink or use drugs, is more strongly associated with an increased likelihood of engaging in substance use.

While the six patterns of risk factors demonstrated unique patterns of delinquency, exposure to violence, and peer pressure, traumatic stress was not useful in distinguishing across the patterns; all six prototypical patterns exhibited nearly the exact sample mean level of traumatic stress. It may be that traumatic stress represents a qualitatively different experience that is unrelated to the type of substance use risk exhibited by the other risk factors studied in the current study. However, it is of interest that there are unique differences across the identified patterns with respect to violence exposure, but not traumatic stress, as exposure to violence may be a catalyst for experiencing traumatic stress. It may be that the effects of violence exposure are expressed in other ways, such as by engaging in other antisocial behaviors (e.g., substance use, delinquency) as was proposed by General Strain Theory (Agnew, 1992, 2001, 2006) or Social Cognitive Theory (Bandura, 2001).

Our risk factor patterns are also distinct from those identified in prior research on prototypical patterns of risk factors for substance use (Lanza & Rhoades, 2013). While our

study uncovered six patterns of risk factors, Lanza and Rhoades (2013) identified only five patterns from indicators assessing unique patterns of household risk (e.g., socioeconomic status, single parent-headed households, household poverty), peer risk (e.g., peer cigarette and alcohol use), and neighborhood risk (e.g., neighborhood unemployment, neighborhood poverty). Once again, our results likely differ due to the different types of items utilized in these studies; while Lanza and Rhoades (2013) focused on near-demographic predictors that may be more objective (e.g., poverty), our study focused on psychosocial variables that may be subjective and take on unique meaning to each individual. Further, the Lanza and Rhoades (2013) study utilized a nationally representative sample, of which 21% was Black or African American, which, as has been previously mentioned, will result in patterns that represent subgroups of broad populations of individuals and may not necessarily be specific to African American adolescents.

When examining conditional patterns of risk factor and protective factor patterns, the combination of the *high parental knowledge* and *low risk* patterns characterized the largest proportion of the sample, meaning that out of the 18 conditional patterns (three protective factor patterns and six risk factor patterns), adolescents are most likely to exhibit low levels of risk factors and high levels of parental monitoring on average. However, this pattern only represented 24% of the total sample; generally, individuals were best characterized by a pattern of risk and protective factors containing either high levels of parental knowledge or average protection, and either low levels of risk factors or low to moderate levels of delinquency. Few individuals exhibited conditional patterns characterized by high levels of risk factors and high levels of protective factors. These general patterns are in line with other conceptualizations of risk and protective factors as discrete dimensions that are likely to fluctuate together (Bogat et al., 2016; Rutter, 1987).

Aim 2: Patterns of etiological factors. We identified six patterns of etiological (i.e., combined risk and protective) factors in the data. We used the term *etiological* factor

in order to avoid an implicit assumption that specific factors would be associated with increased or decreased likelihoods of substance use. However, factors that were traditionally described as "risk" factors and factors that were traditionally described as "protective" factors functioned along these descriptive categories; that is, we identified six discrete patterns of etiological factors wherein patterns that were characterized by higher levels of delinquency, exposure to violence, and peer pressure were also characterized by proportionally lower levels of parental knowledge, self-worth, and neighborhood connectedness.

The most obvious observation when examining the patterns of etiological factors is that the six empirically-derived patterns are strongly reminiscent of the six also empirically-derived patterns of risk factors; that is, the same number of patterns were identified when examining risk factors and protective factors together as etiological factors as were identified when we examined risk factors alone. Further, the average levels of risk etiological factors among the six etiological factor patterns are nearly identical to the levels of risk factors identified among patterns when solely examining risk factors for substance use. In essence, the addition of traditionally-defined protective factors to a model of etiological factors for substance use did not modify the expression of traditionally-defined risk factors among a sample of low-income African American adolescents. This stability in pattern expression infers that the traditionally-defined protective factors evaluated in the present study may not be effective in modifying or reducing the expression of risk for substance use among this sample.

The finding that adding traditionally-defined protective factors to the model did not change the expression of traditionally-defined risk factors is aligned with prior theoretical work suggesting that the dimensions of risk and protection are not unrelated to one another (Bogat et al., 2016). As one's exposure to various risk factors shifts, that individual's exposure to other protective factors shifts as well, suggesting that we should conceptualize patterns of risk and protective factors as unique and integrative wholes that may function

in distinct ways across unique types of individuals in line with a holistic-interactionist approach (Luthar et al., 1993; Magnusson, 1999). This was reflected across the identified patterns of etiological factors wherein levels of etiological risk factors such as delinquency, exposure to violence, and peer pressure increased across unique subgroups of individuals while etiological protective factors such as parental knowledge concurrently decreased across the subgroups; that is, the functioning of risk factors and protective factors appear to be inversely related to each other in specific ways within unique subgroups of individuals, as patterns that were characterized by high levels of delinquency, exposure to violence, and peer pressure demonstrated proportionally lower levels of parental knowledge. As such, the present study demonstrates the usefulness of using person-oriented approaches to identify unique homogeneous subgroups of individuals who may differ in their expression of risk and protective factors, and how changes in one risk or protective factor may be associated with changes in another risk factor across unique types of individuals.

Aim 3: Trajectories of Substance Use

We identified two discrete patterns of alcohol use among low-income African American adolescents, with one pattern exhibiting a high pattern of use, and the other exhibiting an average pattern of use. The average pattern characterized the majority of the sample, while the high use pattern characterized nearly 5% of the sample. Convention suggests that a pattern must characterize at least 5% of the sample in order to be representative of an empirically valid proportion of individuals; after considering the probabilistic nature of these models (i.e., individuals who had a lower likelihood of demonstrating the average use pattern may actually demonstrate the high use pattern), we felt that our derived high alcohol use pattern was likely representative of a sufficiently large proportion of the sample to be considered empirically valid. This determination was also validated after considering other studies of substance use trajectories that typically identify at least two patterns of alcohol use, including a high use pattern and a low or average use pattern (K. A. Bolland

et al., 2016; Komro et al., 2010). The identification of multiple patterns of alcohol use is in line with other research that has identified multiple patterns of alcohol use across adolescence, in addition to findings that alcohol generally increases across adolescence as part of a normative developmental trajectory (Park et al., 2018; White et al., 2006).

However, our results did not provide enough evidence to support the presence of multiple patterns of cigarette or marijuana use across adolescence, which is in contrast to other literature examining nationally-representative samples of adolescents (Park et al., 2018) and other research in primarily-African American samples of low-income, urban adolescents (Lynne-Landsman et al., 2010). The primary reasons for differences in these results is likely due to the time period over which substance use was assessed. The MYS referenced frequencies of substance use within the past 30 days, while other studies examined substance use frequency over the past year. It is likely that 30 days is too narrow of a window in which to adequately assess patterns of substance use, but that past-year frequency provides a wider opportunity to (1) observe substance use, and (2) to identify heterogeneity in substance use patterns (e.g., an individual may not have used cigarettes or marijuana in the past 30 days, but may have done so once or multiple times within the past year). This may indicate differences across substances in availability (e.g., perhaps alcohol is more readily available or easily accessible by adolescents) or indications of severe problem behavior among this specific subgroup of adolescents.

One other reason that this sample may have exhibited multiple patterns of alcohol use but not cigarette or marijuana use may be due to the overall frequency of cigarette and marijuana use relative to that of alcohol. By age 17, half of the sample had initiated into alcohol use, while only 30% and 43% of the sample had initiated into cigarette and marijuana use, respectively. It may also be that lower levels of cigarette and marijuana use overall provided fewer opportunities to observe heterogeneity in use.

Interestingly, while the marijuana use trajectory had a statistically significant linear slope, indicating increasing levels of marijuana use across time, there was no evidence for

significant change in levels of cigarette use across ages 13-17. This may indicate that all individuals who initiate into cigarette use continue to smoke and do not remit until the emerging adult years. If this is the case, those who use cigarettes early in adolescence are likely to continue doing so, but that the risk of initiating into cigarette use does not increase after age 13. These results may suggest that smoking prevention efforts should be placed on the late childhood years. However, an alternate explanation may be that there is an equilibrium in rates of initiation and remission from cigarette use, where an equal number of individuals begin to use as those who stop using in an assessment period. This explanation may also indicate heterogeneity in patterns of cigarette use across adolescence, which have been found in previous research using nationally representative studies but were not replicated in our study, suggesting that additional research is necessary to fully understand trajectories of cigarette use among African American adolescents (Park et al., 2018).

Aim 4: Use of Etiological Factors to Predict Substance Use Trajectories

Use of conditional risk and protective factor patterns in predicting substance use trajectories. We first examined the extent to which conditional patterns of separate risk and protective factors predicted likelihood of demonstrating alcohol use patterns. While we had anticipated that individuals who were likely to demonstrate patterns characterized by low levels of protective factors and high levels of risk factors would be more likely to demonstrate trajectories of high substance use across adolescence, these patterns were only somewhat present. Regardless of the risk factor pattern they were most likely to demonstrate, nearly all individuals who were likely to demonstrate the *average protection* pattern were also more likely to demonstrate the high use alcohol trajectory. The exceptions were that among those most likely to demonstrate the *average protection* pattern, those most likely to demonstrate the *some delinquency* or *elevated delinquency* patterns were no more

likely to demonstrate the *high use* relative to the *average use* alcohol trajectory. These findings do not align with the SDM suggestion that increased exposure to antisocial bonding agents in the absence of prosocial bonding agents will increase likelihood of substance use (Catalano & Hawkins, 1996; Catalano et al., 1996). There are likely other factors not accounted for in our study that differentiate why individuals who are most likely to demonstrate *some delinquency/average protection* or *elevated delinquency/average protection* are not more likely to exhibit high use alcohol trajectories, likely relating to either the peer or school environments which we were unable to account for in our study.

When examining the prediction of substance use among those most likely to demonstrate a patten characterized by high levels of parental knowledge, only those who were likely to demonstrate concurrently high levels of multiple risk factors were more likely to demonstrate the *high use* alcohol trajectory. This indicates that high levels of parental knowledge, while able to buffer the effects of multiple unique patterns consisting of low to moderate levels of risk factors, is not able to adequately protect against the effects of multiple risk factors, including high levels of delinquency, exposure to violence, and peer pressure on likelihood of alcohol use. While this is in contrast to most literature that suggests parental knowledge is associated with decreased levels of alcohol use, some prior research has found a similar lack of relationship between parental knowledge and alcohol use (Tebes et al., 2011). It may be that parental knowledge on its own has little direct influence on the reduction of alcohol use (that is, simply *knowing* where and what your children are up to may have little influence on alcohol consumption), but may rather function as the initiation of a socialization trajectory that bonds adolescents to prosocial agents, which in turn reduces adolescents' likelihood of engaging in alcohol use (Catalano & Hawkins, 1996; Catalano et al., 1996). Further, additional protective factors, as well as a reduction of exposure to risk factors, may be necessary in order to reduce these individuals' likelihood of exhibiting higher levels of alcohol use across adolescence.

We found unexpected results when examining the likelihood of alcohol use among those who were most likely to demonstrate low levels of protective factors. Among those who were most likely to demonstrate few protective factors, only those who were also likely to demonstrate slightly elevated levels of delinquency were more likely to demonstrate the *high use* alcohol trajectory. It may be that those most likely to demonstrate the elevated delinquency and low protection patterns fall into a "sweet spot" of detection for potential risk for substance use. Given that risk factors for substance use are likely more salient and predictive of substance use than protective factors (Cleveland, Feinberg, Bontempo, & Greenberg, 2008), perhaps adolescents with high levels of exposure to risk factors, regardless of exposure to protection, are more closely monitored by other institutions we did not capture in our analysis, such as the school environment. However, perhaps those who are likely to demonstrate the *elevated delinquency* pattern are not considered high-enough risk to warrant special preventative monitoring, so those who are likely to exhibit both the *elevated delinquency* and *low protection* patterns are at unique risk for alcohol use relative to their peers.

When examining the use of conditional risk/protective factor patterns to predict trajectories of cigarette use, we only identified significant differences based on initial levels of use. Individuals who were most likely to demonstrate multiple risk factors had higher levels of initial cigarette use, regardless of most likely protective factor pattern. This suggests that the examined protective factors were not effective in preventing use among those who were also likely to demonstrate high levels of multiple risk factors. Similarly, those with varying levels of exposure to delinquency and the absence of protective factors were likely to have higher levels of cigarette use at age 13, reiterating the importance of delinquency as a prominent risk factor for substance use. There were no differences in the extent to which rates of cigarette use changed over time, which is incongruent with prior research on substance use trajectories across adolescence (Park et al., 2018). However, this also points to

the pre-teen years as a potential critical period for reducing exposure to risk and increasing exposure to protection in reducing substance use among adolescents.

The use of conditional risk and protective factor patterns to predict marijuana trajectories present a similar pattern to those of alcohol use, highlighting the importance of delinquency and exposure to multiple risk factors in predicting higher levels of initial marijuana use. That is, individuals who were likely to demonstrate patterns of risk factors characterized by high levels of delinquency and exposure to violence were more likely to exhibit higher levels of initial marijuana use at age 13, and concurrent likelihood of demonstrating patterns of protective factors characterized by higher mean levels of parental knowledge did not necessarily buffer the effects of those risk factor patterns. However, those most likely to demonstrate high levels of risk factors and low levels of protective factors had decreasing rates of marijuana use across adolescence. This finding is generally in contrast to theorizing in the SDM that would predict individuals with high levels of exposure to antisocial socialization agents (e.g., exposure to multiple levels of risk) and low levels of exposure to prosocial socialization agents (e.g., low levels of exposure to protective factors) would be more likely to engage in behaviors aligned with the values of those antisocial bonded agents; as such, this may be uncovering a subgroup of individuals for whom the socialization processes from the SDM do not adequately describe their internalization of behavior standards (Catalano & Hawkins, 1996; Catalano et al., 1996). While only one unique trajectory of marijuana use was identified in the present study, prior research on marijuana trajectories across adolescence has identified five distinct patterns (Park et al., 2018). It may be that this combination of risk and protective factor patterns may indicate individuals who are likely to demonstrate a unique developmental trajectory of marijuana use that would be more readily uncovered in a larger, more heterogeneous sample. Further, it may be that marijuana use is not a value held by one's socialization agents, and as such, individuals may not be likely to engage in that behavior across adolescence.

Use of etiological factor patterns in predicting substance use trajectories. When considering risk and protective factors as etiological factors (i.e., factors whose effects may either increase or decrease the likelihood of developing substance use depending on the combination of other factors present in that individual's environment) only those who were likely to demonstrate high levels of multiple traditionally-defined risk factors (e.g., delinquency, exposure to violence, or peer pressure) and simultaneously low levels of multiple traditionally-defined protective factors were more likely to demonstrate high levels of alcohol use across adolescence. We found similar results when using the etiological factor patterns to predict cigarette and marijuana use trajectories. A high likelihood of demonstrating etiological factor patterns characterized by high mean levels of delinquency, exposure to violence, or peer pressure, and low mean levels of parental knowledge were associated with higher initial levels of cigarette use. There were no significant differences in rate of change across the etiological factor patterns in line with a nonsignificant slope identified in the unconditional cigarette use trajectory. Results for marijuana use trajectories followed a similar pattern, except that individuals likely to demonstrate any pattern (except those most likely to demonstrate the *some delinquency/average protection*) had a higher initial level of marijuana use. Further, only those who were most likely to demonstrate high levels of risk factors and low levels of protective factors had significantly slower rates of change in marijuana use across time.

These results are congruent with previous research suggesting that higher levels of delinquency, exposure to violence, and peer pressure are associated with higher levels of alcohol, cigarette, and marijuana use (Brook, Lee, Finch, et al., 2013; Fagan et al., 2014, 2015; Geyer et al., 2015; Hoyland et al., 2017; Hunter et al., 2014; Iwamoto & Smiler, 2013; W. A. Mason, Hitchings, McMahon, & Spoth, 2007; Pinchevsky et al., 2013; Studer et al., 2016). It appears that, much as with the use of conditional risk factor and protective factor patterns, high levels of risk factors were generally associated with lower levels of

protective factors, and individuals likely to demonstrate higher risk/lower protection patterns were more likely to have high levels of substance use.

However, we saw a much more sequential progression of risk for substance use across the etiological factor patterns. As the patterns of etiological factors demonstrated higher levels of risk and lower levels of protective factors, individuals likely to demonstrate those higher risk etiological factor patterns were also more likely to exhibit higher levels of substance use. This was not always the case for the conditional risk/protective factor patterns, as there were some instances in which having low levels of protective factors did not increase the likelihood of exhibiting higher levels of substance use. The findings that high levels of risk factors combined with low levels of protective factors were associated with higher levels of substance use is aligned with the SDM, suggesting that individuals who have increased bonding to antisocial socialization agents (e.g., delinquency, violence exposure, peer pressure) and lower levels of bonding to prosocial socialization agents (e.g., parental knowledge) will be more likely to engage in behaviors associated with the standards of those antisocial bonding agents (e.g., substance use; Catalano & Hawkins, 1996; Catalano et al., 1996).

Support for Theoretical and Conceptual Models

Overall, the present study supports the notion that risk and protective factors are distinct, but interacting, constructs, and that there is variation in the extent to which risk and/or protective factors influence outcomes across various types of individuals (Lloyd, 1998; Rutter, 1987). While the present study did not necessarily test a cascade model of socialization processes that may lead to substance use as posited in the SDM, the present study did find that contexts for various types of prosocial or antisocial socialization or bonding (e.g., delinquency, exposure to violence, parental knowledge, peer pressure) varied across subgroups of adolescents and were perhaps more predictive of later substance use trajectories than were traits that were more internal to the individual (e.g., self-worth,

traumatic stress; Catalano & Hawkins, 1996; Catalano et al., 1996). Finally, examining these potential bonding agents or processes using a person-oriented approach supports the conceptual model provided from the holistic-interactionist approach that individuals function as integrated wholes and that the same etiological factors may function differently across multiple types of individuals (Magnusson, 1990, 1999, 2003).

In the first chapter we presented a conceptualization of risk and protective factors as etiological factors so as to not make implicit assumptions about the extent to which risk or protective factors always predict an increased or decreased, respectively, likelihood of substance use, but that the effect of a single etiological factor may be dependent upon the entire combination of etiological factors present in an individuals' environment. We examined this conceptualization by examining (1) conditional patterns of independently-derived risk factor and protective factor patterns and (2) patterns of etiological factors. Generally, the results echo earlier research that risk factors are more salient predictors of later substance use than are protective factors (Cleveland et al., 2008). We identified more heterogeneity in substance use etiology when we considered conditional patterns of independently-identified risk and protective factor patterns as opposed to identifying patterns of combined etiological factors. This may suggest that risk and protective factors serve distinct purposes but that unique interactions among these factors may predict later outcomes among adolescents (Bogat et al., 2016; Jessor et al., 1995).

As mentioned, the results also echo the interactive nature of etiological factors. When we considered risk and protective factors together as etiological factors in a single model, prototypical patterns of etiological factors exhibited lower levels of protective factors as exposure to risk factors increased, which is in line with prior research suggesting that as exposure to one type of vulnerability or resilience shifts, exposure to other types shift as well (Bogat et al., 2016; Luthar et al., 1993). We do not see drastic differences across subgroups with respect to factors exhibiting risk in one context but protection in another; that is, we did not see frequent conditional associations between patterns with high levels of

risk factors and patterns with high levels of protective factors occurring together, and we did not see patterns with high levels of protective factors exhibiting high likelihoods of demonstrating high use or increasing trajectories. However, it is important to continue examining risk and/or protective factors within the context of other etiological factors because of the demonstrated interactive nature of risk and protection.

The present study used the SDM (Catalano & Hawkins, 1996; Catalano et al., 1996) as a framework through which to understand how etiological factors may influence substance use. In essence, the SDM proposes that an individual's characteristics may influence his or her likelihood of later engagement in antisocial behavior (including substance use), but that the extent of this influence may depend on socialization processes that increase one's bonding to either prosocial or antisocial structures in family, peer, school, and neighborhood or community domains (Catalano & Hawkins, 1996; Catalano et al., 1996). Our study suggests that an antisocial bonding process such as engaging with delinquency, being exposed to violence, or experiencing peer pressure may have stronger and more robust effects on adolescents' likelihood of later substance use. Additionally, our results suggest that either (1) the prosocial bonding agents or processes in our study (specifically religiosity, self-worth, and neighborhood connectedness) were not effective in initiating a pattern of bonding that may lead to lower likelihoods of later substance use, or (2) that the socialization and bonding process to these prosocial bonding agents was initiated but not completed.

The specific factors that are likely to co-occur among various subgroups of adolescents indicate domains of influence that are of particular importance to the development of substance use. First, the present study suggests that the peer domain provides a foundation for engagement in antisocial behavior that may lead to later substance use. Patterns of etiological factors that had high mean levels of delinquency and peer pressure, two factors that usually occur within the peer domain, were associated with an increased likelihood of demonstrating a high alcohol use trajectory (relative to an average use trajectory) across

adolescence, and significantly higher initial levels of cigarette and marijuana use. The discrete patterns exhibiting varying levels of delinquency and peer pressure may distinguish subgroups of adolescents who are at varying stages of bonding to or socializing with peers. That is, the SDM proposes that behaviors such as delinquency and substance use arise from a socialization process that results in bonding to peers (Catalano & Hawkins, 1996; Catalano et al., 1996). Given that those who have higher levels of delinquency, but not necessarily high levels of peer pressure, are likely to exhibit later substance use, it may be that delinquent behavior with peers leads to internalizing peers' standards in such a way that promotes later substance use (as another type of antisocial behavior) but that experiencing direct peer pressure to engage in antisocial behavior is not necessarily the socializing process through which this occurs.

Parental knowledge may result from a parent-child socialization process but may also provide opportunities for the parent to function as a prosocial bonding agent for the child, thereby initiating the socialization process. By knowing what is going on with one's child, the parent demonstrates interest and care for the child, which provides a potential opportunity to engage in prosocial behavior for the child (e.g., developing a good relationship with one's parent). If the child perceives an opportunity to engage in a prosocial relationship with one's parent, and the parent continues to foster positive interactions with the child, the child may feel reinforced by those positive interactions, leading the child to internalize their parents' standards (which, presumably, include no or low drug and alcohol use by underage individuals); as such, the adolescent is more likely to engage (or not engage) in behavior in alignment with their parents' values. Given the prominence of parental knowledge as a protective factor for substance use in this study, the socialization processes that occur between parent and child are integral in fostering prosocial behavior among adolescents, in alignment with previous research (Tebes et al., 2011; Tobler & Komro, 2010; Udell et al., 2017). However, further longitudinal work is necessary to elucidate the extent to which the mechanisms of this socialization process differs across adolescents exposed to

various types of risk factors, and the extent to which these mechanisms may change across adolescence.

Combining the theoretical background in the SDM with the conceptual framework of holistic-interactionism provided an opportunity to uncover the extent to which the socialization processes outlined in the SDM were consistent across empirically-derived subgroups of adolescents. Holistic-interactionism proposes that the individual should be conceptualized as an integrated whole and that an individual's behavior can only be understood by examining the simultaneous functioning of all factors that make up the whole of the individual (Bergman et al., 2003; Magnusson, 1999). Given the wide range of factors and mechanisms posited to be associated with substance use according to the SDM, using person-oriented approaches to uncover unique prototypical patterns of etiological factors provided an opportunity to examine the extent to which specific etiological factors were likely to distinguish various types of adolescents who had increased or decreased likelihoods of substance use.

Although the present analyses derived patterns of risk factors and patterns of protective factors that were different from those patterns reported in prior literature, the presence of multiple unique patterns in our sample emphasize the dynamic and specific nature of substance use etiology (Lanza & Rhoades, 2013; Syvertsen et al., 2010). By uncovering unique etiological factor patterns with distinct likelihoods of later substance use, we were able to highlight the especially important nature of specific combinations of delinquency, exposure to violence, peer pressure, and parental knowledge in predicting which types of adolescents were more likely to engage in substance use. The pattern-oriented approach uncovered unique subgroups of adolescents for whom the socialization processes outlined in the SDM differed; that is, adolescents were exposed to unique patterns of potential bonding agents, which influences their likelihood of internalizing standards set by those agents. These unique subgroups of adolescents may respond differently to shifts in exposure to

those bonding agents or socialization processes, including prevention or intervention efforts, which should be accounted for in future research attempting to reduce substance use among adolescents.

Limitations

As with any secondary data analysis, a major limitation concerns the measures available and utilized. The assessment of substance use over the past 30 days may have provided a limited window of opportunity in which to observe substance use among these adolescents, potentially limiting the heterogeneity observed in patterns of substance use and decreasing our ability to compare patterns of substance use identified in the current study to prior literature which has utilized indicators of substance use with respect to the past year. Further, we found that the measure of self-worth provided in the current study had less-than-perfect fit to the data than reported in the validation study, and our results are similar to those of other studies using the same measure with African American adolescents (e.g., Copeland-Linder et al., 2011; Harter, 1982). While we chose to utilize this measure despite less-than-perfect fit because of its availability in the MYS and consistency with other studies using a similar sample, we encourage researchers to be meticulous in their selection of measures for use in large population datasets such that the measures selected have been adequately validated in populations with similar characteristics to those of the targeted sample. The use of a formative measurement procedure, such as principal components analysis may have been utilized to create a self-worth measure with the latent definition of self-worth specifically derived from the indicators used to create the construct, rather than a reflective definition as used in the present study wherein it is thought that the observed indicators reflect the latent construct with a predefined, theoretical definition (Howell, Breivik, & Wilcox, 2007). However, we retained the use of the reflective CFA because we wanted to retain consistency with the procedures used when creating other study measures, and because the self-worth scale utilized in the present analysis was a validated

measure that had a pre-defined theoretical definition that may be used to underlie reflective assessment (Harter, 1982; Howell et al., 2007).

The present study included etiological factors in the family, neighborhood, and peer environments, but we were unable to include potential risk or protective factors from the school environment. Additionally, we were unable to account for deviant peer behavior (e.g., delinquency, substance use). Given the importance of deviant peer associations in the development of substance use among adolescents and in the SDM, this is a notable omission that may have implications for the mechanistic causes of substance use we were able to draw from the current study (Catalano et al., 1996; T. T. Clark et al., 2008; Patrick & Schulenberg, 2013; D. R. Wright & Fitzpatrick, 2004). As such, we were unable to fully test the SDM as a mechanism through which substance use develops, and future research should provide a more rigorous and robust test of this model by adequately identifying potential etiological factors (both theoretically positive and negative) from all four domains of influence. That is, if we include a measure of parental knowledge as a potential protective factor within the family domain, it would be necessary to also include an indicator of potential risk within the family domain, such as primary caregiver's history of mental illness or substance use. Further, empirically-derived patterns of substance use etiology are also completely dependent upon the variables included in the analysis. The omission of certain peer and school-related variables may have implications for the types of risk, protective, and etiological factor patterns that we are able to derive, and these factors should be considered in future research. However, the present study did present an initial example of how the SDM may be combined with holistic-interactionism to predict substance use on which later research may build additional models, theories, and explanations.

While the use of a solely African American adolescent sample is a strength of the current study in identifying multiple patterns of etiological factors among this particular demographic group, there are limitations associated with the use of this particular sample.

First, this is a very specific sample of African American adolescents residing in impoverished neighborhoods in Mobile, AL and may not generalize to African American adolescents residing in other areas, although we did see some consistency across substance use findings with predominately African American samples collected from other urban areas such as Baltimore, MD (Copeland-Linder et al., 2011). Second, the use of a solely African American sample does not provide any type of comparison group to examine differences across various racial/ethnic groups; this was intentional in the current study as we were interested in heterogeneity only within a single racial/ethnic group. It may be of interest in future research to examine unique patterns of etiology in samples of multiple racial/ethnic groups and compare findings across the various groups to determine similarities while still retaining detailed attention to unique heterogeneity within each group. Lastly, the use of such a homogeneous sample may have restricted variation across members of our sample such that there may still exist additional heterogeneity across etiology or substance use that we did not identify in the present study. That is, while we assume there is heterogeneity within a single racial/ethnic group, we may not have provided enough opportunities for variation to be detected.

We also conducted CFA on categorical indicators rather than using item response theory (IRT). IRT is used to account for individuals' item responses by placing the individuals' item responses and their underlying latent trait on a common scale, using the item's properties and the individual's score on a latent trait to predict the individual's item response (Bock, 1997; T. A. Brown, 2014; Lord, 1980). IRT and CFA may be used for similar research questions, including using parameter estimates to provide estimates of an individual's placement on a latent dimension (i.e., the individual's latent trait score in IRT and factor score in CFA; T. A. Brown, 2014, p. 396). While CFA parameters can be used to approximate IRT parameters, this approximation is not exact. Because IRT is meant to be conducted with categorical indicators, whereas CFA is intended for continuous indicators,

we may have obtained a more precise measure of individuals' expressions of our etiological factors had we used IRT in the present study.

The latent profile models specified in this paper are complex analyses, and there were a number of instances throughout the analysis process in which we did not have sufficient computing resources to complete a number of analyses as originally conceived. First, we originally planned to conduct factor mixture analysis (FMA). FMA essentially consists of conducting LPA on a previously identified factor structure, allowing for a latent variable to be simultaneously categorical and continuous (S. L. Clark et al., 2013); that is, FMA simultaneously estimates confirmatory factor models and identifies a latent profile structure in a single model. However, we did not have access to enough computing power to estimate a model with that amount of complexity, and as such we modified the original analysis by conducting the analysis in two steps, first, extracting factor scores and second, conducting LPA. While there is some evidence of bias being introduced when extracting factor scores (Skrondal & Laake, 2001), this procedure likely results in results similar to what would be obtained by a simultaneous FMA with a less computationally-intensive procedure.

Additionally, in *Aim 1.b.* we were forced to construct conditional profile membership by classifying individuals into profiles based on their highest posterior probability of membership and then constructing conditional profiles by examining overlap between most likely latent risk factor profile membership and most likely latent protective factor profile membership. Doing so removes the probabilistic nature of LPA and introduces bias of oversimplification into the results. While this is a common practice when studying latent profiles, it should be noted as removing the probabilistic nature of the results. Another way that we could have examined conditional associations of risk and protective factor patterns in predicting substance use would be to regress the substance use trajectories onto (1) a categorical variable representing the risk factor patterns, (2) a categorical variable representing the protective factor patterns, and (3) the interaction between the risk factor and protective factor patterns. We used a single categorical variable of conditional membership

to predict substance use trajectories in order to provide a more direct comparison to the etiological factor (i.e., combined risk and protective factor) patterns, but future research should consider an interactive approach to provide a more statistically robust assessment of the relationship between separately-identified patterns of risk factors and protective factors.

Lastly, we only examined risk and protective factors cross-sectionally at a single time point in early adolescence. Given that the SDM proposes reciprocal relations across socialization processes and antisocial or prosocial outcomes (Catalano & Hawkins, 1996; Catalano et al., 1996; Cleveland et al., 2008), this is a major limitation of our study. However, given that little attention has been provided to unique patterns of combined (i.e., both risk and protective) etiological factors, we felt it was important to first examine what these patterns look like at a single time point to identify subpopulations of individuals for whom various etiological factors may function in unique ways. After identifying what patterns may look like from an exploratory perspective, we may subsequently assess developmental processes that lead to and extend from these observed patterns, which we propose as one area for future research.

Implications and Future Directions

This assessment of etiological factor patterns among a sample of low-income African American adolescents is among the first to specifically address how indicators of risk and/or protection are exhibited among this population. It is of interest to focus specifically on how etiological factors may function within this population to gain insight into how substance use may develop, and be prevented, in ways that are different from mechanisms associated with substance use among individuals of other racial/ethnic groups and socioeconomic statuses; that is, we sought to expand research on substance use in this understudied group, and to make no assumptions that the same influences of risk or protection on substance use among other racial/ethnic groups may function similarly among members of the studied group. This is a current goal of public health in the United States, to identify factors that

may increase or decrease likelihoods of adverse outcomes among specific cultures or communities, ultimately aiming to develop interventions that may be particularly efficacious among specific types of individuals (M. J. Khoury, Iademarco, & Riley, 2016).

The present study focused solely among a sample of low-income African American adolescents residing in a southern metropolitan area; thus, we can only generalize our findings to other adolescents with similar demographics and geographic locations. However, we did uncover heterogeneity in traditionally-defined risk and protective factors and how these factors relate to trajectories of substance use, even among a population that was homogeneous with respect to race and socioeconomic status. This supports prior research advocating that not all individuals of the same racial/ethnic groups exhibit the same patterns of substance use and related etiological factors (Caetano, Clark, & Tam, 1998). Because we advocate an approach of distinctiveness among various racial/ethnic groups, we do not make specific predictions or implications of how specific combinations of etiological factors may function among individuals of other racial/ethnic groups. However, we do advocate for future research to assess for the presence of heterogeneity in substance use and etiology within other seemingly homogeneous groups, continuing to explore factors that may have important clinical or theoretical links to substance use specifically among individuals who share similar demographic characteristics.

Our results support a theoretical position that risk and protection should be considered separate, but interacting dimensions (Bogat et al., 2016; Jessor et al., 1995; Rutter, 1987). As such, we support continuing to examine distinct risk and protective factor patterns separately, their conditional associations, and relationships to later substance use. Doing so provides a more nuanced perspective on substance use etiology; that is, we found 18 unique patterns of conditional associations between risk factor and protective factor patterns, while only identifying six unique patterns when examining risk and protective factors together as etiological factors. For example, the *elevated delinquency/low protection* conditional association does not have a similar pattern among the etiological factor patterns,

but is still uniquely associated with increased odds of demonstrating the *high use* alcohol trajectory. For this specific group of individuals, increased levels of delinquency (although not as high as those exhibited in the *delinquency & violence exposure* or *multi-risk* patterns) compounds the effects of low parental knowledge to elevate these individuals' likelihood of alcohol use; that is, risk for these individuals does not appear to be proportional to the outcome, but results in an increased likelihood of substance use anyways.

These small nuances in conditional associations of risk and protective factors, combined with the observation of strong similarities between the risk factor patterns and the etiological factor patterns, leads to two questions that will be of theoretical importance in future research: first, what does protection mean for this sample? Or, what factors might successfully reduce substance use risk among this population? We only identified parental knowledge as a potential protective factor when examining patterns of protective factors exclusively, but this does not necessarily buffer the effects of delinquency, exposure to violence, or peer pressure; as such, parental knowledge does not appear to be a protective factor for all individuals in this population. There may be other structural, societal, and sociological factors that influence the complex interactions of risk and protective factors in predicting later substance use, as indicated by prior research on unique patterns of risk and protective factors (Lanza & Rhoades, 2013; Syvertsen et al., 2010). It may be that these sociological factors such as a history of systematic oppression resulting in lower levels of education and employment opportunities among African American communities (which we have attempted to control for in our study by limiting the sample to have little variability with respect to socioeconomic status), still influence substance use risk and should be included in future analyses; every individual's experience is unique and each individual's behavioral outcomes should be understood within the specific context in which that person lives. That is, protection may vary even among individuals who appear to be homogeneous with respect to sociodemographic variables, and protection may mean something slightly different to every type of individual exhibiting even the smallest differences.

While we recognize that each individual is unique, scientific research does aim to draw somewhat general conclusions that may apply to more than one individual. An alternative way that future research may consider examining heterogeneity in the interactive nature of risk and protection may be to first identify unique patterns of risk factors and then to identify trajectories of substance use only among individuals likely to demonstrate each of the risk factor patterns. Doing so may provide a more direct examination of what substance use looks like specifically among individuals who exhibit various patterns of risk and sociodemographic variables. Additionally, examining unique substance use patterns within a defined pattern of risk factors would provide an opportunity to examine how specific factors may modify the effects of risk and sociodemographic variables to reduce likelihood of exhibiting substance use trajectories; that is, among these particular risk patterns, what factors may provide some protection, and do the same factors protect against all patterns of substance use among individuals with similar patterns of risk?

The second major question we are left with is *what* do individuals in this particular sample need protecting from? That is, substance use may not be the most important maladaptive outcome to avoid among this population. Rates of violence are high among the studied neighborhoods (J. M. Bolland, 2007); as such, perhaps it is more important to identify protective factors that may reduce rates of gang membership, violent crime, or drug sales, for instance. If reducing rates of violent crime within a neighborhood is more important than substance use for positive development among adolescents, perhaps our risk and protective profiles would be more informative as to mechanism of risk and/or protection if we focus specifically on preventing the factors that are most strongly associated with negative outcomes within a community (e.g., preventing violent crime). This highlights a need for community-based perspectives to understand interrelationships among antisocial or maladaptive behaviors (e.g., what factors prevent unique patterns of co-occurring community violence, substance use, and drug sales, etc.) and community-focused research

partnerships to identify not only what factors may provide protection, but also what behaviors are most important to protect against.

As such, future research should consider a number of directions. First, continuing to assess patterns of substance use etiology within a social-developmental framework. The SDM proposes that adolescents learn patterns of behavior as a result of bonding to various socialization agents, and in turn modify their own behavior to align with the values of the agents to which they have become bonded (Catalano & Hawkins, 1996; Catalano et al., 1996). This socialization process requires interactions with potential bonding agents across time, so future research should also assess the development of etiological factor patterns longitudinally. The SDM proposes that interactions across various domains of influence function together over time via socialization processes to create dynamic patterns of behavior that will change as agents of socialization change, and this intricate interplay should be fully accounted for in future studies of heterogeneity in substance use etiology using the SDM. Further, the SDM outlines sub-models that describe the most important socialization factors present across multiple developmental periods, suggesting that the most important socialization agent may change across time (Catalano & Hawkins, 1996; Catalano et al., 1996). The present study examined these relationships cross-sectionally in order to gain an initial descriptive overview of the types of etiological factor patterns that may exist among low-income African Americans because (1) little research had been done on unique patterns of risk and protective factors within this specific population and (2) little research had focused on combined patterns of etiological factors. However, theory and practice would benefit from future research that examines dynamic interactions in these etiological factors across time.

Additional direct tests of the SDM and related social-developmental theories should include measurements of factors that may affect substance use development specifically outlined in the model and representing specific domains (e.g., family, peer, school, community) that may influence development (Catalano & Hawkins, 1996; Catalano et al., 1996). In

addition to specific potential sources of socialization, including additional individual-level variables that may influence one's likelihood of bonding to specific socialization agents (e.g., individual cognitive or social skills) in the model may further aid in identifying which types of adolescents may interact with certain socialization agents or aspects of the environment in ways that may indicate an increased likelihood for later substance use.

The present study examined trajectories of alcohol, cigarette, and marijuana use separately. Given the observed rates of use among adolescents in the sample (i.e., nearly half of the adolescents reported using alcohol by age 17), it is likely that co-use or use of multiple substances occurs among adolescents. It is of interest for future research to examine the extent to which heterogeneous patterns of etiological factors for substance use predict trajectories of use of multiple substances across adolescence. Further, it is important to examine trajectories of and the extent to which patterns of etiological factors predict likelihood of demonstrating those trajectories of substance use especially for substances that are on the rise among recently nationally-representative samples of adolescents, including nicotine and vaping (Miech, Johnston, O'Malley, Bachman, & Patrick, 2019).

The MYS provides an excellent in-depth examination of a specific population, but the age of the data (which was collected from 1998-2011) may be a source of differences in findings from studies published with more recent data. It is of interest for future researchers and funding providers to encourage new large-scale data collection among impoverished minority adolescents, including the experiences of adolescents from a wide range of minority racial/ethnic, sexual orientation, or other groups who experience systematic oppression. African Americans continue to experience systematic oppression and discrimination in the United States, and their unique risk for substance use and other health-related consequences must be understood within this historical and societal context (Banton, 1998; Hebl et al., 2002; Rothstein, 2015; Williams et al., 1997; Zapolski et al., 2014). In addition to understanding patterns of substance use (and corresponding etiological factors) it is important

for future research to explore (1) whether the socialization process posited in the SDM adequately describes the factors leading to substance use among African Americans, and (2) what factors may be most effective in promoting socialization processes of prosocial behavior among this population. Methods that include community participation in designing research, as has been done in research on other minority groups, may be an effective way of adequately capturing various factors that are particularly relevant to the communities under study (Dobransky-Fasiska et al., 2009).

As previously mentioned, future research should explore what types of prevention and/or intervention strategies may be most efficacious among various subgroups of individuals who express unique patterns of etiological factors and/or substance use habits. Because we found that various unique patterns of etiological factors are differentially associated with risk for substance use, we may use the SDM to theorize potential mechanisms through which we may be able to reduce instances of substance use among adolescents exhibiting each unique pattern of etiological factors. Parental knowledge may be an important family factor to consider in substance use prevention, so additional research may focus on improving parents' ability to monitor their children's behavior in order to gain knowledge about their whereabouts and activities (Piko & Kovács, 2010; Racz & McMahon, 2011; Stattin & Kerr, 2000). Other parenting variables that were not considered in the present study, but are of interest to substance use prevention, include parent-adolescent communication and parental warmth (Lippold, Hussong, Fosco, & Ram, 2018; Schuster, Mermelstein, & Wakschlag, 2013).

The SDM proposes that perceiving opportunities for prosocial involvement is the first step towards bonding to socializing agents whose values are incongruent with antisocial behavior, including substance use, which in turn reduces the likelihood of engaging in antisocial behavior (Catalano & Hawkins, 1996; Catalano et al., 1996). It may also be of interest to examine how opportunities for prosocial involvement in other domains may function as potential protective factors across various subgroups of individuals, including

neighborhood factors such as playing sports or availability of parks; school factors such as connectedness to one's school, opportunities for extracurricular involvement, opportunities to participate in class; and peer factors such as having prosocial peers (e.g., peers who do not use alcohol or drugs, or peers who do not break the law; Catalano et al., 1996). Identifying how these factors may be improved within a specific community, and which types of individuals are most likely to benefit from increased exposure to these factors, may enhance prevention efforts and reduce the likelihood of substance use across various subgroups of adolescents.

Conclusion

African American adolescents continue to be an important population in which to assess risk for substance use given that (1) early initiation into substance use is associated with an increased likelihood of exhibiting substance-related problems later in life (Grant & Dawson, 1998), and (2) African Americans are at an increased risk of substance-related consequences relative to white Americans at the same levels of use (Zapolski et al., 2014). Substance use etiology is multifaceted and deeply rooted within the social context, functioning as a developmental cascade of social influences interacting with individual characteristics to create change in individuals' behavior across time (Catalano & Hawkins, 1996; Catalano et al., 1996; Masten & Cicchetti, 2010). The expression of substance use and its etiology is heterogeneous across various types of African American adolescents, indicating that adolescents who demonstrate unique patterns of etiological factors may also demonstrate unique patterns of substance use; that is, while most broad population studies compare risk and/or protection for substance use across various racial/ethnic (or other demographic) groups, there is just as much heterogeneity within various racial/ethnic groups as there is between them (Caetano et al., 1998).

We may infer from this heterogeneity that individuals who express unique patterns of etiological factors do also exhibit unique likelihoods for substance use, specifically that

delinquency, exposure to violence, peer pressure, and parental knowledge have particular importance in predicting substance use in unique ways across various types of adolescents. Our findings support other calls for prevention research to focus on both risk and protective factors as discrete, yet interacting, dimensions, improving adolescents' exposure to protective factors while reducing their risk exposure (Hawkins, Catalano, & Arthur, 2002). Future research may utilize these findings, as well as that from other future studies on unique patterns of substance use etiology, to develop prevention and/or intervention efforts that may be most effective when provided to individuals characterized by specific patterns of etiological factors.

APPENDICES

APPENDIX A

List of Items Used in Analysis

Delinquency

All items were dichotomous, where 0 = "no" and 1 = '"yes" (K. A. Bolland et al., 2016; Church et al., 2012; Jaggers et al., 2014). These items reflect lifetime incidence of these behaviors.

(1) Have you ever carried a gun?

(2) Have you ever carried a knife or razor?

(3) Have you ever pulled a knife or gun on someone else?

(4) Have you ever cut or stabbed someone else? Have you ever shot a gun at someone else?

(5) Have you ever been involved in a gang?

(6) Have you ever been arrested?

Exposure to Violence

All items were dichotomous, where 0 = "no" and 1 = "yes," and all items asked (Spano et al., 2006, 2010, 2011, 2008, 2012, 2009). Items reflect lifetime incidence of these behaviors.

(1) Has someone ever pulled a knife or gun on you?

(2) Has someone ever cut or stabbed you bad enough that you had to see a doctor?

(3) Has someone ever shot a gun at you?

(4) Has a friend or anyone in your family ever been shot or stabbed?

(5) Have you ever seen someone being cut, stabbed, or shot?

Peer Pressure

All items were responded to on a three-point scale with response options ranging from 0 = "almost none of them" to 2 = "most of them."

(1) How many of your friends think you are a punk if you don't drink alcohol?

(2) How many of your friends think you are a punk if you don't use drugs?

(3) How many of your friends think you are a punk if you don't carry a weapon?

(4) How many of your friends think you are a punk if you don't want to fight after being insulted or dissed or called out?

(5) How many of your friends think you are a punk if you do well in school?

(6) How many of your friends think you are a punk if you don't have sex?

Traumatic Stress

Items were adapted from the Global Appraisal of Individual Needs (GAIN Dennis et al., 1998; Titus et al., 2008) and were responded to on a scale from 0 = "almost never" to 2 = "very often."

(1) I have bad dreams about the bad things that have happened to a family member or friend.

(2) I have trouble sleeping at night when bad things happen to a family member or friend.

(3) I think I would feel better if I could talk to someone about the bad things that happen to a family member or friend.

(4) When bad things happen to a family member or friend, it feels like they are happening to me.

(5) I think about bad things that have happened to a family member or friend, even when I don't want to.

(6) After bad things happen to a family member or friend, I feel uncomfortable being with them because it reminds me of the bad things that happened.

(7) I worry that bad things might happen to a family member or friend.

Neighborhood Connectedness

All items are dichotomous, where 0 = "disagree" and 1 = "agree" (Glynn, 1981; Perkins et al., 1990).

(1) I feel I am an important part of my neighborhood.

(2) If I moved away from my neighborhood, I would be sorry to leave.

(3) Very few of my neighbors know me.

(4) I have friends in my neighborhood who know they can depend on me.

(5) I do not like living in my neighborhood (*reverse-scored*).

(6) There are people in my neighborhood, other than my family, who really care about me.

(7) I have friends in my neighborhood I can depend on.

(8) If you don't look out for yourself in my neighborhood, no one else will (*reverse-scored*).

(9) No one in my neighborhood takes any interest in what their neighbors are doing (*reverse-scored*).

(10) It is hard to make good friends in my neighborhood.

(11) If I am upset about a personal problem, there are people in my neighborhood I can turn to.

Parental Knowledge

These items were responded to with unique item response categories, which are presented with the item below.

(1) Does your mother or father know who you hang out with? (0 = "no," 1 = "yes")

(2) Does your mother or father know exactly where you are most afternoons (after school) and during the day on weekends and during the summer? (0 = "no," 1 = "yes")

(3) How much does your mother or father really know about what you do most afternoons (after school) and during the day on weekends and during the summer? (0 = "they don't know," 1 = "they know a little," 2 = "they know a lot")

(4) How much does your mother or father really know about where you go at night? (0 = "I don't go out at night," 1 = "they don't know," 2 = "they know a little," 3 = "they know a lot")

(5) Does your mother or father try to find out how you spend your time? (0 = "they don't try," 1 = "they try a little," 2 = "they try a lot")

(6) How much does your mother or father really know about how you spend your time? (0 = "they don't know," 1 = "they know a little," 2 = "they know a lot")

Religiosity

These items were responded to with unique item response categories, which are presented with the item below.

(1) About how often do you go to church, worship services, or other religious activities? (0 = "never," 1 = "once in a while," 2 = "about once a month," 3 = "about 2 or 3 times a month," 4 = "once a week or more")

(2) How important is religion to you? (0 = "not important," 1 = "somewhat important," 2 = "very important")

(3) How often do you read or study a Holy Book (such as The Bible)? (0 = "never," 1 = "once in a while," 2 = "about once a month," 3 = "about 2 or 3 times a month," 4 = "once a week or more")

Self-Worth

These items are structured such that adolescents are asked to select which of two statements is most reflective of how they think about themselves. We will present the instructions for these items, in addition to the two statements that comprise each item.

Now we are interested in how you think about yourself. For each of the following questions, please indicate which of the two statements that are listed is most like you.

(1) "I am usually unhappy with myself" or "I am usually happy with myself"

(2) "I sometimes do things I know I shouldn't do" or 11I hardly ever do things I know I shouldn't do"

(3) "I usually don't like the way I behave" or "I usually like the way I behave"

(4) "I like the kind of person I am" or "I don't like the kind of person I am"

(5) "I usually get into trouble because of the things I do" or "I usually don't do things that get me into trouble"

(6) "I usually make good decisions" or "I usually don't make good decisions"

(7) "I usually behave myself very well" or "I often find it hard to behave myself"

(8) "I am not happy with the way I do a lot of things" or "The way I do things is fine"

(9) "I don't like the way I am leading my life" or "I like the way I am leading my life"

APPENDIX B

Mplus Syntax for Final Analyses

Aim 1

```
TITLE:  Protective Factor LPA
DATA:
  FILE IS MYS_ForAnalysis_121418.dat;
  FORMAT IS F8.0 85F1.0 8F8.3;
  TYPE IS INDIVIDUAL;
VARIABLE:
  NAMES ARE
  ID SEX RACE DEL1-DEL6 EXV1-EXV5 PP1-PP6
  TS1-TS7 NC1-NC11 REL1-REL3 SW1-SW9
  PK1-PK6 CIG13-CIG17 ALC13-ALC17
  MARJ13-MARJ17 ALCT13-ALCT17 CIGT13-CIGT17
  MARJT13-MARJT17 DELFS EXVFS NCFS PKFS
  PPFS RELFS SWFS TSFS;
  MISSING = BLANK;
  USEVARIABLES ARE NCFS PKFS RELFS SWFS ;
  CLASSES = c (2);
  IDVARIABLE = ID;
ANALYSIS:
  TYPE = MIXTURE;
  ESTIMATOR = MLR;
  STARTS = 200 50;
  PROCESSORS = 2 (STARTS);
OUTPUT:
```

```
  TECH1 TECH11; !TECH8
SAVEDATA:
  FILE IS MYS_PROTECT_LPA_3_CPROB.csv;
  SAVE = CPROB;


TITLE:  Risk Factor LPA
DATA:
  FILE IS MYS_ForAnalysis_121418.dat;
  FORMAT IS F8.0 85F1.0 8F8.3;
  TYPE IS INDIVIDUAL;
VARIABLE:
  NAMES ARE
  ID SEX RACE DEL1-DEL6 EXV1-EXV5 PP1-PP6
  TS1-TS7 NC1-NC11 REL1-REL3 SW1-SW9
  PK1-PK6 CIG13-CIG17 ALC13-ALC17
  MARJ13-MARJ17 ALCT13-ALCT17 CIGT13-CIGT17
  MARJT13-MARJT17 DELFS EXVFS NCFS PKFS
  PPFS RELFS SWFS TSFS;
  MISSING = BLANK;
  USEVARIABLES ARE DELFS EXVFS PPFS TSFS ;
  CLASSES = c (6);
  IDVARIABLE = ID;
ANALYSIS:
  TYPE = MIXTURE;
  ESTIMATOR = MLR;
  STARTS = 6400 1600
  PROCESSORS = 2 (STARTS);
OUTPUT:
```

```
  TECH1 TECH11; !TECH8
SAVEDATA:
  FILE IS MYS_RISK_LPA_6_CPROB.csv;
  SAVE = CPROB;
```

Aim 2

```
TITLE:  Risk and Protective Factor LPA
DATA:
  FILE IS MYS_ForAnalysis_121418.dat;
  FORMAT IS F8.0 85F1.0 8F8.3;
  TYPE IS INDIVIDUAL;
VARIABLE:
  NAMES ARE
  ID SEX RACE DEL1-DEL6 EXV1-EXV5 PP1-PP6
  TS1-TS7 NC1-NC11 REL1-REL3 SW1-SW9
  PK1-PK6 CIG13-CIG17 ALC13-ALC17
  MARJ13-MARJ17 ALCT13-ALCT17 CIGT13-CIGT17
  MARJT13-MARJT17 DELFS EXVFS NCFS PKFS
  PPFS RELFS SWFS TSFS;
  MISSING = BLANK;
  USEVARIABLES ARE DELFS EXVFS PPFS TSFS NCFS
  PKFS RELFS SWFS ;
  CLASSES = c (6);
  IDVARIABLE = ID;
ANALYSIS:
  TYPE = MIXTURE;
  ESTIMATOR = MLR;
```

```
  STARTS = 26500 6400;
  PROCESSORS = 2 (STARTS);
  !ALGORITHM = INTEGRATION;
OUTPUT:
  TECH1 TECH11; !TECH8
SAVEDATA:
  FILE IS MYS_RP_LPA_6_CPROB.csv;
  SAVE = CPROB;
```

Aim 3

Alcohol Growth Mixture Model

```
TITLE:  Alcohol GMM
DATA:
  FILE IS MYS_ForAnalysis_120618.dat;
  FORMAT IS F8.0 85F1.0;
  TYPE IS INDIVIDUAL;
VARIABLE:
  NAMES ARE
  ID SEX RACE DEL1-DEL6 EXV1-EXV5 PP1-PP6
  TS1-TS7 NC1-NC11 REL1-REL3 SW1-SW9
  PK1-PK6 CIG13-CIG17 ALC13-ALC17
  MARJ13-MARJ17 ALCT13-ALCT17 CIGT13-CIGT17
  MARJT13-MARJT17;
  MISSING = BLANK;
  USEVARIABLES ARE ALCT13-ALCT17 ;
  CATEGORICAL ARE ALCT13-ALCT17 ;
  CLASSES = ac (2);
```

```
  IDVARIABLE = ID;
ANALYSIS:
  TYPE = MIXTURE;
  ESTIMATOR = MLR;
  STARTS = 100 25;
  PROCESSORS = 2 (STARTS);
  ALGORITHM = INTEGRATION;
MODEL:
  %OVERALL%
  i s | ALCT13@0 ALCT14@1 ALCT15@2 ALCT16@3 ALCT17@4;
  !%ac#1%
  !i@0;
  !s@0;
  !%ac#2%
  !i@0;
  !s@0;
OUTPUT:
  TECH1 TECH11; !TECH8
PLOT:
  TYPE = PLOT3;
  SERIES = ALCT13-ALCT17 (s);
SAVEDATA:
  FILE IS MYS_ALCT_GMM_2_CPROB.csv;
  SAVE = CPROB;
```

Cigarette Latent Growth Model

```
TITLE:  Cigarette Growth Trajectory
```

```
(Polytomous - 3 categories)
DATA:
  FILE IS MYS_ForAnalysis_032119.dat;
  FORMAT IS F8.0 84F1.0 8F8.3 5F1.0;
  TYPE IS INDIVIDUAL;
VARIABLE:
  NAMES ARE
  ID SEX DEL1-DEL6 EXV1-EXV5 PP1-PP6
  TS1-TS7 NC1-NC11 REL1-REL3 SW1-SW9
  PK1-PK6 CIG13-CIG17 ALC13-ALC17
  MARJ13-MARJ17 ALCT13-ALCT17 CIGT13-CIGT17
  MARJT13-MARJT17 DELFS EXVFS NCFS PKFS
  PPFS RELFS SWFS TSFS ALCO13-ALCO17;
  MISSING = BLANK;
  USEVARIABLES ARE CIGT13-CIGT17 ;
  CATEGORICAL ARE CIGT13-CIGT17 ;
  IDVARIABLE = ID;
ANALYSIS:
  ESTIMATOR = MLR;
MODEL:
  i s | CIGT13@0 CIGT14@1 CIGT15@2 CIGT16@3 CIGT17@4;
OUTPUT:
  TECH1 ; !TECH8 TECH11
```

Marijuana Latent Growth Curve Model

```
TITLE:  Marijuana Growth Trajectory
(Polytomous - 3 categories)
```

```
DATA:
  FILE IS MYS_ForAnalysis_032119.dat;
  FORMAT IS F8.0 84F1.0 8F8.3 5F1.0;
  TYPE IS INDIVIDUAL;
VARIABLE:
  NAMES ARE
  ID SEX DEL1-DEL6 EXV1-EXV5 PP1-PP6
  TS1-TS7 NC1-NC11 REL1-REL3 SW1-SW9
  PK1-PK6 CIG13-CIG17 ALC13-ALC17
  MARJ13-MARJ17 ALCT13-ALCT17 CIGT13-CIGT17
  MARJT13-MARJT17 DELFS EXVFS NCFS PKFS
  PPFS RELFS SWFS TSFS ALCO13-ALCO17;
  MISSING = BLANK;
  USEVARIABLES ARE MARJT13-MARJT17 ;
  CATEGORICAL ARE MARJT13-MARJT17 ;
  IDVARIABLE = ID;
ANALYSIS:
  ESTIMATOR = MLR;
MODEL:
  i s | MARJT13@0 MARJT14@1 MARJT15@2 MARJT16@3 MARJT17@4;
OUTPUT:
  TECH1 ; !TECH8 TECH11
```

Aim 4.a.

Conditional patterns predicting most likely latent alcohol use trajectory classes

```
TITLE:  Etiological Factors Predicting
Alcohol Classes
```

```
(Polytomous - 3 categories)
DATA:
  FILE IS MYS_ForFinalAnalysis_042419.dat;
  FORMAT IS F8.0 16F1.0 11F8.3 F1.0 6F8.3
  F1.0 18F4.0 6F11.5 6F1.0 6F11.5 2F1.0;
  TYPE IS INDIVIDUAL;
VARIABLE:
  NAMES ARE
  ID SEX ALCT13-ALCT17 CIGT13-CIGT17
  MARJT13-MARJT17 DELFS EXVFS NCFS PKFS
  PPFS RELFS SWFS TSFS PCP1-PCP3 PCH
  RCP1-RCP6 RCH JCH JC1 JC3-JC18 ECP1-ECP6
  ECH ECD1 ECD3-ECD6 AI AS AC_I AC_S
  ACP1 ACP2 ACH ACD;
  MISSING = BLANK;
  USEVARIABLES ARE SEX JC1 JC3-JC18
  ACD ;
  CATEGORICAL ARE ACD  ;
  IDVARIABLE = ID;
ANALYSIS:
  ESTIMATOR = MLR;
MODEL:
  ACD ON SEX JC1 JC3-JC18;
OUTPUT:
  TECH1 TECH4 ; !TECH8 TECH11
```

Conditional patterns predicting cigarette latent growth curve model

```
TITLE:  Etiological Factors Predicting
Cigarette Growth Trajectory
(Polytomous - 3 categories)
DATA:
  FILE IS MYS_ForFinalAnalysis_042419.dat;
  FORMAT IS F8.0 16F1.0 11F8.3 F1.0 6F8.3
  F1.0 18F4.0 6F11.5 6F1.0 6F11.5 2F1.0;
  TYPE IS INDIVIDUAL;
VARIABLE:
  NAMES ARE
  ID SEX ALCT13-ALCT17 CIGT13-CIGT17
  MARJT13-MARJT17 DELFS EXVFS NCFS PKFS
  PPFS RELFS SWFS TSFS PCP1-PCP3 PCH
  RCP1-RCP6 RCH JCH JC1-JC18 ECP1-ECP6
  ECH ECD1 ECD3-ECD6 AI AS AC_I AC_S
  ACP1 ACP2 ACH ACD;
  MISSING = BLANK;
  USEVARIABLES ARE SEX JC1 JC3-JC18
  CIGT13-CIGT17 ;
  CATEGORICAL ARE CIGT13-CIGT17 ;
  IDVARIABLE = ID;
ANALYSIS:
  ESTIMATOR = MLR;
MODEL:
  i s | CIGT13@0 CIGT14@1 CIGT15@2 CIGT16@3 CIGT17@4;
  i s ON SEX JC1 JC3-JC18;
OUTPUT:
```

```
  TECH1 ; !TECH8 TECH11
```

Conditional patterns predicting marijuana latent growth curve model

```
TITLE:  Etiological Factors Predicting
Marijuana Growth Trajectory
(Polytomous - 3 categories)
DATA:
  FILE IS MYS_ForFinalAnalysis_042419.dat;
  FORMAT IS F8.0 16F1.0 11F8.3 F1.0 6F8.3
  F1.0 18F4.0 6F11.5 6F1.0 6F11.5 2F1.0;
  TYPE IS INDIVIDUAL;
VARIABLE:
  NAMES ARE
  ID SEX ALCT13-ALCT17 CIGT13-CIGT17
  MARJT13-MARJT17 DELFS EXVFS NCFS PKFS
  PPFS RELFS SWFS TSFS PCP1-PCP3 PCH
  RCP1-RCP6 RCH JCH JC1-JC18 ECP1-ECP6
  ECH ECD1 ECD3-ECD6 AI AS AC_I AC_S
  ACP1 ACP2 ACH ACD;
  MISSING = BLANK;
  USEVARIABLES ARE SEX JC1 JC3-JC18
  MARJT13-MARJT17 ;
  CATEGORICAL ARE MARJT13-MARJT17;
  IDVARIABLE = ID;
ANALYSIS:
  ESTIMATOR = MLR;
MODEL:
```

```
  i s | MARJT13@0 MARJT14@1 MARJT15@2 MARJT16@3 MARJT17@4;
  i s ON SEX JC1 JC3-JC18;
OUTPUT:
  TECH1 ; !TECH8 TECH11
```

Aim 4.b.

Etiological factor patterns predicting most likely latent alcohol use trajectory classes

```
TITLE:  Etiological Factors Predicting
Alcohol Classes
(Polytomous - 3 categories)
DATA:
  FILE IS MYS_ForFinalAnalysis_042419.dat;
  FORMAT IS F8.0 16F1.0 11F8.3 F1.0 6F8.3
  F1.0 18F4.0 6F11.5 6F1.0 6F11.5 2F1.0;
  TYPE IS INDIVIDUAL;
VARIABLE:
  NAMES ARE
  ID SEX ALCT13-ALCT17 CIGT13-CIGT17
  MARJT13-MARJT17 DELFS EXVFS NCFS PKFS
  PPFS RELFS SWFS TSFS PCP1-PCP3 PCH
  RCP1-RCP6 RCH JCH JC1 JC3-JC18 ECP1-ECP6
  ECH ECD1 ECD3-ECD6 AI AS AC_I AC_S
  ACP1 ACP2 ACH ACD;
  MISSING = BLANK;
  USEVARIABLES ARE SEX ECD1 ECD3-ECD6
  ACD ;
  CATEGORICAL ARE ACD  ;
```

```
  IDVARIABLE = ID;
ANALYSIS:
  ESTIMATOR = MLR;
MODEL:
  ACD ON SEX ECD1 ECD3-ECD6;
OUTPUT:
  TECH1 TECH4 ; !TECH8 TECH11
```

Etiological factor patterns predicting cigarette latent growth curve model

```
TITLE:  Etiological Factors Predicting
  Cigarette Growth Trajectory
  (Polytomous - 3 categories)
 DATA:
    FILE IS MYS_ForFinalAnalysis_042419.dat;
    FORMAT IS F8.0 16F1.0 11F8.3 F1.0 6F8.3
    F1.0 18F4.0 6F11.5 6F1.0 6F11.5 2F1.0;
    TYPE IS INDIVIDUAL;
 VARIABLE:
    NAMES ARE
    ID SEX ALCT13-ALCT17 CIGT13-CIGT17
    MARJT13-MARJT17 DELFS EXVFS NCFS PKFS
    PPFS RELFS SWFS TSFS PCP1-PCP3 PCH
    RCP1-RCP6 RCH JCH JC1-JC18 ECP1-ECP6
    ECH ECD1 ECD3-ECD6 AI AS AC_I AC_S
    ACP1 ACP2 ACH ACD;
    MISSING = BLANK;
    USEVARIABLES ARE SEX ECD1 ECD3-ECD6
```

```
    CIGT13-CIGT17 ;
    CATEGORICAL ARE CIGT13-CIGT17 ;
    IDVARIABLE = ID;
 ANALYSIS:
    ESTIMATOR = MLR;
    MCONVERGENCE = .001;
    INTEGRATION = 30;
MODEL:
    i s | CIGT13@0 CIGT14@1 CIGT15@2 CIGT16@3 CIGT17@4;
    i s ON SEX ECD1 ECD3-ECD6;
OUTPUT:
    TECH1 ; !TECH8 TECH11
```

Etiological factor patterns predicting marijuana latent growth curve model

```
TITLE:  Etiological Factors Predicting
Marijuana Growth Trajectory
(Polytomous - 3 categories)
DATA:
  FILE IS MYS_ForFinalAnalysis_042419.dat;
  FORMAT IS F8.0 16F1.0 11F8.3 F1.0 6F8.3
  F1.0 18F4.0 6F11.5 6F1.0 6F11.5 2F1.0;
  TYPE IS INDIVIDUAL;
VARIABLE:
  NAMES ARE
  ID SEX ALCT13-ALCT17 CIGT13-CIGT17
  MARJT13-MARJT17 DELFS EXVFS NCFS PKFS
  PPFS RELFS SWFS TSFS PCP1-PCP3 PCH
```

```
  RCP1-RCP6 RCH JCH JC1-JC18 ECP1-ECP6
  ECH ECD1 ECD3-ECD6 AI AS AC_I AC_S
  ACP1 ACP2 ACH ACD;
  MISSING = BLANK;
  USEVARIABLES ARE SEX ECD1 ECD3-ECD6
  MARJT13-MARJT17 ;
  CATEGORICAL ARE MARJT13-MARJT17 ;
  IDVARIABLE = ID;
ANALYSIS:
  ESTIMATOR = MLR;
MODEL:
  i s | MARJT13@0 MARJT14@1 MARJT15@2 MARJT16@3 MARJT17@4;
  i s ON SEX ECD1 ECD3-ECD6;
OUTPUT:
  TECH1 ; !TECH8 TECH11
```

APPENDIX C

Supplementary Tables

Correlations Between All Etiological Factor Scores

Pearson correlations were conducted on all etiological factors after factor scores had been extracted from the confirmatory factor models. The correlations are available in Table C.1.

Overlap in Conditional and Etiological Profile Memberhship

We examined patterns of (1) conditional membership in risk and protective factor patterns and (2) membership in etiological factor patterns. To examine the extent to which individuals likely to be members of each conditional association were also likely to be members in each etiological factor pattern, we examined crosstabs of most likely etiological factor pattern and conditional association membership. The results are presented in Table C.2.

Sample Sizes in Selecting Best-Fitting Profile Models

In latent profile analysis, convention suggests that in the best-fitting model all derived profiles should represent at least 5% of the sample. In the text we reference two instances where we selected a latent profile model due to this criteria. As such, we present the number of individuals most likely to be members of each latent profile across all models tested in Tables C.3-C.5 (i.e., models testing 2-7 latent profiles for risk and etiological factors, and models testing 2-4 profiles for protective factors).

Table C.1

Correlations Between All Etiological Factor Scores

Factor	Delinquency	Exposure to violence	Peer pressure	Traumatic stress	Neighborhood connectedness	Parental knowledge	Religiosity	Self-worth
Delinquency	1.00	-	-	-	-	-	-	-
Exposure to violence	.63**	1.00	-	-	-	-	-	-
Peer pressure	.23**	.21**	1.00	-	-	-	-	-
Traumatic stress	.03	.10**	.07**	1.00	-	-	-	-
Neighborhood connectedness	-.04	-.02	-.11**	.11**	1.00	-	-	-
Parental knowledge	-.27**	-.23**	-.13**	.15**	.13**	1.00	-	-
Religiosity	-.09**	.01	-.06*	.12**	.09**	.21**	1.00	-
Self-worth	-.30**	-.22**	-.23**	-.08**	.10**	.23**	.12**	1.00

Note. $*p < .05$, $**p < .001$.

Table C.2

Crosstabs of Conditional Profile Membership and Etiological Factor Profile Membership

Conditional membership	Low risk/ Average protection	Average risk and protection	Some delinquency/average protection	Elevated risk/decreased protection	Moderate risk/decreased protection	High risk/ low protection
Low risk & high P.K.	376 (24.0%)	0 (0.0%)	0 (0.0%)	0 (0.0%)	0 (0.0%)	0 (0.0%)
Low risk & average protection	255 (16.3%)	0 (0.0%)	0 (0.0%)	0 (0.0%)	0 (0.0%)	0 (0.0%)
Low risk & low protection	57 (3.6%)	0 (0.0%)	0 (0.0%)	0 (0.0%)	0 (0.0%)	0 (0.0%)
Average risk & high P.K.	0 (0.0%)	127 (8.7%)	0 (0.0%)	0 (0.0%)	0 (0.0%)	0 (0.0%)
Average risk & average protection	0 (0.0%)	120 (7.7%)	0 (0.0%)	0 (0.0%)	0 (0.0%)	0 (0.0%)
Average risk & low protection	0 (0.0%)	27 (1.7%)	0 (0.0%)	0 (0.0%)	0 (0.0%)	0 (0.0%)
Some delinquency & high P.K.	0 (0.0%)	0 (0.0%)	71 (4.5%)	0 (0.0%)	0 (0.0%)	0 (0.0%)
Some delinquency & average protection	0 (0.0%)	0 (0.0%)	76 (4.9%)	0 (0.0%)	0 (0.0%)	0 (0.0%)
Some delinquency & low protection	0 (0.0%)	0 (0.0%)	22 (1.4%)	0 (0.0%)	0 (0.0%)	0 (0.0%)
Elev. delinquency & low protection	0 (0.0%)	0 (0.0%)	0 (0.0%)	28 (1.8%)	1 (0.1%)	0 (0.0%)
Elev. delinquency & high P.K.	0 (0.0%)	0 (0.0%)	0 (0.0%)	57 (3.6%)	0 (0.0%)	0 (0.0%)

(*continued*)

Elev. delinquency & average protection	0 (0.0%)	0 (0.0%)	0 (0.0%)	81 (5.2%)	0 (0.0%)	0 (0.0%)
D.V.E. & high P.K.	0 (0.0%)	0 (0.0%)	0 (0.0%)	1 (0.1%)	41 (2.6%)	0 (0.0%)
D.V.E. & average protection	0 (0.0%)	0 (0.0%)	0 (0.0%)	0 (0.0%)	77 (4.9%)	0 (0.0%)
D.V.E. & low protection	0 (0.0%)	0 (0.0%)	0 (0.0%)	0 (0.0%)	27 (1.7%)	0 (0.0%)
Multi-risk & high P.K.	0 (0.0%)	0 (0.0%)	0 (0.0%)	0 (0.0%)	1 (0.1%)	21 (1.3%)
Multi-risk & average protection	0 (0.0%)	0 (0.0%)	0 (0.0%)	0 (0.0%)	0 (0.0%)	66 (4.2%)
Multi-risk & low protection	0 (0.0%)	0 (0.0%)	0 (0.0%)	0 (0.0%)	0 (0.0%)	35 (2.2%)

Note. Percentages reflect percentage of total sample. P.K. = parental knowledge, D.V.E = delinquency & violence exposure.

Table C.3

Number of Individuals Most Likely to be Members of Each Latent Protective Factor Profile

Profile	2	3	4
1	836 (53.4%)	695 (44.4%)	534 (34.1%)
2	731 (46.6%)	675 (43.1%)	487 (31.1%)
3		197 (12.6%)	459 (29.3%)
4			87 (5.6%)

Note. Parentheses indicate percentage of total sample likely to demonstrate each latent profile. There may be rounding error due to the probabilistic nature of LPA.

Table C.4

Number of Individuals Most Likely to be Members of Each Latent Risk Factor Profile

Profile	2	3	4	5	6	7
1	1159 (74.0%)	724 (46.2%)	688 (43.9%)	688 (43.9%)	688 (43.9%)	688 (43.9%)
2	408 (26.0%)	562 (35.9%)	362 (23.1%)	319 (20.4%)	274 (17.5%)	274 (17.5%)
3		213 (13.6%)	304 (19.4%)	260 (16.6%)	169 (10.8%)	169 (10.8%)
4			281 (17.9%)	172 (11.0%)	167 (10.7%)	148 (9.4%)
5				128 (8.2%)	146 (9.3%)	123 (7.8%)
6					123 (7.8%)	110 (7.0%)
7						55 (3.5%)

Note. Parentheses indicate percentage of total sample likely to demonstrate each latent profile. There may be rounding error due to the probabilistic nature of LPA.

Selection of Substance Use Trajectory Models

The text presents the selection of the best-fitting single latent growth curve model, and then testing mixtures of those best-fitting trajectories. The tables below present all models considered when selecting the best-fitting latent growth curve for alcohol, cigarette, and marijuana use, separately.

Table C.5

Number of Individuals Most Likely to be Members of Each Latent Etiological Factor Profile

Profile	2	3	4	5	6	7
1	1128 (72.0%)	733 (46.8%)	688 (43.9%)	688 (43.9%)	688 (43.9%)	688 (43.9%)
2	439 (28.0%)	559 (35.7%)	367 (23.4%)	320 (20.4%)	274 (17.5%)	274 (17.5%)
3		275 (17.5%)	298 (19.0%)	259 (16.5%)	169 (10.8%)	169 (10.8%)
4			214 (13.7%)	171 (11.0%)	167 (10.7%)	150 (9.6%)
5				129 (8.2%)	147 (9.4%)	121 (7.7%)
6					122 (7.8%)	110 (7.0%)
7						55 (3.5%)

Note. Parentheses indicate percentage of total sample likely to demonstrate each latent profile. There may be rounding error due to the probabilistic nature of LPA.

For the alcohol data, we tested models with one through three classes when variance was allowed to be freely estimated within class, and models with one through four classes when variance was fixed within class. This was because when variance was allowed to freely vary either within or across class, the three-class model either 1) suggested that a two-class model had better fit to the data, or 2) had one or more classes that best characterized less than 5% of the sample; as such, we did not explore whether a fourth trajectory could be extracted. We determined that a two-class linear trajectory model wherein variances within class (i.e., intercept variance and slope variance) were allowed to freely vary, but that variances across classes could be fixed to equality, best fit the data given that this model demonstrated the lowest BIC and SSA-BIC across all models, the *p*-values for the LMR-ALRT and VLMR-LRT suggested the addition of a second class significantly improved model fit, and that the second class comprised nearly 5% of the total sample.

We followed the same procedure when examining models for cigarette use. After completing all model tests, we concluded that there was not sufficient evidence to indicate the presence of heterogeneous subgroups of cigarette use, and elected to not include a mixture component, retaining a single growth trajectory. While the VLMR-LRT and LMR-ALRT suggested that a two- or three-class solution may have good fit to the data in certain model tests, these models had low entropy (< 0.7), suggesting less than optimal classification of individuals into trajectory classes. Given that further stages of the analysis required classifying individuals into their most likely latent class membership, we did not feel that such classification was appropriate given the low classification accuracy indicated by entropy. Additionally, the model for a single linear trajectory allowing for individual variation around the growth parameters had the lowest BIC and SSA-BIC of all tested models for cigarette use. As such, we retained a single homogeneous trajectory of cigarette use that allowed for individual variation around the trajectory.

We once again followed an identical procedure when testing models for marijuana use. Similar to the trajectories for cigarette use, while the VLMR-LRT and LMR-ALRT suggested for some models that a two- or three-class solution may have good fit to the data, these models either had low entropy (< 0.7), indicating less than optimal classification of individuals into trajectories, or one of the trajectories contained less than 5% of the total sample. Additionally, the model for a single linear trajectory had the lowest BIC value, while the model for a single quadratic trajectory had the lowest SSA-BIC value of all tested models. We selected the single linear trajectory over the quadratic trajectory because we did not feel that the raw frequencies of use indicated the potential for nonlinear growth to a greater extent than was indicated for either cigarette or alcohol use.

Table C.6

Model Selection Criteria for Growth Models of Alcohol Use Trajectories

Classes	Within class	Between class	Quadratic	BIC	SSA-BIC	Entropy	VLMR-LRT	*p*	LMR-ALRT	*p*
1	Free	-	No	9205.64	9186.58	-	-	-	-	-
	Fixed	-	No	9293.80	9584.27	-	-	-	-	-
	Free	-	Yes	9225.76	9193.99	-	-	-	-	-
	Fixed	-	Yes	9300.71	9288.00	-	-	-	-	-
2	Fixed	Fixed	No	9177.80	9158.74	0.76	138.06	.0000	132.08	.0000
	Fixed	Fixed	Yes	9183.34	9157.92	0.80	146.80	.0000	141.97	.0000
	Free	*Fixed*	*No*	*9168.52*	*9139.92*	*0.78*	*59.20*	*.0001*	*56.63*	*.0001*
	Free	Free	No	9178.67	9143.73	0.67	63.75	.0000	62.06	.0000
	Free	Fixed	Yes	9188.62	9144.15	0.80	66.57	.0000	64.38	.0000
	Free	Free	Yes	9199.30	9148.47	0.51	70.60	.0038	69.04	.0042
3	Fixed	Fixed	No	9172.79	9144.20	0.37	27.08	.0014	25.90	.0018
	Fixed	Fixed	Yes	9179.44	9141.31	0.40	33.33	.0014	32.23	.0018
	Free	Fixed	No	9182.23	9144.11	0.73	8.35	.0028	7.99	.0035
	Free	Free	No	9213.09	9162.26	0.35	2.39	.6673	2.33	.6736
	Free	Fixed	Yes	9208.85	9151.67	0.84	9.20	.0041	8.89	.0048
	Free	Free	Yes	9237.60	9167.71	0.47	11.57	.0628	11.32	.0643
4	Fixed	Fixed	No	9182.51	9144.39	0.44	12.35	.0106	11.81	.0127
	Fixed	Fixed	Yes	9198.54	9147.72	0.51	10.32	.0044	9.98	.0053

Note. Italicized text indicates chosen model. Table presents all models considered. *Within class* refers to whether within-class variability was fixed to equality across all individuals, or allowed to be free (vary) across individuals within the same class. *Between class* refers to whether between-class variability was fixed to equality across all classes, or allowed to be free (vary) across classes.

Table C.7

Model Selection Criteria for Growth Models of Cigarette Use Trajectories

Classes	Within class	Between class	Quadratic	BIC	SSA-BIC	Entropy	VLMR-LRT	*p*	LMR-ALRT	*p*
1	*Free*	-	*No*	*6569.23*	*6577.17*	-	-	-	-	-
	Fixed	-	No	6833.66	6824.13	-	-	-	-	-
	Free	-	Yes	6618.86	6587.09	-	-	-	-	-
	Fixed	-	Yes	6838.73	6826.02	-	-	-	-	-
2	Fixed	Fixed	No	6619.48	6600.42	0.54	236.24	.0000	226.00	.0000
	Fixed	Fixed	Yes	6631.49	6606.07	0.55	236.66	.0000	228.88	.0000
	Free	Fixed	No	6611.49	6582.90	0.87	6.80	.1082	6.51	.1187
	Free	Free	No	6622.34	6587.39	0.75	10.67	.5051	10.38	.5129
	Free	Fixed	Yes	N.C.	N.C.	N.C.	N.C.	N.C.	N.C.	N.C.
	Free	Free	Yes	N.C.	N.C.	N.C.	N.C.	N.C.	N.C.	N.C.
3	Fixed	Fixed	No	6616.02	6587.42	0.50	25.53	.0019	24.43	.0024
	Fixed	Fixed	Yes	6635.78	6597.66	0.51	25.13	.2135	24.30	.2214
4	Fixed	Fixed	No	6628.07	6589.95	0.51	10.01	.0862	9.58	.0946

Note. Italicized text indicates chosen model. Table presents all models considered. N.C. = model was tested, but did not converge. *Within class* refers to whether within-class variability was fixed to equality across all individuals, or allowed to be free (vary) across individuals within the same class. *Between class* refers to whether between-class variability was fixed to equality across all classes, or allowed to be free (vary) across classes.

Table C.8

Model Selection Criteria for Growth Models of Marijuana Use Trajectories

Classes	Within class	Between class	Quadratic	BIC	SSA-BIC	Entropy	VLMR-LRT	*p*	LMR-ALRT	*p*
1	*Free*	-	*No*	*7630.97*	*7611.91*	-	-	-	-	-
	Fixed	-	No	7857.23	7847.70	-	-	-	-	-
	Free	-	Yes	7639.93	7608.16	-	-	-	-	-
	Fixed	-	Yes	7858.95	7846.25	-	-	-	-	-
2	Fixed	Fixed	No	7657.89	7638.83	0.45	221.41	.0000	211.81	.0000
	Fixed	Fixed	Yes	7661.03	7635.61	0.44	227.35	.0000	219.88	.0000
	Free	Fixed	No	7642.50	7613.91	0.88	10.54	.0692	10.08	.0770
	Free	Free	No	7656.42	7621.47	0.86	11.33	.0212	11.03	.0238
	Free	Fixed	Yes	7661.53	7617.06	0.94	7.82	.2853	7.57	.2952
	Free	Free	Yes	7679.81.	7625.81	0.19	11.62	.1129	11.39	.1180
3	Fixed	Fixed	No	7656.73	7628.13	0.34	23.23	.0054	22.23	.0066
	Fixed	Fixed	Yes	7654.48	7616.36	0.38	35.97	.0000	34.79	.0001
4	Fixed	Fixed	No	7659.06	7620.94	0.46	19.74	.0041	18.88	.0052

Note. Italicized text indicates chosen model. Table presents all models considered. *Within class* refers to whether within-class variability was fixed to equality across all individuals, or allowed to be free (vary) across individuals within the same class. *Between class* refers to whether between-class variability was fixed to equality across all classes, or allowed to be free (vary) across classes.

Table C.9

Proportion of Individuals Likely to Demonstrate Conditional Risk and Protective Factor Patterns Endorsing Alcohol Use

Risk Pattern	Protective Pattern	Use	Age 13	Age 14	Age 15	Age 16	Age 17
Low risk	High parental knowledge	No	75.6	64.8	55.9	58.1	57.3
		Yes, just once	18.2	24.1	28.5	26.6	23.2
		Yes, more than once	6.2	11.1	15.6	15.3	19.5
Low risk	Average protection	No	60.9	60.5	61.0	47.3	51.2
		Yes, just once	26.8	27.9	28.8	28.4	28.9
		Yes, more than once	12.3	11.6	10.3	24.3	19.8
Low risk	Low protection	No	68.2	73.2	66.7	67.6	65.5
		Yes, just once	13.6	12.2	19.4	20.6	20.7
		Yes, more than once	18.2	14.6	13.9	11.8	13.8
Average risk	High parental knowledge	No	65.9	63.8	48.6	51.6	45.8
		Yes, just once	26.4	20.2	33.8	28.1	27.1
		Yes, more than once	7.7	16.0	17.6	20.3	27.1
Average risk	Average protection	No	61.1	61.0	44.2	61.8	42.9
		Yes, just once	23.3	23.2	29.9	22.4	28.6
		Yes, more than once	15.6	15.9	26.0	15.8	28.6
Average risk	Low protection	No	57.9	73.7	58.8	64.3	50.0
		Yes, just once	21.1	26.3	17.6	7.1	21.4
		Yes, more than once	21.1	0	23.5	28.6	28.6
Some delinquency	High parental knowledge	No	61.5	62.3	60.0	51.0	42.9
		Yes, just once	25.0	22.6	20.0	24.5	28.6
		Yes, more than once	13.5	15.1	20.0	24.5	28.6
Some delinquency	Average protection	No	60.7	64.0	45.3	27.8	45.2

(*continued*)

		Yes, just once	23.0	20.0	32.1	55.6	16.1
		Yes, more than once	16.4	16.0	22.6	16.7	38.7
Some delinquency	Low protection	No	86.7	60.0	72.2	70.0	50.0
		Yes, just once	13.3	20.0	22.2	10.0	40.0
		Yes, more than once	0	20.0	5.6	20.0	10.0
Elevated delinquency	High parental knowledge	No	74.3	52.5	50.0	45.5	53.1
		Yes, just once	17.1	35.0	25.0	22.7	25.0
		Yes, more than once	8.6	12.5	25.0	31.8	21.9
Elevated delinquency	Average protection	No	62.1	73.6	61.4	51.1	50.0
		Yes, just once	15.2	15.1	25.0	34.0	19.0
		Yes, more than once	22.7	11.3	13.6	14.9	31.0
Elevated delinquency	Low protection	No	50.0	40.0	62.5	50.0	57.1
		Yes, just once	30.0	30.0	18.8	33.3	14.3
		Yes, more than once	20.0	30.0	18.8	16.7	28.6
Delinquency & violence exposure	High parental knowledge	No	58.6	51.5	46.2	43.5	61.9
		Yes, just once	24.1	33.3	38.5	26.1	19.0
		Yes, more than once	17.2	15.2	15.4	30.4	19.0
Delinquency & violence exposure	Average protection	No	51.0	52.2	47.8	48.8	34.5
		Yes, just once	27.5	21.7	23.9	31.7	34.5
		Yes, more than once	21.6	26.1	28.3	19.5	31.0
Delinquency & violence exposure	Low protection	No	55.6	57.1	66.7	54.5	33.3
		Yes, just once	22.2	14.3	16.7	18.2	22.2
		Yes, more than once	22.2	28.6	16.7	27.3	44.4
Multi-risk	High parental knowledge	No	62.5	55.6	70.0	63.6	50.0
		Yes, just once	18.8	27.8	10.0	18.2	10.0
		Yes, more than once	18.8	16.7	20.0	18.2	40.0
Multi-risk	Average protection	No	53.2	60.5	37.5	44.7	46.4
		Yes, just once	29.8	14.0	32.5	31.6	25.0
		Yes, more than once	17.0	25.6	30.0	23.7	28.6

(*continued*)

Multi-risk	Low protection	No	48.0	60.9	52.6	75.0	47.1
		Yes, just once	28.0	13.0	31.6	12.5	29.4
		Yes, more than once	24.0	26.1	15.8	12.5	23.5

Table C.10

Proportion of Individuals Likely to Demonstrate Conditional Risk and Protective Factor Patterns Endorsing Cigarette Use

Risk Pattern	Protective Pattern	Use	Age 13	Age 14	Age 15	Age 16	Age 17
Low risk	High parental knowledge	No	85.6	82.4	83.3	80.8	78.9
		Yes, just once	11.0	11.0	10.3	10.7	9.2
		Yes, more than once	3.4	6.6	6.5	8.5	11.9
Low risk	Average protection	No	83.6	82.9	87.7	75.7	76.1
		Yes, just once	12.4	13.5	8.2	14.9	9.4
		Yes, more than once	4.0	3.5	4.1	9.5	14.5
Low risk	Low protection	No	88.4	64.3	80.6	88.2	86.2
		Yes, just once	7.0	26.2	13.9	8.8	3.4
		Yes, more than once	4.7	9.5	5.6	2.9	10.3
Average risk	High parental knowledge	No	79.1	80.6	80.0	71.9	59.3
		Yes, just once	17.6	11.8	10.7	17.2	16.9
		Yes, more than once	3.3	7.5	9.3	10.9	23.7
Average risk	Average protection	No	80.0	78.0	76.6	72.4	64.2
		Yes, just once	13.3	15.9	10.4	11.8	13.2
		Yes, more than once	6.7	6.1	13.0	15.8	22.6
Average risk	Low protection	No	66.7	94.4	70.6	64.3	73.3
		Yes, just once	16.7	0	5.9	21.4	6.7
		Yes, more than once	16.7	5.6	23.5	14.3	20.0

(*continued*)

Some delinquency	High parental knowledge	No	78.8	83.0	72.0	63.3	72.7
		Yes, just once	13.5	9.4	12.0	14.3	18.2
		Yes, more than once	7.7	7.5	16.0	22.4	9.1
Some delinquency	Average protection	No	73.3	80.0	71.7	58.3	64.5
		Yes, just once	20.0	12.0	17.0	16.7	9.7
		Yes, more than once	6.7	8.0	11.3	25.0	25.8
Some delinquency	Low protection	No	93.3	75.0	72.2	40.0	60.0
		Yes, just once	0	15.0	16.7	20.0	20.0
		Yes, more than once	6.7	10.0	11.1	40.0	20.0
Elevated delinquency	High parental knowledge	No	82.4	75.6	62.9	59.1	77.4
		Yes, just once	14.7	9.8	25.7	13.6	6.5
		Yes, more than once	2.9	14.6	11.4	27.3	16.1
Elevated delinquency	Average protection	No	76.1	78.8	77.3	68.8	57.5
		Yes, just once	17.9	13.5	13.6	12.5	22.5
		Yes, more than once	6.0	7.7	9.1	18.8	20.0
Elevated delinquency	Low protection	No	57.1	71.4	81.3	81.8	57.1
		Yes, just once	23.8	4.8	12.5	9.1	28.6
		Yes, more than once	19.0	23.8	6.3	9.1	14.3
Delinquency & violence exposure	High parental knowledge	No	76.7	67.6	68.0	66.7	52.4
		Yes, just once	16.7	26.5	20.0	12.5	23.8
		Yes, more than once	6.7	5.9	12.0	20.8	23.8
Delinquency & violence exposure	Average protection	No	66.7	73.9	71.7	65.9	58.6
		Yes, just once	21.6	13.0	10.9	12.2	6.9
		Yes, more than once	11.8	13.0	17.4	22.0	34.5
Delinquency & violence exposure	Low protection	No	38.9	50.0	69.2	45.5	57.1
		Yes, just once	33.3	7.1	7.7	27.3	14.3
		Yes, more than once	27.8	42.9	23.1	27.3	28.6
Multi-risk	High parental knowledge	No	68.8	72.2	70.0	81.8	70.0
		Yes, just once	12.5	16.7	0	18.2	10.0

(*continued*)

		Yes, more than once	18.8	11.1	30.0	0	20.0
Multi-risk	Average protection	No	71.7	71.4	65.0	65.8	67.9
		Yes, just once	19.6	14.3	20.0	18.4	14.3
		Yes, more than once	8.7	14.3	15.0	15.8	17.9
Multi-risk	Low protection	No	70.8	56.5	63.2	81.3	52.9
		Yes, just once	25.0	34.8	31.6	0	29.4
		Yes, more than once	4.2	8.7	5.3	18.8	17.6

Table C.11

Proportion of Individuals Likely to Demonstrate Conditional Risk and Protective Factor Patterns Endorsing Marijuana Use

Risk Pattern	Protective Pattern	Use	Age 13	Age 14	Age 15	Age 16	Age 17
Low risk	High parental knowledge	No	87.9	78.1	76.1	69.2	70.1
		Yes, just once	8.6	11.9	11.4	12.5	8.7
		Yes, more than once	3.4	10.0	12.5	18.3	21.2
Low risk	Average protection	No	88.8	77.9	74.0	64.9	60.0
		Yes, just once	5.6	13.4	10.3	12.2	17.5
		Yes, more than once	5.6	8.7	15.8	23.0	22.5
Low risk	Low protection	No	93.0	78.0	63.9	73.5	65.5
		Yes, just once	2.3	12.2	16.7	17.6	13.8
		Yes, more than once	4.7	9.8	19.4	8.8	20.7
Average risk	High parental knowledge	No	81.3	79.8	64.0	57.8	49.2
		Yes, just once	12.1	7.4	18.7	15.6	16.9
		Yes, more than once	6.6	12.8	17.3	26.6	33.9
Average risk	Average protection	No	76.7	71.1	63.6	69.7	50.9
		Yes, just once	13.3	16.9	15.6	13.2	12.7

(*continued*)

		Yes, more than once	10.0	12.0	20.8	17.1	36.4
Average risk	Low protection	No	78.9	68.4	58.8	71.4	78.6
		Yes, just once	15.8	5.3	23.5	7.1	0
		Yes, more than once	5.3	26.3	17.6	21.4	21.4
Some delinquency	High parental knowledge	No	88.2	64.2	74.0	50.0	44.1
		Yes, just once	5.9	17.0	8.0	20.8	20.6
		Yes, more than once	5.9	18.9	18.0	29.2	35.3
Some delinquency	Average protection	No	75.4	64.0	56.6	38.9	38.7
		Yes, just once	18.0	18.0	20.8	27.8	16.1
		Yes, more than once	6.6	18.0	22.6	33.3	45.2
Some delinquency	Low protection	No	93.3	75.0	66.7	50.0	50.0
		Yes, just once	0	10.0	11.1	30.0	30.0
		Yes, more than once	6.7	15.0	22.2	20.0	20.0
Elevated delinquency	High parental knowledge	No	85.7	58.5	62.2	31.8	58.1
		Yes, just once	5.7	12.2	13.5	31.8	6.5
		Yes, more than once	8.6	29.3	24.3	36.4	35.5
Elevated delinquency	Average protection	No	86.6	73.1	65.9	58.3	61.9
		Yes, just once	3.0	9.6	13.6	20.8	11.9
		Yes, more than once	10.4	17.3	20.5	20.8	26.2
Elevated delinquency	Low protection	No	71.4	60.0	43.8	58.3	50.0
		Yes, just once	9.5	20.0	12.5	25.0	33.3
		Yes, more than once	19.0	20.0	43.8	16.7	16.7
Delinquency & violence exposure	High parental knowledge	No	63.3	66.7	46.2	66.7	52.4
		Yes, just once	16.7	9.1	30.8	0	9.5
		Yes, more than once	20.0	24.2	23.1	33.3	38.1
Delinquency & violence exposure	Average protection	No	58.8	58.7	54.3	56.1	30.0
		Yes, just once	21.6	17.4	13.0	19.5	26.7
		Yes, more than once	19.6	23.9	32.6	24.4	43.3
Delinquency & violence exposure	Low protection	No	64.7	61.5	66.7	63.6	22.2

(*continued*)

		Yes, just once	11.8	15.4	16.7	18.2	22.2
		Yes, more than once	23.5	23.1	16.7	18.2	55.6
Multi-risk	High parental knowledge	No	62.5	50.0	40.0	72.7	50.0
		Yes, just once	25.0	22.2	20.0	18.2	20.0
		Yes, more than once	12.5	27.8	40.0	9.1	30.0
Multi-risk	Average protection	No	72.3	58.1	57.5	55.3	53.6
		Yes, just once	12.8	16.3	10.0	15.8	21.4
		Yes, more than once	14.9	25.6	32.5	28.9	25.0
Multi-risk	Low protection	No	44.0	69.6	55.0	62.5	41.2
		Yes, just once	32.0	17.4	20.0	12.5	29.4
		Yes, more than once	24.0	13.0	25.0	25.0	29.4

Table C.12

Proportion of Individuals Likely to Demonstrate Etiological Factor Patterns Endorsing Alcohol Use

Etiological Factor Pattern	Use	Age 13	Age 14	Age 15	Age 16	Age 17
Some delinquency/average protection	No	64.1	62.6	55.4	44.2	44.7
	Yes, just once	22.7	21.1	25.6	34.2	25.0
	Yes, more than once	13.3	16.3	19.0	21.1	30.3
Low risk/average protection	No	69.8	64.0	58.4	55.0	55.8
	Yes, just once	20.8	24.4	27.9	26.7	25.1
	Yes, more than once	9.3	11.6	13.7	18.3	19.1
Average risk and protection	No	63.0	63.6	47.6	57.8	45.0
	Yes, just once	24.5	22.1	30.4	23.4	27.1
	Yes, more than once	12.5	14.4	22.0	18.8	27.9
Elevated risk/decreased protection	No	63.1	60.2	56.7	49.4	52.4
	Yes, just once	18.9	25.7	23.7	30.9	20.7
	Yes, more than once	18.0	14.2	19.6	19.8	26.8
High risk/low protection	No	53.4	60.2	46.4	55.4	48.1
	Yes, just once	27.3	16.9	29.0	24.6	24.1
	Yes, more than once	19.3	22.9	24.6	20.0	27.8
Moderate risk/decreased protection	No	54.6	52.1	50.6	48.0	42.4
	Yes, just once	24.7	23.4	27.7	28.0	27.1
	Yes, more than once	20.6	24.5	21.7	24.0	30.5

Table C.13

Proportion of Individuals Likely to Demonstrate Etiological Factor Patterns Endorsing Cigarette Use

Etiological Factor Pattern	Use	Age 13	Age 14	Age 15	Age 16	Age 17
Some delinquency/average protection	No	78.0	80.5	71.9	58.9	67.6
	Yes, just once	15.0	11.4	14.9	15.8	14.9
	Yes, more than once	7.1	8.1	13.2	25.3	17.6
Low risk/average protection	No	85.2	81.0	84.5	79.6	78.5
	Yes, just once	11.1	13.2	9.9	12.1	8.8
	Yes, more than once	3.7	5.8	5.6	8.4	12.7
Average risk and protection	No	78.4	80.8	77.5	71.4	63.0
	Yes, just once	15.6	12.4	10.1	14.9	14.2
	Yes, more than once	6.0	6.7	12.4	13.6	22.8
Elevated risk/decreased protection	No	74.0	76.3	72.9	67.9	65.8
	Yes, just once	18.7	11.4	17.7	12.3	16.5
	Yes, more than once	7.3	12.3	9.4	19.8	17.7
High risk/low protection	No	70.9	67.1	65.2	72.3	63.0
	Yes, just once	19.8	20.7	20.3	13.8	18.5
	Yes, more than once	9.3	12.2	14.5	13.8	18.5
Moderate risk/decreased protection	No	65.3	68.4	69.9	63.2	56.1
	Yes, just once	21.4	15.8	13.3	14.5	14.0
	Yes, more than once	13.3	15.8	16.9	22.4	29.8

Table C.14

Proportion of Individuals Likely to Demonstrate Etiological Factor Patterns Endorsing Marijuana Use

Etiological Factor Pattern	Use	Age 13	Age 14	Age 15	Age 16	Age 17
Some delinquency/average protection	No	82.7	65.9	65.3	45.7	42.7
	Yes, just once	11.0	16.3	14.0	24.5	20.0
	Yes, more than once	6.3	17.9	20.7	29.8	37.3
Low risk/average protection	No	88.6	78.1	74.4	68.0	66.1
	Yes, just once	7.0	12.4	11.4	12.8	12.3
	Yes, more than once	4.3	9.5	14.1	19.2	21.6
Average risk and protection	No	79.0	75.0	63.3	64.9	53.1
	Yes, just once	13.0	11.2	17.8	13.6	13.3
	Yes, more than once	8.0	13.8	18.9	21.4	33.6
Elevated risk/decreased protection	No	83.1	65.5	61.2	51.2	60.0
	Yes, just once	5.6	12.4	13.3	24.4	11.3
	Yes, more than once	11.3	22.1	25.5	24.4	28.7
High risk/low protection	No	62.5	59.0	54.3	60.0	50.0
	Yes, just once	20.5	18.1	14.3	15.4	22.2
	Yes, more than once	17.0	22.9	31.4	24.6	27.8
Moderate risk/decreased protection	No	61.9	62.4	53.0	60.5	35.0
	Yes, just once	17.5	14.0	19.3	13.2	21.7
	Yes, more than once	20.6	23.7	27.7	26.3	43.3

REFERENCES

Abbey, A., Jacques, A. J., Hayman, L. W., & Sobeck, J. (2006). Predictors of early substance use among African American and caucasian youth from urban and suburban communities. *Merrill-Palmer Quarterly*, *52*(2), 305–326.

Abenavoli, R. M., Greenberg, M. T., & Bierman, K. L. (2017). Identification and validation of school readiness profiles among high-risk kindergartners. *Early Childhood Research Quarterly*, *38*, 33–43. doi: 10.1016/j.ecresq.2016.09.001

Acevedo-Garcia, D., Osypuk, T. L., McArdle, N., & Williams, D. R. (2008). Toward a policy-relevant analysis of geographic and racial/ethnic disparities in child health. *Health Affairs*, *27*(2), 321–333. doi: 10.1377/hlthaff.27.2.321

Agnew, R. (1992). Foundation for a general strain theory of crime and delinquency. *Criminology*, *30*(1), 47–88. doi: 10.1111/j.1745-9125.1992.tb01093.x

Agnew, R. (2001). Building on the foundation of general strain theory: Secifying the types of strain most likely to lead to crime and delinquency. *Journal of Research in Crime and Delinquency*, *38*(4), 319–361. doi: 10.1177/0022427801038004001

Agnew, R. (2006). *Pressured Into Crime: An Overview of General Strain Theory*. Oxford University Press USA.

Agrawal, A., Grant, J. D., Haber, J. R., Madden, P. A. F., Heath, A. C., Bucholz, K. K., & Sartor, C. E. (2017). Differences between White and Black young women in the relationship between religious service attendance and alcohol involvement. *The American Journal on Addictions*, *26*(5), 437–445. doi: 10.1111/ajad.12462

American Psychiatric Association. (2013). *Diagnostic and Statistical Manual of Mental Disorders* (Fifth ed.). American Psychiatric Pub.

Angold, A., Erkanli, A., Farmer, E. M. Z., Fairbank, J. A., Burns, B. J., Keeler, G., & Costello, E. J. (2002). Psychiatric disorder, impairment, and service use in rural African American and white youth. *Archives of General Psychiatry*, *59*(10), 893–901. doi: 10.1001/archpsyc.59.10.893

Asparouhov, T., & Muthén, B. O. (2010). *Weighted least squares estimation with missing data* (Tech. Rep.). Los Angeles, CA: Muthén & Muthén.

Bandura, A. (2001). Social cognitive theory: An agentic perspective. *Annual Review of Psychology*, *52*(1), 1–26. doi: 10.1146/annurev.psych.52.1.1

Bandura, A., & Walters, R. H. (1977). *Social Learning Theory* (Vol. 1). Englewood Cliffs, NJ: Prentice-Hall.

Banton, M. (1998). *Racial Theories*. Cambridge University Press. (Google-Books-ID: xnokRYqmJqgC)

Bem, D. J. (1972). Self-perception theory. In L. Berkowitz (Ed.), *Advances in Experimental Social Psychology* (Vol. 6, pp. 1–62). Academic Press. doi: 10.1016/S0065-2601(08)60024-6

Bergman, L. R., Magnusson, D., & Khouri, B. M. E. (2003). *Studying Individual Development in An Interindividual Context: A Person-oriented Approach*. Mahwah, N.J.: Psychology Press. (Google-Books-ID: 3dp4AgAAQBAJ)

Bierman, A. (2006). Does religion buffer the effects of discrimination on mental health? Differing effects by race. *Journal for the Scientific Study of Religion*, *45*(4), 551–565.

Blustein, E. C., Munn-Chernoff, M. A., Grant, J. D., Sartor, C. E., Waldron, M., Bucholz, K. K., … Heath, A. C. (2015). The association of low parental monitoring with early substance use in European American and African American adolescent girls. *Journal of Studies on Alcohol and Drugs*, *76*(6), 852–861.

Boardman, J. D., Finch, B. K., Ellison, C. G., Williams, D. R., & Jackson, J. S. (2001). Neighborhood disadvantage, stress, and drug use among adults. *Journal of Health and Social Behavior*, *42*(2), 151–165.

Bock, R. D. (1997). A brief history of item response theory. *Educational Measurement: Issues and Practice*, *12*, 21–33.

Boden, J. M., Fergusson, D. M., & Horwood, L. J. (2008). Does adolescent self-esteem predict later life outcomes? A test of the causal role of self-esteem. *Development and Psychopathology*, *20*(1), 319–339. doi: 10.1017/S0954579408000151

Bogat, G. A., Eye, A. v., & Bergman, L. R. (2016). Person-Oriented Approaches. In *Developmental Psychopathology* (pp. 1–49). American Cancer Society. doi: 10.1002/9781119125556.devpsy118

Bohnert, K. M., Anthony, J. C., & Breslau, N. (2012). Parental monitoring at age 11 and subsequent onset of cannabis use up to age 17: Results from a prospective study. *Journal of Studies on Alcohol and Drugs*, *73*(2), 173–177.

Bolland, A. C. (2012). *Representativeness two ways: An assessment of representativeness and missing data mechanisms in a study of an at-risk population* (Unpublished doctoral dissertation). The University of Alabama Tuscaloosa.

Bolland, J. M. (2007). *Overview of the Mobile Youth Survey* (Technical Report). Birmingham, AL: University of Alabama at Birmingham, School of Public Health.

Bolland, K. A., Bolland, J. M., Tomek, S., Devereaux, R. S., Mrug, S., & Wimberly, J. C. (2016). Trajectories of adolescent alcohol use by gender and early initiation status. *Youth & Society*, *48*(1), 3–32. doi: 10.1177/0044118X13475639

Borrell, L. N., Diez Roux, A. V., Jacobs, D. R., Shea, S., Jackson, S. A., Shrager, S., & Blumenthal, R. S. (2010). Perceived racial/ethnic discrimination, smoking and alcohol consumption in the Multi-Ethnic Study of Atherosclerosis (MESA). *Preventive Medicine*, *51*(3-4), 307–312. doi: 10.1016/j.ypmed.2010.05.017

Brenner, A. B., Diez Roux, A. V., Barrientos-Gutierrez, T., & Borrell, L. N. (2015). Associations of alcohol availability and neighborhood socioeconomic characteristics with drinking: Cross-sectional results from the Multi-Ethnic Study of Atherosclerosis (MESA). *Substance Use & Misuse*, *50*(12), 1606–1617. doi: 10.3109/10826084.2015.1027927

Broman, C. L. (2005). Stress, race and substance use in college. *College Student Journal*, *39*(2), 340.

Brook, J. S., Lee, J. Y., Finch, S. J., Brown, E. N., & Brook, D. W. (2013). Long-term consequences of membership in trajectory groups of delinquent behavior in an urban sample: violence, drug use, interpersonal, and neighborhood attributes. *Aggressive Behavior*, *39*(6), 440–452. doi: 10.1002/ab.21493

Brook, J. S., Lee, J. Y., Rubenstone, E., Finch, S. J., Seltzer, N., & Brook, D. W. (2013). Longitudinal determinants of substance use disorders. *Journal of Urban Health*, *90*(6), 1130–1150. doi: 10.1007/s11524-013-9827-6

Brooks, F. M., Magnusson, J., Spencer, N., & Morgan, A. (2012). Adolescent multiple risk behaviour: An asset approach to the role of family, school and community. *Journal of Public Health (Oxford, England)*, *34 Suppl 1*, i48–56. doi: 10.1093/pubmed/fds001

Brown, J. D. (2010). High self-esteem buffers negative feedback: Once more with feeling. *Cognition and Emotion*, *24*(8), 1389–1404. doi: 10.1080/02699930903504405

Brown, T. A. (2014). *Confirmatory Factor Analysis for Applied Research, Second Edition*. New York, NY: Guilford Publications. (Google-Books-ID: JDb3BQAAQBAJ)

Brown, T. L., Flory, K., Lynam, D. R., Leukefeld, C., & Clayton, R. R. (2004). Comparing the developmental trajectories of marijuana use of African American and caucasian adolescents: patterns, antecedents, and consequences. *Experimental and Clinical Psychopharmacology*, *12*(1), 47–56. doi: 10.1037/1064-1297.12.1.47

Bujarski, S. J., Feldner, M. T., Lewis, S. F., Babson, K. A., Trainor, C. D., Leen-Feldner, E., ... Bonn-Miller, M. O. (2012). Marijuana use among traumatic event-exposed adolescents: Posttraumatic stress symptom frequency predicts coping motivations for use. *Addictive Behaviors*, *37*(1), 53–59. doi: 10.1016/j.addbeh.2011.08.009

Bursik, R. J. (1984). Ecological theories of crime and delinquency since Shaw and McKay. In *Proceedings of the annual meeting of the American Society of Criminology.* Cincinnati.

Caetano, R., Clark, C. L., & Tam, T. (1998). Alcohol consumption among racial/ethnic minorities: Theory and research. *Alcohol Health & Research World*, *22*(4), 233–242.

Casement, M. D., Shaw, D. S., Sitnick, S. L., Musselman, S. C., & Forbes, E. E. (2015). Life stress in adolescence predicts early adult reward-related brain function and alcohol dependence. *Social Cognitive and Affective Neuroscience*, *10*(3), 416–423. doi: 10.1093/scan/nsu061

Catalano, R. F., & Hawkins, J. D. (1996). The social development model: A theory of antisocial behavior. In J. D. Hawkins (Ed.), *Delinquency and Crime: Current Theories* (pp. 149–197). Cambridge: Cambridge University Press.

Catalano, R. F., Kosterman, R., Hawkins, J. D., Newcomb, M. D., & Abbott, R. D. (1996). Modeling the etiology of adolescent substance use: A test of the social development model. *Journal of Drug Issues*, *26*(2), 429–455. doi: 10.1177/002204269602600207

Catalano, R. F., Morrison, D. M., Wells, E. A., Gillmore, M. R., Iritani, B., & Hawkins, J. D. (1992). Ethnic differences in family factors related to early drug initiation. *Journal of Studies on Alcohol*, *53*(3), 208–217. doi: 10.15288/jsa.1992.53.208

Celeux, G., & Soromenho, G. (1996). An entropy criterion for assessing the number of clusters in a mixture model. *Journal of Classification*, *13*(2), 195–212.

Cheadle, J. E., & Whitbeck, L. B. (2011). Alcohol use trajectories and problem drinking over the course of adolescence: A study of North American Indigenous youth and their caretakers. *Journal of Health and Social Behavior*, *52*(2), 228–245.

Chen, P., & Jacobson, K. C. (2012). Developmental trajectories of substance use from early adolescence to young adulthood: Gender and racial/ethnic differences. *Journal of Adolescent Health*, *50*(2), 154–163. doi: 10.1016/j.jadohealth.2011.05.013

Chen, P., Voisin, D. R., & Jacobson, K. C. (2016). Community violence exposure and adolescent delinquency: Examining a spectrum of promotive factors. *Youth & Society*, *48*(1), 33–57. doi: 10.1177/0044118X13475827

Christensen, D., Taylor, C. L., & Zubrick, S. R. (2017). Patterns of multiple risk exposures for low receptive vocabulary growth 4-8 years in the longitudinal study of Australian children. *PLOS ONE*, *12*(1), e0168804. doi: 10.1371/journal.pone.0168804

Church, W. T., Jaggers, J. W., Tomek, S., Bolland, A. C., Bolland, K. A., Hooper, L. M., & Bolland, J. M. (2015). Does permissive parenting relate to levels of delinquency? An examination of family management practices in low-income black American families. *Journal of Juvenile Justice*, *4*(2), 95–110.

Church, W. T., Tomek, S., Bolland, K. A., Hooper, L. M., Jaggers, J., & Bolland, J. M. (2012). A longitudinal examination of predictors of delinquency: An analysis of data from the Mobile Youth Survey. *Children and Youth Services Review*, *34*(12), 2400–2408. doi: 10.1016/j.childyouth.2012.09.007

Clark, H. K., Shamblen, S. R., Ringwalt, C. L., & Hanley, S. (2012). Predicting high risk adolescents' substance use over time: The role of parental monitoring. *The Journal of Primary Prevention*, *33*(2-3), 67–77. doi: 10.1007/s10935-012-0266-z

Clark, S. L., Muthén, B., Kaprio, J., D'Onofrio, B. M., Viken, R., & Rose, R. J. (2013). Models and strategies for factor mixture analysis: An example concerning the structure underlying psychological disorders. *Structural Equation Modeling*, *20*(4), 681–703. doi: 10.1080/10705511.2013.824786

Clark, T. T., Belgrave, F. Z., & Nasim, A. (2008). Risk and protective factors for substance use among urban African American adolescents considered high-risk. *Journal of Ethnicity in Substance Abuse*, *7*(3), 292–303. doi: 10.1080/15332640802313296

Clark, T. T., Nguyen, A. B., & Belgrave, F. Z. (2011). Risk and protective factors for alcohol and marijuana use among African-American rural and urban adolescents. *Journal of Child & Adolescent Substance Abuse*, *20*(3), 205–220. doi: 10.1080/1067828X.2011.581898

Clasen, D. R., & Brown, B. B. (1985). The multidimensionality of peer pressure in adolescence. *Journal of Youth and Adolescence*, *14*(6), 451–468. doi: 10.1007/BF02139520

Cleveland, M. J., Feinberg, M. E., Bontempo, D. E., & Greenberg, M. T. (2008). The role of risk and protective factors in substance use across adolescence. *The Journal of Adolescent Health*, *43*(2), 157–164. doi: 10.1016/j.jadohealth.2008.01.015

Colder, C. R., Campbell, R. T., Ruel, E., Richardson, J. L., & Flay, B. R. (2002). A finite mixture model of growth trajectories of adolescent alcohol use: Predictors and consequences. *Journal of Consulting and Clinical Psychology*, *70*(4), 976–985. doi: 10.1037/0022-006X.70.4.976

Collins, L. M., & Lanza, S. T. (2010). *Latent Class and Latent Transition Analysis: With Applications in the Social, Behavioral, and Health Sciences*. New York: Wiley.

Conley, D. J. (1994). Adding color to a black and white picture: Using qualitative data to explain racial disproportionality in the juvenile justice system. *Journal of Research in Crime and Delinquency*, *31*(2), 135–148. doi: 10.1177/0022427894031002003

Copeland-Linder, N., Lambert, S. F., Chen, Y.-F., & Ialongo, N. S. (2011). Contextual stress and health risk behaviors among African American adolescents. *Journal of Youth and Adolescence*, *40*(2), 158–173. doi: 10.1007/s10964-010-9520-y

Corp., I. (2017). *IBM SPSS Statistics for Macintosh, Version 25.0.* Armonk, NY.

Costello, E. J., He, J.-p., Sampson, N. A., Kessler, R. C., & Merikangas, K. R. (2014). Services for adolescent psychiatric disorders: 12-month data from the National Comorbidity Survey-Adolescent. *Psychiatric Services*, *65*(3), 359–366. doi: 10.1176/appi.ps.201100518

Cummings, J. R., Wen, H., & Druss, B. G. (2011). Racial/ethnic differences in treatment for substance use disorders among U.S. adolescents. *Journal of the American Academy of Child & Adolescent Psychiatry*, *50*(12), 1265–1274. doi: 10.1016/j.jaac.2011.09.006

Curran, P. J., Obeidat, K., & Losardo, D. (2010). Twelve frequently asked questions about growth curve modeling. *Journal of cognition and development: Official journal of the Cognitive Development Society*, *11*(2), 121–136. doi: 10.1080/15248371003699969

Dennis, M. L., Titus, J. C., White, M. K., Unsicker, J. I., & Hodgkins, D. (1998). *Global Appraisal of Individual Needs (GAIN): Administration guide for the GAIN and related measures*. Bloomington, IL: Chestnut Health Systems.

Desmond, S. A., Ulmer, J. T., & Bader, C. D. (2013). Religion, self-control, and substance use. *Deviant Behavior*, *34*(5), 384–406. doi: 10.1080/01639625.2012.726170

DeWall, C. N., Pond, R. S., Carter, E. C., McCullough, M. E., Lambert, N. M., Fincham, F. D., & Nezlek, J. B. (2014). Explaining the relationship between religiousness and substance use: Self-control matters. *Journal of Personality and Social Psychology*, *107*(2), 339–351. (WOS:000340466700008) doi: 10.1037/a0036853

Dixon, L. J., Leen-Feldner, E. W., Ham, L. S., Feldner, M. T., & Lewis, S. F. (2009). Alcohol use motives among traumatic event-exposed, treatment-seeking adolescents: associations with posttraumatic stress. *Addictive Behaviors*, *34*(12), 1065–1068. doi: 10.1016/j.addbeh.2009.06.008

Dobransky-Fasiska, D., Brown, C., Pincus, H. A., Nowalk, M. P., Wieland, M., Parker, L. S., ... Reynolds, C. F. (2009). Developing a community-academic partnership to improve recognition and treatment of depression in underserved African American and white elders. *The American Journal of Geriatric Psychiatry*, *17*(11), 953–964. doi: 10.1097/JGP.0b013e31818f3a7e

Donovan, J. E., & Jessor, R. (1985). Structure of problem behavior in adolescence and young adulthood. *Journal of Consulting and Clinical Psychology*, *53*(6), 890–904.

Downing, J., & Bellis, M. A. (2009). Early pubertal onset and its relationship with sexual risk taking, substance use and anti-social behaviour: A preliminary cross-sectional study. *BMC Public Health*, *9*, 446. doi: 10.1186/1471-2458-9-446

Duncan, S. C., Duncan, T. E., & Strycker, L. A. (2002). A multilevel analysis of neighborhood context and youth alcohol and drug problems. *Prevention Science*, *3*(2), 125–133.

D'Amico, E. J., Edelen, M. O., Miles, J. N. V., & Morral, A. R. (2008). The longitudinal association between substance use and delinquency among high-risk youth. *Drug and Alcohol Dependence*, *93*(1–2), 85–92. doi: 10.1016/j.drugalcdep.2007.09.006

Edlund, M. J., Harris, K. M., Koenig, H. G., Han, X., Sullivan, G., Mattox, R., & Tang, L. (2010). Religiosity and decreased risk of substance use disorders: Is the effect mediated by social support or mental health status? *Social Psychiatry and Psychiatric Epidemiology*, *45*(8), 827–836. (WOS:000279775400008) doi: 10.1007/s00127-009-0124-3

Ehlers, C. L., Carr, L., Betancourt, M., & Montane-Jaime, K. (2003). Association of the ADH2*3 allele with greater alcohol expectancies in African-American young adults. *Journal of Studies on Alcohol*, *64*(2), 176–181.

Elkington, K. S., Bauermeister, J. A., & Zimmerman, M. A. (2010). Psychological distress, substance use, and HIV/STI risk behaviors among youth. *Journal of Youth and Adolescence*, *39*(5), 514–527. doi: 10.1007/s10964-010-9524-7

Elliott, D. S., Huizinga, D., & Menard, S. (1989). *Multiple Problem Youth: Delinquency, Substance Use, and Mental Health Problems*. New York: Springer-Verlag.

Ellison, C. G., Musick, M. A., & Henderson, A. K. (2008). Balm in Gilead: Racism, religious involvement, and psychological distress among African-American adults. *Journal for the Scientific Study of Religion*, *47*(2), 291–309.

Fagan, A. A., Wright, E. M., & Pinchevsky, G. M. (2014). The protective effects of neighborhood collective efficacy on adolescent substance use and violence following exposure to violence. *Journal of Youth and Adolescence*, *43*(9), 1498–1512. doi: 10.1007/s10964-013-0049-8

Fagan, A. A., Wright, E. M., & Pinchevsky, G. M. (2015). Exposure to violence, substance use, and neighborhood context. *Social Science Research*, *49*, 314–326. doi: 10.1016/j.ssresearch.2014.08.015

Farmer, A. Y., & Brown, K. M. (2013). Parental religious service attendance and adolescent substance use. *Journal of Religion & Spirituality in Social Work: Social Thought*, *32*(1), 84–101. doi: 10.1080/15426432.2013.749135

Feagin, J. (2013). *Systemic Racism : A Theory of Oppression*. Routledge. doi: 10.4324/9781315880938

Festinger, L. (1964). *Conflict, Decision, and Dissonance*. Palo Alto, CA, US: Stanford U. Press.

Fisher, R. J. (1993). Social desirability bias and the validity of indirect questioning. *Journal of Consumer Research*, *20*(2), 303–315.

Flores, E., Tschann, J. M., Dimas, J. M., Pasch, L. A., & de Groat, C. L. (2010). Perceived racial/ethnic discrimination, posttraumatic stress symptoms, and health risk behaviors among Mexican American adolescents. *Journal of Counseling Psychology*, *57*(3), 264–273. doi: 10.1037/a0020026

Fowler, P. J., Ahmed, S. R., Tompsett, C. J., Jozefowicz-Simbeni, D. M. H., & Toro, P. A. (2008). Community violence and externalizing problems: Moderating effects of race and religiosity in emerging adulthood. *Journal of Community Psychology*, *36*(7), 835–850. doi: 10.1002/jcop.20267

Garner, B. R. (2009). Research on the diffusion of evidence-based treatments within substance abuse treatment: A systematic review. *Journal of Substance Abuse Treatment*, *36*(4), 376–399. doi: 10.1016/j.jsat.2008.08.004

Gerson, R., & Rappaport, N. (2013). Traumatic stress and posttraumatic stress disorder in youth: Recent research findings on clinical impact, assessment, and treatment. *The Journal of Adolescent Health*, *52*(2), 137–143. doi: 10.1016/j.jadohealth.2012.06.018

Geyer, S., Roux, L. l., & Hall, H. (2015). Exposure to substance use in the social environment: The experiences of adolescents in the Tshwane Metropole. *Southern African Journal of Social Work and Social Development*, *27*(3), 322–343. doi: 10.25159/2415-5829/722

Gibbons, F. X., Gerrard, M., Cleveland, M. J., Wills, T. A., & Brody, G. (2004). Perceived discrimination and substance use in African American parents and their children: A panel study. *Journal of Personality and Social Psychology*, *86*(4), 517–529. doi: 10.1037/0022-3514.86.4.517

Gil, A. G., Vega, W. A., & Turner, R. J. (2002). Early and mid-adolescence risk factors for later substance abuse by African Americans and European Americans. *Public Health Reports*, *117*, S15–S29.

Glantz, M. D. (1992). A developmental psychopathology models of drug abuse vulnerability. In M. D. Glantz & R. W. Pickens (Eds.), (pp. 389–418). Washington, DC: American Psychological Association.

Glynn, T. J. (1981). Psychological sense of community: Measurement and application. *Human Relations*, *34*(9), 789–818. doi: 10.1177/001872678103400904

Goldstein, A. L., Wekerle, C., Tonmyr, L., Thornton, T., Waechter, R., Pereira, J., … Team, M. R. (2011). The relationship between post-traumatic stress symptoms and substance use among adolescents involved with child welfare: Implications for emerging adulthood. *International Journal of Mental Health and Addiction*, *9*(5), 507–524. doi: 10.1007/s11469-011-9331-8

Goldstein, S. E., Davis-Kean, P. E., & Eccles, J. S. (2005). Parents, peers, and problem behavior: A longitudinal investigation of the impact of relationship perceptions and characteristics on the development of adolescent problem behavior. *Developmental Psychology*, *41*(2), 401–413. doi: 10.1037/0012-1649.41.2.401

Gottfredson, M. R., & Hirschi, T. (1990). *A General Theory of Crime*. Stanford, Calif: Stanford University Press.

Grant, B. F., & Dawson, D. A. (1998). Age of onset of drug use and its association with DSM-IV drug abuse and dependence: Results from the National Longitudinal Alcohol Epidemiologic Survey. *Journal of Substance Abuse*, *10*(2), 163–173. doi: 10.1016/S0899-3289(99)80131-X

Gutman, L. M., Eccles, J. S., Peck, S., & Malanchuk, O. (2011). The influence of family relations on trajectories of cigarette and alcohol use from early to late adolescence. *Journal of Adolescence*, *34*(1), 119–128. doi: 10.1016/j.adolescence.2010.01.005

Handley, E. D., Rogosch, F. A., Guild, D. J., & Cicchetti, D. (2015). Neighborhood disadvantage and adolescent substance use disorder: The moderating role of maltreatment. *Child Maltreatment*, *20*(3), 193–202. doi: 10.1177/1077559515584159

Harter, S. (1982). The Perceived Competence Scale for Children. *Child Development*, *53*(1), 87–97. doi: 10.2307/1129640

Hawkins, J. D., Catalano, R. F., & Arthur, M. W. (2002). Promoting science-based prevention in communities. *Addictive Behaviors*, *27*(6), 951–976.

Hawkins, J. D., Catalano, R. F., & Miller, J. Y. (1992). Risk and protective factors for alcohol and other drug problems in adolescence and early adulthood: Implications for substance abuse prevention. *Psychological Bulletin*, *112*(1), 64–105. doi: 10.1037/0033-2909.112.1.64

Hebl, M. R., Foster, J. B., Mannix, L. M., & Dovidio, J. F. (2002). Formal and interpersonal discrimination: A field study of bias toward homosexual applicants. *Personality and Social Psychology Bulletin*, *28*(6), 815–825. doi: 10.1177/0146167202289010

Herd, D. (1994). Predicting drinking problems among black and white men: Results from a national survey. *Journal of Studies on Alcohol*, *55*(1), 61–71. doi: 10.15288/jsa.1994.55.61

Hill, G. D., & Atkinson, M. P. (1988). Gender, familial control, and delinquency. *Criminology*, *26*(1), 127–147. doi: 10.1111/j.1745-9125.1988.tb00835.x

Hill, T. D., Ross, C. E., & Angel, R. J. (2005). Neighborhood disorder, psychophysiological distress, and health. *Journal of Health and Social Behavior*, *46*(2), 170–186. doi: 10.1177/002214650504600204

Hirschi, T. (1969). *Causes of Delinquency*. Berkeley: University of California Press.

Hodge, D., Andereck, K., & Montoya, H. (2007). The protective influence of spiritual-religious lifestyle profiles on tobacco use, alcohol use, and gambling. *Social Work Research*, *31*(4), 211–219.

Hooper, L. M., Tomek, S., Bolland, K. A., II, W. T. C., Wilcox, K., & Bolland, J. M. (2015). The impact of previous suicide ideations, traumatic stress, and gender on future suicide ideation trajectories among black American adolescents: A longitudinal investigation. *Journal of Loss and Trauma*, *20*(4), 354–373. doi: 10.1080/15325024.2014.897573

Horton, K. D., & Loukas, A. (2013). Discrimination, religious coping, and tobacco use among white, African American, and Mexican American vocational school students. *Journal of Religion and Health*, *52*(1), 169–183. doi: 10.1007/s10943-011-9462-z

Howell, R. D., Breivik, E., & Wilcox, J. B. (2007). Reconsidering formative measurement. *Psychological Methods*, *12*(2), 205–218. doi: 10.1037/1082-989X.12.2.205

Hoyland, M. A., Rowatt, W. C., & Latendresse, S. J. (2017). Prior delinquency and depression differentially predict conditional associations between discrete patterns of adolescent religiosity and adult alcohol use patterns. *Substance Abuse: Research and Treatment*, *2017*(10), 0–0. doi: 10.4137/SART.S34948

Hu, L., & Bentler, P. M. (1999). Cutoff criteria for fit indexes in covariance structure analysis: Conventional criteria versus new alternatives. *Structural Equation Modeling: A Multidisciplinary Journal*, *6*(1), 1–55. doi: 10.1080/10705519909540118

Hunte, H. E., & Barry, A. E. (2012). Perceived discrimination and DSM-IV–based alcohol and illicit drug use disorders. *American Journal of Public Health*, *102*(12), e111–e117. doi: 10.2105/AJPH.2012.300780

Hunter, S. B., Miles, J. N. V., Pedersen, E. R., Ewing, B. A., & D'Amico, E. J. (2014). Temporal associations between substance use and delinquency among youth with a first-time offense. *Addictive Behaviors*, *39*(6), 1081–1086. doi: 10.1016/j.addbeh.2014.03.002

Huurre, T., Lintonen, T., Kaprio, J., Pelkonen, M., Marttunen, M., & Aro, H. (2010). Adolescent risk factors for excessive alcohol use at age 32 years: A 16-year prospective follow-up study. *Social Psychiatry and Psychiatric Epidemiology*, *45*(1), 125–134. doi: 10.1007/s00127-009-0048-y

Iwamoto, D. K., & Smiler, A. P. (2013). Alcohol makes you macho and helps you make friends: The role of masculine norms and peer pressure in adolescent boys' and girls' alcohol use. *Substance Use & Misuse*, *48*(5), 371–378. doi: 10.3109/10826084.2013.765479

Jaggers, J. W., Bolland, A. C., Tomek, S., Bolland, K. A., Hooper, L. M., Wesley T. Church, I., & Bolland, J. M. (2018). The longitudinal impact of distal, non-familial relationships on parental monitoring: Implications for delinquent behavior. *Youth & Society*, *50*(2), 160–182. doi: 10.1177/0044118X15602415

Jaggers, J. W., Tomek, S., Bolland, K. A., Church, W. T., Hooper, L. M., & Bolland, J. M. (2014). Personal and anticipated strain among youth: A longitudinal analysis of delinquency. *Journal of Juvenile Justice*, *3*(2), 38–54.

Jarrett, R. L. (1995). Growing up poor: the family experiences of socially mobile youth in low-income African American neighborhoods. *Journal of Adolescent Research*, *10*(1), 111–135. doi: 10.1177/0743554895101007

Jessor, R. (1987). Problem-behavior theory, psychosocial development, and adolescent problem drinking. *British Journal of Addiction*, *82*(4), 331–342. doi: 10.1111/j.1360-0443.1987.tb01490.x

Jessor, R. (1991). Risk behavior in adolescence: A psychosocial framework for understanding and action. *Journal of Adolescent Health*, *12*(8), 597–605. doi: 10.1016/1054-139X(91)90007-K

Jessor, R., & Jessor, S. L. (1977). *Problem Behavior and Psychosocial Development: A Longitudinal Study of Youth*. New York: Academic Press.

Jessor, R., Van Den Bos, J., Vanderryn, J., Costa, F. M., & Turbin, M. S. (1995). Protective factors in adolescent problem behavior: Moderator effects and developmental change. *Developmental Psychology*, *31*(6), 923–33. doi: 10.1037/0012-1649.31.6.923

Joe, S., Baser, R. S., Neighbors, H. W., Caldwell, C. H., & Jackson, J. S. (2009, March). 12-Month and lifetime prevalence of suicide attempts among black adolescents in the National Survey of American Life. *Journal of the American Academy of Child and Adolescent Psychiatry*, *48*(3), 271–282. doi: 10.1097/CHI.0b013e318195bccf

Jung, T., & Wickrama, K. a. S. (2008). An introduction to latent class growth analysis and growth mixture modeling. *Social and Personality Psychology Compass*, *2*(1), 302–317. doi: 10.1111/j.1751-9004.2007.00054.x

Kann, L., McManus, T., Harris, W. A., Shanklin, S. L., Flint, K. H., Queen, B., ... Ethier, K. (2018). Youth Risk Behavior Surveillance - United States, 2017. *MMWR Surveillance Summary 2018*, *67*(No. SS-8), 1–479.

Kaynak, O., Meyers, K., Caldeira, K. M., Vincent, K. B., Winters, K. C., & Arria, A. M. (2013). Relationships among parental monitoring and sensation seeking on the development of substance use disorder among college students. *Addictive Behaviors*, *38*(1), 1457–1463. doi: 10.1016/j.addbeh.2012.08.003

Kelly, L. M., Becker, S. J., & Spirito, A. (2017). Parental monitoring protects against the effects of parent and adolescent depressed mood on adolescent drinking. *Addictive Behaviors*, *75*, 7–11. doi: 10.1016/j.addbeh.2017.06.011

Kendler, K. S., Liu, X.-Q., Gardner, C. O., McCullough, M. E., Larson, D., & Prescott, C. A. (2003). Dimensions of religiosity and their relationship to lifetime psychiatric and substance use disorders. *American Journal of Psychiatry*, *160*(3), 496–503. doi: 10.1176/appi.ajp.160.3.496

Kendzor, D. E., Businelle, M. S., Reitzel, L. R., Rios, D. M., Scheuermann, T. S., Pulvers, K., & Ahluwalia, J. S. (2014). Everyday discrimination is associated with nicotine dependence among African American, Latino, and white smokers. *Nicotine & Tobacco Research*, *16*(6), 633–640. doi: 10.1093/ntr/ntt198

Keyes, K. M., Vo, T., Wall, M. M., Caetano, R., Suglia, S. F., Martins, S. S., ... Hasin, D. (2015). Racial/ethnic differences in use of alcohol, tobacco, and marijuana: Is there a cross-over from adolescence to adulthood? *Social Science & Medicine*, *124*, 132–141. doi: 10.1016/j.socscimed.2014.11.035

Khantzian, E. J. (1997). The self-medication hypothesis of substance use disorders: A reconsideration and recent applications. *Harvard Review of Psychiatry*, *4*(5), 231–244. doi: 10.3109/10673229709030550

Khoury, L., Tang, Y. L., Bradley, B., Cubells, J. F., & Ressler, K. J. (2010). Substance use, childhood traumatic experience, and posttraumatic stress disorder in an urban civilian population. *Depression and Anxiety*, *27*(12), 1077–1086. doi: 10.1002/da.20751

Khoury, M. J., Iademarco, M. F., & Riley, W. T. (2016). Precision public health for the era of precision medicine. *American Journal of Preventive Medicine*, *50*(3), 398–401. doi: 10.1016/j.amepre.2015.08.031

Kiesner, J., Poulin, F., & Dishion, T. J. (2010). Adolescent substance use with friends: Moderating and mediating effects of parental monitoring and peer activity contexts. *Merrill-Palmer Quarterly*, *56*(4), 529–556. Retrieved 2018-02-21, from https://www.ncbi.nlm.nih.gov/pmc/articles/PMC3002110/

Kim-Spoon, J., Farley, J. P., Holmes, C., Longo, G. S., & McCullough, M. E. (2013). Processes linking parents' and adolescents' religiousness and adolescent substance use: Monitoring and self-control. *Journal of Youth and Adolescence*, *43*(5), 745–756. doi: 10.1007/s10964-013-9998-1

Kochanek, K. D., Murphy, S. L., Anderson, R. N., & Scott, C. (2004). *Deaths: Final data for 2002* (Tech. Rep. No. 53(5)). Center for Disease Control (CDC) National Vital Statistics Reports.

Komro, K. A., Tobler, A. L., Maldonado-Molina, M. M., & Perry, C. L. (2010). Effects of alcohol use initiation patterns on high-risk behaviors among urban, low-income, young adolescents. *Prevention Science*, *11*(1), 14–23. doi: 10.1007/s11121-009-0144-y

Kornhauser, R. (1978). *Social Sources of Delinquency*. Chicago: University of Chicago Press.

Kraemer, H. C., Kazdin, A. E., Offord, D. R., Kessler, R. C., Jensen, P. S., & Kupfer, D. J. (1997). Coming to terms with the terms of risk. *Archives of General Psychiatry*, *54*(4), 337–343. doi: 10.1001/archpsyc.1997.01830160065009

Lac, A., & Crano, W. D. (2009). Monitoring matters: Meta-analytic review reveals the reliable linkage of parental monitoring with adolescent marijuana use. *Perspectives on Psychological Science*, *4*(6), 578–586. doi: 10.1111/j.1745-6924.2009.01166.x

Lamborn, S. D., Mounts, N. S., Steinberg, L., & Dornbusch, S. M. (1991). Patterns of competence and adjustment among adolescents from authoritative, authoritarian, indulgent, and neglectful families. *Child Development*, *62*(5), 1049–1065. doi: 10.2307/1131151

Lanza, S. T., & Rhoades, B. L. (2013). Latent class analysis: An alternative perspective on subgroup analysis in prevention and treatment. *Prevention Science*, *14*(2), 157–168. doi: 10.1007/s11121-011-0201-1

Larzelere, R. E., & Patterson, G. R. (1990). Parental management: Mediator of the effect of socioeconomic status on early delinquency. *Criminology*, *28*(2), 301–324. doi: 10.1111/j.1745-9125.1990.tb01327.x

Latendresse, S. J., Ye, F., Chung, T., Hipwell, A., & Sartor, C. E. (2017). Parental monitoring and alcohol use across adolescence in black and white girls: A cross-lagged panel mixture model. *Alcoholism, Clinical and Experimental Research*, *41*(6), 1144–1153. doi: 10.1111/acer.13386

Latkin, C. A., & Curry, A. D. (2003). Stressful neighborhoods and depression: A prospective study of the impact of neighborhood disorder. *Journal of Health and Social Behavior*, *44*(1), 34–44.

Lee, C. G., Seo, D.-C., Torabi, M. R., Lohrmann, D. K., & Song, T. M. (2018). Longitudinal trajectory of the relationship between self-esteem and substance use from adolescence to young adulthood. *The Journal of School Health*, *88*(1), 9–14. doi: 10.1111/josh.12574

Lee, R. (2012). Community violence exposure and adolescent substance use: does monitoring and positive parenting moderate risk in urban communities? *Journal of Community Psychology*, *40*(4), 406–421. doi: 10.1002/jcop.20520

Leeies, M., Pagura, J., Sareen, J., & Bolton, J. M. (2010). The use of alcohol and drugs to self-medicate symptoms of posttraumatic stress disorder. *Depression and Anxiety*, *27*(8), 731–736. doi: 10.1002/da.20677

Lehavot, K., & Simoni, J. M. (2011). The impact of minority stress on mental health and substance use among sexual minority women. *Journal of Consulting and Clinical Psychology*, *79*(2), 159–170. doi: 10.1037/a0022839

Leventhal, T., & Brooks-Gunn, J. (2000). The neighborhoods they live in: The effects of neighborhood residence on child and adolescent outcomes. *Psychological Bulletin*, *126*(2), 309–337.

Li, D., Li, X., Zhao, L., Zhou, Y., Sun, W., & Wang, Y. (2017). Linking multiple risk exposure profiles with adolescent internet addiction: Insights from the person-centered approach. *Computers in Human Behavior*, *75*, 236–244. doi: 10.1016/j.chb.2017.04.063

Lippold, M. A., Hussong, A., Fosco, G. M., & Ram, N. (2018). Lability in the parent's hostility and warmth toward their adolescent: Linkages to youth delinquency and substance use. *Developmental Psychology*, *54*(2), 348–361. doi: 10.1037/dev0000415

Little, R. J. A., & Rubin, D. B. (2002). *Statistical Analysis with Missing Data* (2nd ed.). Hoboken, N.J: John Wiley & Sons.

Lloyd, C. (1998). Risk factors for problem drug use: Identifying vulnerable groups. *Drugs: Education, Prevention and Policy*, *5*(3), 217–232. doi: 10.3109/09687639809034084

Lo, Y., Mendell, N. R., & Rubin, D. B. (2001). Testing the number of components in a normal mixture. *Biometrika*, *88*(3), 767–778. doi: 10.1093/biomet/88.3.767

Lockhart, G., Phillips, S., Bolland, A., Delgado, M., Tietjen, J., & Bolland, J. (2017). Prospective relations among low-income African American adolescents' maternal attachment security, self-worth, and risk behaviors. *Frontiers in Psychology*, *8*, 33. doi: 10.3389/fpsyg.2017.00033

Loeber, R., Stepp, S. D., Chung, T., Hipwell, A. E., & White, H. R. (2010). Time-varying associations between conduct problems and alcohol use in adolescent girls: The moderating role of race. *Journal of Studies on Alcohol and Drugs*, *71*(4), 544–553.

Lord, F. (1980). *Applications of item response theory to practical testing problems*. Hillsdale, NJ: Erlbaum.

Luthar, S. S., Doernberger, C. H., & Zigler, E. (1993). Resilience is not a unidimensional construct: Insights from a prospective study of inner-city adolescents. *Development and Psychopathology*, *5*(4), 703–717. doi: 10.1017/S0954579400006246

Lynne-Landsman, S. D., Bradshaw, C. P., & Ialongo, N. S. (2010). Testing a developmental cascade model of adolescent substance use trajectories and young adult adjustment. *Development and Psychopathology*, *22*(4), 933–948. doi: 10.1017/S0954579410000556

Lynne-Landsman, S. D., Graber, J. A., Nichols, T. R., & Botvin, G. J. (2011). Trajectories of aggression, delinquency, and substance use across middle school among urban, minority adolescents. *Aggressive Behavior*, *37*(2), 161–176.

Magnusson, D. (1990). Personality development from an interactional perspective. In L. Pervin (Ed.), *Handbook of Personality* (pp. 193–222). New York: Guliford Press.

Magnusson, D. (1999). Holistic interactionism: A perspective for research on personality development. In L. A. Pervin & O. John (Eds.), *Handbook of Personality: Theory and Research* (pp. 219–247). New York, NY, US: Guliford Press.

Magnusson, D. (2003). The person approach: Concepts, measurement models, and research strategy. *New Directions for Child and Adolescent Development*, *2003*(101), 3–23. doi: 10.1002/cd.79

Mark, T. L., Levit, K. R., Yee, T., & Chow, C. M. (2014). Spending on mental and substance use disorders projected to grow more slowly than all health spending through 2020. *Health Affairs*, *33*(8), 1407–1415. doi: 10.1377/hlthaff.2014.0163

Marotta, P. L., & Voisin, D. R. (2017). Testing three pathways to substance use and delinquency among low-income African American adolescents. *Children and Youth Services Review*, *75*(Supplement C), 7–14. doi: 10.1016/j.childyouth.2017.02.009

Marshall, E. J. (2014). Adolescent alcohol use: Risks and consequences. *Alcohol and Alcoholism*, *49*(2), 160–164. doi: 10.1093/alcalc/agt180

Mason, M. J., Mennis, J., Linker, J., Bares, C., & Zaharakis, N. (2014). Peer attitudes effects on adolescent substance use: The moderating role of race and gender. *Prevention Science*, *15*(1), 56–64. doi: 10.1007/s11121-012-0353-7

Mason, W. A., Hitchings, J. E., McMahon, R. J., & Spoth, R. L. (2007). A test of three alternative hypotheses regarding the effects of early delinquency on adolescent psychosocial functioning and substance involvement. *Journal of Abnormal Child Psychology*, *35*(5), 831–843. doi: 10.1007/s10802-007-9130-7

Mason, W. A., Hitchings, J. E., & Spoth, R. L. (2007). Emergence of delinquency and depressed mood throughout adolescence as predictors of late adolescent problem substance use. *Psychology of Addictive Behaviors*, *21*(1), 13–24. doi: 10.1037/0893-164X.21.1.13

Mason, W. A., & Windle, M. (2002). Reciprocal relations between adolescent substance use and delinquency: A longitudinal latent variable analysis. *Journal of Abnormal Psychology*, *111*(1), 63–76. doi: 10.1037/0021-843X.111.1.63

Masten, A. S., & Cicchetti, D. (2010). Developmental cascades. *Development and Psychopathology*, *22*(3), 491–495. doi: 10.1017/S0954579410000222

Mastrofski, S., Parks, R., Reiss, A., & Worden, R. (1999). *Policing neighborhoods: A report from St. Petersburg* (Tech. Rep.). Washington, DC: National Institue of Justice.

Mayberry, M. L., Espelage, D. L., & Koenig, B. (2009). Multilevel modeling of direct effects and interactions of peers, parents, school, and community influences on adolescent substance use. *Journal of Youth and Adolescence*, *38*(8), 1038–1049. doi: 10.1007/s10964-009-9425-9

McCutcheon, A. L. (1987). *Latent Class Analysis*. Newbury Park, CA: SAGE.

Mereish, E. H., & Bradford, J. B. (2014). Intersecting Identities and substance use problems: Sexual orientation, gender, race, and lifetime substance use problems. *Journal of Studies on Alcohol and Drugs*, *75*(1), 179–188. doi: 10.15288/jsad.2014.75.179

Merikangas, K. R., He, J.-p., Burstein, M. E., Swendsen, J., Avenevoli, S., Case, B., ... Olfson, M. (2011). Service utilization for lifetime mental disorders in U.S. adolescents: Results of the National Comorbidity Survey Adolescent Supplement (NCS-A). *Journal of the American Academy of Child and Adolescent Psychiatry*, *50*(1), 32–45. doi: 10.1016/j.jaac.2010.10.006

Meyers, J. L., Brown, Q., Grant, B. F., & Hasin, D. (2017). Religiosity, race/ethnicity, and alcohol use behaviors in the United States. *Psychological Medicine*, *47*(1), 103–114. doi: 10.1017/S0033291716001975

Miech, R., Johnston, L., O'Malley, P. M., Bachman, J. G., & Patrick, M. E. (2019). Adolescent vaping and nicotine use in 2017–2018 — U.S. national estimates. *New England Journal of Medicine*, *380*(2), 192–193. doi: 10.1056/NEJMc1814130

Miller, R. N., Fagan, A. A., & Wright, E. M. (2014). The moderating effects of peer and parental support on the relationship between vicarious victimization and substance use. *Journal of Drug Issues*, *44*(4), 362–380. doi: 10.1177/0022042614526995

Monahan, K. C., Lee, J. M., & Steinberg, L. (2011). Revisiting the impact of part-time work on adolescent adjustment: Distinguishing between selection and socialization using propensity score matching. *Child Development*, *82*(1), 96–112. doi: 10.1111/j.1467-8624.2010.01543.x

Monahan, K. C., Oesterle, S., Rhew, I., & Hawkins, J. D. (2014). The relation between risk and protective factors for problem behaviors and depressive symptoms, antisocial behavior, and alcohol use in adolescence. *Journal of Community Psychology*, *42*(5), 621–638. doi: 10.1002/jcop.21642

Mulia, N., Ye, Y., Greenfield, T. K., & Zemore, S. E. (2009). Disparities in alcohol-related problems among white, Black and Hispanic Americans. *Alcoholism, Clinical and Experimental Research*, *33*(4), 654–662. doi: 10.1111/j.1530-0277.2008.00880.x

Mulia, N., Ye, Y., Zemore, S. E., & Greenfield, T. K. (2008, November). Social disadvantage, stress, and alcohol use among black, Hispanic, and white Americans: Findings from the 2005 U.S. National Alcohol Survey. *Journal of Studies on Alcohol and Drugs*, *69*(6), 824–833.

Muthén, B. O., & Muthén, L. K. (2000). Integrating person-centered and variable-centered analyses: Growth mixture modeling with latent trajectory classes. *Alcoholism: Clinical and Experimental Research*, *24*(6), 882–891. doi: 10.1111/j.1530-0277.2000.tb02070.x

Muthén, L. K., & Muthén, B. O. (1998-2018). *Mplus User's Guide.* (8.2 ed.). Los Angeles, CA: Muthén & Muthén.

Nash, J. K., & Bowen, G. L. (2002). Defining and estimating risk and protection: An illustration from the school success profile. *Child and Adolescent Social Work Journal*, *19*(3), 247–261. doi: 10.1023/A:1015532132061

Nebbitt, V. E., Lombe, M., Yu, M., Vaughn, M. G., & Stokes, C. (2012). Ecological correlates of substance use in African American adolescents living in public housing communities: Assessing the moderating effects of social cohesion. *Children and Youth Services Review*, *34*(2), 338–347. doi: 10.1016/j.childyouth.2011.11.003

Nonnemaker, J. M., McNeely, C. A., & Blum, R. W. (2003). Public and private domains of religiosity and adolescent health risk behaviors: Evidence from the National Longitudinal Study of Adolescent Health. *Social Science & Medicine*, *57*(11), 2049–2054. doi: 10.1016/S0277-9536(03)00096-0

O'Brien, L., Albert, D., Chein, J., & Steinberg, L. (2011). Adolescents prefer more immediate rewards when in the presence of their peers. *Journal of Research on Adolescence*, *21*(4), 747–753. doi: 10.1111/j.1532-7795.2011.00738.x

Parenteau, S. C., Waters, K., Cox, B., Patterson, T., & Carr, R. (2017). Racial discrimination and alcohol use: The moderating role of religious orientation. *Substance Use & Misuse*, *52*(1), 1–9. doi: 10.1080/10826084.2016.1201840

Park, E., McCoy, T. P., Erausquin, J. T., & Bartlett, R. (2018). Trajectories of risk behaviors across adolescence and young adulthood: The role of race and ethnicity. *Addictive Behaviors*, *76*, 1–7. doi: 10.1016/j.addbeh.2017.07.014

Parra, G. R., DuBois, D. L., & Sher, K. J. (2006). Investigation of profiles of risk factors for adolescent psychopathology: A person-centered approach. *Journal of Clinical Child & Adolescent Psychology*, *35*(3), 386–402. doi: 10.1207/s15374424jccp3503_4

Pascoe, E. A., & Smart Richman, L. (2009). Perceived discrimination and health: A meta-analytic review. *Psychological Bulletin*, *135*(4), 531–554. doi: 10.1037/a0016059

Patrick, M. E., & Schulenberg, J. E. (2013). Prevalence and predictors of adolescent alcohol use and binge drinking in the United States. *Alcohol Research: Current Reviews*, *35*(2), 193–200.

Perkins, D. D., Florin, P., Rich, R. C., Wandersman, A., & Chavis, D. M. (1990). Participation and the social and physical environment of residential blocks: Crime and community context. *American Journal of Community Psychology*, *18*(1), 83–115. doi: 10.1007/BF00922690

Piko, B. F., & Kovács, E. (2010). Do parents and school matter? Protective factors for adolescent substance use. *Addictive Behaviors*, *35*(1), 53–56. doi: 10.1016/j.addbeh.2009.08.004

Pinchevsky, G. M., Fagan, A. A., & Wright, E. M. (2014). Victimization experiences and adolescent substance use: Does the type and degree of victimization matter? *Journal of Interpersonal Violence*, *29*(2), 299–319. doi: 10.1177/0886260513505150

Pinchevsky, G. M., Wright, E. M., & Fagan, A. A. (2013). Gender differences in the effects of exposure to violence on adolescent substance use. *Violence and Victims*, *28*(1), 122–144.

Pirutinsky, S. (2014). Does religiousness increase self-control and reduce criminal behavior? A longitudinal analysis of adolescent offenders. *Criminal Justice and Behavior*, *41*(11), 1290–1307. doi: 10.1177/0093854814531962

Pollard, J. A., Hawkins, J. D., & Arthur, M. W. (1999). Risk and protection: Are both necessary to understand diverse behavioral outcomes in adolescence? *Social Work Research*, *23*(3), 145–158. doi: 10.1093/swr/23.3.145

Pratt, M. E., McClelland, M. M., Swanson, J., & Lipscomb, S. T. (2016). Family risk profiles and school readiness: A person-centered approach. *Early Childhood Research Quarterly*, *36*, 462–474. doi: 10.1016/j.ecresq.2016.01.017

Purnell, J. Q., Peppone, L. J., Alcaraz, K., McQueen, A., Guido, J. J., Carroll, J. K., ... Morrow, G. R. (2012). Perceived discrimination, psychological distress, and current smoking status: Results from the Behavioral Risk Factor Surveillance System Reactions to Race Module, 2004–2008. *American Journal of Public Health*, *102*(5), 844–851. doi: 10.2105/AJPH.2012.300694

Racz, S. J., & McMahon, R. J. (2011). The relationship between parental knowledge and monitoring and child and adolescent conduct problems: A 10-year update. *Clinical Child and Family Psychology Review*, *14*(4), 377–398. doi: 10.1007/s10567-011-0099-y

Ram, N., & Grimm, K. J. (2009). Growth mixture modeling: A method for identifying differences in longitudinal change among unobserved groups. *International Journal of Behavioral Development*, *33*(6), 565–576. doi: 10.1177/0165025409343765

Rhodes, J. E., & Jason, L. A. (1990). A social stress model of substance abuse. *Journal of Consulting and Clinical Psychology*, *58*(4), 395–401.

Richardson, C. G., Kwon, J.-Y., & Ratner, P. A. (2013). Self-esteem and the initiation of substance use among adolescents. *Canadian Journal of Public Health / Revue Canadienne de Santé Publique*, *104*(1), e60–e63.

Ritchwood, T. D., Ford, H., DeCoster, J., Sutton, M., & Lochman, J. E. (2015). Risky sexual behavior and substance use among adolescents: A meta-analysis. *Children and Youth Services Review*, *52*, 74–88. doi: 10.1016/j.childyouth.2015.03.005

Roberts, R. E., Roberts, C. R., & Xing, Y. (2007). Comorbidity of substance use disorders and other psychiatric disorders among adolescents: Evidence from an epidemiologic survey. *Drug and alcohol dependence*, *88*(Suppl 1), S4–13. doi: 10.1016/j.drugalcdep.2006.12.010

Roehler, D. R., Heinze, J. E., Stoddard, S. A., Bauermeister, J. A., & Zimmerman, M. A. (2017). The association between early exposure to violence in emerging adulthood and substance use in early-adulthood among inner-city individuals. *Emerging Adulthood*, 2167696817725455. doi: 10.1177/2167696817725455

Rothstein, R. (2015). The racial achievement gap, segregated schools, and segregated neighborhoods: A constitutional insult. *Race and Social Problems*, *7*(1), 21–30. doi: 10.1007/s12552-014-9134-1

Rubens, S. L., Fite, P. J., Cooley, J. L., & Canter, K. S. (2014). The role of sleep in the relation between vommunity violence exposure and delinquency among Latino adolescents. *Journal of Community Psychology*, *42*(6), 723–734. doi: 10.1002/jcop.21648

Rutter, M. (1987). Psychosocial resilience and protective mechanisms. *American Journal of Orthopsychiatry*, *57*(3), 316–331. doi: 10.1111/j.1939-0025.1987.tb03541.x

Salas-Wright, C. P., Vaughn, M. G., Hodge, D. R., & Perron, B. E. (2012). Religiosity profiles of American youth in relation to substance use, violence, and delinquency. *Journal of Youth and Adolescence*, *41*(12), 1560–1575. doi: http://dx.doi.org/10.1605/01.301-0021318358.2012

Sampson, R. J., & Groves, W. B. (1989). Community structure and crime: Testing social-disorganization theory. *American Journal of Sociology*, *94*(4), 774–802.

Schmidt, L., Greenfield, T., & Mulia, N. (2006). Unequal treatment: Racial and ethnic disparities in alcoholism treatment services. *Alcohol Research & Health*, *29*(1), 49–54.

Schubiner, H., Scott, R., & Tzelepis, A. (1993). Exposure to violence among inner-city youth. *Journal of Adolescent Health*, *14*(3), 214–219. doi: 10.1016/1054-139X(93)90008-D

Schulenberg, J., Patrick, M. E., Maslowsky, J., & Maggs, J. L. (2014). The epidemiology and etiology of adolescent substance use in developmental perspective. In *Handbook of Developmental Psychopathology* (pp. 601–620). Springer, Boston, MA. doi: 10.1007/978-1-4614-9608-3_30

Schuster, R. M., Mermelstein, R., & Wakschlag, L. (2013). Gender-specific relationships between depressive symptoms, marijuana use, parental communication and risky sexual behavior in adolescence. *Journal of Youth and Adolescence*, *42*(8), 1194–1209. doi: 10.1007/s10964-012-9809-0

Schwartz, G. (1978). Estimating the dimensions of a model. *The Annals of Statistics*, *6*(2), 461–464.

Schwinn, T. M., Schinke, S. P., Hopkins, J., & Thom, B. (2016). Risk and protective factors associated with adolescent girls' substance use: Data from a nationwide Facebook sample. *Substance Abuse*, *37*(4), 564–570. doi: 10.1080/08897077.2016.1154495

Sclove, S. L. (1987). Application of model-selection criteria to some problems in multivariate analysis. *Psychometrika*, *52*(3), 333–343. doi: 10.1007/BF02294360

Scull, T. M., Kupersmidt, J. B., Parker, A. E., Elmore, K. C., & Benson, J. W. (2010). Adolescents' media-related cognitions and substance use in the context of parental and peer influences. *Journal of Youth and Adolescence*, *39*(9), 981–998. doi: 10.1007/s10964-009-9455-3

Shaw, C. R., & McKay, H. D. (1942). *Juvenile Delinquency and Urban Areas*. Chicago, IL, US: University of Chicago Press.

Shih, R. A., Parast, L., Pedersen, E. R., Troxel, W. M., Tucker, J. S., Miles, J. N. V., ... D'Amico, E. J. (2017). Individual, peer, and family factor modification of neighborhood-level effects on adolescent alcohol, cigarette, e-cigarette, and marijuana use. *Drug and Alcohol Dependence*, *180*, 76–85. doi: 10.1016/j.drugalcdep.2017.07.014

Simons-Morton, B., Haynie, D. L., Crump, A. D., Eitel, S. P., & Saylor, K. E. (2001). Peer and parent influences on smoking and drinking among early adolescents. *Health Education & Behavior*, *28*(1), 95–107. doi: 10.1177/109019810102800109

Singer, S. I., & Levine, M. (1988). Power-control theory, gender, and delinquency: A partial replication with additional evidence on the effects of peers. *Criminology*, *26*(4), 627–648. doi: 10.1111/j.1745-9125.1988.tb00857.x

Skrondal, A., & Laake, P. (2001). Regression among factor scores. *Psychometrika*, *66*(4), 563–575. doi: 10.1007/BF02296196

Slade, E. P., Stuart, E. A., Salkever, D. S., Karakus, M., Green, K. M., & Ialongo, N. (2008). Impacts of age of onset of substance use disorders on risk of adult incarceration among disadvantaged urban youth: A propensity score matching approach. *Drug and alcohol dependence*, *95*(1-2), 1–13. doi: 10.1016/j.drugalcdep.2007.11.019

Sloboda, Z., Glantz, M. D., & Tarter, R. E. (2012). Revisiting the concepts of risk and protective factors for understanding the etiology and development of substance use and substance use disorders: Implications for prevention. *Substance Use & Misuse*, *47*(8-9), 944–962. doi: 10.3109/10826084.2012.663280

Spano, R., Rivera, C., & Bolland, J. (2006). The impact of timing of exposure to violence on violent behavior in a high poverty sample of inner city African American youth. *Journal of Youth and Adolescence*, *35*(5), 681–692.

Spano, R., Rivera, C., & Bolland, J. M. (2010). Are chronic exposure to violence and chronic violent behavior closely related developmental processes during adolescence? *Criminal Justice and Behavior*, *37*(10), 1160–1179.

Spano, R., Rivera, C., & Bolland, J. M. (2011). Does parenting shield youth from exposure to violence during adolescence? A 5-year longitudinal test in a high-poverty sample of minority youth. *Journal of Interpersonal Violence*, *26*(5), 930–949.

Spano, R., Rivera, C., Vazsonyi, A. T., & Bolland, J. M. (2008). The impact of exposure to violence on a trajectory of (declining) parental monitoring: A partial test of the ecological—transactional model of community violence. *Criminal Justice and Behavior*, *35*(11), 1411–1428.

Spano, R., Rivera, C., Vazsonyi, A. T., & Bolland, J. M. (2012). Specifying the interrelationship between exposure to violence and parental monitoring for younger versus older adolescents: A five year longitudinal test. *American Journal of Community Psychology*, *49*(1-2), 127–141.

Spano, R., Vazsonyi, A. T., & Bolland, J. (2009). Does parenting mediate the effects of exposure to violence on violent behavior? An ecological–transactional model of community violence. *Journal of Adolescence*, *32*(5), 1321–1341.

Squeglia, L. M., Jacobus, J., & Tapert, S. F. (2009). The influence of substance use on adolescent brain development. *Clinical EEG and Neuroscience*, *40*(1), 31–38. doi: 10.1177/155005940904000110

Stattin, H., & Kerr, M. (2000). Parental monitoring: A reinterpretation. *Child Development*, *71*(4), 1072–1085.

Steinman, K. J., & Zimmerman, M. A. (2004). Religious activity and risk behavior among African American adolescents: Concurrent and developmental effects. *American Journal of Community Psychology*, *33*(3-4), 151–161. doi: 10.1023/B:AJCP.0000027002.93526.bb

Stock, M. L., Peterson, L. M., Molloy, B. K., & Lambert, S. F. (2017). Past racial discrimination exacerbates the effects of racial exclusion on negative affect, perceived control, and alcohol-risk cognitions among Black young adults. *Journal of Behavioral Medicine*, *40*(3), 377–391. doi: 10.1007/s10865-016-9793-z

Studer, J., Baggio, S., Deline, S., N'Goran, A. A., Henchoz, Y., Mohler-Kuo, M., … Gmel, G. (2014). Peer pressure and alcohol use in young men: A mediation analysis of drinking motives. *The International Journal on Drug Policy*, *25*(4), 700–708. doi: 10.1016/j.drugpo.2014.02.002

Studer, J., Baggio, S., Grazioli, V. S., Mohler-Kuo, M., Daeppen, J.-B., & Gmel, G. (2016). Risky substance use and peer pressure in Swiss young men: Test of moderation effects. *Drug and Alcohol Dependence*, *168*, 89–98. doi: 10.1016/j.drugalcdep.2016.08.633

Sturman, D. A., & Moghaddam, B. (2011). The neurobiology of adolescence: Changes in brain architecture, functional dynamics, and behavioral tendencies. *Neuroscience and Biobehavioral Reviews*, *35*(8), 1704–1712. doi: 10.1016/j.neubiorev.2011.04.003

Su, J., & Supple, A. J. (2014). Parental, peer, school, and neighborhood influences on adolescent substance use: Direct and indirect effects and ethnic variations. *Journal of Ethnicity in Substance Abuse*, *13*(3), 227–246. doi: 10.1080/15332640.2013.847393

Susman, E. J., & Dorn, L. D. (2009). Puberty: Its role in development. In *Handbook of Adolescent Psychology: Individual Bases of Adolescent Development* (3rd ed., Vol. 1, pp. 116–151). Hoboken, NJ: John Wiley & Sons Inc. doi: 10.1002/9780470479193.adlpsy001006

Sutherland, E. H. (1973). Development of the theory [Private paper published posthumously]. In K. Schuessler (Ed.), *Edwin Sutherland on analyzing crime.* Chicago: University of Chicago Press.

Syvertsen, A. K., Cleveland, M. J., Gayles, J. G., Tibbits, M. K., & Faulk, M. T. (2010). Profiles of protection from substance use among adolescents. *Prevention Science*, *11*(2), 185–196. doi: 10.1007/s11121-009-0154-9

Taylor, K. W., & Kliewer, W. (2006). Violence exposure and early adolescent alcohol use: an exploratory study of family risk and protective factors. *Journal of Child and Family Studies*, *15*(2), 201–215. doi: 10.1007/s10826-005-9017-6

Taylor, R. J., Chatters, L. M., Jayakody, R., & Levin, J. S. (1996). Black and white differences in religious participation: A multisample comparison. *Journal for the Scientific Study of Religion*, *35*(4), 403–410. doi: 10.2307/1386415

Tebes, J. K., Cook, E. C., Vanderploeg, J. J., Feinn, R., Chinman, M. J., Shepard, J. K., ... Connell, C. M. (2011). Parental knowledge and substance use among African American adolescents: Influence of gender and grade level. *Journal of Child and Family Studies*, *20*(4), 406–413. doi: 10.1007/s10826-010-9406-3

Terrell, F., Miller, A. R., Foster, K., & Watkins, C. E. (2006). Racial discrimination-induced anger and alcohol use among black adolescents. *Adolescence*, *41*(163), 485–492.

Thomas, C. R., & Holzer, C. E. (2006). The continuing shortage of child and adolescent psychiatrists. *Journal of the American Academy of Child & Adolescent Psychiatry*, *45*(9), 1023–1031. doi: 10.1097/01.chi.0000225353.16831.5d

Titus, J. C., Dennis, M. L., Lennox, R., & Scott, C. K. (2008). Development and validation of short versions of the internal mental distress and behavior complexity scales in the Global Appraisal of Individual Needs (GAIN). *The Journal of Behavioral Health Services & Research*, *35*(2), 195–214. doi: 10.1007/s11414-008-9107-5

Tobler, A. L., & Komro, K. A. (2010). Trajectories of parental monitoring and communication and effects on drug use among urban young adolescents. *The Journal of Adolescent Health*, *46*(6), 560–568. doi: 10.1016/j.jadohealth.2009.12.008

Tomaka, J., Morales-Monks, S., & Shamaley, A. G. (2013). Stress and coping mediate relationships between contingent and global self-esteem and alcohol-related problems among college drinkers. *Stress and Health*, *29*(3), 205–213. doi: 10.1002/smi.2448

Trucco, E. M., Colder, C. R., Bowker, J. C., & Wieczorek, W. F. (2011). Interpersonal goals and susceptibility to peer influence: Risk factors for intentions to initiate substance use during early adolescence. *The Journal of Early Adolescence*, *31*(4), 526–547. doi: 10.1177/0272431610366252

Udell, W., Hotton, A. L., Emerson, E., & Donenberg, G. R. (2017). Does parental monitoring moderate the impact of community violence exposure on probation youth's substance use and sexual risk behavior? *Journal of Child and Family Studies*, *26*(9), 2556–2563. doi: 10.1007/s10826-017-0769-6

Unger, J. B. (2012). The most critical unresolved issues associated with race, ethnicity, culture, and substance use. *Substance Use & Misuse*, *47*(4), 390–395. doi: 10.3109/10826084.2011.638017

Vidourek, R. A., & King, K. A. (2010). Risk and protective factors for recent alcohol use among African-American youth. *Journal of Drug Education*, *40*(4), 411–425. doi: 10.2190/DE.40.4.f

Volkow, N. D., Baler, R. D., Compton, W. M., & Weiss, S. R. (2014). Adverse health effects of marijuana use. *New England Journal of Medicine*, *370*(23), 2219–2227. doi: 10.1056/NEJMra1402309

Vuong, Q. H. (1989). Likelihood ratio tests for model selection and non-nested hypotheses. *Econometrica*, *57*(2), 307–333. doi: 10.2307/1912557

Wallace, J. M., Brown, T. N., Bachman, J. G., & LaVeist, T. A. (2003). The influence of race and religion on abstinence from alcohol, cigarettes and marijuana among adolescents. *Journal of Studies on Alcohol*, *64*(6), 843–848.

Wallace, J. M., & Muroff, J. R. (2002). Preventing substance abuse among African American children and youth: Race differences in risk factor exposure and vulnerability. *The Journal of Primary Prevention*, *22*(3), 235–261. doi: 10.1023/A:1013617721016

Wallace, S. A., Neilands, T. B., & Sanders Phillips, K. (2017). Neighborhood context, psychological outlook, and risk behaviors among urban African American youth. *Cultural Diversity and Ethnic Minority Psychology*, *23*(1), 59–69. doi: 10.1037/cdp0000108

Watt, T. T., & Rogers, J. M. (2007). Factors contributing to differences in substance use among black and white adolescents. *Youth & Society*, *39*(1), 54–74. doi: 10.1177/0044118X06296701

Wheeler, S. B. (2010). Effects of self-esteem and academic performance on adolescent decision-making: An examination of early sexual intercourse and illegal substance use. *The Journal of Adolescent Health*, *47*(6), 582–590. doi: 10.1016/j.jadohealth.2010.04.009

White, H., McMorris, B. J., Catalano, R., Fleming, C., Haggerty, K., & Abbott, R. (2006). Increases in alcohol and marijuana use during the transition out of high school into emerging adulthood: The effects of leaving home, going to college, and high school protective factors. *Journal of Studies on Alcohol*, *67*(6), 810. doi: 10.15288/jsa.2006.67.810

Williams, D. R., Neighbors, H. W., & Jackson, J. S. (2003). Racial/ethnic discrimination and health: Findings from community studies. *American Journal of Public Health*, *93*(2), 200–208.

Williams, D. R., Yu, Y., Jackson, J. S., & Anderson, N. B. (1997). Racial differences in physical and mental health: Socio-economic status, stress and discrimination. *Journal of Health Psychology*, *2*(3), 335–351. doi: 10.1177/135910539700200305

Wood, M. D., Read, J. P., Mitchell, R. E., & Brand, N. H. (2004). Do parents still matter? Parent and peer influences on alcohol involvement among recent high school graduates. *Psychology of Addictive Behaviors*, *18*(1), 19–30. doi: 10.1037/0893-164X.18.1.19

Wright, D. R., & Fitzpatrick, K. M. (2004). Psychosocial correlates of substance use behaviors among African American youth. *Adolescence*, *39*(156), 653–667.

Wright, E. M., Fagan, A. A., & Pinchevsky, G. M. (2013). The effects of exposure to violence and victimization across life domains on adolescent substance use. *Child Abuse & Neglect*, *37*(11), 899–909. doi: 10.1016/j.chiabu.2013.04.010

Yan, H. (2013). *Does sense of community mediate the effects of neighborhood disadvantage on adolescent drug use?* (Unpublished master's thesis). University of Maryland, College Park, MD.

Yen, I. H., Ragland, D. R., Greiner, B. A., & Fisher, J. M. (1999). Racial discrimination and alcohol-related behavior in urban transit operators: Findings from the San Francisco Muni Health and Safety Study. *Public Health Reports (Washington, D.C.: 1974)*, *114*(5), 448–458.

Yoder, K. A., Whitbeck, L. B., & Hoyt, D. R. (2003). Gang involvement and membership among homeless and runaway youth. *Youth & Society*, *34*(4), 441–467. doi: 10.1177/0044118X03034004003

Yoo, H. C., Gee, G. C., Lowthrop, C. K., & Robertson, J. (2010). Self-reported racial discrimination and substance use among Asian Americans in Arizona. *Journal of Immigrant and Minority Health*, *12*(5), 683–690. doi: 10.1007/s10903-009-9306-z

Zapolski, T. C. B., Pedersen, S. L., McCarthy, D. M., & Smith, G. T. (2014). Less drinking, yet more problems: Understanding African American drinking and related problems. *Psychological Bulletin*, *140*(1), 188–223. doi: 10.1037/a0032113

Zeigler-Hill, V., Dahlen, E. R., & Madson, M. B. (2017). Self-esteem and alcohol use: Implications for aggressive behavior. *International Journal of Mental Health and Addiction*, *15*(5), 1103–1117. doi: 10.1007/s11469-017-9764-9

Zinzow, H. M., Ruggiero, K. J., Hanson, R. F., Smith, D. W., Saunders, B. E., & Kilpatrick, D. G. (2009). Witnessed community and parental violence in relation to substance use and delinquency in a national sample of adolescents. *Journal of Traumatic Stress*, *22*(6), 525–533. doi: 10.1002/jts.20469