ALCOHOL USE, DEPRESSIVE SYMPTOMS, AND PARENTAL ALCOHOL USE PROBLEMS AMONG A NATIONALLY REPRESENTATIVE SAMPLE OF YOUTH INVESTIGATED FOR MALTREATMENT

by

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(Under the Direction of Assaf Oshri)

ABSTRACT

The relations between depressive symptoms and alcohol use among maltreated youth have been well documented. Two contrasting theories about the mechanisms underlying these relations have received extensive support: the *self-medication* and *impaired-functioning hypotheses*. The present study proposed to reconcile these theoretical and empirical inconsistencies using a developmental approach. Specifically, an integrative conceptualization, the *bidirectional hypothesis,* was advanced, which used developmental timing to explain the longitudinal associations between maltreated youth's alcohol use and depressive symptoms. In addition, this investigation examined the role of parental alcohol problems in the etiology of maltreated youth's alcohol use and depressive symptoms. Data of 657 youth were drawn from NSCAW II, a longitudinal, nationally representative sample of maltreated youth. Findings supported the impaired-functioning hypothesis during early-adolescence, and corroborated the self-medication hypothesis during mid- to late-adolescence, thus supporting the bidirectional hypothesis. Lastly, increased youth depressive symptoms mediated the link between parental alcohol problems and youth's alcohol use.

INDEX WORDS: Child Maltreatment, Depressive symptoms, Youth alcohol use, Parental alcohol use problems

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CHAPTER 1

INTRODUCTION

Child maltreatment is a prevalent public health concern in the United States and embodies one of the most toxic relationship environment of child development (Cicchetti & Toth, 2005). Youth who experienced maltreatment during childhood are at significant risk for the development of early alcohol use behaviors (Shin, Edwards, & Heeren, 2009), and depressive symptoms (Kim & Cicchetti, 2006) during adolescence. The etiology of adolescent substance use in relation to these early precursors are important to investigate because early alcohol use has been documented as a robust predictor of substance use persistence across the life-course (Sartor, Lynskey, Heath, Jacob, & True, 2007). According to developmental perspectives on substance use (Schulenberg & Maggs, 2002; Tarter, 2002) and psychopathology (Cicchetti & Toth, 2005), early experiences, the timing of substance use, and associated psychopathology bear critical effects on youth developmental sequelae. Specifically, research suggests that abusive and neglectful parenting can hinder children's attainment of expected stage-salient developmental tasks throughout childhood and adolescence, thereby probabilistically increasing the emergence of maladaptive outcomes such as precocious alcohol use (Brems, Johnson, Neal, & Freemon, 2004) and depressive affect (Kim & Cicchetti, 2006; Oshri, Rogosch, & Cicchetti, 2013).

Although connections between child maltreatment, alcohol use, and depressive symptoms among youth have been established in the empirical literature (Kim & Cicchetti, 2006; Poulin, Hand, Boudreau, & Santor, 2005; Rogosch, Oshri, & Cicchetti, 2010), less is known about the causal temporal order of the effects between alcohol use and depressive symptoms and of the

role of the context of developmental timing on the mechanisms that underlie these associations during adolescence (Johnson et al., 2013). The associations between alcohol use and depressive symptoms have been commonly explained by two competing hypotheses, the *self-medication hypothesis and* the *impaired-functioning hypothesis*, which are contradictory in their purported causal order between alcohol use and depressive symptoms among maltreated adolescents. The self-medication hypothesis suggests that depressive symptoms precede alcohol drinking. Specifically, youth with depressive symptoms consume alcohol to alleviate their negative affects induced by depressive symptoms, thus increasing their risk for higher levels of alcohol use (Khantzian, 1987); whereas the impaired-functioning hypothesis indicates that alcohol consumption exacerbate youth's emotional regulation difficulties and mood swings, thus increasing their risk for higher levels of depressive symptoms (Stice, Burton, & Shaw, 2004). Such contradictory perspective is particularly puzzling because there is empirical research that supports both hypotheses (Blume, Schmaling, & Marlatt, 2000; Gilman & Abraham, 2001; Locke & Newcomb, 2001; Marmorstein, 2009). Thus, an integrative theory backed by empirical studies is necessary. In the present study, a developmental psychopathology perspective was advanced to reconcile the differences between these hypotheses (Cicchetti & Rogosch, 2002) after accounting for developmental timing. In particular, according to developmental psychopathology perspective, the self-medication and the impaired-functioning are not mutually exclusive—youth alcohol use and depressive symptoms can promote each other over time. Therefore, the current study also examined the *bidirectional hypothesis* in addition to the self-medication and the impaired-functioning hypotheses about the direction of the associations between depressive symptoms and alcohol use among maltreated adolescents.

Furthermore, parental alcohol use problems have been documented to highly co-occur with adverse parenting practices, especially child maltreatment (Manly, Oshri, Lynch, Herzog, & Wortel, 2013).Previous studies have established parental alcohol use problems as a significant risk factor for adolescent alcohol use (Chassin, Curran, Hussong, & Colder, 1996; Donovan, 2004) and depressive symptoms (Brook, Brook, Zhang, Cohen, & Whiteman, 2002). Thus, when investigating the direction of the associations between alcohol use and depressive symptoms among maltreated youth, testing the role of parental alcohol use problem is methodologically and theoretically critical. Moreover, the mechanisms underlying the associations between parental alcohol use problems, youth alcohol use and depressive symptoms among maltreated adolescents have not been sufficiently clarified in the empirical literature. Therefore, more research is needed to account for the role of parental alcohol use problems in the developmental pathways between substance use and depressogenic symptoms among maltreated youth. The present study also examined the role of parental alcohol use problems in the self-medication and the impaired-functioning hypotheses.

CHAPTER 2

LITERATURE REVIEW

Theoretical Background: Developmental Psychopathology

The current study is guided by the developmental psychopathology perspective (Cicchetti & Rogosch, 2002), which offers a comprehensive and integrative theoretical perspective for conceptualizing and understanding the emergence of psychopathology as a developmental paradigm. Drawing from the organizational perspective on child development, the developmental psychopathology theory assumes hierarchical organization of development including the initiation and continuity of psychopathology during adolescence and subsequent developmental stages. As a "macroparadigm" (Achenbach, 1990), developmental psychopathology does not espouse a singular theory that would account for all developmental phenomena (Cicchetti, 1993). Instead, it integrates knowledge across multiple levels of analysis and domains that study the origins and course of individual patterns of behavioral maladaptation (Rutter & Sroufe, 2000). According to developmental psychopathology perspective, attention should be paid to identifying adolescents who are at risk for subsequent adult disorders even though they are not currently manifesting disorders because adolescence is an important developmental period that sets the stage for the transition into adulthood roles including adjustment and development of adult disorders (Cicchetti & Rogosch, 2002).

The developmental psychopathology perspective emphasizes the essence to view adolescence in lifespan perspective (Cicchetti & Rogosch, 2002). Specifically, chronological age and developmental stages should be taken into consideration while examining the development

of risky behaviors and psychopathology during adolescence. For example, even though alcohol use and depressive symptoms are often concurrent among adolescents with a history of being maltreated, it still remains unclear whether youth alcohol use promotes depressive symptoms, or youth depressive symptoms function as an etiology of alcohol use. It is possible that, during different developmental stages, alcohol use and depressive symptoms function in different manners, leading to the directions of the causal associations between alcohol use and depressive symptoms change over time (Johnson et al., 2013). Therefore, the present study utilized a developmental psychopathology perspective and incorporated the effect of developmental timing on the nature and direction of the associations between alcohol use and depressive symptoms among maltreated adolescents.

From a developmental psychopathology perspective, risk factors do not function in a static manner (Cicchetti & Nurcombe, 2011). Transactions occur among different individual internal domains, as well as between individuals and external environments. First, risk factors can interact and transact with each other in a bidirectional manner (Cicchetti & Rogosch, 2002). Thus, it is critical to examine both current risk processes, as well as the history of dynamic transactions between various risk factors and their impact on the individual course of development over time (Cicchetti, 1993). Therefore, the current study examined the dynamic interaction of risks processes (i.e., alcohol use and depressive symptoms) in a bidirectional way over time during adolescence. Second, developmental psychopathologists also emphasize the importance of contextual influences on adolescents' development of psychopathology. According to Bronfenbrenner's ecological theory (Bronfenbrenner, 1979), situational and interpersonal influences operate at the microsystem level and play a vital importance in psychosocial development. Thus, the present study also examined the developmental etiology of

adolescents' depressive symptoms and alcohol use in the context of parental alcohol use problems, which has been documented to highly co-occur with child maltreatment (Manly et al., 2013).

Child Maltreatment Prevalence and Operationalization

Child maltreatment is a prevalent major public health concern in the United States. The US Department of Health and Human Services reports that approximately 3.4 million child abuse and neglect cases were filed in the United States in 2015. Specifically, in the year 2015, 683,000 children were maltreated, consisting a rate of 9.2 victims per 1,000 children in the population. These rates have not been subsiding at the national level and bear a significant burden on welfare services as well as the judicial system. For example, in the year of 2015, the National Children's Advocacy Centers (NCAC) served more than 233,532 children who were maltreated, providing victim legal and welfare advocacy and support to these children and their families and thus led to more than 5 million expenditures. Similarly, an estimated of 3.4 million children were under investigation or receiving alternative responses from child protective services agencies, whereas approximately 2.3 million children received prevention services. Lastly, child maltreatment is a major risk factor for child morbidity. In the year 2015, approximately 1,670 children died from child maltreatment in the United States.

Child maltreatment is a multidimensional construct that consists of multiple types of adverse parenting (Manly, 2005). In the empirical literature, child maltreatment is often classified into four types: physical abuse, sexual abuse, emotional abuse, and neglect. Physical abuse refers to the infliction of body injury by non-accidental means which caused or could have caused physical injury to a child, including hitting, beating, kicking, shaking, biting, strangling, scalding, burning, poisoning, and suffocating. Most physical abuse of children in the home is

committed by parents for the purpose of punishment (Friedman et al., 2011). Sexual abuse is defined as sexual or attempted sexual contact between a child and an adult for the purpose of the adult's sexual gratification or financial gain, including contacts for sexual purposes, molestation, statutory rape, prostitution, pornography, exposure, incest, or other sexually exploitative activities (U.S. Department of Health & Human Services, 2017). Emotional abuse involves persistent and extreme dissatisfying a child's basic emotional needs and frequently occurs as verbal abuse or excessive demands on a child's performance (U.S. Department of Health & Human Services, 2017).

Child neglect includes both lack of supervision (i.e., failing to ensure that a child is engaged in safe activities, inadequate supervision) and failure to provide minimum care to a child (i.e., food, clothing, medical care, shelter(Cicchetti & Toth, 2005; English, Thompson, Graham, & Briggs, 2005). Child neglect is the most common subtype of maltreatment in the U.S. In 2015, 75.3% of the victims were reported to CPS for having experienced child neglect. In contrast to physical, sexual and emotional abuse in which overt acts are *committed* against children, children's needs for development are mostly often *omitted* in circumstances of neglect (Mennen, Kim, Sang, & Trickett, 2010). Although any of the subtypes of child maltreatment can be found separately, they often co-occur, i.e., a phenomenon that is called poly-victimization in the child maltreatment literature.

Detecting and operationalizing child maltreatment is complicated by a range of legally sensitive issues. Detection is challenging due to the illegality of child maltreatment perpetration. Perpetrators and victims of child maltreatment are often deterred of reporting because of concern regrading legal prosecution. In addition, families and victims are often discouraged from reporting because of the associated negative stigma (Manly, 2005). There are three main

methodological strategies to identify and assess maltreatment: self-report of either victim or perpetrator, observational paradigms, or utilization of official child protective service (CPS) records (Cicchetti & Toth, 2005). Each of these methods has methodological advantages and disadvantages. Self-report is advantageous in its detection sensitivity, however also has biases related to the willingness of the victim or perpetrator to report honestly. The observational paradigm may be limited by not fully assessing the maltreatment experiences. The CPS report is limited by the propensity to miss out hidden unreported cases but is regarded as the most valid and accurate strategy to evaluate child maltreatment incidences (Cicchetti & Toth, 2005)

Alcohol Use and Depressive Symptomatology Among Maltreated Youth

Alcohol is among the most commonly used substances by adolescents in the U.S. (Johnston, 2017). According to the Monitoring the Future 2016 report (Johnston, 2017), six out of ten (61%) adolescents have consumed alcohol by the 12th grade, and 23% have done so by the 8th grade. In addition, nearly a half (46%) of 12th grade students and 9% of 8th grade students have reported being drunk at least once during their lifetime. Adolescent alcohol use is a serious public health problem as it have been documented to be associated with adolescents' deficits in neurocognitive functioning (Brown, Tapert, Granholm, & Delis, 2000), academic underperformance (Aertgeerts & Buntinx, 2002), physical and mental health problems (Armstrong & Costello, 2002; Oesterle et al., 2004), and delinquency behaviors (Barnes, Welte, & Hoffman, 2002), and thus extracts a high cost in health care, educational failure, mental health services, drug and alcohol treatment, and juvenile crime (Hawkins, Catalano, & Miller, 1992).

Depressive symptomology is also a significant risk factor impeding adolescents' normal development of social, cognitive and psychological competencies and contributes to physical health problems (Wickrama & Wickrama, 2010). Adolescents with depressive symptoms that do

not meet the diagnostic criteria for major depression disorder (MDD) have been documented to be at a higher risk of developing MDD subsequently (Aalto-Setälä, Marttunen, Tuulio-Henriksson, Poikolainen, & Lönnqvist, 2002) which accounts for great mortality, morbidity, and financial costs (Saluja et al., 2004). In addition, depressive symptoms during adolescence are also linked to dysfunctional social and family relationships (Prinstein, Borelli, Cheah, Simon, & Aikins, 2005), difficulties in social status attainment (Wickrama, Conger, Lorenz, & Jung, 2008), lower levels of self-control and self-esteem (Simons, Simons, & Wallace, 2004), and drift towards risky lifestyles characterized by substance use and risky sexual behaviors (Umberson, Liu, & Reczek, 2008).

Alcohol use frequency and depressive symptomology are significantly comorbid during adolescence (Marmorstein, 2009; Poulin et al., 2005) and adulthood (Conner, Pinquart, & Gamble, 2009). Furthermore, adolescents who were maltreated during childhood are at significant risk for the development of early alcohol use behaviors (Shin et al., 2009), and depressive symptoms (Kim & Cicchetti, 2006) during adolescence. According to a developmental psychopathology perspective (Cicchetti & Toth, 2005), abusive and neglectful parenting hinders children's attainment of life-course-expected developmental tasks which can lead to risky behaviors and psychopathology. For example, youth with child maltreatment endure chronic stress which can result in a disruption in the consolidation of self-concept systems (Harter, 1998). Lower self-concepts such as low self-esteem are associated with increased level of depressive symptomatology (Oshri, Carlson, Kwon, Zeichner, & Wickrama, 2016). Such developmental deviations may cascade into future maladaptive behaviors such as precocious alcohol use (Brems et al., 2004), and psychopathology such as depressive affect (Kim & Cicchetti, 2006; Oshri et al., 2013). Therefore, given that adverse parenting poses a significant

threat to the development of self concepts in childhood and adolescence, the comorbidity between alcohol use and depressive symptoms has been conceptualized as a dual ensuing developmental process that is preceded by chronic stress induced by child maltreatment.

Although the connections between child maltreatment, alcohol use, and depressive symptoms among youth have been established empirically, less is known about the temporal precedence and the developmental mechanisms that underlie these associations during adolescence (Johnson et al., 2013). Two prevailing and competing hypotheses in the literature have been used to explain the direction of associations between alcohol use and depressive symptoms during adolescence. The self-medication hypothesis suggests that youth with depressive symptoms utilize alcohol in order to soothe their negative affect (Colder, 2001; Khantzian, 1987). In the context of child maltreatment, youth with depressive symptoms may use alcohol as a means to alleviate the emotional pain emanating from their adverse rearing experiences, thus placing these youth at a higher risk for the development of alcohol use problems (Graham, Massak, Demers, & Rehm, 2007). Indeed, studies suggest that youth who engage in drinking behaviors and substance misuse after experiencing negative affect may do so as a coping strategy to manage distressing emotions (Kuntsche, Knibbe, Gmel, & Engels, 2005). Therefore, in support of the self-medication hypothesis, depressive symptoms during adolescence can subserve the increase in alcohol use following child maltreatment (Grant, Stewart, & Mohr, 2009; Young-Wolff, Kendler, Sintov, & Prescott, 2009).

Alternately, a growing number of studies support the impaired-functioning hypothesis, which states that alcohol use exacerbates the risk for later depressive symptoms (Brook et al., 2002; Delva, Grogan-Kaylor, Steinhoff, Shin, & Siefert, 2007; Dennhardt & Murphy, 2011). Based on the impaired-functioning hypothesis, excessive consumption of alcohol is associated

with the development of depressotypic cognitive organization during adolescence (Stice et al., 2004) and young adulthood (Fergusson, Boden, & Horwood, 2009; Rohde, Lewinsohn, Kahler, Seeley, & Brown, 2001). Recent studies have shown that alcohol use is associated with neurocognitive impairments in emotional regions (Ward, Lallemand, & De Witte, 2009), mood swings (Svikis et al., 2006) and difficulties with regulating emotions (Fox, Hong, & Sinha, 2008). Thus, based on the impaired-functioning hypothesis, consumption of alcohol during adolescence is expected to result in a disruption of emotional functioning leading to symptoms of affect dysregulation—such as depressive symptoms—during adolescence.

Despite the distinct mechanisms proposed by each of these two theories and the contrasting evidence, the self-medication and the impaired-functioning hypotheses are not mutually exclusive. For example, a recent study that examined the effect of adverse childhood experiences on alcohol use and depressive symptoms reported support for both the impaired-functioning and self-medication hypotheses (Johnson et al., 2013). Based on the developmental psychopathology perspective, risk factors interact and transact with each other in a bidirectional manner (Cicchetti & Rogosch, 2002). Several longitudinal studies have reported a transactional relationship wherein alcohol use and depressive symptoms exacerbated the other prospectively (Blume et al., 2000; Gilman & Abraham, 2001; Locke & Newcomb, 2001; Marmorstein, 2009). To reconcile the inconsistencies in the literature about the associations between depressive symptoms and alcohol use among maltreated adolescents, a third integrative hypothesis of bidirectional associations between alcohol use and depressive symptomology is necessary.

Furthermore, based on a developmental psychopathology theoretical formulation, developmental timing is critical for understanding the mechanisms of psychopathology (Cicchetti & Toth, 2005). However, no research to date has examined the self-medication and the

impaired-functioning hypotheses accounting for developmental timing among maltreated adolescents. Youth during early adolescence are particularly vulnerable to the impact of alcohol use. For example, in a prospective study, Odgers et al. (2008) suggested that youth's exposure to alcohol and illicit drugs during early adolescence is a causal factor to subsequent poor mental health and high levels of risky behaviors. Early adolescence is a period when brain regions are still under maturation, thus are more vulnerable to the harmful impact of alcohol use (Bava & Tapert, 2010). Therefore, the connection between alcohol use and impairment of emotion-regulating brain regions might be more salient in early adolescence, during which important neurocognitive changes are occurring. In contrast, compared to early adolescence, alcoholic beverages are more available for youth during middle- to late-adolescence when alcohol use is often considered as normative behavior (Litt & Stock, 2011), thereby increasing youths' likelihood of self-medicating negative affect by drinking alcohol. As a result, it is important to consider the impact of developmental timing while examining the discussed hypotheses regarding the associations between youth alcohol use and depressive symptoms. It is expected that, in early adolescence, alcohol use will precede depressive symptomology, supporting the impaired-functioning hypothesis; whereas during middle- to late-adolescence, depressive symptoms will precede alcohol use, thus supporting the self-medication hypothesis. Therefore, a third hypothesis, called the bidirectional hypothesis, is proposed in the current study to reconcile the literature inconsistencies accounting for the importance of developmental timing. This hypothesis suggests that alcohol use and depressive symptoms can reciprocally influence the development of each other over time. Thus, in addition to testing the self-medication hypothesis and the impaired-functioning hypothesis, the current study also tested the bidirectional hypothesis in the context of developmental timing.

The Role of Parental Alcohol Use Problems

Parental alcohol use problems and risk for youths. Parental alcohol use problems have been extensively documented as a risk factor for adverse family environments, characterized by less cohesive, expressive, and supportive parenting, and more interparental conflicts than families without alcohol abusing parents (Rothenberg, Hussong, & Chassin, 2016). In addition, parental alcohol use problems often negatively affect the quality of marital relationships, thus increasing rates of separation and divorce and potentially leading to family violence (Marshal, 2003). As a result, children of alcohol abusers are reported to be more vulnerable to experienced stress in the family. Empirical findings have supported this proposition. Children of alcohol abusers have been found to be more likely to experience repeated and more severe stressors in the family environment than children of non-alcoholics (Hussong et al., 2008).

Not only does parental alcohol abuse predict future stressors, but parental alcohol use problems are also highly concurrent with child maltreatment (Manly et al., 2013). According to the National Child Abuse and Neglect Data System (NCANDS), in 2014, approximately 9.2% of maltreatment victims, as contrasted with 3.8% of non-victims, were reported to have alcohol-abusing caregivers. Furthermore, the National Center on Addiction and Substance Abuse (NCASA) reports that children of parents who abuse alcohol or other drugs are three times more likely to be neglected than children whose parents do not abuse alcohol and drugs. National child welfare system data also indicates that parental substance use is confirmed or suspected in 66% of all substantiated cases in Child Protective Services (CPS) records (Besinger, Garland, Litrownik, & Landsverk, 1999).

Parental alcohol use problems and youth alcohol use and depressive symptoms. Parental alcohol use problems are a profound risk factor for early onset of alcohol use (Donovan, 2004;

Lieb et al., 2002), increased alcohol use intensity and frequency (Biederman, Faraone, Monuteaux, & Feighner, 2000; Van Zundert, Van Der Vorst, Vermulst, & Engels, 2006), and an accelerated trajectory from alcohol initiation to dependence (Hussong, Bauer, & Chassin, 2008) during adolescence and young adulthood. Parental alcohol use problems also have been documented as a significant risk factor for youth depressive symptoms (Brook et al., 2002; Hussong et al., 2008). However, the mechanisms underlying the associations between parental alcohol use problems, youth alcohol use, and depressive symptoms remain unclear. It is possible that these associations can be explained using the self-medication hypothesis. Specifically, youth negative affect is hypothesized to be induced by the negative home environment caused by the intoxicated parents, leading youth to self-medicate with alcohol use (Chassin et al., 1996). In contrast, the impaired-functioning hypothesis can also indirectly explain the effects of parental alcohol use problems on youth risk for alcohol use and depressive symptoms. Parents with alcohol use problems tend to be less strict about adolescents' alcohol use, leading to youth early initiation of alcohol use (Van Der Vorst, Engels, Meeus, & Deković, 2006). As the early age of onset of alcohol use is linked to brain vulnerability and impaired emotional regulation, it is also plausible that early alcohol use will lead to a higher risk of depressive symptoms in adolescence.

Current Study

The present study had two main goals. The first goal was to reconcile the theoretical and empirically inconsistencies about the associations between youth alcohol use and depressive symptoms and to test the following three hypotheses regarding the associations between alcohol use and depressive symptoms among maltreated youth while accounting for developmental timing: the self-medication, the impaired-functioning, and the bidirectional hypotheses. The

second goal was to test the role of parental alcohol use problems in the etiology of depressive symptoms and alcohol use among maltreated youth.

CHAPTER 3

METHODS

Participants

Data for the present study were obtained from the National Survey of Child and Adolescent Well-Being (NSCAW-II). The NSCAW-II is a nationally representative dataset of children and families who were involved with CPS. This longitudinal dataset consists of information from 5,872 children ranging in age from birth to 17.5 years at the time of sampling in the NSCAW-II cohort. Children were investigated from February 2008 to January 2011 across 30 states in the U.S. Participants were selected from 81 counties across the U.S. through 81 Primary Sampling Units or the geographic area inhabited by the population served by a single Child Protective Services agency. Infants, sexual abuse cases, and open cases were oversampled to allow for adequate analysis of each group. Of the children in the sample, 657 youth were aged 11 to 14 at time-point I (52.7% female) and thus were selected for the current analysis. A majority of children were Caucasian (51.4%), followed by African-American (30.7%), Native American (13.0%), and Other (4.9%). Data were obtained from three time-points with each 18-months apart (time-point I: M_{age} = 12.50, SD_{age} = 1.13; time-point II: M_{age} = 13.70, SD_{age} = 1.19; time-point III: M_{age} = 15.60, SD_{age} = 1.26), and were from multiple reporters (i.e., child and caregiver).

Measures

Youth self-reports.

***Depressive symptoms*.** Depressive symptoms were assessed via 27 items from the Children's Depression Inventory (Kovacs, 1992) for all three time-points with possible responses ranging

from 0 (absence of symptom) to 2 (definite symptom). Youth self-reported their depressive symptoms for the previous two weeks. The depressive symptoms were indicated by five factors: negative mood, interpersonal problems, ineffectiveness, anhedonia, and negative self-esteem. Negative mood included 6 items reflecting feeling sad, feeling like crying, worrying, being bothered or upset by things, and being unable to make up one's mind; interpersonal problems included 4 items indicating that youth have difficulties interacting with people, including trouble getting along with people, social avoidance, and social isolation; ineffectiveness included 4 items showing negative evaluation of one's abilities and school performance; anhedonia included 8 items reflecting impaired ability to experience pleasure; and negative self-esteem included 5 items showing that youth have low self-esteem, self-dislike and feelings of being unloved. Items comprising each of the five indicators were combined, and a sum score was computed with higher scores reflecting greater depressive symptoms (Kovacs, 1992). The reliability of this scale was strong (α_{T1} = .84; α_{T2} = .80; α_{T3} = .79).

Alcohol use. The Youth Risk Behavior Survey (YRBS; Eaton et al., 2006) was used to measure youth alcohol use frequency in the current study. Three self-reported items were used, i.e., "During the past 30 days, how many days have you had at least 1 drink of alcohol?"; "During the past 30 days, how many days have you had at least 5 drinks of alcohol within a couple of hours?"; and "During the past 30 days, how many days have you had at least 1 drink of alcohol on school property?" Data were obtained for all three time-points. The reliability of the three items at every time-point was good (α_{w1} = .89; α_{w2} = .80; α_{w3} = .75).

Parent reports.

Parental alcohol use problems. Data on parental alcohol use problems were obtained by parental report at time-point I. Parents reported on their alcohol use problems through 10 items

on the Alcohol Use Disorders Identification Test (AUDIT; Barbor, Higgins-Biddle, Saunders, & Montero, 2001). The AUDIT was developed by the World Health Organization (WHO) as a simple method of screening for excessive drinking. A total score of 5 or higher has been suggested as a cut point to detect the harmful use of alcohol (Reinert & Allen, 2007; Rumpf, Hapke, Meyer, & John, 2002). The reliability of this scale was acceptable (α = .76).

Youth demographics and controls. Parents reported youth gender, race, and the families' socioeconomic status (SES) at the first time-point. Families' SES was assessed by the question "Is anyone in the household currently receiving child support?"

Analytic Plan

Data were modeled using Mplus 7.4 (Muthén & Muthén, 2012). Maximum likelihood estimation with robust standard errors (Yuan & Bentler, 2000) was used as a model estimator to remedy for data non-normality issues. National-level sampling weights (which accounted for attrition and sample stratification) were used and intra-class correlations were controlled in all analyses. Descriptive statistics showed that the percentage of missing data varied by indicator and time, ranging from 0% to 38.7%, with an average of 20.52% over all indicators and times. Little's Missing Completely at Random (MCAR) test conducted by SPSS suggested that the missing data in the current sample were completely at random (χ^2 (824) = 850.629, p = .253). Thus, missing data were estimated using full-informative maximum likelihood (FIML) algorithm (Rubin & Little, 2002) in Mplus, which allowed individuals to be included in the analysis even if the data were missing in the sample, and yielded more efficient and less biased parameter estimates than traditional methods handling missing data such as case-wise deletion.

To assess the model fits of all models in the current study, a variety of global fit indices were used, including indices of absolute fit, indices of relative fit, and indices of fit with a penalty

function for lack of parsimony. These indices included the chi-square test of model fit, the root mean square error of approximation (RMSEA), the comparative fit index (CFI), the Tucker-Lewis index (TLI), and the standardized root mean square residual (SRMR) (Hu & Bentler, 1999). The stepwise testing of improvement of fit in the cross-lagged models was based on additional absolute model fit indices: the Akaike information criteria (AIC) and the sample-size adjusted Bayesian information criteria (the adjusted BIC), of which lower values indicate a better fit model. In addition to the global fit indices, more focused tests also included modification indices (< 3.84).

Confirmatory factor analysis (CFA) was used to test a measurement model of youth depressive symptoms and alcohol use, respectively. As recommended by Cole and Maxwell (2003), every latent factor was allowed to covariate with other latent factors in both measurement models. The measurement invariance across gender was also tested. Cross-lagged analyses within a structural equation modeling (SEM) framework were used to test the three hypotheses regarding the direction of associations between depressive symptoms and alcohol use among youth. Nested models from Model 1 to Model 4 were evaluated by gradually freeing parameters based on different hypotheses: Model 1 assessed the stability of youth depressive symptoms and alcohol use over time; Model 2 examined the self-medication hypothesis; Model 3 tested the impaired-functioning hypothesis; Model 4 assessed the bidirectional hypothesis. To evaluate the fit improvement of the nested models, each of the more parsimonious models was compared with the next more complex model. A scaled chi-square difference test for nested models was used to evaluate the relative fit (Satorra, 2000). Mediation models within a SEM framework were used to test the role of parental alcohol use problems in the etiology of depressive symptoms and alcohol use among youth. In the mediation models, indirect links were

assessed with the product-of-coefficients approach ($\alpha*\beta$) (Fritz & MacKinnon, 2007), for which the significance and confidence intervals were determined using bias-corrected bootstrapping (BC Bootstrapping) which modified the percentile-based confidence intervals with a bias correction (Efron & Tibshirani, 1986).

CHAPTER 4

RESULTS

Descriptive Statistics and Correlations

Descriptive statistics and bivariate correlations of the variables in the present study are presented in Table 1. Among the 657 youth, about one in third (29.2%) were investigated for physical abuse, one in seven (13.4%) were investigated for sexual abuse, one in ten (10.0%) were investigated for emotional abuse, and 39.0% were investigated for neglect (12.2% failure to provide minimum care and 26.8% lack of supervision). The rest were investigated for other types of maltreatment (e.g., moral maltreatment, education maltreatment, etc.). Overall, youth showed decreasing depressive symptoms (T1 subscale means ranged 1.00-2.96, T2 subscale means ranged .83-2.61, T3 subscale means ranged .67-2.62) and increasing alcohol use (both increasing alcohol use frequency and binge drinking frequency, but decreasing alcohol use on school properties) over the three time-points. At the first time-point, about one in ten (10.3%) parents reported an AUDIT total score equal to or higher than 5, suggesting that they were at risk of harmful alcohol use problems (Reinert & Allen, 2007; Rumpf et al., 2002)

Variables were correlated in the expected directions. Generally, youth depressive symptoms showed significant positive correlations with youth alcohol use. In addition, parental alcohol use problems showed positive correlations with youth depressive symptoms and youth alcohol use.

Measurement Model

Confirmatory factor analysis (CFA) was used to assess the factor structure of youth depressive symptoms and alcohol use (Brown, 2015). Figure 1 presents the measurement model

of youth depressive symptoms in which the latent factors consisting of five indicators (i.e., negative mood, interpersonal problems, ineffectiveness, anhedonia, and negative self-esteem) was assessed at all three time-points. Results (Table 2) showed that all factor loadings were moderate to large (λ>.40) (Brown, 2015) and significant ($p < .001$). The overall measurement model fit was good: χ^2 (82) = 134.826, $p < .001$; RMSEA = .031; SRMR = .054; CFI = .951; TLI = .937 (Hu & Bentler, 1999). Both metric invariance (χ^2 (12) = 2.810, $p = .997$) and scalar invariance (χ^2 (24) = 33.57, $p = .093$) across gender were tested and confirmed (Byrne, 2012).

Figure 2 presents the measurement model of youth alcohol use. The latent factors were constructed of three indicators (i.e. binge drinking, alcohol use frequency, alcohol use at school properties during the past 30 days) at all three time-points. Findings (Table 3) suggested that all factor loadings were moderate to large (λ>.40; Brown, 2015) and significant ($p < .01$). The overall measurement model fit was good: χ^2 (22) = 40.898, $p = .009$; RMSEA = .037; SRMR = .056; CFI = .940; TLI = .902. In both measurement models, the measurement invariance across gender groups was examined. Both metric invariance (χ^2 (6) = 5.210, $p = .517$) and scalar invariance (χ^2 (12) = 6.603, $p = .883$) across gender were tested and confirmed (Byrne, 2012).

Cross-Lagged Analyses: Alcohol and Depressive Symptoms

Following Maxwell and Cole's steps, cross-lagged analyses were used to test the developmental pathways between depressive symptoms and alcohol use over the three time–points. Youth gender, race, and SES were used as covariates. Nested models were gradually modified from model 1 to 4 by freeing parameters based on theories. Table 4 shows the paths freed in each of the successive nested models. In Model 1, the stability of youth depressive symptoms and youth alcohol use was examined. In Model 2, paths were added from depressive symptoms to alcohol use (i.e., the self-medication hypothesis). In Model 3, paths were added

from alcohol use to depressive symptoms (i.e., the impaired-functioning hypothesis). In Model 4, all paths were freed in order to test the bidirectional hypothesis. Relative fit was evaluated with a scaled chi-square difference test for nested models (Satorra, 2000). Each more parsimonious model was compared with the next more complex model (Table 4). Significant chi-square difference indicated a model fit improvement. The AIC, adjusted BIC, and chi-square difference tests confirmed statistically significant improvement of model fits from Model 1 to Model 3 ($\Delta \chi^2(2) = 7.322$) and from Model 2 to Model 4 ($\chi^2(2) = 8.034$). Model fit also improved from Model 1 to Model 2 ($\chi^2(2) = 4.140$) and from Model 3 to Model 4 ($\chi^2(2) = 4.852$), but these improvements were not statistically significant (Table 5).

Model 1: Youth depressive symptoms and youth alcohol use stability model. The analysis supported substantial ranked-ordered stability of youth depressive symptoms across three time-points, with time-point I predicting time-point II ($\beta = .687$, 95% *CI* [.142, .271]) and time-point II predicting time-point III ($\beta = .712$, 95% *CI* [.585, 892]). Youth alcohol use also presented stability across time-point I and time-point II ($\beta = .371$, 95% *CI* [.159, 501]), but the stability across time-point II and time-point III was marginal ($\beta = .374$, 95% *CI* [-.024, 1.225]). Males reported higher levels of alcohol use at time-point III ($\beta = .119$, 95% *CI* [.030, .250]). Model fit was acceptable: $\chi^2 (295) = 536.499$, $p < .001$; RMSEA = .036; SRMR = .081; CFI = .887; TLI = .867 (Hu & Bentler, 1999). At each time-point, youth depressive symptoms and alcohol use were allowed to covary. Youth depressive symptoms and alcohol use were significantly positively covaried at time-point I ($\beta = .157$, 95% *CI* [.026, 260]) and time-point III ($\beta = .254$, 95% *CI* [.003, .128]), but only marginal significant covariance was found at time-point II ($\beta = .146$, 95% *CI* [-.001, .049]).

Model 2: Self-medication hypothesis model. The goal of the second model was to examine the self-medication hypothesis across three time-points. Paths from earlier youth depressive symptoms to later youth alcohol use were freed. There was no significant association between youth depressive symptoms at time-point I and alcohol use in time-point II (β = .048, 95% *CI* [-.016, .031]). However, findings suggested that higher levels of depressive symptoms at time-point II were marginally significantly related to higher levels of alcohol use at time-point III (β = .158, 95% *CI* [-.005 .185]). Model fit was acceptable: χ^2 (293) = 532.359, $p < .001$; RMSEA = .036; SRMR = .077; CFI = .888; TLI = .867 (Hu & Bentler, 1999).

Model 3: Impaired-functioning hypothesis model. The goal of the third model was to examine the impaired-functioning hypothesis across three time-points. Paths from earlier youth alcohol use to later youth depressive symptoms were freed. Findings suggested that youth alcohol use at time-point I was positively associated with youth alcohol use at time-point II significantly (β = .115, 95% *CI* [.054, .345]). In addition, youth alcohol use at time-point II was negatively associated with youth depressive symptoms at time-point III with a marginal significance (β = -.126, 95% *CI* [-.530, .029]). Model fit was acceptable: χ^2 (293) = 529.177, $p < .001$; RMSEA = .036; SRMR = .079; CFI = .890; TLI = .869 (Hu & Bentler, 1999).

Model 4: Bidirectional hypothesis model. In model 4, paths indicating both the self-medication hypothesis and the impaired-functioning hypothesis were freed. In this complete model, males reported higher levels of alcohol use at time-point III (β = .101, 95% *CI* [.003, 160]). Youth depressive symptoms showed rank-ordered stability across three time-points, with time-point I predicting time-point II (β = .670, 95% *CI* [.145, .259]) and time-point II predicting time-point III (β = .749, 95% *CI* [.613, 943]). Youth alcohol use also presented stability across time-point I and time-point II (β = .370, 95% *CI* [.150, .507]) and across time-point II to time-

point III (β = .317, 95% *CI* [.041, .666]). For the self-medication hypothesis, even though no significant association between youth depressive symptoms at time-point I and alcohol use in time-point II was presented (β = .052, 95% *CI* [-.015, .031]), findings suggested that higher levels of depressive symptoms at time-point II was significantly related to higher levels of alcohol use at time-point III (β = .172, 95% *CI* [.001, .196]), lending support to the self-medication hypothesis. For the impaired-functioning hypothesis, results showed that youth's higher levels of alcohol use at time-point I significantly contributed to higher levels of depressive symptoms at time-point II (β = .120, 95% *CI* [.058, .361]), supporting the impaired-functioning hypothesis. However, the association between alcohol use at time-point II and depressive symptoms at time-point III was negative but inconclusive (β = -.129, 95% *CI* [-.541, .015]). Model fit was good: χ^2 (291) = 524.325, p < .001; RMSEA = .036; SRMR = .074; CFI = .891; TLI = .870 (Hu & Bentler, 1999).

Parental Alcohol Use as a Predictor

Figure 4 presents the structural equation model evaluating the self-medication hypothesis with parental alcohol use problems as a risk factor. Gender, race, and SES of youth were adjusted for by using them as covariates. Model fit indices were good: χ^2 (151) = 261.192, p < .001; RMSEA = .034; SRMR = .068; CFI = .914; TLI = .896. Males reported higher levels of alcohol use at time-point III (β = .138, 95% *CI* [.017, .297]). Higher levels of parental alcohol use problems at time-point I contributed to higher levels of depressive symptoms at time-point II (β = .147, 95% *CI* [.012, .053]) after controlling youth depressive symptoms at time-point I (β = .663, 95% *CI* [.142, .259]). Higher levels of depressive symptoms at time-point II significantly contributed to higher levels of youth alcohol use at time-point III (β = .172, 95% *CI* [.014, .268]), after controlling parental alcohol use problems at time-point I (β = .081, 95% *CI* [-.020, .049])

and youth alcohol use at time-point II (β = .381, 95% *CI* [-.223, 1.161]). The results (Table 6) suggested that with parental alcohol use problems as a predictor, the self-medication hypothesis was supported. Youth depressive symptoms mediated the association between parental alcohol use problems and youth alcohol use ($\alpha^*\beta$ = .025, 95%*CI* [.001, .014]).

Figure 5 presents the structural equation model evaluating the impaired-functioning hypothesis with parental alcohol use problems as a predictive risk factor. Youth gender, race, and SES were included as covariates. Model fit was good: $\chi^2(151) = 261.112$, $p < .001$; RMSEA = .034; SRMR = .072; CFI = .909; TLI = .889. No significant differences were found across gender, race, and SES. Parental alcohol use problems at time-point I were not significantly associated with youth alcohol use at time-point II (β = .093, 95% *CI* [-.002, .019]) after controlling for youth alcohol use at time-point I (β = .279, 95% *CI* [-.098, .502]). In addition, findings suggested that the association between youth alcohol use at time-point II and youth depressive symptoms at time-point III was inconclusive (β = -.083, 95% *CI* [-.527, .107]) after controlling parental alcohol use problems at time-point I (β = .087, 95% *CI* [-.017, .058]) and depressive symptoms at time-point II (β = .709, 95% *CI* [.591, .915]). These results regarding parental alcohol use problems as a risk factor (Table 6) suggested that the impaired-functioning hypothesis was not supported. Youth alcohol use did not mediate the association between parental alcohol use problems and youth depressive symptoms ($\alpha^*\beta$ = -.008, 95%*CI* [-.016, .000]).

Table 1.

Descriptive Statistics and Bivariate Correlations among Study Variables

Variables	1	2	3	4	5	6	7	8	9	10	11	12	13	14	15	16	17	18	19	20	21	22	23	24	25	26	27	28
1. Gender	--																											
2. Race	-.65	--																										
3. SES	.03	-.13**	--																									
4. Neg. Mood—T-I	.19**	-.04	.05	--																								
5. Inter. Prob.—T-I	-.01	-.02	.05	.50**	--																							
6. Ineffectiveness—T-I	.00	-.12**	.11**	.53**	.50**	--																						
7. Anhedonia—T-I	.09*	-.10*	.03	.66**	.51**	.56**	--																					
8. Neg. SE—T-I	.14**	-.07	.06	.65**	.42**	.50**	.59**	--																				
9. Neg. Mood—T-II	.19**	.01	.08	.42**	.27**	.34**	.35**	.32**	--																			
10. Inter. Prob.—T-II	.00	-.02	-.01	.25**	.38**	.31**	.27**	.17**	.48**	--																		
11. Ineffectiveness—T-II	.01	-.03	.05	.23**	.21**	.41**	.25**	.25**	.37**	.46**	--																	
12. Anhedonia—T-II	.09*	-.05	.07	.37**	.31**	.35**	.47**	.36**	.61**	.49**	.45**	--																
13. Neg. SE—T-II	.15**	-.05	.05	.40**	.25**	.37**	.35**	.50**	.56**	.38**	.43**	.54**	--															
14. Neg. Mood—T-III	.26**	-.03	.11*	.50**	.30**	.26**	.38**	.35**	.53**	.26**	.28**	.41**	.42**	--														
15. Inter. Prob.—T-III	.07	.11*	.11*	.23**	.25**	.13**	.25**	.15**	.31**	.26**	.21**	.32**	.19**	.46**	--													
16. Ineffectiveness—T-III	.03	-.10*	.08	.28**	.30**	.41**	.24**	.24**	.28**	.26**	.37**	.29**	.32**	.37**	.37**	--												
17. Anhedonia—T-III	.20**	-.05	.13*	.42**	.32**	.28**	.45**	.35**	.40**	.27**	.32**	.49**	.37**	.69**	.41**	.38**	--											
18. Neg. SE—T-III	.18**	-.11*	.06	.39**	.35**	.35**	.38**	.45**	.35**	.24**	.26**	.36**	.58**	.61**	.31**	.40**	.54**	--										
19. ALCM—T-I	.12**	-.01	.05	.17**	.14**	.18**	.18**	.19**	.15**	.11*	.13**	.16**	.14**	.10	.10	.19**	.14**	.11*	--									
20. ALCB—T-I	.07	-.01	.05	.17**	.11*	.15**	.17**	.14**	.13**	.11*	.12*	.16**	.07	.10	.12*	.12*	.12*	.09	.86**	--								
21. ALCSch—T-I	.04	-.00	.03	.18**	.09*	.10*	.14**	.18**	.07	.04	.09*	.06	.05	.13*	.21**	.18**	.12*	.18**	.64**	.69**	--							
22. ALCM—T-II	.11*	-.09	.01	.17**	.14**	.18**	.12*	.17**	.18**	.25**	.19**	.18**	.19**	.05	.03	.11*	.01	.08	.31**	.23**	.14**	--						
23. ALCB—T-II	.04	-.08	.05	.17**	.19**	.21**	.16**	.10*	.12**	.23**	.17**	.11*	.08	.04	.03	.11	.02	.06	.26**	.21**	.15**	.74**	--					
24. ALCSch—T-II	.01	.03	-.06	.01	.07	.03	.03	.01	.04	.17**	.10*	.13**	.06	.04	.05	.05	.00	.05	.08	.12*	.00	.48**	.56**	--				
25. ALCM—T-III	.09	.01	.08	.12*	.14**	.11*	.05	.07	.17**	.13*	.10	.12*	.06	.22**	.26**	.31**	.20**	.18**	.27**	.22**	.07	.07	.07	.01	--			
26. ALCB—T-III	.10*	-.00	-.00	.08	.09	.11*	.03	.02	.13*	.16**	.13*	.09	.04	.10	.19**	.29**	.13*	.09	.31**	.25**	.05	.12*	.12*	.07	.75**	--		
27. ALCSch—T-III	.02	.02	.04	.01	.00	-.05	-.02	-.01	-.01	.09	.10*	.09	.06	.06	.14**	.15**	.11*	.03	.15**	.08	.05	.07	.08	.02	.44**	.48**	--	
28. PAlc—T-I	-.06	-.13**	.05	-.02	.01	.07	-.04	-.04	.02	.01	.04	-.06	.06	.16**	.01	.08	.10	.17**	.03	.01	-.02	.03	.00	-.03	.05	-.01	-.07	--
Mean	1.53	.49	.20	2.09	1.00	1.72	2.96	1.18	1.83	.83	1.60	2.61	.87	1.78	.67	1.42	2.62	.75	.19	.13	.08	.26	.19	.06	.36	.21	.05	1.77
Standard Deviation	.50	.50	.40	2.25	1.35	1.74	2.70	1.60	1.96	1.18	1.66	2.44	1.27	2.11	.95	1.50	2.58	1.30	.62	.56	.47	.70	.71	.38	.80	.67	.26	3.15

Note: Gender was coded as 1 for female and 2 for male; Race was coded as 0 for Caucasian and 1 for minority; SES was coded as 1 for currently receiving child support and 0 as not. T-I = Time-point 1; T-II = Time-point 2; T-III = Time-point 3; Neg. Mood = Negative mood; Inter. Prob. = Interpersonal problems; Neg. SE = Negative self-esteem; ALCM = Alcohol use frequency during the past 30 days; ALCB = Binge drinking during the past 30 day; ALCSch = Alcohol use on school properties during the past 30 days; PAlc=Parental alcohol use problems. $^{**}p < .01$, $^{*}p < .05$.

Table 2.

Measurement Model of Youth Depressive Symptoms

Factor and Indicators	*B (SE)*	*λ (SE)*	R^2	95%*CI*
Depressive symptoms—T-I				
Anhedonia—T-I	1.000 (.000)	.835 (.029)	.697	[.778, .892]***
Neg. Mood—T-I	.271 (.025)	.746 (.035)	.556	[.678, .814]***
Inter. Prob.—T-I	.343 (.038)	.622 (.048)	.387	[.528, .716]***
Ineffectiveness—T-I	.544 (.056)	.677 (.055)	.459	[.569, .786]***
Neg. SE—T-I	.479 (.061)	.650 (.038)	.422	[.575, .725]***
Depressive symptoms—T-II				
Anhedonia—T-II	1.000 (.000)	.755 (.050)	.570	[.657, .853]***
Neg. Mood—T-II	.870 (.064)	.732 (.043)	.536	[.649, .816]***
Inter. Prob.—T-II	1.010 (.118)	.620 (.042)	.384	[.537, .703]***
Ineffectiveness—T-II	.761 (.079)	.617 (.038)	.380	[.542, .691]***
Neg. SE—T-II	1.209 (.203)	.623 (.055)	.378	[.515, .731]***
Depressive symptoms—T-III				
Anhedonia—T-III	1.000 (.000)	.747 (.038)	.559	[.674, .821]***
Neg. Mood—T-III	.925 (.072)	.799 (.037)	.638	[.727, .871]***
Inter. Prob.—T-III	.389 (.065)	.549 (.075)	.211	[.312, .607]***
Ineffectiveness—T-III	.546 (.093)	.497 (.074)	.247	[.352, .642]***
Neg. SE—T-III	.702 (.077)	.682 (.055)	.465	[.574, .790]***

Covariance	*B (SE)*	*β*	95%*CI*
Depressive—T-I & Depressive—T-II	.993 (.149)	.651	[.701, 1.286]***
Depressive—T-I & Depressive—T-III	.889 (.166)	.549	[.564, 1.214]***
Depressive—T-II & Depressive—T-III	.341 (.049)	.701	[.246, .436]***
Inter. Prob.—T-I & Inter. Prob.—T-II	.393 (.100)	.466	[.197, .588]***
Ineffectiveness—T-I & Ineffectiveness—T-II	.237 (.070)	.271	[.100, .374]**
Ineffectiveness—T-I & Ineffectiveness—T-III	.291 (.090)	.319	[.115, .467]**
Neg. SE —T-I & Neg. SE —T-II	.475 (.153)	.366	[.175, .775]**
Neg. SE —T-II & Neg. SE —T-III	.181 (.076)	.325	[.032, .329]*

Note: *SE* = Standard error; *CI* = Confidence interval of *λ* and *B*; T-I = Time-point 1; T-II = Time-point 2; T-III = Time-point 3; Neg. Mood = Negative mood; Inter. Prob. = Interpersonal problems; Neg. SE = Negative self-esteem; Depressive = Youth depressive Symptoms. Model fit was good: χ^2 (82) = 134.826, $p < .001$; RMSEA = .031; SRMR = .054; CFI = .951; TLI = .937. *$p < .05$, **$p < .01$, *** $p < .001$.

Table 3.

Measurement Model of Youth Alcohol Use

Factor and Indicators	*B (SE)*	*λ (SE)*	R^2	95%*CI*
Alcohol—T-I				
ALCB—T-I	1.000 (.000)	.955 (.034)	.912	[.888, 1.023]***
ALCM—T-I	.980 (.064)	.827 (.067)	.684	[.696, .958]***
ALCSch—T-I	.653 (.166)	.802 (.126)	.644	[.555, 1.050]***
Alcohol—T-II				
ALCB—T-II	1.000 (.000)	.947 (.059)	.897	[.831, 1.064]***
ALCM—T-II	1.374 (.231)	.877 (.067)	.769	[.745, 1.009]***
ALCSch—T-II	.394 (.136)	.600 (.108)	.360	[.389, .811]***
Alcohol—T-III				
ALCB—T-III	1.000 (.000)	.897 (.066)	.804	[.767, 1.026]***
ALCM—T-III	.670 (.151)	.768 (.047)	.589	[.676, .859]***
ALCSch—T-III	.152 (.065)	.472 (.137)	.222	[.203, .740]**

Covariance	*B (SE)*	*β*	95%*CI*
Alcohol —T-I & Alcohol —T-II	.042 (.026)	.265	[-.010, .094]
Alcohol —T-I & Alcohol —T-III	.077 (.042)	.347	[-.005, .159]†
Alcohol —T-Ii & Alcohol —T-III	.093 (.054)	.391	[-.013, .199]†
ALCSch—T-I & ALCSch—T-II	-.016 (.008)	-.398	[-.032, .001]*
ALCSch—T-I & ALCB—T-II	-.014 (.006)	-.529	[-.025, -.002]*

Note: *SE* = Standard error; *CI* = Confidence interval of *λ* and *B*; T-I = Time-point 1; T-II = Time-point 2; T-III = Time-point 3; Alcohol = Youth alcohol use; ALCM = Alcohol use frequency during the past 30 days; ALCB = Binge drinking during the past 30 day; ALCSch = Alcohol use in school properties during the past 30 days; PA = Parental alcohol use. Model fit was good: χ^2 (22) = 40.898, p = .009; RMSEA = .037; SRMR = .056; CFI = .940; TLI = .902. †p < .10, *p < .05, **p < .01, *** p < .001.

Table 4.
Structural Equation Models with Cross-Lagged Analysis

	Model 1 (stability model)			Model 2 (self-medication test)			Model 3 (Impaired functioning test)			Model 4 (bidirectional model)		
	B (SE)	*β*	95% *CI*	*B (SE)*	*β*	95% *CI*	*B (SE)*	*β*	95% *CI*	*B (SE)*	*β*	95% *CI*
Stability of depressive symptoms and alcohol use												
Depressive—T-I → Depressive—T-II	.207 (.033)	0.687	[.142, .271]***	.208 (.032)	0.689	[.145, .271]***	.201 (.031)	0.668	[.140, .261]***	.202 (.029)	0.670	[.145, .259]***
Depressive—T-II → Depressive—T-III	.739 (.078)	0.712	[.585, .892]***	.758 (.078)	0.723	[.606, .911]***	.760 (.086)	0.736	[.592, .927]***	.778 (.084)	0.749	[.613, .943]***
Alcohol —T-I → Alcohol —T-II	.330 (.087)	0.371	[.159, .501]***	.320 (.088)	0.362	[.148, .491]***	.340 (.090)	0.379	[.162, .517]***	.329 (.091)	0.370	[.150, .507]***
Alcohol —T-II → Alcohol —T-III	.601 (.319)	0.374	[-.024, 1.225]†	.377 (.154)	0.337	[.075, .679]*	.576 (.325)	0.362	[-.061, 1.214]†	.353 (.159)	0.317	[.041, .666]*
Self-medication hypothesis												
Depressive—T-I → Alcohol —T-II				.007 (.012)	0.048	[-.016, .031]				.008 (.012)	0.052	[-.015, .031]
Depressive—T-II → Alcohol —T-III				.090 (.048)	0.158	[-.005, .185]†				.098 (.050)	0.172	[.001, .196]*
Impaired-functioning hypothesis												
Alcohol —T-I → Depressive—T-II							.200 (.074)	0.115	[.054, .345]**	.210 (.077)	0.120	[.058, .361]**
Alcohol —T-II → Depressive—T-III							-.251 (.142)	-0.126	[-.530, .029]†	-.263 (.142)	-0.129	[-.541, .015]†
Covariance												
Depressive—T-I & Alcohol —T-I	.143 (.060)	0.157	[.026, .260]*	.140 (.061)	0.154	[.020, .261]*	.128 (.059)	0.141	[.012, .243]*	.124 (.060)	0.137	[.005, .242]*
Depressive—T-II & Alcohol—T-II	.024 (.013)	0.146	[-.001, .049]†	.023 (.012)	0.142	[-.001, .047]†	.025 (.013)	0.155	[-.001, .051]†	.024 (.012)	0.151	[.000, .048]†
Depressive—T-III & Alcohol—T-III	.065 (.032)	0.254	[.003, .128]*	.039 (.020)	0.223	[-.001, .079]†	.050 (.028)	0.199	[-.005, .105]†	.028 (.019)	0.166	[-.009, .066]
Gender → Depressive—T-II	.113 (.110)	0.080	[-.102, .329]	.113 (.109)	0.079	[-.101, .328]	.100 (.109)	0.070	[-.113, .313]	.099 (.109)	0.069	[-.114, .312]
Race → Depressive—T-II	.081 (.082)	0.058	[-.078, .241]	.083 (.081)	0.059	[-.076, .242]	.079 (.082)	0.056	[-.082, .240]	.081 (.082)	0.058	[-.080, .241]
SES → Depressive—T-II	.024 (.120)	0.015	[-.210, .259]	.023 (.120)	0.014	[-.211, .257]	.016 (.113)	0.010	[-.206, .237]	.014 (.113)	0.009	[-.208, .235]
Gender → Alcohol—T-II	.027 (.046)	0.037	[-.062, .117]	.023 (.043)	0.032	[-.060, .107]	.028 (.046)	0.038	[-.061, .117]	.024 (.042)	0.033	[-.059, .107]
Race → Alcohol—T-II	.013 (.046)	0.018	[-.078, .104]	.018 (.044)	0.025	[-.068, .103]	.014 (.046)	0.019	[-.077, .104]	.019 (.043)	0.027	[-.066, .104]
SES → Alcohol—T-II	.024 (.069)	0.029	[-.112, .160]	.021 (.067)	0.025	[-.111, .153]	.025 (.070)	0.030	[-.112, .163]	.021 (.067)	0.026	[-.111, .153]
Gender → Depressive—T-III	.134 (.110)	0.091	[-.082, .350]	.130 (.111)	0.087	[-.087, .347]	.135 (.107)	0.092	[-.074, .343]	.131 (.107)	0.089	[-.079, .341]
Race → Depressive—T-III	-.077 (.121)	-0.053	[-.315, .160]	-.079 (.122)	-0.054	[-.317, .159]	-.074 (.120)	-0.051	[-.309, .161]	-.076 (.121)	-0.052	[-.312, .161]
SES → Depressive—T-III	.031 (.129)	0.019	[-.022, .284]	.030 (.130)	0.018	[-.224, .284]	.052 (.123)	0.031	[-.190, .293]	.051 (.124)	0.030	[-.192, .293]
Gender → Alcohol—T-III	.140 (.056)	0.119	[.030, .250]*	.082 (.039)	0.101	[.005, .159]*	.140 (.056)	0.120	[.030, .251]*	.082 (.040)	0.101	[.003, .160]*
Race → Alcohol—T-III	.035 (.073)	0.031	[-.109, .179]	.027 (.054)	0.034	[-.079, .134]	.034 (.073)	0.030	[-.110, .178]	.027 (.055)	0.033	[-.081, .134]
SES → Alcohol—T-III	-.063 (.129)	-0.047	[-.316, .191]	-.043 (.082)	-0.047	[-.204, .117]	-.061 (.128)	-0.046	[-.311, .189]	-.042 (.081)	-0.046	[-.201, .116

Note: *SE* = Standard error; *CI* = Confidence interval of *B*; T-I = Time-point I; T-II = Time-point II; T-III = Time-point III; Depressive = Youth depressive symptoms; Alcohol = Youth alcohol use. Gender was coded as 1 for female and 2 for male; Race was coded as 0 for Caucasian and 1 for minority; SES was coded as 1 for currently receiving child support and 0 as not.
†$p < .10$, *$p < .05$, **$p < .01$, ***$p < .001$.

Table 5.
Fit Statistic and Model Comparisons for Cross-Lagged Analyses

Model	*df*	χ^2	CFI	TLI	SRMR	RMSEA	Model Comparison	AIC	BIC SS Adj.
1	295	536.499***	.887	.867	.036	.081	--	25258.840	25392.609
2	293	532.359***	.888	.867	.036	.077	1 vs. 2 (*p*=.126)	25252.601	25388.918
3	293	529.177***	.890	.869	.036	.079	1 vs. 3 (*p*=.026)*	25246.806	25383.123
4	291	524.325***	.891	.870	.036	.074	2 vs. 4 (*p*=.018)*	25238.795	25377.660
							3 vs. 4 (*p*=.088)†		

Note: *df*=Degree of freedom; CFI = Comparative fit index; SRMR = Standardized root mean square residual; RMSEA = Root mean square error of approximation; AIC = Akaike information criterion; BIC SS Adj. = Bayesian information criterion, sample-size adjusted; Model 1: Stability of depressive symptoms and alcohol use among youth; Model 2: Self-medication hypothesis model; Model 3: Impaired-functioning hypothesis model; Model 4: Bidirectional hypothesis model. †$p < .10$, *$p < .05$.

Table 6.

Mediation Models of the Association between Parental Alcohol Use Problems, Youth Depressive Symptoms, and Alcohol Use

Effects	*B*(*SE*)	*β*	95%*CI*
Model 5: Mediating effects of depressive symptoms among youth			
Direct effects:			
PAlc—T-I → Depressive—T-II	.032 (.011)	.147	[.012, .053]**
Depressive—T-II → Alcohol—T-III	.141 (.065)	.172	[.014, .268]*
PAlc—T-I → Alcohol—T-III	.015 (.018)	.081	[-.020, .049]
Covariates:			
Depressive—T-I → Depressive—T-II	.200 (.030)	.663	[.142, .259]***
Gender→ Depressive—T-II	.119 (.101)	.086	[-.079, .317]
Race→ Depressive—T-II	.069 (.077)	.050	[-.082, .220]
SES→ Depressive—T-II	.010 (.108)	.007	[-.202, .222]
Alcohol —T-II → Alcohol—T-III	.469 (.353)	.381	[-.223, 1.161]
Gender → Alcohol—T-III	.157 (.072)	.138	[.017, .297]*
Race → Alcohol—T-III	.068 (.077)	.061	[-.083, .219]
SES → Alcohol—T-III	-.075 (.121)	-.058	[-.312, .163]
Indirect effects (*α*β*):			
PAlc—T-I → Depressive—T-II→ Alcohol—T-III	.005 (.003)	.025	[.001, .014]*
Model 6: Mediating effects of alcohol use among youth			
Direct effects:			
PAlc—T-I → Alcohol—T-II	.009 (.005)	.093	[-.002, .019]
Alcohol —T-II → Depressive—T-III	-.210 (.162)	-.083	[-.527, .107]
PAlc—T-I → Depressive—T-III	.021 (.019)	.087	[-.017, .058]
Covariates:			
Alcohol —T-I → Alcohol—T-II	.202 (.153)	.279	[-.098, .502]
Gender → Alcohol—T-II	.022 (.019)	.038	[-.015, .058]
Race → Alcohol—T-II	.016 (.023)	.028	[-.029, .061]
SES → Alcohol—T-II	-.016 (.031)	-.024	[-.077, .045]
Depressive—T-II → Depressive—T-III	.753 (.083)	.709	[.591, .915]***
Gender → Depressive—T-III	.176 (.108)	.118	[-.035, .387]
Race → Depressive—T-III	-.093 (.120)	-.064	[-.328, .142]
SES → Depressive—T-III	.052 (.133)	.031	[-.209, .312]
Indirect effects (*α*β*):			
PAlc—T-I → Alcohol—T-II → Depressive—T-III	-.002 (.002)	-.008	[-.016, .000]

Note: *SE* = Standard error; *CI* = Confidence interval of *B*; T-I = Time-point I; T-II = Time-point II; T-III = Time-point III; PAlc = Parental alcohol use problems severity; Depressive = Youth depressive symptoms; Alcohol = Youth alcohol use. Gender was coded as 1 for female and 2 for male; Race was coded as 0 for Caucasian and 1 for minority; SES was coded as 1 for currently receiving child support and 0 as not. Both model fits were acceptable: Model 5: χ^2 (151) = 261.192, $p < .001$; RMSEA = .034; SRMR = .068; CFI = .914; TLI = .896. Model 6: χ^2 (151) =261.112, $p < .001$; RMSEA = .034; SRMR = .072; CFI = .909; TLI = .889. *$p < .05$; **$p < .01$; *** $p < .001$.

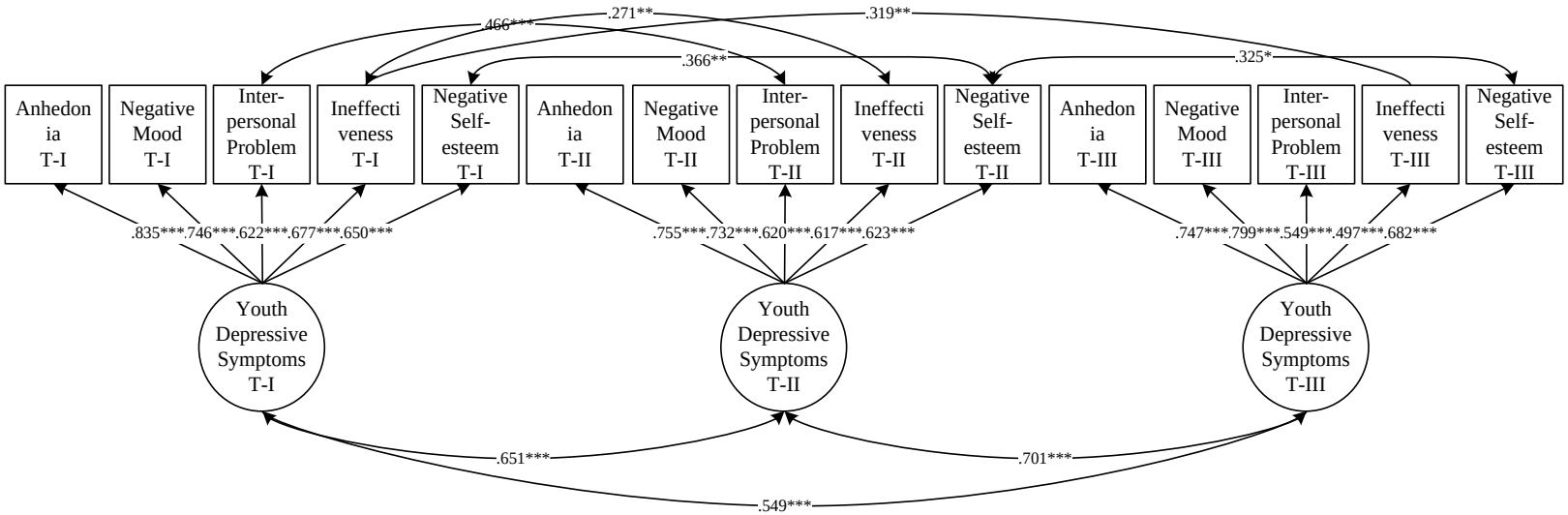

Figure 1. Measurement Model of Youth Depressive Symptoms
Note. T-I=Time-point I; T-II= Time-point II; T-III= Time-point III. Model fit was good: $\chi 2$ (82) = 134.826, $p < .001$; RMSEA = .031; SRMR = .054; CFI = .951; TLI = .937. Standardized coefficients were presented. *$p < .05$, **$p < .01$, ***$p < .001$.

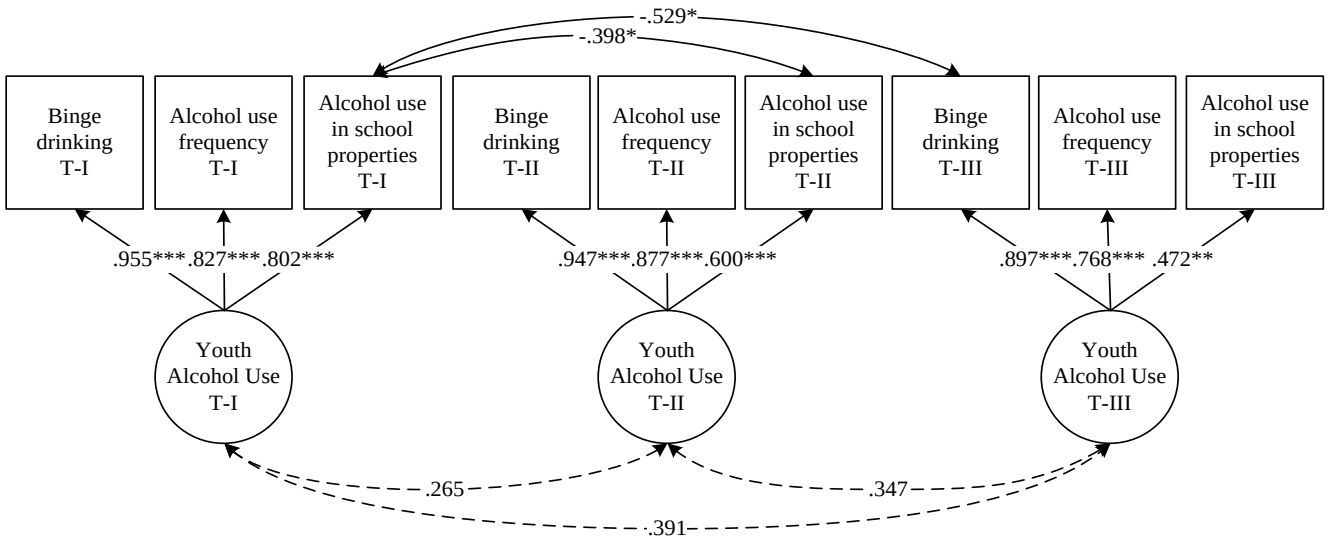

Figure 2. Measurement Model of Youth Alcohol Use.
Note. T-I=Time-point I; T-II= Time-point II; T-III= Time-point III. Model fit was good: $\chi^2(22) = 40.898$, $p = .009$; RMSEA = .037; SRMR = .056; CFI = .940; TLI = .902. Standardized coefficients were presented. *$p < .05$, **$p < .01$, ***$p < .001$.

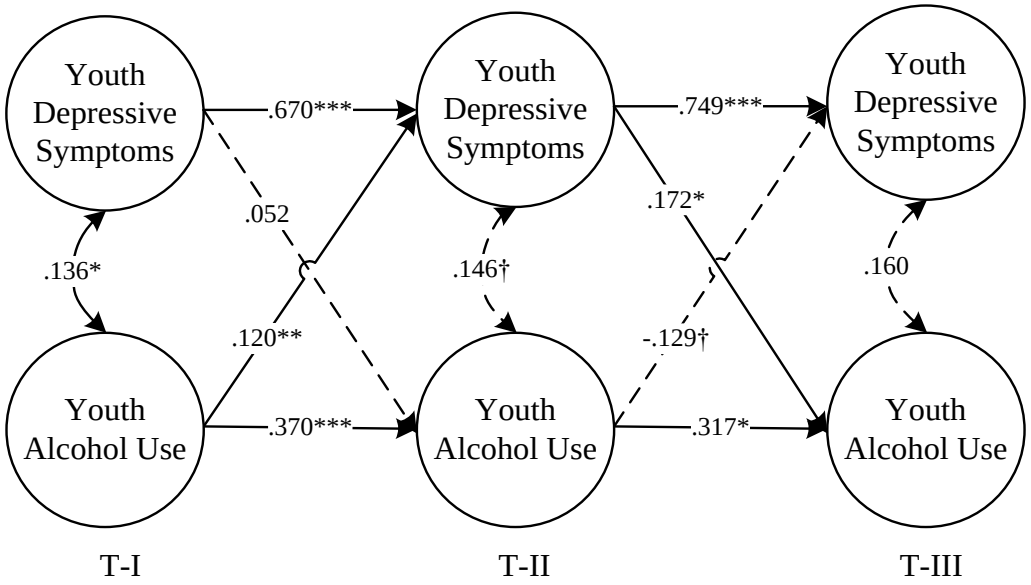

Figure 3. Cross-Lagged Analyses of Relationship Between Youth Depressive Symptoms and Alcohol Use
Note. T-I=Time-point I; T-II= Time-point II; T-III= Time-point III. Model fit was acceptable: χ^2 (291) = 524.325, $p < .001$; RMSEA = .036; SRMR = .074; CFI = .891; TLI = .870. Standardized coefficients were presented. Youth's gender, race and SES were controlled for every path. Covariates were omitted for clarity. †$p < .10$, *$p < .05$, **$p < .01$, ***$p < .001$.

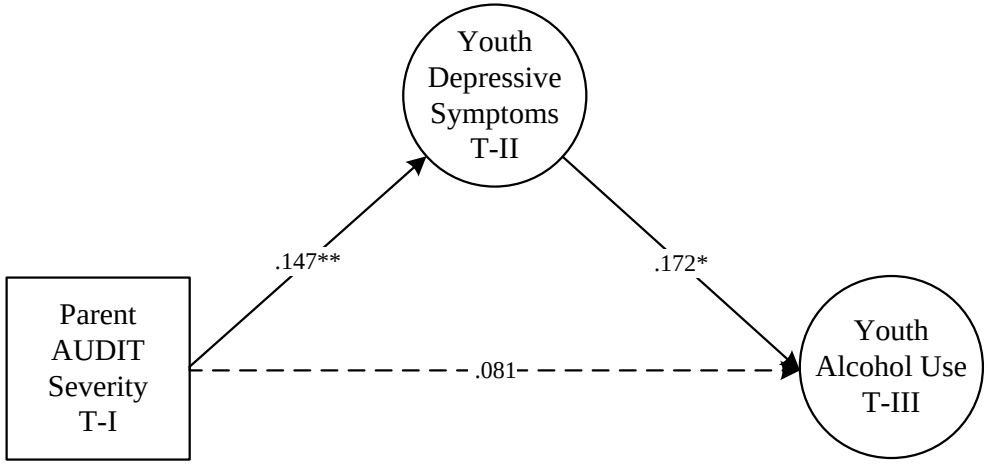

Figure 4. Mediation Model of Depressive Symptoms Linking Parental Alcohol Use Problems and Youth Alcohol Use.
Note. T-I=Time-point I; T-II= Time-point II; T-III= Time-point III. Model fit was acceptable: χ^2 (151) = 261.192, $p < .001$; RMSEA = .034; SRMR = .068; CFI = .914; TLI = .896. Standardized coefficients were presented. Youth's gender, race and SES were controlled for every path. Covariates were omitted for clarity. *$p < .05$, **$p < .01$.

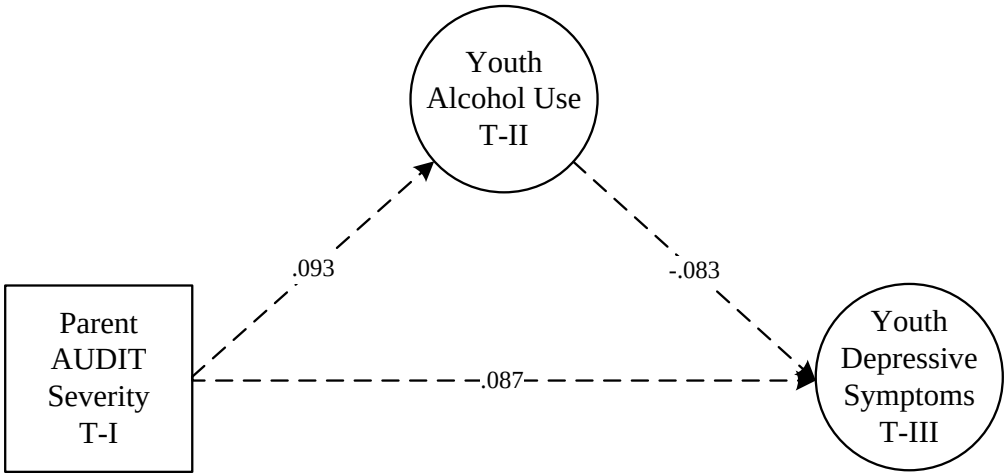

Figure 5. Mediation Model of Youth Alcohol Use Linking Parental Alcohol Use Problems and Depressive Problems.
Note. T-I=Time-point I; T-II= Time-point II; T-III= Time-point III. Model fit was acceptable: χ^2 (151) =261.112, $p < .001$; RMSEA = .034; SRMR = .072; CFI = .909; TLI = .889. Standardized coefficients were presented. Youth's gender, race and SES were controlled for every path. Covariates were omitted for clarity.

CHAPTER 5

DISCUSSION

Maltreated youth are at a significant risk for the development of depressive symptoms (Kim & Cicchetti, 2006) and alcohol use (Shin et al., 2009) during adolescence. Despite the common co-occurrence of alcohol use and depressive symptoms among maltreated youth, evidence about the directionality of associations between the two during adolescence (Saraceno, Heron, Munafò, Craddock, & van den Bree, 2012) remains inconsistent in literature. The current study aimed to reconcile two competing hypotheses, the impaired-functioning and the self-medication hypotheses, on the association between alcohol use and depressive symptoms among maltreated youth. Specifically, using a developmental perspective, the present study proposed and tested an integrative third hypothesis—the bidirectional hypothesis—to explain the associations between depressive symptoms and alcohol use among a nationally representative sample of maltreated youth with CPS records and to examine the role of developmental timing in these associations. Findings supported the impaired-functioning and the self-medication hypotheses at an earlier and older age during adolescence, respectively. Therefore, the current study supported the bidirectional hypothesis whereby youth depressive symptoms and alcohol use were found to promote each other over time. Lastly, parental alcohol use problems were examined as an etiological factor in the associations between youth alcohol and depressive symptoms. When factoring in parental substance use, results supported the self-medication hypothesis in which earlier depressive symptoms precede increased alcohol use during adolescence.

Results from the current study highlight the crucial role of developmental timing on the development of alcohol use and psychopathology during adolescence. The findings regarding the associations between youth alcohol use and depressive symptoms were in accordance with previous studies supporting both the self-medication hypothesis (Paljärvi et al., 2009; Saraceno et al., 2012; Sihvola et al., 2008) and the impaired functioning hypothesis (Brook et al., 2002; Delva et al., 2007; Dennhardt & Murphy, 2011). The impaired-functioning hypothesis was supported at an earlier age, which is corroborated by evidence suggesting that early adolescence is a developmental period with higher neurocognitive vulnerability to the consequences of alcohol consumption (Bava & Tapert, 2010; Spitzer, Kroenke, Williams, & Löwe, 2006). In fact, recent studies have shown that alcohol use is associated with impairment in several brain regions that are involved in affect regulation, including the amygdala (Wrase et al., 2008) and the mesolimbic system (Ward et al., 2009). Specifically, it is possible that alcohol bears a stronger effect on youth brain regions associated with emotional regulation (such as the amygdala and the mesolimbic system) as they undergo maturation (Casey, Jones, & Hare, 2008). Thus, the effects of alcohol use on youth can be exacerbated due to this neurodevelopmental vulnerability during early adolescence, which may disrupt emotional regulation and result in depressive symptoms.

On the other hand, at an older age, depressive symptomatology predicted later increase in alcohol use frequency, supporting the self-medication hypothesis. These findings may reflect the rising availability of alcoholic beverages along with the normative increase in the prevalence of alcohol use during mid to late adolescence. Indeed, national surveys (Johnston, 2016) have reported a significant increase in alcohol use frequency from 8th grade (12-13 years old; 9.7%) to 10th grade (14-15 years old; 21.5%). It is possible that, given the increased accessibility of alcohol in the peer environment, youth have more opportunities to use alcohol to enhance their

mood and cope with negative affects linked to being maltreated during childhood. Interestingly, there was a marginally significant association in which alcohol use at Time II predicted fewer depressive symptoms at Time III, which is not surprising when viewed in light of empirical research on normative adolescent alcohol use. In fact, studies have indicated that some normative use in adolescence is linked with positive outcomes such as increased affiliation with peer groups and peer popularity (Balsa, Homer, French, & Norton, 2011), which helps youths cope with negative affect. In addition, with the increasing prevalence of alcohol use, youth may be able to soothe their negative affect associated with an adverse childhood by consuming alcohol, thus reducing their depressive symptoms during mid- to late-adolescence. Results from the present study further underline the importance of considering developmental timing when studying the mechanisms between alcohol use and depressive symptoms in maltreated adolescence.

Ultimately, the bidirectional hypothesis regarding associations between youth depressive symptoms and alcohol use was supported when considering developmental timing in the context of child maltreatment. This result was consistent with previous evidence suggesting a reciprocal association between depressive symptoms and alcohol use (Blume et al., 2000; Cerdá, Sagdeo, & Galea, 2008; Gilman & Abraham, 2001; Locke & Newcomb, 2001; Marmorstein, 2009) and reconciled the inconsistent evidences of the self-medication and the impaired-functioning hypotheses in literature while accounting for developmental stages. The bidirectional relationship between alcohol use and depressive symptoms was specific, in that the relationship between alcohol use in early (Time I) and late (Time III) adolescence was mediated by depressive symptoms at the second time-point. This finding indicated that alcohol use in early adolescence might be particularly risky for cascading maladaptive development throughout adolescence, resulting in both increases in depressive symptoms and heightened alcohol use.

Further, gender may impact the development of adolescent substance use. Males reported higher levels of alcohol use, but only at the third time-point. This result was consistent with previous studies reporting that males consumed more alcohol and had more alcohol-related problems compared to females (US Department of Health Human Services, 2002). However, this gender effect did not emerge until late adolescence (Young et al., 2002). Differential gender socialization and normative alcohol use amongst teenage boys' peer groups may lead to increased alcohol use in late adolescence (Schulte, Ramo, & Brown, 2009). It is also possible that these results are due to physical and hormonal changes during puberty, and in particular increased muscle mass among males and increased body fat among females. There is evidence that males experience a lower blood alcohol concentration compared to females when given a dose of ethanol proportionate to body weight (Shalala, 1993). This diminished blood alcohol level, in turn, is associated with lower levels of alcohol sensitivity and, consequently, higher levels of alcohol consumption among males compared to females (Schulte et al., 2009).

A second goal of the current study was to test the role of parental alcohol problems in the etiology of depressive symptoms and alcohol use among maltreated youth. Results lent support to the self-medication hypothesis. Depressive symptoms were shown to serve as an underlying mechanism in the association between parental alcohol use problems and youth alcohol use in adolescence. This finding is corroborated by research that has documented parental alcohol use problems as a major source of stress to youth (Hussong et al., 2008; Rothenberg et al., 2016). Furthermore, parental alcohol use problems have been shown to contribute to adverse family environments (Rothenberg et al., 2016). Emotional stress and adverse family environments may induce negative affect, including depressive symptoms (Chassin et al., 1996). Accordingly, the

children of alcohol-abusing parents might self-medicate with alcohol to cope with negative affect linked to their parents' alcohol use.

Limitations and Strengths

It is important to note that there are several limitations to the current study. First, the present study would have benefited from using more than three time-points to test the direction of the association between alcohol and depressive symptoms through young adulthood. However, this dataset is unique for being the only large nationally representative and longitudinal dataset with multiple-reporter measures of youth under investigation by CPS for being maltreated. Second, this study did not take into account service engagement, duration, or service types arranged for the families involved with CPS which might have affected youth depressive symptoms and alcohol use. Lastly, this study comprises a sample of youth involved with CPS, narrowing the generalizability of the findings to this population. However, despite being at significant risk for addiction, maltreated youths are an understudied population that will benefit from empirically informed research.

Implications

The findings of the current study indicated that developmental timing was important to consider while examining the associations between depressive symptoms and alcohol use among maltreated youth. Hence, researchers should utilize a developmental perspective while testing the comorbidity and etiological associations between psychopathology and risk behaviors among adolescents who have been maltreated. Specifically, the transactional relationship between psychopathology and risk behaviors, in which they promote each other over time, should be taken into consideration for future basic and applied research. Importantly, harm reduction programs that seek to prevent early alcohol use among maltreated youth could tailor their content

to account for development timing and should recognize parental alcohol use problems as a risk factor.

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