

THE INTERGENERATIONAL TRANSMISSION OF A STRESS-COPING MODEL OF
ALCOHOL USE AND DISORDER

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ABSTRACT

Jessica M. Solis: The Intergenerational Transmission of a Stress-Coping Model of Alcohol Use and Disorder
(Under the direction of Andrea Hussong)

Substance use disorders are one of the most common psychiatric disorders diagnosed in young adulthood, and previous literature supports the intergenerational transmission of substance use. In order to explain this transmission, the current study focused on the stress-coping model as a potential mechanism. Using data from a multigenerational longitudinal study, it tested whether an individual stress-coping model recurs within alcoholic families over a generation or whether other factors that co-occur with AUDs are responsible for this intergenerational pattern of alcohol use and disorder. Results supported the individual transmission of stress, avoidant coping, and maladaptive coping patterns (e.g., high levels of stress coupled with high levels of avoidant coping) across familial generations. Additionally, these individual pathways negatively predicted adolescent G3 alcohol use and binge drinking. Potential explanations and implications of these findings are considered.

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LIST OF ABBREVIATIONS AND SYMBOLS

AFDP	Adolescent/Adult and Family Development Project
AUD	Alcohol use disorder
α	Cronbach's alpha
β	Standardized regression coefficient
CCSC-R	Children's Coping Strategies Checklist-Reduced
COAs	Children of alcoholics
CFI	Comparative Fit Index
DIS	Diagnostic Interview Schedule
DSM-III	<i>Diagnostic and Statistical Manual for Mental Disorders</i> —Third Edition
FH-RDC	Family History Research Diagnostic Criteria
G1	Generation 1; Parents
G2	Generation 2; Target adolescents (Waves 1-3); Target adults (Waves 4-5)
G3	Generation 3; Targets' offspring (Waves 5-6)
M	Mean
MI	Modification indices
p	P-value
R ²	Proportion of variance in dependent variable explained by independent variable
RMSEA	Root Mean Squared Error of Approximation
SD	Standard deviation
SE	Standard error
SEM	Structural Equation Modeling
SES	Socioeconomic status

SRMR	Standardized Root Mean Square Residual
TLI	Tucker Lewis Index
W4	Wave 4
W5	Wave 5
W6	Wave 6
χ^2	Chi-square

CHAPTER 1: INTRODUCTION

Substance use disorders are one of the most common psychiatric disorders diagnosed in young adulthood (Kessler, Berglund, Demler, Jin, Merikangas, & Walters, 2005). These disorders can be highly impairing, often co-occur with other mental health problems, and demonstrate a strong pattern of intergenerational transmission (Kessler, Chiu, Demler, & Walters, 2005; Sher, Grekin, & Williams, 2005). Intergenerational transmission has been defined as the cross-generational pattern or “cycle” of family dysfunctions, such as substance abuse and disorder (Zuravin et al., 1996). Previous literature supports the intergenerational transmission of substance use, in that having a parent who drinks is positively associated with offspring alcohol use (Yu, 2003) and substance abuse, and having at least one alcoholic parent is typically highly associated with the later development of alcohol dependence (McGue & Iacono, 2004; Sher, 1997); as these findings suggest, alcohol use disorder (AUD) is highly heritable (Chen et al., 2011).

The presence of an intergenerational pattern of AUDs clearly places children of alcoholics (COAs) at greater risk for developing problematic alcohol use. More specifically, COAs initiate substance use earlier than their peers, increase their use more quickly over time, and show a faster escalation from initiation to alcohol use disorder relative to children of non-alcoholic parents (Chassin, Curran, Hussong, & Colder, 1996; Hussong, Bauer, & Chassin, 2008). By young adulthood, COAs also show two times the risk for alcohol and drug use disorders than children of non-alcoholic parents (Chassin, Pitts, DeLucia, & Todd, 1999), with over half evidencing a disorder.

Sher (1991) proffered three potential mechanisms to explain COAs' vulnerability to developing AUDs as captured by a deviance-proneness model, a stress and negative affect or stress-coping model¹, and a substance use effects model. Given its continued empirical support, much of the work investigating the development of AUDs is grounded within the deviance-proneness model. However, Masten and colleagues (2008) argued that there are likely to be multiple risks and roads towards the use of alcohol and the development of AUDs. To disregard these other mechanisms is to ignore the possibility of equifinality towards problematic alcohol use (Hussong et al., 2011). Knowing that, the second of these models, the stress-coping model, depicts an interaction of both genetic and environmental risks such that adolescents with a family history of alcoholism are more likely to experience life stress and ensuing negative affect that leads to the use of alcohol and other drugs as a means to cope with this emotional distress. In turn, the likelihood of developing an AUD increases. Thus, the current study focused on the stress-coping model as a potential explanation for adolescent problematic alcohol use.

A Stress-Coping Model of Alcohol Use and Disorder

Growing out of the Tension Reduction hypothesis (Conger, 1956), the self-medication hypothesis suggests that people turn to alcohol as a means of reducing negative affect resulting from stress. People who consistently drink as a means to regulate negative emotions are thought to be at increased risk for problem-drinking behavior because they are likely to drink more and more often than their peers (Cooper et al., 1995). Even though self-medication may be accelerated by alcohol's stress-dampening effects (Armeli et al., 2003), other research has demonstrated that stress-related drinking does not always lead to problematic alcohol use (Catanzaro & Laurent, 2004; Greeley & Oei, 1999; Young, Oei, & Knight, 1990). Therefore,

¹ Clearly, there are many terms and iterations for a stress and coping interaction leading to alcohol use. In the hopes of avoiding confusion, the term "stress-coping model" will be used throughout this paper to refer to such an interaction.

despite the intuitive appeal of a self-medication mechanism, there is inconsistent evidence (Baker et al., 2004) for the strength (Chassin & Barrera, 1993; Cooper et al., 1995) and longitudinal nature (Hussong et al., 1998; Hussong et al., 2001) of the relationship between negative affect and alcohol use. In response, attempts have been made to reformulate the self-medication model. One such reformulation posits that youth who experience greater life stress and resulting emotional distress are more likely to suffer from emotional dysregulation. In turn, they are less likely to utilize adaptive coping strategies in the face of this distress, which leads to a higher likelihood of engaging in problematic behavior, such as alcohol use (Colder et al., 2002; Cooper et al., 2003; Wills et al., 2001).

COAs are one such group who are more likely to suffer from more frequent stressors and associated maladjustment. For example, COAs experience greater stress throughout the life course (Griffin, Amodeo, Fassler, Ellis, & Clay, 2005; Chassin, Curran, Hussong, & Colder, 1996). More specifically, COAs report more negative family stressors than their peers, particularly for those family stressors that may be a direct consequence of their parents' alcoholism (i.e., parents being arrested, divorced, losing a job, evicted, and other financial difficulties; Chassin, Pillow, Curran, Molina, & Barrera, 1993; Hussong, Bauer, Huang, Chassin, Sher, & Zucker, 2008). However, even when they experience the same life events, COAs rate these family stressors as more severe when compared to their non-COA counterparts (Hussong et al., 2008). Moreover, consistent evidence has demonstrated that adolescents, especially COAs, who experience greater environmental stress, are more likely to use alcohol and to escalate their use over time (Chassin et al., 1996; Hoffman, Cerbone, & Su, 2000). In short, family stressors are particularly influential in COAs' lives as they tend to be more frequent and perceived as more severe.

Researchers have speculated that this increased external life stress forces COAs to access already taxed coping resources (Hussong & Chassin, 2004). Since its infancy, the coping literature has sought to understand the myriad of stressors that challenge individuals across their lifespans, how they do or do not react and/or adapt to the presenting situation, and if and how these responses impact future growth or maladaptation. Beginning with perhaps the most widely cited definition (Compas et al., 2001) of coping, Lazarus and Folkman (1984) defined coping as “constantly changing cognitive and behavioral efforts to manage specific external and/or internal demands that are appraised as taxing or exceeding the resources of the person” (p.141). According to this definition, coping is a continuous, purposeful, and dynamic process that can be divided into problem-focused and emotion-focused strategies. Problem-focused coping is defined as those purposeful actions that are meant to address the stressors arising between a person and his or her environment. Emotion-focused coping entails the alleviation of the resulting negative emotions from that stress. Any of these problem- or emotion-focused actions can be adjusted in response to the continuously evolving demands of the stressful event. As such, Lazarus and Folkman viewed coping as a goal-directed process that has the individual attempting to both resolve the source of the stressor and to manage resulting reactions to the event.

Deeming this and other two-factor characterizations of coping to be too simplistic, Ayers, Sandler, West, and Roosa (1996) generated a four-factor model of child and adolescent coping from a confirmatory factor analysis of 11 theoretically- and empirically-defined coping categories. The four factors were active coping, avoidant coping, support-seeking coping, and distraction. Active coping encompasses those direct problem techniques that incorporate cognitive decision-making, seeking understanding or further information, and employing positive or optimistic thinking. In contrast, avoidant coping involves any wishful thinking or

repression of thoughts or emotions. Pursuing support for one's actions and/or feelings would qualify as support-seeking behavior, and distraction incorporates the utilization of diverting actions or the physical release of emotions. Several studies have claimed and replicated the finding within different samples of youth that this four-factor model of coping actually fits the data better than the more common two-factor models of coping (Ayers et al., 1996; Sandler, Tein, & West, 1994).

From a review of the limited empirical literature that attempts to answer whether COAs cope with stressors differently than their non-COA counterparts, Solis (2013) found that COAs appear to employ a wide range of coping strategies in response to stress. Furthermore, COAs utilize specific coping strategies in response to these stressful situations at significantly different rates than those endorsed by their non-COA peers. In particular, COAs are more likely to use less effective coping strategies across the lifespan, including greater avoidance, denial, and behavioral disengagement (Amodeo et al., 2007; Hussong & Chassin, 2004; Smith et al., 2006; Klostermann et al., 2011). That being said, around adolescence and into young adulthood, COAs are also more likely to use cognitive strategies, such as positive reappraisal, than their peers (Hussong & Chassin, 2004), perhaps as a function of emerging cognitive development. Taken together, COAs appear to have several coping deficits, including a penchant for more avoidant coping strategies. In turn, the use of these more avoidant coping strategies are often seen in adolescents who report the highest levels of alcohol consumption and frequency (Ohannessian et al., 2010).

Therefore, just as their higher likelihood of experiencing greater family stress puts them at risk for problematic alcohol use, adolescent COAs' use of avoidant coping is likely to do the same. Moreover, the interaction between experiencing greater levels of stress and using avoidant

coping strategies in response to said stress, is predictive of alcohol use and alcohol-related problems in adolescents (Laurent, Catanzaro, & Callan, 1997; Wills et al., 2001). As such, for COAs, the stress-coping model of alcohol use may be particularly relevant for explaining alcohol use with particular attention paid to the interaction between *family* stress and *avoidant* coping.

The stress-coping model has received comparatively more empirical attention as an individual risk process within cross-sectional data (Sher et al., 1997) rather than as an intergenerational process within multigenerational, longitudinal data. For example, Sher and colleagues (1997) found that childhood stressors, such as verbal, physical, emotional, and verbal abuse, only partially mediated the relationship between paternal and adolescent and young adult offspring AUDs. However, this mediating relationship was evaluated for parents with a lifetime AUD diagnosis and for stressors that occurred before age 18. Therefore, it was unclear whether the timing of the paternal AUD overlapped with the experience of the childhood stressor, making it difficult to clearly establish the temporal relationship between parental AUD and adolescent AUD via a stressor mechanism. Moreover, even if the stress-coping model does play an explanatory role in the relationship between parental AUD and offspring AUD, research has yet to establish whether this stress-coping mechanism itself recurs over generations and becomes an epidemiological pattern within families over time.

As such, the current study tested whether an individual stress-coping model recurs within alcoholic families over a generation. To do so, I drew on several theoretical perspectives to support the intergenerational component of how an individual stress-coping model is transmitted within alcoholic families. Moreover, data from a landmark longitudinal study (Chassin et al., 1991) was utilized to test whether an individual stress-coping model of alcohol use may recur in an intergenerational context to explain the transmission of problematic alcohol use within

families or whether other factors that co-occur with AUDs are responsible for this intergenerational pattern of alcohol use and disorder.

A Recurring Stress-Coping Model of Alcohol Use and Disorder

Longitudinal studies of high-risk populations, such as COAs, that span developmental periods and generations provide invaluable opportunities to further investigate the developmental processes that underlie the intergenerational transmission of alcohol use disorders within families. Fortunately, the “coming of age” of several of the research community’s esteemed multi-generational longitudinal studies allows for these opportunities to be realized. Seizing this opportunity, the current study combined an intergenerational perspective on alcohol use and disorder with an individual risk perspective focused on the stress-coping model.

More specifically, the current proposal views the intergenerational transmission of alcohol use and disorder as a type of developmental cascade (Masten & Cicchetti, 2010) as predicted by a recurring stress-coping model. More specifically, as Farmer and Farmer (2001) explain, children develop in a system of influences that serve to promote continuity and constrain opportunity over time. These correlated constraints can have either a positive or negative impact depending on the type of developmental factors that are present. In the case of an individual stress-coping model of alcohol use, negative correlated constraints (i.e., an increasingly stressful environment coupled with maladaptive coping strategies) can lead towards the use of alcohol as a means of addressing resulting emotional dysregulation, which in turn, can develop into an AUD. As the alcoholic individual has children, the COA’s developmental system may be dominated by these same and even additional negative, correlated risk factors. Briefly, COAs may possess a genetic risk load to substance abuse (Dick et al., 2002; Prescott et al., 2006), are likely to have more chronic and severe stressors (Hussong et al., 2008), and are also likely to

have more coping deficits as compared to their peers (Amodeo et al., 2007; Hussong & Chassin, 2004; Smith et al., 2006). Moreover, parents and siblings are also more likely to model alcohol use as a means of coping with stress (Barrera et al., 1993; Hansen, 1997). The culmination of these correlated constraints increases risk for a stress-coping pattern of alcohol use in COAs and for eventually developing an AUD themselves. As such, I hypothesized that parents will follow an individual stress-coping pattern of alcohol use in young adulthood leading to the development of an AUD; subsequently, children will model their parents' stress-coping behavior leading to the offspring's own problematic alcohol use. Therefore, *my first hypothesis tested whether the intergenerational transmission of alcohol use and disorder reflects the transmission of a single underlying stress-coping model that is passed across generations.*

The Stressful Environment Selection Model of Alcohol Use and Disorder

A recurring stress-coping model may not be the only mechanism explaining the intergenerational transmission of stress, coping, and alcohol use and disorder. Therefore, complementary hypotheses asserting that selection and socialization are alternative mechanisms in the intergenerational transmission of alcohol use and disorder that were also tested. First, the *stressful environment selection model* argues for the risk mechanism of selecting into stressful environments over time, and it is exposure to this continuously stressful environment, rather than modeling of parental self-medication, that is the putative cause for stress-related alcohol use over generations. For instance, youth who experience negative life and family stressors are at risk for a variety of negative outcomes, including problematic alcohol use, across several developmental time periods (i.e., adolescence, young adulthood, adulthood; Hussong & Chassin, 2004; Low et al., 2012). Unfortunately, previous research has also found a link between early exposure to life stressors and continued stress exposure over the life course. For example, Hammen and

colleagues (2012) found that mothers' reports of higher levels of family stressors during the first five years of their children's lives predicted their adolescents' total acute stress at age 15. Moreover, mothers' higher endorsement of family stress during their children's first five years of life also predicted their offspring's total chronic stress at ages 15 and 20. Similarly, youth-reported chronic stress at age 15 predicted their own later stress at age 20. Turner and Butler (2003) presented similar findings in that higher levels of early adversity in childhood predicted significantly greater levels of stress in adulthood. Therefore, there appears to be stability in individuals' reports of stress over time.

One potential explanation for this continuity in stress exposure comes from the idea that experiences of early stress tend to stem from relatively stable, structural and familial circumstances, such as family conflict or economic uncertainty, which in turn, also increase the child's risk for additional stressors later in life (Pearlin, 1989). Furthermore, early stress exposure is also related to deleterious effects on children's coping, cognitive abilities, and interpersonal styles (Bifulco et al., 2002; Hankin, 2005; Turner & Lloyd, 1995). In turn, previous work has found that the resulting deleterious effects of early exposure to stress are also likely to affect children's selection into different environments later in life and to heighten the likelihood that they will respond to future stressors in ways that intensify and multiply this stress (Campbell, Simpson, Boldry, & Kashy, 2005). This selection process continues, and ultimately recurs, over time as these adolescents and young adults eventually have families of their own, consequently creating an aversive family context for their own children (Agnew, 1997; Thornberry, 2005). Notably, youth who experience more life and family stress are also more likely to drink alcohol and to increase this use, both in quantity and frequency, over time (Chassin et al., 1996; Hoffman et al., 2000). Thus, the selection model posits that early stress

exposure increases the probability of chronic stress exposure over the life course and across familial generations, which in turn, increases the likelihood that problematic alcohol use will occur in response to these stressors over time and across generations. Following this model, *my second hypothesis predicted that the relation between parents' family stress in young adulthood and their offspring's alcohol use in adolescence was mediated by their offspring's family stress in childhood.*

The Socialization of Poor Coping Model of Alcohol Use and Disorder

Switching the focus of intergenerational transmission risk from selection to poor environments to parent socialization practices, the *socialization of poor coping model* recognizes that parents' are often their children's primary agents of socialization (Bronfenbrenner, 1986). One significant means of socialization is parent modeling. Drawing on Social Learning Theory (Bandura, 1977), a small area of research posits that parents' modeling of coping behaviors may influence the development of their children's coping strategies. Kliewer and colleagues have found that parents' utilization of maladaptive coping strategies was associated with their children's own use of similar maladaptive coping strategies in response to dealing with sickle cell disease (Kliewer & Lewis, 1995) and community violence (Kliewer et al., 2006). Likewise, Brook and colleagues (2002) found a similar relationship for more adaptive coping, showing that fathers' use of adaptive coping strategies was related to their adolescents' use of these same strategies. Additionally, in response to invasive medical procedures, children will choose particular coping strategies based on the coping instructions they receive from their parents (Manimala, Blount, & Cohen, 2000). Moreover, children's self-blame attributions and eventual choice of coping strategies were positively correlated with parental criticism, threats, commands,

and psychological control. While this is a growing body of empirical research, the results lend themselves to the premise that parents are socialization agents for their children's coping.

Poor coping strategies are more likely to result in alcohol use. Adolescents who learn ineffective coping strategies for dealing with stress may also be at risk for engaging in alcohol use associated with the stress-coping model. Adolescent heavy drinking (Britton, 2004; Veenstra et al., 2007; Wills et al., 2001) and alcohol-related problems (Laurent, Catanzaro, & Callan, 1997) are typically associated with adolescents' use of more avoidant coping strategies in response to stress. In contrast, the use of more active styles of coping are negatively associated with alcohol use and alcohol-related problems (Wills et al., 2001; Windle & Windle, 1996). The socialization of poor coping model posits that ineffective coping transmitted across generations impacts adolescents' risk for alcohol use as associated with a stress-coping model. Following this model, *my third hypothesis predicted that children's avoidant coping would mediate the pathway from parents' avoidant coping in young adulthood to their offspring's alcohol use in adolescence.*

The Current Study

The current study drew on each of the individual causal pathways in the overarching conceptual model of the recurring stress-coping model of alcohol use. It tested four specific hypotheses that align with the aforementioned pathways.

Hypothesis 1: According to a recurring stress-coping model, the intergenerational transmission of alcohol use and disorder reflected the transmission of an individual, underlying stress-coping process that is modeled by parents and in turn, mimicked by children, to predict intergenerational transmission patterns and mechanisms of alcohol use.

Hypothesis 2: According to a selection into stressful environments model, the relation between parents' family stress in young adulthood and their offspring's alcohol use in adolescence is mediated by their offspring's family stress in childhood.

Hypothesis 3: According to a socialization of poor coping model, children's avoidant coping will mediate the pathway from parents' avoidant coping in young adulthood to their offspring's alcohol use in adolescence.

Hypothesis 4: These three models were posited to present complementary risk for the intergenerational transmission of the individual stress-coping model of alcohol use and disorder. In order to determine whether there were unique effects of the three models, the recurring stress-coping, selection into stressful environments, and socialization of poor coping models were entered into a single analytical model to predict adolescents' alcohol use.

CHAPTER 2: METHODS

Study hypotheses were examined with data from the Adolescent/Adult and Family Development Project (AFDP; Chassin, Lee, Cho, Wang, Agrawal, Sher, & Lynskey, 2012; Chassin, Rogosch, & Barrera, 1991). The AFDP is a community sample that assessed three generations of family members over time including parents (G1s), target adolescents (G2s), and later, these targets' children (G3s).

Procedure

Data were collected with computer-assisted interviews administered to each participant separately either at families' homes or on campus, or by telephone for out-of-state, young adult participants during the later waves of data. Mail-in surveys augmenting the primary battery were added in later waves. Interviews required 1-3 hours, and participants were paid up to \$70 at each wave. To protect the confidentiality of the participants, a Certificate of Confidentiality was obtained from the Department of Health and Human Services.

Participants

A sample of 454 G1-G2 families of mothers (G1s), fathers (G1s), and their children (G2s; 246 COAs and 208 matched controls) completed three annual interviews when the G2 was an adolescent (at ages 10-15 at wave 1) with 449 and 447 completing waves 2 and 3 respectively. Three G2 young adult follow-ups occurred subsequently at 5-year intervals (i.e., waves 4-6 at ages 17-23, 21-30, and 28-36, respectively). G2 partners and G3s were also assessed at 5-year intervals in Waves 5 and 6.

G1 alcoholic parents were identified through court records, HMO wellness questionnaires, and community telephone screenings (Chassin, Barrera, Bech, & Kossak-Fuller, 1992). To participate, at least one G1 parent must have met criteria for lifetime alcohol abuse or dependence according to the *Diagnostic and Statistical Manual for Mental Disorders—Third Edition* (DSM-III; American Psychiatric Association, 1980). To confirm eligibility, diagnostic interviews were conducted during the first wave of data collection with G1 parents using the Diagnostic Interview Schedule (DIS; Robins, Helzer, Croughan & Ratcliff, 1981; Robins, Helzer, Ratcliff, & Seyefried, 1982) or with spousal report, if the alcoholic parent was not interviewed, using the Family History Research Diagnostic Criteria (FH-RDC; Andreasen, Endicott, Spitzer, & Winokur, 1977). Additionally, COA families had to meet the following inclusionary criteria: G1s were either Hispanic or non-Hispanic Caucasian, English-speaking, an Arizona resident and the biological and custodial parent of a residential child between 10.5-15.5 years old at Wave 1; G1s and G2s could not have any cognitive limitations that would preclude the interview process. Control families were recruited via telephone interviews identified from reverse directory searches and were matched to COA families based on ethnicity, family structure, SES and the adolescent's age and sex.

Retention in young adulthood was excellent, with 407 (90%) of the original G2 sample interviewed at wave 4, 411 (91%) interviewed at wave 5, and 409 (90%) interviewed at wave 6. In the G2 young adult follow-up (wave 4), full biological siblings aged 18-26 were included and all of these G2 siblings were again invited to participate at waves 5 and 6, five years later. A total of 327 G2 siblings (78% of eligible participants) were interviewed at wave 4, and 365 G2 siblings (86%) were interviewed at wave 5 (n=378 interviewed at either wave). Beginning in Wave 5, study staff conducted interviews with the children of G2 and G2 sibling participants

(G3s). A total of 433 G3 participants were interviewed at wave 5, and 522 G3 participants (33% Hispanic) were interviewed at wave 6.

Analyses were based on three time points: (1) at Wave 4, when G2 targets and siblings were young adults; (2) at Wave 5, when these G2s' children (G3s) are in late childhood (ages 8-12); and (3) at Wave 6 when G3s are in adolescence (ages 13-17). For G2s to be included in the current study, they must have: (1) participated in Waves 4-6, (2) had a G3 child aged 8-12 during Wave 5, and (3) had a G3 child aged 13-17 in Wave 6. The sample of 203 G2s and their eligible siblings from 140 G1-G2 families was 62% female, 48% COA, 64% Caucasian and 34% Hispanic, ranging in age from 18-26 at Wave 4 (M=22 years, SD=1.38 years), 22-33 at Wave 5 (M=28 years, SD=2.99 years), and 25-40 at Wave 6 (M=34 years, SD=2.79 years). For G3s to be included in the current study, they must have been an eligible family's oldest participating child within the appropriate age ranges at both Waves 5 (ages 8-12) and 6 (ages 13-17). Of those G2s and G2 siblings with children of the appropriate ages, there were 83 G3s at Wave 5 in late childhood (54% female, mean age=9 years, SD=1.33 years) and 112 G3s at Wave 6 in adolescence (70% female, 70% Caucasian and 25% Hispanic, 66% COA, mean age=15 years, SD=1.06 years). The highest education level obtained by G2 parents in this sample at Wave 6 included 26% who had a high school diploma or GED, 34% had some college or vocational/technical school, 16% had completed vocational/technical school or received their AA degree, 11% had received their bachelor's degree, 1% had some graduate/professional school, and 4% had completed graduate/professional school.

Measures

Demographic variables. Demographic variables included participant gender (males=1; females=0) and age (calculated in years from date of birth) as self-reported by G1s, G2s and G3s,

and socioeconomic status as indexed by the highest level of education obtained by G2s. Highest education status was measured on a 10-point scale ranging from (1) 8th grade or less to (11) completed graduate/professional school. Ethnicity was also coded by self-identification as either non-Hispanic Caucasian (0) or Hispanic (1).

Alcoholism. G1 alcohol use was assessed via G1 parent-report. In order to assess lifetime G1 AUD at Wave 1, G1 parents were directly interviewed at baseline using a computerized version of the substance use disorder modules of the DIS (Robins, Helzer, Croughan & Ratcliff, 1981; Robins, Helzer, Ratcliff, & Seyefried, 1982) to assess diagnostic status. G1 AUD status was dichotomized to indicate whether G1s had ever received a diagnosis of AUD. G1 AUD was included as a covariate in subsequent analyses.

All young adult G2 subjects were assessed for alcohol abuse and dependence at Wave 4. Diagnosis was obtained from a computerized version of the Diagnostic Interview Schedule III-R (Robins, L.N., & Helzer, J.E., 1991). A dichotomous variable was created to indicate whether young adults G2s met criteria for an AUD at Wave 4.

Alcohol use. Wave 5 G2 adults' and Wave 6 G3 adolescents' alcohol use (e.g., beer, wine, wine coolers, and hard alcohol) was measured by G2 and G3 participants' self-report of two items assessing their frequency and typical quantity of drinking in the past year. Frequency response options ranged from (0) never to (7) to every day. G2s and G3s then reported on the typical quantity of consumption in a given drinking session with response options ranging from (0) 0 drinks to (8) 9 or more drinks. Scores on the frequency and quantity items were then standardized. To facilitate substantive interpretation, alcohol consumption scores were then obtained by taking the product of the standardized quantity and frequency variables.

Binge drinking. Wave 6 G3 adolescents' binge drinking was measured by G3s' self-report of one item assessing how often they had consumed 5 or more drinks of any alcoholic beverage at one time within the past year. Given the low endorsement rates, this item was then dichotomized to indicate engagement in binge drinking or not.

Family stress. Life stressors were assessed using an adapted version of the General Life Events Schedule for Children (Sandler, Ramirez, & Reynolds, 1986) and Children of Alcoholics Life Events Schedule (Roosa, Sandler, Gehring, Beals, & Cappel, 1988). All items were previously rated in the literature as negative events and were assessed in a past year time-frame. For purposes of the current study, only stressors related to the family were included. G2s reported on their G1-G2 family stress at Wave 4 and on their G2-G3 family stress at Wave 5. G2 respondents indicated whether the same 14 family stressors occurred within the past year at Waves 4 and 5, respectively, and a summary score was created. Given G3s' young age during Wave 5 (aged 8-12) and the likelihood that they may not have been aware of all occurring family stressors, the decision was made to only use G2s' report of G2-G3 family stress items to measure G3s' stress during Wave 5.

Avoidant Coping. The AFDP evaluated a general "trait" approach to parents' and children's coping, and different measures were used for parents and children. G2 assessment of coping during Wave 4 was assessed via 24 items from Zautra, Sheets, and Sandler's (1996) adaptation of Carver, Scheier, and Weintraub's (1989) subscales. For the current study, analyses focused on the 6-item avoidant subscale assessing denial and mental disengagement. Participants rated how often they typically used each coping strategy when faced with a stressful event on a 4-point scale ranging from (1) *I usually don't do this at all* to (4) *I usually do this a lot*. Because internal reliability at Wave 4 for these G2 self-reported avoidant coping strategies was $\alpha=.62$, I

took additional steps to create a more reliable avoidant coping scale. I presented five independent raters with all 24 original coping items and the current study's definition of avoidant coping (i.e., "attempts to reduce stress by distancing oneself mentally and/or physically from the problem at hand"). I instructed each to rate whether the items could be considered avoidant coping. If a rater deemed an item to be an avoidant coping strategy, they then reported, using a 3-point scale, how well it met that definition. Any item that received at least two votes for being an avoidant coping strategy was included in subsequent exploratory factor analysis using promax rotation. A total of 16 G2 coping items were identified by the panel. Analysis of the scree plot and eigenvalues indicated that a one-factor solution of 12 items best represented the avoidant coping data. Internal reliability for these final 12 G2 avoidant coping items was $\alpha=.75$. In order to determine how many avoidant coping strategies were used, use of a coping strategy was dichotomized to indicate no use at all or any use (no matter the frequency). The number of avoidant coping strategies endorsed was then summed to create an avoidant coping score. This type of summary score has been used previously in the literature to demonstrate the importance of the number of different coping strategies employed (Bernzweig, Eisenberg, & Fabes, 1993; Seiffge-Krenke, Aunola, & Nurmi, 2009; Waters & Thompson, 2014). Examples of items included, "refusing to believe that it has happened," "turning to work or other substitute activities to take one's mind off things," and "holding off on doing anything about it until the situation permits."

The Wave 5 assessment of coping for G3s aged 8-12 used 24 items from the Children's Coping Strategies Checklist-Reduced (CCSC-R; Program for Prevention Research, 1999). Nine broad dimensions of coping were assessed, including three, four-item subscales associated with avoidant coping: (a) avoidant actions (attempts to avoid the problem by staying away from or leaving it), (b) repression (suppressed thinking of the problematic situation), and (c) wishful

thinking (utilizing wishful thinking or wishing that the problem was better). However, in order to create a similar avoidant coping item set for G3s in Wave 5 that was used for G2s in Wave 4, the external panel once again rated each of the original 20 G3 coping items. The same rating process yielded 22 items. Analysis of the scree plot and eigenvalues of the exploratory factor analysis indicated that a one-factor solution of 8 items best represented the avoidant coping data for G3s. Internal reliability for these final G3 items was $\alpha=.70$. In order to determine how many avoidant coping strategies were used, use of a coping strategy was dichotomized to indicate no use at all or any use (no matter the frequency). The number of avoidant coping strategies endorsed was then summed to create an avoidant coping score. Example items included “you didn’t think about it,” “you avoided it by going to your room,” and “you just forgot about it.”

Maladaptive Stress/Coping Pattern. In order to determine whether G2s and G3s independently demonstrated a maladaptive pattern of high levels of family stress combined with avoidant coping, a dichotomous (e.g., 0 or 1) maladaptive stress/coping pattern variable was created. This maladaptive stress/coping pattern variable indexed whether a G2 participant reported above average levels of stressful family events and avoidant coping in Wave 4 or whether a G3 participant reported above average levels of stressful family events and avoidant coping in Wave 5. As such, a participant was assigned a score of 1 if (s)he endorsed higher than average levels of both family stress and avoidant coping. 15% of G2s and 29% of G3s demonstrated this maladaptive stress/coping pattern.

CHAPTER 3: RESULTS

Table 1 presents zero-order correlations between the key observed predictor and outcomes variables included in the study models. All analyses were conducted in Mplus Version 6.12. The fit of all models was determined by the examination of multiple indices (e.g., chi-square, Comparative Fit Index (CFI), Tucker Lewis Index (TLI), Root Mean Squared Error of Approximation (RMSEA), and Standardized Root Mean Square Residual (SRMR)). These model fit indices were used for all analyses. In order to address the non-normal distribution of the G3 alcohol use outcome, the MLR estimator was used to produce robust standard errors. A Satorra-Bentler chi-square test was used to perform a Likelihood Ratio Test of model fit. Additionally, in order to account for a nested data structure that included G2 siblings within families, the complex analysis option was used to correct for the standard error biases created by the grouped nature of the data. Similarly, due to the dichotomous nature of the adolescent binge drinking outcome variable, a logit link function was used with the ML estimator.

For the following path models, all G2 predictors were regressed on the following covariates: G1 AUD, G2 SES, G2 age, G2 gender, and G2 ethnicity. Similarly, all G3 predictors and outcomes were regressed on the following covariates: G3 COA status (whether any parent had an AUD), G3 age, G3 gender, and G3 ethnicity.

Hypothesis 1

A path model was used to test the first hypothesis that the intergenerational transmission of alcohol use and disorder reflects the transmission of an individual, underlying stress-coping process that is modeled by parents and in turn, mimicked by children, to predict intergenerational

transmission patterns of alcohol use (see Figure 1). The resulting model did not fit the data well ($\chi^2(28) = 32.06, p = .27, CFI = .87, TLI = .73, RMSEA = .04, SRMR = .05$). Given that the only theoretically viable modification indices related to the transmission of a maladaptive coping pattern (i.e., high family stress coupled with high avoidant coping), sensitivity analyses were conducted to determine the relative contributions of this pattern to adolescents' alcohol use. This sensitivity analysis had G3 alcohol use regressed on G3 childhood maladaptive coping patterns which was in turn regressed on G2 young adult maladaptive coping patterns. The resulting model (see Figure 2; Table 2) fit the data exceptionally well ($\chi^2(15) = 14.41, p = .50, CFI = 1.00, TLI = 1.00, RMSEA = 0.00, SRMR = 0.04$) but did not explain a significant amount of variance in G3 alcohol use ($R^2 = 0.10, p > .05$). According to this model, all of the paths were significant such that G2s who exhibited a maladaptive coping pattern during young adulthood were more likely to later have offspring that exhibited this same maladaptive coping pattern in childhood (standardized $\beta = 0.21, p < .05$). Moreover, a G3 childhood maladaptive coping pattern negatively predicted later alcohol use in adolescence (standardized $\beta = -0.09, p < .05$). Several covariates also significantly predicted adolescent alcohol use. More specifically, older G3s were less likely to exhibit a maladaptive coping pattern in childhood (standardized $\beta = -0.34, p < .001$) and to drink more in adolescence (standardized $\beta = 0.17, p < .05$). Similarly, G3 COAs were also significantly more likely to use alcohol in adolescence (standardized $\beta = 0.40, p < .05$). Additionally, G2s from a higher SES bracket were more likely to exhibit a maladaptive coping pattern in young adulthood (standardized $\beta = 0.22, p < .05$).

The total and indirect effects of G2 young adult maladaptive coping pattern on G3 alcohol use were not significant (standardized $\beta = -0.02, p > .05$). However, in an effort to avoid a misspecified model, further analyses directly regressed G3 alcohol use on G2 maladaptive

coping pattern. In doing so, results indicated that G2s who exhibited a maladaptive coping pattern during young adulthood were less likely to have adolescent offspring who drank (standardized $\beta = -0.11$, $p < .01$). Moreover, the total effect of a G2 young adult maladaptive coping pattern on G3 alcohol use was significant (standardized $\beta = -0.12$, $p = .001$), indicating an indirect effect equaling -0.01 ($p > .05$). Overall, according to these sensitivity analyses, there is evidence for transmission of a maladaptive coping pattern from G2s to G3s that significantly predicted G3 alcohol use.

A path analysis also investigated Hypothesis 1 with regards to adolescent binge drinking (see Figure 3; Table 3). According to this model, even though only covariates were significant predictors, a significant amount of variance in G3 alcohol use was explained ($R^2 = 0.45$, $p < .01$). For instance, older G3s were less likely to exhibit a maladaptive coping pattern in childhood (standardized $\beta = -0.32$, $p < .01$) and more likely to binge drink in adolescence (standardized $\beta = 0.52$, $p < .001$). Additionally, G2 COA status was a significant predictor in that G2s with an alcoholic parent (G1s) in childhood were more likely to develop an AUD themselves in adulthood (standardized $\beta = 0.48$, $p < .05$). Similarly, being a G3 COA increased the log odds of G3s binge drinking in adolescence by 0.53 ($p < .05$). Also, G2s from a higher SES bracket were more likely to exhibit a maladaptive coping pattern in young adulthood (standardized $\beta = 0.22$, $p < .05$).

Hypothesis 2

A path analysis was used to test the hypothesis that the relation between parents' family stress in young adulthood and their offspring's alcohol use in adolescence was mediated by their offspring's family stress in childhood. To test this model, G3 alcohol use was regressed on G3 Wave 5 family stress (i.e., "childhood stress"). In turn, G3 childhood stress was regressed on G2

Wave 4 family stress (i.e., “young adult stress”). The resulting model fit the data moderately well ($\chi^2(15) = 17.08, p = .31, CFI = .96, TLI = .92, RMSEA = 0.04, SRMR = 0.04$). Consequently, modification indices were consulted to re-specify the model. Modification indices indicated that G3 alcohol use should be regressed on G2 age ($MI=4.40$). Previous research has found that younger maternal age is associated with greater frequency of offspring alcohol use (Shaw, Lawlor, & Najman, 2006). Therefore, given the theoretical plausibility of this suggestion, the model was re-specified to include this pathway.

The resulting model (see Figure 4; Table 4) fit the data exceptionally well ($\chi^2(14) = 12.05, p = .60, CFI = 1.00, TLI = 1.00, RMSEA = 0.00, SRMR = 0.03$) and explained a significant amount of variance in G3 alcohol use ($R^2 = 0.15, p < .05$). Moreover, according to a likelihood ratio test, it significantly improved upon the model fit of the original model ($\chi^2(1) = 9.69, p < .01$). Additionally, all of the paths were significant such that G2’s stress in young adulthood significantly predicted their offspring’s stress in childhood (standardized $\beta = 0.57, p < .001$), and G3 childhood stress negatively predicted their later alcohol use in adolescence (standardized $\beta = -0.15, p < .01$). Several covariates also significantly predicted adolescent alcohol use such that older G3s (standardized $\beta = 0.24, p < .01$) and G3 COAs (standardized $\beta = 0.37, p < .05$) were each significantly more likely to use alcohol in adolescence. Moreover, G3s with older parents were less likely to drink alcohol (standardized $\beta = -0.20, p < .01$). The specific indirect effect of G2 young adult stress on G3 alcohol use was significant (standardized $\beta = -0.07, p < .05$). To determine whether the model was misspecified, G3 alcohol use was regressed on G2 young adult stress. Results determined that the total effect (standardized $\beta = 0.01, p > .05$) and direct effect (standardized $\beta = 0.12, p > .05$) were not statistically significant. However, the specific indirect effect of the mediational pathway from young adult stress to childhood stress to

adolescent alcohol use remained significant (standardized $\beta = -0.11$, $p < .05$). Overall, these results support the stressful environment selection model of Hypothesis 2; however, the effect of family stress on adolescent alcohol use was in the opposite direction originally predicted.

A path analysis also investigated Hypothesis 2 with regards to adolescent binge drinking (see Figure 5; Table 5). G3 binge drinking at Wave 6 was regressed on G3 childhood stress. In turn, G3 childhood stress was regressed on G2 young adult stress. The resulting model explained a significant amount of variance in G3 binge drinking ($R^2 = 0.41$, $p < .01$) and indicated that the more family stress experienced by G2s as young adults, the more family stress G3s experienced in childhood. More specifically, with each family stressor G2s experienced in young adulthood, the log odds of G3s experiencing family stress during childhood increased by 0.57 ($p < .001$). G3 family stress during childhood did not predict G3 heavy alcohol use during adolescence. Additionally, older G3s (standardized $\beta = 0.57$, $p < .001$) and G3 COAs (standardized $\beta = 0.53$, $p < .10$) were each significantly more likely to binge drink in adolescence. Therefore, Hypothesis 2 was only partially supported in that family stress continued from one generation to the next, but this transmission did not predict adolescents' heavy alcohol use.

Hypothesis 3

A path analysis estimated whether the relation between parents' avoidant coping in young adulthood and their offspring's alcohol use in adolescence was mediated by their offspring's avoidant coping in childhood. To test this model, G3 alcohol use was regressed on G3 Wave 5 avoidant coping (i.e., "childhood coping"). In turn, G3 Wave 5 avoidant coping was regressed on G2 Wave 4 avoidant coping (i.e., "young adult coping"). The resulting model did not fit the data well ($\chi^2(12) = 13.39$, $p = .34$, CFI = .90, TLI = .75, RMSEA = 0.04, SRMR = 0.04). Consequently, modification indices were consulted to re-specify the model. Modification indices

indicated that G3 childhood coping should also be regressed on G1 AUD status ($MI = 5.51$). The resulting model (see Figure 6; Table 6) fit the data very well ($\chi^2(11) = 7.81, p = .73, CFI = 1.00, TLI = 1.00, RMSEA = 0.00, SRMR = 0.03$) but did not explain a significant amount of variance in G3 alcohol use ($R^2 = 0.11, p > .05$). According to a likelihood ratio test, however, it significantly improved upon the model fit of the original model ($\chi^2(1) = 4.99, p < .05$). Even though G3 childhood coping did not predict G3 alcohol use, G2's coping in young adulthood did predict their offspring's coping in childhood (standardized $\beta = 0.21, p < .05$) such that G2s who endorsed avoidant coping as young adults were more likely to have G3 children who also reported using greater numbers of avoidant coping strategies. Several covariates also significantly predicted both G3 childhood coping and adolescent alcohol use. For example, older G3s (standardized $\beta = -0.29, p < .05$) were less likely to use avoidant coping strategies during childhood and more likely to drink during adolescence (standardized $\beta = 0.23, p < .05$). Additionally, parent and grandparent AUD status were also significant predictors such that G3s were more likely to drink (standardized $\beta = 0.43, p < .05$) if their parent(s) had an AUD. Moreover, they were also more likely to use avoidant coping in childhood if their grandparents (G1s) had an AUD (standardized $\beta = 0.56, p < .05$). In contrast, the total and specific indirect effects from G2 young adult coping did not significantly predict G3 alcohol use (standardized $\beta = 0.02, p > .05$). Further analysis revealed that the total effect (standardized $\beta = -0.04, p > .05$) of G2 young adult coping on G3 alcohol use, a combination of the direct effect (standardized $\beta = -0.07, p > .05$) and the indirect effect (standardized $\beta = 0.03, p > .05$), was not statistically significant. Therefore, Hypothesis 3 was only partially supported in that parents were socialization agents of their children's coping; however, this transmission of maladaptive coping did not lead to increased adolescent alcohol use.

A path analysis also investigated Hypothesis 3 with regards to adolescent binge drinking (see Figure 7; Table 7). G3 binge drinking at Wave 6 was regressed on G3 childhood coping. In turn, G3 childhood coping was regressed on G2 young adult coping. The resulting model explained a significant amount of variance in G3 binge drinking ($R^2 = 0.53$, $p = .001$) and indicated that G3 childhood coping significantly predicted G3 binge drinking such that with each endorsement of an avoidant coping strategy during childhood, the log odds of drinking 5 or more drinks in one sitting during adolescence decreased by 0.24 ($p < .05$). Moreover, G2s' young adult coping predicted their children's coping such that with each G2 endorsement of an avoidant coping strategy during young adulthood, the log odds of their children also endorsing avoidant coping increased by 0.19 ($p < .05$). Several covariates also significantly predicted both G3 childhood coping and adolescent binge drinking. More specifically, older G3s were more likely to binge drink in adolescence (standardized $\beta = 0.56$, $p < .001$) and less likely to use avoidant coping strategies in childhood (standardized $\beta = -0.25$, $p < .01$). Also, having a parent with an AUD increased a G3's log odds of binge drinking by 0.48 ($p < .10$). Therefore, Hypothesis 3 was again only partially supported in that parents were socialization agents of their children's coping; however, this transmission of maladaptive coping led to decreased adolescent binge drinking.

Hypothesis 4

A SEM framework was used to test the final hypothesis that determines whether there are unique effects of the three models, the recurring stress-coping, selection into stressful environments, and socialization of poor coping models, when they were entered into a single analytical model to predict adolescents' alcohol use at Wave 6. To avoid model misspecification, G2 young adult stress and coping were covaried with the G2 young adult maladaptive coping pattern. Similarly, G3 childhood stress and coping were also covaried with the G3 childhood

maladaptive coping pattern. The resulting model did not fit the data well ($\chi^2(48) = 57.10, p = .17, CFI = .94, TLI = .87, RMSEA = 0.04, SRMR = 0.06$). Consequently, modification indices were consulted to re-specify the model. Modification indices indicated that G3 childhood coping should be regressed on G1 AUD status ($M = 5.49$). The resulting model (see Figure 8; Table 8) fit the data well ($\chi^2(47) = 51.08, p = .32, CFI = .97, TLI = .95, RMSEA = 0.03, SRMR = 0.05$) but only explained a moderately significant amount of variance in G3 alcohol use ($R^2 = 0.12, p = .10$). Moreover, according to a likelihood ratio test, it significantly improved upon the model fit of the original model ($\chi^2(1) = 8.27, p < .01$). Overall, age and COA status were significant covariates in this overarching model. For example, older G2s were less likely to develop an AUD (standardized $\beta = -0.17, p < .05$). Moreover, older G3s were more likely to drink alcohol as adolescents (standardized $\beta = 0.21, p < .05$) and less likely to utilize avoidant coping strategies (standardized $\beta = -0.31, p < .05$) and demonstrate a maladaptive coping pattern (standardized $\beta = -0.33, p < .001$) in childhood. Similarly, having an alcoholic parent was associated with G2s being more likely to develop their own AUD (standardized $\beta = 0.53, p < .10$) and for G3s to drink more in adolescence (standardized $\beta = 0.43, p < .05$). Additionally, G3s were also more likely to use avoidant coping in childhood if their grandparents (G1s) had an AUD (standardized $\beta = 0.52, p < .01$).

When looking at the unique effect of the recurring stress-coping model, G2s were more likely to develop an AUD if they endorsed a greater number of avoidant coping strategies in young adulthood (standardized $\beta = 0.23, p < .001$). Furthermore, evidence was attained for the transmission of G2's maladaptive coping pattern in young adulthood to their G3 offspring in childhood (standardized $\beta = 0.26, p < .01$). In turn, G3s that utilized more avoidant coping strategies in the face of high levels of family stress were less likely to drink in adolescence

(standardized $\beta = -0.12$, $p < .10$). Even though a significant amount of variance in the G3 maladaptive coping pattern was explained ($R^2 = 0.18$, $p < .05$), the total and specific indirect effects from G2 maladaptive coping pattern did not significantly predict G3 alcohol use (standardized $\beta = -0.03$, $p > .05$), regardless of the specific meditational path explored. In sum, there does appear to be some evidence for a recurring maladaptive stress-coping pattern that has a relatively unique effect on G3 adolescent alcohol use. However, this effect is in the opposite direction hypothesized.

With regards to the stressful environment selection pathway, G2 young adult stress once again significantly predicted G3 childhood stress (standardized $\beta = 0.50$, $p < .001$). However, childhood stress did not significantly predict adolescent drinking (standardized $\beta = -0.05$, $p > .05$). Additionally, even though a significant amount of variance in G3 childhood stress was explained ($R^2 = 0.32$, $p < .01$), the total and specific indirect effects from G2 young adult stress did not significantly predict G3 alcohol use (standardized $\beta = -0.03$, $p > .05$), regardless of the specific meditational path explored. As such, despite the evidence for the transmission of stressful environments, the unique effect of the relation between parents' family stress in young adulthood and their offspring's alcohol use was limited.

In contrast, the socialization of coping pathway demonstrated that G2s who utilized more avoidant coping strategies in young adulthood were more likely to have offspring who also used avoidant coping in childhood (standardized $\beta = 0.24$, $p < .01$) and who later drank less in adolescence (standardized $\beta = -0.16$, $p < .10$). Moreover, a significant amount of variance in G3 childhood coping was explained ($R^2 = .22$, $p < .05$); however, once again, the total and specific indirect effects from G2 young adult stress did not significantly predict G3 alcohol use (standardized $\beta = 0.03$, $p > .05$), regardless of the specific meditational path explored. Therefore,

relative evidence exists for a socialization of poor coping model, in which there is a unique effect of the relationship between parents' avoidant coping in young adulthood to their offspring's alcohol use in adolescence via their offspring's childhood avoidant coping. However, again, the effect on adolescent alcohol use was in the opposite direction predicted. Overall, given the greater evidence for both the recurring stress-coping and socialization of poor coping models, it appears that avoidant coping is an important and unique predictor of adolescent alcohol use in both of these pathways.

A SEM framework was also used to test the final unique effects hypothesis to predict adolescents' binge drinking at Wave 6. To avoid model misspecification, G2 young adult stress and coping were covaried with the G2 young adult maladaptive coping pattern. Similarly, G3 childhood stress and coping were also covaried with the G3 childhood maladaptive coping pattern. The resulting model (see Figure 9; Table 9) explained a significant amount of variance in G3 binge drinking ($R^2 = 0.53$, $p = .001$). Overall, age and COA status were significant covariates in this overarching model. For example, older G2s were less likely to develop an AUD (standardized $\beta = -0.17$, $p < .10$). Moreover, older G3s were more likely to binge drink as adolescents (standardized $\beta = 0.55$, $p < .001$), more likely to experience childhood stress (standardized $\beta = 0.16$, $p < .10$), and less likely to utilize avoidant coping strategies (standardized $\beta = -0.27$, $p < .01$) and demonstrate a maladaptive coping pattern (standardized $\beta = -0.33$, $p < .001$) in childhood. Similarly, having an alcoholic parent was associated with G2s being more likely to develop their own AUD (standardized $\beta = 0.53$, $p < .05$) and for G3s to binge drink more in adolescence (standardized $\beta = 0.47$, $p < .10$).

When looking at the unique effect of the recurring stress-coping model, with each endorsement of an avoidant coping strategy during young adulthood, the log odds of G2s later

developing an AUD increased by 0.23 ($p < .05$). Furthermore, evidence was attained for the transmission of G2's maladaptive coping pattern in young adulthood to their G3 offspring in childhood (standardized $\beta = 0.25$, $p < .001$) with a significant amount of the variance in G3 childhood maladaptive coping pattern being explained ($R^2 = .17$, $p < .01$). However, the presence of a maladaptive coping pattern in childhood did not predict later adolescent binge drinking (standardized $\beta = -0.04$, $p > .05$). Therefore, it appears that there is only evidence for a recurrent maladaptive coping pattern across familial generations and not for this recurrent pattern's unique effect on adolescent binge drinking.

With regards to the stressful environment selection pathway, G2 young adult stress once again significantly predicted G3 childhood stress (standardized $\beta = 0.50$, $p < .001$), such that with every increase in family stressor experienced by G2s in young adulthood, the log odds of their children also experiencing a family stressor increased by .51 ($p < .001$). Additionally, a significant amount of variance in G3 childhood stress was explained ($R^2 = 0.32$, $p < .001$). However, once again, childhood stress did not significantly predict adolescent binge drinking (standardized $\beta = -0.01$, $p > .05$). As such, despite the evidence for the transmission of stressful environments, the unique effect of the relation between parents' family stress in young adulthood and their offspring's alcohol use is limited.

The socialization of coping pathway demonstrated that G2s' young adult coping predicted their children's coping such that with each G2 endorsement of an avoidant coping strategy during young adulthood, the log odds of their children also endorsing avoidant coping increased by 0.22 ($p < .01$). Of note, a significant amount of variance in G3 childhood coping was explained ($R^2 = .18$, $p < .01$). Furthermore, with each endorsement of an avoidant coping strategy in childhood, the log odds of G3s drinking 5 or more drinks in adolescence decreased by

.23 ($p < .10$). Therefore, evidence exists for the unique contribution of the socialization of poor coping model in the prediction of adolescent binge drinking. Knowing that this pathway seemed to have the most consistent contribution to adolescent binge drinking, it appears that socialization of avoidant coping has the greater unique effect in the prediction of adolescent binge drinking of the three pathways.

CHAPTER 4: DISCUSSION

By drawing on several theoretical perspectives, the current study tested whether an individual stress-coping model of alcohol use may recur in an intergenerational context to explain the transmission of problematic use within families or whether other factors that co-occur with AUDs are responsible for this intergenerational pattern of alcohol use and disorder. Findings provided moderate support for the study hypotheses and supported a more multifaceted approach to the understanding of the intergenerational transmission of stress, coping, and alcohol use across familial generations. Implications of these findings are discussed in turn below.

Intergenerational Transmission of Stress and Coping

Given the transmission of stress, coping, and maladaptive coping patterns across familial generations, support was partially attained for the stressful environment selection, socialization of poor coping, and recurring stress-coping models. More specifically, G2s who experienced higher levels of family stress and used more avoidant coping strategies, respectively, were more likely to have offspring that reported more family stress and use of avoidant coping strategies, respectively, during childhood. Additionally, sensitivity analyses of the recurring stress-coping model revealed that young adults who experienced high levels of family stress coupled with high levels of avoidant coping were more likely to have children who also experienced this same high stress-avoidant coping pattern. This result represents what is likely the first study to investigate the intergenerational transmission of this maladaptive coping pattern.

Previous work on stress proliferation gives credence to the stressful environment selection model such that stressors experienced by parents can proliferate across generations to

impact their children's health and well-being (Thoits, 2010; Turney, 2014). Moreover, this transmission of avoidant coping adds to the small body of literature investigating parents as socialization agents for their children's coping. In fact, to my knowledge, it is the first study to use parents' coping as young adults to predict their offspring's coping in childhood. This positive relationship between parental and child coping is also in response to general stressful situations and not to previously studied more specific contexts such as sickle cell disease (Kliewer & Lewis, 1995) and community violence (Kliewer et al., 2006). Therefore, the current study extends the understanding of the individual intergenerational transmission of stress, coping, and maladaptive stress-coping patterns, within families.

Intergenerational Pathways towards Alcohol Use and Disorder

Interestingly, even though support was attained for the individual intergenerational transmission of family stress and avoidant coping, these individual pathways negatively predicted adolescent G3 alcohol use and binge drinking in Wave 6. Moreover, G3s who demonstrated a maladaptive pattern of coping were less likely to engage in alcohol use and binge drinking in adolescence. Therefore, the recurring stress-coping, stressful environment selection, and socialization of poor coping models did not support the occurrence of problematic alcohol use and disorder. However, even though these findings ran contrary to the original study hypotheses, there are several possible explanations. First, in order to possibly explain the counterintuitive finding of the stressful environment selection model in which childhood stress predicted less adolescent alcohol use, one may want to further consider the model's proven transmission of familial stress from parents to children. More specifically, Seligman (1975) found that when everything else is held constant, predictable stressors are easier to cope with than unpredictable stressful life events. Perhaps, given the current findings supporting the partial

transmission of a stressful environment pathway across familial generations, G2 parents were able to help their G3 children anticipate these stressors, making them less taxing, and leading adolescents to drink less. Unfortunately, little research has studied this area of parent-child relationships (Power, 2004); however, it could be relevant in the current context and future research should further investigate this possible connection.

Alternatively, given the enduring nature of this familial stress, children and adolescents may find this experience to be expected and simultaneously, out of their control. Meaning, perceiving this greater and continued family stress to be uncontrollable may in fact lead to less emotional distress and resulting emotional dysregulation and problematic outcomes. In fact, previous research has suggested that avoidant coping is actually associated with better outcomes when the triggering stressor is considered to be uncontrollable (Creasey et al., 1995; Valentiner et al., 1994). Additionally, avoidant coping has previously been linked to children's more adaptive functioning at higher levels of stress (Gonzales, Tein, Sandler, & Friedman, 2001). As such, this could explain the recurring stress-coping model finding in which G3s who demonstrated a maladaptive coping pattern (e.g., simultaneously experienced high family stress and utilized high avoidant coping) were less likely to drink alcohol. Prior research has also found that childhood stressors only predicted about one-quarter of developing but not the persistence of substance use disorders (Green et al. 2010; McLaughlin et al. 2010). Thus, while important, the experience of childhood stress is not the only factor contributing to the development of AUDs.

For example, as already delineated earlier in this document, coping with said stress is also important. In the case of the current study, the socialization of poor coping pathway indicated that using a greater number of avoidant coping strategies in childhood predicted less alcohol use and binge drinking in adolescence. Interestingly, the evidence for the relationship between

avoidant coping and adjustment outcomes, such as alcohol use, has been mixed (Compas et al., 2001). In fact, literature discrepancies may stem from differences in the operational definition of avoidant coping as either negative (e.g., self-medicating with substances) or as distracting (e.g., going shopping or visiting a friend) (Windle & Windle, 1996). For instance, previous researchers (Cooper et al., 1988; Windle & Windle, 1996) have found a positive relationship between “negative” avoidant coping and alcohol use. In contrast, Gonzales and colleagues (2001) demonstrated that early adolescent girls’ use of distraction coping strategies buffered the relationship between family stress and conduct problems. Given that the majority of the items comprising the present investigation’s avoidant coping construct could be described as distracting, perhaps the socialization pathway’s unexpected inverse relationship with adolescent alcohol use and binge drinking can be attributed to this particular operationalization of avoidant coping. According to this premise, my original hypotheses predicting a positive association between avoidant coping and problematic alcohol use within the socialization and recurring stress-coping models were correct and only fell victim to an incompatible measurement of avoidant coping.

Another explanation could be that the current findings of a negative association between avoidant coping and adolescent alcohol use is accurate, and the original hypothesized mechanism was incorrect. If this is the case, then alternative mechanisms should be considered. For example, Glyshaw and colleagues (1989) demonstrated an inverse relationship between “distracting” avoidant coping and anxiety. Unsurprisingly, previous work has delineated a significant association between exposure to a variety of stressors and the development of internalizing problems, specifically anxiety, in youth and adults (Amato, 2001; Schneiders et al., 2006; Sheidow et al., 2014). When considering the relationship between anxiety and alcohol use,

mixed results have been presented (Blumenthal, Leen-Feldner, Badour, & Babson, 2011 2011). For example, in early adolescence, anxiety may in fact protect youth from initiating alcohol use. Conversely, once an adolescent has begun drinking, anxiety will promote the escalation of alcohol use (Colder, Chassin, Lee, & Villalta, 2010). Given that the G3s in the current sample were, on average, still in early adolescence (mean age=14.2 years) and that most had yet to initiate alcohol use, perhaps the increased stress experienced by these G3s caused protective anxiety against initiating alcohol use. Likewise, it could be that as these adolescents age and consequently are more developmentally likely to begin drinking, the original stress-coping hypothesis may emerge as this increased stress and resulting anxiety intensifies their drinking. Overall, this idea aligns quite nicely with the intergenerational transmission models of affective disorder that suggest that living with a parent with an affective disorder not only entails a genetic risk but may also increase exposure to potential environmental risk factors, such as an increase in perceived stress and the parental modeling of anxious behaviors (Eley et al., 2015). Therefore, the intergenerational transmission of psychopathology may be another possible mechanism in the transmission of problematic alcohol use and disorder.

A final speculation may be that these distraction strategies are the initial steps of distress tolerance that are then followed by more active coping strategies. This speculation may be especially true given Sandler and colleagues' (1994) insistence that the impact of any single coping strategy is best considered in the context of other coping strategies used. Knowing that children may use different types of coping strategies in the face of stress (Gonzales et al., 2001), it is unclear how these strategies may integrate with one another. Therefore, future research should look to further delineate the temporal precedence and possible interactions of different coping strategies so as to more clearly understand how avoidance strategies may impact both

positive and negative outcomes (Rafnsson, Jonsson, & Windle, 2006).

In addition to the aforementioned counterintuitive results with regards to problematic alcohol use and disorder, several findings concerning COA status aligned with previous literature. For example, G3 COAs were especially likely to face high levels of family stress coupled with high levels of avoidant coping—a prime depiction, especially for COAs, of the necessary precursors to a stress-coping model of alcohol use (Wills et al., 2001). Moreover, parent and grandparent AUD status also served as important predictors of problematic G3 alcohol use over time. For instance, having an alcoholic parent was associated with G3s being more likely to not only drink, but to binge drink as adolescents. Additionally, those G2s who had alcoholic parents were also more likely to develop an AUD themselves. Overall, these results are unsurprising given COAs' well documented increased risk for developing problematic alcohol use (Chassin, Pitts, DeLucia, & Todd, 1999).

Knowing that there were several positive associations with increased alcohol use and disorder, a return to the overarching question of how alcohol use and disorder is intergenerationally transmitted is necessary. The current investigation originally proposed that an individual stress-coping model of alcohol use recurs in an intergenerational context to explain this transmission. Complementary hypotheses asserting that selection of stressful environments and socialization of poor coping were also offered as alternative mechanisms. Hypothesis 4 allowed for the investigation of the unique effects of each of these proposed models. According to these analyses, evidence of the intergenerational transmission of stress, coping, and maladaptive coping patterns was once again provided. Also, G3s who used more avoidant coping strategies in childhood were less likely to use and binge drink alcohol in adolescence. These findings were unsurprising given the results of previous analyses. However, within one of the

individual pathways of the recurrent stress-coping model, one notable result is the positive association between G2 avoidant coping in young adulthood and G2s' later development of an AUD. This finding speaks to the significant impact of avoidant coping on alcohol use and disorder. Perhaps, the negative association found between avoidant coping and alcohol use in the socialization and recurrent stress-coping models is a reflection of the timing of the relationship. It could be that the use of avoidant coping is protective in early to mid-adolescence with regards to problematic alcohol use. However, according to this individual pathway, the continued use of avoidant coping strategies into young adulthood increases the likelihood of developing an AUD later in life. As such, one could speculate that the proposed recurring stress-coping, stressful environment, and/or socialization mechanisms do not fully emerge until young adulthood. Moreover, given the evidence supporting the intergenerational transmission of stress, coping, and maladaptive stress-coping patterns, one could argue that each of the three proposed models are not competing with each other. Instead, they could comprise a broader type of developmental cascade that sees the negative correlated constraints of each of the three mechanisms interact with each other to increase the likelihood of developing and transmitting alcohol use and disorder. Therefore, future research should consider investigating the timing of the emergence of these mechanisms in an intergenerational context.

Implications

In terms of clinical intervention, the present results underscore the importance of consistently assessing the presence of familial stress and coping for both parents and youth. Knowing that both of these constructs can be passed down across familial generations and are associated with a host of adaptive and maladaptive outcomes (Amato, 2001; Bifulco et al., 2002; Hammen et al., 2012; Hankin, 2005; Power, 2004; Sheidow et al., 2014). Additionally, even

though it was in the opposite direction hypothesized, the current study did demonstrate a buffering relationship between familial stress, avoidant coping, and adolescent alcohol use. Meaning, the use of what could be considered more distraction-based coping strategies may reduce the likelihood of using alcohol even in the face of higher stress levels. In terms of treatment, there are currently several programs intended to reduce adolescent alcohol use by working from a stress and coping model (Kaminer, Burlison, & Goldberger, 2002; McGillicuddy, Rychtarik, & Papandonatos, 2015). However, many of the coping skills taught in these programs typically involve more problem-solving and cognitive restructuring techniques. While undoubtedly beneficial, being able to offer adolescents, especially those whose cognitive capabilities may not yet have completely matured, a more behaviorally-oriented coping strategy may be an equally appropriate option. As such, the current findings indicate that more distraction-based coping strategies may offer a protective buffer between stress and adolescent alcohol use. In fact, when studying adolescents in residential treatment for substance use disorders, Wei and colleagues (2011) found a significant association between adolescents who endorsed higher levels of avoidant-behavioral coping (e.g., going to a movie, listening to music) and increases in motivation to change their substance use. Previous findings in the coping literature indicate that behavioral strategies that allow an individual to be distracted or help him physically or cognitively avoid a stressor may actually be adaptive (Folkman & Lazarus, 1980). For patients in residential treatment programs, learning more avoidant-behavioral or distraction-based coping skills may help them tolerate their cravings to use alcohol. Moreover, providers who work with substance-abusing adolescents in a variety of treatment contexts (e.g., residential, partial, outpatient) should strive to work closely with their patients to identify those coping strategies, behavioral or otherwise, that may help them in problematic contexts including, home,

school, and/or social environments.

Limitations and Future Directions

Even though the current study represents several examples of previously unstudied intergenerational processes, several limitations should also be noted. First, as already stated, though great pains were taken to create the avoidant coping item set, there are many ways in which to define and measure avoidant coping. This lack of unanimity regarding the core categories of coping leads to a number of negative outcomes, including confusion regarding the overabundance of coping measures; the difficulty in comparing, contrasting and synthesizing results across studies; and struggles with accounting for fundamental individual differences, such as age and family history of alcoholism, in coping processes (Compas et al., 2001). As such, it stands to reason that future researchers should at least attempt to consolidate previous conceptualizations into an overarching developmental framework of coping as “coping needs more detailed specification...because of the bewildering richness of behavior relevant to it” (Pearlin & Schooler, 1978, p. 4). Of course, this is by no means an easy feat. This framework must integrate the unique, related, and dynamic dispositional and situational coping responses individuals employ with the idea that coping is an adaptive process that is integral to individuals’ mental health, physical health, and overall, global functioning (Skinner et al., 2003).

Second, given G3s’ young age during Wave 5 and the likelihood that they may not have been aware of all occurring family stressors, the decision was made for the current study to only use G2s’ report of G2-G3 family stress items to measure G3s’ stress during Wave 5. Despite this justification, one must wonder about whether G3s would have endorsed the same stressful life events as their parents. Correspondingly, the G3s in Waves 5 and 6 were younger than their earlier G2 counterparts. Perhaps, the proposed mediators or moderators (e.g., anxiety) of

escalating alcohol use had yet to emerge in these younger G3s. As such, replication of the current study findings is needed to confirm the directionality of the relationships between stress, coping, and alcohol use.

Finally, similar to the necessity of establishing a more universal definition and measurement of coping, the same requirement can be applied to the stress construct. Even though there is relatively less confusion surrounding the operationalization of stress, there is one particular issue that is especially important to consider: does a researcher's chosen measure of stress operate under a dispositional or situational approach to coping? In other words, is a particular measure more attuned to the way a person typically copes in response to general stressful events or is it attempting to measure the use of different coping strategies in the face of a particular stressor? The current study independently measured dispositional avoidant coping and a specific type of stress (e.g., familial). As such, future studies that are investigating the interaction between avoidant coping and familial stress should perhaps utilize a more situational approach in order to more accurately describe the relationship between these constructs.

Conclusion

In conclusion, the current study is a crucial first step in investigating how stress, coping, and alcohol use is transmitted across familial generations. Evidence for the individual intergenerational transmission of family stress and avoidant coping, and for the interaction between these constructs, was attained. Interestingly, these individual pathways negatively predicted adolescent G3 alcohol use and binge drinking. These findings suggest that, while important, the experience of stress and coping are not the only factors contributing to the development of AUDs.

Table 1. Descriptive statistics for key predictor and outcome variables.

		1	2	3	4	5	6	7	8	9	10	11	12	13	14	15	16	17	18	19	20
1	G1 Lifetime AUD	--																			
2	G2 W5 AUD	.18 +	--																		
3	G2 W6 SES	.00	-.21 *	--																	
4	G2 W6 Age	-.08	-.16	.09	--																
5	G3 W6 Age	.20 +	-.08	-.19 +	.08	--															
6	G2 Gender	-.14	-.11	-.02	.11	-.22 *	--														
7	G3 Gender	.04	.07	.02	-.08	-.25 **	.04	--													
8	G2 Ethnicity	.14	-.04	-.27 **	-.04	.43 ***	-.13	.09	--												
9	G3 Ethnicity	.05	.05	-.03	-.00	.25 *	-.11	.01	.37 ***	--											
10	G2 COA Status	.51 ***	.15	-.04	-.10	.24 *	-.23 *	-.04	.19 +	.12	--										
11	G3 COA Status	.35 **	.31 **	-.18 +	-.04	.18 +	-.02	.06	.04	.08	.31 **	--									
12	G2 W4 Stress	.11	.06	.05	-.05	.05	-.12	-.02	.15	-.03	.05	-.10	--								
13	G3 W5 Stress	.15	.06	.02	-.11	.17 +	-.02	-.11	.16	.05	-.10	-.12	.59 ***	--							
14	G2 W4 Coping	-.10	.10	.07	.13	.05	.08	.00	-.01	.04	.03	-.00	-.21 *	-.21 *	--						
15	G3 W5 Coping	.07	-.11	.16	-.03	-.28 **	.10	-.03	-.21 *	-.14	.03	-.19 +	-.07	-.09	.17 +	--					
16	G2 W4 Mal-adaptive	.10	-.08	.17 +	-.06	.09	-.16	.01	.17 +	.13	.08	-.03	.27 **	.20 *	.32 **	-.09	--				
17	G3 W5 Mal-adaptive	.07	.05	.19 +	-.12	-.29 **	.07	-.08	-.10	-.20 *	-.06	-.06	.21 *	.39 ***	-.03	.43 ***	.17 +	--			
18	G2 W4 AU	.04	.17 +	-.23 *	-.12	-.13	.15	-.06	.14	.05	.11	.10	.01	.04	-.05	.01	.05	-.05	--		
19	G3 W6 AU	.10	.24 *	-.21	-.25 +	.24 *	-.19 +	-.09	.05	.12	.10	.22 *	-.00	-.10	-.04	.01	-.11	-.14	.09	--	
20	G3 W6 Binge	.09	.03	-.10	-.02	.42 ***	-.17 +	-.08	.16	.06	.18 +	.22 *	-.02	.01	-.16	-.25 *	-.18 +	-.17 +	.13	.63 ***	--
	Mean	.25	.18	5.94	33.3	14.2	1.38	1.49	1.39	1.66	.50	.33	.65	.81	11.0	7.50	.14	.29	8.56	.59	.19
	SD	.43	.38	2.48	2.74	1.17	.49	.50	.70	1.16	.50	.47	1.04	1.25	1.45	.93	.35	.45	10.6	2.46	.39

Table 2. Sensitivity analysis of hypothesis 1 predicting adolescent alcohol use from a recurring stress-coping model.

PREDICTORS	OUTCOMES					
	G3 Alcohol Use		G3 Childhood Maladaptive Coping Pattern		G2 Young Adult Maladaptive Coping Pattern	
	β	SE	β	SE	β	SE
G3 Childhood Maladaptive Coping Pattern	-0.09*	.04				
G3 Age	0.17*	.09	-0.34***	.09		
G3 Gender	-0.13	.23	-0.34	.22		
G3 Ethnicity	-0.01	.04	-0.05	.10		
G3 COA Status	0.40*	.19	0.05	.21		
G2 Young Adult Maladaptive Coping Pattern	-0.11**	.04	0.21*	.11		
G2 Age					-0.06	.09
G2 Gender					-0.26	.20
G2 Ethnicity					0.24	.17
G2 COA Status					0.14	.25
G2 SES					0.09*	.04
R ²	0.10	.07	0.16*	.07	0.09	.06

[†]p < .10, *p < .05, **p < .01, ***p < .001

Table 3. Hypothesis 1 predicting adolescent binge drinking from a recurring stress-coping model.

	OUTCOMES									
	G3 Binge Drinking		G3 Childhood Maladaptive Coping Pattern		G2 AUD		G2 Alcohol Use		G2 Young Adult Maladaptive Coping Pattern	
PREDICTORS	β	SE	β	SE	β	SE	β	SE	β	SE
G3 Childhood Maladaptive Coping Pattern	-0.18	.20								
G3 Age	0.52***	.13	-0.32**	.10						
G3 Gender	0.06	.31	-0.33⁺	.20						
G3 Ethnicity	-0.11	.19	-0.02	.10						
G3 COA Status	0.53*	.27	0.04	.10						
G2 Young Adult Maladaptive Coping Pattern							0.09	.10		
G2 Age					-0.13	.10	-0.11	.10	-0.06	.10
G2 Gender					0.24	.20	0.40*	.21	-0.26	.21
G2 Ethnicity					-0.19	.15	0.13	.15	0.24	.15
G2 COA Status					0.48*	.23	0.10	.25	0.14	.24
G2 SES					-0.08*	.04	-0.09*	.04	0.09*	.04
G2 Alcohol Use					0.09	.11				
R ²	0.45**	.15	0.11⁺	.06	0.13*	.07	0.11⁺	.06	0.09	.06

⁺p < .10, *p < .05, **p < .01, ***p < .001

Table 4. Hypothesis 2 predicting adolescent alcohol use from a selection into stressful environments model.

PREDICTORS	OUTCOMES					
	G3 Alcohol Use		G3 Childhood Stress		G2 Young Adult Stress	
	β	SE	β	SE	β	SE
G3 Childhood Stress	-0.15**	.05				
G3 Age	0.24**	.08	0.14	.10		
G3 Gender	-0.15	.21	-0.13	.18		
G3 Ethnicity	0.01	.04	0.04	.10		
G3 COA Status	0.37*	.18	-0.19	.16		
G2 Young Adult Stress	0.12	.11	0.57***	.11		
G2 Age					-0.04	.10
G2 Gender					-0.22	.18
G2 Ethnicity					-0.07	.15
G2 COA Status					0.25	.30
G2 SES					0.01	.05
R ²	0.15*	.07	0.36**	.11	0.03	.03

⁺p < .10, *p < .05, **p < .01, ***p < .001

Table 5. Hypothesis 2 predicting adolescent binge drinking from a selection into stressful environments model.

PREDICTORS	OUTCOMES					
	G3 Binge Drinking		G3 Childhood Stress		G2 Young Adult Stress	
	β	SE	β	SE	β	SE
G3 Childhood Stress	-0.00	.16				
G3 Age	0.57***	.12	0.14	.09		
G3 Gender	0.10	.32	-0.13	.17		
G3 Ethnicity	-0.10	.20	0.04	.09		
G3 COA Status	0.53⁺	.28	-0.19	.18		
G2 Young Adult Stress			0.57***	.07		
G2 Age					-0.04	.10
G2 Gender					-0.22	.21
G2 Ethnicity					-0.07	.16
G2 COA Status					0.25	.25
G2 SES					0.01	.04
R ²	0.41**	.14	0.36***	.08	0.03	.03

⁺p < .10, *p < .05, **p < .01, ***p < .001

Table 6. Hypothesis 3 predicting adolescent alcohol use from a socialization of poor coping model.

PREDICTORS	OUTCOMES					
	G3 Alcohol Use		G3 Childhood Coping		G2 Young Adult Coping	
	β	SE	β	SE	β	SE
G3 Childhood Coping	0.12	.08				
G3 Age	0.23*	.09	-0.29*	.13		
G3 Gender	-0.08	.22	-0.20	.25		
G3 Ethnicity	0.01	.05	-0.14	.12		
G3 COA Status	0.43*	.20	-0.20	.12		
G2 Young Adult Coping	-0.07	.09	0.21*	.10		
G2 Age					0.10	.08
G2 Gender					0.16	.19
G2 Ethnicity					0.10	.13
G2 COA Status			0.56*	.22	-0.25	.32
G2 SES					0.04	.04
R ²	0.11	.07	0.21⁺	.11	0.04	.06

⁺p < .10, *p < .05, **p < .01, ***p < .001

Table 7. Hypothesis 3 predicting adolescent binge drinking from a socialization of poor coping model.

PREDICTORS	OUTCOMES					
	G3 Binge Drinking		G3 Childhood Coping		G2 Young Adult Coping	
	β	SE	β	SE	β	SE
G3 Childhood Coping	-0.24*	0.12				
G3 Age	0.56***	.11	-0.25*	.10		
G3 Gender	0.02	.30	-0.17	.19		
G3 Ethnicity	-0.25	.20	-0.14	.10		
G3 COA Status	0.48⁺	.26	-0.28	.20		
G2 Young Adult Coping			0.19*	.09		
G2 Age					0.11	.10
G2 Gender					0.14	.21
G2 Ethnicity					0.12	.16
G2 COA Status					-0.23	.25
G2 SES					0.03	.04
R ²	0.53**	.15	0.17*	0.07	0.04	.04

⁺p < .10, *p < .05, **p < .01, ***p < .001

Table 8. Unique effects of each hypothesized model predicting adolescent alcohol use.

	OUTCOMES															
	G3 Alcohol Use		G3 Childhood Mal. Coping Pattern		G3 Childhood Stress		G3 Childhood Coping		G2 AUD		G2 YA Mal. Coping Pattern		G2 Young Adult Stress		G2 Young Adult Coping	
PREDICTORS	β	SE	β	SE	β	SE	β	SE	β	SE	β	SE	β	SE	β	SE
G3 Maladaptive Coping Pattern	-.12⁺	.07														
G3 Childhood Stress	-.05	.05														
G3 Childhood Coping	-.16⁺	.09														
G3 Age	.21*	.10	-.33***	.08	.16	.11	-.31*	.13								
G3 Gender	-.12	.22	-.33	.22	-.13	.19	-.20	.25								
G3 Ethnicity	.02	.04	-.06	.10	.05	.10	-.13	.11								
G3 COA Status	.43*	.21	.01	.20	-.28	.16	-.33	.27								
G2 Maladaptive Coping Pattern			.26**	.09					-.16	.10						
G2 Young Adult Stress					.50***	.11			.13	.13						
G2 Young Adult Coping							.24**	.08	.23***	.06						
G2 Age									-.17*	.07	-.06	.09	-.04	.10	.11	.09
G2 Gender									.23	.22	-.25	.20	-.22	.18	.14	.17
G2 Ethnicity									-.16	.13	.24	.17	-.07	.15	.12	.13
G2 COA Status							.52**	.18	.53⁺	.31	.13	.24	.25	.30	-.23	.29
G2 SES									-.09*	.04	.09*	.04	.01	.05	.03	.04
G2 AUD			.06	.12	.09	.08	-.13	.12								
R ²	.12⁺	.08	.18*	.08	.32**	.09	.22*	.11	.18*	.08	.09	.05	.03	.03	.04	.05

⁺p < .10, *p < .05, **p < .01, ***p < .001

Table 9. Unique effects of each hypothesized model predicting adolescent binge drinking.

	OUTCOMES															
	G3 Binge Drinking		G3 Childhood Mal. Coping Pattern		G3 Childhood Stress		G3 Childhood Coping		G2 AUD		G2 YA Mal. Coping Pattern		G2 Young Adult Stress		G2 Young Adult Coping	
PREDICTORS	β	SE	β	SE	β	SE	β	SE	β	SE	β	SE	β	SE	β	SE
G3 Maladaptive Coping Pattern	-.04	.25														
G3 Childhood Stress	-.01	.18														
G3 Childhood Coping	-.23⁺	.14														
G3 Age	.55^{***}	.14	-.33^{***}	.10	.16⁺	.09	-.27^{**}	.10								
G3 Gender	.03	.31	-.34⁺	.19	-.13	.18	-.17	.19								
G3 Ethnicity	-.24	.21	-.07	.10	.05	.09	-.13	.10								
G3 COA Status	.47⁺	.28	.01	.21	-.28	.19	-.21	.21								
G2 Maladaptive Coping Pattern			.25^{***}	.08					-.16	.11						
G2 Young Adult Stress					.51^{***}	.07			.13	.10						
G2 Young Adult Coping							.22^{**}	.08	.23[*]	.10						
G2 Age									-.17⁺	.09	-.06	.10	-.04	.10	.11	.10
G2 Gender									.23	.20	-.25	.21	-.22	.21	.14	.21
G2 Ethnicity									-.16	.15	.24	.15	-.07	.16	.12	.16
G2 COA Status									.53[*]	.22	.13	.24	.25	.25	-.23	.25
G2 SES									-.09[*]	.04	.09[*]	.04	.01	.04	.03	.04
G2 AUD			.06	.10	.09	.09	-.11	.10								
R ²	.53^{***}	.15	.17^{**}	.07	.32^{***}	.08	.18^{**}	.07	.18[*]	.07	.09	.06	.03	.03	.04	.04

⁺p < .10, *p < .05, **p < .01, ***p < .001

Figure 1. Hypothesis 1 testing a recurring stress-coping model of alcohol use and disorder.

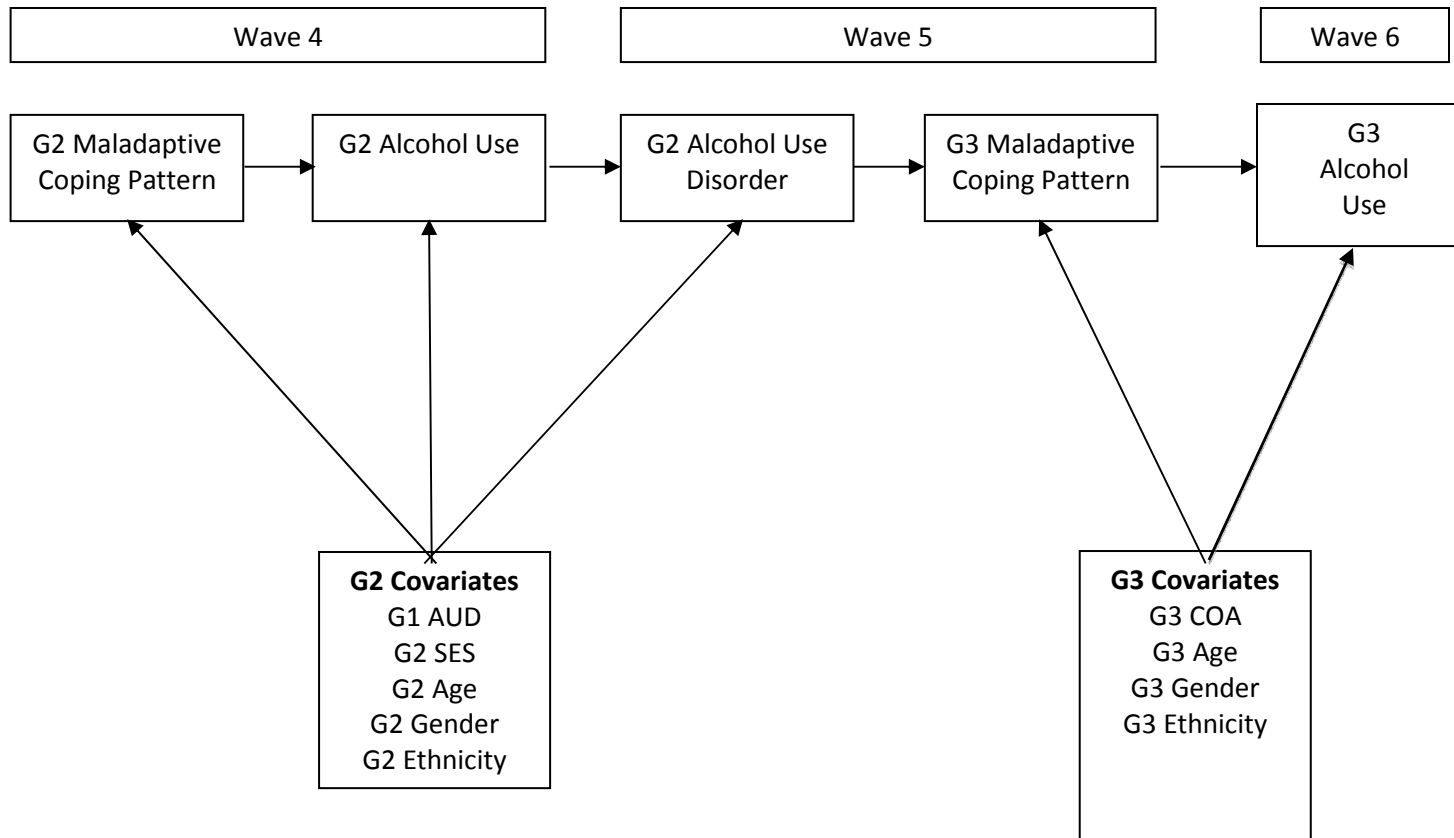
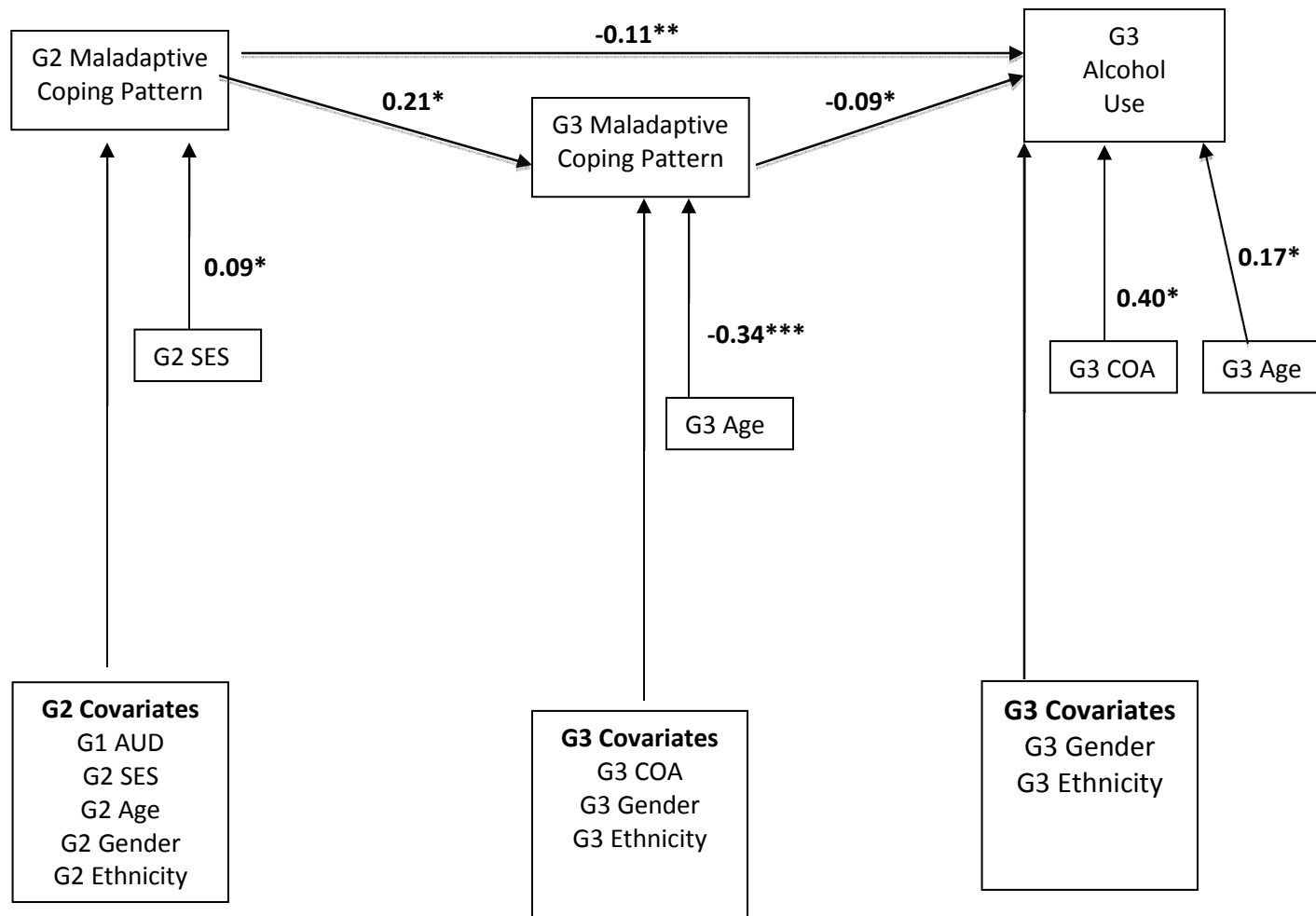
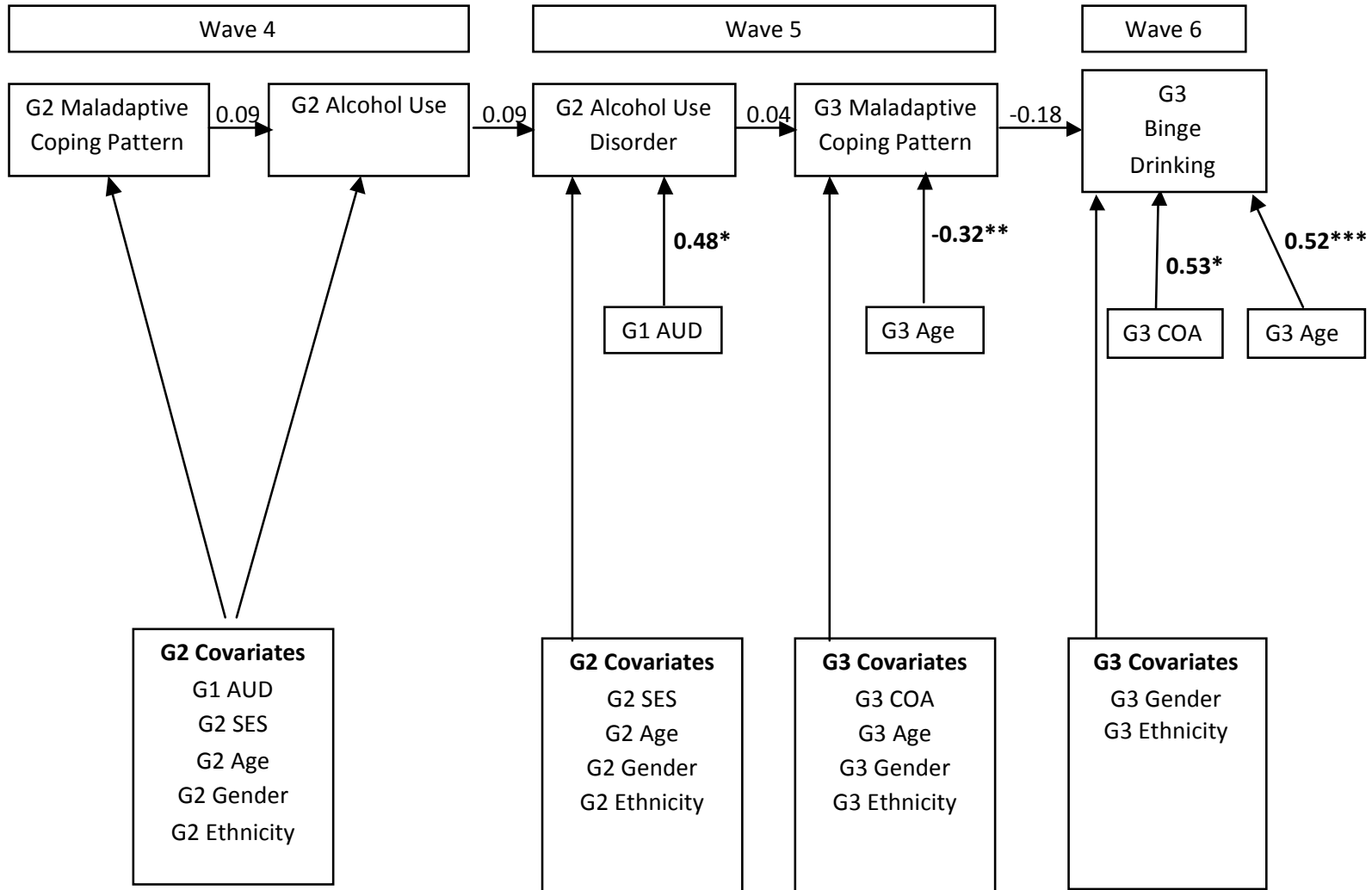


Figure 2. Sensitivity analysis of hypothesis 1 predicting adolescent alcohol use from a recurring stress-coping model.



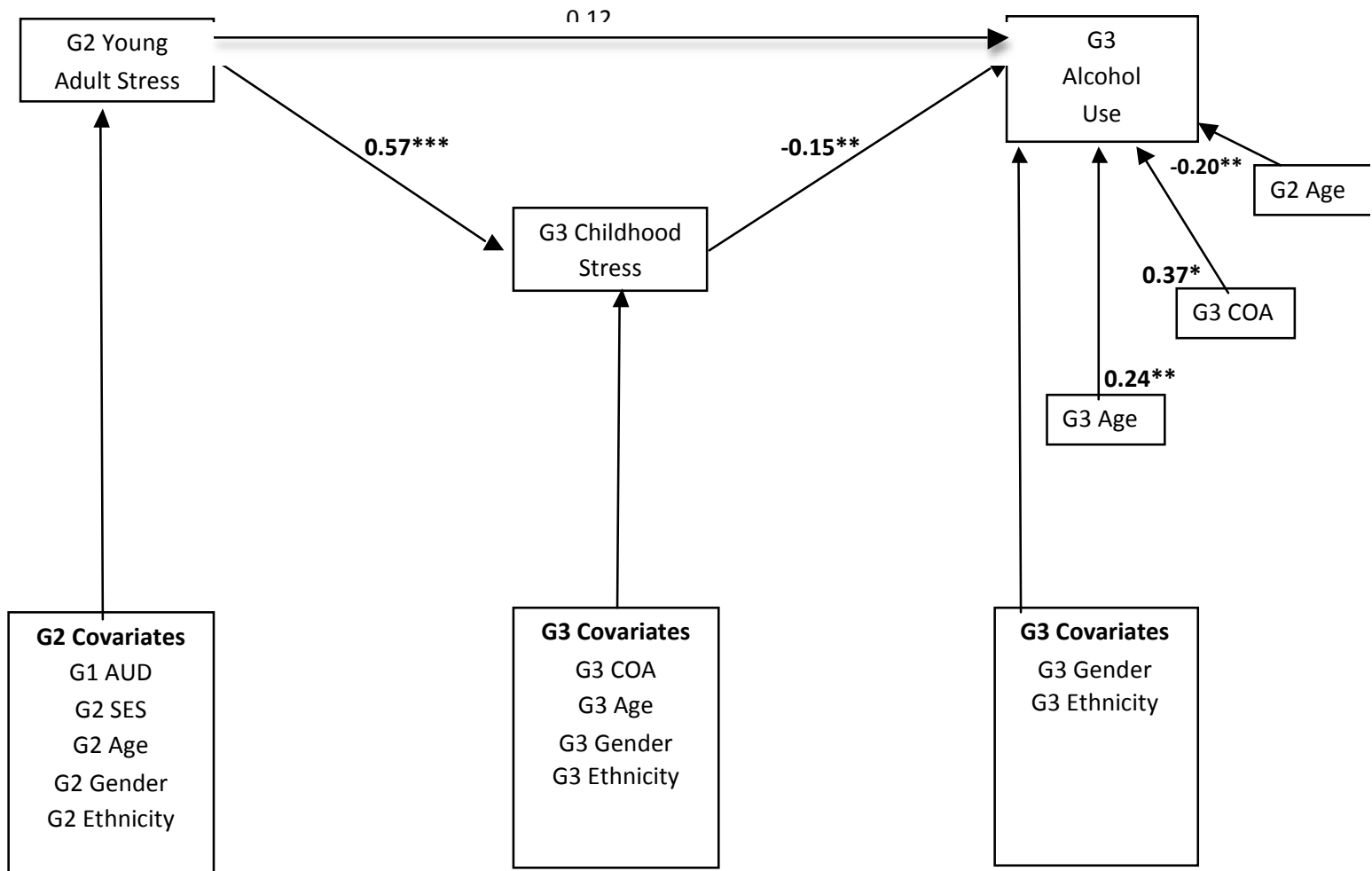
[†]p < .10, *p < .05, **p < .01, ***p < .001

Figure 3. Hypothesis 1 predicting adolescent binge drinking from a recurring stress-coping model.



⁺p < .10, *p < .05, **p < .01, ***p < .001

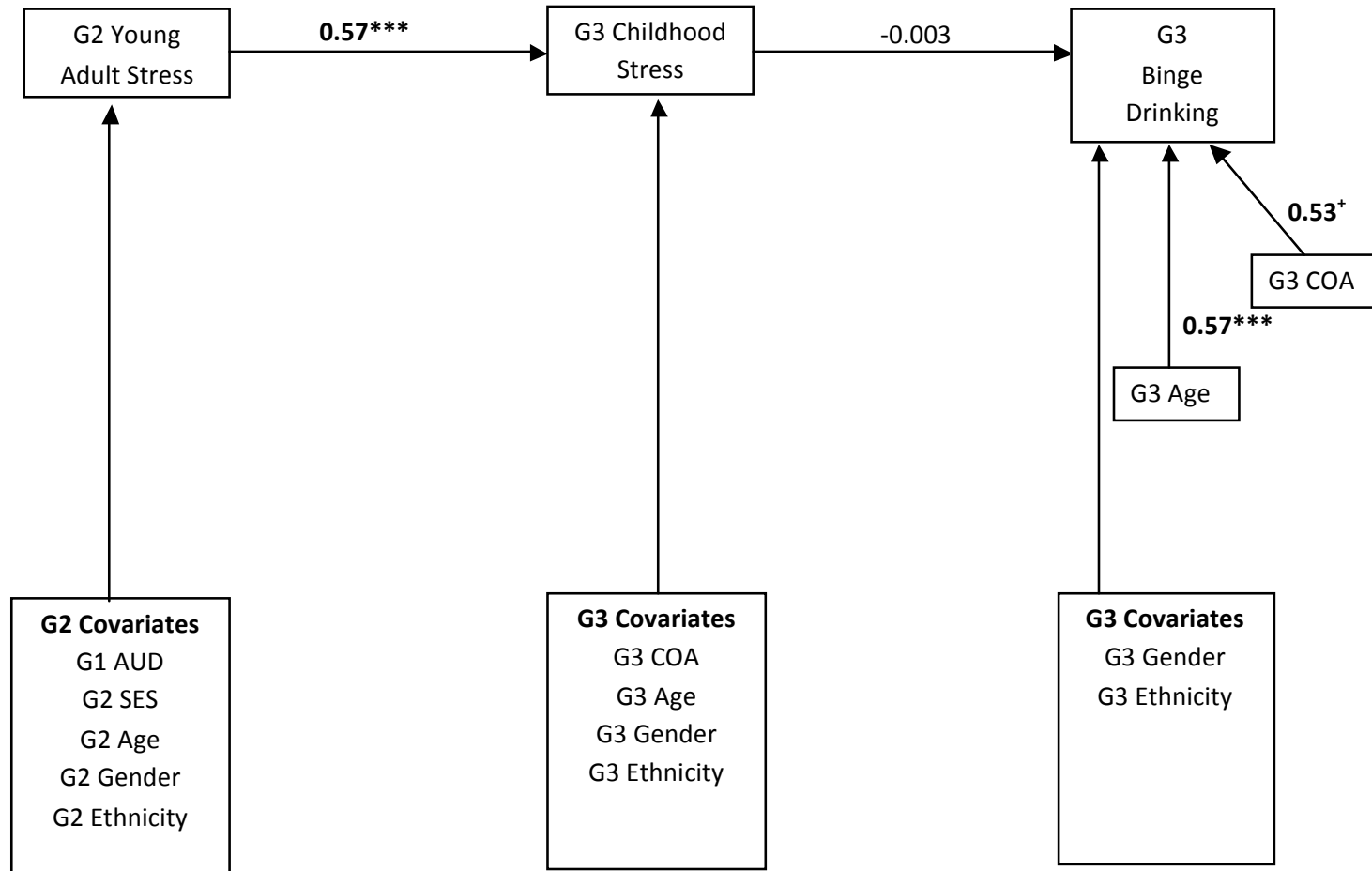
Figure 4. Hypothesis 2 predicting adolescent alcohol use from a selection into stressful environments model.



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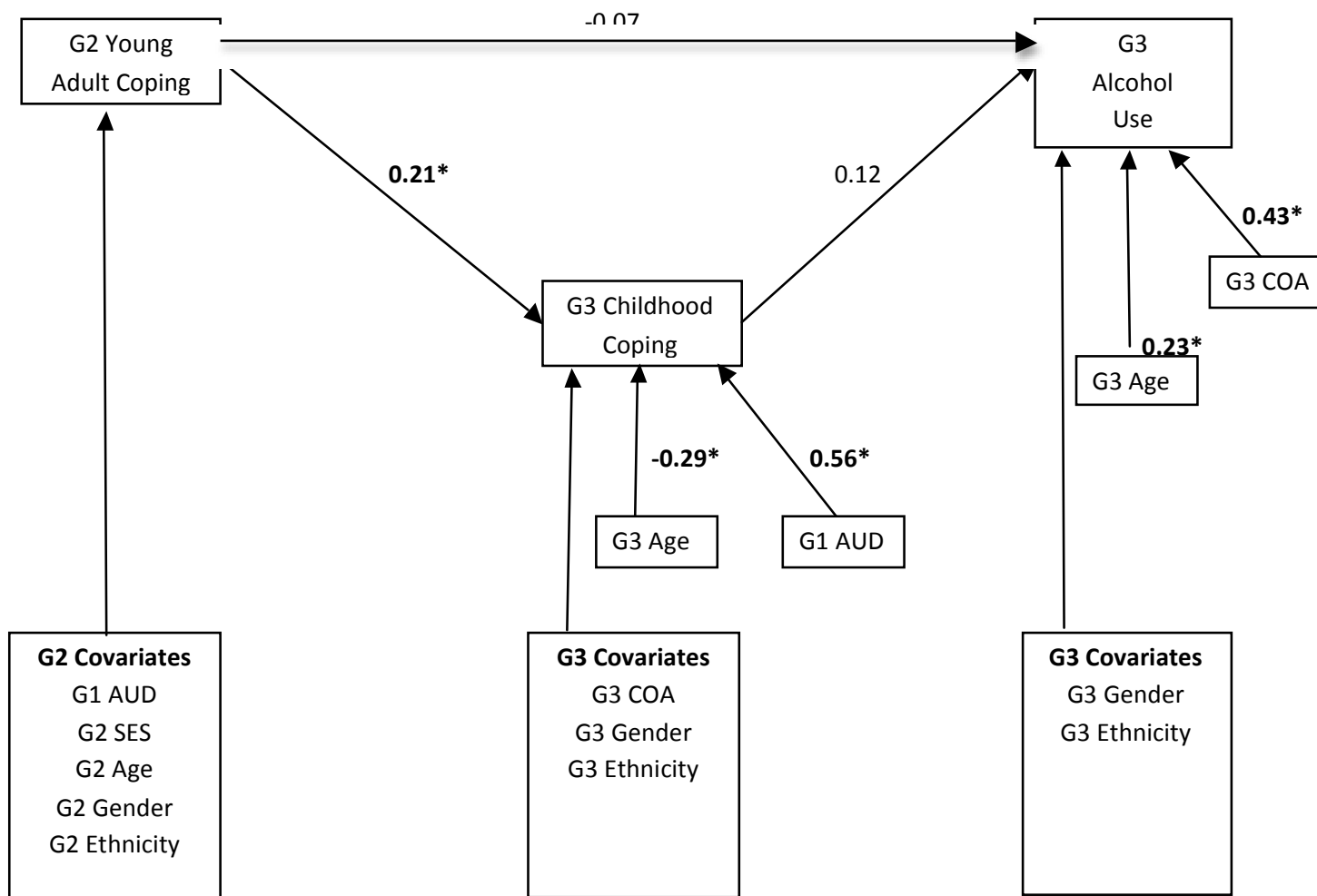
⁺p < .10, *p < .05, **p < .01, ***p < .001

Figure 5. Hypothesis 2 predicting adolescent binge drinking from a selection into stressful environments model.



⁺p < .10, *p < .05, **p < .01, ***p < .001

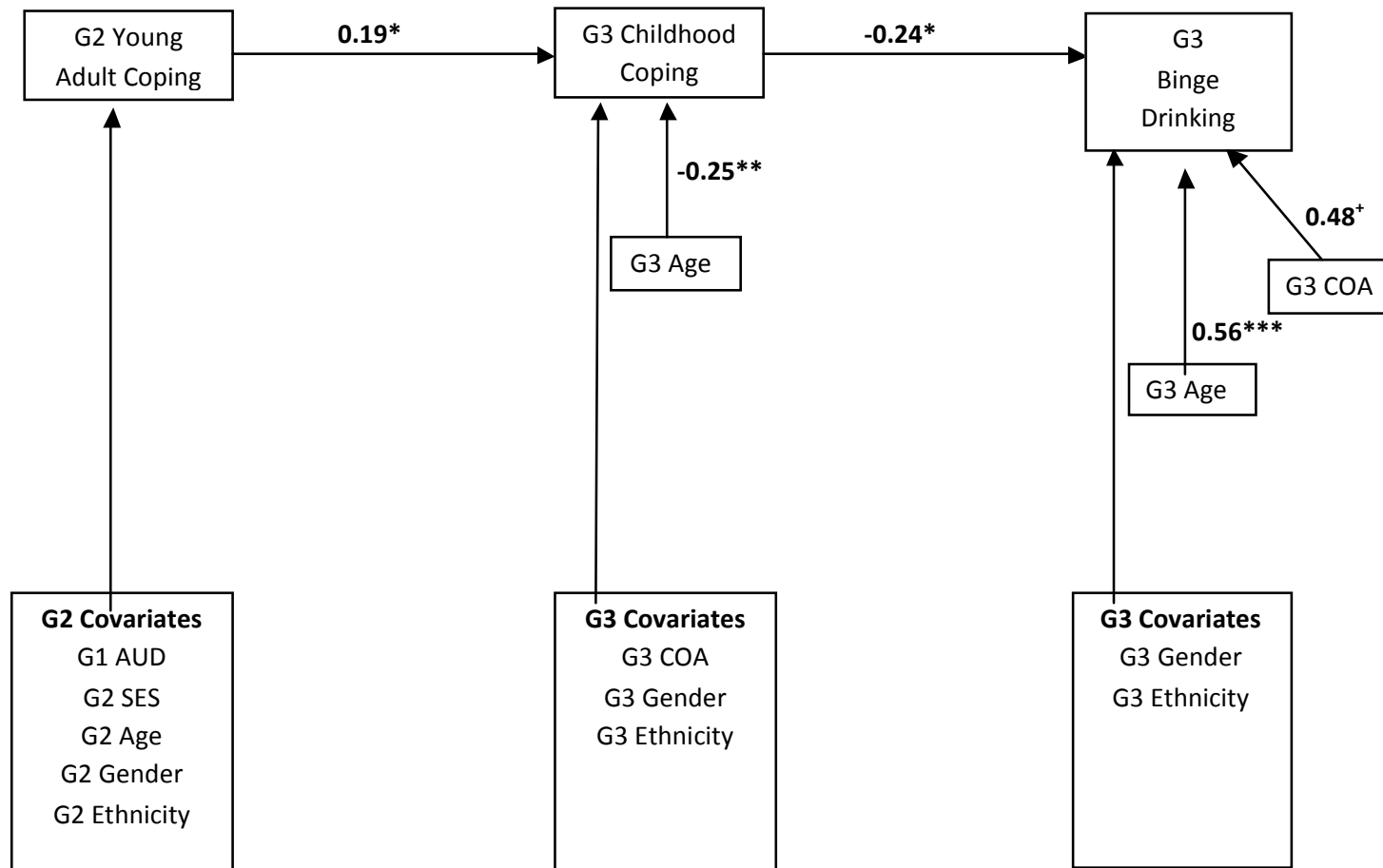
Figure 6. Hypothesis 3 predicting adolescent alcohol use from a socialization of poor coping model.



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⁺p < .10, *p < .05, **p < .01, ***p < .001

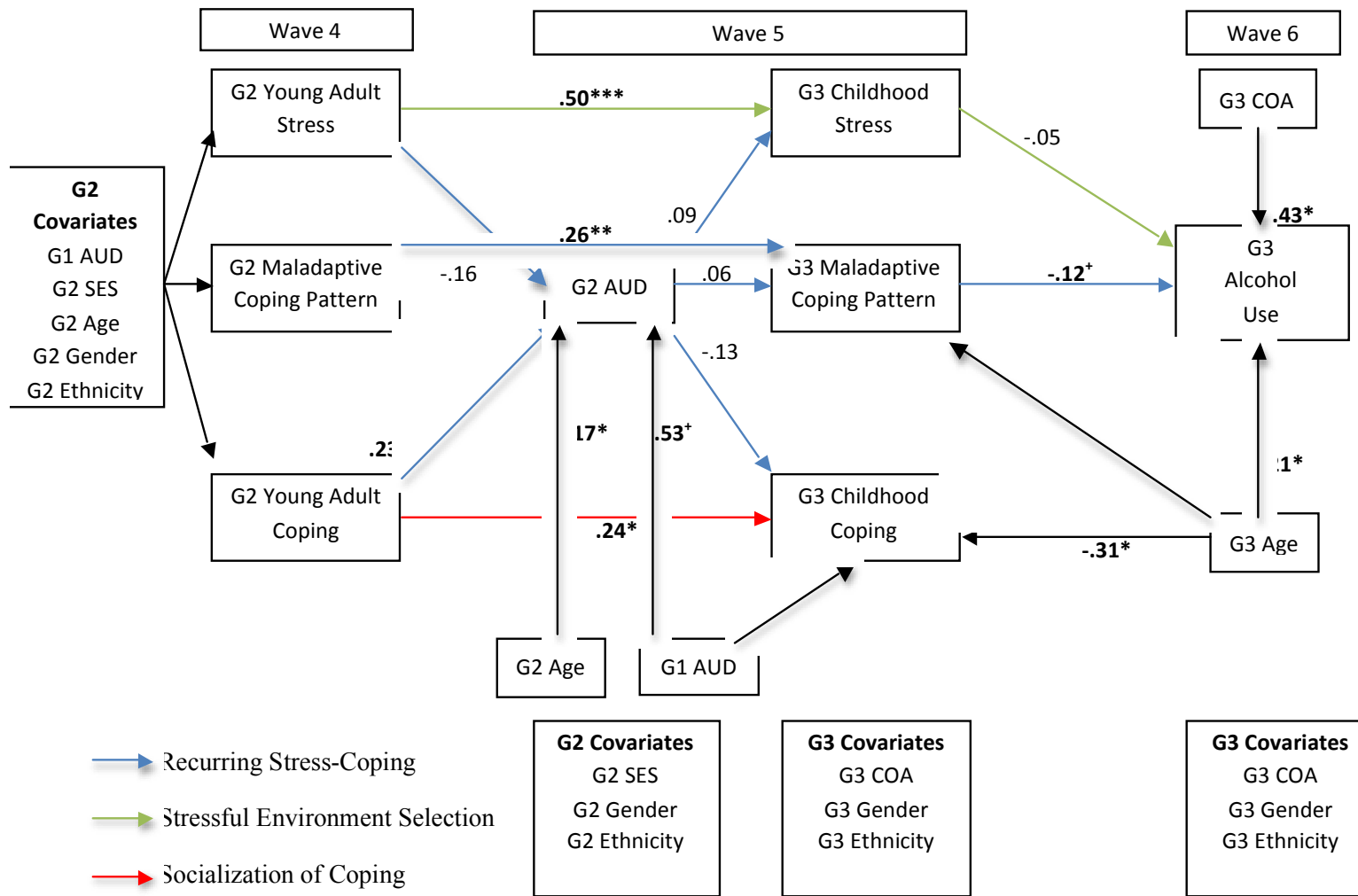
Figure 7. Hypothesis 3 predicting adolescent binge drinking from a socialization of poor coping model.



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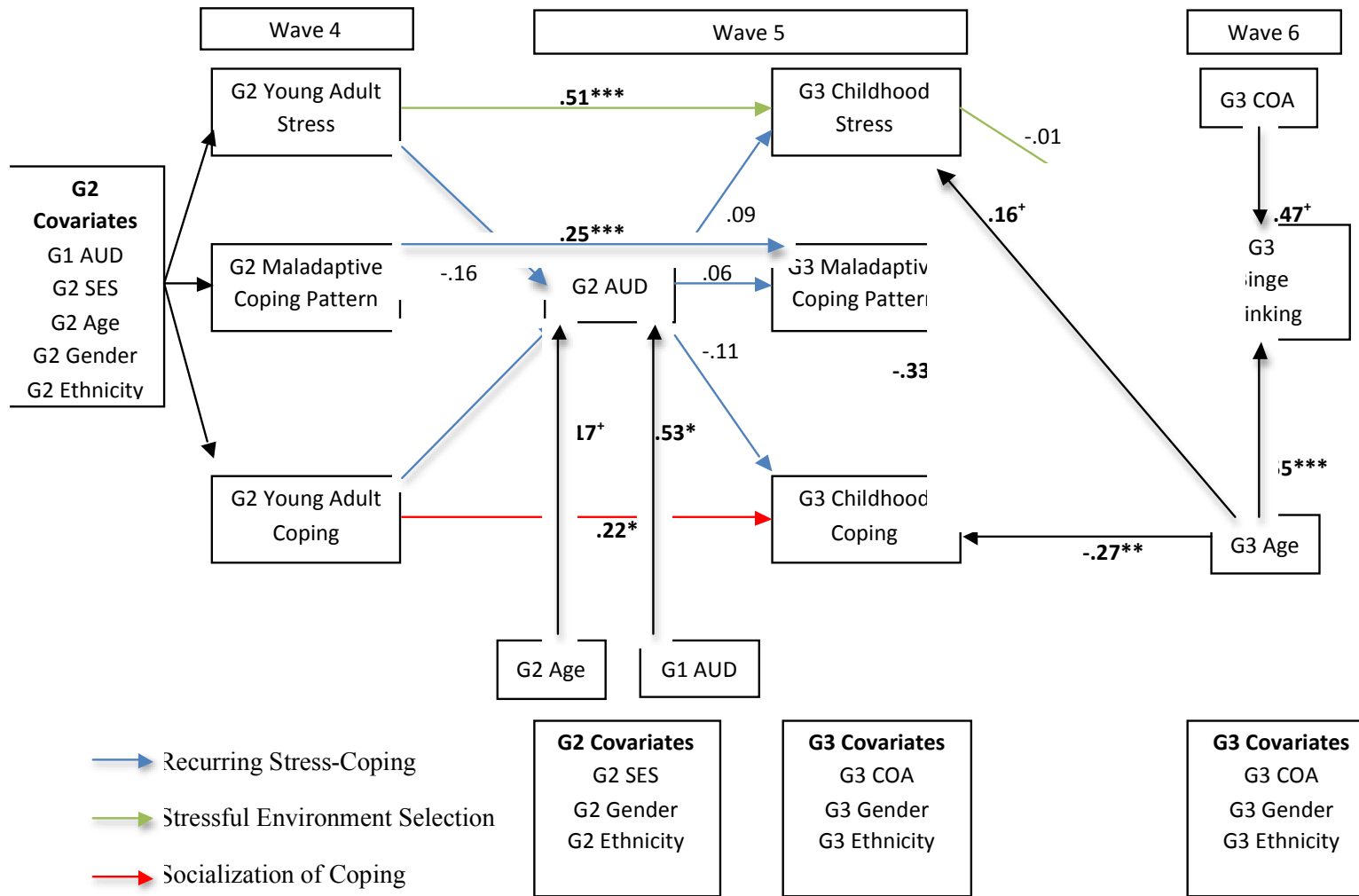
⁺p < .10, *p < .05, **p < .01, ***p < .001

Figure 8. Unique effects model predicting adolescent alcohol use.



⁺p < .10, *p < .05, **p < .01, ***p < .001

Figure 9. Unique effects model predicting adolescent binge drinking.



+p < .10, *p < .05, **p < .01, ***p < .001

REFERENCES

- Agnew, R. (1997). Stability and change in crime over the life course: A strain theory explanation. In T. P. Thornberry (Ed.), *Developmental theories of crime and delinquency* (pp. 101–132). New Brunswick, NJ: Transaction Press.
- Amato, P. R. (2001). Children of divorce in the 1990s: An update of the Amato and Keith (1991) meta-analysis. *Journal of Family Psychology, 15*, 355–370.
- American Psychiatric Association. (1980). *Diagnostic and statistical manual of mental disorders* (3rd ed.). Washington, DC: Author.
- Amodeo, M., Griffin, M.L., Fassler, I., Clay, C., & Ellis, M.A. (2007). Coping with stressful events: Influence of parental alcoholism and race in a community sample of women. *Health & Social Work, 32*, 247-257.
- Andreasen, N. C., Endicott, J., Spitzer, R. L., & Winokur, G. (1977). The family history method using diagnostic criteria. Reliability and validity. *Archives of General Psychiatry, 34*, 1229–1235.
- Armeli, S., Tennen, H., Todd, M., Carney, M., Mohr, C., Affleck, G., & Hromi, A. (2003). A daily process examination of the stress-response dampening effects of alcohol consumption. *Psychology of Addictive Behaviors, 17*, 266-276. doi:10.1037/0893-164X.17.4.266
- Ayers, T. S., Sandler, I. N., West, S. G., & Roosa, M. W. (1996). A dispositional and situational assessment of children's coping: Testing alternative models of coping. *Journal of Personality, 64*, 923-958.
- Baker, T.B., Piper, M.E., McCarthy, D.E., Majeskie, M.R., & Fiore, M.C. (2004). Addiction motivation reformulated: An affective processing model of negative reinforcement. *Psychological Review, 111*, 33-51.
- Bandura, A. (1977). Self-efficacy: Toward a unifying theory of behavioral change. *Psychological Review, 84*, 191-215. doi:10.1037/0033-295X.84.2.191
- Barrera, M., Li, S. A., & Chassin, L. (1993). Ethnic group differences in vulnerability to parental alcoholism and life stress: A study of Hispanic and non-Hispanic Caucasian adolescents. *American Journal of Community Psychology, 21*, 15-35. doi:10.1007/BF00938205
- Bernzweig, J., Eisenberg, N., & Fabes, R. A. (1993). Children's coping in self- and other-relevant contexts. *Journal of Experimental Child Psychology, 55*, 208-226. doi:10.1006/jecp.1993.1012
- Bifulco, A. A., Moran, P. M., Ball, C. C., Jacobs, C. C., Baines, R. R., Bunn, A. A., & Cavagin, J. J. (2002). Childhood adversity, parental vulnerability and disorder: Examining inter-

- generational transmission of risk. *Journal of Child Psychology and Psychiatry*, 43, 1075-1086. doi:10.1111/1469-7610.00234
- Blumenthal, H., Leen-Feldner, E. W., Badour, C. L., & Babson, K. A. (2011). Anxiety psychopathology and alcohol use among adolescents: A critical review of the empirical literature and recommendations for future research. *Journal of Experimental Psychopathology*, 2, 318-353.
- Britton, P. C. (2004). The relation of coping strategies to alcohol consumption and alcohol-related consequences in a college sample. *Addiction Research & Theory*, 12, 103-114. doi:10.1080/16066350310001613062
- Bronfenbrenner, U. (1986). Ecology of the family as a context for human development: Research perspectives. *Developmental Psychology*, 22, 723-742. doi:10.1037/0012-1649.22.6.723
- Brook, D. W., Brook, J. S., Whiteman, M., Arencibia-Mireles, O., Pressman, M. A., & Rubenstone, E. (2002). Coping in adolescent children of HIV-positive and HIV-negative substance-abusing fathers. *The Journal of Genetic Psychology: Research and Theory on Human Development*, 163, 5-23. doi:10.1080/00221320209597965
- Campbell, L., Simpson, J. A., Boldry, J., & Kashy, D. A. (2005). Perceptions of Conflict and Support in Romantic Relationships: The Role of Attachment Anxiety. *Journal of Personality and Social Psychology*, 88, 510-531. doi:10.1037/0022-3514.88.3.510
- Carver, C. S., Scheier, M. F., & Weintraub, J. K. (1989). Assessing coping strategies: A theoretically based approach. *Journal of Personality and Social Psychology*, 56, 267-283.
- Catanzaro, S. J., & Laurent, J. (2004). Perceived family support, negative mood regulation expectancies, coping, and adolescent alcohol use: Evidence of mediation and moderation effects. *Addictive Behaviors*, 29, 1779-1797. doi:10.1016/j.addbeh.2004.04.001
- Chassin, L., & Barrera, M. (1993). Substance use escalation and substance use restraint among adolescent children of alcoholics. *Psychology of Addictive Behaviors*, 7, 3-20. doi:10.1037/0893-164X.7.1.3
- Chassin, L., Barrera, M., Bech, K., & Kossak-Fuller, J. (1992). Recruiting a community sample of adolescent children of alcoholics: A comparison of three subject sources. *Journal of Studies on Alcohol*, 53, 316-319.
- Chassin, L., Curran, P. J., Hussong, A. M., & Colder, C. R. (1996). The relation of parent alcoholism to adolescent substance use: A longitudinal follow-up study. *Journal of Abnormal Psychology*, 105, 70-80.
- Chassin, L., Lee, M. R., Cho, Y. L., Wang, F. L., Agrawal, A., Sher, K. J., & Lynskey, M. T. (2012). Testing multiple levels of influence in the intergenerational transmission of

- alcohol disorders from a developmental perspective: The example of alcohol use promoting peers and μ -opioid receptor M1 variation. *Development and Psychopathology*, 24, 953-967. doi:10.1017/S0954579412000478
- Chassin, L., Pillow, D. R., Curran, P. J., Molina, B. G., & Barrera, M. R. (1993). Relation of parental alcoholism to early adolescent substance use: A test of three mediating mechanisms. *Journal of Abnormal Psychology*, 102, 3-19.
- Chassin, L., Pitts, S. C., DeLucia, C., & Todd, M. (1999). A longitudinal study of children of alcoholics: Predicting young adult substance use disorders, anxiety, and depression. *Journal of Abnormal Psychology*, 108, 106-119. doi:10.1037/0021-843X.108.1.106
- Chassin, L., Rogosch, F., & Barrera, M. (1991). Substance use and symptomatology among adolescent children of alcoholics. *Journal of Abnormal Psychology*, 100, 449-463. doi:10.1037/0021-843X.100.4.449
- Chen, Y., Prescott, C. A., Walsh, D., Patterson, D. G., Riley, B. P., Kendler, K. S., & Kuo, P. (2011). Different phenotypic and genotypic presentations in alcohol dependence: Age at onset matters. *Journal of Studies on Alcohol and Drugs*, 72, 752-762.
- 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, 976-985.
- Colder, C. R., Chassin, L., Lee, M. R., & Villalta, I. K. (2010). Developmental perspectives: Affect and adolescent substance use. In J. Kassel (Ed.), *Substance abuse and emotion* (pp. 109-135). Washington, DC: American Psychological Association. <http://dx.doi.org/10.1037/12067-005>
- Compas, B. E., Connor-Smith, J. K., Saltzman, H., Thomsen, A. H., & Wadsworth, M. E. (2001). Coping with stress during childhood and adolescence: Problems, progress, and potential in theory and research. *Psychological Bulletin*, 127, 87-127.
- Conger, J. J. (1956). Reinforcement theory and the dynamics of alcoholism. *Quarterly Journal of Studies on Alcohol*, 17, 296-305.
- Cooper, M. L., Frone, M. R., Russell, M., & Mudar, P. (1995). Drinking to regulate positive and negative emotions: A motivational model of alcohol use. *Journal of Personality and Social Psychology*, 69, 990-1105.
- Cooper, M.L., Wood, P.K., Orcutt, H.K., & Albino, A. (2003). Personality and the predisposition to engage in risky or problem behaviors during adolescence. *Journal of Personality and Social Psychology*, 84, 390-410.
- Dick, D. M., Nurnberger, J., Jr., Edenberg, H. J., Goate, A., Crowe, R., Rice, J., Bucholz, K. K., Kramer, J., Schuckit, M. A., Smith, T. L., Porjesz, B., Begleiter, H., Hesselbrock, V., &

- Foroud, T. (2002). Suggestive linkage on chromosome 1 for a quantitative alcohol-related phenotype. *Alcoholism: Clinical and Experimental Research*, *26*, 1453-1460. doi:10.1097/00000374-200210000-00001
- Eley, T. C., McAdams, T. A., Rijdsdijk, F. V., Lichtenstein, P., Narusyte, J., Reiss, D., Spotts, E. L., Ganiban, J. M., & Neiderhiser, J. M. (2015). The intergenerational transmission of anxiety: A children-of-twins study. *The American Journal of Psychiatry*, *172*, 630-637. doi:10.1176/appi.ajp.2015.14070818
- Farmer, T. W., & Farmer, E. Z. (2001). Developmental science, systems of care, and prevention of emotional and behavioral problems in youth. *American Journal of Orthopsychiatry*, *71*, 171-181. doi:10.1037/0002-9432.71.2.171
- Folkman, S., & Lazarus, R. S. (1980). An analysis of coping in a middle-aged community sample. *Journal of Health and Social Behavior*, *21*, 219-319.
- Greeley, J., & Oei, T. (1999). Alcohol and tension reduction. In K. E. Leonard & H. T. Blane (Eds.), *Psychological theories of drinking and alcoholism* (pp. 14-53). New York, NY: Guilford Press.
- Griffin, M. L., Amodeo, M., Fassler, I., Ellis, M. A., & Clay, C. (2005). Mediating factors for the long-term effects of parental alcoholism in women: The contribution of other childhood stresses and resources. *The American Journal on Addictions*, *14*, 18-34. doi:10.1080/10550490590899826
- Hammen, C. C., Hazel, N. A., Brennan, P. A., & Najman, J. J. (2012). Intergenerational transmission and continuity of stress and depression: Depressed women and their offspring in 20 years of follow-up. *Psychological Medicine*, *42*, 931-942. doi:10.1017/S0033291711001978
- Hankin, B. L. (2005). Childhood Maltreatment and Psychopathology: Prospective Tests of Attachment, Cognitive Vulnerability, and Stress as Mediating Processes. *Cognitive Therapy and Research*, *29*, 645-671. doi:10.1007/s10608-005-9631-z
- Hansen, W. B. (1997). A social ecology theory of alcohol and drug use prevention among college and university students. In *Designing alcohol and other drug prevention programs in higher education: Bringing theory into practice*. Newton, MA: Higher Education Center for Alcohol and Other Drug Prevention.
- Hoffmann, J. P., Cerbone, F. G., & Su, S. S. (2000). A growth curve analysis of stress and adolescent drug use. *Substance Use & Misuse*, *35*, 687-716.
- Hussong, A., Bauer, D., & Chassin, L. (2008). Telescoped trajectories from alcohol initiation to disorder in children of alcoholic parents. *Journal of Abnormal Psychology*, *117*, 63-78. doi:10.1037/0021-843X.117.1.63

- Hussong, A. M., Bauer, D. J., Huang, W., Chassin, L., Sher, K. J., & Zucker, R. A. (2008). Characterizing the life stressors of children of alcoholic parents. *Journal of Family Psychology, 22*, 819-832.
- Hussong, A. M., & Chassin, L. (2004). Stress and coping among children of alcoholic parents through the young adult transition. *Development and Psychopathology, 16*, 985-1006. doi:10.1017/S0954579404040106
- Hussong, A. M., Curran, P. J., & Chassin, L. (1998). Pathways of risk for accelerated heavy alcohol use among adolescent children of alcoholic parents. *Journal of Abnormal Child Psychology, 26*, 453-466. doi:10.1023/A:1022699701996
- Hussong, A.M., Hicks, R.E., Levy, S.A , & Curran, P.J. (2001). Specifying the relations between affect and heavy alcohol use among young adults. *Journal of Abnormal Psychology, 110*, 449-461.
- Hussong, A. M., Jones, D. J., Stein, G. L., Baucom, D. H., & Boeding, S. (2011). An internalizing pathway to alcohol use and disorder. *Psychology of Addictive Behaviors, 25*, 390-404. doi:10.1037/a0024519
- Kaminer, Y., Burleson, J. A., & Goldberger, R. (2002). Cognitive-behavioral coping skills and psychoeducation therapies for adolescent substance abuse. *Journal of Nervous and Mental Disease, 190*, 737-745. doi:10.1097/00005053-200211000-00003
- Kessler, R. C., Berglund, P., Demler, O., Jin, R., Merikangas, K. R., & Walters, E. E. (2005). Lifetime prevalence and age-of-onset distributions of DSM-IV disorders in the National Comorbidity Survey Replication. *Archives of General Psychiatry, 62*, 593-602. doi:10.1001/archpsyc.62.6.593
- Kessler, R. C., Chiu, W. T., Demler, O., & Walters, E. E. (2005). Prevalence, severity, and comorbidity of 12-month DSM-IV disorders in the National Comorbidity Survey Replication. *Archives of General Psychiatry, 62*, 617-627.
- Kliewer, W., & Lewis, H. (1995). Family influences on coping processes in children and adolescents with sickle cell disease. *Journal of Pediatric Psychology, 20*, 511-525. doi:10.1093/jpepsy/20.4.511
- Kliewer, W., Parrish, K., Taylor, K. W., Jackson, K., Walker, J. M., & Shivy, V. A. (2006). Socialization of coping with community violence: Influences of caregiver coaching, modeling, and family context. *Child Development, 77*, 605-623. doi:10.1111/j.1467-8624.2006.00893.x
- Klostermann, K., Chen, R., Kelley, M. L., Schroeder, V. M., Braitman, A. L., & Mignone, T. (2011). Coping behavior and depressive symptoms in adult children of alcoholics. *Substance Use and Misuse, 46*, 1162-1168.

- Laurent, J., Catanzaro, S. J., & Callan, M. (1997). Stress, alcohol-related expectancies and coping preferences: A replication with adolescents of the Cooper et al. (1992) model. *Journal of Studies on Alcohol*, *58*, 644-651.
- Lazarus, R. S., & Folkman, S. (1984). *Stress, appraisal and coping*. New York, NY: Springer.
- Low, N., Dugas, E., O'Loughlin, E., Rodriguez, D., Contreras, G., Chaiton, M., & O'Loughlin, J. (2012). Common stressful life events and difficulties are associated with mental health symptoms and substance use in young adolescents. *BMC Psychiatry*, *12*, doi:10.1186/1471-244X-12-116
- MacCallum, R. C., Browne, M. W., & Sugawara, H. M. (1996). Power analysis and determination of sample size for covariance structure modeling. *Psychological Methods*, *1*, 130-149.
- Manimala, M., Blount, R. L., & Cohen, L. L. (2000). The effects of parental reassurance versus distraction on child distress and coping during immunizations. *Children's Health Care*, *29*, 161-177. doi:10.1207/S15326888CHC2903_2
- Masten, A. S., & Cicchetti, D. (2010). Developmental cascades. *Development and Psychopathology*, *22*, 491-495. doi:10.1017/S0954579410000222
- Masten, A. S., Faden, V. B., Zucker, R. A., & Spear, L. P. (2008). Underage drinking: A developmental framework. *Pediatrics*, *121*(Suppl4), S235-S251. doi:10.1542/peds.2007-2243A
- McGillicuddy, N. B., Rychtarik, R. G., & Papandonatos, G. D. (2015). Skill training versus 12-step facilitation for parents of substance-abusing teens. *Journal of Substance Abuse Treatment*, *50*, 11-17. doi:10.1016/j.jsat.2014.09.006
- McGue, M., & Iacono, W. G. (2004). The Initiation of Substance Use in Adolescence: A Behavioral Genetics Perspective. In L. F. DiLalla (Ed.), *Behavior genetics principles: Perspectives in development, personality, and psychopathology* (pp. 41-57). Washington, DC, US: American Psychological Association. doi:10.1037/10684-003
- Muthén, L. K., & Muthén, B. O. (1998-2012). *Mplus User's Guide* (Seventh Edition). Los Angeles, CA: Muthén & Muthén.
- Ohannessian, C. M., Bradley, J., Waninger, K., Ruddy, K., Hepp, B. W., & Hesselbrock, V. (2010). An examination of adolescent coping typologies and young adult alcohol use in a high-risk sample. *Vulnerable Children and Youth Studies*, *5*, 52-65.
- Pearlin, L. I. (1989). The sociological study of stress. *Journal of Health and Social Behavior*, *30*, 241-256. doi:10.2307/2136956

- Prescott, C. A., Sullivan, P. F., Kuo, P. H., Webb, B. T., Vittum, J. J., Patterson, D. G., Thiselton, D. L., Myers, J. M., Devitt, M., Halberstadt, L. J., Robinson, V. P., Neale, M. C., Van den Oord, E. J., Walsh, D., Riley, B.P., & Kendler, K. S. (2006). Genomewide linkage study in the Irish affected sib pair study of alcohol dependence: Evidence for a susceptibility region for symptoms of alcohol dependence on chromosome 4. *Molecular Psychiatry, 11*, 603-611. doi:10.1038/sj.mp.4001811
- Program for Prevention Research. (1999). *Manual for the Children's Coping Strategies Checklist and the How I Coped under Pressure Scale*. Tempe, AZ: Arizona State University.
- Robins, L. N., Heltzer, J.E., Croughan, J., & Ratcliff, K.S. (1981). National Institute of Mental Health Diagnostic Interview Schedule: Its history, characteristics, and validity. *Archives of General Psychiatry, 38*, 381-389.
- Robins, L. N., Helzer, J.E., Ratcliff, K.S., & Seyfried, W. (1982). Validity of the Diagnostic Interview Schedule, Version II: DSM-III diagnoses. *Psychological Medicine, 12*, 855-870.
- Roosa, M. W., Sandler, I. N., Gehring, M., Beals, J., & Cappel, L. (1988). The Children of Alcoholics Life-Events Schedule: A stress scale for children of alcohol-abusing parents. *Journal of Studies on Alcohol, 49*, 422-429.
- Sandler, I. N., Ramirez, R., & Reynolds, K. D. (1986). Life stress for children of divorce, bereaved and asthmatic children. Paper presented at the American Psychological Association Convention.
- Sandler, I. N., Tein, J-Y., & West, S. G. (1994). Coping, stress, and the psychological symptoms of children of divorce: A cross-sectional and longitudinal study. *Child Development, 65*, 1744-1763.
- Schneiders, J., Nicolson, N. A., Berkhof, J., Feron, F. J., van Os, J., & deVries, M. W. (2006). Mood reactivity to daily negative events in early adolescence: Relationship to risk for psychopathology. *Developmental Psychology, 42*, 543-554.
- Seiffge-Krenke, I., Aunola, K., & Nurmi, J. (2009). Changes in stress perception and coping during adolescence: The role of situational and personal factors. *Child Development, 80*, 259-279. doi:10.1111/j.1467-8624.2008.01258.x
- Shaw, M., Lawlor, D.A., & Najman, J.M. (2006). Teenage children of teenage mothers: Psychological, behavioural and health outcomes from an Australian prospective longitudinal study. *Social Science & Medicine, 62*, 2526-39.
- Sheidow, A. J., Henry, D. B., Tolan, P. H., & Strachan, M. K. (2014). The role of stress exposure and family functioning in internalizing outcomes of urban families. *Journal of Child and Family Studies, 23*, 1351-1365. doi:10.1007/s10826-013-9793-3

- Sher, K. J. (1991). *Children of alcoholics: A critical appraisal of theory and research*. Chicago, IL: University of Chicago Press.
- Sher, K. J. (1997). Psychological characteristics of children of alcoholics. *Alcohol Health & Research World*, 21, 247-254.
- Sher, K. J., Gershuny, B. S., Peterson, L., & Raskin, G. (1997). The role of childhood stressors in the intergenerational transmission of alcohol use disorders. *Journal of Studies on Alcohol*, 58, 414-427.
- Sher, K. J., Grekin, E. R., & Williams, N. A. (2005). The development of alcohol use disorders. *Annual Review of Clinical Psychology*, 1, 493-523.
- Smith, C. L., Eisenberg, N., Spinrad, T. L., Chassin, L., Morris, A. S., Kupfer, A., Liew, J., Cumberland, A., Valiente, C., & Kwok, O-M. (2006). Children's coping strategies and coping efficacy: Relations to parent socialization, child adjustment, and familial alcoholism. *Development and Psychopathology*, 18, 445-469.
- Solis, J. (2013). *Coping in children of alcoholics across the life span: A review of the literature and a proposed research agenda*. Unpublished manuscript, University of North Carolina-Chapel Hill, Chapel Hill, NC.
- Thornberry, T. P. (2005). Explaining multiple patterns of offending across the life course and across generations. In R. J. Sampson & J. H. Laub (Eds.), *Developmental criminology and its discontents: Trajectories of crime from childhood to old age, The Annals of the American Academy of Political and Social Science*, 602, 156-228.
- Turner, H. A., & Butler, M. J. (2003). Direct and indirect effects of childhood adversity on depressive symptoms in young adults. *Journal of Youth and Adolescence*, 32, 89-103. doi:10.1023/A:1021853600645
- Turner, R., & Lloyd, D. A. (1995). Lifetime traumas and mental health: The significance of cumulative adversity. *Journal of Health and Social Behavior*, 36, 360-376. doi:10.2307/2137325
- Veenstra, M. Y., Lemmens, P. M., Friesema, I. M., Tan, F. S., Garretsen, H. L., Knottnerus, J., & Zwietering, P. J. (2007). Coping style mediates impact of stress on alcohol use: A prospective population-based study. *Addiction*, 102, 1890-1898. doi:10.1111/j.1360-0443.2007.02026.x
- Waters, S. F., & Thompson, R. A. (2014). Children's perceptions of the effectiveness of strategies for regulating anger and sadness. *International Journal of Behavioral Development*, 38, 174-181. doi:10.1177/0165025413515410

- Wei, C. C., Heckman, B. D., Gay, J., & Weeks, J. (2011). Correlates of motivation to change in adolescents completing residential substance use treatment. *Journal of Substance Abuse Treatment, 40*, 272-280. doi:10.1016/j.jsat.2010.11.014
- Wills, T.A., Sandy, J.M., Yaeger, A.M., Cleary, S.D., & Shinar, O. (2001). Coping dimensions, life stress, and adolescent substance use: A latent growth curve analysis. *Journal of Abnormal Psychology, 110*, 309-323.
- Windle, M., & Windle, R. C. (1996). Coping strategies, drinking motives, and stressful life events among middle adolescents: Associations with emotional and behavioral problems and with academic functioning. *Journal of Abnormal Psychology, 105*, 551-560. doi:10.1037/0021-843X.105.4.551
- Young, R. M., Oei, T. P., & Knight, R. G. (1990). The tension reduction hypothesis revisited: An alcohol expectancy perspective. *British Journal of Addiction, 85*, 31-40. doi:10.1111/j.1360-0443.1990.tb00621.x
- Yu, J. (2003). The association between parental alcohol-related behaviors and children's drinking. *Drug and Alcohol Dependence, 69*, 253-262. doi:10.1016/S0376-8716(02)00324-1
- Zautra, A. J., Sheets, V. L., & Sandler, I. N. (1996). An examination of the construct validity of coping dispositions for a sample of recently divorced mothers. *Psychological Assessment, 8*, 256-264.
- Zuravin, S., McMillen, C., DePanfilis, D., & Risley-Curtiss, C. (1996). The intergenerational cycle of child maltreatment: Continuity versus discontinuity. *Journal of Interpersonal Violence, 11*, 315-334. doi:10.1177/088626096011003001