# The Roles of Sensation Seeking and Level of Response to Negative, Sedative Alcohol Effects in the Intergenerational Transmission of Risk for Developing Alcohol Use Disorders

by

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ABSTRACT

The present study tested the respective mediating effects of sensation seeking and initial level of response (LR) to negative, sedative alcohol effects on the relation between the density of familial history of alcoholism and adolescent alcohol use. Additionally, the present study tested the direct effect of LR to negative, sedative alcohol effects on adolescent drinking over and above the effects of sensation seeking; and also tested the moderating effect of sensation seeking on the relation between level of response negative, sedative alcohol effects and adolescent drinking. Specifically, OLS regression models first estimated the effects of sensation seeking, LR to negative, sedative alcohol effects, and their interaction on alcohol outcomes, over and above the influence of covariates. Indirect effects were then tested using the PRODCLIN method through RMediation. Analyses failed to support sensation seeking as a mediator in the relation between familial history of alcoholism and adolescent drinking, and as a moderator of the relation between LR and adolescent drinking. However, analyses did support a robust direct effect of LR to negative, sedative alcohol effects on adolescent alcohol involvement. A significant mediating effect of initial LR to negative, sedative alcohol effects on the relation between familial alcoholism and adolescent drinking was found, however failed to maintain significance in post-hoc analyses attenuating the downward bias of the measure of initial LR. Initial LR to negative, sedative alcohol effects continued to predict adolescent drinking after attenuating measure bias. These findings strengthen research on initial LR to negative, sedative alcohol effects as a risk for greater alcohol involvement in adolescence, and underscore the complexity of studying the familial transmission of alcoholism in adolescent populations.

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# INTRODUCTION

The Centers for Disease Control and Prevention (2012) estimate that excessive alcohol consumption is responsible for 80,000 US deaths a year. Even for individuals without direct exposure, alcohol misuse has a significant impact. In 2006, costs related to excessive alcohol consumption led to an economic burden of $223.5 billion—an impact of $746 per person (Bouchery, Harwood, Sacks, Simon, & Brewer, 2011). For both directly afflicted individuals and society at large, alcohol use disorders (AUDs) pose heavy financial and health burdens. Therefore research on the etiology and development of AUDs is important. Research suggests that both individual differences in the acute pharmacological effects of alcohol, and personality traits contribute significantly to the development of alcohol misuse and to its intergenerational transmission (Sher, 1991; Smith & Anderson, 2004). However, less work has been done investigating how these two risk factors uniquely and interactively affect alcohol use and misuse in adolescents. Investigating these pathways of AUD risk transmission could provide insight into processes that underlie excessive alcohol consumption and AUD development, and inform prevention programs.

## Enhanced Reinforcement and Level of Response to Negative, Sedative Alcohol Effects

Given that alcohol consumption is essential to the development of AUDs, much research has focused on acute alcohol effects and their role in alcohol use. Acute alcohol effects can range from the physiological, such as an elevated heart rate after alcohol consumption, to more subjective experiences, such as the feeling of elation that may accompany drinking to inebriation. Across the range of acute alcohol effects, several

differ significantly between individuals with versus without a family history of alcoholism. For example, individuals with a family history of alcoholism (and thus a greater risk for developing an AUD) have reported less feelings of intoxication (O'Malley & Maisto, 1985), and showed greater reductions in electroencephalogram (EEG) alpha frequencies after alcohol consumption (Pollock et al., 1983).

This variation in responses to alcohol by family history suggests that acute alcohol effects may play a role in the intergenerational transmission of risk for developing an AUD. One model of acute alcohol effects and alcoholism risk is the Enhanced Reinforcement model (Sher, 1991), in which family history of alcoholism causes differences in response to acute alcohol effects, which in turn enhance the reinforcing effects of alcohol. Specifically, differences in sensitivity to the acute pharmacokinetic and/or pharmacodynamic effects of alcohol result in some individuals having a comparatively more enhanced response to alcohol effects, and/or experiencing fewer aversive effects from alcohol. This more pleasant or less unpleasant response is in turn causally related to pathological alcohol involvement. An enhanced experience of alcohol may serve as additional motivation to drink to obtain the positive effects of alcohol, and/or may provide less reason to stop drinking due to diminished experiences of the aversive effects of alcohol. In short, in the Enhanced Reinforcement model, level of response (LR) to the effects of alcohol mediates the relation between family history of alcoholism and pathological alcohol involvement.

What is complex and still unclear about the Enhanced Reinforcement model is how different levels of response to arousing (stimulating versus sedating) and valenced (positive versus negative) acute alcohol effects creates an enhanced response to alcohol.

Valence and arousal are distinct dimensions of acute alcohol effects. Thus, acute alcohol effects can be characterized into four quadrants: negative, stimulating (e.g. aggressive, demanding); negative, sedating (e.g. sleepy, lethargic); positive, sedating (e.g. calming, relaxing) or positive, stimulating (e.g. talkative, excited; Morean, Corbin, & Treat, 2013). Differential LR to any of these four quadrants of alcohol effects may drive the enhancement central to the Enhanced Reinforcement model. For example, for the arousal dimension, alcohol effects align with the biphasic nature of blood alcohol concentrations (BAC) during alcohol consumption, such that stimulating alcohol effects are more pronounced when BAC is increasing/ascending, whereas sedative alcohol effects are more pronounced when the BAC is decreasing/descending (Pohorecky, 1977). Relatively greater sensitivity or insensitivity to one or both limbs of the BAC curve, and their corresponding alcohol effects, may contribute to the enhanced experience of alcohol posited as a risk in the Enhanced Reinforcement model.

In the alcohol effects literature there are two models of level of response to alcohol effects proposed to contribute to this, enhanced experience of alcohol: the Low Level of Response Model (LLRM; Schuckit, 1994) and the Differentiator Model (DM; Newlin & Thomson, 1990). The LLRM originally emerged from anecdotal evidence of alcoholic individuals requiring more alcoholic beverages before experiencing alcohol effects. It is the most studied model of alcohol effects, with much of the supporting literature emerging from the prolific work of Dr. Marc Schuckit and his research group (Morean & Corbin, 2010). According to the LLRM, at-risk individuals have comparatively lower LR to both the positive, stimulating and negative, sedating effects of alcohol. This overall decreased sensitivity results in at-risk individuals consuming more

drinks to attain the desired stimulating effects, without experiencing the negative, sedating effects to slow down or stop consumption (Schuckit, 1994). The DM (Newlin & Thomson, 1990) also proposes that at-risk individuals have a lower LR to the negative, sedative effects of alcohol. However, the DM proposes that at-risk individuals have *greater* LR to positive, stimulating alcohol effects, thereby experiencing greater positive reinforcement. Thus, individuals with the DM response profile will consume more alcohol to experience even greater positive, stimulating effects, and will lack the negative, sedative effects to slow down or inhibit their consumption (Newlin & Thomson, 1990).

Both models provide more specificity to the Enhanced Reinforcement model by predicting the nature of the enhanced response as a function of differential LR to arousal and valence dimensions of alcohol effects. Less clear is whether increased or decreased sensitivity to positive, stimulating alcohol effects during the ascending limb of the BAC curve increases the risk for developing an AUD. However, there is agreement between both models that a low LR to negative, sedative alcohol effects is a risk factor for alcoholism. Accordingly, the current study focused on this low LR to negative, sedative alcohol effects, and its contribution to increased risk for problematic alcohol use in adolescents.

Historically, differential LR to negative, sedative alcohol effects has been investigated through laboratory-based alcohol challenges. In alcohol challenges, participants are administered a set dose of alcohol (typically between 0.4 g/kg to 1.0 g/kg) in the laboratory (Morean & Corbin, 2010). Participants' LR to negative, sedative alcohol effects are recorded at various time points throughout the drinking session. LR is

evaluated through questionnaires, with the Subjective High Assessment Scale (Judd, Hubbard, Janowsky, Leighton, Huey, & Atwell, 1977) being the most commonly used in alcohol challenge studies. Participants are asked to rate the extent to which they are currently experiencing various effects of alcohol—such as feeling clumsy, dizzy, or confused (Schuckit & Gold, 1988; Morean & Corbin, 2010). Reporting a lower score on these items indicates experiencing less of the negative, sedative effects of alcohol, or demonstrating a lower LR to these aversive effects. To control for differences in recent drinking, participants often complete a detailed history of past 30-day alcohol use. Differences in LR to negative, sedative alcohol effects at various time points during the drinking session are compared between individuals with versus without a family history of AUDs.

The results of these alcohol challenge studies suggest that individuals with a family history of AUDs demonstrate significantly lower LR to the same dose of alcohol administered to individuals without a family history (e.g. Schuckit & Smith, 2000). For example, Schuckit, Smith, Kalmijn, and Danko (2005a) compared subjective response to 0.6 g/kg of alcohol among non-alcohol dependent males with and without a family history of AUDs. Participants' subjective responses to the negative, sedative effects of alcohol were evaluated at baseline, 15 minutes after administration, and every following 30 minutes. Participants with a family history of AUDs reported significantly lower LR to negative, sedative alcohol effects at the 60 and 90 minute time points compared to those without a family history (Schuckit et al., 2005a). This relation between LR to negative, sedative alcohol effects and family history is robust as it was found for

daughters of alcoholics (Eng, Schuckit, & Smith, 2005), and in designs where alcohol was administered via infusion (Chiu et al., 2004).

Furthermore, longitudinal studies of children of alcoholics versus non-alcoholics suggest that the relation between family history of alcoholism and LR to negative, sedative alcohol effects is heritable. When LR to negative, sedative alcohol effects was examined across generations during an alcohol challenge, offspring LR in young adulthood positively and significantly correlated with parents' LR in young adulthood. Parent-offspring pairs with a family history of alcoholism had the most robust correlations (Schuckit, et al, 2005a). Further comparison of LR to negative, sedative alcohol effects between parents and their offspring suggest that the genetics underlying LR contribute to 40-60% of variance in the genetic risk for developing an AUD (Schuckit, 1999). Taken together these outcomes implicate family history of alcoholism as an important contributing factor in differing LR to negative, sedative alcohol effects.

These differences in LR to negative, sedative alcohol effects during an alcohol challenge may also predict future alcohol use outcomes. For example, LR to alcohol during an alcohol challenge in young adulthood predicted alcohol use and alcohol-related problems 20 years later. Specifically, lower levels of a composite comprised of LR, body sway, and changes in hormones linked to BAC change, directly related to future heavier drinking (e.g. higher average and maximum number of alcohol beverages in the last 6 months and past 5 years), and more alcohol-related problems (Schuckit, et al., 2011a). Thus, LR to negative, sedative alcohol effects may mediate the relation between family history of alcoholism and offspring alcohol use. Indeed, in the same sample of young adult males with and without a family history of AUDs noted earlier, participants with a

family history of alcoholism who demonstrated a low LR to alcohol had significantly greater risk for developing alcoholism 20 years later (Schuckit & Smith, 1996). In the present study, we sought to replicate this mediating relation. However, given the importance of identifying risk for AUD as early as possible, this study focused on testing whether this mediating relation is present even in emerging alcohol use in the early stages of adolescents' drinking careers.

For the purposes of studying LR to negative, sedative alcohol effects in adolescents, the alcohol challenge design is not appropriate because of ethical concerns with providing alcohol to minors. Additionally, alcohol challenges are costly and each session typically takes several hours to complete. Retrospective self-reports of LRs to alcohol effects are less expensive to obtain and additionally circumvent the problem of administering alcohol to minors. In this design participants recall their drinking experiences, and report the number of drinks they required to reach a certain LR to alcohol effects. The Self-Rating for the Effects of Alcohol (SRE) form is commonly used in retrospective self-report designs (Schuckit, Smith, & Tipp, 1997), and captures LR to negative, sedative alcohol effects during the first five (First 5), heaviest, and most recent drinking experiences. A higher SRE scores indicates requiring more drinks to experience the effects of alcohol, and therefore a lower LR to alcohol and greater risk for alcohol problems. The present study used the First 5 SRE measure given its focus on LRs to the negative, sedative effects of alcohol among specifically adolescents.

Paralleling alcohol challenge study outcomes, research using retrospective self-report designs also suggest a mediating relation between family history of AUDs, offspring LRs, and alcohol use outcomes. Studies using the SRE in adolescents and

young adults found that individuals with a family history of alcoholism had comparatively higher SRE scores (i.e. a lower LR to negative, sedative alcohol effects) compared to individuals without a family history (Schuckit et al., 2005b; Schuckit et al., 1997). These relations were unaffected by recent drinking history, indicating that differences in LR occurred beyond the effects of alcohol tolerance. Additionally, retrospective reports of LRs to negative, sedative alcohol effects during adolescents' first five drinking experiences significantly correlate with parents' LRs (Schuckit, Smith, Danko, Kuperman, Beirut, & Hesselbrock, 2005c). Thus, research relying on retrospective self-reports corroborate alcohol challenge outcomes indicating that LR may be a heritable risk factor.

Greater SRE scores reported in early to late adolescence have also been related to more severe alcohol use outcomes, alcohol-related problems, peer substance use, and greater reports of drinking to cope at the time of interview (Schuckit et al., 2011a; Schuckit, et al., 2005d). This relation between recollections of LR and problematic alcohol use persists over time, with adolescents and emerging adults who report higher SRE scores endorsing worse alcohol use outcomes and AUDs at least five years later in adulthood (Schuckit et al., 2011b; Schuckit, et al., 2007). Finally, also consistent with alcohol challenge outcomes, differences in retrospectively reported LR during adolescents' first five drinking occasions mediates the relation between family history and adolescent drinking/alcohol-related problems (Schuckit, et al., 2005b). The consistency of these findings despite different research designs provides confidence in the mediating role of LR to negative, sedative alcohol effects.

A criticism of these results is that they are limited by the retrospective nature of the data, and participants' recollections may not accurately reflect their actual response to alcohol at the time being recalled. Rather, retrospective reports may instead reflect participants' present-day tolerance. However, there is some evidence that overall SRE scores correlate with LR to negative, sedative alcohol effects demonstrated during alcohol challenges (Schuckit, Smith, & Tipp, 1997). The present study further addressed this problem in three related ways. First, the first-five subscale of the SRE was used to specifically focus on *initial* LR to the negative, sedative effects of alcohol. Although reports may have still been influenced by participants' current tolerance, recollections were focused on a specific time point characterized by no experience, let alone tolerance. Recall may have been improved given that early drinking occasions can be distinct experiences in participants' personal histories. Second, the present study minimized the recall period, or years of retrospection, by assessing initial LR to negative, sedative alcohol effects during adolescence. By using an adolescent sample, the years of retrospection between the time of interview and the participants' first five drinking experiences was reduced, thereby minimizing poor recall (Schuckit et al., 2005b; Schuckit et al, 2005d). Lastly, the present study specifically controlled for participants' years of retrospection, or the number of years between participants' ages of first drink and their ages when responding to the SRE. This strategy for attenuating retrospection bias is a unique contribution to the literature, as little to no LR to alcohol effects studies controlled for years of retrospection despite evidence that the accuracy of recalled reports of alcohol use in adolescence decreases as years of retrospection increases (Brener, Billy, & Grady, 2003). The current study thus sought to replicate literature on the mediating

role of LR to alcohol by using initial drinking SRE scores reported in adolescence, and controlling for years of retrospection, to explore the intergenerational transmission of alcohol risk in late adolescence.

**Personality and Alcohol Use**

Personality traits are relatively stable, internal dispositions that are reflected in differences in external, observable behavior (Sher & Trull, 1994). Individual differences in personality traits have long been studied as risk factors for the development of AUDs. Early research sought to identify the "alcoholic personality" type, or the unique constellation or pattern of personality traits that characterizes an alcoholic (Barnes, 1979; Sher & Trull, 1994). However, recent research has focused instead on elucidating how various personality traits may increase risk for AUDs over the course of development. For example, higher levels of the personality trait extraversion (characterized by sociability, reward-seeking, and gregariousness; Smith & Anderson, 2001) in adolescence were related to heavier alcohol consumption in young adulthood (Ayer et al., 2011). In a sample of young adults with and without a family history of AUDs, low levels of agreeableness increased the odds of having engaged in heavy alcohol and substance use (Chassin, Flora, & King, 2004). These outcomes, and countless other personality-based models of vulnerability (e.g. Sher, 1991; Patterson & Newman, 1993) suggest that different levels of personality traits may increase alcohol use and problems, thereby increasing the risk for developing an AUD.

Behavioral disinhibition is the personality factor with the most consistently supported relation to AUDs. Also commonly referred to as behavioral undercontrol, behavioral disinhibition is a broad vulnerability to externalizing disorders and risk-taking

behavior, and is characterized by such personality constructs as impulsivity, sensation seeking, and aggression (Dindo, McDade-Montez, Sharma, Watson, & Clark, 2009; Sher, 1991; Young et al., 2009). Individuals who meet criteria for an AUD show higher levels of disinhibition compared to individuals who do not meet criteria (McGue, Slutske, Taylor, & Iacono, 1997). Furthermore, disinhibition levels in adolescence and young adulthood are linked to alcohol use outcomes that increase risk for future AUD development. For example, in a longitudinal study with college students, higher disinhibition scores in freshman year (at baseline) predicted comparatively more alcohol use and alcohol-related problems both at baseline and four years later (Sher, Bartholew, & Wood, 2000). In addition, a slower decline in disinhibition levels over the course of development from age 3 to 14 years old relates to early-onset drinking, early-onset drunkenness, and more alcohol-related problems in late adolescence (Wong et al., 2006). Thus, comparatively higher levels of disinhibition over the course of development predicts earlier and more problematic engagement with alcohol.

Twin studies and research on children of alcoholics suggest that behavioral disinhibition is also a heritable personality trait. In a community sample of over 3,000 adult twin-pairs, disinhibition accounted for 40% of the variation of genetic risk for AUDs (Slutske et al., 2002), and accounted for 16-46% of the phenotypic variance for substance experimentation in a community sample of adolescent twin-pairs (Young, Stallings, Corley, Krauter, & Hewitt, 2000). These outcomes are consistent with findings that children of alcoholics show significantly higher levels of disinhibition compared to children of non-alcoholics (Sher, 1991; King et al., 2009).

Behavioral disinhibition may thus mediate the relation between parents' and offspring's alcohol outcomes. Among a sample of 14-year old twin-pairs with and without an alcoholic parent, participants' levels of disinhibition and their own likelihood of early onset drinking increased if their parents engaged in early onset drinking (McGue, Iacono, Legrand, & Elkins, 2001). Further comparison of twin-pairs with identical genes (i.e. monozygotic twins) to twin-pairs with related but *not* identical genes (i.e. dizygotic twins), confirmed that early alcohol use in offspring was partially accounted for by genetic influences on disinhibition. Finally, in a community sample of adolescent children of alcoholics, higher levels of impulsivity in adolescence increased odds of young adult membership in the most severe of three identified alcohol and substance use trajectories: heavy drinking/heavy drug use, moderate drinking/experimental drug use, and light drinking/rare drug use (Chassin, et al., 2004). In short, disinhibition has been found to be a heritable trait related to early alcohol involvement, and a risk factor for developing an AUD (Chassine t al., 2004; Iacono, Malone, & McGue, 2008; McGue et al., 2001; Iacono & McGue, 2002).

Studies specifically testing the mediating effect of disinhibition have mostly found a significant indirect effect of parent AUD status on offspring alcohol use through disinhibition levels. Chassin and colleagues (2004) found that disinhibition significantly mediated the relations between parent alcoholism and offspring alcohol use in adolescence, and parent alcoholism and offspring alcoholism in young adulthood. Consistent with these findings, Tarter, Kirisci, Habeych, Reynolds, & Vanyukov (2004) also found that childhood disinhibition mediated the relation between parents' lifetime and offspring's young adult AUD status. In contrast, a survey conducted with high school

students failed to support disinhibition as a significant partial mediator between parents' alcohol problems and offsprings' alcohol use (Schepis et al., 2008). However, researchers did find significant associations between participants' disinhibition and parents' history of alcohol problems, and participants' disinhibition and past month substance use, which are consistent with previous literature. This study was conducted at a single time-point, and therefore true mediation could not be established. In addition, this study was limited in that parental history of alcoholism was based on offspring self-report, and of those adolescents who responded to the survey, 40% failed to complete the entire survey (Schepis et al., 2008). The selection biases of this study may have diluted the sample of adolescents who could demonstrate the mediating effect of disinhibition on the effects of parent AUD on early drinking. Taken together, these findings suggest that disinhibition may indeed be a heritable mediator of the relation between parents' AUDs and offsprings' alcohol use and misuse. Given its potential importance in the intergenerational transmission of AUDs, the current study tested the role of disinhibition in mediating the effects of family history of AUDs on early alcohol use.

That several studies supported a mediating effect of disinhibition in the transmission of risk for alcoholism is noteworthy given the inconsistent operationalization of this construct. Measures of disinhibition vary across the literature, ranging from counts of externalizing disorders (Tarter et al., 2004; McGue et al., 2001), to measures of impulsivity (Chassin et al., 2004), novelty-seeking, and psychoticism (Sher at al., 2000). Studies testing the mediating effect of disinhibition thus highlight the broad nature of disinhibition as a personality trait. Differentiating the various subfacets of

disinhibition may yield distinct patterns of AUD risk transmission, reflecting the various pathways to pathological alcohol involvement.

Indeed, emerging literature contends that behavioral disinhibition is comprised of five distinct subfacets: positive urgency, negative urgency, (lack of) premeditation, (lack of) perseverance, and sensation seeking (Cyders & Smith, 2007; Whiteside & Lynam, 2001). Lack of premeditation is characterized by a tendency to engage in behavior with little to no prior deliberation or thought of the consequences, and lack of perseverance is characterized by the tendency to disengage from boring or laborious tasks (Whiteside & Lynam, 2001). Sensation seeking is characterized by the tendency to pursue experiences that provide thrilling stimulation, while urgency is characterized by the tendency to act rashly when experiencing negative emotions (Whiteside & Lynam, 2001; Dick et al., 2010). Later research divided urgency into two distinct subfacets based on the type of emotion prompting the rash behavior--negative urgency for the tendency to act rashly when experiencing negative emotions, and positive urgency for acting rashly when experiencing positive emotions (Cyders & Smith, 2007). Each subfacet of disinhibition falls under one of three factors, such that lack of premeditation and lack of perseverance are part of a deficits-in-conscientiousness factor, negative and positive urgency are under a mood-based factor, and sensation seeking is its own distinct factor (Cyders & Smith, 2007).

Beyond a theoretical contribution to personality research, distinguishing among these subfacets may provide a more nuanced understanding of the relation between disinhibition and alcohol use. For example, the negative urgency and sensation seeking subfacets each predict an aspect of alcohol use. In a study exploring the relation between

subfacets of disinhibition and various addictive behaviors among college students, negative urgency predicted the number of alcohol-related problems (e.g. blacking out, problems with work because of drinking), whereas sensation seeking significantly predicted frequency of alcohol consumption (Fischer & Smith, 2008). Individuals with sensation seeking levels that diminish more gradually over the course of development were more likely to demonstrate faster increases in alcohol use level over time (Quinn & Harden, 2013). Thus, studies distinguishing between the subfacets of disinhibition suggest that both negative urgency and sensation seeking are related to alcohol use, but in different ways and each provides insight into the contribution of personality traits to alcohol use and misuse. Reckless incidences of problem drinking may reflect the rash behavior of an individual with high negative urgency who is experiencing an extreme negative emotion, whereas the thrill or novelty of drinking and inebriation may motivate individuals with high levels of sensation seeking to drink more frequently.

Given that increased drinking frequency has been proposed to contribute to eventual alcohol misuse, sensation seeking may play an important role in the development of AUDs by increasing consumption. Indeed, findings for sensation seeking parallel the disinhibition literature in that children of alcoholics demonstrate comparatively higher levels of sensation seeking than do children of non-alcoholics (Finn, Earleywine, & Phil, 1992), and higher levels of sensation seeking predict more severe alcohol use outcomes (Wagner, 2001; Sher et al., 2000). The present study used sensation seeking to explore the role of personality in the intergenerational transmission of AUDs.

**Personality and Level of Response to Negative, Sedative Alcohol Effects**

Personality may not only predict alcohol outcomes, it may also influence the relation between LR to alcohol effects and drinking. According to the Acquired Preparedness Model (APM), personality trait differences impart learning-based risks for a particular behavior. Specifically, trait differences prepare individuals to learn, or acquire, certain interpretations of an experience which may then place them at greater risk for maladaptive behaviors (Smith & Anderson, 2001). This model is an elaboration of Caspi's (2003) reactive person-environment transaction, in which individual differences contribute to different interpretations, and reactions to the same environment or situation. In the case of alcohol, the APM proposes that personality traits linked with greater levels of reward-approach and reward sensitivity, such as sensation seeking, will prepare an individual to be biased towards remembering and/or learning the reward they gain from their drinking experiences. These individuals are therefore at greater risk for gaining reinforcement from their drinking experiences (Smith & Anderson, 2001).

Within the APM framework, personality traits have typically been tested as mediating effects. The present study will expand upon the APM framework by testing the *modifying* effects of reward-approach personality traits. Specifically, the APM suggests that individuals with a low LR to negative, sedative alcohol effects who are also high in sensation seeking are at the greatest risk for an AUD (as compared to individuals with either lower sensation seeking or higher LR). That is, individuals high in sensation seeking will be prepared to learn the positive effects of alcohol. However, because of their low LR they will need to consume comparatively more alcohol to obtain their desired positive, stimulating effect of alcohol, and could drink even more before

experiencing the negative, sedating effects of alcohol. Sensation seeking and LR should thus interact, such that the effect of lower LR to negative, sedative alcohol effects on adolescent alcohol use would be greater as a function of elevated sensation seeking levels. That is, there would be a stronger relation between initial LR and adolescent drinking among those high in sensation seeking, as compared to the relation among those medium to low in sensation seeking (see Figure 1). Personality traits may thus moderate the relation between low LR and drinking, and the APM provides a potential framework for predicting and testing this interplay.

Despite theoretical support, few studies have investigated how the interplay between personality and LR to alcohol effects may contribute to the development of AUDs. Recently, one study focused on the negative emotionality personality trait as it relates to response to amphetamines and drinking. Negative emotionality interacted with LR to amphetamines such that the relation between elevated negative emotionality and heavier alcohol use was stronger among individuals more sensitive to amphetamine effects (Allen & Gabbay, 2013). However, despite substantial support for LR and sensation seeking respectively relating to family history of alcoholism and alcohol use outcomes, there is little to no research on their interplay. To our knowledge, the present study was one of the first to investigate the interaction between sensation seeking and LR to negative, sedative alcohol effects in the context of the intergenerational transmission of risk for developing AUDs.

The few studies that have studied LR to alcohol effects and sensation seeking together investigated both characteristics in the context of a mediating rather than a moderating relation, and yield mixed results. For example, research published over

twenty years ago found that individuals with high levels of disinhibition experienced comparatively greater reductions in cardiovascular and affective responses to stress after consuming alcohol. Disinhibition was thus differentially related to the stress-dampening effects of alcohol (Sher & Levenson, 1982; Levenson, Oyama, & Meek, 1987). In contrast, higher levels of sensation seeking related to comparatively more reports of the positive, stimulating effects of alcohol in a sample of social drinkers in an alcohol challenge (Scott & Corbin, 2014), and also related to higher heart rate (a physiological stimulating response) in a sample of males with low socioeconomic status (Bruelle, 2004).

Some studies have failed to find a relation between LR to alcohol effects and sensation seeking. One study of social drinkers participating in an alcohol challenge failed to find a significant relation between individual differences in disinhibition and LR to either positive, stimulating or negative, sedating alcohol effects (Shannon et al., 2011). Another study investigated the influence of disinhibition through items assessing sensation seeking and boredom susceptibility, and evaluated disinhbition in a hierarchical model of the relation between LR to negative, sedative alcohol effects in young adulthood and AUD status 20 years later. LR was unrelated to disinhibition in this model. Interestingly, disinhibition also failed to relate to family history of alcoholism, which is counter to literature supporting disinhibition as a heritable risk factor for developing an AUD (Schuckit & Smith, 2000). The authors suggest that these results reflect the comparatively lower levels of impulsivity that characterized their study sample. Potential participants were excluded for endorsing antisocial personality disorder, and the resulting sample was primarily comprised of high-functioning individuals of middle to upper-

middle class socioeconomic status. These qualities may thus reflect a study sample with comparatively lower levels of impulsivity than would typically be found among individuals with a family history of alcoholism, thereby making it challenging to investigate disinhibition with the sample (Schuckit & Smith, 2000).

It is difficult to draw conclusions from this developing body of literature. Although significant relations have been found between individual differences in sensation seeking and LR to alcohol effects, the nature of this relation is unclear. Even when distinguishing between physiological versus more subjective response to alcohol effects, the results are inconsistent. Thus, there is little consistent support in the current literature for the mediating role of LR in the relation between sensation seeking and alcohol use outcomes.

The scarcity of research on the interplay of personality with LR highlights a gap in the literature, from which the field could gain valuable insight into the development of AUDs. As such, the current study sought to fill this gap in the literature by clarifying how sensation seeking influences the relation between LR to negative, sedative alcohol effects, and adolescent alcohol use outcomes.

**Summary**

In summary, family history of alcoholism predicts individual differences in LR to the negative, sedative effects of alcohol (Schuckit & Gold, 1988; Schuckit et al., 1997), with a comparatively low LR to aversive effects predicting more severe alcohol use and AUD diagnoses both cross-sectionally and over time (Shuckit et al., 2011a; Shuckit et al., 2005a; Schuckit et al., 2011b; Shuckit et al., 2007). These findings are consistent across alcohol challenge and retrospective self-report designs. Research also supports LR to

negative, sedative alcohol effects as a mediator of the relation between parent's life time AUD status, and offsprings' alcohol use and AUD status in adolescent and adult offspring (Shuckit & Smith, 1996; Schuckit et al., 2005b).

The personality trait disinhibition is also a heritable mediator of the relation between parents' AUD status, and alcohol use and misuse (McGue et al., 2011; Iacono & McGue, 2002; Chassin et al., 2004). Disinhibition is comprised of five subfacets, including sensation seeking which is characterized by seeking out thrilling situations and sensitivity to reward (Whiteside & Lynam, 2001; Cyders & Smith, 2007). Having a family history of alcoholism predicts higher levels of sensation seeking (Finn et al., 1992), and individual differences in sensation seeking predict current and future alcohol use and abuse in young adulthood (Wagner, 2001; Sher et al., 2000).

However, the literature is limited in terms of studies investigating whether and how the interplay between LR and sensation seeking contribute to the development of alcoholism. Testing the effects of LR to negative, sedative alcohol effects and sensation seeking together may deepen understanding of the ways in which personality traits and alcohol effects uniquely and together increase risk for AUD development by contributing to problematic alcohol involvement in adolescence.

*Current Study*

The current study had several research goals. The first goal was to corroborate the LR literature, and test whether initial LRs to negative, sedative alcohol effects reported in adolescence mediate the effect of family history of AUDs on early drinking. Previous literature supports both the heritability of a low LR, and LR predicting future alcohol use and alcohol-related problems. Therefore, it was hypothesized that LR to

negative, sedative alcohol effects would mediate the relation between familial alcoholism and adolescent drinking such that a more dense family history of AUDs would predict a lower LR to negative, sedative alcohol effects, which in turn would predict more severe adolescent drinking outcomes.

The second goal was to confirm disinhibition literature, and test whether sensation seeking scores mediate the effect of family history of AUDs on adolescent alcohol consumption. Elevated sensation seeking levels have been linked to familial alcoholism and have predicted more severe alcohol use outcomes (Finn et al., 1992; Sher et al., 2000; Wagner, 2001). Given these findings, it was hypothesized that sensation seeking scores would mediate the relation between family history density of alcoholism and adolescent drinking such that a more dense family history of alcoholism would predict higher sensation seeking levels, which would predict more severe drinking outcomes.

The third research goal was to test whether initial LR to negative, sedative effects reported in adolescence predicted adolescent drinking uniquely over and above sensation seeking. Literature on LR supports a relation between LR and future alcohol use in both adult and adolescents. Therefore it was hypothesized that LR would predict early drinking over and above sensation seeking, such that a lower LR would predict more severe adolescent drinking outcomes.

The fourth and final research goal of the present study was to test whether sensation seeking and LR to negative, sedative alcohol effects during participants' first five drinking experiences interacted to predict early drinking. The APM posits that possessing higher levels of reward-sensitive personality traits, like sensation seeking, will

prepare an individual to learn the positive effects of drinking. An adolescent with high levels of sensation seeking, and a low LR would therefore drink more in order to obtain these positive effects. It was thus hypothesized that sensation seeking would modify the effects of initial LR to negative, sedative alcohol effects on adolescent alcohol use, such that the relation between lower LR and more severe drinking outcomes would be greater as a function of high levels of sensation seeking. A low initial LR to negative, sedative alcohol effects would predict the most drinking in youth high in sensation seeking, followed by individuals with high LR and high sensation seeking, individuals with low LR and low sensation seeking, while a high initial LR would predict the least drinking in youth low in sensation seeking (see Figure 1).

## METHOD

### The Original Study

#### *Participants*

Participants for the study were a subset from a larger, longitudinal study of familial alcoholism (Chassin, Flora, & King, 2004; Chassin, Pillow, Curran, Molina, & Barrera, 1993; Chassin, Pitts, DeLeucia, & Todd, 1999; Chassin, Rogosch, & Barrera, 1991).

At Wave 1, a total of 454 adolescents and their parents were recruited for the study. Participants are distinguished by generation, with adolescents referred to as generation 2 (G2s) and their parents referred to as generation 1 (G1s). Of the G2s recruited, 246 (54%) had one biological custodial parent with an AUD (COAs) and the remaining 204 (46%) were demographically-matched non COA controls. Both G1s and G2s were interviewed annually for three consecutive years (Waves 1-3). Long-term

follow-up interviews started at Wave 4 and continued every five years until Wave 6. G2s and 327 of their full biological siblings were interviewed at Wave 4. Fifty additional G2 full biological siblings were added at Wave 5, and 610 children of G2s (i.e. G3s) were assessed at Wave 6. Between the ages of 11 and 26 years old, G3s were interviewed at 3 time points: the initial Wave 6 interview (Time 1, T1), a follow-up approximately 18 months after the Wave 6 interview (Time 2, T2), and another assessment 36 months after the Wave 6 interview (Time 3, T3).

Participant attrition at each wave was minimal for G1s and G2s, and moderate for G3s. Of the original 454 G2s, 407 (90%) were interviewed at Wave 4, and 412 (91%) of the original G2s and 300 (92%) of the originally added G2 siblings were interviewed at Wave 5. At Wave 6, 816 (90%) of the G2s provided interviews. Of the original 610 G3s interviewed at T1, 560 (92%) were interviewed at T2, and 479 (78%) responded at T3.

*Recruitment*

COA families were recruited by identifying potential G1s through court records, health maintenance organization (HMO) wellness questionnaires, and telephone surveys. Inclusion criteria included: having a child 11-15 years old, Hispanic or non-Hispanic Caucasian ethnicity, birth dates between 1927 and 1960, and Arizona residency. In addition, one biological parent must have met lifetime DSM-III criteria for an AUD.

Control families were recruited via reverse directories that identified families residing in the same neighborhood as COAs. These families were matched according to demographic characteristics (child age, ethnicity, and socioeconomic status), and family composition (one-parent versus two-parent household). To qualify as a control family, no biological or custodial parent could meet DSM-III or Family History – Research

Diagnostic Criteria (FH-RDC) lifetime diagnosis of alcohol dependence or abuse. To reduce future AUD diagnoses in parent controls, seventeen families who endorsed significant but sub-diagnostic alcohol problems were eliminated.

*Recruitment Biases*

There were two sources of potential recruitment bias: bias between individuals contacted versus not contacted, and bias between those who agreed versus refused to participate in the study.

Contact biases in the COA families were evaluated by comparing the archival records (e.g. court records, HMO surveys) of contacted individuals to noncontacted individuals. Among the court record sample, noncontacted individuals were more likely to have lower SES, be female (15.9% vs 14.0%), unmarried (63.7% vs 48.7%), and Hispanic (21.8% vs 18.0%; all p's < .05). There was no significant difference in BAC at time of arrest, number of prior convictions, or MAST scores by contact status. Similarly, within the HMO sample noncontacted individuals were more likely to be female (33.4% vs 27.2%, $p < .05$), and did not differ significantly from contacted individuals in alcohol indicators.

Refusal biases were minimal. In the court record sample, individuals who were screened and refused to participate were more likely to be married (69% vs 50%) and Hispanic (24% vs 18%), but did not differ by an alcohol indicators (e.g. BAC, MAST scores). In the HMO sample, there were no significant differences between those who agreed versus refused to participate. While recruiting matched controls, 91 families responded to demographic questions but refused to participate. These families were compared to those who agreed to participate as matched controls, and were more likely to

have Hispanic mothers and fathers (41% vs 18%, and 40% vs 22%). There were no other significant differences in SES or family composition by refusal status.

*Procedure*

Informed consent procedures were administered by trained interviewers for both parents and children at each wave of assessment. Families were informed they were participating in a study of why some individuals develop problem with alcohol or drugs and others do not, including questions regarding alcohol and other substance use. Interviews were conducted based on convenience for the family, namely at the Arizona State University campus or at families' residences. Those participants who relocated out of the area during the study were interviewed by telephone. To maintain confidentiality, family members were interviewed separately. Interviews were computer-assisted, with participants having the option of entering their responses into the laptop computer themselves, or having interviewers enter their data based on their verbal response to interview questions. Interview duration ranged from 1-2 hours, and families received financial compensation for their time, with the amount of compensation varying over waves.

The Current Study

*Participants*

The present study focused on G3 participants at T1-T3. Participants were included if they responded and had a score for the Self-Rating of the Effects of Alcohol (SRE) form at any of the three assessment time points. If a participant had responded to the SRE at more than one time point, the score and drinking outcomes from the earliest time point was used (e.g. if a participant had responded to the SRE at T1 and T3, SRE scores and

alcohol measures from T1 were used). An additional inclusion criterion was that participants had to be younger than 19 years of age at the time of SRE report. This maximum age limit was implemented to minimize years of retrospection from time of initial first five drinks, and ensure that analysis was limited to reports provided in adolescence. Participants who reported a maximum number of drinks in one day that exceeded lethal levels (e.g. 30 drinks of alcohol in one day) were also excluded (n=7). A sub-sample of n=99 was obtained for analysis in the current study. A comparison of participants included versus excluded from analysis can be found in Table 1, with excluded participants further divided into abstainers, drinkers, and those who reported lethal doses of alcohol consumption and were thus less reliable reporters. As expected given the age based inclusion criterion, included participants were significantly younger and had fewer years of retrospection between the time of SRE report and drinking onset than did excluded participants who consumed alcohol. Surprisingly, the included sub-sample had significantly higher sensation seeking levels, younger age of drinking onset, and greater past year drinking quantity and frequency as compared to excluded participants who drank alcohol. These differences may reflect a particularly at-risk subsample of younger drinkers. However, as compared to excluded drinkers, the included sub-sample had significantly lower SRE scores, and thus lower risk for alcoholism due to higher initial LR to sedative alcohol effects.

As expected, the included subsample had significantly more dense family histories of alcoholism, and higher levels of sensation seeking compared to excluded participants who abstained from drinking. Finally, the included subsample also had significantly less dense family histories of alcoholism, lower SRE scores, and overall less

drinking than excluded participants who reported lethal doses of alcohol consumption. However, these differences are difficult to interpret as the excluded sub-group is small (n=7) and had potentially inaccurate reports of alcohol consumption.

*Measures*

The measures used for the present study were part of a larger interview battery, and these measures are reviewed below. Detailed descriptive statistics for each predictor variable are in Table 2.

*Demographics*

Participants self-reported gender in Wave 6. Gender was dummy-coded such that 1 = females, and 0 = males. Self-reported ethnicity measures were dummy-coded such that 1 = non-Hispanic Caucasian, and 0 = all other ethnicities. Participant age was calculated in years as a continuous variable from date of birth to date of interview.

The subsample was comprised of slightly more non-Hispanic Caucasians (57.6%, n=57) than other ethnic groups, and about equal number of females (52.4%, n=52) and males. Ages of participants at time of SRE report ranged from 13 to 18 years old, with a mean of 16.60 years old. Participants who met all inclusion criteria, but were age 19 years and older at the time of SRE report were excluded from analysis.

*Family History of AUDs*

Family history of AUDs was captured with a family history density (FHD) score, a weighted composite of lifetime AUD diagnosis status for G3's biological parents and biological grandparents.

To obtain G3 parent and grandparent alcohol statuses, self-report of alcohol abuse or dependence across all waves of data collection were used. Depending on the interview

wave, either DSM-III-R, or both DSM-III-R and DSM-IV criteria were used to determine diagnosis status. Using all diagnostic criteria available across all waves through Wave 6, biological parent and grandparent lifetime alcohol diagnoses were binary coded based on any report of alcohol abuse or dependence, or no report of any alcohol abuse or dependence across all waves.

The G3 FHD score is the weighted sum of all of a G3's biological parent and grandparent lifetime AUD diagnosis status, and ranges from 0 to 2. A higher score reflects a more dense family history of AUDs. Because parents each contribute half to their G3 offspring's genetic material, parent lifetime alcohol variables are weighted 0.5. Grandparents contribute to a quarter of G3 offspring genetic material, therefore grandparent lifetime alcohol variables are weighted 0.25. At minimum, the lifetime AUD diagnosis status of one biological parent and two grandparents had to be available to calculate an FHD score. In such a case, the FHD was calculated by first applying the appropriate weights and summing the available lifetime alcohol variables. This score would then be divided by the maximum possible weighted sum for the available lifetime alcohol variables, and then multiplied by 2 to place the score in the 0 to 2 range.

In the present study, participants' mean FHD score was 0.86, which is comparable to one parent and a grandparent, or three grandparents meeting lifetime criteria for an AUD. Thus, participants on average had moderately dense family history of AUDs.

*Level of Response to Negative, Sedative Alcohol Effects*

Retrospective self-reports of LR to negative, sedative alcohol effects were collected using the "during the first five drinking occasions" sub-scale, or time frame, of the SRE (Schuckit, et al., 1997). Respondents are asked to recall the first five times they

drank, and to report the number of drinks they would need at that time in their drinking career to experience four effects of alcohol: (1) “Feel any different,” (2) “A bit dizzy, or begin to slur your speech,” (3) “Stumbling, or walking in an uncoordinated manner,” and (4) “pass out or fall asleep when you did not want.” If a participant did not experience a particular effect from alcohol, the entry was left blank. Conventional scoring of the SRE involves calculating a summary score of LR by obtaining the mean of all responses provided (Schuckit et al., 1997). Higher summary scores reflect requiring a higher number of drinks to obtain each effect, which indicates a lower LR to negative, sedative alcohol effects.

The SRE was developed as an alternative to the more costly, time-consuming alcohol challenge. After controlling for current alcohol consumption, SRE scores correlate with reports of subjective response to alcohol during alcohol challenges, and have distinguished between individuals at high versus low risk for developing an AUD (Shuckit, 1994; Shuckit & Smith, 1996). Thus, there is support in the literature for the construct validity of this measure.

The present study used the first five drinking experiences subscale. To minimize the years of retrospection between first five drinking experiences and time of interview, SRE scores from the initial Wave 6 interview were used. If the SRE was not completed at the initial interview (e.g. the participant had not yet had a full drink of alcohol), SRE scores for the next earliest available time point were used. Additionally, a log10-transformation was performed to normalize the skewed raw SRE scores (see Table 2).

Participants in the present study reported relatively low LR to negative, sedative alcohol effects during their first five drinking occasions, with a mean raw SRE score of

4.73 (mean transformed SRE=.60). Recall, a higher score on the SRE reflects a lower LR to alcohol, which is linked to a higher risk for developing an AUD. An average SRE score of 4.73 is high compared to studies that also collected first five SRE scores from an adolescent population (First 5 SRE mean=3.9, Schuckit, Smith, Danko, et al 2005b; First 5 SRE mean=2.2, Schuckit, et al, 2005d).

*Sensation Seeking*

Sensation seeking was evaluated with six items from a scale developed by Zuckerman (1980), with responses to items on a Likert-scale ranging from (1) "Strongly agree" to (5) "Strongly disagree." The Thrill Seeking subscale from which these items were adapted has acceptable internal consistency, with Cronbach alpha=.80 (Roberti, Storch, & Bravata, 2003). Scale items were reverse-coded and then averaged into a summary score that represented overall sensation seeking, with a high summary score indicating high levels of sensation seeking. This summary score was used as the index of individual sensation seeking level in the present study's analysis. Participants in the present study had a mean sensation seeking score of 3.45, which represents a moderate level of sensation seeking.

*Alcohol Use Outcomes*

Alcohol use was captured with two items from the same Wave 6 time point at which participants provided a SRE score: average quantity of alcohol consumed per occasion and lifetime maximum number of drinks consumed in one day. The average drinking quantity item was adapted from Sher (1993), "When you drink, about how many drinks do you usually have?" with responses ranging from (0) "Zero drinks," to (8) "Nine or more drinks". Participants reported the greatest number of drinks they had ever

consumed in one 24-hour day for the lifetime maximum number of drinks item ("What is the most drinks that you have ever had in a whole 24-hour day period?"). As mentioned earlier, participants who reported drinking lethal doses of alcohol were excluded from analysis as their responses could not reasonably be interpreted.

On average, participants reported consuming 3 drinks per occasion, and imbibing a maximum of 7 drinks in one day. At the time alcohol use was measured, 4% (n=4) of the sample had not consumed alcohol in the past year. The present sample consumed smaller quantities of alcohol compared to national averages and other adolescent-aged samples (Schuckit et al., 2005b; Newes-Adeyi, Chen, William, & Faden, 2005).

*Age of Onset and Years of Retrospection*

Participants self-reported the age at which they had their first drink. The present sample reported ages averaging at 14.21 years old, which is consistent with U.S. national averages of age of onset (Newes-Adeyi et al., 2005). The number of years between participants' first five drinking experiences, and time of retrospectively reporting LR was estimated by subtracting age of first drink from age at time of SRE report (mean=2.39 years).

## RESULTS

*Preliminary Analyses*

All continuous predictors were centered prior to performing further analyses (Cohen, Cohen, West, & Aiken, 2003). Additionally, all continuous predictors fulfilled the assumption of normality, with the exception of the reported SRE score which was moderately positively skewed (see Table 2). To normalize its distribution, a log10

transformation was performed on SRE scores. Refer to Table 2 for additional descriptive information on study variables.

*Power Analysis*

Power analyses for OLS regression models were conducted using GPOWER software (Faul & Erdfelder, 1992) to determine the effect sizes detectable by the analyses in the present study. For all power analyses, the total sample size was set at N = 99, and the alpha level set to .05.

To test the hypotheses that SRE scores predict alcohol consumption over and above sensation seeking, and the interaction between SRE scores and sensation seeking significantly predicts alcohol consumption, there was sufficient power (>.80) to detect a medium ($f^2$=.15) and large ($f^2$=.35) effect size. There was insufficient power (<.80) to detect a small effect size ($f^2$=.02; Cohen, 1988). To test the mediating effect of LR in the relation between FHD and lifetime maximum number of drinks with the PRODCLIN method in RMediation, there was sufficient power (>.80) to detect medium and large effects for the a-path (Family history density of alcoholism predicting lifetime maximum number of drinks), and the c-path (LR predicting lifetime maximum number of drinks). There was insufficient power at .286 to detect a small effect size. Similarly, there was sufficient power to detect medium and large effects, but insufficient power to detect small effect sizes for the b-path (family history density and LR predicting lifetime maximum number of drinks).

*Covariates*

Covariates are included in models to account for error variance not related to the predictor, thereby increasing the power and sensitivity of the analyses. In the present

study, age, gender, and ethnicity were considered for covariates as they were hypothesized to be correlated with drinking outcomes. Additionally, the total number of completed SRE items was considered as a covariate because SRE scores represent the mean of available item responses, and the number of items completed would thus influence the resulting score. The number of years between participants' ages of first drink and ages at time of reporting, or years of retrospection, was also considered as a covariate as it was hypothesized to have a relation with SRE score. Previous drinking could not be controlled for as participants are retrospectively reporting on their first five drinking occasions, and drinking at this time is undocumented and likely varies for each participant.

Gender was not significantly correlated with any outcome or predictor, and was therefore not included as a covariate in the final analysis. However age, ethnicity, total number of complete SRE items, and years of retrospection were included as covariates due to their significant correlation with at least one of the two alcohol outcomes (see Table 3 for correlations among all covariates, predictors, and criterion). Models were estimated 1) with all covariates, and 2) with covariates not correlated with the specific outcome trimmed. There were no differences in significance between the covariate trimmed versus not trimmed models, except for the "Drinking Quantity" outcome for which a main effect changed from significant to non-significant in a marginally significant model. Only outcomes for models with non-correlated covariates trimmed will be presented below (see Tables 10-11 in Appendix B for outcomes of models with all covariates).

*Outlier Diagnostics*

Regression diagnostics were used to detect problematic extreme data points, or outliers for each of the three alcohol use outcomes. Outliers can significantly affect the outcome of a regression analysis by biasing regression coefficients and increasing their standard error thereby distorting results of regression analyses. In the present study, the leverage of each case (i.e. distance of each case relative to the distribution of the other cases on predictors) was calculated to identify potentially problematic cases. Additionally, the externally studentized residuals of the regression equation with versus without each problem case were calculated to identify cases with particularly high residuals; these residuals are the most potent residuals for identifying extreme cases. Global measures of influence for each case were also computed; outcomes were screened for particularly large standardized changes in the predicted score (i.e. DFFITS), and large standardized changes in the regression coefficient (i.e. DFBETAS) due to the presence of a case. If the inclusion of a case or cases led to a one standard deviation or greater change in the predicted score or regression coefficients, the case was further investigated (Neter, Wasserman, and Kutner, 1989).

For "Lifetime Maximum Number of Drinks" and "Average Drinking Quantity" outcomes, the same case had significantly high leverage (> 3.00) and DFFITS that exceeded 1. Closer inspection of the participant's data revealed no apparent interviewer error or reason to consider the participant's reports unreliable. Furthermore, models were re-estimated for both alcohol outcomes without the outlier case. Although there were changes in the estimated coefficient values, the predictors still contributed significant

prediction over and above covariates, and LR was still a significant main effect when the outlier case was removed. Thus, the case was kept in the data set for final analysis.

*Zero-Order Correlations*

Zero-order correlations among predictors, covariates, and alcohol use outcomes are presented in Table 3. Multi-collinearity was minimal, as sensation seeking and SRE were not significantly correlated. As expected, the covariates years of retrospection and number of complete SRE items were significantly related to the SRE predictor variable. These covariates were included to control for measurement error linked with the design of the SRE measure. No other covariate was significantly related to a predictor. Gender was not significantly related to any outcome or predictor. The covariates age and years of retrospection were significantly related to Lifetime Maximum Number of Drinks (r=.252, $p$=.014; and r=.217, $p$=.035*)*, and number of complete SRE items was significantly related to Past Year Drinking Quantity (r=.248, $p$=.015).

Consistent with the hypothesized mediating effect of SRE on the relation between FHD and alcohol outcomes, significant zero-order correlations were found between FHD and SRE (r=.243, $p$=.015), SRE and Lifetime Maximum number of drinks (r=.409, $p$<.001), and between SRE and Past Year Drinking Quantity (r=.331, $p$=.001). These significant relations are also consistent with the hypothesized direct effect of SRE on alcohol use outcomes. FHD and Lifetime Maximum number of drinks were correlated (r=.200, $p$=.052), thereby fulfilling one criterion for a mediating effect (Kenny, 2014; Baron & Kenny, 1986). However, the relation between FHD and Past Year Drinking Quantity was not significant (r=.130, $p$=.204). And contrary to the hypothesized mediating effect of sensation seeking on the relation between FHD and alcohol outcomes,

no significant relation was found between FHD and sensation seeking, or between sensation seeking and any drinking outcome. *Regression Analyses*

Analyses for the present study occurred in two phases: an initial regression analysis phase, and a mediation analysis phase. In the first regression phase of analysis, family history density, sensation seeking, level of response (SRE), the interaction between sensation seeking and LR, and significantly correlated covariates were entered as predictors in a multiple OLS regression model for the two alcohol use outcomes. Thus, two separate regression models were estimated in the first phase of analysis. These analyses tested: if LR interacted with sensation seeking to affect alcohol outcomes; and whether LR and sensation seeking were appropriate for further testing of mediating effects by preliminarily testing if each predicted adolescent alcohol use.

*Model 1: Predicting Lifetime Maximum Number of Drinks from Family History Density of Alcoholism, Initial LR to Negative, Sedative Alcohol Effects, Sensation Seeking, and the Interaction of Sensation Seeking and Initial LR controlling for Years of Retrospection, Age, and Number of Completed SRE Items.*

The first model predicted participants' maximum number of drinks consumed in a 24-hour period from the covariates: years of retrospection, age, number of completed SRE items (Block 1), and predictors: family history density, LR, sensation seeking, and the interaction of LR and sensation seeking (Block 2).

The Block 2 predictors significantly predicted lifetime maximum number of drinks over and above the Block 1 covariates ($F(4,84) = 3.535$, $p = .01$, $R^2$ change = .121). Initial LR to negative, sedative alcohol effects significantly predicted lifetime maximum number of drinks ($\beta = .403$, $p = .001$), such that a lower initial LR to alcohol (that is, a

greater SRE score) predicted greater number of lifetime maximum number of drinks consumed in a day. The interaction term, FHD, and sensation seeking failed to significantly predict lifetime maximum number of drinks consumed in a day (Table 4). See Table 10 in Appendix B for estimated regression coefficients when all covariates were included in Model 1.

*Model 2: Predicting Drinking Quantity from Family History Density of Alcoholism, Initial LR to Negative, Sedative Alcohol Effects, Sensation Seeking, and the Interaction of Sensation Seeking and Initial LR controlling for Number of Completed SRE Items.*

The second model predicted participants' typical number of drinks consumed per occasion from the covariate: number of completed SRE items (Block 1), and predictors: family history density of alcohol use problems, LR, sensation seeking, and the interaction of LR and sensation seeking (Block 2).

The Block 2 predictors contributed marginally significant prediction over and above the number of completed SRE items covariate ($F(88)=2.261$, $p=.069$, $R^2$ change = .087). Initial LR to negative, sedative alcohol effects significantly predicted drinking quantity ($\beta=.778$, $p=.021$), such that reporting a lower initial LR to negative, sedative alcohol effects (a greater SRE score) predicted typically consuming more drinks per occasion. The interaction term, FHD, and sensation seeking failed to significantly predict typical drinking quantity per occasion (Table 5). See Table 11 in Appendix B for estimated regression coefficients when all covariates were included in Model 2.

*Mediation Analyses*

The second phase of analysis sought to test for significant mediating effects in the relation between family history of alcoholism and adolescent drinking using the

PRODCLIN method in RMediation. The PRODCLIN procedure estimates and calculates the significance of the mediation effect from a pair of regression models estimating the individual paths in a mediation model (MacKinnon et al., 2007; see Figure 2). The relation between the predictor and mediator (Path *a*) is estimated with an OLS regression model of the mediator regressed on the predictor. The relation between the mediator and the criterion (Path *b*), and the direct effect of the predictor on the criterion (Path *c*) are estimated with a model of the criterion regressed on both the predictor and the mediator. The regression coefficient for the *a* path in the first model, and the *b* path (i.e. the coefficient for the mediator variable) in the second model, and both models' standard errors are used to estimate the mediated path, and determine the significance of the mediated path coefficient (MacKinnon et al., 2007; Tofighi & MacKinnon, 2011).

Although the SRE (the mediator) predicted both quantity of drinks per occasion and lifetime maximum number of drinks in 24 hours (the criterions), only the latter had a marginally significant correlation with family history density (the predictor; see Table 3). Furthermore, the small and significant direct effect (r=.200, *p*=.052) of family history density on lifetime maximum number of drinks in 24 hours was reduced to negligible, and non-significant (β=.030, *p*=.755) when controlling for the effects of the mediator (i.e. SRE; see Table 4). Thus, an indirect effect of FHD was estimated for the lifetime maximum number drinks outcome as it met criteria that characterize a mediating effect (Kenny, 2014; Baron & Kenny, 1986). Past Year Drinking Quantity was not significantly related to FHD, and thus the indirect effect of FHD through SRE score was not estimated for this alcohol outcome.

Two OLS regression models were run in the second phase of analysis to test the hypothesized mediating effect of LR to negative, sedative alcohol (with the SRE) in the relation between family history density of alcoholism and lifetime maximum number of drinks. To test the *a* path, years of retrospection, age at SRE, and total number of completed SRE items were included as covariates, family history density of alcoholism was entered as a predictor, and LR was entered as the outcome (see Table 6). To test the *b* path, years of retrospection, age at SRE, and total number of completed SRE items were included as covariates, family history density of alcoholism and LR were entered as predictors into a regression model, and lifetime maximum number of drinks was entered as the criterion (see Table 7).

The estimated *a* and *b* path coefficients and standard errors, and correlation between *a* and *b* were entered into Rmediation, with alpha set at .05 to calculate the coefficient, standard error and asymmetric confidence interval for the indirect effect. LR significantly mediated the relation between family history density and lifetime maximum number of drinks, c'=0.876, 95% CI [0.047, 2.174] (see Table 8 or Figure 2), such that a greater family history density of alcoholism predicted a lower LR to alcohol (a greater SRE score) at first five drinking occasions, which predicted higher reported maximum number of drinks consumed in a day.

*Additional Analyses*

Recent research by Lee, Bartholow, McCarthy, Pedersen, & Sher (2014) indicated that conventional scoring of the SRE is biased, such that missing more items predicts a higher SRE summary score and thus a lower LR to negative, sedative effects, and greater risk for alcoholism. Although we sought to attenuate this bias by including the number of

SRE items completed as a covariate, the elevated variance introduced by participants with fewer completed SRE items persisted and can be better accounted for by standardizing SRE items before calculating the mean of the completed items (i.e. standardized person-mean imputation; Lee et al., 2014).

To further test the robustness of the significant mediation of family history density and adolescent substance use by LR to negative, sedative alcohol effects, the mediation analysis was repeated with standardized person-mean imputed first 5 SRE scores. The mediating effects of LR to negative, sedative alcohol effects were no longer significant using this alternate SRE scoring method, however the significant effect of SRE on lifetime maximum number of drinks remained (Table 9). See Tables 12-13 in Appendix B for the estimated coefficients and additional information for each OLS regression computed with the standardized person-mean imputed SRE scores.

## DISCUSSION

The present study tested the interactive and mediating effects of sensation seeking and initial LR to negative, sedative alcohol effects on the relation between family history of alcoholism and adolescent alcohol use. Specifically, the first goal was to replicate previous research that found a significant mediating effect of initial LR to negative, sedative alcohol effects on the relation between family history density of alcoholism and adolescent alcohol use, with a more dense family history predicting *lower* initial LR, which would predict greater alcohol involvement. This hypothesized relation was partially supported by the present study. Next, the present study sought to corroborate previous disinhibition research that found a significant mediating effect of sensation seeking on the relation between familial alcoholism and adolescent alcohol use, with a

more dense family history predicting *higher* sensation seeking, which would predict greater alcohol involvement. Findings from the present study did not support this hypothesis. The third goal of the present study was to test whether initial LR to negative, sedative alcohol effects would predict drinking over and above the effect of sensation seeking. A priori and post-hoc analyses for the present study robustly supported this hypothesis. And finally, the present study tested the hypothesis that the relation between initial LR to negative, sedative alcohol effects and extent of alcohol use among youth drinkers would differ as a function of sensation seeking, such that the effect of lower initial LR on alcohol use would be stronger at high levels of sensation seeking. Findings from the present study did not support this last hypothesized effect. These findings are discussed in greater detail below.

*Level of Response to Negative, Sedative Effects of Alcohol as a Mediator of the Relation Between Family History Density Of Alcoholism and Adolescent Alcohol Consumption*

Using the typical scoring of the SRE measure, the present study supported the hypothesized mediating effect of initial LR to negative, sedative alcohol effects on the relation between family history of alcoholism and adolescent alcohol use. Specifically, a more dense family history of AUDs was significantly related to a lower LR to negative, sedative alcohol effects, which was, in turn, related to increased alcohol consumption over and above the effects of participant age, and years of retrospection. This outcome replicates previous work that found that initial LR to negative, sedative alcohol effects mediates the effects of family history of alcoholism on adolescent alcohol consumption (Schuckit et al., 2005b). It additionally corroborates research demonstrating that a family history of alcoholism predicts comparatively lower LR to negative, sedative alcohol

effects as compared to individuals without a family history (Schuckit et al., 1997), and that lower LR to negative, sedative alcohol effects relates to more problematic drinking as compared to individuals with a higher LR (Schuckit et al., 2011b; Schuckit et al., 2007).

The significant mediating effect found in the present study is consistent with the Enhanced Reinforcement model, the Differentiator Model, and the Low Level of Response Model of risk for alcoholism. The implication is that an enhanced alcohol experience is not limited to an increase in pleasant effects, but can also be obtained through a decrease in unpleasant consequences.

In addition to replicating the role of LR to negative, sedative alcohol effects in the transmission of alcoholism risk, the significant mediation found in the present study is unique in that significance was maintained over and above the effects of years of retrospection. Some evidence of validity for retrospective self-reports of LR to negative, sedative alcohol effects is provided by research findings that these retrospections correlate with levels reported during alcohol challenge designs (Schuckit, Smith, & Tipp, 1997). However, in general, research on retrospective self-reports of substance use highlights the extent to which relying on memory can compromise recall accuracy. A review of adolescent self-reports of health behaviors concluded that as time between the interview and occurrence of the recalled behavior increases (i.e. as temporal proximity decreases), the accuracy of recalled reports of alcohol use decreases (Brener et al.,2003). Additionally, the illegal nature of under-aged drinking may further compromise the accuracy of retrospective reports. In a study of young adults with serious narcotic use disorders, recall accuracy was diminished for more socially undesirable behaviors

(namely substance use) participants had reported 10 years earlier. For less stigmatized behaviors, recall accuracy was still compromised but significantly more accurate (Hser, Anglin, & Chou, 1992).

Taken together, these findings emphasize the need to control for temporal proximity to accurately evaluate the role of initial LR to alcohol effects in the context of alcohol use in adolescents. However, few studies have sought to control for temporal proximity in the context of retrospective reports of LR to alcohol effects. The one other study that explicitly tested the mediating effect of initial LR to negative, sedative alcohol on family history and alcohol consumption only controlled for effects of age (Schuckit et al., 2005b), and age alone does not affect the reliability of retrospective reports of substance use (Hser et al., 1992). In the present study, the years of retrospection measure represents a unique attempt to control the confounding effects of temporal proximity on retrospective reports of initial LR to alcohol. The years of retrospection measure is thus an important control of temporal proximity and reliance on memory in retrospective reports of initial alcohol use. It should be noted that years of retrospection is also confounded with current age and age of onset, since both age and age of onset serve as markers for temporal proximity. In short, although the years of retrospection measure is useful as a control variable, it also represents a complex combination of memory, age of onset, and age that warrants additional research.

Although there was significant mediation using the typical scoring of the SRE measure, these findings were not maintained when using standardized person-mean imputation scoring to attenuate the measure's bias. Specifically, family history density of alcoholism no longer predicted differences in LR to negative, sedative alcohol effects

(see Tables 12-13 in Appendix B for each OLS regression computed with the alternately scored SRE). The failure to find a relation between family history density of alcohol and initial LR to alcohol when the SRE was standardized person-mean imputation coded, suggests that measurement bias may have been driving the relation between familial alcoholism and LR, and by extension the indirect relation between familial alcoholism and adolescent drinking. Because much of the previous literature used the same biased measure of LR to negative, sedative alcohol effects (Schuckit et al., 2005b, Schuckit et al., 2005c), this outcome additionally suggests that the mediating effect of LR to negative, sedative alcohol effects on the relation between family history of alcoholism and adolescent drinking is less clear than previously indicated. The current findings suggest that this literature needs to be re-examined with methods that correct for the downward bias in the conventional SRE scoring methods.

In contrast to its mediating effect, initial SRE continued to predict adolescent alcohol involvement across both conventional and standardized person-mean imputed scoring methods, indicating a robust relation between LR to negative, sedative alcohol effects and adolescent drinking. This finding strengthens previous literature that tested LR effects on adolescent drinking using conventional SRE scoring methods (Schuckit et al., 2005a). Additionally, it further supports initial LR to negative, sedative alcohol effects as a source of enhanced reinforcement that influences subsequent adolescent alcohol involvement (Sher, 1991). In summary, these post hoc analyses highlight the importance of using scoring methods that attenuate the downward bias of the SRE as a measure of LR to negative, sedative alcohol effects (Lee et al., 2014), and provide robust support for the relation between initial SRE and adolescent alcohol involvement.

*Sensation Seeking as a Mediator of the Relation Between Family History Density of Alcoholism and Adolescent Alcohol Consumption*

There was no support for the hypothesized mediating effect of sensation seeking in the relation between family history density of alcoholism and adolescent alcohol consumption. One potential reason for this non-replication is that the drinking characteristics of the present sample may have restricted the extent to which family history density of alcoholism could distinguish among sensation seeking levels. That is, the sample was comprised solely of adolescents who already experimented with alcohol, and were thus more likely to have higher levels of sensation seeking (Stautz & Cooper, 2013). Indeed, the selected subsample had significantly greater mean levels of sensation seeking compared to both excluded drinking and abstaining participants (see Table 1). Furthermore, plotting sensation seeking scores by lifetime maximum number of drinks revealed that most participants demonstrated moderate to high levels of sensation seeking (see Figure 3 in Appendix B). Thus, participants' sensation seeking levels may have had too little variability. Additionally, the alcohol use outcome measure in the present study (i.e. lifetime maximum number of drinks in 24 hours) did not fit the dimensions of alcohol use that previous literature has linked with sensation seeking (i.e. initial onset, binge drinking; Stautz & Cooper, 2013).

Alternatively, a different dimension or more general measure of impulsivity may better capture the indirect effect of family history of alcoholism on adolescent drinking. For example, Chassin and colleages (2004) found that a more general, parent-reported measure of impulsivity significantly mediated the relation between parental alcoholism and drinking trajectories, such that parental alcoholism acted through higher impulsivity

to increase the odds of membership in a heavy drinking trajectory. A more general impulsivity measure may better capture the transmission of increased risk through the mechanism of elevated drinking, as compared to the mechanism of initial experimentation linked with sensation seeking (Stautz & Cooper, 2013).

*The Effects of Initial Level of Response to Negative, Sedative Alcohol Effects, Sensation Seeking, and Family History Density of Alcoholism on Adolescent Alcohol Use*

Consistent with the hypothesized direct effect of initial LR to negative, sedative alcohol effects and its mediating effect reviewed earlier, initial LR to negative, sedative alcohol effects was linked to adolescent alcohol involvement over and above the effects of sensation seeking. Specifically, as initial LR decreased, the past year quantity and lifetime maximum number of drinks youth reported consuming increased, and this relation was maintained even when correcting for the downward bias of the SRE measure. This finding is consistent with previous studies that found a significant relation between LR to negative, sedative alcohol effects and subsequent alcohol use (Schuckit et al., 2011b; Shuckit et al., 2007). However, it strengthens those findings by correcting for the downward bias of the SRE measure. As posited in the Enhanced Reinforcement model, DM, and LLRM of alcoholism risk, this finding suggests that decreased sensitivity to the negative, sedative effects of alcohol may be one pathway to increased alcohol consumption. Negative, sedative effects of alcohol may act as cues to limit consumption and adolescents who experience less of these negative, sedative effects may lack these cues and thus consume more alcohol.

Surprisingly, sensation seeking levels failed to predict alcohol consumption. This finding is not consistent with our hypotheses or with previous literature. One reason for

this failure to replicate may be that the current subsample was composed solely of adolescents who had consumed alcohol. In contrast, other studies used samples with both drinkers and abstainers, and sensation seeking measures significantly distinguished between the two groups (Wagner, 2001; Sher et al., 2000). Previous research found that sensation seeking is more related to initial experimentation with alcohol than with later stages of problematic drinking (Stautz & Cooper, 2013). The degree to which the current subsample had already experimented may account for the lack of relation between drinking and sensation seeking.

In addition, the relation between family history density of alcoholism and adolescent alcohol use was only marginally significant (r=.200, *p*=.052; see Table 3) One potential explanation for this marginal effect is that it is due to other heterogeneities in familial alcoholism besides density of alcoholism. For example, different trajectories of familial alcoholism may exert differential effects on adolescent drinking. Analyses performed in the same data as the present study, but with a different generation, found that active familial alcoholism predicted the greatest adolescent alcohol involvement, as compared to no or remitted familial alcoholism (Chassin, Rogosch, & Barrera, 1991). Persistent familial alcoholism may be an important factor in driving family history effects on alcohol use. Indeed, in the present sample a post-hoc one-factor ANOVA testing for differences in lifetime maximum number of drinks by familial alcoholism persistence indicated significant group differences F(2,64.79)=5.29, *p*=.007. Specifically, planned comparisons revealed that persistent familial alcoholism predicted significantly greater lifetime maximum number of drinks compared to no or developmentally limited familial alcoholism combined t(70.61)=-2.99, *p*=.004. No and developmentally limited familial

alcoholism did not significantly differ in their prediction of alcohol involvement. Thus, characterization of other sources of heterogeneity within familial alcoholism, beyond density of family history, may be key to a more robust significant replication of familial alcoholism effects on adolescent drinking.

*Modifying Effects of Sensation Seeking on Relation Between Initial Low Level of Response to Negative, Sedative Alcohol Effects on Adolescent Alcohol Consumption*

The present study failed to support the hypothesis that sensation seeking and initial LR to negative, sedative alcohol effects would interact to affect adolescent drinking over and above the effects of the family history density of alcoholism. As described earlier, the Acquired Preparedness model posits that individuals with temperaments characterized by greater reward-approach behavior will be more sensitive to learning the rewarding effects of behavior (Smith & Anderson, 2001). Elaborating on this model, we hypothesized that personality may also modify alcohol effects such that the relation between low LR to negative, sedative effects and greater adolescent drinking would be the strongest among individuals high in sensation seeking. Their sensation seeking levels would make them more sensitive to the rewarding effects of alcohol consumption, and most able to drink large quantities before experiencing the impairing, sedative effects of alcohol that discourage continued use.

One reason for the failure to find this interaction may be that the SRE measure did not capture individual differences in the rewarding effects of alcohol, but rather individual differences in the negative, specifically sedative, effects of alcohol. That is, individuals with decreased sensitivity to the negative, sedative effects of alcohol (measured by our SRE items) may not experience their drinking as any more rewarding

as compared individuals with "normal" sensitivity to negative, sedative alcohol effects. Thus, a low LR may not interact with sensation seeking as hypothesized based on the Acquired Preparedness model, because this characterization of LR may not contribute to a more reinforcing drinking experience. A better test of the interaction between sensation seeking and LR to alcohol on subsequent alcohol use might require a measure of LR to positive alcohol effects—either stimulating or sedating. However, there is little research on the interactive effects of impulsivity and LR to alcohol effects on subsequent drinking, and as far as we are aware, little to no studies on the modifying effects of LR to positive, stimulating or positive, sedating alcohol effects. The few studies that tested both impulsivity and LR to positive, stimulating alcohol effects found that greater levels of impulsivity significantly *predicted* greater LR to stimulating alcohol effects (Scott et al., 2014; Leeman, Ralevski, Limoncelli, Pittman, O'Malley, & Petrakis, 2014; dela Pena et al., 2014). This represents a significant gap in the Acquired Preparedness literature and warrants future research as our findings suggest that initial LR to negative, sedative alcohol effects may not be as relevant to the Acquired Preparedness Models as LR to positive stimulating or sedating alcohol effects.

Relatedly, the non-significant interaction may also indicate that the initial LR to negative, sedative alcohol effects interacts with other facets of impulsivity rather than with sensation seeking. This alternative is consistent with a recent study that tested interactions between LR to negative, sedative alcohol effects and facets of impulsivity. Specifically, researchers found that an overall lower LR to negative, sedative alcohol effects strengthened the relation between negative/positive urgency and alcohol use via impaired control, but did not interact with sensation seeking to predict alcohol use

(Wardell, Quilty, & Hendershot, 2015). Thus, future research should not only test broader measures of LR to alcohol effects (i.e. encompassing arousal and valence) but should also test interactions with a broader range of facets of the propensity for rash action.

*Limitations and Future Directions for Research*

Although the current study improved on past research by measuring First 5 SRE at early ages (thus minimizing faulty recall), controlling for years of retrospection, and using a method to correct for bias in SRE, it is also important to consider its limitations. First, the sample was limited to those who drank alcohol. Additionally, the young age of the adolescent participants, and low prevalence of drinking in all G3 study participants, yielded a fairly small sample size. This sample size may not have provided sufficient power to detect small effects, or effects obscured by measurement error. A larger sample of adolescent drinkers may yield stronger effects and should be used in future research.

Second, due in part to participants' drinking rates and in part to the retrospective nature of the LR measure, the present study was limited by its partially cross-sectional design. Participants' family history had temporal precedence to all other measures, but reports of initial LR to negative, sedative alcohol effects, and some reports of sensation seeking and drinking were collected at the same time point. When we tested the indirect effect of family history on adolescent drinking through LR, the mediating variable was meant to represent *initial* LR to negative, sedative alcohol effects, which precedes the time at which participants reported their alcohol use. However, current alcohol consumption strongly biases recall of past alcohol involvement in adolescents (Collins, Graham, Hansen, & Johnson, 1985), and may have biased the recall of initial LR to

negative, sedative alcohol effects in the current study. Future research on potential mediators of the relation between family history of alcoholism and adolescent alcohol use should seek to establish clearer temporal relations among constructs, perhaps through such innovative methods as ecological momentary assessment.

The use of First 5 SRE as the measure of LR to alcohol effects also limited the study to measuring sensitivity to negative, sedative alcohol effects, such as "slurring your speech" or "stumbling." Emerging research posits that sedative alcohol effects can also manifest as pleasant, reinforcing experiences by providing relaxation or stress-relief (Morean, Corbin, & Treat, 2013). Future research should employ a more complete, nuanced index of differential sensitivity to both pleasant and unpleasant, sedative and stimulating alcohol effects, in order to provide a more complete representation of initial LR. Finally, future research should employ a measure of familial alcohol problems that differentiates among other sources of heterogeneity within familial alcoholism, such as differences in severity and persistence over time.

*Summary and Conclusion*

In summary, the current study replicated and strengthened previous research by finding that initial LR to negative, sedative alcohol effects significantly predicted adolescent alcohol involvement, even after controlling for the downward bias of the SRE and controlling for years of retrospection. However, it failed to replicate previous studies that supported a relation between family history of alcoholism and sensation seeking, and sensation seeking and extent of alcohol use among adolescent drinkers. Additionally, although initial LR to negative, sedative alcohol effects significantly mediated the

relation between family history of alcoholism and adolescent alcohol involvement, this finding was not maintained after using a bias-corrected measure of the First 5 SRE.

Findings from the present study highlight methodological considerations that are important to studying the role of LR to alcohol effects and impulsivity in alcoholism. Namely, the role of different facets of impulsivity may vary by stage of alcohol use, the effects of family history may vary due to unaccounted sources of heterogeneity within familial alcoholism, and the effects of SRE may vary with methods used to correct bias in the measure. Taken together, these findings underscore the complexity of studying the familial transmission of alcoholism in adolescent populations.

Table 1. *Comparison of included to excluded participants*

| | **Included** | | **Excluded - Drinkers** | | | **Excluded - Abstinent** | | | **Excluded – Lethal Doses** | | |
|---|---|---|---|---|---|---|---|---|---|---|---|
| | ***N*** | ***Mean (SD)*** | ***N*** | ***Mean (SD)*** | ***t(p-value)*** | ***N*** | ***Mean (SD)*** | ***t(p-value)*** | ***N*** | ***Mean (SD)*** | ***t(p-value)*** |
| Age | 99 | 16.60(1.45) | 88 | 19.01(4.68) | -4.63***(<.001)***$^{***}$ | 316 | 15.74(3.18) | 3.73***(<.001)***$^{***}$ | 7 | 19.13(3.18) | -2.08(*.08*) |
| Age Onset | 99 | 14.21(1.72) | 81 | 16.62(2.40) | -7.56***(<.001)***$^{***}$ | -- | ---- | ----- | 7 | 15.57(1.81) | -1.92(*.10*) |
| Years of Retrospection | 99 | 2.39(1.35) | 40 | 4.08(3.28) | -3.15***(.003)***$^{**}$ | -- | ---- | ---- | 7 | 4.52(3.75) | -1.49(.19) |
| FHD of Lifetime AUD Diagnoses | 99 | .86(.45) | 76 | .80(48) | .92*(.36)* | 575 | .54(.47) | 6.61***(<.001)***$^{***}$ | 6 | 1.29(.33) | -2.99***(.02)**** |
| SRE (raw) | 99 | 4.73(2.73) | 39 | 6.13(2.74) | -2.90***(.005)***$^{**}$ | -- | ---- | ---- | 7 | 11.13(8.74) | -1.93(*.10*) |
| Sensation seeking | 96 | 3.45(.72) | 77 | 3.22(.67) | 2.21***(.03)**** | 381 | 3.04(.72) | 4.97***(<.001)***$^{***}$ | 6 | 3.11(.90) | .91(*.40*) |
| Past year number of drinking occasions | 99 | 2.27(1.38) | 84 | 2.01(1.72) | -11.37 ***(<.001)***$^{***}$ | -- | --- | --- | 7 | 5.86(2.73) | -4.96***(.008)***$^{**}$ |
| Past year drinking quantity per occasion | 97 | 3.20(2.32) | 88 | 2.11(1.61) | -12.33 ***(<.001)***$^{***}$ | -- | --- | --- | 7 | 3.86(1.46) | -6.97***(<.001)***$^{*}$$^{**}$ |
| Lifetime maximum number of drinks | 95 | 7.28(5.50) | 79 | 6.30(5.64) | 1.15(*.25*) | -- | --- | --- | 7 | 31.71(14.63) | -4.40(*.004*)† |
| | *N* | *%* | *N* | *%* | | *N* | *%* | | *N* | *%* | $\chi^2$*(p-value)* |
| G3 Gender<br>0=Male (M)<br>1=Female (F) | 99 | 47.6% M,<br>52.4% F | 99 | 45.5% M<br>54.5% F | | 432 | 45.5% M<br>54.5% F | | 7 | 71.4% M<br>28.6% F | 5.56 (*.14*) |
| G2 Ethnicity<br>0=Hispanic/<br>Other Ethnicity(H)<br>1=Caucasian (C) | 99 | 42.4% H<br>57.6% C | 94 | 51.1% H,<br>48.9% C | | 565 | 51.1% H<br>48.9% C | | 7 | 57.1% H<br>42.9% C | 9.45(*.02*) |

*Note.* G3 Age reflects participant age in years at time of SRE report. SRE scores are summary of scores of level of response to alcohol during first five drinking occasions where a higher score reflects a lower level of response. Years of Retrospection is the time, in years, between participants age of first drink and age at time of SRE report. Mixed model ANOVAs with planned comparisons between each excluded group versus the included group were run to identify significant group differences. *Significant at *p<.05, ** p<.01, ***p<.001* †This excluded subgroup was created based on the unusually high, lethal number of drinks participants reported consuming in one day.

Table 2. *Descriptive statistics for Subsample of Wave6 G3 Participants*

| | N | Min. | Max. | Mean (SD) | Skewness (SE) | Kurtosis (SE) |
|---|---|---|---|---|---|---|
| G3 Age | 99 | 13 | 18.94 | 16.60(1.45) | -.29(.24) | -.50(.48) |
| G3 Age of Onset | 99 | 11 | 18 | 14.21(1.72) | -.08(.24) | -.52(.48) |
| G3 Years of Retrospection | 99 | 0 | 6.87 | 2.39(1.35) | 1.10(.24) | 1.10(.48) |
| G3 SRE Item Count Total | 99 | 1 | 4 | 2.53(1.06) | .01(.24) | -1.22(.48) |
| G3 Family History Density of Lifetime AUD Diagnoses | 99 | 0 | 1.75 | .86(.45) | -.21(.24) | -.59(.48) |
| G3 report of SRE | 99 | 1 | 13.67 | 4.73(2.73) | .86(.24) | .40(.48) |
| G3 report of SRE (Transformed) | 99 | 0 | 1.14 | .60(.27) | -.40(.24) | -.37(.48) |
| G3 report of sensation seeking | 96 | 1.33 | 4.83 | 3.45(.72) | -.30(.25) | .32(.49) |
| G3 report of past year drinking quantity per occasion | 97 | 0 | 9 | 3.20(2.32) | 1.09(.25) | .47(.49) |
| G3 report of lifetime maximum number of drinks | 95 | 0 | 22 | 7.28(5.50) | 1.03(.25) | .17(.49) |
| | **N** | **%** | | | | |
| G3 Gender | 99 | 52.4% (N=52) Female | | | | |
| G3 Ethnicity | 99 | 57.6% (N=57) Non-Hispanic Caucasian | | | | |

*Note.* G3 Age reflects participant age in years at time of SRE report. SRE scores are summary of scores of level of response to alcohol during first five drinking occasions where a higher score reflects a lower level of response. Years of Retrospection is the time, in years, between participants age of first drink and age at time of SRE report.

Table 3. *Correlations among variables*

| | | G3 Age | Years of Retro | # SRE Items | Gender | Ethnicity 1= Cauc 0 = Other | FHD | Sensation Seeking | SRE Score | SRE Score (Trans) | Past Year Drinking Quantity | Life Max # of Drinks |
|---|---|---|---|---|---|---|---|---|---|---|---|---|
| G3 Age | Pearson Correlation | 1 | .247* | -.138 | .048 | .010 | -.032 | -.114 | .100 | .071 | .123 | .252* |
| | Sig. (2-tailed) | | .014 | .172 | .639 | .921 | .755 | .270 | .322 | .483 | .230 | .014 |
| | N | 99 | 99 | 99 | 99 | 99 | 99 | 96 | 99 | 99 | 97 | 95 |
| Years of Retro | Pearson Correlation | .247* | 1 | .086 | -.100 | .043 | .103 | .127 | .199* | .148 | .004 | .217* |
| | Sig. (2-tailed) | .014 | | .398 | .326 | .675 | .310 | .218 | .048 | .145 | .971 | .035 |
| | N | 99 | 99 | 99 | 99 | 99 | 99 | 96 | 99 | 99 | 97 | 95 |
| # SRE Items | Pearson Correlation | -.138 | .086 | 1 | .102 | .001 | .129 | .049 | .512** | .525** | .248* | .176 |
| | Sig. (2-tailed) | .172 | .398 | | .317 | .991 | .202 | .633 | .000 | .000 | .015 | .088 |
| | N | 99 | 99 | 99 | 99 | 99 | 99 | 96 | 99 | 99 | 97 | 95 |
| Gender | Pearson Correlation | .048 | -.100 | .102 | 1 | -.125 | .039 | -.026 | .041 | .065 | .089 | .070 |
| | Sig. (2-tailed) | .639 | .326 | .317 | | .217 | .699 | .799 | .686 | .520 | .384 | .499 |
| | N | 99 | 99 | 99 | 99 | 99 | 99 | 96 | 99 | 99 | 97 | 95 |
| Ethnicity 1= Cauc 0 = Other | Pearson Correlation | .010 | .043 | .001 | -.125 | 1 | .117 | .046 | -.162 | -.183 | -.088 | -.106 |
| | Sig. (2-tailed) | .921 | .675 | .991 | .217 | | .248 | .656 | .108 | .069 | .391 | .306 |
| | N | 99 | 99 | 99 | 99 | 99 | 99 | 96 | 99 | 99 | 97 | 95 |
| FHD | Pearson Correlation | -.032 | .103 | .129 | .039 | .117 | 1 | .027 | .243* | .250* | .130 | .200 |
| | Sig. (2-tailed) | .755 | .310 | .202 | .699 | .248 | | .792 | .015 | .012 | .204 | .052 |
| | N | 99 | 99 | 99 | 99 | 99 | 99 | 96 | 99 | 99 | 97 | 95 |
| Sensation Seeking | Pearson Correlation | -.114 | .127 | .049 | -.026 | .046 | .027 | 1 | .121 | .130 | .142 | .003 |
| | Sig. (2-tailed) | .270 | .218 | .633 | .799 | .656 | .792 | | .242 | .207 | .172 | .976 |
| | N | 96 | 96 | 96 | 96 | 96 | 96 | 96 | 96 | 96 | 94 | 92 |
| SRE Score | Pearson Correlation | .100 | .199* | .512** | .041 | -.162 | .243* | .121 | 1 | .941** | .331** | .409** |
| | Sig. (2-tailed) | .322 | .048 | .000 | .686 | .108 | .015 | .242 | | .000 | .001 | .000 |
| | N | 99 | 99 | 99 | 99 | 99 | 99 | 96 | 99 | 99 | 97 | 95 |
| SRE Score (Trans) | Pearson Correlation | .071 | .148 | .525** | .065 | -.183 | .250* | .130 | .941** | 1 | .331** | .435** |
| | Sig. (2-tailed) | .483 | .145 | .000 | .520 | .069 | .012 | .207 | .000 | | .001 | .000 |
| | N | 99 | 99 | 99 | 99 | 99 | 99 | 96 | 99 | 99 | 97 | 95 |
| Past Year Drinking Quantity | Pearson Correlation | .123 | .004 | .248* | .089 | -.088 | .130 | .142 | .331** | .331** | 1 | .618** |
| | Sig. (2-tailed) | .230 | .971 | .015 | .384 | .391 | .204 | .172 | .001 | .001 | | .000 |
| | N | 97 | 97 | 97 | 97 | 97 | 97 | 94 | 97 | 97 | 97 | 93 |
| Life Max# Drinks | Pearson Correlation | .252* | .217* | .176 | .070 | -.106 | .200† | .003 | .409** | .435** | .618** | 1 |
| | Sig. (2-tailed) | .014 | .035 | .088 | .499 | .306 | .052 | .976 | .000 | .000 | .000 | |
| | N | 95 | 95 | 95 | 95 | 95 | 95 | 92 | 95 | 95 | 93 | 95 |

†p<.1, *p < .05, **p < .001 Note: Due to the skewedness/kurtosis of the SRE scores, a log10 transformation was performed. The resulting log10 transformed SRE scores are represented as SRE Score (Transformed) in the correlation table.

Table 4.

*Regression Analysis Predicting Lifetime Maximum Number of Drinks from Family History Density of Alcoholism (FHD), Initial Level of Response to Alcohol (LR), Sensation Seeking (SS), and the potential interaction of LR x SS with non-significantly correlated covariates trimmed.*

| Variable | *B* | *SE B* | β | $R^2$ |
|---|---|---|---|---|
| Block 1 | | | | .159** |
| Years of retrospection | .716 | .423 | .171 | |
| Age | .903 | .369 | .250* | |
| Number of Completed SRE Items | 1.276 | .514 | .246* | |
| Block 2 | | | | .280* |
| Years of retrospection | .584 | .416 | .139 | |
| Age | .683 | .359 | .189 | |
| Number of Completed SRE Items | .188 | .567 | .036 | |
| Family History Density | .374 | 1.195 | .030 | |
| Sensation Seeking | -.302 | .709 | -.041 | |
| Level of Response | 8.184 | 2.292 | .403** | |
| Level of Response x Sensation Seeking | -.084 | 2.783 | -.003 | |

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

Table 5.

*Regression Analysis Predicting Drinking Quantity from Family History Density of Alcoholism (FHD), Initial Level of Response to Alcohol (LR), Sensation Seeking (SS), and the potential interaction of LR x SS with non-significantly correlated covariates trimmed.*

| Variable | *B* | *SE B* | β | $R^2$ |
|---|---|---|---|---|
| Block 1 | | | | .065* |
| Number of Completed SRE Items | .576 | .227 | .256* | |
| Block 2 | | | | .153† |
| Number of Completed SRE Items | .251 | .257 | .111 | |
| Family History Density | .147 | .548 | .027 | |
| Sensation Seeking | .278 | .325 | .086 | |
| Level of Response | 2.403 | 1.019 | .278* | |
| Level of Response x Sensation Seeking | 1.661 | 1.268 | .131 | |

†$p$<.1, *$p$ < .05

Table 6.

*Regression Analysis Predicting Initial Level of Response to Alcohol from Family History Density of Alcoholism Controlling For Years Of Retrospection, Age At SRE, And Total Number Of Completed Items*

| Variable | *B* | *SE* | β | $R^2$ |
|---|---|---|---|---|
| Block 1 | | | | .302** |
| Years of retrospection | .014 | .018 | .070* | |
| Age | .024 | .017 | .128 | |
| Number of Completed SRE Items | .138 | .022 | .087 | |
| Block 2 | | | | .334* |
| Years of retrospection | .010 | .018 | .051 | |
| Age | .026 | .017 | .136 | |
| Number of Completed SRE Items | .133 | .022 | .516** | |
| Family History Density | .111 | .052 | .183* | |

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

Table 7.

*Regression Analysis Predicting Lifetime Maximum Number of Drinks from Family History Density of Alcoholism and Initial Level of Response to Alcohol Controlling For Years Of Retrospection, Age At SRE, And Total Number Of Completed Items*

| Variable | *B* | *SE* | β | $R^2$ |
|---|---|---|---|---|
| Block 1 | | | | .126** |
| Years of retrospection | .579 | .423 | .139 | |
| Age | .913 | .381 | .245* | |
| Number of Completed SRE Items | 1.028 | .524 | .196 | |
| Block 2 | | | | .252*** |
| Years of retrospection | .357 | .400 | .086 | |
| Age | .741 | .361 | .199* | |
| Number of Completed SRE Items | -.060 | .572 | -.011 | |
| Family History Density | 1.154 | 1.159 | .095 | |
| Level of Response | 7.893 | 2.307 | .383** | |

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

Table 8.
*Mediation analysis using the PRODCLIN method in RMediation predicting an indirect effect of Family History Density of Alcoholism on Lifetime Maximum Number of Drinks through Initial Level of Response to Alcohol.*

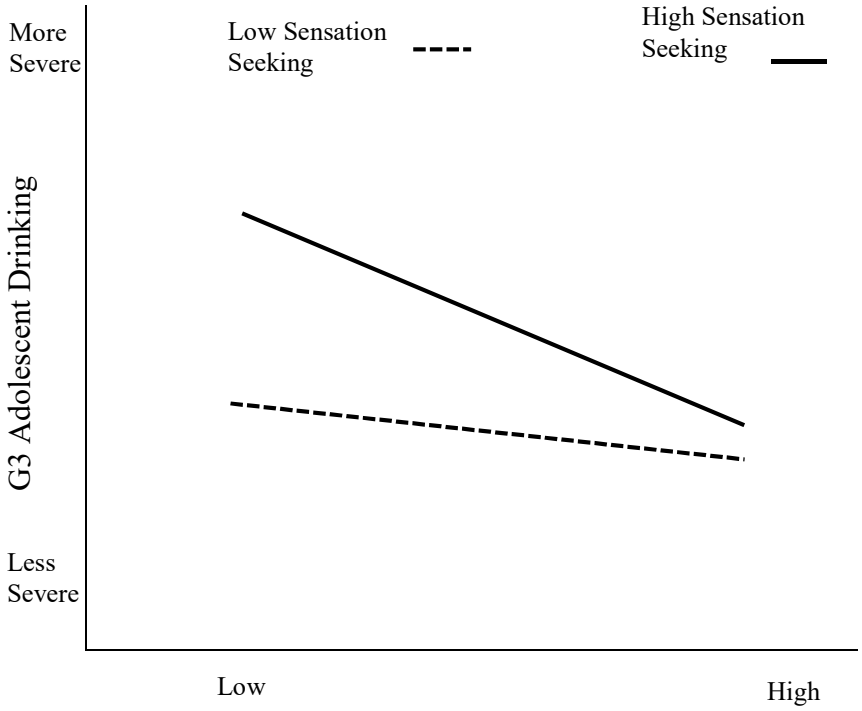
| Model | *a* coefficient (S.E.) | *b* coefficient (S.E.) | $\rho_{ab}$ | 95% CI | *c′* coefficient (S.E.) |
|---|---|---|---|---|---|
| FHD → SRE → Lifetime Max Drinks | .111* (.052) | 7.893** (2.307) | .262 | [.047, 2.174] | 0.876* (.552) |

Table 9.
*Mediation analysis using the PRODCLIN method in RMediation predicting an indirect effect of Family History Density of Alcoholism on Lifetime Maximum Number of Drinks through Initial Level of Response to Alcohol using standardized person-mean imputed SRE scores.*

| Model | *a* coefficient (S.E.) | *b* coefficient (S.E.) | $\rho_{ab}$ | 95% CI | *c′* coefficient (S.E.) |
|---|---|---|---|---|---|
| FHD → SRE(Alt Scoring) → Lifetime Max Drinks | .279 (.184) | 2.277* (.639) | .163 | [-.16, 1.776] | 0.635 (.496) |

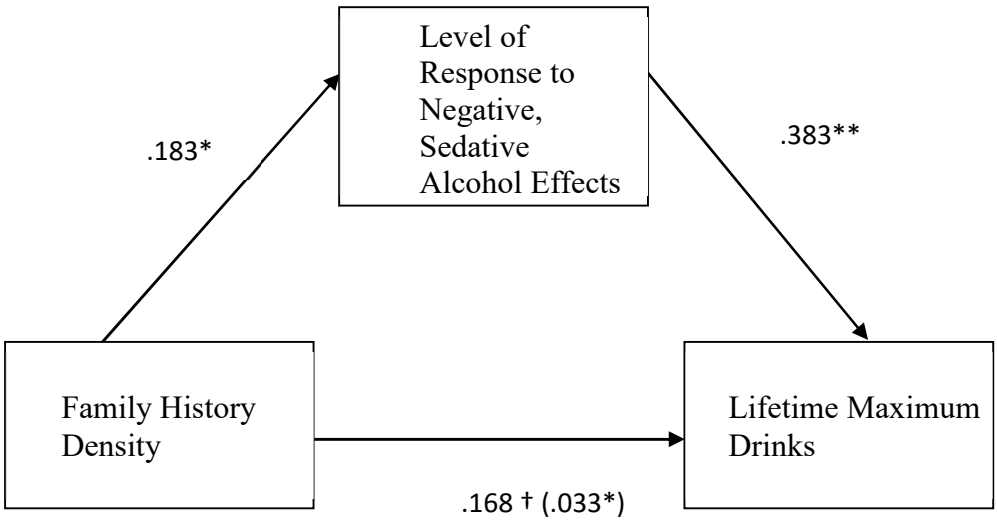
Figure 1.

*Graph of the hypothesized relation between level of response to negative, sedative effects of alcohol and G3 adolescent drinking as a function of sensation seeking levels.*




**Note: Sensation seeking is hypothesized to moderate the effect of LR during first five drinking occasions on adolescent drinking outcomes, such that the relation between initial LR to negative, sedative alcohol effects and adolescent drinking would be stronger among individuals high on sensation seeking as compared to individuals low on sensation seeking.*

Figure 2.
*Model testing hypothesis that initial LR to negative, sedative alcohol effects mediates the relation between family history density of alcoholism and lifetime maximum number of drinks, using standardized regression coefficients.*




*†p<.1, *p<.05, **p<.001*
*Note: the indirect effect is represented in parentheses*

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# APPENDIX A

## SRE SUB-SCALE, SENSATION SEEKING SCALE & ALCOHOL CONSUMPTION ITEMS

## SELF-RATING OF THE EFFECTS OF ALCOHOL (SRE): FIRST-FIVE DRINKING OCCASIONS SUB-SCALE ITEMS

Think about the first five occasions you had a full drink of alcohol.
How many standard alcoholic drinks did it take for you to:

1. Feel any different?

2. Feel a bit dizzy, or to begin to slur your speech?

3. Begin stumbling, or walking in an uncoordinated manner?

4. Pass out or fall asleep when you did not want?

SENSATION SEEKING SCALE

Please rate how much you agree with the following statements:

1.) I like wild parties
   (1) Strongly Agree
   (2) Agree
   (3) Neither Agree nor Disagree
   (4) Disagree
   (5) Strongly Disagree

2.) I like being where there is something going on all the time
   (1) Strongly Agree
   (2) Agree
   (3) Neither Agree nor Disagree
   (4) Disagree
   (5) Strongly Disagree

3.) I would do almost anything on a dare.
   (1) Strongly Agree
   (2) Agree
   (3) Neither Agree nor Disagree
   (4) Disagree
   (5) Strongly Disagree

4.) I like work that has lots of excitement.
   (1) Strongly Agree
   (2) Agree
   (3) Neither Agree nor Disagree
   (4) Disagree
   (5) Strongly Disagree

5.) I like to have new and exciting experiences, even if they are a little unconventional.
   (1) Strongly Agree
   (2) Agree
   (3) Neither Agree nor Disagree
   (4) Disagree
   (5) Strongly Disagree

6.) I often long for excitement.
   (1) Strongly Agree
   (2) Agree
   (3) Neither Agree nor Disagree
   (4) Disagree
   (5) Strongly Disagree

ALCOHOL CONSUMPTION ITEMS

1. When you drink about how many cans of beer, glasses of wine, bottles of wine cooler, or "shots" of hard liquor do you usually have?

| | |
|---|---|
| 0 ..... | 0 |
| 1 ..... | 1 |
| 2 ..... | 2 |
| 3 ..... | 3 |
| 4 ..... | 4 |
| 5 ..... | 5 |
| 6 ..... | 6 |
| 7-8 ..... | 7 |
| 9 or more .. | 8 |
| . ..... | 9 |

2. What is the most drinks that you have ever had in a whole 24-hour day period?

# APPENDIX B

# REGRESSION MODELS WITH ALL COVARIATES, REGRESSION MODEL PREDICTING SRE FROM ALL COVARIATES, AND MODELS FOR PRODCLIN PROCEDURE USING ALTERNATE SRE SCORE METHOD

Table 10. *Regression Analysis Predicting Lifetime Maximum Number of Drinks from Family History Density of Alcoholism (FHD), Initial Level of Response to Alcohol (LR), Sensation Seeking (SS), and the potential interaction of LR x SS with all covariates.*

| Variable | *B* | *SE B* | B | $R^2$ |
|---|---|---|---|---|
| Block 1 | | | | .170** |
| Ethnicity | -1.141 | 1.064 | -.105 | |
| Years of Retrospection | .736 | .424 | .176 | |
| Age | .908 | .368 | .251* | |
| Number of complete SRE Items | 1.283 | .514 | .247* | |
| Block 2 | | | | .111* |
| Ethnicity | -.451 | 1.051 | -.041 | |
| Years of Retrospection | .590 | .419 | .141 | |
| Age | .693 | .361 | .192 | |
| Number of completed SRE Items | .216 | .574 | .042 | |
| Level of Response | 7.948 | 2.368 | .391** | |
| Sensation Seeking | -.279 | .714 | -.038 | |
| Level of Response x Sensation Seeking | -.058 | 2.798 | -.002 | |
| Family History Density | .460 | 1.218 | .037 | |

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

Table 11. *Regression Analysis Predicting Average Drinking Quantity from Family History Density of Alcoholism (FHD), Initial Level of Response to Alcohol (LR), Sensation Seeking (SS), and the potential interaction of LR x SS with all covariates.*

| Variable | *B* | *SE B* | B | $R^2$ |
|---|---|---|---|---|
| Block 1 | | | | .106* |
| Ethnicity | -.494 | .476 | -.104 | |
| Years of Retrospection | -.082 | .184 | -.047 | |
| Age | .292 | .167 | .183 | |
| Number of complete SRE Items | .639 | .229 | .284** | |
| Block 2 | | | | .082† |
| Ethnicity | -.346 | .487 | -.073 | |
| Years of Retrospection | -.208 | .186 | -.117 | |
| Age | .283 | .168 | .178 | |
| Number of completed SRE Items | .368 | .265 | .163 | |
| Family History Density | .290 | .555 | .054 | |
| Sensation Seeking | .401 | .330 | .123 | |
| Level of Response | 1.953 | 1.080 | .226 | |
| Level of Response x Sensation Seeking | 1.873 | 1.282 | .148 | |

**$p < .01$, *$p < .05$, †$p<.1$

Table 12. *Regression Analysis Predicting Initial Level of Response to Alcohol from Family History Density of Alcoholism Controlling For Years Of Retrospection, Age At SRE, And Total Number Of Completed Items using standardized person-mean imputed SRE scores*

| Variable | *B* | *SE* | β | $R^2$ |
|---|---|---|---|---|
| Block 1 | | | | .016 |
| Years of retrospection | .070 | .063 | .116 | |
| Age | .019 | .058 | .033 | |
| Block 2 | | | | .040 |
| Years of retrospection | .059 | .063 | .098 | |
| Age | .024 | .058 | .043 | |
| Family History Density | .279 | .184 | .154 | |

Table 13. *Regression Analysis Predicting Lifetime Maximum Number of Drinks from Family History Density of Alcoholism and Initial Level of Response to Alcohol Controlling For Years Of Retrospection, Age At SRE, And Total Number Of Completed Items using standardized person-mean imputed SRE scores*

| Variable | *B* | *SE* | β | $R^2$ |
|---|---|---|---|---|
| Block 1 | | | | .299* |
| Years of retrospection | .690 | .425 | .166 | |
| Age | .791 | .382 | .212* | |
| Block 2 | | | | .482*** |
| Years of retrospection | .423 | .401 | .102 | |
| Age | .772 | .355 | .207* | |
| Family History Density | 1.644 | 1.145 | .135 | |
| Level of Response | 2.277 | .639 | .327** | |

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

Figure 3. *Plot of self-reported levels of sensation seeking in early adolescence by lifetime maximum number of drinks consumed in 24 hours.*