COMMON AND SPECIFIC ALCOHOL RISK FACTORS IN AFRICAN AMERICANS
AND CAUCASIANS

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SARAH PEDERSEN

Dr. Denis McCarthy, Dissertation Supervisor

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The undersigned, appointed by the Dean of the Graduate School, have examined the dissertation entitled

COMMON AND SPECIFIC ALCOHOL RISK FACTORS IN AFRICAN AMERICANS AND CAUCASIANS

Presented by Sarah Pedersen

A candidate for the degree of Doctor of Philosophy

And hereby certify that in their opinion it is worthy of acceptance.

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Denis McCarthy, Ph.D.

_____

Glenn Good, Ph.D.

_____

Anna Bardone-Cone, Ph.D.

_____

Timothy Trull, Ph.D.

_____

Bruce Bartholow, Ph.D.

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ABSTRACT

Considerable research evidence shows that the alcoholism risk process differs for African Americans and Caucasians (Bachman et al., 1991; Wallace et al., 2003). The current study was designed to test a model examining racial differences in alcohol use. I examined both common factors that function similarly across race and context specific factors that differ in their association with alcohol across race.

One hundred and seventy eight participants (mean age = 21.87, SD = 1.23; 57% African descent) completed an alcohol administration study, receiving a moderate dose of alcohol (0.72g/kg alcohol for males, 0.65g/kg for females). Acute alcohol response was measured at 8 time points (i.e., baseline, 15, 30, 45, 60, 90, 120, and 150 minutes).

Latent variable growth models largely supported acute alcohol response as a common risk factor that functions similarly across racial groups. Regression analyses showed support for a mean difference model where African Americans had lower levels of risk factors (e.g., disinhibition) and higher levels of protective factors (e.g., religiosity) compared to Caucasians. The implications and limitations of these findings are discussed.

INTRODUCTION

Differences in Alcohol Use between African Americans and Caucasians

Considerable research evidence shows that the alcoholism risk process differs for African Americans and Caucasians. Results from the National Comorbidity Survey Replication data found that the lifetime prevalence of alcohol use disorders was lower for African Americans (9.5%) compared to Caucasians (13.4%) and that African Americans have a lower lifetime risk of developing a substance use disorder (Breslau, Aguilar-Gaxiola, Kendler, Su, Williams, & Kessler, 2006). Additionally, African American adolescents begin drinking later (Bachman, Wallace, O'Malley, Johnston, Kurth, & Neighbors, 1991; Barnes & Welte, 1986), engage in less heavy drinking (Wallace, Bachman, O'Malley, Schulenberg, Cooper, & Johnston, 2003; Bachman et al., 1991), show slower increases in rates of drinking (Warheit, Vega, Khoury, Gil, & Elfenbein, 1996), and have higher abstention rates (Substance Abuse and Mental Health Services Administration, 2003). Differences between these groups have been found to be quite large in adolescence and early adulthood. However, this gap diminishes significantly in adulthood, with alcohol use peaking in the early twenties for Caucasians but not peaking until the early thirties for African Americans (Herd, 1989). Despite lower rates of alcohol use disorders, there is some evidence for increased persistence of alcohol dependence and more frequent engagement in heavy drinking occasions in African Americans (Dawson, 1998). Also, at similar levels of alcohol consumption, African Americans are more likely to experience alcohol-related problems than Caucasian drinkers (Caetano & Kaskutas,

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1995).

There are several potential explanations for these between-group differences in alcohol use patterns. One is that African Americans may have lower mean levels of common risk factors, such as disinhibited personality characteristics or alcohol expectancies (McCarthy, Miller, Smith, & Smith, 2001). African Americans may also have higher mean levels of protective factors, such as religiosity. However, the complexity of observed differences between African American and Caucasian drinking patterns and alcohol-related problems indicate the need for an integrative model to account for differences in the alcoholism risk process.

Smith and colleagues (Smith, Spillane, & Annus, 2006) put forth a broad model for psychologists that draws a contrast between universal and relative risk factors. Smith and colleagues identify universal or common risk factors as factors that have been found in a wide range of cultures and have been found to function similarly across cultures. Affect and personality are two examples provided by Smith of constructs that are relatively invariant across cultures (e.g., individuals across cultures experience pleasure, neuroticism is found in multiple cultures). On the other hand, relative or context specific factors are culturally bound or differ by culture. For example, different events or experiences produce pleasure in different cultures and the expression of pleasure may differ across groups.

Alcohol use disorders and eating disorders are discussed within this model as examples where a common risk factor, such as impulsivity, results in a disorder (Bulimia), only in Western cultures where there is unlimited access to food combined

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with a societal thin-ideal (context specific risk factor). Recently, Spillane and Smith (2007) applied this cross-cultural model to alcoholism risk in reservation-dwelling American Indians. In this model, contextual factors are presented as a way to explain the higher mean levels of drinking seen in American Indian populations compared with Caucasians. For example, Spillane and Smith (2007) argue that perceived discrimination, modeling, and socioeconomic status are risk factors that are more likely to be experienced by this group and can explain higher mean levels of drinking behavior. However, Spillane and Smith (2007) also identify common factors that are thought to explain individual differences in drinking seen within American Indians. These common risk factors (e.g., alcohol expectancies, emotional distress, family history) are thought to increase risk for alcohol-related problems and heavy use similarly across racial and ethnic groups. The current study represents an extension of the model discussed by Smith and colleagues (2006), by examining alcohol response as a common risk factor that occurs across (all drinking) cultures but where context specific factors (e.g., peer use) may constrain or facilitate alcohol response's association with alcohol use across African Americans and Caucasians.

The current study incorporated both common and context specific risk and protective factors to account for both mean level differences between African Americans and Caucasians and individual differences within groups in drinking behavior and alcohol-related problems. Response to alcohol, disinhibition, and alcohol expectancies were tested as common risk factors that were thought to have similar influence on alcohol use in each racial group. While alcohol response has been shown to function similarly

across a variety of racial groups (e.g., Asian; Luczak, Elvine-Kries, Shea, Carr, & Wall, 2002; Native American; Garcia-Andrade, Wall, & Ehlers, 1997; Latino; Schuckit, Smith, & Kalmijn, 2004), only one study has examined the validity of alcohol response as a marker of risk for heavy alcohol use in African Americans (Pedersen & McCarthy, 2009). These results demonstrated that alcohol response functioned similarly for African Americans as has been found in other racial/ethnic groups. However, a limitation of that study was that there was not a Caucasian sample to allow for direct racial comparisons. As part of the current study, racial differences in response to alcohol were directly examined to determine if it indeed functions as a common risk factor for African American alcohol use and related problems. Socioeconomic status, religiosity, peer drinking, and family modeling were examined as social/contextual factors that may differ across groups in both mean levels and in the prediction of drinking behavior. These social/contextual factors were also tested as potential moderators of the association between common risk factors and alcohol-related behavior. Lastly, alcohol expectancies were examined as a mediator for both common and contextual factors and alcohol-related behavior.

Response to Alcohol

The proposed study tested response to alcohol as a common risk factor that functions similarly across African Americans and Caucasians. Models of alcoholism risk have increasingly focused on integrating genetic and psychosocial etiologic factors. One way in which genetics influences risk for alcoholism is through response to alcohol. Alcohol response has been found to be heritable (Heath & Martin, 1991; Heath et al.,

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1999; Viken, Rose, Morzorati, Christian, & Li, 2003) and to differentiate between children of alcoholics and controls (Schuckit, 1985; Schuckit & Gold, 1988). Additionally, alcohol response has been shown to influence risk for alcohol dependence (Schuckit & Smith, 2001) and to relate to increased heavy alcohol use (e.g., Conrod, Peterson, & Pihl, 2001). Recently, it has been shown that having a low level of response assessed by both retrospective report (Schuckit et al., 2007) and through alcohol challenge paradigms (e.g., Schuckit & Smith, 2000), prospectively predicts alcohol related problems and heavy use.

However, response to alcohol is a complex construct as both dampened (e.g., Schuckit, 1980, 1984; Schuckit & Smith, 2001) and heightened (e.g., Finn, Zeitouni, & Pihl, 1990, Conrod, Pihl, Ditto, 1995; Conrod et al., 2001) response have been found to be associated with risk. Newlin and Thompson (1990) synthesized these findings by showing that in sons of alcoholics, dampened response to the sedating effects of alcohol on the descending limb, when blood alcohol levels are decreasing, and increased response to alcohol on the ascending limb, when blood alcohol levels are increasing, are related to increased risk. Additionally several different ways of assessing alcohol response have been developed (e.g., retrospective vs. acute; subjective vs. physiological) and these differences in assessment methodology may influence results.

Response to alcohol has been studied in Asian (e.g., Luczak, et al., 2002), Native American (Garcia-Andrade, et al., 1997), and Latino (Schuckit, et al., 2004) populations primarily to assess ethnic differences in ADH and ALDH polymorphisms. However, despite recent findings showing that ADH polymorphisms (ADH1B*3) may be linked to

decreased risk for alcoholism in African Americans (Ehlers, Carr, Betancourt, & Montane-Jaime, 2003) studies have not directly compared African American and Caucasian response to alcohol. Assessing response to alcohol both retrospectively and in an alcohol-challenge paradigm may provide a clearer picture of the validity of this endophenotype for African Americans. Additionally, African Americans have also been found to have lower levels of other common risk factors for drinking, such as disinhibited personality characteristics and positive alcohol expectancies (McCarthy et al., 2001) and these risk factors have been found to influence response to alcohol (Erblich & Earlywine, 2003; Brunelle, et al., 2004; Schuckit, 1998). Incorporating alcohol response into a model with other common risk factors may provide a more complete picture of the alcohol risk process for African Americans.

Other Common Risk Factors

Disinhibited Personality Traits

The personality domain of impulsivity/disinhibition has been found to be the personality construct with the strongest and most consistent association with alcohol-related and antisocial behaviors (Sher & Trull, 1994). A number of different conceptual models of impulsivity/disinhibition have been proposed, based in structural models of personality (e.g., Eysenck, 1990; Goldberg, 1990; Tellegen, 1988; Zuckerman, Kuhlman, Joireman, Teta, & Kraft, 1993), neurobiological or neuropsychological models (Cloninger, 1987; Gray, 1987), empirically based integrative models (Barrett, 1993), and behavioral performance (Reynolds, Ortengren, Richards, & de Wit, 2006). The proposed project will examine a multi-factor model of impulsivity (Whiteside & Lynam, 2001)

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based on the Five Factor model of personality (Costa & McCrae, 1990).

Studies have tested how individual differences in disinhibition relate to alcohol response. High heart rate in response to alcohol on the ascending limb of the blood alcohol curve has been associated with higher levels of self-reported reward sensitivity (Brunelle et al., 2004) and increased intoxicated behavioral undercontrol (Assaad, Pihl, Seguin, Nagin, Vitaro, & Tremblay, 2006). Similarly, Erblich and Earleywine (2003) found that disinhibition was related to increased subjective stimulation from alcohol and that the association between disinhibition and self-reported alcohol use was strengthened for individuals who reported increased stimulation on the ascending limb.

Recently, Lynam and colleagues (Whiteside & Lynam 2001; Lynam & Miller, 2004) have advanced a model disaggregating impulsive/disinhibited personality traits within the framework of the Five Factor model. Whiteside and Lynam (2001) identified five factors: Sensation Seeking, (lack of) Perseverance, (lack of) Premeditation, Negative Urgency, and Positive Urgency. Each of these five factors is associated with different types and aspects of externalizing behavior (Fischer, Smith, & Anderson, 2003; Lynam & Miller, 2004; Miller, Flory, Lynam, & Leukfeld, 2003; Smith, Fischer, Cyders, Annus, Spillane, & McCarthy, 2007; Whiteside, Lynam, Miller, & Reynolds, 2005). Prior studies on racial differences have focused on the broad construction of disinhibition (McCarthy et al., 2001), however focusing on more specific facets of disinhibition may reveal potentially important racial differences that could explain differences in alcohol use behavior and alcohol-related problems for African Americans and Caucasians.

Alcohol Expectancies

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Considerable research has focused on outcome expectancies of alcohol consumption (see Goldman, Darkes, & Del Boca, 1999; Jones, Corbin, & Fromme, 2001 for reviews). Alcohol expectancies are broadly defined as positive and reinforcing or negative and punishing and range over numerous content domains (sexual, social, physical, cognitive impairment, aggression, etc.). Expectancies have been found to be strong predictors of drinking (Goldman, 1994), to predict drinking prospectively, and to change based on drinking experience (Aas, Klepp, Laberg, & Edvard, 1995; Sher, Wood, Wood, & Raskin, 1996; Smith, Goldman, Greenbaum, & Christiansen, 1995). Expectancies have also been shown to develop prior to experience with drinking (Miller, Smith, & Goldman, 1990; Dunn & Goldman, 2000). Family history of alcoholism has been found to relate to differences in alcohol expectancies (Brown, Craemer, & Stetson, 1987; Sher, 1991), suggesting that environmental, physiological, and genetic factors may influence the formation of expectancies.

Relatively few studies have tested the role of alcohol expectancies in the alcoholism risk process in African Americans. Studies have found that African Americans report lower mean levels of both global positive expectancies about alcohol (McCarthy et al., 2001) and specific subscales: physical/social pleasure, social assertiveness, and tension-reduction (Reese & Friend, 1994). However the results are inconsistent about differential roles expectancies may play in drinking behavior for these two groups. Reese and Friend (1994) found that alcohol expectancies are more predictive of Caucasian drinking behavior whereas McCarthy and colleagues (2001) did not find differences in the association between alcohol expectancies and drinking behavior when

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comparing Caucasians and African Americans.

Social/Contextual Factors

Socioeconomic Status

Socioeconomic status has been found to be associated with many medical conditions, including psychiatric disorders (Adler & Snibbe, 2003). Referred to as the SES-health gradient, findings have consistently shown that having higher SES reduces risk for illness. It has been proposed that SES interacts with genes (G X E) in the prediction of health problems, such that among individuals with high SES the development of illness is more strongly related to genetic factors but in individuals with lower SES genetics plays less of a role (Johnson, 2007).

A recent review (Galea, Nandi, & Vlahov, 2004) of epidemiological studies that assessed social factors, including socioeconomic status, in the prediction of substance use concluded that the association between SES and substance use remains unclear. Galea and colleagues (2004) noted a limitation of most studies, which was difficulty disentangling race from SES. Further complicating the picture are findings indicating that SES may function differently across racial groups (Jones-Webb, Hsiao, & Hannan, 1995). Jones-Webb and colleagues (1995) found that lower SES African American males experienced more alcohol-related problems compared to their Caucasian counterparts. However, at higher SES, Caucasian males experienced more alcohol-related problems than African Americans. These results highlight the importance of exploring the SES-alcohol use association within a framework that considers other alcoholism risk factors (e.g., alcohol expectancies).

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Religiosity

Religiosity has been examined as a protective factor for psychiatric disorders in general, including alcohol use disorders. Michalak, Trocki, and Bond (2007) using the National Alcohol Survey data from the year 2000 examined several demographic variables, including ethnicity and religion. They found that individuals who identify as more religious and hold to their religion's views on alcohol had higher rates of abstention and reported less heavy drinking. African Americans, compared to Hispanics and Caucasians, were the most religious, most adherent to religious beliefs, and had the highest rates of abstention. Additionally, the authors found differences among religious denominations and drinking. For example, almost 45% of Catholic drinkers were classified as heavy drinkers compared to about 13% of Jewish drinkers. Data from the Monitoring the Future project (Wallace, Brown, Bachman, & Laveist, 2003) found that lower levels of alcohol involvement seen in African American youth could be partially explained by increased religiosity among African Americans compared with Caucasians. However, despite lower mean levels of religiosity, being religious was more strongly related to abstention for Caucasians. Contrary to the findings with youth, Darrow, Russell, Cooper, Mudar, and Frone (1992) found that church attendance is a protective factor for African American women but was not related to drinking for Caucasian women. These inconsistent findings may be due to differences in the assessment of religiousness.

Peer Drinking

Peer influences are commonly considered one of the most important determinants

of adolescent and young adult substance use. Peer alcohol use has been consistently found to predict alcohol involvement (Curran, Stice, & Chassin 1997; D'Amico & McCarthy, 2006; Farrell & Danish, 1993; Pedersen & Skondral, 1998; Schulenberg et al., 1999; Wills & Cleary, 1999). Peer attitudes toward substance use are also highly predictive of use of alcohol and other drugs (Hawkins, Catalano, & Miller, 1992; Zhang, Welte, & Wieczorek, 1997).

In a sample of women, Herd (1997) found mean level racial differences in normative beliefs about drinking, with African Americans reporting less social acceptance about alcohol use compared to Caucasians. Different aspects of peer influence have also been found to relate to alcohol use for these two racial groups. Perceived peer use is associated with drinking for African Americans whereas peer approval is related to drinking for Caucasian youth (Vega, Zimmerman, Warheit, Apospori, & Gil, 1993). These findings indicate that peer drinking may be differentially related to drinking behavior for African Americans and Caucasians.

Familial Modeling

Family alcohol use can increase risk for problem drinking through both biological and environmental mechanisms. The environmental component of family modeling is important, as children begin learning vicariously about the effects of alcohol from their family before actually initiating drinking behavior. For example, Brown, Tate, Vik, Haas, and Aarons (1999) found that increased exposure to heavy alcohol use in the family was associated with the increased development of positive alcohol expectancies, which in turn predicted drinking behavior. Given different drinking behaviors between African

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Americans and Caucasians (Breslau et al., 2006), it is likely that African American youth experience different modeling of alcohol use than Caucasian youth. This modeling may also have different effects on alcohol consumption for these groups.

Gender X Race

It is well established that males consume more alcohol, have higher rates of alcohol use disorders and more alcohol-related consequences than females, and this is consistent across racial groups (Dawson, 1996). However, epidemiological studies also highlight the possibility of an interaction between race and gender, where the difference in drinking behavior is larger between African American and Caucasian women than men. For example, 6.2% of Caucasian men and 4.5% of African American men reported heavy drinking behavior in the last year compared to 5.2% of Caucasian women and 1.9% of African American women (National Health Interview Survey, 2005). A similar pattern is seen for abstention rates.

Studies on response to alcohol have focused largely on men, but studies conducted assessing response to alcohol in women have produced similar results (e.g., Schuckit et al., 2004; Schuckit, Smith, Kalmijn, Tsuang, Hesselbrock, & Bucholz, 2000). One important future direction for this area of research is to examine response to alcohol separately by gender and race. Schuckit and colleagues (2004) found no overall gender differences in alcohol response, but found that Latina women had a more intense reaction to alcohol than other ethnic/gender groups (i.e., Latina men, Caucasian women, Caucasian men). A similar process may occur in African Americans, such that African American women are more sensitive to the sedating effects of alcohol than Caucasian

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women. Cultural factors may also restrict alcohol consumption more for African American women than men; that is, it may be especially non-normative for African American women to consume large amounts of alcohol.

Integrating Common and Social/Contextual Differences

The overarching goal of the current study was to expand Smith and colleagues (Smith et al., 2006) conceptual model by incorporating response to alcohol as a common risk factor for alcoholism and applying this cross-cultural model to the alcoholism risk process of African Americans and Caucasians. To examine this model, the current project had four primary aims.

Aim 1: Test response to alcohol, disinhibited personality characteristics, and alcohol expectancies as common risk factors, with similar within-group associations with drinking behavior for Caucasians and African Americans.

These common risk factors were hypothesized to have similar prediction of within-group differences in drinking behavior and negative consequences from alcohol for African Americans and Caucasians. Mean differences in these factors were thought to partially explain between group differences in alcohol-related behavior. For example, I hypothesized that African Americans would have lower mean levels of disinhibited personality characteristics compared to Caucasians but that these personality traits would be related to drinking behavior for both racial groups.

Since alcohol response in African Americans has not been directly compared to Caucasians, an important aspect of this first aim was to examine potential racial differences in alcohol response. I hypothesized that compared to Caucasians, African

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Americans would respond to alcohol in a pattern consistent with their reduced drinking behavior: they would experience decreased stimulation and increased sedation from alcohol.

Aim 2: Test both mean level differences in social/contextual factors (socioeconomic status (SES), religiosity, family modeling, and perceived peer drinking) and differences in the association with these risk factors and drinking behavior across racial groups.

Several aspects of the social environment relevant to alcohol use have been shown to differ between African Americans and Caucasians (e.g., SES, religiosity) and there is some evidence that these contextual factors may differentially relate to drinking behavior for these two groups. For example, African Americans have been found to be more religious as a whole (Michalak et al., 2007), but identifying as religious was more strongly associated with decreased drinking for Caucasians (Wallace et al., 2003). In the current study I hypothesized that while African Americans would have higher mean levels of religiosity compared to Caucasians, religiosity would have a stronger association with drinking behavior for Caucasians. This highlights the potential for contextual factors to have both mean level differences and differential association with drinking behavior.

Aim 3: Integrate common and social/contextual factors in the prediction of racial differences in drinking behavior.

While response to alcohol and disinhibition are strong predictors of drinking behavior, environmental factors may constrain or facilitate this association. One aspect of the hypothesized model was to test social/contextual factors as moderators of the

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association of response to alcohol and disinhibition with drinking behavior. For example, increased sensitivity to the stimulating effects of alcohol may not increase risk for alcohol consumption for members of peer groups with norms against drinking. These social/contextual factors may be particularly likely to constrain the influence of risk factors for African Americans.

Aim 4: Examine alcohol expectancies as a mediator for both common and contextual factors.

Another aspect of the hypothesized model was to test alcohol expectancies as proximal risk factors that mediate the association between both common and social/contextual factors and drinking behavior (Goldman et al., 1999). McCarthy and colleagues (2001) found that alcohol expectancies mediated the relation between disinhibition and drinking similarly for African Americans and Caucasians. Within this model it was hypothesized that the lower levels of positive expectancies often found in African Americans are due to differences in other risk factors (e.g., lower levels of disinhibition) that influence the development of these beliefs. These expectancy differences, in turn, will lead to observed differences in drinking behavior across groups.

METHOD

Participants

Participants were 178 young adults. The sample was 47% male and had a mean age of 21.78 (*SD* = 1.16, range 21-26). One hundred and two participants (57%) were of African American descent, while seventy-six participants (43%) were of European American descent. To meet inclusion criteria for the study, those who identified as Hispanic or mixed race were required to have at least one biological parent who was African American or European American. The majority of the sample (76%) had some college education and 15% reported being college graduates. Participants were required to be between the ages of 21 and 26 years old and to be current drinkers. Additionally, participants were excluded if they were currently an abstaining alcoholic, had significant medical or psychiatric illness (e.g., psychotic disorders, past head injury with loss of consciousness > 5 minutes) or were currently taking medication for which use of alcohol is contraindicated.

Procedures

Participants were recruited from the University of Missouri, the city of Columbia, Missouri and the surrounding area (Boone County, Missouri). Fliers were placed at various locations at the University of Missouri-Columbia and at local businesses. Potential participants receive a basic phone screening to determine eligibility.

Participants who met eligibility criteria were scheduled for an interview. On the scheduled interview day, participants were provided with an informed consent form. It

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took an average of 1½ hours to complete the interview and study questionnaires. Interviews, study tasks, and questionnaire completion were conducted in a private office. African American participants received $40 for their participation in the interview. Caucasians completed an abbreviated assessment battery and received $10 for their participation in the interview.

Participants that were eligible for the alcohol challenge were then scheduled for a second appointment. Appointments were scheduled within 1 week of the initial interview. Participants were given an information packet prior to their laboratory appointment. They were instructed to refrain from alcohol for 24 hours before the session and to refrain from other drug use for 48 hours. They were also instructed to fast for 8 hours prior to their session (starting at midnight the evening prior).

The alcohol challenge session took approximately five hours. Participants arrived at the laboratory at 8:00 a.m. A questionnaire was administered to verify compliance with pre-session instructions. A breath alcohol analysis was used to verify abstinence from alcohol. Females were given a urine pregnancy test and excluded from the study if they tested positive. During the baseline period a low-fat breakfast was provided.

Between 8:30 and 9:00 a.m., baseline measures were taken. At 9:00 a.m., participants received an alcoholic beverage (vodka and tonic). Participants received a dose of alcohol equivalent to 0.72g/kg alcohol for males, 0.65g/kg alcohol for females, designed to reach a peak blood alcohol concentration of approximately 0.075 to 0.080 mg% (Sher & Walitzer, 1986). Dosing was based on weight and gender (range of total vodka consumed = 70mls – 248 mls). The alcohol drinks were made using 50% alcohol

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(vodka), in 20% solution with non-caffeinated soda (tonic). Beverages were consumed over a 15 minute period.

Participants were assessed prior to beverage consumption, in 15 minute intervals for the first hour following consumption, and 30 minute intervals thereafter. The alcohol administration and assessment were conducted in a private office, with a semi-recumbent chair, separate from that used for interviews. This office was equipped with a vital signs monitor and computer. At approximately 12:00 noon, each participant was provided lunch. To minimize risk, the following procedures outlined in the NIAAA Recommended Council Guidelines on Ethyl Alcohol Administration in Human Experimentation were used (NIAAA, 2005). Participants were not allowed to leave the laboratory until their observable behavior had returned to normal and until their BAC fell below 0.02%. Each participant was also required to travel home by taxi (provided by the study), or with a friend. Participants were required to state in writing that he or she would not drive a car or operate other machinery for three hours after leaving the laboratory. African Americans were reimbursed $100. Caucasians completed a reduced assessment battery and were therefore reimbursed $50 for participation in the session.

Measures (Interview Session)

The Semi-Structured Assessment for the Genetics of Alcoholism (SSAGA-II). This clinical interview was used to collect demographic information, medical history, substance abuse and dependence symptoms, and alcohol and substance use behaviors (Bucholz, Cadoret, Cloninger, Dinwiddle, Hesselbrock, & Numberger, 1994; Hesselbrock, Easton, Bucholz, Schuckit, & Hesselbrock, 1999). The Family History

Assessment Module assessed first degree family history of alcohol dependence (Rice et al., 1995).

Alcohol Use Behavior. The Drinking Styles Questionnaire (DSQ: Smith, McCarthy, & Goldman, 1995) was used to provide additional information on alcohol involvement. The DSQ was used to collect information about drinker/nondrinker status, age of first drink, past month and typical frequency and quantity of drinking, frequency of drunkenness, problems perceived to be caused by one's drinking, and typical drinking contexts. The DSQ has demonstrated good reliability and validity in adolescent samples (Smith et al., 1995) and has been used in Caucasian and African American college samples (McCarthy et al., 2001).

Two separate dependent variables were created using items from the DSQ. A factor score was created using past month quantity, frequency, number of times having 5 or more drinks, and largest number of drinks consumed on one occasion. A second dependent variable, alcohol-related problems, was created by summing 8 true/false items that assessed negative consequences of alcohol use (e.g., experienced legal problems, blackouts). The DSQ contains 10 true/false items that are designed to look at alcohol-related problems (e.g., blackout, trouble with friends, legal problems). We excluded 2 items (felt nauseous, had a hangover) due to lack of variability in our sample (> 80% of participants endorsed these items).

Personality Characteristics. Personality characteristics were assessed using the UPPS impulsive behavior scale. The UPPS is a 59-item questionnaire measure designed to distinguish between distinct personality traits that might lead to impulsive behavior

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(Whiteside & Lynam, 2001). The UPPS has been found to have five factors, (lack of) Premeditation, Sensation Seeking, (negative) Urgency, (positive) Urgency, and (lack of) Perseverance. UPPS scales have been found to differentially relate to different types of impulsive behaviors (Whiteside et al., 2005; Cyders, Smith, Spillane, Fischer, Annus, &, Peterson, 2007) including substance use (Miller et al., 2003).

Alcohol Expectancies. The Alcohol Expectancies Questionnaire (AEQ: Brown, Goldman, Inn, & Anderson, 1980; Goldman, Greenbaum, & Darkes, 1997) was used to assess explicit expectancies concerning the positive effects of alcohol consumption. Participants were asked to respond to questions using a five-point Likert scale (1-strongly disagree; 5- strongly agree), with higher scores representing greater agreement with positive alcohol expectancies. Subscales of the AEQ have been found to have moderate to good internal consistency reliability (coefficient alphas ranging from .67 to .87), and validity evidence indicates that the AEQ is related to various aspects of drinking behavior (Goldman, Brown, Christiansen, & Smith, 1991).

The Comprehensive Effects of Alcohol questionnaire (CEOA: Fromme, Stroot, & Kaplan, 1993) was used to measure negative alcohol expectancies. The CEOA is a well-established measure of alcohol outcome expectancies. The CEOA has 38 expectancy items, which have been found to load on two higher order factors of positive and negative expectancies (Fromme et al., 1993). The CEOA has demonstrated good test-retest and internal consistency reliability, and has been found to be associated with alcohol use in adolescent and college student populations (Fromme & D'Amico, 2000; Fromme et al., 1993). Seven factors have been found for the CEOA measure, four classified as positive

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expectancies and three as negative expectancies. The negative expectancy scales were used in the current study.

Socioeconomic Status. A composite variable representing socioeconomic status was computed using Hollingshead's (1975) four factor index of social status. This information was assessed as part of the self-report demographics questionnaire which included questions about the occupation for the mother and father, educational attainment of both parents and the participants, and annual household income.

Religiosity. Several questions from the Semi-Structured Assessment for the Genetics of Alcoholism (Bucholz et al., 1994; Hesselbrock, et al., 1999) were used to assess religiosity. These questions assessed religious preference, whether or not the participant's religion has rules forbidding the use of alcohol, and attendance of religious services. I focused on attendance of religious services in the past 12 months as the primary measure of religiosity in this study.

Perceived Peer Drinking. Two questions were used to assess perceived peer alcohol use. These open-ended questions were "What percentage of people your age do you think drank alcohol in the last month?" and "When people your age drink alcohol, on average, how many drinks do you think they have?" An overall perceived peer drinking variable was created by standardizing these two questions and taking the sum.

Family Modeling. Two items (1 Never Uses – 5 Uses Daily) were used to assess father alcohol use: "How often does your father drink beer or wine?" and "How often does your father drink liquor?" Parallel questions were asked for the participant's mother and siblings. The average of these 6 items was used as a composite measurement of

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overall family modeling/alcohol use.

Measures (Alcohol Challenge Session)

 Breath Alcohol Concentration. Breath alcohol readings were taken using a breathalyzer device (Intoximeters, inc.) at baseline, and all measurement points (i.e., 15, 30, 45, 60, 90, 120, and 150 minutes) after consumption of the beverage.

 Subjective Feelings of Intoxication. Subjective feelings of intoxication were evaluated at baseline, and at all measurement points following beverage consumption, using the Biphasic Alcohol Effects Scale (BAES: Martin, Earleywine, Musty, Perrine, & Swift, 1993). This measure assesses separate sedating and stimulating effects of alcohol on both the ascending and descending limb of the blood alcohol curve. This measure has been used to discriminate sedating and stimulating effects of alcohol in alcohol challenge studies and to have a four factor structure (type of response x blood alcohol curve limb: Earleywine & Erblich, 1996).

 Additionally, the Self-Rating Effects of Alcohol form (SRE: Schuckit, Smith, & Tipp, 1997) was used to assess retrospective sensitivity to alcohol. The SRE asks participants to report the number of drinks required to feel the effects of alcohol (i.e., feel different, feel a bit dizzy or slur speech, begin stumbling, pass out) the first 5 times the participant drank, after 3 months drinking once a month, and during period of heaviest drinking. Schuckit and colleagues (1997) found the SRE to be a valid indicator of subjective response to alcohol in adults.

 Cardiovascular Measurements. Blood pressure (diastolic and systolic) was obtained using an automatic blood pressure measurement system at baseline and all

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measurement points after beverage consumption. Conrod and colleagues (2001) conducted a series of studies demonstrating that heart rate response can be a reliable and valid way to assess individual differences in physiological response to alcohol.

Data Analytic Plan

Aim 1: Test the association of response to alcohol, disinhibition, and alcohol expectancies with drinking behavior and negative consequences from alcohol. I hypothesized that while these risk factors may have different mean levels between groups (African Americans lower, Caucasians higher), they would function similarly in the prediction of alcohol use behaviors within each racial group. For retrospective sensitivity to alcohol, disinhibition, and alcohol expectancies, hierarchical regression analyses were conducted following recommendations outlined by Aiken and West (1991). All predictor variables were centered before analyses were conducted. Two-way interaction terms were then created between race (African Americans = 0, Caucasians = 1) and each predictor. Separate regression analyses were run with the predictor, race, and sex on Step 1 and the interaction term between the predictor and race on Step 2.

Subjective and physiological response to alcohol during the alcohol challenge is a repeated measurement, assessed at 8 different time points. To account for the nesting of multiple assessments within person, a latent variable growth model using Mplus 5.2 (Muthén & Muthén, 2009) was used to examine this risk factor. Models were estimated using a maximum likelihood estimator, and model fit was assessed using the Chi-square goodness of fit test, comparative fit index (CFI), Tucker-Lewis index (TLI), and root-mean-square error of approximation (RMSEA). For the CFI and TLI, I used the

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conventional cutoff values .90 or greater for acceptable fit, and .95 or greater for good fit. RMSEA values between .05 and .08 represent acceptable fit, while values less than .05 indicate a good fit (McDonald & Ho, 2002). Models were fit separately for the ascending and descending limbs of the BAC curve. The slope or change parameter was used to predict past month drinking behavior and alcohol-related problems. A multi-group model was run for each alcohol response measure to examine if changes in alcohol response following alcohol consumption were similar for both racial groups. A chi-square difference test was used to test the effect of such constraints on model fit. A significant change in chi-square when constraints are imposed indicates a loss of model fit, suggesting that equality constraints are not justified, and prediction differs across groups. This approach allowed for the evaluation of multiple equality constraints simultaneously as well as focused tests of the equality of specific predictive relations across groups. Chi-square difference tests were also used to test the appropriateness of constraining the path from alcohol response growth (slope parameter) to drinking behavior to equal across African Americans and Caucasians.

Aim 2: Test social/contextual factors that may differ in both mean level and association with alcohol for African Americans and Caucasians. I hypothesized that African Americans and Caucasians would differ in mean levels of social/contextual factors (religiosity, SES, family modeling, perceived peer drinking) and that these factors would be differentially related to drinking for these two racial groups. For example, African Americans on average have lower SES than Caucasians, but SES may not be as strongly related to alcoholism risk for African Americans. To test this aim, a series of

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hierarchical regression analyses were conducted with religiosity, SES, family modeling, and perceived peer drinking as predictors of alcohol use and alcohol-related problems. As in aim 1, Step 1 included sex, race, and the predictor and Step 2 included the interaction term between the predictor and race.

Aim 3: Integrate common and social/contextual factors in the prediction of racial differences in drinking behavior. Test social/contextual factors as moderators of disinhibition and alcohol response. I hypothesized that social/contextual factors would interact with acute response to alcohol, retrospective sensitivity to alcohol, and disinhibition to predict past month drinking and alcohol-related problems. For example, increased stimulation from alcohol may only be related to drinking behavior for individuals who have a peer group that drinks alcohol regularly. Response to alcohol would not be related to drinking behavior for individuals embedded in a peer group with infrequent alcohol use or abstention. To test this hypothesis, three-way interactions were tested in a hierarchical regression framework for disinhibition and retrospective sensitivity to alcohol. Path analyses were used to test the interaction of acute response to alcohol with social/contextual factors (e.g., religiosity, SES) separately for African Americans and Caucasians. As in aim 1, acute response to alcohol was modeled in Mplus 5.2 to take advantage of repeated measurements. Models were run separately for each alcohol response measure and for each social/contextual factor. Mplus 5.2 allows for testing interaction effects between latent (i.e., slope) and measured variables.

Aim 4: Test alcohol expectancies as mediators of both social/contextual and common factors.

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Aim 4 tested alcohol expectancies as a mediator of other risk factors associations with drinking behavior. Mediation hypotheses were tested using path analyses. Mplus provides statistical significance tests of indirect (mediated) effects. Bootstrapping was used to estimate the standard errors. A significant indirect effect when only the predictor and mediator are included in the model indicates at least partial mediation.

RESULTS

Descriptive Information

Tables 1 and 2 present correlations between study variables for African

Americans and Caucasians, respectively. Table 3 presents means and standard deviations

for study variables separately by race.

T-tests were used to test mean level racial differences in drinking behavior,

alcohol-related problems and common (e.g., alcohol expectancies) and context specific

factors (e.g., SES). Caucasians reported higher past month drinking levels (e.g., drinking

alcohol more frequently, consuming a higher quantity of drinks) and endorsed

experiencing a higher number of alcohol-related problems. Caucasians were also found to

have significantly higher levels of positive and negative alcohol expectancies,

disinhibition, increased family alcohol use, and SES. Additionally, they reported reduced

sensitivity to alcohol (higher tolerance) and perceived their same age peers to be

consuming more alcohol. African Americans reported attending more religious services

in the past 12 months.

Table 1

Correlations among study variables for African Americans

	1.	2.	3.	4.	5.	6.	7.	8.	9.	10.	11.	12.	13.	14.	15.	16.
1. Drinking																
2. Alc. Problems	.37**															
3. Initial LS	.43**	.15														
4. 3 months LS	.55**	**.25***	.78**													
5. Heaviest LS	.67**	**.33**	.65**	.87**												
6. Neg. Urgency	.11	.45**	.07	.13	.12											
7. Sens. Seek	**.43**	**.21***	.31**	.35**	.38**	.03										
8. Pos. Urgency	**.42**	.34**	.17	.26*	.26*	.55**	.23*									
9. Perseverance	.09	**.25***	.20*	.14	.20	.30**	.08	.34**								
10. Premeditation	.22*	.16	.18	.11	.17	.29**	.21*	.15	.55**							
11. Pos. Expect.	**.27**	.30**	.08	.13	.12	.34**	.32**	.39**	-.03	-.07						
12. Neg. Expect.	.17	**.44**	.14	.17	.11	.46**	.29**	.34**	.14	.07	.54**					
13. SES	.12	.12	.10	.12	.18	.04	.20	.11	-.16	.02	.17	.11				
14. Religiosity	**-.25**	-.15	-.12	-.18	-.24*	.00	-.02	-.06	.01	.09	-.02	-.04	.08			
15. Peer Drinking	**.18**	.13	.07	.10	.21*	.19	.17	.01	-.03	.17	.04	-.10	.12	.02		
16. Family Model	.18	.04	.05	.06	-.01	.17	.01	.19	.00	.17	.15	.09	-.12	.08	.08	

Notes: * *p* < .05; ** *p* < .01. Bolded correlation coefficients are those that differ in their association with past month drinking ('Drinking') and alcohol-related problems ('Alc. Problems') across races. Low retrospective sensitivity to alcohol is abbreviated 'LS.'

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Table 2

Correlations among study variables for Caucasians

	1.	2.	3.	4.	5.	6.	7.	8.	9.	10.	11.	12.	13.	14.	15.	16.
1. Drinking																
2. Alc. Problems	.54**															
3. Initial LS	.49**	.18														
4. 3 months LS	.51**	.16	.74**													
5. Heaviest LS	.48**	.19	.54**	.77**												
6. Neg. Urgency	.08	.30**	-.02	-.12	.01											
7. Sens. Seek	.08	.02	.24*	.20	.14	-.04										
8. Pos. Urgency	.13	.25*	-.04	-.10	-.08	.46**	.15									
9. Perseverance	-.02	-.04	-.04	-.03	-.02	.47**	-.12	.19								
10. Premeditation	.09	.08	.04	.04	.01	.49**	.17	.32**	.38**							
11. Pos. Expect.	.17	.33**	-.01	-.14	-.07	.52**	.14	.33**	.17	.18						
12. Neg. Expect.	-.20	.06	-.06	-.04	-.17	.24*	.01	.07	.04	.14	.33**					
13. SES	.03	.09	.05	-.17	-.11	.19	-.02	.13	-.09	.18	.10	-.01				
14. Religiosity	-.17	.01	.09	-.04	-.04	.00	.15	-.08	-.19	-.16	-.10	.00	.07			
15. Peer Drinking	.35**	-.02	.25*	.23*	.28*	-.03	-.12	.00	-.03	.03	.05	-.18	.08	-.21		
16. Family Model	-.03	.00	-.12	-.15	-.08	.15	-.26*	.06	.12	.08	-.03	-.01	.16	-.08	-.01	

Notes: * $p < .05$; ** $p < .01$.

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Table 3

Means (standard deviations) for study variables across African Americans and Caucasians

1.	2.	3.	4.	5.	6.	7.	8.	9.	10.	11.	12.	13.	14.	15.	16.
African Americans															
-.37	1.36	3.87	4.84	6.10	2.10	2.63	1.46	1.84	1.78	3.00	2.18	46.56	15.77**	-.26	2.32
(.79)	(1.43)	(1.56)	(2.10)	(2.84)	(.61)	(.60)	(.50)	(.49)	(.44)	(.66)	(.54)	(11.50)	(21.87)	(1.41)	(.83)
Caucasians															
.49**	2.88**	4.24	5.94**	8.02**	2.27*	3.08**	1.66*	1.89	1.98**	3.29**	2.47**	50.89**	8.04	.34**	2.58*
(1.04)	(1.74)	(1.75)	(2.45)	(3.02)	(.55)	(.45)	(.56)	(.55)	(.46)	(.53)	(.45)	(9.12)	(11.89)	(1.43)	(.78)

Notes: * mean differences across race ($p < .05$). ** mean difference across race ($p < .01$). Racial group with higher mean is denoted with asterisk. 1=*Drinking*; 2=*Alc. Problems*; 3=*Initial LS*; 4=*3 months LS*; 5=*Heaviest LS*; 6=*Neg. Urgency*; 7=*Sens. Seek*; 8=*Pos. Urgency*; 9=*Perseverance*; 10=*Premeditation*; 11=*Pos. Expect*.; 12=*Neg. Expect*.; 13=*SES*; 14=*Religiosity*; 15=*Peer Drinking*; 16=*Family Model*.

Acute Alcohol Response Growth Models

Separate base models were run on the full sample, where race was specified as a grouping variable, for stimulation, sedation, and blood pressure on the ascending and descending limbs of the blood alcohol curve. Time was coded so that the intercept reflected the model implied baseline assessment (pre-drinking assessment for ascending limb, 60-minutes post drinking for descending limb), and was used to control for baseline differences in blood pressure and subjective measurements. These models controlled for sex, BrAC (45 minutes post-drinking for ascending limb analyses, 60 minutes post-drinking for descending limb analyses), and weight. Sex and weight were used as control variables for both the intercept and slope parameters while BrAC and the model implied intercept were used as control variables for the slope parameter.

The fit indices for these base models indicated at least adequate fit for all unconstrained models (see Table 4). The Chi-squares for stimulation on the ascending limb and all descending limb models were not significant, indicating the models fit the data well, and the CFI and TLI indices also indicated good fit. The RMSEA for these models indicated acceptable to good fit. While sedation and blood pressure on the ascending limb had significant Chi-squares, indicating a lack of model fit, the CFI and TLI indicated these models fit the data at an acceptable level (> .90). RMSEA values indicated acceptable fit for blood pressure but not sedation on the ascending limb.

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Table 4

Model fit indices for unconstrained base models

	Chi-Square	CFI	TLI	RMSEA
Ascending Limb				
Stimulation	28.23, *ns*	0.99	0.98	0.06
Sedation	47.31, *p* < .001	0.96	0.93	0.12
Blood Pressure	40.62, *p* < .05	0.98	0.97	0.09
Descending Limb				
Stimulation	24.28, *ns*	0.99	0.99	0.04
Sedation	32.62, *ns*	0.98	0.97	0.08
Blood Pressure	22.47, *ns*	0.99	0.99	0.02

To test for racial differences in the intercept and slope parameters, I examined a series of nested models. As shown in Tables 5 and 6, each of these nested models constrained a single parameter (i.e., intercept mean, slope mean, intercept variance, slope variance, intercept predicting slope) to be equal for African Americans and Caucasians. I used a Chi-square difference test to compare each nested model with the base model (i.e., the model that freely estimated the parameter for each race). If the constraint did not result in a significantly worse model fit over the base model, the parameter was assumed to be equal for both races. After each parameter of interest had been tested individually, all constraints that did not result in significantly worse model fit were tested simultaneously against the base model (i.e., omnibus tests). The omnibus test was then used as the new baseline model to increase the stringency of the test of model fit. This sequence was repeated first examining differences in intercept and slope means, intercept and slope variances, and then intercept predicting slope. These analyses largely supported subjective and physiological response to alcohol as an invariant construct across racial groups, with relatively few parameters differing between groups.

Table 5

Multi-group chi-square difference test across racial groups for sedation on the ascending limb

Type of Model	Model fit X^2	df	Model Comparison	X^2 Difference Test ΔX^2	Δdf	*p*-value
Ho: Base Model (no constraints across race)	47.68	22				
Equivalence of factor means when covariates are constant at 0						
1. Constrain intercept mean	47.69	23	1 vs. Ho	0.01	1	*ns*
2. Constrain slope mean	48.57	23	2 vs. Ho	0.89	1	*ns*
3. Omnibus test of all (i.e., Models 1 and 2) nonsignificant constraints	48.94	24	3 vs. Ho	1.26	2	*ns*
Equivalence of growth factor variances						
4. Model 3 + constrain intercept variance	49.38	25	4 vs. 3	0.44	1	*ns*
5. Model 3 + constrain slope variance	57.9	25	5 vs. 3	8.96	1	**< .01**
Equivalence of intercept predicting slope						
6. Model 4 + constrain intercept predicting slope	52.43	26	6 vs. 4	3.05	1	*ns*
Equivalence of relation between predictors and linear growth model parameters						
7. Model 6 + constrain sex predicting slope	57.84	27	7 vs. 6	5.41	1	**< .05**

Table 6

Multi-group chi-square difference test across racial groups for stimulation on the descending limb

	Model fit		Model Comparison	X^2 Difference Test		
Type of Model	X^2	*df*		ΔX^2	Δdf	*p*-value
Ho: Base Model (no constraints across race)	29.34	22				
Equivalence of factor means when covariates are constant at 0						
1. Constrain intercept mean	40.59	23	1 vs. Ho	11.25	1	**< .001**
2. Constrain slope mean	37.45	23	2 vs. Ho	8.11	1	**< .01**
Equivalence of growth factor variances						
4. Constrain intercept variance	32.10	23	4 vs. Ho	2.76	1	*ns*
5. Constrain slope variance	29.62	23	5 vs. Ho	0.28	1	*ns*
6. Omnibus test of Models 4 and 5	32.26	24	6 vs. 1	2.92	2	*ns*
Equivalence of intercept predicting slope						
7. Model 6 + constrain intercept predicting slope	34.48	25	7 vs. 6	2.22	1	*ns*
Equivalence of relation between predictors and linear growth model parameters						
8. Model 7 + constrain sex predicting intercept	34.54	26	8 vs. 7	0.06	1	*ns*
9. Model 8 + constrain sex predicting slope	40.08	27	9 vs. 8	5.54	1	**< .05**

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Both stimulation and blood pressure on the ascending limb did not differ across groups (i.e., intercept and slope means and variances and the association between intercept and slope were equivalent). For sedation on the ascending limb, constraining the slope variance to be equivalent across groups resulted in a decrement in model fit (see Table 5). Follow-up analyses indicated that African Americans had significant intra-group variability around the slope parameter, while Caucasians did not. For stimulation on the descending limb, the intercept and slope means were not equivalent for African Americans and Caucasians (see Table 6). These results indicated that Caucasians had a significantly higher intercept mean for stimulation on the descending limb compared to African Americans and that Caucasians had significant increases in stimulation on the descending limb while African Americans did not have a significant rate of change. Blood pressure on the descending limb also significantly decreased for Caucasians but not African Americans. Additionally, the intercept significantly predicted the slope in this model for Caucasians but not African Americans.

Potential sex differences in acute response to alcohol were then examined across racial group. Stimulation and sedation differed across race and sex. African American males experienced a sharper increase in stimulation on the ascending limb compared to African American females. Additionally, African American females experienced sharper increases in sedation on the ascending limb compared to African American males (see Table 5). Sex differences were not found for Caucasians in either of these models. On the descending limb, Caucasian males experienced a sharper increase (shallower decrease) in stimulation compared to Caucasian females while sex did not predict changes in stimulation on the descending limb for African Americans (see Table 6). The association

between sex and rate of change in sedation on the descending limb and blood pressure on either the ascending or descending limb of the BAC curve did not differ by race.

Acute Alcohol Response Predicting Drinking Behavior

To examine the association between alcohol response and drinking behavior, I first ran models using the slope parameter to predict past month drinking behavior and alcohol-related problems within each racial group. For African Americans, experiencing a sharper increase in stimulation on the ascending limb was related to higher levels of past month drinking ($\beta = .32$, $p < .01$) and experiencing more alcohol-related problems ($\beta = .23$, $p < .05$). Additionally, experiencing a sharper increase in sedation on the ascending limb was associated with lower levels of past month drinking ($\beta = -.33$, $p < .001$; see Figure 1) and experiencing fewer alcohol-related problems ($\beta = -.24$, $p < .05$). Experiencing a sharper increase (shallower decrease) in blood pressure on the ascending limb was associated with decreased past month drinking ($\beta = -.50$, $p < .0001$) and alcohol-related problems ($\beta = -.22$, $p < .05$). Experiencing a sharper increase (shallower decrease) in blood pressure on the descending limb was also related to decreased past month drinking ($\beta = -.24$, $p < .05$).

For Caucasians, experiencing a sharper increase (shallower decrease) in blood pressure on the ascending limb was associated with decreased past month drinking ($\beta = -.39$, $p < .0001$). Additionally, experiencing a sharper increase in sedation on the ascending limb was marginally related to lower levels of past month drinking ($\beta = -.26$, $p = .06$, see Figure 1) but was not related to alcohol-related problems. Stimulation on the ascending and descending limb as well as sedation on the descending limb were also not related to either outcome variable for Caucasians.

Figure 1

Sedation on the ascending limb predicting past month drinking behavior
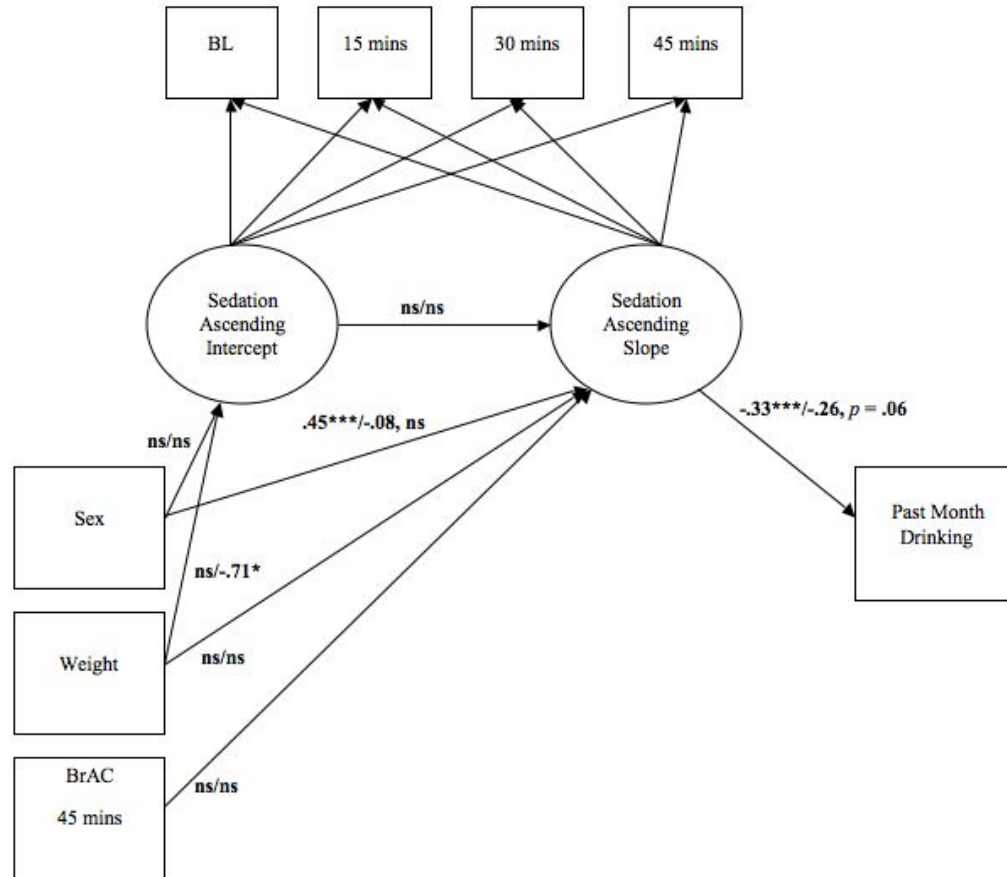
Note: Values = African Americans/Caucasians; *** *p* < .001; ** *p* <.01; * *p* < .05

To test racial differences in the pathway from alcohol response slope to past month drinking behavior and alcohol-related problems, separate unconstrained models were re-run for each of these outcome variables for alcohol response growth parameters on the ascending and descending limbs. I then constrained the association between the slope parameters and the outcome variables to be the same across groups. Despite differences in significance level and beta weights for some of these pathways (see above) constraining these paths to be equivalent across racial groups did not result in a significant decrement in model fit.

Other Common Risk Factors, Race, and Drinking Behavior

Retrospective Sensitivity to Alcohol

In addition to examining acute response to alcohol as a potential common risk factor, aim 1 was also designed to test retrospective sensitivity to alcohol, alcohol expectancies, and disinhibition as common risk factors for both racial groups. I first examined if the association between retrospective alcohol sensitivity (initial, after 3 months of drinking regularly, and during the period of heaviest drinking) and past month drinking and alcohol-related problems differed by race. The interaction between race and the three retrospective sensitivity to alcohol variables in the prediction of either past month alcohol use or alcohol-related problems was not significant indicating that low sensitivity to alcohol (high tolerance) as measured by retrospective self-report functions similarly as a risk factor for both African Americans and Caucasians. For the full sample, initial low sensitivity, low sensitivity after drinking for 3 months, and during the period of heaviest drinking were significant predictors of past month drinking (β's ranged from .33 to .46, p's < .0001) and low sensitivity after drinking for 3 months and during the

period of heaviest drinking were related to alcohol-related problems (β = .17, p < .05; β = .23, p < .01, respectively). Initial low sensitivity was marginally related to alcohol-related problems (β = .13, p = .09).

Alcohol Expectancies

Next, I examined if the association between alcohol expectancies (positive and negative) and drinking behavior differed by race. Separate regressions were run for positive and negative expectancies. The interaction between race and positive expectancies was not significant for either past month drinking behavior or alcohol-related problems. Main effects of positive expectancies were found for past month drinking behavior (β = .17, p < .01) and alcohol-related problems (β = .22, p < .01).

Negative expectancies interacted with race to predict past month drinking (β = -.16, p < .05) but not alcohol-related problems. Partial correlations controlling for sex were run within each racial group to probe the interaction between race and negative alcohol expectancies. For Caucasians, there was a trend for higher levels of negative alcohol expectancies to be related to *decreased* past month drinking (r = -.17, *ns*). However, for African Americans, there was a trend for higher levels of negative expectancies to be related to *increased* past month drinking (r = .12, *ns*). While race did not interact with negative expectancies to predict alcohol-related problems, a main effect of negative expectancies was found (β = .34, p < .001), with higher levels of negative expectancies being associated with increased alcohol-related problems.

Disinhibited Personality Characteristics

As the final part of aim 1, I examined several different aspects of disinhibition (i.e., sensation seeking, lack of perseverance, lack of premeditation, negative urgency,

positive urgency) in the prediction of alcohol use and related problems. Disinhibited personality characteristics largely functioned as a common risk factor across races with non-significant interactions being found between disinhibition and race in the prediction of past month drinking. For the full sample, main effects of positive urgency ($\beta = .22$, $p <$ .001), lack of premeditation ($\beta = .14$, $p < .05$), negative urgency ($\beta = .13$, $p < .05$), and sensation seeking ($\beta = .14$, $p < .05$) were found in the prediction of past month drinking behavior. Lack of perseverance was not related to past month drinking. When examining disinhibition and alcohol-related problems a similar pattern of results was found. Positive ($\beta = .27$, $p < .0001$) and negative ($\beta = .36$, $p < .0001$) urgency were positively associated with alcohol-related problems. Lack of premeditation, sensation seeking, and lack of perseverance were not associated with alcohol-related problems.

Aim 2: Contextual Factors, Race, and Drinking

Aim 2 was designed to examine risk/protective factors that were hypothesized to not only differ in mean level across race but also in association with drinking behavior. To test this aim hierarchical regression analyses were run examining main effects and interactions with race for socioeconomic status (SES), number of times an individual attended religious services in the past 12 months (religiosity), family alcohol use, and perceived peer use. All analyses controlled for sex. Perceived peer use was associated with past month drinking behavior ($\beta = .24$, $p < .0001$). Socioeconomic status ($\beta = .12$, $p = .08$) and religiosity ($\beta = -.12$, $p = .06$) were marginally related to past month drinking behavior. Family modeling was not related to past month drinking behavior and none of the contextual risk/protective factors were associated with alcohol-related problems. While mean differences in contextual factors were found, contrary to hypothesis, all

interactions between race and contextual variables were not significant. Indicating that while contextual factors differ in mean level across racial groups, the association with drinking behavior was similar for African Americans and Caucasians.

Aim 3: Common Risk Factors Interacting with Contextual Factors

Aim 3 was designed to test whether contextual factors facilitated or constrained the association between common risk factors (e.g., sensation seeking) and drinking behavior. For acute alcohol response, interactions were run in Mplus. Two-way interactions between acute response to alcohol and contextual factors were run within racial group, as this type of analysis does not allow for a direct multi-group comparison. Interactions involving retrospective sensitivity to alcohol (SRE) and disinhibition were examined within a hierarchical regression framework. I first tested all possible 2-way interactions between retrospective sensitivity to alcohol/disinhibition and contextual factors. I then examined 3-way interactions entered on Step 3 (e.g., race X sensation seeking X peer use) with all possible 2-way interactions (e.g., race X sensation seeking, race X peer use, sensation seeking X peer use) entered on Step 2. As previous analyses, in aims 1 and 2, already examined differential prediction for common and contextual factors by race (e.g., race X sensation seeking, race X peer use) I will only be presenting results from the 2-way interaction between common and contextual factors (e.g., sensation seeking X peer use) and the 3-way interaction incorporating race. All significant 2-way interactions were probed at 1 standard deviation above and below the mean for the contextual variable (Aiken & West, 1991). Significant 3-way interactions were probed within each racial group using the same recommended method by Aiken and West (1991).

See table 7 for a summary of these interactions presented separately by race.

Acute Alcohol Response X Contextual Factors

I used the slope parameter from the base models (stimulation, sedation, and blood pressure) and created an interaction term between this parameter and the contextual variable for all possible combinations (e.g., slope stimulation ascending limb X family modeling, slope blood pressure descending limb X SES). The interaction term was then used to predict past month drinking behavior and alcohol-related problems. The slope parameter and the contextual variable were also used as control variables in the prediction of drinking behaviors.

Stimulation on the ascending limb interacted with SES to predict past month drinking behavior for African Americans ($b = .12$, $p < .05$). Probing this interaction revealed stimulation on the ascending limb was related to past month drinking at high SES levels ($b = 3.43$, $p < .001$) but not low SES levels ($b = -.10$, *ns*). Stimulation on the ascending limb did not interact with any other contextual variable to predict either outcome variable for African Americans. All interactions for stimulation on the ascending limb were not significant for Caucasians. Only one significant interaction was found for sedation on the ascending limb for either racial group: in the African American sample sedation on the ascending limb interacted with number of religious events attended to predict alcohol-related problems ($b = .04$, $p < .05$). Follow-up analyses showed that experiencing increased growth in sedation on the ascending limb was related to fewer alcohol-related problems at low levels of religious event attendance ($b = -2.70$, $p < .01$). Sedation on the ascending limb was not related to alcohol-related problems at high levels of religious event attendance ($b = -.51$, *ns*).

Table 7

Interactions between common and contextual factors presented separately by race

African American Drinking	Caucasian Drinking	African American Alcohol Problems	Caucasian Alcohol Problems
Stim. Ascend. X SES[a]	ns	ns	ns
ns	ns	Sed. Ascend. X Relig. [a]	ns
BP Ascend. X SES[a]	ns	ns	BP Ascend. X SES[a]
BP Ascend. X Relig. [a]	BP Ascend. X Relig. [a]	BP Ascend. X Relig. [a]	ns
ns	ns	ns	BP Ascend. X Family Modeling[a]
Sed. Descend. X SES[a]	ns	ns	ns
ns	ns	Sed. Descend X Family Modeling[a]	ns
ns	ns	ns	BP Descend. X Peer Use[a]
Low Sens. (3 months) X SES[b]	Low Sens. (3 months) X SES[b]		
Low Sens. (heaviest) X SES[b]	Low Sens. (heaviest) X SES[b]		
Low Sens. (3 months) X Peer[b]	Low Sens. (3 months) X Peer[b]		
Low Sens. (heaviest) X Relig. [b]	Low Sens. (heaviest) X Relig. [b]		
Pos.Urgency X Family Modeling[b]	Pos. Urgency X Family Modeling[b]		
Perseverance X Peer Use[b]	Perseverance X Peer Use[b]		
ns	ns	ns	Neg. Urgency X Family Modeling[c]
ns	ns	ns	Pos. Urgency X Family Modeling[c]
ns	Sensation Seeking X Peer[c]	ns	ns
ns	ns	ns	Premeditation X Relig. [c]
ns	Premeditation X SES[c]	ns	Premeditation X SES[c]

Note. [a] Two-way interactions run within racial group for acute response to alcohol. [b] Two-way interactions between common risk factors and contextual factors that did not differ by race. [c] Three-way interactions probed separately by racial group.

Blood pressure on the ascending limb interacted with SES to predict past month drinking for African Americans (b = -1.88, p < .05) and alcohol-related problems for Caucasians (b = -8.11, p < .001). For African Americans, ascending limb blood pressure was related to past month drinking at high SES levels (b = -77.18, p < .001) but not low SES levels (b = -9.52, ns). Follow-up analyses with Caucasians, indicated that blood pressure on the ascending limb was associated with alcohol-related problems at both high (b = -272.72, p < .001) and low SES levels (b = -79.33, p < .001). Blood pressure on the ascending limb also interacted with number of religious events attended in predicting past month drinking (b = 1.40, p < .001) and alcohol-related problems for African Americans (b = 1.51, p < .001). Follow-up analyses again showed that blood pressure on the ascending limb was related to past month drinking and alcohol-related problems for both high (b = -28.82, p < .001; b = -116.46, p < .001, respectively) and low religious event attendance (b = -88.53, p < .001; b = -173.11, p < .001, respectively). This model of blood pressure and religious events would not converge for Caucasians. The interaction between blood pressure on the ascending limb and family modeling was also significant for alcohol-related problems in the Caucasian sample (b = -110.29, p < .001). Blood pressure on the ascending limb was associated with alcohol-related problems at high levels of family alcohol use (b = -266.77, p < .001) but was not associated with alcohol-related problems at low levels of family alcohol use (b = -1.71, ns).

On the descending limb, sedation interacted with SES to predict past month drinking (b = .32, p < .05) and family modeling to predict alcohol-related problems (b = -2.79, p < .01) for African Americans. Sedation was related to past month drinking at high levels of SES (b = 8.59, p < .001) but not at low levels of SES (b = -.49, ns). Follow-up

analyses for the interaction between sedation and family modeling revealed the opposite pattern. Sedation on the descending limb was associated with alcohol-related problems at low levels of family alcohol use ($b = 3.74$, $p < .05$) but not at high levels of family alcohol use ($b = -1.90$, ns). For Caucasians, blood pressure on the descending limb interacted with perceived peer use in predicting alcohol-related problems ($b = -.79$, $p < .05$). Experiencing a sharper increase (shallower decrease) in blood pressure on the descending limb was related to fewer alcohol-related problems at high levels of perceived peer drinking ($b = -1.07$, $p < .05$) but was related to more alcohol-related problems at low levels of perceived peer drinking ($b = 2.48$, $p < .001$).[1]

*Retrospective Sensitivity to Alcohol X Contextual Factors*

I examined whether the association between retrospective sensitivity to alcohol and drinking behavior was moderated by SES, family modeling, perceived peer use, and religiosity and if this moderation differed for African Americans and Caucasians. Low sensitivity to alcohol after 3 months and during the period of heaviest use interacted with SES in predicting past month drinking behavior ($\beta = .13$, $p < .05$, see Figure 2a; $\beta = .17$, $p < .01$, see Figure 2b; respectively).

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[1]Unstandardized coefficients are presented for ease of interpretation. Several follow-up analyses conducted to probe interactions with acute response produced standardized Beta coefficients greater than 1 or less than -1. These analyses were primarily when examining blood pressure. While uncommon, Beta coefficients greater than 1/-1 can occur when covariates are included in analyses. Analyses using median splits instead of 1 standard deviation above and below the mean as well as analyses without any covariates in the model support the pattern of results presented above.
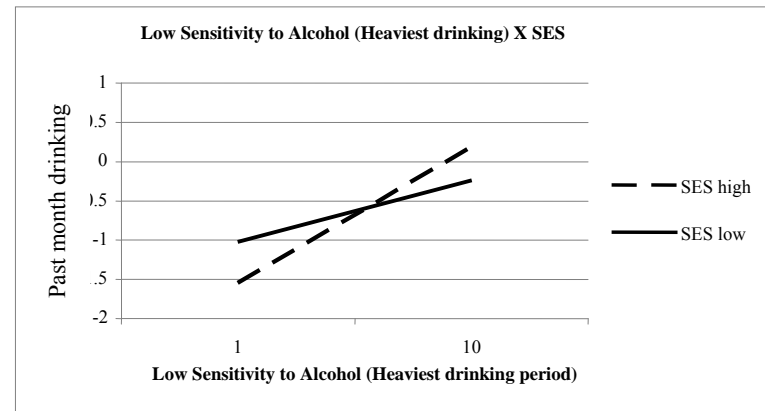
Figure 2

Low sensitivity to alcohol interacting with SES in the prediction of past month drinking behavior
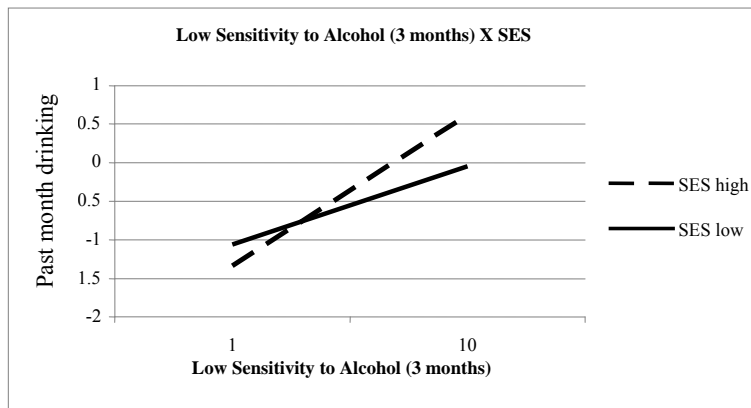
2a.   2b.

Probing these interactions revealed that low sensitivity to alcohol (after 3 months, during the period of heaviest drinking) was more strongly related to past month drinking at high SES levels ($\beta = .50$, $p < .001$, $\beta = .59$, $p < .001$, respectively) compared to low SES levels ($\beta = .26$, $p < .01$, $\beta = .26$, $p < .01$, respectively). Additionally, the interaction between perceived peer use and low sensitivity after 3 months ($\beta = .11$, $p < .05$) predicted past month drinking. Follow-up analyses showed that low sensitivity to alcohol was more strongly related to past month drinking at high levels of perceived peer alcohol use ($\beta = .46$, $p < .001$) compared to low peer use ($\beta = .26$, $p < .01$). Finally, the interaction between number of religious events attended and low sensitivity to alcohol during the period of heaviest drinking was significantly related to past month drinking ($\beta = -.19$, $p < .01$). Probing this interaction revealed that low sensitivity to alcohol was related to *increased* drinking at high levels of religious event attendance ($\beta = .26$, $p < .01$) but was related to *decreased* drinking at low levels of religious event attendance ($\beta = -.27$, $p < .01$).

Initial low sensitivity to alcohol did not interact with any of the contextual factors to predict past month alcohol use. All 2 way interactions between retrospective sensitivity to alcohol (i.e., initial, 3 months, period of heaviest drinking) and contextual factors were not significant for alcohol-related problems. Three-way interactions between race, retrospective sensitivity to alcohol, and contextual factors were also not significant.

Disinhibited Personality Characteristics X Contextual Factors

Next, I examined if disinhibition (i.e., lack of perseverance, negative urgency, lack of premeditation, sensation seeking, positive urgency) was moderated by contextual factors in the prediction of drinking behavior and alcohol-related problems and if this

interaction differed by race. Urgency and sensation seeking did not significantly interact with any of the four contextual factors to predict drinking behavior or alcohol-related problems. However, several significant 3-way interactions were found and all other facets of disinhibition interacted with at least one contextual factor to predict either past month drinking behavior or alcohol-related problems.

The interaction between positive urgency and family modeling was significantly associated with past month drinking ($\beta = .14$, $p < .05$). Probing this interaction indicated that positive urgency was related to drinking behavior at high levels of family alcohol use ($\beta = .32$, $p < .001$) but not low levels of family alcohol use ($\beta = .07$, *ns*). Additionally, a 3-way interaction between negative urgency, family modeling and race ($\beta = .20$, $p < .05$) and a 3-way interaction between positive urgency, family modeling, and race ($\beta = .36$, $p < .0001$) were significantly associated with alcohol-related problems. Two-way interactions, run within racial group, showed that family modeling interacted with negative and positive urgency for Caucasians but not African Americans. For Caucasians, negative and positive urgency was strongly related to increased alcohol-related problems at high levels of family alcohol use (negative urgency: $\beta = .57$, $p < .001$; positive urgency: $\beta = .68$, $p < .001$) and was not related to alcohol-related problems at low levels of family alcohol use (negative urgency: $\beta = -.24$, *ns*; positive urgency: $\beta = -.34$, *ns*; see Figures 3a, 3b).

Lack of perseverance was moderated by perceived peer use in the prediction of past month alcohol use ($\beta = -.16$, $p < .01$).

Figure 3

Urgency interacting with family modeling in the prediction of alcohol-related problems for Caucasians

3a.

3b.
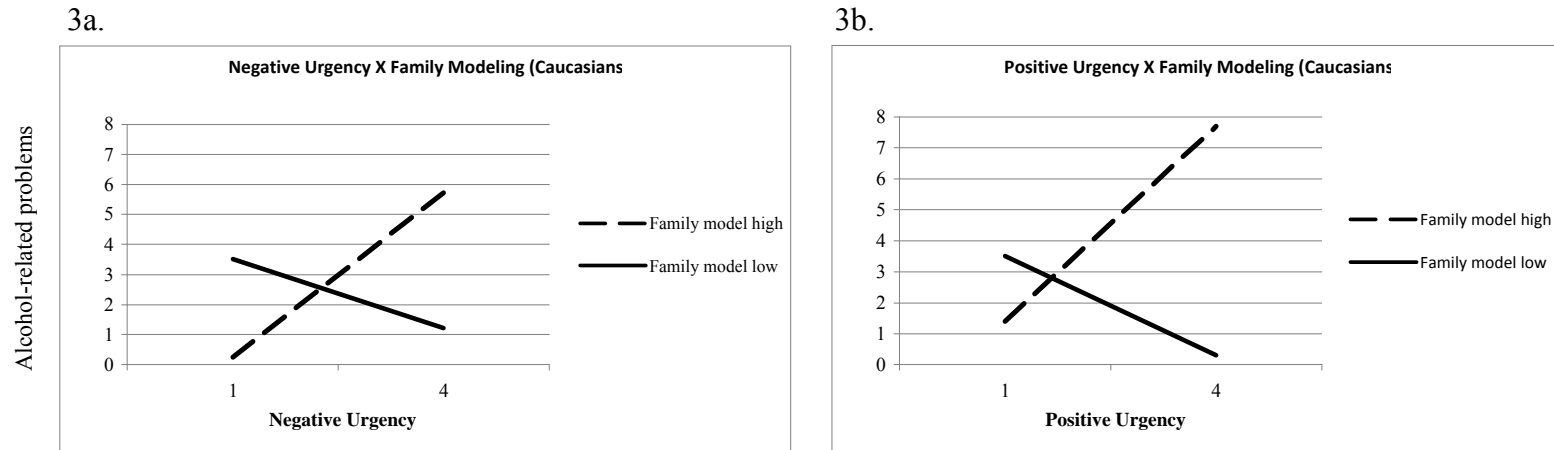
Probing this interaction showed that lack of perseverance was related to past month drinking behavior at low levels of perceived peer use (β = .17, p < .05), but was not related to drinking at high levels of perceived peer use (β = -.19, ns). Additionally, a 3-way interaction between sensation seeking, perceived peer use, and race was found to be associated with past month alcohol use (β = .19, p < .05). Probing this interaction showed that for Caucasians sensation seeking was marginally related to *heavier* past month drinking at high levels of perceived peer alcohol use (β = .23, p = .11). However, sensation seeking was marginally related to significantly *less* drinking at low levels of peer use (β = -.28, p = .10).

Lack of premeditation interacted with number of religious events attended in predicting alcohol-related problems (β = -.15, p < .05). However, the interaction between lack of premeditation and religion was qualified by a significant 3-way interaction with race (β = -.20, p < .05). Follow-up analyses showed that lack of premeditation significantly interacted with religious event attendance for Caucasians but not African Americans. However, simple slopes for 1 standard deviation above and below the mean were not significant.

Two-way interactions between disinhibited personality characteristics and SES were not significant, however, several 3-way interactions were found between facets of disinhibition and SES. Lack of premeditation, SES, and race interacted to predict past month drinking (β = .21, p < .05) and alcohol-related problems (β = .24, p < .05). For Caucasians, lack of premeditation was marginally related to *increased* past month drinking (β = .25, p = .09) and alcohol-related problems (β = .25, p = .11) at high SES levels and was marginally related to *decreased* drinking and alcohol problems (β = -.40, p

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$= .09; \beta = -.38, p = .13$) at low SES levels.

Aim 4: Alcohol expectancies as mediators of both social/contextual and common factors.

Aim 4 tested alcohol expectancies as a mediator of other risk factors (common and context specific) and drinking behavior. To test mediation in the present study, Mackinnon's recommended guidelines for testing mediation were used (e.g., Mackinnon, Lockwood, Hoffman, West, & Sheets, 2002).

As presented in aim 1, positive and negative expectancies were related to past month drinking and alcohol related problems so could be considered potential mediators. However, the recommendation that the predictor be related to the mediator eliminated numerous common and context specific factors from further analysis. None of the contextual factors were related to positive or negative alcohol expectancies, but several common risk factors were used to further test the possibility of mediation. Positive urgency, negative urgency, and sensation seeking were related to both positive and negative alcohol expectancies and were used in further mediation analyses of past month drinking/alcohol-related problems. Blood pressure on the ascending limb was the only measure of acute alcohol response that was related to alcohol expectancies (positive and negative). However, blood pressure was only related to alcohol expectancies for African Americans. Follow-up mediation analyses were run within African Americans for this risk factor.

Once variables were selected I bootstrapped the standard errors to examine the significance of the indirect effect of disinhibition and alcohol response through alcohol expectancies on drinking behavior/alcohol-related problems. Several significant indirect

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effects were found for African Americans in the prediction of alcohol-related problems. Negative expectancies mediated the association between several common risk factors and alcohol-related problems: positive urgency (β = .12, p < .01; 95% CI: 0.045 – 0.197), negative urgency (β = .12, p < .01; 95% CI: 0.030 – 0.217), sensation seeking (β = .12, p < .01; 95% CI: 0.038 – 0.197), and blood pressure on the ascending limb (β = -.12, p < .05; 95% CI: -0.215 – -0.017). Positive alcohol expectancies also significantly mediated the association between sensation seeking and alcohol-related problems (β = .08, p < .05; 95% CI: 0.010 – 0.152). Models examining mediation for blood pressure and past month drinking would not converge. Results did not support disinhibition being mediated by positive or negative expectancies for Caucasians for past month drinking behavior or alcohol-related problems.

Follow-up analyses were conducted for each significant indirect effect that was found to examine which specific mediation paths differed by race. Analyses were run within each racial group examining the path from the predictor to the mediator (path a) and the path from the mediator to the outcome (path b). Results showed that all possible disinhibited personality characteristics (predictor variables: i.e., negative urgency, positive urgency, sensation seeking) were strongly related to both positive and negative expectancies (mediator) for African Americans (path a: β's = .26 - .47, p's < .01). However, for Caucasians, only negative urgency (β = .22, p < .05) was related to negative expectancies. For the path b (mediator to outcome), positive expectancies were related to alcohol-related problems for both racial groups (Caucasians: β = .33, p < .001; African Americans: β = .28, p < .001) but negative expectancies were only associated with alcohol-related problems for African Americans (β = .43, p < .001). These results

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provide further indication that negative expectancies may function as a mediator for African Americans but not Caucasians.

DISCUSSION

The current study was designed to take an initial step towards developing an integrative model of alcohol use and alcohol-related problems in African Americans. Both common, risk/protective factors that were thought to function similarly across racial groups (e.g., disinhibition), and context specific factors, risk/protective factors that were thought to have differential associations with drinking across racial groups (e.g., religiosity) were examined. Prior studies have found that African Americans drink less alcohol (Wallace et al., 2003; Bachman et al., 1991), have lower mean levels of common risk factors (e.g., positive alcohol expectancies and disinhibition; McCarthy et al., 2001) and may have higher levels of protective context specific factors (e.g., religiosity, low family alcohol use) compared to Caucasians. However, relatively few studies have attempted to incorporate multiple risk and protective factors into a model of alcohol use for African Americans. Smith and colleagues (2006) outlined a broad framework to facilitate the integration of common and context specific factors for the study of psychological disorders across different cultures. This framework was used as a foundation to develop the primary aims of this study. The current study extended Smith and colleague's proposed model to African Americans and tested alcohol response as a common risk factor that functions similarly for Caucasians and African Americans.

Racial Differences in Acute Alcohol Response

I examined potential racial differences in blood pressure, stimulation, and sedation across the ascending and descending limbs of the blood alcohol curve. While relatively few significant differences were found, the differences that were significant

may indicate that Caucasians have a higher risk profile for acute response (increased stimulation, decreased sedation) compared to African Americans. Specifically, Caucasians had higher levels of stimulation 60 minutes post-drinking and continued to increase in stimulation on the descending limb while African Americans did not. Additionally, sex differences emerged that were consistent with males having a higher risk profile compared to females. African American males experienced a sharper increase in stimulation and decreased growth in sedation on the ascending limb compared to females. For Caucasians, males experienced a sharper increase in stimulation on the descending limb compared to females.

Acute response to alcohol was largely supported as a common risk factor that functioned similarly across groups in the prediction of past month drinking behavior and alcohol-related problems. Model comparisons across African Americans and Caucasians showed that the association between growth in acute response (i.e., stimulation, sedation, and blood pressure) and drinking behavior was similar across groups. However, when models were run within each racial group the size of the effects as well as the pattern of significance was different for African Americans and Caucasians. Experiencing a sharper increase in stimulation was related to increased past month drinking and alcohol-related problems for African Americans. Also, experiencing a sharper increase in blood pressure on the descending limb was related to decreased past month drinking for African Americans. For both racial groups, experiencing a sharper increase in sedation and blood pressure on the ascending limb was related to decreased drinking behavior.

Racial Differences in other Common Risk Factors

Previous research has found that African Americans have lower mean levels of

disinhibition and positive alcohol expectancies compared to Caucasians and that these risk factors function similarly to predict drinking behavior within each racial group (McCarthy et al., 2001). I extended these findings by examining specific facets of disinhibition, both positive and negative alcohol expectancies, and retrospective report of sensitivity to alcohol. Results from the current study largely parallel these previous findings with African Americans having lower mean levels of risk factors, such as disinhibited personality characteristics, positive alcohol expectancies, and increased sensitivity to alcohol (lower tolerance). Additionally, within each racial group these risk factors were similarly associated with past month drinking and alcohol-related problems. These results support the hypothesis that mean differences in these risk factors may at least partially explain the mean level differences in drinking behavior that has been found across these two racial groups.

Interestingly, African Americans also had lower mean levels of negative alcohol expectancies compared to Caucasians. Previous research on negative expectancies has found that having higher levels of negative expectancies is related to decreased alcohol use in Caucasian samples but that this association is not as strong as positive expectancies with alcohol use (Jones et al., 2001). In the current study, consistent with previous research, higher negative expectancies were related to decreased drinking for Caucasians. However, for African Americans higher negative expectancies were related to increased alcohol use. Additionally, for the entire sample, higher levels of negative expectancies were related to experiencing more alcohol-related problems.

Learning based models support the idea that individuals act in accordance with their expectations. Based on this model individuals with higher levels of negative

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expectancies would drink less alcohol and individuals with lower levels would drink more. For example, if you expect drinking alcohol will cause you to be sick or become impaired then you would drink less alcohol. This is the opposite pattern of what was found for African Americans. One potential explanation for this finding is that negative expectancies that are related in a positive direction to drinking behavior may be a function of lifetime exposure to alcohol. This may be particularly relevant for African American adolescents and young adults due to the later initiation and reduced drinking behavior seen in this racial group. Additionally, young adults may still be developing these cognitions given their relatively short drinking history compared to mature adults. Future studies including abstainers and light drinkers as well more established (older) drinkers from both racial groups would allow for further examination of this finding.

Racial Differences in Context Specific Risk/Protective Factors

Another focus of the study was to examine several risk (e.g., low SES) and protective (e.g., religiosity) factors that were thought to differ not only in mean level but also in association with drinking behaviors across racial groups. Results from the current study supported a mean difference model. African Americans had lower SES, perceived their peers as lighter drinkers, reported lower family alcohol use, and attended religious events more frequently compared to Caucasians.

The hypothesis that these context specific factors would differ in their association with drinking across racial groups was not supported. Perceived peer drinking, religiosity, and SES were related to past month drinking behavior similarly for both racial groups. The higher mean levels of religiosity and lower levels of perceived peer drinking are congruent with the drinking behavior reported by these two racial groups (African

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Americans consuming less alcohol than Caucasians). Interestingly, higher levels of SES were marginally related to increased past month drinking behavior. This finding may be a result of the current sample being primarily college students, where the range in SES is restricted to include only people who can afford attending college. Additionally, previous studies on SES, race, and substance use have had difficulty disentangling SES from race (Galea et al., 2004). In this study, Caucasians had higher mean levels of SES and alcohol use which may further complicate the interpretation of the interplay between SES, race, and drinking behavior.

Integrating Common and Context Specific Factors

The final aim of the current study was to integrate common and context specific factors to begin the development of a cross-cultural model of African American alcohol use. I proposed that context specific factors would constrain or facilitate the association between common risk factors and drinking behavior and that this may differ by race. Additionally, I examined alcohol expectancies as proximal risk factors that are influenced by more distal factors (e.g., disinhibition) and that this indirect path to drinking behavior is important to examine across race.

Several interesting patterns were found in these analyses. First, for both Caucasians and African Americans high levels of socioeconomic status increased the association with drinking behavior for several common risk factors. Increased response to alcohol was related to drinking behavior at high levels of SES but was not related to drinking behavior at low levels of SES. A similar pattern was found for retrospective report of alcohol sensitivity: low sensitivity to alcohol was more strongly related to past month drinking at high levels of SES compared to low levels of SES. The mechanism

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that explains these findings is unclear in the current study. However, one potential reason for this pattern of results may be that SES influences other factors that are related to alcohol use. For example, having high SES may give you more disposable income which may increase alcohol accessibility or ability to afford alcohol in college/student populations. This in turn, may increase the importance of individual risk factors, like response to alcohol, to determine one's drinking level. For, individuals without the means to purchase alcohol (less disposable income, low SES), response to alcohol may become a less important determinant of drinking behavior. This is consistent with the finding in this sample that high SES is related to increased drinking behavior. Future research that focuses on recruiting participants from a wider range in SES is important to build on these results.

The role of SES and common risk factors largely did not differ across race, with one exception. For Caucasians, one facet of disinhibition, lack of premeditation, was related to increased drinking behavior at high levels of SES and decreased drinking behavior at low levels of SES. While this finding was race specific, high SES facilitating the relation between a common risk factor and drinking behavior is consistent with the larger findings on SES from this study.

Family modeling also facilitated the association between several common risk factors and drinking behavior for both races. For example, one facet of disinhibition, positive urgency, was related to drinking behavior for individuals who reported having heavy drinking family members (i.e., mother, father, sibling) and was not related to drinking behavior for individuals with light drinking family members. This interaction did not differ by race, indicating that being high in positive urgency and perceiving

having heavy drinking family members increases risk for heavy alcohol use for both Caucasians and African Americans.

Important racial differences were also found when incorporating family modeling into a model with common risk factors. Results from this study indicate that family modeling may play a relatively more important role for Caucasians when incorporating this risk factor into a larger model. For Caucasians, both negative and positive urgency were related to increased alcohol-related problems when heavy family alcohol use was reported but not at low levels of family alcohol use. A similar pattern was found for blood pressure on the ascending limb in Caucasians: blood pressure was related to alcohol-related problems at high levels of family alcohol use but not low levels of family use. Interestingly, only one significant race specific interaction with family alcohol use was found for African Americans and it was in the opposite direction than what was found for Caucasians. Sedation on the descending limb was related to alcohol-related problems at low levels of family alcohol use but not high levels of family alcohol use. Future research incorporating diagnostic family alcohol use indicators (e.g., parent alcohol use disorder diagnosis) as well as parent/sibling report is needed to better understand this finding in African Americans. One possibility is that due to the relatively low levels of family alcohol use in African Americans, this risk factor relates differently to other risk factors, such as response to alcohol.

Perceived peer use produced a less consistent pattern of results with both high and low perceived peer use facilitating the association between common risk factors and drinking behavior. For example, lack of perseverance was related to increased past month drinking behavior at low levels of perceived peer use but not high levels of perceived use.

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On the other hand, sensation seeking was related to increased drinking in Caucasians at high levels of perceived peer use but not low levels of perceived use. Peer use is a widely studied risk factor for alcohol use (e.g., Curran et al., 1997) and recent research has shown that one way sensation seeking may increase risk for alcohol and drug use is by increasing association with deviant peers (Yanovitzky, 2005). While findings in the current study were consistent with this for sensation seeking, the opposite was found for another aspect of disinhibition, lack of perseverance. Examining this transaction over time may allow for the integration of these results.

Follow-up analyses with religious event attendance also did not reveal a clear pattern of results. Similar to what was found for perceived peer use, both high and low levels of religious event attendance facilitated the association between common risk factors and drinking behavior. One potential reason for these seemingly inconsistent findings is that the current study focused on religious event attendance. The pattern of results may become clearer if other aspects of religiosity, such as adherence to religious rules and identification with religious group, were also examined.

Finally, alcohol expectancies were examined as proximal risk factors that were influenced by more distal risk factors to predict drinking behavior. Negative expectancies were found to mediate the association between several common risk factors and alcohol-related problems. Importantly, this was only found for African Americans. Previous research on racial differences and alcohol expectancies have focused primarily on positive expectancies. Results from this study indicate that negative expectancies may be particularly important as both direct and indirect risk factors for alcohol use in African Americans. If this finding is replicated, further research is required to examine the

development of these cognitions in this racial group.

Limitations

The current study has several limitations. First is the restriction of the sample to current drinkers (a requirement of this type of alcohol challenge study). Limiting the sample to current drinkers restricted the range of drinking variables and potentially reduced the range of drinking risk factors such as alcohol expectancies and disinhibited personality characteristics. Future studies could examine differences in risk/protective factors between racial groups that include a wider range of drinker categories (e.g., abstainers) to see if results replicate in a more generalizable sample. Of particular importance would be to include abstainers from both racial groups as African Americans have been found to have significantly higher abstention rates compared to Caucasians (Substance Abuse and Mental Health Services Administration, 2003).

Further, because this is a cross-sectional study of current drinkers, there is no empirical basis for the temporal sequencing of study variables. This may be particularly important for acute response to alcohol, as it has been largely unstudied in African Americans. This sample also contained individuals who already reported having a lifetime diagnosis of an alcohol use disorder which limits my ability to determine if response to alcohol is a risk factor for alcoholism or a result. I have interpreted these findings within the theoretical framework that posits level of response to alcohol as a risk factor for alcohol use disorders (Schuckit & Smith, 2001), and this is consistent with the format of the sensitivity to alcohol measure used in the current study, which asks participants to retrospect about their response to alcohol at an earlier period. Longitudinal studies, including individuals who onset to drinking, are required to test whether level of

response predicts later alcohol use and alcohol use disorders in African Americans.

Additionally, the current sample consisted primarily of college students at a predominantly Caucasian university. It is important to examine different age groups (adolescents, mature adults) as well as recruit from a wider range of demographic characteristics (e.g., African Americans in primarily African American communities) to further extend this model. For example, context specific factors could play a more significant role for African Americans embedded in a larger African American community with more conservative cultural norms about alcohol use.

Another limitation is that the alcohol administration consisted of a single dose level of alcohol and did not contain a placebo condition, which prevented testing dose effects on acute alcohol response. Results could differ at lower (e.g., BrAC = .04%) or higher (e.g., BrAC = .10%) doses of alcohol and it is also important for future studies to account for possible placebo effects and how these may differ by race. Examining different beverages (e.g., beer) and contexts (e.g., bar, party) is an important next step in this research.

Finally, while the sample size for this study is large for alcohol administration studies, the sample size may have reduced my power to detect significant effects, particularly for multi-group comparisons where models constrained specific paths to be equivalent across groups. These analyses may be more prone to sample size issues as they are testing differences across groups, opposed to analyses that were run examining a specific risk factor predicting drinking behavior. Estimated power for multi-group comparisons computed using RMSEA (Preacher & Coffman, 2006) further suggest that the current study may have been underpowered for this type of analysis. For example, the

estimated power to detect a significant racial difference ($p < .05$) is only .58 for the multi-group model examining stimulation on the ascending limb predicting alcohol-related problems. For models with past month drinking as the outcome of interest the estimated power is even lower.

Overall Conclusion and *Implications*

The current study was designed to identify and integrate common and context specific factors that could explain the differences in drinking behavior seen in African Americans and Caucasians. This study largely found support for a mean difference model where African Americans were found to have reduced levels of risk factors and higher levels of protective factors. Importantly, this was found in a sample that excluded abstainers and light drinkers, so findings could be more pronounced in a sample with a wider drinking range. However, I largely did not find support for a differential association model, where risk factors would be related to drinking behavior more strongly for one racial group. While some differences were found across race, both hypothesized context specific and common risk/protective factors were similar in their association with alcohol use and alcohol-related problems. One exception to this is the finding that negative expectancies serve a more proximal role for other more distal risk factors (personality characteristics) for African Americans.

This study provides an important first step in the integration of several risk/protective factors in the explanation of alcohol use in African Americans. Findings of this study lend further support for the validity of alcohol response as a risk factor for alcohol use in African Americans by directly comparing it with response to alcohol in Caucasians. Further work is required to examine how other risk factors, such as

disinhibited personality characteristics and alcohol expectancies, relate to response to alcohol. This may be especially important given the mean level differences seen in risk factors like disinhibition and the differences in the role of negative expectancies found across racial groups.

REFERENCES

Aas, H., Klepp, K., Laberg, J. C., & Edvard, L. (1995). Predicting adolescents' intentions to drink alcohol: Outcome expectancies and self-efficacy. *Journal of Studies on Alcohol, 56,* 293-299.

Adler, N. E. & Snibbe, A. C. (2003). The role of psychosocial processes in explaining the gradient between socioeconomic status and health. *Current Directions in Psychological Science, 12,* 119-123.

Aiken, L. S. & West, S. G. (1991). Multiple Regression: Testing and Interpreting Interactions. Thousand Oaks, CA: Sage.

Assaad, J., Pihl, R. O., Seguin, J. R., Nagin, D. S., Vitaro, F., & Tremblay, R. E. (2006). Intoxicated behavioral disinhibition and the heart rate response to alcohol. *Experimental and Clinical Psychopharmacology, 14*, 377-388.

Bachman, J. G., Wallace, J. M., O'Malley, P. M., Johnston, L. D., Kurth, C. L., & Neighbors, H. W. (1991). Racial/ethnic differences in smoking, drinking, and illicit drug use among American high school seniors, 1976-89. *American Journal of Public Health, 81*, 372-377.

Barrett, E. S. (1993). Impulsivity: Integrating cognitive, behavioral, biological and environmental data. In W. McCowan, J. Johnson, & M. Shure (Eds.), The impulsive client: Theory, research, and treatment. Washington, DC: American Psychological Association.

Barnes, G. M. & Welte, J. W. (1986). Adolescent alcohol abuse: Subgroup differences and relationships to other problem behaviors. *Journal of Adolescent Research, 1,*

79-94.

Breslau, J., Aguilar-Gaxiola, S., Kendler, K. S., Su, M., Williams, D., & Kessler, R. C. (2006). Specifying race-ethnic differences in risk for psychiatric disorder in a USA national sample. *Psychological Medicine, 36,* 57-68.

Brown, S. A., Creamer, V. A., & Stetson, B. A. (1987). Adolescent alcohol expectancies in relation to personal and parental drinking patterns. *Journal of Abnormal Psychology, 96,* 117-121.

Brown, S. A., Goldman, M. S., Inn, A., & Anderson, L. R. (1980). Expectations of reinforcement from alcohol: Their domain and relation to drinking patterns. *Journal of Consulting and Clinical Psychology, 43*, 419-426.

Brown, S. A., Tate, S. R., Vik, P. W., Haas, A. L., & Aarons, G. A. (1999). Modeling of alcohol use mediates the effect of family history of alcoholism on adolescent alcohol expectancies. *Experimental Clinical Psychopharmacology, 7,* 20-27.

Brown, S. A., Vik, P. W., & Creamer, V. A. (1989). Characteristics of relapse following adolescent substance abuse treatment. *Addictive Behaviors, 14,* 291-300.

Brunelle, C., Assaad, J., Barrett, S. P., Avila, C., Conrod, P. J., Tremblay, R. E., & Pihl, R. O. (2004). Heightened heart rate response to alcohol intoxication is associated with a reward-seeking personality profile. *Alcoholism: Clinical and Experimental Research, 28*, 394-401.

Bucholz, K. K., Cadoret, R., Cloninger, C. R., Dinwiddle, S. H, Hesselbrock, V. M., & Nurnberger, J. I. (1994). A new semi-structured psychiatric interview for use with genetic linkage studies: A report on the reliability of the SSAGA. *Journal of Studies on Alcohol, 55*, 149-158.

Caetano, R. & Kaskutas, L. (1995). Changes in drinking patterns among Whites, Blacks and Hispanics, 1984-1992. *Journal of Studies on Alcohol, 56,* 558-565.

Cloninger, C. R. (1987). Neurogenetic adaptive mechanisms in alcoholism. *Science, 236,* 410-416.

Conrod, P. J., Peterson, J. B., & Pihl, R. O. (2001). Reliability and validity of alcohol-induced heart rate increase as a measure of sensitivity to the stimulant properties of alcohol. *Psychopharmacology, 157*, 20-30.

Conrod, P. J., Pihl, R. O., & Ditto, B. (1995). Autonomic reactivity and alcohol-induced dampening in men at risk for alcoholism and men at risk for hypertension. *Alcoholism: Clinical and Experimental Research, 19,* 482-489.

Costa, P. T. & McCrae, R. R. (1990). Personality disorders and the five-factor model of personality. *Journal of Personality Disorders, 4,* 362-371.

Curran, P. J., Stice, E., & Chassin, L. (1997). The relation between adolescent alcohol use and peer alcohol use: A longitudinal random coefficients model. *Journal of Consulting and Clinical Psychology, 65*, 130-140.

Cyders, M. A., Smith, G. T., Spillane, N. S., Fischer, S., Annus, A. M., & Peterson, C. (2007). Integration of Impulsivity and Positive Mood to Predict Risky Behavior: Development and Validation of a Measure of Positive Urgency. *Psychological Assessment, 19,* 107-118.

D'Amico, E. J. & McCarthy, D. M. (2006). Escalation and initiation of younger adolescents' substance use: The impact of perceived peer use. *Journal of Adolescent Health, 39*, 481-487.

Darrow, S. L., Russell, M., Cooper, M. L., Mudar, P., & Frone, M. R. (1992).

Sociodemographic correlates of alcohol consumption among African-American and White women. *Women and Health, 18,* 35-51.

Dawson, D. A. (1996). Gender differences in the risk of alcohol dependence: United States, 1992. *Addiction, 91*, 1831-1842.

Dawson, D. A. (1998). Beyond Black, White and Hispanic: Race, ethnic origin and drinking patterns in the United States. *Journal of Substance Abuse, 10,* 321-339.

Dunn, M. E. & Goldman, M. S. (2000). Drinking-related differences in expectancies of children assessed as first associates. *Alcoholism: Clinical & Experimental Research, 24,* 1639-1646.

Earleywine, M. & Erblich, J. (1996). A confirmed factor structure for the biphasic effects of alcohol scale. *Experimental and Clinical Psychopharmacology, 4,* 107-113.

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

Erblich, J. & Earleywine, M. (2003). Behavioral undercontrol and subjective stimulant and sedative effects of alcohol intoxication: Independent predictors of drinking habits? *Alcoholism: Clinical and Experimental Research, 27,* 44-50.

Eysenck, H. J. (1990). Genetic and environmental contributions to individual differences: The three major dimensions of personality. *Journal of Personality, 58,* 245-261.

Farrell, A. D. & Danish, S. J. (1993). Peer drug associations and emotional restraint: Causes or consequences of adolescents' drug use? *Journal of Consulting and Clinical Psychology, 61,* 327-334.

Finn, P. R., Zeitouni, N. C., & Pihl, R. O. (1990). Effects of alcohol on

psychophysiological hyperreactivity to nonaversive aversive stimuli in men at high risk for alcoholism. *Journal of Abnormal Psychology, 99,* 79-85.

Fischer, S., Smith, G. T., & Anderson, K. G. (2003). Clarifying the role of impulsivity in bulimia nervosa. *International Journal of Eating Disorders, 33,* 406-411.

Fromme, K. & D'Amico, E. J. (2000). Measuring adolescent alcohol outcome expectancies. *Psychology of Addictive Behaviors, 14,* 206-212.

Fromme, K., Stroot, E. A., & Kaplan, D. (1993). Comprehensive effects of alcohol: Development and psychometric assessment of a new expectancy questionnaire. *Psychological Assessment, 5*, 19-26.

Galea, S., Nandi, A., & Vlahov, D. (2004). The social epidemiology of substance use. *Epidemiologic Reviews, 26,* 36-52.

Garcia-Andrade, C., Wall, T. L., & Ehlers, C. L. (1997). The firewater myth and response to alcohol in Mission Indians. *American Journal of Psychiatry, 154,* 983-988.

Goldberg, L. R. (1990). An Alternative "Description of Personality": The Big-Five Factor Structure. *Journal of Personality and Social Psychology, 59,* 1216-1229.

Goldman, M. S. (1994). The alcohol expectancy concept: Applications to assessment, prevention, and treatment of alcohol abuse. *Applied & Preventive Psychology, 3,* 131-144.

Goldman, M. S., Brown, S. A., Christiansen, B. A., & Smith, G. T. (1991). Alcoholism and memory: Broadening the scope of alcohol-expectancy research. *Psychological Bulletin, 110,* 137-146.

Goldman, M. S., Darkes, J., & Del Boca, F. K. (1999). Expectancy mediation of biopsychosocial risk for alcohol use and alcoholism. In I. Kirsch (Ed.), How

 Expectancies Shape Experience. American Psychological Association: Washington, DC.

Goldman, M. S., Greenbaum, P. E., & Darkes, J. (1997). A confirmatory test of hierarchical expectancy structure and predictive power: Discriminant validation of the Alcohol Expectancy Questionnaire. *Psychological Assessment, 9,* 145-157.

Gray, J. A. (1987). The psychology of fear and stress. Cambridge, England: Cambridge University Press.

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

Heath, A. C., Madden, P. A. F., Bucholz, K. K., Dinwiddie, S. H., Slutske, W. S., Bierut, L. J., Rohrbaugh, J. W., Statham, D. J., Dunne, M. P., Whitfield, J. B., & Martin, N. G. (1999). Genetic differences in alcohol sensitivity and the inheritance of alcoholism risk. *Psychological Medicine, 29,* 1069-1081.

Heath, A. C. & Martin, N. G. (1991). Intoxication after an acute dose of alcohol: an assessment of its association with alcohol consumption patterns by using twin data. *Alcoholism: Clinical and Experimental Research, 15,* 122-128.

Herd, D. (1997). Racial differences in women's drinking norms and drinking patterns: A national study. *Journal of Substance Abuse, 9,* 137-149.

Herd, D. National Institute on Alcohol Abuse and Alcoholism (1989). The epidemiology of drinking patterns and alcohol-related problems among US Blacks. (DHHS Publication No. ADM 89-1435). Washington, DC: U.S. Government Printing Office.

Hesselbrock, M., Easton, C., Bucholz, K. K., Schuckit, M., & Hesselbrock, V. (1999). A
     validity study of the SSAGA: A comparison with the SCAN. *Addiction, 94,* 1361-
     1370.

Hollingshead, A. B. (Unpublished Working Paper, 1975). Four factor index of social
     status.

Johnson, W. (2007). Genetic and environmental influences on behavior: Capturing all the
     interplay. *Psychological Review, 114,* 423-440.

Jones, B. T., Corbin, W., & Fromme, K. (2001). A review of expectancy theory and
     alcohol consumption. *Addiction, 96,* 57-72.

Jones-Webb, R. J., Hsiao, C., & Hannan, P. (1995). Relationships between
     socioeconomic status and drinking problems among black and white men.
     Alcoholism: Clinical and Experimental Research, 19, 623-627.

Luczak, S. E., Elvine-Kreis, B., Shea, S. H., Carr, L. G., & Wall, T. L. (2002). Genetic
     risk for alcoholism relates to level of response to alcohol in Asian-American men
     and women. *Journal of Studies on Alcohol, 63,* 74-82.

Lynam, D. R. & Miller, J. D. (2004). Personality Pathways to Impulsive Behavior and
     Their Relations to Deviance: Results from Three Samples. *Journal of
     Quantitative Criminology, 20,* 319-341.

MacKinnon, D. P., Lockwood, C. M., Hoffman, J. M., West, S. G., & Sheets, V. (2002).
     A comparison of methods to test the significance of mediation and other
     intervening variable effects. *Psychological Methods, 7,* 83-104.

Martin, C. S., Earleywine, M., Musty, R., Perrine, M., & Swift, R. (1993). Development
     and validation of the biphasic effects of alcohol scale. *Alcoholism: Clinical and*

Experimental Research, 17, 140-146.

McCarthy, D. M., Miller, T. L., Smith, G. T., & Smith, J. A. (2001). Disinhibition and expectancy in risk for alcohol use: comparing black and white college samples. *Journal of Studies on Alcohol, 62,* 313-321.

McDonald, R. P. & Ho, M. H. (2002). Principles and practice in reporting structural equation analyses. *Psychological Methods, 7,* 64-82.

Michalak, L., Trocki, K., & Bond, J. (2007). Religion and alcohol in the U.S. national alcohol survey: How important is religion for abstention and drinking? *Drug and Alcohol Dependence, 87,* 268-280.

Miller, J., Flory, K., Lynam, D., & Leukefeld, C. (2003). A test of the four-factor model of impulsivity-related traits. *Personality and Individual Differences, 34,* 1403-1418.

Miller, P. M., Smith, G. T., & Goldman, M. S. (1990). Emergence of alcohol expectancies in childhood: A possible critical period. *Journal of Studies on Alcohol, 51,* 343-349.

Muthén, L. K. & Muthén, B. O. (2007). Mplus User's Guide. Fifth Edition. Los Angeles, CA: Muthen and Muthen.

National Advisory Council on Alcohol Abuse and Alcoholism. (2005). Recommended council guidelines on ethyl alcohol administration in human experimentation. Maryland: National Institute on Alcohol Abuse and Alcoholism.

National Health Interview Survey (2005). Retrieved July 30, 2007 from http://www.niaaa.nih.gov/Resources/DatabaseResources/QuickFacts/AlcoholCon sumption/dkpat26.htm.

Newlin, D. B. & Thompson, J. B. (1990). Alcohol challenge with sons of alcoholics: A
critical review and analysis. *Psychological Bulletin, 108,* 383-402.

Pedersen, S.L. & McCarthy, D.M. (2009). An examination of subjective response to
alcohol in African Americans. *Journal of Studies on Alcohol and Drugs, 70,*
288-295.

Pedersen, W. & Skrondal, A. (1998). Alcohol consumption debit: Predictors and
consequences. *Journal of Studies on Alcohol, 59,* 32-42.

Preacher, K. J. & Coffman, D. L. (2006, May). Computing power and minimum sample
size for RMSEA [Computer software]. Available from http://www.quantpsy.org/.

Reese, F. L. & Friend, R. (1994). Alcohol expectancies and drinking practices among
Black and White undergraduate males. *Journal of College Student Development,
35,* 319-325.

Reynolds, B., Ortengren, A., Richards, J. B., & de Wit, H. (2006). Dimensions of
impulsive behavior: Personality and behavioral measures. *Personality and
Individual Differences, 40,* 305-315.

Rice, J. P., Reich, T., Bucholz, K. K., Neuman, R. J., Fishman, R., Rochberg, N., et al.
(1995). Comparison of direct interview and family history diagnoses of alcohol
dependence. *Alcohol Clinical Experimental Research, 19,* 1018-1023.

Schuckit, M. A. (1980). Self-rating of alcohol intoxication by young men with and
without family histories of alcoholism. *Journal of Studies on Alcohol, 41,* 242-
249.

Schuckit, M. A. (1984). Subjective responses to alcohol in sons of alcoholics and
controls. *Archives of General Psychiatry, 41,* 879-884.

75

Schuckit, M. A. (1985). Ethanol-induced changes in body sway in men at high

   alcoholism risk. *Archives of General Psychiatry, 42,* 375-379.

Schuckit, M. A. (1998). Biological, psychological and environmental predictors of the

   alcoholism risk: A longitudinal study. *Journal of Studies on Alcohol, 59,* 485-494.

Schuckit, M. A. & Gold, E. O. (1988). A simultaneous evaluation of multiple markers of

   ethanol/placebo challenges in sons of alcoholics and controls. *Archives of General*

   Psychiatry, 45, 211-216.

Schuckit, M. A. & Smith, T. L. (2001). The clinical course of alcohol dependence

   associated with a low level of response to alcohol. *Addiction, 96,* 903-910.

Schuckit, M. A. & Smith, T. L. (2000). The relationships of a family history of alcohol

   dependence, a low level of response to alcohol, and six domains of life

   functioning to the development of alcohol use disorders. *Journal of Studies on*

   Alcohol, 61, 827-835.

Schuckit, M. A., Smith, T. L., Danko, G. P., Pierson, J., Hesselbrock, V., Bucholz, K. K.,

   Kramer, J., et al., (2007). The ability of the Self-Rating of the Effects of Alcohol

   (SRE) scale to predict alcohol-related outcomes five years later. *Journal of*

   Studies on Alcohol and Drugs, 68, 371-378.

Schuckit, M. A., Smith, T. L., & Kalmijn, J. (2004). Findings across subgroups regarding

   level of response to alcohol as a risk factor for alcohol use disorders: A college

   population of women and Latinos. *Alcoholism: Clinical and Experimental*

   Research, 28, 1499-1508.

Schuckit, M. A., Smith, T. L., Kalmijn, J., Tsuang, J., Hesselbrock, V., & Bucholz, K.

   (2000). Response to alcohol in daughters of alcoholics: a pilot study and a

comparison with sons of alcoholics. *Alcohol and Alcoholism, 35,* 242-248.

Schuckit, M. A., Smith, T. L., & Tipp, J. E. (1997). The self-rating of the effects of
alcohol (SRE) form as a retrospective measure of the risk for alcoholism.
Addiction, 92, 979-988.

Schulenberg, J., Maggs, J. L., Dielman, T. E., Leech, S. L., Kloska, D. D., Shope, J. T., &
Laetz, V. B. (1999). On peer influences to get drunk: A panel study of young
adolescents. *Merrill-Palmer Quarterly, 45,* 108-142.

Sher, K. J. (1991). Characteristics of children of alcoholics. In D. Gallant and E. Goltheil
(Eds.), Recent developments in alcoholism, Volume 9: Children of alcoholics.
(pp. 301-326). New York: Plenum.

Sher, K. J. & Trull, T. J. (1994). Personality and disinhibitory psychopathology:
Alcoholism and antisocial personality disorder. *Journal of Abnormal Psychology,
103,* 92-102.

Sher K. J. & Walitzer K. S. (1986). Individual differences in the stress-response-
dampening effect on alcohol: a dose-response study. *Journal of Abnormal
Psychology, 95,* 159-67.

Sher, K. J., Wood, M. D., Wood, P. K., & Raskin, G. (1996). Alcohol outcome
expectancies and alcohol use: A latent variable cross-lagged panel study. *Journal
of Abnormal Psychology, 105,* 561-574.

Smith, G. T., Fischer, S., Cyders, M. A., Annus, A. M., Spillane, N. S., & McCarthy, D.
M. (2007). On the validity and utility of discriminating among impulsivity-like
traits. *Assessment, 14,* 155-170.

Smith, G. T., Goldman, M. S., Greenbaum, P. E., & Christiansen, B. A. (1995).

Expectancy for social facilitation from drinking: The divergent paths of high-expectancy and low-expectancy adolescents. *Journal of Abnormal Psychology, 104,* 32-40.

Smith, G. T., McCarthy, D. M., & Goldman, M. S. (1995). Self-reported drinking and alcohol-related problems among early adolescents: Dimensionality and validity over 24 months. *Journal of Studies on Alcohol, 56,* 383-394.

Smith, G. T., Spillane, N. S., & Annus, A. M. (2006). Implications of an emerging integration of universal and culturally specific psychologies. *Perspectives on Psychological Science, 1,* 211-233.

Spillane, N. S. & Smith, G. T. (2007). A theory of reservation dwelling American Indian alcohol use risk. *Psychological Bulletin, 133,* 395-418.

Substance Abuse and Mental Health Services Administration (2003). Results from the 2002 National Survey on Drug Use and Health: National Findings. Retrieved July 30, 2007 from http://www.niaaa.nih.gov/Resources/DatabaseResources/QuickFacts/AlcoholConsumption/dkpat2.htm.

Tellegen, L. (1988). The analysis of consistency in personality assessment. *Journal of Personality, 56,* 621-663.

Vega, W. A., Zimmerman, R. S., Warheit, G. J., Apospori, E., & Gil, A. G. (1993). *American Journal of Public Health, 83,* 185-189.

Viken, R. J., Rose, R. J., Morzorati, S. L., Christian, J. C., & Li, T. (2003). Subjective intoxication in response to alcohol challenge: Heritability and covariation with personality, breath alcohol level, and drinking history. *Alcoholism: Clinical and*

Experimental Research, 27, 795-803.

Wallace, J. M., Bachman, J. G., O'Malley, P. M., Schulenberg, J. E., Cooper, S. M., & Johnston, L. D. (2003). Gender and ethnic differences in smoking, drinking and illicit drug use among American 8th, 10th and 12th grade students, 1976-2000. *Addiction, 98,* 225-234.

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

Warheit, G. J., Vega, W. A., Khoury, E. L., Gil, A., & Elfenbein, P. H. (1996). A comparative analysis of cigarette, alcohol, and illicit drug use among an ethnically diverse sample of Hispanic, African American, and Non-Hispanic white adolescents. *Journal of Drug Issues, 26,* 901-922.

Whiteside, S. P., Lynam, D. R., Miller, J. D., & Reynolds, S. K. (2005). Validation of the UPPS impulsive behaviour scale: A four-factor model of impulsivity. *European Journal of Personality, 19,* 559-574.

Whiteside, S. P. & Lynam, D. R. (2001). The Five Factor Model and impulsivity: Using a structural model of personality to understand impulsivity. *Personality and Individual Differences, 30,* 669-689.

Wills, T.A. & Cleary, S.D. (1999). Peer and adolescent substance use among 6th-9th graders: Latent growth analyses of influence versus selection mechanisms. *Health Psychology, 18,* 453-463.

Yanovitzky, I. (2005). Sensation seeking and adolescent drug use: The mediating role of association with deviant peers and pro-drug discussions. *Health Communication,*

17, 67-89.

Zhang, L., Welte, J. W., & Wieczorek, W. F. (1997). Peer and parental influences on

    male adolescent drinking. *Substance Use & Misuse, 32,* 2121-2136.

Zuckerman, M., Kuhlman, D. M., Joireman, J., Teta, P., & Kraft, M. (1993). A

    comparison of three structural models for personality: The big three, the big five

    and the alternative five. *Journal of Personality and Social Psychology, 65,* 757-

    768.

VITA

Sarah Pedersen was born in Saxonburg, Pennsylvania, on July 25, 1981. After finishing high school in 1999, she went to the University of Montana to complete her undergraduate studies emphasizing psychology. She received a M.A. in psychology in December 2005 from the University of Missouri-Columbia, and a Ph.D. in clinical psychology from the University of Missouri-Columbia in August 2010. She will complete her clinical internship at Western Psychiatric Institute and Clinic at the University of Pittsburgh Medical School in August 2010.