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Turning To The Bottle For Answers: Identifying Racial Differences in Predictors of Risk Drinking Among College Women

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TURNING TO THE BOTTLE FOR ANSWERS: IDENTIFYING RACIAL DIFFERENCES IN PREDICTORS OF RISK DRINKING AMONG COLLEGE WOMEN

A thesis submitted in partial fulfillment of the requirements for the degree of Master of Science at Virginia Commonwealth University

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Abstract

TURNING TO THE BOTTLE FOR ANSWERS: IDENTIFYING RACIAL DIFFERENCES IN PREDICTORS OF RISK DRINKING AMONG COLLEGE WOMEN

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Epidemiological data suggest that alcohol use and related problems have increased among college women. The current study examined psychosocial predictors of risk drinking in a sample of college women (N=360), whether race moderated this relationship. Potential predictors included: daily smoker; premenstrual syndrome (PMS) symptom severity; age at first alcohol use, negative affect, parental history of alcohol problems and minority status. Analyses found that somatic PMS symptom severity score, age of first alcohol use, daily smoking, age of first alcohol use and non-minority status were related. Findings suggest that minority group membership was associated with low risk drinking, while somatic PMS severity scores were associated with high-risk alcohol use among White women. While further research is needed, current study findings suggest that screening college women for somatic symptoms of PMS and alcohol use may identify women at greater risk of developing alcohol use disorders.
Turning to the Bottle For Answers: Identifying Racial Differences in Predictors of Risk Drinking Among College Women

In 2002, the National Institute on Alcohol Abuse and Alcoholism (NIAAA) held a workshop to discuss racial and ethnic health disparities in alcohol use research. This “call to arms” (Russo, Purohit, Foudin & Salin, 2004) was an opportunity for researchers to discuss findings, which suggest that racial and ethnic minorities suffer greater health consequences and had poorer alcohol treatment outcomes despite lower consumption rates (Russo et al., 2004). After this meeting, many investigators focused on gathering epidemiological data to demonstrate the types of health disparities experienced by various racial and ethnic groups. For example, higher rates of fetal alcohol spectrum disorders and liver disease were identified among African-Americans and Hispanics (Flores, Yee, Leng, Escarce, Bastani, Salmeron & Morales, 2008; Russo et al., 2004). In addition to pinpointing which disparities individual racial and ethnic groups experience, researchers focused on identifying correlates and potential causal factors such as physiological aspects and sociocultural influences that may account for these disparities. Overall, many of the findings suggest that alcohol related health disparities experienced by racial and ethnic minorities are correlated with lower socioeconomic status, perceived discrimination, cultural mistrust of health care professionals, physiological processes and increased access to alcohol within neighborhoods (Chartier & Caetano, 2010).

With increasing knowledge about the types of disparities experienced by different racial and ethnic groups, researchers have focused on identifying developmental trajectories in order to explain the progression from regular alcohol use to adverse alcohol related health problems. Much of the research with racial and ethnic individuals has focused on two developmental
periods: adolescence, usually early to middle adolescence, when individuals are enrolled in secondary school and experiment with alcohol use, and middle to late adulthood, when alcohol related diseases such as cancer, liver disease and cardiovascular diseases are often diagnosed. Studying these developmental periods has helped researchers identify psychosocial protective factors that account for less drinking among racial and ethnic minority youth during middle and high school years, and to examine risk factors that may lead to adverse health consequences later in life because of earlier alcohol use.

Ironically, researchers have not had the same enthusiasm for studying racial and ethnic college students, despite evidence that the highest rates of alcohol abuse and alcohol related injury and death occur among 18-25 year olds enrolled in college. Estimates identify that 31% of college students meet Diagnostic and Statistical Manual of Mental Disorders, Fourth Edition, Text Revision (DSM-IV-TR) criteria for alcohol abuse (Knight, Wechsler, Kou, Seibering, Weitzman & Schuckit 2002) while additional studies report that the quantity and frequency of alcohol use on college campuses exceeds national averages (Johnston, O’Malley, Bachman & Schulenberg, 2009). Moreover, when compared to their same aged peers who are not enrolled in college full-time or not enrolled at all, college students report higher rates of alcohol abuse and alcohol consumption overall (Johnston et al., 2009).

Given the high injury rates and high rates of alcohol abuse among college students, it is odd that researchers with an interest in understanding the development of racial and ethnic health disparities have not examined this population to determine some of the risk and protective factors that may lead to or prevent the incidence of these disparities later in life. Some investigators have argued that when they attempted to study racial and ethnic minority college students’ alcohol use, few participants reported drinking at dangerous levels and that in many cases, their
drinking behaviors were much lower than White students’ (Centers for Disease Control, 1997; O’Malley & Johnston, 2002; Wechsler, Lee, Kuo & Lee, 2000). Such findings have dissuaded additional researchers from examining alcohol use among racial and ethnic minority college students, even with projected increases in the U.S. minority population. However, several studies examining alcohol use among college students demonstrate that there are differences in alcohol consumption rates between racial groups and within specific racial groups (Hatchett & Holmes, 2004; O’Hare, 1995; Wechsler et al., 2000). This small subset of studies not only demonstrates the importance of further inquiry into the alcohol consumption patterns and associated psychosocial correlates among racial and ethnic minority college students, but the findings, which suggest within-group differences, beg for researchers to continue studying these in order to better understand why some racial and ethnic groups are disproportionately affected by negative health outcomes later in life.

This current gap in the literature leaves much to be desired and numerous unanswered questions regarding the importance of risk and protective factors in alcohol use among college students and how they interact among racial and ethnic minority college students. The purpose of the proposed study is to add to the current body of literature by examining racial and ethnic differences in rates of risk drinking among a sample of ethnically diverse college women, and to identify predictors of risk drinking among the different racial and ethnic groups represented in the current sample. Recent research suggests that racial and ethnic minority women are at greater risk of experiencing adverse consequences such as higher mortality rates from liver and cardiovascular diseases (Russo et al., 2004; Stinson, Grant & Dufour, 2001), higher prevalence of fetal alcohol spectrum disorders (Abel, 1995; Russo et al., 2004), increased reports of injuries from intimate partner violence (Cunradi, Caetano, Clark & Schafer, 1999) and untreated alcohol
use disorders as a result of their drinking (Schmidt, Ye, Greenfield & Bond, 2007). Additionally, data suggest that that in the eight year time period between 1993 and 2001, the number of female college students who reported drinking to intoxication three or more times during the past month increased from 18% to 24%, while the number of college women reporting three more binge drinking episodes in the past month increased from 35% to 42% (O’Malley & Johnston, 2002). Despite the increasing alcohol use among college women and the prevalence adverse health outcomes related to alcohol use for women and especially for racial and ethnic minority women, researchers have not fully explored the relationship between race, ethnicity, psychosocial factors and risk drinking in college women. As a result, limited information exists to assist researchers and clinicians with developing efficient and reliable screening tools that identify racial and ethnic minority college women at highest risk for experiencing immediate or long-term alcohol related problems. As with any understudied group in research, a lack of information about the prevalence of risk drinking among racial and ethnic minority women produces an inaccurate portrayal of the extent and nature of the problem. As a result, limited knowledge may delay intervention and exacerbate alcohol related health disparities. Because of a national interest in prevention of alcohol-related health disparities, further research is warranted to understand how drinking at the college level may negatively impact racial and ethnic minority women’s health later on in life, and to determine how race moderates the relationship between known psychosocial risk factors associated with alcohol related health problems.

The proposed study will use the NIAAA’s definition of risk drinking, which is alcohol use by a woman that exceeds three alcoholic beverages daily or seven alcoholic beverages weekly, when one alcoholic beverage is defined as one 12-ounce beer, 8 ounces of malt liquor, a 5-ounce glass of wine or one 1.5-ounce shot of 80-proof distilled spirits or liquor (NIAAA,
According to the NIAAA’s guidelines, alcohol consumption at or above this level places a woman at an increased risk of experiencing immediate adverse consequences in addition to increasing the probability that she will also experience long-term alcohol related health problems if she continues consuming alcohol at this quantity and frequency (NIAAA, 2004). Quantitatively, risk drinking will also be measured using a sum score of equal to or greater than eight on the Alcohol Use Disorder Identification Test (AUDIT), which is a measure used to differentiate high-risk drinkers from individuals whose drinking may not put them at risk for experiencing negative consequences as result of their drinking.

First, the literature on alcohol use and abuse will be summarized with a focus on the prevalence of high-risk alcohol use among college women. In addition, psychosocial correlates associated with high-risk alcohol use in this population will be examined and critiqued. Then, the following aims of the proposed study will be examined: 1) to identify differences in risk drinking among the racial and ethnic groups represented in the sample of participants; 2) to identify predictors of risk drinking, defined as having an AUDIT score of greater than or equal to eight; and 3) to determine whether race moderates the relationship between significant predictors and risk drinking.

Review of the literature

Alcohol Use

Description of the substance and operational definition of an alcoholic beverage.

Alcohol, which is known as ethyl alcohol or ethanol, is a drug categorized as a Central Nervous System depressant that is commonly consumed in liquid form and administered orally (Julien, 2008). Ethyl alcohol is water and fat soluble, and drinks that contain it usually contain a percentage of the molecule in its pure form (Julien, 2008). Because of its high solubility, alcohol
is quickly absorbed in the gastrointestinal tract and stomach. In many cases, absorption and metabolism occur so quickly that an individual’s blood alcohol concentration (BAC) peaks 30-90 minutes after ingestion of an alcoholic beverage (Julien, 2008).

As demonstrated in Figure 1, the NIAAA has defined alcoholic beverages in the following way: a beverage containing 13.7 grams, also known as 0.6 ounces of pure alcohol. This quantity of alcohol is often found in one 12-ounce beer, 8 ounces of malt liquor, a 5-ounce glass of wine or one 1.5-ounce shot of 80-proof distilled spirits or liquor (NIAAA, 2010). While many individuals report using alcohol frequently, only a subset of these users will eventually go on to experience alcohol-related impairments in daily functioning, adverse health consequences and/or other sequelae of Alcohol Use Disorders (AUDs; Hasin, Stinson, Ogburn & Grant, 2007).

**Alcohol Use Disorders**

**Diagnostic criteria for alcohol use disorders.** The DSM-IV-TR has identified two specific types of AUDs: Alcohol Dependence and Alcohol Abuse.

**Alcohol dependence criteria.** Alcohol Dependence is a disorder characterized as “a maladaptive pattern of [alcohol] use that leads to clinically significant impairment or distress” (American Psychiatric Association, 2000, p. 197). More specifically, individuals must have experienced three or more of the following in the past 12 months to meet criteria for Alcohol Dependence: 1) tolerance, or needing increasing amounts of alcohol to become intoxicated; 2) withdrawal, which is defined as meeting DSM-IV-TR criteria for substance withdrawal; 3) increased consumption; 4) unsuccessful attempts to quit use despite a high motivation to quit; 5) spending “a great deal of time using, obtaining alcohol or recovering from the effects of alcohol”; 6) neglecting important obligations because of use; and 7) continued use “despite knowledge of a medical or psychological problem exacerbated by use” (APA, 2000, p.197).
Because Alcohol Dependence includes symptoms of physiological dependence (e.g. withdrawal and tolerance), a diagnosis of Alcohol Dependence is generally considered to represent the more severe form of alcohol use disorder.

**Alcohol abuse criteria.** If an individual does not meet the criteria for Alcohol Dependence, but continues to drink at a level resulting in legal, medical, psychological or interpersonal problems, a diagnosis of Alcohol Abuse is considered. Alcohol Abuse is described as “a maladaptive pattern of alcohol use manifested by recurrent and significant adverse consequences related to the repeated use of alcohol” (APA, 2000, p.198). In order to meet criteria for Alcohol Abuse, this maladaptive period of alcohol use must occur within a 12-month period and result in clinically significant impairment or distress as characterized by at least one of the following: 1) alcohol use that results in “failure to fulfill role obligations at work, school or home”; 2) repeated alcohol use in potentially harmful situations; 3) repeated alcohol related legal problems; 4) continued alcohol use despite social or interpersonal problems that follow alcohol consumption or have been aggravated by alcohol consumption (APA, 2000).

Despite the presence of standard diagnostic criteria for Alcohol Abuse that specifically delineate how alcohol consumption can negatively impact an individual’s life, clinicians and researchers have found that these diagnostic criteria do not specify crucial information such as the number of beverages that need to be consumed to result in impairment, quantity, frequency of use and years of regular use. Although many of these critical components are assumed, non-specific information may mislead clinicians and researchers when assessing an individual’s patterns of alcohol use. Additionally, much of this information is necessary to identify individuals whose drinking may put them at risk for developing an AUD. As a result, the National Institutes of Alcohol Abuse and Alcoholism (NIAAA) have offered more
comprehensive definitions of Alcohol Abuse that incorporate this missing information and posit a more complete set of criteria for current drinkers.

*Risk Drinking.* According to the NIAAA’s guidelines, alcohol consumption should be conceptualized as high or low-risk use, where risk refers to the probability that an individual may experience immediate negative consequences after consuming alcohol (NIAAA, 2010). The NIAAA cautions that individuals vary in their ability to metabolize alcohol, and as such, even low-risk alcohol consumption may result in negative outcomes for some individuals. However, after reviewing nationally based population studies, the NIAAA suggests that a daily consumption of three or more drinks for women and four or more drinks for men may result in negative outcomes for most individuals, and, as such, should be considered high-risk drinking. Figure 2 depicts the NIAAA recommended guidelines for alcohol use. The NIAAA posits that high-risk drinking, defined as exceeding daily and or weekly consumption guidelines, will result in immediate and long term negative health, social, occupational and legal outcomes for individuals who consume alcohol (NIAAA, 2010). The NIAAA has divided the category of high-risk drinking into two smaller sub-categories: at risk drinking and highest risk drinking. At risk drinking occurs when a female consumes more than three drinks daily or seven weekly, and when a male consumes more than four drinks daily or fourteen drinks weekly (NIAAA, 2010). Highest risk drinking occurs when a woman consumes more than three drinks daily and more than seven drinks weekly, and it also occurs when a man drinks more than four drinks daily and over 14 drinks weekly (NIAAA, 2010).

By contrast, low-risk drinking has been defined as a woman consuming less than three drinks daily and less than seven drinks during the week and a man consuming less than four drinks daily and less than 14 drinks during the week (NIAAA, 2010). According to the NIAAA,
consumption at this level typically does not result in harmful consequences for most individuals who consume alcohol (NIAAA, 2010). By incorporating a specific definition of what constitutes an alcoholic beverage as well as a quantities and frequencies that typically result in the clinical impairments outlined by the DSM-IV-TR, the NIAAA guidelines given researchers and clinicians a way to assess for individuals who may be at risk of eventually developing AUDs and who may benefit from early intervention.

Binge drinking is another definition of alcohol abuse that has been suggested by researchers who study patterns of alcohol consumption, especially among samples of college students. Currently, binge drinking is defined as women consuming four or more alcoholic beverages during a single occasion and men consuming five or more alcoholic beverages during a single occasion (NIAAA, 2010). The definition of binge drinking has been revised several times. Traditionally, a binge referred to “an extended period of time (usually two or more days) during which a person repeatedly administers alcohol or another substance to the point of intoxication, and gives up his/her usual activities and obligations in order to use the substance” (Schuckit, 1998, p.124). For decades this definition has been used by clinicians to describe individuals meeting the criteria for alcohol abuse because it described a pattern of alcohol consumption that resulted in several of the adverse consequences outlined in the DSM-IV-TR’s diagnostic criteria for alcohol abuse.

Researchers within the field have debated over whether the new definition of women consuming four or more drinks in one setting and men consuming five or more drinks in one setting truly constitutes a “binge”. The NIAAA defines binge drinking as “a pattern of drinking alcohol that brings blood alcohol concentration (BAC) to 0.08 gram percent or above” (National Council on Alcohol Abuse and Alcoholism, 2004). The NIAAA (2004) has identified that this
BAC typically occurs when women consume more than four alcoholic beverages in two hours or when men consume more than five alcoholic beverages in a two-hour period. Epidemiological studies also confirm that consumption of at least five drinks during the course of several hours results in individuals experiencing alcohol related problems such as driving while under the influence, nausea, dizziness, physical injury and absences at work (Midanik, Tam, Greenfield & Caetano, 1996).

Although the five-four drinking cut-off is clearly associated with negative outcomes in the general population, many researchers hesitate to characterize this as binge drinking. They suggest characterizing alcohol consumption that is at or above the four-drink cutoff for women and five-drink cut-off for men as heavy episodic drinking. Researchers in the field who contribute regularly to the body of literature on patterns of alcohol consumption have used both terms interchangeably to refer to alcohol use that exceeds daily consumption guidelines, results in a blood alcohol concentration of 0.08 gram percent or higher and leads to impairment and negative outcomes for the individual consuming the alcohol. As such, the definition of binge or heavy episodic drinking provides additional clarification on the amount of alcohol that needs to be consumed, as well as the frequency with which this amount must be consumed in order to achieve the level of clinically significant impairment as described in the Alcohol Abuse criteria put forth in the DSM-IV-TR.

The remainder of this literature review will focus on alcohol use and abuse as it occurs in a college population. However, instead of relying solely on the DSM-IV-TR’s diagnostic criteria for Alcohol Abuse, the author has chosen to adopt the NIAAA’s risk drinking guidelines as the definition of Alcohol Abuse discussed and examined throughout this paper. These guidelines include the criteria for binge drinking and when violated, could lead an individual to reach a
BAC of 0.08 gram percent or higher and subsequently experience adverse outcomes as a direct result of their alcohol consumption. As such, the NIAAA risk categories provide an alternative index for assessing the relationship between alcohol consumption and the negative effects of this consumption among a college population.

**Prevalence of Alcohol Consumption and Alcohol Use Disorders**

**Prevalence in the general population.** According to the 2009 National Survey on Drug Use and Health (NSDUH), 51.9% of Americans age 12 and older reported consuming at least one alcoholic beverage in the past 30 days (Substance Abuse and Mental Health Services Administration; SAMSHA, 2010). However, for many individuals, their consumption of alcohol exceeds recommended guidelines and puts them at greater risk of experiencing negative health, social, occupational and legal outcomes. Data from the National Epidemiologic Survey on Alcohol and Related Conditions (NESARC) found that a little over one third of the general population met DSM-IV criteria for an alcohol use disorder (AUD) in their lifetime (Hasin, Stinson, Ogburn & Grant, 2007). Of this percentage, 41% reported meeting DSM-IV criteria for Alcohol Dependence and the remaining 59% met DSM-IV criteria for Alcohol Dependence (Hasin et al., 2007).

In addition, alcohol use and AUDs are not randomly distributed in the general population. Instead, research has consistently stated that prevalence rates vary across a variety of subgroups. In particular, rates of alcohol use and related problems vary across gender, race/ethnicity and age. The following section summarizes key findings in these three domains, with specific focus on how each one relates to the central purpose of this study.

**Gender differences in alcohol use.** The prevalence of alcohol use and AUDs in men and women has varied over time. While the majority of epidemiological studies continue to report
that men consume greater quantities of alcohol and report higher prevalence rates of AUDs (Hasin et al., 2007; SAMSHA, 2010), these rates have gradually converged, especially as younger women engage in more alcohol use, (Keyes, Grant & Hasin, 2008; Keyes, Martins, Blanco & Hasin, 2010). To illustrate, the 2004 NSDUH survey found that 44.0% of women and 56.9% of men over age 12 reported alcohol use in the past month (SAMHSA, 2005). Five years later, the 2009 NSDUH survey reported rates of alcohol use (past month) of 46.5% for women and 57.6% for men (SAMHSA, 2010). While the gender gap persists, women showed a larger change over the five-year period than did men (2.5% increase and 0.7% increase, respectively).

Researchers have identified a myriad of factors that contribute to differences in prevalence rates among men and women. Specifically, findings demonstrate that when given the same amount of alcohol, women achieve a much higher blood alcohol level or concentration (BAL or BAC) than men because of enzymatic differences that lead women to absorb alcohol differently than men (Julien, 2008). Women also contain lower percentages of total body water, which contributes to a higher blood concentration compared to men (Wilsnack, Vogelstanz, Wilsnack & Harris, 2000). Because women often achieve a higher BAL than men, they reach a desired state of intoxication at a lower quantity of alcohol than men, whereas men may imbibe more in order to achieve their desired state of inebriation. Previously, researchers assumed that women drank less because these physiological differences allowed them to experience a prolonged state of intoxication. However, as current epidemiological data demonstrate, rates of alcohol use and AUDs among women are increasing despite physiological factors assumed to discourage women from drinking at the same level that men do.

In addition to examining physiological factors that may account for gender divergence in alcohol use, researchers have identified that men and women possess different developmental
trajectories for AUDs. Specifically, when comparing the onset age of regular use and the amount of time from regular use to dependence or abuse, numerous studies report that women begin using alcohol regularly at a later age and that they develop AUDs during a shorter time period than men (Hussong & Bauer, 2008; Randall, Roberts, Del Boca, Carroll, Connors & Mattson, 1999; Zilberman, Tavares, et-Guebaly, 2003). This effect, first described by Lisanksy (1958), is known as telescoping and describes “the rapid acceleration of alcohol-related problems in women following drinking onset” (Hussong & Bauer, 2008, p.64). Telescoping may account for some of the differences in the rates of AUDs, especially considering that the average age at onset of alcohol abuse was 22.5 years of age and the age of alcohol dependence onset was 21.9 years old for participants in the NESARC study (Hasin et al., 2007). Because women begin drinking regularly later, they typically meet diagnostic criteria for AUDs at a later age, while men often meet diagnostic criteria for AUDs at an earlier age. When comparing AUD prevalence rates at specific age points, men often outnumber women because of their earlier ages of onset, especially when researchers compare individuals in the young adult age (e.g. 18-25 year old) range.

Finally, research has focused on the effect of gender role expectations on alcohol consumption. Specifically, in societies where alcohol consumption is associated with an expression of masculinity, women consume less alcohol and are often chastised or judged out for fear that intoxicated women will neglect household duties and become reckless, uninhibited and promiscuous (Purcell, 1994; Warner, 1997; Wilsnack et al., 2000). Overall, as the data demonstrate, rates of alcohol use among women and men differ, but are currently converging despite the presence of physiological and cultural factors once believed to maintain divergent drinking rates. With an increasing number of women who drink, especially an increasing
number of women who binge drink, rates of alcohol related problems have increased. These data demonstrate the need for researchers to continue to study this population in order to identify and treat those at greatest risk of developing an AUD or an adverse health outcome as a result of their alcohol use.

**Gender differences in alcohol related problems.** It appears that women are also more susceptible to a variety of alcohol-related health problems (NIAAA, 1999). For example, while consuming lower quantities of alcohol for fewer years than men, women were at greater risk for developing alcohol-related liver problems (Mezey, Kolman, Diehl, Mitchell & Herlong, 1988). This risk was not limited to those who consume alcohol at high-risk levels (i.e., those set by NIAAA). Instead, risks for developing liver problems remain elevated even among those who drink below NIAAA-set guidelines, when compared to non-drinking women (Gronbaek, Deis, Sorensen, Becker, Schnohr and Jensen, 1995). Investigators also found that women were more likely to die from complications of liver cirrhosis than men and they were particularly vulnerable to alcoholic hepatitis (Klatsky, Armstrong & Friedman, 1992; NIAAA, 1999; Rehm, Taylor, Mohapatra, Irving, Baliunas, Patra et al., 2010).

Some studies have found that heavy drinking confers greater risk for development of cardiovascular disease (CVD) in women (Murray, Connett, Tyas, Bond, Ekuma, Silversides et al., 2002). NIAAA (1999) reported heavy alcohol consumption was more commonly associated with CVD in women as compared to men. Likewise, Jepson, Fowkers, Donnan and Housley (1995) found that alcohol use increased rates of premature death from CVDs among women. More research is needed, however, as patterns of risk appear to be non-linear, with light or moderate alcohol use potentially serving as a protective factors for cardiovascular health in
Alcohol use in women is also associated with increased risk for certain cancers, including those of the oral cavity, pharynx, esophagus, larynx, rectum, breast and liver (Allen, Beral, Casabonne, Kan, Reeves, Brown & Green, 2009). Further, even low to moderate levels of drinking have been related to neurocognitive deficits among women. Specifically, in the short term, at comparable levels of alcohol ingestion, women are more likely to experience blackouts and other memory impairments than their male counterparts (NIAAA, 2004). Neurological and neuropsychological studies of men and women with long term alcohol dependence have found decreased brain volume as well as similar reports of memory and learning impairments (Hommer, Momenan, Kaiser & Rawlings, 2001; Mann, Batra, Gunther & Schroth, 1992). Hommer and colleagues (2001) found, however, that many female study participants reported a shorter duration of drinking time than did male participants, suggesting that women’s brains may be more susceptible to the adverse effects of alcohol.

Heavy alcohol consumption has also been associated with lower bone density and increases in the incidence of osteoporosis among women (National Institute of Arthritis and Musculoskeletal and Skin Diseases, 2000). In human and animal studies, researchers have observed that alcohol appears to lower estrogen levels, thus increasing the risk for osteoporosis, especially among post-menopausal women, as well as increasing irregular menstrual cycles and infertility in women of child-bearing age (Becker, Tonneson, Kass-Claesson & Glud, 1989; Valimaki, Pelkonen, Salaspuro, Harkonen, Hirvonen & Ylikari, 1984). Any alcohol use during pregnancy and while breast-feeding has been associated Fetal Alcohol Spectrum Disorders (CDC, 2002); low birth weight (Little & Wendt, 1991); birth defects (Abel, Jacobsen
& Sherwin, 1983); behavioral and cognitive disorders (Moore, Jones & Lewis, 2002); and increased risk of miscarriage in the first trimester (Kesmodel, Wisborg, Olsen, Henriksen & Sechler, 2002).

In addition to the physical health consequences associated with alcohol use, research demonstrates that women who consume alcohol are at increased of experiencing psychological and interpersonal problems such as depression (Dixit & Crum, 2000; Wilsnack, Klassen, Schur & Wilsnack, 1991); increased use of nicotine and illicit substances (Floyd, Sobell, Velasquez, Nettleman, Sobell et al, 2007; Lejuez, Bornovalova, Reynolds, Daughters & Curtin, 2007); and sexual assault, rape and physical violence (NIAAA, 2004; Hingson, Heeren, Zakocs, Kopstein & Wechsler, 2005). Finally, women who consume alcohol, especially moderate to heavy amounts are at risk of developing Alcohol Abuse and Dependence (NIAAA, 2004).

Overall, extant research documents the association between alcohol consumption and adverse health outcomes for women. While the majority of the research emphasizes that moderate and heavy consumption increase the risk that women will experience some of the above illnesses, nearly all research reporting on the negative health outcomes associated with alcohol consumption references the casual relationship between women’s increased BAL and levels of impairment. Ultimately, it is believed that physiological differences in metabolism, absorption and reactivity to alcohol contribute greatly to the adverse health outcomes that women drinkers experience. Because of their increased vulnerability to these negative outcomes and reported increases in alcohol use among women, this group is in need of research that will assist with the identification and treatment of those at greatest risk.

**Racial and ethnic differences in alcohol use and AUDs.** Epidemiological data demonstrate that in addition to gender differences, there are also racial and ethnic differences in
the prevalence rates of alcohol consumption. Namely, 2008 NSDUH data (SAMSHA, 2009) suggested that a larger percentage Caucasian or White participants age 12 or older reported current (past month) alcohol use than any other racial group (56.1%), followed by American Indians and Alaska Natives (44.7%), Hispanics (42.1%), Blacks (39.3%) and Asians (35.2%). As with differences in alcohol use, data from the NESARC demonstrate that AUDs are not normally distributed across the population. Racial and ethnic differences are also prominent in the lifetime reports of AUDs, as demonstrated by the 43% percent of Native Americans, the 34.1% of Whites, the 21.6% of Hispanics, the 20.6% of Blacks, and the 11.6% of Asians who reported a lifetime diagnosis of at least one AUD (Hasin et al., 2007). These data suggest that when comparing past month use with AUD prevalence rates, it appears that a high percentage of racial and ethnic minorities go on to develop and AUD despite lower reported use.

Several factors are associated with the differences in consumption patterns and AUD prevalence rates among various racial and ethnic groups assessed. Much of the research on correlates of alcohol use among racial and ethnic minorities has focused on identifying biological and sociocultural factors that may account for differences in alcohol consumption across racial and ethnic groups. This review will focus primarily on the most commonly cited factors such as genetic and biological differences as well as socio-cultural differences that influence alcohol consumption patterns.

Extant research posits that among the general population, differences in epidemiological data on AUDs and consumption rates may be the result of genetic factors, which influence the metabolism and absorption of alcohol after it is ingested (Edenberg, 2007; Eng, Luczak & Wall; 2007). Edenberg (2007) has identified one of the genes for aldehyde dehydrogenase (ALDH2), an enzyme used in the metabolism of alcohol, as a gene with variations that differ across ethnic
groups. Specifically, individuals who have an inactive copy of the ALDH2*2 allele tend to be of Asian descent, are rarely from European or African descent (Edenberg, 2007 & Oota, Pakstis, Boone-Tamir, Goldman, Grigorenko, Kajuna et al., 2004), and often experience unpleasant side effects, such as facial and body flushing, after consuming alcohol. The prevalence of this allele in Asian individuals may explain why some Asians and Asian Americans report the lowest levels of alcohol and AUD consumption in the general population and in some college samples.

Another possible explanation for racial and ethnic variability in drinking levels and AUD diagnoses include socio-cultural beliefs and norms regarding alcohol consumption. For example, religion, ethnic identity, adherence to cultural values and community immersion are protective factors that have been associated with decreasing levels of alcohol use and abuse within racial and ethnic minority communities (Brown, Parks, Zimmerman & Phillips, 2001; Herd and Grube, 1996). It is believed that these protective factors buffer against stressors such as assimilating to the social and cultural climate of the United States, socioeconomic barriers, racism and discrimination (Al-Issa, 1997).

Similarly, researchers posit that cultural norms, which discourage inebriation and drinking outside of appropriate social settings, assist with the regulation of alcohol consumption and result in lower rates of alcohol abuse and dependence (Caetano et al., 1998). However, one caveat to the acceptance of this belief, is that with acculturation and assimilation to Western values and norms about drinking, many immigrants and U.S. born individuals no longer ascribe to the cultural values that admonish against drinking to intoxication or outside of appropriate social situations (Caetano et al., 1998). The most recent data from the NSDUH (SAMSHA, 2010) has documented that Asians and Hispanics born in the United States report higher rates of past-month alcohol use and past-month binge drinking than their foreign born Asian and
Hispanic counterparts, which seems to support the premise that acculturation influences alcohol consumption patterns in some racial and ethnic minority groups. In summary, racial and ethnic differences in alcohol use and AUD prevalence rates persist, with Whites reporting higher rates of alcohol use and AUDs. Findings continue to support genetic and sociocultural factors that maintain these differences and reduce the number of racial and ethnic minorities who report drinking.

However, despite their lower alcohol use and AUD prevalence rates, racial and ethnic minorities who do drink are disproportionately affected by adverse health consequences that have been associated with alcohol use (Flores et al., 2008; Russo et al., 2004). Like women, racial and ethnic minorities appear to be more vulnerable to the toxic effects of alcohol and at greater risk of experiencing unhealthy consequences when they drink. Despite the NIAAA’s “call to arms”, these vulnerabilities and the limited research that exists to explain the association between minority status and alcohol related health disparities demonstrate that this is an area that researchers need to explore in order to prevent or decrease these disparities.

**Racial and ethnic differences in alcohol related problems.** Current findings in this area have identified where alcohol related health disparities occur and some of the correlates associated with them. Disparities have been identified in utilization of alcohol treatment programs (Schmidt, Ye, Greenfield & Bond, 2007), severity and duration of AUD symptoms (Schmidt et al., 2007; Chartier & Caetano, 2010), social consequences of alcohol use (Chartier & Caetano, 2010; Mulia, Ye, Greenfield & Zemore, 2009; Sloan, Malone, Kertsez, Wang & Costanzo, 2009;) and physiological sequelae of alcohol use (Mulia et al., 2009).

Research suggests that when diagnosed with an AUD, Hispanics and Blacks are least likely to seek treatment for alcohol use problems (Schmidt & Weisner, 2000). Several possible
explanations abound. First, unemployment and lack of health insurance drastically limit one’s ability to pay for private sector alcohol treatment services and disproportionately more Hispanics and Blacks are uninsured, have lower incomes or are unemployed (Schmidt et al., 2007). Second, individuals may not seek treatment until their symptoms are severe and if no care is available, these symptoms may increase in severity and lead to the experience of additional consequences (Weisner et al., 2002).

Research also suggests that racial and ethnic minority group members experience more social consequences for their drinking than their White peers. Specifically, in a longitudinal study of the relationship between heavy drinking during young adulthood and employment in mid-life, Sloan and colleagues (2009) found that Blacks who engaged in heavy drinking during young adulthood were more likely to be unemployed mid-life. Findings also demonstrate that Black and Hispanic couples report higher rates of alcohol-attributed intimate partner violence (Caetano et al., 2000). Identified medical sequelae include higher mortality rates from liver and cardiovascular disease (Stinson, 2001; Yoon et al., 2001; Russo et al., 2004), higher prevalence of fetal alcohol spectrum disorders (Abel, 1995; Russo et al., 2004), increased reports of injuries from intimate partner violence (Cunradi, Caetano, Clark & Schafer, 1999) and a greater occurrence of untreated alcohol use disorders (Schmidt et al., 2007).

Ultimately, these data demonstrate that in the United States, racial and ethnic minority status is often associated with socioeconomic disadvantage and barriers to care (Schmidt et al., 2007). Because of these barriers, racial and ethnic minority groups may suffer greater consequences for their drinking. More important for this study is the cumulative effect of being both a woman and a racial/ethnic minority. As previously delineated, both women and racial minorities are more likely to experience negative consequences from their alcohol use. As such,
racial and ethnic minority women represent an extremely vulnerable population that warrants additional attention in order to reduce their risk of experiencing adverse outcomes as a result of their alcohol use.

Age group differences in alcohol use and AUD prevalence. In addition to gender and racial differences in alcohol use, AUDs and alcohol-related consequences, researchers have also identified age as an important variable. Most notably, individuals in the 18-29 age group continue to report the highest rates of alcohol consumption as well as lifetime and past year diagnoses of AUDs followed by individuals in the 30-44 age group, the 45-64 age group and individuals reporting their age as 65 and over (Johnston & O’Malley, 2002; Hasin et al., 2007).

Given the high prevalence rates of AUDs among individuals in the 18-29 age range, researchers have shifted their focus to examining predictors and factors influencing increased alcohol consumption among this age group. One pertinent factor seems to be educational attainment and college attendance. In the 2001-2002 NESARC dataset, individuals who reported attending college had higher rates of AUDs than any other educational group assessed. Data from the 2001 National Household Survey on Drug Abuse (NHSDA), sponsored by SAMSHA reported that college students were more likely to be diagnosed with an AUD than their non-college peers (Slutske, 2005). Specifically, 18% of college students met criteria for either Alcohol Abuse or Dependence, while only 15.1% of their non-college attending peers met this same criteria (Slutske, 2005).

However, differences in disorder prevalence rates were also observed. College students and non-college attending peers reported similar percentages of past year Alcohol Dependence (Slutske, 2005). Despite a lack of differences in reports of Alcohol Dependence among both samples, college students were more likely to report experiencing tolerance and withdrawal
symptoms than their non-college attending peers (Slutske, 2005). College students were also more likely to meet the diagnostic criteria for Alcohol Abuse: 11.9% of college attending respondents reported experiencing symptoms of Alcohol Abuse in the past year, compared to 8.5% of their non-college attending peers (Slutske, 2005).

However, data from several studies, both of the general population and within college samples, demonstrate that college students report heavier alcohol consumption and more alcohol related problems than their same aged, non-college attending peers. In 2008, 61% of all full-time college students who participated in the NSDUH reported drinking in the past month, and 41% of this sample reported binge drinking (SAMSHA, 2009). Comparatively, 54% of same age peers who were either not in college or enrolled part-time reported current drinking, while and 38% reported binge drinking (SAMSHA, 2009). Additional analyses revealed that within the 18-25 age group, those who had graduated college reported higher levels of drinking in the past month than those who had less than a high school diploma (SAMSHA, 2009).

**Prevalence of alcohol consumption and AUDs among college students.** Researchers interested in the alcohol consumption patterns of college students have collected their own epidemiological data on this population. Three well known studies include Monitoring the Future (MTF), sponsored by the National Institutes on Drug Abuse (NIDA), the College Alcohol Study (CAS), sponsored by the Robert Wood Johnson Foundation, and the National College Health Assessment (NCHA-II), sponsored by the American College Health Association (ACHA). Investigators of these studies have focused their efforts on documenting the prevalence of alcohol consumption among college students, in addition to noting prevalence rates of binge drinking, and drinking that often exceeds NIAAA guidelines for low-risk drinking or leads to negative consequences for those who drink. Data from the most recent wave of young
adults surveyed as part of MTF (e.g. individuals in the 18-25 age group) demonstrates that in the two-weeks prior to being surveyed, 40% of college student respondents reported in engaging in at least one episode of binge drinking, while only 30% of same age, non-college enrolled peers reported the same behavior (Johnston & O’Malley, 2009).

Investigators for the CAS have collected data in four different years: 1993, 1997, 1999 and 2001 (Wechsler & Nelson, 2008). Wechsler, Molnar, Davenport and Baer (1999) reported that college student respondents who engaged in binge drinking consumed 91% of the alcohol that the entire sample reported drinking. Wechsler and Wuethrich (2002) have also characterized drinking patterns as “polarized” with increasing amounts of college students reporting that they either abstain from drinking or that they engage in binge drinking (Wechsler & Nelson, 2008, p. 3). Wechsler and Nelson (2008) characterize the drinking pattern of college students surveyed as “one of excess and intoxication” (p.3) because 48% of college drinkers reported that a motivating factor for their drinking behavior was reaching a level of intoxication. Similarly, 29% of college student respondents reported that they were intoxicated three or more times a month (Wechsler, Lee, Kuo, Seibring, Nelson & Lee, 2002).

This characteristic pattern of drinking to intoxication or binge drinking often results in academic problems, legal problems and negative health consequences. Several researchers have identified an association between binge drinking behavior and increased absences at class, lower grade point averages, and a decreased amount of time spent studying (Powell, Williams & Wechsler, 2004; Wechsler, Lee, Kuo, Seibring, Nelson & Lee, 2002). Hingson, Heeren, Zakocs, Kopstien & Wechsler (2005) estimate that close to two million college students have driven while under the influence, and that as many as 1,700 students die from accidents that happen while under the influence of alcohol. The combination of students who report drinking to
intoxication and data that demonstrates that college students experience a disproportionate amount of adverse consequences as a result of their drinking, both speak to the conclusion that Alcohol Abuse is the most prevalent AUD among college students.

Finally, NCHA-II data collected in the fall of 2010 demonstrate that 59.1% of female students and 61.2% of male students reported alcohol use in the past 30 days (ACHA, 2011). Additionally, 24.4% reported driving after consuming at least one alcoholic beverage (ACHA, 2011).

All of these studies, while assessing different samples of college students and different domains of alcohol use (e.g. use, AUD prevalence, alcohol related problems and binge drinking), have demonstrated that alcohol use among college students varies significantly from rates reported in the general population. Additionally, they underscore the importance of studying alcohol use and predictors of risk drinking in this population because college students are more likely to consume greater amounts of alcohol and experience serious consequences as a result of their drinking (Hingson et al., 2008; Wechsler & Nelson, 2008; & Johnston & O’Malley, 2009).

Consequences experienced by college students who consume alcohol. As noted in the introduction to this literature review, it is important not to restrict the focus of research to a categorical variable such as Alcohol Abuse. This is particularly true for college students, as they may experience negative outcomes from alcohol consumption at sub-threshold levels of Abuse. Examples include unprotected sexual encounters, sex with multiple sexual partners and contraction of sexually transmitted diseases (Cooper, 2002), alcohol poisoning, blackouts, vomiting and hangovers (Wechsler, Dowdall, Davenport & Castillo, 1995), depression, increased suicide attempts and suicides (Presley, Leichliter & Meilman, 1998) and an increased risk of developing AUDs (Knight, Wechsler, Kou, Seibering, Weitzman & Schuckit, 2002).
College students who report consuming alcohol are also more likely to be victims of crimes as well as perpetrators of crimes (Abbey, 2002; Hingson et al., 2009). Hingson and colleagues (2009) report that 696,000 students are victims of physical assault committed by another student who has been drinking. Hingson and colleagues (2009) report that 97,000 students have reported being sexually assaulted while drinking, while 100,000 students reported engaging in non-consensual sex while under the influence of alcohol (Hingson, Heeren, Zakocs, Kopstein & Wechsler, 2002). Koss (1988) reported that in a national sample of college students, 74% of rape perpetrators and 55% of rape victims had consumed alcohol prior to the act. Close to four million college students report driving while intoxicated and 110,000 of those college students who reported driving under the influence have been arrested for DWI or DUI (Hingson et al., 2002; Hingson et al., 2009). Colleges have also reported increased arrests due to property damage and vandalism committed by students under the influence of alcohol (Wechsler et al., 1995; Wechsler et al., 2002).

Alcohol use among college students has also been associated with academic problems, such as declining grade point averages and increased class absences (Engs et al., 1996; Presley et al., 1996; and Wechsler et al., 2002). Several researchers have found that alcohol use, especially binge or heavier use, by adolescents and young adults often results in problems with attention, concentration, learning and memory (Zeigler, Wang, Yoast, Dickinson, McCaffree, Robinowitz, Sterling et al., 2005). Many of these cognitive difficulties often manifest as problems with studying, decreasing grade point averages and academic failure. Researchers believe that alcohol-related neurodegeneration in areas of the brain that are responsible for attention, learning and memory is a potential mechanisms for these cognitive problems. Continued and increasing use of alcohol seems to exacerbate these impairments and eventually produces cognitive deficits.
(Zeigler et al., 2005). Overall, these findings demonstrate that because of their increased use of alcohol, college students are highly susceptible to immediate and long-term alcohol related problems. Because of their greater risk, it is important that researchers examine correlates, risk and protective factors in order to identify those in need of risk reduction and other forms of intervention.

**Gender differences in rates of college alcohol use and AUDs.** While gender differences in rates of alcohol use and AUDs in the general population were reviewed above, the unique alcohol consumption rates in college students warrant specific review of gender differences in alcohol use prevalence rates and consequences in college students. The 2010 NCHA-II report found that 47.9% of female college students survey participants in fall, 2010 reported drinking 1-9 times in the past 30 days, while only 43.2% of males reported drinking this frequently in the past 30 days (ACHA, 2011). Taken together, recent data on college students demonstrate that college women are catching up to or in some cases, exceeding the consumption rates of their male peers. Among college students, researchers have noted that binge drinking has increased among college women at a rate faster than it has among college men (Wechsler & Wuethrich, 2002). Similarly, college women are more likely to report drinking heavily during their freshman year (McCabe, 2002), and they tend to be more likely to embrace binge-drinking behaviors while enrolled in college (Reifman & Watson, 2003). However, in spite of recent increases in drinking rates among college women, AUD rates continue to diverge: 5% of female college students endorsed enough criteria to warrant a diagnosis of Alcohol Dependence in the past 12-months, while 8% of male college students met enough criteria to receive this diagnosis in the past 12-month (Slutske, 2005). Additionally, 9% of female college students received a diagnosis of Alcohol Abuse in the past 12-months compared to 16% of their male college student
counterparts (Slutske, 2005). Despite the divergence among rates of AUDs in college students, increasing rates of binge drinking among college women demonstrate that with time, college women’s AUD rates may converge or eventually surpass rates reported by college men.

As the numbers of female students reporting binge drinking have increased, researchers investigating explanations for gender convergence among college students have predominately emphasized the role of peer influence and norms towards drinking on the alcohol use and AUD rates in this population. Findings demonstrate that the actual, observed drinking behavior of close friends greatly influences the alcohol consumption of both women and men (Borsari & Carey, 2006; Talbott et al., 2008; Mallett, Bachrach & Turrisi, 2009). Additionally, studies on the environmental contexts of drinking in college have surmised that college students report increased rates of drinking in social situations (e.g. parties, gatherings, dates) than when they are alone (Clapp, Shillington & Segars, 2000). Researchers have attributed this increase in drinking while in social settings to the influential aspects of injunctive peer norms about drinking. In addition to noting the relationship between observed drinking and alcohol consumption rates, this area of research has focused on the misperception of peer drinking norms. Lewis and Neighbors (2004) identified that both male and female college students overestimated the quantity and frequency of alcohol consumed among their same-sex peers, but that the misperception of these drinking norms tended to be a stronger predictor of alcohol consumption among college women when compared to college men. Specifically in a review of numerous studies, Borsari and Carey (2003) found that college women often compare their own drinking behaviors to their male peers in a social situation, and may see their male peers’ consumption levels as normative. Because college women may perceive their alcohol consumption to be much lower than that of their male peers, they may also perceive that when drinking in the
presence of male peers it is socially acceptable to drink more and that others expect for them to
drink at a similar level (Borsari & Carey, 2003; Orcutt, 1991).

Overall, data on college drinking demonstrate that gender differences are beginning to
dissipate as college women increase their use of alcohol. Because of their increase in drinking,
especially binge drinking, and physiological vulnerabilities that increase their risk of
experiencing alcohol related health problems, college women have distinguished themselves as a
group whose alcohol use requires further inquiry in order to provide early intervention to prevent
long-term consequences.

**Gender differences in alcohol related problems among college students.** As stated in
previous sections, alcohol use, especially heavy or binge drinking, often results in negative
consequences for both women and men. However, because of their increased sensitivity and
reactivity to alcohol, women often experience different adverse outcomes or varying degrees of
adverse outcomes as a result of their alcohol consumption. College women represent a unique
subset of women and the following paragraphs will examine alcohol related-problems specific to
college women instead of focusing on adverse consequences that women may experience across
developmental stages.

Numerous studies have identified that college women who consume alcohol are
especially vulnerable to experiencing rape and sexual assault. Abbey (2002) has explained the
relationship between alcohol consumption and increased victimization by examining socio-
cultural and physiological factors that influence college men’s perceptions of intoxicated women.
In reviewing studies that have examined this phenomenon and correlates of it, Abbey (2002)
surmised that college men often perceive situations where both men and women are drinking as
situations where the woman is agreeing to any sexual advances (McAuslan, Abbey, Zawacki,
Abbey (2002) concludes that alcohol hinders communication about appropriate limits for sexual behavior and increases males’ aggressive behaviors because of the disinhibitory effects of the drug. By contrast, when under the influence of alcohol, women are often unable to physically defend themselves due to the effect that alcohol has on cognition and motor functioning (Abbey, 2002).

Increased sexual violence towards college women who have been drinking and increases in unprotected sexual activity while under the influence may result in unintended pregnancies (Naimi, Lipscomb, Brewer & Gilbert, 2003) and contracting sexually transmitted diseases (Thomas, Brodine, Shaffer, Shafer, Boyer, Putnam et al., 2001). Additionally, college women who consume alcohol and do not practice safer sex, not only increase their risk of becoming pregnant, but also risk exposing the fetus to alcohol, thus increasing the risk of having a child with a Fetal Alcohol Spectrum Disorder (Ingersoll, Ceperich, Nettleman, Karanda, Brocksen & Johnson, 2005).

Alcohol use among college women has also been associated with an increased rate of depression among this population, however, it remains unclear whether the depressive symptoms precede alcohol use or vice versa. The majority of studies suggest that women may use alcohol to regulate their emotional states (Cooper, Frone, Russell & Mudar, 1995; Kassel, Jackson and Unrod, 2000; Kuntsche, Knibbe, Gmel & Engels, 2006). Additional findings suggest that a history of depression predicts moderate to heavy alcohol use among women (Dixit & Crum, 2000; Helzer & Pryzbeck, 1988; Wilsnack, Klassen, Schur & Wilsnack, 1991).

Finally, college women who consume alcohol, especially those who binge drink, are at a greater risk of developing alcohol abuse and dependence (O’Neill, Parra & Sher, 2001). College women who drink at moderate to heavy levels throughout college are also more likely to
experience the longer term health consequences of alcohol use such as various cancers, liver diseases, CVD and may eventually have an increased mortality rate of CVD. In sum, college women who drink, especially at heavier levels, increase their risk of experiencing adverse consequences as a result of their drinking. Because college women may be more likely to engage in heavier drinking than their non-college peers, their alcohol use increases their vulnerability to experience more immediate and delayed alcohol-related problems. However more research is needed to fully understand the biopsychosocial factors that may specifically implicate the health outcomes of college women drinkers.

**Racial and ethnic differences in alcohol use and AUDs in the college population.** As with gender, racial and ethnic differences in rates of alcohol use and AUDs have been observed among college students in the United States. More specifically, data from the CAS, MTF and the National College Health and Risk Behavior Survey (NCHRS, CDC, 1997) all indicate a national trend where Caucasian students reported the highest rates of binge drinking and alcohol consumption overall, followed by Hispanic, Black and Asian students (CDC, 1997; O’Malley & Johnston, 2002; Wechsler et al., 2000). Similarly, among respondents who completed the CAS, the majority of students who reported a diagnosis of either Alcohol Abuse or Alcohol Dependence in the past 12 months identified as Caucasian, followed by identified as “other”, Asian and Black (Knight et al., 2002).

Because of the lower percentage of racial and ethnic minority students who report alcohol use, researchers have focused on identifying rates of abstinence and protective factors associated with decreased alcohol use instead of examining risk factors and rates of risk drinking. When analyzing data from the three waves of the CAS, Wechsler and colleagues (2000) reported that among students who reported abstaining from alcohol use in the past year, all racial and ethnic
groups demonstrated an increase in the number of abstainers across the three waves assessed. Specifically, Black/African American students and Asian students have reported the highest rates of abstinence across all ethnic groups represented during all four years and when compared to the average rate of abstainers in the population (Wechsler et al., 2000).

Additional studies have identified the following protective factors that seem to account for lower rates of drinking among racial/ethnic minority college students: attendance at a four-year university, religiosity, spirituality, adherence to racial/ethnic cultural beliefs that disparage alcohol use and intoxication, having fewer friends who drink or attending a Historically Black College/University (Cateano et al., 1998; Hatchett and Holmes, 2004; Meilman et al., 1995; O’Hare, 1995; Paschall, Bersamin & Flewelling, 2005). Other socio-cultural factors that appear to implicate reduced drinking included feeling disconnected from the predominately white culture and associated cultural values on predominately white college campuses (Peralta, 2005), perceived pressure to negate stereotypes that assume heavier alcohol and substance use among racial/ethnic minorities (Peralta & Steele, 2009), fear of receiving harsher punishments for alcohol infractions (Peralta & Steele, 2009), and fear of experiencing racism when drinking among inebriated White peers (Peralta & Steele, 2009).

While researchers have been able to identify numerous protective factors associated with abstinence, they have avoided examining factors associated with binge or risk drinking among racial and ethnic minority college students. As demonstrated by data from past studies such as the CAS, 39.5% of Hispanic students, 23.1% of Asian students and 15.5% of Black students reported binge drinking in the past 12 months (Wechsler et al., 2000). Even though these numbers are much smaller than the 49.2% of Caucasian students reporting this behavior, the racial and ethnic minority students who drink at this level may be placing themselves at greater
risk for experiencing immediate and delayed health consequences as a result of their drinking. Currently, few risk factors for alcohol use and related problems have been identified among racial/ethnic minority college students. Those that have been studied include the effects of assimilation and acculturation on adherence to cultural values that discourage drinking and drinking to intoxication (Cateano et al., 1998; Hatchett and Holmes, 2004), co-morbid substance use/abuse (Chen, Unger, Boley and Cruz, 2005; Epstein, Botvin & Diaz, 2002), and experiences of discrimination (Martin, Tuch & Roman, 2003). Unfortunately, researchers have not yet incorporated this information into clinical practice when screening and treating those in this subgroup whose drinking puts them at risk for serious harm. Similarly, even with the current knowledge about protective factors, there few, if any interventions that promote these protective factors and encourage racial/ethnic minority college students to continue these behaviors in order to prevent alcohol use and its associated problems later in life. Moreover, from information currently in the literature, it seems as though the research community has assumed that racial and ethnic minority college students are not at risk and should remain unstudied until enough suffer adverse consequences to warrant further inquiry.

Because of an overemphasis on protective factors and rates of abstinence among racial/ethnic minorities, the needs of these students continue to remain unacknowledged and unmet. Additionally researchers have yet to determine when identified protective factors lose their buffering effects or how drinking at the college level may eventually lead to some of the alcohol related health disparities observed among racial/ethnic minorities. More research is needed, not only to identify factors associated with risk drinking among racial/ethnic minority college students, but also to understand how college alcohol use and its associated protective/risk factors fits into a developmental trajectory for alcohol related health disparities. Ultimately, by
studying racial/ethnic minority college students who engage in risk drinking, researchers can identify crucial time points for intervention as well as behaviors to target in these interventions.

**Gender differences in alcohol consumption and AUDs among college students belonging to various racial and ethnic groups.** The previous sections have examined gender and racial differences in alcohol use and related problems among all college students. The following section will review what is known at present about alcohol consumption and problems in college students as a function of gender and racial/ethnic group membership. In particular, patterns and findings unique to such cohorts will be described.

Gender differences in alcohol use, AUD rates and adverse health consequences have been observed within racial/ethnic minority college students. Nearly every study on college drinking behaviors has acknowledge that Caucasian males report the highest rates of alcohol consumption and AUDs when compared to males in other racial and ethnic groups, and when compared to females of all racial and ethnic groups (Hasin et al., 2007; O’Malley & Johnston, 2002; Wechsler et al., 2000; Wechsler et al., 2002). Among data collected from the 1995 NCHRS and the 1999 CAS, Hispanic males appear to be the group with the next highest rate of heavy alcohol use, followed by Caucasian females, Hispanic females, black males and then black females (O’Malley & Johnston, 2002). Similar trends have emerged in additional studies: data from the CORE Alcohol and Drug Study, a national study of drinking behaviors among college students at 10 colleges, demonstrates that Caucasian women report higher rates of alcohol consumption than their African American female counterparts (Madison-Colmore, Ford, Cooke & Ellis, 2003).

Because of the limited information on risk drinking among racial and ethnic minority college students, there is little knowledge as to why these gender differences exist. In one study,
Corbin and colleagues (2008) were able to identify an indirect relationship between perceptions of family and peer norms about drinking, personal drinking values and reported alcohol use. More specifically, these findings suggest that cultural values may discourage some racial and ethnic college women from engaging in risk drinking. However, there is a paucity of research that delineates the prevalence of gender differences in alcohol consumption among all racial groups, but especially among Latinos, Asians, Native Americans, and bi/multi-racial individuals. Without such data, it is impossible to observe if there are gender differences in alcohol consumption among these groups, and far greater to assess the prevention and intervention needs of college women from these ethnic and racial backgrounds. Unfortunately, racial and ethnic women represent a subgroup of the population that is highly susceptible to experiencing adverse alcohol related consequences. Much research is needed to identify racial and ethnic women in need of early intervention or to encourage some of the protective behaviors that they engage in so as to prevent future problems.

In summary, all of the previous sections have focused on epidemiological information regarding alcohol use and alcohol related problems in the general population and then among college students. In both populations, women consume less alcohol, but are more vulnerable to the toxic effects of alcohol. Despite this vulnerability, women, especially college women, continue to engage in drinking that leads to adverse health outcomes. Similarly, fewer percentages of racial and ethnic minority groups report alcohol consumption, but many who do often go on to report disproportionately higher rates of alcohol related problems. Overall, both women and racial/ethnic minorities represent populations that are much more vulnerable to experiencing adverse consequences from alcohol use even though fewer individuals from these two groups report drinking as much as men or Caucasians. As such, racial/ethnic women,
especially those enrolled in colleges and universities, are a group at greater risk of experiencing adverse consequences if they choose to drink. For that reason, more research is necessary to identify risk factors correlated with alcohol use and alcohol related problems. By identifying these risk factors, researchers and clinicians are then able to better recognize individuals who may benefit from early intervention to prevent the occurrence of these problems.

The following sections will focus on specific correlates and factors associated with risk drinking among college women. Included in the subsequent sections is a review of domains that the literature has identified as having potential importance in the development of heavy drinking and alcohol related problems. The purpose of examining these specific predictors is to identify women at risk for developing alcohol related problems so that early intervention and prevention can be offered.

**Predictors of Risk Drinking Among College Women**

Risk drinking is defined as women consuming three or more drinks daily or seven or more drinks weekly (NIAAA, 2010). To-date, little research has been conducted examining predictors of risk drinking in college settings. Instead, research on the drinking patterns of college students has focused primarily on identifying predictors of binge drinking, heavy alcohol consumption, alcohol related problems, problem drinking and AUDs. Given the dearth of information about predictors of high risk drinking among college samples, this study will evaluate correlates of this behavior in hopes of adding to the literature on risk drinking among college students. Further, the present review will be restricted to risk factors that the author has found to be pertinent to the population of interest, based on the literature. These include family history of alcohol abuse or dependence, age of first alcohol use, depression, tobacco use and premenstrual symptomatology.
Family history of alcohol abuse or dependence. Numerous researchers have identified a relationship between a family history of AUDs and eventual onset of an AUD (Cloninger, Sigvardson, Gilligan, von Knorring, Reich & Bohman, 1988; Cotton, 1979; Dawson, Harford & Grant, 1992; Nurnberger et al., 2004). To gather this information, researchers often use four methodologies: family history, family study, twin study or adoption study. In a family history study, researchers interview one family member about the alcohol use/abuse of their first and second degree biological relatives. The family study method involves interviewing multiple family members about the drinking behaviors of biological first and second-degree relatives (Andreasen Endicott, Spitzer & Winokur, 1977). While both of these methodologies are useful, methodological issues abound and include: under or over-reporting of problems in family members, social desirability bias and lack of contact with specific family members. Some of these threats to validity can be minimized by using standardized measures such as the Family History-Research-Diagnostic Criteria (FH-RDC; Andreasen, Endicott, Spitzer & Winokur, 1977), the Family Alcohol and Drug Survey (FADS; Pickens, Svikis, McGue, Lykken, Heston & Clayton, 1991), the Semi-Structured Assessment for the Genetics of Alcohol (SSAGA; Helzer & Robbins, 1988), and the Family History Module of the Addiction Severity Index (ASI: McClellan et al., 1992).

Findings from studies that have used family history measures offer evidence supporting the relationship between a family history of AUDs and experiencing symptoms of an AUD at some point during an individual’s lifespan (Cotton, 1979; Dawson, Harford & Grant, 1992; Grant, 1998). Respondents from the National Longitudinal Alcohol Epidemiological Study (NLAES) who reported having a family member with alcoholism or problem drinking were more likely to meet DSM-IV criteria for lifetime alcohol dependence (Grant, 1998). Using a mixed
sample of individuals from both community and alcohol treatment settings, Curran and colleagues (1998) found that family history of alcohol abuse/dependence was a stronger predictor of alcohol use problems among women than socioeconomic status (Curran et al., 1998). Among college women, a similar trend has been identified. LaBrie, Kenney, Lac and Migliuri (2009) assessed first semester freshmen women (N=72) to determine whether they had a family history of alcohol abuse/dependence. They then collected prospective data on participant alcohol use over a five-week period of time. Women who reported a family history of alcohol abuse/dependence consumed significantly more alcohol over the course of 5 weeks than women without a family history of alcohol abuse/dependence.

Although family history studies of alcohol use provide researchers with information about potential genetic risk factors of AUD development, they do not account for the interaction between genetics and environment (Ball and Murray, 1994). While genetics may account for differences in alcohol response, sensitivity to alcohol, tolerance, drug metabolism and emotional functioning that may lead an individual to engage in drinking for self-medicating purposes (Eng, Shuckit & Smith, 2005; Khantzian, 1985; Tsuang et al., 1996), environmental factors such as access to alcohol, norms and values about alcohol consumption and stressful life events that may exacerbate predisposing factors are also important to consider (Weiss, Griffith & Mirin, 1992). To pursue such research, other more complex research strategies are needed. They include twin studies and adoption studies.

**Twin studies.** Twin studies allow researchers to examine genetic contributions to AUD by comparing monozygotic (MZ) and dizygotic (DZ) twins on their rates of AUD development and alcohol consumption (Goodwin, 1978). Using samples of twins, researchers have been able
to calculate heritability indices after examining concordance rates (e.g. the presence of an AUD in the individual’s twin) for MZ and DZ twins (Fuller & Thompson, 1978).

Studies with samples of male and female twins have found that MZ twins are twice as likely to be at risk for developing an AUD than DZ twins (Pickens et al., 1991; Kendler, Heath, Neale Kessler & Eaves, 1992). Similarly, in a meta-analysis of population-based twin studies, Kendler and colleagues (1992) reported heritability estimates for AUDs that suggested that more than half of the variance in AUDs is due to genetic factors and that genetics are an influential factor in AUD development. Prescott and colleagues (1999) reported that among women, the relationship between a family history of AUD and incidence of an AUD is more salient among a sample of female twins obtained from the general population than female twins sampled from a treatment population. Results from this large, population based study suggest that among women, genetic factors may have a greater influence on the development of AUD than in men. Twin studies restricted to female samples support and replicate these findings.

While these studies suggest strong genetic factors in the development of AUDs among women, they often assume similar environmental factors, but don’t take the interaction of environment and genes into account. To further examine the interaction between these two factors, researchers have used adoption studies.

**Adoption studies.** Adoption studies compare adopted individuals to their biological and adoptive parents to examine genetic and behavioral contributions to target behaviors. Because adoptees and their adoptive parents share environments, similar findings between adopted children and their biological parents are believed to be the result of genetics. As noted by Goodwin, Schulsinger, Hermansen, Guze & Winokur (1973) similarities in behavior (e.g., alcohol use and abuse) that occur between an adoptee and her biological parents are assumed to
be genetically influenced, while similarities observed among adoptees and their adoptive parents are considered to be primarily environmental in nature (Goodwin et al., 1973). Researchers using the adoption study method have found that adoptees with a biological parent who has an AUD were more likely to have an increased rate of AUDs even when raised by an adoptive parent with no history or current diagnosis of AUDs (Bohman, Sigvardsson & Cloninger, 1981; Cadoret & Gath, 1978; Newlin, Miles, van den Bree, Gupman & Pickens, 2000; Yates, Cadoret, Troughton & Stewart, 1996). These data support the conclusion that genetic liability greatly influences AUD development in offspring. However, adoption studies have also supported the role of environmental factors such as family norms and family environment in the role of AUD development (Cadoret et al., 1986; Cloninger et al., 1988). In sum, the results of these family history, twin and adoption studies demonstrate that there is a strong genetic component involved in the development of AUDS, especially among women; however, genetic factors do not always act in isolation, so there is a need for researchers to more explicitly identify environmental mechanisms and how the two interact.

Even though many of the aforementioned studies found strong associations between a family history of alcoholism and future alcohol problems among offspring and descendants, many of these studies were conducted with treatment and community samples. As such, these findings cannot be generalized to college students, so researchers have tried to find similar associations among college students. Current findings both negate and support higher rates of alcohol use and abuse among college students with family histories of AUDs (Engs, 1990; Alterman et al., 1989; Havey & Dodd, 1993; Bogart et al., 1995; Kushner & Sher, 1993; Perkins & Berkowitz, 1991; Pullen, 1994). Possible explanations for these inconsistent findings include variability in family history methodology, recall bias, social desirability bias and small sample
sizes (Baer, 2002). Researchers have also posited that individuals with a family history of alcohol, especially those who are children of alcoholics (COAs) may not attend college, especially if they have already begun to experience clinical impairment due to their alcohol use (Baer, 2002). Because the influence of genetic factors is not as clear among college students as it is with epidemiological and treatment studies, further research is warranted to determine the role of genetic factors in problematic drinking among college women.

Overall, numerous clinic and community based studies have demonstrated a relationship between family history of AUDs and AUDs or alcohol problems among offspring and descendants. These studies provide evidence for genetic influence in the development of AUDs, but only among treatment or community samples. Because this relationship remains unclear among college women, additional research is warranted to understand why a phenomenon that occurs in the general population does not appear to be as salient in a college population.

**Age of First Drink.** Initiation of alcohol use is one of the most commonly studied predictors of binge drinking and AUD development among all age groups. Current literature utilizing longitudinal studies has found that earlier initiation of alcohol use, especially use before the age of 14, is predictive of future alcohol problems (Hawkins, Catalano & Miller, 1992). Data from the NLAES demonstrated that nearly 40% of individuals who reported taking their first drink before the age of 14 were four times more likely to report a diagnosis of alcohol dependence than those who reported taking their first drink in their 20’s (Grant and Dawson, 1997). Individuals who consume their first drink during adolescence are also more likely to consume increasing amounts of alcohol as they get older (Barnes, Welte & Dintcheff, 1992). Among women, alcohol initiation that occurs sometime within the 15-17 age range was most
highly associated with an increased risk of developing alcohol dependence (Dawson, Goldstein, Chou, Ruan & Grant, 2008). 

Early experimentation with alcohol is often predictive of future alcohol problems, while delayed onset of regular drinking decreases the risk of alcohol dependence and abuse (Grant, Stinson & Harford, 2001). Both age of first drink and age of drinking onset (i.e. regular drinking) represent important predictors for researchers interested in identifying critical time points in the development of AUDs among college women.

Co-morbid Psychopathology. Both epidemiological and clinical studies have consistently supported a connection between AUDs and psychological disorders such as depression, anxiety, eating disorders and co-morbid substance use disorders among women (Carpenter & Hasin, 1999; Conway et al., 2006; Harrell, Slane & Krump, 2009; Kessler, Crum, Warner, Nelson, Schulenberg & Anthony, 1997; Kushner, Abrams, Borchardt, 2000; Sonne, Back, Zuniga, Randall & Brady, 2003). Women with symptoms or a diagnosis of a psychological illness or who report negative affect are often at higher risk of increased alcohol and substance use than women without symptoms/diagnosis of a psychological disorder (Conway, Compton & Stinson, 2006; Smith, 2009). As such, psychopathology of any type, is a risk factor frequently studied in the AUD research. The current review will only focus on depression and nicotine use as risk factors of high risk drinking among college women, as these are of interest to the manuscript author.

Depression and alcohol use. Research on the relationship between negative affect and AUDs has typically focused on studying depression. Findings have consistently identified two different patterns: 1) depression that precedes drinking, and 2) drinking that precedes depression. Schuckit (2000), defines these two types as primary depression, or depression that occurs
independently of alcohol use, and alcohol induced/secondary depression, respectively. According to Schuckit (2000), secondary depression is often the result of pharmacological changes that result from extensive alcohol use and withdrawal. By contrast, primary depression has no direct casual relationship to the onset of the AUD even though it is strongly associated with increased drinking and AUD onset (Schuckit, 2000). Gender differences in depressive symptomatology have also been identified. More specifically, men are more likely to experience alcohol-induced/secondary depression, while women are more likely to have pre-morbid depression or depressive episodes that precede AUDs (Brown, Inaba, Gillin, Schuckit, Stewart & Irwin, 1995; Davidson, 1995; Schuckit, Tipp, Bergman, Reich, Hesselbrock & Smith, 1997). Both treatment and community samples have found that women with current or a prior history of depression were more likely to consume heavier amounts of alcohol and report AUD symptoms than women with no past or current diagnoses of depression (Dixit & Crum, 2000; Kessler et al., 1997; Sanniball & Hall, 2001).

While the causal pathways from depression to alcohol use and eventually, AUD, are unclear, researchers speculate that women may use alcohol as a coping strategy to self-medicate (Cooper, Frone, Russell & Mudar, 1995; Kuntsche, Knibble, Gmel & Engels, 2006) or to aid in emotional regulation (Kassel, Jackson & Unrod, 2000). Research participants often report using alcohol as a way to escape emotional problems and stressful situations (Carey & Correia, 1997). Among college women, research consistently purports that depression and depressive symptoms are risk factors for heavy drinking and alcohol-related problems (Harrell & Karim, 2008; Nolen-Hoeksema, 2004; Harrell, Slane & Klump, 2009). Studies utilizing both cross-sectional and longitudinal designs have been able to identify a progression from pre-morbid depressive symptoms to heavier drinking and increased alcohol-related problems among college women.

While causal mechanisms for this relationship remain unclear, current research suggests that women with a history of depressive symptomatology may engage in heavier drinking as a way of regulating and coping with negative affect (Armeli, Conner & Tennen, 2008; Goldsmith, Tran, Smith & Howe, 2009), coping with stress (Carey & Correia, 1997) and as a way to increase social support and engagement with peers (Gleason, 1994; LaBrie, Huchting, Lac, Tawalbeh, Thompson & Larimer, 2009). Given the strength of these associations and the numerous data supporting the relationship between pre-existing symptoms of depression and subsequent alcohol related problems, more research is warranted to identify causal mechanisms between depression symptoms and increased drinking among college women.

**Nicotine and alcohol use.** Research has consistently identified a relationship between cigarette smoking and alcohol consumption. More specifically, when compared to non-smokers, smokers are more likely to report drinking alcohol (Istvan & Matarazzo, 1984; Zacny, 1990), and smokers are more likely to smoke more while drinking (Mello, Mendelson & Palmieri, 1987). Smoking among college students is of particular concern as many students enter college and initiate smoking sometime during their first year in college. Data from the 1995 National College Health Risk Behavior Survey, suggests that 70% of student respondents had tried smoking and 42% considered themselves as current smokers (Everett, Husten, Kann, Warren, Sharp & Crossett, 1999). Recent epidemiological data suggests that smoking among college students, especially college women, has greatly decreased since 1999, when rates of smoking among college students reached peak levels (Johnston, O’Malley, Bachman & Schulenberg, 2009). More recently, the 2008 Monitoring the Future report demonstrates that close to 18% of college
students report smoking, and that more male students report smoking than female students (Johnston et al., 2009).

While the numbers of college students who smoke are significantly lower than the number of their same age peers not enrolled in college who smoke (Johnston et al., 2009), college students appear to initiate smoking while in college instead of arriving to college with smoking habits. After following a sample of first year students throughout college, Wetter and colleagues (2004) found that 11.5% of students who reported never smoking prior to entering college reported themselves as social smokers four years later. Costa, Jessor and Turbin (2007) reported that 22% of freshmen who reported never smoking were reporting regular smoking behaviors by the spring of their sophomore year. These findings demonstrate that college students are susceptible to initiating smoking behaviors while in college and continue to maintain these behaviors beyond their college years.

Research in both adolescent and young adult smokers describes an interchangeable relationship between smoking and alcohol consumption. Breslau’s (1995), study of 1000 young adult smokers, found a strong relationship between nicotine dependence and an increased likelihood of developing alcohol dependence. Similarly, findings from a recent study revealed that individuals with early onset of smoking (i.e. before age 14) were two to three times more likely to become heavy drinkers as adults (Dierker, Lloyd-Richardson, Stolar, Flay, Tiffany, Collins et al., 2006).

Several studies have also identified that among females, smoking is often a risk factor for high risk drinking and alcohol related problems. Mackey, McKinney and Tavakoli (2008) revealed that female college students who categorized themselves as “smokers” were more likely to engage in high-risk behaviors such as sex with multiple partners, binge drinking, and illicit
drug use. Research also suggests that among women who smoke and drink, an increase in the use of one substance tends to increase use of the other (Harrison and McKee, 2008). McKee and colleagues (2004) found that female college student “smokers” also reported that they most often smoked while drinking, and while drinking they smoked more cigarettes.

Explanations for co-occurring use among female college students vary greatly. Across the literature is a consistent characterization of college smokers as “social” smokers versus daily or regular smokers. Levinson and colleagues (2007) examined how college students characterize their smoking status and found that the majority of the students they interviewed characterized their smoking behavior as social in nature because it occurred in bars, nightclubs or at parties and with others (Moran, Wechsler, Rigotti, 2004; Philpot et al., 1999). Ironically, Levinson and colleagues (2007) reported that most social smokers did not identify themselves as “smokers”. Levinson and colleagues’ findings demonstrate a prominent trend among college women who smoke and drink: both behaviors increase social engagement and facilitate social bonding. Additionally, as noted in Nichter and colleagues’ (2010) study, participants spoke about the perceived benefits of smoking while drinking, namely, that smoking allowed partying to continue longer because they stayed awake longer. Ultimately, smoking has been shown to increase the amount of alcohol consumed by college women. As such, it is an important risk factor that needs to be studied in order to understand who is at risk for engaging in high risk drinking.

Premenstrual symptomatology. Researchers focused on identifying risk factors for increased alcohol use among women, have examined changes in a woman’s menstrual cycle and how these fluctuations may affect her drinking patterns. Findings suggest that many women experience affective and somatic symptoms during late luteal phase or premenstrum (Rome,
and that they may increase their drinking during this phase of their menstrual cycle, possibly in response to these symptoms (Allen, 1996; Leonhardt, Benedict, Svikis, McCaul & Kornstein, 2001; Podolosky, 1963). The following section will review the relationship between premenstrual symptomatology and increased alcohol use among women in order to highlight premenstrual symptoms as potential risk factors for risk drinking.

During the late luteal phase or the one to two week period prior to menstruation, many women report experiencing somatic and affective symptoms (Rome, 1992). According to the International Classification of Disease, Tenth Edition (ICD-10), these symptoms comprise a syndrome known as Premenstrual Syndrome or PMS (World Health Organization, 1996). The term premenstrual syndrome was coined by Greene and Dalton in 1931 (Kessel, 2000) and has since been used to describe the experience of the following symptoms in the week or two weeks prior to menstruation: fatigue, irritability, mood lability, depression, food cravings, breast tenderness, bloating, tension and anxiety (Mortola, Girton, Beck & Yen, 1990). These symptoms emerge during the late luteal phase of a woman’s menstrual cycle, peak and then disappear (Mortola et al., 1990).

According to the ICD-10 diagnostic criteria, a woman only needs to report experiencing at least one of the above symptoms during multiple menstrual cycles in order to meet criteria for PMS (Kessel, 2000). However, the diagnostic criteria for PMS as put forth by the ICD-10 have been criticized as they rely on subjective report and have no objective measure that health care providers can use to validate and quantify the occurrence of these symptoms (Braverman, 2007). However, nearly 90% of women currently menstruating report experiencing at least one of the above symptoms during the premenstrual period (Braverman, 2007). More stringent criteria for PMS that has been published by the American College of Obstetricians and Gynecologists
(2000) requires women to report at least one affective symptom (e.g. depression, anxiety, withdrawal, irritability, angry outbursts) and one somatic symptom (e.g. breast tenderness, bloating, headache, swelling) five days prior to the onset of their menstrual period, and that a pattern of these symptoms be observed for at least 3 consecutive menstrual cycles.

A more severe premenstrual disorder, with diagnostic criteria put forth by the American Psychiatric Association (APA) is Premenstrual Dysphoric Disorder, also known as PMDD. While similar to PMS, PMDD is a disorder characterized by severe affective symptoms that cause clinically significant impairment (Braverman, 2007). Close to 10% of women of childbearing age report a diagnosis of PMDD (Braverman, 2007). Women’s health researchers have identified a relationship between PMS/PMDD symptoms and alcohol use. More specifically research suggests that women who experience premenstrual symptoms that interfere with daily living may be more likely to increase their alcohol consumption premenstrually (Allen, 1996; Leonhardt et al., 2001).

Some of the earliest research in the area of PMS and alcohol use focused primarily on samples of women diagnosed with alcoholism (Perry, 2004). Wall (1937) and Lolli (1953) were two of the first researchers to identify a connection between drinking among women who experienced affective and somatic symptoms during the premenstrum. Lolli, and later, Podolosky (1963), conducted a series of case studies of women with alcoholism whose drinking reportedly increased during the premenstrual period. Both researchers posited that women who drank during the premenstrual period did so to alleviate some of the affective and somatic symptoms they were experiencing during the premenstrual period.

Subsequent studies continue to demonstrate that women with premenstrual symptoms reported increased alcohol consumption during the premenstrual period (Allen, 1996; Leonhardt
et al., 2001). While the majority of these studies used samples of women with AUDs, studies conducted with samples of women without AUDs found similar results among women reporting severe PMS symptoms (Chuong & Burgos, 1995; Rossignol & Bonnlander, 1991).

The above studies emphasize a significant relationship between PMS symptoms and increased alcohol consumption among women. Their findings demonstrate that PMS symptom severity is a risk factor for increased alcohol consumption that may increase high risk drinking among women (Halliday et al., 1996; Allen, 1996). There is currently little research on the causal relationship between PMS symptoms and increased drinking during the premenstrual phase. However, what research is available suggests that women experiencing severe PMS symptoms and PMDD may experience increased psychosocial stress and a decreased quality of life (Lustyk, Widman, Paschane & Ecker, 2004), which may lead them to increase their drinking as way to cope. More research is warranted to clarify the relationship between PMS symptoms and increased drinking during the premenstrual phase, as well as to identify causal factors for this relationship.

In sum, research on alcohol use and alcohol use problems at the college level has found several correlates that indicate increased risk for alcohol related problems. Among these, robust associations have been found for age of alcohol initiation, depression and nicotine use (Armeli, Conner & Tennen, 2008; Goldsmith, Tran, Smith & Howe, 2009; Hawkins et al., 1992; Istvan & Matarazzo, 1984; Zacny, 1990). Other correlates such as family history of alcohol use and premenstrual symptoms appear to have inconsistent findings and require more research in order to fully flesh out the relationship between these predictors and risk drinking.
Statement of the Problem

Rationale

High risk drinking, defined in women as consuming more than three drinks daily or more than seven drinks weekly (NIAAA, 2004), is a behavior that occurs frequently on college campuses. In 2008, 34% of college women reported engaging in high risk drinking (Johnston et al., 2009). Extant data demonstrate that high risk drinking has been associated with the following detrimental consequences for college women: incidence of AUDs (Hasin et al., 2007), co-morbid substance use and abuse (Bradley et al., 1998), risky sexual behaviors (Bradley et al., 1998), unwanted sexual advances and sexual assault (Abbey, 2002), and accidental death and injury (Hingson et al., 2005).

A growing body of research suggests that women may be particularly vulnerable to the negative effects of high risk drinking because of physiological differences in alcohol metabolism that may lead to increased levels of impairment (Julien, 2008) and greater physiological and psychological harm as result. These physiological and psychosocial sequelae have prompted clinicians to focus on prevention efforts that reduce high risk drinking and its consequences. To date, research has identified several correlates of women who engage in high risk drinking, and subsequent researchers have translated these predictors into screening measures developed to assist clinicians with identifying women for intervention purposes. Since resources are often limited, researchers have emphasized the importance of identifying those at the greatest levels of risk. Subsequent studies have focused on the development of reliable and valid screening tools that economically and efficiently identify women at greatest risk.
Historically, the majority of these studies focused on predominately Caucasian samples of women. More recently, greater focus on minority women suggest that non-White minority women may be at an even greater risk for adverse consequences due to heavy drinking. Identified sequelae among minority women include higher mortality rates from liver and cardiovascular disease (Stinson, 2001; Yoon et al., 2001; Russo et al., 2004), higher prevalence of fetal alcohol spectrum disorders (Abel, 1995; Russo et al., 2004), increased reports of injuries from intimate partner violence (Cunradi, Caetano, Clark & Schafer, 1999) and untreated alcohol use disorders. Such widespread disparities in combination with an increasing number of non-White minorities in the U.S. population prompted NIAAA and the National Institutes of Health to encourage research on the correlates of alcohol related health disparities (NIH, 1994; NIAAA, 2001; Russo et al., 2004), and eventually led to establishing the National Center on Minority Health and Health Disparities in 2000, which recently became the National Institute on Minority Health and Health Disparities (NCMHD, 2010).

In spite of this impetus, the paucity of research focused on predictors of high risk drinking at the college level among non-White minority women is of concern, especially since researchers have identified this developmental phase as the period of time when individuals from all racial and ethnic groups engage in the highest rates of drinking. Ultimately, the increasing alcohol related health disparities among racial and ethnic minority women demonstrate the need for researchers to identify critical developmental periods when non-White minority women engage in drinking behaviors that increase their risk of developing adverse consequences later in life.

Researchers cite several factors that contribute to this gap in research. These include higher abstinence rates among non-white minority college women and limited access to the non-
white minority college women who drink at a high-risk level. Numerous studies have demonstrated that White students engage in higher rates of drinking than all minority groups, while Hispanic and Latino college students engage in moderate rates of drinking, followed by African American or Black students and then Asian students, who report the least amount of drinking (Dowdall, Coawford & Wechsler, 1998; Keeling, 2000; Wechsler et al., 2000; O’Malley & Johnston, 2002; Cranford, McCabe & Boyd, 2006). Additional research findings show that Black and Asian college students reported the highest rates of abstention from alcohol and that Whites reported the lowest rates of abstention (Wechsler et al., 2000). These findings also demonstrate that non-white minority women are less likely to engage in high risk drinking even when compared to their white female counterparts. Because of these findings and a consistent trend towards lower rates of high risk drinking among college women there has been little incentive for researchers to study high risk drinking and its correlates among this group. However, even though high risk drinking occurs less frequently among racial and ethnic minority college women, the data do reflect the fact that it occurs (Ham & Hope, 2003) and that the delayed consequences of high risk drinking are more severe for non-white minority women (Gavaler, Deal & Rosenblum, 2004).

After justifying the need to study high risk drinking among racial and ethnic minority women, researchers are still challenged by methodological issues. Specifically, researchers often experience difficulty with recruiting a large enough sample of non-white minority women who engage in high risk drinking and must rely on convenience samples, which may over or under sample individuals who engage in this behavior (Burlew, Feaster, Brecht & Hubbard, 2009). They also experience difficulty with assessing both within and between group differences in high risk drinking, which demonstrate similarities, as well as the heterogeneity present among racial
and ethnic minority groups that engage in high risk drinking. Researchers seeking to identify between group differences often succeed in comparing racial and ethnic minority groups to their Caucasian counterparts, however, overreliance on this statistical strategy has undermined the importance of acknowledging intra-racial or intra-ethnic differences in high risk drinking or predictors of high risk drinking among this population. Few researchers have been able to recruit enough participants so that both within group and between group analyses can be completed and meaningfully interpreted.

Researchers interested in studying racial and ethnic differences in high risk drinking also struggle with establishing measurement equivalence, as many, but not all, of the measures have been validated and standardized with predominately Caucasian samples (Burlew et al., 2009). While many of the measures have since been validated in racially and ethnically diverse samples, a lack of measurement equivalence is a commonly cited limitation, especially when the underlying construct that they are attempting to assess may not be culturally relevant or may have another cultural epithet that the measures do not assess (Burlew et al., 2009). Overall, because of these challenges and because of an overarching disconnect between high risk drinking among racial and ethnic minority college women and subsequent alcohol-related health disparities, researchers have neglected this area and many unanswered questions remain.

Aims and Hypotheses

The present study used a cross sectional design to examine racial differences in predictors of risk drinking among college women. The present study sought to contribute to the research literature about risk drinking among racial and ethnic minority college women. An additional purpose of the proposed study was to help researchers better understand that identified factors of
risk drinking may vary by race and ethnic group membership, and that addressing these differences may lead to more accurate screening measures and targeted interventions.

The specific aims of this study were 1) to identify differences in risk drinking among the racial and ethnic groups represented in an urban sample of college women; 2) to identify predictors of high risk drinking, which was defined as an AUDIT score of greater than or equal to eight; and 3) to determine whether race moderates the relationship between significant predictors and risk drinking.

It was hypothesized that 1) a model of the following psychosocial predictors: daily smoking, premenstrual symptoms, race, age of first drink and family history of alcoholism would significantly predict high risk drinking and that 2) that variables found to significantly predict risk drinking would vary by minority group membership.
Methods

The present study is a secondary analysis of data that was collected as part of a doctoral dissertation funded by an NIAAA F31 pre-doctoral training grant awarded to Bridget Perry, Ph.D. The original study was a cross-sectional study that recruited participants from the fall, spring and summer semesters of the 2003-2004 academic year. Participants for the original study were undergraduate women enrolled in introductory psychology courses at a large urban university. Female students were eligible to participate in this study if they were currently enrolled at the university during study recruitment and if they could consent to study participation. A total of four hundred and eighty one women completed the initial survey battery.

Participants

Participants for the current secondary analysis were selected based on their responses to items that assessed their alcohol use in the 12 months prior to study participation. Participants who denied alcohol use in the previous year (N=121) were deemed ineligible and excluded from further analyses. Thus, the final sample for this secondary analysis included data from N=360 women who reported consuming alcohol in the 12 months prior to study participation.

Procedure

Recruitment. For the original study, the investigator posted a description of the study on a website managed by the university’s psychology department. All students enrolled in an introductory psychology course received a hyperlink from their instructors to an internet database of research studies available to complete for academic research credits in the
psychology department. By clicking on the hyperlink, women could read the description of the study, which was advertised as study of stress and coping behaviors in college women. The description advised interested women to contact the researcher to sign up for one of several pre-selected dates to complete a series of questionnaires. The description notified participants that they would be completing the assessments during a 45-minute group time so that the PI could be available to answer questions. Interested women who met the inclusion criteria contacted the researcher and signed up for a time to complete the questionnaires.

**Data collection.** Participants came to an identified location on campus where they and the PI met as a group. On average, there were 10-12 participants per group session, as well as the PI and one research assistant. Upon arrival, participants were given a consent form that described the study, what would be required of the participants, their rights as research participants, risks, benefits of the study and contact information for the investigator and her mentor, Dace Svikis, Ph.D. The handout informed participants that by completing and turning in the questionnaires in the assessment battery, they were consenting to study participation, and that upon study completion, they would receive class research credit for their participation.

After reviewing the handout, participants were then given a packet of self-report questionnaires, which they completed over the course of an hour during the group session. After completing the assessments, participants turned in the packet to the PI or research assistant, who briefly reviewed their answers to ensure that the participants had not inadvertently skipped items or sections of the battery. Participants were awarded one undergraduate research credit for participating in the study.
Measures

The full assessment battery administered to study participants included the following self-report measures, which are listed in the order that they were administered: Smoking Questionnaire that included items from the Fagerström Test for Nicotine Dependence (FTND; Heatherton, Kozlowski, Frecker & Fagerstrom, 1991) and the Smoking CAGE (Ewing, 1984); Perceived Stress Scale (PSS; Cohen, Kamarck & Mermelstein, 1983); Alcohol Use Disorders Identification Test (AUDIT; Babor, De La Fuente, Saunders & Grant, 1989); Brief COPE (Carver, Weintraub & Scheier, 1989); Shortened Premenstrual Assessment Form (SPAF; Allen, McBride & Pirie, 1991); General Health Information measure created by the investigator; (GHI; Perry, 2004); Reasons for Drinking scale (RFD; Cronin, 1997); Health Survey (Perry, 2004); Positive and Negative Affect Scale (PANAS; Watson, Clark & Tellegen, 1988); Complaints Checklist (CC; Center on Alcoholism, Substance Abuse and Addictions, 1995); Family History of Alcohol and Drug Abuse Module of the Addiction Severity Index (ASI; McLellan, Kushner, Metzger, Peters, Smith, Grissom, et al., 1992); Eating Health Questionnaire (EHQ; Coker & Rogers, 1990); Short Form Perceived Social Support (SFPSS; Rice & Longabaugh, 1996) and Demographics questionnaire (Perry, 2004).

Measures in the assessment battery were selected to either test the PI’s hypotheses or to disguise the true purpose of the study, which was to identify relationships between alcohol use, PMS symptom severity and family history of alcoholism. Because of the sensitive nature of the PI’s original research questions (e.g. the relationship between PMS symptoms, alcohol use and a family history of alcoholism), additional measures were included to reduce participant reactivity to the constructs of interest.
The present study examined a subset of these measures. Specific domains were selected after reviewing findings from studies of alcohol use and problems in college women, as well as areas that were not assessed in the current literature. Overall, many of these studies reported correlations between high risk drinking and a number of demographic and psychosocial variables. Specifically, college women who engaged in high risk alcohol use were more likely to: report a family history of alcohol problems; smoke cigarettes (use tobacco); report symptoms of PMS; initiate drinking at a younger age; report negative affect and belong to a Caucasian ethnic group. However few previous studies how race moderates this relationship.

**Measures Used to Assess Independent Variables**

For the present study, independent variables were selected from the following domains:
- a) tobacco use;
- b) PMS symptomatology;
- c) age of alcohol initiation;
- d) negative affect;
- e) family history of alcohol problems; and
- f) race/ethnic group membership. The assessment battery for the original study contained several measures that assessed these respective domains. However, in order to narrow the scope of the present project, specific measures were chosen using a systematic set of procedures. Descriptions of the measures and subsequent variables assessed in the present secondary analysis are summarized below and presented in the order that they were administered to the participants.

**Smoking Questionnaire.** The smoking questionnaire was constructed by the P.I. (i.e. Bridget Perry) and consisted of a 42-item measure that assessed past and current tobacco use. Participants identified their current smoking pattern (e.g. non-smoker/never smoker, past/no-longer smoker, social smoker [e.g. smoking when drinking or smoking during social occasions, but not daily] or daily smoker), lifetime daily cigarette use (yes or no) and information about the smoking behaviors of their friends and significant others. Women who reported smoking at
some point in their lifespan, were then asked to complete items from the FTND (Heatherton et al., 1991), items from the Smoking CAGE (Ewing, 1984) and questions about attempts at smoking cessation. Women who did not report any lifetime tobacco use skipped additional items and proceeded to the next questionnaire in the assessment packet.

The FTND (Heatherton et al., 1991) is a six item self-report measure of nicotine dependence that is a revised version of the Fagerström Tolerance Questionnaire (FTQ; Fagerstrom & Schneider, 1989). Total scores range from 0-10 with scores equal to or greater than six indicating that an individual may be experiencing physiological symptoms of nicotine dependence as described in the DSM-IV. A factor analysis of the measure demonstrated that two items contributed a significant portion of the variance on each of the two factors identified (Richardson & Ratner, 2005). Item number one, time to the first cigarette of the day (TTF, e.g. “How soon after you wake up do you smoke your first cigarette?”) contributed significantly to both factor one and factor two, while item number four, average daily consumption of cigarettes (CPD, e.g. “How many cigarettes a day do you smoke?”) contributed significantly to factor two (Radzius, Gallo, Epstein, Gorelick, Cadet, Uhl & Moolchan, 2003). Response choices for TTF consist of four categories, each with a descending point value: within five minutes, 6-30 minutes, 31-60 minutes and after 60 minutes. Response choices for CPD consist of four categories, each with a descending point value: 10 or less, 11-20, 21-30 and 31 or more.

The CAGE (Ewing, 1984) is a four-item measure originally developed to identify persons at risk for alcohol problems. A modified version of this measure, known as the Smoking CAGE, was created to screen for nicotine dependence (Lairson, Harrist, Martin, Ramby, Rustin, Swint et al., 1992; Mallin, 2002). The Smoking CAGE was included in the Smoking Questionnaire included in the original battery of assessments that participants completed. Items that assessed
whether an individual felt the need to cut-down on their smoking, felt annoyed by friends and relatives’ criticism of their smoking, experienced guilt for the smoking and smoke first thing in the morning after waking (Ewing, 1984).

**Shortened Premenstrual Assessment Form (SPAF).** The SPAF (Allen, 1991) is an abbreviated version of the Premenstrual Assessment Form (PAF). The SPAF consists of 20 items that retrospectively assesses premenstrual symptoms experienced by female respondents. Unlike other measures that assess the presence of symptoms that comprise Premenstrual Syndrome (PMS) or Premenstrual Dysphoric Disorder (PMDD), the SPAF assesses the amount of change that an individual experiences in symptom severity. Each item lists a symptom (i.e. feeling sad or blue, breast tenderness or swelling) and asks respondents to report the amount of change that they experience in that symptom during the week before the onset of menses (Perry, 2004). Participants respond by choosing an option from one of six answer choices (0=no change/ symptom not present to 6=extreme change) for each symptom.

The SPAF demonstrates high reliability, internal consistency and validity (Allen, 1991). A factor analysis of the items revealed a three-factor structure consisting of three subscales: affective symptoms, water retention and pain. Two subscales, water retention and pain, have been combined to form a larger subscale of somatic symptoms.

**General Health Questionnaire.** The General Health Questionnaire (Perry, 2004) is a 45 item self report assessment that measured a participant’s health and wellbeing within 30 days of beginning the study. Participants answered items that measured several domains including: a) physical symptoms experienced in the past four weeks (e.g. breathing problems, nausea, insomnia, headaches, back pain, digestive problems and allergies); b) estimated hours of sleep each night and restfulness of sleep; c) healthy eating behaviors (e.g. number of meals eaten daily,
daily water consumption, taking a multi-vitamin); d) caffeine consumption; e) menstrual cycle (e.g. onset of menses, date of last period, frequency of periods); f) past pregnancies and current/past contraceptive use; and g) alcohol use (e.g. weekend and weekday quantity/frequency measures over the course of the past 12 months and within the 30 days prior to completing the assessment and age of self-initiated alcohol use). The items on alcohol use were modeled after items used in the NLAES interview instrument (Grant, Peterson, Dawson & Chou, 1994). Participants responded to the above items by selecting from specific response categories or by writing in their answers (e.g. number of hours slept nightly, number of menstrual periods annually, age of alcohol initiation and the number of drinks consumed).

**Family History Module of Addiction Severity Index (ASI).** The Family History Module of the ASI (McLellan et al., 1980; 1991) focuses on biological first-degree (i.e. parents and siblings) as well as second-degree (i.e. biological aunts, uncles and grandparents) relatives. For each family member (or category of family members), the interviewer asks the participant if the relative(s) had “a significant drinking or alcohol problem-one that did or should have led to treatment”. Similar questions were asked for the other drugs and for psychiatric/mental health disorders. The full module queried participants about maternal and paternal grandparents, maternal and paternal aunts and uncles, mother, father and siblings, in addition to the participant herself. For the present study, only maternal, paternal and parental alcohol use/problems were examined. For each parent, potential responses included: yes (relative met criteria); no (relative did not meet the criteria) or don’t know (participant had insufficient information to categorize the relative).

Previous research has used the family history module of the ASI to categorize individuals’ family history of alcohol use (Pickens, Preston, Miles, Gupman, Johnson, Newlin et
al., 2001). While the ASI has been studied extensively and found to have high reliability and validity (McLellan, Luborsky, Cacciola, Griffith, Evans, Barr et al., 1985) much less research has focused on the Family History module. Two studies compared rates of maternal, paternal and parental alcohol problems in biological parents using the ASI Family History module as compared to Family Alcohol and Drug Survey (FADS; Pickens et al., 1991). The FADS was developed for use in a twin/family study of alcoholism (Pickens et al., 1991). It is a semi-structured interview that uses Family History-Research Diagnostic Criteria (FH-RDC; Andreasen, Rice, Endicott, Reich & Corynell, 1986) to assign diagnoses of Alcohol and Other Drug Abuse in first-and-second-degree relatives. Two previous studies found comparable levels of specificity and sensitivity (Pickens et al., 1991; Smith, 2009). They concluded that the ASI parental history module provided a reliable and valid method for collecting parental alcohol abuse and problem data.

Positive and Negative Affect Schedule (PANAS). The PANAS (Watson, Clark & Tellegen, 1988) is a 20 item self-report measure that assesses affective mood states as well as trait emotionality. The PANAS asks about a respondent’s experiences one week prior to completing the assessment. Each item lists an emotion (e.g. interested, ashamed) and asks participants to report the extent to which they have felt that emotion in the past week using a 5-pt rating scale (from 1=very slightly or not at all to 5=extremely). Participants use a pen or pencil to complete the measure and respond to all items. The PANAS consists of two subscales: a positive affective (PA) state subscale and a negative affect state (NA) subscale. High scores on the PA subscale indicate that an individual experiences elevated energy and a high level of engagement and pleasure. High scores on the NA scale indicate low energy, disengagement and a tendency to experience mood states associated with feelings of anger and sadness. The
PANAS demonstrates high internal consistency and reliability (Watson, Clark & Tellegen, 1988). The NA subscale scores were used in the current study as a measure of negative affective and emotional states.

**Demographics Questionnaire.** The demographics questionnaire included in the battery is a seven item self-report measure. Items assess a respondent’s age, marital status, race/ethnicity, year in school, employment status, current living situation, current smoking status, past smoking status, age of first alcoholic drink and Greek organization membership. Participants a) wrote in their age; b) chose their marital status from one of four categories (e.g. single/never married, married, divorced/separated or widowed); c) selected their race or ethnicity from five categories (e.g. African-American, Asian-American, Hispanic, Other or White-American); d) chose their year in school from one of four categories (e.g. freshman, sophomore, junior or senior); e) selected their employment status from one of three categories (e.g. full-time, part-time or unemployed); f) selected yes or no to indicate Greek affiliation; and g) selected one of six categories to describe their current living situation (e.g. on campus alone, on campus with a roommate, off campus alone, off campus with friends, off campus with parents or off campus with other relatives).

**Independent Variable Selection Process**

Potential independent variables were selected by reviewing the items that comprised the measures for each domain. Table 1 provides a final list of the selected measures and corresponding items that were identified as independent and dependent measures.

Several items that comprised the Smoking Questionnaire measured tobacco use. Specific items included current smoker status and the following measures of nicotine dependence from the FTND: daily cigarette consumption (CPD) and time to first cigarette (TTF). Prior to
selecting the final items, all of the participants’ responses to these measures of tobacco use were reviewed. Because 48.1% of the final sample reported that they had never smoked in their lifetime, and did not complete the remaining questions in the Smoking Questionnaire, an item that assessed lifetime history of daily smoking was included as a measure of tobacco use in further analyses.

The SPAF assessed the Premenstrual Symptom domain. Responses on the SPAF generate three scores: a total score, an affective PMS symptom score and a somatic PMS symptom score. The somatic and affective subscale scores were selected as measures of PMS symptoms.

The General Health Information Questionnaire included several items that assessed current and past alcohol use. For the present study, the number of weekdays in the past month the participant drank alcohol, number of weekend days in the past month the participant drank alcohol, average number of drinks consumed during the weekend and weekdays, and age (years) at first self-initiated alcohol use. Age at first use of alcohol use was included in the pool of the psychosocial variables for the present study, while quantity and frequency measures (for weekend and weekday drinking) of alcohol use were used to describe study participants.

The PANAS was the only measure that assessed a participant’s affective state. Because the construct of interest was negative affective symptoms, the total Negative Affect subscale score was selected as a potential predictor of high risk drinking.

The Family History Module of the ASI provided information about parental alcohol abuse/problems. Responses were tallied to construct a parental history of alcohol problems variable. Specifically, each participant was categorized as either: Parental History positive (PH+) if they reported at least one biological parent with an alcohol problem or Parental History
negative (PH-) if neither biological parent had an alcohol problem. Previous research on alcohol use among college students has used these criteria to summarize parental alcohol abuse (VanVoorst & Quirk, 2003; Jackson & Sher, 2003). Finally, one item from the Demographics Questionnaire assessed self-reported racial and ethnic group membership, and was included as one of the potential predictors.

Summary of Independent Variable Selection Process

The final pool of potential independent variables included a) lifetime history of daily tobacco use (categorical); b) summary scores of premenstrual symptom severity for affective symptoms (continuous; range 0 to 60); c) summary scores of premenstrual symptom severity for somatic symptoms (continuous; 0 to 60); d) self-reported age at first self-initiated alcohol use (continuous; measured in years); e) negative affect summary score (continuous; range from 0 to 50); f) family history of alcoholism (categorical; yes/no to one or more biological parents meeting criteria for Alcohol Abuse); and g) race/ethnic group membership (categorical).

Measures Used to Assess Dependent Variables

Alcohol Use Disorders Identification Test (AUDIT). The AUDIT (Babor, De La Fuente, Saunders & Grant, 1989) is a 10-item self-report questionnaire that assess a respondent’s problematic alcohol use. Items comprise four separate domains: frequency and amount of alcohol consumption, symptoms of physiological dependence, adverse psychological reactions to alcohol consumption, and alcohol related problems. The AUDIT is scored from 0-40 with scores greater than or equal to eight indicating alcohol abuse, dependence and high-risk alcohol use. Several studies have demonstrated that the AUDIT has high sensitivity and specificity in identifying alcohol use and dependence in college students (Conigrave, Hall & Saunders, 1995;
Dependent Variable Selection Process

The AUDIT assessed alcohol use severity in study participants. Two dependent measures were created from AUDIT responses. The first, risk drinking, was created by summing all 10 items on the AUDIT. Scores ranged from 0 to 39, with higher scores indicating riskier drinking. The second, high-risk drinking, was a categorical measure, created from the first variable (risk drinking). Based on published studies with the AUDIT risk scores greater than or equal to eight were defined as positive for high risk drinking, while scores of seven or less were defined as negative for high risk drinking. Participants positive for high risk drinking were assigned a value of one, while those negative for such risk were assigned a value of zero.

Summary of Dependent Variable Selection Process

Two dependent variables of risk drinking were created for the current study: a) a Total AUDIT variable (continuous) and b) a categorical AUDIT variable. The categorical score provided information on the severity of participants’ risk drinking and classified participants as either high-risk drinkers (e.g. AUDIT scores of greater than or equal to eight) or low risk drink (e.g. AUDIT scores of less than or equal to seven).
Table 1

*Variables Used in Statistical Tests of Hypotheses*

<table>
<thead>
<tr>
<th>Predictor/Independent Variable</th>
<th>Measure</th>
</tr>
</thead>
<tbody>
<tr>
<td><strong>Domain</strong></td>
<td></td>
</tr>
<tr>
<td>Tobacco use</td>
<td>Lifetime daily cigarette use</td>
</tr>
<tr>
<td></td>
<td>Smoking Questionnaire</td>
</tr>
<tr>
<td>Premenstrual symptoms</td>
<td>Affective PMS symptom score</td>
</tr>
<tr>
<td></td>
<td>SPAF Affective symptom subscale</td>
</tr>
<tr>
<td></td>
<td>Somatic PMS symptom score</td>
</tr>
<tr>
<td></td>
<td>SPAF Somatic symptom subscale</td>
</tr>
<tr>
<td>Onset of drinking</td>
<td>Age of first self-initiated alcohol use</td>
</tr>
<tr>
<td></td>
<td>General Health Information</td>
</tr>
<tr>
<td>Negative affect</td>
<td>Negative affect score</td>
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<td></td>
<td>Negative Affect (NA) subscale of PANAS</td>
</tr>
<tr>
<td>Family history of alcohol problems</td>
<td>Parental alcohol use problem</td>
</tr>
<tr>
<td></td>
<td>Family History Module of ASI</td>
</tr>
<tr>
<td>Racial and ethnic group membership</td>
<td>Racial/ethnic group category</td>
</tr>
<tr>
<td></td>
<td>Demographics Questionnaire</td>
</tr>
<tr>
<td><strong>Outcome/Dependent Variable</strong></td>
<td></td>
</tr>
<tr>
<td><strong>Domain</strong></td>
<td></td>
</tr>
<tr>
<td>Risk drinking</td>
<td>Total AUDIT score (continuous)</td>
</tr>
<tr>
<td></td>
<td>AUDIT</td>
</tr>
<tr>
<td></td>
<td>High/Low Risk AUDIT score (categorical)</td>
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<tr>
<td></td>
<td>AUDIT</td>
</tr>
</tbody>
</table>
Data Analysis Plan

Data in the current study were analyzed using SPSS v. 19.0 for Macintosh (SPSS, Chicago, IL.). Prior to any statistical analyses, frequency distributions of all variables of interest were computed and screened for any missing values. Frequency distributions revealed that 331 cases of data were missing data on at least one of the eight variables. Due to the high prevalence of missing data, the data were reviewed to determine the pattern of missing cases and correlates of the missing data. Categorical variables were created for each of the variables of interest to indicate if missing values existed or not. These categorical variables were then analyzed using a series of chi-square analyses, t-tests and logistic regression analyses to determine participant characteristics potentially associated with missing data.

Two techniques were used to treat cases of missing data. Multiple regression was used to predict values for three of the continuous independent variables: SPAF Somatic score, SPAF Affective score and Negative Affect subscale score from the PANAS (Gelman & Hill, 2007; Wayman, 2003). These predicted values were then imputed in place of the original missing data. Additionally, mean imputation was used for missing cases of age of first-self initiated alcohol use (Gelman & Hill, 2007; Wayman, 2003). Analyses were run twice, once with the missing data excluded and another with the imputed values replacing the missing data.

Standardized z-score values of the continuous variables of interest (e.g. age of alcohol initiation, total AUDIT score, somatic PMS symptom severity score, affective PMS symptom severity score and negative affective symptom score) were reviewed for the presence of univariate outliers (e.g. z-score absolute values greater than 3.29, Tabachnick & Fidell, 2007). The analyses were run twice: once with the outliers and then without (L. Thacker, personal communication, March 9, 2011). If the results did not differ, the outliers remained in the final
sample. Similarly, multivariate outliers were identified by reviewing Mahalanobis distances that exceeded a critical value for an alpha level of \( p<.001 \) (Tabachnick & Fidell, 2007).

Skewness and kurtosis statistics were reviewed to assess for non-normality among continuous variables. Variables that violated the assumption of normality were transformed using either a square root or inverse transformation (Tabachnick & Fidell, 2007). As cited in Perry (2004), continuous variables that were a part of the interaction terms entered into multiple regression analyses, were centered so as to reduce multicollinearity (Cohen, Cohen, West & Aiken, 2003). After exploratory analyses were conducted to assess normality, descriptive analyses were computed to identify and summarize demographic characteristics of the sample. Additionally, descriptive and exploratory analyses were run to assess differences between participants with missing data and those with complete data.

The hypothesized relationship between the independent variables and risk drinking were examined using logistic regression for a categorical dependent measure of risk drinking (e.g. dichotomous AUDIT score) and a standard multiple regression for the continuous dependent measure of risk drinking (e.g. total AUDIT score). Significant predictors from these initial analyses were then created into interaction terms to test the moderating effects of minority status on risk drinking. The second hypothesized relationship between the interaction terms and risk drinking was examined using direct logistic regression and multiple regression analyses.

**Results**

**Data Preparation**

Four hundred and eighty-one women completed the initial assessment battery. Of these, \( N=121 \) women reported no alcohol use in the past 12-months and were excluded from subsequent analyses. Data from the remaining sample of \( N=360 \) participants (74.8% of the initial
sample) were reviewed for missing cases among the seven independent variables (three
categorical and four continuous) and the two dependent variables (one categorical and one
continuous).

Initial review of both hard-copy participant data and the partially-completed computer
database for all assessment measures found overall rates of missing data at time of study
completion were quite low. This was due in large part to careful review by PI and her research
assistant, of each assessment packet prior to participants leaving the testing area. The
researchers would review the data packets to make sure no pages had been missed and that no
items were inadvertently left blank.

More often, however, participants could not answer some of the questions in the
assessment battery. For example, when asked about family history of alcohol use and problems,
some participants had no knowledge about one or more family members. In such cases, a
response could not be coded; these “don’t know” responses were then classified as “missing” in
the computer database. Similarly, a number of participants could not recall the age at which they
first began drinking alcohol. Again, this inability to give an accurate self-report (e.g., age to the
nearest year) resulted in a code of “missing” within the database.

The only exception to this pattern was found for the PANAS questionnaire. In this case,
N=125 of the 360 women who completed the full assessment battery did not have item or scale
scores. This was because raw data were apparently misplaced between the time of original study
completion and conduct of the present study. When all such “missing data” were combined for
review, the frequencies of missing data were summarized in Table 2. Taken together, there was
a total of N=331 cases of missing data points for at least one of the seven independent variables
and two dependent variables.
Due to the large number of missing data, mean imputation and imputation of predictive values generated by multiple regression were used to provide estimates of missing values for the continuous independent variables as well as to preserve the sample size and reduce response bias (Gelman & Hill, 2007; Tabachnick & Fidell, 2007). Using multiple regression, values were predicted for the missing cases of the SPAF Somatic, SPAF Affective and Negative Affect Subscale scores. Mean values were imputed for the missing cases of age of first self-initiated alcohol use. Next, standardized scores were generated for the continuous outcome variable to assess the presence of outliers. Using a critical value of +/- 3.29 (Tabachnick & Fidell, 2007), eight univariate outliers were identified in the AUDIT outcome variable. Because these outliers
<table>
<thead>
<tr>
<th>Type of variable</th>
<th>Variable name</th>
<th>Number of missing cases (% of N=331)</th>
</tr>
</thead>
<tbody>
<tr>
<td>Independent</td>
<td>Daily Smoker (categorical)</td>
<td>57 (17.2%)</td>
</tr>
<tr>
<td></td>
<td>Affective PMS symptom score (continuous)</td>
<td>11 (3.3%)</td>
</tr>
<tr>
<td></td>
<td>Somatic PMS symptom score (continuous)</td>
<td>13 (3.9%)</td>
</tr>
<tr>
<td></td>
<td>Age of first self-initiated alcohol use (continuous)</td>
<td>66 (19.9%)</td>
</tr>
<tr>
<td></td>
<td>Negative affect score (continuous)</td>
<td>125 (37.7%)</td>
</tr>
<tr>
<td></td>
<td>Parental history of alcohol problem (categorical)</td>
<td>50 (15%)</td>
</tr>
<tr>
<td></td>
<td>Race (categorical)</td>
<td>7 (2.1%)</td>
</tr>
<tr>
<td>Dependent</td>
<td>AUDIT (continuous)</td>
<td>13 (3.9%)</td>
</tr>
<tr>
<td></td>
<td>AUDIT (categorical)</td>
<td>13 (3.9%)</td>
</tr>
</tbody>
</table>
were not the result of data entry errors and reflected an expected range of alcohol use scores across a sample of college women, the decision was made to include these cases in the analyses (L. Thacker, personal communication, March 9, 2011). After reviewing Mahalanobis distances, no multivariate outliers were identified. Frequency distributions of the remaining cases indicated no violations of normality. After imputation of the means and predicted values for the continuous variables, analyses were completed using a final sample of \( N = 360 \).

**Summary of Participants with Missing Data**

Chi-square, t-tests and logistic regression analyses were used to characterize participants with missing data and to identify demographic and other correlates of missing values on independent variables. Predictors for the chi-square analyses included: parental history of alcohol use, daily smoking, minority group membership, year in school and high risk drinking (i.e. AUDIT score \( \geq 8 \)). Variables for the continuous analyses included age of first self-initiated alcohol use, current age, AUDIT score, Spaf somatic score, Spaf affective score and negative affect score. Findings for each analysis are summarized below.

**Missing data on age of first self initiated alcohol use.** Significant differences were identified on year in school, \( \chi^2(3, 347)=12.03, p = .01 \), with freshman (69.2%) more likely to have missing data when compared to the number of sophomore, junior and senior women with missing data.

For the continuous outcome, significant age differences were identified, \( t (353)=-2.89, p = .01 \) with younger women (\( M = 18.91, SD = 2.13 \)) were more likely to have missing values on age of first self-initiated alcohol use.

Finally, a logistic regression analysis was used to identify predictors of missing data on age of first self-initiated alcohol use. A categorical variable, “missing age of first self-initiated
alcohol use” was created and used as the dependent variable for the logistic regression. Independent variables included missing daily smoking, missing minority group membership, missing negative affect, missing Spaf somatic score, missing Spaf affective score and missing family history of alcohol problems. None of the independent variables were significant predictors of missing age of first-self-initiated alcohol use.

**Missing data on daily smoking.** No significant differences were identified when assessing correlates of missing data on daily smoking. However, t-tests identified significant age differences, \( t(353) = -2.58, p = .01 \) with younger women (\( M = 18.93, SD = 1.50 \)) were more likely to have missing values on daily smoker.

Logistic regression analysis was used to identify predictors of missing data on daily smoking. A categorical variable, “daily smoking missing” was created and used as the dependent variable for the logistic regression. Independent variables included missing age of first self-initiated alcohol use, missing minority group membership, missing negative affect, missing Spaf somatic score, missing Spaf affective score and missing family history of alcohol problems. One independent variable, missing negative affect, significantly predicted missing values on daily smoking, \( \chi^2(6) = 241.48, p = .001 \).

**Missing data on minority group membership.** No statistically significant differences were found on any of the categorical or continuous variables used to assess for missing data on minority group membership.

Logistic regression analysis was used to identify predictors of missing data on minority group status. A categorical variable, “minority group status missing” was created and used as the dependent variable for the logistic regression. Independent variables included missing age of first self-initiated alcohol use, missing daily smoking, missing negative affect, missing Spaf
somatic score, missing Spaf affective score and missing family history of alcohol problems. No significant predictors of missing minority group status were identified.

**Missing data on negative affect.** For the categorical analysis, two significant differences were identified. First, freshmen women completed the survey were more likely to have missing data on negative affect (54.8%) when compared to other classes, $\chi^2(3, 347) = 24.34, p = .001$. Additionally, non-smokers were more likely to have missing data on negative affect (83.9%) when compared to smokers who completed the survey, $\chi^2(1, 360) = 9.08, p = .001$.

For the continuous outcomes, significant age differences were identified, $t(353) = -4.41, p = .001$, with younger women ($M = 18.93, SD = 1.84$) more likely to have missing data.

Logistic regression analysis was used to identify predictors of missing data on the negative affect variable. A categorical variable, “negative affect score missing” was created and used as the dependent variable for the logistic regression. Independent variables included missing age of first self-initiated alcohol use, missing daily smoking, missing minority group membership, missing Spaf somatic score, missing Spaf affective score and missing family history of alcohol problems. Missing daily smoking was identified as a significant predictor of missing values on negative affect score, $\chi^2(6)=262.25, p = .001$.

**Missing data on Spaf somatic symptom score.** For the categorical outcomes, no significant differences were identified. Similarly, when assessing predictors of the continuous outcomes no significant differences were identified.

Logistic regression analysis was used to identify predictors of missing data on the Spaf somatic symptom score. A categorical variable, “Spaf somatic symptom score missing” was created and used as the dependent variable for the logistic regression. Independent variables included missing age of first self-initiated alcohol use, missing daily smoking, missing minority
group membership, missing negative affect score, missing Spaf affective score and missing family history of alcohol problems. No significant predictors were identified for missing values on Spaf somatic symptom scores.

**Missing data on Spaf affective symptom score.** Categorical analyses demonstrated that non-smokers were more likely to have missing data on negative affect (54.5%) than their smoking peers who completed the survey, $\chi^2(1, 360)=7.23, p = .01$.

Continuous analyses demonstrated that older women ($M = 21.27, SD = 4.41$) were more likely to have missing data than their younger peers, $t(353)=2.36, p = .05$. Similarly, significant differences were identified on age of first alcohol use, $t(290) = 2.00, p = .001$ with women who reported higher ages of self-initiated alcohol use ($M = 16.78, SD = 3.15$) more likely to have missing data on Spaf affective symptom scores.

Logistic regression analysis was used to identify predictors of missing data on the Spaf affective symptom scores. A categorical variable, “Spaf affective symptom score missing” was created and used as the dependent variable for the logistic regression. Independent variables included missing age of first self-initiated alcohol use, missing daily smoking, missing minority group membership, missing Spaf somatic score, missing negative affective score and missing family history of alcohol problems. Missing Spaf somatic score was identified as a significant predictor of missing values on Spaf affective symptom score, $\chi^2(6)=17.71, p = .01$.

**Missing data on parental history of alcoholism.** For both categorical and continuous outcomes no significant differences were identified. Logistic regression analysis was used to identify predictors of missing data on the parental history of alcoholism. A categorical variable, “parental history of alcoholism missing” was created and used as the dependent variable for the logistic regression. Independent variables included missing age of first self-initiated alcohol use,
missing daily smoking, missing minority group membership, missing Spaf somatic score and missing Spaf affective score. No significant differences were identified.

Final Sample Characteristics

Demographic information. The mean age for the final sample of participants (N = 360) was 19.65 years (SD = 2.33). The majority of participants (65.2%) self-identified as White American, with 21% identifying as African-American, 6.2% identifying as Asian-American, 1.7% identifying as Hispanic and 5.9% identifying as “Other”. A review of the “Other” responses revealed that three women wrote in nationalities that are often included as “Asian” in the United States (e.g. Filipino/Iranian; Pacific Islander/Native American; and Indian). Additionally, one woman identified as “West Indian”, which is a national group included under the Black race and three women self-identified as “biracial”. One woman identified as “European”, a group often characterized as White.

Recoding of Race Variable

Because many of the women who self-identified as “Other” reported belonging to nationalities that have been conceptualized as one of the following races in the United States: Asian, Black, Hispanic or White, their responses were recoded and they were grouped into respective racial categories. A new variable, “RaceBlackWhiteOther” was created with the following racial categories: Black, White and Other, so that the groups could be compared to assess for differences on the independent and dependent variables. Women who identified as Asian, biracial and Hispanic were consolidated to create the “other” category of “RaceBlackWhiteOther”.

In order to assess racial and ethnic differences in the independent and dependent variables across the three racial groups, the potential predictors were analyzed using a one-way
analysis of variance (ANOVA) for the continuous independent variables (i.e. affective PMS score, somatic PMS score, age of first self-initiated alcohol use and negative affect score), and a chi-square analysis for the categorical predictors (e.g. daily smoker and parental history of alcohol problems).

Results of the one-way ANOVA demonstrated that there were statistically significant differences between the three racial groups on the continuous AUDIT score, $F(2, 350) = 10.18, p = .001$. Tukey post-hoc comparisons demonstrated that significant mean differences were observed between Black ($M = 3.77, SD = 4.16$) and White women ($M = 7.12, SD = 5.60$) and Black ($M = 3.77, SD = 4.16$) and women categorized as Other ($M = 7.31, SD = 7.19$). No statistically significant differences were identified for age of first self-initiated alcohol use, somatic PMS score, affective PMS score and negative affect score. Results are included in Table 3.

A chi-square test identified significant racial differences between women who reported AUDIT scores in the high-risk range (e.g. greater than or equal to eight) and women whose scores indicated low-risk drinking, $\chi^2(2, 353)=14.54, p = .001$. Table 3 presents a summary of the aforementioned results.

Because the racial and ethnic subgroups responded similarly on measures of the independent variables and only differed significantly from White women, the decision was made to consolidate the responses of Black women and women categorized as “other”. As a result, a new categorical variable, “race/ethnic minority status” was created, and women who self-identified as non-White were coded as minorities (categorical score of one), while White women were coded as non-Hispanic white (categorical score of zero). After creating this additional
group, independent t-tests were run to assess for differences in mean scores of continuous variables. No significant differences were found between the mean scores of minority or white non-minority women on any of the independent variables (e.g. somatic PMS symptoms, affective PMS symptoms, negative affect and age of first drink). The groups differed significantly on the AUDIT, $t(194) = 4.04, p = .001$, and when analyzed using a chi-squared analysis, the groups differed significantly on the AUDIT, $\chi^2(1, 194) = 16.36, p = .001$, with 79.1% of non-minority White women reporting high-risk drinking compared to 20.9% of minority women. Additional chi-square analyses revealed that the groups did not differ significantly on smoker status or family history of alcohol problems. Table 4 includes a summary of the final sample’s demographic and psychosocial characteristics.

**Domain Specific Participant Characteristics**

**Prevalence of tobacco use.** Thirty-five percent of participants reported smoking daily at some point in their lifetimes. In this subgroup, 50.9% reported that they were daily smokers at the time of assessment, while 26.4% reported only smoking during social occasions or while drinking.

**Prevalence of premenstrual symptomatology.** Participant scores on the affective PMS symptom subscale of the SPAF ranged from values of 10 to 60 ($M = 32.17, SD = 12.87$). A review of item responses revealed that many participants reported severe changes in the following symptoms during the week prior to their periods: tendency to argue (20.4%) and the presence of mood swings (22.1%). Moderate changes were noted for affective PMS symptoms: feeling dissatisfied with personal appearances (22.3%); increased stress (22.4%) and sadness (23.7%). Table 6 includes a summary of the item responses and means for both the affective and somatic subscales of the SPAF.
Table 3

Comparison of Predictor and Outcome Measures by Racial Group

<table>
<thead>
<tr>
<th>Variable</th>
<th>Black M (S.D.) or %</th>
<th>Other M (S.D.) or %</th>
<th>White M (S.D.) or %</th>
</tr>
</thead>
<tbody>
<tr>
<td>Tobacco Use ***</td>
<td></td>
<td></td>
<td></td>
</tr>
<tr>
<td>Daily smoker</td>
<td>4.8%</td>
<td>16.2%</td>
<td>79%&lt;sup&gt;a&lt;/sup&gt;</td>
</tr>
<tr>
<td>Affective PMS symptom score</td>
<td>29.49 (13.57)</td>
<td>31.98 (12.12)</td>
<td>32.80 (12.59)</td>
</tr>
<tr>
<td>Somatic PMS symptom score</td>
<td>28.12 (11.40)</td>
<td>28.19 (10.76)</td>
<td>30.36 (9.86)</td>
</tr>
<tr>
<td>Age of self-initiated first alcohol use</td>
<td>15.55 (2.61)</td>
<td>14.83 (2.82)</td>
<td>14.86 (2.25)</td>
</tr>
<tr>
<td>Negative affect score</td>
<td>20.10 (5.31)</td>
<td>21.75 (6.05)</td>
<td>21.05 (5.72)</td>
</tr>
<tr>
<td>Parental history of alcohol problems</td>
<td></td>
<td></td>
<td></td>
</tr>
<tr>
<td>One parent PH+</td>
<td>25.4%</td>
<td>10.4%</td>
<td>64.2%</td>
</tr>
<tr>
<td>Both parents PH+</td>
<td>28.6%</td>
<td>0%</td>
<td>71.4%</td>
</tr>
<tr>
<td>AUDIT (continuous)</td>
<td>2.79 (3.94)&lt;sup&gt;***&lt;/sup&gt;</td>
<td>5.14 (6.89)</td>
<td>6.25 (6.08)</td>
</tr>
<tr>
<td>High risk alcohol use (AUDIT score ≥ 8)&lt;sup&gt;***&lt;/sup&gt;</td>
<td>9.7%</td>
<td>13.3%</td>
<td>77.0%&lt;sup&gt;a&lt;/sup&gt;</td>
</tr>
</tbody>
</table>

* <sup>p = .05; ** p = .01; *** p = .001</sup>

<sup>a</sup> White women differed significantly from both Black women and women identified as “Other”.

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Scores on the somatic PMS symptom subscale of the SPAF ranged from values of 10 to 60 ($M = 30.08$, $SD = 10.53$). Approximately one fourth of the sample reported moderate changes in one or more of the following symptoms during the week prior to menses: swelling and breast tenderness (27.8%); decreased energy and fatigue (26.3%); malaise (25.8%) and abdominal pain and discomfort (23.8%).

**Prevalence of negative affect.** Scores on the Negative Affect subscale of the PANAS ranged from values of 10 to 41 ($M = 21.15$, $SD = 5.92$). Participants reported that in the week prior to completing the assessment, they experienced minimal levels of the following emotions: nervousness (38.7%), irritability (35.3%), distress (33.6%) and feeling upset (38.3%).

**Prevalence of parental history of alcohol problems.** As reported in Table 4, 21.9% of participants reported at least one biological parent with an alcohol problem. Additionally, 5.2% of these participants also reported that both parents had a history of alcohol problems. Of the women who reported only one parent affected, 78.1% reported that their biological fathers had an AUD.

**Prevalence of alcohol use.** All of the 360 women included in the sample used to complete the statistical analyses reported drinking at least one alcoholic beverage in the 12 months prior to completing the assessment battery for the present study. Average age of reported first self-initiated alcohol use was 14.99 years ($SD = 2.47$). Overall, 67.4% of participants reported consuming alcohol one or fewer weekdays in the 30-day period prior to completing the assessment battery. The number of drinks that they reported consuming on at least one weekday ranged from 0 to 12 drinks ($M = 1.51$, $SD = 1.91$).
<table>
<thead>
<tr>
<th>Characteristic</th>
<th>Final Sample (n=360)</th>
</tr>
</thead>
<tbody>
<tr>
<td>Tobacco Use</td>
<td></td>
</tr>
<tr>
<td>Daily Smoker</td>
<td>35%</td>
</tr>
<tr>
<td>Affective PMS symptom score</td>
<td>32.17 (12.86)</td>
</tr>
<tr>
<td>Somatic PMS symptom score</td>
<td>30.10 (10.53)</td>
</tr>
<tr>
<td>Age of first self-initiated alcohol use (years)</td>
<td>14.99 (2.47)</td>
</tr>
<tr>
<td>Negative affect score</td>
<td>21.15 (5.92)</td>
</tr>
<tr>
<td>Parental history of alcohol problems</td>
<td></td>
</tr>
<tr>
<td>One parent PH+</td>
<td>21.9%</td>
</tr>
<tr>
<td>Both parents PH+</td>
<td>5.2%</td>
</tr>
<tr>
<td>Race/ethnicity</td>
<td></td>
</tr>
<tr>
<td>Minority women</td>
<td>34.6%</td>
</tr>
<tr>
<td>AUDIT Score (continuous)</td>
<td>6.40 (5.94)</td>
</tr>
<tr>
<td>High risk alcohol use (AUDIT score ≥ 8)</td>
<td>32.2%</td>
</tr>
<tr>
<td>Age at time of assessment (years)</td>
<td>19.65 (2.33)</td>
</tr>
</tbody>
</table>
However, the reported number of drinks consumed on a single day during the weekend ranged from 0 to 16 ($M = 3.45$, $SD = 2.46$), and the largest reported number of drinks consumed on a single day during the weekend ranged from 0 to 16 ($M = 3.48$, $SD = 2.67$). Participants reported feeling the effects of the alcohol after consuming approximately three drinks ($M = 2.94$, $SD = 1.45$). Eighteen percent of participants reported that injuries to self or others occurred as a result of their drinking.

**Hypothesis Testing**

In order to reduce potential response bias and to preserve the sample size, missing values were imputed for continuous variables (Gelman & Hill, 2007; Tabachnick & Fidell, 2007). However, because two of the categorical variables had missing data (i.e. daily smoker and parental history of alcohol use), which could not be, imputed using the mean or predicted values, the actual sample size used for analyses decreased to $N=256$ women.

**Hypothesis one.** The first hypothesis posited that a model of psychosocial variables (e.g. tobacco use, affective PMS symptoms, somatic PMS symptoms, age of first self-initiated alcohol use, negative affect, family history of alcohol problems and minority status) would significantly predict engagement in risk drinking among this sample of college women. To test this hypothesis, a standard logistic regression was performed, using a categorical AUDIT score as the measure of risk drinking. Additionally, a standard multiple regression was computed and used the continuous AUDIT score as a measure of risk-drinking.
Table 5

*Means and Standard Deviations for Item Responses on the Shortened Premenstrual Assessment Form (SPAF)*

<table>
<thead>
<tr>
<th>Domain</th>
<th>Item</th>
<th>M (S.D.)</th>
<th>Level of Change</th>
</tr>
</thead>
<tbody>
<tr>
<td>Affective PMS symptoms</td>
<td>2. Feel anxious or more nervous</td>
<td>2.75 (1.41)</td>
<td>Minimal</td>
</tr>
<tr>
<td></td>
<td>5. Feel that I just can’t cope or am overwhelmed by ordinary demands</td>
<td>2.74 (1.54)</td>
<td>Minimal</td>
</tr>
<tr>
<td></td>
<td>6. Tend to nag or quarrel over unimportant issues</td>
<td>3.48 (1.61)</td>
<td>Mild</td>
</tr>
<tr>
<td></td>
<td>7. Feel dissatisfied with personal appearances</td>
<td>3.48 (1.63)</td>
<td>Mild</td>
</tr>
<tr>
<td></td>
<td>8. Tend to be tearful, weep or cry</td>
<td>3.39 (1.75)</td>
<td>Mild</td>
</tr>
<tr>
<td></td>
<td>9. Feel under stress</td>
<td>3.46 (1.55)</td>
<td>Mild</td>
</tr>
<tr>
<td></td>
<td>10. Have mood swings from high to low or low to high</td>
<td>3.55 (1.65)</td>
<td>Mild</td>
</tr>
<tr>
<td></td>
<td>11. Have outbursts of “irritability” or bad temper</td>
<td>3.26 (1.65)</td>
<td>Mild</td>
</tr>
<tr>
<td></td>
<td>12. Feel sad or blue</td>
<td>3.17 (1.51)</td>
<td>Mild</td>
</tr>
<tr>
<td></td>
<td>15. Tend to be intolerant or impatient or to lose the ability to respond or understand the faults, needs or errors of others</td>
<td>2.99 (1.54)</td>
<td>Minimal</td>
</tr>
<tr>
<td>Somatic PMS symptoms</td>
<td>1. Have decreased energy or tend to easily fatigue</td>
<td>2.99 (1.32)</td>
<td>Minimal</td>
</tr>
<tr>
<td></td>
<td>3. Have a feeling of malaise</td>
<td>3.17 (1.45)</td>
<td>Mild</td>
</tr>
<tr>
<td></td>
<td>4. Have pain, tenderness, enlargement and swelling breasts</td>
<td>3.24 (1.62)</td>
<td>Mild</td>
</tr>
</tbody>
</table>
(Table 5 continued)

<table>
<thead>
<tr>
<th></th>
<th>Description</th>
<th>Score</th>
<th>Severity</th>
</tr>
</thead>
<tbody>
<tr>
<td>14</td>
<td>Have weight gain</td>
<td>2.68</td>
<td>Minimal</td>
</tr>
<tr>
<td></td>
<td></td>
<td>(1.47)</td>
<td></td>
</tr>
<tr>
<td>16</td>
<td>Have relatively steady abdominal heaviness, discomfort or pain</td>
<td>3.18</td>
<td>Mild</td>
</tr>
<tr>
<td></td>
<td></td>
<td>(1.64)</td>
<td></td>
</tr>
<tr>
<td>17</td>
<td>Have increased sexual activity or desire</td>
<td>2.99</td>
<td>Mild</td>
</tr>
<tr>
<td></td>
<td></td>
<td>(1.66)</td>
<td></td>
</tr>
<tr>
<td>18</td>
<td>Have skin problems such as acne, pimples, etc.</td>
<td>3.00</td>
<td>Mild</td>
</tr>
<tr>
<td></td>
<td></td>
<td>(1.55)</td>
<td></td>
</tr>
<tr>
<td>19</td>
<td>Have edema, swelling, stiffness or water retention</td>
<td>2.45</td>
<td>Minimal</td>
</tr>
<tr>
<td></td>
<td></td>
<td>(1.50)</td>
<td></td>
</tr>
<tr>
<td>20</td>
<td>Feel bloated</td>
<td>3.23</td>
<td>Mild</td>
</tr>
<tr>
<td></td>
<td></td>
<td>(1.56)</td>
<td></td>
</tr>
</tbody>
</table>
**Logistic regression analysis.** Seven potential predictors were entered into the logistic regression: tobacco use, affective PMS symptom score, somatic PMS symptom score, age of first-self initiated alcohol use, negative affect score, parental history of alcohol problems and minority status. Compared to the constant-only model, a model with all seven predictors was statistically significant, $\chi^2(7) = 68.67, p = .001$. Table 6 provides a summary of the model as well as odds ratios for the predictors. Wald criteria demonstrate that daily smoking significantly predicted high risk drinking at the $p = .001$ level, $\chi^2(1) = 15.69, p = .001$ and that two other independent variables also significantly predicted high risk drinking at the $p = .05$ level: 1) negative affect, $\chi^2(1) = 6.16, p = .05$ and 2) minority group membership, $\chi^2(1) = 5.40, p = .05$. Predictive ability for the model was modest: Nagelkerke’s $R$-square $= .326$ and Cox and Snell’s $R$-square $= .235$.

**Multiple regression analysis.** Daily smoker, affective PMS symptom score, somatic PMS symptom score, age of first self-initiated alcohol use, negative affect score, parental history of alcohol problems and minority status were also entered as potential predictors into a stepwise multiple regression equation. The full model, with all predictors, was compared with a constant-only model and found to be statistically significant, $F(7, 248) = 8.47, p = .001$. An adjusted $R$-squared value indicated that the full model accounted for 17% of the variance in AUDIT scores. Significant predictors in the model included somatic PMS symptom score, $t(248)=1.97, p = .05$; negative affect score, $t(248) = 3.19, p = .01$; age of first self-initiated alcohol use, $t(248) = -3.09, p = .01$; and minority status, $t(248) = -2.17, p = .05$. Table 7 includes a summary of the multiple regression results.
<table>
<thead>
<tr>
<th></th>
<th>B</th>
<th>S.E.</th>
<th>Wald</th>
<th>Df</th>
<th>Sig.</th>
<th>Exp(B)</th>
<th>95% Confidence Interval</th>
</tr>
</thead>
<tbody>
<tr>
<td>Daily Smoker</td>
<td>1.25</td>
<td>.31</td>
<td>15.69</td>
<td>1</td>
<td>.000***</td>
<td>3.50</td>
<td>1.88-6.51</td>
</tr>
<tr>
<td>Affective PMS symptom score</td>
<td>.026</td>
<td>.020</td>
<td>1.82</td>
<td>1</td>
<td>.177</td>
<td>1.01</td>
<td>.962-1.06</td>
</tr>
<tr>
<td>Somatic PMS symptom score</td>
<td>.010</td>
<td>.025</td>
<td>.166</td>
<td>1</td>
<td>.683</td>
<td>1.03</td>
<td>.988-1.07</td>
</tr>
<tr>
<td>Age of first self-initiated</td>
<td>-.125</td>
<td>.068</td>
<td>3.38</td>
<td>1</td>
<td>.066</td>
<td>.883</td>
<td>.773-1.01</td>
</tr>
<tr>
<td>alcohol use</td>
<td></td>
<td></td>
<td></td>
<td></td>
<td></td>
<td></td>
<td></td>
</tr>
<tr>
<td>Negative affect score</td>
<td>.067</td>
<td>.027</td>
<td>6.16</td>
<td>1</td>
<td>.013*</td>
<td>1.07</td>
<td>1.01-1.13</td>
</tr>
<tr>
<td>Parental history of alcohol</td>
<td>.247</td>
<td>.261</td>
<td>.899</td>
<td>1</td>
<td>.343</td>
<td>1.28</td>
<td>.768-2.13</td>
</tr>
<tr>
<td>problems</td>
<td></td>
<td></td>
<td></td>
<td></td>
<td></td>
<td></td>
<td></td>
</tr>
<tr>
<td>Minority status</td>
<td>-.817</td>
<td>.352</td>
<td>5.40</td>
<td>1</td>
<td>.020*</td>
<td>.442</td>
<td>.222-0.880</td>
</tr>
<tr>
<td>Constant</td>
<td>-.817</td>
<td>.352</td>
<td>5.40</td>
<td>1</td>
<td>.020*</td>
<td>.442</td>
<td>.222-0.880</td>
</tr>
</tbody>
</table>

*p = .05; **p = .01; ***p = .001
Table 7

Summary of Standard Multiple Regression Analysis of High Risk Drinking using Continuous AUDIT-Scores

<table>
<thead>
<tr>
<th>Variable</th>
<th>B</th>
<th>SE B</th>
<th>β</th>
<th>p-value</th>
<th>95% Confidence Interval</th>
</tr>
</thead>
<tbody>
<tr>
<td>Daily Smoker</td>
<td>-.001</td>
<td>.006</td>
<td>-.012</td>
<td>.838</td>
<td>-.014-.011</td>
</tr>
<tr>
<td>Affective PMS symptom score</td>
<td>.001</td>
<td>.044</td>
<td>.002</td>
<td>.983</td>
<td>-.086-.088</td>
</tr>
<tr>
<td>Somatic PMS symptom score</td>
<td>.116</td>
<td>.059</td>
<td>.190</td>
<td>.050*</td>
<td>.000-.231</td>
</tr>
<tr>
<td>Age of first self-initiated alcohol use</td>
<td>-.472</td>
<td>.153</td>
<td>-.181</td>
<td>.002**</td>
<td>-.773-.171</td>
</tr>
<tr>
<td>Negative affect score</td>
<td>.201</td>
<td>.063</td>
<td>.206</td>
<td>.002**</td>
<td>.077-.326</td>
</tr>
<tr>
<td>Parental history of alcohol problems</td>
<td>.477</td>
<td>.633</td>
<td>.044</td>
<td>.452</td>
<td>-.769-1.72</td>
</tr>
<tr>
<td>Minority status</td>
<td>-1.62</td>
<td>.744</td>
<td>-.125</td>
<td>.031*</td>
<td>-3.08-.151</td>
</tr>
<tr>
<td>Constant</td>
<td>6.19</td>
<td>2.83</td>
<td>-</td>
<td>.030*</td>
<td>.612-11.77</td>
</tr>
</tbody>
</table>

*p = .05; **p = .01; ***p = .001
Hypothesis two. The second hypothesis posited that that variables found to significantly predict risk drinking would vary by minority group membership. To test this hypothesis, a series of interaction terms (e.g. minority status*predictor) were entered into both a logistic regression equation where the dependent variable was the categorical AUDIT score, and a multiple regression equation where the dependent variable was an individual’s continuous AUDIT score.

Logistic regression analysis of moderation. The following independent variables were entered into the logistic regression equation: daily smoker, negative affect, minority status and two interaction terms (e.g. minority status*daily smoker; minority status*negative affect). When compared to a constant-only model, the model with all five predictors was statistically significant, $\chi^2(5) = 53.77, p = .001$. Significant predictors in the final model were daily smoking, $\chi^2(1) = 14.86, p = .001$ and negative affect, $\chi^2(1) = 13.40, p = .001$. None of the interaction variables significantly predicted high risk drinking. Model effect sizes were modest: Nagelkerke’s $R$-square =.231 and Cox and Snell’s $R$-square =.165. Table 8 provides a summary of the coefficients.

Multiple regression analysis of moderation. Independent variables entered into a multiple regression equation included somatic PMS symptom score, age of first self-initiated alcohol use, negative affect score, minority status and three interaction terms: somatic PMS symptom score*minority status, negative affect*minority status and age of first self-initiated alcohol use*minority status. The overall model with all seven predictors was statistically significant, $F(7, 342) = 8.89, p = .001$. An adjusted $R$-squared value indicated that the full model accounted for 13.7% of the variance in AUDIT scores. Statistically significant predictors included 1) somatic PMS symptom score*minority status interaction, $t(342) = 2.06, p = .05$; 2)
Table 8

*Summary of Logistic Regression with Categorical AUDIT Score as the Dependent Measure of Risk Drinking*

<table>
<thead>
<tr>
<th></th>
<th>B</th>
<th>S.E.</th>
<th>Wald</th>
<th>Df</th>
<th>Sig.</th>
<th>Exp(B)</th>
<th>95% Confidence Intervals</th>
</tr>
</thead>
<tbody>
<tr>
<td>Daily smoker</td>
<td>1.26</td>
<td>.33</td>
<td>14.86</td>
<td>1</td>
<td>.001***</td>
<td>3.545</td>
<td>1.86-6.74</td>
</tr>
<tr>
<td>Minority status</td>
<td>-.705</td>
<td>.389</td>
<td>3.28</td>
<td>1</td>
<td>.070</td>
<td>.494</td>
<td>.230-1.06</td>
</tr>
<tr>
<td>Daily smoker*Minority status</td>
<td>.486</td>
<td>.633</td>
<td>.589</td>
<td>1</td>
<td>.445</td>
<td>1.62</td>
<td>.470-5.62</td>
</tr>
<tr>
<td>Negative Affect</td>
<td>.100</td>
<td>.027</td>
<td>13.40</td>
<td>1</td>
<td>.001***</td>
<td>1.10</td>
<td>1.05-1.17</td>
</tr>
<tr>
<td>Negative affect*Minority status</td>
<td>-.491</td>
<td>.284</td>
<td>2.99</td>
<td>1</td>
<td>.084</td>
<td>.612</td>
<td>.351-1.07</td>
</tr>
<tr>
<td>Constant</td>
<td>-3.20</td>
<td>.656</td>
<td>23.85</td>
<td>1</td>
<td>.001***</td>
<td>.041</td>
<td></td>
</tr>
</tbody>
</table>

*p = .05; **p = .01; ***p = .001
negative affect score, $t(342) = 3.21, p = .01$; and minority status, $t(342) = -2.73, p = .01$. A summary of the coefficients is included in Table 9.
Table 9

Summary of Multiple Regression Analysis of Risk Drinking using Continuous AUDIT Scores

<table>
<thead>
<tr>
<th>Variable</th>
<th>B</th>
<th>SE B</th>
<th>β</th>
<th>p-value</th>
<th>95% Confidence Interval</th>
</tr>
</thead>
<tbody>
<tr>
<td>Somatic PMS symptom Score</td>
<td>.039</td>
<td>.044</td>
<td>.068</td>
<td>.378</td>
<td>-.684-11.02</td>
</tr>
<tr>
<td>Negative Affect score</td>
<td>.237</td>
<td>.074</td>
<td>.233</td>
<td>.001***</td>
<td>.092-.383</td>
</tr>
<tr>
<td>Age of first self-initiated alcohol use</td>
<td>-.287</td>
<td>.165</td>
<td>-.116</td>
<td>.083</td>
<td>-.612-.037</td>
</tr>
<tr>
<td>Minority status</td>
<td>-1.72</td>
<td>.632</td>
<td>-.136</td>
<td>.007**</td>
<td>-2.965-.480</td>
</tr>
<tr>
<td>Somatic PMS symptom score*Minority status</td>
<td>1.445</td>
<td>.702</td>
<td>.154</td>
<td>.05*</td>
<td>.064-2.83</td>
</tr>
<tr>
<td>Negative affect symptom score* Minority status</td>
<td>-.868</td>
<td>.701</td>
<td>-.087</td>
<td>.216</td>
<td>-2.25-.511</td>
</tr>
<tr>
<td>Age of first self-initiated alcohol use*Minority status</td>
<td>-.281</td>
<td>.627</td>
<td>-.030</td>
<td>.654</td>
<td>-1.51-.952</td>
</tr>
<tr>
<td>Constant</td>
<td>5.17</td>
<td>2.97</td>
<td>-</td>
<td>.083</td>
<td>-.684-11.02</td>
</tr>
</tbody>
</table>

*p = .05; **p = .01; ***p = .001
Discussion

The goal of the present study was threefold. It sought to: 1) examine racial differences in risk drinking among college women; 2) identify psychosocial predictors of risk drinking among a sample of college women; and 3) determine if racial/ethnic minority status moderated the relationship between significant predictors of risk drinking. Potential predictors included current daily smoking, affective PMS symptom score, somatic PMS symptom score, age of first self-initiated alcohol use, negative affect score, parental history of alcohol problems, and minority status. Risk drinking was operationalized using the AUDIT to create both a continuous variable (total AUDIT score) and a categorical variable (cutoff score greater than or equal to eight). In the later case, participants were labeled either “high risk” (AUDIT score equal to or greater than eight) or “low risk” (AUDIT score equal to or less than eight).

It was hypothesized that a model of selected psychosocial factors would significantly predict risk drinking, and that minority status would moderate the relationship between significant predictors and risk drinking. The full model (with all potential psychosocial predictors) was tested to identify correlates of risk drinking. Significant predictors of both categorical and continuous outcome measures were then analyzed to determine minority status moderated in these relationships.

Summary of Study Findings

Racial and ethnic differences in risk drinking. A primary aim of this study was to better understand racial and ethnic group differences in risk drinking. As expected, the present study found that significantly fewer minority women engaged in risk drinking and their drinking was more likely to be characterized as low-risk compared to alcohol use by a similar group of White female participants. Additionally, the present study found that White women obtained
higher AUDIT scores and were more likely to be classified as high-risk drinkers than minority women.

**Predictors of risk drinking.** As hypothesized, the full model that included seven psychosocial variables significantly predicted risk drinking both as a categorical and as a continuous measure. For the categorical variable, daily smoking, negative affect and non-minority status predicted high risk drinking. For the continuous variable, somatic PMS symptom score, age of first alcohol use, negative affect and non-minority status predicted risk drinking. These findings suggest that higher AUDIT scores were associated with more somatic PMS symptomatology and greater negative affect. However, as age of first alcohol use increased, women were less likely to report high-risk alcohol use. Both analyses confirmed that minority status was associated with low-risk drinking, while non-minority status was associated with high-risk drinking.

**Racial/ethnic minority status as a moderator of the relationship between predictors and risk drinking.** Next, minority status was examined as a potential moderator of the relationship between the independent measures and both categorical and continuous AUDIT scores. The data showed that minority women were more likely to report low-risk drinking regardless of their somatic PMS symptom scores, while non-minority women were more likely to report high-risk drinking as their somatic PMS symptom scores increased. However, race only moderated the relationship for the continuous outcome measure, and moderation analyses were not significant when using a categorical outcome measure of risk drinking.

**Discussion of Study Findings**

**Premenstrual symptomatology and alcohol use.** The relationship between premenstrual symptoms and alcohol use/abuse has received considerable research attention
Findings are inconsistent, however, with some studies confirming and others negating an association between reported PMS symptom severity and levels of alcohol consumed during the premenstrual phase in both clinical (e.g. women with AUDs or diagnosed PMS) and non-clinical samples (Allen, 1996; Leonhardt et al., 2001; Chuong & Burgos, 1995).

Most previous studies focused on women who met diagnostic criteria for Alcohol Abuse/Dependence. Findings from such studies demonstrated that women with AUDs reported increased drinking during the premenstrual cycle when compared to women without AUDs (Podolsky, 1963; Belfer, Shader, Carroll & Harmatz, 1971). Further, this finding was confirmed when participants’ alcohol use during the month was compared to their use during the premenstrum (Allen, 1996). A review of studies with women who did not have AUDs found evidence that women who report severe symptoms of PMS also report increased consumption of alcohol, during the premenstrual phase (Deuster, Adera & South-Paul, 1999; Kritz-Silverstein, Wingard & Garland, 1999; Strine, Chapman & Ahluwalia, 2005). Some researchers posit that such fluctuations in drinking may place women at greater risk for developing AUDs. Overall, data from these studies indicate that women who report more severe symptoms of PMS are also more likely to report increases in their alcohol use during the premenstrum. However, these findings should be interpreted with caution due as several methodological issues limit their generalizability. These issues include use of retrospective data, participant awareness of possible hypotheses and limited racial diversity in previous samples.

Retrospective studies have been criticized because some women report inflated estimates of their PMS symptom severity (Marvan, Cortes-Iniestra, 2001; McFarland, Ross &
DeCourville, 1989; Nash & Chrisler, 1997). In order to limit the influence of recall bias, researchers have gathered prospective data on menstrual cycles and variations in alcohol use throughout various phases of the cycle. Findings from many prospective studies contradict the association between retrospective reports of severe PMS symptoms and increased alcohol use during the premenstrum (Ascher-Svanum, 1982; Harvey & Beckman, 1985; Sutker, Libet, Allain & Randall, 1983). In the present study, retrospective data were used to assess all of the domains. Because of the risk of recall bias, the findings of the present study should be regarded with caution and used as exploratory findings that may guide future research.

Another limitation to some studies of the relationship between PMS symptoms and alcohol use is participants’ knowledge of the study hypotheses. As noted by researchers studying this area, when participants have knowledge of the hypotheses, they may underreport their drinking behaviors or over-report their PMS symptoms (Abplanalp, 1983; Brooks-Gunn & Ruble, 1980). In the present study, women had no knowledge about study hypotheses. Instead, participants were told that this was a study of healthy habits. Because the participants of the original study were blind to the actual hypotheses, they were immune to the effects of reactivity to the actual study’s purpose.

Another factor to consider is that many studies to date have focused on homogenous samples with limited racial and ethnic diversity. While discussed further in the Study Limitations section, this lack of racial and ethnic diversity participants limits generalization of previous study findings to predominately Caucasian women.

Despite these methodological limitations, findings from the current study emphasize an important distinction between PMS affective versus somatic PMS symptoms and the relationship between these individual domains and risk drinking. In the present study, the SPAF included
PMS symptoms that were clustered into separate affective and somatic symptom categories. When analyzed separately, somatic PMS symptoms were associated with risk drinking, while affective PMS symptoms were not significantly associated with risk drinking in this sample. Much of the research on PMS symptoms has not distinguished between affective and somatic PMS symptoms and their relationship to alcohol use; instead, researchers have relied on measures that often combine severity scores for symptoms comprising both domains into a sum total score. However, this study’s findings demonstrate that somatic, but not affective symptoms, are positively associated with risk drinking. Additionally, results from the present study demonstrate that race moderates the relationship between somatic PMS symptoms and risk drinking, such that minority women, even those with severe somatic PMS symptoms reported low risk drinking while White peers’ risk drinking increased as their somatic PMS symptom severity scores increased.

Previous reports have found a positively associated relationship between somatic symptoms (e.g. bloating, weight gain and fatigue) and increased drinking during the premenstrual phase (Svikis et al., 2006). Research on the relationship between somatic symptoms and alcohol use has demonstrated that women who report numerous somatic complaints such as back pain, weight gain, bloating, and abdominal pain may increase their alcohol intake to alleviate their somatic pain (Tien, Schlaepfer & Fisch, 1997). Because the literature provides limited information from previous studies about the relationship between somatic PMS symptoms and alcohol use separate from the co-morbid affective PMS symptoms, the present study findings support the utility of examining the relationship between premenstrual alcohol use and PMS symptom severity separately for the two domains.
**Age of first self-initiated alcohol use.** Research has shown that individuals who initiate alcohol use at an earlier age (e.g. less than or equal to age 14) are at an increased risk of developing alcohol dependence and related problems later in life (NIAAA, 1998; DeWit, Adlaf, Offord & Ogbourne, 2000). The present study revealed a strong association between age of first self-initiated alcohol use and risk drinking, when defined by a continuous measure. Specifically, as age of first-self initiated alcohol use increased, AUDIT continuous scores decreased overall with some increases (e.g. increased response frequencies) reported at ages 12, 17-18 and 20 in the current sample. The mean age of self-initiated alcohol use for the study participants was 14.99 years. As suggested by previous findings, women who reported alcohol use prior to age 14 reported some of the higher AUDIT scores, which indicated that their alcohol consumption was in the high-risk range (DeWit et al., 2000).

Within the research, there are several explanations for the relationship between early alcohol initiation and eventual alcohol problems. One hypothesis is that early initiation of alcohol may be associated with impulsivity and poor emotion regulation skills (Glanz & Leshner, 2000). Additional research supports the influence of family history of alcohol problems and environmental factors, such as social learning processes and social norms towards alcohol use, on age of first drink (Prescott & Kendler, 1999; Poelen, Derks, Engels, van Leeuwe, Scholte, Willemsen & Boomsma, 2008). While the exact causal mechanisms and processes remain unknown, research findings continue to demonstrate that among women, early initiation of alcohol is associated with increased alcohol dependence later on in life and that because of this effect, early intervention and screening is warranted.

Somewhat surprisingly, in this sample, race did not moderate the relationship between age of first-self initiated alcohol use and risk drinking, suggesting that younger age at initiation of
alcohol use may be associated with risk for alcohol problems regardless of the genetic or cultural
depth factors subsumed under minority group membership. In other studies, findings on the
relationship between race and age at initiation of alcohol have typically shown that Caucasian
youth are more likely to initiate alcohol use during adolescence when compared to non-white
same age samples (Flewelling & Bauman, 1990; Hawkins, Graham, Maguin, Abbott, Hill, &
Caetano, 1997; Johnston, O’Malley & Bachman, 2002; Donovan, 2004). Additional analyses of
these studies have revealed that when the non-white or minority groups were subdivided into
raced and ethnic groups, differences were noted with Asian American participants reporting
delays in age of alcohol of initiation when compared to white participants (Kosterman, Hawkins,
Guo, Cateano & Abbott, 2000; Donovan, 2004). By contrast no differences were identified in
the age of alcohol initiation reported by African American and Native American participants
when compared to white participants (Kosterman et al., 2000; Kaplow, Curran, Angold &
Costello, 2001; Donovan, 2004). These findings in addition to the current study’s findings may
demonstrate the effect of two processes. First, the age of initiation may be converging between
the races. For example, in the present study’s sample, women from different racial groups did
not differ statistically when comparing their age of alcohol initiation. Second, when examiners
collapse several racial and ethnic groups into one homogenous minority group, these individual
differences become eclipsed, thus giving the effect of no within group or between group
differences. In the present study, the minority group was comprised of predominately African
American women. However, because all ethnicities were grouped after conducting analyses to
assess for between group differences, there’s no way of knowing how responses by African
American women may have influenced the relationships between potential predictors and
assessed outcomes.
In the present study both within and between group comparisons were used to assess for differences between racial and ethnic groups before they were grouped together to form a larger minority group. Many researchers do not report this step or their findings to demonstrate that their groups are more alike than different before creating a non-white group. However, as findings from Kosterman and colleagues (2000) and Kaplow and colleagues (2001) demonstrate, within group differences may exist and researchers would be wise to evaluate these potential differences before consolidating groups.

**Racial and ethnic minority group status.** Studies of college students have consistently demonstrated that racial and ethnic minority women report lower rates of binge drinking, risk drinking, alcohol abuse and alcohol related consequences than non-minority peers (CDC, 1997; Wechsler et al., 2000; O’Malley & Johnston, 2002). In many studies of undergraduates, Black and Asian women reported the highest rates of abstention when compared to Hispanic, Biracial and White peers (Wechsler et al., 2000). By contrast, all of the previously cited studies (e.g. MTF, CAS, YRBSS-NCHRBS) reported that White students endorsed the highest percentage of items pertaining to alcohol abuse, alcohol dependence, binge drinking and alcohol related problems. racial group with the highest rates of alcohol abuse, alcohol dependency, binge drinking and alcohol related problems.

Consistent with larger studies, the present study found that White women reported higher rates of risk drinking than racial/ethnic minority women. Further, present findings demonstrate that minority status moderates the relationship between somatic PMS symptoms and risk drinking, such that minority women, even those who reported severe symptoms of PMS, were less likely to report high risk drinking. These findings are also consistent with the current
literature and suggest that minority women are less likely than their White peers to engage in risk drinking at the college level.

Minority status or minority group membership has often been identified as a protective factor against risk drinking during adolescence and young adulthood. Researchers have identified sociocultural and genetic factors associated with decreased alcohol consumption among minority women that may explain the protective relationship of minority group membership. Sociocultural factors, which may affect alcohol consumption include cultural norms that disparage inebriation and alcohol consumption (Caetano et al., 1998). These sociocultural factors often overlap, but can vary by racial or ethnic group. For example, extant literature posits that among Black women, religiosity and spirituality, having fewer friends who drink, attendance at a four year college or university, and attendance at a Historically Black College or University are associated with decreases in alcohol use (Meilman et al., 1995; Paschall et al., 2005). Other socio-cultural factors associated with reduced rates of alcohol consumption among college educated Black women include disconnection from the predominately White culture and associated cultural values on predominately White college campuses (Peralta, 2005), perceived pressure to negate negative stereotypes of Blacks as prone to heavier alcohol and substance use (Peralta & Steele, 2009), the perception that Black students are under increased surveillance and receive harsher punishments for alcohol infractions (Peralta & Steele, 2009), and fear of experiencing racism when drinking among inebriated White peers (Peralta & Steele, 2009). Whereas with Asian, Hispanic or Latina college students, some of the sociocultural factors associated with decreased alcohol may include racial/ethnic identity consolidation, and the effects of assimilation and acculturation on non-adherence to cultural
values that discourage drinking and drunkenness (Hatchett and Holmes, 2004; Warner et al., 2006).

In addition to the sociocultural factors associated with abstinence from alcohol among minority college students, some Asian students may have a biological factor that decreases their use of alcohol. Specifically, individuals who have an inactive copy of the aldehyde dehydrogenase (*ALDH2*2) allele tend to be of Chinese, Korean or Japanese descent, and are rarely from European or African descent. Individuals with this allele typically report adverse side effects of alcohol (e.g. facial and body flushing) within an hour of consuming it (Edenberg, 2007; Oota et al., 2004). Individuals with copies of this allele may be less inclined to drink as often or as frequently as individuals without this allele, who do not experience these effects. As such, the prevalence of the *ALDH2*2 allele in Chinese, Korean or Japanese individuals may explain why some Asians report lower levels of alcohol consumption in the general and college samples. However, this biological factor has only been identified in samples of Asian individuals and no additional biological factors have been identified or associated with decreased alcohol use among Black or Hispanic individuals.

Overall, these factors suggest that sociocultural beliefs and norms may prevent the minority college women from engaging in alcohol use. In the case of some groups such as Asian American college women, biological and sociocultural factors may interact to produce a general protective factor against alcohol related problems during their college years. However, the effects of sociocultural processes such as acculturation and assimilation may be weakening this protective factor for Latina and Asian women. Specifically, extant literature posits acculturation appears to be responsible for increased rates of drinking among U.S. born young adults who identify as Latino/Latina, Hispanic or Asian-American (Cateano et al., 1998). Researchers
hypothesize that the effects of assimilation and acculturation on non-adherence to cultural values (Hatchett and Holmes, 2004; Warner et al., 2006) may be responsible for the increased alcohol consumption among Hispanic and Asian college women when compared to their Black peers.

**Daily Smoking.** Present findings support the well-established relationship between tobacco use and risk drinking. However, this relationship was found only for the broad categorical measure of high versus low risk drinking. That is, daily smokers were more likely to report high risk drinking than their non-smoking peers. One possible explanation for the absence of this relationship with a continuous outcome includes the use of non-normal data. Despite transformations, the data did not approximate a normal curve. Multiple regression analysis, which was used to identify predictors of the continuous measure of risk drinking is highly sensitive to non-normal data (Tabachnick & Fidell, 2007), whereas logistic regression analysis is able to tolerate the presence of non-normal data (Tabachnick & Fidell, 2007).

When compared to non-smokers, smokers are more likely to report drinking alcohol (Istvan & Matarazzo, 1984; Zacny, 1990), and smokers are more likely to increase the number of cigarettes they smoked if they smoke while drinking (Mello, Mendelson & Palmieri, 1987). Traditionally, many students entered college and initiated smoking sometime during their first year in college. The recent proliferation of smoke-free college campuses, restaurants and bars has been associated with lower rates of cigarette smoking among college students. Despite such overall declines in smoking among college women, studies continue to find an association between cigarette smoking and heavier drinking in college women (McKee et al., 2004; Harrison and McKee, 2008).

In the present study, daily smoking served as the primary indicator of tobacco use and potential nicotine dependence. It was selected over more moderate patterns of tobacco use (i.e.,
less than daily smoking) because daily smokers tend to be more accurate in their self reports of cigarette smoking. Further, it was posited that daily smokers would be more likely to report symptoms of nicotine dependence with higher severity levels. Present study findings affirmed that daily cigarette smokers were more likely to also report high risk drinking. Future studies may want to differentiate between different types of tobacco (e.g. cigarettes, cigarillos, bidis and hookah) when assessing the relationship between daily or frequent tobacco use and risk drinking. As these other types of tobacco become more popular, rates of cigarette smoking in women may decline, while these alternative forms of tobacco use show an increase.

**Negative affect.** Findings from the current study support previous research and demonstrate a statistically significant association between negative affect and risk drinking in this sample of college women. Research with college women has consistently identified a significant relationship between symptoms of depression and anxiety and increased alcohol use (Davidson, 1995; Brown, Inaba, Gillin, Schuckit, Stewart & Irwin, 1995; Schuckit, Tipp, Bergman, Reich, Hesselbrock & Smith, 1997). Researchers have found support for pre-morbid and co-morbid mood and anxiety disorders that have been associated with increases in alcohol use among women (Kessler et al., 1997; Dixit & Crum, 2000; Sanniball & Hall, 2001).

**Summary of Findings Not Supported**

While present findings offer support for the relationships between somatic PMS symptoms, non-minority group membership, tobacco use, negative affect and risk drinking, there were two potential predictors that were supported. These non-significant findings and their implications for the current study as well as other studies will be delineated below.

**Parental history of alcohol problems.** Findings on the influence of family history of alcohol problems on drinking have been inconsistent in a college sample due to variability in
family history methodology, recall bias, and small sample sizes (Baer, 2002). Some studies found no differences in rates of drinking among college students with or without a family history of AUDs (Engs, 1990; Alterman et al., 1989; Harvey & Dodd; 1993; Bogart et al., 1995), while others report that children of parents with AUDs are more likely to endorse higher rates of AUDs than children whose parents do not have AUDs (Kushner & Sher, 1993; Perkins & Berkowitz, 19991; Pullen, 1994). Such variability in the literature warrants additional research to clarify the relationship between family history of alcohol problems and alcohol use among college women.

Results of the present study did not support a relationship between parental alcohol problems and risk drinking. Several factors may account for the lack of association between this predictor and risk drinking. First, a previous study demonstrated that when compared to peers not enrolled in college, college students were less likely to report family alcohol problems (Helmkamp, Hungerford, Williams, Furbee, Manley & Horn, 2000; Perry, 2004). Second, the present study utilized a self-report assessment of family alcohol problems, which is highly susceptible to recall bias. Third, family history of alcohol problems was restricted to parental history. While the research supports the use of parental alcohol problems as a measure of family alcohol problems (Barnes et al., 1992), a family density approach may have increased the likelihood of discovering a significant effect, as more family members’, both distal (e.g. second degree) and proximal (e.g. first degree), alcohol use patterns would have been reported.

Affective PMS Symptoms. While the current study did not find a positive association between affective PMS symptom change and risk drinking among this sample, past researchers noted that women who report symptoms of anxiety and depression or meet DSM-IV criteria for mood and anxiety disorders often endorse symptoms of PMS. When reviewing case studies of alcoholic women, Podolsky’s findings (as cited in Perry, 2004) demonstrated a relationship
between personality traits and increases in premenstrual drinking. He characterized these women as “passive, dependent” personality types (Perry, 2004, p. 27) and their drinking as self-medicating (Perry, 2004). More recently, Ross, Coleman and Stojanovska (2003) found an association between reported symptoms of PMS and neuroticism, a trait often associated with depression, anxiety and anger. After examining the relationship between depression, smoking and PMS symptoms among adolescent girls, Dorn and colleagues (2009), found that adolescent girls who endorsed symptoms of depression and anxiety were more likely to report PMS symptoms when compared to same aged peers who did not endorse mood or anxiety symptoms.

Few studies have examined the specific relationship between affective PMS symptoms and risk drinking. Much of the previous research may have focused on both the somatic and affective symptoms instead of looking at either domain separately. In the current study, affective and somatic PMS symptoms were assessed independently as predictors of risk drinking in this sample. As such, several reasons may account for the absence of significant findings. First, many of the women in the current study endorsed symptoms of negative affect as measured by the PANAS. It is possible that some of the women who reported affective symptoms of PMS may consistently experience negative affect, so they did not notice or report a change in these symptoms. Similarly, as noted in previous research, women who report negative affect symptoms before, during and after the late-luteal phase often endorse PMS symptoms. It is possible that these women may have reported somatic PMS symptoms instead of affective PMS symptoms and that the previous researchers did not assess which PMS symptom items were more commonly endorsed. Because of the strong relationship between negative affect and reports of PMS symptoms, more research is needed to fully understand how affect influences PMS symptoms and then how this relationship affects risk drinking.
Study Limitations

Present findings should be considered in light of several limitations. These include 1) use of a poorly defined construct of race, 2) use of self-report measures, 3) lack of information about socioeconomic and sociocultural influences, 4) limited diversity in sample, 5) the correlational nature of findings and 6) the handling of “missing” and misplaced cases of data. First, the original construct of race was poorly defined and restricted to self-categorization by participants according to socially constructed categories with no conceptual meaning. Because this was a crucial part of the current author’s research question, race should have constructed in terms of cultural practices, beliefs, practices, acculturation and ethnic identity. However, this construct was not a primary measure for the original author’s study. Measuring these constructs instead of relying on a socially constructed definition of race would have allowed the author to conclude which factors may have produced the protective effect against risk drinking among racial/ethnic minority women. However, because this study was a secondary data analysis of previously collected data, the author was limited in how she could meaningfully conceptualize race using the variables available to her.

Second, the research relied primarily on retrospective measures that were self-report. All self-report measures may be affected by recall bias. Research on alcohol use and premenstrual symptoms has discouraged the use of retrospective self-report measures because respondents may unintentionally or intentionally over or underreport information. As a result, use of prospective, timeline follow back or interview methods have been recommended to reduce the effects of recall bias. Third, a measure to assess for socioeconomic differences (e.g. neighborhood disorganization, family income, and use of social services) or sociocultural practices (e.g. on campus culture immersion, religiosity) was not included. Such information
may have identified differences in the sample that were masked. Fourth, the sample size, while initially large, did not include much racial and ethnic diversity. While the numbers present were representative of the University’s student population during the 2002-2003 school year, they were not diverse enough to adequately measure within-group differences among racial/ethnic minority students.

Fifth, the findings, while important and informative, are correlational and not causal. Questions continue to remain about the underlying mechanisms associated with affective PMS symptoms and risk drinking, as well as the relationship between minority group status as a protective factor against risk drinking. Additional research that includes measures of cultural factors, a more representative sample, and fewer retrospective self report measures would strengthen the original methodology and potentially answer some of the questions that remain.

Sixth, because of misplaced raw data on the PANAS, inability to give accurate self report on age of first alcohol use and responses that could not be coded (e.g. “don’t know” responses on the family history of alcohol problems measure), there was a total of 331 missing cases of data across seven independent and two dependent variables. Because of the high number of missing cases, mean imputation and imputation of regressed values were used to provide estimated values for the missing cases. While statistically sound and appropriate for the current sample, these strategies only provide estimates of what a participant might have answered and it is possible that the actual data may have yielded completely different responses.

While the aforementioned limitations are overarching, there are also minor, construct specific limitations that may have influenced this study’s findings. These limitations will be delineated by content area below.
*Premenstrual symptomatology findings.* While findings from the current study support extant data on the relationship between affective PMS symptoms and alcohol consumption, several limitations exist. First, the results of this study are correlational and do not establish a causal relationship between affective PMS symptoms and risk drinking. Second, the current study used retrospective self-report to measure participants’ PMS symptoms. Use of retrospective reporting has been associated with recall bias and over-endorsement of severe PMS symptoms (Paige, 1980; MacFarland, Ross & DeCourville, 1989; Marvan, Cortes-Iniestra, 2001). As a result, the reported severity of PMS symptoms may not be accurate. Third, the retrospective reports only focused the premenstrum phase of a woman’s menstrual cycle, when current research encourages researchers to evaluate women’s drinking across the phases of a woman’s menstrual cycle, as changes in alcohol use may occur during any given phase of the menstrual cycle (Tobin et al., 1994).

Fourth, the majority of research on PMS and the relationship between PMS symptoms and alcohol use has been studied in primarily Caucasian samples. Few studies in this area report samples with enough racial diversity to examine and comment on racial differences in PMS symptoms. For example, in a sample of only 46 women recruited by Svikis and colleagues (2006) for a PMS and alcohol use study, 73% self-identified as White with 22% self-identified as Black (Svikis et al., 2006). Additionally, validation samples for many of the measures used to assess PMS symptoms often lack racial and ethnic diversity. As a result, researchers using these measures run the risk of assessing a construct that may manifest itself differently among various racial and ethnic groups or may not even exist among these groups of women.

Finally, while a measure of negative affect was included, no clinical measures of mood or anxiety symptoms (e.g. Beck Depression Inventory or Beck Anxiety Inventory) were included to
definitively diagnose participants with co-morbid mood and anxiety symptoms in addition to reporting affective PMS symptoms. Without more clinical measures of mood/affective disorders, it is impossible to distinguish between women with co-morbid mood disorders and those whose affective symptoms vary during the premenstrum.

**Age of self-initiated alcohol use.** While the research demonstrates that an earlier age of alcohol initiation is related to alcohol dependence and related problems later on in life (DeWit et al., 2000), there is a difference between age of first self-initiated alcohol use and age of onset for regular alcohol use. The current study asked participants how old they were when they first drank an alcoholic beverage and the information obtained from this question identifies the developmental time period when someone first experiments with alcohol. However, responses to this question do not reveal whether or not someone continued to use alcohol or if they used it once and may have used it regularly at a later age. Research demonstrates that while early self-initiated use is associated with increased rates of alcohol dependence later on, women who began drinking regularly at an older age (e.g. late 20’s) typically develop symptoms of alcohol abuse and dependence during a shorter time period than men who initiate regular alcohol use around this same age (Randall et al., 1999). Overall, age of first self-initiated alcohol use is an important and useful variable because it suggests critical time points for interventions aiming to reduce incidence rates of AUDs. By contrast, when used in isolation, this variable does not provide a researcher much information about whether or not a person will engage in risk drinking. Many researchers assume that because an individual begins drinking during their early teenage years, they will be at greater risk for developing an AUD or experiencing adverse effects from their alcohol consumption. However, additional information, such as age of regular use, could
strengthen the predictive relationship between age of first use and increased risk for engaging in high risk drinking.

**Racial and ethnic minority status findings.** While previous studies have identified a link between genetic and sociocultural factors that interact and protect racial and ethnic minorities from engaging in alcohol use at the level of their White peers, findings from this study are limited because no biological or sociocultural measures were included in the initial battery to assess for the influence of these factors on risk drinking in this sample. Unfortunately, many researchers have made this same mistake when conceptualizing the relationship between race or ethnicity and psychosocial predictors or constructs. Namely, they have assumed that a single item, usually “what is your race or ethnicity” is enough to measure an individual’s level of cultural integration, racial identity and racial socialization. However, race or ethnic categorization provides little information from which to draw conclusions.

Numerous researchers have criticized psychology’s use of race as a poorly defined and then, poorly measured categorical variable that leads to misinterpreted implications. Specifically, Helms, Jernigan & Mascher (2005) characterize the use of race in psychological literature as lacking “consensual theoretical or scientific meaning in psychology” (p. 27) as “some psychologists contend that race refers to biological characteristics as reflected in physical appearance, some argue that it is a pseudonym for impoverished backgrounds… others assert that race is a social construction” (Helms et al., 2005; p. 27). Because of the inconsistent meanings construed by race, Helms and colleagues (2005) propose that researchers interested in examining the relationship of race follow these guidelines: 1) measure aspects of culture, such as beliefs, values, customs and practices; 2) provide a rationale and conceptualization of racial categories that focuses on the attributes of interest (e.g. racism related stress or effects of racial
socialization). Helms and colleagues (2005) posit that the use of these guidelines ground findings in theory and provide meaning to the misidentified relationships that psychologists often attribute to racial differences (e.g. differences in cultural values, perceived discrimination).

Within the area of substance abuse research, several investigators have taken a similar approach to studying the influence of culture, cultural factors, and cultural identity on tobacco and marijuana use among Black adolescents and young adult African American women (Wallace and Fisher, 2006; Nasim, Corona, Belgrave, Utsey & Fallah, 2007). Instead of examining the influence of race on substance use among Black adolescents, Wallace and Fisher (2006) specified three cultural factors from Black American culture that had “been empirically found to be related to outcomes specially for Black Americans” (Wallace and Fisher, 2006; p. 442). These three factors were ethnic identity, racial socialization and extended family support. From these constructs and others, Wallace and Fisher (2006) were able to report that children with higher scores on a measure of ethnic identity reported disapproving attitudes towards substance use and that children who received socialization on discrimination buffered by messages about racial pride, were also less likely to endorse favorable attitudes towards substance use. By focusing on attributes of what other researchers conceptualize as “race”, Wallace and Fisher (2006)’s findings highlight cultural factors that may specifically provide protection against substance use.

Nasim and colleagues (2007) focused their research question on cultural orientation and practices as protective factors against smoking. Cultural orientation was conceptualized as level of immersion in African American culture (e.g. engagement in practices and beliefs), family interdependency, and cultural interdependency (Nasim et al., 2007). Results suggested that non-smokers (tobacco and marijuana) reported the following protective factors: cultural immersion
(e.g. traditional family values and religious beliefs and practices) and family interdependency (Nasim et al., 2007). Once again, a specific conceptualization of cultural factors led to factors to go beyond identifying how “race” influenced smoking behaviors.

In the present study, race was included as a way to measure the racial/ethnic representativeness of the sample and to compare it demographically with statistics for the undergraduate student body. The author of the current manuscript initially conceptualized race as a way to demonstrate the influence of cultural variation on psychosocial predictors of risk drinking in a sample of college women. However, no measures of cultural factors (e.g. ethnic identity, religiosity, acculturation, individualism/collectivism) or measures indicating the effects of discrimination or disadvantage (e.g. SES, neighborhood disorganization, cultural mistrust) were included in the initial battery, so the findings of this study are ambiguous, as they do not identify cultural factors that may account for the observed differences in risk drinking. Additionally, because the final sample size (n=194) lacked ethnic representation, and self-reported racial/ethnic categories were more similar than not, the author of the current manuscript created a minority group so that between-group analyses could be completed.

In spite of these limitations, the present study possessed several strengths. To-date, it is one of few studies that has examined psychosocial predictors of risk drinking and minority status as a moderator of the relationship between affective PMS symptoms and risk drinking. Findings from this study may assist future researchers with improving the methodology used to assess racial health disparities and “racial” differences in substance abuse research. Finally, study findings may improve methods used to identify women at risk for AUDs and their consequences.
Implications and Future Directions

The present study was novel and made a unique contribution to the literature in several ways. First, by using “risk drinking” as the outcome measure and looked at categorical and continuous measures of this construct. Many previous studies focus on binge drinking, problem drinking or drinking-related consequences. Risk drinking was chosen because it identifies individuals whose current level of drinking may lead to immediate or delayed adverse consequences. As such, risk drinkers are often targets for brief interventions that hope to reduce risk and prevent longer-term consequences. Second, this study examined subcategories of PMS symptoms (e.g. somatic and affective) separately. Many researchers have not looked at the two categories of symptoms separately, but by isolating them in this study, the author was able to demonstrate a significant relationship between somatic symptoms of PMS and risk drinking in the absence of a significant relationship between affective PMS symptoms and risk drinking.

Another implication is the identification of risk factors associated with risk drinking among this population. The findings from this study suggest that women who report somatic PMS symptoms, especially White women, may be more likely to engage in high-risk drinking, or drinking with a greater likelihood of resulting in either immediate or delayed adverse consequences. Additionally, minority group status was identified as a protective factor. Because minority group status was identified as a protective factor among this age group, it is possible that future studies should focus on identifying ways to maintain this protective factor. Further research is warranted to investigate these relationships, as the findings from this study are preliminary and do not infer causation.
Finally, because minority status was identified as a protective factor, more research is needed to identify correlates of this protective factor. Additionally, as previously mentioned, only one item assessed “race”. Future studies, especially those that seek to understand the development of racial/ethnic health disparities, should include measures of racial/ethnic identity, cultural identity, acculturation, discrimination/racism, social disadvantage and sociocultural factors.

Despite the correlational nature of the study, the study findings do have practical implications. For example, future researchers and clinicians could use these risk factors, especially the somatic PMS symptoms and age of first self-initiated alcohol use to identify women who may benefit from alcohol use screenings and brief interventions at primary care and OB-GYN appointments. Additionally, by differentiating affective from somatic symptoms of PMS, future researchers may be able to understand which set of symptoms has a stronger correlation with increased alcohol use.

In summary, results of the current study suggest that age of first self-initiated alcohol use, daily smoking, negative affect and somatic PMS symptoms were associated with risk drinking among college students. Minority status was identified as a moderator of the relationship between somatic PMS symptoms and risk drinking among college women. Specifically, for women identifying as racial and ethnic minorities, minority status functioned as a protective factor, while White women with somatic PMS symptoms were more likely to report engaging in high-risk drinking. Results of the current study did not support associations between parental history of alcohol use and risk drinking. Results are correlational and not causal in nature. Additional research is warranted to determine the protective effect of minority status and to fully understand the relationship between somatic PMS symptoms and risk drinking.
References
References


doi:10.1080/09595230600741339


2002, 37, 87–92.


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sample in Germany. *Addiction, 95*, 1389-1401.


National Institute of Alcohol Abuse and Alcoholism (1998). *Age of Drinking Onset Predicts*


use among college students. *Educ. Econ.*, 12, 135-149.


Substance Abuse and Mental Health Services Administration, Office of Applied Studies. (June 24, 2010). *The NSDUH Report: Substance Use among American Indian or Alaska Native Adults*. Rockville, MD.


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