

Caffeine is often consumed in efforts to offset behavioral decrements produced by alcohol intoxication (Liguori & Robinson, 2001). Drinking a cup of black coffee to sober up best exemplifies this belief. The concept of caffeine antagonizing alcohol's deleterious effects was first presented in research dating back to the early 1900s (Mackay, Tiplady, & Scholey, 2002). Currently, the effectiveness of caffeine's ability to counteract alcohol's effects is generally considered a myth (Fudin & Nicastro, 1988). However, several studies do indicate that caffeine can antagonize alcohol's effects on some tasks of psychomotor performance (Burns & Moskowitz, 1990; Hasenfratz, Bunge, Dal Pra & Battig, 1993; Rush, Higgins, Hughes, Bickel & Wiegner, 1993; Hasenfratz, Buzzini, Cheda & Battig, 1994; Mackay, et al., 2002). Mixed results prevent conclusive interpretation. However, even though it is unclear that caffeine can antagonize alcohol impairment, people still consume both drugs in combination. One possible explanation for why people attempt to "sober up" with caffeine may be related to the propagation of this idea by the media; alternatively, caffeine may change one's perceptions of sobriety via the activation of expectancies or via pharmacological mechanisms. The present study examines the interactive effects of caffeine and alcohol consumption on people's perception of sobriety.

#### HISTORY OF ALCOHOL

As summarized by Maisto, Galizio, and Connors (2004), alcohol is one of the first documented drugs to be used by humans. Its discovery is likely to have been accidental. Consumption of fruit juice contaminated with yeast and microbes is thought to have resulted in the first intoxication (Edwards, 2002). The first wine was documented in early Egyptian times, around 5000 to 600 B.C. Early preparation consisted of filling earthen containers with barley. These containers were placed underground until a germination process occurred. The barley was

then exhumed, crushed and baked into a cake. The resulting cake was then soaked in water until fermentation was complete. This early acidic beverage was called “boozah” (Maisto, et al., 2004).

The production and use of distilled spirits was documented around 1000 B.C. in China. In Europe around 800 A.D., the discovery of distillation increased the potency of alcohol and its popularity around the world (Pittman & White, 1991).

Excessive alcohol consumption became particularly prevalent in Europe. This problem was so engrained during this period that it was depicted during various artwork of the time, such as Hogarth’s “Gin Lane” (Maisto, et al., 2004). As immigrants left Europe in response to British rule, the problems associated with alcohol were quickly inherited by colonial America. Many historians believed that the pilgrims landing on Plymouth Rock was no more than the byproduct of a glorified “beer run”; they stopped because they were out of alcohol (Lender & Martin, 1982).

As discussed by Maisto et al. (2004), alcohol was a central economic staple in new world colonies. Taverns were central locations in which pleasure, politics and business was conducted. By 1830, average alcohol consumption had risen to seven gallons per capita, or roughly five standard drinks per day. During the nineteenth century America began to expand westward. Expansion resulted in increased economic opportunities, partially due to the ease of opening alcohol-serving establishments.

Originating from the French word salon, saloons became a popular American icon in the 1800s. In England, they were public meeting places. Its American counterpart served as a means to serve massive quantities of alcohol. Initially, saloons were fairly primitive structures, shacks or tents for which a row of barrels was used as a bar. This setup allowed for an individual to enter this business with minimal investment. Such cheap and potentially lucrative

establishments of the time added to their proliferation. With such high economic turn over, the saloon evolved into a social institution, which became an icon in America. In fact its popularity became so great that saloons became forums where meetings and business took place. The significant societal and economic presence of the saloon during this time set the stage for modern alcohol consumption.

Trends in alcohol consumption have varied since then. Beginning in 1935, the federal government has taken an active role in tracking and examining alcohol use prevalence rates. Alcohol consumption across all age groups has become a salient social concern. One of the most common government measures examines per-capita alcohol consumption, or how many drinks the average person drinks. Census measures have examined how many gallons per year, individuals fourteen and older have consumed. The consumption analysis has been examined for every type of alcoholic beverage, beer, wine and hard liquor from 1935 to 2001. According to a 2001 survey, conducted by the National Institute on Drug Abuse (NIDA), 9.2% of males and 2.6% of females in the United States reported heavy alcohol use per month (NIDA, 2001). Heavy alcohol use was defined as five or more drinks per occasion in the last 30 days. Self-reports of heavy use were also examined for individuals below the legal drinking age. It was found that, 3.1% of males and 1.9% of females between the ages of 12 to 17 years reported to have consumed alcohol in the past 30 days. Other studies have also supported that U.S. citizens consume a considerable amount of alcohol each year. Although amounts vary for different ethnicities and age groups, individuals of 18 to 25 years of age are particularly vulnerable to the effects of alcohol, and its potential for abuse and misuse. Trends in alcohol consumption across all age groups have become a salient social concern; it is often a primary factor in the incidence of traffic accidents, suicide, and spousal abuse (Pittman & White, 1991).

## ALCOHOL PHARMACOLOGY

Alcohol is most commonly consumed in beverage form, where it travels from the stomach to the small intestine and is absorbed into the bloodstream. The circulation of alcohol in the bloodstream alone is not sufficient to produce intoxication. Its chemical structure allows as much as 90 percent to cross the protective glial sheath known as the blood-brain barrier (Julien, 1992).

Alcohol exerts depressant effects on the Central Nervous System (CNS). A prominent hypothesis is that alcohol works on gamma-aminobutyric acid (GABA) systems in the brain (Maisto, et al., 2004). In particular, alcohol is thought to alter GABA-mediated neurotransmission.

GABA is an inhibitory neurotransmitter found throughout the brain and spinal cord. It is essential for creating inhibitory neuronal responses. The activity of this neurotransmitter is responsible for a variety of behaviors including motor coordination, sedation and anxiety relief (Mihic & Harris, 1997). The presence of GABA in the central nervous system is critical for mediation of neuronal firing. For example, some researchers hypothesize that abnormalities in GABA release can lead to over excitation of neuronal firing, resulting in disorders such as Epilepsy (Carlson, 2001). Currently, two types of GABA receptors have been identified in the human brain, GABA<sub>A</sub> and GABA<sub>B</sub> (Valenzuela, 1997). GABA<sub>A</sub> has perhaps shown to play the most critical role in explaining alcohol pharmacology. This receptor is an ion channel forming protein; it permits the passage of chloride ions into a cell. Once negatively charged chloride molecules cross a cell's membrane, a hyperpolarization results in a decrease in the decrease cell's excitability. The functional effect is sedation and intoxication.

As summarized by Mihic and Harris (1997), the GABA<sub>A</sub> receptor is comprised of three subunit groups  $\alpha$ ,  $\beta$ , and  $\gamma$ . Although the exact compositions of the subunits are unknown, the activity of each subunit shows binding specificity. For example,  $\alpha$  and  $\gamma$  subunits are activated by benzodiazepines. The specific activity characteristic of each subunit determines the overall characteristic of the GABA<sub>A</sub> receptor. Medications such as benzodiazepines and barbiturates act on GABA<sub>A</sub> receptors and increase receptor effects on the neuron, resulting in anxiolytic and anesthetic properties. Although the GABA theory is the most prominent theory of alcohol intoxication, knowledge of alcohol pharmacology is far from complete.

#### THE EFFECTS OF ALCOHOL ON PERFORMANCE

The depressant effect of alcohol is a function of BAC, it is typically expressed as a ratio of weight of alcohol per 100 units of blood volume (Sobell & Sobell, 1981) or, milligrams of alcohol per 100 milliliters of blood. The legal limit of intoxication for most countries varies from .08 to .10% BAC (Pittman & White, 1991). Hand-held breathalyzers are the most accurate methods to assess intoxication, however BAC can also be estimated in the following equation:  $BAC = \text{number of standard drinks} \times (.025\%) - \text{number of drinking began} \times (.015\%)$  (Maisto, et al., 2004). On average, one standard drink typically increases BAC by .025%, within 45 to 60 minutes of drinking. Depressant effects of alcohol are more evident at higher BAC concentrations, leading to a gross decrease in motor ability.

Evidence suggests that moderate and high doses of alcohol produce significant effects on an array of tasks involving divided attention, information processing, learning, and reaction time (Mackay, et al., 2002). Alcohol also produces decrements in tasks that require divided attention (Maisto, et al., 2004). Scholey (2004) reports increased error rates are most commonly observed at higher BACs. Using a within group design, participants intoxicated at a .08% BAC had 50%

increased error rates on the Gibson Spiral Maze task versus control condition. While the effects at low doses are not as robust, some suggest low doses can produce detriments in driving behavior and performance on complex tasks (Osborne & Rogers, 1983).

A variety of tasks including reaction time tasks and driving simulator tasks have been used to assess alcohol's detrimental effects on behavior. Generally impairment is positively correlated with BAC. Beginning in 1950, Bjerver and Goldberg demonstrated that alcohol intoxication of .04 to .06% BAC impaired driving ability. However deterioration in driving performance has been observed at BACs as low as .02 - .03% BAC, well below the legal limit of intoxication (Drew, Colquhoun, & Long, 1958; Levine, Kramer, & Levine, 1975). Maisto and others (2004) report abnormalities in psychomotor functioning at BACs of 0.15% and higher.

When compared to the no alcohol control, Liguori, D'Agostino, Dworkin, Edwards and Robinson (1999) reported a .10% BAC significantly increased braking latency in driving simulators. The same was also found at .06% BAC, which is below the legal limit of driving intoxication in all states.

Although it is a common belief that alcohol intoxication may at least be partially counteracted by caffeine, this belief has not been supported consistently by previous findings. Results are mixed at best. Conflicting results may be due to the gross variability of dependent measures and drug doses used in testing (Azcona, Barbanoj, Torrent & Jane, 1995). Many studies lack adequate control of confounding variables including body weight, and drug history (Liguori & Robinson, 2001; Marsden & Leach, 2000; Rush, et al., 1993). However Filmore and Vogel-Sprott (1994) suggest that alcohol-caffeine performance may be independent of pharmacological effects and may depend more upon drug expectancy. Vicarious learning is one source in which to acquire such expectancies. For example, television characters are often

portrayed drinking a cup of black coffee in an effort to sober up quickly (Fudin & Nicastro, 1988). Expectancies are most often acquired when a consumed drug is reliably associated with a particular response (Vogel-Sprott, 1992). In the case of alcohol and caffeine, subjective intoxication may contribute to drug expectancies, thereby influencing their interactions. The purpose of this study is to examine subjective intoxication as a result of alcohol and caffeine consumption.

### HISTORY OF CAFFEINE

Children and adults commonly use caffeine for non-medical purposes. It is found in a variety of products including, coffee, carbonated drinks, desserts, and medications. Many have argued that it is the most widely used drug in the world; coffee, tea and cola, the world's most popular drinks all contain caffeine (Weinburg & Bealer, 2001). Caffeine's popularity as a food additive makes estimates of use difficult. However, Americans consume approximately 211 milligrams of caffeine per capita, usually in beverage form (Maisto, et al., 2004).

The exact origin of caffeine use is unknown, however according to Weinberg and Bealer (2001) an Ethiopian man, named Kaldi first observed the effects of caffeine on goats. According to the legend, he awoke in the middle of the night to find his goats jumping. He later noticed that they had been eating coffee beans. After linking the two events, he attempted to use the beans in order to help him stay awake during long prayer vigils. This may have been the prelude to the first cup of coffee.

The biological origin of caffeine is arguably the result of an evolutionary adaptation in coffee plant; it is a natural pesticide, producing sterility in many insects. Caffeine can also permeate the soil and inhibit the growth of locally competing plants and fungi. This natural defense is so potent however that the chemical may kill the coffee plant itself. This phenomenon

partially explains why coffee plantations degenerate every ten to twenty-five years (Weinberg & Bealer, 2001).

The coffee plant is a tropical evergreen that belongs to the *Coffea* genus. This family is comprised of 25 species of coffee plants, which thrive in the tropics of the eastern hemisphere. The plant that is prevalently used for today's cup of coffee is known as *Coffea arabica*, or coffee shrub of Arabia. Although indigenous to Ethiopia, 18th century European traders first introduced the plant to Latin America and today it accounts for one of Latin America's primary exports and 75% of coffee consumption worldwide (Weinberg & Bealer, 2001).

## CAFFEINE PHARMACOLOGY

Upon digestion, caffeine is quickly absorbed. As summarized by Julien (1992), significant caffeine blood levels are typically achieved in 15-45 minutes after the drug is taken. Complete absorption is estimated at 90 minutes and its half-life ranges from 2.5 to 7.5 hours. Peak caffeine levels may partly depend on the consumed beverage source. Using 155 milligrams of caffeine, Marks and Kelly (1973) report men's peak caffeine plasma levels were achieved within 30 minutes in coffee and tea, whereas the peak for Coca-Cola did not occur for an hour. However, another study reports no time differences in peak caffeine plasma levels in coffee, cola drinks, and caffeine capsules (Liguori, Hughes, & Grass, 1997). In human beings, caffeine metabolism is influenced by many factors; it has been shown that liver disease, pregnancy, and the use of oral contraception can slow this process, whereas cigarette smoking can potentiate absorption.

Caffeine acts as a CNS stimulant. It first affects the cortex tissue, resulting in cerebral-cortical stimulation of the brain stem (Julien, 1992). Activation of these sites can produce mental alertness and reduced fatigue.

The mechanisms by which caffeine operates are complex and are still unclear. The Adenosine Blockade theory is currently the dominant theory, used to explain caffeine's on neurotransmitters. Caffeine's ability to antagonize adenosine via the A<sub>2</sub> receptor is thought to play a role in alcohol interaction (Yacoubi, Parimeter, Costentin & Vaugeois, 2003). It can attenuate alcohol's depressant effects, partially substantiating the belief that a cup of coffee can antagonize intoxication.

Adenosine is a neuromodulator and exerts its effects by decreasing the rate at which neurons fire. It also may have indirect inhibitory actions by reducing the production of

neurotransmitters that cause excitability. Caffeine is recognized as an antagonist for A<sub>1</sub> and A<sub>2</sub> adenosine receptors (Olah & Stiles, 2000). In other words, caffeine can block the adenosine receptors, which then prevents adenosine uptake; thereby preventing adenosine's normal hypnotic-sedative effects (Yacoubi et al., 2003). The competitive antagonism of adenosine receptors may then result in increased alertness and mood.

#### THE EFFECTS OF CAFFEINE ON PERFORMANCE

Although it is most commonly consumed in a cup of coffee, caffeine is used as an additive in products such as sodas, chocolate candies, yogurts, and cereals. Many medications also contain caffeine including analgesics, cold remedies, and appetite suppressants. Alert aids have as much as 200 mg per capsule (Weinberg & Bealer, 2001). The pervasive use of caffeine has stimulated much research on its effects on human behavior. Overall, literature on caffeine's effects on human behavior has reported mixed results (Smith, Rusted, Eaton-Williams, Savory & Leathwood, 1990; Loke, 1990). However there is reliable evidence that caffeine increases vigilance and reduces fatigue.

Caffeine's apparent effect on vigilance has become of particular interest of the United States military. The United States Army Institute of Environmental Medicine recently examined the effects of caffeine on vigilance and sleep deprivation (Bovill, Tharion, Lieberman, 2003). Sixty-eight US Navy SEAL trainees were sleep deprived for 72 hours and randomly assigned to three doses: 100, 200 or 300 mg of caffeine. At 200 and 300 mg, it was found that caffeine significantly improved reaction time, visual vigilance, and alertness. Others have also reported similar findings. A recent study reports that after sleep depriving participants for a continuous 64 hours, a 600 mg dose of caffeine was found to antagonize cognitive impairments due to profound sleep deprivation (Beaumont et al., 2001).

Caffeine's effect on behavior has been also been documented at far lower doses. For example, Durlach (1998) assessed caffeine's effect on cognitive performance at 60 mg, the typical caffeine dose of one cup of tea. Participants in this study viewed complex visual patterns and were then required to match it to one of four corresponding patterns. Despite administering one tenth of the amount of caffeine used in the previously mentioned studies, significantly faster reaction time was observed in pattern recognition, delayed match to sample and visual search tasks.

The degree to which caffeine enhances cognitive performance may vary with a variety of factors. For example, performance on simple reaction time and visual search tasks is enhanced in individuals with higher levels of habitual caffeine use (Smit & Rogers, 2000). Personality characteristics may mediate effects; extroverts appear more likely to benefit than introverts (Gupta, 1988). Other studies indicate that impulsiveness can produce differential caffeine effects (Gupta, Singh, & Gupta, 1999). When compared to placebo controls, caffeine reliably improves the performance of impulsive individuals more than non-impulsive participants.

Caffeine's effects additive effects on performance are also probably dose-dependent. Osborne and Rogers (1983) suggest performance on reaction time and vigilance tasks increase with 80 to 200 mg doses, whereas doses of 500 mg and up impair performance (Rees, Allen, & Lader, 1999).

## ALCOHOL AND CAFFEINE PERFORMANCE INTERACTIONS

The reinforcing effects of caffeine and alcohol make them two of the world's most used drugs and may be one reason that they are often consumed together. The fact that caffeine acts as a stimulant, whereas alcohol is a depressant is likely to have led to its use as a home remedy for alcohol intoxication. It is a popular belief that caffeine can antagonize the intoxicating effects of alcohol (Hasenfratz, et al., 1993). For example, television media often portrays characters sobering up after drinking a cup of black coffee, even though there is a lack of conclusive evidence that this is effective for "sobering up" (Fudin & Nicasastro, 1988). Although separate physiological and behavioral effects are known both alcohol and caffeine, a better understanding of their combined effects presents a variety of practical implications.

Pilcher (1911) was among the first pioneers who examined the interaction effects of alcohol and caffeine. Using cats, he observed that interaction of the two drugs resulted in both synergy and antagonism. In particular, the behavioral effects of low doses of alcohol were antagonized, whereas, larger doses of alcohol and caffeine produced synergistic behavioral decrements on a complex performance task.

Subsequent methods to examine alcohol and caffeine interaction have varied. For example, Osborne and Rogers (1983) used a microcomputer to measure simple reaction time to letter stimuli. Because caffeine is likely to be consumed with alcohol in preparation for driving, others have examined interactions of alcohol and caffeine in simulated driving tasks (Fudin & Nicasastro, 1988). Using a randomized, double blind, within subject design, Liguori and Robinson (2001) examined the effects of 0, 200, and 400 mg caffeine capsules on brake latency and performance in nine females and six males intoxicated at .08% BAC. Latency was measured as the amount of time to press a brake pedal following the appearance of a fence across ten trials. The examined caffeine doses were relatively high and were simultaneously administered with

alcohol. It was reported that both 200 and 400 mg of caffeine significantly decreased brake latency, yet this does not necessarily mean better driving when compared to the administration of alcohol alone. Brake latency, is only one of a number of behaviors required to operate an automobile. Practical interpretations are further cautioned, despite caffeine's apparent antagonism of alcohol effects, brake latency was still on average 9% longer when compared to placebo controls, which may prove to be fatal in real-life situations.

Many studies have examined the effects of alcohol and caffeine on test batteries that may be related to driving. When compared to alcohol alone, Burns and Moskowitz (1990) found a 4.4 mg/kg caffeine capsules administered ten minutes after a 0.58 g/kg dose of alcohol dose reduced average number of errors and increased accuracy in Compensatory Tracking, Divided Attention and Critical Tracking Tests.

Rush et al. (1993) administered three doses of alcohol (0, 0.5 and 1.0 g/kg) and caffeine (0, 250, 500 mg/70 kg) alone and in combination to eight adult humans in a double blind repeated measures design. Participants received all possible combinations twice; performance was examined with a Digit-Symbol Substitution Test (DSST), and Repeated Acquisition and Performance task. Caffeine was consumed sixty minutes prior to alcohol. When compared to alcohol alone, caffeine significantly offset alcohol related performance decrements on both tasks. A more recent study also supports evidence for antagonism on DSST and reaction time tasks. Mackay, Tiplady, and Scholey (2002) administered alcohol (0.66 g/kg) and caffeine (110-120 mg), both or neither to sixty-eight healthy volunteers. Caffeine was consumed twenty minutes after alcohol, in the form of a 170 ml cup of Nescafe Original coffee; decaffeinated coffee was used for the no caffeine condition. The combination of caffeine and alcohol reduced the number of DSST errors compared to alcohol alone.

Other researchers have found inconsistent alcohol and caffeine interaction effects. For example, Marsden and Leach (2000) investigated the effects of alcohol and caffeine on maritime navigational skills. When consumed independently, both drugs produced opposite effects on performance; caffeine enhanced performance, whereas alcohol produced impairments on both visual search and navigational tasks. When taken together, the combination of both substances did not produce any differences in performance, when compared to the alcohol alone, suggesting that caffeine does not antagonize alcohol.

The drug classification of alcohol and caffeine intuitively produces a hypothesis inferring that their combination may suggest an antidote for intoxication; however, there exists evidence that is contrary to this reasoning. Many studies suggest that caffeine does not ameliorate alcohol impairment (Fudin & Nicastro, 1988; Nuotto, Mattila, Seppala, & Konno, 1982). There is some evidence that caffeine exaggerates the impairing effects of alcohol. Osborne and Rogers (1983) administered alcohol (2.2 ml/kg and placebo) followed by caffeine (150 mg in coffee and decaff) to eight subjects (4 males and 4 females). Each participant was tested once a week, over a four-week period. Subjects were required to memorize sets of four memory sizes (1,2,3, or 4 letters). After each of the test stimuli were memorized, subjects determine if stimuli were shown in the previous set. The combination of alcohol and caffeine significantly increased mean reaction time, when compared to alcohol and no caffeine.

Evidence for and against antagonism may suggest that the interaction effects of alcohol and caffeine are dosage-dependent and task-specific. For example, Azcona, Barbanoj, Torrent and Jane (1995) claim that an antagonism will occur only in tasks in which coffee is known to enhance performance, such as simple reaction time and vigilance. Order effects may partially explain discrepancies in alcohol and caffeine interaction. Several researchers theorize that

evidence for behavioral antagonism is found only when caffeine is consumed prior to alcohol (Hasenfratz, et al., 1994).

The lack of consensus regarding alcohol and caffeine antagonism may be a result of a variety of issues, such as differential doses of the drugs used, administration, lack of biological screening of drug intake, differential tasks used in experimentation and drug expectancy effects. The examination of both alcohol and caffeine solely at a pharmacological level is inadequate to understand their combined effects; this has lead researchers to examine drug expectancy as an issue. When taken separately, placebo doses of both drugs can produce different effects on psychomotor tasks. As demonstrated by Fillmore, Mulvihill and Vogel-Sprott (1994), psychomotor performance can be altered by the expectancy of drug effects, rather than the direct pharmacological effect of the drugs. Fifty male participants were randomly assigned to placebo or no-treatment groups and performed twelve trials on a pursuit rotor task. Prior to testing, each group received information regarding the expected drug effect on the task. The caffeine placebo (C+) and alcohol placebo (A+) were led to expect enhanced performance, whereas C- and A- expected impaired performance. It was found that placebo caffeine improved performance only when performance enhancement expectancy was activated. Placebo alcohol however, demonstrated the opposite effect; psychomotor performance deteriorated when the performance enhancement expectancy was activated.

A follow-up study using a 0.56 g/kg dose of alcohol and a 2.93 mg/kg dose of caffeine indicate that regardless of drug condition, participants who expected the most impairment, performed the most poorly (Fillmore & Vogel-Sprott, 1994). When alcohol and caffeine were combined, it has been found that participants who expect the most psychomotor impairment, performed most poorly (Fillmore & Vogel-Sprott, 1995).

One interpretation of these findings is that caffeine may antagonize alcohol-related effects based upon an individual's expectations of their combined effects. However, impairment is likely to be dominated by the expectancy of alcohol impairment alone, drinkers who expect an antagonistic effect of caffeine are less likely to compensate for alcohol impairment (Fillmore, Roach, & Rice, 2002). These data suggest that evidence for antagonism relies less on interactive pharmacology of both drugs, but more on perception (i.e. drug expectancies).

### PERCEPTIONS OF INTOXICATION

There a number of ways to assess intoxication levels empirically in humans. Despite the ease of use of most BAC analyzers, few individuals employ such tools to examine their own intoxication. Instead, they rely on their own self-perceived level of impairment as a component for decision-making during intoxication. Although self-perception of intoxication is not a precise measure of sobriety, it is often the only indicator through which individuals can assess their ability to accomplish tasks such as driving successfully (Nicholson, Wang & Mahoney, 1994).

Perception of the intoxication of other individuals also plays a role in alcohol-related decision making. This concept is especially salient in law enforcement, since alcohol consumption is a factor in many crimes (Pagano & Taylor, 1979). Police officers must often judge a person's intoxication in an array of settings. The deployment of field sobriety and breath tests is generally dependent on an officer's perception of intoxication. Pagano and Taylor (1979) examined officer's accuracy to assess intoxication. Officer's subjective assessments of a person's intoxication were compared to actual BAC measurements of volunteers. Thirty-six college students were randomly assigned to a low (.04%) or a high (.08%) alcohol condition. Using a 5-point Likert scale, officers, made three consecutive assessments of the two groups of

students. Results indicated that officers were moderately accurate at judging students in the low condition, however they grossly underestimated the degree of intoxication of high alcohol subjects.

Other studies have examined perception of intoxication using only breath odor (Moskowitz, Burns, & Ferguson, 1999). Police officers were asked to estimate the degree of intoxication of an individual blowing in to a plastic tube, standing behind a screen. During these ideal laboratory conditions, officers were able to accurately detect alcohol only two-thirds of the time for individuals under .08 BAC and 85% for participants over .08 BAC. After participants consumed food, correct breath odor detection fell below chance levels.

The degree to how well individuals can accurately estimate their own level of intoxication has also been examined. Researchers interested in adults convicted of driving under the influence (DUI) have been particularly interested in this issue. In fact, Lewis and Merz (1995) used the Microcomputer-based Assessment System (MAS) to examine DUI convicts' perceptions of their own intoxication at the time of arrest. It was found that when compared to measured BAC at the time of arrest, convictees made generally accurate estimates of intoxication. Yet, despite this positive relationship, they still chose to drive.

Several studies have examined the relationship of BAC and perceived intoxication in a more immediate manner. Werch (1990) examined the perceived intoxication of 156 individuals at various drinking establishments. After a BAC breath sample was obtained, a survey was administered. Participants indicated how intoxicated they felt on a scale of 0 to 30, with 0 being cold sober and 30 representing feeling very high. Perception of intoxication was highly correlated with BAC ( $r = .52, p < .001$ ). It was also reported that the higher the BAC, the less accurate the judgment of perceived intoxication. Even though greater alcohol consumption was

associated with greater perceived intoxication it was also associated with greater errors in estimating intoxication. Individuals with higher BAC typically underestimated their intoxication.

A number of other studies support positive linear relationships between perceived intoxication measured by questionnaires and actual BAC readings (Moss, Yao, & Maddock 1989; Jones & Neri, 1985, Lex, Greenwald, Lukas, Slater, & Mendelson, 1988). Several authors propose perceived intoxication is a greater predictor of alcohol impairment than BAC (Nicholson, Wang, Collins, Airhihenbuwa, Mahoney & Maney, 1992). In this study it was found that perceived intoxication throughout the experiment was correlated more closely with reaction time than BAC, suggesting there is a link between psychomotor performance and perceived intoxication. The validity of perceived intoxication is potentially so strong that many alcohol educators suggest that this be incorporated into alcohol education curriculum (Nicholson, et al., 1994).

As with all subjective decisions, errors exist in BAC estimation or the perception of one's intoxication. These types of errors can be classified into three groups, mixed pattern, overestimators and underestimators (Beirness, 1987). Overestimators are individuals who estimate their intoxication in excess during both absorption and elimination phases of the BAC curve. Underestimators tend to chronically undershoot their own BAC, especially during the elimination process. Those in the mixed pattern tend to overestimate during absorption and underestimate on the ascending limb on the BAC curve.

Few studies examine the subjective effects of alcohol or caffeine. Despite the overwhelming belief in the myth of caffeine as a sobering agent, fewer studies have directly examined subjective reports of combined alcohol and caffeine effects in a laboratory setting (Fudin & Nicasastro, 1988; Rush, et al., 1993; Ligouri, & Robinson, 2001; Maisto, et al., 2004;

Burns & Moskowitz, 1990; Marsden & Leach, 2000; Osborne & Rogers, 1983). However, one study used visual analog scales to assess perceived intoxication. The authors reported no change in subjective intoxication as a result of caffeine (Liguori, & Robinson, 2001; Rush et al., 1993). Inconsistent findings may be attributed to several confounds: Liguori, & Robinson (2001) administered 200 and 400 mg caffeine doses, however they failed to control for body weight. The ecological validity of the caffeine doses is questionable; participants received 0, 200, or 400 mg capsules, which is roughly equivalent to 2 to 3 standard cups of coffee (Maisto, et. al, 2004). The method used for drug administration may also reduce validity; caffeine was administered in capsule form and was consumed immediately with alcohol. Rush et al. (1993) only examined high caffeine doses. Participants received 0, 250 or 500 mg/kg in capsule form 60 minutes before alcohol administration. When caffeine is used as a sobering agent, it is usually consumed after alcohol intoxication, in a cup of coffee.

A better understanding of subjective intoxication as a result of caffeine and alcohol consumption with ecologically doses and administration can provide researchers insight as to why the social myth of using caffeine to sober up exists so pervasively in American society. If it is the case that intoxicated individuals perceive themselves as being more sober after a cup of coffee, this may partly explain why individuals choose to drive while under the influence of alcohol. Also, if caffeine can counter some of alcohol's effects on cognition, then perhaps subjective intoxication is independent of motor decrements.

## HYPOTHESIS

Within alcohol and caffeine research fields, there are a number of studies pertaining to the interaction of drugs on performance, however, currently few thoroughly examine perceptions of sobriety as a result of their interaction at varying doses. The goal of the present study was to

examine the interactive effects of alcohol and caffeine on perceived intoxication. Caffeine is often consumed in efforts to offset alcohol intoxication (Liguori & Robinson, 2001). Therefore, it was predicted that participants would perceive themselves more sober after consuming a cup of caffeinated coffee. This finding was also expected for alcohol placebo, previous literature indicates the alcohol placebo method used in this study is effective in making participants believe they received alcohol (Noel, Lisman, Schare, & Maisto, 1992). Many attempts have been made in this study to control for factors that may have contributed to mixed results of previous studies. In caffeinated coffee conditions, administering a 2.0 mg/kg dose of caffeine controlled dose and body weight. Participants were tested on weekday evenings to minimize time of day effects. A pre-experimental questionnaire excluded participants that used psychoactive substances 24 hours prior to testing, and a pre-experimental breath test ensured a .00% BAC.

## METHODS

### Design

The present study used 2 X 4 repeated measures design. The independent variables were alcohol (water, placebo, .04, and .08), and caffeine (placebo [C-]: 8 ounces of decaffeinated coffee and [C+]: 2.0 mg / kg of anhydrous caffeine dissolved in 8 ounces of decaffeinated coffee). Participants were administered an intoxication questionnaire three times throughout the study.

### Participants

Participants used in this study came from a larger study funded by a grant from the National Institute on Alcohol and Alcohol Abuse (NIAAA) (1-R01-AA13471 Noel, et al., 2004). One hundred and thirty-three males between the ages of 21 and 30 were recruited via posters,

flyers and newspaper advertisements in the Wilmington, NC area. Prior to eligibility, participants were administered a preliminary questionnaire which screened for drinking, drug and psychiatric problems. Those eligible were compensated \$15 per hour and were randomly assigned in a double fashion to one of the following eight conditions: water / caffeine, water / no caffeine, alcohol placebo / caffeine, alcohol placebo / no caffeine, .04 alcohol / caffeine, .04 alcohol / no caffeine, .08 alcohol / caffeine, and .08 alcohol / no caffeine. The experiment was conducted in the evenings at an alcohol research laboratory located in the psychology department at the University of North Carolina at Wilmington. The University Institutional Review Board approved this project.

## Materials

Informed consent forms (Appendix A) were administered to the initiation of the study. An Alco-Sensor III, (Intoximeters, Inc.) hand-held intoximeter was used to examine BAC prior to and throughout the testing session. A weighted scale was used to measure the participant's body height and weight. A computer program by John Curtin, titled Blood Alcohol Calculator 2.1.0, was used to estimate the amount of vodka and tonic necessary to produce the assigned BAC based upon height and weight. Alcohol beverage consisted of a mixture 80 proof Smirnoff Vodka, tonic water and lime. Participants were administered eight ounces of decaffeinated coffee containing either no caffeine or 2.0 mg/kg FDA approved anhydrous caffeine.

The dependent measures consisted of breath tests, an estimation of standard drinks consumed and a ten-point intoxication questionnaire, where one indicates no intoxication and ten indicates being extremely intoxicated (Appendix B). The intoxication questionnaire was administered three times during testing 38, 74 and 98 minutes after the last consumed alcohol beverage.

## Procedure

Prior to initiation of the experiment, a valid driver's license was checked to authenticate age. After obtaining an informed consent, keys, pagers and mobile phone were held for safe keeping. A hand-held Breathalyzer examined the subjects' blood alcohol level. The experiment was immediately terminated if the Breathalyzer detected any breath alcohol. This apparatus was used for measure BACs throughout the study.

Upon arrival, participants are told that they were chosen to participate in a three-part study. The purpose of the first study was to examine the influence of alcohol on perceptual processes (especially visual acuity). The video study examined social memory and judgment and involved a couple on a date. Finally in the caffeine study, they were told they may be consuming an average dose of caffeine and participate in a number of cognitive tasks. The amount of alcohol they were to receive was no more than the equivalent of four to five standard drinks and as a result, they must stay in the lab until their blood alcohol concentration returned to zero. All participation was completely voluntary. Participants could stop the experiment at anytime by saying stop, out loud. If alcohol had been administered, they were required to consent to stay in the lab until blood alcohol level reached .00. During the entire experiment, a participant sat in a comfortable chair with feet reclined during all tasks.

Body height and weight were measured and used as variables for proper alcohol titration. In an adjacent observation room separated by a one-way mirror, the drink mixer prepared the mixture of alcohol according to the subject's randomly assigned condition and body variables. The beverage consisted of a mixture of 80 proof Smirnoff Vodka, tonic water and lime. In both placebo conditions, flattened tonic water was substituted for vodka and poured into a vodka bottle. Drops of vodka are sprayed onto the serving cart; previous research indicates this method

is effective in making subjects think they received alcohol (Noel, et al., 1992; Johnson, Noel, & Sutter, 2000). In the water condition, a pitcher of cold tap water was served.

All drinks were placed on a serving cart in their appropriate containers with a knife, cutting board, latex gloves, timer, pitcher and serving cups. This cart was wheeled out of the control room to the experimenter. A double blind was maintained in that the experimenter in contact with the participant did not know the drink content. Drinks were mixed and served by experimenter 1, in front of the participant. After proper mixing, Experimenter 1 instructed the participant:

*“You will have 25 minutes to drink these vodka tonics. During that time, we will be watching you from the observation room to monitor your progress. Since you must consume all the drinks in 25 minutes, you will be prompted if you need to speed up or slow down a little. This timer should help. Do you have any questions? We will synchronize timers now”.*

After the 25-minute drinking period, an additional 25 minutes was allowed for alcohol absorption. During this period, the participant completed several computerized cognitive tasks. At the end of the absorption period, a third experimenter entered. The participant watched a video of a couple on a date. After the video, a breath test was administered and the participant completed an Intoxication Questionnaire (Time 1), approximately 40 minutes after drinking ended.

In the adjacent room, the drink mixer titrated one of two doses of caffeine according to randomly assigned active or placebo condition. In the active conditions, the participant’s weight was used to calculate a dose of 2.0 mg of FDA approved anhydrous caffeine / kg. Eight ounces of decaffeinated coffee brewed with Folgers Coffee Singles was used as a vehicle to administer

powdered caffeine. The participant had ten minutes to consume the coffee, with the option of one cream and artificial sweetener.

An additional twenty minutes elapsed for caffeine absorption. During this time, participants were allowed to read magazines. At the end of the waiting period, the drink mixer entered and administered a breath test. The participant completed another intoxication questionnaire (Time 2).

The participants were then administered computer tasks for the remainder of the session, and received a third intoxication questionnaire (Time 3). Once they reached a .02 BAC, participants were debriefed, completed payment paperwork and were provided a large one topping pizza. The experiment ended when the participant registered a .00 BAC.

## RESULTS

### Time of Administered Measures

One hundred and thirty-three participants were tested (Table 1). Two independent variables, alcohol and caffeine were manipulated in order to examine their effects on subjective intoxication. As a limitation of being a part of a larger study, dependent measures were administered three times throughout the study 38 (Time 1), 74 (Time 2), and 98 (Time 3) minutes after last consumed alcohol drink. Assessment times were also chosen based upon the amount of time required to reach peak blood concentrations for both alcohol and caffeine. Rall (1990) suggests peak blood alcohol concentration is achieved 30 to 90 minutes of the last consumed drink, while caffeine reaches peak blood levels 30 minutes after ingestion (Maisto, et al., 2004). The dependent measures consisted of a perceived intoxication questionnaire and an estimate of the number of standard drinks administered during the study. Time one served as a baseline estimate of perceived intoxication approximately 38 minutes after last consumed alcohol beverage, independent of caffeine. After time one, participants consumed a cup of coffee; approximately 36 minutes elapsed to allow for caffeine absorption. Times two and three allowed measures of perceived intoxication with both alcohol and caffeine present. Small deviations in the schedule of dependent measures occurred however, no significant time differences were found across assessment times of each condition,  $F(6,178) = .727, p = .629$ .

Table 1

Number of Participants

Number of Participants			
Alcohol	Caffeine	Decaff	Total
Water	18	17	35
Placebo	18	15	33
.04	16	16	32
.08	18	15	33
	70	63	133

## BAC Measures

The physiological measure of blood alcohol concentration was effective for each condition; significant differences between alcohol groups were maintained throughout the study,  $F(2,80) = .52, p < .001$ . Although no physiological caffeine measures were used in the study, during the debriefing all participants reported they believed they had one cup of caffeinated coffee, suggesting an effective manipulation. Participants in the alcohol placebo condition reported a greater number of estimated drinks than the water group across all times, also suggesting an effective alcohol placebo manipulation. No alcohol-caffeine interaction on BAC was found across time,  $F(9, 283) = 1.50, p = .147$  (Figure 1).

Blood alcohol concentrations reached their average peak 38 minutes after the end of the drinking period. Table 2 shows means and standard deviations of mean BACs for each assessment time, significant differences occurred across time,  $F(9,285) = 21.39, p < .001$ . Corresponding values are plotted in Figure 1.

At time one, a Tukey's post-hoc showed the .08 group had greater BACs than corresponding .04 ( $M_{diff} = .032, SE = .002, p < .001$ ), placebo ( $M_{diff} = .061, SE = .002, p < .001$ ) and water conditions ( $M_{diff} = .061, SE = .003, p < .001$ ). Likewise, the .04 condition had higher BACs than placebo ( $M_{diff} = .028, SE = .002, p < .001$ ). BACs for water and placebo conditions did not differ.

For time two, the .08 condition BACs were higher than .04 ( $M_{diff} = .036, SE = .002, p < .001$ ), placebo ( $M_{diff} = .056, SE = .002, p < .001$ ) and water groups ( $M_{diff} = .056, SE = .002, p < .001$ ). .04 BACs were greater than both placebo ( $M_{diff} = .020, SE = .002, p < .001$ ) and water conditions ( $M_{diff} = .020, SE = .002, p < .001$ ).

Time three BACs followed the identical trend, .08 was greater than .04 ( $\underline{M}_{diff} = .036$ ,  $\underline{SE} = .002$ ,  $p < .001$ ), placebo ( $\underline{M}_{diff} = .048$ ,  $\underline{SE} = .002$ ,  $p < .001$ ) and water groups ( $\underline{M}_{diff} = .048$ ,  $\underline{SE} = .002$ ,  $p < .001$ ). .04 was significantly greater than placebo ( $\underline{M}_{diff} = .012$ ,  $\underline{SE} = .002$ ,  $p < .001$ ) and water BACs ( $\underline{M}_{diff} = .012$ ,  $\underline{SE} = .002$ ,  $p < .001$ ).

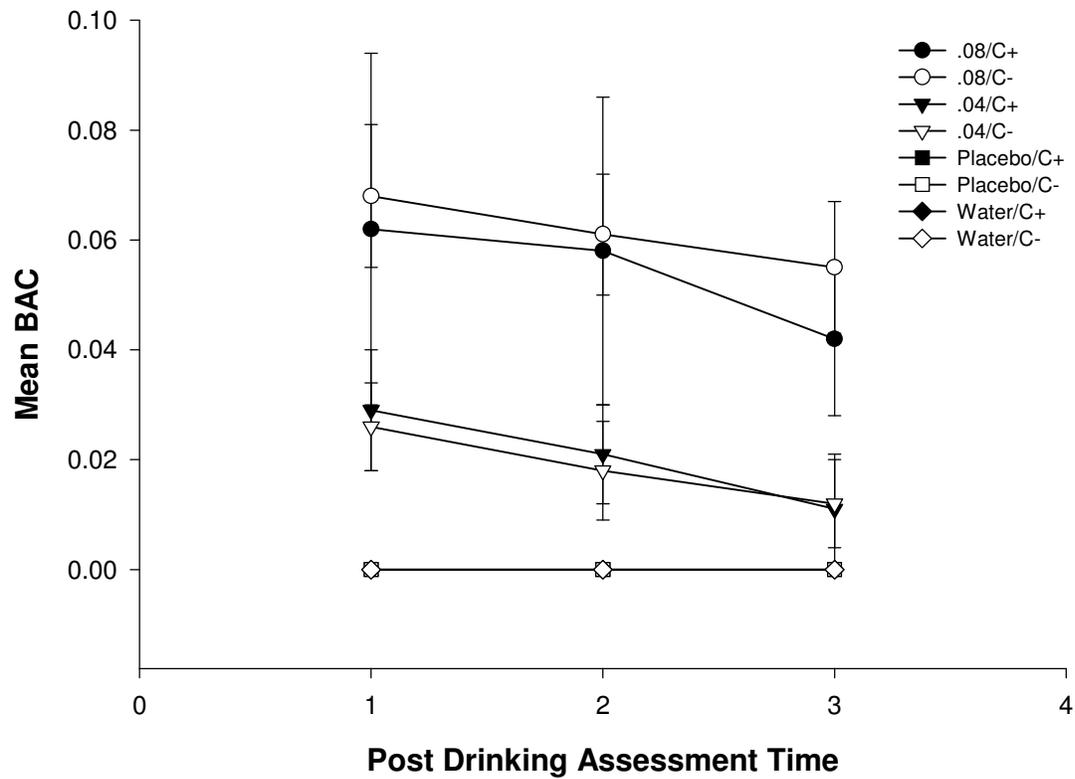


Figure 1. Mean Blood Alcohol Concentrations for each experimental condition at post drinking assessment times. C+ = caffeinated coffee; C- = decaff coffee. No alcohol-caffeine interaction on BAC was found across time,  $F(9,283) = 1.50$ ,  $p = .147$ .

Table 2

Means and Standard Deviations of Blood Alcohol Concentrations

Condition	BAC					
	Time 1		Time 2		Time 3	
	<u>M</u>	<u>SD</u>	<u>M</u>	<u>SD</u>	<u>M</u>	<u>SD</u>
Water / Caffeine	.000	.000	.000	.000	.000	.000
Water / Decaff	.000	.000	.000	.000	.000	.000
Placebo / Caffeine	.000	.000	.000	.000	.000	.000
Placebo / Decaff	.000	.000	.000	.000	.000	.000
.04 / Caffeine	.029	.011	.021	.009	.011	.010
.04 / Decaff	.026	.008	.018	.009	.012	.008
.08 / Caffeine	.062	.032	.058	.028	.042	.014
.08 / Decaff	.068	.013	.061	.011	.055	.012

## Number of Estimated Drinks

Table 3 displays the mean number of estimated standard drinks consumed for each alcohol condition. Significant differences were found across all three time periods;  $F(3,122) = 34.77, p < .001$ ,  $F(3,119) = 33.27, p < .001$ ,  $F(3,98) = 21.82, p < .001$ , respectively. A Tukey's post-hoc found .08 participants had a significantly greater number of reported drinks than were reported by the water condition ( $M_{diff} = 4.46, SE = .49, p < .001$ ). The same trend was found when .04 ( $M_{diff} = 4.14, SE = .49, p < .001$ ) and placebo ( $M_{diff} = 3.82, SE = .49, p < .001$ ) were compared to water conditions. At time two, the .08 group reported more drinks than the water condition ( $M_{diff} = 4.63, SE = .50, p < .001$ ). .04 ( $M_{diff} = 4.16, SE = .50, p < .001$ ) and placebo ( $M_{diff} = 3.39, SE = .50, p < .001$ ) were also greater than the water condition. Time three differences were identical, .08 ( $M_{diff} = 4.63, SE = .60, p < .001$ ), .04 ( $M_{diff} = 4.25, SE = .61, p < .001$ ) and placebo differed from water condition ( $M_{diff} = 3.50, SE = .64, p < .001$ ).

Table 4 displays the means and standard deviations for the number of estimated drinks across assessment times, corresponding values have been plotted in Figure 2. No caffeine-alcohol interactions across time were found.

When collapsed across alcohol conditions, an ANOVA at time two showed a greater number of estimated drinks in caffeine ( $M = 3.85, SD = 2.49$ ) versus decaff conditions ( $M = 2.76, SD = 2.64$ ),  $F(1,121) = 5.47, p = .021$ . This same trend also approached significance at time three,  $F(1,100) = 3.74, p = .056$ , a greater number of estimated drinks for caffeine ( $M = 4.02, SD = 2.32$ ) versus decaff ( $M = 3.03, SD = 2.81$ ) conditions.

Table 3

Means and Standard Deviations of Number of Estimated Standard Drinks Consumed by Alcohol Condition

Alcohol Condition	Number of Estimated Standard Drinks Consumed					
	Time 1		Time 2		Time 3	
	<u>M</u>	<u>SD</u>	<u>M</u>	<u>SD</u>	<u>M</u>	<u>SD</u>
Water	.20	.81	.17	.77	.08	.35
Placebo	4.03	2.28	3.57	2.34	3.58	2.17
0.04	4.35	2.45	4.33	2.48	4.32	2.56
0.08	4.66	1.67	4.81	1.63	4.71	2.00

Table 4

Means and Standard Deviations of Estimated Drinks

Condition	Number of Estimated Drinks					
	Time 1		Time 2		Time 3	
	<u>M</u>	<u>SD</u>	<u>M</u>	<u>SD</u>	<u>M</u>	<u>SD</u>
Water / Caffeine	.42	1.15	.38	1.12	.21	.56
Water / Decaff	.00	.00	.00	.00	.00	.00
Placebo / Caffeine	4.47	2.29	4.23	2.39	4.20	2.08
Placebo / Decaff	3.50	2.24	2.72	2.05	2.90	2.16
.04 / Caffeine	4.63	1.36	4.70	1.36	4.70	1.36
.04 / Decaff	4.08	3.18	4.00	3.21	3.90	3.44
.08 / Caffeine	4.83	1.68	5.29	1.61	4.88	2.14
.08 / Decaff	4.46	1.68	4.26	1.53	4.53	1.88

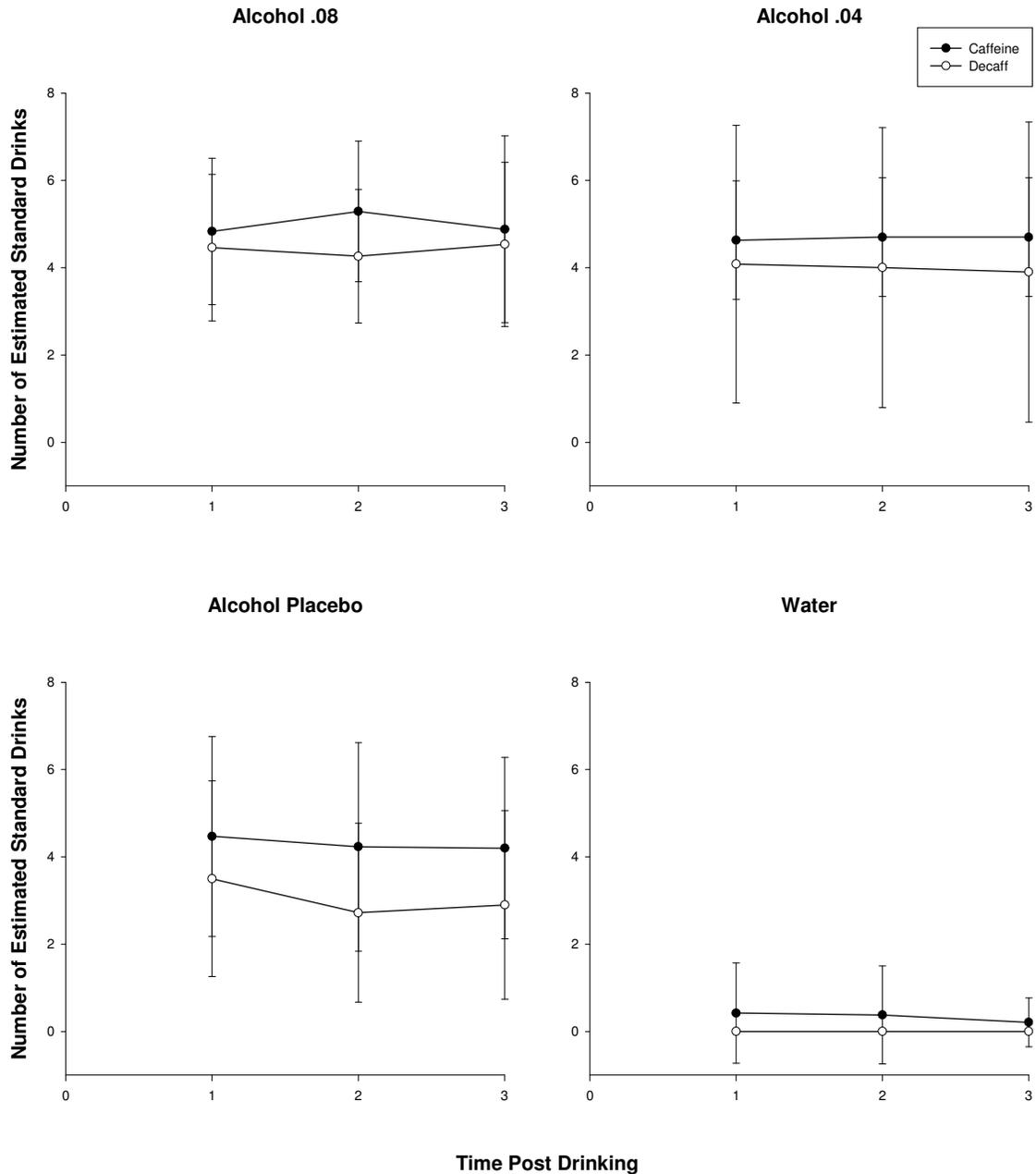


Figure 2. Mean Number of Estimated Standard Drinks for each experimental condition, at all three times of assessment. No significant alcohol-caffeine interactions for each group were observed across time.

## Perceived Intoxication

Subjective assessments of perceived intoxication data is presented in Table 5. When collapsed across caffeine conditions, significant differences in the first intoxication questionnaire were found between alcohol groups,  $F(3,122) = 38.82, p < .001$ . Post-hoc analysis using a Tukey's test revealed significant differences in all four-alcohol conditions.

At time one participants in the .08 alcohol condition reported greater perceived intoxication than .04 ( $M_{diff} = .952, SE = .363, p = .048$ ), placebo ( $M_{diff} = 2.39, SE = .35, p < .001$ ), and water condition ( $M_{diff} = 3.70, SE = .36, p < .001$ ). The .04 group reported greater perceived intoxication than placebo ( $M_{diff} = 1.44, SE = .36, p < .001$ ), and water condition ( $M_{diff} = 2.75, SE = .37, p < .001$ ). Placebo condition revealed greater perceived intoxication than the water condition ( $M_{diff} = 1.31, SE = .36, p = .003$ ).

Perceived intoxication at time two revealed a similar outcome, the .08 group reported greater perceived intoxication than the .04 ( $M_{diff} = 1.18, SE = .28, p < .001$ ), placebo ( $M_{diff} = 2.07, SE = .28, p < .001$ ), and water condition ( $M_{diff} = 2.32, SE = .29, p < .001$ ). The .04 participants reported greater perceived intoxication than placebo ( $M_{diff} = .88, SE = .28, p = .011$ ), and water condition ( $M_{diff} = 1.14, SE = .29, p = .001$ ). However, at time two, the placebo did not significantly differ from the water condition.

Subjective intoxication at time three was assessed approximately 98 minutes after the end of the drinking period. Differences were again observed for the .08 group. Participants reported feeling more intoxicated than .04 ( $M_{diff} = .99, SE = .29, p = .005$ ), placebo ( $M_{diff} = 1.54, SE = .30, p < .001$ ), and water condition ( $M_{diff} = 1.67, SE = .33, p < .001$ ).

A significant alcohol-caffeine interaction was found for perceived intoxication across time. Repeated measures ANOVAs demonstrated an interaction effect for the .04 condition,  $F$

(2,26) = 3.30,  $p = .050$  (see Figure 3). Within this group, interactions were specifically found from times one to two,  $F(1,29) = 6.22$ ,  $p = .019$ ; after drinking a cup of caffeine coffee, participants reported greater perceived intoxication versus the decaffeinated group. Alcohol placebo group approached a significant interaction from time one to two,  $F(1,30) = 3.12$ ,  $p = .088$ , suggesting participants feels more intoxicated after consuming caffeinated coffee.

Table 5

Means and Standard Deviations of Perceived Intoxication by Alcohol Condition

Perceived Intoxication (0 to 10)						
Alcohol Condition	Time 1		Time 2		Time 3	
	<u>M</u>	<u>SD</u>	<u>M</u>	<u>SD</u>	<u>M</u>	<u>SD</u>
Water	.03	.18	.00	.00	.00	.00
Placebo	1.34	1.31	.25	.62	.13	.34
0.04	2.79	1.47	1.14	1.16	.67	.98
0.08	3.74	2.06	2.33	1.77	1.67	1.75

Table 6

Means and Standard Deviations of Participants' Perceived Intoxication Assessments

Condition	Perceived Intoxication (0 to 10)					
	Time 1		Time 2		Time 3	
	<u>M</u>	<u>SD</u>	<u>M</u>	<u>SD</u>	<u>M</u>	<u>SD</u>
Water / Caffeine	.00	.00	.01	.02	.00	.00
Water / Decaff	.09	.30	.00	.00	.00	.00
Placebo / Caffeine	1.54	1.30	.18	.39	.08	.29
Placebo / Decaff	.91	1.14	.18	.40	.18	.40
.04 / Caffeine	2.60	1.40	1.40	1.40	.93	1.16
.04 / Decaff	3.03	1.64	.89	.92	.39	.68
.08 / Caffeine	4.00	1.96	2.38	1.75	1.88	1.89
.08 / Decaff	3.83	2.07	2.37	1.88	1.57	1.66

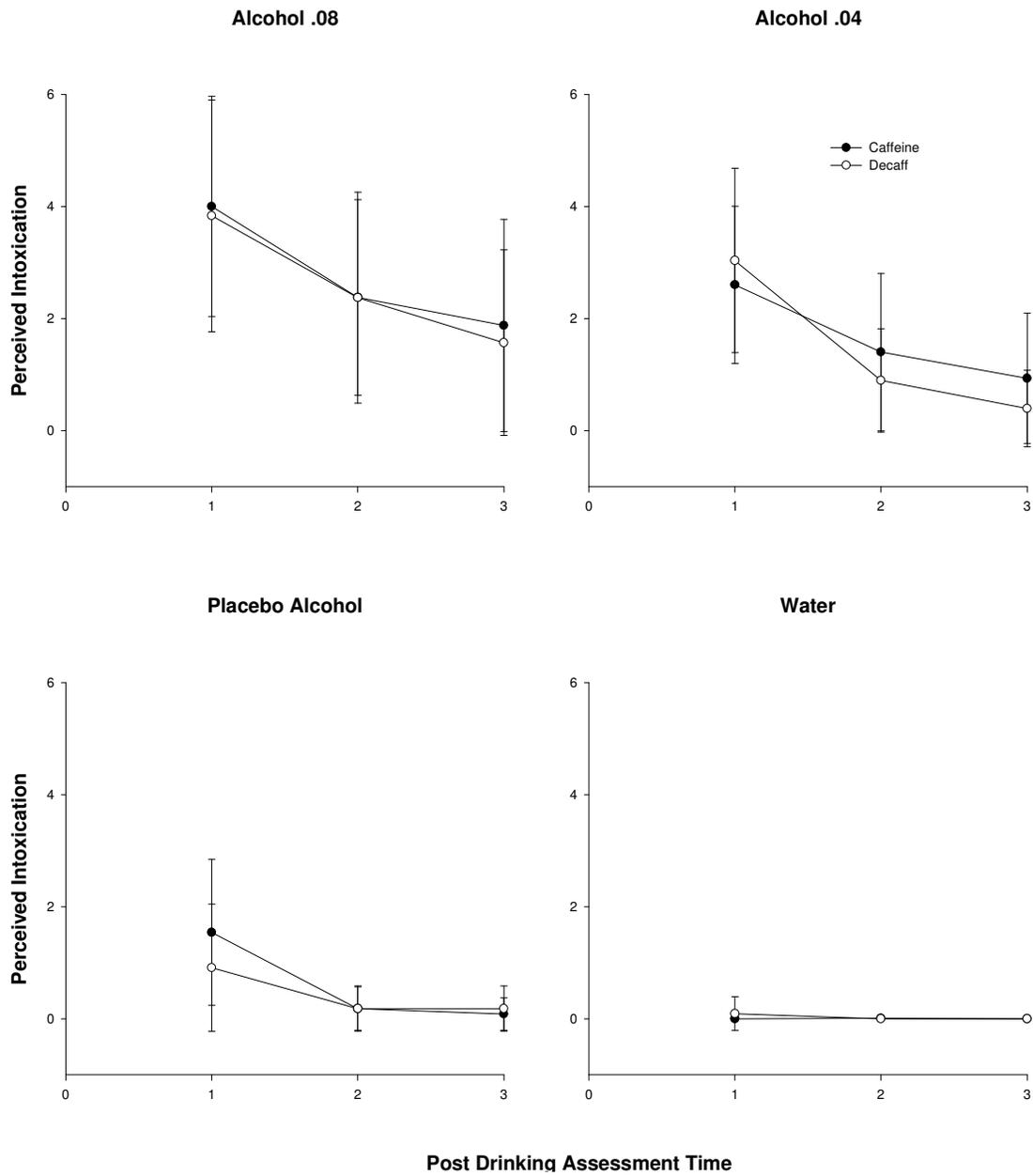


Figure 3. Perceived Intoxication for each experimental condition at all three times of assessment. A significant alcohol-caffeine interaction was found from time one to time two for the .04 alcohol group,  $F(1,29) = 6.22, p = .019$ . Alcohol placebo approached significance from time one to time two,  $F(1,30) = 3.12, p = .088$ .

## DISCUSSION

This research used an intoxication questionnaire comprised of a zero to ten-point scale to assess participants' perceived intoxication under the joint effects of alcohol and caffeine.

Contrary to prediction, the results suggest that caffeine may increase ratings of subjective intoxication.

### Subjective Intoxication

In the present research, the data suggest that alcohol independent of caffeine, increased subjective intoxication. This change appeared to be a function of rising and declining BAC. Measured BAC for active .08 and .04 alcohol conditions peaked at time one, and declined across time, significant differences across dose however were maintained at each assessment time (Table 5). Similar to the BAC data, perceived intoxication was highest for the .08 group across time. The .04 group reported greater intoxication than placebo and water condition at time one and two.

It was predicted that after consuming a cup of caffeinated coffee a decrease in participants' subjective intoxication would result. Data in the .04 condition in fact show the opposite outcome. Within in this group, it appeared that the interaction of alcohol and caffeine significantly increased intoxication scores from time one to time two (Table 7 and Figure 3). The data suggest that consuming a caffeinated cup of coffee (C+) after alcohol intoxication, increases estimates of perceived intoxication across time when compared to decaffeinated coffee (C-).

Conclusive interpretation of the results is difficult, however several possibilities may explain interaction effects. A significant alcohol-caffeine interaction was not found for the .08

group. An interaction approached significance similar to the .04 group for the placebo condition from times one to two ( $p = .088$ ).

Conflicting support for caffeine antagonism of alcohol may partially be due to differences in experimental methods. Azcona et al. (1995) conclude that caffeine does not antagonize the effect of alcohol on perceived drunkenness, however unlike the present study, they failed to control for bodyweight. Caffeine was also simultaneously co-administered with alcohol in capsule form. Similar results from Liguori and Robinson (2001), and Fillmore (2003) suggest that subjective interaction may not occur in alcohol-caffeine research designs in which caffeine is administered in capsule form and mixed into alcohol.

The result of the current study is consistent with the only other examination of alcohol-caffeine that included comparable methods (Fillmore, et al., 2002). Caffeine conditions in both the present and the Fillmore study were administered via a cup of decaffeinated coffee. The administration of caffeine via a coffee beverage is arguably a more ecologically valid method to study alcohol-caffeine interactions. First, coffee ranks among the most popular method in which to obtain caffeine (Weinburg & Bealer, 2001). Media portrayal of characters drinking a cup of coffee to “sober up” suggests it is likely to be consumed after alcohol in preparation for tasks such as driving (Fudin & Nicastro, 1988).

Ironically, the data suggest the presence of caffeine on board with alcohol produces greater feelings of intoxication. Higher intoxication ratings in response to drug combination may suggest participants were unable to subjectively distinguish separate drug effects and used an additive-effect drug rating strategy. This implies the combination of alcohol and caffeine would produce a greater magnitude of subjective intoxication versus alcohol alone. The lack of a no-caffeinated coffee condition in the present study precludes such a conclusion. However, a

similar study reported an alcohol-caffeine treatment yielded lower ratings of subjective intoxication when compared to alcohol alone (Fillmore, et al., 2002).

Evidence for and against subjective interaction is at best unclear. Several studies attribute discrepancies to an expectancy effect for alcohol-caffeine interactions (Fillmore et al., 1994, Fillmore & Vogel-Sprott, 1996; Fillmore & Blackburn, 2002). It has been reported that alcohol-related task impairment is absent when information of alcohol's effect on task impairment is given prior to testing (Fillmore, et al., 2002).

When alcohol is consumed in isolation, Fillmore proposes that reduced impairment can be attributed to the activation of compensatory response, which counteracts alcohol-related impairment. He extends the theory to explain subjective alcohol-caffeine effects. Drinkers who expect an antagonist effect of caffeine on perceived intoxication would be less likely to compensate for alcohol-induced impairment and report greater intoxication ratings. In the debriefing of the present study, all participants reported having had one cup of caffeinated coffee, suggesting that participants expected caffeine would be administered. This expectancy may have contributed to greater perceived intoxication ratings in that participants may have compensated less for alcohol-induced impairments. The proliferation of the alcohol-caffeine myth may significantly contribute to such expectancy.

Fillmore's ironic expectancy theory may partially explain results in the .04 alcohol condition in the present study, however similar results were not found for .08 alcohol condition. Due to increased blood alcohol levels, participants may have been unable to distinguish separate subject effects of caffeine, regardless of condition. As blood alcohol levels approached the legal limit, a 2.0 mg/kg dose of caffeine may have been ineffective to render a subjective change.

Data from a previous study reporting that a 4.0 mg/kg caffeine dose and .076 BAC increased subjective intoxication suggests that this is likely the case (Fillmore et al., 2002).

On a final note, a cup of caffeinated coffee significantly increased perceived intoxication in the .04 alcohol condition, whereas the placebo group approached the similar trend.

Exploratory results suggest caffeine-increased rates of subjective intoxication from time one to two. Pharmacological effects of caffeine may have been misinterpreted as perceived alcohol intoxication. The lack of statistical significance prohibits a conclusive understanding.

#### Limitations

Interpretations of these results are limited due to a small sample size. Other factors may have also influenced the results of this study. Before, participating, subjects were asked to refrain from eating, consuming caffeine three hours prior to the beginning of the study. The use of alcohol and psychoactive substances was also prohibited twenty-four hours of initiation. The absence a biological screening prevents guaranteed compliance.

Expectancy may have also influenced results; alcohol and caffeine expectancies were not assessed. Fillmore hypothesizes that when individuals expect alcohol-caffeine antagonism, they are less likely to compensate for impairment and report greater intoxication. The present study failed to examine and control for participants' alcohol-caffeine expectancies.

#### Future Studies

Alcohol and caffeine are the two most popular non-prescribed drugs in the world. Despite the prevalence of combined use, their behavioral interactions are undeniably complex. Data from the current study suggest that caffeinated coffee increases subjective ratings of intoxication when compared to decaffeinated coffee. Confounding factors such as small sample size and the absence of drug expectancy assessment limits interpretations. Future studies should

investigate alcohol and caffeine expectancy effects, alone and in combination. An increased sample size may demonstrate significant effects for placebo condition. The addition of a no caffeine group is desirable; it would allow researchers to examine the magnitude of subjective intoxication in alcohol alone.

Researchers should also investigate subjective alcohol-caffeine interactions using various doses. The current study failed to find an effect for .08 alcohol BAC and 2.0 mg/kg caffeine, however Fillmore (2002) reported significant interactions with .076 BAC and 4.0 mg/kg caffeine dose.