Summer 5-7-2010

The effects of caffeine on delay discounting in humans

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The Effects of Caffeine on Delay Discounting in Humans

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A thesis submitted to the Graduate Faculty of

JAMES MADISON UNIVERSITY

In

Partial Fulfillment of the Requirements

for the degree of

Master of Arts

Psychological Sciences

August 2010

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Abstract

A behavioral form of impulsivity, delay discounting, has been used to examine the effects of drug consumption on individuals' abilities to delay gratification. However, delay discounting has not been used to examine one of the most commonly used drugs in the world, caffeine. Nor has delay discounting been used to examine the effects of drug influence on impulsivity. This study examined the influence of 200 mg caffeine on delay discounting in a collegiate sample. 15 participants underwent two experimental sessions: a caffeine condition and a placebo condition. Although participants were more impulsive under caffeine than under placebo, this trend was non-significant. This study does however provide a good model for evaluating the influence of drug state on impulsivity.

Introduction

Impulsivity is a characteristic of various mental disorders including mania, obsessive-compulsive disorder, attention-deficit/hyperactivity disorder, and substance abuse disorders (APA, 1994). Despite prevalence in not only psychopathic, but everyday behaviors, no one singular definition of impulsivity exists (Evenden, 1999). Researchers have operationally defined impulsivity as the inability to inhibit inappropriate responses, the tendency to act without considering consequences, and the inability to delay gratification, to name a few (Ainslie, 1975; Barkley, 1997). While most researchers agree that impulsivity is multidimensional, researchers still disagree as to which components should be included in the definition of impulsivity (Whiteside & Lynam, 2001; Zuckerman, 1971). Furthermore, debate has risen over whether impulsivity is a stable personality trait (e.g. Moeller, Barratt, Dougherty, Schmitz, & Swann, 2001) or a description of a malleable state (e.g., Lowenstien, 1996). Other researchers have argued that impulsivity is both a state and trait (Wingrove & Bond, 1996).

Due to the lack of consensus of a definition of impulsivity, multiple assessments have been developed, which operate under various models of impulsivity. These measures generally fall under two categories: self-report questionnaires and behavioral tasks (Reynolds, Ortengren, Richards, & de Wit, 2006). Both types of measures have advantages and disadvantages (Moeller et al., 2001). Self-report measures, for instance, can help assess an individual's long-term patterns of behaviors. Additionally, self-reports allow researchers to obtain data on a variety of different behaviors and thought processes. One weakness of self-report measures is that they rely on self-report data, which may not accurately reflect individuals' true impulsivity levels. Behavioral measures of

impulsivity are able to circumvent the flaws in self-report. However, behavioral tasks assess a narrower spectrum of behaviors than self-reports. Further complicating the decision of how to measure impulsivity, behavioral tasks and self-report questionnaires do not correlate well (Lane, Cherek, Rhoades, Pietras, & Tcheremissine, 2003). It is possible that the two types of measures assess different kinds of impulsivity. Interestingly both self-report questionnaires and behavioral tasks assessing impulsivity have been found to be associated with substance abuse (e.g., Zuckerman, 1990; Vuchinich & Simpson, 1998).

In studying substance abuse and addiction, researchers have used various measures of impulsivity. One of the more common behavioral measures used is delay discounting.

Delay Discounting

Delay discounting is a behavioral model of impulsivity that operates under a definition that could be called an inability to delay gratification. Specifically, delay discounting refers to the reduction in subjective value of a reward that is delayed over time. This process relates to impulsivity because people may sometimes choose a sooner reward over a later reward, even if the later reward is greater in objective value. Such a choice (choosing the smaller, sooner reward) is considered impulsive, whereas the inverse is considered self-control.

Models of delay discounting. Several mathematical models of delay discounting are described in the literature to date. Exponential discounting (Fishburn & Rubinstien, 1982; Lancaster, 1963) assumes that the subjective value of a reward drops by a fixed

amount, or exponentially, per unit time delayed. Samuelson (1937) proposed the most common function used to model exponential discounting:

$$V = Ae^{-kD} \quad (1)$$

where V is the subjective value of the reward, A is the objective value of the reward, D is the length of the delay until the reward is given, and k is a discounting constant representing the rate of discounting. Higher k values indicate that an individual discounts rapidly, or that rewards lose subjective value more quickly for that individual.

Although an exponential model of delay discounting is intuitively sufficient to explain choice, evidence has suggested that this model may not best describe decision-making between immediate and delayed rewards (Chung, 1965; de Villiers & Herrnstein, 1976). Instead, a hyperbolic model of discounting seems to fit behavioral data better. One of the commonly used models of hyperbolic discounting was posited by Mazur (1987):

$$V = A/(1 + kD). \quad (2)$$

According to this function, a reward will lose value more quickly at shorter delays than at longer delays. Similarly to the exponential model, the hyperbolic model of choice can account for an individual choosing a smaller more immediate reward over a larger delayed reward.

Given the variety of mathematical models for delay discounting, research has focused on which models to use. For instance, strong evidence has been gathered against the

usage of exponential discounting, or Equation 1. Namely, the exponential model cannot account for preference reversals (Ainslie & Haendel, 1983). If an individual must wait some time before either reward can be obtained, he or she may initially choose the delayed but greater reward. Yet as time grows closer to the sooner but smaller reward, the individual may opt to switch preference to the reward closer in time. For example, an individual may have the choice between a concert delayed by sixth months, and a car delayed by a year. At first, the individual may choose the car, which is the objectively more valuable choice. However, as time passes, the individual may impulsively switch preferences to the concert in one month, as opposed to the car in seven months. While exponential models of discounting cannot mathematically account for preference reversals, the hyperbolic model can (Kirby & Herrnstein, 1995).

Assessing delay discounting. Assessments of delay discounting vary. Commonly, research participants will be requested to indicate a series of hypothetical preferences for a later, larger reward or a smaller, sooner reward (Rachlin, Raineri, & Cross, 1991). However, studies have also given real rewards to participants. In these studies, researchers will randomly select responses from participants and give them the amount of money they choose at the delay indicated (Kirby, Petry, & Bickel, 1999). The purpose of utilizing a real-rewards delay discounting task is to have participants treat each choice as personally relevant. This procedure has the advantage of high external validity; participants are encouraged to demonstrate impulsivity with real rewards. However, the procedure does have drawbacks. Because any one trial could be the response selected to have real consequences, researchers must constrain themselves to offer rewards that are feasible (e.g. $100 instead of $1,000). Furthermore, delays need to be tailored for

practicality, such that delivery of rewards for delays of 25 years, for example, would be problematic.

As the usage of real-rewards discounting tasks are designed to have participants exhibit genuine impulsivity, concern has been raised about whether the usage of hypothetical rewards generates different discounting rates than real rewards. Indeed, Kirby (1997) compared hypothetical-rewards studies and real-rewards studies, finding that individuals discounted real money more than hypothetical money, though both were still described well by hyperbolic models. However, other studies comparing real and hypothetical rewards within subjects have not found a difference between real and hypothetical rewards. (Johnson & Bickel, 2002; Madden, Begotka, Raiff, & Kastern, 2003). Few other studies have compared these two methods of delay discounting assessment.

Another method in which delay discounting tasks can differ is in the commodity of the reward. Research participants may be asked to indicate intertemporal preference for small and large health gains (Chapman & Elstein, 1995), drugs (Field, Santarcangelo, Sumnall, Goudie, & Cole, 2006), or other relevant rewards (Van den Bergh, Dewitte, & Warlop, 2008) instead of or in addition to money. For instance, Baker, Johnson, and Bickel (2003) compared smokers and non-smokers on discounting rates for health, money, and in the case of smokers, cigarettes. In order to obtain an equivalent value of health gains or cigarettes for a given value of money, the researchers asked participants during the first experimental session to associate amounts of money to number of cigarette packs and length of improved health. No significant differences were found

within the two groups between monetary and health gains. However, smokers discounted cigarettes at a higher rate than they discounted both money and health gains. Therefore, delay-discounting tasks may be modified to incorporate rewards more personally relevant to participants.

Analysis of delay discounting. Analysis of delay discounting involves calculating indifference points, where the larger delayed reward and the smaller immediate reward are subjectively worth the same to the individual. For example, $600 available immediately may be worth the same as $1000 available after a month for a given individual. The given individual will be equally likely, then, to choose $600 available immediately as to choose $1000 available after a month. In terms of Equations 1 through 4, the indifference point is the *V* value for a given delay. Graphing indifference points over multiple delays yields an indifference curve, where steeper curves indicate more impulsive decision-making. These curves are fit to the data using nonlinear regression analyses, from which *k* and *s* values are derived.

Myerson, Green, and Warusawitharana (2001) noted problems with using *k* and *s* values. Firstly, k and *s* values are tied to the mathematical model researchers utilize, which may differ across studies. Indeed, researchers still debate over which function best describes delay discounting. Second, distribution of *k* values is not normal but positively skewed, which results in statistical problems. Because standard parametric tests assume normal distribution, non-parametric tests are necessary to make inferences about the population. Yet non-parametric tests are less powerful than parametric tests (Hays, 1994)

and are not able to compare groups when other experimental factors also differ (i.e., commodity of reward).

To remedy these problems, Myerson and colleagues (2001) have suggested graphing indifference curves and analyzing the area underneath the curve (AUC). In order to calculate AUCs, reward values and delays are calculated as proportions of the maximum used reward and delay, respectively. Then, lines are drawn from data points down to the *x*-axis, segmenting the curve into a series of trapezoids. Assuming two data points that have coordinates of (x_1, y_1) and (x_2, y_2), the area of each trapezoid is $(x_2 - x_1)\ [(y_1 + y_2)/2]$. The areas of all trapezoids are then summed to form an AUC. Lesser areas underneath the curve of indifference denote more impulsivity, with 0.0 indicating maximum discounting (highest impulsivity) and 1.0 indicating no discounting (lowest impulsivity). Unlike *k* values, AUCs are more normally distributed and are thus suitable for parametric testing. Furthermore, AUCs are computed using observed data points instead of a curve fit to the data, which results in a model- and theory-free parameter of delay discounting.

However, Myerson and colleagues (2001) warned that two separate indifference curves may have different rates of discounting, but the same areas. For instance, an individual may discount steeply at shorter delays and shallowly at longer delays. Another individual may discount shallowly at shorter delays and steeply at longer delays. If the AUCs were computed for these two individuals, the same value may be obtained. To avoid such confounds, the authors suggest examining the raw data and potentially computing two AUCs for shorter and longer delays. Despite this potential issue, AUCs provide a valuable method with which to evaluate delay discounting statistically.

A variety of delay-discounting models, assessments, and analyses have been described in the literature. One common usage of delay-discounting tasks has been to understand the role of impulsive decision making in addiction.

Using delay discounting to study addictive behaviors. Impulsive decision making occurs frequently among those who exhibit addictive behaviors. As an example, an individual may often choose to engage in drug use (a small immediate reward) over delayed but larger incentives such as good health or financial stability. Due to the parallels between delay discounting and drug use, many studies have used delay discounting as a framework for understanding the relation between addiction and impulsivity.

Gambling. Dixon, Marley, and Jacobs (2003) administered a delay-discounting task to individuals gambling at an off-track betting facility for horse races. Potential participants were first screened using the South Oaks Gambling Screen (SOGS). Individuals scoring above a 4 on the SOGS (which is associated with pathological gambling) were asked to indicate preference between a series of two hypothetical money amounts, one immediate and one delayed. Immediate amounts ranged between $1 and $1000, and delays ranged from 1 week to 10 years. The researchers found that the hyperbolic model of delay discounting did not always provide a good fit, due to individual variability. However, gamblers generally had higher rates of discounting when compared to non-gambling controls sampled from the surrounding community. Furthermore, the mean AUC for aggregate indifference points was significantly lower for

gamblers than non-gamblers. These results suggest that gamblers are more impulsive than non-gamblers.

Delay discounting has also been used to examine comorbid gambling and substance use. For instance, Petry (2001) examined gamblers with and without substance use problems as compared to controls. Petry found that gamblers without comorbid substance abuse discounted more rapidly than non-gambling controls. Furthermore, gamblers with comorbid substance abuse discounted more rapidly than gamblers without comorbid substance abuse. In other words, there is an interaction between substance-use and gambling associated with higher rates of discounting than simply gambling. This finding replicates results of a previous study (Petry & Casarella, 1999).

Illicit drugs. Cocaine abusers have also been studied with respect to delay discounting. Hiel, Johnson, Higgins and Bickel (2006) studied cocaine abusers, former cocaine abusers, and controls. Participants were recruited from outpatient treatment at a university-based research clinic. Hiel and colleagues found that there were no significant differences in the discounting of money between all three groups. However, when collapsing the current cocaine users and the former cocaine users, researchers found that individuals who had ever used cocaine discounted money more steeply than non-using controls.

Another study examined delay discounting in heroin addicts. Kirby and colleagues (1999) recruited heroin addicts from a campus-based outpatient clinic, whereas controls were recruited from the local community. Participants completed self-report measures of

impulsiveness, as well as a delay-discounting task. The researchers found that heroin addicts discounted roughly twice the rate of non-drug-using controls.

Cigarette smoking. Other studies have examined delay discounting in cigarette smokers. Bickel, Odum, and Madden (1999) examined discounting in current, never, and ex-smokers. To qualify as a current smoker, individuals must have reported smoking at least 20 cigarettes a day for the past 5 years; ex-smokers must have abstained from similar levels of smoking for a year. Participants completed a delay discounting task involving monetary values. Current smokers completed an additional delay discounting task involving amounts of cigarettes instead of monetary values. The researchers found that current smokers discounted money more steeply than never or ex-smokers. Furthermore, smokers discounted cigarettes at a steeper rate than they discounted money. The researchers also found that ex-smokers discounted very similarly to never-smokers, suggesting that the impulsivity associated with current smokers is dynamic.

Johnson, Bickel, and Baker (2007) replicated these findings in individuals smoking fewer than 20 cigarettes a day, more than 20 cigarettes a day, and non-smokers. Participants completed three laboratory sessions, one assessing health gains and losses and cigarette gains and losses, and two sessions using hypothetical monetary choices. During these later sessions, participants performed a computer-administered delay discounting task. Gains and losses were represented in the delay-discounting paradigm. The researchers found that light smokers discounted cigarettes similarly to heavy smokers, and both discounted money more than non-smokers.

The studies mentioned above used discounting of cigarettes to compare groups of smokers. Yet the researchers did not control for cigarette satiation and deprivation, which could influence how quickly smokers discount cigarettes. To investigate satiation and deprivation, Field and colleagues (2006) conducted a study examining the effects of nicotine deprivation on delay discounting. Individuals who smoked daily completed two sessions, one after at least 13 hrs of cigarette deprivation, and one where participants could smoke freely beforehand. The researchers found that individuals discounted both money and cigarettes more rapidly under the deprivation condition than under the free condition. This suggests that impulsive decision-making may be more evident under deprived conditions. Conversely, self-control may be greatest in addicted individuals after, or perhaps during, drug usage, due to the reinforcing effects of the drug.

Problem drinking. Researchers have used delay discounting as a framework for understanding problematic alcohol drinking. Petry (2001) studied alcoholics, currently abstinent alcoholics, and non-abusers. Participants responded to a delay-discounting task with delayed rewards consisting of $1000, $100, 15 bottles of preferred wine or liquor, and 5 bottles of preferred wine or liquor. Petry found that alcoholics discounted money more quickly than controls. Currently abstaining alcoholics discounted less than alcoholics, yet more than non-abusers. When comparing the discounting of money to the discounting of bottles of alcohol within groups, Petry found that alcohol was discounted more rapidly than money. In another study, Field, Christiansen, Cole, and Goudie (2007) examined adolescent light and heavy drinkers in the United Kingdom. Drinking endorsement was collected from potential participants and was used to form the light drinking group (lowest 25%) and the heavy drinking group (highest 25%). Participants

then completed a paper version of the delay discounting task. The researchers found that heavy drinkers discounted more rapidly than light drinkers.

Not all studies have found that alcohol abuse is associated with increased discounting. Wilhelm, Reeves, Phillips, and Mitchell (2007) compared two strains of rats on a delay discounting task and a go/no-go task, another measure of behavioral impulsivity. Rodents were bred to voluntarily drink either low or high amounts of 10% ethanol. Rats could choose via nose-poke between a large amount (20 µl) of sucrose after a delay ranging from 0 to 12 s or a small amount (10 µl) immediately. The go/no-go task used a light to signal *go* trials and a tone and light to signal *no-go* trials. Rodents were to refrain from nose-poking on no-go trials and were to nose-poke on go trials; more nose-pokes on no-go trials indicated higher impulsivity. Wilhelm and colleagues found that light drinking and heavy drinking rodents did not differ on the delay-discounting task, though heavy drinking rodents did make more pre-cue responses to go trials than light drinking rodents. The researchers suggested that the genetic manipulation of drinking preference in rodents may be unrelated to the inability to delay gratification.

In a study with humans, Richards, Zhang, Mitchell, and de Wit (1999) assessed the effects of alcohol on delay discounting. Participants recruited from a university and the surrounding community attended four sessions: an orientation, two experimental sessions (alcohol/placebo), and debriefing. In experimental sessions, participants completed a predrug computerized delay-discounting task. This task was programmed to titrate towards individuals' indifference points for given delays. Then, participants drank 0.5 g kg^{-1} alcohol, 0.8 g kg^{-1} alcohol, or placebo. Participants were given twenty minutes for

the drug to take effect. Finally, participants completed the same computerized delay-discounting task, and were driven to their homes. Experimental conditions were counterbalanced between participants. The researchers found no significant differences between alcohol conditions and the placebo condition. The researchers suggested that the effects of alcohol may be more evident under conditions of high conflict, where both gains and potential losses are larger.

One study found that alcohol intoxication reduces delay discounting. Ortner, MacDonald, and Olmstead (2003) examined the effects of alcohol, placebo, and control (sober) on delay discounting. Undergraduate students were given either three mixed alcoholic drinks, non-alcoholic drinks with rims dipped in alcohol (placebo), or were assigned to control. Then, they completed a delay-discounting task with a delayed reward of $10 and immediate values ranging from $.01 to $10.50. The researchers found that indifference points for students in the alcohol and placebo conditions were higher overall than for students in non-alcohol conditions. Ortner and colleagues suggested that perhaps the artificialness of the experimental session inhibited individuals from behaving uninhibitedly in regards to choice in rewards.

Caffeine and impulsivity. Few studies have looked at caffeine's influence on impulsivity and even fewer have used a delay discounting paradigm. Roehrs, Greenwald, and Roth (2004) examined the effects of ethanol, sleepiness, and caffeine on a risk-taking measure, the Stop-Light Task. This task involves typing various *x-y* key combinations during a "green light" for actual monetary gain. Once the "yellow light" appeared, participants could choose to not complete the remainder of the combinations, which

would conserve their earnings, or they could choose to continue, risking their current earnings. Adult participants were identified as either alert or sleepy, based on monitored sleep patterns. Participants then attended multiple sessions where they received a combination of caffeine (0.0 mg, 150.0 mg, or 300.0 mg) and ethanol (0.0 g kg^{-1} or 0.5 g kg^{-1}). Sessions were separated by 3 to 7 days so participants could be tested within-subjects, as well as between groups (alert or sleepy). After the combination of ethanol and caffeine took effect, participants completed the Stop-Light Task. However, no significant effects for risky choices were found for ethanol or caffeine or any of their interactions.

Jones and Lejuez (2005) conducted a study examining the personality correlates of caffeine consumption. College students were screened for high and low levels of caffeine consumption, as well as caffeine dependence, using the Caffeine Consumption Questionnaire (Shohet & Landrum, 2001). Two groups of participants were then formed: non-caffeine-dependent low consumers and caffeine-dependent high consumers. Participants then completed three personality measures of impulsivity: the Sensation-Seeking Scale (Zuckerman, Eysenck, & Eysenck, 1978), the Eysenck Impulsiveness Scale (I_7; Eysenck, Pearson, Easting, & Allsopp, 1985), and the Balloon Analogue Risk Task (BART). The researchers found that high sensation-seeking and impulsivity scores were significantly correlated with being caffeine-dependent high consumers. However, when sensation-seeking and impulsivity scores were entered into a logistic regression, only sensation-seeking could significantly predict group status. These findings suggest that caffeine consumption may be associated with sensation-seeking, though not with risk-taking or impulsivity.

Marczinski and Fillmore (2003) examined the effects of different doses of caffeine (0.0 mg kg^{-1}, 2.0 mg kg^{-1}, and 4.0 mg kg^{-1}) combined with different doses of alcohol (0.0 g kg^{-1}, and 65.0 g kg^{-1}) on a cued go/no-go behavioral inhibition task. Cues were consistent with go/no-go targets on roughly 80% of trials. Participants consisted of social drinkers recruited from the local community. Alcohol generally lengthened the response times for go targets with the no-go cue. However, the researchers found that 4.0 mg kg^{-1} caffeine shortened back to baseline the response times to go targets with no-go cues under the alcohol condition. The researchers also found that caffeine alone shortened response times to go targets with go cues. Caffeine was not found to have any main effect or interaction effect for failure to suppress responses on no-go targets. These findings suggest that while caffeine may not influence impulsivity in terms of failing to suppress a predominant motion, caffeine may aid in decreasing response speed.

Childs and de Wit (2006) investigated the effects of caffeine (placebo, 50 mg, 150 mg, or 450 mg) on subjective, physiological, and behavioral measures. Participants were college students who endorsed low and non-dependent usage of caffeine, due to the difficulty of assessing the effects of caffeine in individuals who have developed a tolerance. Participants were given a version of the go/no-go task as a measure of behavioral disinhibition. However, no significant effects of caffeine were found for failure to inhibit responses.

Meyer and Caston (2004) examined the interactive and main effects of stress and caffeine on investigatory behaviors and behavioral disinhibition (impulsivity) in mice. Eighty mice were divided into two groups: stressed and unstressed. Stressed mice

underwent stressful procedures for six consecutive days, which included: submersion in cold water; placement in an open, lit field; and placement in a confined tube. Furthermore, the two groups were divided into four caffeine groups (saline, 30 mg kg^{-1}, 60 mg kg^{-1}, or 120 mg kg^{-1}). Stressed and unstressed mice were injected with either saline or caffeine on the sixth day following the stressful procedure. Behavioral measures of investigation and inhibition were assessed by placing mice in a parallelepipedic container. Behaviors that indicated investigation included walking time and rearing behaviors, whereas time spent in the center of the device and time spent frozen in the periphery of the device served as measures of disinhibition. Specifically, more time spent in the center and less time spent frozen in the periphery indicated higher levels of disinhibition. The researchers found that unstressed mice injected with 30 mg kg^{-1} caffeine walked more often in the center zone of the device (behavioral disinhibition) than mice injected with saline, though this was not found for higher levels of caffeine. However, this effect did not quite meet statistical significance. Furthermore, stressed mice showed only non-significant increases in behavioral disinhibition. The results from this study imply that caffeine may increase behavioral disinhibition in the rodent, though more studies need to be conducted to find this effect.

Diller, Saunders, and Anderson (2008) conducted one of the few delay discounting studies of caffeine. The researchers injected acute levels (10 mg, 17 mg, and 30 mg, counterbalanced) of caffeine into male rats trained to engage in delay-discounting tasks. Specifically, rodents had the opportunity to press one lever to obtain 1 pellet of food immediately, or another lever to obtain 3 pellets after a delay. Delays ranged from 0 to 16 s. Rodents injected with caffeine preferred the delayed reward more often than

controls, resulting in higher indifference points and areas under the curve (AUC). Indifference points and AUCs were higher in higher doses. In a second phase of the study, Diller and colleagues examined the effects of chronic administration of 30 mg of caffeine on delay discounting. They found that chronic administration resulted in lowered mean indifference points and AUCs, though both were still higher than saline controls. These results suggest that acute administrations of caffeine in rodents decrease impulsivity in a dose-dependent manner. Chronic administration of high doses heightens impulsivity, though not to baseline levels.

Upon examination, the literature suggests that a relation exists between drug abuse (or other addictive behaviors) and impulsivity. Problematic drug users are generally more likely to choose a smaller, sooner reward over a greater, delayed reward. However, few studies have experimentally examined the effects of drug state on delay discounting. In other words, little research has been done on individuals while intoxicated by a given drug. Furthermore, one of the most commonly used drugs in the world, caffeine, has not been studied under the delay-discounting model of impulsivity in humans. Caffeine is an important drug to consider under the delay-discounting paradigm for reasons that will become apparent after a short review of the drug.

Caffeine

Caffeine, or 1,3,7-trimethylxanthine, is found naturally in various forms, such as in coffee, chocolate, and tea. It is also added to other consumables such as soda and energy drinks. An estimated 80% of the American population consumes caffeine daily, regardless of source (Barone & Roberts, 1996; Bonham & Leaverton, 1979). Daily levels

of caffeine consumption for the average individual in the US range from 275 mg (Lane, 1983) to 3 mg kg^{-1} (Barone & Roberts, 1996).

Few studies have assessed the reasons individuals consume caffeine. Graham (1988) modified an alcohol consumption model for usage for caffeine. Motives included two "personal" reasons (stimulant and relief) and two "social" reasons (beverage and to be sociable). She found that both personal motives were the best predictors of caffeine consumption, with beverage motives being a strong predictor. Heinz, Kassel, and Smith (2009) developed a Caffeine Expectancy Questionnaire (CEQ) under the theory that expectancy of outcomes underlies caffeine's reinforcing effects. This measure comprised of four factors including withdrawal symptoms, positive effects, acute negative effects, and mood effects. The researchers found adequate validity for the measure, though cautioned that more studies are needed to assess the instrument's reliability.

Physiological effects. Caffeine has a wide variety of physiological effects. Caffeine exerts its effects in the brain through the blockage of adenosine receptors (Daly & Fredholm, 1998). Adenosine is a neurotransmitter that modulates the responses mediated by other neurotransmitters and commonly produces sedative effects that caffeine inhibits (Cauli & Morelli, 2005; Dunwiddie & Worth, 1982). In roughly 30-45 min, caffeine reaches peak concentration levels (Denaro & Benowitz, 1991). Caffeine metabolizes into three main compounds (Benowitz, 1990). The primary metabolite of caffeine is paraxanthine, with other metabolites being theobromine and theophylline. These metabolites are pharmacologically active in the body, which may contribute to the effects of caffeine.

One of the more commonly recognized effects of caffeine is on sleep. Evidence has found that, when consumed before trying to sleep, caffeine can delay the onset of sleep (Zwyghuizen-Doorenbos, Roehrs, Lipschutz, Timms, & Roth, 1990), as well as reduce the duration of sleep (Hicks, Hicks, Reyes, & Cheers, 1983). However, wide individual differences exist. Levy and Zylber-Katz (1982) found that individuals who reported no difficulty sleeping after caffeine intake metabolized caffeine more quickly than individuals who reported sleep difficulties after caffeine consumption. Other studies found that even morning caffeine intake can influence sleep at night (Landolt, Werth, Borbely, & Dijk, 1995). Some authors have suggested that heavy coffee drinkers develop a tolerance to the effects of caffeine on sleep (Colton, Gosselin, & Smith, 1968), though the literature has been ambiguous on the topic (Smith, 2002).

Caffeine can aversively affect the cardiovascular system at some doses. Specifically, while caffeine has little effect on heart rate, both systolic and diastolic blood pressure rise by 5 – 15 mmHg and 5 – 10 mmHg respectively, usually at higher doses (Noordzij et al, 2005). Although some research has suggested that physiological tolerance of the drug can moderate the effects on blood pressure, empirical support for this claim is conflicted (James, 2004). Research has also examined the effects of long-term caffeine use on heart disease through epidemiological studies (Cornelis & El-Sohemy, 2007). Generally, results show that caffeine may serve as a risk factor in coronary heart disease. Findings, however, have been conflicted due to methodological differences in data collection and not controlling for confounds.

Psychological effects of caffeine. Research on the effects of caffeine has not been limited to physiological domains; the psychological actions of caffeine have been examined as well. General psychological effects of caffeine include improved alertness (Regina, Smith, Keiper, & McKelvey, 1974), memory (Smith, Sturgess, & Gallagher, 1999), and reaction time (Durlach, 1998) at some doses. Caffeine has also been shown to increase subjective ratings of stress (Lane, 1983). Research has commonly examined the effects of caffeine on mood, anxiety, and whether caffeine is addictive.

Mood. Studies on the effects of caffeine on mood states have yielded conflicting results. Some studies report that caffeine elevates mood. For instance, Childs and de Wit (2008) conducted a study in which volunteers from the community and university were deprived of sleep for over 12 hrs. Participants were required to wake up at 7:00 am and to attend the laboratory sessions beginning at 5:00 pm. At 3:30 am, participants completed mood ratings and performance tasks and were given either caffeine pills (200 mg) or placebo pills. Then, mood ratings were assessed every 30 min after until 5:00 am. The researchers found that caffeine raised positive mood and friendliness ratings and lowered depression ratings.

However, Veleber and Templar (1984) found the opposite. Participants recruited from colleges and businesses were given 0, 150, or 300 mg of caffeine per 45.36 kg of body weight. Mood ratings were collected before and after the drug took effect. The researchers found that depression, anxiety, and hostility ratings increased from pre-consumption to post-consumption. In discussing these results, the researchers postulated

that caffeine produces an initial elation in mood which is followed by a depression, similar to other psychoactive stimulants such as amphetamine.

Anxiety. Studies generally show that caffeine in moderate to large doses can induce anxiety in non-clinical samples (Hughes, 1996). For example, Loke (1988) assessed the effects of 0, 200, and 400 mg of caffeine on anxiety in undergraduates. After caffeine administration, participants completed subjective mood ratings and memory tasks. Participants reported dose-dependent increases in nervousness, tension, and anxiety. Few studies have found a non-effect of caffeine on anxiety, though exceptions exist (James, Crosbie, & Paull, 1987).

Research has also been conducted in individuals with anxiety disorders. Intuitively, individuals with anxiety disorders may consume less caffeine than others, as the effects of caffeine may mimic or induce anxiety. Indeed, Lee, Cameron, and Greden (1984) found that 84% of anxious inpatients consumed less than 249 mg of caffeine per day, as opposed to 41% of other inpatients. When such individuals are exposed to moderate levels of caffeine (3.3 mg kg^{-1}) they report significantly higher anxiety responses than non-anxious controls (Totten & France, 1995).

Caffeine as an addictive drug. With the widespread usage of caffeine, concern has been raised over whether caffeine is addictive. Indeed, some have considered caffeine as a model drug of abuse (Holtzman, 1990). In general, the research suggests that usage of caffeine at low to moderate doses is non-problematic (James & Paull, 1985). However, at high doses, individuals can become intoxicated by caffeine.

Caffeine intoxication has also been referred to in the literature as caffeinism, caffeine overdose, and caffeine poisoning. Generally, caffeine intoxication is associated with symptoms such as irritability, nervousness, headache, reflex hyperexcitability, and tachypnea (APA, 1994). These symptoms raise important diagnostic concerns, as they can resemble symptoms associated with anxiety disorders (Greden, 1974). Caffeine intoxication can occur in some individuals at doses exceeding 500 mg, while the lethal dose in human is estimated to be roughly 10 g (Stavric, 1988). Cases of death resulting from caffeine intake have been reported, both accidentally and as a method of suicide (Kerrigan & Lindsey, 2005; Holmgren, Norden-Pettersson, & Ahlner, 2004).

Individuals who consume caffeine regularly may also experience withdrawal symptoms upon abrupt cessation. Schuh and Griffiths (1997) conducted a study on the symptoms of caffeine withdrawal in humans. On average, participants recruited for the study consumed an average daily amount of 379 mg of caffeine. Upon abstinence from caffeine for 19 hrs, participants reported headaches, fatigue, lack of well-being, and unhelpfulness. Such symptoms are commonly reported in the literature, along with depression, nausea, muscle aches, and difficulty concentrating (Griffiths & Woodson, 1988a). These symptoms occur roughly 12-24 hrs after termination of chronic or acute caffeine consumption, though the symptoms can vary individually in severity (Juliano & Griffiths, 2004).

Research on tolerance to caffeine's effects has been inconclusive. Much research has studied whether tolerance can be developed for caffeine's effects on blood pressure. An early study by Robertson, Wade, Workman, Woosley, and Oates (1981) found partial

tolerance to caffeine-related increases in blood pressure over a period of 3 days. Other studies have replicated this finding and suggested possible mechanisms of tolerance (Colton et al., 1968; Shi, Benowitz, Denaro, & Sheiner, 1993). However, a large body of literature also exists finding no tolerance to the cardiovascular effects of caffeine (Hartley, Lovallo, Whitsett, Sung, & Wilson, 2001; James, 2004). More research on the tolerance of caffeine's hemodynamic effects is needed. Other research has focused on the tolerance to effects on sleep (Snel, 1993), finding equivocal results. Studies of tolerance to caffeine's effects on mood have generally found either no tolerance (Estler, 1982) or tolerance to some negative effects such as headache and fatigue (Evans & Griffiths, 1992).

Studies have shown that caffeine reinforces its usage at low and moderate doses (Rush, Sullivan & Griffiths, 1994; Hughes, Oliveto, Bickel, Higgins, & Badger, 1995). At higher doses (300 mg 70 kg^{-1}), caffeine can induce discomfort and aversive effects (Garrett & Griffiths, 1998). The reinforcing properties of caffeine can be associated with avoidance of withdrawal symptoms, but can also be independent of withdrawal (Silverman, Mumford, & Griffiths, 1994). However, these reinforcing effects are often weaker than other drugs (Griffiths & Woodson, 1988b).

Present Study

Caffeine is the most commonly consumed drug in the world. Furthermore, a review of the literature suggests that consumption of caffeine can have potentially adverse psychological, as well as physiological, effects. Though the research is not clear whether caffeine is addictive, effort should be made to understand possible implications of

caffeine addiction. While many drugs are correlated with high levels of impulsivity, research suggests that caffeine may only be related to certain types or measures of impulsivity, if at all. Only one study to date has used delay-discounting tasks to model the effects of caffeine on impulsivity (Diller et al., 2008). It is important to examine delay discounting in humans to obtain a behavioral measure of inability to delay gratification, which may underlie addictive behaviors. Furthermore, delay discounting should be examined while individuals are under the effects of caffeine.

The goal of this study is to examine caffeine's influence on delay discounting in humans. Participants performed a delay-discounting task after having ingested a placebo or caffeine. Comparisons were made within participants as each individual underwent both placebo and caffeine trials. As assignments to placebo and caffeine were cross-balanced, comparisons were made between orders of assignments as well.

Method

Participants

Participants were 28 undergraduate students (18 men, 10 women) at James Madison University (JMU). The majority of participants were Caucasian (54% White, 4% Black/African American, 4% Hispanic, 7% Other, 32% Not-Specified). Inclusion criteria included being at least 18 years of age and endorsing little to no caffeine consumption on an online screener survey. Potential participants who endorsed consuming more than an average of 12 oz of cola or tea per day or 5 oz of coffee per week were excluded from participation. Furthermore, individuals were excluded if they reported smoking, illicit drug use, or taking prescription drugs (with the exception of birth control). Women who were pregnant or were attempting to become pregnant were also excluded, as were individuals whose body mass index (BMI) was below "normal" or above "overweight." If individuals endorsed having medical conditions that might be exacerbated by caffeine consumption (e.g., allergy to caffeine, high blood pressure), they were excluded from the study.

Participants were recruited through the JMU Psychology Department's online participant pool. If potential participants met inclusion criteria based on responses to the online screener, they were contacted via email to schedule an appointment for two laboratory sessions. Those who completed both sessions of the study were given 3 hrs of course credit: 1 hr for the first session and 2 hrs for the second session.

Design

Participants completed two laboratory sessions in a repeated-measures design. In one session, participants were given 200 mg of caffeine, dissolved in orange juice. In the other session, participants were given a placebo, consisting of only the orange juice. Participants were randomly assigned to receive either caffeine or placebo the first session, and the order of conditions was counterbalanced across participants. Caffeine administration was double-blind; neither the participant nor the administrator knew to which condition the participant was assigned. Between 4 days and 1 week passed between sessions; participants were encouraged to schedule sessions on the same day of the week and at the same time.

Measure

During the laboratory sessions, seven participants completed a computerized version of the Hypothetical Money Choice Task (HMCT: Richards et al, 1999), which is a titrating version of the delay-discounting task (Holt, Myerson, & Green, 2003), and is designed to converge towards an individual's indifference point for a given delay and delayed amount of money. On each trial, participants were given the choice between two different amounts of hypothetical money: a small reward available immediately, and a large reward delayed by an amount of time ranging from 1 week to 25 years.

On this task, participants' responses determined the next immediate reward presented (Estle, Green, Myerson, & Holt, 2006). If participants chose the smaller but more immediate reward, for instance, the next immediate reward would be reduced in amount by one-half the difference between the two reward values. Likewise, if participants chose the larger delayed reward, the next immediate reward would be increased in amount by

one-half the difference. For instance, if the participant first chose $500 available immediately over $1000 available after a delay, the next choice would be between $250 available immediately (one-half the difference between $500 and $1000) and $1000 available after a delay. Alternatively, if the same participant instead first chose $1000 delayed in time over $500 available immediately, the next choice would be between $750 available immediately or $1000 available after a delay.

After the first choice, the adjustment in the immediate choice was one-half of the previous adjustment. Therefore, if the second choice was between $250 available immediately or $1000 delayed, because the last adjustment was $250 subtracted from $500, the next adjustment would be $125 added to or subtracted from the immediate amount. If the participant chose $250 for the second choice, then the third choice would be between $125 available immediately and $1000 delayed in time. Alternatively, the choice could be between $375 available immediately versus $1000 delayed. The final indifference point was one-half of the previous adjustment added to or subtracted from the last amount available immediately. Each delay is represented by six choices, or six adjustments (see the Appendix for an example of the titration process).

The other 21 participants received an alternate version of the computerized HMCT (Field et al., 2006). This version, instead of titrating towards an individual's indifference point for a given delay, uses a crossover method. In this method, a series of 27 choices for each delay is presented to participants at random. When data are sorted by immediate amount and delay, participants will often choose the larger later reward up to a certain point, where they crossover and begin choosing the smaller sooner reward. The average

of the amount before and after the crossover serves as the indifference point (see the Appendix for an example sorted sequence of immediate amounts).

Procedure

At the start of a session, a research assistant read and presented informed consent to the participant. Then the participant was weighed (for BMI) and connected to Criticare Systems for physiological measurement. A baseline reading of blood pressure and heart rate was taken while the participant watches episodes of the television show "Friends." After 10 min, placebo or 200 mg caffeine was given to the participant in orange juice. Participants were given 5 min to consume the orange juice. The participant then continued to watch "Friends" for 30-45 min while the caffeine took effect. After participants finished watching "Friends," they began the HMCT computerized delay-discounting task. Sessions ran between 1-1.5 hrs. If the participant experienced discomfort or had an elevated blood pressure or heart rate, the research assistant offered to play a relaxation tape until vitals returned to a safe level.

Results

Due to technical complications, complete data from two participants (15 and 16) were lost, as were partial data from two other participants (13 and 21). Furthermore, two participants (18 and 28) were excluded for other reasons (failure to meet BMI criteria and apparent intoxication, respectively). Indifference points for the remaining 24 participants were calculated (as described above). The hyperbolic discounting model (Mazur, 1987) was then fit to the group and individual data. The group R^2 values under caffeine and placebo were .99 and .96, respectively. Furthermore, the median R^2 values for participants under caffeine and placebo were .83 and .87, respectively. Together, these data suggest that Mazur's hyperbolic model did a nice job of describing the group data as well as most individual cases (see Table 1 for individual k and R^2 values). In 11 cases involving 8 participants, though, fit indices were less than .10. These poor fits demonstrated that some participants discounted delayed rewards in a non-systematic fashion; they also show that the hyperbolic model does not always do a good job describing individual data (e.g., Green, Myerson, & McFadden, 1997).

Next, to further screen for individuals who exhibited non-systematic discounting (i.e., response patterns that did not follow the typical negatively accelerated pattern observed in many studies of delay discounting), Johnson and Bickel's (2008) algorithm was utilized. In accordance with their algorithm, participants with (a) any indifference point greater than the one preceding it by an amount of 20% of the delayed reward (i.e., an indifference point that was at least $200 greater than the preceding indifference point), or (b) participants having a final indifference point not less than the first indifference point by an amount of 10% of the delayed reward (i.e., if the last indifference point was

not at least $100 less than the first indifference point), were excluded. In using this algorithm, if a participant demonstrated non-systematic discounting in either or both lab sessions, that participant was excluded from further analysis. This resulted in the exclusion of an additional seven participants (1, 4, 7, 12, 14, 20, and 23), many of whom had indices of fit near zero. Therefore, the following analyses were performed on the remaining 15 participants (8 men, 7 women). Like the full sample, the majority of participants were Caucasian (47% White, 7% Black/African American, 7% Hispanic, 13% Other, 27% Not-Specified).

Although the derived *k* parameters from the discounting functions are useful for describing the form and fit of a curve, as previously mentioned, they do not always do a good job of describing individual cases of discounting. Moreover, derived *k* values make theoretical assumptions about the shape of the discounting function; the purpose of this study, however, was not to provide the best fit to the data, but rather to compare discounting rates under caffeine and placebo (i.e., no caffeine) conditions. Therefore, instead of using *k* values, AUCs (described above), which provide an atheoretical measure of discounting (Myerson et al., 2001), were used to compare rates of discounting between conditions.

First, an independent-samples *t* test was performed to see whether there was an order effect (i.e., whether participants discounted differently depending on the order in which they experienced the conditions). No significant difference in AUC was found between those participants who received caffeine first and those who received the placebo first, $t(13) = 0.63$, $p = .53$. Another independent *t* test was performed to determine if there was

a significant difference between AUCs of those who completed the titrating version of the HMCT and those who completed the crossover version of the task. No significant difference was found, $t(13) = 0.92$, $p = .38$. Therefore, the data were collapsed into two conditions: caffeine and placebo.

Next, a dependent-samples *t* test was performed to examine differences in AUC between the caffeine and placebo conditions. Average AUC for the caffeine condition was .30 (*SD* = .26) and was .36 (*SD* = .31) for the placebo condition, suggesting that under caffeine, participants discounted slightly faster (i.e., were more impulsive) than when they responded under placebo. This difference, however, was not statistically significant, $t(14) = 1.03$, $p = .32$. The results of this analysis are portrayed graphically in Figure 1.

Discussion

To assess the differences in rates of delay discounting under the influence of caffeine versus placebo, 15 undergraduate participants completed two laboratory sessions. In one laboratory session, participants were given 200 mg of caffeine via orange juice. In the other laboratory session, participants were given just orange juice. In both sessions, participants completed a delay-discounting task after allowing the caffeine to take effect. There was no significant difference between caffeine session AUCs and placebo session AUCs.

This study had two goals: to examine the relation between caffeine and delay discounting, and to examine the influence of drug state on delay discounting. Although more research will be needed to clarify the relation between caffeine and delay discounting, the present study demonstrated that delay discounting can be assessed while under the influence of a drug. Potentially other measures of impulsivity can be measured under drug influence as well. Few studies have researched whether influence of a drug increases or decreases delay discounting. In one of these few studies that examined drug state, Ortner and colleagues (2006) compared delay discounting in college students given three mixed drinks (alcohol), non-alcoholic drinks with the rims dipped in alcohol (placebo), or students assigned to control. The researchers found that individuals in the placebo and alcohol conditions were less impulsive than controls. More research will be needed in order to investigate the influence of drug state on delay discounting and impulsivity in general, and the model used in the present study is a good way to examine such influences.

This study suggests that caffeine may not influence impulsivity. These observations support other findings in experimental studies examining the effects of caffeine on impulsivity. For instance, Roehrs et al. (2004) compared the effects of 0, 150, and 300 mg of caffeine on a risk-taking task and found no significant differences. Likewise, Marczinski and Fillmore (2003) gave 0, 2, and 4 mg kg^{-1} of caffeine to participants and evaluated their performances on a cued go/no-go behavioral inhibition task. Again, no differences were observed.

If caffeine does not have an effect on impulsivity, as the present study suggests, it would differ from other drugs that have been found to influence impulsivity, including alcohol (Petry, 2001), opiates (Kirby et al., 1999), and cocaine (Hiel et al., 2006). One possible reason for these differences is that caffeine seems to affect different areas of the brain. For instance, Nehlig (1999) noted that compared to two other stimulants, amphetamine and cocaine, caffeine activates different areas in the brain associated with reward. Specifically, whereas classical drugs of abuse lead to dopamine release in the nucleus accumbens (the key structure for reward and addiction), caffeine releases dopamine in the prefrontal cortex. Thus, perhaps this difference in dopamine pathways may explain drug differences in reward-related behaviors, such as delay discounting.

Although the present findings support some previous research, they also conflict with other studies. For instance, these results did not support the findings in Diller et al. (2008), which has been one of few studies to examine caffeine's effect on delay discounting. They found that rodents given an acute dose of caffeine (10, 17, and 30 mg) exhibited less impulsivity as dosage increased. When examining chronic administration

of caffeine, they found that this effect was lowered, but not back to baseline. This may indicate a species difference between how humans and rodents are affected by caffeine. This is doubtful, however, as a body of research has demonstrated that the effects of caffeine in rodents are comparable to the effects of caffeine in humans (Fredholm, Battig, Holmen, Nehlig, & Zvartau, 1999; Nehlig, 1999). More research is needed to determine if caffeine decreases impulsivity, and if so, how.

Differing research methods may provide one possible explanation for these conflicting bodies of research. Correlative studies have more often found significant associations between levels of caffeine intake and impulsivity than experimental studies (e.g., Jones & Lejuez, 2005; Landrum, 1992; Waldeck & Miller, 1997). It may be that caffeine alone does not cause impulsivity, but another variable, group of variables, or an interaction of variables mediates the effect. Furthermore, the studies finding a correlation between caffeine use and impulsivity often used self-report questionnaires, whereas studies not finding differences between caffeine conditions often used behavioral measures. Research has shown that behavioral measures of impulsivity may not correlate with self-report measures (e.g., Lane et al., 2003), which suggests that the two types of measures may assess different kinds of impulsivity, different constructs altogether, or different elements of the same construct. Perhaps caffeine may contribute to only certain kinds of impulsivity.

Another explanation for conflicting research findings is that perhaps a high enough dosage of caffeine was not used. The effects of caffeine on impulsivity may be evident in humans only at higher doses. Such dose-dependent responses have been shown in prior

literature (Diller et al., 2008; Marczinski & Fillmore, 2003). The present study used 200 mg of caffeine, roughly the equivalent of two cups of coffee (Barone & Roberts, 1996). However, the present study did not ask participants if they experienced the effects of caffeine on a given trial. These data would be useful because if participants could not accurately guess whether they received caffeine or placebo, then it is possible they were not given enough caffeine to experience its effects, which might lead to no differences between conditions. This explanation is unlikely, though, because participants reported not being regular consumers of caffeine. Nevertheless, a manipulation check should be considered for future research.

It is still unclear whether caffeine influences impulsivity. Furthermore, if it does, the direction of the effect has not yet been confirmed. If caffeine does not affect impulsivity, as the present study suggests, there may be important clinical implications. Perhaps simply using a drug may not affect impulsivity. Rather, maybe it is an addiction to drugs that influences impulsivity. This study and others (e.g., Richards et al., 1999) suggest that drug state alone may not influence delay discounting, but addiction may (Dixon et al., 2003). If this is the case, then successful treatments of addiction should be applicable to any drug addiction or any behavioral addiction.

A secondary implication of this study is that no significant difference was found in AUCs between a titration delay-discounting task and a crossover delay-discounting task. This is important experimentally because titration requires fewer trials in order to arrive at an indifference point. Potentially, students presented with a large number of trials may lose motivation to complete an experimental task, which may then affect the validity of

the results (Arvey, Strickland, Drauden, & Martin, 1990; Wolf, Smith, & Birnbaum, 1995). However, because no difference was found in AUCs between the two tasks, researchers may not have to worry about affecting validity when choosing which delay-discounting task to use.

This study has a few limitations that need to be considered. This study had a small sample size, due in part to difficulty finding and recruiting caffeine-naïve students. This is a small limitation, however, because of the repeated-measures design of the study. Furthermore, data from some participants were completely or partially lost. Because of the within-participants design of this study, any participant with only partial data was completely excluded. Data lost from these individuals may have strengthened or altered current findings.

Another limitation is that this study did not account for individual variance in intelligence. Research has suggested that individuals with lower cognitive ability are more impulsive on delay-discounting tasks than those with higher cognitive ability (see Shamosh & Gray, 2007). Therefore, it is important to take intelligence into account when analyzing delay discounting. Future research may consider measuring intelligence and including the measure as a covariate to verify this assumption.

There is the potential limitation that the homogeneity of the sample influenced results, as the sample was predominantly Caucasian. Some research has suggested that Caucasian and African American individuals differ on delay discounting (e.g., de Wit, Flory, Acheson, McCloskey, & Manuck, 2007). However, due to the repeated measures

design of the present study, each individual served as their own control. In other words, individual differences were controlled for experimentally.

The present study has suggested that individuals given a placebo or 200 mg of caffeine do not differ in delay discounting. However, more research into the effects of caffeine on delay discounting is needed. Future research should include higher doses to test for dose-dependent effects. In examining the influence of drug state on delay discounting or impulsivity, caffeine consumption should be checked chemically through biological measures. This would ensure that participants only consume caffeine within the study and that any effect of is due to the given dosage of caffeine, rather than a combination of caffeine dose from both within and outside the study. Other measures of impulsivity, both behavioral and self-report, should be compared, and intelligence will need to be assessed in future studies. Different populations should also be studied to determine if similar results are found outside of college-aged students.

Furthermore, in order to assess whether drug addiction or drug state influences impulsivity, a future study could compare caffeine addicts under placebo and under caffeine. If it is drug addiction, rather than the drug itself that influences impulsivity, individuals under placebo could be more impulsive due to caffeine deprivation (e.g., Field et al., 2006). Together, these potential future studies should provide useful information on the effects of caffeine on impulsivity.

Table 1

Indices of Fit and Derived Parameters for the Hyperbolic Discounting Function

ID	Condition	R^2	k
1	No Caffeine	0.000	0.000
	Caffeine	0.795	64.230
2	No Caffeine	0.993	0.004
	Caffeine	0.980	0.003
3	No Caffeine	0.979	0.003
	Caffeine	0.895	0.003
4	No Caffeine	0.929	0.001
	Caffeine	0.000	0.000
5	No Caffeine	0.902	0.818
	Caffeine	0.587	0.623
6	No Caffeine	0.956	12.599
	Caffeine	0.899	20.074
7	No Caffeine	0.980	0.023
	Caffeine	0.000	6.969
8	No Caffeine	0.979	0.081
	Caffeine	0.876	0.155
9	No Caffeine	0.987	0.111
	Caffeine	0.962	0.064
10	No Caffeine	0.000	0.003
	Caffeine	0.950	0.065
11	No Caffeine	0.000	0.001
	Caffeine	0.000	0.005
12	No Caffeine	0.000	318.173
	Caffeine	0.696	2.333
14	No Caffeine	0.955	0.000
	Caffeine	0.910	0.000
17	No Caffeine	0.841	0.033
	Caffeine	0.770	0.007
19	No Caffeine	0.884	0.010
	Caffeine	0.981	0.168

20	No Caffeine	0.000	30.616
	Caffeine	0.000	35.025
22	No Caffeine	0.640	0.701
	Caffeine	0.825	0.673
23	No Caffeine	0.021	0.000
	Caffeine	0.000	0.000
24	No Caffeine	0.871	2.249
	Caffeine	0.890	2.345
25	No Caffeine	0.690	0.561
	Caffeine	0.871	0.147
26	No Caffeine	0.974	0.012
	Caffeine	0.974	0.017
27	No Caffeine	0.868	0.006
	Caffeine	0.947	0.015
28	No Caffeine	0.581	0.044
	Caffeine	0.721	0.015

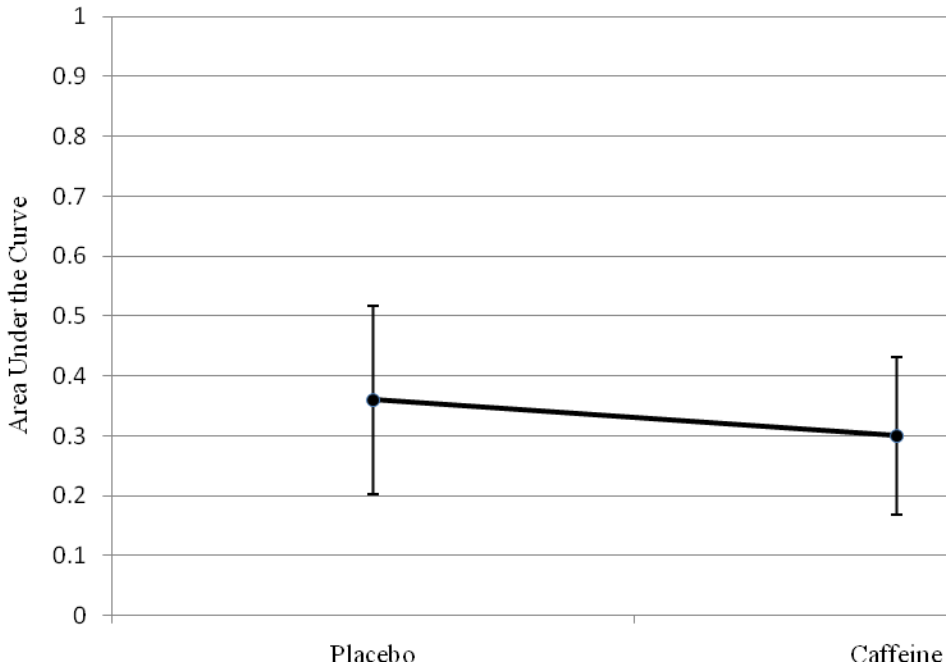

Figure 1. Areas Under the Curve for Placebo and Caffeine Conditions with 95% Confidence Intervals

Appendix

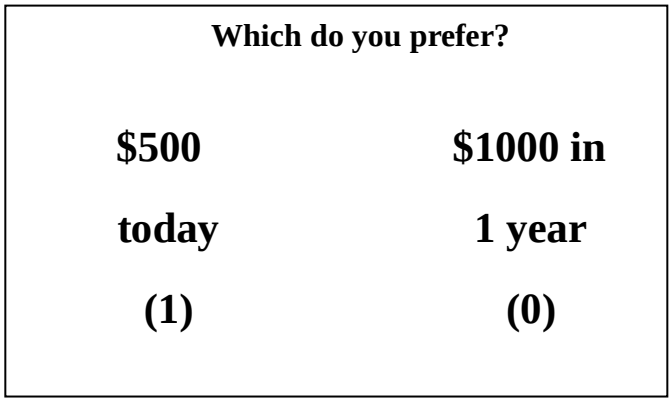

Figure 2. Example Hypothetical Money Choice Task (HMCT) stimuli

Table 2

Example Progression through HMCT Titration Trials for 1 Delay

Trial	Immediate Amount	Delayed Amount	Amount of Previous Adjustment	Participant's Choice
Trial 1	500.00	1000.00		Delayed
Trial 2	750.00	1000.00	250.00	Delayed
Trial 3	875.00	1000.00	125.00	Immediate
Trial 4	812.50	1000.00	62.50	Delayed
Trial 5	843.75	1000.00	31.25	Delayed
Trial 6	859.38	1000.00	15.63	Immediate
Final Indifference Point	851.57	1000.00	7.81	

Table 3

Example Progression through HMCT Crossover Trials for 1 Delay

Immediate Amount	Delayed Amount	Participant's Choice	Indifference Point if Participant Crosses Over Here
1	1000	Delayed	0.5
5	1000	Delayed	3
10	1000	Delayed	8
20	1000	Delayed	15
40	1000	Delayed	30
60	1000	Delayed	50
80	1000	Delayed	70
100	1000	Delayed	90
150	1000	Delayed	125
200	1000	Delayed	175
250	1000	Delayed	225
300	1000	Delayed	275
350	1000	Delayed	325
400	1000	Delayed	375
450	1000	Delayed	425
500	1000	Delayed	475
550	1000	Delayed	525
600	1000	Delayed	575
650	1000	Delayed	625
700	1000	Delayed	675
750	1000	Delayed	725
800	1000	Delayed	775
850	1000	Immediate	825
920	1000	Immediate	885
960	1000	Immediate	940
990	1000	Immediate	975
1000	1000	Immediate	995

References

Ainslie, G. (1975). Specious reward: A behavioral theory of impulsiveness and impulse control. *Psychological Bulletin, 82*(4), 463-496.

Ainslie, G., & Haendel, V. (1983). The motives of the will. In E. Gottheil, K. Druley, T. Skodola, & H. Waxman (Eds.), *Etiologic Aspects of Alcohol and Drug Abuse* (pp. 119–140). Springfield, IL: Charles C. Thomas.

American Psychiatric Association. (1994). Diagnostic and statistical manual of mental disorders (4th ed.). Washington, DC: Author.

Arvey, R. D., Strickland, W., Drauden, G., & Martin, C. (2006). Motivational components of test taking. *Personnel Psychology, 43(4),* 695-716.

Baker, F., Johnson, M. W., & Bickel, W. K. (2003). Delay discounting in current and never-before cigarette smokers: Similarities and differences across commodity, sign, and magnitude. *Journal of Abnormal Psychology*, *112*(3), 382-392.

Barkley, R. A. (1997). Behavioral inhibition, sustained attention, and executive functions: Constructing a unifying theory of ADHD. *Psychological Bulletin, 121*, 65-94.

Barone, J. J., & Roberts, H. R. (1996). Caffeine consumption. *Food and Chemical Toxicology*, *34*(1), 119-129.

Benowitz, N. L. (1990). Clinical pharmacology of caffeine. *Annual Review of Medicine*, *41*(1), 277-288.

Bickel, W. K., Odum, A. L., Madden, G. J. (1999). Impulsivity and cigarette smoking: Delay discounting in current, never, and ex-smokers. *Psychopharmacology, 146,* 447-454.

Bonham, G. S., & Leaverton, P. E. (1979). Use habits among adults of cigarettes, coffee, aspirin, and sleeping pills. *Vital and Health Statistics Series 10, Data from the National Health Survey, 131*, 1-48.

Cauli, O., & Morelli, M. (2005). Caffeine and the dopaminergic system. *Behavioural Pharmacology, 16*(2), 63-77.

Chapman, G. B., & Elstein, A. S. (1995). Valuing the future: Temporal discounting of health and money. *Medical Decision Making, 15*(4), 373-386.

Childs, E., & de Wit, H. (2006). Subjective, behavioral, and physiological effects of acute caffeine in light, nondependent caffeine users. *Psychopharmacology, 185*(4), 514-523.

Childs, E., & de Wit, H. (2008). Enhanced mood and psychomotor performance by a caffeine-containing energy capsule in fatigued individuals. *Experimental and Clinical Psychopharmacology, 16*(1), 13-21.

Chung, S. (1965). Effects of delayed reinforcement in a concurrent situation. *Journal of the Experimental Analysis of Behavior, 8*(6), 439-444.

Colton, T., Gosselin, R. E., & Smith, R. P. (1968). The tolerance of coffee drinkers to caffeine. *Clinical Pharmacology and Therapeutics, 9*(1), 31-39.

Cornelis, M. C., & El-Sohemy, A. (2007). Coffee, caffeine, and coronary heart disease. *Current Opinion in Lipidology, 18*(1), 13-19.

Daly, J. W., & Fredholm, B. B. (1998). Caffeine—an atypical drug of dependence. *Drug and Alcohol Dependence, 51*(1-2), 199-206.

de Villiers, P. A., & Herrnstein, R. J. (1976). Toward a law of response strength. *Psychological Bulletin, 83*(6), 1131-1153.

de Wit, H., Flory, J. D., Acheson, A., McCloskey, M., & Manuck, S. B. (2007). IQ and nonplanning impulsivity are independently associated with delay discounting in middle-aged adults. *Personality and Individual Differences, 42(1),* 111-121.

Denaro, C. P., & Benowitz, N. L. (1991). Caffeine metabolism: Disposition in liver disease and hepatic-function testing. *Drug and Alcohol Abuse Reviews.Liver Pathology and Alcohol, 2*, 513–539.

Diller, J. W., Saunders, B. T., & Anderson, K. G. (2008). Effects of acute and repeated administration of caffeine on temporal discounting in rats. *Pharmacology, Biochemistry and Behavior, 89,* 546-555.

Dixon, M. R., Marley, J., & Jacobs, E. A. (2003). Delay discounting by pathological gamblers. *Journal of Applied Behavior Analysis, 36,* 449-458.

Dunwiddie, T. V., & Worth, T. (1982). Sedative and anticonvulsant effects of adenosine analogs in mouse and rat. *Journal of Pharmacology and Experimental Therapeutics, 220*(1), 70-76.

Durlach, P. J. (1998). The effects of a low dose of caffeine on cognitive performance. *Psychopharmacology, 140,* 116-119.

Estle, S. J., Green, L., Myerson, J., & Holt, D. D. (2006). Differential effects of amount on temporal and probability discounting of gains and losses. *Memory and Cognition, 34*(4), 914-928.

Estler, C. J. (1982). Caffeine. *Psychoptropic Agents, Part III: Alcohol and Psychotomimetics: Psychotropic Effects of Central Acting Drugs,* 369-389.

Evans, S. M., & Griffiths, R. R. (1992). Caffeine tolerance and choice in humans. *Psychopharmacology, 108*(1), 51-59.

Evenden, J. (1999). Varieties of impulsivity. *Psychopharmacology, 146*(4), 348-361.

Eysenck, S. B. G., Pearson, P. R., Easting, G., & Allsopp, J. F. (1985). Age norms for impulsiveness, venturesomeness and empathy in adults. *Personality and Individual Differences, 6*(5), 613-619.

Field, M., Christiansen, P., Cole, J., & Goudie, A. (2007). Delay discounting and the alcohol stroop in heavy drinking adolescents. *Addiction, 102*(4), 579-586.

Field, M., Santarcangelo, M., Sumnall, H., Goudie, A., & Cole, J. (2006). Delay discounting and the behavioural economics of cigarette purchases in smokers: The effects of nicotine deprivation. *Psychopharmacology, 186*(2), 255-263.

Fishburn, P. C., Rubinstein, A. (1982). Time preference. *International Economic Review, 23*(3), 677-694.

Fredholm, B. B., Battig, K., Holmen, J., Nehlig, A., & Zvartau, E. E. (1999). Actions of caffeine in the brain with special reference to factors that contribute to its widespread use. *Pharmacological Reviews, 51(1),* 83-133.

Garrett, B. E., & Griffiths, R. R. (1998). Physical dependence increases the relative reinforcing effects of caffeine versus placebo. *Psychopharmacology, 139*(3), 195-202.

Graham, K. (1988). Reasons for consumption and heavy caffeine use: Generalization of a model based on alcohol research. *Addictive Behaviors, 13*(2), 209-214.

Greden, J. F. (1974). Anxiety or caffeinism: A diagnostic dilemma. *American Journal of Psychiatry, 131*(10), 1089-1092.

Green, L., Myerson, J., McFadden, E. (1997). Rate of temporal discounting decreases with amount of reward. *Memory & Cognition, 25,* 715-723.

Griffiths, R. R., & Woodson, P. P. (1988a). Caffeine physical dependence: A review of human and laboratory animal studies. *Psychopharmacology, 94*(4), 437-451.

Griffiths, R. R., & Woodson, P. P. (1988b). Reinforcing effects of caffeine in humans. *Journal of Pharmacology and Experimental Therapeutics, 246*(1), 21-29.

Hartley, T. R., Lovallo, W. R., Whitsett, T. L., Sung, B. H., & Wilson, M. F. (2001). Caffeine and stress: Implications for risk, assessment, and management of hypertension. *Journal of Clinical Hypertension, 3*(6), 354-361.

Hays, W. L. (1994). *Statistics.* Fort Worth, TX: Harcourt Brace College.

Heinz, A. J., Kassel, J. D., & Smith, E. V. (2009). Caffeine expectancy: Instrument development in the rasch measurement framework. *Psychology of Addictive Behaviors, 23*(3), 500-511.

Hicks, R. A., Hicks, G. J., Reyes, J. R., & Cheers, Y. (1983). Daily caffeine use and the sleep of college students. *Bulletin of the Psychonomic Society, 21*(1), 24-25.

Hiel, S. H., Johnson, M. W., Higgins, S. T., & Bickel, W. K. (2006). Delay discounting in currently using and currently abstaining cocaine-dependent outpatients and non-drug-using matched controls. *Addictive Behaviors, 31,* 1290-1294.

Holmgren, P., Nordén-Pettersson, L., & Ahlner, J. (2004). Caffeine fatalities—four case reports. *Forensic Science International, 139*(1), 71-73.

Holt, D. D., Green, L., & Myerson, J. (2003). Is discounting impulsive? evidence from temporal and probability discounting in gambling and non-gambling college students. *Behavioural Processes, 64*(3), 355-367.

Holtzman, S. G. (1990). Caffeine as a model drug of abuse. *Trends in Pharmacological Sciences, 11*(9), 355-356.

Hughes, J. R., Oliveto, A. H., Bickel, W. K., Higgins, S. T., & Badger, G. J. (1995). The ability of low doses of caffeine to serve as reinforcers in humans: A replication. *Experimental and Clinical Psychopharmacology, 3*, 358-363.

Hughes, R. N. (1996). Drugs which induce anxiety: Caffeine. *New Zealand Journal of Psychology, 25*, 36-42.

James, J. E. (2004). Critical review of dietary caffeine and blood pressure: A relationship that should be taken more seriously. *Psychosomatic Medicine, 66*(1), 63-71.

James, J. E., & Paull, I. (1985). Caffeine and human reproduction. *Reviews on Environmental Health, 5*(2), 151-167.

James, J. E., Crosbie, J., & Paull, I. (1987). Symptomatology of habitual caffeine use amongst psychiatric patients. *Australian Journal of Psychology, 39*(2), 139-149.

Johnson, M. W., & Bickel, W. K. (2002). Within-subject comparison of real and hypothetical money rewards in delay discounting. *Journal of the Experimental Analysis of Behavior, 77*(2), 129-146.

Johnson, M. W., & Bickel, W. K. (2008). An algorithm for identifying nonsystematic delay-discounting data. *Experimental and Clinical Psychopharmacology, 16(3),* 264-274.

Johnson, M. W., Bickel, W. K., & Baker, F. (2007). Moderate drug use and delay discounting: A comparison of heavy, light, and never smokers. *Experimental and Clinical Psychopharmacology, 15*(2), 187-194.

Jones, H. A., & Lejuez, C. W. (2005). Personality correlates of caffeine dependence: The role of sensation seeking, impulsivity, and risk taking. *Experimental and Clinical Psychopharmacology, 13*, 259-266.

Juliano, L. M., & Griffiths, R. R. (2004). A critical review of caffeine withdrawal: Empirical validation of symptoms and signs, incidence, severity, and associated features. *Psychopharmacology, 176*(1), 1-29.

Kerrigan, S., & Lindsey, T. (2005). Fatal caffeine overdose: Two case reports. *Forensic Science International, 153*(1), 67-69.

Kirby, K. N. (1997). Bidding on the future: Evidence against normative discounting of delayed rewards. *Journal of Experimental Psychology-General, 126*(1), 54-70.

Kirby, K. N., & Herrnstein, R. J. (1995). Preference reversals due to myopic discounting of delayed reward. *Psychological Science, 6*(2), 83-89.

Kirby, K. N., Petry, N. M., & Bickel, W. K. (1999). Heroin addicts have higher discount rates for delayed rewards than non-drug-using controls. *Journal of Experimental Psychology General, 128*, 78-87.

Lancaster, K. (1963). An axiomatic theory of consumer time preference. *International Economic review, 4*(2), 221-231.

Landolt, H. P., Werth, E., Borbély, A. A., & Dijk, D. J. (1995). Caffeine intake (200 mg) in the morning affects human sleep and EEG power spectra at night. *Brain Research, 675*(1-2), 67-74.

Landrum, R. E. (1992). College students' use of caffeine and its relationship to personality. *College Student Journal, 26(2),* 151-155.

Lane, J. D. (1983). Caffeine and cardiovascular responses to stress. *Psychosomatic Medicine, 45*(5), 447-451.

Lane, S. D., Cherek, D. R., Rhoades, H. M., Pietras, C. J., & Tcheremissine, O. V. (2003). Relationships among laboratory and psychometric measures of impulsivity: Implications in substance abuse and dependence. *Addictive Disorders & their Treatment, 2*(2), 33-40.

Lee, M. A., Cameron, O. G., & Greden, J. F. (1985). Anxiety and caffeine consumption in people with anxiety disorders. *Psychiatry Research, 15*(3), 211-217.

Levy, M., & Zylber-Katz, E. (1983). Caffeine metabolism and coffee-attributed sleep disturbances. *Clinical Pharmacology & Therapeutics, 33*(6), 770-775.

Loewenstein, G. (1996). Out of control: Visceral influences on behavior. *Organizational Behavior and Human Decision Processes, 65*(3), 272-292.

Loke, W. H. (1988). Effects of caffeine on mood and memory. *Physiology & Behavior, 44*(3), 367-372.

Madden, G. J., Begotka, A. M., Raiff, B. R., & Kastern, L. L. (2003). Delay discounting of real and hypothetical rewards. *Experimental and Clinical Psychopharmacology, 11*(2), 139-145.

Marczinski, C. A., & Fillmore, M. T. (2003). Dissociative antagonistic effects of caffeine on alcohol-induced impairment of behavioral control. *Experimental and Clinical Psychopharmacology, 11*(3), 228-235.

Mazur, J. E. (1987). An adjusting procedure for studying delayed reinforcement. *Quantitative Analyses of Behavior, 5*, 55–73.

Meyer, L., & Caston, J. (2004). Stress alters caffeine action on investigatory behaviour and behavioural inhibition in the mouse. *Behavioural Brain Research, 149*(1), 87-93.

Moeller, F. G., Barratt, E. S., Dougherty, D. M., Schmitz, J. M., & Swann, A. C. (2001). Psychiatric aspects of impulsivity. *American Journal of Psychiatry, 158*(11), 1783-1793.

Myerson, J., Green, L., & Warusawitharana, M. (2001). Area under the curve as a measure of discounting. *Journal of the Experimental Analysis of Behavior, 76*(2), 235-243.

Nehlig, A. (1999). Are we dependent upon coffee and caffeine? A review on human and animal data. *Neuroscience and Biobehavioral Reviews, 23,* 563-576.

Noordzij, M., Uiterwaal, C. S. P. M., Arends, L. R., Kok, F. J., Grobbee, D. E., & Geleijnse, J. M. (2005). Blood pressure response to chronic intake of coffee and caffeine: A meta-analysis of randomized controlled trials. *Journal of Hypertension, 23*(5), 921-928.

Ortner, C. N. M., MacDonald, T. K., & Olmstead, M. C. (2003). Alcohol intoxication reduces impulsivity in the delay-discounting paradigm. *Alcohol and Alcoholism, 38*(2), 151-156.

Petry, N. M. (2001). Delay discounting of money and alcohol in actively using alcoholics, currently abstinent alcoholics, and controls. *Psychopharmacology, 154,* 243-250.

Petry, N. M. (2001). Substance abuse, pathological gambling, and impulsiveness. *Drug and Alcohol Dependence, 63*(1), 29-38.

Petry, N. M., & Casarella, T. (1999). Excessive discounting of delayed rewards in substance abusers with gambling problems. *Drug and Alcohol Dependence, 56*(1), 25-32.

Rachlin, H., Raineri, A., Cross, D. (1991). Subjective probability and delay. *Journal of the Experimental Analysis of Behavior, 55(2),* 233-244.

Regina, E. G., Smith, G. M., Keiper, C. G., & McKelvey, R. K. (1974). Effects of caffeine on alertness in simulated automobile driving. *Journal of Applied Psychology, 59*(4), 483-489.

Reynolds, B., Ortengren, A., Richards, J. B., & de Wit, H. (2006). Dimensions of impulsive behavior: Personality and behavioral measures. *Personality and Individual Differences, 40*(2), 305-315.

Richards, J., Zhang, L., Mitchell, S., & De Wit, H. (1999). Delay or probability discounting in a model of impulsive behavior: Effect of alcohol. *Journal of the Experimental Analysis of Behavior, 71*(2), 121-143.

Robertson, D., Wade, D., Workman, R., Woosley, R. L., & Oates, J. A. (1981). Tolerance to the humoral and hemodynamic effects of caffeine in man. *Journal of Clinical Investigation, 67*(4), 1111-1117.

Roehrs, T., Greenwald, M., & Roth, T. (2004). Risk-taking behavior: Effects of ethanol, caffeine, and basal sleepiness. *Sleep, 27*(5), 887-893.

Rush, C. R., Sullivan, J. T., & Griffiths, R. R. (1995). Intravenous caffeine in stimulant drug abusers: Subjective reports and physiological effects. *Journal of Pharmacology and Experimental Therapeutics, 273*(1), 351-358.

Samuelson, P. A. (1937). A note on measurement of utility. *The Review of Economic Studies, 4*(2), 155-161.

Schuh, K. J., & Griffiths, R. R. (1997). Caffeine reinforcement: The role of withdrawal. *Psychopharmacology, 130*(4), 320-326.

Shamosh, N. A., & Gray, J. R. (2008). Delay discounting and intelligence: A meta-analysis. *Intelligence, 36(4),* 289-305.

Shi, J., Benowitz, N. L., Denaro, C. P., & Sheiner, L. B. (1993). Pharmacokinetic-pharmacodynamic modeling of caffeine: Tolerance to pressor effects. *Clinical Pharmacology & Therapeutics, 53*(1), 6-14.

Shohet, K. L., & Landrum, R. E. (2001). Caffeine consumption questionnaire: A standardized measure for caffeine consumption in undergraduate students. *Psychological Reports, 89*(3), 521-526.

Silverman, K., Mumford, G. K., & Griffiths, R. R. (1994). Enhancing caffeine reinforcement by behavioral requirements following drug ingestion. *Psychopharmacology, 114*(3), 424-432.

Smith, A. (2002). Effects of caffeine on human behavior. *Food and Chemical Toxicology, 40*(9), 1243-1255.

Smith, A., Sturgess, W., Gallagher, J. (1999). Effects of a low dose of caffeine given in different drinks on mood and performance. *Human Psychopharmacology: Clinical and Experimental, 14,* 473-482

Snel, J. (1993). Coffee and caffeine: Sleep and wakefulness. In S. Garattini (Ed.), *Coffee, Caffeine and Health* (pp 255–290). New York: Raven Press.

Stavric, B. (1988). Methylxanthines: Toxicity to humans. 2. Caffeine. *Food and Chemical Toxicology, 26*(7), 645-662.

Totten, G. L., & France, C. R. (1995). Physiological and subjective anxiety responses to caffeine and stress in nonclinical panic. *Journal of Anxiety Disorders, 9*(6), 473-488.

Van den Bergh, B., Dewitte, S., & Warlop, L. (2008). Bikinis instigate generalized impatience in intertemporal choice. *Journal of Consumer Research, 35*(1), 85-97.

Veleber, D. M., & Templer, D. I. (1984). Effects of caffeine on anxiety and depression. *Journal of Abnormal Psychology, 93(1), 120-122.*

Vuchinich, R. E., & Simpson, C. A. (1998). Hyperbolic temporal discounting in social drinkers and problem drinkers. *Experimental and Clinical Psychopharmacology, 6*(3), 292-305.

Waldeck, T. L., & Miller, L. S. (1997) Gender and impulsivity differences in licit substance use. *Journal of Substance Abuse, 9,* 269-275.

Whiteside, S. P., & Lynam, D. R. (2001). The five factor model and impulsivity: Using a structural model of personality to understand impulsivity. *Personality and Individual Differences, 30*(4), 669-689.

Wilhelm, C., Reeves, J., Phillips, T., & Mitchell, S. (2007). Mouse lines selected for alcohol consumption differ on certain measures of impulsivity. *Alcoholism-Clinical and Experimental Research, 31*(11), 1839-1845.

Wingrove, J., & Bond, A. J. (1997). Impulsivity: A state as well as trait variable. does mood awareness explain low correlations between trait and behavioural measures of impulsivity? *Personality and Individual Differences, 22*(3), 333-339.

Wolf, L. F., Smith, J. K., & Birnbaum, M. E. (1995). Consequence of performance, test, motivation, and mentally taxing items. *Applied Measurement in Application, 8(4),* 341-351.

Zuckerman, M. (1971). Dimensions of sensation seeking. *Journal of Consulting and Clinical Psychology, 36*(1), 45-52.

Zuckerman, M. (1990). Influences of sensation seeking, gender, risk appraisal, and situational motivation on smoking. *Addictive Behaviors, 15*(3), 209-220.

Zuckerman, M., Eysenck, S., & Eysenck, H. J. (1978). Sensation seeking in england and america: Cross-cultural, age, and sex comparisons. *Journal of Consulting and Clinical Psychology, 46*(1), 139-149.

Zwyghuizen-Doorenbos, A., Roehrs, T. A., Lipschutz, L., Timms, V., & Roth, T. (1990). Effects of caffeine on alertness. *Psychopharmacology, 101*(1), 36-39.