ABSTRACT

AROUSAL THEORY AND THE INTERRELATIONSHIPS OF CAFFEINE, NICOTINE, AND IMPULSIVITY

Thomas James White, M.A., 2006

Directed By: Professor Barry D. Smith, Ph.D., Department of Psychology

Nicotine, caffeine, impulsivity, and arousal are all intercorrelated: both drugs increase arousal, and impulsivity is theoretically related to arousal. However, the independent and joint effects of nicotine and caffeine on impulsive behavior are unclear. In this study, male college students ($N = 63$) were administered either caffeine or lactose placebo (double-blind) and either nicotine or placebo cigarettes (double-blind). Participants engaged in three behavioral tasks: the Stop Signal Task (SST), the Stroop Color-Word Test (SCWT), and the Delay Discounting Task (DDT). Drug intake did not produce significant changes across conditions on any of the three tasks. The hypothesis that caffeine and nicotine have an interactive effect on impulsivity in men was not supported by the data. Potential reasons for the lack of significant findings include variability within the sample on consumption history.

AROUSAL THEORY AND THE INTERRELATIONSHIPS OF CAFFEINE, NICOTINE, AND IMPULSIVITY

By

Thomas James White

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Advisory Committee:

Professor B.D. Smith, Ph.D., Chair

Dr. C.W. Lejuez, Ph.D.

Dr. Kevin E. O'Grady, Ph.D.

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Introduction

Arousal theory postulates that impulsive individuals are under-stimulated at rest, have high sensitivity to signals of reward, and seek behavioral activation to raise their cortical arousal (H. J. Eysenck, 1947; Gray, 1975; McDougall, 1929; Pavlov, 1927/1960; Smith, 1994). As such, arousal or activation is an important contributor to a wide range of behaviors. A current arousal model suggests that an individual's overall level of arousal at a given moment is an interactive function of a number of factors, including genetic predispositions, personality characteristics, drugs, and situational stimuli in the environment (Smith, 1994; Wilken, Smith, Tola, & Mann, 1999). The purpose of the present study was to examine the main and interactive effects of two drugs (caffeine and nicotine), a personality factor (impulsivity), and experimental stimuli on aspects of arousal. A number of studies have examined the place of caffeine use within arousal theory (e.g. Bullock & Gilliland, 1993; Davidson, Fedio, Smith, Aurielle, & Martin, 1992; Gupta, Singh, & Gupta, 1999; Smith, Kline, & Meyers, 1990). However, fewer studies have examined the relationship of smoking to arousal (Rose, 1986; Rose & Behm, 1989; Rose, et al., 2003), and none have examined the effects of both drugs in combination on impulsive behavior and with respect to impulsive personality, within an arousal theory framework.

Hans Eysenck's seminal work on personality identifies impulsivity as an important personality factor deriving from extraversion, a broader typological dimension that is one of three principal factors in his personality model (H. J. Eysenck, 1947; S. B. G. Eysenck & H. J. Eysenck, 1977); in this model, extraversion is a dimension of activation that reflects cortical arousal potentials. Eysenck's model proposes that the

reticular activating system is inherently sensitive to stimulation, and thus, extraverts have more capacity for responding to stimuli at high intensities than do introverts (Zuckerman, 1985). Another definition of impulsivity, similar but not identical to Eysenck's definition, is the oversensitivity to signals of reward and undersensitivity to signals of punishment (Patterson & Newman, 1993; Wallace, Newman, & Bachorowski, 1991; Zuckerman, 1985). This model posits that impulsive individuals are those who consistently seek out stimulation, focusing on the rewards and not focusing on punishment. Zuckerman's *sensation seeking* factor strongly correlates with both of these definitions of impulsivity, both conceptually and empirically, defining sensation seeking as a "personality trait directly measuring the seeking of arousal stimulation" (Zuckerman, 1985).

Impulsivity is a normal personality trait (Gillespie, Cloninger, Heath, & Martin, 2003; S. B. G. Eysenck, Pearson, Easting, & Allsop, 1985; Gray, 1987; Zuckerman, 1993). However, due to the very nature of impulsivity as being undersensitive to punishment, impulsivity is also a hallmark feature of a number of DSM-IV psychiatric diagnoses; these include Antisocial Personality Disorder, Attention Deficit/Hyperactivity Disorder (ADHD), Bipolar Disorder, Borderline Personality Disorder (BPD), Bulimia Nervosa, Intermittent Explosive Disorder, Kleptomania, Pathological Gambling, Pyromania, Substance Use Disorders, and Trichotillomania (American Psychiatric Association, 1994). Impulsivity is seen in higher levels in those with any DSM-IV Axis I or Axis II diagnoses than in normals (Moeller, Barratt, Dougherty, Schmitz, & Swann, 2001), and is considered by some to be the defining feature of ADHD (Rubia, 2002). Personality measures of impulsivity predict a wide variety of psychiatric disorders and other risky or problematic behaviors (S. B. J. Eysenck & H. J. Eysenck, 1978; Sher &

Trull, 1994; Stanford, Greve, Boudreaux, Mathias, & Brumbelow, 1996; Zuckerman & Kuhlman, 2000).

Any substance that systematically affects arousal may well differentially affect those low and high on a personality dimension that is based on arousal differentials (i.e. impulsivity). Nicotine, a drug that produces physiological arousal, is one such substance. While the relationship of impulsivity to illegal arousal drugs has been studied, the relationship of impulsivity to smoking, a common and legal form of drug use, deserves examination (Acton, 2003). Nicotine is a powerful drug that reaches its maximum brain concentrations within one minute of smoke inhalation (United States Department of Health and Human Services [USDHHS], 1988) and smoking one to two cigarettes (with a typical nicotine yield of 0.6 to 1.4mg per cigarette) reduces subjective distress while increasing arousal of the nervous system (Gilbert, 1979; Parrott, 1998). At typical doses of one to two cigarettes in short succession, nicotine affects nicotinic acetylcholine receptors (nAChRs) to activate reward centers in the central nervous system (CNS), increasing dopamine (DA) and epinephrine release in the cerebral cortex of the brain (Mansvelder & McGehee, 2002; National Institute on Drug Abuse [NIDA], 2005; Nutt, 1997). Additionally, nicotine increases regional cerebral blood flow (rCBF) in the left frontal region of the cortex, and decreases rCBF in the left amygdala and the right hemisphere of the cortex (Rose, et al., 2003). Laboratory studies of nicotine (e.g. Garrett & Griffiths, 2001) have shown a dose-dependent positive effect on mood, subjective "high," and liking for drug, at a rate as high as 3.0 mg per 70 kg of body weight – roughly the equivalent of two cigarettes smoked in succession for an average weight male college student. Thus, nicotine may be considered a stimulant drug in terms of its effects

on physiological arousal, although it has a calming effect on the orthogonal construct of mood (Parrott, 1998), Dose effects on activation have been found for nicotine, such that lower doses equivalent to those received from smoking one or two cigarettes, increase reticular activation, but doses higher than those obtained from normal smoking, when administered in the laboratory, decrease reticular activation (Rose, et al., 2003). Such physiological findings are consistent with the psychological findings that low doses of nicotine (such as those received from the first cigarette of the day) produce a stimulant effect, but high doses of nicotine, equivalent to smoking a high number of cigarettes in short succession, produce a sedative effect (Ashton, et al., 1980; Clarke, 1987). These physiological and psychological dose effects for nicotine are consistent with the inverted-U-shaped arousal function, which states that the amount of arousal produced as a function of stimulant intake follows an inverted-U-shaped pattern. The optimal level of arousal is achieved by a specific dose of the drug (in this case, typical doses of nicotine); greater doses will actually reduce arousal in accordance with the Yerkes-Dodson law (Smith, 1994).

Understanding reasons for nicotine use (i.e. smoking), including the effects of nicotine on arousal and impulsivity, are important for a number of reasons. Not the least of these reasons is the fact that cigarette smoking is the number one cause of preventable death in the United States and other developed countries, causing over 440,000 deaths annually in the Unites States alone and resulting in over $75 billion in direct medical costs (Bergen & Caporaso, 1999; Centers for Disease Control and Prevention [CDC], 2002; NIDA, 2005). It is also a highly addictive habit, and reflecting this, Nicotine Dependence and Nicotine Withdrawal are listed as mental disorders in the Diagnostic and

Statistical Manual of Mental Disorders (DSM-IV; American Psychiatric Association, 1994). Over 90% of teenagers who smoke four cigarettes will develop nicotine dependence, and will smoke (on average) for a period of 30 to 40 years (Russell, 1990).

Caffeine, like nicotine, is a legal substance that systematically affects physiological arousal, acting as a strong CNS and skeletal muscle stimulant: in doses of 50-300 mg, caffeine has been shown to stimulate the cerebral cortex (Clementz & Dailey, 1988; Bullock & Gilliland, 1984). As doses of caffeine exceed 300mg, stimulation of the medulla, vagus, and vasomotor centers also occurs, and doses of caffeine exceeding 500mg should be avoided to reduce the risk of decrements in performance (Bullock & Gilliland, 1984). Caffeine also stimulates the cardiac muscle, increasing heart rate, and dilates blood vessels and constricts blood flow in the cerebral cortex, which can relieve headaches (Bullock & Gilliland, 1984). The primary effect of caffeine is to block adenosine receptors, which improves cognitive and psychomotor performance and improves affect (Nutt, 1997; Smith, Osborne, Mann, Jones, & White, 2004). Moderate levels of chronic caffeine use can reduce the effects of acute exposure to caffeine; however, high chronic levels can exacerbate the effects of acute exposure (Smith, 1994; Smith, et al. 2002).

No significant health risks are associated with moderate caffeine use. However, excessive caffeine use can have harmful side effects, and these should be taken seriously. Caffeine is the most widely available and commonly used drug in the world, with 80% of the world's population consuming caffeine daily (R. M. Gilbert, 1984; Pendergrast, 1999). Case studies have shown that some individuals experience a syndrome of anxiety, irritability, headache, dizziness, and other symptoms, known as "caffeinism" (Greden,

1974). Caffeine has also been shown to disproportionately increase anxiety in panic patients compared with normals (Lee, Flegel, Greden, & Cameron, 1988), and can cause panic attacks in those with or without panic disorder (Krystal, Deutsch, & Charney, 1996; Kuboki & Suematsu, 1994). The DSM-IV recognizes Caffeine Intoxication, Caffeine-Induced Anxiety Disorder, and Caffeine-Induced Sleep Disorder; it also recommends more research be conducted on the validity of Caffeine Withdrawal as a diagnosis. Despite these potential side effects, however, the results appear to be mixed for caffeine; unlike nicotine, caffeine is considered to have a low dependence potential (Feinstein, et al., 2000; Nehlig, 1999), and the overall results appear to be mixed regarding the benefits and dangers of caffeine use (Smith & Tola, 1998; Smith, et al. 2004).

Nicotine and caffeine may affect behavior in a variety of ways related to arousal. This study was designed to discover whether caffeine and nicotine use increase, decrease, or have no effect on impulsive behavior. Additionally, the potential interactive effects of caffeine and nicotine intake on impulsive behavior were assessed, as was the relationship between impulsive personality and responses to caffeine and nicotine. Replicated research has shown that male smokers experience heightened sensitivity to the pharmacological effects of nicotine compared with female smokers (Benowitz & Hatsukami, 1998; Daurignac, Perez-Diaz, Grillon, & Jouvent, 2001; Ikard & Tomkins, 1973; Perkins, 1996; Perkins, Jacobs, Crow, & Blackburn, 2002). For this reason, men were recruited as the study population.

The reason for conducting the present study was to determine potential reasons for the use of caffeine and nicotine, independently and in conjunction. If caffeine use or cigarette smoking increases impulsive behavior, it is possible that extraverted individuals

intentionally use these drugs as a means of increasing impulsivity, perhaps for pleasure associated with this increase. Such findings would be consistent with H.J. Eysenck's (1947) theory that impulsivity is a subfactor of arousal, and thus, extraverted individuals are impulsive individuals; and that extraverted, impulsive individuals are chronically underaroused, and seek external excitement to raise their level of arousal (H.J. Eysenck, 1947; H. J. Eysenck & M. W. Eysenck, 1985). However, if caffeine use or cigarette smoking decreases impulsive behavior, it is possible that impulsive individuals seek out these drugs as a means of regulating their behavior and reducing their excessive baseline impulsivity. Alternatively, perhaps impulsive people are differently affected by arousal than nonimpulsives and therefore are more likely to seek out these drugs. Consistent with arousal theory, impulsives and nonimpulsives have been shown to experience different effects from caffeine (e.g. Craig, Humphreys, Rocklin, & Revelle, 1979; Gupta, et al., 1999; Revelle, Humphreys, Simon, & Gilliland, 1980) and nicotine (D. G. Gilbert, 1979; D. G. Gilbert & B. O. Gilbert, 1995). For this reason, baseline impulsive personality was assessed to examine potential interactions of baseline impulsivity and drug effects.

Study Hypotheses

For the present experiment, the following hypotheses were made:

1. Nicotine intake decreases impulsive behavior compared with placebo;
2. Caffeine intake increases impulsive behavior compared with placebo;
3. The combined intake of nicotine and caffeine decreases impulsive behavior compared with caffeine.

These hypotheses reflected several considerations. First, nicotine has been shown in previous research to decrease impulsive behavior in the absence of caffeine (D.G.

Gilbert, 1979), and thus, it was hypothesized that nicotine intake decreases impulsivity. Second, caffeine should, theoretically, increase impulsive behavior in the absence of nicotine, due to its well-established effect on cortical and sympathetic activation (e.g. Nehlig, Daval, & Debry, 1992), to which impulsivity is theoretically tied. Thus, it was hypothesized that caffeine intake increases impulsivity as a function of its increasing arousal. Third, it was hypothesized that nicotine intake blocks the increase in impulsivity from caffeine, such that individuals who receive both caffeine and nicotine together do not experience the increase in impulsivity associated with caffeine intake in the absence of nicotine. These hypotheses are consistent with prior research findings that suggest caffeine has an arousing effect on behavior (Smith, et al., 2004), nicotine has a paradoxical calming effect on behavior despite producing autonomic arousal (Parrott, 1998), and nicotine intake blocks the arousal effects of caffeine (Rose, 1986; Rose & Behm, 1991), presumably including its effects on impulsivity. Thus, according to these hypotheses, individuals who wish to increase their impulsivity use caffeine in the absence of nicotine, but those who wish to increase their arousal but not experience an increase in impulsivity use nicotine in conjunction with caffeine.

Method

The research methods used in the present study were approved by the University of Maryland Institutional Review Board (IRB). Data for the present study was collected under two different IRB approvals: #01524, data collected between August 1, 2004 and July 31, 2005; #050428, data collected between October 7, 2005 and October 6, 2006. The IRB approval memos can be found in Appendix A.

Experimental Design Considerations

A power analysis (Cohen & Cohen, 2001) showed that for large effect sizes (f^2 = 0.40), it is necessary to have 15 subjects in each cell. With four cells, an N of 60 was needed to have adequate power ($1\text{-}\beta \geq .8$).

Male smokers experience heightened sensitivity to the pharmacological effects of nicotine compared with female smokers (Benowitz & Hatsukami, 1998; Daurignac, Perez-Diaz, Grillon, & Jouvent, 2001; Ikard & Tomkins, 1973; Perkins, 1996; Perkins, Jacobs, Crow, & Blackburn, 2002). To avoid confounds based on gender differences, only male participants were recruited. Further, the subject pool from which participants were selected was comprised of students in introductory psychology courses at the University of Maryland. Because most students in these courses are between the ages of 18 and 25, the age of 30 was chosen as an upper cutoff to avoid a skewed distribution with a few outliers. Because smoking is illegal for those 17 years of age or younger, the age of 18 was chosen as a lower cutoff.

A number of adverse health effects are associated with acute caffeine intake and cigarette smoking. These include dry mouth, increased heart rate or blood pressure, irregular heartbeat, and dizziness. To minimize the potential for unforeseen adverse

health effects, only participants who reported prior smoking of two cigarettes within a half-hour period, and prior caffeine intake of 300mg or more in a half-hour period, were allowed to participate in the study.

Because the half-life of caffeine has been found to average five hours (James, 2000), and because nicotine withdrawal has not been associated with significant decrements in laboratory study performance at periods under 24 hours of abstinence (Hatsukami, Fletcher, Morgan, Keenan, & Amble, 1989), participants were asked to abstain from products containing either caffeine or nicotine for a period of six hours prior to participation. This six-hour abstinence period is consistent with previous studies conducted in our laboratory measuring the effects of caffeine (e.g. Davidson & Smith, 1989, 1991; Smith, Davidson, & Green, 1993), as well as a number of similar studies in the literature (e.g. Craig, et al., 1979).

Caffeine generally reaches peak plasma levels between 30 and 60 minutes following administration (Benowitz, 1990; Mandel, 2002), and nicotine generally reaches peak levels immediately. As such, this study was designed such that participants completed the behavioral tasks during the period between 30 and 60 minutes following caffeine administration, such that they were likely experiencing peak caffeine levels during their completion of the behavioral measures. Likewise, nicotine levels peak within minutes of smoking, and as such, the study was designed such that participants began the behavioral measures immediately after smoking their second of two cigarettes. Thus, participants' caffeine and nicotine levels were likely peaking concurrently as they began the outcome measures. The combination of two cigarettes following caffeine consumption maintains an analogue to real-world smoking, which may involve smoking

two cigarettes in short succession, and generally subsequent to caffeine intake (Emurian, Nellis, Brady, & Ray, 1982). The double-dose of nicotine administered in this study is roughly equivalent to the high-dose conditions of nicotine administered intravenously in previous laboratory studies (3.0 mg/70 kg body weight is roughly equivalent to two 0.8 mg cigarettes smoked by a 150-lb. college male). Additionally, it should be noted that cigarette smoking has been found to produce stronger effects in the laboratory than in the natural environment, perhaps due to situational characteristics of the testing environment (Mucha, Mutz, Stephan, & Pauli, 1996); this was predicted to create a drug effect large enough to detect any differences in impulsive behavior resulting from the treatment.

Because the presence of other participants could have potentially affected arousal (and thus, theoretically, affected impulsivity), each participant was run individually, in the presence of one research assistant. To avoid any potentially arousing effects of opposite-sex research assistants on participants, all research assistants involved with running participants were male.

Participants

Participant demographics are summarized in Table 1. Sixty-three male college students were recruited from lower-level psychology courses at the University of Maryland. Participants were provided with extra credit in their psychology courses in exchange for their participation. Inclusion criteria were (1) male; (2) 18-30 years old; (3) have previously consumed 300 mg of caffeine with no adverse effects; (4) have previously smoked two cigarettes within a half-hour period with no adverse effects. These criteria were assessed via brief screening surveys assessing current level of caffeine use and smoking status (see “Caffeine Survey” and “Smoking Survey” in the

"Measures" section and Appendices B and C). Participants ranged in age from 18 to 29 (M = 19.86, SD = 2.45). Fifty-seven participants (90%) were full-time students enrolled in an introductory psychology course at the University of Maryland. The study sample was diverse with regard to race and ethnicity: 41 participants (65%) identified as White/Caucasian; 8 (13%) identified as Asian/Asian American; 5 (8%) identified as Black/African American; and 9 (14%) identified with other ethnic identifications, or identified as multiracial.

Table 1. Demographics and past-week caffeine use across drug conditions.

	Caffeine + Nicotine (n=16)	Caffeine (n=16)	Nicotine (n=16)	Placebo (n=15)
Age	19.69 (2.39)	19.44 (2.34)	20.75 (3.02)	19.53 (1.88)
Race	11 White (69%)	9 White (56%)	10 White (63%)	11 White (73%)
Class Standing	8 Freshmen (50%)	10 Freshmen (63%)	7 Freshmen (44%)	8 Freshmen (53%)
Past-Week Caffeine (mg)	814.1 (607.0)	937.3 (1079.7)	904.7 (963.8)	661.5 (399.8)
Smoking Status	10 Non-Smokers (63%)	13 Non-Smokers (81%)	9 Non-Smokers (56%)	8 Non-Smokers (53%)

Although inclusion criteria required that all participants had previously smoked two cigarettes in a half-hour period, only 19 participants (30%) reported regular daily smoking. These 19 daily smokers' average score on the FTND was 2.9 (SD = 1.45); due to not being daily smokers, all other participants were asked not to complete the FTND. Participants' mean caffeine intake for the 7 days prior to participation was 832.1 mg (SD = 802.5).

Self-Report Measures

Demographic Survey. The Demographic survey used (Appendix B) assessed the following self-report items: age, racial and/or ethnic identity, and year in school.

Caffeine Survey. Using a list of typical sources of caffeine from various foods and drinks (Smith, White, & Shapiro, in preparation), the Caffeine Survey (Appendix C) lists a variety of caffeinated products under the categories of "coffee/cocoa," "teas," "soft drinks," "caffeinated water," "over the counter drugs," and "chocolate candy." Participants were instructed to write the number of times they consumed each product in the last seven days. Participants' past-week caffeine consumption in mg was computed for each participant based on the typical levels of caffeine found in these sources (Smith, White, & Shapiro, in preparation).

Smoking Survey. Because history of cigarette use could potentially moderate the effect of nicotine on impulsivity, the Smoking Survey (Appendix D) contained a number of items related to smoking history: the age at which the participant first smoked a cigarette, the age of first smoking regularly, the current average number of cigarettes per day, and the average number of cigarettes smoked per day during the heaviest usage period.

Fagerstrom Test of Nicotine Dependence. The Fagerstrom Test of Nicotine Dependence (FTND; Heatherton, Kozlowski, Frecker, & Fagerstrom, 1991; Appendix E) is a modification of Fagerstrom's (1978) original Nicotine Tolerance Questionnaire (Fagerstrom, 1978). The FTND consists of six forced-choice questions regarding smoking preferences, and has been shown to be a reliable and valid instrument for assessing level of nicotine dependence and distinguishing heavier smokers from lighter

smokers: the coefficient alpha for the FNTD is 0.61 (Heatherton et al., 1991), and each item has been biochemically validated as a measure of nicotine dependence (Heatherton, et al., 1991). Reliability could not be accurately assessed in this study because there are few items (six), and only a small portion of the sample completed the FTND (n = 19). Further, range restriction was evident: the FTND yields scores ranging from 0 to 10; the mean FTND score for the 19 regular smokers in this study was 2.9 (SD = 1.45), and none received a score greater than 4.

Eysenck Impulsiveness-Venturesomeness-Empathy Questionnaire (I_7). The I_7 (S. B. G. Eysenck, et al. 1985) provides a self-report measure of impulsive, venturesome, and empathic personality traits. This scale could not be reproduced in the appendixes because it is not in the public domain. The scale consists of 54 items in three subscales: Impulsiveness (Imp; 19 items), Venturesomeness (Vent; 16 items), and Empathy (Emp; 19 items). The I_7 scales have shown to be reliable (Cronbach's α: Imp, .84 males, .83 females; Vent, .85 males, .84 females; Emp, .69 males, .69 females; S. B. G. Eysenck, et al., 1985). Emp was never considered theoretically related to Imp and Vent, and the Emp scale was only included in the I_7 to interrupt the monotony of the 35 Imp and Vent items (Caci, et al., 2003). Thus, for the purposes of the present study, only Imp and Vent were considered as theoretically relevant covariates of impulsive behavior. Reliability in the current study was good (Cronbach's α=.79) for Imp and acceptable (Cronbach's α=.67) for the Vent scale.

Barratt Impulsiveness Scale. The Barratt Impulsiveness Scale (BIS-11; Patton, Stanford, & Barratt, 1995; Appendix F) provides an additional self-report measure of impulsive personality. The BIS-11 consists of 30 items that are answered on a four-point

scale (Rarely/Never, Occasionally, Often, Almost Always/Always). Eleven items are reverse-scored to reduce response bias. The BIS-11 has been found to be reliable with both normal and impulsive populations (Cronbach's α for undergraduates: .82; substance-abusers: .79; general psychiatric patients: .83; inmates: .80; Patton, et al., 1995). Both the I_7 and the BIS-11 were administered because they have previously shown to measure slightly different domains within the realm of impulsivity. The administration of both measures served to tap a wider variety of impulsivity than either measure alone. Reliability in the current study was good (Cronbach's α =.71).

Positive and Negative Affect Schedule. The Positive and Negative Affect Schedule (PANAS; Watson, Clark, & Tellegen, 1988; Appendix G) is a brief self-report measure of positive and negative affect. The PANAS consists of two 10-item scales, one of which measures positive affect and one of which measures negative affect. Items from the two scales are intermixed to decrease response bias. Participants were instructed to fill in a number from 1 to 5 for each item; the numbers correlate with descriptions ranging from "very slightly/not at all" (1) to "extremely" (5). The positive and negative scales of the PANAS have been shown to be largely uncorrelated, have high internal consistency, and test-retest reliability (Watson, et al., 1988; also cf. Tellegen, Watson, & Clark, 1999a, 1999b). Reliability for the positive affect scale in this study was excellent (pre-drug: Cronbach's α = .83; post-drug: Cronbach's α = .91). Reliability for the negative affect scale was good for the pre-drug administration (Cronbach's α = .70) and acceptable for the post-drug administration (Cronbach's α = .59).

Behavioral Measures

Stroop Color-Word Test. Golden's revised version of the Stroop Color-Word Test (SCWT) contains three subtests, each of which consists of 100 words or stimuli printed in a somewhat random order, in 14-point font, on white paper (Stroop, 1935; Golden, 1976; Appendix H). The stimuli were arranged in five columns of 20 items each. In the first subtest, the words RED, GREEN, and BLUE were printed in black ink and displayed in a semi-random order. In the second subtest, the stimulus XXXX was printed in red ink, blue ink, or green ink and displayed 100 times in a semi-random order. In the third subtest, the words RED, GREEN, and BLUE were printed in incongruous colors of ink (RED in blue or green ink; BLUE in green or red ink; GREEN in red or blue ink), comprising 100 different stimuli, which were presented in a semi-random order. In the first subtest, participants were instructed to read the word printed; in the second and third subtests, they were instructed to state the color ink in which the stimuli were printed. For all three subtests, the total number of seconds to completion and the total number of errors made were recorded. Interference scores for time and accuracy were then calculated for each participant by subtracting his total time and total number of errors on the non-interference condition (the first subtest) from his total time and total number of errors in the interference condition (the third subtest). Like the SST, the SCWT has been found to have test-retest reliability for number of correct words and colors (squared partial correlations: words correct, .67; colors correct, .57; interference score: .47), and has been found to discriminate normal children and adolescents from those with impulse-control disorders (Kindlon, Mezzacappa, & Earls, 1995).

The Stop Signal Task. The Stop Signal Task (SST; Logan & Cowan, 1984; Logan, Cowan, & Davis, 1984; Logan, Schachar, & Tannock, 1997) measures individuals'

ability to inhibit motor responses. In this task, the letter "X" or "O" appears on the screen and the participants were instructed to press a corresponding key for each letter. On 25% of trials dispersed randomly throughout the task, participants hear a tone that signals inhibiting the response (i.e. not pressing either key). The delay between the stimulus letter (X or O) and the inhibitory tone is varied systematically until the participant inhibits responses on 50% of signal trials. After the stop signal delay has been adjusted, the signal response time (SRT) is calculated by subtracting the final mean delay from the final mean reaction time. The mean probability of inhibition on signal trials (corrected for omission errors on nonsignal trials) has been found to show moderate to high temporal stability, as well as discriminant validity in distinguishing normal children and adolescents from those with impulse-control disorders (Kindlon, et al., 1995). The SST runs in the disc operating system (DOS) and was administered on a Dell computer attached to a 38 cm flat-screen LCD monitor. In the current study, five participants' SST data had to be excluded from analysis because they apparently ignored or misunderstood the directions, and failed to inhibit any responses in the presence of the stop signal

Delay Discounting Task. The Delay Discounting Task (DDT; Bickel, Odum, & Madden, 1999; Madden, Bickel, & Jacobs, 1999; Madden, Petry, Badger, & Bickel, 1997) measures the relative value of delayed rewards to immediate rewards. The DDT involves participants being given a series of choices between hypothetical monetary rewards.

Administration of the DDT was completed with the participant seated at a table. On the participant's left were a stack of 27 index cards, above which lay a card that read "Right Now," indicating hypothetical rewards that could be received immediately. These

monetary amounts were $1000, $990, $960, $920, $850, $800, $750, $700, $650, $600, $550, $500, $450, $400, $350, $300, $250, $200, $150, $100, $80, $60, $40, $20, $10, $5, and $1. To the participant's right lay two cards: one was a duplicate $1000 card, and above it was a stack of seven cards with various lengths of time printed on them, representing hypothetical delays to receiving rewards. These delays were 1 week, 2 weeks, 1 month, 6 months, 1 year, 5 years, and 25 years. The following instructions were then read (adapted from Bickel, et al., 1999): "Now I am going to ask you to make some hypothetical money decisions. You will not actually receive the money that you choose, but just make your decisions as though you were really going to get the money you choose. Please just pick the option that you prefer, not the option that you think I might want you to prefer. The card on your left is always going to show some amount of money that you can have, in cash, right now. The card on the right is always going to show some money that you can have at some point in the future. So now you are being asked to choose between getting $1000, cash, today, versus $1000, cash, in one week. Point to the one that you would prefer." After the choice was made, the stack to which the participant pointed was flipped. Thus, if he selected "$1000 today," then the top card on the "money today" stack was flipped and the choice became "$960 today" versus "$1000 in one week." At this point, if he chose "$1000 in one week," then the "time to $1000 reward" stack was flipped, and the choice became "$960 today" versus "$1000 in two weeks." The flipping was continued until the participant chose the final card of one of the two stacks: either "$1 today" over $1000 at any point in the future, or "$1000 in 25 years" over any amount of money today.

Each participant's monetary "switch point," defined as the reduction in immediate reward offered that caused them to prefer the $1000 delayed reward. This dollar amount is considered the participant's rate of discounting (k) at that point in time, defined by the formula $V = A/(1+kD)$, in which V is the dollar value of the delayed reward ($1000), A is the dollar value of the immediate reward, and D is the delay (measured in weeks). Participants' k values were measured across groups using repeated-measures ANOVA.

Drug Preparation

Caffeine. To maintain the double-blind, placebo-controlled design, doses of 300 mg of powdered caffeine were measured individually via an electronic balance into sealed plastic bottles, and identical-looking doses of lactose placebo powder, of an equal volume to 300 mg of caffeine, were measured into identical plastic bottles and stored in a refrigerator. Caffeine or lactose placebo were administered to participants via a plastic cup filled with 240 mL of water and 10 mL Tang™ brand sweetened orange drink powder. After participants drank the entire cup of Tang™, research assistants verified that there was no residual powder in the bottom of the cup.

Nicotine. Further keeping with the double-blind, placebo-controlled study design, participants received either nicotine, via two Marlboro Lights™ cigarettes (0.8 mg nicotine per cigarette; Federal Trade Commission, 1999), or two denicotinized placebo cigarettes (QuestCigs, Inc.). Denicotinized cigarettes are regular-looking and -tasting cigarettes, with the exception that the tobacco in them has been treated to remove almost all nicotine. As such, they deliver similar amounts of smoke and tar compared with regular cigarettes, yet they only deliver a very small amount of nicotine (0.05 mg as measured by the FTC method; QuestCigs, Inc.), and do not affect plasma nicotine levels

(Rose, et al., 1999). To ensure the maintenance of the double blind, the brand names of the Marlboro and Quest cigarettes were obscured by affixing an opaque white paper sticker around the base of the tobacco column near the filter (i.e., the area where the brand names were printed), causing the cigarettes to appear identical.

Procedure

This study used a double-blind, placebo controlled design. Prior to starting, participants were assigned to one of four experimental conditions via block randomization: nicotine and caffeine (NC); nicotine and placebo drink (N); caffeine and placebo cigarettes (C); or placebo drink and placebo cigarettes (P). To ensure that participants would abstain from caffeine and nicotine for six hours prior to their participation, each participant was e-mailed by a research assistant after an appointment time was scheduled, and participants were asked to abstain from caffeine, nicotine, alcohol and other drugs for six hours prior to their appointment time. They were told that they would provide a saliva sample at the visit that could be measured for caffeine and nicotine content. If a participant indicated at any point during this procedure that he had consumed nicotine or caffeine within the previous six hours, he was to be rescheduled. No participants reported recent use (past six hours) of caffeine or nicotine.

Procedures for this study are summarized in Table 2. Participation in this study consisted of one session, lasting approximately 75 minutes, scheduled between the hours of 9:00 AM and 4:00 PM. Upon arriving at the laboratory, participants were given the opportunity to provide informed consent (Appendix I). Following informed consent, they were asked to give the time of their last use of nicotine and their last use of caffeine. To ensure that participants believed that the level of nicotine was being verified, we asked

them to provide a saliva sample by sucking on a cotton swab for five seconds and placing it in a numbered plastic bag, which was placed in a refrigerator. Participants were then given a packet containing the self-report measures, which
were counterbalanced to control for order effects. These measures took approximately 15 minutes to complete. Upon completion of these measures, participants were administered the cup of Tang™ containing either caffeine or lactose placebo powder. Consumption of the Tang was followed by the research assistant starting a stopwatch, handing the participant the first of his two cigarettes, and leading him to a designated smoking area 4 m outside the side door of the building. Participants smoked the cigarettes at a typical pace, unprompted by the research assistant. Because the pace of smoking varies between individuals, research assistants remained outside with each participant for six minutes, until the stopwatch showed time +8 minutes. At this point, participants were brought back inside the laboratory (arriving at time +10 min) for a ten-minute waiting period, during which they were instructed to sit in a comfortable armchair, and had the opportunity to read a magazine or newspaper. Following this waiting period (time +20 min), participants were handed their second cigarette and were brought outside for a second six-minute period (time +22 min to time +28 min), at which time they smoked the second cigarette.

Upon returning inside the laboratory following the second cigarette (time +30 min), participants were administered the PANAS for a second time. Following the PANAS, the behavioral measures were administered in a counterbalanced order. Completion of the behavioral measures took approximately 20 minutes. Following completion of the behavioral measures, participants were administered a manipulation

check (Appendix J), which asked them to state if they believe they received nicotine or placebo, and to report their confidence in this belief (0 to 100%). Participants were asked the same question about caffeine. Finally, participants were given a receipt to confirm the award of extra credit, and thanked for their participation. They were asked if they felt any adverse effects of drug intake, and told to call the lab if they experienced any effects after leaving. No participants reported any adverse effects or called. Participants were told that they would be debriefed as to their drug condition following the completion of data collection.

Table 2. Procedure Chart.

Relative Time (min)	Procedures
-15 to +0	Informed consent; baseline measures
+0	Administration of caffeine/placebo
+0 to +2	Walk outside
+2 to +8	Smoke first cigarette
+8 to +10	Walk inside
+10 to +20	Wait inside; read magazines/newspapers
+20 to +22	Walk outside
+22 to +28	Smoke second cigarette
+28 to +30	Walk inside
+30 to +32	Complete second PANAS
+32 to +58	Administration of three behavioral measures
+58 to +60	Manipulation check; debriefing

Data Analysis

Prior to the data analysis, data screening was performed. This included descriptive statistics to check for data-entry errors, imputation of missing data, and determination of skew and kurtosis. The baseline measures of impulsivity, the mood measure (PANAS), and the outcome measures (SCWT, SST, DDT) were all normally distributed. Skew and kurtosis were acceptable.

Preliminary Analyses. To check for potential confounds across the four conditions (NC, C, N, and P), a series of univariate ANOVAs for the continuous variables, and χ^2 tests for the dichotomous variables, were conducted for all demographic, variables, as well as mood (PANAS), and baseline impulsivity (I_7 Imp and Vent; BIS-11) variables. *Post hoc* comparisons were conducted using Scheffe's correction. Results indicated that none of the baseline variables significantly differed across conditions. Next, the reliability of the baseline measures of impulsivity was measured using Cronbach's α and found to be good (Imp: .79; Vent: .67; BIS-11: .71). The baseline measures of impulsivity were then correlated using Pearson's product correlation. Imp was significantly correlated at the $p<.05$ level with Vent ($r^2 = .30$) and BIS-11 ($r^2 = .66$). The correlation of Vent and BIS-11 ($r^2 = .12$) was not statistically significant at the $p<.05$ level. Finally, the double-blind was tested using χ^2 analysis. The belief that participants received nicotine was significantly greater among those who received nicotine (i.e., the N and NC groups) than those who did not (i.e., the C and P groups): χ^2 (1, $N = 63$) = 16.171, $p = .000$. However, no significant difference was found for the participants' receiving caffeine between those who received caffeine (i.e., the C and NC groups) compared with those who did not (i.e., the N and P groups): χ^2 (1, $N = 63$) = .021, $p = .884$.

Hypothesis Testing. Univariate ANOVA was conducted to test for differences across conditions on the SST and SCWT. A repeated-measures ANOVA was conducted to test for differences across the seven time levels of the DDT. *Post hoc* comparisons were conducted using Scheffe's correction. Because four ANOVAs were planned, the significance level for each of the four tests was set at $p<.0125$ to account for familywise error and thus make the significance level $p<0.05$ across the outcome measures.

Results

Manipulation Check

χ^2 analysis of the manipulation check found that the double-blind administration of nicotine was compromised. Participants in the N and N+C groups were significantly more likely to guess that they received nicotine than were those in the C and P groups: χ^2 (1, N = 63) = 16.171, p = .000. However, χ^2 analysis revealed no significant difference between those in the C and N+C groups' belief that they received caffeine compared to those in the N and P groups: χ^2 (1, N = 63) = .021, p = .884. Thus, the double-blind procedure was maintained for participants with regard to caffeine but not with regard to nicotine.

The Relationship Among Demographic Measures Across Groups

Table 3. Statistical Testing of Baseline Measures.

Baseline Measure	Statistics
Age	F (3, 59) = .978, p = .409, η^2 = .047
Age of Smoking First Cigarette	F (3, 59) = .560, p = .644, η^2 = .028
Age of Smoking Regularly	F (3, 42) = 1.604, p = .203, η^2 = .103
Baseline Average Smoking Rate	F (3, 59) = 1.163, p = .332, η^2 = .056
Past-Week Caffeine Use (mg)	F (3, 59) = .353, p = .787, η^2 = .018
Race/Ethnicity	χ^2 (9, N = 63) = 2.160, p = .989
Class Standing	χ^2 (15, N = 63) = 10.559, p = .783

Analysis of the baseline demographic variables using univariate ANOVA for the continuous variables (age, age of first cigarette, age of first regular smoking, FTND scores, baseline smoking rate, baseline caffeine use, baseline positive and negative affect)

and χ^2 analysis for the nominal and ordinal variables (race/ethnicity, class standing) found that none of these variables significantly differed across conditions at the $p<.05$ level. As such, these variables could not be considered confounding variables regarding drug effects on impulsivity. The statistics for the baseline measures are found in Table 3.

Table 4. Mood and baseline impulsivity across drug conditions.

	Caffeine + Nicotine (*n*=16)	Caffeine (*n*=16)	Nicotine (*n*=16)	Placebo (*n*=15)
Mood				
Pre-Tx. PA	27.6 (5.8)	28.3 (8.0)	30.1 (5.6)	27.4 (6.19)
Pre-Tx. NA	12.5 (2.3)	13.6 (3.7)	11.8 (1.8)	13.8 (4.0)
Post-Tx. PA	29.3 (8.1)	29.0 (9.9)	27.8 (6.0)	28.1 (6.4)
Post-Tx. NA	12.8 (2.5)	12.0 (1.8)	12.4 (3.4)	12.4 (2.9)
Impulsivity				
Imp	8.1 (3.9)	7.9 (4.8)	7.9 (3.9)	8.4 (4.2)
Vent	10.8 (2.9)	12.1 (2.6)	12.3 (2.7)	11.4 (2.6)
BIS-11	70.7 (8.1)	72.3 (8.8)	74.1 (9.1)	72.7 (7.2)

Key:
Pre-Tx. PA: Positive Affect (PANAS) prior to drug intake
Pre-Tx. NA: Negative Affect (PANAS) prior to drug intake
Post-Tx. PA: Positive Affect (PANAS) following drug intake
Post-Tx. NA: Negative Affect (PANAS) following drug intake
I_7 Imp: Raw Score on Impulsivity Subscale of Eysenck's I_7 Questionnaire
I_7 Vent: Raw Score on Venturesomeness Subscale of Eysenck's I_7 Questionnaire
BIS-11: Total Raw Score on the Barratt Impulsivity Scale (11th Edition)

Mood

Table 4 summarizes the results of pre- and post-treatment mood as measured by the PANAS. Participants' mean positive affect at baseline was 28.35 (*SD* = 6.41), and their mean negative affect at baseline was 12.89 (*SD* = 3.09). Univariate ANOVA revealed that post-treatment PANAS positive affect (*M* = 28.52, *SD* = 7.61) and negative

affect (M = 12.41, SD = 2.63) did not significantly differ across conditions at the p<.05 level. Thus, pre-treatment mood could not be considered a confounding factor on the results.

Baseline Measures of Impulsivity

Table 4 summarizes the baseline impulsivity scores from the I_7 and BIS-11. I_7 Imp (M = 8.06, SD = 4.11), Vent (M = 11.63, SD =2.68), and BIS-11 (M = 72.45, SD = 8.32) scores did not significantly differ across treatment conditions.

Table 5. Correlations Among Baseline Impulsivity Measures and Outcome Measures.

	PA pre	NA pre	BIS-11	I_7 Imp	I_7 Vent	CW Time	CW Errors	SST Avg Delay	DDT Avg *k*
PA pre	1.000	.325*	.243	.132	.195	.077	.002	.053	.083
NA pre	.325*	1.000	.161	.238	-.089	.175	.046	.074	.026
BIS-11	.243	.161	1.000	. 658*	.117	.101	.263*	-.030	-.057
I_7 Imp	.132	.238	.658*	1.000	.296*	-.042	.186	-.112	.066
I_7 Vent	.195	-.089	.117	.296*	1.000	-.046	.079	-.086	.126
CW Time	.077	.175	.101	-.042	-.046	1.000	.308*	.176	.212
CW Errors	.002	.046	.263*	.186	.079	.308*	1.000	-.116	.172
SST Avg Delay	.053	.074	-.030	-.112	-.086	.176	-.116	1.000	.101
DDT Avg *k*	.083	.026	-.057	.066	.126	.212	.172	.101	1.000

Key:
*Statistically significant correlation (p<.05).
PA pre: Raw Positive Affect Score on the PANAS at baseline
NA pre: Raw Positive Affect Score on the PANAS at baseline
BIS-11: Total Raw Score on the Barratt Impulsivity Scale (11th Edition)
I_7 Imp: Raw Score on the Impulsivity Subscale of Eysenck's I_7 Questionnaire
I_7 Vent: Raw Score on the Venturesomeness Subscale of Eysenck's I_7 Questionnaire
CW Time: Total time (sec) on the color-word administration of the Stroop Color-Word Task
CW Errors: Total errors on the color-word administration of the Stroop Color-Word Task
SST Avg Delay: The average delay (ms) across all four measured blocks of the Stop Signal Task
DDT Avg *k*: The average *k* value across all seven time levels of the Delay Discounting Task

Correlations Among Variables of Interest

Table 5 summarizes the statistical relationships among the baseline measures of mood and impulsivity, and the outcome measures, across all four drug conditions. Among the baseline measures of mood, positive and negative affect at baseline were positively correlated with each other (r = .325, n = 63, p = .009). Among the baseline measures of impulsivity, I_7 Imp was positively correlated with BIS-11 total scores (r = .658, n = 63, p = .000) and with I_7 Vent (r = .296, n = 63, p = .018). SCWT color-word time and color-word errors were positively correlated with each other (r = .308, n = 62, p = .015), and BIS-11 total score was correlated with color-word errors (r = .263, n = 62, p = .020). None of the other baseline impulsivity measures were correlated with any of the other outcome measures, nor were any of the other outcome measures correlated with each other across conditions.

Behavioral Measures

Stroop Color-Word Task. A univariate ANOVA was conducted to examine differences in SCWT color-word time and Color-Word errors as a function of treatment condition. No significant effects of overall treatment condition were found for color-word time [$F(3, 59) = 1.6$, $p = .18$, $\eta^2 = .08$] or color-word errors [$F(3, 58) = 0.67$, $p = .58$, $\eta^2 = .03$].

Stop Signal Task. A univariate ANOVA was conducted to examine differences in stop reaction time as a function of treatment condition. No significant effect of average stop signal delay was found [$F(3, 54) = 1.5$, $p = .22$, $\eta^2 = .08$].

Delay Discounting Task. A repeated measures ANOVA was conducted to examine rates of discounting (*k*) as a function of treatment condition. No significant differences in *k* were found [$F(3, 59) = 1.45$, $p = .24$, $\eta^2 = .07$].

Unplanned Analyses

Because the planned analyses yielded no significant results, a series of unplanned analyses were conducted to explore potential trends in the data meriting future study. First, different approaches to measuring the outcome data were attempted. For the SCWT, total time across the three trials was computed, as was total errors across the three trials. Following this, a time difference score was computed (Color-Word Time – Word Time), as was an error difference score (Color-Word Errors – Word Errors). Univariate ANOVA found no significant results using any of these four variables (total time; total errors; change in time; change in errors) as dependent variables. For the SST, the delay time for each of the four experimental blocks was analyzed independently; univariate ANOVA found no significant results using any of the four individual delays as a dependent variable. Finally, for the DDT, repeated-measures ANOVA was conducted using the raw monetary amounts (instead of the *k* slopes) as the dependent variable, but no significant differences were found across drug conditions.

Following these unplanned analyses, an empirical power analysis was conducted to determine what sample size would be needed to find significant differences between groups in future research. Using the assumption that the observed power in the current study sample ($\eta^2 = .08$ for the SST and SCWT, and $\eta^2 = .07$ for the DDT) accurately reflects the percentage of variance in the outcome measures accounted for by the experimental manipulation, an empirical (i.e. *post hoc*) power analysis was conducted to

determine the appropriate sample size for future studies. Acting as if a regression would be conducted, with a significance level of $p < .05$, and three predictor variables (N, C, N+C), the sample sizes needed to have adequate power with a correlation coefficient of $r^2 = .08$ were determined. For power $(1 - \beta) = .80$, a sample size of 130 would be needed. For power $(1 - \beta) = .90$, a sample size of 165 would be needed.

Discussion

The three study hypotheses – that (1) nicotine intake decreases impulsive behavior compared with placebo, that (2) caffeine intake increases impulsive behavior compared with placebo, and that (3) the combined intake of nicotine and caffeine decreases impulsive behavior compared with caffeine – were not supported by the results of the present study.

No significant increases or decreases in impulsivity on any of the three behavioral measures of impulsivity used in this study, following caffeine intake compared with placebo, were found. Although no prior studies assessing the effects of caffeine on the DDT and the SST have been located in the extant literature, numerous studies have found effects of caffeine on SCWT performance, and so the null results of the present study pertaining to the SCWT are particularly inconsistent with the literature. The SCWT has been used in similar studies, and caffeine has been shown to produce effects on SCWT performance. This has been true even when caffeine doses and experimental procedures have been similar to those in the present study (Foreman, et al., 1989). In Foreman and colleagues' (1989) study, participants in the high-caffeine condition (10 male undergraduates) received 250 mg caffeine, a dose only slightly lower than that used in the current study. Compared to 11 male undergraduates who received 125mg caffeine, and 11 male undergraduates who received placebo, these participants exhibited impairments in their performance on a numerical version of the Stroop Task (Foreman, Barraclough, Moore, Mehta, & Madon, 1989). These participants completed the modified Stroop task and two other behavioral measures within 30 and 60 minutes after receiving caffeine or placebo, the same time period used in the current study. Thus, the failure of the present

study to find significant differences related to caffeine intake, using the traditional SCWT and sampling a larger number of participants from a similar population, does not suggest that the SCWT is a poor measure of impulsivity sensitive to caffeine intake. Additionally, the other behavioral measures of impulsivity (the DDT and the SST) showed no differences between those receiving caffeine and placebo, creating an overall pattern of null findings from caffeine use on impulsivity in the present study. Whether or not caffeine affects behavioral impulsivity, it did not measurably increase or decrease impulsivity on any of the measures used in this study on this study sample. The lack of findings for caffeine on impulsivity in the present study, compared with similar studies' positive findings for caffeine on impulsivity, suggests that extraneous sources of variance may have reduced statistical significance in the present study. Potential extraneous sources of variance will be discussed below.

No significant increases or decreases in impulsivity on any of the three measures used in this study were found following nicotine intake as compared with placebo. These three measures (the DDT, the SCWT, and the SST) have not been used in the extant literature to examine the results of acute nicotine intake on impulsivity, so it is not clear whether they are sensitive to measuring the effects of nicotine intake. The DDT used in the present study was specifically chosen because it is more sensitive than similar delay-discounting procedures in distinguishing differences in delay discounting between smokers and non-smokers (Bickel, et al., 1999; Epstein, et al., 2003; Mitchell, 1999). However, these three studies in the extant literature used the DDT to measure differences between current, former, and ex-smokers; the present study attempted to measure differences in the acute effects of nicotine. It is possible that the DDT is simply not

sensitive to the acute effects of nicotine. Likewise, the SCWT and the SST have also not been used to study the acute effects of nicotine, and it is possible that they, too, are not sensitive to the effects, if they exist, of nicotine on impulsivity.

Interpreting the null results of the C+N condition for all three behavioral measures of impulsivity is more challenging. On its face, the hypothesis that the combined intake of nicotine and caffeine decreases impulsive behavior compared with the intake of caffeine alone, was not supported by the results. However, the spirit of this hypothesis was that nicotine and caffeine have antagonistic effects on each other with regard to impulsivity: that caffeine alone increases impulsivity, nicotine alone decreases impulsivity, and together, their effects are either closer to the effects of nicotine (i.e., a decrease in impulsivity from placebo), or closer to the effects of placebo (i.e., akin to abstaining from both drugs, with respect to impulsivity). Because the entire study yielded null results among all four drug conditions (i.e., neither caffeine, nor nicotine, nor the conjunction of the two effected any change on impulsivity), it is difficult to confirm or deny the notion that the drugs have an antagonistic relationship on impulsivity. In the absence of effects for either caffeine or nicotine on impulsivity, it is premature to make generalizable assumptions about this specific hypothesis. What is perhaps more relevant is to examine the null findings across all drug conditions.

The hypotheses for the present study were developed based on the extrapolation of two general effects found in the literature: that (1) personality measures of impulsivity are positively correlated with caffeine and nicotine use, which themselves are correlated, and (2) that caffeine and nicotine are drugs that increase arousal. These effects were tied together with arousal theory, which states that impulsivity is a personality trait that is

related to arousal-seeking. Thus, it was hypothesized that because higher- and lower-impulsive individuals seek out caffeine and nicotine use in different patterns to modulate their arousal, and because caffeine and nicotine intake affect arousal, that caffeine and nicotine intake would also affect impulsive behavior. The null findings of the present study, then, suggest that caffeine and nicotine do not directly affect impulsive behavior. These results suggest that although individuals higher on the personality trait of impulsivity are more likely to use caffeine to increase arousal, and that some individuals use nicotine to block the effects of caffeine on arousal, that these changes in arousal do not correspond with changes in impulsive behavior. Perhaps becoming more aroused does not lead a person to behave more impulsively, even though those with impulsive personalities are more likely to seek out these changes in arousal. Although arousal theories (e.g. Smith, 1994; Wilken, Smith, Tola, & Mann, 1999) suggest that a number of factors affect arousal in complex patterns, it may be that the acute independent and joint effects of caffeine and nicotine on arousal (e.g. Rose, 1986; Rose & Behm, 1991; Rose, et al., 1993) do not affect impulsivity *per se*.

Personality measures of impulsivity and behavioral measures of impulsivity reflect differing ways of operationalizing the construct. Both types of measures are based on verbal definitions of impulsivity, but the methodology (i.e. items on a survey versus verbal performance on laboratory tasks, or motor performance on a computerized task) used to measure the construct varies. Method variance is a known source of extraneous variance in psychological research (Campbell & Fiske, 1959). Method variance has likely led to low or nonsignificant correlations in the literature between personality and behavioral measures of impulsivity, as is discussed at length in the literature review

(following the references section). In fact, no single personality or behavioral measure has been universally agreed upon to measure impulsivity (McDonald, et al., 2003), and perhaps the two or more very different ways of operationally defining the construct are only minimally associated with each other, and reflect problems in operational definitions of a phenomenon across different forms of measurement. The present study utilized three different behavioral measures of impulsivity, tapping different facets of the construct of behavioral impulsivity, and still no significant findings were observed. A correlation matrix of the measures of interest in the present study, run *post hoc* on the data collapsed across conditions, showed that the only significant correlation among the behavioral measures was between SCWT color-word time and SCWT color-word errors. No correlations were found between the three behavioral measures. Further, the only correlation between a baseline variable and an outcome measure was that observed between the BIS-11 and SCWT color-word errors.

It is possible that the behavioral measures used in the present study are not sensitive to the effects of nicotine. It is also possible that the DDT and the SST are not sensitive to the effects of caffeine, although it is difficult to say that the SCWT is not sensitive to the effects of caffeine, given the extant literature. It is possible that method variance decreased the ability to find statistical significance in the present study. It is also possible that the study hypotheses were simply wrong, and the extrapolation that led to the hypotheses should not be made. Perhaps the associations between those individuals who are cortically and sympathetically under-aroused and the construct of extraversion, and the definitional association of extraversion and impulsivity on personality measures, and the correlation of impulsivity on personality measures and caffeine/nicotine use, and

the effects of caffeine on increasing arousal, simply do not add up to an acute effect of caffeine on impulsive behavior. And perhaps the differences on an impulsive behavioral task (the DDT) between smokers and non-smokers, and the differences on impulsive personality measures between smokers and non-smokers, do not translate to acute effects of nicotine on impulsivity. And perhaps the interactive effects of caffeine and nicotine on arousal (i.e., that nicotine blocks the increase in arousal that results from caffeine use) do not translate to effects on behavioral measures of impulsivity.

Limitations

The limitations of the present study fall into several categories: (1) aspects of the study design related to caffeine and nicotine dosing and their relation to the study sample, (2) the failure of the double-blind with respect to nicotine; (3) the lack of control over time-of-day, an arguably arousal-affecting factor, (4) the potential ceiling effect, as the sample had heightened BIS-11 impulsivity compared to the general population, and (5) the poor correlation between personality and behavioral definitions and measurement of impulsivity. These limitations will be addressed in this order.

Although this study controlled for the confounding effects of differential baseline caffeine and nicotine use across groups, and participants' reported smoking histories and baseline rates of smoking and caffeine use did not significantly differ across conditions, their baseline caffeine and nicotine consumption varied widely, and this may have introduced error variance. It is possible that the correlations between impulsivity and nicotine and caffeine use may be related to chronic, and not acute, use of these drugs (for example, in Bickel and colleagues' [1999] differentiation of smokers and non-smokers using the DDT). The inclusion criteria in the present study were loosely defined, allowing

any young men who smoked or consumed caffeine, even sporadically, to participate. As a result, participants reflected a broad spectrum of casual to moderate users of these drugs. Although the issue of varying intake was explored in the unplanned analyses on the data, and no differences were found across those with varying baseline levels of either or both drugs, the study was not designed to adequately examine such differences. It is possible that men who generally smoke and drink caffeine at different rates experience different effects from the drugs. Perhaps long-term caffeine or nicotine use, across time, leads individuals to experience different effects of these drugs from acute administration. If this is indeed the state of nature, then sampling users of these drugs who typically use varying amounts, and have used the drug for varying amounts of time, may have increased error variance in the present study and contributed to the nonsignificant results.

Another potential source of error variance in the present study was that the participants were requested to abstain from caffeine and nicotine intake for six hours prior to participation, and it is possible that the some participants did not abstain from smoking or drinking caffeine for six hours prior to their participation. If this factor confounded the results, it could not be measured. Nonetheless, although participants' actual adherence could not be assessed, the deception involving the collection of saliva samples, and the belief of participants that these levels would be measured, was likely sufficient to motivate participants to abstain. Participants in this study reported low baseline rates of smoking, and with none of the participants scoring higher than 4 on the ten-point-scale FTND, it is unlikely that participants in this study were dependent enough to be unable to abstain from smoking for six hours prior to their participation. Six hours is also not long enough for caffeine withdrawal syndrome to occur, so it is unlikely that

participants were unable to abstain from drinking caffeine, and equally improbable that participants who did abstain were experiencing caffeine withdrawal. However, it cannot be said with certainty that all participants abstained from smoking or caffeine use for six hours prior to their participation. It is also not possible to state that unequal violations of the abstinence requirement occurred across drug conditions.

Yet another factor that may have confounded the study results was the failure of the double-blind with regard to nicotine. Although participants were not able to accurately determine whether or not they had received caffeine, they were able to state whether or not they received nicotine. Nicotine is a drug that, compared with placebo, is easily detected by participants when they are asked to guess (Mooney, White, & Hatsukami, 2003). Thus, because those participants in the present study who received nicotine were aware of their receiving nicotine, this may have confounded the results. However, the unblinding of nicotine-versus-placebo is generally associated with an increase, not a negation, of the effects of nicotine when compared with placebo (Mooney, et al., 2003). Thus, if anything, the unblinding of participants to their nicotine status should have created an effect, not negated one. Further, the double-blind with regard to caffeine was maintained, and those receiving caffeine also did not behave significantly different than those receiving placebo. Null results were found for both drugs on the behavioral measures for this study, regardless of the blind. Finally, because the double-blind administration of nicotine fails regularly, this is a problem across studies, including those that have found differential effects of nicotine and placebo on behavior.

Another potential limitation of the current study was the lack of control over the time of day. Participants in the present study completed the behavioral measures at

various times of the day (between 9:30 AM and 4:00 PM), and therefore, the time of day (i.e. morning versus afternoon) was not consistent for all participants. The lack of control over the time of day in the present study may have contributed to the lack of significant findings. One possible reason that no significant results were found in the present study is that an uncontrolled factor contributed to error variance and obscured any effects that would otherwise have been found. A controversial factor in the literature, argued by some to affect arousal and performance on arousal-related tasks, is the time of day. If performance on these measures is significantly impacted by the time of day, then the lack of control of this factor may have contributed to the null results. Executive control of actions, one important facet of impulsivity assessed with the SST and SCWT, has been shown to be associated with circadian rhythms in humans (Manly, Lewis, Robertson, Watson, & Datta, 2002). The ability to withhold responses until a thoughtful response has been shown in some studies to be greater in the afternoon than in the morning. For example, Manly and colleagues (2002) assessed such executive functioning at various times of day (1 AM, 7 AM, 1 PM, and 7 PM) and found significantly more impulsive responding in participants at 1 AM and 7 AM than at 1 PM and 7 PM. However, other studies using the SST, DDT, and SCWT (e.g. Epstein, et al., 2003; Kindlon, et al., 1995; Ortner, MacDonald, & Olmstead, 2003; van Mourik, Oosterlan, & Sergeant, 2005) have yielded significant differences across various conditions without controlling for this factor. Thus, the time of day does not appear to affect performance on these measures to the extent of obscuring the effects of other experimental manipulations.

Alternately, it is possible that this same factor (the time of day) interacts with caffeine or nicotine intake such that it confounded the results. The literature on the effects

of caffeine intake on behavior at various times of the day is inconsistent: some studies have shown caffeine x time-of-day interactions (e.g. Revelle, et al., 1980), whereas other studies have shown that these effects are inconsistent and that caffeine x time-of-day effects are weak and require significant manipulations to show any effects (Miller, Lombardo, & Fowler, 1995). Revelle and colleagues (1980) interpreted their caffeine x time-of-day findings as requiring a re-evaluation of arousal theory, suggesting that low impulsives are more aroused in the morning and less aroused at night, and vice-versa for high impulsives. However, Eysenck and Folkard (1980) convincingly argued that this interpretation was suspect due to its being based on poor measures of impulsivity, and extreme false-dichotomization of participants. The literature on the potential interaction of caffeine and time-of day is thus inconclusive. The literature on the potential interaction of nicotine and time-of-day is nonexistent.

In yet another limitation, the study sample was higher on the measure of impulsive personality than the general population. With respect to the personality measures of impulsivity, only baseline BIS-11 impulsivity predicted SCWT impulsivity, and none of the baseline impulsivity measures correlated with impulsivity on the SST or the DDT. BIS-11 total scores in the present study (M = 72.44, SD = 8.22) also did not differ across treatment conditions, but were noticeably higher than those reported by studies on non-impulsive populations: Stanford, Greve, and Dickens (1995) reported lower mean BIS-11 scores for a non-aggressive college-student sample (M = 59.96, SD = 8.93) and for an aggressive college-student sample (M = 63.90, SD = 9.21). Bayle and colleagues (Bayle, et al., 2003) reported lower BIS-11 scores for adult psychiatric inpatients without impulse-control disorders (M = 57.3, SD = 15.7), whereas adult

psychiatric inpatients with kleptomania of (M = 72.1, SD = 18.3) had BIS-11 scores much closer to the means for participants in all four drug conditions of the present study. Thus, it is likely that the study sample experiences heightened impulsivity compared with the general population. Whether this heightened impulsivity is typical of caffeine- and nicotine-consuming male college students, or is specific to the study sample, merits future research. If the sample chosen for the present study experiences greater impulsivity than the population of caffeine- and nicotine-consuming male college students at large, this may have introduced a bias that canceled out any typical behavioral effects of drug intake on impulsivity. However, if the general population of caffeine- and nicotine-using college men experiences heightened impulsivity, these findings are more generalizable to the real world. In either case, whether or not the sample was typical of the population, it is possible that a ceiling effect occurred, such that participants' higher baseline impulsivity, across all conditions, eliminated the typical effects of caffeine and nicotine on impulsivity.

The conceptual problems associated with the measurement of impulsivity are immense and cannot be clarified with a single study. As noted in the literature review of this thesis, impulsivity as a broad concept relates to a plethora of constructs, many of which relate so tangentially to each other that they can hardly be said to be similar in anything but name. One major limitation to the present study was the attempt to relate personality measures of impulsivity from baseline to the effects found from the behavioral measures of impulsivity administered following drug intake. Impulsivity as a personality construct and impulsivity as a cross-sectionally measured description of behavior are conceptually related, and have shown some correlations in factor analysis

(e.g. Gerbing, Ahadi, & Patton, 1987). However, method variance between paper-and-pencil measures of personality, (and, in this case, between computer- and human-administered measures of behavior), is great (Campbell and Fiske, 1959). Nevertheless, the findings across methods in this study were strikingly consistent across measures of impulsivity: for all three measures, each drug caused more impulsive behavior than no drug, and the caffeine-nicotine combination caused the most impulsive behavior. Thus, any method variance that may have occurred did not change the general effect of caffeine and nicotine on impulsivity.

Future Directions

The limitations of the present study related primarily to inadequate control of several key factors related to the study constructs: assessment of abstinence prior to participation, time of day, baseline nicotine and caffeine intake, and psychopathology. Future research investigating the acute effects of caffeine and nicotine ought to better control for all of these variables. Inclusion and exclusion criteria should focus more specifically on one population with regard to baseline drug use. Perhaps these factors could be restricted to modal use patterns in the population, such as those who smoke between 10 and 30 cigarettes per day, and consume between 50 and 300 mg of caffeine per day. Participants should be better screened for baseline impulsivity. Although the present study used baseline measures of impulsivity as covariates for the outcome measures, the I_7 and BIS-11 could be used as pre-screening measures to select a specific population (e.g. low impulsives, high impulsives, or those with scores in the mid-range, most similar to the general population). Those with impulse-control disorders (e.g. ADHD, Bipolar Disorder, BPD) could also be screened out and excluded from

participation. Finally, actual chemical measurement of plasma caffeine and nicotine levels prior to drug intake could be measured, and the time of day at which participants complete the study procedures could be standardized.

Additionally, the goal of untangling the messy construct of impulsivity and its relationship to caffeine, nicotine, and arousal could be measured in other methods. The longitudinal time-sequence of scores on personality measures of impulsivity, behavioral measures of personality, and natural use of caffeine and nicotine could be measured in adolescents. The progression of these constructs' covariance, across time, using a developmental perspective, would greatly increase our knowledge of if and how they affect each other. In addition, the various effects of caffeine, nicotine, and their interaction, on a wide plethora of impulsivity measures, should be studied. Impulsivity measures are theoretically designed to measure the construct of impulsivity, and the experimentation of which measures show effects from caffeine and nicotine use is somewhat suspect from a theoretical perspective. This is to say, "fishing" for a measure that finds results favorable to a hypothesis may be somewhat scientifically dishonest in the sense of testing a theory. However, determining which measures of impulsivity show significant findings following drug intake may give us a better idea of the specific types of effects, if any, these drugs produce. Knowing that caffeine and nicotine intake affects specific speed-versus-accuracy measures may help us understand which facets, if any, of impulsivity are related to stimulation by chemical intake.

In conclusion, it is possible that the intercorrelations among impulsive personality, impulsive behavior, arousal, the seeking of arousal, the acute effects of caffeine and nicotine, and the chronic effects of caffeine and nicotine, do not imply that

all of these factors are correlated with all of the other factors. A complex picture emerges from prior literature and from the results of the present study. The questions suggested by this complex picture are many: should *impulsivity* refer to impulsive personality only? Should behavioral impulsivity be given a different name? Is behavioral impulsivity associated with increases in arousal at all? Does the chronic use of caffeine and nicotine affect impulsive behavior in a way that acute use of the drugs do not? These questions will require a good deal of future exploration in a variety of areas, to attempt to explain such divergent patterns among a convergent cluster of factors.

Appendix A: Literature Review

Arousal Theory and Impulsivity

Arousal refers to the overall level of excitation in the cerebral cortex and activation of the autonomic nervous system (Gray, 1964) and is the primary mechanism involved in impulsivity. Arousal can range from sleep to extreme behavioral activation (Humphreys & Revelle, 1984). Arousal theory postulates that impulsive individuals are chronically underaroused, and seek external excitement to raise their level of arousal (H. J. Eysenck & M. W. Eysenck, 1985; Humphreys & Revelle, 1984; Revelle, et al., 1980). In Gray's (1975, 1987) model of arousal, two systems regulate arousal: the *behavioral activation system* (BAS), which is sensitive to signals of reward, and the *behavioral inhibition system* (BIS), which is sensitive to signals of punishment. Two personality dimensions (Impulsivity and Anxiety) are proposed to account for individual differences in arousal. For example, those high on impulsivity and low on anxiety have an overactive BAS and underactive BIS, and those high on both impulsivity and anxiety will have an overactive BAS and an overactive BIS. Therefore, while both of these types of individuals will exhibit impulsivity, those also high on anxiety will experience more stress due to the consequences of their behavior.

Psychophysiological research has tended to support the notion of individual differences in resting arousal: Mathias and Stanford (2003) found that healthy males high in impulsivity experienced lower baseline arousal, stronger reactions to arousal-eliciting stimuli, and declining arousal after prolonged exposure to such stimuli (Mathias & Stanford, 2003). Houston and Stanford (2001) have produced contradictory results, showing that those high in BIS impulsivity experience increased reactivity in response to

increased stimulus intensity. However, one potential confound with this finding was that the individuals studied were aggressive impulsives, and thus aggression may have mediated their electrodermal arousal (Houston & Stanford, 2001).

Defining Impulsivity

Impulsivity (sometimes called impulsiveness) is a complex construct that has been assigned differing meanings across areas of psychological research (Evenden, 1999a, Fernandez & Bravo, 2003; Hollander & Evers, 2001, Hollander & Rosen, 2002, Swann, Bjork, Moeller, & Dougherty, 2002; J.L. White, et al., 1994). Some of the many definitions of impulsive people, impulsive behavior, or impulsive personality in the literature include “a dimension of personality [that] is the failure to resist an impulse, drive, or temptation that is harmful to oneself or others” (Hollander, Posner, & Cherkasky, 2002); “given to sudden, imprudent, and predominately affective action” (Twain, 1957); “driven by the desire to obtain pleasure, arousal, and gratification” (Hollander & Rosen, 2000); "inappropriate use of speed and time, causing a premature style and lack of persistence, and weak control mechanisms" (Rubia, 2002); and “actions that appear poorly conceived, prematurely expressed, unduly risky, or inappropriate to the situation and that often result in undesirable consequences” (Daruna & Barnes, 1993). The last two definitions attempt to include almost all facets of impulsivity currently researched. Not surprising from the wide variety of actions and traits included in these definitions is that there appear to be several unique aspects of impulsivity, without a common neurobiological basis (Evenden, 1999b): this is to say, the term *impulsivity* is used to describe a great deal of different syndromes, not all of which are related to increases in cortical arousal.

Kindlon and colleagues (1995) identified the two most researched domains of impulsivity as the motivational domain, encompassing individual differences in the sensitivity to signals of reward and punishment, and the cognitive domain, encompassing impairments in executive control of inhibition (Kindlon, Mezzacappa, & Earls, 1995). The study of the motivational domain of impulsivity stems primarily from arousal theory, defining impulsivity as the high sensitivity to signals of reward coupled with low sensitivity to signals of punishment (Patterson & Newman, 1993; Wallace, et al., 1991; Zuckerman, 1985). More recent studies of motivational impulsivity focus on the delay discounting paradigm, measuring the level at which smaller, immediate reinforcers are preferred to larger, delayed reinforcers (e.g. Bickel, Odum, & Madden, 1999; Epstein, et al., 2003; Mitchell, 1999). The cognitive domain of impulsivity has primarily been studied from a cognitive science perspective, and thus the research focus has been on acting before thinking (e.g. Kagan, 1965), or failures in information processing (e.g. Dickman & Meyer, 1988; Hinson, Jameson, & Whitney, 2003). These syndromes may be unrelated to arousal. Laboratory measures of impulsivity generally address either the motivational domain or the cognitive domain. Measures of impulsive personality attempt to include both behavioral and cognitive aspects of impulsivity as they relate to stable, consistent traits (e.g. Cloninger, 1986, 1987; S. B. G. Eysenck, Pearson, Easting, & Allsopp, 1985; Patton, et al., 1995; Herpertz & Sass, 1997).

Personality Measures of Impulsivity and Their Theoretical Origins

Impulsiveness-Venturesomeness-Empathy Scale (I_7). Hans Eysenck originally subsumed Impulsivity (called Impulsiveness in his model) under Extraversion (E) in his two-factor model of personality (Eysenck, 1947). He cited a number of earlier

dichotomous personality traits, which can be seen as the forerunners of impulsivity: "Non-perseveration vs. Perseveration" (Spearman, 1927); "Slow oscillation vs. Quick oscillation" (McDougall, 1926); "Careless vs. Careful" (Downey, 1923); Slow personal tempo vs. Quick personal tempo" (Kretschmer, 1926); and "Lacking in persistence vs. Persistent" (Downey, 1923; all references as cited in Eysenck, 1947). Thus, the "personality factor" of impulsivity, located under the "personality style" of E, was created. Impulsivity comprised non-perseverative, non-persistent, quickly changing behavior accompanied by a rapid personal tempo. H. J. Eysenck and S. B. G. Eysenck (1975) later developed a three-factor model of personality, adding Psychoticism (P) to the factors of Neuroticism (N) and E. Factor analysis of the 43 impulsivity items from the E, N, and P scales of the Eysenck Personality Questionnaire (EPQ; H. J. Eysenck & S. B. G. Eysenck, 1975) yielded four factors: *narrow impulsiveness, risk-taking, non-planning,* and *liveliness* (S. B. G. Eysenck & H. J. Eysenck, 1977). The Eysencks then decided to create a separate inventory, apart from the EPQ, for measuring impulsivity (Imp), Venturesomeness (Vent), and empathy (Emp). This measure, the I_7 (sometimes called the IVE) was created to acknowledge that Imp, Vent, and Emp remain subfactors of personality, but they nonetheless occupy a unique space within personality and are worth measuring directly (S. B. G. Eysenck & H. J. Eysenck, 1978). The I_7 has been slightly revised since its initial incarnation to improve reliability and factor loadings (Cronbach's α: Imp, .84 males, .83 females; Vent, .85 males, .84 females; Emp, .69 males, .69 females; S. B. G. Eysenck, et al., 1985). Of these three factors, Imp and Vent are conceptually related to impulsivity, whereas Emp is not. The theoretical universality of Imp and Vent across cultures was bolstered by a recent study that replicated these

impulsivity constructs using a French-language translation of the I_7 with a French sample (Caci, Nadalet, Bayle, Robert, & Boyer, 2003).

Barratt Impulsiveness Scale (BIS-11). The Barratt Impulsiveness Scale measures impulsivity based on the assumption that impulsivity and restraint lie at opposite poles of the same construct (Barratt, 1965), and that impulsivity encompasses both cognitive and behavioral components (Barratt & Patton, 1983). Barratt relates his construct of impulsivity to "behavioral oscillation," i.e. weak "habit strength" in the Hull/Spence classic S-R behaviorist paradigm (Patton, et al., 1995). This theory assumes that impulsivity is a third-order dimension, under the first- and second-order dimensions of response speed, risk-taking, acting before thinking, and nonplanning. Additionally, Barratt's theory assumes that impulsivity is related to the similar concepts of extraversion, sensation seeking, and general inhibition (Barratt & Patton, 1983). Finally, this theory assumes that motor skills are more affected by impulsivity than sensory discrimination (Barratt, 1967). The items on the original Barratt Impulsiveness Scale (BIS) were selected to fit Barratt's conceptualization of impulsivity as a personality trait, orthogonal to anxiety, influencing efficiency in performance of motor tasks (Barratt, 1959). After four revisions, Barratt republished the BIS-5 (Barratt, 1965) accompanied by factor analysis results showing the BIS-5's discriminant validity from anxiety measures. The BIS-5 was not significantly correlated with any of the primary anxiety or emotional stability measures of the time (Barratt, 1965). The BIS was most recently revised (BIS-11; Patton, et al., 1995) to allow easier comparison to the Eysencks' I_7. The initial exploratory factor analysis of the BIS-11 found three second-order factors, two of which (Motor Impulsiveness and Nonplanning Impulsiveness) correlated respectively with two

of the I_7 subfactors (Impulsiveness in the narrow sense and Nonplanning); the last second-order factor, Attentional Impulsiveness, was not correlated with any of the I_7 subfactors (Patton, et al., 1995). Despite this factor analysis, the BIS-11 does not yield consistent subscales, and accordingly, the BIS-11 total score is generally used without regard to scores on subfactors. The BIS-11 total score has shown to be reliable with a number of diverse populations (Cronbach's α for undergraduates: .82; substance-abusers: .79; general psychiatric patients: .83; inmates: .80; Patton, et al., 1995).

Behavioral Measures of Impulsivity and Their Theoretical Origins

Delay Discounting Tasks. Delay discounting tasks (DDTs) measure the extent to which delayed consequences are devalued, which is an operational definition of impulsivity from a behaviorist perspective (Ainslie, 1975; Rachlin, Raineri, & Cross, 1991). Unlike many behaviorist paradigms in psychology, delay discounting is also used in cognitive psychology for assessing impulsivity (e.g. Hinson, et al., 2003). Delay discounting bridges both the cognitive frames model and the behavioral choice model of impulsivity, because both models can convincingly explain the phenomenon (Rachlin, Logue, Gibbon, & Frankel, 1986). The behaviorist model implicates the learning of consequences of choosing small, immediate rewards compared with large, delayed rewards (Ainslie, 1975); the cognitive model emphasizes the expected value of a delayed reward based on Prospect Theory, the notion that certain gains are favored over uncertain gains of equal or greater value (Mill, 1829; Tversky & Kahneman, 1981). A number of DDT paradigms have been created and used; the DDT used in this study is described in the "Methods" section, with full instructions found in Appendix H. Delay Discounting Tasks have been shown to be effective instruments for measuring drug effects on

impulsivity: for example, they have been used to measure the influence of alcohol intoxication on impulsivity (Ortner, MacDonald, & Olmstead, 2003). A review of the delay discounting literature reported that measures of delay discounting consistently show a positive correlation with impulsive personality as measured by the I_7 and BIS-11 (Bickel & Marsch, 2001). Although there are a variety of similar measures of delay discounting, none of them is considered the "gold standard," as the measures are intercorrelated (the correlation between two major DDTs was .82), and there is no objective standard of what an individual's "true" level of discounting is (Epstein, et al., 2003). Thus, it seems at the present time that any of the available DDTs are appropriate for use in laboratory studies.

Stroop Color-Word Test. The Stroop Color-Word Test (SCWT) was originally developed to study the process of interference in verbal reactions to stimuli (Stroop, 1935). The SCWT measures interference of one type of stimulus (word names) with another (color names) in a series of words printed in a color ink that is a different color than the word itself (e.g. RED printed in blue ink). An interference score on the SCWT, measuring an individual's level of stimulus interference, can be computed by subtracting his or her number of errors on the non-interference task (naming words) from his or her number of errors on the interference task (van Mourik, Oosterlan, & Sergeant, 2005). The most commonly used version of the SCWT is that developed by Golden (1976); it has been shown to discriminate impulsive children and adolescents from normals, and to have high temporal stability (squared partial correlations: words correct, .67; colors correct, .57; interference score: .47; Kindlon, et al., 1995).

Stop Signal Task. The Stop Signal Task (SST) is a computerized task that was designed to measure individuals' ability to inhibit motor responses (Logan & Cowan, 1984; Logan, Cowan, & Davis, 1984; Logan, Sachar, & Tannock, 1997). The SST is based upon the notion that conflicting "stop" and "go" stimuli may be present in the natural environment simultaneously, such as when a response is triggered but other cues suggest it may be inappropriate (Bedard, et al., 2003; Schachar & Logan, 1990). In the SST, the letter "X" or "O" appears on the screen and the participant is asked to press (as quickly as possible) a corresponding key for each letter. On 25% of trials dispersed randomly throughout the task, participants hear a tone that signals inhibiting the response (i.e. not pressing either key). The delay between the stimulus letter (X or O) and the inhibitory tone is varied systematically until the participant inhibits responses on 50% of signal trials. After the stop signal delay has been adjusted, the signal response time (SRT) is calculated by subtracting the final mean delay from the final mean reaction time. The mean probability of inhibition on signal trials, (corrected for omission errors on nonsignal trials) has been found to show moderate to high temporal stability, as well as discriminant validity in distinguishing normal children and adolescents from those with impulse-control disorders (Kindlon, et al., 1995).

The Relationship Between Behavioral and Personality Measures of Impulsivity

Gerbing, Ahadi, and Patton conducted a factor analysis of all the major personality and behavioral measures of impulsivity, and identified twelve self-report components and three behavioral components of impulsivity (Gerbing, Ahadi, & Patton, 1987). These 15 components load onto three factors: *spontaneous, not persistent*, and *carefree*. A more recent factor analysis of nine personality measures of impulsivity found

eight factors: *concentration, decision-making, thinking, money, excitement, temper control, future orientation*, and *complexity avoidance* (Harmstead & Lester, 2000). It is possible that while some individuals may exhibit multiple facets of impulsivity, any given individual may exhibit some facets of impulsivity and not others.

The Role of Impulsivity in Psychiatric Disorders

Impulsivity is considered a normally distributed personality trait in all models of impulsive personality (e.g. Gillespie, Cloninger, Heath, & Martin, 2003; S. B. G. Eysenck, et al., 1985; Gray, 1987; Zuckerman, 1993). However, impulsivity is also considered a hallmark feature of a number of DSM-IV psychiatric diagnoses, including Antisocial Personality Disorder, ADHD, Bipolar Disorder, Borderline Personality Disorder (BPD), Bulimia Nervosa, Intermittent Explosive Disorder, Kleptomania, Pathological Gambling, Pyromania, Substance Use Disorders, and Trichotillomania (American Psychiatric Association, 1994). Buss and Plomin (1975) called impulsivity "the most troublesome of the four temperaments" in their developmental model of personality, due to the very definition of impulsivity as lacking control.

It appears that in some limited cases, impulsivity can be a protective factor for psychiatric disorders: in the absence of anxiety, impulsivity is associated with lower rates of depression in a sample of college males (Farmer, 1998). However, as a general principle, impulsive personality is seen in higher levels in those with any DSM-IV Axis I or Axis II diagnoses than in normals (Moeller, et al., 2001). Personality measures of impulsivity predict a wide variety of psychiatric disorders and other problem behaviors: impulsivity has been shown to predict suicidality in those diagnosed with BPD (Brodsky, Malone, Ellis, Dulit, & Mann, 1997; Moeller, et al., 2001), degree of self-mutilation in

those diagnosed with personality disorders (Simeon, et al., 1992), bulimia-type binge eating in adolescents (Kaltiala-Heino, Rissanen, Rimpela, & Rantanen, 2003), gambling treatment dropout (LeBlond, Ladouceur, & Blaszcynski, 2003), relapse after eating disorder treatment (Keel, Mitchell, Miller, Davis, & Crow., 2000), MDMA ("ecstasy") use in college students (Morgan, 1998), driving violations in the elderly (Owsley, McGwin, & McNeal, 2003); delinquency in adolescent boys (Cooper, Wood, Orcutt, & Albino, 2003; White, et al., 1994); poor grades in adolescent boys (Cooper, et al., 2003) and general risk-taking (S. B. G. Eysenck & H. J. Eysenck, 1978; Sher & Trull, 1994; Stanford, et al., 1996; Zuckerman & Kuhlman, 2000).

Research into the impulsive disorder of gambling addiction has examined the relationship between gambling and Zuckerman and colleagues' construct of *sensation seeking* (Zuckerman, 1971, 1983; Zuckerman, Bone, Neary, Magelsdorff, & Brustman, 1972; Zuckerman, Kolin, Price, & Zoob, 1964). Sensation seeking is an arousal subfactor closely correlated with impulsivity as measured by the BIS, I_7, and TPQ scales (Zuckerman & Cloninger, 1996; Zuckerman & Kuhlman, 2000). For example, the "impulsive sensation seeking" (ImpSS) scale of Zuckerman's Sensation Seeking Scale (SSS) correlates at r=.68 with Cloninger's "Novelty Seeking" impulsivity construct on his TPQ scale (Zuckerman & Cloninger, 1996). Arousal is considered the primary motivation for gambling (Anderson & R. I. F. Brown, 1984; R. I. F. Brown, 1986; Coventry & R. I. F. Brown, 1993; Dickerson & Adcock, 1987; Leary & Dickerson, 1985), and increased arousal when gambling is significantly positively correlated with gambling withdrawal symptoms (Wray & Dickerson, 1981). Under the arousal theory umbrella, sensation seeking and impulsivity can be seen to largely account for the same

portion of variance in impulse-control disorders such as gambling and SUDs. For example, one recent study found that both SSS and I_7 scores distinguished between college students who were low and high on caffeine abuse and dependence symptoms (Jones & Lejuez, 2004). Additionally, McCormick (1993) directly measured BIS impulsivity in substance abusers and found that those with severe gambling problems had significantly higher BIS-10 scores than those without a severe gambling problem. These data also support the notion that studies of sensation seeking and impulsivity in impulse-control disorders are primarily measuring the same aspects of arousal seeking.

Not surprisingly, DSM-IV Substance Use Disorders (SUDs) often co-occur with other impulse control disorders (e.g. Lacey & Evans, 1986; O'Boyle & Barratt, 1993; Stanford & Barratt, 1992); in prospective studies, impulsivity is a good predictor of SUDs (Sher, Bartholow, & Wood, 2000) and severity of problem gambling (Steel & Blaszczynski, 1998). McGue and colleagues found that impulsive personality distinguishes alcoholics who use other drugs from those who do not use other drugs (McGue, Slutske, & Iacono, 1999). Bayle and colleagues found that impulsive personality distinguishes kleptomaniacs from psychiatric controls (Bayle, Caci, Millet, Richa, & Olie, 2003). Novelty-seeking impulsivity, as measured by Cloninger's Tridimensional Personality Questionnaire (TPQ; Cloninger, 1987), distinguishes alcoholics from non-alcoholics and smokers from nonsmokers (Howard, Kivlahan, & Walker, 1997). McCormick (1993) found that within a population of those diagnosed with SUDs, impulsivity predicted the number of substances abused, and distinguished those who had gambling problems from those who did not. Bickel and Marsch have made the argument that the co-occurrence of SUDs and other impulse-control disorders reflect

the shared influence of impulsivity on a cluster of impulse-control disorders (Bickel & Marsch, 2001).

Buss and Plomin (1975) proposed that impulsive temperament is a developmental risk factor for SUDs in general. However, they later argued against this view, because impulsivity cannot be measured in neonates and infants, and as such, they could not show that impulsivity occurs prior to the onset of SUDs (Buss & Plomin, 1984). It is well accepted that SUDs are determined by a variety of environmental and genetic causes (e.g. O'Brien, 2003), and in light of the multiple causation of SUDs, Tarter (1988) argued that impulsivity cannot be considered a primary risk factor in the development of SUDs. Nonetheless, impulsivity remains a common factor across SUDs. While there appears to be little basis for a single general predisposition to become addicted to drugs (Rozin & Stoess, 1993), dysfunctional emotional regulation and poor impulse control appear to predispose adolescents to a variety of risk behaviors, accounting for nearly 30% of the variance in general problem behavior in adolescents (Cooper, et al., 2003).

The seeming co-occurrence of impulse control disorders is consistent with personality models of mental illness, in which persons with similar personality profiles may exhibit one or more disorders from a spectrum of similar disorders. Bayle and colleagues found that in a population diagnosed with kleptomania, common co-diagnoses were bipolar disorder, recurrent depression, compulsive buying, trichotillomania, alcohol or nicotine dependence, personality disorders (PDs), and suicidal behavior (Bayle, et al., 2003). A recent French study found that the impulse control disorders most likely to co-occur were kleptomania, trichotillomania, and bulimia in one cluster, and compulsive buying, pathological gambling, and trichotillomania in another cluster (Lejoyeux,

Arbaretaz, McLoughlin, & Ades, 2002). In this study, BIS impulsivity was not found to differentiate those with and without DSM-IV Axis I impulse control disorders. Additionally, Antisocial Personality Disorder and BPD did not cluster with the DSM Axis I impulse control disorders. This suggests that impulsive personality disorders, reflecting a general inability to tolerate boredom, do not share a similar etiology with specific behavioral syndromes of impulsivity (Lejoyeux, et al., 2002). Steel and Blaszczynski (1987) maintain the opposite: that within a population of pathological gamblers (i.e., those with an Axis I impulse control disorder), their construct of the "antisocial impulsivist" (Blaszczynski, Steel, & McConaghy, 1997) receives high principal-component analysis loadings from all four Cluster B and three of the four Cluster C PDs, including Antisocial PD (+. 62), BPD (+. 53), Histrionic PD (+. 70), Narcissistic PD (+. 45), Dependent PD (+. 44), Avoidant PD (+. 60), and Passive-Aggressive PD (+. 41; Steel & Blaszczynski, 1997). This would suggest that virtually all the non-schizophrenia spectrum DSM PDs share factors under the umbrella construct of impulsivity and antisociality, and that these disorders share an etiology with the Axis I disorder of Pathological Gambling.

Neuropsychological Correlates of Impulsivity

Impulsivity has been associated with frontal lobe dysfunction (White, et al., 1994). Additionally, populations of impulse-control disordered patients show frontal lobe impairment on neuropsychological tests. Kunert and colleagues (2003) measured BIS-10 impulsivity n a population diagnosed with BPD. In this population, BIS-10 cognitive impulsiveness was significantly negatively correlated with Wechsler Adult Intelligence Scale (WAIS) performance IQ scores (r=-.56) and with the capacity to learn on a

reminding test (*r*=-.56). BIS-10 motor impulsiveness was significantly negatively correlated with mean reaction times on an alertness task (*r*=-.63), WAIS performance IQ (*r*=-.57), and age-corrected WAIS block design scores (*r*=-.56). BIS-10 nonplanning was significantly negatively correlated with WAIS picture completion scores (*r*=-.60). BIS-10 total scores were significantly negatively correlated with WAIS full-scale IQ (*r*=-.57) and WAIS performance IQ (*r*=-.66). These findings suggest a general inverse relationship between impulsivity and frontal lobe functioning (Kunert, Druecke, Sass, & Herpertz, 2003). Regard and colleagues (2003) found problem gamblers performed significantly worse on a variety of tests of frontal lobe functioning, including concentration, verbal fluency, figural fluency, interference on the Stroop Task, concept learning and identification, verbal learning, and visuospatial recall memory (Regard, Knoch, Gutling, & Landis, 2003). It should be noted that these tests are measures of cognitive impulsivity, and as such, these frontal lobe syndromes may or may not be related to general cortical arousal.

Impulsivity and Aging

Levels of impulsivity appear to remain fairly consistent throughout adolescence and young adulthood, and to decrease throughout older adulthood. Cooper and colleagues (2003) measured behavioral impulsivity via items taken from the NEO-PI (Costa & McCrae, 1985) in a cross-sectional sample of teenage boys aged 13-19, and then measured impulsivity again 4.5 years later. The temporal reliability of the NEO-PI-derived impulsivity scale was .63 (Cooper, et al., 2003). These results suggest that impulsivity remains stable throughout adolescence and into young adulthood. Lejoyeux and colleagues (2002) found that in a sample of depressed patients, those with co-

occurring impulse control disorders were significantly younger than those with a single impulse-control disorder (mean age = 37.7 versus 42.8 years; Lejoyeux, et al., 2002). These results suggest that impulsivity decreases throughout adulthood. Hurt and Oltmanns (2002) found similar results in a sample of incarcerated adult women with personality disorders: impulsivity was significantly negatively correlated with age (r=-.18). Finally, Zuckerman's Sensation-Seeking factor, a construct closely related with impulsivity (Zuckerman & Cloninger, 1996), is also significantly negatively correlated with age, decreasing across adulthood (Zuckerman, S. B. G. Eysenck, & H. J. Eysenck, 1978; Zuckerman & Neeb, 1980).

Drug Effects of Nicotine

Nicotine is a powerful drug that reaches its maximum brain concentrations almost immediately after smoking a cigarette (NIDA, 2004) and produces a variety of stimulant and depressant effects (Parrott, 1998). Nicotine affects nicotinic acetylcholine receptors (nAChRs) to activate reward centers in the central nervous system (CNS), increasing dopamine (DA) and epinephrine release in the cerebral cortex of the brain (Mansvelder & McGehee, 2002; NIDA, 2004; Nutt, 1997). Additionally, nicotine increases regional cerebral blood flow (rCBF) in the left frontal region of the cortex, and decreases rCBF in the left amygdala and the right hemisphere of the cortex (Rose, et al., 2003). Dose effects on activation have been found for nicotine, such that lower doses increase reticular activation, but higher doses decrease reticular activation (Rose, et al., 2003). Such physiological findings are consistent with the psychological findings that low doses of nicotine produce a stimulant effect, but high doses of nicotine produce a sedative effect (Ashton, et al., 1980; Clarke, 1987). Electroencephalography (EEG) studies of

participants who have consumed nicotine consistently show that nicotine produces cortical arousal; however, individual analysis in some studies has shown that some individuals experience reduced cortical arousal response to nicotine (Conrin, 1980). Levin and colleagues found that heavy smokers experience increased resting heart rate and decreased heart rate variability, which are factors that can lead to sudden death. Thus, it is possible that these changes are a mechanism of mortality in smokers (Levin, Levin, & Nagoshi, 1992). Masson and Gilbert found that the magnitude of heart rate and diastolic blood pressure changes due to smoking was significantly higher in those with Type A personality than those with Type B personality (Masson & Gilbert, 1990). This effect further underscores the differential effects of nicotine for those with different personality types. Nicotine also has been shown to have differential effects for men and women in laboratory studies: replicated research has shown that male smokers experience heightened sensitivity to the pharmacological effects of nicotine compared with female smokers (Benowitz & Hatsukami, 1998; Daurignac, et al., 2001; Ikard & Tomkins, 1973; Perkins, 1996; Perkins, et al., 2002).

Strong relationships have consistently been found between smoking and a number of mental disorders: de Leon and colleagues found significantly higher frequencies of nicotine dependence in both U.S. and Spanish inpatients with diagnoses of Bipolar Disorder or Schizophrenia (de Leon, Becona, Gurpegui, Gonzalez-Pinto, & Diaz, 2002). Breslau and colleagues found that nicotine-dependent young adults were at significantly increased risk for alcohol disorders, SUDs, major depression, and anxiety disorders (Breslau, Kilbey, & Andreski, 1994). In those with Panic Disorder, smoking is a risk factor for panic attacks (Breslau & Klein, 1999; Isensee, Wittchen, Stein, Hofler, & Lieb,

2003). These findings suggest that nicotine is commonly used as self-medication for a variety of mental disorders. Nicotine has been found to potentate the effects of neuroleptic drugs in treating the impulsive symptoms associated with Tourette's disorder (Sanberg, et al., 1997); however, the exact mechanism by which nicotine reduces impulsivity in Tourette's patients is not clear. Nicotine increases the levels of serotonin (5-HT) in the CNS, including the cortex; this could potentially effect a reduction in impulsivity (Seth, Cheeta, Tucci, & File, 2002). It is clear that impulsivity increases as a result of nicotine withdrawal (Sommese & Patterson, 1995); what is not clear is whether this effect suggests that nicotine intake decreases impulsivity in and of itself.

Drug Effects of Caffeine

Caffeine is a methylxanthine compound that acts as a strong CNS and skeletal muscle stimulant; in doses of 50-200 mg, it stimulates the cerebral cortex; in doses over 500 mg, it stimulates the medulla, vagus, and vasomotor centers (Gilliland & Bullock, 1984). Caffeine blocks adenosine receptors in the brain (Nutt, 1997). Caffeine stimulates the cardiac muscle, increasing heart rate; it also dilates blood vessels and constricts blood flow in the cerebral cortex, which can relieve headaches; and it functions as a mild diuretic, increasing urination (Gilliland & Bullock, 1984).

Smith, Rypma, and Wilson (1981) found that caffeine produced stronger skin conductance response for low impulsives than for high impulsives. This finding is consistent with findings with introverts and extraverts (e.g. Fowles, Roberts, & Nagel, 1977; Smith, Wilson, & Davidson, 1984; Smith, Wilson, & Jones, 1983), and suggests that the effects of caffeine on impulsivity may be closely related to the effects of caffeine on arousal in general.

Caffeine dependence is neither formally recognized in DSM-IV, nor included as a criteria set for further study. This may have been due in part to the work of Hughes and colleagues (Hughes, Oliveto, Helzer, Higgins, & Bickel, 1992), who did not recommend that caffeine dependence be added to the DSM, as the diagnosis fails to meet DSM standards for Substance Dependence. However, Hughes and colleagues did acknowledge the existence of a caffeine dependence syndrome, and its heuristic value for clinicians (Hughes, et al., 1992). More assertively, Strain and colleagues (1994) reported that caffeine dependence syndrome could and should be diagnosed using DSM criteria for Substance Dependence as they apply to caffeine (Strain, Mumford, Silverman, & Griffiths, 1994). Caffeine dependence has been observed in teenagers (Bernstein, Carroll, Thuras, Cosgrove, & Roth, 2002), and has been created in a laboratory study involving adults (Silverman, Evans, Strain, & Griffiths, 1992). The actual neurochemical mechanisms by which caffeine withdrawal occurs are unknown (Hughes, 1992). The relationship between caffeine use and dependence may be compared to alcohol use and dependence: most individuals will use caffeine without experiencing problems, but a substantial minority will become dependent (Glass, 1994).

Interactive Effects of Nicotine and Caffeine on Arousal

Results are mixed on the interactive effects of nicotine and caffeine on arousal. One study found that when nicotine and caffeine were administered jointly, they produced an additive effect on physiological arousal, as seen in marked increases in blood pressure during task performance after the consumption of both nicotine and caffeine (Ray, Nellis, Brady, & Foltin, 1986). However, another line of research by Rose and colleagues (Rose, 1986; Rose & Behm, 1991; Rose, et al., 1993) found that while

caffeine alone increases arousal, nicotine blocks this effect when jointly administered. However, it is possible that this interaction is due to the arousal input-output function; as an inverted-U, increasing arousal input beyond a certain point results in a decrease of arousal (Smith, 1994).

The Relationships Among Smoking, Caffeine Intake, and Impulsivity

Cigarette smoking and caffeine intake are moderately to strongly related with each other (Brown & Benowitz, 1989; Istvan & Matarazzo, 1984; Tanda & Goldberg, 2000); the correlation between caffeine intake and smoking has been found to range from .14 to .41 (Cameron & Boehner, 1982; Conway, Vickers, Ward, & Rahe, 1981; Prineas, Jacobs, Crow, & Blackburn, 1980). Additionally, smoking is moderately to strongly correlated with impulsive personality as measured with a variety of inventories (Dinn, Aycicegi, & Harris, 2004; Mitchell, 1999; Schubert, 1965). Much less published research has examined the relationship between caffeine intake and impulsivity. Waldeck & Miller (1997) found that caffeine intake and impulsivity were significantly positively correlated in college men, but not significantly correlated in college women.

Different studies of smoking and impulsivity have used different measures of impulsivity to measure this relationship. Spinella (2002) found smoking status and inhibition errors on the Go-No Go Task correlated at r=. 35 (df=X) and smoking status correlated with Barratt Impulsivity Scale (Patton, et al., 1995) non-planning at r=. 30 (df=X; Spinella, 2002). Heath and colleagues (1995) found the Novelty Seeking Scale of the Tridimensional Personality Questionnaire (Cloninger, Przybeck, & Svrakic, 1991) to correlate with smoking status (r= .28, df=X for women; r=. 27, df=X for men; Heath, Madden, Slutske, & Martin, 1995). Williams (1973) found smoking in ninth-grade boys

to be significantly positively correlated (*r*=. 30, *df*=X) with impulsivity as measured by the Jackson Personality Research Form (Jackson, 1967). Lipkus and colleagues administered the Minnesota Multiphasic Personality Inventory (MMPI) to college students; at a 25-year follow-up, they divided never-smokers from ever-smokers (i.e. current and former smokers) and found that the ever-smokers had received significantly higher *Pd* (Psychopathic Deviate) scores as college students, indicating increased impulsivity and rebelliousness (Lipkus, Barefoot, Williams, & Siegler, 1994). Many studies have found a positive correlation between sensation seeking and lifetime smoking (e.g. Carton, Jouvent, & Widlocher, 1994; Zuckerman, Ball, & Black, 1990). Smoking is also positively correlated with general risk-taking behavior (Hines, Steenberg, & DePew, 1995). An alternative hypothesis is that smoking addiction causes individuals to consider themselves more impulsive people due to their engaging in an unhealthy habit (Kassel, Shiffman, Gnys, Paty, & Zettler-Segal, 1994).

Some early studies examining personality correlates of smoking found a relationship between smoking and the broad construct of negative affect: smokers had significantly higher negative affect than nonsmokers (e.g. Ikard, Green, & Horn, 1969). Other early studies based on Eysenck's original two-factor model of personality found significantly higher extraversion scores across smoking status (e.g. non-, light, heavy) and with number of cigarettes smoked daily (H. J. Eysenck, Tarrant, Woolf, & England, 1960; H. J. Eysenck, 1963, 1964; Kanekar & Dolke, 1970; Rae, 1975). Since Eysenck moved the subfactor of impulsivity from E to P, a number of studies have found P to be positively correlated with smoking (e.g. Arai, Hosokawa, Fukao, Izumi, & Hisamichi, 1997; Golding, Harpur, & Brent-Smith, 1983). This suggests that both before and after

the change in Eysenck's model, impulsivity accounted for the majority of the variance in smoking accounted for by personality, despite his original assertion to the contrary (i.e. that extraversion itself accounted for this difference – cf. Eysenck, 1963). From negative affect to extraversion to psychoticism to sensation seeking to impulsivity, the specificity of what aspects of personality account for smoking behavior has narrowed over the years. It is hoped that this study can shed light on how impulsivity is related to smoking, and how caffeine fits into this equation.

Smokers commonly drink coffee or other caffeinated beverages while smoking cigarettes (Marshall, Epstein, & Green, 1980; Marshall, Green, et al., 1980; Swanson, Lee, & Hopp, 1994) and often report pleasurable sensations from this combination substance use. In general, caffeine use appears to act as a cue triggering smoking: smoking is twice as likely to occur after drinking coffee than before (Emurian, Nellis, Brady, & Ray, 1982). Despite the fact that smoking, caffeine intake, and impulsivity appear to be intercorrelated, the nature of their interaction has not been well researched. The reasons for the positive relationship between smoking and caffeine consumption are not known (Gilliland & Bullock, 1984); nor is it known if and how the two drugs interact to produce changes in arousal and impulsive behavior.

Appendix B: Demographics

1. What is your age? _____ years

2. What is your racial or ethnic identity? _____
    a. African American/Black
    b. Asian American or Pacific Islander
    c. Caucasian/White
    d. Native American or Alaskan Native
    e. Multiracial or other: _____

3. What is your class standing? _____
    a. High school/pre-college
    b. Freshman
    c. Sophomore
    d. Junior
    e. Senior
    f. College grad/Graduate student
    g. Other (please specify):_____

Appendix C: Caffeine and Smoking Survey

Instructions: Please indicate the number of times IN THE PAST WEEK that you consumed any of these products. That is, if you drank one latte on Tuesday morning, and another one on Thursday afternoon, write the number "2" in the box for "latte." Also be sure to note the size of the drink. Please try to answer as accurately as you can. Thanks!

PRODUCT	#	PRODUCT	#
COFFEE/COCOA		**SOFT DRINKS**	
Regular coffee AT HOME		Coca-Cola	
Regular coffee AT COFFEE SHOP		Diet Coke	
Decaffeinated coffee		Pepsi-Cola	
Café Mocha – list size:_____		Diet Pepsi	
Café Latte – list size:_____		Jolt Cola	
Instant Coffee		Afri Cola	
Instant Cappuccino		RC Cola	
Frozen coffee drink – list size:_____		Any Other Cola (brand: _____)	
Caramel Macchiato		Mountain Dew	
Any other espresso drink: _____		Diet Mountian Dew	
Hot Chocolate (Cocoa)		Dr. Pepper	
Other:_____		Diet Dr. Pepper	
TEAS		Mr. Pibb	
Green Tea (made from a tea bag)		Barq's Root Beer	
Black Tea (made from a tea bag)		Any Other Root Beer (brand: _____)	
Orange Tea (made from a tea bag)		Sunkist Orange Soda	
White Tea (made from a tea bag)		Any Other Orange Soda (brand:_____)	
Any Herbal Tea		Sprite/7UP	
Arizona Ice Tea (any kind) – bottle		Any Other Soda (brand: _____)	
Celestial Seasonings Ice Tea – bottle		**OVER-THE-COUNTER DRUGS**	
Celestial Seasonings Herbal Iced Tea		NoDoz, Maximum strength	
Nestea Ice Tea (any kind) – bottle		NoDoz, regular strength	
Lipton Ice Tea (any kind) – 16 oz. Bottle		Vivarin	
Lipton Brisk Ice Tea – 12 oz. Can		Excedrin, regular	
Snapple Ice Tea (any kind) – bottle		Excedrin PM	
Bigelow Raspberry Royale – bottle		Vanquish	
Any other tea: _____		Anacin	
CAFFEINATED WATER		Other drug with caffeine: _____	

Caffeinated Water (brand:_____)		**CHOCOLATE CANDY**	
FROZEN TREATS		Butterfinger	
Coffee Flavored Ice Cream:_____		Nestle Crunch	
Coffee Flavored Yogurt:_____		Other:_____	

(see next page for smoking questions)

1. At what age did you try your first cigarette? _____ years

2. At what age did you first start smoking regularly? _____ years

3. How many cigarettes do you CURRENTLY smoke, per day, on average? _____

4. Did you ever smoke MORE than you do now, on average? (circle) YES NO

5. If "YES," how many cigarettes did you smoke, on average, when you were smoking the most? _____

Appendix D: I-7

Instructions: Please answer each question by marking the "YES" or "NO" box following each question. There are no right or wrong answers, and no trick questions. Work quickly and do not think too long about the exact meaning of the questions.

1.	Would you enjoy water skiing?	**YES** ☐	**NO** ☐
2.	Usually do you prefer to stick to brands you know are reliable, rather than trying new ones on the chance of finding something better?	**YES** ☐	**NO** ☐
3.	Would you feel sorry for a lonely stranger?	**YES** ☐	**NO** ☐
4.	Do you enjoy taking risks?	**YES** ☐	**NO** ☐
5.	Do you often get emotionally involved with your friends' problems?	**YES** ☐	**NO** ☐
6.	Would you enjoy parachute jumping?	**YES** ☐	**NO** ☐
7.	Do you often buy things on impulse?	**YES** ☐	**NO** ☐
8.	Do unhappy people who are sorry for themselves irritate you?	**YES** ☐	**NO** ☐
9.	Do you generally do and say things without stopping to think?	**YES** ☐	**NO** ☐
10.	Are you inclined to get nervous when others around you seem to be nervous?	**YES** ☐	**NO** ☐
11.	Do you often get in a jam because you do things without thinking?	**YES** ☐	**NO** ☐
12.	Do you think hitch-hiking is too dangerous a way to travel?	**YES** ☐	**NO** ☐
13.	Do you find it silly for people to cry out of happiness?	**YES** ☐	**NO** ☐
14.	Do you like diving off the high-board?	**YES** ☐	**NO** ☐
15.	Do people you are with have a strong influence on your moods?	**YES** ☐	**NO** ☐
16.	Are you an impulsive person?	**YES** ☐	**NO** ☐
17.	Do you welcome new and exciting experiences and sensations, even if they are a little frightening and unconventional?	**YES** ☐	**NO** ☐
18.	Does it affect you very much when one of your friends seems upset?	**YES** ☐	**NO** ☐
19.	Do you usually think carefully before doing anything?	**YES** ☐	**NO** ☐
20.	Would you like to learn to fly an airplane?	**YES** ☐	**NO** ☐
21.	Do you ever get deeply involved with the feelings of a character in a film, play, or novel?	**YES** ☐	**NO** ☐
22.	Do you often do things at the spur of the moment?	**YES** ☐	**NO** ☐
23.	Do you get very upset when you see someone cry?	**YES** ☐	**NO** ☐
24.	Do you sometimes find someone else's laughter catching?	**YES** ☐	**NO** ☐
25.	Do you mostly speak without thinking things out?	**YES** ☐	**NO** ☐
26.	Do you often get involved in things you later wish you could get out of?	**YES** ☐	**NO** ☐
27.	Do you get so "carried away" by new and exciting ideas that you never think of possible snags?	**YES** ☐	**NO** ☐

28.	Do you find it hard to understand people who risk their necks climbing mountains?	**YES** ☐	**NO** ☐
29.	Can you make decisions without worrying about other people's feelings?	**YES** ☐	**NO** ☐
30.	Do you sometimes like doing things that are a little bit frightening?	**YES** ☐	**NO** ☐
31.	Do you need to use a lot of self-control to keep out of trouble?	**YES** ☐	**NO** ☐
32.	Do you become more irritated than sympathetic when you see someone cry?	**YES** ☐	**NO** ☐
33.	Would you agree that almost everything enjoyable is illegal or immoral?	**YES** ☐	**NO** ☐
34.	Generally, do you prefer to enter cold sea water gradually rather than diving or jumping straight in?	**YES** ☐	**NO** ☐
35.	Are you often surprised at people's reactions to what you do or say?	**YES** ☐	**NO** ☐
36.	Would you enjoy the sensation of skiing very fast down a high mountain slope?	**YES** ☐	**NO** ☐
37.	Do you enjoy watching people opening presents?	**YES** ☐	**NO** ☐
38.	Do you think an evening out is more successful if it is unplanned or arranged at the last moment?	**YES** ☐	**NO** ☐
39.	Would you like to go scuba diving?	**YES** ☐	**NO** ☐
40.	Would you find it very hard to break bad news to someone?	**YES** ☐	**NO** ☐
41.	Would you enjoy fast driving?	**YES** ☐	**NO** ☐
42.	Do you usually work quickly, without bothering to check your work?	**YES** ☐	**NO** ☐
43.	Do you often change your interests?	**YES** ☐	**NO** ☐
44.	Before making up your mind, do you consider all the advantages and disadvantages?	**YES** ☐	**NO** ☐
45.	Can you get very interested in your friends' problems?	**YES** ☐	**NO** ☐
46.	Would you like to go exploring in very small caves?	**YES** ☐	**NO** ☐
47.	Would you be put off by a job involving quite a bit of danger?	**YES** ☐	**NO** ☐
48.	Do you prefer to "sleep on it" before making decisions?	**YES** ☐	**NO** ☐
49.	When people shout at you, do you shout back?	**YES** ☐	**NO** ☐
50.	Do you feel sorry for shy people?	**YES** ☐	**NO** ☐
51.	Are you happy when you are with a cheerful group and sad when others are glum?	**YES** ☐	**NO** ☐
52.	Do you usually make up your mind quickly?	**YES** ☐	**NO** ☐
53.	Can you imagine what it must be like to be very lonely?	**YES** ☐	**NO** ☐
54.	Does it worry you when others are worrying and panicky?	**YES** ☐	**NO** ☐

Appendix E: BIS-11

Please answer each question below as it applies to you, as best you can, on the 1-4 scale.

		1. Rarely/ Never	2. Occasionally	3. Often	4. Almost Always/ Always
1.	I plan tasks carefully.	1. ☐	2. ☐	3. ☐	4. ☐
2.	I do things without thinking.	1. ☐	2. ☐	3. ☐	4. ☐
3.	I make up my mind quickly.	1. ☐	2. ☐	3. ☐	4. ☐
4.	I am happy-go-lucky.	1. ☐	2. ☐	3. ☐	4. ☐
5.	I don't "pay attention."	1. ☐	2. ☐	3. ☐	4. ☐
6.	I have "racing" thoughts.	1. ☐	2. ☐	3. ☐	4. ☐
7.	I plan trips well ahead of time.	1. ☐	2. ☐	3. ☐	4. ☐
8.	I am self-controlled.	1. ☐	2. ☐	3. ☐	4. ☐
9.	I concentrate easily.	1. ☐	2. ☐	3. ☐	4. ☐
10.	I save regularly.	1. ☐	2. ☐	3. ☐	4. ☐
11.	I "squirm" at plays or lectures.	1. ☐	2. ☐	3. ☐	4. ☐
12.	I am a careful thinker.	1. ☐	2. ☐	3. ☐	4. ☐
13.	I plan for job security.	1. ☐	2. ☐	3. ☐	4. ☐
14.	I say things without thinking.	1. ☐	2. ☐	3. ☐	4. ☐
15.	I like to think about complex problems.	1. ☐	2. ☐	3. ☐	4. ☐
16.	I change jobs.	1. ☐	2. ☐	3. ☐	4. ☐
17.	I act "on impulse."	1. ☐	2. ☐	3. ☐	4. ☐
18.	I get easily bored when solving thought problems.	1. ☐	2. ☐	3. ☐	4. ☐
19.	I have regular health check-ups.	1. ☐	2. ☐	3. ☐	4. ☐
20.	I act on the spur of the moment.	1. ☐	2. ☐	3. ☐	4. ☐
21.	I am a steady thinker.	1. ☐	2. ☐	3. ☐	4. ☐
22.	I change residences.	1. ☐	2. ☐	3. ☐	4. ☐
23.	I buy things on impulse.	1. ☐	2. ☐	3. ☐	4. ☐
24.	I can only think about one problem at a time.	1. ☐	2. ☐	3. ☐	4. ☐
25.	I change hobbies.	1. ☐	2. ☐	3. ☐	4. ☐
26.	I walk and move fast.	1. ☐	2. ☐	3. ☐	4. ☐
27.	I solve problems by trial-and-error.	1. ☐	2. ☐	3. ☐	4. ☐
28.	I spend or charge more than I earn.	1. ☐	2. ☐	3. ☐	4. ☐
29.	I talk fast.	1. ☐	2. ☐	3. ☐	4. ☐
30.	I often have extraneous thoughts when thinking.	1. ☐	2. ☐	3. ☐	4. ☐
31.	I am more interested in the present than the future.	1. ☐	2. ☐	3. ☐	4. ☐
32.	I am restless at the theater or lectures.	1. ☐	2. ☐	3. ☐	4. ☐
33.	I like puzzles.	1. ☐	2. ☐	3. ☐	4. ☐

Appendix F:

Positive and Negative Affect Scale

This scale consists of a number of words that describe different feelings and emotions. Read each item and mark the appropriate answer in the space next to that word. Indicate to what extent you FEEL THIS WAY RIGHT NOW.

1	**2**	**3**	**4**	**5**
very slightly/ not at all	**a little**	**moderately**	**quite a bit**	**extremely**

_____ interested	_____ irritable
_____ distressed	_____ alert
_____ excited	_____ ashamed
_____ upset	_____ inspired
_____ strong	_____ nervous
_____ guilty	_____ determined
_____ scared	_____ attentive
_____ hostile	_____ jittery
_____ enthusiastic	_____ active
_____ proud	_____ afraid

Appendix G: FTND

Please check the appropriate box for each question.

1. Do you smoke cigarettes on a daily basis?	❑ Yes ❑ No (Skip this page)
2. How soon after you wake up do you smoke your first cigarette?	❑ within 5 minutes ❑ 6-30 minutes ❑ 31-60 minutes ❑ After 60 minutes
3. Do you find it difficult to refrain from smoking in places where it is forbidden? (e.g. in church, at the library, in a movie theater)	❑ Yes ❑ No
4. Which cigarette would you hate most to give up?	❑ The first one in the morning ❑ A different one
5. How many cigarettes per day do you smoke?	❑ 10 or less ❑ 11-20 ❑ 21-30 ❑ 31 or more
6. Do you smoke more frequently during the first hours after waking up than during the rest of the day?	❑ Yes ❑ No
7. If you are so sick that you are in bed most of the day, do you still smoke?	❑ Yes ❑ No

Appendix H (**Consent): The Effects of Coffee and Cigarettes on Behavior**

Statement of Age of Participant: I state that I am over 18 years of age and in good physical health, and wish to participate in a program of research being conducted by Dr. Barry D. Smith, Department of Psychology, University of Maryland, College Park, MD 20742.

Purpose: The purpose of this study is to examine the effects of caffeine and cigarette smoking, alone and in combination, on people's behavior.

Procedures: My participation in this study should last less than one hour. I will be asked to complete a series of questionnaires regarding my demographic information, impulsivity, and mood. I will be asked to drink two six-ounce cups of coffee, which may or may not contain caffeine. I will also be asked to smoke two cigarettes, which may or may not contain nicotine, while drinking the coffee. Coffee drinking and cigarette smoking will take place outside the Biology-Psychology building, 15 feet away from the side door of the building. Coffee drinking and cigarette smoking will occur in two five-minute periods, separated by 15 minute periods inside the building. The total time spent outside will be ten minutes. Inside the laboratory, I will complete a computer task, a hand-eye task, and a time-estimation task, as well as some questionnaires including questions about personal characteristics such as impulsivity and mood. The results of this study may be used to advance research about why people use caffeine and nicotine.

Confidentiality: All information collected in the study is confidential. My name will appear on only a consent form and a receipt indicating that I was paid for participation. My name will not be connected to my data at any time "to the extent permitted by law." I realize that possible exceptions to confidentiality include cases in which evidence of abuse to children or impaired persons is uncovered, or if a court of law issues a subpoena for my research records. The data I provide in this research study, without my name attached, will be grouped with data from other participants if the results of the study are used in scientific reports or presentations.

Risks: I understand that although risks in the current study are low, I may experience dry mouth and increased heart rate from the coffee and/or cigarette smoking. Additionally, cigarette smoking is a known risk factor for lung cancer, heart disease, and stroke. I understand that these are the same risks I encounter from my own use of coffee and cigarette smoking. It is not the intent of the researchers to promote cigarette smoking as a lifestyle choice. I may experience temporary emotional discomfort as a result of completing the questionnaires, computer task, hand-eye task, or time estimation task.

Benefits, Freedom to Withdraw, & Ability to Ask Questions: I understand that this experiment is not intended to help me personally, but that the investigators hope to learn more about what effects caffeine use and cigarette smoking have on people's behavior. I understand that I may ask any questions about the study without penalty. I understand that I am free to withdraw from participation at any time without penalty.

Medical Care: I understand that the University of Maryland does not provide any medical or hospitalization coverage for participants in this research study. I understand that the University of Maryland will not provide and compensation for injury sustained as a result of participation in this research study except as required by law.

Rights of Research Participants: If I have questions about my rights as a research subject or I wish to report a research-related injury, I will contact: Institutional Review Board Office, University of Maryland, College Park, Maryland, 20742; (e-mail) irb@deans.umd.edu; (telephone) 301-405-4212

For questions about this study, contact:
Barry D. Smith, Ph.D.
University of Maryland
Department of Psychology
College Park, Maryland 20742
(301) 405-5860

Printed Name of Participant: _____

Signature of Participant: _____

Date: _____

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