Summer 2012

Assessing the utility of a brief Abstinence Test for initiating caffeine abstinence

Bradley T. Joachim
James Madison University

Assessing the Utility of a Brief Abstinence Test for Initiating Caffeine Abstinence

Bradley T. Joachim

A thesis submitted to the Graduate Faculty of

JAMES MADISON UNIVERSITY

In

Partial Fulfillment of the Requirements

for the degree of

Master of Arts

Department of Graduate Psychology

August 2012

Dedication

I dedicate my thesis project to my loving parents, Richard and Beverly Joachim, as a small token of my appreciation for all that they have done for me throughout my life.

Acknowledgements

I would like to acknowledge my parents for providing me with the supportive environment that has allowed me to reach this point in my academic career. I would like to thank Dr. Jessica Irons for taking me into her research lab, showing me the value and joy of teaching, and for being patient with me over the last 4 years leading up to my thesis. I am grateful for the opportunities she has extended to me and for what she has done for me as a professional, academic, and mentor; I wish I could contribute as significantly and positively to her life as she has done to mine during my time at JMU. I would also like to thank Dr. Dan Holt and Dr. Jeff Dyche for their informative feedback throughout my thesis project. I would like to thank Dr. Kyle Seifert for allowing me to use his lab to analyze samples and for teaching me things I did not anticipate learning during my thesis project. I am grateful for the many hours given by the undergraduate research assistants in Dr. Irons' lab who made completing my thesis possible. I am also gracious for the influence of Tracy Zinn and Bryan Saville in all aspects of my academic career, including on my thesis. I would like to thank Sherry Serdikoff for opening my eyes to behavior analysis and for being the first to recognize my passion for psychological research. Finally, I must acknowledge Brantley Jarvis and Stephen Robertson for their invaluable impact on my understanding of research and teaching today.

Table of Contents

List of Tables

List of Figures

Abstract

Caffeine is the most commonly consumed drug in the world. Although its effects are relatively mild when consumed in moderate amounts, there exist cases where caffeine use is problematic. Currently no behavioral intervention for problematic caffeine consumption exists in which caffeine use is verified beyond self-reports. No measures of caffeine dependence and withdrawal exist either. The current study examined the viability of contingency management, an empirically supported behavioral intervention for reducing drug use, for initiating abstinence from caffeine consumption among college students of varying levels of use, as well as validity evidence for novel measures of caffeine dependence and withdrawal. Participants (N = 39) came in to the lab for 3 experimental sessions in an ABA design over the course of 5 to 7 days to complete the AUTOC, CWS, and SCEWS and to provide saliva samples. During the BAT participants could earn a higher magnitude reward ($20) for abstaining from caffeine. 95% of participants met criteria for abstinence during the BAT. The ELISA appeared to work at an aggregate level, though individual samples were inconsistent enough to prevent these results from being used as a criterion for caffeine abstinence. AUTOC, CWS, and SCEWS scores functioned moderately well for measuring caffeine dependence and withdrawal. These results indicate CM of caffeine use may be effective for intervening with problematic caffeine consumption.

Introduction

Caffeine is a stimulant and the most consumed psychoactive drug in the world. Up to 80% of adults in the United States consume caffeine on a daily basis (Julien, 2008). Average daily caffeine intake among caffeine consumers has been estimated to be around 280 mg (Gilbert, 1984). Caffeine consumption typically occurs relatively consistently throughout the span of a lifetime; however little is known about its long term effects (James, 2004). It is naturally occurring in many foods and drinks commonly consumed in western culture, including coffee, tea, and cocoa (i.e., chocolate). Similarly, caffeine is added to a variety of consumable items such as soft drinks, energy drinks, gum, and soap, and is included as an ingredient in various over the counter and prescription drugs. In the United States caffeine is most frequently consumed in coffee and soda (Hughes & Oliveto, 1997).

Caffeine reaches peak blood concentration in 30 to 45 min. Caffeine's half-life varies as a function of individual factors, ranging from 2.5 to 10 hr, and it is longer for infants, pregnant women, and elderly persons (Julien, 2008). Once in the bloodstream, caffeine exerts excitatory effects as an adenosine antagonist. Adenosine is thought to build up during wakefulness, ultimately contributing to feelings of fatigue. By blocking adenosine receptors, caffeine causes an alleviation of sleepiness (Carlson, 2011).

Caffeine consumption, at both high and low doses, has been associated with increased alertness if consumed during low levels of arousal (e.g., early morning; Nawrot, Eastwood, Rotstein, Hugenholtz, & Feeley, 2003). Wesensten, Killgore, and Balkin (2005) demonstrated that a single large dose of caffeine (600 mg) administered to adults (N = 48) under 85 hr sleep deprivation improved response time on a simple

vigilance task. Similarly, Smith (2009) showed that a single administration of chewing gum containing a small amount of caffeine (40 mg) to college students (N = 118) led to significantly better performance on reaction tasks compared to those who consumed regular chewing gum or no gum.

Other subjective effects of caffeine are not as clearly established. For example, regular caffeine consumers may demonstrate greater improvements in memory and visiospatial reasoning (Jarvis, 1993; Smith, Sturgess, & Gallagher, 1999), although some research has shown caffeine to have no effect on memory or even negative effects (e.g., Erikson et al., 1985; Terry and Phifer, 1986). Not surprisingly Goldstein, Kaizer, and Warren (1965) found that self-reported effects of caffeine on alertness task performance did not correlate with actual performance, indicating that subjective self-reports of caffeine's affects may not be valid indicators of the actual behavioral effects of the drug. As such, subjective effects of caffeine have also been examined independently of behavioral effects. Because many people have experience with caffeine and its effects, there is often an expectancy effect associated with the drug. Oei and Hartley (2005) told participants they would be consuming caffeine or placebo prior to actually consuming one of either caffeinated or decaffeinated coffee. Participants who were told they had consumed caffeine made more correct detections in a signal detection task than those participants who were told they had consumed placebo, regardless of what they actually consumed.

Most desirable effects of caffeine (e.g., increased alertness) occur following consumption of small doses (e.g., less than 200 mg). Consuming larger quantities of caffeine (e.g., greater than 300 mg), however, is typically associated with the onset of

adverse symptoms, including nervousness and anxiety (Strain, Mumford, Silverman, & Griffiths, 1994). Research has suggested that administration of larger doses of caffeine (e.g., greater than 200 mg) is associated with increased anger and decreased drowsiness (Roache & Griffiths, 1987), although others have found no effect (Swift & Tiplady, 1988). Likewise, research shows that large doses (e.g., 1 g; Nawrot et al., 2003) of caffeine can produce anxiety in adults with anxiety disorders (Nawrot et al., 2003; Bruce, Scott, Shine, & Lader, 1982). Caffeine is also related to an increase in time taken to fall asleep as well as overall duration of sleep (Brenesova, Oswald, & Loudon, 1975).

Most research has focused on the acute effects of caffeine however there are some notable findings related to the long-term effects of caffeine consumption. Caffeine is associated with an increase in occurrence of reproductive complications when consumed in doses of 400 mg or more daily during pregnancy (e.g., SIDS; Ford et al., 1998), as well as with accelerated decrease in bone density among post-menopausal women who consume more than 450 mg daily (Harris & Dawson-Hughes, 1994). There is also an established relation between regular caffeine consumption and elevated blood pressure (James, 2004). At the population level, any increases in high blood pressure (including those attributed to regular caffeine consumption) are usually associated with increased rates of cardiovascular disease (James, 2004), though the link between regular caffeine consumption and cardiovascular disease is unknown. Additionally, long-term caffeine use may lead to a dependence syndrome analogous to other substance use disorders described in the DSM-IV-TR (APA, 2000). Although not formally recognized in the DSM-IV-TR as a substance of abuse, caffeine can lead to intoxication, withdrawal, and dependence according to the literature (e.g., Strain et al., 1992). Symptoms of

intoxication (also referred to as caffeinism; Greden, 1974) include exaggerated experience of caffeine withdrawal effects, including nervousness, irritability, agitation, and headache (Greden, 1974). Rates of excessive caffeine use leading to intoxication among psychiatric inpatients may be as high as 40% (MacKay & Rollins, 1989); however, rates of caffeinism in the population are uncertain due to underdiagnoses (Greden, 1974).

Caffeine dependence and withdrawal

According to the DSM-IV-TR, drug use must meet at least three of the following seven criteria to be considered drug dependence (relevant examples with caffeine included in parentheses): (a) tolerance (Griffiths & Woodson, 1988); (b) withdrawal (Silverman, Evans, Strain, & Griffiths, 1992); (c) substance often taken in larger amounts over a longer period of time than intended (Greden, 1974); (d) persistent desire or unsuccessful efforts to cut down or control use (Greden, 1974); (e) a great deal of time spent in activities necessary to obtain, use, or recover from the effects of the substance (Strain et al., 1994); (f) important social, occupational, or recreational activities given up or reduced because of substance use; (g) continued use despite knowledge of a permanent or recurrent physical or psychological problem that is likely to have been caused or exacerbated by substance use (Greden et al., 1978). Data have shown that caffeine use can lead to a situation in which an individual meets criteria for dependence (Hughes, Oliveto, Liguori, Carpenter, & Howard, 1998).

Caffeine dependence has been scarcely studied explicitly but research has yielded data that support the notion that dependence can and does occur. Several efforts have been made to characterize caffeine dependence among caffeine users. Strain, Mumford,

Silverman, and Griffiths (1994) examined 99 adults who self-reported psychological and physical dependence on caffeine. The authors used a structured clinical interview that included assessment of caffeine dependence based on DSM-IV (APA, 1994) criteria for substance dependence. Strain et al. (1994) found that 16 participants met criteria for caffeine dependence. Hughes, Oliveto, Liguori, Carpenter, and Howard (1998) reported prevalence of caffeine dependence criteria among a sample of 162 caffeine users via telephone interviews. Using DSM-IV criteria for substance dependence, Hughes et al. (1998) found a substantial proportion of participants self-reported experiences consistent with meeting criteria for caffeine dependence. Taken together with Strain et al. (1994) and other research on the potential for caffeine abuse (e.g., Ogawa & Ueki, 2007; Bernstein, Carroll, Thuras, Cosgrove, & Roth, 2002), these results provide compelling evidence for caffeine dependence.

An aspect of dependence that is of particular interest with respect to caffeine is withdrawal (e.g., Juliano & Griffiths, 2004). The greatest occurrence of undesirable effects of caffeine consumption occurs following cessation of use. Indeed, the effects of caffeine vary by latency since last consumption (Yeomans et al., 2002; Addicott & Laurienti, 2009; Christopher, Sutherland, & Smith, 2005), suggesting that many of the appetitive effects of caffeine are due to relief of caffeine withdrawal. Onset of withdrawal symptoms varies but generally occurs around 12 hr after last caffeine consumption (Griffiths & Woodson, 1988), with peak intensity of withdrawal symptoms occurring at 20 to 50 hr after last consumption (Juliano & Griffiths, 2004), continuing with progressively severity for 2 to 9 days (McKim, 2007). Symptoms are quickly alleviated by consuming caffeine (Julien, 2008; Juliano & Griffiths, 2004).

Juliano and Griffiths (2004) conducted a comprehensive review of the literature on caffeine withdrawal and found that headache was the most common symptom of withdrawal. Fatigue, decreased energy and activity, decreased alertness, drowsiness, decreased well-being, depressed mood, decreased concentration, irritability, and fogginess also met the authors' criteria for valid symptoms of caffeine withdrawal.

Despite evidence for caffeine dependence and withdrawal, there currently exists no standardized method for measuring these phenomena. Taken together, their lack of specific inclusion in the DSM-IV-TR (APA, 2000) and the fact that caffeine is a legal, widely available drug, among other potential reasons, limit the perceived prevalence and importance of caffeine dependence and withdrawal syndromes.

Contingency management

Just as there are few tools for measuring and characterizing caffeine dependence and withdrawal, there are fewer available treatment options, presumably because of a relative lack of concern related to excessive caffeine consumption. Unlike many other types of drugs (e.g., opioids, nicotine, alcohol) there are currently no effective pharmacotherapies for stimulants, including caffeine. As such, treatment of problematic caffeine consumption (e.g., dependence) may be best approached similarly to how dependence for other stimulants is treated—with behavioral interventions. Behavioral interventions involve manipulation of environmental variables that influence drug use, such as drug cost or availability (Bickel, Madden, & Petry, 1998), in order to decrease or cease drug consumption.

Contingency management (CM) has been a well-studied behavioral intervention strategy. In CM interventions for substance use, participants earn access to incentives

(e.g., money, prizes) contingent upon providing evidence of reduction or abstention from substance use. A wide range of studies have demonstrated the feasibility of CM for reducing use of a variety of substances (Petry, 2000), including nicotine (Roll, Higgins, & Badger, 1996), cocaine (Silverman, Chutuape, Bigelow, & Stitzer, 1999), opiates (Higgins, Stitzer, Bigelow, & Liebson, 1986), methadone (Iguchi, Belding, Morral, & Lamb, 1997), alcohol (Miller, Hersen, Eisler, & Watt, 1974), marijuana (Budney, Moore, Higgins, & Rocha, 2006), and benzodiazepines (Stitzer, Bigelow, Liebson, & Hawthorne, 1982).

Recent meta-analyses support the efficacy of CM and suggest that the effect sizes for CM interventions consistently exceed other treatment options for substance intervention (i.e., Lussier, Heil, Mongeon, Badger, & Higgins, 2006; Prendergast, Podus, Finney, Greenwell, & Roll, 2006; Dutra et al., 2008). Few studies to date have examined the efficacy of CM for intervening with caffeine use.

Several studies have applied CM to caffeine use; however, there are several critical deficits that suggest a need for further study. Foxx and Rubinoff (1979) investigated the utility of an incentive-based behavioral procedure for reducing excessive coffee drinking among three normal-functioning adults who self-reported interest in reducing their coffee drinking and consuming at least eight cups of brewed coffee per day. A changing criterion design (Hartmann & Hall, 1976) was implemented to reduce caffeine consumption to the equivalent of five cups of coffee or fewer over the course of 4 weeks. Coffee drinking was monitored via participant self-report and self-reports from a significant other who frequently interacted with the participant and was familiar with the participant's coffee drinking. Overall, the changing criterion design reduced coffee

drinking to moderate levels of use: Two participants never exceeded criterion levels of caffeine consumption and the third participant only exceeded criterion on 2 days (Foxx & Rubinoff, 1979). Follow-up at 40 months indicated participants continued to consume fewer than the terminal criterion goal of the initial study (Foxx, 1982). Bernard, Dennehy, and Keefauver (1981) performed a systematic replication of Foxx and Rubinoff (1979) in a single-case research procedure and yielded similar results. Although seemingly compelling, caffeine consumption was not verified beyond self-report, a key component of CM.

James, Stirling, and Hampton (1985) studied 27 adults who consumed at least eight cups of coffee or tea daily. Participants provided self-reports of caffeine consumption supplemented by independent reports of caffeine consumption by significant others. Participants were assigned to either a self-initiation regimen (i.e., an active treatment control group) or a fading procedure, with criterion determined based on individual levels of consumption. Participants in the fading group reduced consumption the most from baseline and maintained lowest consumption levels throughout treatment and again at 6- and 18-week follow-up.

CM procedures are effective for initiating and maintaining reduced caffeine consumption (Foxx & Rubinoff, 1979; Bernard et al., 1981; James et al., 1985). However, findings from studies to date are significantly limited in their reliance on self-report for verification of meeting criterion levels of caffeine use and lack of verification of caffeine use or abstinence. Additionally, CM can be costly to implement in terms of funding, researcher time, and the relatively high behavioral cost for participants.

The brief abstinence test

A brief abstinence test (BAT) can be used to model CM and to induce abstinence for a short period of time in effort to examine potential for efficacy (Robles et al., 2000). Participants in a BAT receive high-magnitude incentives contingent on providing evidence (self-report and biological samples) of brief (i.e., hours, or days) drug abstinence. Further, the BAT allows for experimental manipulation of use such that abstinence and related phenomena, such as withdrawal, may be examined. As such, a BAT is an ideal method for preliminary analysis of the potential efficacy of CM for caffeine use. The BAT has been used to initiate abstinence from opiates among opiate and cocaine dependent community adults (Sigmon, Correia, & Stitzer, 2004; Robles et al., 2000) as well as among nicotine dependent college student smokers (Irons & Correia, 2008).

Robles et al. (2000) evaluated the effectiveness of brief abstinence test for cocaine among cocaine-dependent methadone maintenance outpatients. Seventy-two participants enrolled in a 13-week methadone maintenance program were informed at the beginning of week 9 that if they returned in 2 days and had abstained from cocaine use since the beginning of the week that they would earn a $100 voucher. Participants who met abstinence criteria received payment 2 days after providing urine samples for analysis. No new use of cocaine was assumed by either a low benzoyclecgonine concentration (i.e., $\leq$ 300 ng/ml) analyzed from a frozen urine sample or a minimum 50% reduction in benzoyclecgonine concentration in the sample. Among those participants with complete data sets (n = 50), abstinence during BAT (84%) was significantly greater than abstinence during days prior to and immediately following the test date. Results demonstrated that

combining a high value reinforcer ($100) with a low cost behavioral requirement (i.e., abstinence from cocaine use for 2 days) yielded clinically significant rates of substance abstinence, particularly when compared to abstinence rates prior to and following the intervention. These results provide evidence for the utility of a brief abstinence reinforcement procedure for reliably initiating drug abstinence.

Katz et al. (2002) obtained results similar to Robles et al. (2000) that demonstrated the utility of a BAT. They extended the findings of Robles et al. (2000) via systematic replication and by introducing two new payment conditions, each of which included subsequent payments for additional periods of brief drug abstinence. Katz et al. (2000) used a within-subjects design in which participants experienced one of four conditions in counterbalanced order, each separated by 4 weeks of a "washout period," over 23 weeks. In addition to the control and single voucher conditions used in Robles et al. (2000), Katz et al. (2002) included both a continuous and an interrupted voucher schedule, each of which offered reinforcers over more prolonged time periods ($300 total) compared to Robles et al. (2000). All three experimental conditions produced significantly higher rates of abstinence than the control condition; additionally, the continuous and interrupted voucher conditions produced higher prolonged rates of abstinence than the control and single voucher conditions, neither of which differed in abstinence rate by the end of the 11-day experimental period. These results demonstrate that a BAT can not only initiate abstinence but that the initial abstinence experience can show influence on use in subsequent sessions during which response requirements are more difficult to reach.

Sigmon, Correia, and Stitzer (2004) examined the generalizability of repeated occurrences of abstinence initiation during a BAT to periods for which no incentives were available contingent upon drug abstinence. Their results further demonstrated the effectiveness of a BAT for reliably initiating cocaine abstinence among methadone patients. Participants in each of two experimental groups provided significantly more cocaine-negative urine samples on earning days compared to non-earning days.

Irons and Correia (2008) extended the application of a BAT to another pattern of drug use and to a different population. They used a 3-week ABA-design intervention in which 12 college students could earn a substantial monetary reward ($65) contingent on providing self-reports indicative of abstinence from smoking and negative urine (urinalysis) and breath (CO levels indicative of abstinence) samples of nicotine. Participants returned to the lab 1 week following an initial baseline phase for the BAT and again 1 week after that for a return to baseline session. Overall, both mean urinalysis and CO levels, as well as self-reported measures of abstinence, significantly decreased from baseline to the BAT, indicating the generalizability of a BAT to multiple drugs and to different populations. However, none of these measures returned to original baseline levels at the return to baseline session. Although the causal relation between the BAT and change in smoking behavior was confounded by the inconsistent rates of behavior across baselines, a failure to return to baseline rates of smoking may represent a clinically significant pattern of behavior resulting from the intervention. Being able to initiate continued lower levels of drug use is significant from a clinical perspective for which the goal of intervention is to reduce or eliminate drug use. As such, more research is

necessary to assess further utility of a BAT as a clinical intervention for initiating (and possibly maintaining) drug abstinence.

The current study

The purpose of the current study was to examine the potential viability of CM (via use of a BAT) for experimentally inducing caffeine abstinence long enough to induce peak experience of withdrawal symptoms (i.e., 2 days abstinence) among college students. Part of examining the viability of CM for caffeine use includes assessing the utility of an ELISA for verifying caffeine use in saliva: The current study therefore supplemented self-reports of caffeine use with saliva samples in order to examine the potential of verifying behavior with a biological measure. A secondary goal included examining the validity of novel measures of caffeine dependence and caffeine withdrawal. Based on previous research, I hypothesized that when offered incentives to abstain, caffeine consumption would decrease among participants. I also hypothesized that experience of caffeine withdrawal symptoms would be greater following abstinence relative to following regular consumption. Finally, I hypothesized that scores on a novel measure of caffeine dependence would be positively correlated with baseline levels of caffeine consumption.

Method

Participants

Inclusion criteria. Participants were 39 undergraduate students (M_{age} = 19.41, *SD* = 0.94, 74% female, 95% White) enrolled at JMU who were at least 18 years of age, self-reported consuming at least 25 mg caffeine per day (*M* = 122.73, *SD* = 116.08), and who did not currently take any prescription medications that contain caffeine during the study. A power analysis using G*Power revealed a minimum sample size of 7 to detect an effect using the effect size for CM (*d* = 0.58) obtained by Dutra et al. (2008) with moderate power set to .80 for $\alpha = .05$.

Recruiting procedures. Participants were recruited from the pool of respondents to a central screener that was emailed to the entire student body at James Madison University (JMU) during the fall semester. The central screener consists of 21 questionnaires about various health-related topics. Respondents were invited 40 to 60 at a time to participate in an enrollment session during which inclusion criteria were verified. During the enrollment session, those respondents that met all inclusion criteria were invited to participate in the current study.

Materials

Demographics questionnaire. A demographics questionnaire was used to assess demographic information of the participants, including age, gender, and race.

Daily Caffeine Consumption Questionnaire (DCCQ). This questionnaire was used to obtain participants' self-reported use of caffeine. Three researcher-generated items were included to characterize most recent consumption of caffeine. The DCCQ includes the Caffeine Consumption Questionnaire (Landrum, 1992), a measure developed

to precisely and consistently assess caffeine use among college students by accounting for the number of days and servings that an individual consumes various foods and beverages containing caffeine.

Autonomy Over Caffeine Scale (AUTOC). Currently there is no standard measure of dependence for caffeine. The AUTOC measure was adapted from the Autonomy Over Smoking Scale (AUTOS; DiFranza et al., 2009) that was developed from DSM-IV-TR (APA, 2000) criteria for smoking dependence. The AUTOC includes the same 12 items as the AUTOS except wording was altered from tobacco consumption to reflect caffeine consumption. The AUTOS is used to assess current tobacco withdrawal symptom intensity that represents autonomy, or lack thereof, from nicotine. Items are descriptions of experiences of smoking withdrawal symptoms to which the participant records how well each describes themselves; responses are recorded on 4-point Likert scales (0 = *not at all*, 3 = *very well*). Scores can range between 0-36, with higher scores corresponding to increased experience of withdrawal symptoms, cue-induced cravings, and psychological dependence. The AUTOS is highly internally reliable (α = .91 - .97) and is concurrently valid with Hooked On Nicotine Checklist scores (DiFranza et al., 2002) and DSM-IV TR (APA, 2000) diagnostic criteria for nicotine dependence.

Caffeine Withdrawal Scale (CWS). Currently there is no standard measure for assessing experience of symptoms of withdrawal from caffeine. This measure was adapted from the Wisconsin Smoking Withdrawal Scale (WSWS; Welsh et al., 1999) that was developed to assess severity and type of smoking withdrawal symptoms. The CWS includes 23 of the same 28 items as the WSWS except wording was altered from

descriptions of smoking withdrawal symptoms to reflect those of caffeine withdrawal, and five items relating to food consumption were removed. Items on the WSWS consist of statements related to clinical symptoms of smoking withdrawal (e.g., "I have felt impatient") and are rated on a 5-point Likert-type scale (1 = *strongly disagree*, 5 = *strongly agree*). The WSWS provides an overall withdrawal score [range = 0 (no withdrawal) – 140 (extreme withdrawal)] and scores for each of its 7 subscales, which reflect clinical symptoms of nicotine withdrawal: anger, anxiety, sadness, concentration, craving, sleep, and hunger (APA, 1994). Internal consistency for measuring overall withdrawal is high (α = .90 - .91), whereas subscale internal consistencies are slightly lower (α = .75 - .93). Additionally, overall measures on the WSWS are predictive of smoking cessation outcomes (Welsch et al., 1999).

Stimulant and Caffeine Experience of Withdrawal Symptoms (SCEWS). There are currently no standard criteria for measuring symptoms of caffeine withdrawal. This measure was created using summaries of symptoms of caffeine withdrawal reported by Julien (2008) and McKim (2007), as well as DSM-IV TR criteria for withdrawal from small doses of another relatively mild stimulant, amphetamine withdrawal syndrome (APA, 2000). Criteria for another stimulant (amphetamine) other than caffeine were used because the DSM-IV-TR does not include caffeine withdrawal symptoms.

Enzyme-linked immunosorbent assay (ELISA). A caffeine ELISA (Neogen Corporation, Lexington, KY) was used to validate self-reported measures of caffeine consumption by revealing the presence of caffeine metabolites in saliva samples. After samples are obtained, they will be frozen at -40°C until enough samples are collected to run a single ELISA (up to 45 samples, each in duplicate and including controls, can be

analyzed at once). Once enough samples have been collected, they are thawed and assayed. During an incubation period, samples are placed in dishes pre-coated with binding sites. Following incubation the dishes are washed and K-Blue® Substrate is added to the dish; the antibodies from the substrate and drug (i.e., caffeine) molecules compete for binding sites on the dish. Approximately 30 min after incubation with the substrate the samples may be analyzed using a spectrophotometer; color change is inversely related with the presence of the drug metabolite in the sample. Results are presented as a logarithmic function of light absorbance at 450 nm.

Design

The current study was an ABA reversal design. Following enrollment, participants experienced a baseline condition. All participants then experienced a BAT 2 days after the baseline session, followed by a return to baseline 2 to 4 days after the BAT session.

Procedure

All sessions occurred between 12:30 p.m. and 6:30 p.m. in a psychology research lab.

(A) Baseline 1/Enrollment. All participants earned a $10 voucher for attending and completing the Baseline 1/enrollment session. This session included verification of inclusion criteria (see above) by oral report, followed by informed consent procedures, and collection of self-report measures (DCCQ, AUTOC, CWS, and the SCEWS) and a saliva sample. Participants were informed that if they returned to the lab 2 days following this initial session and demonstrated evidence of abstinence (saliva sample indicative of abstinence AND self-report not consuming any caffeine) that they would earn $20, and

that if they returned to the lab 2 days following this initial session and provided biological and self-report measures but failed to demonstrate evidence of abstinence they would earn $2. Participants were told that any caffeine use would result in a failed biological test.

(B) Brief Abstinence Test (BAT). Participants returned to the lab 2 days following the Baseline 1 session and completed the self-report measures and provided a saliva sample. The researcher asked participants if they abstained from caffeine since Baseline 1 and then asked probing questions about the experience. Participants who demonstrated abstinence were provided a $20 voucher. Participants who did not demonstrate abstinence were provided a $2 voucher for attendance. Participants were then asked to return to the lab for a return to baseline session for which they could earn $5 for attendance, regardless of their caffeine consumption prior to the session, and a $10 bonus for completing all three sessions with perfect attendance.

(A) Baseline 2. A final session occurred 2 days following the BAT phase and it included completion of the self-report measures and a saliva sample. Participants earned $5 for attending the session and a $10 bonus for perfect attendance to all three sessions. Participants were able to earn a maximum of $45 if they attended all sessions and achieved abstinence criteria during the BAT. The time period for the study for individual participants was between 5 to 7 days (Baseline 2 occurred 2 to 4 days after the BAT, depending on the day of the week of the BAT). All participant payments were made in the form of vouchers until the end of the experiment so that the saliva samples could be collectively analyzed using the ELISA. Participants were then instructed to return to the lab 1 to 5 days after their final session (i.e., once the saliva samples have been analyzed)

to receive cash payment for all sessions in one sum. Research has shown high magnitude reinforcers are effective for changing drug use in a CM analog even at delays of 1 week (Packer, Howell, McPherson, & Roll, 2012).

Results

Thirty-nine of 42 participants who enrolled completed all three sessions of the experiment. One participant withdrew after completing the baseline session and re-entered later. Analyses excluded data from this participant's first baseline session as well as data from individuals who did not complete all three sessions. Two respondents who completed one session did not return for any further sessions and one respondent who completed two sessions did not return for the final session; data for these three respondents were not used in any analyses.

Abstinence during the BAT was defined as a verbal self-report of 0 caffeinated products consumed since the baseline session supplemented with responses to probing questions consistent with experience of withdrawal from caffeine (e.g., "I felt sleepier during classes than usual"). Percentage of light transmittance, as measured by the ELISA, was used as a supplement to self-reported caffeine for verifying caffeine abstinence. A spectrophotometer analyzed all saliva samples in duplicate; as such, values of percentage of light transmittance used for statistical analyses were mean values of each pair of duplicate samples. In most cases percentage of light transmittance appeared to support self-reports, though measurement error (e.g., bad wash during the rinsing process) from the ELISA overall was too substantial to be used alone as verification of caffeine abstinence. Well readings that seemed to be erroneous (e.g., two samples of one replicated sample were notably different) were not excluded in analyses.

See Table 1 for a summary of descriptive statistics for the dependent measures. See Table 2 for a summary of intercorrelations among the novel measures of caffeine dependence and withdrawal.

Primary analyses

Self-report. Among the 39 participants, 37 (95%) met self-report criterion for abstinence during the BAT and 18 (46%) during Baseline 2. A repeated-measures ANOVA revealed statistically significant differences in self-reported latency (hr) since last consuming caffeine, $F(1.05, 39.90) = 23.42$, $p < .001$, partial $\eta^2 = .38$. Planned contrasts revealed that latency since last consuming caffeine at the BAT ($M = 54.23$, $SD = 14.59$) and Baseline 2 ($M = 49.65$, $SD = 56.93$) sessions were significantly longer than for Baseline 1 ($M = 7.11$, $SD = 10.14$, ps $< .001$). Participants who met criteria for abstinence did not significantly differ from participants who did not meet criteria for abstinence at either BAT or Baseline 2 in terms of self-reported average daily caffeine consumption (ps $> .05$).

ELISA. Thirty-two of 39 participants (82%) met absolute reduction criterion for abstinence during the BAT using self-report and results from the ELISA (i.e., a reduction in caffeine use as indicated by the ELISA; no clear cutoff score was evident from the analyses nor existed in the literature for caffeine).

Twenty-four participants (62%) met reduction criterion during the BAT using duplicate samples *SD* (i.e., reduction of ≥ 1 *SD* for the mean difference between duplicate samples across all three sessions). An independent *t*-test revealed participants who met this reduction criterion from Baseline 1 to BAT significantly differed from participants who did not meet this criterion in light transmittance during the BAT, $t(36.76) = 4.03$, $p < .001$, $d = 1.20$, CI 95% [0.047, 0.142]. Another independent *t*-test revealed participants who met this reduction criterion from BAT to Baseline 2 did not significantly differ from

participants who did not meet this criterion in light transmittance during Baseline 2, $t(37) = 1.10$, $p = .280$, $d = 0.37$, CI 95% [-0.032, 0.108].

A repeated-measures ANOVA revealed statistically significant differences in light transmittance through the saliva samples among the three sessions, $F(2, 76) = 8.36$, $p = .001$, partial $\eta^2 = .18$. Planned contrasts revealed the BAT ($M = 79.58\%$, $SD = 9.23\%$) and Baseline 2 ($M = 80.16\%$, $SD = 10.49\%$) yielded significantly less light transmittance compared to Baseline 1 ($M = 85.57\%$, $SD = 6.38\%$, ps = .001 and .005, respectively). Neither percentage light transmittance nor latency since last consuming caffeine differed significantly between BAT and Baseline 2 sessions (ps = 1.0).

A median split of self-reported average daily caffeine consumption (median = 90 mg) yielded high and low caffeine users, neither of which differed in light transmittance at Baseline 1, BAT, or Baseline 2 (ps > .05). Participants in the upper and lower quartiles of self-reported average daily caffeine use did not significantly differ in light transmittance at Baseline 1, BAT, or Baseline 2 (ps > .05).

Pearson Product-Moment correlations revealed no significant correlations between light transmittance and self-reported latency since last consuming caffeine during Baseline 1 [$r(37) = -.02$, $p = .882$], the BAT [$r(37) = -.02$, $p = .919$], or Baseline 2 [$r(37) = -.22$, $p = .178$]. A point-biserial correlation revealed that light transmittance significantly negatively correlated with whether or not participants met criteria for abstinence from caffeine across all three study sessions such that likelihood of meeting criteria for abstinence from caffeine was negatively related with light transmittance, $r(117) = -.295$, $p = .001$.

Dependence analyses

AUTOC. Pearson Product-Moment correlations revealed AUTOC scores and CWS scores significantly correlated during Baseline 1 [$r(37) = .46$, $p = .003$], the BAT [$r(37) = .71$, $p < .001$], and Baseline 2 [$r(37) = .72$, $p < .001$]. Pearson Product-Moment correlations also revealed AUTOC scores and SCEWS scores significantly correlated during Baseline 1 [$r(37) = .37$, $p = .020$], the BAT [$r(37) = .75$, $p < .001$], and Baseline 2 [$r(37) = .64$, $p < .001$].

A repeated-measures ANOVA revealed that AUTOC scores significantly differed during the three study sessions, $F(1.66, 63.23) = 5.27$, $p = .011$, partial $\eta^2 = .122$. Planned contrasts showed AUTOC scores increased significantly from Baseline 1 ($M = 8.72$, $SD = 5.99$) to BAT ($M = 10.62$, $SD = 8.77$, $p = .023$), and declined slightly from BAT to Baseline 2 ($M = 10.28, 8.46$, $p = 1.0$). AUTOC scores did not differ significantly between Baseline 1 and Baseline 2 ($p = .098$).

Pearson Moment-Product correlations revealed AUTOC scores and percentage light transmittance were significantly negatively correlated during each Baseline 1 [$r(37) = -.34$, $p = .032$] and Baseline 2 [$r(37) = -.42$, $p = .008$], but were not significantly correlated during the BAT [$r(37) = -.11$, $p = .502$]. Additionally, Baseline 1 AUTOC scores were not significantly related with self-reported average daily caffeine consumption, $r(37) = .05$, $p = .802$. Using a median split of self-reported average daily caffeine consumption, high and low caffeine users did not significantly differ in AUTOC scores at Baseline 1, $t(37) = 1.67$, $p = .104$, $d = 0.55$, CI 95% = [-6.98, 0.68].

Withdrawal analyses

Pearson Product-Moment correlations revealed CWS and SCEWS scores significantly correlated during Baseline 1 [$r(37) = .36$, $p = .024$], the BAT [$r(37) = .67$, $p < .001$], and Baseline 2 [$r(37) = .58$, $p < .001$].

CWS. A repeated-measures ANOVA revealed that CWS scores did not significantly differ across the three study sessions, $F(2, 76) = 0.56$, $p = .576$, partial $\eta^2 = .014$. CWS scores slightly increased from Baseline 1 ($M = 64.38$, $SD = 6.77$) to BAT ($M = 65.26$, $SD = 8.82$) and declined slightly from BAT to Baseline 2 ($M = 65.15$, 8.98), though not back to Baseline 1 levels.

Using a median split of self-reported average daily caffeine consumption, high and low caffeine users did not significantly differ in CWS scores at BAT or Baseline 2 (ps > .05).

Independent *t*-tests revealed participants who met reduction criterion from Baseline 1 to BAT using duplicate samples *SD* significantly differed in CWS scores from participants who did not meet this criterion during the BAT, $t(37) = 2.76$, $p = .009$, $d = 0.93$, CI 95% [1.95, 12.81], and likewise from BAT to Baseline 2 during Baseline 2, $t(37) = 3.21$, $p = .003$, $d = 1.12$, CI 95% [3.23, 14.31].

Pearson Moment-Product correlations revealed CWS scores and percentage light transmittance were not significantly correlated during Baseline 1 [$r(37) = -.10$, $p = .546$] or the BAT [$r(37) = .129$, $p = .435$], but were significantly negatively correlated during Baseline 2 [$r(37) = -.33$, $p = .038$].

SCEWS. A repeated-measures ANOVA revealed that SCEWS scores significantly differed across the three study sessions, $F(2, 76) = 6.88$, $p = .002$, partial η^2

= .153. Scores on the SCEWS significantly increased from Baseline 1 (M = 3.05, SD = 3.04) to BAT (M = 5.00, SD = 4.48, p = .010) and significantly declined from BAT to return to baseline (M = 2.97, SD = 3.96, p = .012). SCEWS scores did not differ across the two baseline sessions (p = 1.0).

Using a median split of self-reported average daily caffeine consumption, high and low caffeine users did not significantly differ in SCEWS scores at BAT or Baseline 2 (ps > .05).

Independent t-tests revealed participants who met reduction criterion from Baseline 1 to BAT using duplicate samples SD did not significantly differ in SCEWS scores from participants who did not meet this criterion during the BAT, $t(37) = 1.03$, p = .310, d = 0.35, CI 95% [-1.47, 4.50], but SCEWS scores did significantly differ between these groups from BAT to Baseline 2 during Baseline 2, $t(13.71) = 2.64$, p = .020, d = 1.20, CI 95% [1.67, 6.48].

Pearson Moment-Product correlations revealed SCEWS scores and percentage light transmittance were not significantly correlated during Baseline 1 [$r(37) = -.19$, p = .248] or the BAT [$r(37) = -.02$, p = .895], but were significantly negatively correlated during Baseline 2 [$r(37) = -.46$, p = .003].

Discussion

The current study examined the potential efficacy of CM for initiating caffeine abstinence among college students of varying levels of typical caffeine consumption using a BAT, and to induce abstinence from caffeine long enough for participants to experience peak withdrawal symptoms so that novel measures of caffeine withdrawal and dependence could be examined. Results indicated the BAT was effective for initiating abstinence from caffeine among participants. A majority of participants (95%) met criteria for abstinence from caffeine when offered a high magnitude monetary incentive, and nearly half of all participants continued to abstain from caffeine at the return to baseline session. Scores on the novel measure of caffeine dependence, the AUTOC, unexpectedly varied across sessions contrary to what would be expected of levels of dependence over the course of approximately 1 week. AUTOC scores were not related to baseline levels of caffeine consumption. Scores on one novel measure of caffeine withdrawal, the CWS, did not systematically vary across sessions as predicted based on the apparent success of the BAT for initiating abstinence from caffeine and thus inducing withdrawal; however, scores on the other novel measure of caffeine withdrawal, the SCEWS, did vary across sessions as predicted. SCEWS scores differed across sessions as a function of whether participants met criteria for abstinence from caffeine whereas CWS scores did not.

Aggregated results from an ELISA matched aggregated self-reported levels of abstinence throughout the study, though results for individual samples sometimes varied nonsystematically from self-reports. The use of an ELISA for providing a biological measure of caffeine use was an improvement upon previous CM of caffeine studies

which only used self-report measures to verify caffeine use (e.g., Foxx & Rubinoff, 1979); however, because individual data from the ELISA were sometimes incongruent with self-reports it is unclear how well this technology measures caffeine use for the purposes of the current research design. Results of the ELISA were moderately inconsistent within and between samples. Individual factors (e.g., metabolic rate, caffeine use prior to experimental time periods) possibly influenced ELISA results, though how and to what degree are not clear and warrant further research. As such, ELISA results themselves were not sufficient as a criterion for determining caffeine use in the current study. Further research is warranted to examine exactly how these factors influence the ELISA so that criterion values or percent change may be established to discern caffeine abstinence over a certain period of time. Additionally, future research on caffeine consumption would benefit from advances in technology for determining levels of caffeine use similar to simple techniques available for smoking (e.g., urinalysis), alcohol (e.g., breathalyzer), and other drugs. Such a technology would not only aid in caffeine research but could also be used as a medical tool for screening caffeine consumption as a potential contributing factor for numerous health-related problems (e.g., anxiety symptoms, insomnia, or headaches for which there is no clear underlying cause).

Caffeine consumption reduced during the BAT but did not increase to baseline levels during the return to baseline. Although this is a challenge to inferring a causal relation, it is a clinically preferred outcome relative to observing a complete return to baseline levels of behavior and one that has been reported using a BAT in the literature (Irons & Correia, 2008). That is, to observe the success of the BAT coupled with the continued behavior change in the desired direction (i.e., a reduction in drug use) in a

clinical setting would likely be considered a success. Because no participants met criteria for abstinence at baseline and 95% of participants met criteria for abstinence during the BAT, it is unlikely that the phase changes did not exert experimental control. Despite the apparent effectiveness of the intervention, the lack of a return to baseline levels of behavior confounds a causal relation between the BAT and the change in caffeine consumption. Participants may have continued to remain abstinent from caffeine because they encountered the appetitive effects of abstinence such as the cessation of experience of withdrawal symptoms. Future research on the BAT could use multiple baseline designs or between-subjects designs to discern the apparent causal relation between the BAT and changes in caffeine consumption.

If participants continued to abstain from caffeine use due to encountering the appetitive effects of abstinence then a BAT may be an effective but also sufficient treatment method for reducing caffeine consumption. Unlike other drugs (e.g., nicotine) cue-induced relapse for caffeine use is likely not an important post-intervention risk. Although past researchers (e.g., James, Stirling, & Hampton, 1985) suggested treatment for reducing caffeine use should fading components to avoid initiating aversive withdrawal symptoms, the observed results indicate that a single contingency is sufficient for initiating reduced caffeine consumption. Thus a BAT may be a more cost-effective treatment option for problematic caffeine consumption than a fading procedure or a large-scale CM intervention. However, the long-term implications of reduced caffeine use among individuals for whom caffeine consumption is problematic are not clear. Given similarities in caffeine consumption and use of other drugs it is unlikely that the long-

term benefits of reduced caffeine use would be less than the cost of implementing BATs to induce the change in consumption.

Participants did not receive payment until 2 to 6 days following their final session. Delay of delivery of contingencies did not appear to influence behavior to a greater degree than did the experimental design. These results are consistent with those of Packer, Howell, McPherson, and Roll (2012), who found that high magnitude reinforcers can cause reduced drug use even when delivered at delays of up to 1 week. Two participants did not meet criteria for caffeine abstinence during the BAT. One participant reported that they forgot about the instructions given during Baseline 1. The other participant who did not abstain from caffeine during the BAT reported they "needed caffeine" because they had a test the morning of the day of the BAT session. Despite the fact that these observed limitations were beyond experimental control, the intervention achieved the primary goal of the study as evident by the majority of the sample who met criteria for abstinence from caffeine. Future research on CM of caffeine consumption could control for these specific issues by manipulating saliency of the contingencies (e.g., sending verbal reminders via text message or E-mail).

The novel measure of caffeine dependence and one of the novel measures of withdrawal (CWS) were developed from analogous measures for smoking. No such measures exist for caffeine; smoking measures were adapted for caffeine because caffeine consumption is moderately analogous to cigarette smoking (legal, occurs over the course of the day, effects are relatively mild, CM works for both). AUTOC scores differed across experimental phases contrary to what would be expected of drug dependence over such a brief period of time (e.g., cigarette dependence; DiFranza et al.,

2009). Additionally, baseline AUTOC scores were not significantly related to self-reported average daily caffeine consumption contrary to what would be expected based on previous attempts to measure caffeine dependence (Hughes et al., 1998; it is important to note here that self-reported average daily caffeine consumption did not appear to vary meaningfully in any statistical analyses using any variables in the current study). Thus the AUTOC either does not effectively measure caffeine dependence or caffeine dependence may change rapidly relative to dependence for other drugs such as nicotine (DiFranza et al., 2009). Given the sparse literature on measuring dependence for caffeine further research is necessary to define the construct. Stronger validity evidence may be gathered for the AUTOC once the construct of interest is more clearly defined. Additionally, the AUTOC may be adapted to more validly measure caffeine dependence: Items on the AUTOC were derived from a measure of smoking dependence, a construct that may not be as similar to caffeine dependence as originally hypothesized.

The majority of participants met criteria for abstinence during the BAT, which allowed for caffeine withdrawal to be examined. CWS scores did not differ across sessions as would be expected given the majority of participants experienced abstinence (and thus withdrawal) at least once during the study. As predicted, SCEWS scores significantly differed during the BAT when experience of abstinence was greatest among the sample. However, CWS and SCEWS scores significantly correlated at all three time points. The CWS was derived from a measure of smoking withdrawal that assessed responses to experience of symptoms of withdrawal on a 5-point Likert scale. The SCEWS was derived from lists of common symptoms of caffeine withdrawal in two drugs text books to which respondents answer yes or no as to whether they were currently

experiencing the symptoms. Interestingly, CWS scores significantly differed at both the BAT and return to baseline between participants who reduced and did not reduce caffeine use as evident by reduction of light transmittance by at least one SD derived from duplicated saliva samples across all three sessions. SCEWS scores only significantly differed at Baseline 2. It is conceivable that both the CWS and the SCEWS measure experience of caffeine withdrawal to varying degrees such that the SCEWS may provide more valid responses than the CWS. The current study provides preliminary evidence for the utility of the CWS, and SCEWS; however, further validity evidence is necessary (e.g., using large sample sizes than the one used in the current study) before either the CWS or the SCEWS may be used for clinical purposes.

It is important to develop sound measures of caffeine dependence and withdrawal so that these constructs may be validly and reliably measured. Despite previous attempts to characterize these constructs (Hughes et al., 1998; Juliano & Griffiths, 2004), currently no such measures exist for caffeine dependence or withdrawal. Thus it is difficult to determine the extent and severity of these phenomena. Likewise the potential utility of a BAT may be fully realized only after problematic caffeine consumption (e.g., incidences of caffeine dependence) is better characterized in order to identify individuals for whom treatment would be beneficial. Additionally, valid measures of caffeine dependence and withdrawal would allow for caffeine to serve as a model for studying dependence and withdrawal phenomena in other drugs.

Given the promising results observed in the current study, future research should examine CM of caffeine over a longer time period. Because caffeine consumption can be experimentally manipulated, CM of caffeine may be useful for not only initiating but also

reinforcing continued abstinence from caffeine beyond experience of peak withdrawal symptoms, although the current results indicate such generalization of treatment outcomes may supersede the need for continued reinforced abstinence. Long-term compliance with the dietary changes associated with reduced caffeine use may be optimally pursued through such methods (Griffiths & Reissig, 2008). Additionally, the overall success observed in the current study using a sample of users of a wide range of caffeine suggests similar research may be fruitful among other populations of interest (e.g., users of high levels of caffeine, users advised to avoid caffeine). Specific aspects of the BAT (e.g., payment amount) could be manipulated to determine optimally cost-effective conditions for implementing CM for caffeine.

The current study showed caffeine use can be experimentally manipulated by a CM procedure similarly to how many other drugs have been shown to do (e.g., alcohol, Miller et al., 1974). These results indicate CM may be a viable avenue for treating problematic caffeine consumption. Novel measures of caffeine dependence and withdrawal provide a preliminary attempt to assess these phenomena. It is hopeful that further research on treatment of caffeine consumption will allow for the measurement of caffeine dependence and withdrawal so that individuals who need treatment may be identified and have an empirically-based solution via CM.

Table 1

Descriptive Statistics for Dependent Measures

	Baseline 1		BAT		Baseline 2	
	M	*SD*	*M*	*SD*	*M*	*SD*
Caffeine Latency (hr)	7.11	10.14	54.23	14.59	49.65	56.93
% Light Transmittance	85.57	6.38	79.58	9.23	80.16	10.49
AUTOC	8.72	5.99	10.62	8.77	10.28	8.46
CWS	64.38	6.77	65.26	8.82	65.15	8.98
SCEWS	3.05	3.04	5.00	4.48	2.97	3.96

Note. $N = 39$. Caffeine Latency (hr) = self-reported latency since last consuming caffeine in hours. AUTOC scores can range from 0 to 36 with higher scores indicative of greater caffeine dependence. CWS scores can range from 23 to 115. SCEWS scores can range from 0 to 19.

Table 2

Summary of Intercorrelations for AUTOC, CWS, and SCEWS

Measure	*1*	*2*	*3*	*4*	*5*	*6*	*7*	*8*	*9*
1. AUTOC1	–								
2. AUTOC2	.91**	–							
3. AUTOC3	.87**	.94**	–						
4. CWS 1	.46**	.52**	.60**	–					
5. CWS 2	.56**	.71**	.77**	.75**	–				
6. CWS 3	.52**	.59**	.72**	.73**	.85**	–			
7. SCEWS 1	.37*	.25	.34*	.36*	.29	.37*	–		
8. SCEWS 2	.67**	.75**	.78**	.38*	.67**	.60**	.52**	–	
9. SCEWS 3	.57**	.50**	.64**	.50*	.40*	.58**	.51**	.52**	–

Note. Intercorrelations for novel measures of caffeine dependence and withdrawal (N = 39) at Baseline 1(1), BAT (2), and Baseline 2 (3).
*p < .05. **p < .01.

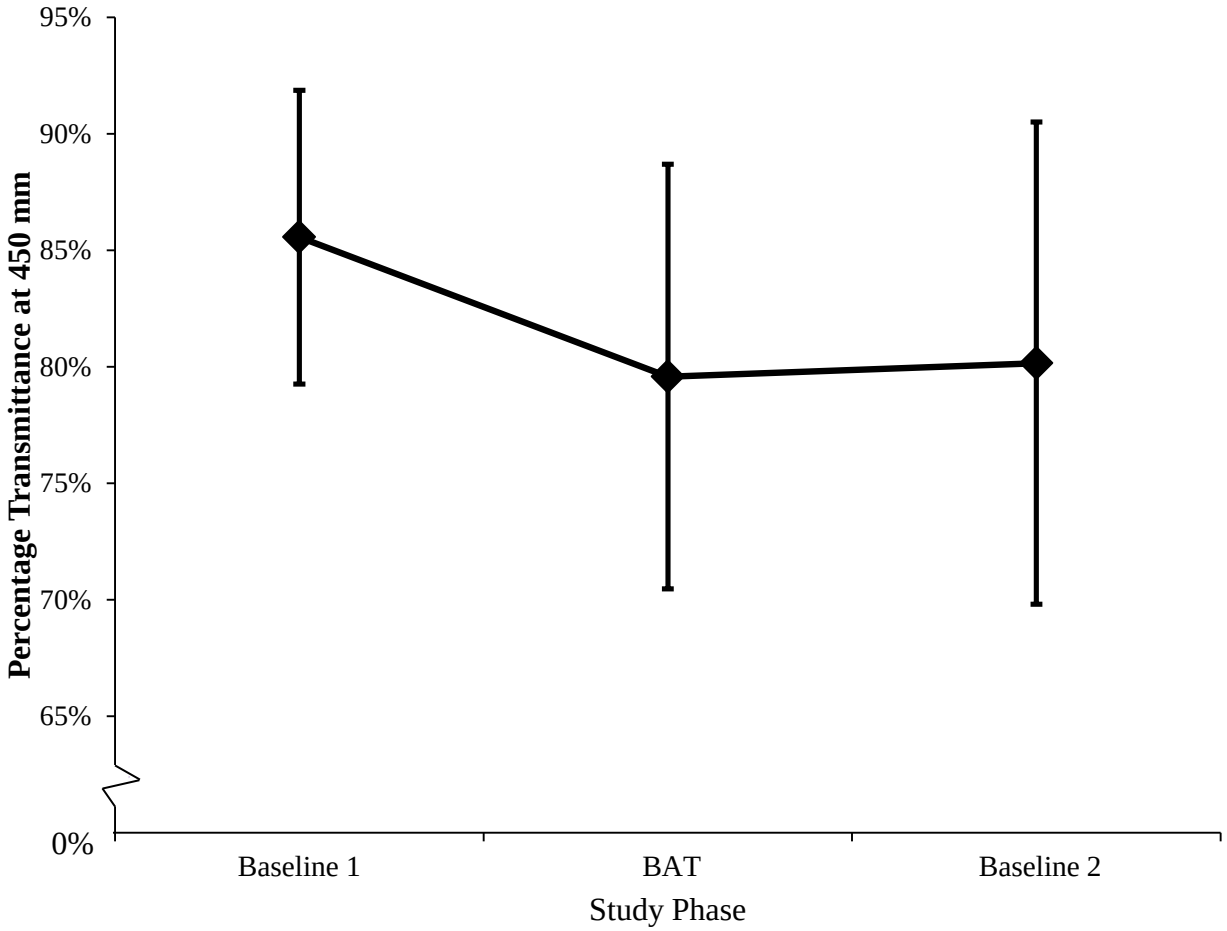

Figure 1. Percentage of light transmittance at 450 nm. Lower levels of transmittance are associated with lower levels of caffeine use.

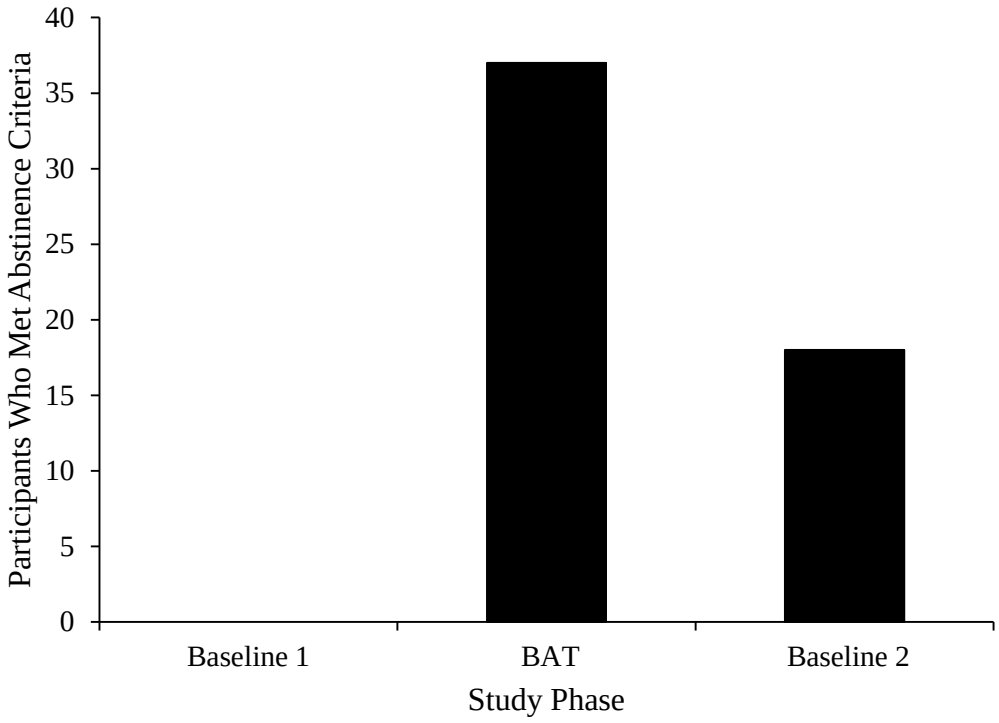

Figure 2. Number of participants who met criteria for abstinence during each study phase. No participants met criteria during Baseline 1, 37 participants met criteria during the BAT, and 18 participants met criteria during Baseline 2 (N = 39).

Appendix A

Informed Consent
Contingency Management of Caffeine Consumption Cessation (IRB#12-0199)

You may be eligible to participate in a study on caffeine consumption behavior. This study is being conducted Dr. Jessica Irons, an assistant professor of psychology at James Madison University along with Brad Joachim, a graduate student in psychology. Your responses to a recent web-based survey indicate that you are a student at James Madison University and you consume at least 25 mg caffeine per day. If either of these is incorrect, please tell the researcher at this time.

Participation in this study is voluntary. If you decide to participate, you will sign this informed consent form. Please ask any questions before you sign. If you choose to participate in this study, you will complete several questionnaires and answer some questions about your caffeine consumption today. You will also be asked to provide a saliva sample, which is necessary to verify that your caffeine consumption level meets a required level to participate in this study. This intake session will take approximately 20 minutes of your time, and you will be compensated for your participation with monetary payment ($10).

The remainder of the study will last approximately 1 week. During the remainder of the study you will be asked to attend the lab two more times in addition to today. During each of these visits, you will be asked to provide a saliva sample and to complete a brief self-report measure. Each of these visits will take approximately 15 minutes and will not exceed 30 minutes. Depending on the session, you may earn access to money depending on your caffeine consumption behavior; however, you do NOT have to change your caffeine consumption behavior to be enrolled in this study or to earn money in this study (i.e., you may earn some money regardless of your caffeine consumption).

You will be compensated for your participation with monetary payment. Payments will be made in lump sum following sample analysis, which will occur 1 – 3 days after the last session of the study. Payment amounts will vary by session. In total, you may be able to earn up to $45.00 over the course of the study. You will be compensated for attendance, regardless of your caffeine consumption behavior. You are NOT required to change your caffeine consumption behavior to participate in this study. If you choose to change your caffeine consumption behavior, you may earn access to payment as a result. In the event that you miss one scheduled session, you will be contacted to reschedule an appointment. Should you miss more than two appointments throughout the study, you will be removed from the study.

All responses will be confidential, and to maintain your privacy, your questionnaires will be labeled by a code number that we assign to you. Your name will not be linked to your data in any way. The master code list will be kept in a locked filing cabinet separate from the data that is accessible only to the researcher. The master code list will be destroyed after data analysis is complete. This informed consent will be destroyed after 3 years.

The risks of participating in this intake session are minimal. You may find answering questions about your use of caffeine distressing. Abstinence from caffeine consumption may be associated with mild symptoms such as headache, drowsiness, fatigue, irritability, or negative mood state. Breaches of confidentiality are highly unlikely because your identifying information will be kept separately from the questionnaires that you complete, and your questionnaires will be identified by your code number. You have the option to withdraw your consent to participate at any point of this session. If you decide to withdraw from the study you will not be penalized.

The direct benefit to you, the participant, is the opportunity to qualify for a study in which you will have the opportunity to engage in brief caffeine consumption abstinence, and you may be able to use the study as a way of initiating longer term caffeine consumption abstinence. Potential to engage in brief substance is predictive of successful longer term abstinence in the future. We cannot promise you that you will achieve either short- or long-term abstinence from caffeine consumption. Your participation has the potential to benefit the public by helping psychologists develop better methods of substance use cessation.

Information collected through your participation may be published in a professional journal, and/or presented at a professional psychology conference. However, your name and any other identifying information will not be associated with the data collected, and you will thus remain confidential.

_____ Participant's Initials

Your decision whether or not to participate will not jeopardize your future relations with James Madison University, or the Department of Psychology. If you have any further questions or would like to receive a copy of the final aggregate results of this study, please contact Dr. Jessica Irons (568-6565, ironsjg@jmu.edu, Department of Psychology).

For more information regarding your rights as a research participant you may contact the James Madison University IRB Chair, Dr. David Cockley (540-568-2834, cocklede@jmu.edu).

HAVING READ THE INFORMATION PROVIDED YOU MUST DECIDE WHETHER OR NOT YOU WISH TO PARTICIPATE IN THIS INTAKE SESSION. YOUR SIGNATURE INDICATES YOUR WILLINGNESS TO PARTICIPATE.

By signing this form, I certify that I am at least 18 years of age.

_____		_____	
Participant's signature	Date	Investigator's signature	Date
_____		_____	
Participant's printed name		Co-investigator's signature	Date

Appendix B

Demographics Questionnaire

Please answer the following questions as completely as you can to the best of your ability.

Gender: ___ Female ___ Male
Age: _____ years
Race: (select all that apply)
_____Asian
_____Black or African American
_____White
_____Hispanic or Latino
_____American Indian or Alaska Native
_____Native Hawaiian or Other Pacific Islander
_____Multi-ethnic
_____Other

How many years of school have you completed (e.g., graduated high school = 12 years)?
_____

How regularly do you smoke (answer in days per week)?
_____

Do you suffer from any cardiovascular health problems?
Yes _____ No _____

Do you suffer from any medical conditions?
Yes _____ No _____

Do you have any diagnosed psychological/psychiatric conditions?
Yes _____ No _____

Do you currently take any prescription medications (please do not include birth control when answering this question)?
Yes _____ No _____

Do you take any recreational drugs other than alcohol or nicotine?
Yes _____ No _____

Are you currently pregnant or attempting to get pregnant?
Yes _____ No _____

How regularly do you exercise (please answer in number of days per week)?

_____ days per week

Are you particularly sensitive or allergic to the effects of caffeine?
Yes _____ No _____

Have you ever been advised to avoid caffeine?
Yes _____ No _____

What is your height (report in inches)?
_____ inches

What is your weight?
_____ lbs.

Please indicate how stressful you feel your daily life is on average (scale of 1-10, 1 = not stressed at all, 10 = completely stressed):
_____

On average, how much expendable money (i.e., money that you can spend for personal reasons – not bills, insurance, etc.) do you have *per month*?

$_____

Are you willing to be contacted about opportunities to participate in future studies about caffeine consumption where you would have the opportunity to earn money or gift certificates and coupons? ____ Yes **_____** No

If yes, are you willing to provide biological samples to verify your level of caffeine consumption? These measures will be used ONLY to assess caffeine consumption and ALL results will remain confidential. ____ Yes **_____** No

Appendix C

Daily Caffeine Consumption Questionnaire (DCCQ)

What is your most consumed source of caffeine? (e.g., coffee, tea, soft drinks, energy drinks, chocolate, caffeinated gum)

_____

How long has it been since you last consumed a caffeinated product?

_____

How much caffeine did you consume then? (e.g., 2 cups of coffee, a 20 oz. bottle of soda, a large iced tea from a restaurant)

_____

Appendix D

Autonomy Over Caffeine Scale (AUTOC)

Please indicate to what degree the following statements describe you:	Not at all	A little	Pretty well	Very well
1. When I go too long without a caffeinated product, I get impatient.				
2. When I got too long without a caffeinated product, I get strong urges that are hard to get rid of.				
3. When I go too long without a caffeinated product, I lose my temper more easily.				
4. When I go too long without a caffeinated product, I get nervous or anxious.				
5. I rely on caffeinated products to focus my attention.				
6. I rely on caffeinated products to take my mind off being bored.				
7. I rely on caffeinated products to deal with stress.				
8. I would go crazy if i couldn't have a caffeinated product.				
9. When I feel stressed, I want a caffeinated product.				
10. When I see other people consuming caffeinated products, I want to do so as well.				
11. When I smell a caffeinated product, I want to consume a caffeinated product.				
12. While eating, I want to consume a caffeinated product.				

Appendix E

Caffeine Withdrawal Scale

Please answer the following questions based on how you have felt or what you have noticed [over the last 24 hours/over the last week]. Answer based on how you have felt in general during this time:	Strongly Disagree	Disagree	Neither Agree nor Disagree	Agree	Strongly Agree
1. I am getting restful sleep					
2. I have been tense or anxious.					
3. My level of concentration is excellent					
4. I awaken from sleep frequently during the night.					
5. I have felt impatient.					
6. I have felt upbeat and optimistic.					
7. I have found myself worrying about my problems.					
8. I have had frequent urges to consume caffeinated products.					
9. I have felt calm lately.					
10. I have been bothered by the desire to consume caffeinated products.					
11. I have felt sad or depressed.					
12. I have been irritable, easily angered.					
13. I have been bothered by negative moods such as anger, frustration, and irritability.					
14. I am satisfied with my sleep.					
15. I have felt frustrated.					
16. I have felt hopeless or discouraged.					
17. I have thought about consuming caffeinated products a lot.					
18. I feel that I am getting enough sleep.					
19. It is hard to pay attention to things.					
20. I have felt happy and content.					
21. My sleep has been troubled					
22. I have trouble getting caffeinated products off my mind.					
23. It has been difficult to think clearly.					

Appendix F

Stimulant and Caffeine Experience of Withdrawal Symptoms

Indicate whether you have recently (i.e., in the previous couple hours) experienced the following more so than you normally may experience each:	Yes	No
1. Headache		
2. drowsiness		
3. fatigue		
4. negative mood state		
5. impaired intellectual performance		
6. impaired motor performance		
7. difficulty with concentration		
8. decreased energy		
9. weakness		
10. lethargy		
11. decreased motivation for work		
12. impaired concentration		
13. increased irritability		
14. flu-like symptoms (aches, muscle stiffness, hot/cold spells, nausea, heavy feelings in the limbs)		
15. vivid, unpleasant dreams		
16. insomnia or hypersomnia		
17. increased appetite		
18. psychomotor retardation or agitation		
19. experience of one of the above has caused significant distress or impairment in social, occupational, or other important areas of functioning		

Appendix G

Debriefing Statement

The purpose of this study was to examine an intervention designed to help individuals reduce their caffeine consumption. If participation in this study caused you any discomfort or distress, you should visit one of the following:

- Your personal general practitioner.
- University Health Center located next to Burruss Hall, at the corner of Mason and Grace St, adjacent to Rockingham Memorial Hospital, (540.568.6178; http://www.jmu.edu/healthctr/)

References

Addicott, M. A., & Laurienti, P. J. (2009). A comparison of the effects of caffeine following abstinence and normal caffeine use. *Psychopharmacology, 207*, 423-431. doi:10.1007/s00213-009-1668-3

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

American Psychiatric Association. (2000). *Diagnostic and statistical manual of mental disorders* (4th ed., text rev.). Washington, DC: American Psychiatric Association.

Bernard, M. E., Dennehy, S., & Keefauver, L. W. (1981). Behavioral treatment of excessive coffee and tea drinking: A case study and partial replication. *Behavior Therapy,12*, 543-548.

Bernstein, G. A., Carroll, M. E., Thuras, P. D., Cosgrove, K. P., & Roth, M. E. (2002). Caffeine dependence in teenagers. *Drug and Alcohol Dependence, 66*, 1-6.

Bickel, W. K., Madden, G. J., & Petry, N. M. (1998). The price of change: The behavioral economics of drug dependence. *Behavior Therapy, 29*, 545-565.

Brenesova, V., Oswald, I., & Louden, J. (1975). Two types of insomnia: Too much waking or not enough sleep. *British Journal of Psychiatry, 126*, 439-445.

Bruce, M., Scott, N., Shine, P., & Lader, M. (1992). Anxiogenic effects of caffeine in patients with anxiety disorders. *Archives of General Psychiatry, 49*, 867-869.

Budney, A. J., Moore, B. A., Higgins, S. T., & Rocha, H. L. (2006). Clinical trial of abstinence-based vouchers and cognitive behavioral therapy for cannabis dependence. *Journal of Consulting and Clinical Psychology, 74*, 307-316.

Carlson, N. R. (2011). *Foundations of behavioral neuroscience*. Boston, MA: Allyn & Bacon.

Christopher, G., Sutherland, D., & Smith, A. (2005). Effects of caffeine in non-withdrawn volunteers. *Human Psychopharmacology, 20*, 47-53. doi:10.1002/hup.658

DiFranza, J. R., Savageau, J. A., Fletcher, K., Ockene, J. K., Rigotti, N.A., McNeil, A. D., … Wood, C. (2002). Measuring the loss of autonomy over nicotine use in adolescents: The DANDY (Development and Assessment of Nicotine Dependence in Youths) study. *Archives of Pediatric & Adolescent Medicine, 156*, 397-403.

DiFranza, J. R., Wellman, R. J., Ursprung, W. W. S. A., & Sabiston, C. (2009). The Autonomy Over Smoking Scale. *Psychology of Addictive Behaviors, 23*(4), 656-665. doi:10.1037/a0017439

Dutra, L., Stathopoulou, G., Basden, S. L., Leyro, T. M., Powers, M. B., & Otto, M. W. (2008). A meta-analytic review of psychosocial interventions for substance use disorders. *American Journal of Psychiatry, 165*, 179-187.

Erikson, G. C., Hager, L. B., Houseworth, C., Dungan, J., Petros, T., & Beckwith, B. E. (1985). The effects of caffeine on memory for word lists. *Physiology & Behavior, 35*(1), 47-51.

Evans, S. M., & Griffiths, R. R. (1999). Caffeine withdrawal: A parametric analysis of caffeine dosing conditions. *The Journal of Pharmacology and Experimental Therapeutics, 289*(1), 285-294.

Ford, R., Schulter, P., Mitchell, E., Taylor, B., Scragg, R., & Stewart, A. (1998). Heavy caffeine intake in pregnancy and sudden infant death syndrome: New Zealand cot death study group. *Archives of Disease in Childhood, 78*, 9-13.

Foxx, R. M. (1982). Behavioral treatment of caffeinism: A 40-month follow-up. *The Behavior Therapist, 5*, 23-24.

Foxx, R. M., & Rubinoff, A. (1979). Behavioral treatment of caffeinism: Reducing excessive coffee drinking. *Journal of Applied Behavior Analysis, 12*(3), 335-344.

Goldstein, A., Kaizer, S., & Warren, R. (1965). Psychotropic effects of caffeine in man: II. Alertness, psychomotor coordination and mood. *Journal of Pharmacology and Experimental Therapeutics, 150*, 146-151.

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

Greden, J. F., Fontaine, P., Lubetsky, M., & Chamberlin, K. (1978). Anxiety and depression associated with caffeinism among psychiatric inpatients. *American Journal of Psychiatry, 135*(8), 963-966.

Griffiths, R. R., & Reissig, C. J. (2008). Substance abuse: Caffeine use disorders. In A. Tasman, J. Kay, A. Lieberman, L. Kolb, M. First & M. Maj (Eds.), *Psychiatry* (pp. 1019-1040). Chichester, UK: John Wiley & Sons, Ltd. doi:10.1002/9780470515167.ch56

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

Harris, S., & Dawson-Hughes, B. (1994). Caffeine and bone loss in healthy post menopausal women. *American Journal of Clinical Nutrition, 60*, 573-578.

Hartmann, D. P., & Hall, R. V. (1976). The changing criterion design. *Journal of Applied Behavior Analysis, 9,* 527-532.

Higgins, S. T., Stitzer, M. L., Bigelow, G. E., & Liebson, I. A. (1986). Contingent methadone delivery: Effects on illicit-opiate use. *Drug and Alcohol Dependence, 17*(4), 311-322.

Hughes, J. R., & Oliveto, A. H. (1997). A systematic survey of caffeine intake in Vermont. *Experimental and Clinical Psychopharmacology, 5*(4), 393-398. doi:10.1037/1064-1297.5.4.393

Hughes, J. R., Oliveto, A. H., Liguori, A., Carpenter, J., & Howard, T. (1998). Endorsement of DSM-IV dependence criteria among caffeine-users. *Drug and Alcohol Dependence, 52*, 99-107.

Iguchi, M. Y., Belding, M. A., Morral, A. R., Lamb, R. J., & Husband, S. D. (1997). Reinforcing operants other than abstinence in drug abuse treatment: An effective alternative for reducing drug use. *Journal of Consulting and Clinical Psychology, 65*, 421-428.

Irons, J. G., & Correia, C. J. (2008). A brief abstinence test for college student smokers: a feasibility study. *Experimental and Clinical Psychopharmacology, 16*, 223-229. doi:10.1037/1064-1297.16.3.223

James, J. E. (2004). Critical review of dietary caffeine and blood pressure: A relationship that should be taken more seriously. *Psychosomatic Medicine, 66*(1), 63-71. doi:10.1097/10.PSY.0000107884.78247.F9

James, J. E., Stirling, K. P., & Hampton, B. A. M. (1985). Caffeine fading: Behavioral treatment of caffeine abuse. *Behavior Therapy, 16*, 15-27.

Jarvis, M. J. (1993). Does caffeine intake enhance absolute levels of cognitive performance? *Psychopharmacology, 110*, 45-52.

Juliano, L. M., & Griffiths, R. R. (2004). A critical review of caffeine withdrawal: Empirical validation of symptoms and signs, incidence, severity, and associated features. *Psychopharmacology, 176*(1), 1-29. doi:10.1007/s00213-004-2000-x

Julien, R. M. (2008). Caffeine and nicotine. In C. Advokat and J. Comaty (Eds.), *A primer of drug action* (pp. 479-511). New York, NY: Worth Publishers.

Katz, E. C., Robles-Sotelo, E., Correia, C. J., Silverman, K., Stitzer, M. L., & Bigelow, G. (2002). The brief abstinence test: Effects of continued incentive availability on cocaine abstinence. *Experimental and Clinical Psychopharmacology, 10*(1), 10-17. doi:10.1037//1064-1297.10.1.10

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

Lussier, J. P., Heil, S. H., Mongeon, J. A., Badger, G. J., & Higgins, S. T. (2006). A meta-analysis of voucher-based reinforcement therapy for substance use disorders. *Addiction, 101,* 192-203.

Miller, P. M., Hersen, M., Eisler, R. M., & Watts, J. G. (1974). Contingent reinforcement of lowered blood/alcohol levels in an outpatient chronic alcoholic. *Behaviour Research Therapy, 12*(3), 261-263.

Nawrot, P., Jordan, S., Eastwood, J., Rotstein, A., Hugenholtz, A., & Feeley, M. (2003). Effects of caffeine on human health. *Food Additives and Contaminants, 20*(1), 1-30. doi:10.1080/0265203021000007840

Oei, A., & Hartley, L. R. (2005). The effects of caffeine and expectancy on attention and memory. *Human Psychopharmacology, 20*, 193-202. doi:10.1002/hup.681

Ogawa, N., & Ueki, H. (2007). Clinical importance of caffeine dependence and abuse. *Psychiatry and Clinical Neuroscience, 61*, 263-268. doi:10.111/j.1440-1819.2007.01652.x

Packer, R. R., Howell, D. N., McPherson, S., & Roll, J. M. (2012). Investigating reinforcer magnitude and reinforcer delay: A contingency management analog study. *Experimental and Clinical Psychopharmacology*. Advance online publication. doi:10.1037/a0027802

Petry, N. M. (2000). A comprehensive guide to the application of contingency management procedures in clinical settings. *Drug and Alcohol Dependence, 58*, 9–25.

Prendergast, M., Podus, D., Finney, J., Greenwell, L., & Roll, J. (2006). Contingency management for treatment of substance use disorders: A meta-analysis. *Addiction, 101*, 1546-1560. doi:10.1111/j.1360-0443.2006.01581.x

Roache, J. D., & Griffiths, R. R. (1987). Interactions of diazepam and caffeine: behavioral and subjective dose effects in humans. *Pharmacology, Biochemistry, and Behaviour, 26*, 801-812.

Robles, E., Silverman, K., Preston, K. L., Cone, E. J., Katz, E., Bigelow, G. E., & Stitzer, M. L. (2000). The brief abstinence test: Voucher-based reinforcement of cocaine abstinence. *Drug and Alcohol Dependence, 58*, 205-212.

Rogers, P. J., Heatherly, S. V., Hayward, R. C., Seers, H. E., Hill, J., & Kane, M. (2005). Effects of caffeine and caffeine withdrawal on mood and cognitive performance degraded by sleep restriction. *Psychopharmacology, 179,* 742-751.

Roll, J. M., Higgins, S. T., & Badger, G. J. (1996). An experimental comparison of three different schedules of reinforcement of drug abstinence using cigarette smoking as an exemplar. *Journal of Applied Behavior Analysis, 29,* 495-205.

Siegel, S. (2005). Drug tolerance, drug addiction, and drug anticipation. *Current Directions in Psychological Science, 14*(6), 296-300.

Sigmon, S. C., Correia, C. J., & Stitzer, M. L. (2004). Cocaine abstinence during methadone maintenance: Effects of repeated brief exposure to voucher-based reinforcement. *Experimental and Clinical Psychopharmacology, 12*(4), 269-275. doi:10.1037/1064-1297.12.4.269

Silverman, K., Chutuape, M. A., Bigelow, G. E., & Stitzer, M. L. (1999). Voucher-based reinforcement of cocaine abstinence in treatment-resistant methadone patients: Effects of reinforcement magnitude. *Psychopharmacology, 146,* 128-138.

Silverman, K., Evans, S. M., Strain, E. C., & Griffiths, R. R. (1992). Withdrawal syndrome after the double-blind cessation of caffeine consumption. *New England Journal of Medicine, 327*(16), 1109-1114.

Smith, A. (2009). Effects of caffeine in chewing gum on mood and attention. *Human Psychopharmacology: Clinical and Experimental, 24,* 239-247. doi:10.1002/hup.1020

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

Stitzer, M. L., Bigelow, G. E., Liebson, I. A., & Hawthorne, J. W. (1982). Contingent reinforcement for benzodiazepine-free urines: Evaluation of a drug abuse treatment intervention. *Journal of Applied Behavior Analysis, 15*(4), 493-503.

Strain, E. C., Mumford, G. K., Silverman, K., & Griffiths, R. R. (1994). Caffeine dependence syndrome. Evidence from case histories and experimental evaluations. *Journal of the American Medical Association, 272*(13), 1043-1048.

Swift, C. G., & Tiplady, B. (1988). The effects of age on the response to caffeine. *Psychopharmacology, 94*, 29-31.

Terry, W. S., & Phifer, B. (1986). Caffeine and memory performance on the AVLT. *Journal of Clinical Psychology, 42*(6), 860-863.

Welsch, S. K., Smith, S. S., Wetter, D. W., Jorenby, D. E., Fiore, M. C., & Baker, T. B. (1999). Development and validation of the Wisconsin Smoking Withdrawal Scale. *Experimental and Clinical Psychopharmacology, 7*(4), 354-361.

Wesensten, N. J., Killgore, W. D. S., & Balkin, T. J. (2005). Performance and alertness effects of caffeine, dextroamphetamine, and modafinil during sleep deprivation. *Journal of Sleep Research, 14*, 255-266.

World Health Organization (1992). *International statistical classification of diseases and related health problems (10th ed.)*. Geneva: World Health Organization.

Yeomans, M. R., Ripley, T., Davies, L. H., Rusted, J. M., & Rogers, P. J. (2002). Effects of caffeine on performance and mood depend on the level of caffeine abstinence. *Psychopharmacology, 164*, 241-249. doi:10.1007/s00213-002-1204-1