

**The Effects of Caffeine on Cardiovascular Reactivity, Performance and Mood
during an Extended Stressor Task**

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University of Manitoba**

**A Thesis
Submitted to the Faculty of Graduate Studies
in Partial Fulfillment of the REquirements
for the Degree of
MASTER OF ARTS**

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PERFORMANCE, AND MOOD DURING AN EXTENDED STRESSOR TASK

BY

JAMES M. EDIGER

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Abstract

Recent research has consistently found that cardiovascular (CV) adjustments to stress are exacerbated by the prior consumption of caffeine. If experienced frequently, these exacerbations may increase an individual's risk of developing CV pathologies. Although most studies in the area have employed short-term laboratory stressors, it is likely that many individuals are exposed to prolonged periods of stress following caffeine consumption (e.g., in the workplace). The purpose of the present study was to determine whether caffeine's effects on CV stress reactivity are observable throughout the entire course of a prolonged laboratory stressor task. In addition, the study examined the effects of caffeine on subjects' performance of, and subjective responses to, the prolonged stressor task.

In a double-blind, randomized-factorial experiment, 16 male habitual caffeine users participated in four laboratory sessions. During these sessions, subjects rested or performed a stressor task for 48 m following the consumption of either 200mg caffeine or placebo. Indices of CV activity (heart rate, systolic, diastolic, and mean arterial blood pressure, and digital blood volume pulse), task performance (number of correct responses, number of errors, and mean response time), and affective state (positive and negative affect) were recorded throughout these sessions.

In general, the results indicated that caffeine increased blood pressure over placebo levels under both resting and stressful conditions and that these responses were maintained throughout the duration of the sessions. Prolonged stress, both by itself and in combination with caffeine, produced sustained increases in heart

rate and elevations in blood pressure, which decreased over time. No effects of caffeine on task performance were noted, but the drug was found to increase subjects' ratings of both positive and negative affect. These findings suggest that caffeine consumption prior to engaging in prolonged stressful activities may increase the risk of developing stress-related pathologies by exacerbating the physiological cost of stress for prolonged periods.

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The Effects of Caffeine on Cardiovascular Reactivity,
Performance, and Mood during an Extended Stressor Task

Thus we come to the [caffeine] paradox -- the question of how a drug so fraught with potential hazards can be consumed in the United States at the rate of more than a hundred billion doses a year without arousing the kind of hostility, legal repression, and social condemnation aroused by the illicit drugs. (p. 205).

(Brecher, 1972)

Although caffeine is generally regarded as a relatively harmless stimulant, controversy over its possible consequences for human health has existed almost since the introduction of caffeinated beverages. In North America, this controversy surfaced during the late 19th century following several reports of health problems resulting from coffee drinking (e.g., "Untitled Editorial," 1878). Since then, the popular and medical literatures have been saturated with numerous claims that habitual caffeine use constitutes a major etiological factor in a host of diseases and disorders (Troyer & Markle, 1984). Despite these assertions, caffeine has remained one of the most widely consumed drugs in the world.

On average, North Americans consume approximately 200mg of caffeine per person per day (Graham, 1978). The bulk of this caffeine is consumed in the form of caffeinated beverages including coffee, tea, caffeinated soft drinks, and cocoa (Graham, 1978; Shapiro, Lane, & Henry, 1986). In addition, a small amount of caffeine enters the North American diet through certain food products

such as chocolate (Wells,1984). Coffee, the primary source of caffeine for North Americans (Bonham & Leaverton, 1980), is made from the seeds of *Coffea Arabica* and related species and contains between 60mg and 150mg caffeine per 225ml serving (Dews, 1982; Ritchie, 1975). Cola drinks, made from the nuts of the tree *Cola Acuminata* constitute the second most important source of caffeine for North Americans, and typically contains 50mg caffeine per 360ml serving. Tea also contains approximately 50mg caffeine per serving (225ml) and is brewed from the leaves of *Thea Sinensis*. Finally, both cocoa and chocolate are derived from the seeds of *Theobroma Cacao* and contain 10mg and 6mg caffeine per 225ml and 30g serving, respectively.

The only other significant source of caffeine in North America comes from nonprescription medications (Graham,1978). Murray (1988) reports that over 100 nonprescription medications list caffeine as an active ingredient. The reason for the drug's inclusion in these products depends largely on the intended purpose of the medication. For example, many diet pills contain caffeine because of its diuretic effects. By inducing water excretion, caffeine reduces bloating, thus appearing to promote quick weight loss. Caffeine is also included in many headache and cold medications, where it is used as an analgesic and to counteract the sedative effects in "non-drowsy" formulations. Several medications developed to reduce menstrual symptoms also contain caffeine, both for its diuretic and analgesic effects. Finally, over-the-counter stimulants, such as "No Doze" and "Wake Ups" tablets contain large doses of caffeine to increase arousal and reduce fatigue. The amount of caffeine in these medications varies, but typically does not exceed 250mg per dose (Wells, 1984).

Several factors have been suggested to explain the high rate of caffeine consumption in North America. For example, although caffeine is a major constituent of many popular beverages and medications, its presence in these products may be largely unknown by the general population (Johnson-Greene, Fatis, Sonnek, & Shawchuck, 1988). As a result, many people may overuse the drug unintentionally. The high rates of caffeine use in North America may also reflect the fact that the consumption of caffeinated products is associated in our culture with desirable social consequences or events (Troyer & Markle, 1984). Indeed, the "coffee break" is generally regarded as a time for relaxed conversation and fellowship during the work day. Further, young people may view coffee and tea drinking as a sign of maturity, since children do not typically drink these beverages. Thus, young people may begin to drink these beverages in order to appear "grown up." Finally, high levels of caffeine intake may be maintained by some individuals as a means of avoiding unpleasant somatic sensations or cognitions (Graham, 1988). Habitual caffeine users, for example, may consume the drug in order to alleviate the headaches, muscle spasms, and heightened anxiety that accompany caffeine withdrawal (Greden, Victor, Fontaine, & Lubetsky, 1980).

Perhaps the most likely explanation for the overuse of caffeine in North America is that, in general, caffeine is assumed to influence behaviour in a positive way. Such an assumption has been promoted for the past several decades both by the caffeine-producing industries and by the medical profession. Early claims of caffeine's virtues include its proposed ability to "improve health," to increase "vitality and longevity," and to "spark intellect" (for a review, see Troyer

& Markle, 1984). More recently, caffeine has been reported to act on the central nervous system to promote clear thinking and improve cognitive processes (Ritchie, 1975).

Although several investigations indicate that caffeine can produce positive behavioural effects, much of this research is conflicting and equivocal. Furthermore, recent investigations suggest that when ingested in moderate to large doses, caffeine can have adverse effects on human behaviour. In the next section, research exploring both the positive and negative effects of caffeine on human behaviour will be reviewed.

Caffeine and Behaviour

Positive Behavioural Effects of Caffeine

Research dating back to the early 1900's suggests that caffeine can enhance performance in a variety of settings. In one of the first empirical investigations of caffeine's behavioural effects, Hollingsworth (1912) found that low doses of caffeine improved the psychomotor performance of subjects required to insert a stylus successively into three holes. At higher doses, however, subjects' performance on this task declined. Similar results have been reported more recently in studies employing reaction-time tasks. For example, Paroli (1972) found that while one cup of coffee (approximately 105mg of caffeine) increased rapidity and accuracy of motor behaviour of subjects engaged in a reaction-time task, two cups produced hand tremors that interfered with task performance. In contrast, Smith, Tong, and Leigh (1977) reported that performance on a choice reaction-time task was improved by 200mg caffeine, despite the occurrence of hand tremors. These authors noted that decision-time

scores (ie., the amount of time required to decide how to respond) were decreased following the caffeine, which may have resulted in an overall improvement in reaction time.

Performance on vigilance tasks has also been reported to be improved by caffeine. Baker and Theologous (1972) found that subjects engaged in a visual vigilance task analogous to night driving had significantly fewer lapses of attention following caffeine ingestion than following placebo. Similarly, Childs (1978) found that 400mg of caffeine improved performance on a visual target-scanning task in high-caffeine users compared to low users. Lieberman, Wurtman, Emde, Roberts, and Coviella (1987) recently found that small doses of caffeine (32mg - 250mg) significantly improved auditory vigilance scores in subjects required to identify signal tones among extraneous auditory stimuli. These authors further report that no aversive behavioural effects accompanied this improvement, suggesting that caffeine can enhance vigilance performance without producing the negative effects associated with higher doses.

In addition to enhancing psychomotor and vigilance performance, caffeine has also been found to improve performance on tests of mental ability (Paroli, 1972; Truitt, 1971). This effect may depend on the personality characteristics of the individual being tested, however (Sawyer, Julia, & Turin, 1982). For example, Revelle, Amaral, and Turiff (1976) measured the performance of introverts and extroverts on a verbal ability test under conditions of time stress. These authors found that 200mg caffeine improved the performance of extroverts, but had detrimental effects on the performance of introverts. Similarly Gilliland (1980) found that scores on Graduate Record Examination practice tests were

improved with increasing doses of caffeine for extroverts. Introverts, on the other hand, demonstrated reduced scores at higher doses. In general, these findings are consistent with Eysenck's (1962) prediction that stimulant drugs will alter performance in an introverting direction (ie., produce increased accuracy and performance). These findings suggest, however, that caffeine's effects on mental task performance may be beneficial only to individuals who do not already possess introverted personality characteristics.

Although studies investigating the effects of caffeine on performance have typically employed tasks of short duration, a growing body of literature suggests that caffeine's ability to enhance performance is more pronounced when exposure to the task is prolonged. In a review of the early literature on caffeine's behavioural effects, Weiss and Laties (1962) concluded that caffeine's ability to enhance performance is most apparent during later stages of an experiment when subjects have become fatigued. This conclusion was based, in part, on the results from studies conducted by Seashore and Ivy (1953) investigating the effects of caffeine under simulated battlefield conditions. These authors consistently demonstrated that caffeine was most effective in improving human performance in situations where performance was likely to deteriorate as the result of excessive work stress, boredom, or fatigue.

Caffeine's ability to alleviate fatigue has been well documented in studies investigating the drug's effects on sleep. In general, these studies indicate that moderate doses of caffeine can delay the onset of sleep and reduce the quality of sleep (Dews, 1982; Goldstein, Kaizer, & Whitby, 1969; Revelle, Humphreys, Simon, & Gilliland, 1980). The intensity of these effects appear to depend on

several factors, however, including personal history of caffeine use (Goldstein & Kaizer, 1969).

Caffeine may also increase physical endurance in tasks requiring prolonged muscular work (Ritchie, 1975; Truitt, 1971). Costill, Dalsky, and Fink (1972), for example, found that nine competitive cyclists took significantly longer to pedal to exhaustion on a bicycle ergometer following 330mg caffeine compared to placebo. Again, caffeine's effects on the performance of these tasks may depend on characteristics of the individual, such as level of physical fitness or gender (Perkins & Williams, 1975; Sawyer et al., 1982).

In addition to research involving tests of fatigue and endurance, studies employing extended vigilance and psychomotor tasks also suggest that caffeine's beneficial effects are more pronounced during later stages of an experiment. Regina, Smith, Keiper, and McKelvey (1974) found that performance on a simulated, but highly realistic, automobile driving task was significantly enhanced by 200mg caffeine. This effect was not apparent, however, until after the task was performed for 60 minutes. Similar results were reported previously by Hauty and Payne (1955), who found that caffeine improved visual vigilance and motor performance during the later stages of a three-hour perceptual-motor task. These effects deteriorated, however, when exposure to the task was extended for an additional four hours.

Caffeine's effects on extended task performance has also been investigated in studies using tests of mental ability. Buzzie (1986), for example, reported that performance on an extended subject-paced information processing task was improved by 150mg-450mg caffeine. However, these effects did not interact

with the effects of fatigue, suggesting that caffeine's ability to prolong performance may entail more than the simple restoration of fatigue-induced decrements in performance. In another study, Broverman and Casagrande (1982) found that 113mg caffeine impaired performance on a perceptual-restructuring (embedded figures) task when the task was novel. Once the task was rehearsed, however, caffeine tended to improve performance. Thus, caffeine may improve performance on these types of tasks only if the subject has had previous experience with the task and/or has become bored with it (Broverman & Casagrande, 1982; Loke & Meliska, 1984).

Negative Behavioural Effects of Caffeine

In 1974, Greden (1974) described three case histories in which clinical levels of anxiety resulted from the overuse of caffeine. In each case, the individual was seeking treatment for severe anxiety, restlessness, and nervousness. Although initially diagnosed with "anxiety disorder," a closer examination of the patients' dietary habits revealed that each had been ingesting between 1000mg and 1500mg of caffeine on a daily basis. Treatment consisted of restricting dietary caffeine and resulted in complete or near complete alleviation of all symptoms. In addition, when the subjects were later challenged with large doses of caffeine, the symptoms reoccurred, thus reinforcing the hypothesis that excessive caffeine intake produced the anxiety.

Although case studies describing caffeine-induced anxiety similar to those reported by Greden (1974) have appeared in the literature since the early 1900's, the American Psychiatric Association has only recently included caffeine toxicity, or "caffeinism" in its diagnostic manual (DSM III: American Psychiatric

Association, 1980). The DSM III describes caffeinism as severe anxiety resulting from the consumption of large amounts (usually more than 250mg) of caffeine. Symptoms of caffeinism include restlessness, nervousness, excitement, muscle twitches, insomnia, flushed face, diuresis, cardiovascular (CV) disturbances, rambling flow of thought or speech, and gastrointestinal complaints. Because these symptoms are similar to those associated with anxiety disorders, people experiencing caffeinism are often misdiagnosed. As a result, clear estimates of the prevalence of caffeinism are not available. Kaplan and Shadock (1981) have suggested, however, that at least one in ten people suffer from caffeinism.

In general, empirical research has supported the hypothesis that caffeine, when consumed in moderate to large doses, can produce clinical or near clinical levels of anxiety. For example, Velber and Templer (1984) had subjects rate their moods prior to and following the ingestion of either placebo, 150mg, or 300mg caffeine. Analysis of the mood ratings indicated that subjects receiving caffeine rated themselves as more anxious, depressed, and hostile than placebo controls. Further, the amount of caffeine consumed was a significant predictor of post-drug mood ratings. In an earlier study, Goldstein, Kaizer and Whitby (1969) found that subjects' mood ratings were differentially effected by caffeine depending on their history of caffeine use. In that study, habitual caffeine users and nonusers were given either caffeine (150mg or 300mg) or placebo. Mood ratings were obtained from the subjects at half hour intervals for a total of two hours following drug ingestion. The results suggested that caffeine produced an increase in anxiety in nonusers but decreased irritability and increased alertness in habitual users. When caffeine was withheld from the habitual users, however, ratings of

irritability and sluggishness, and reported incidence of headaches tended to increase.

Several survey studies provide evidence to suggest an association between caffeine use and level of anxiety. For example, Greden, Fontaine, Lubetsky, and Chamberlin (1978) surveyed the caffeine use habits of 83 psychiatric inpatients and found that both moderate and heavy caffeine users scored significantly higher on measures of state and trait anxiety than low users. In addition, 50% of the heavy users and 34% of the moderate users scored above 23 (severe) on the Beck Depression Inventory, compared to 17% of the low users. Similar results were reported by Gilliland and Andress (1981) using low, moderate, and heavy caffeine-using college students. These authors found that both moderate and heavy users scored significantly higher on measures of trait anxiety and depression than did low users. Further, moderate and heavy users also tended to have lower grade-point averages and higher frequencies of psychophysiological disorders compared to low users.

In a more recent study, James and Crosbie (1987) compared subjective reports of state and trait anxiety and depression among heavy caffeine users and moderately using psychiatric patients and college students. These authors found that while students scored significantly lower than psychiatric patients on measures of anxiety, the scores of heavy caffeine users were not different from those of patients. A similar pattern was also observed for measures of depression. These authors concluded that since psychiatric patients tend to score higher on subjective ratings of anxiety and depression, and since heavy caffeine users and

patients demonstrated a similar "psychological profile," heavy caffeine use may lead to poor psychological health.

In addition to increasing anxiety levels, caffeine has also been reported to affect other aspects of behaviour adversely. Indeed, several of the studies already discussed report that moderate to high doses of caffeine may impair psychomotor coordination, and induce feelings of depression and hostility. Investigations with psychiatric inpatients suggest that caffeine use may be associated with undesirable behaviors within this population. In one study (Podboy & Mallory, 1977), decaffeinated coffee was substituted secretly for regular coffee for a group of 15 severely retarded women. During a six-week period of decaffeinated coffee, staff reports of nocturnal wakings and aggressive outbursts decreased significantly. In a similar study, Defreitas and Schwartz (1978) found that staff ratings of hostility-suspiciousness, anxiety and irritability for 14 male inpatients were significantly lower during a three week period of decaffeinated coffee. As with the Podboy and Mallory study, decaffeinated coffee was substituted secretly for regular coffee so that neither the staff nor the patients were aware of the type of coffee they (the patients) were drinking. When regular coffee was reintroduced to the patients, these behaviours tended to increase, as did the incidence of manifest psychosis.

Overall, the literature suggests that caffeine, when ingested in moderate to large doses, may lead to undesirable changes in behaviour. Research also suggests that caffeine may play a central role in the etiology of certain disease states. For example, recent epidemiological studies have linked caffeine consumption with an increased incidence of pancreatic cancer (MacMahon, Yen,

Trichopolous, Warren, & Nardi, 1981) and cancer of the lower urinary tract (Cole, 1971; Morrison, Buring, & Verhock, 1982). Teratological investigations suggest that the use of caffeine during pregnancy may increase the risk of congenital birth defects (Collings, 1979; Sun, 1980). Although this evidence is inconclusive for humans, the Surgeon General of the United States has advised expectant mothers to avoid caffeinated products. Perhaps the most widely investigated health consequence of habitual caffeine use has been its association with CV diseases. In the next section, research linking caffeine to CV pathology will be reviewed briefly.

Caffeine and the Cardiovascular System

Caffeine and Cardiovascular Disease

The role of caffeine in the development of CV disease states has been debated in the epidemiological literature for the past two decades. For example, early case-control studies (Boston Collaborative Drug Surveillance Program, 1972; Jick, Miettinen, Neff, Shapiro, Heinonen, & Sloane, 1973) reported that the consumption of 1-5 and 6 or more cups of coffee per day was associated with an increased risk of myocardial infarction (MI) of approximately 1.4 and 2.2 times respectively. These findings were later disputed by Klatsky, Friedman, and Siegelau (1973) who found no association between coffee consumption and CV disease risk in a larger sample of MI patients. Since then, a number of case-control studies have reported both positive (La Vecchia, Gentile, Negri, Parazzini, & Franceschi, 1989; Rosenberg, Palmer, Kelly, Kaufman, & Shapiro, 1988; Rosenberg, Werler, & Kaufman, 1987; Wilhelmsen, Tibblin, Elmfeldt, Wedel, &

Werko, 1977) and negative (Hennekens, Drolette, Jesse, Davies, & Hutchison, 1976; Rosenberg, Slone, Shapiro, Kaufman, Stolley, & Miettinen, 1980) findings.

Prospective epidemiological research has also produced conflicting results with regard to caffeine's role in CV disease. For example, Dawber, Kannel, and Gordon (1974) found no significant association between coffee/caffeine consumption and the incidence of CV disease in a cohort of 4492 men and women during a 12 year follow up period. Coffee drinking was associated with an increased risk of "deaths from all causes," but this association was accounted for by the relationship between coffee drinking and cigarette smoking, itself a significant predictor of CV disease. Several other prospective studies have also failed to find a significant role for caffeine/coffee consumption in the development of CV disease (Murray, Bjelke, Gibson, & Schuman, 1987; Welin, Svardsudd, Tibblin, & Wilhelmsen, 1984; Yano, Rhoads, & Kagan, 1977).

Many of these studies have been criticized by LaCroix, Mead, Liang, Thomas, and Pearson (1986) for measuring the coffee-drinking habits of the subjects only once during the course of the investigation, and at a time remote from the coronary event. These authors suggest that changes over time in caffeine/coffee consumption and other important lifestyle variables (ie., alcohol or nicotine use) were not considered adequately in earlier investigations. In their study, LaCroix et al., (1986) repeatedly monitored the health and coffee-drinking habits of 1130 men for 19 to 35 years. Their results indicated that, on average, the consumption of 2-4 and 5 or more cups of coffee per day was associated with an increased risk of developing CV disease of 2.04 and 2.73 times, respectively. Further, these authors noted that the strongest associations were found when the

time between assessment of coffee drinking and the coronary event was shortest, suggesting that the adverse effects of caffeine/coffee on the CV system may be particularly evident within a short time period following the CV event. Since then, other prospective studies utilizing a short follow-up period have found similar results (LeGrady, Dyer, & Shekelle, 1987; Tverdal, Stensvold, Solvoll, Foss, Lund-Larsen, & Bjartveit, 1990).

In contrast to these findings, a recent large-scale prospective study by Grobbee, Rimm, Giovannucci, Colditz, Stampfer, and Willett (1990) found no evidence of caffeine/coffee relationship with CV disease states. In this study, the health of over 45,000 men was monitored approximately three years after an initial assessment of coffee drinking habits was made. Although these authors found no association between caffeinated coffee consumption and CV endpoints, they did discover a small but statistically significant relationship between decaffeinated coffee consumption and coronary heart disease. No clear explanation for this finding could be identified.

Overall, epidemiological research has provided no clear picture of the role of caffeine in the development of CV disease. However, the fact that a similar number of investigations have reported positive findings as have reported negative findings indicates that habitual caffeine use may not be totally innocuous. On the contrary, this body of literature suggests that caffeine may indeed be related to the development of CV disease states, but that the nature of this relationship is obscure and likely influenced by a number of factors including sex, age, and lifestyle habits.

Caffeine's Cardiovascular Effects

The link between caffeine and CV disease is strengthened by the fact that caffeine has a pronounced effect on all aspects of the CV system. Caffeine directly stimulates the myocardium, producing increases in heart rate (HR), contractility, and cardiac output (Rall, 1980; Ritchie, 1975). These effects may be masked in intact organisms, however, since caffeine also stimulates the medullary vagal nuclei, producing a decrease in cardiac activity (Stephenson, 1977). Further inhibition of caffeine's effects on the myocardium may result from the activation of the baroreceptor reflex circuit in response to increases in blood pressure (BP) levels (Shapiro et al., 1986). Thus, caffeine may produce a variety of myocardial responses including bradycardia, tachycardia, or no change in HR.

In addition to its myocardial effects, caffeine also influences the peripheral vasculature, producing vasodilation in coronary, pulmonary, and general musculature (Dews, 1982; Ritchie, 1975). At the same time, caffeine increases vascular resistance by direct stimulation of medullary vasomotor centres (Rall, 1980). Regardless of its effects in the periphery, caffeine causes a marked constriction of cerebral blood vessels and a significant decrease in cerebral blood flow (Rall, 1980).

Once ingested, caffeine is absorbed quickly into circulation, reaching peak plasma levels within 15-45m (Neims & von Borstel, 1983; Robertson et al., 1978). Although biotransformation rates vary between individuals, caffeine's metabolic half-life is estimated at 5-6h (Shapiro et al., 1986). Caffeine's primary CV effect is to produce an increase in resting BP of about 14/10mmHg

(Robertson et al., 1978). In addition, caffeine may increase circulating catecholamines, plasma renin activity, and respiration rate; factors which may influence its effects on CV activity.

Although the mechanisms mediating caffeine's influence on physiological processes are largely unknown, several lines of evidence suggest that caffeine's ability to block the actions of endogenous adenosine may underlie some of its physiological and behavioural effects. For example, systemically administered adenosine tends to have a sedative effect on behaviour. Thus, caffeine blockade of adenosine may produce apparent stimulation (Neims & von Borstel, 1983). Caffeine will also compete for occupancy of adenosine receptor sites, and the potency of caffeine in causing behavioural excitement correlates highly with its potency in blocking the binding of adenosine to neural membranes (Snyder, Katims, Annau, Burns, & Daly, 1981). In addition, chronic caffeine consumption has been found to increase neural adenosine receptor density as well as increase the sensitivity of these receptors to adenosine (Boulenger, Patel, Post, Parma & Manangos, 1983). Such effects may account for the phenomena of tolerance and withdrawal often associated with habitual caffeine use.

With regard to the CV system, adenosine is known to have at least three actions that may be responsible for its hypotensive effects (Verhaeghe, Vanhoutte, & Shepard, 1977). Adenosine can depress cardiac and vascular smooth muscle directly, attenuate the responsiveness of myocardial and smooth muscle cells to catecholamines, and act presynaptically to inhibit catecholamine release from sympathetic terminals. Caffeine, on the other hand, has been found to produce increased myocardial activity (Rall, 1980; Ritchie, 1975) and

enhanced catecholaminergic activity at CV alpha and beta receptors (Kalsner 1971; Kalsner, Frew, & Smith, 1975). Thus, caffeine may produce its CV effects by antagonizing the inhibitory action of endogenous adenosine on CV activity.

In addition to providing an explanation for some of caffeine's behavioural and physiological effects, the adenosine-blockade hypothesis also provides a theoretical basis for the link between caffeine and CV disease. Shapiro et al. (1986) suggest that by inhibiting the normal adenosine-mediated blockade of sympathetic nervous system (SNS) activity, caffeine may produce increases in resting sympathetic arousal. When SNS activity increases, such as during psychological stress, caffeine's enhancement of SNS activity may become even more pronounced. In this case, caffeine may exacerbate the effects of stress on CV and other stress response systems by eliminating the usual inhibitory effects of endogenous adenosine. Because physiological responses to stress are thought to play an important role in the etiology of CV diseases (Manuck & Krantz, 1986; Taggart & Carruthers, 1981), exacerbation of these potentially pathogenic effects by caffeine may further increase the risk for developing these pathologies. Opportunities for such exacerbations would be quite frequent, since research suggests that caffeine consumption is increased during stressful situations (Conway, Vickers, Ward, & Rahe, 1981). Thus, the link between caffeine and CV disease may involve caffeine's effects on CV reactivity to stress.

Caffeine and Stress

Perhaps the most convincing demonstration of caffeine's ability to enhance physiological responses to stress comes from animal research. In one study, Henry and Stevens (1980) observed mice living either peacefully as

siblings in boxes or interacting under conditions of chronic psychosocial stress in population cages. These cages were designed specifically to maximize contact between rival males, thereby producing a chronically stressful environment for the animals. Mice in both environments were given either plain water or water containing caffeine to drink. These conditions were maintained for three to five months, during which several measures of CV and adrenal-medullary activity were recorded. As expected, highly stressed animals demonstrated increases in plasma renin activity, arterial BP, adrenal weight, and plasma corticosterone levels compared to minimally stressed mice. However, the addition of caffeine to the animals' diet intensified these effects. Further, highly stressed mice receiving caffeine had a significantly higher monthly mortality rate and demonstrated more evidence of renal damage than the highly stressed mice receiving water. Regardless of living environment, caffeine tended to increase the animals' excitability and the intensity of their social interactions. Based on these findings, the authors concluded that chronic caffeine consumption enhances the physiological cost of psychosocial stress, thereby increasing the risk of stress-related disease states.

Although somewhat less dramatic, research with humans also suggests that caffeine can intensify physiological responses to stress. In an early study, Lane (1983) measured the CV responses of ten "caffeine-naive" subjects under conditions of relaxation and experimentally induced stress (ie., a mental arithmetic task). Subjects were tested on two separate occasions during which they received either 250mg caffeine or placebo prior to physiological recordings. The results indicated that both caffeine and stress independently produced

significant elevations in systolic and diastolic BP levels. When combined, the CV effects of caffeine added to those of stress so that BP responses to stress were significantly higher if the subject had ingested caffeine prior to the task. In contrast, caffeine had little or no effect on HR under either resting or stressful conditions.

Using a similar design, Lane and Williams (1985) investigated the effects of caffeine and stress on the HR, BP, and forearm bloodflow (FBF) of caffeine-naive males. As in the Lane (1983) study, caffeine was found to elevate resting systolic and diastolic BP levels but not HR. Again, this effect added to the BP elevations produced by stress. In addition, although caffeine did not influence resting FBF, it did potentiate the FBF response during the stressor. The authors suggest that this potentiated FBF response may represent a mechanism through which caffeine enhances the pathogenic effects of stress. They suggest that caffeine may augment the already inappropriately increased flow of oxygenated blood to peripheral vascular beds during periods of stress (i.e., cardiac-somatic decoupling; Obrist, 1981). This increase in blood flow may induce a compensatory autoregulatory response in the periphery which, if activated repeatedly, may result in chronic elevations in BP.

Research in this area has not been restricted to investigations of CV changes in caffeine-naive subjects. Subsequent studies have found that caffeine and stress produce cumulative increases in BP in habitual caffeine users (Lane & Williams, 1987; Ratliff-Crain, O'Keeffe & Baum, 1989), treated hypertensives (Goldstein & Greenberg, 1987), Chinese students (Yang, Greenstadt, & Shapiro, 1983), black males (Myers, Shapiro, McClure, & Daims, 1989), and both black

and white females (Strickland, Myers, & Lahey, 1989). In addition, some studies suggest that a family history of hypertension may influence caffeine's effects on CV reactivity. Lane and Williams (1987), for example, found that caffeine enhancement of FBF responses during stress were most pronounced in subjects with hypertensive parents. Similarly, Yang et al., (1983) reported that caffeine had a greater effect on BP levels in Chinese students with a positive family history of hypertension. In contrast, however, neither Lane and Williams (1985) nor Greenberg and Shapiro (1987) found evidence to suggest that family history of hypertension influences caffeine's effects on CV reactivity. Finally, the Type A behaviour pattern has also been investigated in studies of caffeine and stress (Lane & Williams, 1985; 1987; Yang et al., 1983). In general, differences in CV reactivity have not been found between Type A and Type B subjects within this context. Research in this area is somewhat limited, however, and further investigations are warranted.

Several recent studies have attempted to extend the findings of early caffeine-stress research by focusing on physiological measures other than HR or BP. For example, France and Ditto (1988) investigated the effects of caffeine on vascular activity under conditions of rest and stress. These authors found that under both conditions caffeine's effects on BP were mediated by an increase in vascular resistance. In contrast, Pincomb, Lovallo, Passey, and Wilson (1988) found that although caffeine's primary hemodynamic effect during rest was to increase vascular resistance, this effect completely disappeared when the subjects were faced with a stressful challenge. The cumulative effects of caffeine and stress on BP were mediated under these conditions by a caffeine potentiation of

stress-induced increases in HR, ejection acceleration, and stroke volume. Based on these findings, Pincomb et al.(1988) concluded that the effects of caffeine on CV functioning are dependent on the behavioural state of the individual. Under resting conditions, caffeine produces increased BP levels by increasing vascular resistance. If the individual is in a state of behavioural arousal, however, caffeine induced increases in BP are mediated by a potentiated cardiac output. More recently, France and Ditto (1992) also reported results which indicated that caffeine may potentiate cardiac output under conditions of stress. These authors contend, however, that caffeine may produce its pressor effects during stress through several CV adjustments including enhanced cardiac output and/or changes in vascular resistance.

In a somewhat different study, Lane, Adcock, Williams and Kuhn (1990) measured the effects of caffeine on both CV and neuroendocrine responses to a stressful challenge. As in previous studies, these authors found that caffeine and stress combined in an additive fashion to produce heightened levels of BP. In addition, the combination of caffeine and stress were found to produce elevations in circulating norepinephrine, epinephrine, and cortisone. It is interesting to note that these results are consistent with those reported by Henry and Steven (1980) in their research with chronically stressed mice.

Rationale of Present Study

Research investigating the combined effects of caffeine and stress clearly demonstrates that caffeine will increase CV responses to stressful challenges. The conclusion generally drawn from these findings is that habitual caffeine use may increase an individual's risk of developing CV disease states by exacerbating

the physiological cost of stress. This conclusion is based on the assumption that conditions set up in the laboratory approximate those that exist in the natural environment. However, an examination of the procedures used in most caffeine-stress studies to induce stress in subjects leads one to question the validity of this assumption.

In the majority of caffeine-stress studies reported to date, stress was induced by having subjects perform standard laboratory "stressor" tasks such as mental arithmetic. Although effective in producing large CV responses, such tasks bear very little resemblance to the stressors that people encounter in real life. Tasks such as mental arithmetic differ from naturally occurring stressors in at least two ways. The first difference is related to the nature of the task, or what the subject is asked to do. In general, mental arithmetic tasks require subjects to perform simple mathematical computations repeatedly without the aid of paper, pencil, or calculator under conditions of peer competition and/or time constraints. Although a few individuals may encounter similar situations during the course of work or study, most people are rarely faced with such a challenge.

A second difference between laboratory tasks and naturally occurring stressors is related to the duration of stress exposure. For the most part, subjects in caffeine-stress studies are asked to perform the stressor task for periods ranging from 5 to 15 m. Although the use of such short-term stressors may be necessary in some studies because of procedural constraints, experience suggests that under more natural conditions individuals are exposed to prolonged periods of stress following caffeine consumption. A typical example might be a data-entry clerk who drinks a cup of coffee prior to starting his or her work day. This individual

may be exposed to stressors associated with his or her job (eg., having to perform a largely repetitive and unstimulating task quickly while maintaining a high degree of accuracy in order to avoid reprimands from superiors) for several hours before he or she is allowed to take a break. Although the nature of the stressor may vary from job to job, for many, prolonged exposure to stress is a regular part of every working day.

Recently, two studies have attempted to validate the results of laboratory investigations under conditions that more closely approximate the natural environment. In the first study, Pincomb, Lovallo, Passey, Brackett, and Wilson (1987) investigated the effects of caffeine on the CV reactivity of medical students engaged in quiet study during naturally occurring periods of high stress (final exam week) or low stress (a week during which no exams were scheduled). In the second study, France and Ditto (1989) recorded the CV responses of telemarketing representatives under conditions of caffeine or placebo prior to and while they worked. The results of both studies were consistent with those obtained in the laboratory; caffeine exacerbated the BP responses induced by the naturally occurring stressful situation.

Although commendable in their use of naturalistic testing environments, neither Pincomb et al., (1987) nor France and Ditto (1989) adequately addressed the issue of duration of stress exposure. In both of these studies, subjects experienced the combined effects of caffeine and stress for only a short period of time. The duration of stress exposure following caffeine consumption may be of little concern if the drug's effects on CV reactivity during short term stressors are

similar to its effects when exposure to stress is prolonged. Unfortunately, this may not be the case.

Recently Miller and Ditto (1988; 1989) reported that the hemodynamic responses of subjects engaged in an extended stressor task varied as the task progressed. In these studies, subjects were required to play a video game task for a one-hour period while measures of HR, BP, digital blood volume pulse (DBVP), and ear pulse transit time (EPTT) were recorded continuously. During early stages of the task, subjects demonstrated an increase in HR, BP, and EPTT, and a decrease in DBVP. As the task progressed, however, HR and EPTT responses became significantly less pronounced while BP and DBVP responses remained stable. In interpreting these results, the authors concluded that the observed reductions in HR and EPTT during later stages of the task reflected an habituation of cardiac output responses to the stress. Vascular resistance (as reflected by DBVP), on the other hand, showed no signs of altering during any stage of the experiment.

The results reported by Miller and Ditto (1988; 1989) are of significance when assessing the potential effects of caffeine on CV responding during an extended stressor task. As described above, Pincomb et al., (1988) report that the mechanisms underlying caffeine's effects on CV reactivity vary depending on the behavioural state of the individual. Under resting conditions, caffeine increases BP by enhancing vascular resistance. During periods of stress, however, caffeine exacerbates BP responses by potentiating stress-induced increases in cardiac output. If caffeine's pressor effects during stress result from a potentiation of cardiac output, and if cardiac output habituates during later stages of an extended

stressor task, then it is possible that caffeine's ability to exacerbate CV stress responses are decreased as exposure to stress is increased. Unfortunately, since there have been no reported investigations of the effects of caffeine on CV reactivity during an extended stressor task, the validity of this hypothesis cannot be determined.

The purpose of the present study is to determine whether caffeine's pressor effects during the early stages of an extended stressor task will differ from its effects during later stages of the task. According to MacDougall, Musante, Castillo, and Acervado (1988), resolution of this issue may have important implications for the role of caffeine as a health risk factor. These authors suggest that if caffeine's CV effects are observable only during short term stressors or only during the early stages of prolonged stress, the health risks associated with caffeine use may be somewhat limited. In this case, the physiological costs of caffeine's pressor effects would add little to the overall pathogenic consequences of prolonged exposure to stress. On the other hand, if caffeine's CV effects are maintained during prolonged exposure to stress, its role as a health risk factor may be more significant. In this case, caffeine would enhance the physiological cost of the stress for extended periods, and thus would significantly increase the individual's risk of developing stress-related pathologies.

A second purpose of this study is to determine whether the performance of a stressor task will be influenced by the prior consumption of caffeine. As mentioned, previous research suggests that caffeine's performance-enhancing effects may be more pronounced when exposure to the task is prolonged. In the present study, such a finding may have important implications, particularly in

view of caffeine's potential CV effects. For example, if caffeine is found to enhance performance during later stages of the extended stressor task while exacerbating CV reactivity only temporarily during early stages of the task, its use under these conditions may be warranted. In this case, caffeine's performance-enhancing effects may outweigh the risks associated with transient increases in CV reactivity. On the other hand, if caffeine's CV effects are maintained throughout the duration of the task, then its use under conditions of prolonged stress should be avoided. In this case, the potential health consequences associated with the prolonged exacerbation of the physiological costs of stress would greatly outweigh any possible benefits caffeine may have on performance.

The final purpose of the present study is to assess the effects of caffeine on subjects' mood states. Although previous research suggests that caffeine, when consumed in moderate to large doses, may increase anxiety, a recent study by Ratliff-Crain et al. (1989) found that habitual caffeine users react to behavioural challenges with fewer negative mood effects if they have consumed caffeine prior to engaging in the task. These findings are in accord with an earlier study by Goldstein et al. (1969) in which habitual caffeine users demonstrated a deterioration in mood over a two hour period following abstinence from their usual morning coffee. In the present study, habitual caffeine users will receive either caffeine or placebo before resting or engaging in an extended stress task. It is expected that these subjects will report more positive mood states if they consume caffeine prior to these activities.

A unique feature of the present study is the use of an unconventional laboratory task which more closely resembles naturally occurring stressors than tasks typically used in previous caffeine-stress studies. Briefly, the task involves having subjects continuously enter data into a computer over an extended period of time. Subjects will be required to perform this task both quickly and accurately in order to avoid an aversive tone. The task thus requires a high degree of concentration on the part of the subject while he performs a largely repetitive and unstimulating activity for a prolonged period of time. Naturally occurring examples of comparable "stressful" conditions can be found readily in a variety of occupational settings, and in particular, in those jobs that involve assembly line or keyboard work. Indeed, with the increased use of computers in the workplace, a computer data-entry task seems more relevant to real life than do more conventional types of laboratory stressor tasks such as mental arithmetic.

Method

Subjects

Seventeen male students from the University of Manitoba were recruited through posted advertisements to serve as subjects for the present experiment. Prior to entry into the experiment, all subjects were prescreened by telephone interview for the following characteristics; habitual caffeine use for the previous 12 months with an average estimated daily caffeine intake of 100-500mg caffeine per day, good physical health, no history of caffeine intolerance or CV disease, no family history of hypertension, nonsmokers, and not presently taking any prescription or nonprescription medications.

All subjects accepted into the study participated in four separate 2 h laboratory sessions. For participating in the experiment, subjects received either a \$20.00 honorarium or credit towards their introductory psychology course experiment participation requirement.

Experimental Design

The design of the experiment was a double-blind, within-subjects, randomized factorial with three independent variables; drug condition (caffeine versus placebo), stress condition (task versus rest), and time of measure (early versus late). Dependent variables included indices of CV activity (heart rate [HR], systolic blood pressure [SBP], diastolic blood pressure [DBP], mean arterial pressure [MAP], and digital blood volume pulse [DBVP]), measures of affective state (positive and negative affect), and task performance (number of correct responses, number of errors, mean response time). Subjects were assigned randomly to one of four groups to determine the order in which they would experience the four experimental conditions (ie., caffeine-task, caffeine-rest, placebo-task, and placebo-rest) over the course of four separate laboratory sessions. Group assignments were counterbalanced over sessions so that an equal number of subjects was assigned to each experimental condition during each session.

Materials

Caffeine administration. During each of the four laboratory sessions, the subject was asked to consume 230ml of grapefruit juice which, when appropriate, contained 200mg caffeine (2 dissolved "Wake-Ups" tablets, Arden Limited). Caffeine was administered in a double-blind fashion, with order of presentation

counterbalanced across subjects and sessions. Grapefruit juice was chosen as both the vehicle and the placebo because previous research has suggested that its taste effectively masks the bitter flavour of caffeine (Pincomb, Lovallo, Passey, Whitsett, Silverstein, & Wilson, 1985).

Physiological measures. Measures of HR, SBP, DBP, and MAP were recorded from the subject's nondominant arm using a Critikon Dinamap 845XT vital signs monitor. The unit was attached to the subject via an arterial occluding cuff which automatically inflated at preprogrammed intervals. DBVP was recorded from the index finger on the subject's nondominant hand using a Grass model PPS-B photoplethysmograph. The unit was secured to the subject's finger with an adhesive collar and a small Velcro strap and coupled with an AC amplifier (time constant = .04 seconds, gain adjusted on a session by session basis) on a Grass model 6 electroencephalograph. The signal was output to a pen driver and recorded on a single channel of the electroencephalograph writer. DBVP measures were always recorded at least three ms after any inflation of the blood pressure cuff.

Affective state ratings. Subjects rated their affective state several times during each laboratory session using the Positive and Negative Affect Schedule (PANAS: Watson, Clark, & Tellegen, 1988). This paper and pencil scale requires subjects to rate their current emotional state according to 20 mood descriptor terms. The scale provides scores for both positive affect (PA) and negative affect (NA) which, according to the authors, are highly distinctive emotional dimensions. Whereas PA reflects the extent to which the individual feels enthusiastic, active, and alert, NA reflects aversive mood states including

anger, contempt, disgust, guilt, fear, and nervousness. Data supporting the validity and reliability of the scale are provided by Watson et al. (1988). A copy of the PANAS is presented in Appendix A.

Extended stressor task. During two of the laboratory sessions, the subject performed an extended "stressor" task based loosely on that used by Miller & Ditto (1988; 1989). The task consisted of four 12-m time blocks during which the subject was required to enter, using his dominant hand, several seven-digit figures into a Mind model AI-AT personal computer. The task was translated into an avoidance paradigm by requiring the subject to achieve a certain prescribed level of performance in order to avoid exposure to an aversive tone. Pilot data indicated that the task was effective in producing physiological and subjective responses consistent with those produced by more conventional laboratory stressor tasks (see Appendix B).

Each of the 12-m time blocks was divided into four 3-m trials. At the beginning of each trial, the subject was informed of the number of figures he had to enter during the trial (i.e., the criterion score) in order to avoid triggering the tone. Criterion scores for each trial were based on pilot data collected from a sample of male university students and were selected to reflect performance that was difficult but not impossible (see Appendix C). The subject was then presented with the first seven-digit figure and was prompted to enter it. Once the figure had been entered, the subject was informed of the accuracy of his entry and of the number of figures that he had yet to enter to avoid hearing the tone. This process continued until the 3-m trial ended.

At the end of each trial, the subject's score was compared to the criterion score. If the criterion score was met, the subject received a brief congratulatory message on the computer monitor. If the criterion score was not met, the subject was exposed to a 85db, 1000hz tone for 2 s. The tones were presented from one of two speakers mounted in front and to either side of the subject. Two speakers were used to increase the uncertainty of the situation for the subjects. The decision of which speaker to use was made on a random basis.

Approximately 30s after the presentation of the congratulatory message or the tone, the subject was prompted to begin the next trial. This process continued until subjects had completed all four trials in each of the four time blocks. A copy of the task instructions are presented in Appendix D.

Procedure

During the prescreening interview, information regarding the subject's age, height, and weight was collected. The subject was also asked to estimate his daily consumption of caffeinated products. These data were converted into estimates of daily caffeine intake using values supplied by Dews (1982). Other prescreening information was collected at this time to ensure that subjects met the desired characteristics. If accepted into the study, the subject was informed of the experimental rationale and all procedures involved were explained. Informed consent was then obtained (see Appendix E) and an initial laboratory session was scheduled. The subject was asked to abstain from caffeine for a minimum of 12 h and all food for a minimum of 3 h prior to the session. The subject was telephoned the night before the initial session (and the night before all subsequent sessions) and reminded of the dietary restrictions.

Upon entering the laboratory for the initial session, the subject was seated in a reclining chair and familiarized with the protocol for that day's session. The physiological recording apparatus was then attached and the subject was left alone for a 12-m adaptation period. At the end of this adaptation period, physiological measures were recorded and the subject was asked to complete the first copy of the PANAS. Next, the subject consumed the vehicle/placebo beverage and was left alone for 40 ms to allow for maximal caffeine absorption (Robertson et al., 1978). During this waiting period, the subject was allowed to read, listen to music, and, if applicable, was familiarized with the stressor task.

Following the waiting period, subjects assigned to the task condition were seated in front of the computer where they were instructed to proceed with the task. Subjects in the rest condition remained in the reclining chair and continued to read or listen to music. These subjects were instructed to relax but to remain awake throughout the session. Regardless of condition (ie., task performance or rest), the subject's physiological measures were recorded every 4 m during each of the four 12-m time blocks. At the end of each time block, the subject was asked to rate his current affective state on the PANAS. Following the final time block and mood rating, the subject was asked to indicate whether he thought he had received caffeine or placebo during that session. Once the subject's response was noted, the physiological recording apparatus was detached and the subject was scheduled for the next laboratory session.

The experimental protocol during subsequent sessions was identical to those of the first session according to the subject's group assignment. At the end

of the fourth laboratory session, the subject was fully debriefed and received either the honorarium or the experimental credits.

All sessions were conducted in a 234 x 295cm chamber and took place between 7:00am and 7:00pm. Sessions were scheduled at least two days apart and at approximately the same time of day for each subject. The physiological recording apparatus and other equipment used in the experiment was stored in a room adjacent to the experimental chamber. These rooms were connected by intercom to allow for communication between the experimenter and the subject.

Data Reduction

Measures of HR, SBP, DBP, and MAP were recorded once at the end of the adaptation period and no reduction of these data were performed. During the task performance/rest phase of the sessions, CV measures were recorded 12 separate times (three times during each of four time blocks). For statistical purposes, these data were reduced by computing the average of the first six (ie., early) and the last six (ie., late) measures recorded during the session. Heart rate was measured in beats per minute (bpm) and blood pressure was measured in mmHg.

DBVP was measured for 30 seconds near the end of the adaptation period and every 4 m during the task performance/rest phase of the sessions. These samples were scored by measuring the amplitude of each pulse wave generated during the sampling period and then averaging over these values. This process resulted in one estimate of DBVP during adaptation and 12 estimates of DBVP during task performance/rest. As with other physiological measures, these data were reduced by averaging over the first six (ie., early) and the last six (ie., late)

samples recorded during the sessions. The resulting two estimates were then converted into percent change from adaptation score using the following formula provided by Stern, Ray, and Davis (1980):

$$[(\text{estimate score} - \text{adaptation score})/\text{adaptation score}] \times 100$$

Task performance was measured as the number of correct responses and the number of errors made by the subject during the first two (ie., early) and the last two (ie., late) time blocks of task performance. Mean response time was computed as the average latency to enter the seven-digit figure presented on the monitor during that trial. Individual response latencies were averaged over the early and late periods of task performance.

Responses to the PANAS items were scored as outlined by Watson et al. (1988). Responses recorded at the end of the adaptation period were not reduced further. PA and NA scores recorded at the end of time blocks 1 and 2 were averaged to produce the early estimates of positive and negative affect. Similarly, PA and NA scores recorded at the end of time blocks 3 and 4 were averaged to produce the late estimate of positive and negative affect.

Results

Preliminary Analyses

Subject characteristics. The ages, height, weight, and daily caffeine intake for the subjects included in the study population are summarized in Table 1. Data from one subject were dropped from this and further analyses because of difficulty in obtaining valid DBVP measurements. Effectiveness of the

Table 1
Mean Age, Weight, Height, and Estimated Daily Caffeine Intake (EDCI) of
Subjects included in the Data Analysis

Characteristic	Statistic	
	Mean	Standard Error
Age (years)	22.1	1.10
Weight (kg)	74.8	2.78
Height (cm)	117.6	1.76
EDCI (mg)	216.1	29.15

double - blind administration was confirmed by comparing the percentage of correct guesses subjects made regarding drug condition (45%) to the percentage of erroneous guesses (55%). Correct predictions of drug condition were not better than chance, $t(15) = 1.85, p > .10$.

Adaptation data. Measures of HR, SBP, DBP, MAP, DBVP, PA, and NA collected at the end of the adaptation period were analyzed separately by a series of $4 \times 2 \times 2$ (Session x Drug x Stress) repeated measures multivariate analyses of variance (MANOVAs). No main effects or interactions were found for any of these dependent measures (all $ps > .10$).

Physiological Data

Measures of HR, SBP, DBP, MAP, and DBVP were initially analyzed by separate $2 \times 2 \times 2$ (Drug x Stress x Time of Measure) repeated measures analyses of variance (ANOVAs). In addition, a series of *a posteriori* contrasts were conducted on specific subsets of the data to delineate and clarify the interactive effects of caffeine, task performance, and time on cardiovascular activity. Table 2 provides descriptive statistics for all physiological measures.

Heart rate. The ANOVA performed on the HR data revealed a main effect for stress, $F(1, 15) = 37.97, p < .001$, indicating that subjects demonstrated higher HR during task performance ($M = 72.9$ bpm, $SE = 3.16$) than during rest sessions ($M = 59.9$ bpm, $SE = 2.42$). The lack of a significant Stress x Time, $F(1, 15) = 2.33, p = .15$, or Drug x Stress x Time interaction, $F(1, 15) = 1.34, p = .26$, in the main ANOVA suggests that HR remained elevated throughout the course of task performance.

Table 2

Mean (SE) Values of Cardiovascular Measures during the Early and Late Trial Blocks during Task Performance and Rest Sessions

Session	Measure									
	Heart rate		Systolic Blood pressure		Diastolic Blood Pressure		Mean Arterial Pressure		Digital Blood Volume Pulse	
	Early	Late	Early	Late	Early	Late	Early	Late	Early	Late
Task Performance										
Caffeine	73.8 (4.03)	72.4 (3.36)	130.1 (3.16)	127.6 (3.13)	80.6 (2.19)	78.7 (2.48)	95.8 (2.50)	94.7 (2.55)	50.0 (9.00)	42.8 (11.28)
Placebo	73.7 (2.83)	71.8 (2.40)	125.9 (3.05)	123.7 (2.38)	75.7 (1.84)	73.9 (2.16)	92.2 (1.89)	91.2 (2.09)	41.1 (10.08)	35.8 (9.89)
Rest										
Caffeine	59.8 (2.37)	61.3 (2.66)	122.9 (1.81)	123.2 (2.12)	74.4 (1.82)	76.4 (1.73)	89.2 (1.53)	90.9 (1.78)	39.9 (11.30)	48.6 (12.17)
Placebo	59.7 (2.47)	58.9 (2.16)	115.2 (1.76)	116.6 (1.66)	67.7 (2.54)	69.2 (2.25)	82.8 (1.67)	84.4 (1.81)	22.7 (11.86)	41.7 (9.13)

Note. Heart rate measured in beats per minute, blood pressure measured in mm/Hg, and digital blood volume pulse measured as percent change from baseline

The ANOVA yielded no main effect for drug, $F(1, 15) = 0.42, p = .53$, or time, $F(1, 15) = 1.11, p = .37$, and the Drug x Time interaction did not reach significance, $F(1, 15) = 3.11, p = .10$, indicating that neither caffeine nor passage of time influenced HR in any appreciable way.

Systolic blood pressure. The ANOVA performed on the SBP data revealed a drug main effect, $F(1, 15) = 19.84, p < .001$, indicating that subjects had higher SBP levels during caffeine sessions ($M = 125.9\text{mm/Hg}, SE = 2.06$) than during placebo sessions ($M = 120.3\text{mm/Hg}, SE = 2.20$). The main effect of stress was also significant, $F(1, 15) = 13.02, p < .01$, and confirmed that SBP was higher during performance of the stress task ($M = 126.8\text{mm/Hg}, SE = 2.93$) as compared to the rest session ($M = 119.5\text{mm/Hg}, SE = 1.84$). The combination of caffeine and task performance resulted in SBP levels that were higher than those associated with either caffeine alone, $F(1, 15) = 5.21, p < .05$, or task performance alone, $F(1, 15) = 4.76, p < .05$. However, the lack of a Drug x Stress interaction in the main ANOVA, $F(1, 15) = 1.21, p = .28$, suggests that their combined effect was additive in nature. That is, the joint effect of combining caffeine with performance of the stress task was equivalent statistically to summing the elevations produced by each of these conditions independently.

The ANOVA revealed no main effect of time, $F(1, 15) = 1.17, p = .30$, but did yield a Stress x Time interaction, $F(1, 15) = 7.22, p < .05$. This interaction reflected the fact that when subjects were engaged in the stress task, they showed higher SBP during the early trial block than during the late trial block, $F(1, 15) = 4.87, p < .05$. During the extended rest periods, however, SBP

remained relatively constant throughout the sessions, $F(1, 15) = 1.42, p = .25$. Nevertheless, even though SBP declined during the course of the stress session, it remained higher during the later time blocks than at the corresponding time period of the rest session, $F(1, 15) = 8.48, p < .01$.

Neither the Drug x Time, $F(1, 15) = 0.49, p = .50$, nor the Drug x Stress x Time, $F(1, 15) = 0.80, p = .78$, interactions were significant, indicating that under both resting and task conditions, caffeine produced increases in SBP that were maintained for the duration of the recording sessions.

Diastolic blood pressure. The results of the ANOVA conducted on DBP data were similar to those found for SBP. A drug main effect was revealed, $F(1, 15) = 39.68, p < .001$, indicating that subjects demonstrated higher DBP levels during caffeine sessions ($M = 77.5\text{mm/Hg}, SE = 2.06$) than during placebo sessions ($M = 71.6\text{mm/Hg}, SE = 2.20$). The analysis also revealed a stress main effect, $F(1, 15) = 17.94, p < .001$, and confirmed that DBP levels were higher during performance of the stress task ($M = 77.2\text{mm/Hg}, SE = 2.17$) than during rest sessions ($M = 71.9\text{mm/Hg}, SE = 2.09$). As with SBP, the combination of caffeine and task performance produced DBP levels that were higher than those associated with caffeine alone, $F(1, 15) = 5.50, p < .05$, or task performance alone, $F(1, 15) = 9.52, p < .01$. However, the failure of the main ANOVA to yield a Drug x Time interaction, $F(1, 15) = 0.76, p = .40$, suggests that these factors again combined in an additive fashion.

The ANOVA revealed no main effect of time, $F(1, 15) = 0.01, p = .96$, but did yield a Stress x Time interaction, $F(1, 15) = 8.48, p < .01$. In contrast to the results of the SBP analysis, this interaction reflected the fact that subjects

demonstrated both a decrease in DBP levels over time during performance of the stressor task, $F(1, 15) = 4.68, p < .05$, and an increase in DBP over time during rest sessions, $F(1, 15) = 8.54, p < .01$. Although subjects' DBP levels were higher when they performed the task than when they rested during both the early trial block, $F(1, 15) = 21.96, p < .001$, and the late trial block, $F(1, 15) = 7.62, p < .05$, task-rest differences during the early trial block were greater than during the late trial block, $F(1, 15) = 8.48, p < .05$

Neither the Drug x Time, $F(1, 15) = 0.09, p = .76$, nor the Drug x Stress x Time, $F(1, 15) = 0.12, p = .74$, interactions were significant, indicating that under both resting and task conditions, caffeine produced increases in DBP that were maintained for the duration of the recording sessions.

Mean arterial pressure. The ANOVA performed on the MAP data revealed a drug main effect, $F(1, 15) = 43.21, p < .001$, and a stress main effect, $F(1, 15) = 26.71, p < .001$. These findings indicate that subjects demonstrated higher MAP levels during caffeine sessions ($M = 92.7\text{mm/Hg}, SE = 2.09$) than during placebo sessions ($M = 87.6\text{mm/Hg}, SE = 1.87$), and during task performance ($M = 93.5\text{mm/Hg}, SE = 2.26$) than during rest ($M = 86.8\text{mm/Hg}, SE = 1.70$). As with other measures of blood pressure, the combination of caffeine and task performance produced MAP levels that were higher than those associated with either caffeine alone, $F(1, 15) = 4.68, p < .05$, or task performance alone, $F(1, 15) = 7.90, p < .05$. The lack of a Drug x Stress interaction, $F(1, 15) = 1.35, p = .26$, indicates that the combined effect of caffeine and stress was additive. No other main effects or interactions were revealed.

Digital blood volume pulse. The ANOVA performed on the DBVP data yielded no main effects. A Stress x Time interaction was revealed however, $F(1, 15) = 9.52, p < .01$. As suggested in Figure 1, although DBVP levels remained stable over time during task performance, $F(1, 15) = 1.42, p = .25$, they were significantly lower during the early trial block as compared to the late trial block under resting conditions, $F(1, 15) = 9.24, p < .01$. No other interactions were revealed.

Task Performance Data

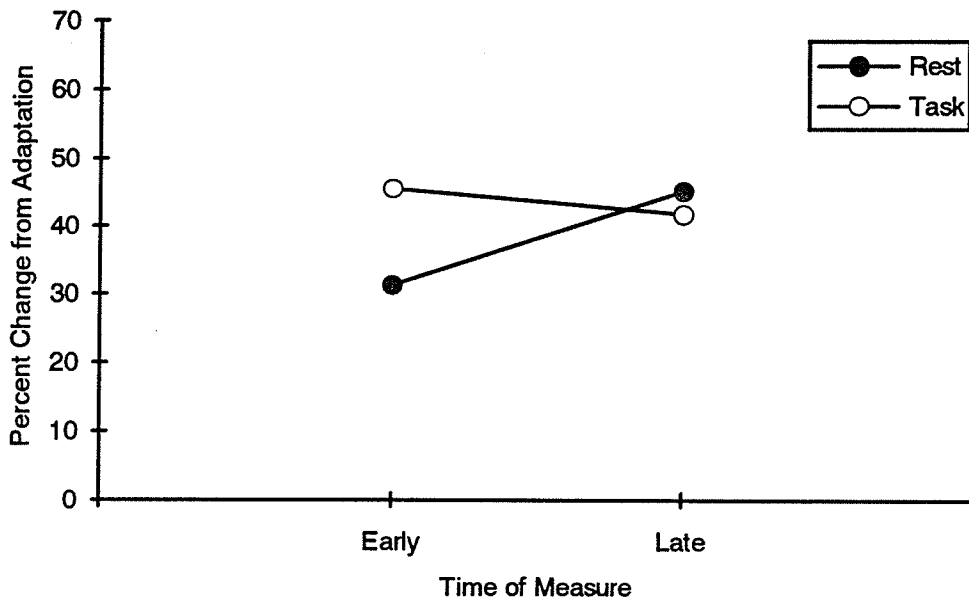
The number of correct responses and errors made by the subjects and the subjects' mean response times were analyzed separately by a series of 2 x 2 (Drug x Time of Measure) repeated measures ANOVAs. The analyses revealed a time main effect for both number of correct responses, $F(1, 15) = 17.26, p < .001$, and mean response time, $F(1, 15) = 26.77, p < .001$. These findings indicate that subjects made fewer correct responses ($M = 274, SE = 15.61$) and demonstrated slower response times ($M = 5.1$ s, $SE = 0.23$) during the early time blocks than during later time blocks ($M = 286, SE = 15.35$, and $M = 4.8$ s, $SE = 0.42$ for correct responses and mean response time respectively). No other main effects or interactions were revealed by the analyses.

Mood Ratings Data

Subjects' ratings of positive affect and negative affect were analyzed by separate 2 x 2 x 2 (Drug x Stress x Time of Measure) repeated measures ANOVAs. *A posteriori* contrasts were conducted on specific subsets of the data when necessary to delineate interactions.

Figure 1

Mean Percent Change from Adaptation of Digital Blood Volume Pulse during Task Performance and Resting Sessions as a Function of Time of Measure



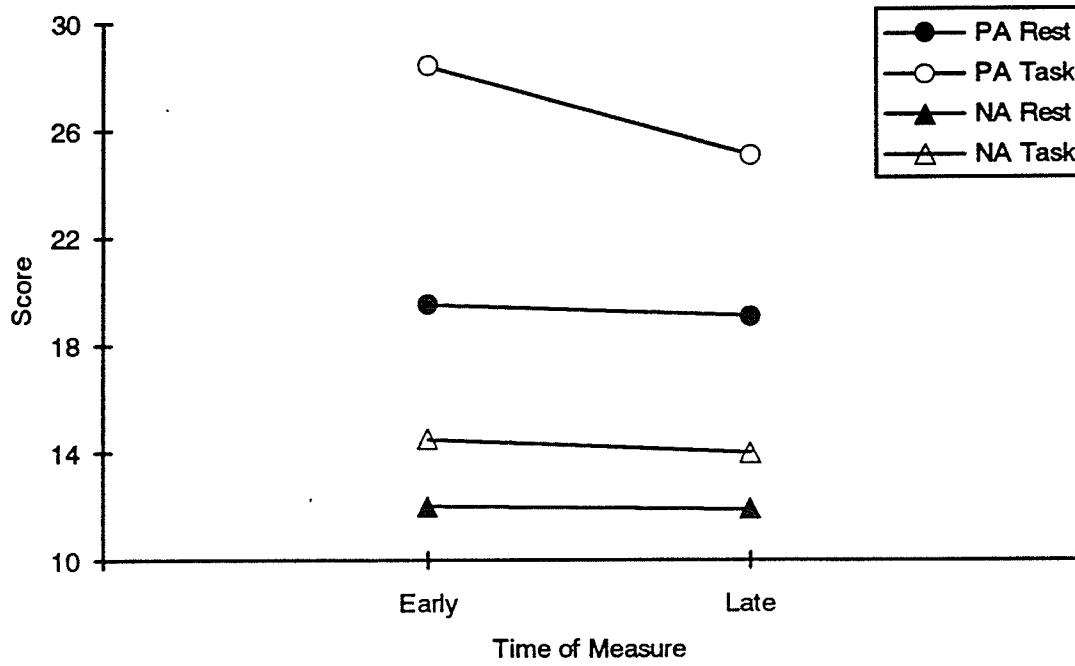
Positive affect. Analysis of the PA scores yielded a main effects for drug, $F(1, 15) = 5.31, p < .05$, stress, $F(1, 15) = 23.40, p < .001$, and time, $F(1, 15) = 16.45, p < .001$. On average, subjects demonstrated higher PA scores during caffeine sessions ($M = 24.4, SE = 1.72$) than during placebo sessions ($M = 21.6, SE = 1.92$), during task sessions ($M = 26.8, SE = 1.48$) than during rest sessions ($M = 19.3, SE = 1.60$), and during early time blocks ($M = 23.9, SE = 1.92$) than during late time blocks ($M = 22.1, SE = 1.75$).

The analysis also revealed a Stress x Time interaction, $F(1, 15) = 10.05, p < .01$. This interaction reflected the fact that during performance of the stress task, subjects demonstrated higher PA scores during early trial blocks than during late trial blocks, $F(1, 15) = 22.13, p < .001$. In contrast, PA scores did not change during the extended rest sessions, $F(1, 15) = 0.56, p = .46$. This interaction is depicted in Figure 2. No other interactions were revealed.

Negative affect. A stress main effect was revealed by the ANOVA performed on the NA scores, $F(1, 15) = 11.32, p < .01$, indicating that subjects demonstrated higher NA scores during task sessions ($M = 14.2, SE = 1.20$) than during rest sessions ($M = 12.0, SE = 0.70$). There was a trend towards a drug main effect as well, $F(1, 15) = 4.31, p = .056$. This finding suggests that subjects tended to have higher NA scores during caffeine sessions ($M = 13.6, SE = 1.09$) than during placebo sessions ($M = 12.6, SE = 1.01$). No other main effects or interactions approached significance.

Figure 2

Positive Affect (PA) and Negative Affect (NA) Scores during Task Performance and Resting Sessions as a Function of Time of Measure



Discussion

The Effects of Caffeine's on Cardiovascular Reactivity

The results of the present study are consistent with those of previous caffeine-stress research; the independent pressor effects of caffeine and stress combine in an additive fashion to produce blood pressure elevations that are greater than those produced by either caffeine alone or by stress alone (France & Ditto, 1989; 1992; Lane, 1983; Lane & Williams, 1985; Meyers, et al., 1989; Pincomb et al., 1988; Strickland et al., 1989). Further, since subjects in the present study were selected on the basis of their habitual use of caffeine, the present findings lend support to the growing body of literature which suggests that caffeine's CV effects are apparent in regular caffeine users after only short periods (i.e., overnight) of abstinence (Goldstein & Shapiro, 1987; Greenberg & Shapiro, 1987; Lane et al., 1990; Lane & Williams, 1987; Ratliff-Crain et al., 1989).

Also consistent with previous research is the finding that caffeine had little effect on HR during both resting and task sessions. Although some studies investigating the hemodynamic responses to caffeine have found that the drug produces increases in HR under resting conditions (Robertson et al., 1978; Robertson et al., 1984; Whitsett, Manion, & Christenson, 1984), most report either a decrease (France & Ditto, 1988; Pincomb et al., 1985), or no effect at all (Goldstein, Shapiro, Hui, & Yu, 1990; Lane & Manus, 1989; Pincomb et al., 1988). For the most part, caffeine-stress research has reported that while stress exposure produces large changes in HR, caffeine has very little effect under these conditions. In general, most authors attribute the absence of clear caffeine effects

in HR to the drug's influence on central and peripheral mechanisms that may increase or decrease myocardial activity.

Of greater interest is the finding that the additive effects of caffeine and stress are maintained when exposure to stress is prolonged. In the present study, subjects were exposed to stress in the form of a realistic computer data-entry task for a period of just under one h. During early stages of stress exposure, subjects demonstrated significant increases in SBP, DBP, and MAP that were further elevated by the prior consumption of caffeine. As the exposure to the stress continued, both SBP and DBP effects became less pronounced, but remained above resting levels. Despite this decrease, BP responses during caffeine sessions remained above placebo levels throughout the entire duration of stress exposure. Although previous studies have found additive effects of caffeine and stress, this study is apparently the first to report that these effects are sustained when exposure to stress is prolonged.

The finding that caffeine's ability to exacerbate CV reactivity is maintained during prolonged stress may be of particular significance to individuals whose occupations or lifestyles involve the regular exposure to extended periods of stressful activity. By itself, prolonged stress may elicit the activation of CV and other stress response systems for extended periods of time (Carroll, Cross, & Harris, 1990; Carroll & Roy, 1989; Miller & Ditto, 1988; 1989). This alone could potentially increase the individual's risk of developing stress-related pathologies. The prior consumption of caffeine in such situations would likely magnify the physiological cost associated with the extended activation of stress response systems, thus making the onset of stress-related

pathologies more imminent (MacDougall et al., 1988). Given the nature of the stress task used in the present study -- a data-entry task performed under deadline pressure which simulates conditions in many work place settings -- it seems likely that relatively common job demands interspersed with occasional coffee breaks may be sufficient to produce sustained, large magnitude elevations in BP.

Although subjects in the present study were exposed to the stressful task for just under one hour, there is reason to believe that caffeine exacerbation of CV stress reactivity may be experienced by some individuals for a large portion of the day. In one of the first double-blind studies to investigate caffeine's hemodynamic effects, Robertson et al. (1978) found that under resting conditions, caffeine's pressor effects were sustained for the duration of a 3-h recording session. These findings were in accord with earlier reports that caffeine's effective half-life was between 4 and 10 h (Horning, Brown, & Nowlin, 1977). Given the drug's half-life and the stability of its CV effects, it seems likely that caffeine's ability to exacerbate CV reactivity may also be maintained in situations where exposure to stress exceeds one hour.

This is particularly significant in view of recent findings reported by Goldstein, Shapiro, Hui, and Yu (1990). These authors found that in a sample of habitual caffeine users, a second dose of caffeine produced increases in resting BP that were similar in magnitude to those produced by the first dose, even though the second dose followed the first dose by just a few hours. These results suggest that an individual who has a cup of coffee first thing in the morning and then another cup a few hours later during coffee break may experience elevated BP levels throughout the entire morning. If this pattern holds true for individuals

who regularly face prolonged periods of stress, the repeated consumption of caffeine between episodes of stress exposure may result in these individuals experiencing caffeine exacerbation of CV stress responses throughout most of the day. Under these conditions, the consequences associated with caffeine use would be maximized.

Although the findings of the present study require replication, they may have relevance to the etiology and treatment of CV disease in at least two ways. First, the finding that caffeine exacerbation of CV reactivity can be long lived may help clarify its role in the development of some forms of CV disease. For example, Julius and Weder (1989) have argued recently that the development of human hypertension involves the progressive raising of the body's BP "set point." Although the factors which lead to the resetting of set point are not entirely clear, prolonged elevation in BP, such as those observed in the present study, may be of important in this process. It seems possible that when elevations in BP are sustained for prolonged periods, the body may come to adjust its homeostatic mechanisms accordingly. By enhancing and maintaining elevated BP levels during prolonged exposure to stress, caffeine may make this readjustment of the set point more imminent.

Secondly, the ability of caffeine to maintain exacerbated CV responses during prolonged periods of stress may be of clinical significance for individuals suffering from hypertension. For some hypertensives, the combination of caffeine and prolonged exposure to stress may be responsible for maintaining BP responses at or above pathogenic levels. Besides promoting and enhancing the deleterious consequences associated with chronic elevations in BP, such an effect

may negate any benefits these individuals are receiving from antihypertensive medications. By limiting their consumption of caffeine and/or their exposure to stress, these individuals may be able to reduce their BP to less dangerous levels.

Although the present study indicates that caffeine's effects on CV reactivity are maintained when exposure to stress is prolonged, the results provide little information about the mechanisms underlying this effect. A previous study by Pincomb et al., (1988) suggests that the mechanisms responsible for caffeine's pressor effects are dependent on the behavioural state of the subject. In that study, caffeine's pressor effects under resting conditions were mediated by an increase in vascular resistance. Under conditions of stress, however, BP increases were produced by an exacerbation of cardiac output while vascular resistance remained at placebo levels. France and Ditto (1988) also found that caffeine produced increases in resting BP by enhancing vascular resistance. However, these authors failed to find any evidence of a caffeine induced exacerbation of cardiac output during stress exposure.

In the present study, caffeine appeared to have very little effect on vascular resistance during either the resting or task-performance sessions. A closer examination of the DBVP data reveals that, although not significant, caffeine-placebo differences at time 1 were greater under resting than stressful conditions (differences of approximately 17% and 9% respectively). As the sessions progressed however, the caffeine-placebo differences under resting conditions became more similar to those observed under stressful conditions (under both resting and task conditions, caffeine-placebo differences at time 2 were approximately 7%). These findings suggest the possibility that the results

reported by Pincomb et al., (1988), that vascular resistance is more strongly affected by caffeine under resting conditions than under stressful conditions, may be limited in duration and only observable during short-term stress exposure. Indeed, the stress exposure used by those authors lasted approximately 15 minutes, a duration considerably shorter than what would have been experienced by subjects in the present study at time 1. Clearly, more research is needed in this area before any definitive conclusions can be reached.

Effects of Caffeine on Task Performance

The present study found no evidence of a performance-enhancing effect of caffeine. The analyses found that number of correct responses, errors, and mean response time did not differ significantly between caffeine and placebo sessions. Research investigating the effects of caffeine on task performance generally reports that caffeine's performance-enhancing effects may depend on the type of task being tested. This body of literature suggests that although caffeine tends to improve performance on vigilance and reaction time tasks (Baker & Theologus, 1972; Lieberman et al., 1987; Smith et al., 1977), it has either no effect or detrimental effects on tasks that require fine motor movement (Hollingsworth, 1912; Kuznicki & Turner, 1986; Lieberman et al., 1987; Paroli, 1972; Putz-Anderson et al, 1981). Given that the task used in the present study involved both a reaction time component (subjects had to perform at a certain rate to avoid the tone), and a fine motor component (subjects had to manipulate a numerical keypad), the finding of no significant drug effect might have been foreseen.

The finding of no significant drug by time interaction for the task performance data was surprising and contrary to the research hypothesis.

Previous research suggests that caffeine's performance enhancing effects are most noticeable in situations where performance is likely to deteriorate as the result of boredom or fatigue (Weiss & Laties, 1962; Buzzie, 1986; Loke & Meliska, 1984). Although subjects in the present study performed the stressor task for an extended period of time, their performance during the latter stages of the task did not differ as the result of prior caffeine consumption. One possible explanation for this discrepancy may be that the duration of the task used in the present study was not long enough for caffeine's performance-enhancing effects to be realized. In previous studies, performance was found to be enhanced by caffeine after the subject engaged in the task for periods greater than one h (Baker & Theolougus, 1972; Hauty & Payne, 1955; Regina et al, 1974; Weiss & Laties, 1962). In the present study, subjects performed the task for a total of 48 m.

A significant main effect of time was found for measures of correct responses and mean reaction time. In general, subjects made more correct responses and responded more quickly as the task progressed. This effect was expected, since pilot data indicated that subjects demonstrate improved performance of the task over time. In fact, criterion scores for task performance were increased progressively over successive trials in order to counter any practice effects.

Effects of Caffeine on Mood

The present study provided partial support for the hypothesis that subjects would demonstrate more positive mood states and less negative mood states during caffeine sessions than during placebo sessions. Although subjects

demonstrated higher positive affect following caffeine consumption, they also tended to have higher negative affect scores under these conditions.

The finding that caffeine consumption increased positive affect scores is consistent with a previous study (Goldstein et al., 1969) which found that habitual caffeine users reported less irritability and greater alertness and contentedness following caffeine consumption than following placebo. Although the mechanisms by which caffeine increases positive mood states is unclear, these effects may be dependent on an individual's personal history of caffeine use. In an earlier study, Goldstein and Kaizer (1969) found that regular caffeine users reported being more affected by the desirable stimulant actions of caffeine and less affected by the drug's unpleasant side effects than nonusers. Because subjects in the present study were selected on the basis of habitual caffeine use, increases in positive mood state were expected.

The trend towards a significant caffeine effect on negative affect scores is in contrast to the findings of Ratliff-Crain et al., (1989). These authors reported that caffeine was effective in reducing negative mood states of regular caffeine users engaged in a laboratory stressor task. However, the instrument used in that study to measure the subjects emotional state assessed two aspects of negative mood; "stress effects" (ie., how nervous, restless and stressed the subjects felt) and "withdrawal effects" (ie., items related to physical discomfort). An examination of the results reveals that while subjects rated the withdrawal items lower following caffeine consumption than following placebo, the drug had little effect on ratings of the stress items. In the present study, the instrument used to measure negative mood focused primarily on affective dimensions related to

nervousness, jitteriness, and hostility (Watson et al., 1988). Several studies have reported that caffeine consumption, even in habitual users, increases these mood states (Chiat & Griffiths, 1983; France & Ditto, 1992; Greden, 1974; Velber & Templer, 1984). Thus, the discrepancy between the findings of the present study and those of Ratliff-Crain et al., (1989) may be due to differences in the types of instruments used to assess negative mood.

The finding that task performance increased both positive and negative affect scores seem at first to be contradictory. However, Watson et. al., (1988) have demonstrated that positive and negative affect, as measured by the PANAS, are highly distinctive and orthogonal dimensions of mood. An examination of the items on the PA scale suggest that PA scores reflect how determined, excited, alert, and active the subject feels. NA scores, on the other hand, appear to reflect the degree to which the subject feels distressed, upset, hostile, and ashamed. Given the nature of the stressor task used in the present study, it seems reasonable to assume that subjects felt more active, alert, and excited during task sessions than during rest sessions. Despite these "positive affect" feelings, subjects were probably more likely to experience feelings of distress, upset, and hostility when they performed the task than when they rested.

General Conclusions

The purpose of the present study was to determine whether caffeine's effects on CV stress responses during the early stages of a prolonged stressor task are similar to its effects during later stages of the task. Previous research indicates that the pressor effects of caffeine add to those produced by stress so that BP responses to stress are greater when caffeine is consumed. The present

study also found that the pressor effects of caffeine and stress add together to produce BP responses greater than those produced by stress alone. In addition, this study found that these heightened BP responses are maintained throughout the duration of the extended stressor task. This finding indicates that caffeine's effects on CV stress responses are maintained when exposure to stress is prolonged.

Although the findings of this study require replication before they can be accepted fully, they do caution against the use of caffeine prior to engaging in prolonged periods of stress. Under such conditions, caffeine may increase an individual's risk of developing stress-related pathologies by exacerbating the physiological cost of stress for prolonged periods. Further, the finding that caffeine may increase negative mood states during the performance of prolonged stressor tasks also argues against the use of the drug in these situations. The only beneficial effect of caffeine noted in this study was an increase in positive affect. Given the potential health consequences associated with the drug, these effects do not seem great enough to warrant the consumption of caffeine prior to prolonged stress exposure.

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Appendix A

The Positive and Negative Affect Schedule

This questionnaire consists of a number of words that describe different feelings and emotions. Read each word and then circle the number to the right of that word which best describes the way you feel right now, that is, at the present moment.

	Slightly or not at all	a little	moderately	quite a bit	extremely
interested	1	2	3	4	5
distressed	1	2	3	4	5
excited	1	2	3	4	5
upset	1	2	3	4	5
strong	1	2	3	4	5
guilty	1	2	3	4	5
scared	1	2	3	4	5
hostile	1	2	3	4	5
enthusiastic	1	2	3	4	5
proud	1	2	3	4	5
irritable	1	2	3	4	5
alert	1	2	3	4	5
ashamed	1	2	3	4	5
inspired	1	2	3	4	5
nervous	1	2	3	4	5
determined	1	2	3	4	5
attentive	1	2	3	4	5
jittery	1	2	3	4	5
active	1	2	3	4	5
afraid	1	2	3	4	5

Appendix B

Pilot Study: Determining the "Stressfulness" of the Computer Data-Entry Task

The purpose of this pilot study was to determine whether a newly developed computer "data-entry" task would elicit physiological and subjective responses in male subjects that are similar to those associated with more conventional laboratory stressors.

Subjects in this pilot study were 15 male university students enrolled in an introductory psychology course. Each subject participated in a 1 h laboratory session for which he received an experimental credit.

All apparatus used in this study has been explained in the manuscript (see Method). The computer task used in this study differed from the one used in the study proper in that subjects performed the task for the first 12 minute time block only (ie., they performed four consecutive trials).

Upon arriving for the experiment, each subject was told that the purpose of the study was to examine physiological and subjective responses to a computer data-entry task. The subject was informed that his heart rate and blood pressure would be measured several times during the study.

Next, the subject was left alone for a 12-m adaptation period during which physiological measures (ie., HR, SBP, DBP, and MAP) were recorded every 4 m. After this adaptation period, the subject was asked to complete a copy of the PANAS. The subject then performed the computer data-entry task for 12 m during which physiological measures were recorded every 4 m.

Once the subject completed the task, he was asked to complete a second copy of the PANAS and then to rate, on individual 5-point scales, how "challenging," "stressful," "frustrating," and "tiring" he thought the task was. The subject was then debriefed and given an experimental credit.

The physiological data recorded at the fourth, eighth, and twelfth minute of adaptation and task performance were averaged to produce one measure each of HR, SBP, DBP, and MAP for the adaptation and task performance phases of the experiment. These values were then compared separately using repeated measures t -tests (two tailed). In brief, all cardiovascular measures were significantly higher during the task phase of the experiment than during the adaptation phase (see table B-1 for a summary of these results).

Pre- and post-task ratings on the PANAS scales were scored to produce one pre- and one post-task rating of each positive affect and negative affect. These ratings were then compared separately with a repeated measures T-test. This analysis revealed that subjects demonstrated significantly higher negative affect scores following performance of the task than prior to task performance ($t(14) = 3.62, p < .05$). An increase in negative affect is characterized by increasing feelings of anger, contempt, disgust, guilt, fear, and/or nervousness. No significant effects were found for the positive affect scores.

Ratings of how challenging, stressful, frustrating, and tiring the task was were averaged across all subjects. These mean responses are reported in table B-2. On average, subjects rated the task as somewhat challenging, moderately stressful, moderately frustrating, and slightly to moderately tiring.

Table B-1

Mean Heart Rate, Systolic, Diastolic, and Mean Arterial Blood Pressure, and Positive and Negative Affect Scores During Adaptation and Task Performance Phases of the Experiment.

Measure	Adaptation		Task		Diff.
	Mean	SE	Mean	SE	
Heart rate	69.6	2.09	84.6	2.37	-15.01*
Systolic BP	123.2	2.07	137.3	3.10	-14.11*
Diastolic BP	73.0	1.74	83.5	2.69	-10.47*
Mean Arterial BP	88.5	2.07	101.7	2.99	-13.21*
PA score	23.3	1.71	23.2	1.56	0.10
NA score	13.7	0.86	20.1	2.13	-6.40**

* $p < .001$

** $p < .01$

Table B-2
Mean Ratings of how Challenging, Stressful, Frustrating, and Tiring the
Computer Task Was

Item	Mean	Standard Error
Challenging	2.0	0.26
Stressful	3.0	0.32
Frustrating	3.1	0.35
Tiring	3.5	0.27

Note. Scale:

- 1 very much so
- 2 somewhat
- 3 moderately so
- 4 only slightly
- 5 not at all

Overall, physiological and subjective responses resulting from the performance of the computer data-entry task are consistent with those associated with more conventional laboratory stressor tasks. As with these tasks, the computer data-entry task was found to produce increases in cardiovascular activity as well as increases in negative mood states. Further, when asked to rate how stressful the task was, subjects generally rated the task as moderately stressful. Based on these findings, it seems reasonable to assume that this task is at least comparable to other laboratory tasks in terms of its ability to induce acute stress.

Appendix C

Pilot Study: Determining the Computer Data-Entry TaskCriterion Scores

The purpose of this pilot study was to determine criterion scores for each trial of the computer data-entry task. It was decided prior to conducting this study that the criterion scores for each trial would be the mean number of entries made per trial by the pilot study sample.

Subjects in this pilot study were 20 male university students enrolled in an introductory psychology course. Each subject participated in a 1-h laboratory session for which he received an experimental credit.

All apparatus used in this study has been explained in the manuscript (see Method). The computer task used here differed from the one used in the study proper in that no criterion scores were present to subjects and no congratulatory message or tone followed the trial. Task instructions were altered accordingly.

Upon arriving for the experiment, each subject was told that the purpose of the study was to determine how quickly and accurately people could enter data into a computer. The subject was then informed of all procedures involved in the study and the computer data-entry task was explained fully with the instructions that the subject should try to perform the task as quickly as possible. The subject was then left alone for 48 m during which he performed the four 12-m time blocks that made up the task. Once the subject had finished the task, he was debriefed and given an experimental credit.

The correct number of entries made during task performance was averaged across subjects. These mean responses served as the criterion scores for the study proper. The means and standard deviations of the subjects responses are presented in Table C-1.

Table C-1

Mean Number of Correct Responses, Standard Errors, and Criterion Scores for each Trial of the Task

Time Block	Trial	Mean	SE	Criterion
1	1	32.7	1.01	33
	2	35.3	1.19	35
	3	36.6	0.82	37
	4	38.5	1.20	39
2	1	33.1	1.55	33
	2	34.9	1.09	35
	3	36.2	0.71	36
	4	38.0	1.86	38
3	1	32.4	1.45	32
	2	33.7	1.17	34
	3	37.4	1.55	37
	4	38.8	0.84	39
4	1	34.5	1.00	35
	2	36.9	1.25	37
	3	38.4	1.17	38
	4	40.2	1.17	40

Appendix D

Instructions for the Computer Data-Entry Task

Thank you for participating in our experiment. During this part of the experiment, we are interested in determining how quickly and accurately you can enter data into a computer over an extended period of time. To do this we have developed a computer "data-entry" task that we would like you to perform over the next 50 minutes.

The data-entry task consists of four-12 minute time blocks. Each of these time blocks is divided into four-3 minute trials. During each trial, the computer will present several seven digit figures to you one at a time. Your job is to input the figures back into the computer as quickly and as accurately as possible.

While performing the task, you will see a number displayed near the top of the computer monitor labeled "YOUR SCORE". This number will tell you how many figures you have accurately entered so far during that trial. Above "YOUR SCORE" will be another number labeled "TARGET SCORE." This number refers to how many figures an average person with your background (ie., a male university student) can enter under similar conditions.

At the end of each trial, the computer will pause for a moment to inform you if you have reached the "TARGET SCORE" for that trial. If "YOUR SCORE" is equal to or greater than the "TARGET SCORE," a brief congratulatory message will be displayed. The next trial will begin a few seconds later, so be prepared.

If, on the other hand, "YOUR SCORE" is less than the "TARGET SCORE," a loud, high pitched tone will be emitted from one of the speakers on either side of the monitor. This tone indicates that you are performing the task too slowly and that you must work more quickly and accurately during the next trial to avoid hearing the tone again. The next trial will begin shortly after this, so be prepared.

At the end of each 12 minute time block (ie., after four consecutive trials), the computer will pause while you complete a questionnaire. To proceed to the next block of trials, you must press the <ESCAPE> key. You may take a short break here, but don't wait too long or your performance level will deteriorate.

Before you begin the task, there are just a few more points that you should be aware of:

1. You must use only one hand to enter figures into the computer and you must use the numerical keypad on the right hand side of the keyboard.
2. Once you have typed in a figure, you must press the <ENTER> key so that the computer can accept your response.
3. If you notice that you have made a typing error in you entry, you may correct your response before you press the <ENTER> key by using the <BACKSPACE> key. Please note, however, making such corrections often take more time than they save.
4. During the task, the computer will respond only when you press numeric keys, the <ENTER> key, or the <BACKSPACE> key. If you press any other

key the computer will emit a beep and an error message will be displayed (this will cost you time).

5. Your blood pressure will continue to be measured periodically during this part of the experiment. When you feel the blood pressure cuff start to inflate, limit your arm and body movements as much as possible.

If you have any questions about these instructions or how you are suppose to perform the task, please ask them now.

The task will begin immediately after you press the enter key. **PLEASE WAIT UNTIL THE EXPERIMENTER TELLS YOU TO PROCEED BEFORE YOU START THE TASK.**

Appendix E

Informed Consent Form

The purpose of this study is to examine the relationship between caffeine and physiological activity under conditions of rest and behavioural arousal. The study consists of four 2-hour laboratory sessions. During each session, you will be asked to drink a glass of grapefruit juice which may or may not contain 200mg caffeine (approximately equivalent to two cups of coffee). In addition, you will be asked to perform a computer task for 48 minutes during two of the sessions. During the remaining two sessions, you will simply be required to sit and relax in a reclining chair for 48 minutes.

Each of the four sessions will follow the same basic protocol. Each session will begin with a brief rest period during which you only have to sit and relax. After this, you will be asked to drink the grapefruit juice and then to rest for an additional 40 minutes. Finally, you will be asked to either perform the computer task or to continue relaxing for 48 minutes.

At several points throughout each session your heart rate and blood pressure will be measured. In addition, you will be periodically asked to make ratings of how you are feeling.

For participating in this study, you will receive either 8 experimental credits or a \$20.00 honorarium at the end of the fourth session. Please remember that you are under no obligation to complete all four sessions and that you may terminate your involvement in this study at any time. If you do choose to terminate your involvement with this study before completing all four sessions, you will receive 2 experimental credits or \$5.00 for each session that you have partially or fully completed.

Please feel free to ask any questions you may have.

Thank you for your participation.

Signature: _____

Date: _____