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Effects of Caffeine Consumption on Cardiovascular Reactivity to Social Stress

Suzanne L. Keller

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EFFECTS OF CAFFEINE CONSUMPTION ON CARDIOVASCULAR
REACTIVITY TO SOCIAL STRESS

by

Suzanne L. Keller

A Thesis
Submitted to the
Faculty of The Graduate College
in partial fulfillment of the
requirements for the
Degree of Master of Arts
Department of Psychology

Western Michigan University
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EFFECTS OF CAFFEINE CONSUMPTION ON CARDIOVASCULAR
REACTIVITY TO SOCIAL STRESS

Suzanne L. Keller, M.A.

Western Michigan University, 1988

Several risk factors have been linked to cardiovascular diseases; among them are exposure to psychological stressors and caffeine consumption.

The objective of this study was to examine the effects of caffeine consumption on cardiovascular reactivity to role play simulations of social stressors. A double-blind alternating treatment design was employed. Eight subjects (five female and three male) received either caffeine-containing or placebo beverages. Each subject received four samples of each beverage over a total of eight sessions. The results include: (a) stressors increased systolic and diastolic blood pressure along with heart rate in baseline, placebo and drug conditions; (b) caffeine produced increases in blood pressure over placebo but was not statistically significant; and (c) caffeine did not increase cardiovascular reactivity when presented in conjunction with stressful social interactions. An additive effect between caffeine consumption and stressful social interactions was evidenced by increases in blood pressure.

ACKNOWLEDGEMENTS

I would like to dedicate this thesis to my family: Joe, Josh and Becky whose understanding and patience throughout this study have made its completion possible. Without them by my side, it would not have been worthwhile.

I would also like to express my deep appreciation to the many people who were involved in the preparation of this study. Without the editing and feedback of Dr. R. Wayne Fuqua, my graduate advisor, this study would not have been as effective. Bernie Pinto's diligence in measurement and preparation of the drug vehicle was invaluable.

I am grateful to The Graduate College for financial support for this study and to the Department of Psychology for the assistantships I have received.

Suzanne L. Keller

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CHAPTER I

INTRODUCTION

Cardiovascular diseases are the leading cause of death, accounting for more than one half of all deaths in the United States (American Heart Association, 1981). One of the cardiovascular diseases, coronary heart disease (CHD), is the greatest single cause of death in the United States (Davison & Neale, 1986). CHD is a narrowing of the arteries in or near the heart resulting in a restriction or blockage of oxygenated blood flow to the heart (Sue, Sue & Sue, 1986). This narrowing of the arteries may result in cardiac arrest.

There are several known risk factors associated with CHD. Hypertension, diabetes, obesity, cigarette smoking, elevated serum cholesterol and physical inactivity have all been found to increase the risk of CHD (Sue et al., 1986). Other research suggests that specific behavior patterns and lifestyles might increase the risk of CHD. There is evidence that Type A behavior is associated with increased risk of CHD even though the underlying psychological components are not fully understood or defined (Rosenman et al., 1975; Glass et al., 1980). Of these factors, elevated blood pressure (BP), (hypertension), is estimated to affect 15 to 33% of the adult population of

the United States (Davison & Neale, 1986).

Only a small percentage of hypertensive individuals can concretely attribute their elevated BP to physical causes. When there is no known organic cause, essential hypertension is diagnosed. Essential hypertension is estimated to account for 90% of all hypertension diagnoses (Orton, Beiman & Ciminero, 1982). Among the factors that have been postulated to play a role in the etiology and maintenance of elevated BP are high sodium consumption, obesity, and heavy use of caffeine and alcohol (Sue et al., 1986).

Pharmacological treatment, although shown to be effective for hypertension, remains problematic. Many antihypertensive medications prescribed have unpleasant side effects such as impotence and dry mouth and are costly. Treatment is of long duration and often incorporates a complex medical regimen (Herd & Weiss, 1984). These factors lessen the likelihood of compliance which then lessens the potential effect. These concerns lead to a continuing interest in developing alternatives or adjuncts to pharmacological treatment.

The etiology and maintenance of essential hypertension has been the source of much speculation, but has eluded experimental proof. Psychological stress is one of the factors thought to be linked in the development and maintenance of hypertension (Obrist, 1981). Weiner

(1979) suggests a number of possible physiological mechanisms that when activated by stress may be correlated to the development and maintenance of essential hypertension, although those mechanisms have not been clearly established. Lacey and Lacey (1958) suggest that some individuals respond to stress with a specific physiological response pattern. This pattern involves sympathetic nervous system activity which regulates both heart rate (HR) and BP and can be likened to the increased levels of HR and BP in the fight-or-flight response. This physiological response pattern is also known as "cardiovascular reactivity." Krantz and Manuck (1984) suggest that injury to the inner walls of coronary arteries may occur either mechanically (through increased cardiac output) or chemically (through release of toxic catecholamines and endocrines). It is this arterial injury exacerbated under certain stressful situations that can then lead to accumulation of atherosclerotic plaque (cholesterol). This accumulation is believed to contribute to hypertension and the artery restriction and blockage of CHD.

These hypothesized mechanisms linking stress, cardiovascular reactivity and the development of essential hypertension have yet to be experimentally validated for a variety of ethical and practical reasons. Nevertheless, attempts have been made to analyze and modify

reactivity under the supposition that such research may eventually have implications for prevention and control of hypertension (Kallinke, Kulick, & Heim, 1982).

A number of behavioral techniques such as relaxation, yoga and biofeedback have been found to attenuate cardiovascular reactivity to laboratory stressors (e.g., Agras & Jacob, 1979; English & Baker, 1983; Steptoe & Ross, 1982). These techniques were tested by initiating stressors in experimental settings to evoke cardiovascular reactivity, then determining intervention effect. Among the laboratory based stressors that have been employed are the structured interview (MacDougall, Dembroski, & Krantz, 1981), socially relevant role play tasks (Twentyman & McFall, 1975), competitive video/computer type games (Fettes, 1986) and mental arithmetic tasks (Lane, 1983).

These stressors espouse to approximate naturalistic situations. But one must consider the artificiality of these stressors within the laboratory analog setting. The length of exposure to the stressor, as well as the inability to withdraw from it may alter otherwise naturalistic reactions. The external validity of the effects across time, setting and population must remain suspect.

Shapiro, Lane & Henry (1986) suggest the need to identify factors that potentiate the effects of stress on

cardiovascular reactivity. Among the factors proposed to have a potentiating effect on cardiovascular reactivity is caffeine ingestion, which has also been proposed to be a risk factor for hypertension (LaCroix, Mead, Liang, Thomas, & Pearson, 1986). While there is ample research on the effects of caffeine on cardiovascular functioning (Shapiro et al., 1986), there is a general paucity of research on the effects of caffeine on cardiovascular reactivity to stress. Given the ubiquity of stressful situations in everyday life, knowledge of the effects of caffeine on stress reactions may have implications for the etiology of hypertension.

Caffeine is one of the most commonly used drugs in the western world today. Average intake of caffeine in the United States has been estimated at greater than 200 mg. daily per person (Graham, 1978). This intake is equivalent to two to three cups of coffee for every man, woman and child. Caffeine is consumed in the caffeine-containing beverages of coffee, tea and many soft drinks. Caffeine is also found to a lesser concentration in cocoa and chocolate. Many non-prescription drugs used for pain relief, weight loss, premenstrual cramps and alertness have significant amounts of caffeine added to them, with little scientific evidence of its effectiveness.

Caffeine has a variety of effects on human beings. It is a central nervous system stimulant that has been

found to increase physical and mental activity levels (Calhoun, 1971). Caffeine has also been correlated with fatigue reduction, alertness enhancement and increased vigilance (Kuznicki & Turner, 1985; Weiss & Laties, 1962). Along with these seemingly benign effects, caffeine has some rather negative side effects associated with its use. In experimentation with low dosages (300 mg. or less), caffeine has been found to increase self report measures of anxiety, depression and hostility in normal adults (Veleber & Templer, 1984). Gilbert (1976) observed a dependence after continued use, which brought on various withdrawal symptoms such as irritability, fatigue, headache and depressed mood when caffeine was withheld. By ingesting more caffeine these withdrawal symptoms were eliminated.

Caffeine has been linked with disorders in the gastrointestinal system (Curatola & Robertson, 1983). It has been speculated to be a factor in promoting certain types of cancer (Rosenberg, 1985). Several animal studies have indicated a correlation between caffeine ingestion and birth defects (Watkinson, 1985; Ohnishi et al., 1986). Other studies link caffeine use to breast disease and heart rhythm problems (Rosenberg, 1985). Caffeine has been linked to essential hypertension (Sutherland, McPherson, Renton, Spencer, & Montague, 1985), which may lead to other cardiovascular diseases.

Whitsett, Manion and Christensen (1984) found that caffeine altered HR, and systolic and diastolic blood pressure (SBP and DBP respectively) in caffeine users. This alteration consisted of marked increases in both SBP and DBP along with a maximal decrease in HR from 71 to 46 beats per minute. Robertson et al. (1978) studied the effect of a single administration of 250 mg. of caffeine and also showed marked changes in the cardiovascular indices. BP increased by 14/10 mmHg. along with a marked decrease in HR from baseline levels. In 1985, Izzo, Ghosal, Kwong, Freeman and Jaenike found that non-users of caffeine (caffeine-naive) had an accentuated reactivity to caffeine when compared to chronic caffeine users. This suggested that tolerance might be an effect of continued caffeine use lessening the changes in HR and BP over time. These studies all measured increases in blood pressure but did not address cardiovascular reactivity between placebo and drug conditions.

In 1983, Lane questioned the role that psychological stressors had on cardiovascular reactivity when paired with caffeine ingestion. Lane found a significant elevation to SBP and DBP both at rest and during stress after ingestion of 250 mg. of caffeine. The results confirmed that BP during a stressful situation is higher after consumption of caffeine. This suggested an additive effect might be present. An additive effect

means that the increases in BP obtained by caffeine consumption add to the increases obtained under stress to get a BP elevation greater than when either variable was employed in isolation. Further research (Lane & Williams, 1985; Lane & Williams, 1987) confirmed the additive effect on blood pressure increases, while the additive effect on cardiovascular reactivity remained inconclusive. Caffeine consumption does affect the cardiovascular system, but to what extent when presented in combination with other risk factors such as stress remains complicated.

In all of his studies, Lane used mental arithmetic as the laboratory stressor. This task was chosen because it had been previously shown to produce elevation in heart rate and blood pressure (e.g., Greenberg & Shapiro, 1987). But mental arithmetic tasks used as stressors do have certain limitations. The tasks are not characteristic of many naturalistic stressors in that the task is completed in isolation. The task is also predictable. No new elements are introduced once the task is set. Mental arithmetic seems to assess only one type of stress, that of performance anxiety. Mental arithmetic does not deal with interpersonal types of stress wherein one shares in the control of the situation. It also does not take into account the spontaneity of many situations in which individuals are placed daily. To approximate a

more naturalistic environmental exposure, competitive social interactions and social situations with the potential of provoking anger, anxiety and/or impatience might better test caffeine's role in cardiovascular reactivity. The data obtained using naturalistic stressors might better show the cumulative effects of caffeine and stress on cardiovascular reactivity in individuals.

This study attempted to assess the effects of caffeine on cardiovascular reactivity to stressors. The research compared caffeine and non-caffeine states under both rest and stress conditions in a laboratory analogue situation much like the work of Robertson et al., (1984) and Lane (1983). But this research extended the previous work by incorporating naturally occurring stressors through approximated role plays. The differences in stressors employed, could impact subjects, by being closer to everyday situations which might better represent everyday life. Results of this study could further determine caffeine's role in cardiovascular diseases and aid in determining interventions to help alleviate cardiovascular reactivity. This could aid in interventions to be used in place of or in conjunction with pharmacological treatments for the hypertensive population as well as preventive measures for the general population using caffeine in their everyday lives. If in

fact, stressors do play a role in CHD and can not be avoided in one's environment, then a reduction or elimination of caffeine might for some individuals lower the risk of increased BP and the possible consequences of hypertension and cardiovascular disease.

CHAPTER II

METHODS

Subjects

Nine subjects were recruited by personal solicitation, among Psychology Department, Western Michigan University, students and faculty, for participation in a caffeine consumption study. For inclusion, a subject had to meet the criterion of consuming less than 300 mg. of caffeine (less than three cups of brewed coffee or equivalent) daily. This was determined by having potential subjects fill out a questionnaire on typical caffeine consumption incorporating a list of all common caffeine containing products. The second criterion for inclusion required a 10% increase over resting levels in the combined mean of SBP, DBP and HR, when exposed to a three minute laboratory role play of a stressful situation. A third criterion required that subjects had never been told to avoid caffeine for medical reasons and that there was no known personal history of cardiovascular disease.

Three men and six women met the criteria and began participation in the study. One female subject (S9) dropped out after the baseline phase, leaving eight

others to participate to the conclusion of the study. The ages ranged from 43 to 22 years old with a mean age of 32 years old. None of the subjects reported taking any antihypertensive medications. Subjects were required to sign an Informed Consent (Appendix A) prepared in accordance with Western Michigan University's human subject guidelines (see Appendix B for HSIRB application and approval). These consent forms specified potential risks and benefits of participating along with outlining precautions for confidentiality. Additionally, participants agreed to make no major changes in their dietary or exercise patterns during the course of the study and to notify experimenters if such changes were unavoidable. Each subject was given the choice of being paid \$3.00 per session or \$36.00 upon completion of the experiment.

Setting

All sessions were conducted in the Behavioral Medicine Laboratory at Western Michigan University, Kalamazoo. The subjects consumed the caffeine containing/placebo beverages in the outer office, containing comfortable chairs and a table, that was used as a waiting room. A small inner room (6' X 10') equipped with physiological monitoring equipment was used for both baseline and experimental phases. This room contained a carrel housing physiological monitoring equipment,

located in such a way as to be shielded from the subject. There were also two comfortable chairs for the subject and the role play actor. An Olympus videotape camera (model VX 304) on a tripod, was located in an adjoining room behind a one-way mirror. The role play actor was only visible during the three minute role play stressors. The technician monitoring physiological measures could be heard but not seen except when giving the subject directions.

Apparatus/Materials

Physiological Recording

SBP, DBP, and HR were measured with a self-inflating digital sphygmomanometer (Carolina Biological Supply, Model 69-1118). The monitoring cuff was placed about 1-2 cm above the elbow joint of the subject's left arm. Detection of Korotkoff sound, BP and HR readings and cuff inflation/deflation were displayed digitally following each determination of BP.

Frontalis electromyographic activity (EMG) and finger electrodermal activity (EDG) were monitored with a three component system manufactured by J & J Electronics (EMG Model M-52; EDG Model R-72). Readings were displayed digitally on the digital integrator unit (J & J Dual Integrator Unit Model D-200). The EMG electrodes

were placed parallel to the frontalis muscle of the forehead to measure muscle tension levels. The electrodes were attached to adhesive discs with electrode gel. The EDG electrodes were attached to the tips of both the index and middle fingers of the left hand with electrode gel to measure galvanic skin responses.

Drug Vehicle

Powdered caffeine was dissolved in a solution of eight ounces of canned pineapple juice. Pineapple juice was found through taste tests to be the most effective vehicle for masking the bitterness of the caffeine. The caffeine was administered to the subjects on the basis of 2 mg. of powdered caffeine per pound body weight. The maximal dosage of caffeine given any subject was 300 mg. due to possible adverse side effects of a greater concentration. This solution or the placebo solution (pineapple juice alone) was packaged in nine ounce white opaque styrofoam cups covered with a white plastic lid. The cocktails were then distributed to subjects in a double-blind schedule so that neither experimenter nor subjects were aware of the conditions. The juice cocktails were kept in a refrigerator in the laboratory at approximately 36 degrees Fahrenheit. The solutions were shaken well before administration because of precipitation of solids within the pineapple juice, and given to

the subjects thirty minutes before the scheduled appointment. After ingestion the subject removed a yellow adhesive label attached to the glass, placed it on the data sheet and initialed it noting the time ingestion was completed on the label.

Intake Information

Subjects were asked to complete a Participant Information Form (Appendix C) during the screening session. This form requested information relating to medical history (personal/familial), current medications, behaviors regarding tobacco, alcohol, and sodium use along with weekly exercise. The form also ascertained methods of dealing with stress and present weight and height of each participant.

Questionnaires

A Caffeine Consumption Checklist (Appendix D) was administered at the beginning of each session to assist the subject in remembering if any caffeine had been consumed within 24 hours prior to the session. This checklist was then signed and dated by the subject.

A Participant Feedback Form (Appendix E) was administered at the end of each session to determine stressfulness of the role plays utilized within the session and to obtain suggestions for additional stress-

ful situations that might be utilized in future sessions.

Construction of Stimuli

Subject-specific vignettes representing potentially stressful situations were determined on the basis of answers given by the subjects on the Jenkins Activity Scale (Jenkins, Zyzanski & Rosenman, 1971) and the Hassles and Uplift Scale (Kanner, Coyne, Schaefer & Lazarus, 1981). Information obtained during the intake interview was also employed in creation of the individualized role plays. Role plays developed by Eisler, Hersen, Miller and Blanchard (1975) that require appropriate responses to general social stressors were also utilized across subjects for consistency.

Both generalized and subject-specific role plays represented interactions from four common relationship categories including: (1) friends/acquaintances, (2) spouse/family members, (3) co-workers/colleagues, and (4) strangers. From these categories, role plays approximating three minutes were developed. Each subject was introduced to 12 novel role plays throughout the study. An example of a representative role play follows: "You have been waiting in line at a store for some time and someone pushes his/her way in front of you. You are already late for an important appointment. Confront this person."

Safeguards

Although the role plays incorporating social stressors were naturalistic in origin, as well as typical of daily exposure, the physiological measures were monitored carefully with instructions to terminate the session if the BP rose to 200/110 mmHg. Along with immediate termination, relaxation techniques were available for the subject. As an additional safeguard a person trained in cardiac pulmonary resuscitation was present during every session. These precautionary measures were not utilized throughout the study.

Selection / Training of Role Players

Actors/actresses (4 male, 3 female) were recruited and trained to play certain roles with sample vignettes. Training consisted of becoming acquainted with and rehearsing the sample vignettes until specific criteria were achieved. While rehearsing, the investigator suggested verbal and non-verbal responses appropriate for the specific situation. Written prompts were also provided on ways to be effectively stressful. Training was terminated when the role player displayed the suggested verbal and nonverbal responses that were outlined on the vignette script and was consistent with the other actors. The actors/actresses were asked to

personalize each situation by using the subject's name. Feedback on the actor's performance was given throughout the study to maintain consistent performance.

Experimental Design

A double-blind alternating treatment design across subjects was utilized. Caffeine cocktails or placebo cocktails were introduced in each of eight experimental sessions to all subjects following a multiple baseline condition in which no beverage was given. The multiple baseline was incorporated to determine possible reactivity to introduction of the beverage. The beverage was administered in such a way that each subject would receive four placebo and four caffeine cocktails overall, with no more than two of the same type of beverage being administered consecutively. All subjects participated in either twelve or thirteen sessions.

Procedure

The following procedures were generic to all experimental sessions regardless of phase. Procedures specific to the experimental phases will be detailed in later sections. The subject first filled out a Caffeine Consumption Checklist (Appendix D) regarding their consumption of caffeine during the previous 24 hours. Only those subjects who had not ingested any caffeine for

the required time participated in an experimental session; otherwise they were rescheduled for another time when caffeine abstinence had been maintained for the required 24 hours. Once in the experimental room the subject was asked to sit down and relax. The physiological recording equipment was attached and a test BP reading taken. At this time the following instructions were read to the subject by the experimenter.

During this period you should sit quietly and relax. We will occasionally take readings during this phase and all other rest phases, but we can't talk to you or answer questions until the completion of this experimental session.

The subject was then asked for any physical sensations that he/she was experiencing and for an estimate of current BP. This phase consisted of a ten-minute adaptation phase in which BP and HR were measured at the end of the first, fifth, seventh and ninth minute. Along with these physiological measurements, one minute averages of EDG and EMG were sampled at the end of the third, sixth, eighth and tenth minute. Following the adaptation period, the experimenter obtained an estimate of BP and any physical sensations the subject was experiencing.

The first stress period was initiated by the experimenter coming out from behind the carrel and reading the following directions:

During this phase, we will read a description of a potentially stressful interaction with _____ (role players name). Please attempt to respond as you typically would in such a situation. We are interested in your typical response patterns and not in your acting ability. Begin the interaction when I say "Begin" and continue interacting with _____ (role player's name) until we ask you to stop. We will occasionally take readings during this phase.

The description of the vignette was then read and the actor/actress would enter the room. The experimenter would say "begin" and start timing the three minute role play. BP and HR were sampled at the end of the first and third minute, and EMG and EDG were averaged at the end of each minute, over the three minute period. When the experimenter concluded the role play by saying "stop" the actor/actress exited and the subject was again asked for an estimate of BP and awareness of any physical sensations.

The session continued with rest periods of five minutes alternating with stress periods identical to the first, with one exception, a different vignette was presented each time. In the rest periods BP and HR were sampled at the end of every second and fourth minute and EDG and EMG were averaged at the end of every first, third and fifth minute. These periods alternated until the subject had participated in three different role plays. At the end of each session the equipment was disconnected and the subject was asked to fill in a

Participant Feedback Form (Appendix E). An appointment time was set up for the next session.

Experimental Phases

During the eight experimental sessions the subject was asked to report thirty minutes before the session began. At this time, the juice cocktail (caffeine/placebo) was administered. After the Caffeine Consumption Checklist (Appendix D) was filled in to determine abstinence, the client was given the cocktail and observed drinking it. After the cocktail was completely ingested, the label from the beverage container was transferred to the data sheet. The subject was then asked to relax until the session was to begin. After the session, the subject was asked whether he/she felt caffeine had been included in the cocktail. This information along with any physical sensations experienced, was entered on the data sheet.

Dependent Variables

Physiological Responses

BP, HR, EMG and EDG were recorded during all phases of the experiment, both in the rest and stressor periods. The HR and BP readings were recorded by the experimenter at two minute intervals during each period. The EMG and

EDG readings were recorded at the end of successive one minute cycles. To assess physiological reactivity, the average of each of the physiological readings was calculated separately for each rest and stress period within each session. The percentage change (mean stress/mean rest x 100%) and the absolute change mean stress minus mean rest levels) were calculated for each session.

Subjective Measures

Subjects were asked to estimate their BP and to report any physical sensations experienced, following each rest and stressor phase. They were also asked at the conclusion of each experimental session to determine whether they had consumed caffeine and if in fact, they could determine any specific effects that might be attributed to the caffeine ingestion.

Scoring

Charting of SBP, DBP, HR along with EMG and EDG was accomplished after each session and maintained to determine if any phase changes should be made. Subjective ratings were also scored to determine subject's awareness of effects.

Self-Report Measures

At the end of each session, subjects completed the Client Feedback Form (Appendix E). A five-point scale rating was obtained for the level of perceived stress associated with each of the three role plays. Five-point scales were also used to determine the extent to which the subject's responses to each role play were typical of responses he/she might make in the natural environment and to whether the actor/actress responses were characteristic of ways in which others would respond in a similar natural situation.

Independent Variables

The primary independent variables included caffeine ingestion and social stressor (generalized and individualized) presentation. Both of the listed independent variables were fully described under the Apparatus/Materials section of this chapter.

CHAPTER III

RESULTS

Physiological Measures

Systolic Blood Pressure

Graphic Analysis

Figure 1 displays mean levels of SBP in baseline, placebo and drug conditions. This information is shown in all conditions for both rest and stressor phases. The graph in the upper left presents a group average while the remaining graphs present averages for individual subjects. Group data show reactivity to the stressor phase compared to the rest phase with a mean increase of 8.7 mmHg across conditions. The mean increases in mmHg for individual conditions were (113.7 (Stress) - 104.6 (Rest)) for baseline, (112.5 (stress) - 104.4 (rest)) for placebo and (118.0 (stress) - 109.1 (rest)) for drug condition. This suggests that stressful social situations may have a role in increased SBP regardless of condition. In the rest phase, the effects of caffeine compared to placebo showed a (109.1 (drug) -

Legend. = Rest Period Levels
 = Stress Period Levels

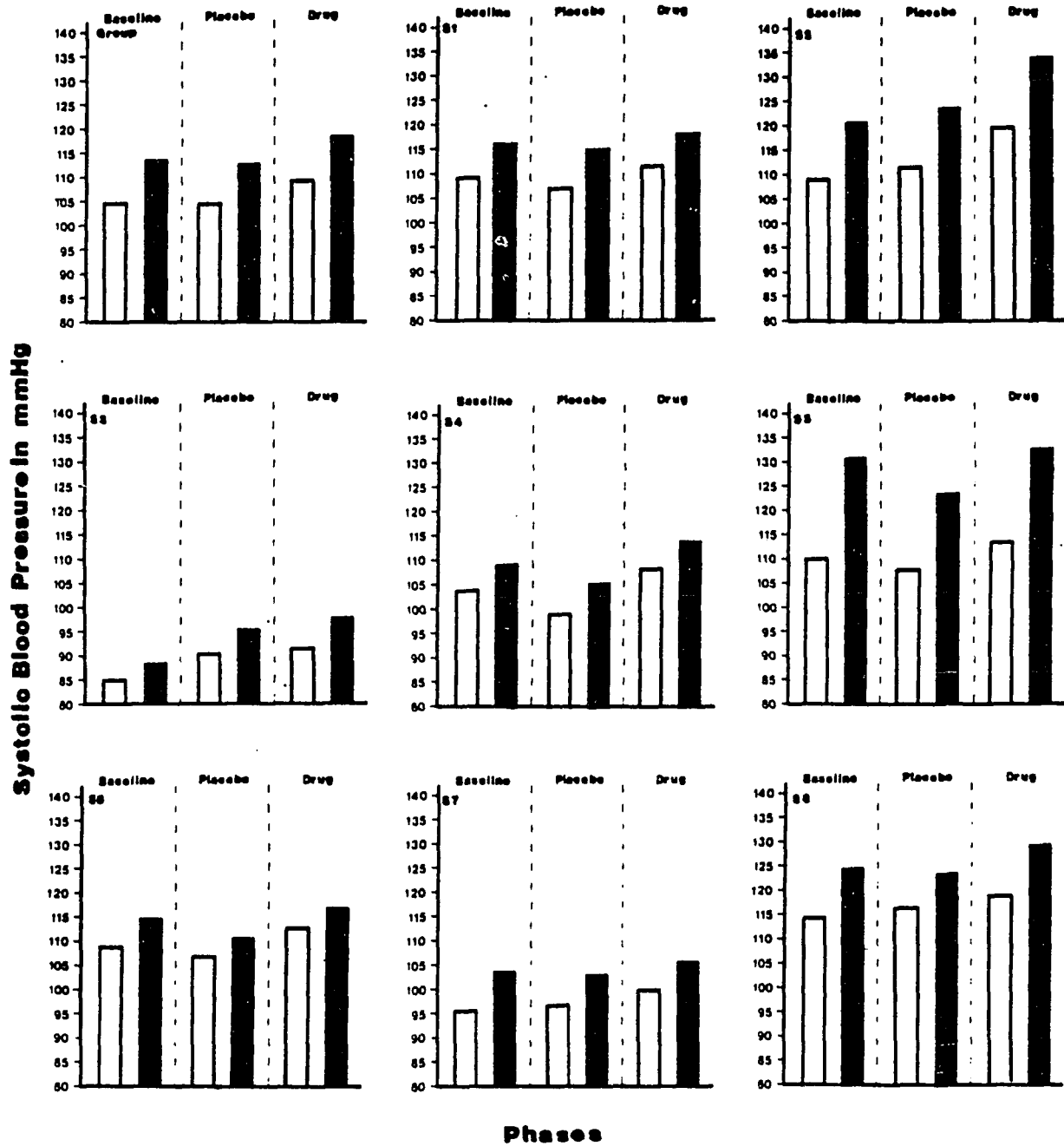


Figure 1. Mean Changes in Systolic Blood Pressure From Rest to Stressor Periods Across Phases by Subject and by Group.

104.4 (placebo)) mmHg mean increase (displayed as the white bars) while in the stressor phase the same effects showed (118.0 (drug) - 112.5 (placebo)) mmHg mean increase (displayed as the black bars). These results indicate an additive effect. When in the caffeine condition, SBP was elevated both during the rest and stress phases. There seems to be very little combined reactivity of the stressor and caffeine together.

Individual data showed considerable between-subject variability. All subjects except subject 2 and subject 3 showed no change or a decrease between baseline and placebo conditions both at rest and at stress. The decline might suggest some physiological habituation to the stressors, thus causing less reactivity overall. Reactivity to stressor situations never totally dropped out as is shown between rest and stress phase differences across all conditions, including those that were presented later in the experiment.

All subjects showed an increase in mean SBP levels in the drug condition. The greatest changes seem to be in subject 2 and subject 3. The fluctuations in amount of change seem to suggest individual variability that may be masked when merged into group data. This must be taken into consideration when reviewing the findings.

Statistical Analysis

Table 1 shows a summary of analysis of variance on SBP comparing placebo and drug conditions between rest and stressor phases. Multivariate repeated measures analysis of variance were also conducted between overall rest and stressor phases and between overall placebo and drug conditions.

Table 1
Summary of Analysis of Variance on SBP

Source	Mean	SD	df	MS	F	p
Differences between conditions						
At rest	P=104.4 D=109.1	8.0 9.0	1	89.3	1.07	.3172
At stress	P=112.5 D=118.0	10.0 12.0	1	121.0	.87	.3775
Overall differences						
Across phases			1	572.9	5.15	.0312
Across condition			1	209.1	1.88	.1814
Main effects			2	391.0	3.51	.0435

P = Placebo condition
D = Drug condition

Statistical analysis of the data shows no significant effects of caffeine, while there is a significant

difference ($<.05$) between rest and stressor phases. The difference in results between conditions and phases could be due to small sample size and high between subject variability.

Diastolic Blood Pressure

Graphic Analysis

Figure 2 displays mean levels in DBP in baseline, placebo and drug conditions for both rest and stressor phases. Group data are displayed in the upper left graph while individual data are presented in the remaining graphs. Group data show reactivity to the stressor phase compared to the rest phase with a mean increase of 7.5 mmHg across conditions. The mean increases in mmHg for individual conditions were (77.3 (stress) - 67.6 (rest)) for baseline, (72.9 (stress) - 66.6 (rest)) for placebo and (79.4 (stress) - 72.9 (rest)) for drug condition. As the case with data on SBP, these results suggest that stressful social situations may play a role in increased DBP regardless of condition. In the rest phase, the effects of caffeine compared to placebo showed (72.9 (drug) - 66.6 (placebo)) mmHg mean increase, while in the stressor phase there was (79.4 (drug) - 72.9 (placebo)) mmHg mean increase. As with SBP, there is an indication of an additive effect. DBP is increased across both

Legend. = Rest Period Levels
 = Stress Period Levels.

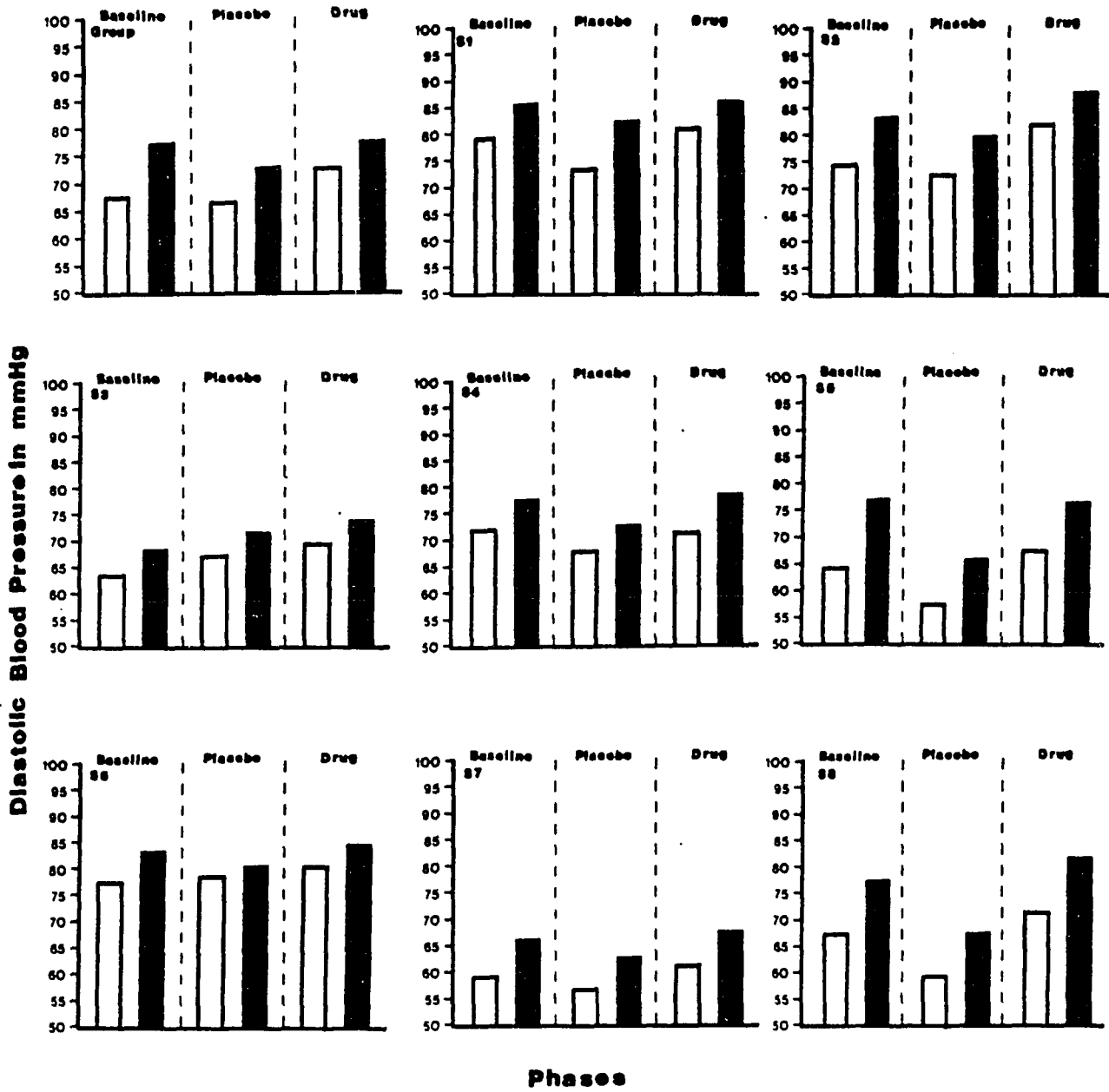


Figure 2. Mean Changes in Diastolic Blood Pressure From Rest to Stressor Periods Across Phases by Subject and by Group.

rest and stressor phases after caffeine ingestion.

Individual subject data showed variability as were also shown in SBP data. All subjects except for subject 3, showed a decrease in DBP levels from baseline phase to placebo phase. Individual increases in both rest and stressor phases from placebo to drug conditions are much greater. Subject 5 and subject 8 showed the greatest change in DBP between placebo and drug condition.

These data suggest that again as in SBP all subjects showed an increase in DBP across rest and stress phases. All subjects also showed an increase from placebo to drug conditions. The decrease in DBP levels for all but one subject in baseline to placebo condition could be attributed to habituation to the experimental situation.

Statistical Analysis

Table 2 shows a summary of analysis of variance on DBP comparing rest and stress phases between conditions. Multivariate repeated measures analysis of variance were conducted between phases and between conditions. Statistical analysis of the differences between conditions show no significant effects of caffeine, when analyzed separately by placebo vs. drug condition, although under the stress phase the findings are nearing ($<.05$) significance.

Table 2

Summary of Analysis of Variance on DBP

Source	Mean	SD	df	MS	F	P
Differences between conditions						
At rest	P= 66.6 D= 72.9	7.5 6.9	1	157.5	2.67	.1244
At stress	P= 72.9 D= 79.4	6.9 6.3	1	169.7	3.38	.0874
Overall differences						
Across phases			1	325.8	5.97	.0211
Across condition			1	327.0	5.99	.0209
Main effects			2	326.4	5.98	.0069

P = Placebo condition
D = Drug condition

The multivariate repeated measures analysis of variance conducted across phases and conditions were significant as shown. These results suggest that the stressor had an effect and that caffeine also had a significant effect. These results although favorable could be biased due to small sample size and high between subject variability.

Heart Rate

Graphic Analysis

Figure 3 displays mean HR levels during baseline, placebo and drug conditions for both rest and stressor phases. The graph in the upper left presents a group average while the remaining graphs present averages for individual subjects. Group data show reactivity to the stressor phase compared to the rest phase with a mean increase of 8.3 BPM across conditions. The mean increases in BPM for individual conditions were (82.4 (stress) - 74.7 (rest)) for baseline, ((82.4 (stress) - 74.3 (rest)) for placebo and (82.5 (stress) - 73.3 (rest)) for drug condition. As with the results of SBP and DBP, this suggests that stressful social situations may have a role in increased HR regardless of condition. Group data showed no differences in HR sampled under rest phases across three experimental conditions; HR under stress phases showed similar stability across all three conditions. This suggests that caffeine played no role in HR change either at rest or at stress. There is an increase across all three phases between rest and stressor periods. This suggests that HR is affected by stressor situations.

Individual data showed considerable between-subject variability such that no suggestion of effectiveness is

Legend. □ = Rest Period Levels
 ■ = Stress Period Levels

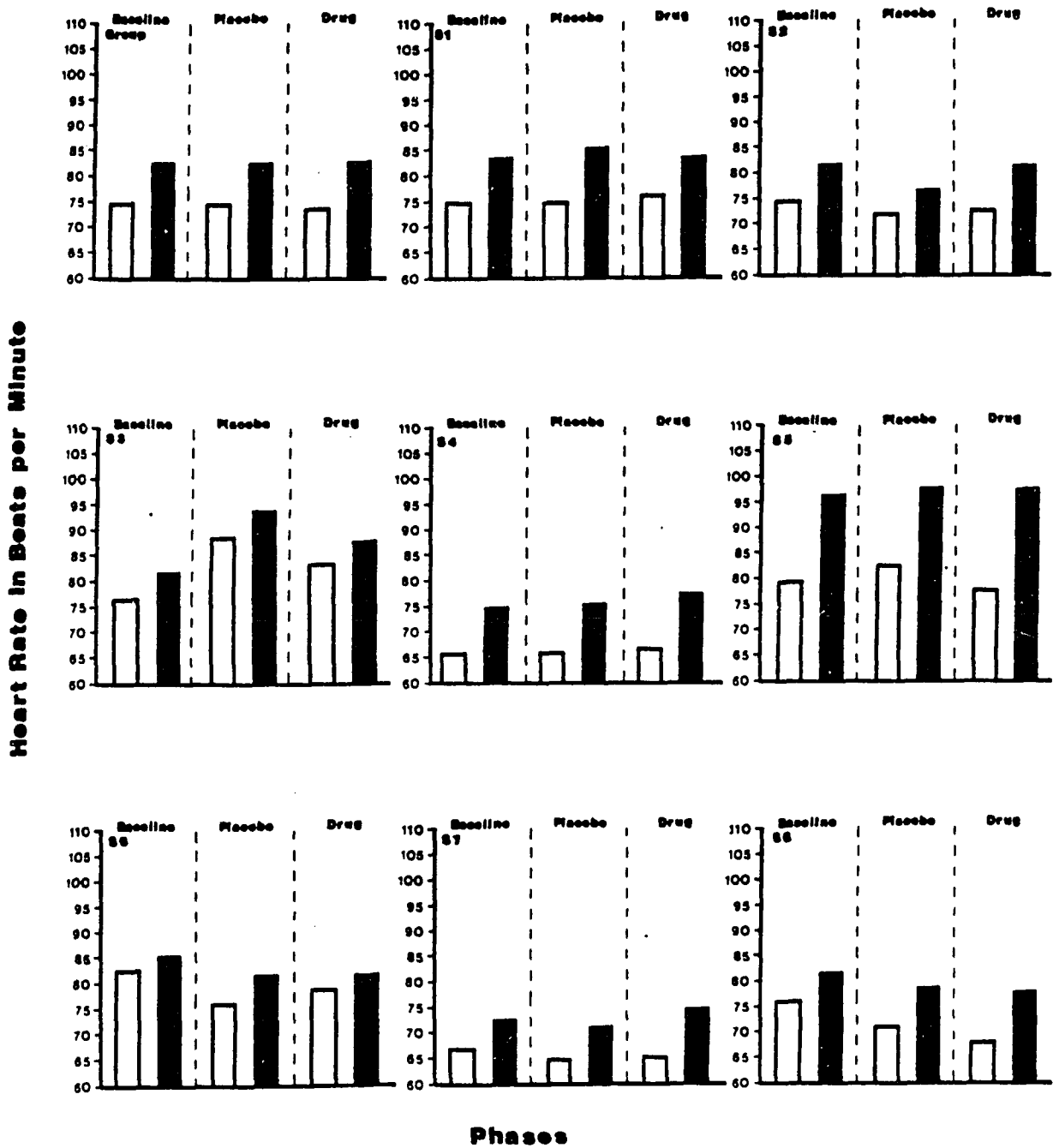


Figure 3. Mean Changes in Heart Rate From Rest to Stressor Periods Across Phases by Subject and by Group.

able to be made.

Statistical Analysis

Table 3 shows a summary of analysis of variance on HR comparing placebo and drug conditions between rest and stressor phases. Multivariate repeated measures analysis

Table 3

Summary of Analysis of Variance on HR

Source	Mean	SD	df	MS	F	p
Differences between conditions						
At rest	P= 74.3 D= 73.3	7.5 6.1	1	4.3	.08	.7842
At stress	P= 82.4 D= 82.5	8.7 6.8	1	.04	.001	.9815
Overall differences						
Across phases			1	604.7	9.81	.0040
Across condition			1	1.8	.03	.8689
Main effects			2	303.2	4.92	.0148

P = Placebo condition

D = Drug condition

of variance were conducted comparing overall rest and stressor phases and overall placebo and drug conditions. As with SBP and DBP between group data comparing caffeine to placebo levels within a phase (rest and stress) show

no significant effect.

Multivariate repeated measures analysis of variance across phases (rest to stressor phase) were significant while those same measures across condition (placebo to drug) were not significant.

Table 4

Summary of Analysis of Variance on EDG

Source	Mean	SD	df	MS	F	p
Differences between conditions						
At rest	P= 12.7 D= 16.4	7.7 6.4	1	55.9	.98	.3506
At stress	P= 15.9 D= 19.8	8.6 7.2	1	60.8	.85	.3833
Overall differences						
Across phases			1	92.1	1.42	.2438
Across condition			1	120.5	1.85	.1841
Main effects			2	106.3	1.64	.2128

P = Placebo condition
D = Drug condition

Additional Physiological Measures

The additional physiological measures results were not displayed graphically because significant effects on these measures were not obtained.

Statistical Analysis

Table 4 and Table 5 summarize an analysis of variance on EDG and EMG comparing placebo and drug conditions between rest and stressor phases.

Table 5

Summary of Analysis of Variance on EMG

Source	Mean	SD	df	MS	F	p
Differences between conditions						
At rest	P= 2.6	0.8				
	D= 3.1	1.0	1	1.2	1.21	.2901
At stress	P= 5.2	1.5				
	D= 5.3	1.9	1	.01	.002	.9684
Overall differences						
Across phases			1	45.12	20.85	.0001
Across condition			1	.66	.31	.5907
Main effects			2	22.89	10.58	.0004

P = Placebo condition
D = Drug condition

Multivariate repeated measures analysis of variance were conducted between rest and stressor phases and between placebo and drug conditions.

Statistical analysis of all data shows no significant effects in any comparison except for overall

differences across phases for EMG. This suggests that neither caffeine nor stress have any significant effects on these physiological measures. But one must be cautioned due to small sample size and high between-subject variability.

Additional Graphic Analysis

Figure 4 displays mean SBP and DBP levels along with HR levels during rest and stress periods across experimental sessions for subject 5. These data were chosen due to the visual clarity of the findings. One can observe the distinct separation of physiological measures associated with each condition and each phase. The graphs displaying the other subjects results were more difficult to discriminate individual effects. As depicted in Figure 4, stressor phases show increases in the physiological measures being assessed. This figure also displays drug conditions as increases (although smaller) in the measures as well. Other individuals showed larger effects of stress or caffeine on some physiological measures but the presence of substantial between-subject variability precludes generalizations across subjects.

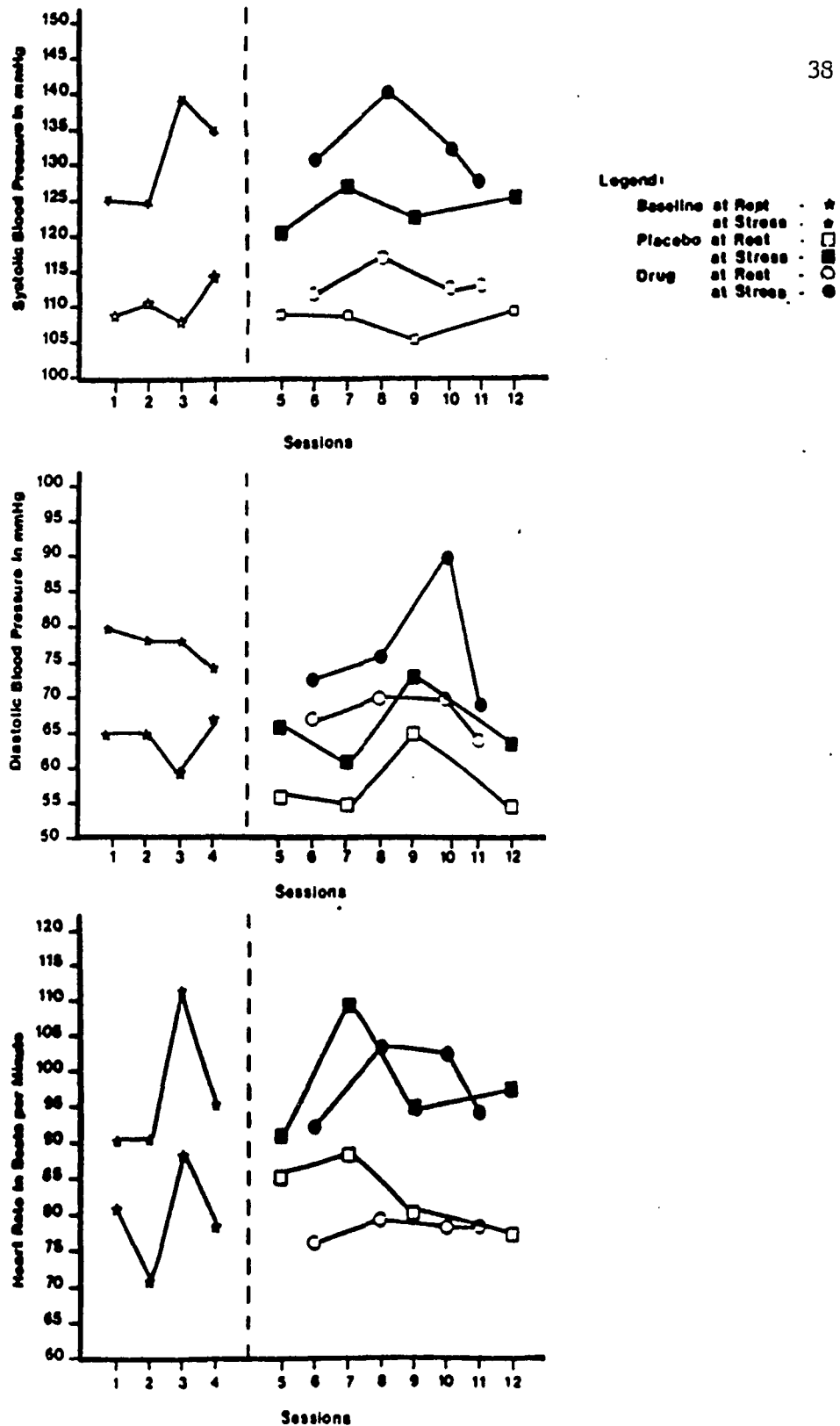


Figure 4. Mean Systolic and Diastolic Blood Pressure Levels and Mean Heart Rate Levels During Rest and Stress Periods Across Experimental Sessions for Subject 5.

Self-Report Measures

Participant Information Form

Only small changes were reported by all subjects in the following areas throughout the entire study: alcohol, caffeine, tobacco and sodium consumption; daily stress, exercise and weight. None of these systematically varied with presentation of the independent variable.

Caffeine Consumption Checklist

With two exceptions, all subjects reported abstinence from caffeine for 24 hours prior to each session. On the two occasions in which a subject had consumed caffeine, the session was rescheduled to allow for abstinence to take place. The checklists provided a broad enough basis as to cover all areas of possible caffeine ingestion.

Client Feedback Form

All subjects filled out Client Feedback Forms as to how stressful each of the role plays was. Each subject was also asked to rate how typical each of the situations was, how typical the subject's reactions to the situations were, and how typical the actors responses in the situations were. Table 6 displays mean ratings from a 5

point rating scale (1 = not stressful/typical to 5 = very stressful/typical) per subject pertaining to each category.

The subjects perceived the situations to be just over average stressfulness (mean 3.5). The situation (mean 3.2), subjects reaction (mean 3.9) and actor's response (mean 3.3) were all within the average range as well. These data suggest that the roleplayed interactions were not unlike natural situations and could thus be considered similar to daily environmental responses.

Table 6
Summary of Client Feedback

Category	S1	S2	S3	S4	S5	S6	S7	S8
How Stressful	4.1	3.0	3.6	3.3	3.3	3.3	3.3	3.7
Typical Situation	4.5	2.4	3.3	3.5	3.0	2.6	3.0	3.2
Typical Reaction	4.4	3.9	3.7	4.0	4.2	3.2	4.3	3.7
Actors Response	4.3	2.4	3.0	3.8	3.9	2.1	3.2	3.6

Caffeine Ingestion Estimates

At the end of each experimental session the subjects were asked to determine if they had ingested caffeine in

the cocktail. The subjects were scored for the accuracy of their estimates. Table 7 displays the results of these estimates by subject and the mean of all subjects.

Inaccurate reports of ingestion seemed to come early in the experimental phases with most disagreement being false positives. As sessions progressed accuracy regarding detection of caffeine ingestion improved. This may suggest an added awareness of physical sensations as the sessions progressed.

Table 7

Summary of Caffeine Ingestion Estimates

Subject	Agreement	Percentage Agreement
#1	6	75%
#2	7	88%
#3	6	75%
#4	8	100%
#5	8	100%
#6	5	63%
#7	7	88%
#8	6	75%
Mean	6.6	83%

Subjects were also asked immediately after inges-

tion of the beverage whether or not they had just consumed caffeine. Out of 64 ingestions of the beverage only 23 beverages were correctly identified as to whether or not they had contained caffeine. The fact that this identification accuracy is below chance responding (32%-50%) suggests that the beverage vehicle was adequate to mask caffeine.

CHAPTER IV

DISCUSSION

This study attempted to assess the effects of caffeine consumption on cardiovascular reactivity to laboratory simulations of stressful social interactions. Such simulations are assumed to be more representative of naturalistic stressors than the types of stressful tasks (e.g., mental arithmetic, cold pressor, video game tasks) utilized in previous studies (Lane, 1983; Robertson et al., 1984).

The data on the effect of stress on SBP show an average increase of 8.1 mmHg (112.5 (stress) - 104.4 (rest)) during the placebo condition and an average increase of 8.9 mmHg (118.0 (stress) - 109.1 (rest)) during the drug condition. This suggests that stressful social interactions increase SBP both in the caffeine and placebo condition.

The data also show an overall increase in SBP under the caffeine condition an average of 5.5 mmHg (118.0 (caffeine) - 112.5 (placebo)) in the stress phase and an average of 4.7 mmHg (109.1 (caffeine) - 104.4 (placebo)) in the rest phase. This suggests that caffeine elevates SBP both at rest and during stressful situations.

Although the effects of caffeine do seem to increase

SBP levels, it does not seem to increase the cardiovascular reactivity which is the absolute amount of change between stress and rest phases. This can be determined by looking at the average increases during the stress phases under both caffeine and placebo condition which remain constant.

When tested statistically, the stressor phase increases of SBP are shown to be significant while the caffeine consumption condition increases are not significant.

The data on the effects of stress also show an average 6.3 mmHg increase in DBP (72.9 (stress) - 66.6 (rest)) during the placebo condition and an average 6.5 mmHg increase in DBP (79.4 (stress) - 72.9 (rest)) during the drug condition. This again, suggests that stressful social interactions increase DBP both in the caffeine and placebo condition.

An average of 6.5 mmHg increase in DBP (79.4 (caffeine) - 72.9 (placebo)) is evident in the stress phase as is an average of 6.3 mmHg increase in DBP (72.9 (caffeine) - 66.6 (placebo)) during the rest phase, showing an overall increase in DBP in the caffeine condition. These data suggest that caffeine elevates DBP both at rest and during stressful situations.

As with SBP, the effects of caffeine seem to increase overall DBP levels, but caffeine does not seem

to increase the cardiovascular reactivity between stress and rest.

When tested for statistical significance, both the stress phase and the caffeine condition were proven significant suggesting that results seen were most likely not due to chance but were due to the experimental manipulation of stress and rest phases and drug and placebo conditions.

These findings are relevant to the hypothesis of Henry and Stephens (1980). Henry and Stephens hypothesize a synergistic interaction of caffeine and stress on the cardiovascular system, suggesting a combined effect greater than the sum of the effects. Results from this study show no synergistic interaction. But cautiously, due to lack of statistical significance, this researcher suggests that an additive effect may be present, wherein the elevation from the stress phase adds to the elevation of the caffeine condition to create a higher elevation than possible under either variable in isolation.

There were no changes evidenced in HR between drug and placebo conditions. This replicates the findings of Lane (1983), Lane and Williams (1985, 1987), and Robertson et al. (1984). In those studies as with this study, increases in HR were observed between stress and rest phases suggesting reactivity to stressful situations. The mechanisms proposed to account for HR increases in a

stressful situation do not seem to be further affected by caffeine.

A note of caution must accompany the results of this study. Although individual data show increases in individual subject's SBP and DBP in response to caffeine ingestion during both the stressor and rest phases, one must consider the lack of statistical significance for the effects of caffeine. This lack of significance suggests that the effects of caffeine may be due to chance rather than manipulation of drug condition and stressor phase. The lack of results could also be due to small subject number and high individual variability. With additional subjects statistical significance might have been obtained.

Although conclusions regarding the effects of caffeine on BP and reactivity are limited due to the lack of statistical significance, the possibility of clinical significance remains. For a patient in the borderline hypertension category (140/90 mmHg) who consumes caffeine and is exposed to stressful situations; the increases in BP shown to occur in this study could potentially place the person in the hypertension category (160/95 mmHg). This risk, although pertinent to any person being exposed to both stressful situations and caffeine, is more problematic for individuals already at a higher BP level.

Those individuals placed in the borderline hypertension

category tend to receive the least amount of benefit from pharmacological intervention for hypertension (Agras & Jacob, 1979). This reduction in caffeine intake and stress reduction/elimination could prove especially beneficial on a long term basis for such individuals who are not candidates for medication-based management of hypertension. Caffeine reduction/withdrawal could also prove beneficial as an adjunct to medical regimens, possibly adding to the medications effectiveness in decreasing BP. Any BP decrease in the patient over time could then, theoretically decrease the risk of developing CHD (Sue et al., 1986).

One must consider procedural variables when evaluating the results of this study. The population was composed of, for the most part, college professors, students and professionals in the field of Psychology. Skills, such as assertiveness, progressive muscle relaxation, social skills and problem-solving, obtained in educational pursuits and professional practice may have alleviated some reactivity to the simulated stressors although there is no direct evidence to verify this because application of these skills during the session was not directly assessed or controlled for in this study. Two subjects (S2, S5) coming from different backgrounds did exhibit more reactivity. These particular skills (e.g., progressive muscle relaxation, assertive-

ness, social skills, problem-solving) attained from educational and professional pursuits may play a part in attenuating cardiovascular reactivity (Fettes, 1986; Pinto, 1987). Further research might evaluate the importance of utilizing these skills during stressful situations.

Another variable that could have accounted for some between subject variability is the level of tolerance for caffeine that subjects might have been exhibiting. Five subjects (S3, S4, S6, S7, S8) were moderate caffeine users (not more than 300mg of caffeine daily) while the other three subjects (S1, S2, S5) were basically abstinent. Subjects abstained from caffeine for 24 hours prior to each session. Lane (1983) suggests that withdrawal from caffeine overnight is sufficient to reduce tolerance effects on cardiovascular reactivity. No significant differences in BP were evident between subjects who were moderate caffeine users and subjects who had been previously abstinent. But to further rule out possible tolerance effects, varied periods of abstentions as well as different levels of previous use should be more closely examined for their effect on resting levels of BP and reactivity to stress.

Based on the similarity between the role plays and naturally-occurring stressful events, it is assumed that stressful social interactions are more naturalistic than

mental arithmetic tasks and that results will generalize to the natural environment. This could be empirically tested by manipulating both social interactions and mental arithmetic tasks in a future study to determine comparative effects. Social interactions are also more difficult to control because of the unique nature of each interaction. Due to the inherent uniqueness evidenced in socially stressful interactions, differences in individual reactivity may have occurred. The factors that merit further research to determine their effects on physiological reactivity to a role play session include: (a) the effectiveness of individual actors/actresses to the role play situations, (b) the inherent variables involved in social interactions, such as verbalizations, affect, possible cognitions (positive/negative) and the role each plays on the an individual subject, (c) the nature of the stressor involved in each role play and potential effect on each subject, and (d) extraneous current variables in a subject's personal environment that may affect role play interactions. These elements all deal with the way a uniquely stressful situation is handled both by the actor/actress and by the subject. When dealing with social interactions, an individual's learning history must be taken into account. A more thorough assessment of historical variables such as previous interactions, individual stressors, methods of

dealing with stress, may prove to be beneficial for more realistic representation. One must also look at the extraneous current variables that may affect the interaction. Asking the subject at the time of each session if there are any variables (e.g., stress, emotions, outstanding problems) currently influencing his/her interactions may better define experimental effect. Further research isolating some of these components of social interaction may be able to determine how an individual's history and current environment play a role in immediately stressful situations.

Along the same dimension, it could be argued that the social interactions are too contrived to be useful, thus posing a threat to external validity. The subjects in this study were not allowed to resolve the conflict, with the role player being instructed to continue with the aversive situation for the entire time period. Whether the inability to resolve or terminate the conflict increased reactivity to a level that is not representative of naturally occurring stressors is a topic for further research. While this is a possibility, the following precautions were undertaken. Self-report measures along with personal interviews were administered to individualize stressful situations for each client. In addition, the Participant Feedback Form (Appendix E) was administered after every session to determine how

realistic the role plays had been.

Along the same line, the repetition of the social situations may have allowed for habituation thereby decreasing the amount of reactivity to the role play. The results showed relatively constant levels of reactivity to stressors throughout the study, thus suggesting that habituation was only a minor concern. Further work could be done on creating more realistic and novel stressful situations to avoid habituation and artificiality of the situation. One way that this might be achieved could be through some type of ambulatory monitoring that would assess degree of reactivity during natural daily stressors thereby helping to rule out artificiality and possible habituation.

The actors/actresses may not have been equally stressful. Although attempts were made to standardize the "stressfulness" of the performances and no major differences in the characteristics of the performance between actors or across sessions were subjectively noted by experimenters, there may well have been variations present. These variations in performance could lead to alterations in the indices measured. This possibility seems remote in that seven different actors/actresses were utilized with interactions being based on each subject's self-report of stressful situations.

One additional finding of this study deserves a

final note. When asked to report physical sensations, three subjects reported an elevated activity level (e.g., physiological arousal) and increased verbal response (e.g., loudness, shorter latency) level when they thought that they had ingested caffeine. Conversely they felt tired, and at low energy levels without caffeine. These sensations were reported after each stressor. It is unclear whether perceptions of caffeine ingestion, whether accurate or inaccurate, might have influenced subject performance in the role plays in a placebo-like effect. Further study of the phenomena of "cognitive anticipation" would be beneficial in that it may play a role in how an individual perceives that he/she should interact in a situation.

None of the subjects was able to discriminate increases and decreases in BP during rest and stress periods. When asked what their blood pressure at the end of each rest and stress phase was, there was no accurate estimates of the reading or of the increase/decrease in level. In essence, the subjects detected physiological effects under each condition but were unable to correlate those changes with autonomic responses.

In summary, these results are important for two reasons. First, if an individual has elevated BP and consumes caffeine, reducing or eliminating caffeine should decrease BP levels. Second, if an individual who

consumes caffeine is under stressful conditions that can not be altered, reduces or eliminates caffeine, the individual may reduce the effects of stress on BP. Caffeine has been postulated to play a role in development and maintenance of hypertension (Sue et al., 1986) and through this study has been shown to elevate both resting and at stress SBP and DBP. This researcher is not claiming that caffeine causes hypertension or that avoidance of caffeine will alleviate hypertension. But if implementation of a health program to control or prevent hypertension is of prime concern, reduction or elimination of caffeine might be a component to be considered.

To determine caffeine's role in cardiovascular reactivity, hypertension and CHD, one must continue to manipulate environmental variables that best exemplify naturalistic situations. Tolerance issues must be addressed more carefully. Both historical and current individual variables must be taken into account to help improve on internal validity. Research should incorporate higher levels of caffeine presentation due to the number of caffeine users who exceed the 300 mg dosage daily and may represent a larger population to be studied. Finally, research should also be extended to children where there seems to be a paucity of information available. Many children are consuming large amounts of

caffeine in soft drinks and chocolate. The effects on children should be monitored and evaluated both from a physiological and performance perspective. Caffeine is a drug that can effectively be dealt with when its effects become empirically known.

APPENDICES

Appendix A
Informed Consent

Western Michigan University

Department of Psychology

You are invited to participate in a research study on the body's reactions to stress with caffeine ingestion. We hope to learn if caffeine ingestion compounds the blood pressure elevations commonly experienced during various types of psychological stress.

As a participant, you will first be asked to complete two questionnaires concerning the degree and types of stress in your everyday life. We will then ask you to abstain from caffeine for 24 hours prior to each of the 11 to 15 assessment sessions that you will attend. In each of those sessions, we will measure your heart rate, blood pressure, muscle tension and skin electrical activity during mild stress tasks comprised of three minute role plays depicting naturalistic situations. The equipment used to perform these measurements is non-invasive and painless. Thirty minutes prior to each session you will drink a juice cocktail which may or may not contain caffeine. These sessions will be one hour in length, with attendance of approximately 2 to 3 session per week. You will be paid \$3.00 per assessment session.

The research involves minimal risk to you. The caffeine will be limited to 300 mg. which is equivalent to less than 3 cups of dripolated coffee. The stress tasks are mild and not unlike the situations you experience in everyday life. For example, you might be asked to role play a situation in which you are late for a meeting and waiting in line to purchase an item. Another person steps in front of you. You will then be asked to confront that person about the situation. In the unlikely event that you become extremely stressed, the tasks will be immediately terminated and relaxation exercises commenced. Other than the caffeine abstention prior to each session, we ask that you make no major diet or exercise changes in your life that may influence the results. In addition to payment, you may experience the added benefit of a heightened awareness of how your body reacts to caffeine consumption and social stress. You will also have the opportunity to participate in a volunteer program to help you decrease your caffeine consumption, at the conclusion of the investigation.

Information obtained in this study will be confidential with access restricted to experimenters. By signing this Informed Consent document, you give permission for

Appendix B

**Human Subjects Institutional Review Board
Application and Approval**

Protocol #:

DIRECTIONS: Please type or print the requested information - except signatures. You may attach additional sheets as necessary, and reference the appropriate page.

PRINCIPAL INVESTIGATOR: R. Wayne Fugua, Ph.D. Date October 29, 1986

TITLE OF PROJECT: Effects of caffeine consumption on cardiovascular reactivity to social stress

ABSTRACT: Briefly describe the purpose, research design, and site of the proposed research activity.

Reducing caffeine consumption may be effective in attenuating cardiovascular reactivity to stress. This study will examine the effects of caffeine consumption on blood pressure reactivity to a group of laboratory stress tasks developed to approximate stress in the natural environment.

Subjects will be assessed under caffeine and no caffeine states while being presented with individual stressors. These stressors (role plays) will be presented in the initial assessment and following phases of the study. Novel stressor probe trials will help disclose any generalization effects to situations not specifically used in initial assessment.

A per-session alternating-treatment design across subjects will be utilized. Caffeine will be introduced randomly in a double-blind schedule with each subject being exposed to the same amounts of caffeine over time. The research site will be the Behavior Medicine Laboratory Wood Hall, Western Michigan University, where equipment to monitor physiological reactivity is located.

CHARACTERISTICS OF SUBJECTS: Briefly describe the subject population (e.g., age, sex, prisoners, people in mental institutions, etc.). Also indicate the source of subjects.

Eight to twelve subjects interested in obtaining better awareness of what effect caffeine consumption has on their cardiovascular systems will participate in this study. Previous research does indicate that reducing caffeine consumption could attenuate cardiovascular reactivity to unpleasant social situations. Subjects will be recruited by means of public advertisement, from the community.

SUBJECT SELECTION: How will the subjects be selected? Approximately how many subjects will be involved in the research?

The following inclusion criteria will be used: 1) reactivity to laboratory stressors (mean blood pressure change of 10% from rest periods), 2) evaluation of consuming no more than 300 mg. caffeine daily, 3) agreement to abstain from all sources of caffeine for 24 hours prior to each session, 4) agreement to make no major changes in diet or exercise over the course of the research study. Subjects who display blood pressure readings greater than 160mmHg (systolic blood pressure) and 95mmHg. (diastolic blood pressure) will be excluded from the study

CONFIDENTIALITY OF DATA: Briefly describe the precautions that will be taken to ensure the privacy of subjects and confidentiality of information. Be explicit if data is sensitive.

Subjects will sign an informed consent form stating that data may be used in scientific presentations and publications provided that subjects' identities remain confidential. Each subject will be assigned a letter for purposes of data identification - no data will reflect the subject's name. Actors participating in the study will also be required to sign a confidentiality of information form. Records will be stored in a secure place and treated with standard confidentiality.

BENEFITS OF RESEARCH: Briefly describe the expected benefits of the research.

Subjects will be paid \$3.00 per assessment session for participation. Indirect benefits from added awareness of caffeine and social stressor effects are expected. A voluntary program to decrease caffeine consumption will be offered at the conclusion of investigation.

RISKS TO SUBJECTS: Briefly describe the nature and likelihood of possible risks (e.g., physical, psychological, social) as a result of participation in the research.

Minimal risk is involved. Caffeine ingestion will be limited to 300 mg. per session, which is equivalent to 3 cups of brewed coffee. The proposed stressors are mild and not unlike situations experienced in everyday life. The equipment used to monitor the physiological levels is non-invasive, painless and electrically safe. A subject could conceivably overreact either physiologically or psychologically to a particular stress, although the likelihood of this risk is extremely low.

PROTECTION FOR SUBJECTS: Briefly describe measures taken to protect subjects from possible risks, if any.

If physiological levels approach extreme levels, or if a subject appears to be experiencing unwarranted anxiety, all stressors and monitoring will cease. A session will be terminated if blood pressure readings exceed 200/110 mmHg. Relaxation techniques will be implemented. An extensive debriefing will also follow. Continued participation in the investigation will be determined by the subject and the experimenter. Equipment operators will be trained to recognize and prevent such a situation. Operators will also be trained in cardiopulmonary resuscitation as an added precaution.

INFORMED CONSENT: Please attach a copy of the informed consent form. If oral consent will be obtained, describe procedures for obtaining and documenting such consent. (Subject should be given a copy of the consent form).

Please see attached.

QUESTIONNAIRES OR INTERVIEW SCHEDULES: If questionnaires, interview schedules or data collection instruments are used, please identify them and attach a copy of what will be used in the project.

Two psychological self-report scales will be administered prior to the treatment phase: Jenkins Activity Scale by Jenkins, Zyzanski and Rosenman (1979) and the Hassles and Uplift Scale by Kanner, Coyne, Schaefer and Lazarus (1981). These self-report measures will help identify individual natural stressors which will later serve as the basis for role play situations.

Reviewed:

Western Michigan University
Human Subjects Institutional Review Board
Human Subjects Approval Form

Protocol #:

Received:

DIRECTIONS: Please type or print each response - except signatures. Refer to the Western Michigan University Policy for the Protection of Human Subjects to determine the appropriate level of review.

PRINCIPAL INVESTIGATOR R. Wayne Fugua, Ph.D. DEPARTMENT Psychology
Home Phone 375-1030 Office Phone 383-6052
Home Address 405 N. 8th Office Address 312 Wood Hall
Kalamazoo, MI Western Michigan University

PROJECT TITLE: Effects of caffeine consumption on cardiovascular reactivity
to social stress

SUBMISSION DATE: _____ PROPOSED PROJECT DATES 1/5/97 TO 4/25/97

Note: The principal investigator should not initiate the research project until the protocol has been reviewed and approved by the Human Subjects Institutional Review Board.

APPLICATION IS: New Renewal Continuation Supplement

SOURCE OF FUNDING: _____
(if applicable)

Signature of Investigator

STUDENT RESEARCH (Fill out if applicable.)

Name of Student Suzanne L. Keller Phone 349-3323 Address 1624 Texel Dr. Kalamazoo

The research is: Undergraduate Level Graduate Level

Faculty Advisor R. Wayne Fugua, Ph.D. Department Psychology

Signature of Faculty Advisor R. Wayne Fugua Phone 383-6052

VULNERABLE SUBJECT INVOLVEMENT (Fill out if applicable.)

Research involves subjects who are: (check as many as apply)

- | | |
|--|--|
| 1. <input type="checkbox"/> children
approximate age _____ | 6. <input type="checkbox"/> Other subjects whose life
circumstances may interfere
with their ability to make
free choices in consenting
to take part in research |
| 2. <input type="checkbox"/> mentally retarded persons
<input type="checkbox"/> check if institutionalized | _____ |
| 3. <input type="checkbox"/> mental health patients
<input type="checkbox"/> check if institutionalized | _____ |
| 4. <input type="checkbox"/> prisoners | _____ |
| 5. <input type="checkbox"/> pregnant women | (Describe Please) |

HSIRB

Protocol #: _____

Received: _____

LEVEL OF REVIEW: Please indicate here if you think that the research project is exempt from review, subject to expedited review, or subject to full review.

Exempt (Forward 1 application to IRB Chair)

Which category of exemption applies? # _____

Expedited (Forward 3 applications to IRB Chair)

Subject to full IRB review (Forward 9 applications to IRB Chair)

Comments:

Your application was reviewed and the Human Subject Institutional Review Board (HSIRB) has determined that:

1. The proposed activities, subject to any conditions and/or restrictions indicated in Remarks below, have (a) provided adequate safeguards to protect the rights and welfare of human subjects involved, (b) established appropriate procedures and/or documents to obtain informed consent, and (c) demonstrated that the potential benefits of the research substantially out-weigh the risks.
2. The proposed activities, for reasons indicated in Remarks below do not provide adequate protection for the rights and welfare of the human subjects.

At its meeting on _____, the HSIRB (approved) (provisionally approved... see remarks) this application with regard to the treatment of human subjects. The HSIRB categorized this application as:

1. Involving subjects at no more than minimal risk.
2. Involving subjects at more than minimal risk.

REMARKS:

Signature HSIRB Chair

Date



Western Michigan University
Kalamazoo, Michigan 49008-3899

*Human Subjects
Institutional Review Board*

TO: R. Wayne Fuqua
Suzanne L. Keller

FROM: Ellen Page-Robin, Chair *EP-R*

RE: Research Protocol #87-01-01

DATE: January 14, 1987

This letter will serve as confirmation that your research protocol, "Effects of Caffeine Consumption on Cardiovascular Reactivity to Social Stress," has been approved by the HSIRB.

As the Board discussed with you, you may not need a statement from a physician if a statement saying that the subject has never been told to avoid caffeine for medical reasons is included in the consent form. If you choose to use a physician, we would like a copy of the form you give him/her.

Also, the Board requests that an example of a stressor be included in the informed consent.

Please send a copy of the revised informed consent for your file.

If you have any questions, please contact me at 383-4917.

Appendix C

Participant Information Form

PARTICIPANT INFORMATION FORM

Among the factors that are thought to influence blood pressure are: exercise, body weight, blood pressure medication, excessive consumption of salt, caffeine or alcohol and stress. We are requesting that you make no systematic changes in any of the above listed factors that can influence blood pressure during the course of this study except for caffeine abstinence 24 hours prior to session as such changes could influence the outcome of the study. If you make life style changes that could influence your blood pressure, please notify the experimenters. Additionally, please complete the attached questionnaire concerning blood pressure risk factors. Information that you provide on this form will be held in the strictest of confidence. The identity of individual participants will be completely protected in any publications or presentations emanating from this research.

NAME: _____ Date of Birth: _____

Address: _____ Phone: _____

Physician's

Name: _____

1. Have you ever been told that your blood pressure was high?
yes _____; no _____. If yes, how long ago? _____
2. Have you ever taken medication for high blood pressure?
yes _____; no _____. If yes, what and how often?
3. Do you currently take medication for high blood pressure?
yes _____; no _____. If yes, what and how often?

4. Do any of your family members or close relatives have high blood pressure or heart disease?
yes _____; no _____.
5. Has any close relative died of stroke, heart failure or kidney failure before age 65? yes _____; no _____.

Please read and sign the following statement: I understand that systematic alterations in my diet, blood pressure medication, exercise, body weight, and levels of stress may influence my blood pressure and obscure the results of this experiment. I agree to hold these factors constant to the best of my ability and to notify the experimenters should unavoidable changes occur.

Signature

Date

Appendix D

Caffeine Consumption Checklist

CAFFEINE CONSUMPTION CHECKLIST

Please list the items and the amounts of each item that contained caffeine that you have consumed in the last 24 hours. Be specific on the amounts.

1. How much coffee did you consume:

Instant regular	_____
Instant decaf.	_____
Instant freeze dried	_____
Percolated regular	_____
Percolated decaf.	_____
Dripolated regular	_____
Dripolated decaf.	_____

2. How many ounces were in each cup of coffee? _____

3. How much tea did you consume:

Regular tea, bagged	
brewed 1 minute	_____
brewed 3 minutes	_____
brewed 5 minutes	_____
Instant iced tea	_____
Herb tea	_____

4. What brand of tea did you consume? _____

5. How many ounces were in each cup/glass of tea?

6. How many bottles/cans of soft drinks did you consume:

Coca Cola	_____
Pepsi Cola	_____
Pepsi Light	_____
Dr. Pepper	_____
Mr. Pibb	_____
Mountain Dew	_____
Mello Yellow	_____
Tab	_____
Sunkist Orange	_____
Royal Crown Cola	_____
Jolt Cola	_____

Others: _____

7. How many total ounces of soft drinks did you consume: _____

8. How much chocolate did you consume:

Hot chocolate _____
 Chocolate milk _____
 Milk chocolate _____
 Baking chocolate _____

9. Are there any other foods/drinks that you consumed that contained chocolate? _____ If so, what were they? _____

10. How much chocolate did you consume in total? _____

11. Did you take any non-prescription drugs that contain caffeine:

No Doz _____
 Vivarin _____
 Anacin _____
 Midol _____
 Dristan _____
 Sinarest _____
 Coryban-D _____
 Caffedrine _____
 Other: _____

12. How many pills/capsules did you ingest? _____

13. Are there any other items that you consumed in the last 24 hours that you know contain caffeine. Please list the item and the amount below.

To the best of my knowledge the above is an accurate accounting of the items and amounts of caffeine that I have consumed in the last 24 hours.

Signature

Date

Appendix E

Participant Feedback Form

CLIENT FEEDBACK FORM

CLIENT#

To help us to assess the personal relevance of the interactions represented in the role plays you have participated in, please circle the appropriate response categories below:

1. How stressful was role play #1? 1 2 3 4 5
Not Very
Stressful Stressful

How stressful was role play #2? 1 2 3 4 5
Not Very
Stressful Stressful

How stressful was role play #3? 1 2 3 4 5
Not Very
Stressful Stressful

2. Was role play #1 typical of situations you generally encounter?

1 2 3 4 5

Not typical Very typical

Was role play #2 typical of situations you generally encounter?

1 2 3 4 5

Not typical Very typical

Was role play #3 typical of situations you generally encounter?

1 2 3 4 5

Not typical Very typical

3. Were your responses in role play #1 similar to your responses to such situations in everyday life?

1 2 3 4 5

Not similar Very similar

BIBLIOGRAPHY

- Agras, S., & Jacob, R. (1979). Hypertension. In O. F. Pomerleau & J. O. Brady (Eds.), Behavioral Medicine: Theory and Practice (pp.205-232). Baltimore: Williams & Wilkens.
- American Heart Association (1981). American Heart Association Heart Facts, 1982. Dallas, TX.
- Calhoun, W. H. (1971). Central nervous system stimulants. In E. Furchtgott (Ed.), Pharmacological and biophysical agents and behavior (pp. 181-268). New York: Academic Press.
- Curatola, P. W., & Robertson, D. (1983). The health consequences of caffeine. Annals of Internal Medicine, 98, 641-653.
- Davison, G. C., & Neale, J. M. (1986). Abnormal psychology (4th ed.). New York: John Wiley.
- Eisler, R. M., Hersen, M., Miller, P. M., & Blanchard, E. B. (1975). Situational determinants of assertive behaviors. Journal of Consulting Clinical Psychology, 43, 330-334.
- English, E. J., & Baker, T. B. (1983). Relaxation training and cardiovascular response to experimental stressors. Health Psychology, 2, 239-259.
- Fettes, P. (1986). Effects of instructions and cue controlled relaxation training on cardiovascular reactivity to social stressors. Unpublished master's thesis, Western Michigan University, Kalamazoo, MI.
- Gilbert, R. M. (1976). Caffeine as a drug of abuse. In R. J. Gibbins (Ed.), Research advances in alcohol and drug problems (pp. 49-176). New York: John Wiley.
- Glass, D. C., Krakoff, L. F., Contrada, R., Hilton, W. F., Kehoe, K., Manucci, E. G., Collins, C., Snow, B., & Elting, E. (1980). Effect of harassment and competition upon cardiovascular and plasma catecholamine responses in Type A and Type B individuals.

Psychophysiology, 17, 453-462.

Graham, D. M. (1978). Caffeine: its identity, dietary sources, intake and biological effects. Nutritional Review, 36, 97-102.

Greenberg W., & Shapiro, D. (1987). The effects of caffeine and stress on blood pressure in individuals with and without a family history of hypertension. Psychophysiology, 24, 151-155.

Henry, J. P., & Stephens, P. M. (1980). Stress, health, and the social environment: a sociobiologic approach to medicine. New York: Springer-Verlag.

Herd, J. A., & Weiss, S. M. (1984). Overview of hypertension: Its treatment and prevention. In J. D. Matarazzo, S. M. Weiss, J. A. Herd, & N. E. Miller (Eds.), Behavioral health. A handbook of health enhancement and disease prevention (pp. 789-805). New York: John Wiley.

Izzo, J. L., Ghosal, A., Kwong, T., Freeman, R. B., Jaenike, J. R. (1983). Age and prior caffeine use alter the cardiovascular and adrenomecullary responses to oral caffeine. American Journal of Cardiology, 53, 918-922.

Jenkins, C. D., Zyzanski, S. J., & Rosenman, R. H. (1971). Progress toward validation of a computer scored test for the type A coronary prone behavior pattern. Psychosomatic Medicine, 33, 193-202.

Kallinke, D., Kulick, B., & Heim, P. (1982). Behavior analysis and treatment of essential hypertension. Journal of Psychosomatic Research, 26, 541-549.

Kanner, A. D., Coyne, J. C., Schaefer, C., & Lazarus, R. S. (1981). Comparison of two modes of stress measurement: Daily hassles and uplifts versus major life events. Journal of Behavioral Medicine, 4, 1-39.

Krantz, D. S., & Manuck, S. B. (1984). Acute psychophysiological reactivity and risk of cardiovascular disease: A review and methodological critique. Psychological Bulletin, 96, 435-464.

Kuznicki, J. T., & Turner, L. S. (1985). The effects of caffeine on caffeine users and non-users. Physiology & Behavior, 37, 397-408.

- Lacey, J. I., & Lacey, B. C. (1958). Verification and extension of the principle of autonomic response-stereotype. American Journal of Psychology, 71, 50-73.
- LaCroix, A. Z., Mead, L. A., Liang, K., Thomas, C. B., & Pearson, T. A. (1986). Coffee consumption and incidence of coronary heart disease. The New England Journal of Medicine, 315, 977-982.
- Lane, J. D. (1983). Caffeine and cardiovascular responses to stress. Psychosomatic Medicine, 45, 447-451.
- Lane, J. D., & Williams, R. D. (1985). Caffeine affects cardiovascular responses to stress. Psychophysiology, 22, 648-655.
- Lane, J. D., & Williams, R. D. (1987). Cardiovascular effects of caffeine and stress in regular coffee drinkers. Psychophysiology, 24, 157-164.
- MacDougall, J. M., Dembroski, T. M., & Krantz, D. S. (1981). Effects of types of challenge on pressor and heart rate responses in Type A and B women. Psychophysiology, 18, 1-9.
- Obrist, P. A. (1981). Cardiovascular psychophysiology: A perspective. New York: Plenum Press.
- Ohnishi, A., Branch, R. A., Jackson, K., Hamilton, R., Biaggioni, I., Deray, G., & Jackson, E. K. (1986). Chronic caffeine administration exacerbates renovascular, but not genetic, hypertension in rats. Journal of Clinical Investigation, 78, 1045-1050.
- Orton, I. K., Beiman, I., Ciminero, A. R. (1982). The behavioral assessment and treatment of essential hypertension. Behavioral medicine assessment and treatment strategies (pp. 175-198). New York: Plenum Press.
- Pinto, B. M. (1987). Effects of assertiveness training and cue controlled relaxation training on cardiovascular reactivity to social stressors. Unpublished master's thesis, Western Michigan University, Kalamazoo, MI.
- Robertson, D., Frolich, J. C., Carr, R. K., Watson, J. T., Hollifield, J. W., Shannel, D. G., & Oates, J.

- A. (1978). Effects of caffeine on plasma renin activity, catecholamines and blood pressure. The New England Journal of Medicine, 298, 181-186.
- Robertson, D., Hollister, A. S., Kincaid, D., Workman, R., Goldberg, M. R., Tung, C., & Smith, B. (1984). Caffeine and hypertension. American Journal of Medicine, 77, 54-60.
- Rosenberg, L. (1985). Breast cancer and the consumption of coffee. American Journal of Epidemiology, 122, 391-399.
- Rosenman, R. H., Brand, R. J., Jenkins, D. D., Friedman, M., Strauss, R., & Wrum, M., 1975. Coronary heart disease in the western collaboration group study: Final follow-up experience of 8 1/2 years. Journal of the American Medical Association, 233, 872-877.
- Shapiro, D., Lane, D. L., & Henry, J. P. (1986) Caffeine, cardiovascular reactivity, and cardiovascular disease. In K. A. Matthews, S. M. Weiss, T. Detre, T. M. Dembroski, F. Balkner, S. B. Manuck, & R. B. Williams (Eds.), Handbook of stress reactivity, and cardiovascular disease (pp.311-327). New York: John Wiley.
- Stephens, A., & Ross, A. (1982). Voluntary control of cardiovascular reactions to demanding tasks. Biofeedback and Self-Regulation, 7, 149-166.
- Sue, D., Sue, D., & Sue, S. (1986). Understanding abnormal behavior. Dallas: Houghton Mifflin.
- Sutherland, D. J., McPherson, D. D., Renton, K. W., Spencer, C. A., & Montague, T. J. (1985). The effect of caffeine on cardiac rate, rhythm, and ventricular repolarization. Chest, 87, 319-324.
- Twentyman, C. T., & McFall, R. M. (1975). Behavioral training of social skills in shy males. Behavior Therapy, 43, 54-59.
- Veleber, D. M., & Templar, D. I. (1984). Effects of caffeine on anxiety and depression. Journal of Abnormal Psychology, 93, 120-122.
- Watkinson, B. (1985). Maternal caffeine use before during and after pregnancy and effects upon offspring. Neurobehavioral Toxicology and Teratology, 7, 9-17.

- Weiner, H. (1979). Psychobiology of essential hypertension. New York: Elsevier.
- Weiss, B., & Laties, V. G. (1962). Enhancement of human performance by caffeine and the amphetamines. Pharmacological Review, 14, 1-36.
- Whitsett, T. L., Manion C. V., & Christensen, H. D. (1984). Effects of coffee and caffeine. American Journal of Cardiology, 53, 918-922.