THE DIFFERENTIAL EFFECTS OF HARASSMENT AND SEX ON CARDIOVASCULAR AND SALIVARY CORTISOL STRESS REACTIVITY AND RECOVERY

by

TRACEY LYNN EARLE

B.Comm., Dalhousie University, 1986

B.Sc.(Hon.), Dalhousie University, 1989

A THESIS IN PARTIAL FULFILLMENT OF

THE REQUIREMENTS FOR THE DEGREE OF

MASTER OF ARTS

in

THE FACULTY OF GRADUATE STUDIES

(Department of Psychology)

We accept this thesis as conforming

to the required standard

THE UNIVERSITY OF BRITISH COLUMBIA

September 1994

In presenting this thesis in partial fulfilment of the requirements for an advanced degree at the University of British Columbia, I agree that the Library shall make it freely available for reference and study. I further agree that permission for extensive copying of this thesis for scholarly purposes may be granted by the head of my department or by his or her representatives. It is understood that copying or publication of this thesis for financial gain shall not be allowed without my written permission.

Department of Psychology

The University of British Columbia
Vancouver, Canada

Date Oct. 6/94

Abstract

To explore the differential effects of harassment on cardiovascular and neuroendocrine stress reactivity and recovery, 28 males and 32 females were randomized to a harassment or no-harassment control condition (4 groups in total). The harassment consisted of 3 scripted statements delivered during performance of a mental arithmetic stress task. The harassing statements were delivered on a fixed schedule during the task by a same-sexed experimenter. Cardiovascular, salivary cortisol and subjective state affect measures were taken at baseline, immediately post-task and throughout an extended recovery period. On all measures it was found that subjects in the harassed condition exhibited larger stress reactivity responses compared to the non-harassed subjects. The harassed males showed the largest reactivity in the physiological measures, as well as significant changes on subjective measures. Compared to the harassed males, the harassed females showed a more pronounced response on the subjective state affect measure of hostility, and a smaller, but still significant cardiovascular reactivity response. The only cardiovascular index to show significant changes for any of the subjects was systolic blood pressure (SBP); diastolic blood pressure and heart rate showed similar trends as SBP, but did not quite reach statistical significance. The harassed males were the only group to show significant cortisol responses. Sex differences during the recovery periods were observed: the harassed males exhibited attenuated cardiovascular and cortisol recovery, while females, overall tended to exhibited an over-compensation response on cardiovascular measures. It may be that cardiovascular and cortisol sex differences in reactivity to emotional stressors, and recovery from, them indicate sex-specific pathways which may link negative affect (i.e., hostile or angry feelings) to development of cardiovascular disease.

TABLE OF CONTENTS

List of Tables

List of Figures

The Differential Effects of Harassment and Sex on Cardiovascular and Salivary Cortisol Stress Reactivity and Recovery

In our modern society, the concept of "stress" has received much attention. Since the seminal work of Hans Selye in the 1930's, stress has been thought to play a role in the development of many physical diseases (Selye, 1980). Stress can be more precisely defined as any physical, psychological, or social factor which is perceived as causing distress. The development and progression of cardiovascular diseases (CVD), for example, atherosclerosis and hypertension, are thought to be mediated by stress responses (Fredrikson & Matthews, 1990; Markovitz & Matthews, 1991; Manuck, Kasprowicz & Muldoon, 1990). Yet, despite decades of research, the etiologies of hypertension and other CVDs are not well understood. Myriad studies have established that various types of stressors, physical, psychological, and emotional, can elicit short-term cardiovascular and neuroendocrine reactions in both normotensive and hypertensive subjects (termed stress reactivity). However, the physiological mechanisms by which the cumulative effects of such stress reactivity can lead to sustained cardiovascular pathology are still largely speculative and widely debated (Eliot, 1988; Manuck, et al., 1990; Fredrikson & Matthews, 1990; Markovitz & Matthews, 1991; Pickering & Gerin, 1990).

One popular theory linking stress reactivity and CVD purports that enhanced cardiovascular stress reactivity is a risk marker for some other correlated factor that is itself the causal agent in the disease process (Manuck, et al., 1990). Another theory postulates a direct link between stress induced cardiovascular reactivity and cardiovascular disease. Taking hypertension as an example of cardiovascular disease, repeated activation of the sympathetic nervous system in response to numerous stressors in daily life causes frequent, and often extreme changes in hemodynamic parameters. Over time it is thought that hypertension develops as a

result of this 'over use', via either homeostatic mechanisms being reset to a higher basal level, or arterial wall smooth muscle hypertrophy (Manuck, et al., 1990). For atherosclerosis, the processes are believed to be similar, in that injury to the arterial wall is also involved, which ultimately results in the formation of atheromatous plaques. These injuries can be due to mechanical forces (i.e., extreme hemodynamic variability) or chemical forces (i.e., exaggerated or frequent neuroendocrine changes; Krantz & Manuck, 1984). The two disorders, hypertension and atherosclerosis, are not unrelated cardiovascular diseases; hypertension may accelerate the development of atherosclerosis (Krantz & Manuck, 1984).

The nature of the stressful situation which elicits the heightened reactivity response can lead to varied physiological outcomes; some stressors elicit only cardiovascular responses, while other stressors elicit both cardiovascular and hormonal responses (Eliot, 1988; Krantz & Manuck, 1984). Laboratory studies have attempted to isolate the various deleterious aspects of stress tasks which lead to increased stress reactivity; several factors seem to be relevant: the type of task, the nature of the response required, and the emotional tone elicited by the stressor. Several previous studies concur that borderline hypertensives and male normotensives with a positive family history for hypertension, show greater blood pressure reactivity to mental or psychological stressors (e.g., mental arithmetic) compared to physical stressors (e.g., isometric handgrip or cold pressor tasks; Schneiderman & McCabe, 1989). As well, different physiological responses are produced by psychological stressors which require an active or effortful coping response, as opposed to stressors requiring only a passive coping response, in which the subject is required only to be vigilant and process sensory stimuli (Williams et al., 1982). It has been suggested that the elicitation of an emotional response, such as feelings of anger, hostility, or some other kind negative affect, will result in broader sympathetic responding, including both hemodynamic and neuroendocrine changes (Schneiderman & McCabe, 1989; Mason, 1968a & 1968b, Frankenhaeuser, 1991).

Most psychophysiological studies investigating stress reactivity have assessed only the sympathetic aspects of the stress response, focusing mainly on hemodynamic measures, and as blood pressure and heart rate in particular. More recently, increasing numbers of studies have begun to incorporate neuroendocrine measures, but still the theoretical focus has been mainly on the sympathetic hormones (i.e., catecholamines) because of their link to cholesterol levels via lipid mobilization (Williams, 1989). Both blood pressure and catecholamines are indices of the sympathetic-adrenal medullary system, which is responsible for the classic "fight or flight" response (Van Doornen, 1991). Sympathetic arousal is reflected in heart rate, blood pressure, or norepinephrine changes, while adrenal activity is marked mainly by epinephrine fluctuations (Dimsdale, 1986) .While catecholamines, both epinephrine and norepinephrine, are certainly responsive to stress, they are very short-lived (Manger, 1980) and their effects may be too fleeting to be only the mechanism linking stress reactivity to sustained CVD.

Other than the sympathetic-adrenal cortical system, the hypothalamic-adrenal- cortical system is also responsive to stress (Mason, 1968a; Williams, 1989). Frankenhaeuser has long taken a multi-modal approach to the study of stress reactivity (Collins & Frankenhaeuser, 1978), incorporating measures of both the sympathetic-adrenal medullary system and the hypothalamic-adrenal- cortical system. She has suggested that stress reactivity should be analyzed using a model with two distinct dimensions (Frankenhaeuser, 1991). One dimension is the "activity" axis (including mental or physical effort, engagement and determination); this axis ranges from active to passive coping. The second dimension is the "affect" axis, ranging from negative to positive emotional states. Stress-induced changes in the activity axis can be assessed via sympathetic-adrenal medullary indices (e.g., hemodynamic or catecholamine measures). Similarly, cortisol measures, one index of pituitary-adrenal cortical activity, can be used to reflect changes in the affect axis. Frankenhaeuser asserts that it is the combined sympathetic-adrenal medullary, and the pituitary-adrenal cortical response(s) which

gives a more clinically relevant estimation of the stress experience, both with respect to the immediate and the long term impact on the cardiovascular system.

Frankenhaeuser (1991) therefore hypothesized that the most detrimental stressor would be one which elicits an increase in measures of both axes. Such a stressor would require not only an active coping response, would also elicit a negative emotional response. A low-control, joyless mental activity is how she describes such a stressor. Similar models to Frankenhaeuser's (e.g., Karasek, Russell & Theorell, 1982) concur that stressors requiring highly demanding, or active, effortful coping in combination with low control, passivity or helplessness will produce increases in not only epinephrine but also cortisol. Both models agree that stress situations with high demands and high control produce increased epinephrine, but decreased cortisol levels. It seems to be the "unpleasant" emotional tone of a stressor which is associated with a subsequent increase in cortisol (Mason, 1968a; Van Doornen, 1991).

Cortisol's Role in the Stress Response

Cortisol is the main glucocorticoid secreted by the adrenal cortex. It has a role in "almost every physiologic function" (Goodman, 1988), namely: it interacts with insulin to maintain blood glucose levels, it is involved in lipid, protein and nucleic acid synthesis, it interacts with the immune system and can suppress T & ß cell production, and it is essential for the maintenance of normal cardiac function (an insufficiency leads to circulatory collapse). Cortisol is also an integral hormone in the stress response; cortisol levels are directly increased by both physical or psychological/ emotional stressors, and cortisol also influences the effects of the catecholamines (Williams, 1989; Herd, 1986).

Because of the role cortisol plays in the neuroendocrine response to stress, and its influence on, catecholamine function, lipid metabolism and cardiac function, cortisol could be a critical factor in explaining the relationship between stress and cardiovascular disease. How-

ever, the long term consequences of a chronically exaggerated cortisol response to stress are unknown. There are several lines of evidence which are plausible explanations for how cortisol, alone or in combination with epinephrine, might lead to heart disease. First, pathology of the adrenals (e.g., Cushing's disease) causing excess cortisol production can result in elevated blood pressure. Approximately 80 percent of patients with Cushing's disease (a disorder characterized by abnormally high levels of cortisol which are maintained constantly without the normal circadian rhythmicity) develop hypertension (Irony, Biglieri & Kater, 1989). Second, increased cortisol levels may inhibit the metabolism of norepinephrine thereby potentiating the effects of epinephrine (Oparil & Katholi, 1990; Gravanis, 1987). The half-life of cortisol is at least one hour (Kirschbaum & Hellhammer, 1989) while epinephrine's actions last only seconds (Manger, 1980).

The interactions of epinephrine and cortisol have a variety of possible mechanisms by which stress induced elevations in these hormones could lead to CVD. Epinephrine has been hypothesized to have a detrimental effect on the arterial musculature , eliciting concurrent cortisol responses could lead to more extensive damage. Epinephrine not only stimulates cardiac rate and contractility, it also mobilizes lipids from adipose tissue (thereby increasing free fatty acids), and stimulates the liver to produce glucose for fuel for muscular response (flight or fight response). Cortisol is also involved in synthesis of cholesterol and is the main glucocorticoid, enabling metabolism (Herd, 1986; Krantz & Manuck, 1984; Williams, 1989; Goodman, 1988).

Elevations in cortisol can be elicited by a variety of emotional or psychological stressors such as: films containing themes of suspense or tension (Hellhammer, Röttger, Lorenzen & Hubert, 1986), public speaking (Bassett, Marshall & Spillane, 1987; Lehnert, et al., 1989; Kirschbaum, Bartussek & Strasburger, 1992), anticipation of surgical procedures (Ben-Aryeh, et al., 1985) or venipuncture for blood sampling (Hubert, Möller & Nieschlag, 1989), written and oral examinations (Semple, Gray, Borland, Espie & Beastall, 1988; Meyerhoff,

Oleshansky & Mougey, 1988). Various cognitive stressors may also elicit a cortisol increase, including: Stroop-like tasks, mirror-tracing, choice-reaction tasks and mental arithmetic (Frankenhaeuser, Lundberg & Forsman, 1980; Bohnen, Houx, Nicholson & Jolles, 1990; Bohnen, Nicolson, Sulon & Jolles, 1991). As early as 1968, Mason suggested that the salient components of emotional or psychological stressors responsible for elicitation of cortisol responses may be unpredictability, uncertainty and novelty. Hellhammer, Kirschbaum and Belkien (1987) agree and also add that suspense or anticipation, but not necessarily tension, are also important factors for effective stimuli. Loss of control over the stressful situation also seems to be a salient feature (Van Doornen, 1991; Karasek et al., 1982).

Those studies employing cognitive stressors such as mental arithmetic have had mixed results with respect to cortisol responses, ranging from no significant results (Lehnert et al., 1989; Collins & Frankenhaeuser, 1978) to significant stress-induced cortisol changes (Pomerleau & Pomerleau, 1990; Williams, et al., 1982). There are several possible explanations for the variability in results: 1) there is a large amount of unexplained inter-subject variability in the normal levels of cortisol (Kirschbaum & Hellhammer, 1989), 2) time of day, collection and assay procedures differed, 3) different media was used to sample cortisol (saliva, urine and plasma), and 4) the arithmetic task itself was presented to the subjects differently. With respect to variability due to the math task, the studies with significant results (Pomerleau & Pomerleau, 1990; Williams, et al., 1982), made the arithmetic task more competitive by offering a prize for the best performance and emphasized that speed and accuracy would be judged. None of the tasks however, was designed to directly manipulate the emotional state of the subjects as Frankenhaeuser (1991) and Mason (1968a) suggested.

Advantages of Salivary Cortisol

Determining cortisol responses to stress ideally requires a method of sampling that is not in itself stressful, while still being sensitive to short term hormonal changes. According to these criteria, salivary cortisol measures are clearly superior over both urinary and plasma

sampling. While it is true that urinary cortisol measures have been popular (Lundberg et al., 1989; Lundberg et al., 1990; Bassett et al., 1987), and collection of samples is not stressful to the subject, this medium is less than optimal . In terms of precise determination of cortisol levels, urinary sampling is the least desirable medium because many factors, other than those experimentally controlled for, can influence the urinary metabolites of cortisol (Kuhn, 1989). Moreover, the procedural requirements of this method are cumbersome: an extended experimental stress condition should be employed as changes in cortisol are not immediately reflected in the urine; in order to optimize the accuracy of the assay, all urine from a 24 hour period should be collected (Kuhn, 1989); and collection is inconvenient in terms of speed and the requirements for privacy (subjects may need to leave the stressful situation to produce the sample). Therefore, the use of urinary assays to assess stress-related changes in hormones is suitable only for populations under chronic, long-term stress, not acute laboratory stress paradigms (Dimsdale, 1986).

Many studies have also employed plasma sampling to assess cortisol levels (Bohlin, et al. 1986; Meyerhoff et al., 1988; Lovallo et al., 1989). Until the 1970's with the advent of sensitive assays, plasma was the most sensitive medium. However, plasma sampling is fraught with many disadvantages (Vining & McGinley, 1987), the most critical is the painful, invasive, and stressful nature of the procedure required to obtain the plasma (most people are at least moderately upset with having an intravenous catheter inserted). Saliva, unlike plasma, is a medium also very sensitive to short-term fluctuations and offers many advantages plasma does not.

Salivary sample collection is painless, non-invasive, virtually stress free, suitable for repeated sampling, technically easy, undemanding for storage, and less complicated for reliable and sensitive assays (Kirschbaum & Hellhammer, 1989; Laudat, et al., 1988; Vining & McGinley, 1987; Vining, McGinley, Maksvytis & Ho, 1983). Cortisol normally travels through the blood in a bound state (Brien, 1980). Salivary cortisol levels, however, are unaf-

fected by the many factors which alter levels of corticosteroid binding globulin (such as pregnancy, oral contraceptive or aspirin use), because bound cortisol is too large to pass through the acinar cells of the salivary glands (Vining & McGinley, 1987). With plasma, these spurious elevations in total cortisol level if these factors are unaccounted for. Because salivary cortisol levels reflect only the unbound fraction of cortisol, the biologically active portion, salivary assays directly yield a more meaningful number than plasma assays. The unbound fraction is only 5-10% of the total amount of circulating cortisol. Despite the small amount, it has been well established that the detectable levels of cortisol in saliva accurately, and virtually immediately, reflect circulating plasma cortisol levels (Kirschbaum & Hellhammer, 1989; Vining & McGinley, 1985; 1987). The only disadvantage to assessing cortisol via saliva is the size of the normative database against which to compare results; there are a limited number of published studies which have reported unstimulated, absolute salivary cortisol values from normal men and women. While the number of studies is not large, the results have shown increasing convergence, as assay methodology has become refined over the last decade (Kirschbaum & Hellhammer, 1989).

Several studies have found no sex differences between normal men and women in unstimulated levels of salivary cortisol (Riad-Fahmy, Read, Walker & Griffiths 1982; Kirschbaum & Hellhammer, 1989). Normal values morning (8 a.m.) values for healthy adults range from 0.45 to 0.78 μg/dl (Kirschbaum, Strasburger, Jammers & Hellhammer, 1989). Despite the advantages of this method, the increasing popularity of salivary cortisol measures through the 1980's, and the increasing interest in the neuroendocrine aspects of the cardiovascular stress response, few studies have combined both cardiovascular and salivary cortisol measures. Moreover, as described earlier, many studies which have collected cortisol measures did not employ a stress paradigm which would optimize the chances for eliciting a cortisol response; namely, a stressor with a negative emotional component.

Cortisol and Negative Emotional Responses to Stress

The question remains as to why not all people exposed to stressful situations succumb to illness. Several studies have suggested that one of the most valuable predictors of both the development and severity of cardiovascular disease is the propensity to experience and express hostility (Williams, 1985; Barefoot, Dahlstrom, & Williams, 1983). The mechanism by which hostility leads to risk for CVD is also unknown. Many studies have investigated the role of hostility and cardiovascular reactivity (Smith & Houston, 1987; Suarez & Williams, 1989; Smith & Allred, 1989; Houston, Smith & Cates, 1989; Everson, McKey & Lovallo, in press). In general, the cumulative findings suggest that people with higher levels of trait hostility show increased cardiovascular reactivity to tasks which are designed to provoke some kind of negative affect such as anger or hostility (Suls & Wan, 1993).

Pope and Smith (1991) found that trait hostility is significantly correlated with urinary cortisol response, however, they did not employ a lab stressor or empirically assess levels stress in any way. Linden and Long (1987) examined expressed hostile mood, plasma cortisol and blood pressure, in response to two stressors, neither of which was specifically designed to elicit hostile feelings. Despite the limitations of the stress tasks, subjects who expressed more hostility showed more cortisol reactivity from baseline and at minute 30 of the post-task recovery period. The evidence suggests that there may be a significant link between hostility (both trait and state), cardiovascular reactivity, and stress. Until very recently, most of the work concerning hostility used only male subjects, thereby limiting the generalizability of results (Suarez, Harlan, Peoples & Williams, 1993).

Lai and Linden (1992) examined the effects of gender, and opportunity for anger release, using a task which could provoke hostile feelings. Their task was a mental arithmetic task with false feedback on performance (i.e., harassing statements). Lai and Linden (1992) found sex differences during both the reactivity phase and the recovery phase. Males showed larger cardiovascular reactivity responses than did the females. During the post-task recovery phase,

males who were permitted to express their negative feelings showed more rapid return of heart rate to baseline (termed recovery). Opportunity to express emotion did not similarly facilitate the cardiovascular recovery for females.

The math task with harassment as used by Lai & Linden (1992) increased cardiovascular responses by a ratio of approximately 2:1 compared to responses obtained in previous studies in the same laboratory using an identical paradigm without the harassment condition (cf., Linden, 1987). Addition of harassment to the mental arithmetic paradigm should not only make the task more potent for eliciting cortisol responses, but it also adds a negative emotional tone to this cognitively challenging task. According to Frankenhaeuser's model (1991), this task should elicit response from both the sympathetic-adrenal medullary axis and the hypothalamic-pituitary-adrenal cortical axis.

There does not seem to be one study which has incorporated all of the potentially relevant factors and measures reviewed here in one protocol. The present study used Lai & Linden's math task both with and without harassment to examine varied negative emotional tone during a stressor. The present study also differs from previous investigations in that it examined the both the stress response in reaction to the task, and extended post-task recovery period; both males and females subjects were included; and cardiovascular and salivary cortisol parameters were measured.

Study Objectives:

1) To examine the effects of harassment (i.e., scripted, negative feedback) versus no harassment during a standard mental arithmetic stress task, on both cardiovascular (i.e., blood pressure and heart rate) and salivary cortisol reactivity and recovery.
2) To explore any sex differences in cardiovascular or cortisol reactivity and recovery, and any possible interactions (e.g., Sex x Harassment Condition).
3) To explore the relationships between subjective state measures of affect and cortisol and cardiovascular response patterns.

Method

Subjects

60 university students (30 females and 30 males), recruited from undergraduate psychology classes at the University of British Columbia (U.B.C.), completed the study. In return for participation, subjects received points toward course credit. Only one subject, a male in the harassment condition, requested that the experiment be stopped part way through the protocol; his data were not included in the subsequent analyses. The student population at the U.B.C. is multicultural; the ethnic background of the subjects in this study was: 65% Asian (39/60), 25% Western European (15/60), and 10% East Indian (6/60). Ages of the subjects ranged from 18 to 33 with a mean age of all subjects 20.7 years.

Inclusion and Exclusion Criteria

Inclusion criteria were: 1) no known history of hypertension or use of anti-hypertensive medication, 2) baseline, resting blood pressures of less than 140 mmHg SBP and 90 mmHg DBP, 3) no known conditions which may cause cortisol abnormalities. The third criterion was assessed using a medical history screening questionnaire (see Appendix 1) which was administered as part of a larger battery of questionnaires. Factors assessed included: serious medical conditions (e.g., history of: hypercortisolism, adrenal insufficiency , kidney disease, pituitary tumors, other recent surgeries or serious illnesses, severe clinical depression, diabetes, and hypoglycemia (Brien, 1980)), and more other variables which acutely may alter cortisol (e.g., food, exercise, nicotine, alcohol, caffeine consumption; Kirschbaum & Hellhammer, 1987). Assessing the acute factors also served as a secondary check on compliance with pre-study restrictions, detailed below.

Procedure

Prior to the study visit, subjects received an information sheet which detailed participation requirements and compliance instructions for the study (see Appendix 2). The written instructions explained that subjects must: (1) refrain from consuming alcohol or caffeine, smoking, or exercising vigorously for at least 12 hours prior to their scheduled visit; (2) on the morning of the visit, eat a light breakfast at least one hour prior to their visit; (3) be very careful in their dental hygiene the night before, so as not to make their gums bleed, and on the morning of the visit to not brush their teeth. The night before the study visit each subject was also contacted by telephone to remind them of the pre-study restrictions.

Because the purpose of the study was to investigate the effects of harassment during a mental arithmetic stress task, some deception was involved. Within each gender, half of all subjects were randomized to the harassment condition, which entailed receiving negative feedback on their task performance (i.e., harassing comments). The remaining subjects, randomized to the non-harassment, control condition, performed the math task without feedback. Subjects obviously could not know that they might be randomized to a condition in which they would be harassed or the manipulation would be ineffectual. Therefore, subjects were told that the study purpose was to investigate changes in thoughts and feelings during a cognitive stress task.

In order to control for the circadian rhythm of cortisol, all study visits were scheduled in the morning: beginning at 10:00 a.m. and concluding by 1:30 p.m. (Vining, 1984). On the day of the study visit, the experimental procedures and confidentiality issues were explained before obtaining written consent. Compliance with instructions for meals and abstinence from alcohol, caffeine, smoking and exercise and last meal time was confirmed. Weight and height were measured. The subject was then asked drink 250 ml of water, to ensure that no food debris contaminated saliva samples and to ease the generation of saliva samples.

Once comfortably seated, subjects' non-dominant arm was attached to an automated digital blood pressure and heart monitor (Dinamap 845 Vital Signs Monitor, Critikon, Inc.,

Tampa, FL.) and 12 minutes of baseline recording began. Subjects were instructed to relax and keep movement to a minimum. Systolic blood pressure (SBP), diastolic blood pressure (DBP), and heart rate (HR) were sampled frequently throughout the baseline at minutes 0, 3, 7, and 11. (see Appendix 3 for a sample data collection sheet detailing minute by minute activity). During the baseline, subjects were required to fill out a battery of questionnaires. The questionnaires ensured that the subjects were quietly and consistently occupied during the baseline and the range of the content of the questionnaires helped to hide the purpose of the study.

Questionnaires included: a medical screening questionnaire and several state affect measures. The state affect measures were administered both pre-and post-task to assess changes in emotional state which may have been elicited by the experimental manipulation. These measures were: the Multiple Adjective Checklist - Hostility subscale (MAACL-H; Zuckerman & Lubin, 1965) which assesses changes in negative affect, namely feelings of hostility and anger, the Activity- Affect Grid (Russell, 1989) which assesses general changes in physical arousal and pleasantness of affect, and the Current Thoughts state self esteem scale (Heatherton & Polivy, 1991), which assesses self esteem changes.

At the end of the baseline, the first saliva sample was taken (minute 12). Forty-five seconds before minute 12, subjects were given a warning and asked to collect saliva in their mouth. When producing the sample, subjects were asked to gently expectorate into a pre-labelled collection vial, one for each of the 5 samples required. Subjects were given approximately 1 minute to produce 2 ml of saliva (most subjects have little difficulty producing 3 ml, at 10 minute intervals; Vining, 1984). If subjects had difficulty producing a saliva sample, citric acid swabs were available to touch to the tongue to increase saliva flow. The use of external stimuli, such as citric acid, does not influence cortisol levels, nor does saliva flow rate (Vining & McGinley, 1987).

The mental arithmetic stress task began at minute 13 and continued for 12 minutes. The task consisted of mentally performing serial subtractions of seven from a starting point of 9000, with the answers being given aloud. Subjects were told that both speed and accuracy were important, and that if they made a mistake or lost their place, they were to start again from 9000. For subjects in the harassment condition, the scripted harassing comments (see Appendix 4 for script) were delivered on a fixed schedule at minutes 2, 6, and 10 of the task by a same-sexed experimental assistant. Blood pressure and heart rate were measured the minute following the delivery of each harassing statement, and at similar times into the task for the control subjects.

The experimental assistants were trained to deliver the harassing comments in a firm, but neutral, authoritative tone of voice. The assistant , along with the experimenter, were located in a separate room from the subject; all communications were delivered over an intercom. At minute 25, immediately after the math stress task, a second saliva sample was obtained; again 1 minute was allowed to produce the sample.

From minutes 26-63, the recovery phase, subjects completed the same state affect questionnaires as they did pre-task, and continued to work on the remainder of the questionnaire battery. In total, 3 more saliva samples were taken at minute 38 (12 minutes into recovery), minute 50 (24 minutes into recovery), and minute 62, the end of recovery. Blood pressure and heart rate were measured at minutes 1, 5, and 9 of the recovery phase and following each saliva sample.

The experimental protocol was complete following the recovery period. Subjects were then debriefed by the experimenter. They received both a verbal and written explanation of the actual study purpose and rationale for deception. All subjects were insured that their performance on the math task was not abnormal in any way, and that the harassing statements were a standardized script delivered to all subjects in the harassment condition (See Appendix 5 for a copy of the consent and debriefing forms).

Assay Procedures

After collection, saliva samples were refrigerated (4°C) and transferred daily to a -20° C freezer where they remained until all samples were collected. Cortisol levels have been shown to be unaffected by storage temperatures ranging from 4° to -70° C (Kahn, Rubinow, Davis, Kling & Post, 1988).

Assay procedures were based on the methods outlined by Kirschbaum, Strasburger, Jammers and Hellhammer (1989), which detailed adaptation of several commercially available serum cortisol radioimmunoassay kits for use with salivary cortisol samples. The Magic Cortisol (^{125}I) Radioimmunoassay was the kit used (CIBA Corning Diagnostics Corp., Medfield, MA). The assay procedures for saliva differ from those used for plasma in only one respect: the standards, against which the samples are compared, are diluted 1:10 with a buffered saline solution (pH 8.0). The dilution is necessary because the concentrations of cortisol found in saliva are estimated to be 5 - 10% of those in plasma (Al-Ansari, Perry, Smith & Landon, 1982; Brien, 1980).

Prior to assay, the samples were thawed and then centrifuged at 3000 r.p.m. for 2 minutes in order to precipitate out any debris and reduce the viscosity of the saliva, making the samples easier to pipette. Procedures then followed manufacture's instructions for incubation times, temperatures, and magnetic separation (to remove the labelled antibody from the solution). Samples were counted for five minutes in a gamma counter.

The separation step in the assay imposed batch size was limits of 60 samples per batch. Therefore care was taken not to systematically include all samples from a particular group or time in a given batch, so as to disperse the effects of inter-assay variability over all samples.

Analytic Strategies

The primary objective of the present study was to investigate the differential physiological patterns, both cardiovascular and endocrinological, elicited by a standard psychological

stressor (a mental arithmetic task) with and without a negative affect component (scripted harassing feedback during the math task). As well, exploration of the sex differences in the physiological patterns of reactivity to and recovery from the stressor was also an objective. Reactivity to a stressor is defined as the change in mean pre-task to post-task levels for a given variable. Recovery is defined as return to baseline level for a given variable, and was assessed by comparing post-task levels to the mean baseline . Cardiovascular variables were measured frequently throughout the protocol, baseline and task means were calculated based on the last two readings of each period, recovery measures are singular readings at specific time points.

The study can be conceptualized as a 2 (harassed vs. non-harassed, control conditions) x 2 (sex) x K (repeated cardiovascular and cortisol measures over time) factorial design. Because of the varying number of repeated measures over time (K = 8 time points for the cardiovascular measures and K = 5 time points for cortisol measures), and the p number of dependent measures (systolic & diastolic blood pressures, heart rate, and cortisol), four separate 3-way between-within, repeated measures univariate analyses of variance (ANOVAs) were done; one ANOVA each for systolic blood pressure, for diastolic blood pressure, for heart rate and for cortisol. Computing one large MANOVA (multivariate analysis of variance) instead would have little overall power because the number of subjects per cell is limited in comparison to the total number of measures; moreover, combining all dependent measures in one analysis can obscure real differences in some factors due to error variance on others (Stevens, 1992, pp. 152-153, 454-456).

To assess changes in affect pre- and post-task (a check on the effectiveness of the harassment manipulation), another Sex x Condition x Time 3-way repeated measures ANOVA was conducted on MAACL-H questionnaire scores.

In higher order factorial designs it is recommended that interactions beyond two factors are initially evaluated using a liberal alpha ($p \leq 0.25$) to avoid Type II errors if the term is dropped; but final conclusions about the data must also pass the more traditional alpha ($p \leq$

0.05; Howell, 1987, p.390). Therefore, three-way interactions were first tested at $p \leq 0.25$, any significant 3-way interactions at this level were then followed up with simple interaction action effects analysis at each time point using a more stringent criterion ($p \leq 0.05$). Evaluation of simple interactions involved conducting a 2-way (Condition x Sex) univariate ANOVA at each level of the repeated measure factor. These analyses were then broken down into simple simple effects of two, non-parallel, 2-way interactions at a given time *k* (i.e., the effect of Harassment Condition at each level of Sex, and the effect of Sex at each level of Harassment Condition). Simple simple effect analyses yielded pairwise comparisons which were tested using an alpha of $p \leq 0.01$. Because of the number of tests on the repeated measures that are necessary to fully explore physiological recovery from the stressor (i.e., all of the repeated physiological measures), a fairly conservative approach is justified in order to limit experiment-wise Type I error. Any further pairwise comparisons of interest were conducted using Tukey's HSD procedure, with a familywise Type I error rate of $p \leq 0.05$.

When the omnibus F of the overall 2 x 2 x K ANOVA did not reveal a significant three-way interaction, 2-way interactions and/ or main effects were explored (using $p \leq 0.05$). As explained above, any significant 2-way interactions were followed-up in the same way, with simple effects analyses at each time point for the repeated measure, and then simple simple effects. Again, as there were only two levels of each between subjects factors, further *post hocs* tests beyond simple simple effects were unnecessary.

All analyses were performed using SPSS (Macintosh version 4.0), which automatically tests for the major assumptions underlying between-within designs to which analysis of variance models are not robust to violation: 1) homogeneity of covariance matrices for the *p* dependent variables (repeated measures) in each group (tested with Box's M test), and 2) sphericity (tested with the Mauchly test). These tests are considered sensitive to even slight departures from normality and can lead to quite conservative conclusions, (Levine, 1991, pp. 28-29; Stevens, 1992, pp. 448-449). Therefore, significance of Box's M was tested using an

alpha level of $p \leq 0.001$ (Tabachnick & Fidell, 1989, p. 379). When cell sizes are approximately equal, ANOVA is moderately robust to violation of this assumption, although it may result in a decrease in power and a slight increase in Type I error (Stevens, 1992, pp. 256-268). Mauchly's test was evaluated against $p \leq 0.05$, and when necessary, subsequent adjustments of degrees of freedom for non-sphericity were made using the most stringent estimate of epsilon possible. The following strategy was used: the omnibus F was tested against the most conservative ε, the lower bound of epsilon; if it passed, these degrees of freedom were reported and the test is unambiguous, if it did not pass using the most conservative test, the Greenhouse Geisser ε was used, failing that the slightly more moderate Huynh-Feldt estimate of ε was used. There is no consensus as to which is the better estimate of epsilon, the Greenhouse Geisser or the Huynh-Feldt but generally Greenhouse Geisser is considered overly conservative unless the assumption of sphericity is severely violated (Levine, 1991; Stevens, 1992). Epsilon ranges from 1 for spherical data ,or no assumption violations, to 1/k-1 (where k = levels of the repeated measure) for maximal violations; epsilons above 0.75 are considered mild, and epsilons below 0.50 or 0.40 are considered severe.

Results

Baseline Control Variables

There were no significant differences between the female control and harassed groups, nor the male control and harassed groups on any demographic or physiological variable measured (see Table 1). Over all four groups, cell sizes and number of subjects per group with a positive family history for hypertension were approximately equal. No significant groups differences in age ($F(3,56) = 1.46$, $p = 0.235$) were present. As would be expected, there were statistically significant differences between males and the females in height ($F(3,56) = 17.04$, $p < 0.0001$) and weight ($F(3,56) = 10.81$, $p < 0.0001$); *post hoc* follow-up revealed no

significant pairwise group differences between the female control and harassed groups nor the male control and harassed groups (using $p \leq 0.05$) on these variables.

With respect to other exclusion factors assessed in the screening questionnaire, no subjects had any medical condition serious enough to warrant exclusion. Three subjects reported being regular smokers, all were males randomized to the control condition. A total of four females reported using oral contraceptives (2 randomized to control and 2 randomized to harassed conditions), and two female subjects reported that they might be pregnant (both randomized to control). As discussed previously, salivary cortisol levels do not appear to be significantly influenced by pregnancy or oral contraceptive use. These uncontrollable factors (smoking, pregnancy and oral contraceptive use) were largely balanced over the groups by randomization. In cases where randomization did not counterbalanced the variable, the bias was a conservative one to controls were somewhat neutralized by random group assignment to control conditions would lead to a conservative bias for detection of differences between control and harassed conditions.

Baseline Cardiovascular Variables

Group means and standard deviations for the cardiovascular measures of systolic blood pressure (SBP), diastolic blood pressure (DBP), and heart rate (HR) over all 8 time points are presented in Table 2.

Table 1. Group Demographics

Group	Number of Subjects	Age (years)	Height (cm)	Weight (kg)	Family History of Hypertension
Control Females	16	19.94	161.81	53.21	8
(st. dev.)		(2.11)	(6.48)	(7.66)	
Harassed Females	16	21.13	166.22	59.06	7
(st. dev.)		(4.03)	(7.42)	(9.76)	
Control Males	14	22.00	175.72	67.76	8
(st. dev.)		(4.30)	(4.52)	(9.46)	
Harassed Males	14	19.79	175.85	67.92	6
(st. dev.)		(1.81)	(7.42)	(7.83)	

Tests of ANOVA Assumptions

The assumption of homogeneity of covariance matrices was tenable for systolic and diastolic blood pressures, but not for heart rate and cortisol. Because cell sizes are approximately equal in this study, the ANOVA results can still be considered robust. All Mauchly's tests for sphericity proved significant, therefore adjusted degrees of freedom were used to determine the significance of all F ratios. The specific epsilons appear in each ANOVA table.

Table 2. Group Means for Cardiovascular Measures Over Time: Blood Pressure & Heart Rate

Total minutes	12 Baseline	24 Task	26 Rec1*	31 Rec5	35 Rec9	39 Rec13	51 Rec25	63 Rec37
Systolic Blood Pressure (mmHg)								
Control Females	108.9	117.3	110.2	107.8	107.9	106.9	108.4	110.6
(st. dev.)	(9.5)	(9.9)	(9.6)	(10.6)	(5.9)	(9.4)	(9.3)	(11.0)
Harassed Females	110.2	122.8	117.5	114.4	113.9	109.2	109.7	109.5
(st. dev.)	(7.5)	(13.0)	(12.9)	(9.7)	(9.3)	(9.6)	(10.0)	(9.8)
Control Males	122.1	125.6	126.0	122.9	123.3	120.6	121.9	119.1
(st. dev.)	(14.9)	(14.2)	(11.5)	(11.9)	(12.0)	(14.6)	(12.7)	(13.7)
Harassed Males	122.5	140.2	130.1	127.7	123.5	123.8	122.5	122.5
(st. dev.)	(12.8)	(16.5)	(14.4)	(15.1)	(12.3)	(10.6)	(12.4)	(12.5)
Diastolic Blood Pressure (mmHg)								
Control Females	71.4	77.6	70.8	71.4	70.4	71.1	70.2	74.5
(st. dev.)	(6.3)	(6.9)	(5.9)	(6.5)	(4.5)	(6.2)	(5.2)	(11.2)
Harassed Females	67.3	79.7	70.2	67.9	66.5	66.0	66.5	65.5
(st. dev.)	(7.4)	(11.0)	(7.7)	(8.0)	(5.3)	(5.5)	(6.4)	(8.3)
Control Males	71.4	76.8	73.0	70.7	72.8	72.1	72.6	73.4
(st. dev.)	(9.2)	(9.6)	(8.8)	(9.1)	(8.5)	(8.8)	(10.3)	(7.9)
Harassed Males	70.3	86.9	76.5	74.3	73.6	73.1	72.4	71.3
(st. dev.)	(10.6)	(10.7)	(11.6)	(10.7)	(8.5)	(9.0)	(9.2)	(8.3)
Heart Rate (bpm)								
Control Females	75.8	80.5	71.4	71.9	71.0	70.0	70.6	68.8
(st. dev.)	(11.0)	(14.1)	(9.4)	(10.0)	(8.7)	(8.6)	(10.0)	(11.1)
Harassed Females	70.1	84.2	69.4	68.9	68.2	65.8	65.3	63.7
(st. dev.)	(10.3)	(15.1)	(9.3)	(9.7)	(10.6)	(9.8)	(8.5)	(9.1)
Control Males	76.4	77.1	75.3	70.9	73.9	70.2	70.5	69.4
(st. dev.)	(9.6)	(10.9)	(8.4)	(7.3)	(9.1)	(8.2)	(9.5)	(7.9)
Harassed Males	74.2	93.1	76.6	74.8	75.9	76.1	75.1	68.2
(st. dev.)	(17.9)	(22.3)	(19.5)	(16.7)	(16.5)	(17.8)	(17.1)	(13.9)

* Rec = Recovery phase, or minutes post-task.

Effects of Harassment and Sex on Stress Reactivity — Cardiovascular Measures

As can be seen from Table 2 and the graphs of SBP, DBP and HR, shown as Figures 1, 2 and 3, respectively, the harassed subjects appeared to react more strongly than the control subjects in response to stress task. Moreover, the males and the females showed differing post-task recovery responses. The harassed groups, but particularly the harassed males, exhibited attenuated return to baseline on several measures, while the females showed a more rapid return to baseline and then what appears to be an over-compensation response.

Repeated measures ANOVAs over all eight time points (baseline, task, recovery minutes 1, 5, 9, 13, 25, and 37) revealed significant main effects for SBP, DBP and HR, meaning that each of these cardiovascular indices showed significant changes over time. Each of the cardiovascular measures also had significant interactions present. There were no significant 3-way interactions (Sex x Condition x Time) for SBP, DBP or HR, nor were there any significant overall 2-way interactions of Sex x Time for any of these measures. As expected however, the harassment manipulation resulted in significant 2-way interactions of Condition x Time for each of the three cardiovascular measures; again, these 2-way interactions included all 8 time points of each repeated measure. Even with conservative degrees of freedom corrections for non-sphericity there are significant differences between the harassed and control groups on all cardiovascular indices: **SBP** F (5.81, 325.09) = 2.89, p = 0.010 (using GG ε); **DBP** F (1.00, 56.00) = 4.77, p = 0.033 (using lower bound ε); **HR** F (1.00, 56.00) = 5.61, p = 0.021 (using lower bound ε). The harassed subjects showed larger stress reactivity responses than the control subjects. Simple interaction analyses are needed to differentiate where over the 8 repeated measures, the differences exist between the groups.

SBP Group Means Over Time

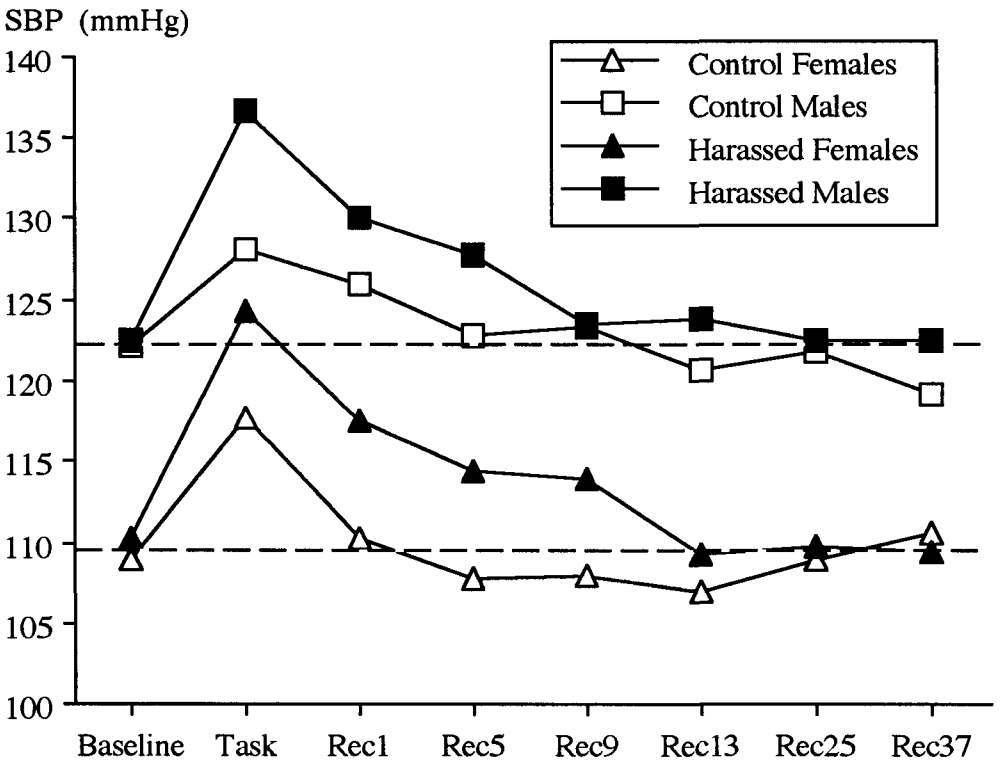

Figure 1. Systolic Blood Presssure group means (Control Females, Control Males, Harassed Females, and Harassed Males) over all time points: baseline, task, and recovery periods. Baseline to task changes were significantly different for harassed and control groups within each gender. Harassed groups each exhibited attenuated recovery until Recovery minute 13. Values correspond to means reported in Table 2.

DBP Group Means Over Time

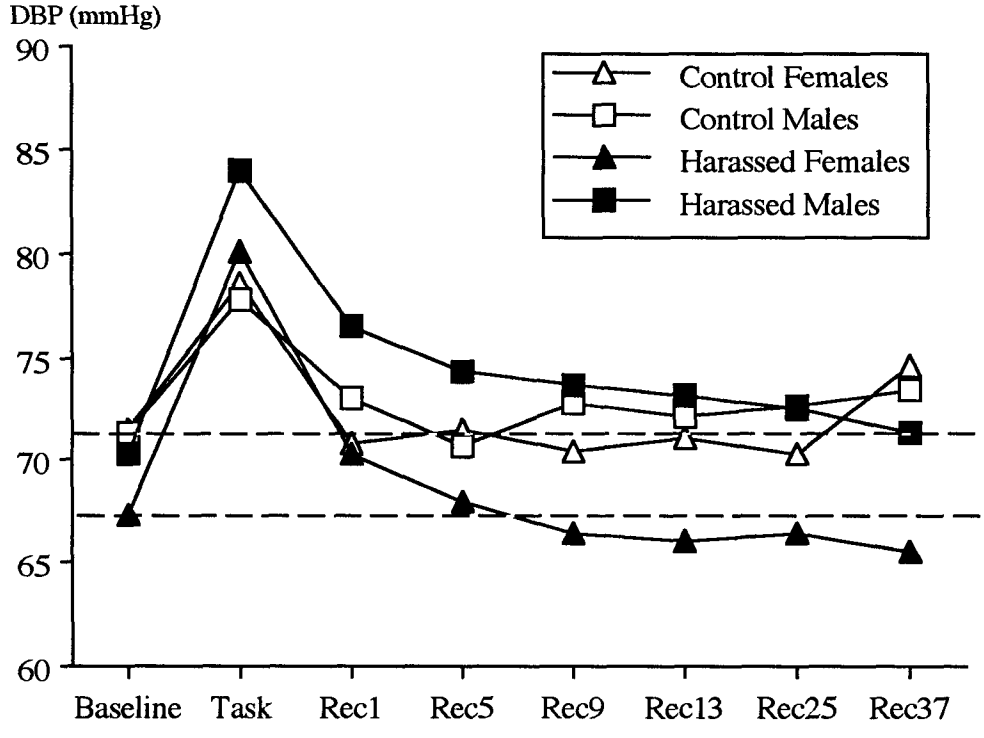

Figure 2. Diastolic Blood Presssure group means (Control Females, Control Males, Harassed Females, and Harassed Males) over all time points: baseline, task, and recovery periods. Baseline to task changes were not significantly different for harassed and control groups. During recovery, harassed males exhibited attentuated return to baseline, while the females exhibited an over-compensation response. Values correspond to means reported in Table 2.

HR Group Means Over Time

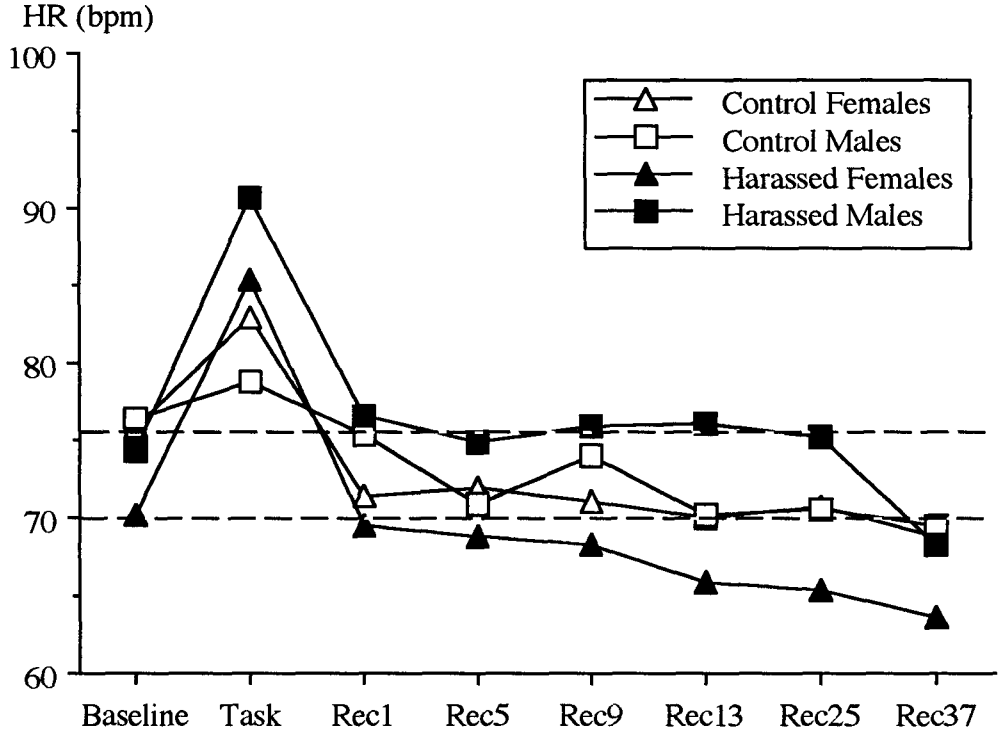

Figure 3. Heart Rate group means (Control Females, Control Males, Harassed Females, and Harassed Males) over all time points:baseline, task, and recovery periods. Baseline to task changes were not significantly different for harassed and control groups. During recovery, harassed males exhibited attentuated return to baseline, while the females exhibited an over-compensation response. Values correspond to means reported in Table 2.

Table 3. 2 x 2 x 8 Repeated Measures ANOVA Table for Systolic Blood Pressure

Source	df	MS	F	p
BETWEEN SUBJECTS				
Sex	1	19820.64	24.72	0.0001
Condition	1	1432.60	1.79	0.187
Sex x Condition	1	11.23	0.014	0.906
Subjects Within Groups	56	801.96		
WITHIN SUBJECTS				
SBP	7	933.64	27.47	0.0001
GG	5.81			0.0001
HF	6.90			0.0001
lb	1.00			0.0001
Sex x Time	7	25.48	0.75	0.630
GG	5.81			0.606
HF	6.90			0.628
lb	1.00			0.390
Condition x Time	7	98.11	2.89	0.006
GG	5.81			0.010
HF	6.90			0.006
lb	1.00			0.095
Sex x Condition x Time	7	36.73	1.08	0.375
GG	5.81			0.373
HF	6.90			0.375
lb	1.00			0.303
Within Cells	392	33.98		
GG	325.09			
HF	386.36			
lb	56.00			

Epsilons: indicating mild non-sphericity

Greenhouse-Geisser:	GG = 0.82930
Huynh-Feldt:	HF = 0.98561
lower bound (1/K-1)	lb = 0.14286

Table 4. 2 x 2 x 8 Repeated Measures ANOVA Table for Diastolic Blood Pressure

Source	df	MS	F	p
BETWEEN SUBJECTS				
Sex	1	1207.12	3.36	0.072
Condition	1	125.58	0.35	0.556
Sex x Condition	1	753.77	2.10	0.153
Subjects Within Groups	56	358.74		
WITHIN SUBJECTS				
DBP	7	651.14	25.68	0.0001
GG	5.04			0.0001
HF	5.90			0.0001
lb	1.00			0.0001
Sex x Time	7	24.97	0.98	0.442
GG	5.04			0.428
HF	5.90			0.435
lb	1.00			0.335
Condition x Time	7	120.92	4.77	0.0001
GG	5.04			0.0001
HF	5.90			0.0001
lb	1.00			0.033
Sex x Condition x Time	7	8.80	0.35	0.932
GG	5.04			0.885
HF	5.90			0.909
lb	1.00			0.558
Within Cells	392	25.36		
GG	282.42			
HF	330.34			
lb	56.00			

Epsilons: indicating mild to moderate non-sphericity

Greenhouse-Geisser:	GG = 0.72045
Huynh-Feldt:	HF = 0.84270
lower bound (1/K-1)	lb = 0.14286

Table 5. 2 x 2 x 8 Repeated Measures ANOVA Table for Heart Rate

Source	df	MS	F	p
BETWEEN SUBJECTS				
Sex	1	1575.55	1.61	0.210
Condition	1	0.02	0.00	0.996
Sex x Condition	1	1252.89	1.28	0.263
Subjects Within Groups	56	980.73		
WITHIN SUBJECTS				
HR	7	1514.15	50.94	0.0001
GG	3.68			0.0001
HF	4.18			0.0001
lb	1.00			0.0001
Sex x Time	7	49.52	1.67	0.116
GG	3.68			0.164
HF	4.18			0.156
lb	1.00			0.202
Condition x Time	7	166.65	5.61	0.0001
GG	3.68			0.0001
HF	4.18			0.0001
lb	1.00			0.021
Sex x Condition x Time	7	33.03	1.11	0.355
GG	3.68			0.351
HF	4.18			0.353
lb	1.00			0.296
Within Cells	392	29.72		
GG	205.88			
HF	233.95			
lb	56.00			

Epsilons: indicating moderate to severe non-sphericity

Greenhouse-Geisser:	GG = 0.52522
Huynh-Feldt:	HF = 0.59681
lower bound (1/K-1)	lb = 0.14286

Foremost it is necessary to evaluate reactivity to the stress task (changes from baseline to post-task). There were no significant differences among the four groups on baseline levels of diastolic blood pressure or heart rate, as evidenced by non-significant interactions at baseline for Sex and for Condition (**DBP** Sex x Time(1) $F(1,56) = 0.54$, $p = 0.466$; Cond x Time(1) $F(1,56) = 1.67$, $p = 0.202$; Sex x HR(1) $F(1,56) = 0.56$, $p = 0.459$; Cond x HR(1) $F(1,56) = 1.56$, $p = 0.217$), and non-significant pairwise comparisons between all groups (see Tables 7 & 8). Males had significantly higher baseline systolic blood pressures than did females (**SBP** Sex x Time(1) $F(1,56) = 21.27$, $p \leq 0.0001$), differences were present overall for all males and females and were not restricted to either the control and harassed condition (**SBP** Cond x Time(1) $F(1,56) = 0.10$, $p = 0.753$; see also Table 6 for all pairwise comparisons). It is typical to see sex differences in resting systolic or diastolic blood pressure for adults between the ages of 18 and 44 (Saab, 1989); with the males exhibiting a systolic reading averaging 4.4 - 9 mmHg higher than that of females.

At time point 2, immediately following the stress task, differences, other than those present at baseline, were evident for systolic blood pressure. The larger stress reactivity responses of the harassed subjects emerged as a significant interaction at time 2 with Condition (**SBP** Cond x Time(2) $F = (1,56) = 5.48$, $p = 0.023$). Simple simple effects follow-up revealed that male subjects overall exhibited significantly higher SBPs than females overall (**SBP** Sex x Time(2) $F = (1,56) = 12.49$, $p = 0.001$). This sex difference was evident in both the harassed and control conditions, but was stronger in the harassed (CF vs. CM $F = (1,56) = 5.76$, $p = 0.020$; HF vs. HM $F = (1,56) = 6.78$, $p = 0.012$). Among the four groups, the harassed males showed the largest SBP reactivity to the stress task.

Although the omnibus ANOVAs for both diastolic blood pressure and heart rate showed significant main effects for these two measures, differences between the harassed and control groups in response to the task were only approaching significance (**DBP** Cond x Time(2) $F = (1,56) = 2.85$, $p = 0.097$; **HR** Cond x Time (2) $F = (1,56) = 3.17$, $p = 0.080$). Pairwise

comparisons for the task time point were examined, to see if a similar trend was emerging for DBP and HR as was evident in SBP. Indeed, the trend was similar; the harassed males had reactivity responses that were larger than the control males on both DBP and HR. Because a more stringent alpha ($p \leq 0.01$) is applied at this level of analysis, neither of these contrasts can be considered significant (CM vs. HM: **HR** Time(2) $F = (1,56) = 4.08$, $p = 0.048$, **DBP** Time(2) $F = (1,56) = 3.44$, $p = 0.069$).

Table 6. Pairwise Group Comparisons Over Time: Systolic Blood Pressure

Total minutes	12 Baseline	24 Task	26 Rec1	31 Rec5	35 Rec9	39 Rec13	51 Rec25	63 Rec37
Pairwise Comparisons								
CF vs. CM	***	†	***	†	***	***	***	*
CF vs. HF	n.s.	n.s.	n.s.	n.s.	n.s.	n.s.	n.s.	n.s.
CF vs. HM	*'	*'	*'	*'	*'	*'	*'	*'
CM vs. HF	*'	n.s.	n.s.	n.s.	n.s.	*'	*'	n.s.
CM vs. HM	n.s.	n.s.	n.s.	n.s.	n.s.	n.s.	n.s.	n.s.
HF vs. HM	**	*	**	**	*	***	**	**

*** $p \leq 0.001$ ** $0.001 > p \leq 0.01$ * $0.01 > p \leq 0.05$ † $0.005 > p < 0.01$

n.s. $p > 0.1$ *' $p \leq 0.050$, but exact p unknown (may be *, **, or ***)

Table 7. Pairwise Group Comparisons Over Time: Diastolic Blood Pressure

Total minutes	12 Baseline	24 Task	26 Rec1	31 Rec5	35 Rec9	39 Rec13	51 Rec25	63 Rec37
Pairwise Comparisons								
CF vs. CM	n.s.	n.s.	n.s.	n.s.	n.s.	n.s.	n.s.	n.s.
CF vs. HF	n.s.	n.s.	n.s.	n.s.	n.s.	†	n.s.	**
CF vs. HM	n.s.	n.s.	n.s.	n.s.	n.s.	n.s.	n.s.	n.s.
CM vs. HF	n.s.	n.s.	n.s.	n.s.	n.s.	n.s.	n.s.	n.s.
CM vs. HM	n.s.	n.s.	n.s.	n.s.	n.s.	n.s.	n.s.	n.s.
HF vs. HM	n.s.	n.s.	†	†	**	**	*	†

*** $p \leq 0.001$ ** $0.001 > p \leq 0.01$ * $0.01 > p \leq 0.05$ † $0.005 > p < 0.01$

n.s. $p > 0.1$ *' $p \leq 0.050$, but exact p unknown (may be *, **, or ***)

Table 8. Pairwise Group Comparisons Over Time: Heart Rate

Total minutes	12 Baseline	24 Task	26 Rec1	31 Rec5	35 Rec9	39 Rec13	51 Rec25	63 Rec37
Pairwise Comparisons								
CF vs. CM	n.s.	n.s.	n.s.	n.s.	n.s.	n.s.	n.s.	n.s.
CF vs. HF	n.s.	n.s.	n.s.	n.s.	n.s.	n.s.	n.s.	n.s.
CF vs. HM	n.s.	n.s.	n.s.	n.s.	n.s.	n.s.	n.s.	n.s.
CM vs. HF	n.s.	n.s.	n.s.	n.s.	n.s.	n.s.	n.s.	n.s.
CM vs. HM	n.s.	*	n.s.	n.s.	n.s.	n.s.	n.s.	n.s.
HF vs. HM	n.s.	n.s.	n.s.	n.s.	†	*	*	n.s.

*** $p \leq 0.001$ ** $0.001 > p \leq 0.01$ * $0.01 > p \leq 0.05$ † $0.005 > p < 0.01$

n.s. $p > 0.1$ *' $p \leq 0.050$, but exact p unknown (may be *, **, or ***)

Effects of Harassment and Sex on Recovery from Stress — Cardiovascular Measures

As can be seen by the graphical representations (Figures 1 - 3) of the cardiovascular data, subjects' heightened blood pressures and heart rates do not return immediately to baseline levels after the end of the stress task. Condition x Time interactions of diminishing marginal significance support that the effects of the harassment manipulation on systolic blood pressure fade gradually over the first two post-task recovery measures (**SBP** Cond x Time(3) $F = (1,56) = 3.23$, $p = 0.078$; **SBP** Cond x Time(4) $F = (1,56) = 3.48$, $p = 0.067$). At both of these time points (recovery minutes 1 and 5), the harassed subjects were no longer significantly differed from their same-sexed control counterparts (see Table 6 for pairwise comparisons). By minute 9 of the recovery phase, the 2-way interaction between Condition and Time for SBP had completely disappeared as subjects had generally returned to baseline levels (**SBP** Cond x Time(5) $F = (1,56) = 1.39$, $p = 0.243$). Looking at Table 6 with the pairwise contrasts, the same patterns of group differences that were present at baseline were again present, with one exception. At baseline, all male groups had significantly higher SBPs than all female groups, however, even 9 minutes after the stressor, the female harassed and the male control and harassed groups did not show enough recovery to regain the normal pattern of sex differences. These groups did not differ enough to reach statistical significance at time 5 whereas they easily did at baseline (**SBP** CM vs. HF and HM vs. HF Time(5): $F = (1,56) = 2.61$, $p > 0.050$ (Tukey's HSD)). The marginal reactivity effects seen in DBP and HR between the groups had vanished by minute 5 of recovery (**DBP** Cond x Time(4) $F = (1,56) = 0.00$, $p = 0.987$; **HR** Cond x Time(4) $F = (1,56) = 0.02$, $p = 0.892$).

Another distinct pattern began to emerge in the recovery phase, aside from the normal post-stress decline in blood pressure and heart rate to baseline levels; some subjects showed an over-compensation response with levels declining well below baseline means as recovery continued. Beyond minute 9 of recovery, stronger sex differences than those observed at baseline (increasing trend in F's, and smaller p's) began to emerge as the females began to

over-compensate in their recovery on all cardiovascular indices. Pairwise comparisons found more pronounced differences between the harassed females and harassed males on DBP, SBP, and weakly on HR at recovery minute 13 than at baseline. With respect to SBP, the statistical and graphical results, suggest that the difference between the harassed groups may be attributable to the recovery of initial baseline sex differences; the harassed males were slow to complete recovery. The harassed females' SBP did not fall much below baseline. However, for DBP and heart rate more seems to be happening than recovery to baseline values. The harassed males again exhibited attenuated recovery on both DBP and HR, but as well, the harassed females showed over-compensation beyond baseline levels from minute 9 of recovery to near the end of the protocol at recovery minute 25 (**DBP:** HF vs. HM DBP(5) $F = (1,56) = 8.47$, $p = 0.005$; HF vs. HM DBP(6) $F = (1,56) = 7.18$, $p = 0.010$; HF vs. HM DBP(7) $F = (1,56) = 4.40$, $p = 0.041$; **HR:** HF vs. HM HR(5) $F = (1,56) = 3.40$, $p = 0.070$; HF vs. HM HR(6) $F = (1,56) = 5.80$, $p = 0.019$; HF vs. HM DBP(7) $F = (1,56) = 5.38$, $p = 0.024$). By recovery minute 25, the differences were not quite statistically significant using an alpha criterion of 0.01. The harassed males and the harassed females did not show significant baseline differences for either DBP or HR so this pattern seems to be different from that observed in SBP.

Table 9. Group Means for Cortisol Responses (μg/dl)

Total minutes	12 Baseline	26 Task	39 Rec13	51 Rec25	63 Rec 37
Control Females	0.4467	0.3821	0.3726	0.3541	0.3125
(st. dev.)	(0.2988)	(0.1985)	(0.1679)	(0.1641)	(0.1258)
Control Males	0.4144	0.3789	0.3942	0.3787	0.3317
(st. dev.)	(0.2200)	(0.1806)	(0.2716)	(0.3201)	(0.2310)
Harassed Females	0.3979	0.4398	0.4140	0.4159	0.3905
(st. dev.)	(0.1533)	(0.1732)	(0.1637)	(0.2007)	(0.1772)
Harassed Males	0.6760	0.9946	0.7980	0.8823	0.5808
(st. dev.)	(0.4625)	(0.8136)	(0.5162)	(0.7745)	(0.3681)

Effects of Harassment and Sex on Stress Reactivity — Salivary Cortisol Measures

As can be seen from the graphed cortisol values over time (see Figure 4), there were pronounced differences between the harassed males and all other groups in response to the stress task and in the recovery phase .

Repeated measures ANOVAs over all five time points (baseline, task, recovery minutes 13, 25, and 37) revealed a significant main effect for cortisol, indicating that cortisol levels varied significantly over time. There were also significant 2-way and 3-way interactions present, suggesting again, differences between the sexes and harassment conditions. The 3-way interaction was significant using a liberal alpha of 0.25 even applying the most stringent lower bound ε degrees of freedom correction for non-sphericity (Sex x Condition x Cortisol $F(1.00, 56.00) = 1.85$, $p = 0.180$). Simple interaction effects and simple simple effects are necessary to determine where over the five time points this interaction remains significant.

Although Figure 4 suggests significant differences in cortisol at baseline between the harassed males and the other three groups, tests of all pairwise comparisons using Tukey's

HSD (with a familywise error rate of 0.05) reveals no significant differences between any of groups. The standard deviation of the harassed male group was larger than those for the other groups. Therefore the 3-way interaction can not be predominantly attributable to initial group differences.

Looking at cortisol reactivity to the stress task (changes from baseline to post-task) strong 2-way interactions emerge at time 2 with Sex and with Condition; the 3-way interaction remains significant (Sex x Time(2) $F(1,56) = 6.32$, $p = 0.015$; Cond x Time(2) $F(1,56) = 9.42$, $p = 0.003$; Sex x Condition x Time(2) $F(1,56) = 6.46$, $p = 0.014$). Pairwise comparisons to follow up these interactions found that the differences were due to 3 significant differences between the harassed males and each of the other groups (CF vs. HM $F = (1,56) = 0.1097$, $p < 0.050$ {Tukey's}; CM vs. HM $F = (1,56) = 14.76$, $p = 0.0001$; HF vs. HM $F = (1,56) = 11.79$, $p = 0.001$). No other pairwise comparisons were significant. The harassed females did not differ from the control females (CF vs. HF $F = (1,56) = 0.15$, $p = 0.702$), therefore it was not the harassment alone contributing to the cortisol response. The results clearly support a significant Sex x Condition interaction with cortisol; the harassed males were the only subjects to show a significant increase in cortisol in reaction to the stressor. Examining the groups means in Table 9, confirms that the other groups did not show similar stress-evoked changes.

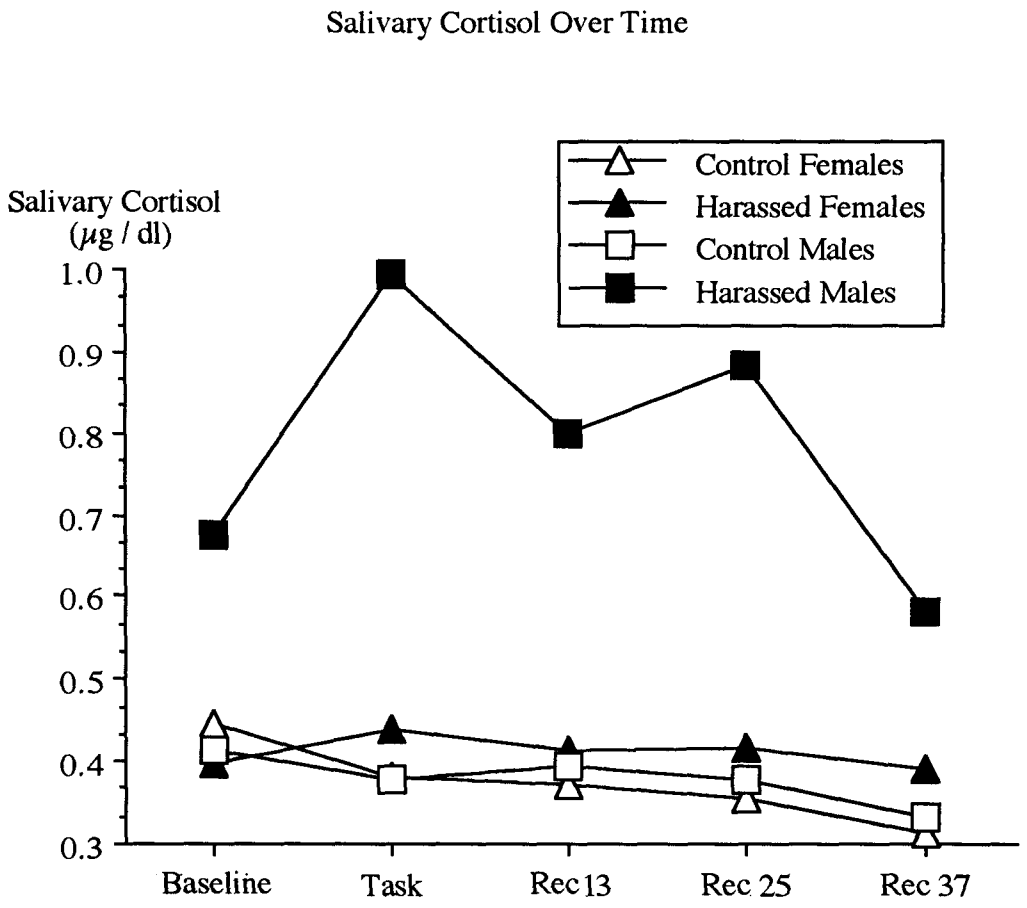

Figure 4. Salivary cortisol group means (Control Females, Control Males, Harassed Females, and Harassed Males) over all time points: baseline, task, and recovery periods. Significant baseline to task changes are evident only in the harassed males, who also exhibit significantly attentuated recovery until after Recovery minute 25. Values correspond to means reported in Table 9.

Table 10. 2 x 2 x 5 Repeated Measures ANOVA Table for Cortisol

Source	df	MS	F	p
BETWEEN SUBJECTS				
Sex	1	2.67	6.05	0.017
Condition	1	3.66	8.28	0.006
Sex x Condition	1	2.57	5.81	0.019
Subjects Within Groups	56	0.44		
WITHIN SUBJECTS				
Cortisol	4	0.16	4.01	0.004
GG	2.71			0.011
HF	3.02			0.009
lb	1.00			0.050
Sex x Cortisol	4	0.09	2.17	0.073
GG	2.71			0.100
HF	3.02			0.093
lb	1.00			0.146
Condition x Cortisol	4	0.13	3.19	0.014
GG	2.71			0.030
HF	3.02			0.025
lb	1.00			0.080
Sex x Condition x Cortisol	4	0.07	1.85	0.121
GG	2.71			0.147
HF	3.02			0.140
lb	1.00			0.180
Within Cells	224	0.04		
GG	151.93			
HF	168.96			
lb	56.00			

NOTE: 3-way interactions tested at $p \leq 0.25$, follow-ups to interaction (see Table 11) tested at $p \leq 0.05$ and $p \leq 0.01$.

Epsilons: indicating moderate non-sphericity

Greenhouse-Geisser:	GG = 0.67824
Huynh-Feldt:	HF = 0.75430
lower bound (1/K-1)	lb = 0.14286

Effects of Harassment on Stress Recovery — Salivary Cortisol Measures

The same differences that were evident in response to the stress task were maintained until minute 25 of recovery. The harassed males showed attenuated recovery on cortisol measures (as well as the previously reported cardiovascular measures). At minutes 13 and 25 of recovery there were significant interactions for Sex, Condition and Sex x Condition at each of the time points for cortisol (Sex x Time(3) $F = (1,56) = 6.55$ $p = 0.013$; Condition x Time(3) $F = (1,56) = 7.90$, $p = 0.007$; Sex x Condition x Time(3) $F = (1,56) = 5.23$, $p = 0.026$; Sex x Time(4) $F = (1,56) = 6.55$ $p = 0.013$; Condition x Time(4) $F = (1,56) = 7.90$, $p = 0.007$; Sex x Condition x Time(4) $F = (1,56) = 5.23$, $p = 0.026$). As before, the differences were due to significant pairwise contrasts between the harassed males and each of the three other groups (see Table 11). By the end of recovery at minute 37, only a significant Condition x Time interaction remained attributable to a significant difference between the harassed males and the control males (CM vs. HM Time(5) $F(1,56) = 7.66$, $p = 0.008$). Unlike the cardiovascular measures, there was no apparent over-compensation response by any of the groups.

Table 11. Pairwise Group Comparisons Over Time: Salivary Cortisol

Total minutes	12 Baseline	24 Task	39 Rec13	51 Rec25	63 Rec37
Pairwise Comparisons					
CF vs. CM	n.s.	n.s.	n.s.	n.s.	n.s.
CF vs. HF	n.s.	n.s.	n.s.	n.s.	n.s.
CF vs. HM	n.s.	*'	*'	*'	*'
CM vs. HF	n.s.	n.s.	n.s.	n.s.	n.s.
CM vs. HM	*	***	***	**	**
HF vs. HM	*	***	**	**	*

*** $p \leq 0.001$ ** $0.001 > p \leq 0.01$ * $0.01 > p \leq 0.05$ † $0.005 > p < 0.01$

n.s. $p > 0.1$ *' $p \leq 0.050$, but exact p unknown (may be *, **, or ***)

Effects of Harassment and Sex on Stress Reactivity — Subjective Measures

With respect to the subjective measures, as compared to the physiological measures, an overall reversed pattern was found for the sexes. With all physiological measures, the males showed larger responses than did the females. On the subjective self-report measures, the females showed larger pre- to post-task changes. The harassing statements did appear to elicit negative affect, as the harassed subjects showed larger and statistically significant pre- to post-task changes on the MAACL-H, a subjective self-report measure designed to tap hostile feelings.

At baseline there were no differences among the groups on the MAACL-H (**MAACL-H** Time(1)F (3,56) = 0.3181, p = 0.8122). After the task, there were significant differences on MAACL-H both between the sexes (**MAACL-H** Sex x Time(2) F (1,56) = 6.27, p = 0.015) and between the conditions (**MAACL-H** Cond x Time(2) F (1,56) = 12.76, p = 0.001). With respect to the other state measures that were administered, neither the Activity-Affect Grid (F (3,56) = 0.54, p = 0.658), nor the Current Thoughts self esteem scale (F (3,56) = 0.99, p = 0.404), showed significant changes from pre- to post-task. The lack of significant change on the Activity-Affect Grid and Current Thoughts scale, coupled with the significant changes on the MAACL hostility measure, implies that the harassing statements delivered during the stressor had a specific impact on subjects' affective state (i.e., aroused anger or hostility) and did not cause more general changes in mood, such as changes in self-esteem or general physical arousal.

Looking more closely at the significant interactions on the MAACL-H, the Sex x Time interaction reflects that overall, the females reported more change in negative affect in reaction to the stress task than did the males. While the Condition x Time interaction reflects the impact of the harassment; the harassed subjects reported more negative affect in response to the stressor than did the control subjects. Pairwise comparisons following up these interactions revealed that the male harassed subjects did not significantly differ from the male or the female control groups on the post-task MAACL-H , while the female harassed group showed

significantly greater emotional response than each of the two control groups (see Table 12). The difference between the male and female harassed groups was not quite large enough to reach statistical significance using an alpha criterion of 0.01 (HF vs. HM Time(2) $F(1,56) = 5.67$, $p = 0.021$).

Table 12. Pairwise Group Comparisons Pre- and Post- Task on the MAACL-H

Total minutes	12 Baseline	24 Task
Pairwise Comparisons		
CF vs. CM	n.s.	n.s.
CF vs. HF	n.s.	*
CF vs. HM	n.s.	n.s.
CM vs. HF	n.s.	*
CM vs. HM	n.s.	n.s.
HF vs. HM	n.s.	n.s.

* $p \leq 0.01$ n.s. $p > 0.1$

Discussion

The main objective of this study was to explore the additional effects of a negative emotional stress (harassment), during a standard psychological stress task, mental arithmetic. On all measures, cardiovascular, cortisol and subjective, it was found that subjects in the harassed condition exhibited larger stress reactivity (pre- to post-task change) responses compared to the non-harassed control subjects. However, not all harassed subjects responded equally

across all measures. The amplified stress response in the harassed condition was particulary pronounced for male subjects. The harassed males showed the largest reactivity in the physiological measures, and they exhibited significant changes on every type of measure. In descending order of response magnitude, the harassed males showed significant reactivity on cortisol, cardiovascular, and subjective state measures. Compared to the harassed males, the harassed females showed a more pronounced response on the subjective state hostility indices and, a smaller, but still significant response on cardiovascular measures. The control subjects, who performed the mental arithmetic stress task without harassing feedback, showed significant reactivity to the task on SBP, and subjective state indices. Again, within the control condition, the SBP changes were larger for the males than the females. The two control groups did not differ on subjective measures. Like the harassed females, neither of the control groups showed significant changes in cortisol over baseline levels. In sum, the addition of harassment to the math stressor produced physiological reactivity responses that were approximately twice those obtained with the math task alone (more than double for males, less for females).

Both the sex difference in stress reactivity and the overall size of the systolic blood pressure changes elicited by the addition of harassment to the math stressor were in line with results obtained by Lai and Linden (1992) and many other studies which attempted to trigger angry or hostile feelings with harassment (Everson, et al., in press; Suarez & Williams, 1989; Smith & Allred, 1989). These latter studies, however, chose to study only male subjects so no sex differences could be examined. The present study, and Lai and Linden (1992) both used the same stress paradigm, however, Lai and Linden obtained significant changes on diastolic blood pressure and heart rate which the present study did not. The more limited results on cardiovascular parameters in the present study could be due to the delivery of the harassing statements. While subjects in the present study reported at the time of debriefing that they did not suspect that the feedback was false, the experimental confederates reported much distress

themselves in having to give such negative feedback to the subjects, and may not have sounded as "negative" as the confederates used in Lai and Linden (1992). The manipulation in the present study was potent enough, however, to elicit very significant changes on other measures, namely salivary cortisol and subjective state measures of hostility. Cortisol measures were not taken in Lai and Linden, nor any of the other studies mentioned above using harassment paradigms, so there is no direct basis of comparison for endocrine measures. However, as early as 1978, Collins and Frankenhaeuser provided evidence which suggested that females might not show cortisol increases in response to the same stressors which elicited cortisol increases in males; this obsevation was confirmed by the present findings. The present study seems to be the only one which combined both cardiovascular and cortisol measures, subjects of both sexes, and employed a laboratory stress paradigm that manipulated negative affect (harassed vs. non-harassed conditions).

Because cortisol can have slower response, and remains at an elevated level for far longer than cardiovascular indices, the recovery phase in the present study was longer than is standard in most stress reactivity protocols. During this extended observation period two different recovery patterns were observed. First, compared to the control groups, both the female and male harassed subjects exhibited an attenuated return to baseline on SBP until minute 9 of recovery. After this time, the females of both groups began to show an over-compensation response. The harassed females had levels of SBP, DBP and HR that were significantly lower than those of the harassed males during the later part of the recovery, minutes 13 to 25 (HR was only approaching significance). This significance was due to the twofold pattern described, the attenuated return to baseline of the harassed males and the over-compensation of the harassed females. The salivary cortisol levels in the harassed males also remained elevated until after minute 25 of recovery.

Like the present study, Lai and Linden (1992) also found differential recovery patterns between males and females over their ten minute recovery period. The recovery period in the

former study was of a different nature than that used in the present study. Lai and Linden's protocol gave half of the subjects the opportunity to express in writing any negative feelings aroused by the manipulation; the other half copied a neutral paragraph. In the present study, subjects completed a battery of questionnaires or read light material during the recovery period and were not given the opportunity to express their feelings. Lai and Linden (1992) found that the harassed males who did not have an opportunity to express their feelings showed a significantly attenuated HR and DBP cardiovascular recovery until minute 10. The females did not show this pattern; their return to baseline was complete by the end of 10 minutes.

Because most studies examining cardiovascular responses to stress have focused mainly on the reactivity response to the stressor, extended recovery periods have seldom been employed. Recovery has traditionally been defined as being complete when the subject returned to previously observed baseline levels. It was assumed that the normal pattern of post-stress "recovery" was return to baseline. An over-compensation response, as observed in many subjects (especially female) in the present study, may be part of the normal pattern of recovery and indeed may be indicative of a truly completed recovery. As most previous studies were only interested in cardiovascular measures, and had no reason to extend recovery observations, this pattern was not observed before.

Theoretical and Clinical Implications

Epidemiological data indicate a lower incidence of CVD among females (Watkins & Eaker, 1986). It could be that females have a healthier stress response which confers some long term benefits with respect to development of cardiovascular pathology. Frankenhaeuser (1983) has suggested that physiological responses to a stressor could be similar to a learned coping response. It makes sense then to study a "healthier" stress response, especially if it might be a learnable coping strategy. Although CVD is a major health risk in females, it develops at a much later age (Watkins & Eaker, 1986), yet most studies investigating psycho-

logical and behavioral links to cardiovascular disease have focused on the almost exclusively the "unhealthier" stress responses of males. The present study has shown that emotional stressors which are potent for males, can fail to prduce a similarly exaggerated response in females. This is an important finding because it may suggest two or more different pathways by which response to a stressor leads to pathology.

It may be that the female style of recovery, the over-compensation response, is healthier, protective, or a normal response that is lost over time with repeated stress activation responsive (i.e., as in the male response). Stressors which elicit negative affect and require an active coping response are thought to activate both the sympathetic-adrenal medullary axis and the hypothalamic-pituitary-adrenal cortical axis (cf., Frankenhaeuser's model, 1991). Recovery is an important phase to explore as it may indicate the pathway by which heightened stress responsivity leads to sustained cardiovascular pathology. As described earlier, there are many potential mechanisms by which chronic or repeated elevations in cortisol could lead to pathology; moreover, the half-life of cortisol is at least one hour (Kirschbaum & Hellhammer, 1989), therefore any stressor which elicits a long-lasting cortisol response is worthy of further study.

In light of the present findings, it appears that females, as compared to males, assess emotional situations differently and may, therefore have different physiological responses to many stressors. Indeed, Frankenhaeuser herself reported in 1978, that females show less of cortisol response than do males in reaction to cognitive challenges (Collins & Frankenhaeuser, 1978). The math task used in the present study was not purely a psychological or cognitive stressor; half of the subjects had the additional emotional component of harassing feedback. However, as reported above, only the males responded on both physiological axes.

Future Challenges

Designing a task that elicits a neuroendocrine response from females will be particularly challenging as self-report on the emotional impact of the task is of limited use. The harassed females did perceive the emotional stress of the harassment condition, as they did report significantly more negative affect after the task than the non-harassed control females. Frankenhaeuser's later work suggest two possible avenues (Frankenhaeuser, 1991): she was able to obtain significantly increased cortisol levels in females at stressful home and work situations. The real life stressors of home seem to be particularly potent for women, for example, taking a child for a medical appointment. Laboratory protocols, even with extended paradigms (e.g., 60 minutes to 4 hours) yielded mixed results for female subjects (Lundberg, et al., 1990; Lundberg, et al., 1989; Bohnen, Houx, Nicolson & Jolles, 1990; Bohnen, Nicolson, Sulon & Jolles, 1991). The lack of the proper affective component in these protocols could be the critical factor. Negative affect is predictive of development of CVD (Williams & Anderson, 1987; Suls & Wan, 1993), but the mechanism by which emotions such as anger and hostility lead to disease is unknown. In studies attempting to further investigate this link, it is important to have a task which elicits negative affect. Harassment during a mental arithmetic is such a task; it produces strong physiological changes in males, but markedly less so in females. Stressors eliciting negative affect may have a unique physiological finger-print; the cortisol results of this study support that idea.

Both Hellhammer and colleagues (1987) and Mason (1968a) have pointed out several psychological components that are particularly indicative of a cortisol response: novelty, unpredictability, uncertainty, suspense, ruminating thoughts/behaviors. As advised by Suls and Wan in their review article (1993), studies attempting to link reactivity to negative affect, such as hostility, should employ stress tasks which are designed to provoke the affective state in question. Particularly for hostility, the tasks should be inter-personal in nature. This was the case in our study.

Some researchers in the field have criticized the concept of cardiovascular reactivity to laboratory stressors as too limited and too narrowly focused to really explain the development of cardiovascular disease (Pickering & Gerin, 1990). The current findings support the idea that reactivity should not necessarily be abandoned but rather expanded to look at more than cardiovascular measures. More routine inclusion of cortisol measures in stress reactivity paradigms with carefully designed tasks (i.e., those shown to have ecological validity), and subject pools which include both males and females, would give additional information which may give valuable insight into the etiological mechanisms by which stress leads to pathology. The present study suggests the entire stress response is important, both the reaction and the recovery phases.

If future studies were able to show consistent sex differences on cortisol responsivity or recovery, such evidence could be suggestive of differing pathways to cardiovascular disease for males and females. It is therefore important to include both sexes in future studies and to limit generalization from previous studies that have included only males (or females).

The sample of the present study was approximately two-thirds Asian-Canadian and therefore may limit the generalizability of these findings. Because the acceptability of expression of negative affect varies across cultures (Russell, 1994), it is important to collect demographic information about the cultural background of the subject pool under study. Responsivity to harassment, or any negative emotion, is likely to vary by culture, and may partially explain the observed sex differences, as it is less acceptable for females in many cultures to express negative emotions. While it is true that the subjects in the present study were not asked to express their feelings, it may be that if it was never acceptable for one to express a certain emotion, one becomes less responsive to it (i.e., a learned coping response).

In sum, cortisol measures could be particular informative if they were more commonly included in paradigms exploring the physiological relationship of negative affect, such as anger, hostility, and stress, as it could potentially explain the apparent sex-specific pathways

of negative affect and cardiovascular disease.

References

Al-Ansari, A. A. K., L. A. Perry, D. S. Smith & J. Landon. (1982). Salivary cortisol determination: adaptation of a commercial serum cortisol kit. Annals of Clinical Biochemistry, 19, 163-166.

Barefoot, J. C., G. Dahlstrom & R. B. Williams. (1983). Hostility, CHD incidence, and total mortality: A 25-year follow-up study of 255 physicians. Psychosomatic Medicine, 45(1), 59-63.

Bassett, J. R., P. M. Marshall & R. Spillane. (1987). The physiological measurement of acute stress (public speaking) in bank employees. International Journal of Psychophysiology, 5, 265-273.

Ben-Aryeh, H., R. Roll, L. Kahana, E. Malberger, R. Szargel, & D. Gutman. (1985). Saliva as an indicator of stress. International Journal of Psychosomatics, 32(3), 3-8.

Bohnen, N., P. Houx, N. Nicolson & J. Jolles. (1990). Cortisol reactivity and cognitive performance in a continuous mental task paradigm. Biological Psychology, 31, 107-116.

Bohnen, N., N. Nicolson, J. Sulon & J. Jolles. (1991). Coping style, trait anxiety and cortisol reactivity during mental stress. Journal of Psychosomatic Research, 35(2/3), 141-147.

Brien, T. G. (1980). Free cortisol in human plasma. Hormone and Metabolic Research, 12, 643-650.

Collins, A. & M. Frankenhaeuser. (1978). Stress responses in male and female engineering students. Journal of Human Stress, 4, 43-48.

Dimsdale, J. E. (1986). New dimensions in studying sympathetic nervous system responses to stressors. In T. H. Schmidt, T. M. Dembroski, & G. Blümchen (Eds.), Biological and Psychological Factors in Cardiovascular Disease, (pp. 343-354). Heidelberg: Springer-Verlag Berlin.

Eliot, R. S. (1988). Physiology of stress. Stress and the Heart: Mechanisms, Measurements and Management. (pp. 31-39). Mount Kisco, N.Y.: Futura Publishing Company, Inc.

Frankenhaeuser, M. (1983). The sympathetic-adrenal response to challenge: Comparison between the sexes. In T. M. Dembroski & T. H. Schmidt (Eds.), Biobehavioral Basis of Coronary Heart Disease, (pp.91-105). New York: Karger.

Frankenhaeuser, M. (1991). The psychophysiology of workload: Comparison between sexes. Annals of Behavioral Medicine, 13(2), 197-204.

Frankenhaeuser, M., U. Lundberg & L. Forsman. (1980). Dissociation between sympathetic-adrenal and characterized by high controlability: comparison Type A and Type B males and females. Biological Psychology, 10, 79-91.

Fredrikson, M. & K. A. Matthews. (1990). Cardiovascular responses to behavioral stress and hypertension: A meta-analytic review. Annals of Behavioral Medicine, 12(1), 30-39.

Goodman, H. M. (1988). Basic Medical Endocrinology. New York: Raven Press.

Gravanis, M. B. (1987). Cardiovascular Pathophysiology. New York: McGraw-Hill Book Company.

Heatherton, T. F. & J. Polivy. (1991). Development and validation of a scale for measuring state self-esteem. Journal of Personality and Social Psychology, 60(6), 895-910.

Hellhammer, D. H., C. Kirschbaum, & L. Belkien. (1987). Measurement of salivary cortisol under psychological stimulation. In J. N. Hingtgen, D. Hellhammer & G. Huppmann (Eds.), Advanced Methods in Psychobiology (pp. 281-289). Toronto: C. J. Hogrefe, Inc.

Hellhammer, D. H., K. Röttger, J. Lorenzen & W. Hubert. (1986). Suspense increases salivary cortisol. In R. C. A. Frederickson, H. C> Hendrie, J. N. Hingtgen & M. H. Aprison (Eds.), Neuroregualtion of Autonomic, Endocrine and Immune Systems, (pp. 521-524). Boston: Martinius Nijhoff Publishing.

Herd, J. (1986). Neuroendocrine mechanisms in coronary heart disease. In K. A. Matthews, S. M. Weiss, T. Detre, T. M. Dembroski, B. Falkner, S. B. Manuck & R. B. Williams (Eds.), Handbook of Stress, Reactivity, and Cardiovascular Disease. (pp. 49-70). New York: John Wiley & Sons.

Houston, B. K., M. A. Smith & D. S. Cates. (1989). Hostility patterns and cardiovascular reactivity to stress. Psychophysiology, 26, 337-342.

Howell, D. C. (1987). Statistical Methods for Psychology, Second Edition. Boston: Duxbury Press.

Hubert, W., M. Möller & E. Nieschlag. (1989). Stress reactions in response to the procedures of LHRH tests as measured by salivary and serum cotisol and psychological variables. Hormone Reasearch, 32, 198-202.

Irony, I., E. G. Biglieri & C. E. Kater. (1989). The adrenocortical hormones in hypertensive disorders. In J. H. Laragh, B. M. Brenner & N. M. Kaplan (Eds.), Endocrine Mechanisms in Hypertension, (pp. 1-8). New York: Raven Press.

Kahn, J. P., D. R. Rubinow, C. L. Davis, M. King & R. M. Post. (1988). Salivary cortisol: A practical method for evaluation of adrenal function. Biological Psychiatry, 23, 335-349.

Karasek, R. A., R. S. Russell & T. Theorell. (1982). Physiology of stress and regeneration in job related cardiovascular illness. Journal of Human Stress, 8, 29-42.

Kirschbaum, C., D. Bartussek & C. J. Strasburger. (1992). Cortisol responses to psychological stress and correlations with personality traits. Personality and Individual Differences, 13(12), 1353-1357.

Kirschbaum, C. & D. H. Hellhammer. (1989). Salivary cortisol in psychobiological research: An overview. Neuropsychobiology, 22, 150-169.

Kirschbaum, C., C. J. Strasburger, W. Jammers & D. H. Hellhammer. (1989). Salivary cortisol in psychobiological research: An overview. Pharmacology, Biochemistry & Behavior, 34, 747-751.

Krantz, D. S. & S. B. Manuck. (1984). Acute psychophysiologic reactivity and risk of cardiovascular disease: A review and methodologic critique. Psychological Bulletin, 96(3), 435-464.

Kuhn, C. A. (1989). Adrenocortical and gonadal steroids in behavioral cardiovascular medicine. In N. Schneiderman, S. P. Weiss & P. G. Kaufmann (Eds.), Handbook of Research Methods in Cardiovascular Behavioral Medicine. (pp. 185-204). New York: Plenum Press.

Lai, J. Y., & W. Linden. (1992). Gender, anger expression style, and opportunity for anger release determine cardiovascular reaction and recovery from anger provocation. Psychosomatic Medicine, 54, 297-310.

Laudat, M. H., S. Cerdas, C. Fournier, D. Guiban, B. Guilhaume & J. P. Luton. (1988). Salivary cortisol measurement: A practical approach to assess pituitary-adrenal function. Journal of Clinical Endocrinology and Metabolism, 66(2), 343-348.

Lehnert, H., J. Beyer, P. Walger, R. Murison, C. Kirschbaum & D. H. Hellhammer. (1989). Salivary cortisol in normal men: The effects of corticotropin releasing factor and different psychological stimuli. In H. Weiner. I. Florin, R. Murison & D. Hellhammer (Eds.), Frontiers of Stress Research, (pp. 392-394). Toronto: Hans Huber Publishers.

Levine, G. (1991). A Guide to SPSS for Analysis of Variance. Hillsdale, N.J.: Lawrence Erlbaum Associates, Publishers.

Linden, W. (1987). A microanalysis of autonomic activity during human speech. Psychosomatic Medicine, 49, 562-578.

Linden, W. & B. C. Long. (1987). Repression, hostility, and autonomic recovery from a laboratory stressor. Journal of Clinical Hypertension, 3, 567-578.

Lovallo, W. R., G. A. Pincomb, B. H. Sung, S. A. Everson, R. B. Passey & M.F. Wison. (1989). Hypertension risk and caffeine's effect on cardiovascular activity during mental stress. Health Psychology, 10, 236-243.

Lundberg, U., M. Fredrikson, L. Wallin, B. Melin & M. Frankenhaeuser. (1989). Blood lipids as related to cardiovascular and neuroendocrine functions under different conditions in healthy males and females. Pharmacology, Biochemistry & Behavior, 33, 381-386.

Lundberg, U., B. Melin, M. Fredrikson, M. Tuomisto & M. Frankenhaeuser. (1990). Comparison of neuroendocrine measurements under laboratory and naturalistic conditions. Pharmacology, Biochemistry & Behavior, 37, 697-702.

Manger, W. M. (1980). Catecholamines and the heart. In G. H. Bourne (Ed.), Hearts and Heart-like Organs, Volume 2, (pp.161-262). New York: Academic Press, Inc.

Manuck, S. B., A. L. Kasprowicz & M. F. Muldoon. (1990). Behaviorally-evoked cardiovascular reactivity and hypertension: Conceptual issues and potential associations. Annals of Behavioral Medicine, 12(1), 17-29.

Markovitz, J. H. & K. A. Matthews. (1991). Platelets and coronary heart disease: Potential psychophysiologic mechanisms. Psychosomatic Medicine, 53, 643-668.

Mason, J. W. (1968a). A review of psychoendocrine research on the pituitary-adrenal cortical system. Psychosomatic Medicine, 3(5), 576-607.

Mason, J. W. (1968b). A review of psychoendocrine research on the sympathetic-adrenal medullar system. Psychosomatic Medicine, 3(5), 631-653.

Meyerhoff, J. L., M. A. Oleshansky & E. H. Mougey. (1988). Psychologic stress increases plasma levels of prolactin, cortisol, and POMC-derived peptides in man. Psychosomatic Medicine, 50, 295-303.

Oparil, S. & R. Katholi. (1990). Humoral control of the circulation. In O. B. Garfin (Ed.), Current Concepts in Cardiovascular Physiology, (pp. 226-229). New York: Academic Press, Inc.

Pickering, T. G. & W. Gerin. (1990). Cardiovascular reactivity in the laboratory and the role of behavioral factors in hypertension: A critical review. Annals of Behavioral Medicine, 12(1), 3-16.

Pomerleau, O.F. & C. S. Pomerleau. (1990). Cortisol response to a psychological stressor and/ or nicotine. Pharmacology, Biochemistry & Behavior, 36, 211-213.

Pope, M. K. & T. W. Smith. (191). Cortisol excretion in high and low cynically hostile men. Psychosomatic Medicine, 53, 386-392.

Riad-Fahmy, D., G. F. Read, R. F. Walker & K. Griffiths. (1982). Steroids in saliva for assessing endocrine function. Endocrine Reviews, 3(4), 367-395.

Russell, J. A., A. Weiss & G. A. Mendelsohn. (1989). Affect Grid: A single-item scale of pleasure and arousal. Journal of Personality and Social Psychology, 57(3), 493-502.

Russell, J. A. (1994). Is there universal recognition of emotion form facial expressions? A review of the cross-cultural studies. Psychological Bulletin, 115(1), 102-141.

Saab, P. (1989). Cardiovascular and neuroendocrine responses to challenge in males and females. In N. Schneiderman, S. P. Weiss & P. G. Kaufmann (Eds.), Handbook of Research Methods in Cardiovascular Behavioral Medicine. (pp. 453-484). New York: Plenum Press.

Schneiderman, N. & P. M. McCabe. (1989). Psychophysiologic strategies in laboratory research. In N. Schneiderman, S. P. Weiss & P. G. Kaufmann (Eds.), Handbook of Research Methods in Cardiovascular Behavioral Medicine. (pp. 349-364). New York: Plenum Press.

Selye, H. (1980). The nature if stress and its relation to cardiovascular disease. In G. H. Bourne (Ed.), Hearts and Heart-like Organs. Volume 2, (pp. 289-332). New York: Academic Press, Inc.

Semple, C. G., C. E. Gray, W. Borland, C. A. Espie & G. H. Beastall. (1988). Endocrine effects of examination stress. Clinical Science, 74, 255-259.

Smith, M. A. & K. D. Allred. (1989). Blood pressure responses during social interaction in high and low cynically hostile males. Journal of Behavioral Medicine, 11, 135-143.

Smith, M. A. & B. K. Houston. (1987). Hostility, anger expression, cardiovascular responsivity, and social support. Biological Psychology, 24, 39-48.

Stevens, J. (1992). Applied Multivariate Statistics for the Social Sciences. Hillsdale, N.J.: Lawrence Erlbaum Associates, Publishers.

Suarez, E. C., E. Harlan, M. C. Peoples & R. B. Williams. (1993). Cardiovascular and emotional responses in women: The role of hostility and harassment. Health Psychology, 12(6), 459-468.

Suarez, E. C. & R. B. Williams. (1989). Situational determinants of cardiovascular and emotional reactivity in high and low hostile men. Psychosomatic Medicine, 51, 404-418.

Suls, J. & C. K. Wan. (1993). The relationship between trait hostility and cardiovascular reactivity: A quantitiative review and analysis. Psychophysiology, 30, 615-626.

Tabachnick, B. G. & L. S. Fidell. (1989). Using Multivariate Statistics, Second Edition. Northridge, CA: Harper Collins Publishers.

Van Doornen, L. (1991). Stress and the dynamics behind hypertension, cholesterol and atherosclerosis. In A. Appels, J. Groen, J. Koolhaas, J. van Dixhoorn, L. Van Doornen, C. Mendes de Leon & C. Meesters (Eds.), Behavioral Observations in Cardiovascular Research,. (pp. 79105). Rockland, MA: Swets and Zeitlinger Inc.

Vining, R. F. (1984). Closing Session Discussion. Proceedings of the Ninth Tenovus Workshop, 1982. In G. F. Read, D. Riad-Fahmy, R. F. Walker & K. Griffiths (Eds.), Immunoassays of Steroids in Saliva, (pp. 343-346). Cardiff: Alpha Omega Publishing Ltd.

Vining, R. F. & R. A. McGinley. (1985). Hormones in saliva. CRC Critical Reviews in Clinical Laboratory Sciences, 23(2), 95-145.

Vining, R. F. & R. A. McGinley. (1987). The measurement of hormones in saliva: Possibilities and pitfalls. Journal of Steroid Biochemistry, 27(1-3), 81-94.

Vining, R. F., R. A. McGinley, J. J. Maksvytis & K. Y. Ho. (1983). Salivary cortisol: A better measure of adrenal cortical function than serum cortisol. Annals of Clinical Biochemistry, 20, 329-335.

Watkins, L. O. & E. Eaker. (1986). Population and demographic influences on reactivity. In K. A. Matthews, S. M. Weiss, T. Detre, T. M. Dembroski, B. Falkner, S. B. Manuck & R. B. Williams (Eds.), Handbook of Stress, Reactivity, and Cardiovascular Disease,. (pp.236-239). New York: John Wiley & Sons.

Williams, R. B. (1989). Biological mechanisms mediating the relationship between behavior and coronary heart disease. In A. W. Siegman & T, M, Dembroski (Eds.), In Search of Coronary-Prone Behavior: Beyond Type A, (pp. 195-205). Hillsdale, N.J.: Lawrence Erlbaum Associates, Publishers.

Williams, R. B. & N. B. Anderson. (1987). Hostility and coronary heart disease. In J. W. Elias & P. H. Marshall (Eds.), Cardiovascular Disease and Behavior. (pp.17-37). New York: Hemisphere Publishing Corporation.

Williams, R. B., J. D. Lane, C. M. Kuhn, W. Melosh, A. D. White & S. M. Schanberg. (1982). Type A behavior and elevated physiological and neuroendocrine responses to cognitive tasks. Science, 218(29), 483-485.

Zuckerman, M. & B. Lubin. (1965). Manual for the Multiple Affect Adjective Check List. San Diego, CA: Educational and Industrial Testing Service.

Appendix 1: Medical History Screening Questionnaire

	YES	NO
1. Have you had any of the following major illnesses or surgeries:		
• liver disease	[ ]	[ ]
• kidney disease	[ ]	[ ]
• thyroid disease	[ ]	[ ]
• adrenal or pituitary tumors	[ ]	[ ]
• diabetes	[ ]	[ ]
2. Do you have high blood pressure?	[ ]	[ ]
3. Does anyone in your family have high blood pressure?	[ ]	[ ]
4. Do you smoke?	[ ]	[ ]
5. Do you regularly consume tea, coffee, cola drinks, chocolate products?		
6. Do you regularly exercise on a weekly basis?	[ ]	[ ]
• how many times per week ?	____	
7. How many hours do you usually sleep each night?	____	
• approximate bedtime last night	____	
• approximate waking time this morning	____	
8. What medications are you currently taking on a regular basis?		
• include both prescription and over-the-counter (e.g., aspirin)		

If you are female:

9. Are you currently taking oral contraceptives?	[ ]	[ ]
10. Are you or could you be pregnant?	[ ]	[ ]

Appendix 2: Instructions to subjects

THE NIGHT BEFORE YOUR STUDY VISIT:

- Try to get your usual amount of sleep and at the usual time
- Be careful when brushing and flossing your teeth the night before, try not to make your gums bleed

For at least 12 hours prior to study visit, DO NOT :

- EXERCISE VIGOROUSLY
- CONSUME ALCOHOL
- SMOKE
- CONSUME CAFFEINE

- Get up at least one hour before your study visit
- Eat a light breakfast (e.g., juice, milk or yogurt, and toast, muffin, or cereal; **REMEMBER no coffee, tea, hot chocolate, colas or any food containing chocolate!**)

- **Do not brush or floss your teeth the morning of the study visit**, just rinse your mouth with mouth wash or water or chew gum (your gums cannot have recently bled)

Appendix 3: Data Collection Form

Data Sheet: Condition H

Date of Visit _____ Time _____

Subject # _____ Initials _____

Gender: F [ ] M [ ] Age _____

Height _____ Weight _____

ADAPTATION	**SBP**	**DBP**	**HR**
Minute 0	_____	_____	_____
Minute 3	_____	_____	_____
Minute 7	_____	_____	_____
Minute 10:30	**Experimenter —**	**Warning for Sample 1**	
Minute 11	_____	_____	_____
Minute 12	**Experimenter —** *"Stop filling out questionnaires now & gently spit into Vial 1."*		

TASK	**SBP**	**DBP**	**HR**
Minute 13	**Assistant —** *"Begin subtracting by 7's starting from 9000; give your answers aloud."*		
Minute 13	_____	_____	_____
Minute 15	**Assistant —**	**Statement 1**	
Minute 16	_____	_____	_____
Minute 19	**Assistant —**	**Statement 2**	
Minute 20	_____	_____	_____
Minute 23	**Assistant —**	**Statement 3**	
Minute 24	_____	_____	_____
Minute 25	**Experimenter —** *"Stop subtracting now & gently spit into Vial 2. When you have finished with the saliva sample, return to completing the questionnaires."*		

RECOVERY	**SBP**	**DBP**	**HR**
Minute 26	_____	_____	_____
Minute 31	_____	_____	_____
Minute 35	_____	_____	_____
Minute 37:00	**Experimenter —**	**Warning for Sample 3**	
Minute 38	**Experimenter —**	*"Please take Vial 3 and give another saliva sample."*	
Minute 39	_____	_____	_____
Minute 49:00	**Experimenter —**	**Warning for Sample 4**	
Minute 50	**Experimenter —**	*"Please take Vial 4 and give another saliva sample."*	
Minute 51	_____	_____	_____
Minute 61:00	**Experimenter —**	**Warning for Sample 5**	
Minute 62	**Experimenter —**	*"Please take Vial 5 and give the last saliva sample."*	
Minute 63	_____	_____	_____

Appendix 4: Script of Harassing Statements

All harassing statements will be read by a same-sexed Experimental Assistant at the following times:

MINUTE 15 (2 minutes into task)

"Look (*insert subject's name*), you are subtracting *way too slow*. You've got to do it *much faster*. Continue where you stopped.

MINUTE 19 (6 minutes into task)

(*Insert subject's name*), you're still *too slow* and *also inaccurate*. This *can't* be your best. Now try it again from where you left off.

MINUTE 23 (10 minutes into task)

You're obviously not good enough at doing this, *now try harder*. Keep going!

MINUTE 24:15

EXPERIMENTER: In about 45 seconds I'll ask you give another saliva sample so please allow the saliva to collect in your mouth.

MINUTE 25 AFTER BLOOD PRESSURE MEASUREMENT

Stop subtracting.

EXPERIMENTER: Please produce a saliva sample in the vial marked 2.

Appendix 5: Consent and Debriefing Forms

CONSENT FORM

I _____ agree to participate in a research project entitled "Intellectual Performance and Physiological Arousal: Part II" which is to be conducted in the cardiovascular psychophysiology lab in the Psychology Department at the University of British Columbia with Dr. Wolfgang Linden as the Principal Investigator, (phone: 822-4156). This study will involve the completion of various questionnaires during a one hour lab session (+ 5 to 10 minutes for explanations). The procedures to be followed and their purposes have been explained to me. As I understand it, the study requires me to relax for 12 minutes, engage in a mental arithmetic task for 12 minutes while being monitored, and then relaxing and completing questionnaires for another 36 minutes (total time 62 minutes). I also understand multiple cardiovascular functions will be monitored throughout the session and I am to give five samples of my saliva for analysis. All monitors are non-invasive in nature, are harmless, and lead to no foreseeable risks to my health or safety. I understand that I may refuse to participate or withdraw from the study at any time without influence on my class standing. All information is strictly confidential. While findings may be used in future studies, there will be no identification of me personally on any permanent records. All information will be recorded in group form and will remain strictly anonymous. I am aware that I have the right to ask and receive answers to any questions related to the experiment. Questions, if any, have been answered to my satisfaction.

If you had problem with the experiment, you can contact either Dr. Linden at 822-4156 or Dr. James Russell at 822-2549. Dr. Russell is in charge of the subject recruitment committee. I have read and understood the content of this form. I have received a copy of this consent form.

Research Participant Witness

_____ _____

Date: _____ Laboratory : 822-3800

DEBRIEFING

Now that you have completed participation in our study, we want to explain it's full purpose.

We are not surprised if you found part of the study unpleasant; we are now going to explain why we frustrated you with our negative comments on your performance.

Research shows that anger coping skills (keeping anger in vs. letting anger out) and high levels of hostility are associated with blood pressure and heart rate responses. Extreme cardiovascular responses are believed to be involved in the development of heart disease. Previous studies have shown an association between the development of heart disease and high hostility scores (from a questionnaire measure). The findings obtained in this study may tell us about the relationships between gender , how people express anger and other negative emotions, and cardiovascular and endocrine (hormonal) responses under stress. In addition, we also wanted to find out how the negative evaluation of the frustrator/ experimental assistant changed the cardiovascular and hormonal response. Therefore, half of all subjects received the same negative comments as you were given, and the other half received no feedback at all.

Thus, the goals of the study were:

1) To examine the effect of harassment versus no harassment during a stressful task (mental arithmetic) on cardiovascular (i.e., blood pressure and heart rate) and salivary cortisol responses and recovery.
2) To explore any gender differences in cardiovascular or cortisol response or recovery.
3) To explore the relationship between anger expression style and cortisol &/ or cardiovascular response patterns.

Since the subject matter we are studying is anger and anger-coping skills, we view it as essential to annoy subjects somewhat, in order to make the task similar to a realistic situation. The frustration and negative comments obviously would not have worked if you were informed before the task.

So once again, we apologize for the frustration and negative feeling you may have had during parts of the study. The negative comments on your performance were from a standard scripts and by no means reflect your actual performance on the serial subtraction task. It is important that you do not share this information with others or else our study will be contaminated. We welcome your input so that we can improve our study design. We really appreciate your participation in this study to further our knowledge of how stress influences cardiovascular and hormonal responses. Coronary heart disease is the leading cause of death in the western world; therefore studies such as ours which help us to understand how coronary heart disease develops and who might be at risk could provide valuable information.

I would like to know how you feel now about the experiment after I explained the design and purpose to you. Please share your feelings with us and feel free to make suggestions you may have about the design of the study. Thanks again for participating.

If you would like to know the results of the study, a copy of the report will be available in approximately six months from the following address:

Dr. W. Linden
Department of Psychology
2136 West Mall
University of British Columbia
Vancouver, B.C. V6T 1Z4

(Phone: 822-3800)

If you are interested in this area of research and would like to read more about it, you could start with:

Gentry, W., A. Chesney, H. Gray, R. Hall, & E. Harburg. (1982). Habitual anger coping styles: Effect on mean blood pressure and risk for essential hypertension. Psychosomatic Medicine, 44: 195-202.

Lai, J. Y. & W. Linden. (1992). Gender, anger expression style, and opportunity for anger release determine cardiovascular reaction to and recovery from anger provocation. Psychosomatic Medicine, 54: 297-310.