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Lifetime Stress, Blood Pressure, Heart Rate, and Salivary Cortisol Levels in Post-Menopausal Women.

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A Thesis

in

The Department

of

Psychology

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For the Degree of Master of Arts at
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Abstract

Lifetime Stress, Blood Pressure, Heart Rate, and Salivary Cortisol Levels in Post-Menopausal Women.

Wendy Smith

Post-menopausal women are a unique group who may be at increased risk for the development of coronary heart disease. As such, more research is needed to understand the physiology of these women. This study had two purposes. The first was to assess the effects of past stress levels on blood pressure (BP), heart rate (HR), and salivary cortisol excretion in post-menopausal women. The second purpose of this study was to examine the interactions between the hypothalamic-pituitary-adrenal (HPA) axis and the cardiovascular system in these women. Two measures of past stress were examined in relation to BP, HR, and cortisol levels. The Concordia Lifetime Stress Graph (CLSG) provided a measure of lifetime stress, and scores on the Life Experiences Survey (LES) provided a measure of stress in the past year. Salivary cortisol levels, BP, and HR were examined both at baseline and in response to a laboratory stress test comprised of an anticipatory period, an oral defense, and a mental arithmetic task. Results indicated that neither stress experienced over the lifetime or over the past year was related to baseline or reactive BP, HR, or cortisol levels. Other results assessing the interaction between the HPA axis and the cardiovascular system indicated a negative relationship between cortisol reactivity and baseline BP. This negative relationship is inconsistent with previous findings of a positive relationship in younger men. The findings of this study suggest that past levels of stress in post-menopausal women do not influence current functioning of the HPA axis or the cardiovascular system. In addition, the relationship

between the cardiovascular system and the HPA axis may be different in post-menopausal women than it is in younger men.

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Stress is the body's reaction to a variety of arousing situations and experiences that can be as different from one another as sexual arousal, pain, fatigue, or loss of blood. The primary goal of the stress response is to mobilize energy for immediate use. In order to conserve energy, the stress response temporarily inhibits certain processes such as digestion, reproduction, and functioning of the immune system. In addition, pain perception and inflammation are suppressed (Sapolsky, 1990). The stress response functions primarily through the functioning of two systems; the hypothalamic-pituitary-adrenal (HPA) axis, and the sympathetic nervous system. Research has investigated the functioning of each of these systems, both separately and in relation to one another. A lot is known about the interaction between the two systems in rats, but less is known about the interaction between the cardiovascular system and the HPA axis in humans.

In humans and rats, when a stressor is perceived, a chain reaction begins within the HPA axis. Corticotrophin-releasing hormone (CRH) is released by the hypothalamus within a few seconds of the stressor. About 15 seconds later, CRH causes the secretion of adrenocorticotropic hormone (ACTH) from the pituitary gland, which, in turn, results in increased glucocorticoid release from the adrenal cortex within a few minutes (Sapolsky, 1990). The secretion of glucocorticoids in response to a stressor has been called the general adaptation syndrome (Selye, 1936). The main role of glucocorticoids is to increase the availability of glucose to provide energy to the muscles. The primary glucocorticoid in humans is cortisol.

At the same time that the HPA axis is activated by a stressor, so is the sympathetic nervous system (SNS). There are two major components to this system, the SNS nerves

that regulate cardiovascular activity, and those that innervate the adrenal medulla (Cryer, 1980). The SNS nerves regulating cardiovascular activity innervate the heart, vasculature, skeletal muscles, and major organs. In times of stress, the SNS nerves regulating cardiovascular functioning show increased activity. There is an increase in neurotransmitter release, the primary of which is norepinephrine (NE; Cryer, 1980). NE binds primarily to α-adrenergic receptors producing vasoconstriction in many organs, resulting, for example, in pupil dilation. NE also binds to β-adrenergic receptors, among other effects, increasing the force and rate of heart contractions, resulting in accelerated HR, and a rise in BP (Tortora, 1995).

Although the adrenal medulla component of the SNS also responds to stress with NE release, its primary response is increased epinephrine (EPI) secretion. EPI binds nonspecifically to both α- and β-adrenergic receptors. This increased catecholamine binding serves to intensify the actions of NE on the cardiovascular system, thus sustaining increased BP and HR (Schneiderman, Ironson & McCabe, 1987). The adrenal medulla also has other roles, for example, it participates in glucose regulation and metabolism (Ganong, 1987).

The differential response pattern elicited by α-adrenergic and β-adrenergic binding is dependent in part on the nature of the stressor. For example, reaction time tasks and mental arithmetic are considered to be β-adrenergic tasks, thus inducing changes in HR, BP, catecholamine release, and muscle blood flow (Allen, Boquet & Shelley, 1991). The cold pressor task elicits an α-adrenergic response pattern, demonstrated by vasoconstriction (Allen et al., 1991).

A number of factors have been studied in relation to cardiovascular expression of the stress response. These factors include, but are not limited to, the type of coping required for the task (i.e. active or passive), the personality and mood of the subject, and the subjective judgement of the impact of the stressor (see Krantz, Manuck & Wing, 1986; Krantz & Lazar, 1987 for reviews). Further, researchers have found evidence that people tend to show a specific pattern of response to stress across different tasks. For example, subjects who are "β–responders" tend to show the same physiological responses associated with β-adrenergic activity across tasks such as cold pressor and mental arithmetic, which generally elicit different response patterns (Allen et al., 1991).

As well as examining cardiovascular reactivity to laboratory stress, researchers investigate ambulatory cardiovascular activity and reactivity in real life situations. Comparisons between cardiovascular data collected in the laboratory and in the field are important in determining the external validity of laboratory measures. Studies suggest that there is some correlation between laboratory and ambulatory measures. For example, Fredrikson, Blumenthal, Evans, Sherwood and Light (1989) exposed 55 men to a 15 minute mental arithmetic task while measuring SBP, DBP and HR. The researchers also obtained baseline measures of these variables. The participants were asked to wear a BP-HR monitor the day following the laboratory session, and three BP and HR readings were taken each hour for 12 hours. Results indicated that significant relationships existed between laboratory baseline measures and stress measures and ambulatory measures for SBP, DBP, and HR. The reactivity laboratory measure was not related to any of the ambulatory measures. The authors suggest that mental arithmetic is a task eliciting

primarily increased β-adrenergic activity resulting in increased BP and HR, and this pattern may be more reflective of real life stress than the α-adrenergic pattern of increased vasoconstriction.

Cardiovascular functioning also appears to be influenced by age. β-adrenergic activity decreases as a function of age, maximal HR declines, and resistance to blood flow increases (see Ucino, Kiecolt-Glaser & Cacioppo, 1992). This results in changes in cardiovascular activity and reactivity. For example, studies report that HR reactivity decreases as a function of age in men (Ginter, Hollandsworth & Intrieri, 1986) and in women (Ditto, Miller & Maurice, 1987). In addition, an increase in baseline SBP and DBP with age has been noted (Steptoe, Moses & Edwards, 1990; Uchino, Kiecolt-Glaser & Cacioppo, 1992), as well as increases in BP reactivity (Ditto et al., 1987; Garwood, Engel & Capriotti, 1982). A recent longitudinal and cross-sectional study suggests that baseline SBP and DBP are stable in adults up to the age of approximately 45 years then they gradually increase until they plateau at age 70 (Pearson, Morrell, Brant, Landis & Fleg, 1997).

Interestingly, menopausal state may interact with age in determining cardiovascular functioning in women. Post-menopausal women have higher SBP than pre-menopausal women (Bonithon-Kopp, Scarabin, Darne, Malmejac & Guize, 1990). This increase in SBP in women following menopause may be due to lack of estrogen. In fact, SBP can be decreased in post-menopausal women by administration of estradiol (Lindheim et al., 1992). This finding illustrates the importance of studying the effects of stress on cardiovascular activity in post-menopausal women. Their hormonal status

renders them a unique group and they have not been studied in detail. It is important to research this group as they may be at increased risk for the development of coronary heart disease (Bonithon-Kopp, et al., 1990).

In addition to increased cardiovascular functioning, stress also leads to increased cortisol release which can be detected in the blood, urine, or saliva a few minutes after onset of a stressor. The stressor can be physical, such as exercise (Tacker, Leach, Owen & Rummel, 1978) or psychological, such as an oral presentation (Evans, Bistow, Hucklebridge, Clow & Pang, 1994), a short-term memory task, or a multiple-choice test (Wittersheim, Brandenberger & Follenius, 1985). One laboratory task which is reported to increase cortisol release as well as BP and HR in younger adults is comprised of an anticipation and planning period of a simulated job interview lasting 10 minutes (a 5 minute interview where the subject must explain to a committee of three confederates why he should be hired while being video- and audio-taped, followed by 5 minutes of mental arithmetic; counting backwards from 1022 by 13 being told to start over every time a mistake is made (Kirschbaum, Pirke & Hellhammer, 1993)).

Researchers have investigated gender differences in cortisol levels, both at baseline and in response to stress. Within the population of young and middle-aged adults, baseline cortisol levels are not found to be different as a function of gender (e.g. Kirschbaum, Wüst & Hellhammer, 1992). In addition, gender differences are found in young and middle-aged adults in the magnitude of cortisol increases in response to stress. Males of these ages show a larger cortisol increase in response to stress than females do (e.g., Kirschbaum, Klauer, Filipp & Hellhammer, 1995; Kirschbaum, Wust & Hellhammer, 1992). The relationship between cortisol levels and gender may be different

in older adults. Research findings from a study examining cortisol levels in adults aged between 60 and 80 years indicate that men of this age have higher basal cortisol levels than women (Olsson et al., 1989).

Age differences in cortisol levels have also been examined. Many researchers have found increased cortisol release with increasing age (e.g., Halbreich, Asnis, Zumoff, Nathan & Shindledecker, 1984; Jacobs, Mason, Kosten, Brown & Ostfeld, 1984), whereas other researchers have not found this increase (e.g., Ohashi, Fujio, Nawata & Ibayashi, 1986; Lindheim et al., 1992; Sherman, Washam & Pfohl, 1985). Results of other studies suggest that differences in cortisol concentrations as a function of age may only be detectable under conditions of stress. For example, one study reported that 2 months after a spouse was hospitalized for a major illness, older adults had higher cortisol levels than younger adults (Jacobs et al., 1984). A more recent study reported that although basal cortisol levels did not differ between younger and older participants, cortisol secretion during a cognitive challenge task (signal detection) was higher in the older group (Gotthardt et al., 1995).

Although HPA activity in response to stress is well documented, the long-term effects of stress on HPA axis activity are less understood. The prevailing view is that once a stressor is terminated the body returns to a state of homeostasis, or an internal balance. Upon termination of a stressor, there is a negative feedback loop which halts the production of CRH so that secretion of cortisol decreases back to basal levels, restoring the internal environment to homeostasis. More recent research suggests, however, that while this negative feedback loop may exist in certain brain regions such as the paraventricular nucleus of the hypothalamus, other regions, notably the amygdala, do not

seem to be under the same homeostatic control. In fact, when CRH increases in response to increased levels of glucocorticoids the amygdala adapts to keep levels high instead of trying to stop production of the hormone (Schulkin, McEwen & Gold, 1994). The amygdala is involved in emotional arousal, so increased glucocorticoids in this structure may result in increased fear and anxiety, along with an increase in negative expectations (Schulkin, et al., 1994). This is an example of what has been called allostasis; the constant changing of the internal environment, and the belief that the internal mileu is affected by both what has happened in the past, and what the individual anticipates happening in the future (Sterling & Eyer, 1981). The more stress experienced, the more stress the individual expects to experience, resulting in even higher elevations in glucocorticoid release in response to subsequent stressors. This may eventually lead to higher basal levels of cortisol, according to the glucocorticoid cascade hypothesis (Sapolsky, Krey & McEwen, 1986). This hypothesis states that repeated exposure to stress results in constant high levels of glucocorticoids, which, in turn, leads to over-activity of the glucocorticoid receptors in the hippocampus, eventually resulting in loss of receptor capacity, and HPA axis disinhibition. Thus, it would be expected that individuals who have experienced more stress throughout the lifetime would not only respond with increased cortisol release to a subsequent stressor, but also would eventually have higher basal levels of this hormone.

There is little support for the theory of allostasis, primarily because relatively few researchers have looked at the long-term effects of stress on glucocorticoid release. One study, however, examined the effects of a major life stressor on cortisol release 6 months later (Willis, Thomas, Garry & Goodwin, 1987). Participants were already part of another

study, so baseline measures of pre-crisis cortisol levels were available. Major life stressors experienced were death of a child or a spouse, diagnosis of a major illness of a family member, or divorce. The researchers found that serum cortisol levels rose above baseline at the time of the stressful event, and although cortisol concentrations were decreased 6 months after the crisis, they had still not returned to baseline levels. Unfortunately, no follow-up study was done on these subjects to see if cortisol concentrations eventually returned to pre-crisis levels, or remained elevated years later. It is possible that the elevated glucocorticoid release six months after the crisis is actually in response to many smaller stressors due to the stressful changes in lifestyle brought about by the crisis, and not in response to the crisis itself. These results do, however, show a need for more research investigating long-term effects of stressful life experiences.

Perhaps a reason for the lack of research investigating effects of stress throughout the lifetime on HPA axis activity is the lack of an adequate measure of lifetime stress. Most of the available instruments to assess the occurrence and impact of major life stressors assess only the events which occurred within a year prior to assessment. This is especially problematic when these measures are used with an elderly population, given a finding of an inverse relationship between number of events experienced on the Lifetime Events Survey (LES) and age (Lazarus & Delongis, 1983). The stressful events listed on the LES are less likely to have happened within the past year in the life of older adults, and a measure of the events experienced over the lifetime is perhaps more appropriate for this population. The Concordia Lifetime Stress Graph (CLSG) is a retrospective measure of past experiences over the lifetime and allows subjects to rate how stressful these experiences are remembered as being (Bonneville, 1995).

Although it is not known whether or not ratings of past stress are related to current cortisol levels, research findings suggest that ratings of current stress do correlate with current cortisol levels. For example, cortisol release during a typical workday was found to be higher in subjects with high scores on the Perceived Stress Scale (PSS) than in subjects with low PSS scores (van Eck & Nicolson, 1994). In another study, participants completed the Daily Stress Inventory (DSI), which provides measures of both the frequency and the subjective intensity of stressors, on 10 consecutive days. In addition, urinary cortisol levels were obtained for each day. The cortisol levels on the two days with the highest DSI scores (i.e. subjectively rated as the most stressful) were significantly higher than cortisol levels on the 2 days with the lowest DSI scores (i.e. subjectively rated as the least stressful; Brantley, Dietz, McKnight, Jones & Tulley, 1988). The results of these studies indicate that people are able to subjectively rate their current stress level such that it is correlated with HPA axis activity. Perhaps people are also able to judge the severity of past stressors such that these ratings would correlate with cortisol concentrations at the time the stressors were experienced. Although the relationship between subjective stress and cortisol response to stress cannot be assessed post-hoc, the glucocorticoid cascade hypothesis postulates that current baseline cortisol levels are a function of past life stress (Sapolsky et al., 1986). According to this hypothesis, if people are accurately rating the severity of their past stress it should be related to current baseline cortisol levels.

In addition to influencing HPA axis activity, past stress may also have some influence on cardiovascular reactivity. For example, one study found that BP of students who scored high on the Life Experiences Survey (LES; a measure of frequency and

intensity of stress over the past year) took more time to return to baseline after a laboratory stressor than the BP of students who scored low on the LES (Pardine & Napoli, 1983). These results suggest that the amount of stress experienced in the past year influences subsequent cardiovascular functioning. In addition, the animal literature shows that rats exposed to chronic stress are more likely to develop hypertension (Lawler, Cox, Sanders & Mitchell, 1988). This may suggest that people who have experienced more stress throughout the lifetime are more at risk for the development of hypertension.

Although the cardiovascular system and the HPA axis both respond to stress, it is unclear in what way, if any, these systems are linked. One study provides some support for an interaction between the HPA axis and the cardiovascular system. Men who were higher in HR reactivity showed a significant increase in cortisol release following an aversive shock avoidance task whereas those who were low in HR reactivity did not show this increase (Lovallo, Pincomb, Brackett & Wilson, 1990). The researchers note that all subjects rated the task as equally stressful, so the difference in cortisol level observed between HR reactivity groups was probably not due to differences in perception of level of stress. They speculate that the differences in HR and cortisol reactivity are due to differential responses of the hypothalamic and medullary control centres to equal amounts of cortical stimulation.

Hypertensives or borderline hypertensives are often used in research to investigate the possible relationship between the cardiovascular system and cortisol levels. In one study which investigated the functioning of the HPA axis and the cardiovascular system, borderline hypertensive (140/90 < BP < 160/96 mm Hg) and normotensive (BP < 130/85 mm Hg) males were subjected to mental stressors (Al'Absi, Lovallo, McKey & Pincomb,

1994). A reaction time task interspersed with mental arithmetic produced an increase in cortisol release in the borderline hypertensive men, but not in the normotensive men. This suggests that differences exist in reactivity to stress in cortisol levels as a function of hypertensive status.

The mechanism of this difference in cortisol levels as a function of hypertensive state has been investigated in humans and animals. It has been known for some time that spontaneously hypertensive rats have higher levels of adrenal hormones than normal rats (Aoki, Takikawa & Hotta, 1973). In human hypertensives, however, it may be that cortisol levels are normal, but tissue sensitivity to the hormone may be altered. Cortisol may retard catecholamine degradation in tissues and increase sensitivity of adrenergic receptors (Al'Absi, Lovallo, McKey & Pincomb, 1994). Walker, Best, Schackleton, Padfield and Edwards (1996) speculate that hypertensives have increased vasoconstrictor sensitivity to glucocorticoids that may initiate increased peripheral vascular resistance which is a characteristic of hypertension. The mechanism of this increased sensitivity is, however, uncertain. There may be a genetic component to this increased sensitivity, because hypertensives often have a family history of hypertension (Cinciripini, 1986; Light, 1987).

The goal of the current study was to increase the understanding of the relationships between stress, SBP, DBP, HR, and cortisol. Given the shortage of research assessing these relationships in post-menopausal women as well as the increased risk of this population for the development of coronary heart disease (Bonithon-Kopp et al., 1990), the specificity of the relationships between these variables in older women was examined.

The stressful laboratory task was modified from that of Kirschbaum and his colleagues (1993). The subject was told to imagine that she had been wrongly accused of shoplifting (Miller & Sita, 1994) and she had to defend herself to an accomplice. She then had to count backwards from 1022 by 13 and was required to correct mistakes she made. The modified stressful task was evaluated in this study to assess its ability to increase physiological measures of stress in an elderly population. It was expected that the task would cause an increase in all physiological measures, SBP, DBP, HR and salivary cortisol levels.

The relationship between current and past stress and physiological responses was investigated. Given the research showing people's ability to rate their stress such that it correlates with physiological measures (e.g., Brantley et al., 1988; van Eck & Nicolson, 1994), it was expected that cortisol samples taken at various times throughout the course of a typical day (providing a cortisol diurnal record) would correlate with subjects' stress ratings for the same points. In addition, the relationships between lifetime stress levels and physiological responses were investigated. According to the glucocorticoid cascade hypothesis (Sapolsky et al., 1986), more cortisol release during the diurnal record as well as at baseline measures was anticipated for those participants who had experienced more stress throughout the lifetime.

It was also expected that the women reporting more lifetime stress would have a higher increase in cardiovascular measures when they were anticipating stress. The theory of allostasis states that the experience of stress is moderated in part by the experience of past stressors and the anticipation of the current stressor (Schulkin et al., 1994, Sterling & Eyer, 1981). Further, subjects with higher scores on the LES were expected to have

higher SBP following the stressful task for longer than the subjects with lower LES scores as was previously shown in college students (Pardine & Napoli, 1983).

Another purpose of this study was to examine the effects of completing the CLSG on cortisol levels. Preliminary research suggests that completion of the CLSG causes an increase in SBP, DBP and HR. When a subject is discussing an event rated as being more stressful, her SBP, DBP and HR are higher than when she is discussing an event she rates as being less stressful (unpublished observations). This type of comparison of physiological responses to discussing a particular stressor was not examined with relation to cortisol levels because the increase is not detected immediately, but rather 15 to 20 minutes after the onset of a stressor. Changes in cortisol levels as a result of completing the CLSG were examined.

Another purpose of this study was to investigate the relationship between cortisol reactivity and cardiovascular activity, as measured by resting blood pressure and heart rate. Previous research suggests that individuals who have higher resting blood pressure (Al'Absi et al., 1994) have higher cortisol reactivity to a stressful task. The relationship between cortisol and cardiovascular measures has not been studied in post-menopausal women, and an important aim of this study was to explore this relationship in this population.

Method

Participants

Thirty postmenopausal women between 55 and 70 years of age (M = 61.83, SD = 4.77) were recruited by word of mouth, participation in previous research projects at Concordia University, newspaper stories, and flyers placed throughout the Montreal Urban Community. Individuals on blood pressure medication or hormone replacement therapy were excluded from this study to avoid possible confounds of these medications on cortisol and cardiovascular activity and reactivity. The procedure of this study involved some writing, so the blood pressure cuff had to be placed on the non-dominant arm. For consistency in cuff placement, only right-handed women participated in this study. The average level of education of the participants was 13.97 years (SD = 2.62). Participants were paid 40 dollars each as an honorarium.

Of the 30 participants, two refused to complete the laboratory stress procedure. All other data for these subjects was collected.

Apparatus

Measurements of systolic (SBP) and diastolic (DBP) blood pressure (in mm Hg) and heart rate (HR in beats per min) were obtained using an IBS Model SD-700A automated blood pressure monitor (Waltham, Mass, USA). This unit employs a ruggedized sensor to detect arterial wall motion and audible as well as inaudible Korotkoff vibrations. A special filter identifies specific vibrations associated with obstructed blood flow between the SBP and DBP range. The filtered signals correlate closely with those obtained by standard ausculatory methods using a stethoscope (Matthews, Manuck & Saab, 1986: Matthews, Rakaczky, Stoney & Manuck, 1987). The

blood pressure cuff was placed on the left arm which was kept immobile during all testing.

Cortisol Analysis

Salivary cortisol samples were taken using Whatman #1 filter paper. The paper was cut into 7 x 3 cm strips with a line drawn at the 2 cm point to mark the saturation area. The strips were pre-marked with the sample number in the space outside the area of saturation. All preparation of the filters was done with surgical gloves. After saturation, the filters were placed individually in small plastic bags and frozen until they were assayed for cortisol. Frozen samples were transported by the subjects to the laboratory in a small insulated bag with a small freezer ice pack.

The assays were performed at the Douglas Hospital Research Laboratories. Cortisol levels in saliva were determined using competitive protein binding radioimmunoassay. Previous studies have shown an intra-(3.5%) and inter-(5%) assay variability which is within acceptable limits for the purpose of this study.

Psychological Measures

The Life History interview (Appendix A) was used to obtain basic demographic data and information on past and present occupation, present living conditions, and lifestyle. Subjects were weighed and their height was measured to produce a Body Mass Index (weight/height^2).

Health (Appendix B) was assessed by a checklist of past illnesses and hospitalizations similar to that used by physicians at intake interviews with new patients.

Participants also rated their current health status, and answered questions about prescription and non-prescription drug use, and menopause.

The Hassles Scale (Kanner, Coyne, Schaefer & Lazarus, 1981) was previously modified to reflect the concerns of an elderly population. The version used consists of 35 items which describe possible hassles in the areas of family, friends, health, finances, home and work environment, and chance occurrences. Subjects were asked to indicate how frequently each event occurred in the past month on a five-point Likert-type scale ranging from 1 (did not occur) to 5 (occurred extremely often). Test-retest reliability for frequency of hassles has been shown to be .79 (Kanner et al., 1981). The Hassles Scale was used to establish the current level of stress for the starting point on the CLSG.

The Life Experiences Survey (LES, Sarason, Johnson & Siegel, 1978) measures stress that occurred in the past year. Eight of the items on the original questionnaire were removed because they were judged not relevant to an older population, so the LES used consisted of 43 major positive and negative life events such as death of a spouse, retirement, or birth of a new family member. The subject was required to indicate which of the events occurred in the past year, and to rate the impact each event had on her life at the time it occurred on a Likert-type scale ranging from 7 (extremely negative) to 1 (extremely positive). The LES yields a score for the total negative life events which is the weighted impact scores of the negative events, and a score for the positive events which is the weighted positive events score. Subtraction of the positive events from the negative yields a measure of the negative stress experienced with the positive stress removed. Test-retest reliability of the LES over a five week period was found to be .64, and discriminant validity was found by comparing scores on the LES to scores on the Beck

Depression Inventory ($r = .06$) and scores on the Marlowe-Crowne Social Desirability Scale ($r = .01$) (Sarason et al., 1978). This test was given between the Hassles scale and the CLSG and, in addition to providing a measure of recent stressful life events, serves to bring to a subject's attention a standard list of major life events.

The Concordia Lifetime Stress Graph (CLSG, Bonneville, 1995, Appendix C) is a measure of lifetime stress. The subject was asked to construct a graphical representation of lifetime stress on a piece of graph paper. The background colour varies in intensity of red, from white at the abscissa though successively increased saturations of red to a deep red at the top of the page. The ordinate, which runs from the origin to the top of the page, is marked into six equal intervals, but no numeric values are assigned to the markings. The abscissa is marked in one year intervals with every fifth year labeled from 1935 through to 2000. It was explained to the subject that the amount of stress that she had experienced over her adult lifetime would be plotted, with greater stress being indicated by more saturated red. The year of the interview (1996 or 1997) was the end point of the graph, and was calculated as the subject's mean score on the Hassles Scale (ranging from 0.0 to 5.0). This was used as the starting point for plotting stressful events because previous research indicates that hassles may be an appropriate measure of current health and psychological well-being (e.g. DeLongis, Coyne, Dakof, Folkman & Lazarus, 1982; Kanner et al., 1981). The subject was able to change this point if she did not feel it correctly represented her average level of stress for the past year. The subject was then encouraged to think through the events of her life and approximate the level of stress she experienced at those times. The measure of stress derived from the graph is mean stress experienced since age 16. The CLSG has a test-retest reliability for a group of 49 older

women of .68 after approximately 15 months and has been shown to have good discriminant and convergent validity (Bonneville, 1995).

Procedure

Participation in the experiment consisted of two sessions with the interviewer: a session in the subject's home, and a session in the laboratory. Both of these sessions were scheduled between 12.30pm and 4pm to avoid the early morning fluctuations in cortisol. The participants were also asked to gather cortisol samples for one entire day between the two sessions. They were asked to take the first sample immediately upon awakening, and then take subsequent samples every two hours thereafter until they went to bed. This provided a diurnal record of cortisol levels. Samples were obtained by placing a filter in the mouth for a few seconds until it was saturated with saliva.

The in-home session took approximately 2 hours (see Appendix D). The subject first signed the consent form (Appendix E). She was asked to sit with the blood pressure cuff on for 10 minutes, during which time she relaxed and chatted quietly with the interviewer. After these 10 minutes, 5 baseline measures were taken, one per minute. A baseline cortisol sample was also taken at this time.

The cuff was then removed, and the subject completed the life history interview, the health questionnaire, the Hassles and the LES. The cuff was replaced and another baseline was taken. The subject then completed the CLSG, during which time blood pressure and heart rate were recorded every 3 minutes. Fifteen minutes after the start of the CLSG a cortisol sample was taken, followed by 3 more samples, at 10 minute intervals. To fill in the time during cortisol samples, subjects were given several paper and pencil psychosocial questionnaires including the Eysenck Personality Inventory

(Eysenck & Eysenck, 1968), the Buss-Durkee Hostility Inventory (Buss & Durkee, 1957), the Coping Strategies Inventory (Tobin et al., 1982), the Centre for Epidemiology Studies Depression Scale (Radloff, 1977) and the Social Support Questionnaire (Sarason et al., 1983). The psychosocial questionnaires are part of another study.

The subject was thanked for her participation in the first part of the experiment, and asked to continue to participate by collecting cortisol samples for an entire day. She was given 10 pre-marked filters, each in a small plastic bag. She was asked to put each filter back in the bag when she had saturated it, and to put each sample in the freezer as soon as possible after taking it. At the time of each sample, she was also asked to indicate her stress-level on a stress-graph similar to the CLSG. The participant was given both written and oral instructions and an insulated bag and an ice pack to transport the frozen samples to the lab (Appendix F). The day chosen to collect samples was left up to the subject. The next session was scheduled at least 5 days following the first part of the study, at the subject's convenience. She was asked to bring her frozen filters with her at this time.

Upon arrival into the laboratory for the second testing session (see Appendix G), the subject relaxed for 10 minutes, then blood pressure and heart rate baselines were obtained each minute for five minutes following the same procedure as in the home visit. A baseline cortisol measure was taken immediately following the blood pressure and heart rate measures.

The stress protocol used was divided into a 10 minute anticipation period and 5 minutes each of speech and mental arithmetic (Kirschbaum et al., 1993). The subject was told to imagine that she had been wrongly accused of shoplifting (Miller & Sita, 1994),

and had to verbally defend herself to a woman she does not know (an accomplice) for 5 minutes, and that she may be asked some questions. She was told that her defense would be videotaped for later viewing, and an overhead camera focused on the subject was pointed out to her. She was given paper and a pencil to outline her defense, but was told that she could not have the paper with her during her plea (see Appendix H). She was left alone for 10 minutes to prepare the talk. Blood pressure and heart rate were measured at 2 min intervals during this time.

After the preparation time, the accomplice entered the room. The subject was asked for her defense to the charge of shoplifting. If she stopped talking before the 5 minutes were up, she was asked a series of questions by the accomplice (Appendix I). Immediately following the 5 minute defense, the subject was asked to count backwards from 1,022 by 13 (Kirschbaum et al.,1993). Whenever an error was made, the subject was asked to correct it. This mental arithmetic continued for 5 minutes. Blood pressure and heart rate were measured every 2 minutes during the tasks.

Immediately following the tasks, a saliva sample was taken, and then three more samples were taken at 10 minute intervals. Heart rate and blood pressure were measured every 2 minutes for the first 10 minutes, and then at 5 minute intervals. During this 30 minute recovery period, a tape with music rated by college students as inducing a neutral mood (Pignatiello, Camp & Rasar, 1986) was played. The subject also had access to reading material such as cooking, gardening, and animal books. The examiner entered the room every 10 minutes to take a cortisol sample.

Following this 30 minutes, the cuff was removed, and the subject completed a variety of cognitive tests, including the Vocabulary and Picture Completion subtests of

the WAIS-R, the Self-Ordered Pointing Task (Petrides & Milner, 1982), and the Continuous Attention Task (Cegalis & Bowlin, 1991). The participants also completed the Concordia Lifetime Drinking Questionnaire (Chaikelson, Arbuckle, Lapidus & Gold, (1994) and the Concordia Lifetime Smoking Questionnaire (Chaikelson, Arbuckle & Lapidus, 1995) and the Habitual Physical Activities Questionnaire (Voorrips, Ravelli, Dongelmans, Deurenberg & Van Staveren, 1991). These questionnaires and cognitive tasks are part of another study. The subject was weighed and her height measured. She was told that her defense was not really videotaped and any questions she had were answered. She was then paid and thanked for her participation.

Statistical Analysis

A repeated measures ANOVA was conducted on the cortisol levels obtained on the home test day to determine whether differences existed as a function of sample time. Similarly, a repeated measures ANOVA was conducted on the cortisol levels from the laboratory test day. When significant effects were detected, post hoc analysis of the individual means was made using Tukey's honestly significant difference (Tukey) tests.

Means were generated for each subject for each of the SBP phases of the laboratory test day (i.e. baseline, anticipation, stress, post-stress 2 minutes, and post-stress 5 minutes). Means were then obtained across subjects for each of these phases. A repeated measures ANOVA was conducted on these means at each of the phases to assess differences in SBP as a function of time. The same statistical procedures were conducted for DBP data and HR data.

Results

All data were examined for skewness. The SBP, DBP, and HR data along with measures of stress, health, and diurnal cortisol were normally distributed. The home and laboratory day cortisol home and lab samples were skewed in the positive direction. A square root transformation was performed to make the distribution of scores fit a normal curve to allow analyses to be conducted. Outliers existed on the baseline home and laboratory SBP measures as well as on some measures of home and laboratory cortisol. These outliers were given a score equivalent to the mean of the variable plus three standard deviations.

The two subjects who did not complete the laboratory stress procedure did not differ from the rest of the sample on baseline SBP, DBP, HR and cortisol measures obtained on the home test day, or on the baseline measures of the laboratory test day. Because these subjects did not differ from the others on these measures and because other data used in various analyses was available for these subjects, they were not dropped from the sample. In addition to these data being missing for the laboratory test day, the home day baseline cortisol sample was missing for one subject. These missing data resulted in different sample sizes being used for different analyses.

Figure 1 shows the transformed cortisol data for the home test day. The ANOVA detected a significant main effect of time, $F(4,112) = 3.88$, $p<.05$. Post hoc analyses did not reveal any significant differences between the individual means.

The transformed cortisol data for the laboratory test day is shown in Figure 2. The times at which cortisol samples were taken during the laboratory test day are different from the times at which cortisol samples were taken during the home test day. During the

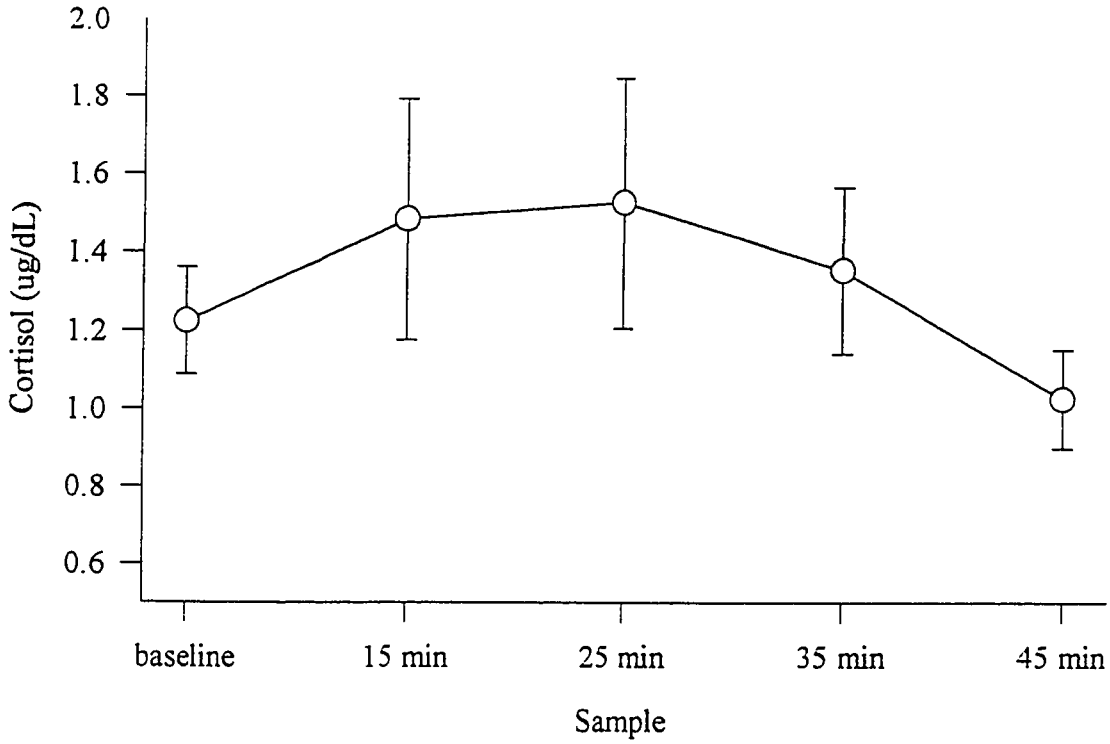

Figure 1. Cortisol levels during the home testing day. Times refer to time since beginning to complete the CLSG. Points represent the mean cortisol level; vertical bars depict standard errors of the means.

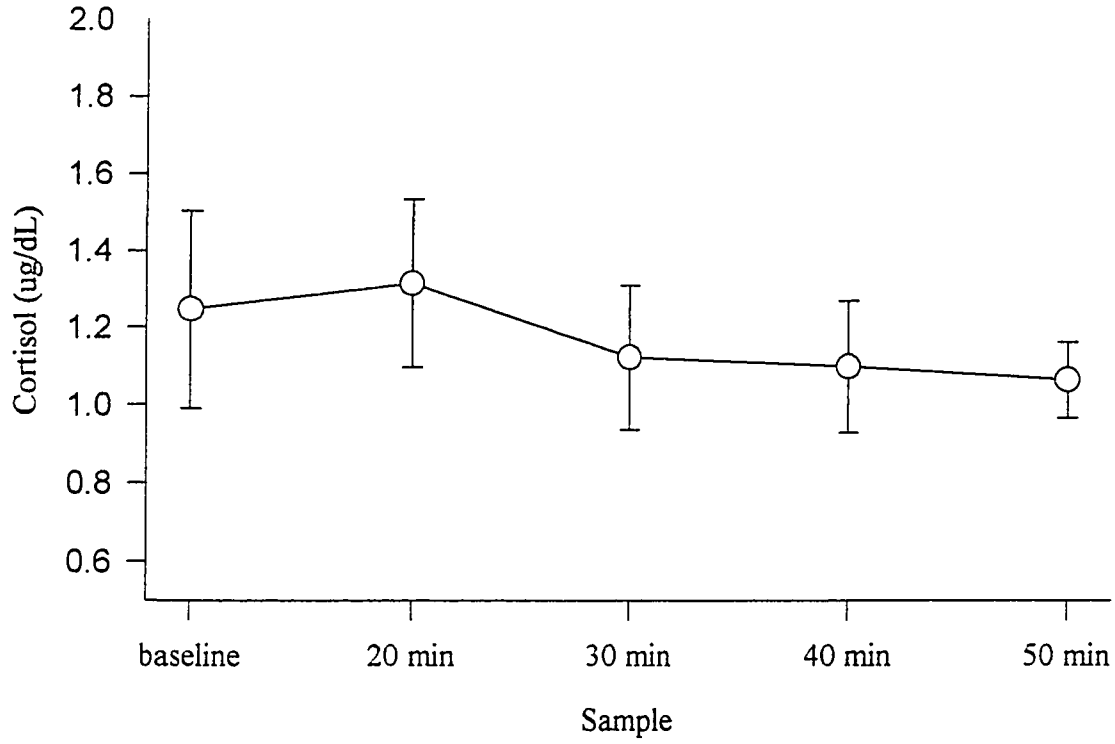

Figure 2. Cortisol levels during the laboratory testing day. Times refer to time since beginning the stress procedure. Points represent the mean cortisol level; vertical bars depict standard errors of the means.

home test day a baseline sample was taken, following which samples were taken at 15, 25, 35, and 45 minutes after the subject began to complete the CLSG (see abscissa for Figure 1). On the laboratory testing day a baseline sample was taken, then cortisol samples were taken 20, 30, 40, and 50 minutes after the subject began the stress task (see abscissa for Figure 2). This difference in sample times was because the laboratory stress procedure lasted for 20 minutes so the cortisol sample was taken after completion of the task on that day. Because cortisol has been shown to increase within 15 minutes of the onset of stress, during the home test day the first sample was taken 15 minutes after beginning the stress procedure. The home day cortisol samples taken at 15 minutes and 25 minutes after beginning completion of the CLSG were highly correlated ($r = .84$, $p<.001$) and not significantly different from each other ($t(29) = -.21$, n.s.).

The ANOVA for the cortisol samples obtained during the laboratory test day did not detect any differences between cortisol levels as a function of sample time ($F(4,108) = 1.2$, n.s.).

Table 1 shows the correlations of all cortisol laboratory and home test day samples. As can be seen, cortisol data within and across test days was highly correlated.

Figure 3 shows the mean pattern of blood pressure responses for the laboratory test day. The ANOVA conducted on the SBP means during the laboratory day (laboratory baseline, anticipation, stress, post-stress 2 minutes, and post-stress 5 minutes) detected a significant effect of time $F(4,104) = 71.34$, $p<.001$. Post-hoc tests revealed that each mean was significantly different from the mean before (e.g. baseline and anticipation, anticipation and stress, stress and post stress) except the means for post stress and post-stress 5 minutes which were not significantly different from each other. The direction of

Table 1

Pearson Intercorrelation Matrix for Cortisol Home and Laboratory Test Day Samples.

	Home Day Samples					Laboratory Day Samples				
	1	2	3	4	5	1	2	3	4	5
Home1	--	.75*** (29)	.77*** (29)	.85** (29)	.75*** (29)	.64*** (29)	.70*** (27)	.69*** (27)	.71*** (27)	.53** (27)
Home2		--	.84*** (30)	.83*** (30)	.59*** (30)	.78*** (30)	.58*** (28)	.64*** (28)	.72*** (28)	.36 (28)
Home3			--	.89*** (30)	.67*** (30)	.74*** (30)	.68*** (28)	.75*** (28)	.72*** (28)	.35 (28)
Home4				--	.84*** (30)	.71*** (30)	.65*** (28)	.71*** (28)	.79*** (28)	.59*** (28)
Home5					--	.44* (30)	.55** (28)	.64*** (28)	.71*** (28)	.59*** (28)
Lab1						--	.81*** (28)	.72*** (28)	.74*** (28)	.16 (28)
Lab2							--	.80*** (28)	.73*** (28)	.38* (28)
Lab3								-- (28)	.82*** (28)	.51**
Lab4									-- (28)	.61*** (28)

* $p<.05$
** $p<.01$
*** $p<.001$
n for each cell in parantheses

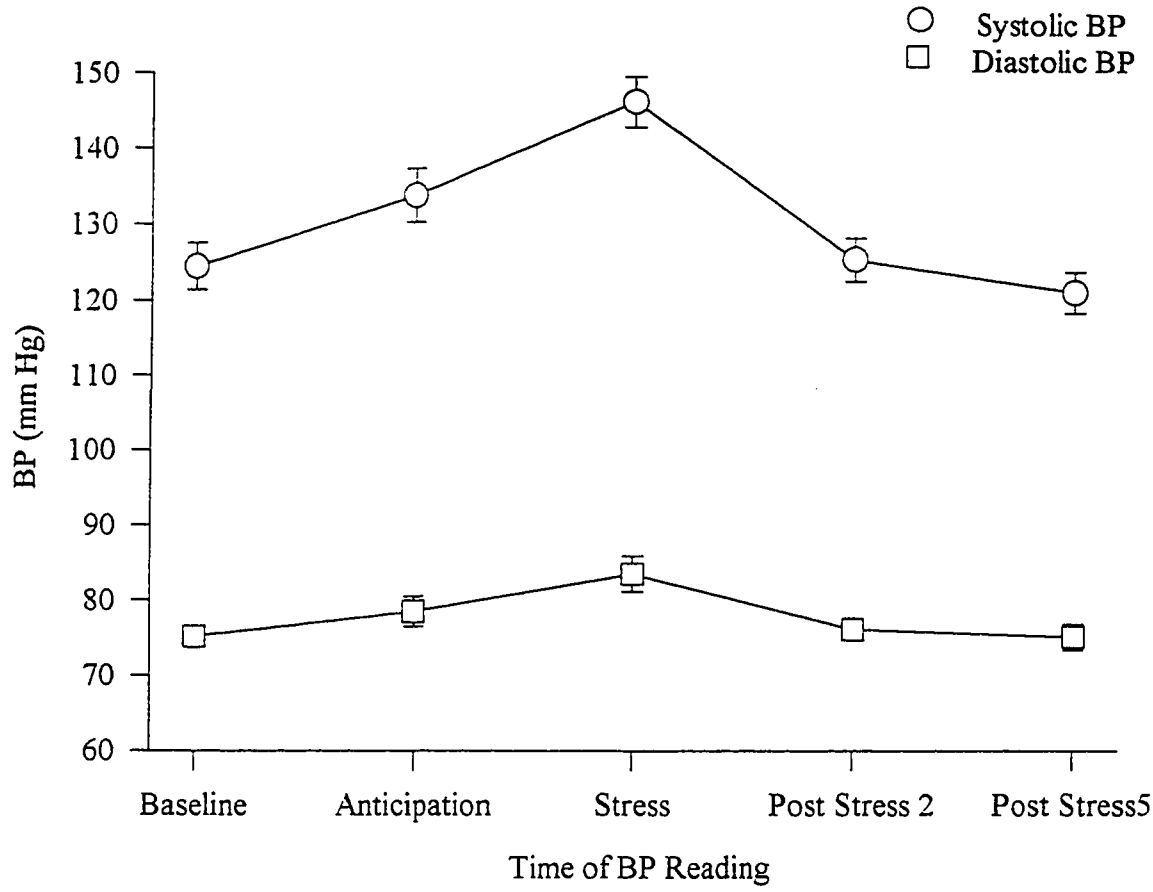

Figure 3. Blood pressure during the laboratory testing day. Points represent the mean blood pressure; vertical bars depict standard errors of the means.

the mean differences in SBP can be seen in Figure 3: SBP increased from baseline to anticipation and anticipation to stress, then decreased from stress to post-stress 2 minutes.

The ANOVA conducted on the mean DBP points during the laboratory test day detected a significant main effect of time ($F(4,104) = 19.27$, $p<.001$). Post-hoc analyses revealed that each mean DBP was significantly different from the mean before (e.g. baseline and anticipation, anticipation and stress, stress and post stress) except for the means for post stress and post stress 5 minutes which were not significantly different from each other. The pattern of these differences can be seen in Figure 3. As with SBP, there was an increase in DBP from baseline to anticipation and from anticipation to stress, then a decrease from stress to post-stress 2 minutes.

Figure 4 shows the laboratory day HR data plotted as mean HR at each phase in the laboratory stress procedure. The ANOVA detected a significant effect of time ($F(4.104) = 28.50$, $p<.001$). Post-hoc analyses revealed that at each phase the mean HR was significantly different from the mean before (e.g. baseline and anticipation, anticipation and stress, stress and post stress) except for the difference between the HR means post stress and post stress 5 minutes. The pattern of these differences can be seen in Figure 4. As in the case of BP, there was an increase in mean HR from baseline to anticipation and anticipation to stress, then a decrease from stress to post-stress 2 minutes.

The home and laboratory test day baselines were compared for each of the physiological variables (SBP, DBP, HR and cortisol level). The SBP home baseline and lab baselines were significantly different from each other ($t(28) = 3.53$, $p<.001$) with the home baseline being higher than the lab baseline ($M=129.701$, $SD=17.80$ and

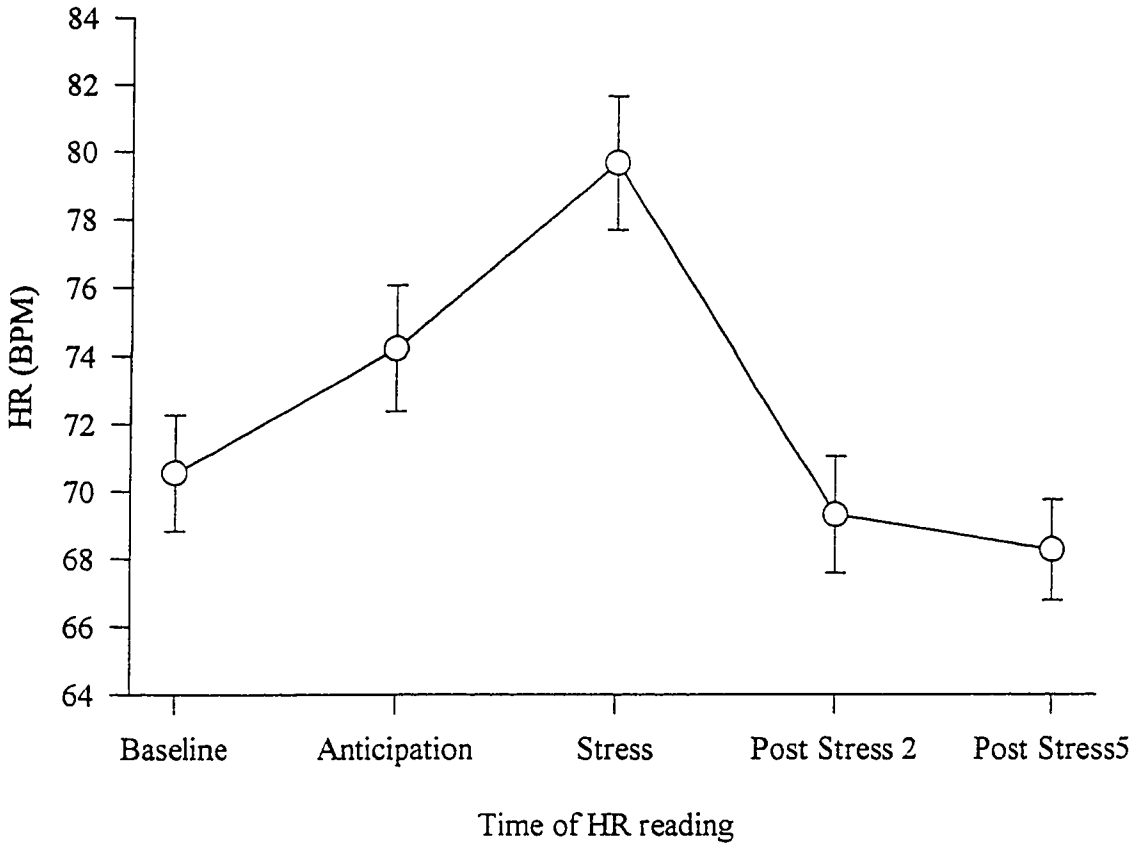

Figure 4. Heart rate during the laboratory testing day. Points represent the mean heart rate; vertical bars depict standard errors of the means.

M=124.604, SD=15.304 respectively), and they were also highly correlated (r=.901, p<.001). The DBP home baseline and lab baselines were also significantly different from each other (t(28) = 2.48, p<.05) with the home baseline being higher than the lab baseline (M = 78.00, SD = 9.856 and M = 75.437, SD = 1.459 respectively). They were also highly correlated (r =.826, p<.001). The HR baselines from home and laboratory test days were significantly correlated (r =.489, p<.01), however, there was no significant difference found between the two means (t(28)=-.81, p>.05).

The home and laboratory cortisol level data were examined in relation to the diurnal record of cortisol data. Subjects had collected between five and 10 samples each for the day, with the average number of samples per subject being eight. Values for the times falling between samples for each subject were obtained using extrapolation, and the resulting mean cortisol level by time of day is shown in Figure 5. The cortisol baselines were compared to the cortisol level of the same time (1 p.m.) from the subject's own cortisol collection day. A repeated measures ANOVA revealed significant differences between the means (F(2,56)=5.8, p<.01), and these differences can be attributed to the day sample (M = .80, SD = .624) being lower than either the laboratory baseline sample (M = 1.029, SD = .407) or the home baseline (M = 1.066, SD = .305). Although the sample from the subject's own cortisol day was not correlated with the home and laboratory baselines, the area under the curve in Figure 5 (representing total cortisol secretion during the day) was found to be significantly correlated with the average of the home and the laboratory baseline cortisol samples (r = .58, p<.001).

Figure 6 shows the subjects' self-reported stress levels by time of day corresponding to the cortisol samples. In order to examine the hypothesis that self-

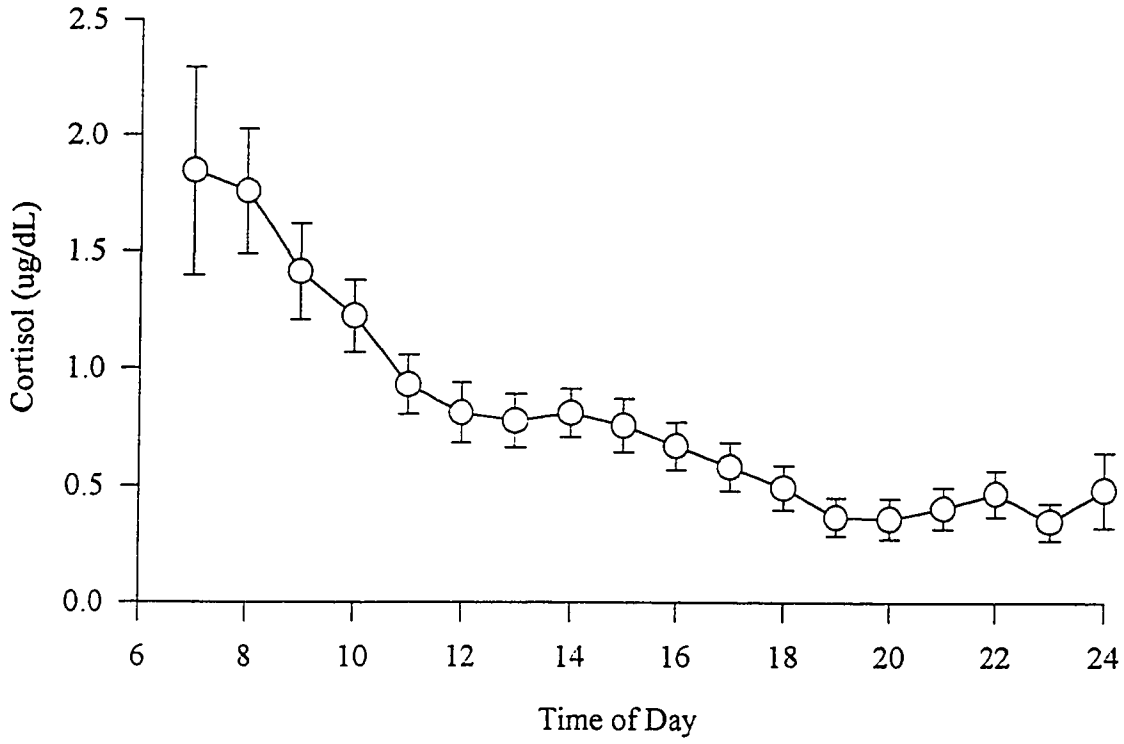

Figure 5. Diurnal rhythm of cortisol throughout a day. Time of day is in hours. Points represent the mean cortisol levels; vertical bars depict standard errors of the means.

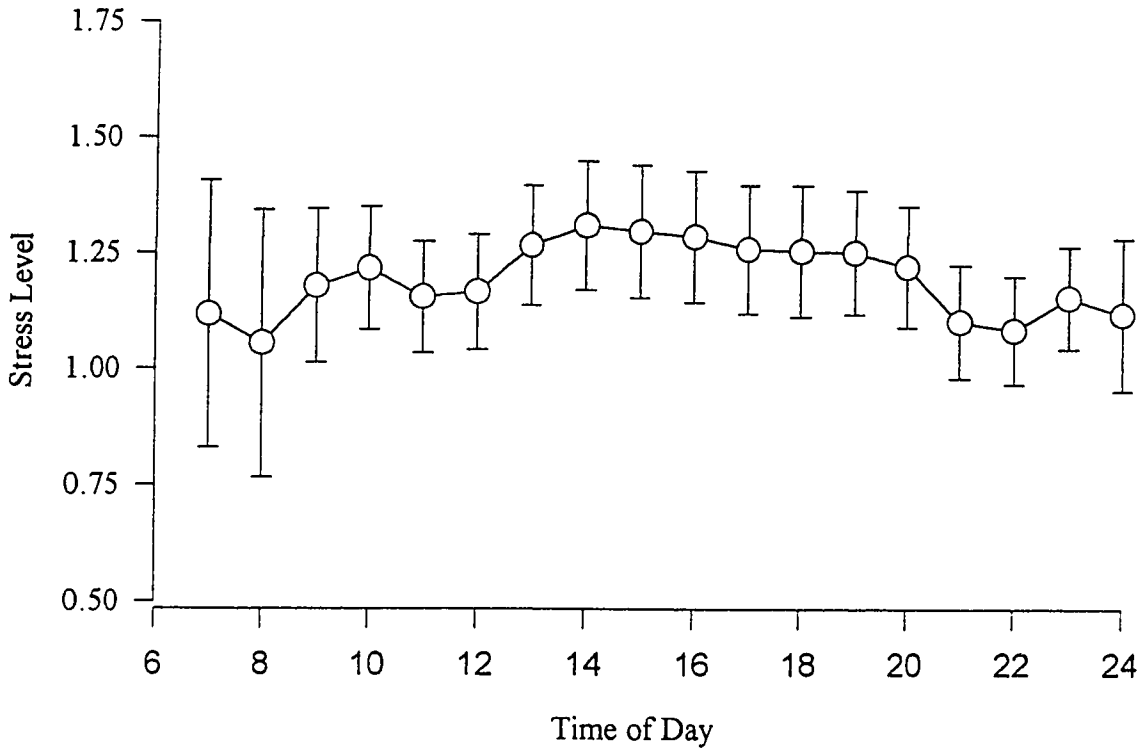

Figure 6. Subject-rated stress points throughout a day. Time of day is in hours. Points represent the mean stress level; vertical bars depict standard errors of the means.

ratings of stress would be related to cortisol levels, correlations between these two sets of variables were examined. For each subject, a score was obtained for total cortisol secretion by measuring the area under the curve for the daily cortisol graph. Similarly, the area under the curve for daily stress was measured for each subject. Mean area under the curve was calculated (i.e. the mean of all subjects' total areas under the curve) for stress and for cortisol. A correlation of these two areas under the curve (stress and cortisol) was found to be not significant ($r = .02$, n.s.). Correlations were also conducted between the means of each stress point and each cortisol point at each time (see Figures 5 and 6), and none of these correlations were significant.

To examine the glucocorticoid hypothesis that more lifetime stress would be correlated with higher baseline and total daily secretion of cortisol, total lifetime stress (measured as area under the curve for the CLSG) was examined in relation to total cortisol release during the diurnal record (area under the curve for cortisol) ($r = .01$, n.s.), and the mean of cortisol baselines for the home and laboratory test days ($r = .23$, n.s.). None of these correlations was significant.

To examine the hypothesis of the allostasis perspective that more lifetime stress would lead to increased anticipatory responses to subsequent stress, a median split was performed on the variable of total lifetime stress (area under the curve on the CLSG). Anticipatory difference scores were obtained for each of the cardiovascular variables (SBP, DBP, and HR) by subtracting the laboratory baseline from the anticipation score for each variable. T-tests were performed between the low lifetime stress and the high lifetime stress groups on each of the anticipatory difference scores. No significant differences were found between the groups on the SBP anticipatory difference score

($t(26) = .16$, n.s.) or the HR anticipatory difference score ($t(26) = .83$, n.s.). The ability of the lifetime stress groups to differentiate DBP anticipatory difference scores approached significance ($t(26) = 1.94$, $p=.055$). The group means for the lifetime stress groups suggest that the participants who experienced less stress over the lifetime were more likely to show a decrease or no change from baseline DBP when anticipating a stressful task ($M = -.0718$, $SD = 3.503$), whereas the participants who experienced more stress over the lifetime were more likely to show a rise in DBP from baseline when anticipating a stressful task ($M = 3.876$, $SD = 3.375$).

The hypothesis of stress in the past year being related to recovery (i.e. return to baseline) of SBP following a stressful task was investigated. A median split was performed on the LES variable of negative life events, which provides a measure of the participant's subjective impact of negative life events that occurred over the past year. To investigate differences in time taken to return to baseline SBP following a stressor as a function of self-reported negative stress in the past year, post-stress task SBP measures at each 2-minute interval were compared by LES group. None of these comparisons were found to be significant. A post-stress difference variable was created by subtracting the average of the 2-minute post-stress SBP measures from the average of the stress SBP measures. The two LES groups were compared on this recovery variable, and the groups were not found to be significantly different ($t(25) = .45$, n.s.).

Correlations between baseline SBP, DBP, HR and cortisol reactivity were conducted to investigate the hypothesis that women with higher baselines would have higher cortisol reactivity. Average baselines were calculated as the mean of the home day and laboratory day baselines for each of the cardiovascular variables. Cortisol reactivity

was measured by subtracting the baseline cortisol level on the laboratory test day from the cortisol level obtained immediately following completion of the laboratory stress task. Table 2 shows a correlation matrix of these variables and others. As can be seen, average baseline SBP and cortisol reactivity were correlated ($r = -.42$, $p<.05$) and the correlation between average baseline DBP and cortisol reactivity approached significance ($r = -36$, $p=.06$). Average baseline HR and cortisol reactivity were not related ($r = -.12$, n.s.).

In addition to a significant correlation between cortisol reactivity and baseline SBP, Table 2 shows that other variables were significantly correlated with cortisol reactivity, specifically, months since menopause ($r = -.39$, $p<.05$), LES ($r = .52$, $p<.01$), and Body Mass Index (BMI) ($r = -.40$, $p<.05$). To determine whether these variables accounted for separate portions of the variance in cortisol reactivity, a direct entry multiple regression was conducted. As can be seen in Table 3, the combination of baseline SBP, months since menopause, BMI, and negative life events accounted for 47% of the variance in cortisol reactivity with baseline SBP and BMI being the only significant predictors of cortisol reactivity.

Table 2

Pearson Intercorrelation Matrix for Selected Variables.

	SBP Base	DBP Base	HR Base	BMI	LES	Hassles	Life Stress	Age	Mths since menopause
Cort React.	-.42* (28)	-.36 (28)	-.12 (27)	-.40* (28)	.52** (28)	.05 (28)	.01 (28)	-.23 (30)	-.39* (28)
SBP Base	--	.65*** (30)	.02 (29)	.04 (30)	-.04 (30)	.23 (30)	-.03 (30)	-.05 (30)	-.06 (30)
DBP Base		--	.36 (29)	.39* (30)	.07 (30)	.32 (30)	-.13 (30)	-.34 (30)	-.23 (30)
HR Base			--	.24 (29)	.24 (29)	.26 (29)	-.02 (29)	-.33 (29)	-.16 (29)
BMI				--	-.25 (30)	.11 (30)	-.25 (30)	.00 (30)	.03 (30)
LES					--	.46** (30)	-.29 (30)	-.48** (30)	-.47** (30)
Hassles						--	-.03 (30)	-.47** (30)	-39* (30)
Life Stress							--	.24 (30)	.28 (30)
Age								--	.70*** (30)

* $p<.05$
** $p<.01$
*** $p<.001$
n for each cell in parentheses

Table 3

Multiple Regression of Predictors of Cortisol Reactivity (N=27).

Predictor	ß	r	sr^2	t	p
Baseline SBP	-.40	-.42	.26	-2.87	<.01
BMI	-.30	-.40	.16	-2.12	<.05
Months Since Menopause	-.25	-.25	.09	-1.52	
Negative Events	.31	.31	.13	1.85	

Note $R = .74$ $R^2 = .55$ Adjusted $R^2 = .47$ $F(4,23) = 7.03$, $p < .001$

Discussion

The present study assessed the relationships between levels of past stress and cardiovascular and HPA axis activity at baseline and during laboratory stress in post-menopausal women. The interaction between the cardiovascular system and the HPA axis was also examined at baseline and during laboratory stress. No relationship was found between levels of past stress and SBP or HR and only a trend towards a relationship existed between past stress and DBP. This trend was towards total lifetime stress being higher in women who had a higher increase in DBP from baseline to anticipation of the stressful laboratory test consisting of an oral defense and a mental arithmetic task. Cortisol reactivity was found to be negatively related to baseline SBP and showed a trend towards being negatively related to baseline DBP. In addition, baseline SBP, life events experienced over the past year, length of time since menopause, and BMI were found to predict 47% of the variance in cortisol reactivity.

The relationship found in the current study between cardiovascular activity and cortisol reactivity was inconsistent with previous studies of this relationship. This relationship has been studied in younger men, and has been shown to be positive. For example, men who have high HR reactivity also have high cortisol reactivity (Lovallo et al., 1990). In addition, borderline hypertensive males show an increase in cortisol levels following a stressful laboratory task, whereas normotensive males do not (Al'Absi et al., 1994). In the current study, however, the relationship between baseline BP and cortisol reactivity was negative. Post-menopausal women with higher BP were found to have less of an increase in cortisol levels following the laboratory stress task than women with lower BP.

The finding that post-menopausal women who have higher BP show less cortisol reactivity to stress must be interpreted with caution because of a methodological flaw in this study. Although the stressful laboratory task was associated with increased BP and HR, there was no significant increase in cortisol associated with the task. For this study, the task was modified from its original version shown to be associated with increased cortisol levels in younger adults (Kirschbaum et al., 1993), and it is probable that the modifications rendered the task too low in stress to affect cortisol levels. Although the possibility that cortisol excretion in post-menopausal women is influenced differently by stress than it is in younger men can not be ruled out, the design of this study does not allow investigation of this possibility. A more accurate replication of the Kirschbaum et al stress task is necessary to examine group differences in salivary cortisol reactivity.

BMI was found to be related to cortisol reactivity. Although some researchers control weight in their studies (e.g. Al'Absi et al., 1994), others do not (e.g. Bohnen, Nicholson, Sulon & Jolles, 1990). Researchers have found a positive relationship between basal cortisol levels and waist to hip circumference ratios (Marin et al, 1992), suggesting that larger people have higher basal cortisol levels. In the current study, no relationship was found between BMI and baseline cortisol levels, and the relationship found between cortisol reactivity and BMI was negative. Larger people were more likely to have lower cortisol reactivity to the laboratory stressor. This finding is tainted by the problem with the cortisol reactivity measure (i.e. the insignificant increase in cortisol as a result of the stress task), but it does suggest that the relationship between BMI and HPA axis activity should be investigated in more detail.

Although there was a significant correlation between length of time since menopause and cortisol reactivity, the correlation was in the different direction from what was expected based on previous research. Lindheim et al (1992). found no differences in cortisol reactivity between pre- and post-menopausal women, but post-menopausal women treated with estrogen showed blunted cortisol reactivity. These results suggest that estrogen decreases cortisol reactivity, whereas in the present study the women who had been without estrogen (i.e. post-menopausal) for longer showed less cortisol reactivity. These discrepant findings may indicate that the relationship between cortisol reactivity and length of time since menopause is associated with something other than estrogen such as dehydroepiandrosterone (DHEA) or androgen levels (Haffner, Newcomb, Marcus, Klein & Klein, 1995). The methodological problem of the lack of increased cortisol excretion from baseline to stress in this study results in a problem with the cortisol reactivity measure, so any conclusions based on this measure are tentative. In addition, length of time since menopause does not account for any unique variance in the multiple regression, suggesting that the correlation is dependent on a third variable. This variable is not age, because age was not related to the cortisol measure. The present study does not allow us to investigate what the length of time since menopause variable may be measuring, but it does suggest the need for more research into the interactions between the physiological changes associated with menopause.

The allostatic model of past stressors affecting current reactions to stress received only minimal support in this study, as did the glucocorticoid cascade hypothesis of repeated exposure to stress resulting in increased basal cortisol levels. The only support found was that self-reported stress levels in the past year were positively related to

cortisol reactivity to the laboratory stress test. This finding may be contaminated by the methodological problem affecting the stress reactivity measure. A trend was also found towards women reporting higher levels of lifetime stress having more of an increase in DBP over baseline when anticipating the stressful task. These findings do not alter the fact that most of the results do not support any influence of past stress levels on current physiological functioning. A possible confound stems from using subjective measures of stress, such as the LES and the CLSG, and comparing these to objective physiological measures, such as BP, HR, and cortisol levels. Whereas objective measures can be placed on a continuum across individuals, subjective measures can not (Raphael, Cloitre & Dohrenwend, 1991). In all, the findings of this study suggest that current physiological functioning is not affected by past stress levels.

Baseline cortisol measures taken during the home and laboratory test days were found to be significantly higher than the cortisol level obtained on the subject's cortisol collection day. This suggests that baseline measures of cortisol obtained in an experimental situation may not be representative of true basal cortisol excretion. This increase in cortisol levels from the subject's own sample collection to the samples taken with the experimenter may reflect some form of anticipation. Participants may have increased cortisol activity because of their expectations of participation in the research. The stress task by Kirschbaum et al. (1993) is comprised of an anticipatory period as well as the stress tasks, and the results of the current study suggest that the baseline cortisol level obtained in the laboratory is already tainted with an increase in cortisol release due to anticipation. Perhaps future studies involving cortisol levels should use a true baseline such as one collected by a subject on a typical day.

The hypothesis that cortisol levels and self-rated stress at regular intervals throughout a typical day in the participant's life would be correlated was not supported. Previous research has shown a correlation between cortisol release and ratings of stress (Brantley et al., 1988; van Eck et al & Nicholson, 1988). Those studies differ from the current one because they examined stress ratings on different days and found more cortisol release on the highest stress day than on the lowest stress day. It is possible that the relationship between stress and cortisol is not strong enough to be detected at different points during the same day.

In addition to the problem of the stress task not being arousing enough, there are at least two other possible problems in interpreting the data obtained from this study. The first is the sample size. A large amount of variability was found on many of the physiological measures, suggesting that the sample was not homogeneous. This heterogeneity was especially evident in cortisol samples, suggesting that other factors influence cortisol levels in post-menopausal women. A larger sample size is necessary both to reduce the variability, and to examine some of the reasons for it. Past research has identified a host of variables associated with differences in cortisol excretion, including such factors as coping style (Bohnen et al., 1990) and personal well-being (Brandtstädter, Baltes-Götz, Kirschbaum & Hellhammer, 1991). These factors and others should be examined in post-menopausal women in an attempt to identify the sources of the variability in cortisol excretion.

Another possible problem in interpretation and generalizability of this data involves the lifestyles of the participants. Most of the women in this sample were retired, and many of them reported during the interview that their daily routine is relatively free

of stress. In addition, a large number of the participants had a score of zero on the LES, suggesting they had no major stress over the past year. Perhaps a more complete picture of the effects of levels of past stress on current HPA axis and cardiovascular system functioning in post-menopausal women would be obtained if the sample contained some women who were currently experiencing stressful life events, such as recent death of a husband.

The goals of this study were to examine how the existing literature of HPA-axis and cardiovascular functioning applies to post-menopausal women, and how past levels of stress relate to current physiological functioning. Although methodological problems make the specific research hypotheses difficult to confirm, overall the results suggest that the experience of past stress is not related to current physiological responses to stress. In addition, the relationship between cardiovascular system activity and HPA axis reactivity may be affected by time since menopause and BMI in older women. These conclusions are tentative owing to the problems in this study, and more research is needed to elucidate these issues in post-menopausal women.

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Appendix A

Life History Interview

INTERVIEW

ID: _____

Date of interview _____

Gender: F( ) M( ) Tester: _____

Language of interview: French ( ) English ( )

What is your first language? _____

What language did you attend school in? _____

If first language other than English/French, at what age did you first start speaking English/French? _____

What is your birth date? _____ Present age: _____

Were you born in Canada? Yes ( ) No ( )

If not, where were you born? _____

Where were your parents born? _____

Are your parents living?

If YES, how old are they? Mother _____
Father _____

If NO, how old were they when they died? Mother _____
Father _____

Present marital status:

(1) single-never married _____ (2) married _____
(3) separated _____ (4) divorced _____
(5) widowed _____ (6) common-law _____
(7) cohabitating (<1 year) _____ (8) "divorced" from common-law/cohab. _____

CURRENTLY MARRIED, DIVORCED, ETC...:

In what year were you married, divorced, etc.? _____

MARRIED, COMMON-LAW OR COHABITATING, DIVORCED, WIDOWED OR SEPARATED:

You've told me that you are currently _____. Is this your only marriage/significant relationship or have you been married/involved before? Yes ( ) No ( ) How many times? _____
(excluding the current one)

(Although it states "relationship", we are referring to having lived with someone before.)

****

Children:

Do you have any children? Yes ( ) No ( )
If YES:
How many sons? _____ How many daughters? _____

(**Note:** Write S in brackets if subject's child, or P if partner's.)

What is/ are the birth date(s) of your sons? _____ _____

_____ _____ _____ _____

(Indicate where the children live)

What is/are the birth date(s) of your daughters? _____ _____

_____ _____ _____ _____

(Indicate where the children live)

Education:

How many years of education do you have (i.e., what was the highest level achieved)? _____

Based on answer, circle highest grade attained:
1 2 3 4 5 6 7 8 9 10 11 12 13 14 15 16 17 18 19 20 21 22 23 24 25
Elementary Secondary CEGEP—1er cycle Graduate--Professional

At present, do you work for pay outside the home? Yes ( ) No ( )
If No,
Have you ever worked for pay outside the home? Yes ( ) No ( )

What do (or did) you do? _____

What is (or was) your spouse's occupation? Please give me as exact a description as possible. _____

****

How financially comfortable would you say you are currently?
___ (1) Very comfortable
___ (2) Comfortable
___ (3) Neither comfortable nor uncomfortable
___ (4) Uncomfortable
___ (5) Very uncomfortable

How financially comfortable would you say you are compared to other people you know of the same age?
___ (1) Much worse off than most
___ (2) Worse off than most
___ (3) Slightly worse off than most
___ (4) About the same as most
___ (5) Slightly better off than most
___ (6) Better off than most
___ (7) Much better off than most

If you don't mind telling me, what was your household's total income from all sources before taxes and deductions for this past year?

____ (1) Less than $10,000
____ (2) $10,000 - $29,999
____ (3) $30,000 - $49,999
____ (4) $50,000 - $74,999
____ (5) $75,000 - $99,999
____ (6) Over $100,000

How many people contributed to this income? _____

On average how many social functions do you attend per month (parties, get-togethers with friends or relatives, social club meetings, etc.)?

____ (1) None
____ (2) Less than once a month
____ (3) About one a month
____ (4) Two or three a month
____ (5) About one a week
____ (6) Two or more a week

How important is your social life to you? 1 2 3 4 5 6 7
Not at all Somewhat Extremely

How often do you attend religious services?

____ (1) Never
____ (2) Less than once a month
____ (3) About once a month
____ (4) Two or three times a month
____ (5) Once a week
____ (6) More than once a week

How religious are you? 1 2 3 4 5 6 7
Not at all Somewhat Devoutly (very much so)

During the past 12 months, would you describe your life as:

____ (1) Very stressful
____ (2) Fairly stressful
____ (3) Not very stressful
____ (4) Not at all stressful

How many cigarettes on average do you smoke per day? _____

How many drinks (beer, wine, or liquor) on average do you have per week? _____ drinks

Overall, how happy was your childhood?

1 2 3 4 5 6 7
extremely happy somewhat happy not happy at all

In your estimation, how happy were your parents together when you were a child?

1 2 3 4 5 6 7
extremely happy somewhat happy not happy at all

Overall, how happy has your adult life been?

1 2 3 4 5 6 7
extremely happy somewhat happy not happy at all

Appendix B

Health Questionnaire

Code # _____

HEALTH QUESTIONNAIRE

This questionnaire deals with your health. First we are going to ask you some general questions.

How would you rate your overall health at the present time?
Very good ☐ Fair ☐ Poor ☐

Compared to 5 years ago, is your health:
Better ☐ About the same ☐ Worse ☐

How much do your health problems stand in the way of your doing the things you want to do?
Not at all ☐ A little ☐ A great deal ☐

On average, how often do you visit a doctor?
Once a week or more ☐
Once every 2 weeks ☐
Once a month ☐
Between 2 and 12 times a year ☐
Once a year ☐
Less than once a year ☐

How many times in your life have you been hospitalized? _____

How many of these were pregnancy related? _____

How many times have you undergone surgery? _____

When do you think your menopause started? _____

When did you last menstruate? _____

Have you undergone surgical menopause? Yes ☐ No ☐
If so, when? _____

Are you currently receiving hormone replacement therapy? Yes ☐ No ☐
If so, which form of hormone replacement therapy are you taking and how long have you been on it? _____

_____

Please indicate which of the following conditions you currently have or have suffered from in the past and the times when you suffered from them:

	From - To (Mth/Yr)	
Headache	_____	
Migraine	_____	
Dizziness	_____	
Varicose veins	_____	
Hemorrhoids	_____	
Drug allergy	_____	Which drug(s): _____
Bronchitis	_____	
Bursitis	_____	
Heart condition	_____	
Chest pains	_____	
Overweight	_____	
Anemia	_____	
Emotional problems	_____	Specify: _____
Kidney infection	_____	
Asthma	_____	
Arthritis	_____	
Ulcers	_____	Specify: _____
High blood pressure	_____	
Diabetes	_____	
Emphysema	_____	

Do you or have you suffered from any other conditions in the past? If so, please explain which condition and when you had it. _____

_____

_____

Do you have high cholesterol? Yes ☐ No ☐

If so, are you currently taking medication to lower your cholesterol? Yes ☐ No ☐

Does anyone in your family suffer from high cholesterol? If so, please indicate the person's relationship to you, and their age. _____

_____

Does anyone in your family suffer from high blood pressure? If so, please indicate the person's relationship to you, and their age. _____

_____

In the past 30 days, have you taken any of the following medications?

		If so, was this with a doctor's prescription?
Aspirin or similar pain reliever	Yes ☐ No ☐	Yes ☐ No ☐
Tranquilizers (i.e. valium)	Yes ☐ No ☐	Yes ☐ No ☐
Stimulants	Yes ☐ No ☐	Yes ☐ No ☐
Diet pills	Yes ☐ No ☐	Yes ☐ No ☐
Anti-depressants	Yes ☐ No ☐	Yes ☐ No ☐
Codeine, demerol or morphine	Yes ☐ No ☐	Yes ☐ No ☐
Allergy medication	Yes ☐ No ☐	Yes ☐ No ☐
Cough or cold remedies	Yes ☐ No ☐	Yes ☐ No ☐
Antibiotics (i.e. penicillin)	Yes ☐ No ☐	Yes ☐ No ☐
Heart medication	Yes ☐ No ☐	Yes ☐ No ☐
Blood pressure medication	Yes ☐ No ☐	Yes ☐ No ☐
Insulin or other diabetic medication	Yes ☐ No ☐	Yes ☐ No ☐
Sleeping pills	Yes ☐ No ☐	Yes ☐ No ☐
Stomach remedies	Yes ☐ No ☐	Yes ☐ No ☐
Laxatives	Yes ☐ No ☐	Yes ☐ No ☐
Arthritis medication	Yes ☐ No ☐	Yes ☐ No ☐
Cortisone	Yes ☐ No ☐	Yes ☐ No ☐
Thyroid pills	Yes ☐ No ☐	Yes ☐ No ☐
Seizure pills	Yes ☐ No ☐	Yes ☐ No ☐
Blood thinner pills	Yes ☐ No ☐	Yes ☐ No ☐
Pills for breathing	Yes ☐ No ☐	Yes ☐ No ☐
Circulation pills	Yes ☐ No ☐	Yes ☐ No ☐
Other (list):		

_____

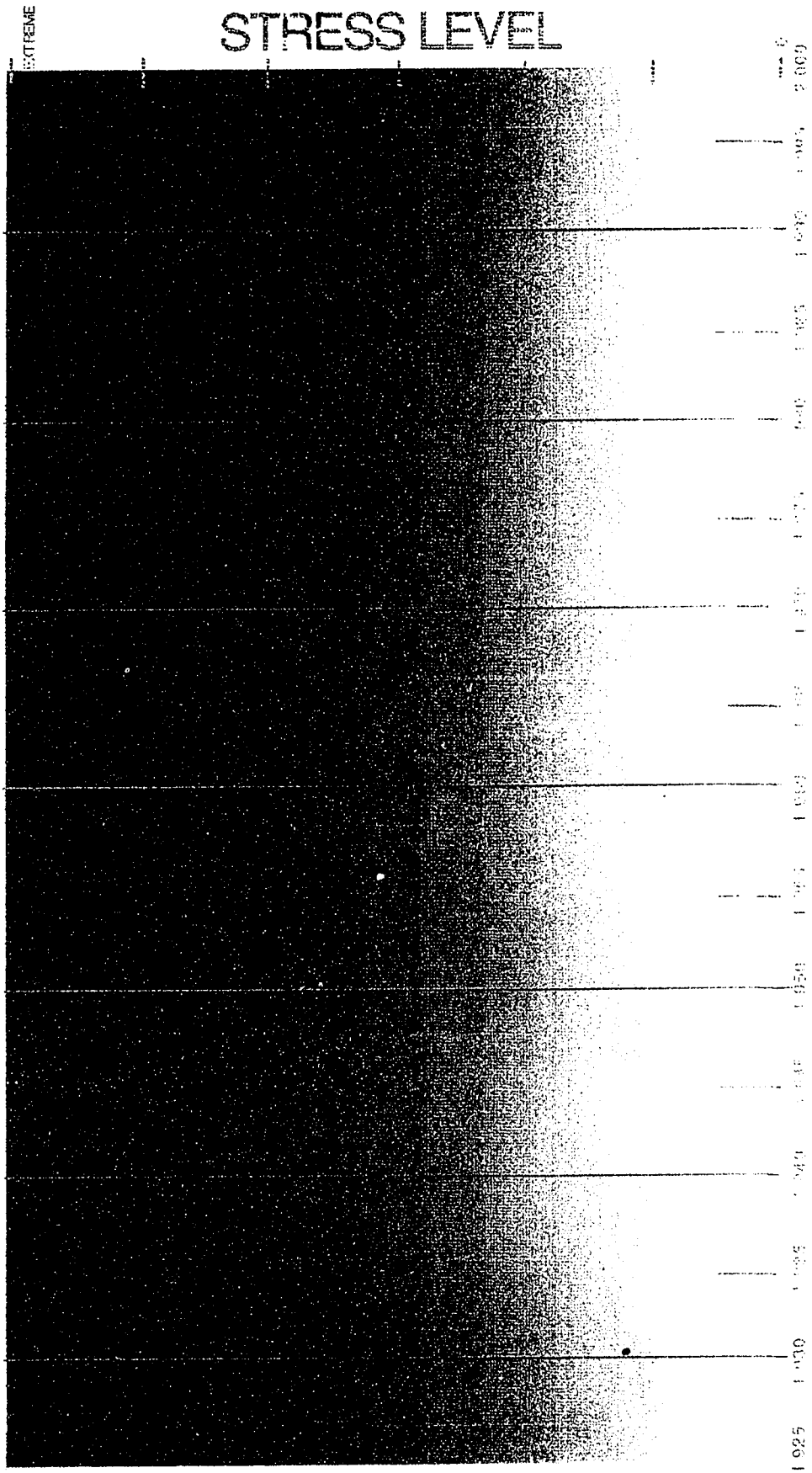
_____

_____

_____

Appendix C

Concordia Lifetime Stress Graph


STRESS LEVEL
EXTREME
1925

Appendix D

In Home Testing Day Procedure

Subject # _____

IN HOME PROCEDURE

- Consent form
- Subject sits for 10 min. with BP cuff on arm, then 5 baselines (1 per min.)
- Cortisol sample (1) time _____
- Interview
- Health
- Hassles
- LES
- CLSG: BP every 3 min.
  Cortisol 15 min. after start, then 3 more at 10 min. intervals.
  Time (2) _____
  Time (3) _____
  Time (4) _____
  Time (5) _____
- Coping
- SSQ
- Habitual Activities
- Buss-Durkee
- Eysenck
- CES-D

- Explain daily cortisol
- Schedule laboratory apt

Appendix E

Consent Form

Code no. _____

CONSENT FORM TO PARTICIPATE IN RESEARCH
Department of Psychology, Concordia University

Title of Research: Impact of lifetime experiences on cardiovascular function and the HPA axis in elderly women.

I consent to participate in the first part of the study on the influence of lifetime events on physiological responses to stress which is under the supervision of Drs. Arbuckle, Chaikelson, and Miller.

1. With respect to the study itself, I understand that there are 3 phases: a home session, a salivary cortisol measurement day, and a laboratory session.

The home session will last approximately 3 hours and involve:

- Answering a number of questionnaires including health, personality, coping, and social support measures.
- Measuring various cardiovascular responses (e.g. heart rate and blood pressure). These recordings will be painless, safe, and non-invasive (no needles are involved), requiring only the placement of a blood pressure cuff around the arm. Saliva levels of cortisol will also be measured, meaning that a small filter will be placed in the mouth until saturated with saliva. During the completion of some questionnaires, temporary changes such as increases in blood pressure, heart rate and cortisol secretion may occur. These will return to normal after the end of the visit causing no adverse effects.

The salivary cortisol measurement day involves:

- On one day of my choice over the next week I will measure my salivary cortisol level upon awakening and every 2 hours thereafter until I go to sleep. I will do this by placing a small filter in my mouth until it is saturated with saliva. I will place each filter in the corresponding bag and keep these samples in my freezer until I go to the laboratory. When I go to the laboratory, I will bring the samples with me. All of the filters and the bags will be supplied by the experimenter.

The laboratory session will last approximately 3 hours and involve:

- Filling out alcohol, smoking, and exercise questionnaires
- A testing session during which I will engage in two tasks which will be videotaped:
  (1) A five minute mental arithmetic task involving subtraction.
  (2) A five minute speech task where I must verbally defend myself.
  I will be given ten minutes to prepare this speech.
- As in the home session, blood pressure, heart rate, and cortisol will be measured.
- Completing some tasks of memory and vocabulary knowledge.

2. With respect to my participation in the study, I understand that:

- Any information learned about me or anyone else through my responses to the questionnaire will be kept confidential.
- I will receive a full explanation of the findings when they become available.
- I understand that participation in the experiment is voluntary and that if I refuse to participate, it will not prejudice in any way my potential participation in other experiments in the Department of Psychology and that I am free to discontinue my participation at any time.
- I understand that I may ask any questions about the experiment prior to signing this consent form and can expect a satisfactory explanation.
- I will receive a $40 honorarium after completion of the study as a thank you for my participation in this experiment.
- A medical assessment of cardiovascular function is not provided, but a description of my responses will be given.

I have carefully studied and understood this agreement, and therefore I freely consent and agree to participate in this experiment.

_____	_____
Signature	Experimenter's Signature
_____	_____
Print your name	Date

Appendix F

Instructions for Day Cortisol Samples

Thank you again for your participation in this experiment examining how lifetime events affect physiology. In this part of the experiment, we want to look at your cortisol levels every couple of hours for a complete day. The day you choose to do this is up to you, as long as it is one day before you come into the laboratory.

In order for us to look at your daily cortisol rhythm, we need you to take a saliva sample when you first wake up, and every 2 hours thereafter, taking a final saliva sample right before you go to sleep. You will remember that to get a saliva sample, you place a supplied filter in your mouth up to the cut mark, and leave it for a few seconds until it is saturated with saliva. You may find it useful to imagine yourself biting into a lemon. You will then place the filter in one of the small plastic bags and put it in a freezer. If you can't get to a freezer right away just put it in when you can, it doesn't have to be immediately. Remember to put each sample in a separate bag, and to use them in order according to the number written on each filter.

When you take each saliva sample, please write on the attached sheet the time of day, the place you are in, and if you have smoked, eaten or drank anything in the past hour. Please try to wait at least 10 minutes after eating, drinking or smoking before taking a saliva sample. We would also appreciate you rating your stress level using the piece of red paper provided. As in the graph we made of your stress levels throughout your life, the deeper red means higher stress, and the paler red mean low stress. Along the bottom are numbers reflecting the sample number. We ask you to draw a point on the graph according to how stressed you feel each time you take a sample, so every 2 hours. This will enable us to create a mini-graph of your daily stress.

On the day that you come into the laboratory, please bring your samples with you. Place them in the lunch bag we gave you with the ice pack. Don't forget to also bring the attached sheet of paper too, and the mini-graph of your daily stress levels.

As in every part of the experiment, all of the information we obtain from the cortisol samples and from the attached sheet will be kept confidential.

Appendix G

In Lab Testing Day Procedure

Subject # _____

LAB PROCEDURE

- Subject sits for 10 min. with BP cuff on arm, then 5 baselines (1 per min.)
- Cortisol sample (1) time _____

Has anything major happened since I was at your home? Any major life change or stressful event? Any minor event but which was out of the ordinary or caused you a lot of stress?

_____

Stress tasks:

- give instructions, 10 min. alone, BP every 2 min.
- accomplice enters room, start stress task, BP every 2 min.

- Cortisol sample (2) after stress tasks. Time _____
- BP every 2 min. for 10 mins., then reset BP for every 5 mins.
- Put neutral music on, tell subject to read books, explain will enter every 10 min. for saliva sample.
- Take cortisol 10 min. from last sample, then 2 more at 10 min. intervals.
  Time (3) _____
  Time (4) _____
  Time (5) _____

- Digit-digit, digit-symbol
- Continuous Attention
- Emerging Complex Figure: Time finished:_____
  Recall time (add 40 min): _____
- Picture Completion
- Vocabulary
- Hebb repeated digit string
- Serial-order pointing
- Smoking graph
- Alcohol graph
- Daily activities, part 2

- Height: _____ Weight: _____

- Explain not really videotaped, just to increase stress level.
- Pay subject, sign payment form.

Appendix H

Laboratory Stress Task – Subject Instructions

Subject ID: _____

We would like you to imagine that you have been accused of shoplifting. You were in a store, and a salesperson says she saw you take an item and put it in your purse. Indeed, the item is in your purse, but that is because you bought it previously at another store. Unfortunately, you do not have the receipt.

Please take the next 10 minutes to plan your verbal defense to the accusation of shoplifting. You will be required to defend yourself in front of a woman. You will have 5 minutes to convince her of your innocence. You must talk during the entire 5 minutes, and you may be asked some questions by her. Your defense will be videotaped for later viewing.

Feel free to write down notes to yourself on this sheet. You will not, however, be able to keep this sheet with you while you defend yourself.

Appendix I

Laboratory Stress Task – Experimenter Instructions

Subject ID: _____

Questions:
(write the subject's answers)

1. Please explain again how this item came to be in your purse.

2. Where did you buy the item?

3. Why do you not have the receipt?

4. Were you alone or with someone else?

5. Did anyone else witness your purchase?

6. Why do you think the salesperson says you stole the item?

If time is still remaining, start from question 1 and ask them all in order again.

Number of last question asked: _____
Number of times the list of questions was asked: _____

When 5 min. is up:

Thank you. We now require you to count backwards from 1022 by 13. So you will say 1022, 1009, 996, 983, 970, and so on. Do you understand?
You must do this for 5 minutes. Whenever you make a mistake we will ask you to start over from 1022.
Go ahead now please.

1022 - 1009 - 996 - 983 - 970 - 957 - 944 - 931 - 928 - 915 - 902 - 889 - 876 - 863 - 850 - 837 - 824 - 811 - 798 - 785 - 772 - 759 - 746 - 733 - 720 - 707 - 694 - 681 - 668 - 655 - 642 - 629 - 616 - 603 - 590 - 577 - 564 - 551 - 538 - 525 - 512 - 499
....

Write down responses: