

**Autonomic Characteristics of Defensive Hostility:
Reactivity and Recovery to Active and Passive Stressors**

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(ABSTRACT)

Defensive hostility has been attributed as an early risk factor of coronary heart disease. The autonomic characteristics of high defensive, high hostile (HD) and low defensive, high hostile (LD) men and women were assessed with a variety of cardiovascular (CV) measures. Reactivity and recovery to an active laboratory stressor (video game, VG) and a passive laboratory stressor (hand cold pressor, CP) of 15 HD men, 16 LD men, 16 HD women, and 16 LD women were recorded. It was predicted that the CV patterning associated with the HD participants would display more sympathetic and less vagal control as well as the least pronounced recovery from the stressors in comparison to LD participants. Results revealed differential CV responses to the lab tasks by group. HD women displayed consistently high levels of low frequency power heart rate variability (HRV) during baseline and across conditions. HD men exhibited significantly pronounced heart rate reactivity and reduced high frequency power HRV to the CP task in comparison to LD men. Interestingly, LD women displayed weaker blood pressure (BP) recovery to the VG in comparison to HD women, whereas the opposite pattern was observed in BP recovery to the CP. These results suggest that defensiveness and sex may moderate the CV reactivity and recovery to different types of stressors in hostile participants.

DEDICATION

This thesis is dedicated most lovingly to my family, without whose encouragement, love, and support this project would not be possible.

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INTRODUCTION

A substantial amount of research has attributed hostile tendencies as an early risk factor of coronary heart disease (CHD) (see Miller, Smith, Turner, Guijaro, & Hallet, 1996 for a review). Hostility has been described as an enduring and general personality trait that involves a devaluation of the worth and motives of others, an expectation that others are likely sources of wrongdoing, a relational view of being opposed to others, and a desire to inflict harm or see others harmed (Smith, 1994).

Hostile people are prone to cynical attitudes and a mistrust of others, which may give rise to the frequent experience of anger and a variety of associated behaviors. Situations requiring anger inhibition may be more prevalent in the daily life experiences of hostile individuals than encounters permitting anger expression. Some evidence suggests that hostile persons who inhibit their anger expression are more likely to develop significant coronary atherosclerosis than hostile individuals who express their anger (Dembroski, MacDougal, Williams, Haney, & Blumenthal, 1985).

The construct known as *defensiveness* has been associated with the inhibition of anger expression through the ongoing avoidance of the recognition, reporting, and behavioral display of angry feelings to maintain social desirability (Crowne & Marlowe, 1964; Weinberger, 1990; Jamner, Shapiro, Goldstein, & Hug, 1991). Defensiveness, in combination with cynical hostility, creates an 'approach-avoidance' conflict characterized by a desire to obtain social approval while distrusting those capable of providing such support. This chronic state of social conflict may be reflected in a cardiovascular (CV) patterning that ultimately contributes to the development of CHD (Jorgensen, Abdul-Karim, Kahan, & Frankowski, 1995; Helmers & Krantz, 1996).

In light of research indicating that when hostility is associated with anger inhibition the risk of coronary dysfunction is increased, the focus of the present study is on the autonomic nervous system (ANS) patterns associated with this tendency. Individuals rating high in both hostility and defensiveness may be considered as high emotional inhibitors, suppressing the expression of angry feelings to maintain social desirability. High defensive, high hostile (HD) individuals may be more reactive to lab

stressors than individuals from any other combination of these two traits (Larson & Langer, 1997). It was hypothesized that HD individuals ruminate while inhibiting their anger expression and that the rumination process may be instrumental in prolonging the CV activity associated with the experience of anger.

A discussion of the ANS as a medium to transfer psychological variables to end organ output will provide a foundation to elucidate the relationship between defensive hostility and CHD. Hostility may be considered a chronic tendency to perceive daily life experiences as threatening and to respond with aggression. The mental state of conjuring up threatening images may activate aggressive responses that have a detrimental impact on CV health status. The following issues will be addressed to form a theoretical rationale for the present study: the various models used to explain the hostility/CHD relationship, the ways in which the hostility construct has been measured, and the potential mediating variables cogent to this literature.

Autonomic Activity, Hostility, and CHD

The ANS, a branch of the peripheral nervous system, is bifurcated into sympathetic and parasympathetic branches. Heart rate and rhythm are controlled by the ANS despite the well established automaticity of the various intrinsic pacemaker tissues (Randall & Ardell, 1990). Generally, parasympathetic (cholinergic) influences on the heart reduce the force of contraction and slow heart rate (HR), whereas the sympathetic influences (adrenergic) have opposite effects. Complex interactions between tonic sympathetic and parasympathetic activity continuously modulate cardiac performance (Urather, Neely, & Hageman, 1986).

The tenth cranial nerve, known as the vagus, emits acetylcholine onto the sinoatrial node of the heart by the neural firing of the medullary nucleus ambiguus (Porges, 1995); the net effect of this vagus nerve activity is a reduction in HR. Beat to beat variations in HR are influenced most heavily by vagal activity, because the heart responds more gradually to input from the sympathetic nervous system (Saul, 1990). Therefore, enhanced short-term HRV generally reflects greater cardiac vagal control, essential to self-regulation and ability to effectively cope with environmental demands

(Porges, 1992). Individuals who exhibit enhanced resting HRV may be described as highly responsive and sensitive to changing environmental demands (Goldberger, 1991), or demonstrating autonomic flexibility (Friedman & Thayer, 1998a).

Spectral analysis of the electrocardiogram (ECG) has been used to designate the intrinsic sources of variability in the HR time series (e.g., Stein et al., 1994). Heart rate fluctuations occur at different frequencies, allowing for differential mapping of the components of the power spectra on to the autonomic indices. The high frequency (HF) (0.15-0.40 Hz) component corresponds to respiration rate, whereas the low frequency (LF) (0.04-0.14 Hz) component primarily reflects baroreceptor-mediated regulation of blood pressure (BP), referred to as Mayer waves that correspond to approximately 6 breaths per minute (Saul, 1990). The autonomic branches mediate fluctuations of HR at different frequency bands (Askelrod et. al., 1981). Fluctuations at frequencies > 0.15 Hz reflect changes mediated by vagal activity, whereas fluctuations at frequencies < 0.15 Hz reflect changes mediated by cardiac vagal and sympathetic activity.

The respiratory component of HRV corresponding to frequencies > 0.15 Hz are relatively fast acting and have been established to reflect HF spectral power as an index of cardiac vagal tone (Malliani, Pagani, Lombardi, & Cerutti, 1991). However, the autonomic underpinnings of the slower, LF component are more controversial. Baroreceptor regulation of BP can be achieved through sympathetic or parasympathetic influence (Friedman & Thayer, 1998b). However, a number of studies suggest that low frequency power (LFP) is a fair estimator of cardiac sympathetic influences (Friedman, Thayer, & Tyrell, 1996; Kamada et al., 1992; Bronis, 1983).

Vagal withdrawal is an indicator of stress on the CV system (Porges, 1991; 1995). An increase in cardiac vagal tone, or the effect of vagus nerve activity on the heart, has been associated with a reduction in HR, rapid CV recovery after exposure to aversive stimuli, and an increase in HRV (Saul, 1990; Porges, 1992). Vagal tone is suppressed during stressful states and this suppression has been associated with slow CV recovery from chronic stressors that may play a key role in the development of CHD (Broschott & Thayer, 1998).

HRV provides psychophysiologicalists with a contextually sensitive index of autonomic activity. Studies utilizing HRV as an indicator of autonomic activity have consistently demonstrated a strong negative correlation with CV dysfunction. Therefore, extreme HR stability is generally an indicator of pathology (Pincus et al., 1991; Latson, 1994; Peng et al., 1994), whereas HRV is associated with healthy CV functioning (Friedman & Thayer, 1998b). A strong association has been determined between reduced vagally mediated HRV and CHD mortality in middle-aged and elderly men (Stein & Kleiger, 1999). Healthy individuals exhibit resting HR activity in both low (mixed sympathetic and vagal influences) and high (predominantly vagal influence) frequency physiological oscillations, whereas individuals with severe congestive heart failure exhibit resting HR activity in only very low frequency (<0.04 Hz) oscillations (Saul, 1990), reflecting consistently high sympathetic influence with depressed parasympathetic activity.

Decreased HRV is associated with lower post-MI survival (Bigger, Fleiss, Rolnitsky, & Steinman, 1993) and has been shown to predict the development of CAD (Liao et al., 1997) as well as cardiac events (Tsuji et al., 1996). In a study involving 808 patients who survived acute myocardial infarction (AMI), patients exhibiting low HRV approximately 2 weeks post AMI experienced a relative mortality risk 5.3 times greater at a 3 year follow-up than patients with high post AMI HRV (Kleiger, Miller, Bigger, & Moss, 1987). The authors hypothesized that the association between diminished vagal tone and HRV may predispose patients to ventricular fibrillation.

Reduced HRV has been reported in a variety of psychopathologic conditions. Individuals who experienced frequent bouts of panic anxiety have demonstrated the highest HR and lowest HRV in comparison to non-anxious controls for various laboratory stressors (Friedman & Thayer, 1998a, b). Decreased HRV has also been documented in post-traumatic stress disorder (Klein, Cnaai, Harel, Braun, & Ben-Haim, 1995; Cohen et al., 2000) and worry associated with generalized anxiety disorder (Thayer, Friedman, & Borkovec, 1996). These studies substantiate HRV as an

informative measure for a variety of psychological conditions related to anxiety disorders, as well as stressful states in general (Friedman, Thayer, & Tyrell, 1996).

High frequency heart period variability (HF-HPV), an index of cardiac vagal modulation, has been demonstrated as inversely related to hostility in a healthy sample, but only during waking hours (Sloan et al., 1994). This finding supports the transactional theory of disordered autonomic regulation, characterized by the idea that the cynical attitudes held by hostile individuals prompts them to create and maintain negative interactions with others (Smith & Christensen, 1992,a), because hostile individuals can only actively engage with their stressors and create interpersonal conflict while awake. Elevated Ho scores are associated with a decrease in vagal modulation of RR intervals, as evidenced by reduced HF power and increased LF/HF ratio (Sloan et al., 1994), indicating an autonomic imbalance towards sympathetic dominance. Recent evidence has corroborated this finding by indicating an inverse relationship between hostility and HF power in response to mental arithmetic stressors involving harassment (Shapiro et al., 2000; Sloan et al., 2001).

Sympathetic influence on CV activity may also be assessed with impedance cardiography (ICG), a non-invasive technique for measuring thoracic blood volume changes (Sherwood et. al., 1990). Whereas HRV analysis is reflective of mixed vagal-sympathetic chronotropic (cardiac rate) influences on the heart, ICG provides a sympathetic inotropic (cardiac contractility) assessment. Measures derived from ICG may include pre-ejection period (PEP), a systolic time interval variable that reflects beta-adrenergic influences on myocardial contractility. Systolic intervals refer to various measures of cardiac events that occur between the onset of ventricular depolarization to the completion of blood ejection from the left ventricle.

A closer examination of the relationship between hostility and CHD will help to form a rationale for investigating CV activity in the present study. The following discussion outlines this literature, highlighting the various theories that have been proposed to explain the nature of this relationship.

Hostility and CHD

Coronary heart disease is an insidious disorder, since a patient may suffer from substantial coronary artery narrowing over decades without any overt symptoms (Smith & Leon, 1992). However, clinical manifestations of CHD, as in myocardial infarction, can occur quite suddenly and without warning. Since CHD is a pervasive disorder, it is prudent to recognize individual risk factors contributing to the onset of this disease.

The traditional risk factors for CHD include high blood cholesterol, hypertension, cigarette smoking, obesity, lack of physical activity, and blood glucose levels, accounting for approximately half the CHD cases each year (Jenkins, 1988). Research psychologists have approached the study of risk factors by exploring potential psychological phenomena that may affect CHD onset. A prominent risk factor emerging from this literature involves the investigation of the detrimental health impact associated with stress. For example, a physiological aspect of a prolonged stress response is injury to the lining of coronary arteries from enhanced blood flow, providing sites vulnerable to subsequent development of atherosclerosis, or narrowing of coronary arteries (Smith & Leon, 1992). Therefore, stress-related increases in HR and BP may contribute to CHD through the promotion of endothelial injury.

Stress may be viewed as a subjective phenomenon, arising from primary and secondary appraisal processes regarding the potential personal threat of a stressor and ability to cope with circumstances surrounding a stressor, respectively (Lazarus, 1966). Hence, individual differences in response to stressors are evident within this framework. The study of the Type A behavior pattern, characterized by competitiveness, impatience, hostility, an over-commitment to work, and a loud, rapid vocal style (Friedman & Rosenman, 1959), has been a major influence on studies linking stress responsivity and CHD. In contrast to the Type A behavior pattern, Type B behavior pattern is simply the absence of Type A characteristics. Thus, Type A personalities tend to respond to mild stressors more vigorously than their Type B counterparts, as reflected in physiological recordings of BP and HR (Harbin, 1989). The constellation of behaviors comprising the Type A personality is quite varied, with some components more predictive of CHD

incidence and mortality than others (Greenglass, 1996). Hostility has been demonstrated to be the most powerful aspect of the Type A behavior pattern for predicting CHD (Dembroski & Costa, 1987; Matthews, 1988).

Models of Hostility and CHD

Various models have been proposed over the past two decades to examine the relationship between trait hostility and CHD. The most prominent models, which should not be seen as mutually exclusive, include the psychophysiological reactivity model, the psychosocial vulnerability model, and the transactional model (Smith & Christensen, 1992a).

The psychophysiological reactivity model. The psychophysiological reactivity model for assessing the autonomic characteristics of trait hostility postulates that hostile individuals experience anger more often and more intensely than their non-hostile counterparts (Williams, Barefoot, & Shekelle, 1985), presumably causing them to experience more frequent, intense activation of the sympathetic-adrenomedullary system (Guyll & Contrada, 1998). It has been hypothesized that this activation promotes the development of coronary artery disease, precipitating clinical CHD (Kamarck & Jennings, 1991; Krantz & Manuck, 1984). Therefore, it appears as if the autonomic characteristics of trait hostility may serve as a valuable physiological marker for predicting the potential onset of coronary dysfunction.

However, basic research assessing the CV activity associated with the psychophysiological reactivity model has revealed inconsistent findings (Smith & Christensen, 1992b). Meta-analytic reports of this literature suggest that these inconsistencies may be due to variations in the type and intensity of laboratory stressors (Suls & Wan, 1993) and a neglect to report recovery data analyses from the lab stressor tasks (Schuler & O'Brian, 1997). In another review of 105 studies assessing physiological reactivity to stress, only 23% reported recovery data (Linden, Gerin, & Christenfeld, 1997). The results from studies that include recovery data have revealed a tendency for participants rating high on hostility to exhibit longer lasting blood pressure responses following an anger provoking stressor (e.g., Fredrickson et al., 2000). The

impact of defensiveness on the CV recovery to lab stressors for hostile subjects is unclear and has yet to be tested. Theoretically, the anger inhibition quality of defensiveness should prolong CV reactivity to stressors via the process of post stressor rumination. Analysis of CV recovery data may provide crucial insights into the basic underlying autonomic processes surrounding exposure to various stressors.

The psychosocial vulnerability model. A frequent finding of studies testing the psychophysiological reactivity model is that hostile individuals demonstrate heightened diastolic blood pressure (DBP) reactivity to stressors involving interpersonal provocation (Suls & Wan, 1993). Another result often observed concerns hostile individuals experiencing less social support from peers than nonhostile individuals, potentially accounting for enhanced CV reactivity to stressors (Davidson, Prkachin, Lefcourt, & Mills, 1996). These findings are associated with the psychosocial vulnerability model, which positions the factors of interpersonal conflict and lack of social support as instrumental mediating variables in the relationship between hostility and CV health status.

The transactional model. Whereas the psychosocial vulnerability model regards hostile individuals as being subjected to interpersonally challenging social environments, the transactional model emphasizes hostile individuals as having cynical attitudes that prompt them to actively engage with others in a way that reinforces subsequent negative interactions (Smith & Christensen, 1992a). The transactional model is a blending of the psychophysiological reactivity and psychosocial vulnerability models, asserting hostile individuals to demonstrate heightened CV reactivity to mundane daily life stressors as well as to the more frequent, prolonged and severe interpersonal stresses ensue by the social and cognitive correlates of hostility.

The defensiveness construct is cogent to the discussion of the psychosocial vulnerability and transactional models of hostility. In the context of the psychosocial vulnerability model, interpersonal conflict and a perceived lack of social support may be particularly relevant to the CV health status of defensively hostile individuals. The tendency for defensively hostile individuals to cater their social interactions to be viewed

in a socially desirable light, coupled with the frequent experience of anger and frustration arising from the distrust of others may exacerbate daily physiological stress responses to relatively mundane events in comparison to purely hostile individuals. Further, the qualities of anger suppression and rumination that characterize defensiveness are likely to have an indirect impact of continuing subsequent frustrating interactions related to the transactional model.

Hostility Assessment

In addition to the various models proposed to account for the relationship between hostility and CHD, there also exists multiple ways to assess the construct of hostility. The Cook & Medley Hostility Scale (Ho) (1954) is perhaps the most widely used measure of hostility in the literature involving CV reactivity to stressors. Hostile individuals have been described as creating a self-fulfilling prophecy by not only responding to everyday stressors with heightened physiological reactivity, but also reinforcing negative interactions with other people through cynical attitude, increasing the probability of subsequent stressful interactions (Smith & Pope, 1990). The Ho scale was found to provide a stronger association to severity of coronary artery disease (CAD) among patients undergoing coronary angiography than the association between Type A behavior assessments and CAD in the same sample (Williams et al., 1980).

Numerous studies have established a relationship between the Ho scale and coronary dysfunction. In a prospective study of 255 male physicians, Barefoot, Dahlstrom & Williams (1983) reported that physicians scoring above 13 on the Ho scale in medical school were approximately six times more likely to be diagnosed with CHD 25 years later than physicians scoring below 13 on the Ho scale. Further, the test/retest reliability of the Ho scale across these 25 years was determined to be .85, indicating that this scale measures a stable psychological trait. Individuals rating the sample median of 20 on the Ho scale were more than twice as likely to experience coronary calcification, a critical marker variable for the subsequent development of CHD, than individuals who rated below the median (Iribareen et al., 2000). Since this study investigated coronary calcification in individuals 18-30 yrs of age, these results suggest that a high level of trait

hostility may predispose young adults to CHD. Another study utilizing 424 angiography patients revealed that 70% of those with Ho scores above 10 had clinically significant CAD (Williams et al., 1980).

One problem in reviewing studies that directly link the Ho scale to coronary pathology is the difficulty in determining direction of causality. Because enhanced CV reactivity to laboratory stress may be associated with increased risk of subsequent CV morbidity and/or mortality (e.g., Boone, 1991), a host of studies have investigated the potential relationship between the Ho scale and CV reactivity to establish a causal link between hostility and CHD (e.g., Christensen & Smith, 1993). However, this research has yielded inconsistent results (Felston, 1995; Suls & Wan, 1993). For example, CV reactivity has not consistently differentiated between high and low hostile individual to active coping tasks (Kamarck, Manuck, & Jennings, 1990). The multidimensionality of the Ho scale may account for some of the inconsistencies in this literature.

An important consideration in evaluating the degree to which the Ho scale assesses the construct of hostility relates to the original intention behind the selection of item content. The 50 item Ho scale was initially devised to identify instructors experiencing difficulty in establishing a rapport with their students (Cook & Medley, 1954). Hence, the scale is bound to tap into areas outside the hostility construct, potentially causing variability in the literature regarding the present utility of the Ho scale as a means for determining trait hostility and any associations with CV activity and coronary pathology.

Hostility has been described as a multifaceted construct including cognitive, affective, and behavioral elements (Eckhardt, Barbour, & Stuart, 1997). The Ho scale has been analyzed as comprised of six subscales: hostile affect, hostile attributions, cynicism, aggressive responding, social avoidance, and a miscellaneous category termed 'other' (Barefoot, Dodge, Peterson, Dahlstrom, & Williams, 1989). The combination of the hostile affect, cynicism, and aggressive responding subscales, termed the composite hostility scale (Chost), has been found to be a better predictor of mortality (Barefoot et. al., 1989), myocardial ischemia (Helmets, et. al., 1993), and CV responses to active lab

stress (Larson & Langer, 1997) compared with the total Ho score. Therefore, the Chost scale represents a theoretically coherent combination for hostility: cynicism items are belief statements referring to cognitions, hostile affect items reflect emotional experience, and aggressive responding items refer to behavior.

Potential Mediators of the Hostility/CHD Relationship

It is important to obtain a strong understanding of the link between hostility and CHD by considering variables other than hostility assessment that may exert a significant influence on this relationship. Certain variables, as in inhibition of emotional expression, aspects of CV regulation, exposure to different types of stressors, and recovery from stressors may affect the hostility/CHD link. A discussion of these variables may highlight the parameters of this relationship.

Defensiveness. Hostile individuals prone to inhibiting their anger expression may be more vulnerable to coronary dysfunction than hostile individuals that express their anger (Dembroski et. al., 1985; Helmers et. al., 1995). The combination of trait hostility and defensiveness, a quality associated with the inhibition of expressing aggressive tendencies, may provide a more accurate depiction of the hostility-CHD relationship than utilizing the Ho scale alone. The Marlowe-Crowne Social Desirability Scale (MC) is a defensiveness inventory that assesses the likelihood individuals are to change their attitudes in response to social pressure, to cheat following poor test performance, and to inhibit their expression of anger (Crowne & Marlowe, 1964; Millham, 1974). Individuals with high MC scores tend to deny socially undesirable characteristics of self, a stable individual difference quality. (McCrae & Costa, 1983; Paulhus, 1984).

A series of investigations in both laboratory and field settings have revealed that HD men exhibit greater CV responses to stressors in comparison to high hostile, low defensive (LD) men (Helmers & Krantz, 1996; Jorgenson et al., 1995; Jamner et. al., 1991). Furthermore, defensive hostility has been demonstrated to predict CAD. In two independent samples, HD male CAD patients exhibited the greatest duration and most frequent episodes of ambulant ischemia during daily life activities and the most severe ischemia in response to two active coping stressors (Helmers et al., 1995). A similar

pattern of findings to The Kuopio Ischemia Heart Disease Study emerged from a recent investigation whereby HD male patients demonstrated more than a two-fold increase in the number of coronary arteries blocked in comparison to male patients scoring low on both scales (Jorgensen et al., 2001). Defensive-Hostile men have also been known to display greater systolic blood pressure (SBP) levels than LD men during baseline and active coping stressors (Helmers & Krantz, 1996). Another study revealed enhanced SBP reactivity to mental arithmetic and the Stroop task for hostile men who suppress their anger (Houston, Smith, & Cates, 1989).

Autonomic patterning may vary as a function of trait hostility (Schuler & O'Brian, 1997) and exposure to different types of stressors (Obrist, 1981) with an increase in sympathetic activity accompanied by a reduction in parasympathetic activity to active coping tasks. Examination of these differences may illustrate how certain types of stressors can mediate the hostility-CHD relationship.

Active and Passive Coping. A central issue surrounding the study of CV responses to various stressors relates to the evolutionary significance of the fight or flight response. The ability to quickly prepare the body for defensive activity has obvious survival relevance. However, life-threatening situations are relatively unusual occurrences in contemporary society. Certain individuals may be prone to respond inappropriately to moderate daily stressors by preparing the body for intensive activity when in fact little is required (Folkow & Neil, 1971). Over time, this chronic behavioral tendency may contribute to the development of coronary dysfunction through more frequent and prolonged stress responses.

The analysis of CV responsivity to laboratory stressors provides a medium to assess whether defensively hostile individuals are prone to physiological patterning likely to be associated with the subsequent development of CAD. One possible way to classify laboratory stressors is as active and passive coping tasks (Obrist, 1981). A primary distinction between active and passive coping stressor tasks is the degree of psychological engagement required for performance completion. The cold pressor task has been classified as a passive coping task because it requires relatively little continuous

mental effort to execute (Obrist, 1976). Reaction time and arithmetic tasks require considerable mental effort to execute and are considered active coping in nature.

Numerous studies have indicated significant CV reactivity exclusive to active coping tasks. A recent investigation on hostility and autonomic control, defined as an increase in HF-HPV during exposure to laboratory stressors, revealed an inverse relationship between this physiological measure and hostility for mental arithmetic and the Stroop task, but a non-significant relationship for orthostatic tilt (Sloan et al., 2001). Other studies have demonstrated stronger CV reactivity for hostile individuals exposed to active stressors involving harassment than active stressors without provocation (see Suls & Wan, 1993 for review). These studies support the transactional model of the relationship between hostility and CHD, suggesting that hostile individuals need to actively engage with their stressors to exhibit the pronounced physiological patterning likely to lead to coronary pathology.

However, a recent investigation on the CV reactions to a passive coping task, the forehead cold pressor, revealed a faster respiration rate and shorter PEP for high hostiles compared to low hostiles (Ruiz, Smith & Uchino, 2001). To test the transactional model in the defensively hostile population, it may be important to analyze differences in CV patterning based upon exposure to both types of stressors.

Cardiovascular reactivity to lab stressors may also vary as a function of sex. In general, men tend to demonstrate more CV reactivity to lab stressors than women (Guyll & Contrada, 1998). However, hostile women who suppress their anger have been found to demonstrate significantly elevated DBP reactivity to an active stressor in comparison to hostile men who suppress their anger (Harralson, Suarez, & Lawler, 1997). Therefore, anger suppression in combination with hostility may influence CV responses to active stressors between men and women.

In addition to reviewing CV reactivity to active and passive stressors, it may be helpful to include an analysis on recovery data to attain a comprehensive view on the relationship between physiological patterning and defensive hostility. Recovery data may

provide insights into a pathogenic process whereby HD individuals maintain elevated physiological activity after stressor removal.

Reactivity and Recovery. Early investigations of the underlying autonomic processes associated with stressful emotional states have asserted an overall sympathetic nervous system (SNS) dominance paradigm, in accord with Cannon's (1929) traditional fight or flight model. This perception of global SNS functioning has led researchers to the currently dominant SNS hyperreactivity hypothesis, whereby hostile individuals are considered to demonstrate hyperreactive sympathetic activity during frustrating situations.

The traditional model of SNS hyperreactivity treats autonomic reactivity as a temporary response perturbation from a relatively static norm, or homeostasis. The primary assumption behind the hyperreactivity hypothesis is that a negative correlation exists between sympathetic and parasympathetic influences on end organs (Suls & Wan, 1993). Therefore, under conditions of stress, it is believed that sympathetic activity is higher while parasympathetic activity is relatively low. This view is similar to the recurrent activation hypothesis of reactivity, linking chronically large changes from CV baseline to stress with disease risk (Manuck, Kasprowicz, Monroe, Larken, & Kaplan, 1989). However, the positive association between HRV and healthy cardiac dynamics (Goldberger, 1991) challenges these views by suggesting that resting measurements of heart rate are unstable and therefore do not represent a static norm. Further, coactivation of sympathetic and vagal influence on the heart may occur in response to certain stressors (Bernston et al., 1991). If the ANS does not operate in an exclusively reciprocal fashion with respect to sympathetic and parasympathetic influences on the heart, the hyperreactivity hypothesis is less compelling in explaining the mechanisms under which trait hostility exerts an influence on CHD development.

Autonomic assessments tracking both sympathetic and vagal activation and withdrawal may provide a more inclusive perspective on the hostility-CHD link than focusing primarily on sympathetic activity, as is common with the hyperreactivity model. The primary emphasis in the hyperreactivity model is SNS reactivity to stress,

characterized by chronic deviations from a homeostatic baseline norm. The hyperreactivity model predicts a positive association between the severity and frequency of stress induced CV changes from baseline and the development of CHD. An implicit assumption underlying this model is the notion that the CV baseline levels of hostile individuals are not by themselves pathogenic; rather, chronically enhanced sympathetically mediated CV reactivity to daily stressors is believed to contribute to coronary dysfunction. Therefore, the hyperreactivity model leaves the impression that ANS variability may be the toxic element linking hostility to CHD.

An alternative dynamical systems model of ANS functioning has been referred to as homeodynamic (Appel et al., 1989), suggesting that fluctuating processes, as in HRV, maintain organismic stability. A main conflict between the homeodynamic model and the hyperreactivity or recurrent activation models concerns the assumption of a steady state baseline. However, the homeodynamic model extends the emphasis from baseline perturbations to include autonomic activity after stressor removal, focusing on whether the overall autonomic pattern is rigid or flexible. According to this model, a rigid pattern in HRV from baseline to post-stress is considered detrimental to coronary health. Slow CV recovery after exposure to frustrating events may be a critical factor in the hostility-CHD link instead of the SNS hyperreactivity hypothesis (Brosschot & Thayer, 1998), since the vagus nerve produces rapid changes in HR (Saul, 1990). One potential explanation for the slow CV recovery hypothesis is that hostile individuals may experience a tendency to inhibit their anger expression in a way that prolongs the SNS response and maintains low vagal control.

Hostile individuals have been known to display larger and longer lasting BP responses to anger provoking situations (Fredrickson et al., 2000). Investigations involving the hand cold pressor have also demonstrated that hostile individuals maintain significant post-stressor elevations in HR and SBP in comparison to individuals rating low on hostility, who display non-significant post-stressor differences from CV baseline measurements (Demaree & Harrison, 1997; Demaree, Harrison, & Rhodes, 2000).

One focus of the current investigation was to determine the relationship between CV reactivity and recovery to active and passive coping tasks in defensive-hostile men and women from a young, non-clinical population. The mixture of defensiveness with high Chost scores prescribes a conflicted behavior pattern involving the tendency to respond aggressively to frustrating situations as well as a need to mold activity in a socially desirable fashion. The conflict between a need for approval and a hostile attitude may be associated with stressor-induced CV reactivity and potentially stress related CHD. The inhibition of aggressive tendencies under conditions of laboratory stress is likely to be reflected in depressed parasympathetic activity and elevated sympathetic responsivity. Also, HD men were expected to experience the most pronounced carry-over effect from lab stress reactivity, manifesting in exaggerated physiological activity during recovery. This study will add to the defensive hostility literature by providing analyses utilizing HRV as an indication of cardiac vagal tone. The assessment of recovery from the laboratory stressors may highlight the importance of reduced vagal control as a pathogenic marker potentially linking defensive hostility with CHD.

Design and Hypotheses

The present experiment was conducted to determine the CV patterning associated with defensive hostility. This relationship was examined by administering the Ho scale accompanied by the MC scale as screening devices to create two groups by sex: high defensive, high hostile (HD) men, low defensive, high hostile (LD) men, HD women, and LD women. Each participant was exposed to a video game (active task) and the hand-cold pressor (passive task) while continuous physiological measurements were assessed via electrocardiography (ECG), impedance cardiography (ICG), and a BP monitoring device. The design for this study is a 2 X 2 X 6 (group X sex X condition) mixed design, with condition as the within subjects factor.

In the present experiment, a baseline period was followed by an active task, a recovery period, a second baseline period, a passive task, and a second recovery period. The ordering of the active and passive tasks was counterbalanced. Change scores, task

minus baseline for reactivity and post-stress minus baseline for recovery, were calculated for all physiological measures and compared between groups.

HD participants were expected to exhibit the least vagal control and the most sympathetic alpha and beta adrenergic activity. As a marker of low vagal control, HD participants were predicted to display the least HF-HRV across all six conditions. Further, impedance measures were expected to reveal decreased PEP, accompanied by increases in HR from ECG, across all conditions with HD participants. In accord with previous investigations (eg., Mente & Helmers, 1999), HD men were expected to present elevated baseline BP. Post hoc analyses on reactivity scores were expected to confirm previous findings (eg., Jorgenson et al., 1995) with HD men presenting the most pronounced HR increases from baseline in response to the VG. Finally, the HD group was expected to display attenuated recovery scores with the least change in CV activity from baseline in comparison to the LD group.

METHOD

Participants

Participants were 63 right-handed undergraduate psychology students (age range: 18-27 years) at Virginia Polytechnic Institute & State University: 15 HD male participants; 16 LD male participants; 16 HD female participants; and 16 LD female participants. This sample size was acceptable for a large effect size with power = 0.7 (Cohen, 1988). Due to counterbalancing of tasks, half of each group received the active stressor first. However, other studies in the HD literature have not found order effect to be an issue in CV reactivity to lab stressors (e.g., Mente & Helmers, 1999).

Participants were selected on the basis of scores obtained via the Ho scale and MC scale. In accord with a recent investigation in the HD literature, high vs. low defensive participants were identified as having MC scores ≥ 16 or < 16 and hostile participants were identified as having Chost scores ≥ 15 , to determine the two groups in the present experiment (Larson & Langer, 1997). All participants in this experiment abstained from caffeine and alcohol for 12 hours before the study, and received extra credit in a psychology course for their participation. Average questionnaire scores, age, weight, and alcohol and caffeine intake by group and sex are reported in Table 1.

Apparatus

The Ho scale consists of 50 true-false items from the Minnesota Multi-phasic Personality Inventory (see Appendix A). In combined samples of more than 600 men and 600 women, high levels of internal consistency has been determined for the Ho scale, with Cronbach's alphas ranging from .80 to .82 for both men and women (Smith & Frohm, 1985). The test-retest correlations are high ($r > .8$) over periods of 1-4 years (Barefoot et al., 1983; Schekelle et al., 1983). Example items include, "It is safer to trust nobody," "I am not easily angered," and "I have at times had to be rough with people who were rude or annoying."

The MC scale is a 33 item true-false questionnaire designed to measure both social desirability and avoidance of disapproval (Crowne & Marlowe, 1964; see Appendix B). Scores range from 0 to 33, with higher scores representing a need for

approval and social defensiveness (Weinberger, 1990). This widely used measure is reported to be reliable (alpha coefficient = 0.88; 1 month test-retest correlation = 0.88) (Crowne & Marlowe, 1964).

Numerous studies have cited that resting baseline measurements may be contaminated by recent exercise, metabolic activity, and/or emotional excitement (eg., Jennings, Kamarck, Stewart, Eddy, & Johnson, 1992). Moreover, there is evidence to suggest that resting baseline measurements are less stable than that of task levels (Murphy, Alpert, & Walker, 1991). To ameliorate this possibility, a vanilla baseline condition has been standardized for comparison to physiological activity during lab tasks and was used in the current study. A vanilla baseline is a resting condition that involves exposure to neutral visual stimuli and may serve as a comparison condition to lab tasks when it shares all the situational characteristics of the experimental conditions except for the factor(s) the experimenter believes induces reactivity. Participants should be alert and inactive throughout the baseline procedure, which should not be complex or exciting.

The visual stimulus used as a ‘vanilla baseline’ was a 3 minute segment of a video by Godfrey Reggio (coproduced by Francis Ford Coppola and George Lucas) entitled *Powaqqatsi: Life in Transformation*. *Powaqqatsi* is a multicultural documentary portraying daily life events in various countries. There are no words in the film, and the scenes come from all over the world. The selected section of *Powaqqatsi* provided a neutral visual stimulus to serve as a vanilla baseline because it does not portray people under stress.

The electrocardiogram and impedance cardiography were recorded with the Ambulatory Monitoring System (AMS) v 4.4 (Vrije Universiteit Amsterdam, the Netherlands), using Ag-AgCl electrodes; the validity and reliability of this device has been established (Willemsen et al., 1996). BP was monitored by usage of the Vasotrac (Medwave, Inc.) system, a non-invasive continuous blood pressure recording device placed over the radial artery and held in place by a Velcro wrist strap. This device has been found not to produce significant discomfort or distraction to participants during

extended use (Friedman, Christie, Weaver, & Sargent, 2003). A car race game task (Test Drive 4, Accolade, Inc., 1997) was used in the active coping procedure.

Dependent Variables

The measure derived from the ECG was HR, reflecting sympathetic beta-adrenergic and parasympathetic influences (Saul, 1990). The ECG inter-beat interval (IBI) data was analyzed with a spectral analysis procedure (SAS for Win98, v.8.2, 2001). IBI's represent the amount of time in milliseconds that elapses between the R spikes from the cardiac waveform. The differences between adjacent IBI values were computed and subjected to an ordinary least squares regression procedure for detrending. Output residuals were used to create power spectral density units ($\text{ms}^2 \text{Hz}^{-1}$). LF (0.04-0.15 Hz) and HF (0.15-0.40 Hz) ranges were extracted from the power spectral density units, with the HF component serving as a measure of cardiac vagal activity. A natural logarithm procedure was employed (SPSS, v.10, 2001) to correct for skewed raw score distributions in the spectral data.

The non-invasive ICG technique provided a measure of PEP through the introduction of a very low amplitude high frequency alternating current, completely harmless to biological tissues, into the thorax to track blood volume changes (Sherwood, Royal, Hutcheson, & Turner, 1992). SBP and DBP were assessed with the BP monitoring device.

Procedure

The conduction of this study has been approved by the Institutional Review Board at Virginia Polytechnic Institute (#02-025). Each participant signed an informed consent and completed a health screening information form (see appendices C and D). Upon arrival at the lab, each participant had six thoracic electrodes applied to the torso to record ECG and ICG in accordance with configuration guidelines described in the AMS user manual v 1.2 (Vrije Universiteit, Amsterdam, the Netherlands). The BP cuff was placed on the wrist of the non-dominant (left) hand.

All task instructions were given to the participants prior to each task administration. Participants engaged in the following 3-minute laboratory tasks:

(1) Vanilla Baseline (VB): The participant sat quietly and relaxed in a comfortable lounge chair while observing a video screen showing a neutral segment of *Powaqqatsi*. This procedure served as a resting baseline.

(2) (VG): The participant played a challenging race car video game that has been reported as ‘arousing’ and ‘engaging’ as a stressor (Santucci & Friedman, 2003). This challenging task exemplified “active coping” that elicited sympathetic beta-adrenergic activity and parasympathetic withdrawal (Obrist, 1981).

(3) Recovery (RC): The participant was instructed to sit quietly and relaxed. This procedure allowed for assessment of CV recovery from the previous laboratory task.

(4) Vanilla Baseline 2 (VB2): The participant was again instructed to sit quietly in the lounge chair while viewing another neutral segment of *Powaqqatsi*.

(5) Hand cold pressor (CP): The participant placed the right hand up to the wrist in a bucket of iced water kept between 0-3 degrees C. The hand was removed from the water after 1.5 minutes, and then re-emerged after 10 seconds to allow immersion to be maintained for three minutes with minimal discomfort. The hand cold pressor task is a passive coping procedure characterized by sympathetic alpha-adrenergic activation (Saab et al., 1999).

(6) RC: The participant was instructed to again sit quietly and relaxed. Recording equipment was removed after completion of this RC procedure. The ordering of active and passive coping tasks were counterbalanced between participants.

Statistical Analyses

A series of multivariate analyses of variance (MANOVA's) on repeated measures were conducted to test significant differences in CV activity by group. A 2 X 2 X 2 (group X sex X baseline) MANOVA assessed any significant baseline differences in CV activity across groups and between baseline conditions. In the context of significant differences in CV activity at baseline as a function of sex, separate analyses were conducted for men and women. A 2 X 6 (group X condition) MANOVA was conducted on mean values to detect any significant differences in CV activity by group during the individual tasks. A 2 X 2 (group X condition) MANOVA was conducted on reactivity

difference scores to evaluate CV activity in response to the two stressors by group. A 2 X 2 (group X condition) MANOVA assessed significant changes in recovery difference scores by group. Multiple comparison Bonferroni procedures were used as post hoc tests to control for error rates.

RESULTS

Baseline Tests

The 2 X 2 X 2 MANOVA on baseline data yielded a significant main effect for sex, $F(6, 54)=5.160$, $p<.001$, indicating differential CV activity during baseline periods by sex. The condition and group terms were ns, indicating that the CV activity exhibited by the groups did not differ as a function of baseline period or group. Univariate analyses yielded a significant group effect for LFP at baseline, $F(1,59)=7.450$, $p=.008$. Bonferroni multiple comparisons revealed HD subjects to exhibit higher LFP than LD subjects at both baseline periods, $t'(61)=2.610$, $p<.05$ for baseline 1 and $t'(61)=2.346$, $p<.05$ for baseline 2. Figure 1 displays mean log-transformed LFP scores by group for each baseline condition.

Univariate sex effects were observed for PEP, $F(1,59)=6.635$, $p=.013$; SBP, $F(1,59)=5.199$, $p=.026$; DBP, $F(1,59)=5.338$, $p=.024$; and LFP, $F(1,59)=6.965$, $p=.011$. Sex effects for HR and high frequency power (HFP) were ns. Bonferroni multiple comparisons revealed men to display higher BP and LFP at both baseline periods in comparison to women, whereas women showed higher PEP than men at both baseline periods.

Results for Men

Mean Task Values. The 2 X 6 MANOVA on mean scores for all conditions revealed a significant main effect for condition, $F(30, 562)=6.183$, $p<.001$, indicating differential CV activity as a function of condition. The group term was ns. A condition X group interaction was observed, $F(30, 562)=1.706$, $p=.012$.

All univariate analyses for condition were significant, p 's $<.006$. A condition X group interaction was observed for HR, $F(5, 145)=3.945$, $p=.002$. Figure 2 displays mean HR by group across all conditions for men. LD men displayed higher HR than HD men across all conditions except during the CP, when HD men exhibited higher HR than LD men. A trend for a condition X group interaction was observed for SBP, $F(5, 145)=2.229$, $p=.054$. Figure 3 displays SBP by group across all conditions for men. LD men showed higher SBP across all conditions compared to HD men, with the exception of during the

CP when SBP was essentially the same. Multiple comparison Bonferroni analyses revealed no differences between groups for HR and SBP across conditions.

Reactivity Scores. The 2 X 2 MANOVA on reactivity scores yielded no values for group, condition, and condition X group interaction. However, univariate analyses on reactivity scores revealed main group effects for HR, $F(1, 29)=8.006$, $p=.008$ and HFP, $F(1, 29)=5.765$, $p=.023$. All other univariate group effects were ns. Figure 4 displays HR reactivity by group for the two stressors. HD men displayed significantly elevated HR reactivity to the CP in comparison to LD men, $t'(29)=3.084$, $p<.01$. Although HD men also showed pronounced HR reactivity to the VG in comparison to LD men, the difference was ns. Figure 5 displays mean log-transformed HFP by group for the two stressors. LD men showed a slight increase in HFP to the CP, whereas HD men showed the opposite with a reduction in HFP, $t'(29)=2.727$, $p<.05$. Both HD men and LD men exhibited reduced HFP in response to the VG and the difference between the groups was ns.

Recovery Scores. The 2 X 2 MANOVA on recovery scores was ns. However, univariate analyses on recovery scores revealed a significant condition effect for LFP, $F(1,29)=4.727$, $p=.038$, indicating differential post stressor LFP activity by stressor. All other univariate analyses were ns. Post-stressor LFP was higher following the CP in comparison to the VG.

Results for Women

Mean Task Values. The 2 X 6 MANOVA on mean scores for all conditions revealed a significant main effect for condition, $F(30, 582)=8.316$, $p<.001$, indicating differential CV activity as a function of condition. The group and interaction terms were ns. However, univariate analyses yielded a significant group term for LFP, $F(1,30)=6.291$, $p=.018$. Figure 6 displays mean log-transformed LFP by group across all conditions. HD women showed consistently higher LFP across conditions in comparison to LD women. Multiple comparison Bonferroni analyses revealed HD women to exhibit higher LFP in comparison to LD women during the first baseline condition, $t'(30)=3.075$, $p<.05$. All univariate condition terms were significant, $p's <.001$, except a ns PEP term.

Reactivity Scores. The 2 X 2 MANOVA on reactivity scores for women yielded a significant condition term, $F(6, 25)=5.158$, $p=.001$, indicating differential CV activity as a function of stressor. The group and interaction terms were ns. Univariate analyses revealed significant condition terms for SBP, $F(1, 30)=12.855$, $p=.001$; DBP, $F(1,30)=12.690$, $p=.001$; LFP, $F(1, 30)=16.449$, $p<.001$; and HFP, $F(1, 30)=11.765$, $p=.002$. The CP task elicited increases in BP and HRV, whereas the VG elicited decreases in these CV domains.

Recovery Scores. The 2 X 2 MANOVA on recovery scores for women yielded ns group and condition terms. However, the condition X group interaction was significant, $F(6, 25)=2.778$, $p=.033$. Univariate analyses revealed significant condition X group interaction terms for SBP, $F(1, 30)=7.735$, $p=.009$ and DBP, $F(1, 30)=17.477$, $p<.001$.

Figure 7 displays mean SBP recovery scores by group for the two stressors. LD women showed significantly elevated post-stress SBP following the VG in comparison to HD women, $t'(30)=2.514$, $p<.05$. Interestingly, HD women exhibited essentially the same post-stress SBP compared to baseline. An opposite ns pattern was observed with SBP recovery from the CP task. HD women displayed elevated post-stress SBP following the CP task, whereas LD women showed lower post-stress SBP following the CP task than at baseline.

Figure 8 displays mean DBP recovery scores by group for the two stressors. The overall CV patterning is roughly the same as that displayed in Figure 7 for SBP recovery. LD women showed elevated post-stress DBP following the VG in comparison to HD women, $t'(30)=2.912$, $p<.05$. HD women exhibited post-stress DBP slightly less than their respective baseline. An opposite ns tendency was observed in DBP recovery from the CP task, whereby LD women displayed decreased post-stress DBP compared to baseline and HD women showed increased post-stress DBP compared to baseline.

DISCUSSION

This experiment was intended to provide useful information regarding the hostility construct and how it may interact with defensiveness in predicting CV reactivity and recovery to laboratory stressors. A primary finding from this experiment revealed that HD men displayed the most pronounced HR increase and HFP decrease in response to the CP task. A central hypothesis of this experiment was that HD participants would exhibit a rigid CV patterning, with the least pronounced recovery to both stressors in comparison to LD participants. However, this hypothesis was not supported; rather, it was found that type of stressor may be of particular importance in predicting the CV reactivity and recovery of HD participants or LD participants, respectively.

Although these differences were ns, visual inspection of tables 2 and 3 reveals that LD men exhibited the highest resting SBP and DBP for baseline and recovery periods. Further, LD men displayed the most SBP and DBP reactivity to the VG, but the least SBP and DBP reactivity to the CP. However, HD men displayed just the opposite pattern, with the most SBP, DBP and HR reactivity to the CP.

Baseline data revealed HD participants to display significantly higher LFP levels in comparison to LD participants. Further, HD women maintained high LFP scores across all conditions. Although there is substantial evidence that HFP represents a non-invasive index of cardiac vagal modulation (eg, Pagani et al, 1986), the physiological substrates of LF oscillations are less well understood. However, it has been argued from multiple sources that LFP reflects a considerable amount of sympathetic influences on cardiac activity (eg., Friedman, Thayer, & Tyrell, 1996). The finding that LD women exhibit lowered LFP at baseline and across conditions is not outside the predictions for CV activity in this experiment when considering the sympathetic contribution to LF oscillations.

HD participants were expected to display the least HF-HRV across conditions, marking a tendency toward reduced vagal control, but this prediction was not entirely supported. However, HD men were found to exhibit reduced HF-HRV in response to

both stressors. Moreover, the reduction in HF-HRV displayed by HD men in response to the CP task was significantly lower than the slight increase exhibited by LD men.

In accord with research indicating that HD men display heightened HR reactivity to active stressors (eg., Jorgenson et al., 1995), HD men were expected to display the most pronounced HR reactivity to the VG in comparison to LD men. Although HD men did display more HR reactivity to the VG in comparison to LD men, the difference was ns. Rather, the HD men were found to present significantly elevated HR reactivity to the CP in comparison to LD men.

Condition X group interactions in blood pressure recovery revealed that LD women exhibited weak BP recovery following the VG task, whereas HD women displayed weak BP recovery following the CP task. Exposure to the VG task may be a good opportunity for LD participants to engage in an exciting task, providing support for the description of this stressor in a recent investigation (Santucci & Friedman, 2003). Although these interactions were not predicted, they are somewhat in accord with a CV patterning emerging from the present experiment.

Defensiveness may moderate the CV patterning of hostile individuals, characterized by a tendency for HD participants to display enhanced sympathetic alpha and beta adrenergic activity and decreased vagally mediated HRV in response to passive stressors. Alternatively, LD participants appear to exhibit enhanced sympathetic alpha and beta adrenergic activity and decreased vagally mediated HRV in response to active stressors. Gender may differentiate the CV patterning of HD participants, whereby HD men display pronounced HR and decreased HF-HRV reactivity to passive stressors and HD women display a tendency toward weak BP recovery from passive stressors.

A key discrepancy between this experiment and the majority of studies in the HD literature concerns the use of harassment or some form of interpersonal frustration as an essential quality defining the lab stressors. For example, Jorgenson and colleagues (1995) exposed participants to an oral mental arithmetic stressor involving interpersonally frustrating inaccurate feedback of poor performance and produced significant SBP and HR reactivity in HD men. In accord with the psychosocial vulnerability model of

hostility, an argument has been made by various investigators that it is necessary to harass hostile individuals, thereby invoking the critical feelings of anger and frustration, to give rise to significant elevations in CV responding to lab stress (eg, Suarez & Williams, 1989; Suls & Wan, 1993). However, these studies do not specifically address the influence of defensiveness on the relationship between hostility and harassment in predicting CV reactivity and recovery to lab tasks.

A primary characteristic of the defensiveness construct is the tendency to inhibit the expression of angry feelings while catering social interactions to be viewed in a socially desirable light by others. Therefore, interpersonal provocation may be particularly relevant to the 'approach-avoidance' conflict that typifies defensive hostility, but not necessarily hostility alone. HD men may exhibit significant elevations in HR and reductions in HF-HRV as well as poor CV recovery in comparison to LD men when exposed to active coping stressors involving harassment.

Another critical limitation needing to be addressed with this experiment concerns laterality issues related to hostility. There is evidence to suggest that individuals rating high in hostility present significantly lower regional blood flow to the prefrontal cortex during mental stress in comparison to those rating low on hostility (Shapiro et al., 2000). Further, the orbital-frontal cortex has connections to amygdaloid bodies that seem to decrease hostility levels (Butter, Snyder, & McDonald, 1970). In particular, the right anterior cerebrum may be responsible for the inhibition or regulation of hostile behaviors. Moreover, hostile individuals exhibit a heightened left ear advantage to identifying auditory stimuli during a dichotic listening task after exposure to the CP, suggesting that right temporal activation is of particular importance to the hostility construct (Demaree & Harrison, 1997).

In the present experiment, all participants inserted their right hands up to the wrist into the ice water for the CP task. Other studies that observed significantly elevated HR and SBP recovery scores in hostile men had participants submerge their left hands for the CP task (Demaree & Harrison, 1997; Demaree, Harrison, & Rhodes, 2000). The relationship between defensiveness and cerebral laterality is currently unknown.

However, it is possible that the results in the current experiment would be more pronounced if participants inserted their left hands into the ice water for the CP task. An argument could be made that the current experiment stressed the ‘wrong brain’ during the CP task by having participants insert their right hands into the ice water.

Finally, it is important to discuss the issue of recovery period assessment. The procedure used in the present study was to subtract mean baseline values from mean post-stress values for each dependent variable. This procedure is the same method used in a recent investigation of CV recovery from active and passive stressors (Glynn, Christenfeld, & Gerin, 2002). Another method for assessing recovery concerns the time it takes for a participant to resume baseline levels within a specified period (Linden, Gerin, & Christenfeld, 1997). One of the primary variables of interest for this investigation, HF-HRV, is strongly affected by temporal factors. The premise behind the homeodynamic model is embedded within the consideration of CV changes over time. Future studies of this nature may consider using a recovery assessment procedure more sensitive to temporal issues than the one employed in the present study.

Conclusions

The results of this experiment provide at least partial support for both the recurrent activation hypothesis or hyperreactivity hypothesis and the homeodynamic model. The finding that HD men exhibit the most pronounced HR reactivity to the CP task is in accord with the hyperreactivity model. However, the finding that HD women exhibit weak BP recovery to the CP supports the homeodynamic model.

Overall, the results for this investigation suggest that LD participants are alpha adrenergic responders to active stressors, whereas HD participants are alpha and beta adrenergic responders to passive stressors. This pattern of CV activity was supported for female participants in the context of CV recovery and male participants in the context of CV reactivity. Future studies should incorporate harassment or interpersonal provocation as part of the active stressors and have participants sub-merge their left hands in ice water as part of the CP task.

REFERENCES

- Appel, M.V., Berger, R.D., Saul, J.P., Smith, J.P., & Cohen, R.J. (1989). Beat to beat variability in cardiovascular variables: Noise of music? *Journal of the American College of Cardiology*, 14, 1139-1148.
- Amerena, J. & Julius, S. (1995). Role of the nervous system in human hypertension. In N.K. Hollenberg (ed), *Hypertension: Mechanisms and Therapy*. Pennsylvania: Current Medicine.
- Askelrod, S., Gordon, D., Ubel, F.A., Shannon, D.C., Barger, A.C., & Cohen, R.J. (1981). Power spectrum analysis of heart rate fluctuations: A quantitative probe of beat-to-beat cardiovascular control. *Science*, 213, 220-222.
- Barefoot, J.C., Dahlstrom, G., & Williams, R.B. (1983). Coronary heart disease incidence and total mortality: A 25 year follow-up study of 255 physicians. *Psychosomatic Medicine*, 45, 59-63.
- Barefoot, J.C., Dodge, K.A., Peterson, B.L., Dahlstrom, W.G., & Williams, R.B. (1989). The Cook-Medley hostility scale: Item content and ability to predict survival. *Psychosomatic Medicine*, 46-57.
- Bernston, G.G., Cacioppo, J.T., Quigley, K.S. (1991). Autonomic determinism: The modes of autonomic control, the doctrine of autonomic space, and the laws of autonomic constraint. *Psychological Review*, 98, 459-487.
- Bigger, J.T., Fleiss, J.L., Rolnitzky, L.M., & Steinman, R.C. (1993). The ability of several short-term measures of R-R variability to predict mortality after myocardial infarction. *Circulation*, 88, 927-934.
- Bongard, S., Pfeiffer, J.S., Al'Absi, M., Hodapp, U., & Linnenkemper, G. (1997). Cardiovascular responses during effortful active coping and acute experience of anger in women. *Psychophysiology*, 34, 459-466.
- Boone, J.L. (1991). Stress and Hypertension. *Primary Care*, 18, 623-645.
- Bronis, M. (1983). Heart rate variability and autonomous system: Some possible relations. *Automedica*, 4, 223-225.

- Brosschot, J. F. & Thayer, J.F. (1998). Anger inhibition, cardiovascular recovery, and vagal function: a model of the link between hostility and cardiovascular disease. *Annals of Behavioral Medicine*, 20(4), 326-32.
- Butter, C.M., Snyder, D.R., & McDonald, J.A. (1970). Effects of orbital frontal lesions on aversive and aggressive behaviors in rhesus monkeys. *Journal of Comparative Physiology and Psychology*, 72, 132-144.
- Cannon, W.B. (1929). *Bodily Changes in Pain, Hunger, Fear, and Rage*. 2nd ed. New York: D. Appleton.
- Christensen, A.J. & Smith, T.W. (1993). Cynical hostility and cardiovascular reactivity during self-disclosure. *Psychosomatic Medicine*, 55, 193-202.
- Cohen, H., Benjamin, J., Geva, A.B., Matar, M.A., Kaplan, Z., & Kotler, M. (2000). Autonomic dysregulation in panic disorder and in post-traumatic stress disorder: Application of power spectrum analysis of heart rate variability at rest and in response to recollection of trauma or panic attacks. *Psychiatry Research*, 96, 1-13.
- Cohen, J. (1988). *Statistical Power Analysis for the Behavioral Sciences* (2nd ed). New Jersey: Erlbaum.
- Cook, W.W. & Medley, D. M. (1954). Proposed hostility and pharisaic-virtue scales for the MMPI. *Journal of Applied Psychology*, 238, 414-18.
- Crowne, D.P. & Marlowe, D. (1964). *The Approval Motive: Studies in Evaluative Dependence*. New York: Wiley.
- Davidson, D.W., Prachkin, K.M., Lefcourt, H.M., & Mills, D.E. (1996). Towards a psychosocial mediator model of hostility and CHD. *Psychology and Health*, 11, 421-429.
- Demaree, H.A. & Harrison, D.W. (1997). Physiological and neuropsychological correlates of hostility. *Neuropsychologia*, 35(10), 1405-1411.
- Demaree, H.A., Harrison, D.W., & Rhodes, R.D. (2000). Quantitative electroencephalographic analyses of cardiovascular regulation in low- and high-hostile men. *Psychobiology*, 28(3), 420-431.

- Dembroski, T.M. & Costa, P.T. (1987). Coronary prone behavior: Components of the Type A pattern and hostility. *Journal of Personality*, 55, 211-235.
- Dembroski, T.M., MacDougal, J.M., Williams, R.B., Haney, T.L., & Blumenthal, J.A. (1985). Components of Type A, hostility, and anger-in: Relationship to angiographic findings. *Psychosomatic Medicine*, 47(3), 219-233.
- Eckhardt, C.I., Barbour, K.A., & Stuart, G.A. (1997). Anger and hostility in martially violent men: Conceptual distinctions, measurement issues, and literature review. *Clinical Psychology Review*, 17, 333-359.
- Felston, G. (1995). Cynical hostility influences anger, but not cardiovascular reactivity during competition with harassment. *International Journal of Psychophysiology*, 19, 223-231.
- Folkow, B. & Neil, E. (1971). *Circulation*. New York: Oxford University Press.
- Fredrickson, B.L, Maynard, K.E., Helens, M.J., Haney, T.L., Siegler, I.C., & Barefoot, J.C. (2000). Hostility predicts magnitude and duration of BP response to anger. *Journal of Behavioral Medicine*, 23(3), 229-243.
- Friedman, B.H., Christie, I.C., Weaver, J.B, & Sargent, S.L. (2003). *Self-reported sensitivity to continuous non-invasive blood pressure monitoring via the radial artery*. Manuscript submitted for publication.
- Friedman, B.H. & Thayer, J.F. (1998a). Anxiety and autonomic flexibility: A cardiovascular approach. *Biological Psychology*, 49, 303-23.
- Friedman, B.H. & Thayer, J.F. (1998b). Autonomic balance revisited: Heart rate variability and panic anxiety. *Journal of Psychosomatic Research*, 44, 133-151.
- Friedman, B.H., Thayer, J.F., & Tyrell, R.A. (1996). Spectral characteristics of heart period variability in shock avoidance and cold face stress in normal subjects. *Clinical Autonomic Research*, 6, 147-152.
- Friedman, M. & Rosenman, R.H. (1959). Association of a specific overt behavior pattern with blood and cardiovascular findings. *Journal of the American Medical Association*, 169, 1286-1296.

- Fukudo, S., Lane, J.D., Anderson, N.B., Kuhn, C.M., Shanberg, S.M., McCowan, N., Muranaka, M., Suzuki, J., & Williams, R.B. (1992). Accentuated vagal antagonism of beta-adrenergic effects on ventricular repolarization. *Circulation*, 85(6), 2045-2053.
- Glynn, L.M., Christenfeld, N., & Gerin, W. (2002). The role of rumination in recovery from reactivity: Cardiovascular consequences of emotional states. *Psychosomatic Medicine*, 64, 714-726.
- Goldberger, A.L. (1991). Is the normal heart beat chaotic or homeostatic? *News in Psychological Sciences*, 6, 87-91.
- Greenglass, E.R. (1996). Anger and coronary heart disease. In C.D. Spielberger, I.G. Sarason (eds). *Stress and Emotion: Anxiety, Anger, & Curiosity*, v(16). Washington DC: Taylor & Francis.
- Guyll, M., & Contrada, R.J. (1998). Trait hostility and ambulatory cardiovascular activity: Responses to social interaction. *Health Psychology*, 17(1), 30-39.
- Harbin, T.J. (1989). The relationship between Type A behavior pattern and physiological responsivity: A quantitative review. *Psychophysiology*, 26, 110-119.
- Harralson, T.L., Suarez, E.C., & Lawler, K.A. (1997). Cardiovascular reactivity among hostile men and women: The effects of sex and anger expression. *Women's Health: Research on Sex, Behavior, and Policy*, 3(2), 151-164.
- Helmers, K.F. & Krantz, D.S. (1996). Defensive hostility, sex, and cardiovascular levels and responses to stress. *Annals of Behavioral Medicine*, 18(4), 246-254.
- Helmers, K.F., Krantz, D.S., Baniz, C.N., Klein, J., Kop, W.J., Gottdeiner, J.S., & Rozanski, A. (1995). Defensive hostility: Relationship to multiple markers of cardiac ischemia in patients with coronary artery disease. *Health Psychology*, 14(3), 202-209.
- Helmers, K.F., Krantz, D.S., Howell, R., Klein, J., Bairey, N., & Rozanski, A. (1993). Hostility and myocardial ischemia in coronary artery disease patients: Evaluation by sex and ischemia index. *Psychosomatic Medicine*, 55, 29-36.

- Helmrs, K.F., Krantz, D.S., Merz, C.N.B., Klein, J., Kop, W.J., Gottdiener, J.S., & Rozanski, A. (1995). Defensive hostility: Relationship to multiple markers of cardiac ischemia in patients with coronary disease. *Health Psychology, 14*, 202-209.
- Houston, B.K., Smith, M.A., & Cates, D.S. (1989). Hostility patterns and cardiovascular reactivity to stress. *Psychophysiology, 26*, 337-342.
- Iribarren, C., Sidney, S., Bild, D.E., Liu, K., Markovitz, J.H., Roseman, J.M., & Matthews, K. (2000). Association of hostility with coronary artery calcification in young adults. *Journal of the American Medical Association, 283*(19), 2546-2552.
- Jamner, L., Shapiro, D., Goldstien, I., & Hug, R. (1991). Ambulatory blood pressure and heart rate in paramedics: Effects of cynical hostility and defensiveness. *Psychosomatic Medicine, 53*, 393-406.
- Jenkins, C.D. (1988). Epidemiology of cardiovascular diseases. *Journal of Consulting and Clinical Psychology, 56*, 324-332.
- Jennings, J.R., Kamarck, T., Stewart, C., Eddy, M., & Johnson, P. (1992). Alternate cardiovascular baseline assessment techniques: Vanilla or resting baseline. *Psychophysiology, 29*(6), 742-750.
- Jorgenson, R.S., Abdul-Karim, K., Kahan, T.A., & Frankowski, J.J. (1995). Defensiveness, cynical hostility and cardiovascular reactivity: A moderator analysis. *Psychotherapy and Psychosomatics, 64*, 156-161.
- Jorgenson, R.S., Frankowski, J.J., Lantinga, L.J., Phadke, K., Sprafkin, R.P., & Abdul-Karim, K.W. (2001). Defensive hostility and CHD: A preliminary investigation of male veterans. *Psychosomatic Medicine, 63*, 463-469.
- Kamada, T., Miyake, S., Kumashiro, M., Monou, H., & Inoue, K. (1992). Power spectral analysis of heart rate variability in type A's and type B's during mental workload. *Psychosomatic Medicine, 54*, 462-470.
- Kamarck, T. & Jennings, J.R. (1991). Biobehavioral factors and sudden cardiac death. *Psychological Bulletin, 109*, 42-75.

- Kamarck, T.W., Manuck, S.B., & Jennings, J.R. (1990). Social support reduces cardiovascular reactivity to psychological challenge: A laboratory model. *Psychosomatic Medicine*, 52, 42-58.
- Kleiger, R.E. Miller, J.P., Bigger, J.T., & Moss, A.J. (1987). Decreased heart rate variability and its association with increased mortality after acute myocardial infarction. *American Journal of Cardiology*, 59, 256-262.
- Klein, E., Cnaai, E., Harel, T., Braun, S., & Ben-Haim, S.A. (1995). Altered heart rate variability in panic disorder patients. *Biological Psychiatry*, 37, 18-24.
- Krantz, D.S. & Manuck, S.B. (1984). Acute psychophysiologic reactivity and risk of cardiovascular disease: A review and methodological critique. *Psychological Bulletin*, 96, 535-64.
- Latson, T.W. (1994). Principles and applications of heart rate variability analysis. In Lynch, C. III (Ed.), *Clinical Cardiac Electrophysiology: Preoperative Considerations* (pp. 307-49). Philadelphia: J.B. Lippincott.
- Larson, M.R. & Langer, A.W. (1997). Defensive hostility and anger expression: Relationship to additional heart rate reactivity during active coping. *Psychophysiology*, 34, 177-184.
- Lazarus, R.S. (1966). *Psychophysiological Stress and the Coping Process*. New York: McGraw Hill.
- Liao, D., Cai, J., Rosamond, W.D., Barnes, R.W., Hutchinson, R.G., Whitsel, E.A., Rautaharju, P. & Heiss, G. (1997). Cardiac autonomic function and incident CHD: A population-based case-cohort study. *American Journal of Epidemiology*, 145, 696-706.
- Linden, T.L.W., Gerin, E.W., & Christenfeld, N. (1997). Physiological stress reactivity and recovery: Conceptual siblings separated at birth? *Journal of Psychosomatic Research*, 42(2), 117-135.
- Malliani, A., Pagani, M., Lombardi, F., & Cerutti, S. (1991). Cardiovascular neural regulation explored in the frequency domain. *Circulation*, 84, 482-492.

- Manuck, S.B., Kasprovicz, A.L., Monroe, S.M., Larkin, K.T., & Kaplan, J.R. (1989). Psychophysiologic reactivity as a dimension of individual differences. In N. Schneiderman, S.M. Weiss, & P.G. Kaufmann (Eds.), *Handbook of research methods in cardiovascular behavioral medicine*. New York: Plenum Press, pp. 365-382.
- Matthews, K.A. (1988). Coronary heart disease and Type A behaviors: Update on and alternative to the Booth-Kewley and Friedman quantitative review. *Psychological Bulletin*, 104, 373-380.
- McCrae, R.R. & Costa, P.T. (1983). Social desirability scales: More substance than style. *Journal of Consulting and Clinical Psychology*, 51, 882-888.
- Mente, A. & Helmers, K.F. (1999). Defensive hostility and cardiovascular responses to stress in young men. *Personality and Individual differences*, 27, 683-694.
- Miller, T.Q., Smith, T.W., Turner, C.W., Guijaro, M.L., & Hallet, A.J. (1996). A meta-analytic review of research on hostility and physical health. *Psychological Bulletin*, 119, 332-48.
- Millham, J. (1974). Two components of need for approval score and their relationship to cheating following success and failure. *Journal of Research in Personality*, 8, 378-392.
- Murphy, J.K., Alpert, B.S., & Walker, S.S. (1991). Whether to measure change from baseline or absolute label in studies of children's cardiovascular reactivity: A two year follow-up. *Journal of Behavioral Medicine*, 14, 409-414.
- Obrist, P.A. (1976). The cardiovascular-behavioral interaction as it appears today. *Psychophysiology*, 13, 95-107.
- Obrist, P.A. (1981). *Cardiovascular Physiology*. New York: Plenum Press.
- Pagani, M., Lombardi, F., Guzzetti, S., Rimoldi, O., Furlan, R., Pizzinelli, P., Sandrone, G., Malfatto, G., Dell'Orto, S., Piccaluga, E., Turiel, M., Basselli, G., Cerutti, S., & Malliani, A. (1986). Power spectral analysis of heart rate and arterial pressure variabilities as a marker of sympatho-vagal interaction in man and conscious dog. *Circulation Research*, 59, 178-193.

- Paulhus, D. (1984). Two-component models of socially desirable responding. *Journal of Personality and Social Psychology*, 46, 598-609.
- Peng, C.K., Buldyrev, S.V., Hausdorff, J.M., Havlin, S., Mietus, J.E., Simons, M., Stanley, H.E., & Goldberger, A.L. (1994). Non-equilibrium dynamics as an indispensable characteristic of a healthy biological system. *Integrated Physiological and Behavioral Science*, 29, 283-93.
- Pincus, S.M., Gladstone, I.M., & Ehrenkranz, R.A. (1991). A regularity statistic for medical data analysis. *Journal of Clinical Monitoring*, 7, 335-45.
- Porges, S.W. (1991). Vagal tone: An autonomic mediator of affect. In J. Barber & K.A. Dodge (Eds.), *The Development of Emotion Regulation and Dysregulation* (pp. 111-28). Massachusetts: Cambridge University Press.
- Porges, S.W. (1992). Autonomic regulation and attention. In B.A. Campbell, H. Hayne, & R. Richardson (Eds.), *Attention and Information Processing in Infants and Adults* (pp. 201-23). New Jersey: Erlbaum.
- Porges, S.W. (1995). Cardiac vagal tone: A physiological index of stress. *Neuroscience and Biobehavioral Reviews*, 19(2), 225-233.
- Randall, W.C. & Ardell, J.L. (1990). Nervous control to the heart: Anatomy and pathophysiology. In D.P. Zipes, J. Jalife (eds.). *Cardiac Electrophysiology: From Cell to Bedside*. Philadelphia: W.B. Saunders, pp. 291-299.
- Ruiz, J.M., Smith, T.W., & Uchino, B.N. (2001, March). *Differences in magnitude and duration of vagal response for high and low hostile individuals during cold stimulation*. Poster session presented at the annual meeting of the American Psychosomatic Society, Monterey, California.
- Santucci, A.K. & Friedman, B.H. (2003). *Affective responses to autonomic tasks: An idiodynamics approach*. Manuscript submitted for publication.
- Saul, J.P. (1990). Beat-to-beat variations of heart rate reflect modulations of cardiac autonomic outflow. *News in Physiological Sciences*, 5, 32-37.
- Schekelle, R.B., Gale, M., Ostfeld, A.A., & Paul, O. (1983). Hostility, risk of coronary heart disease, and mortality. *Psychosomatic Medicine*, 45, 109-14.

- Schuler, J.L.H. & O'Brian, W.H. (1997). Cardiovascular recovery from stress and hypertension risk factors. *Psychophysiology*, 34, 649-659.
- Shapiro, P.A., Sloan, R.P., Bagiella, E., Kuhl, J.P., Anjilvel, S., & Mann, J.J. (2000). Cerebral activation, hostility, and cardiovascular control during mental stress. *Journal of Psychosomatic Research*, 48, 485-491.
- Sherwood, A., Allen, M.T., Fahrenberg, J., Kelsey, R.M., Lovallo, W.R., & van Doornen, L.J.P. (1990). Methodological guidelines for impedance cardiography. *Psychophysiology*, 27, 1-23.
- Sherwood, A., Royal, S.A., Hutcheson, J.S., & Turner, J.R. (1992). Comparison of impedance cardiographic measurements using band and spot electrodes. *Psychophysiology*, 29, 734-741.
- Sloan, R.P., Bagiella, E., Shapiro, P.A., Kuhl, J.P., Chernikhova, D., Berg, J., & Myers, M.M. (2001). Hostility, sex, and cardiac autonomic control. *Psychosomatic Medicine*, 63, 434-440.
- Sloan, R.P., Shapiro, P.A., Bigger, T., Bagiella, E., Steinam, R.C., & Gorman, J.M. (1994). Cardiac autonomic control and hostility in healthy subjects. *American Journal of Cardiology*, 74, 298-300.
- Smith, T.W. (1994). Concepts and methods in the study of anger, hostility, and health. In A.W. Siegman & T.W. Smith (Eds.), *Anger, hostility, and the heart*. Hillsdale, N.J.: Lawrence Erlbaum Associates, Inc, pp. 23-43.
- Smith, T.W. & Christensen, A.J. (1992a). Hostility, health, and social contexts. In H.S. Friedman (Ed.). *Hostility, Coping, and Health*. Washington D.C., American Psychological Association, pp. 33-48.
- Smith, T.W. & Christensen, A.J. (1992b). Cardiovascular reactivity and interpersonal relations: Psychosomatic processes in social context. *Journal of Social and Clinical Psychology*, 11, 279-301.
- Smith, T.W. & Frohm, K. (1985). What's so unhealthy about hostility? Construct validity and psychosocial correlates to the Cook & Medley hostility scale. *Health Psychology*, 4, 503-520.

- Smith, T.W. & Leon, A.S. (1992). *Coronary Heart Disease: A Behavioral Perspective*. Illinois: Research Press.
- Smith, T.W. & Pope, M.K. (1990). Cynical hostility as a health risk: Current status and future directions. *Journal of Social Behavior and Personality*, 5, 77-88.
- Stein, P.K., Bosner, M.S., Kleiger, R.E., & Conger, B.M. (1994). Heart rate variability: A measure of cardiac autonomic tone. *American Heart Journal*, 127, 1376-1381.
- Stein, P.K. & Kleiger, R.E. (1999). Insights from the study of heart rate variability. *Annual Review of Medicine*, 50, 249-261.
- Suarez, E.C. & Williams, R.B. (1989). Situational determinants of cardiovascular and emotional reactivity in high and low hostile men. *Psychosomatic Medicine*, 51, 401-418.
- Suarez, E.C. & Williams, R.B. (1990). The relationship between dimensions of hostility and cardiovascular recovery as a function of task characteristics. *Psychosomatic Medicine*, 52, 558-570.
- Suls, J. & Wan, C.K. (1993). The relationship between trait hostility and cardiovascular reactivity: A quantitative review and analysis. *Psychophysiology*, 30(6), 615-626.
- Thayer, J.F., Friedman, B.H., & Borkovec, T.D. (1996). Autonomic characteristics of generalized anxiety disorder and worry. *Biological Psychiatry*, 39, 255-266.
- Tsuji, H., Larson, M.G., Venditti, F.J., Manders, E.S., Evans, J.C., Feldman, C.L., & Levy, D. (1996). Impact of reduced heart rate variability on risk for cardiac events: The Framingham Heart Study. *Circulation*, 94, 2850-2855.
- Urather, F., Neely, B.H., & Hageman (1986). Differential sympathetic-parasympathetic interactions in sinoatrial and atrioventricular junction. *American Journal of Physiology*, 250, H43.
- Weinberger, D.A. (1990). The construct validity of the repressive coping style. In: J.L. Singer, (ed). *Repression and Dissociation: Implications for Personality Theory, Psychopathology, and Health*. Chicago: University of Chicago Press, pp. 337-386.

- Willemsen, G.H.M., De Geus, E.J.C., Klaver, CHAM, Van Doornen, LJP, & Carroll, D. (1996). Ambulatory monitoring of the impedance cardiogram. *Psychophysiology*, 33, 184-193.
- Williams, R.B., Barefoot, J.C., & Shekelle, R.B. (1985). The health consequences of hostility. In Chesney, M.A. & Rosenman, R.H. (Eds.), *Anger and Hostility in Cardiovascular and Behavioral Disorders* (pp 173-185). Washington D.C.: Hemisphere.
- Williams, R.B., Haney, T.L., Lee, K.L., Kong, Y., Blumenthal, J.A., & Whalen, R.E. (1980). Type A behavior, hostility, and coronary atherosclerosis. *Psychosomatic Medicine*, 42, 539-549.

APPENDIX A
FIGURES

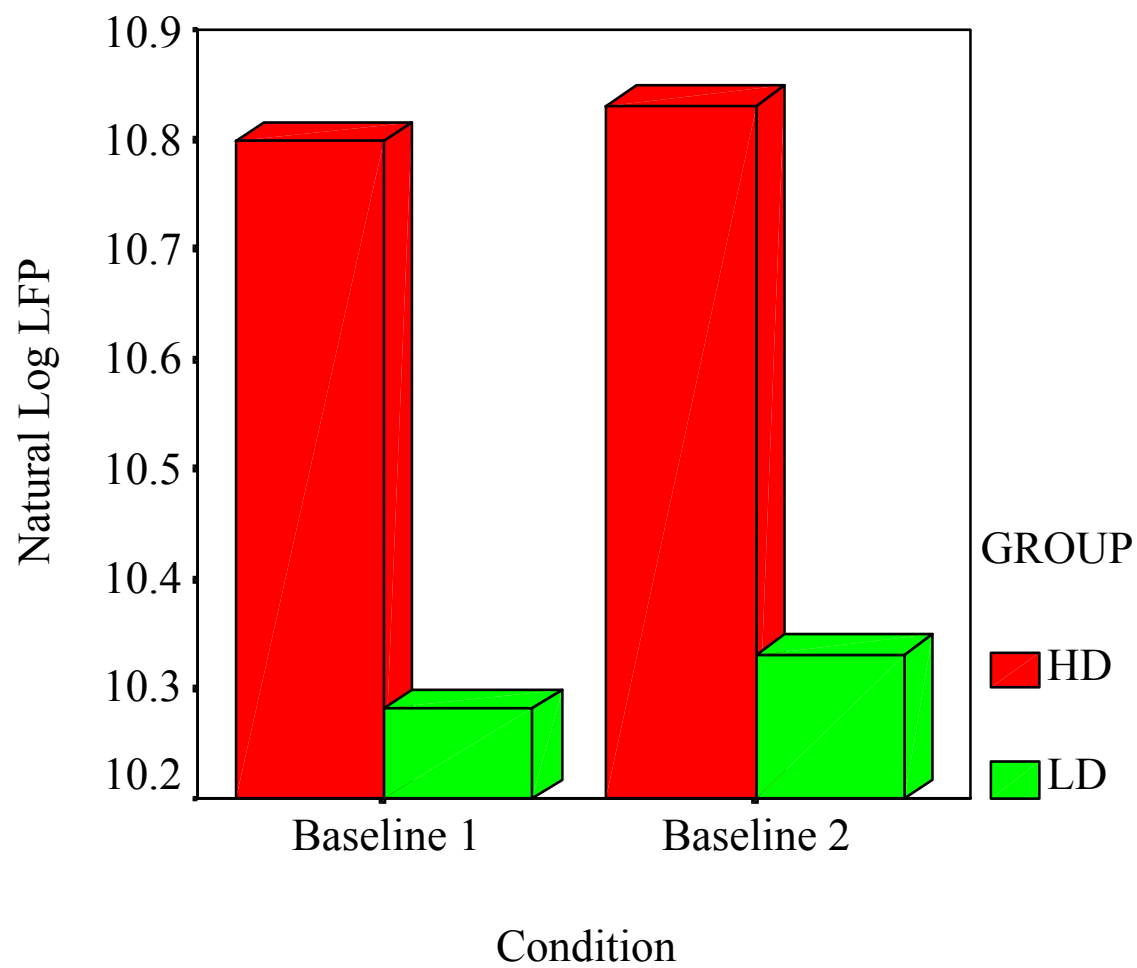


Figure A-1. Mean Low Frequency Power During Baseline Periods by Group

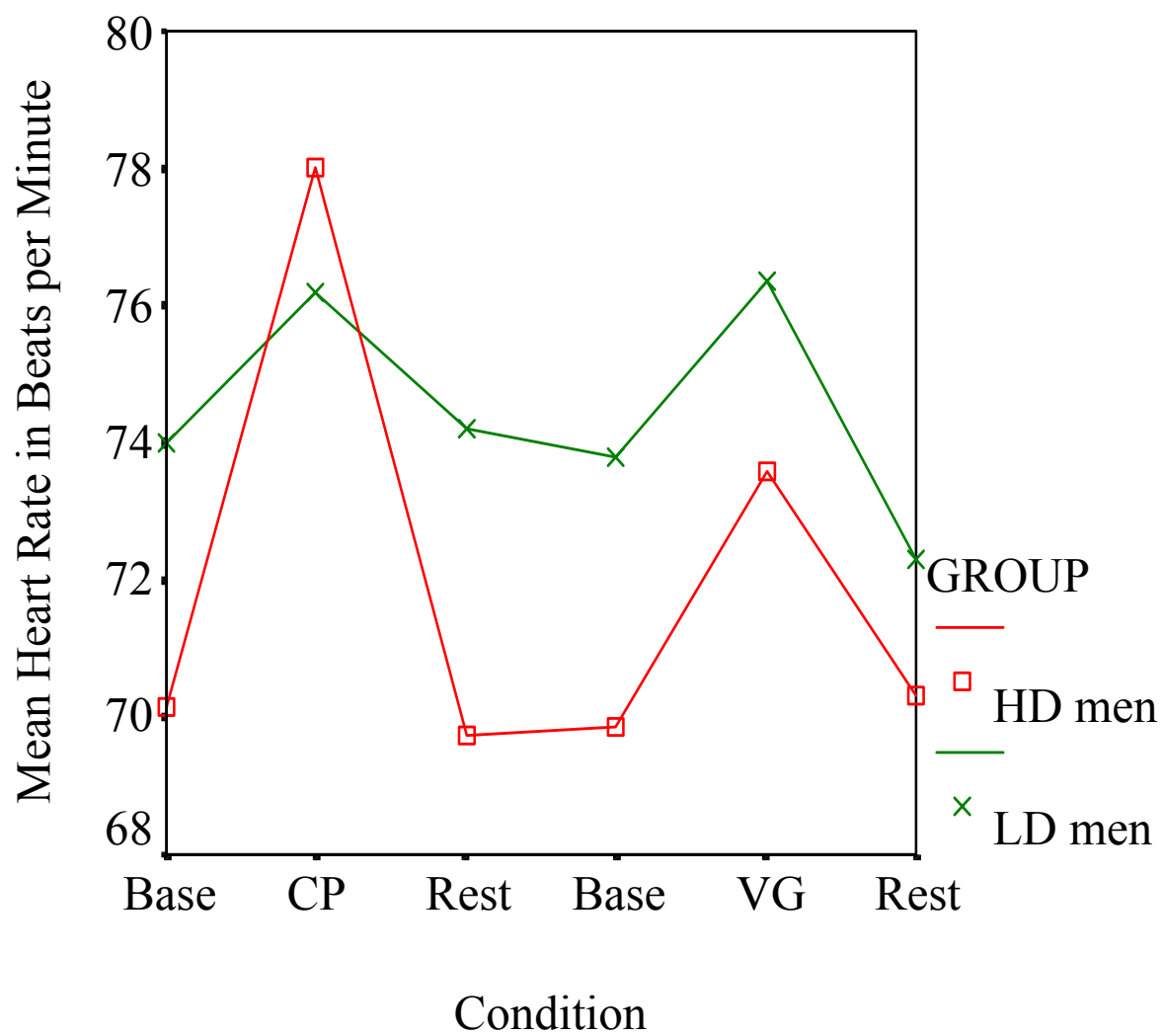


Figure A-2. Mean Heart Rate Across Conditions by Group for Men

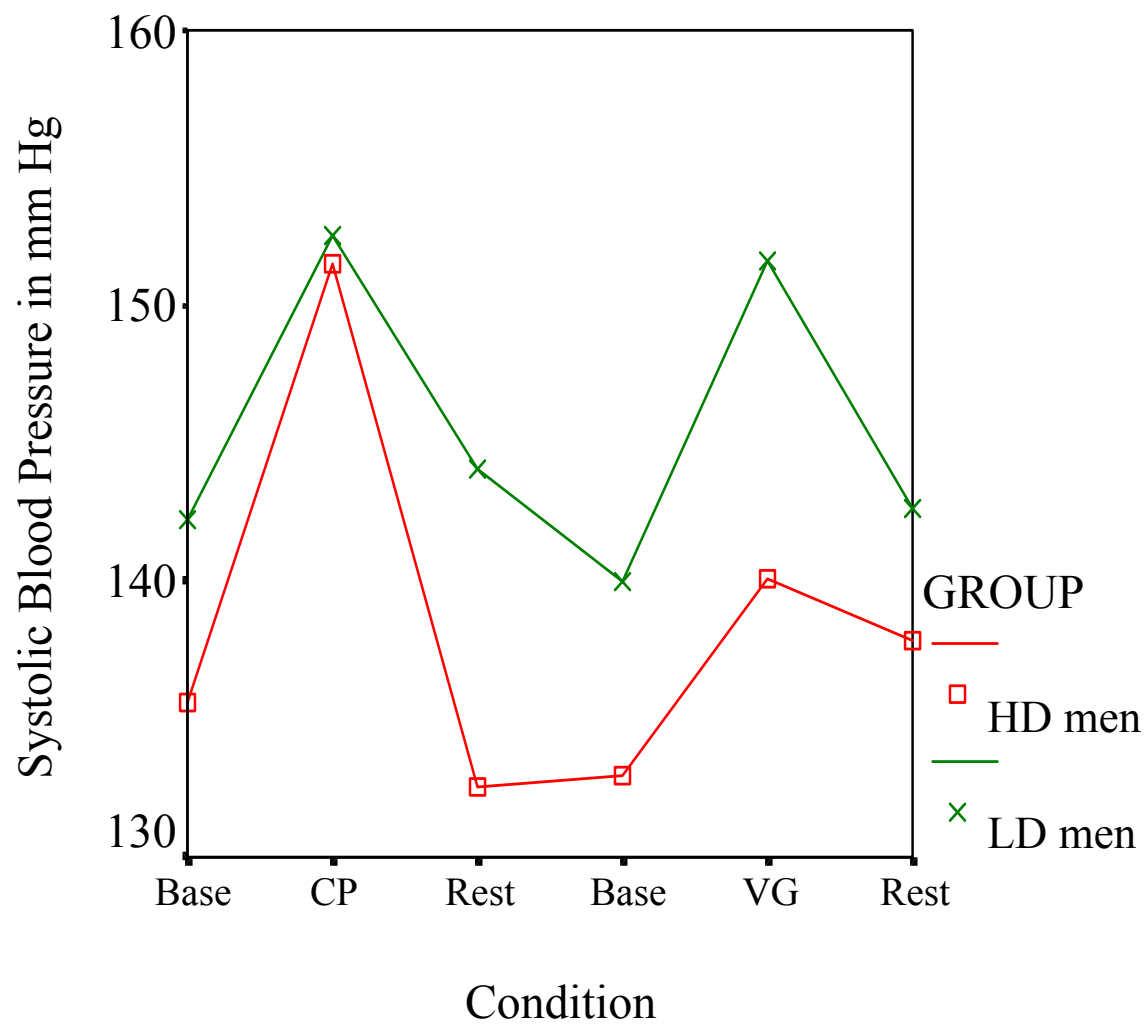


Figure A-3. Mean Systolic Blood Pressure Across Conditions by Group for Men

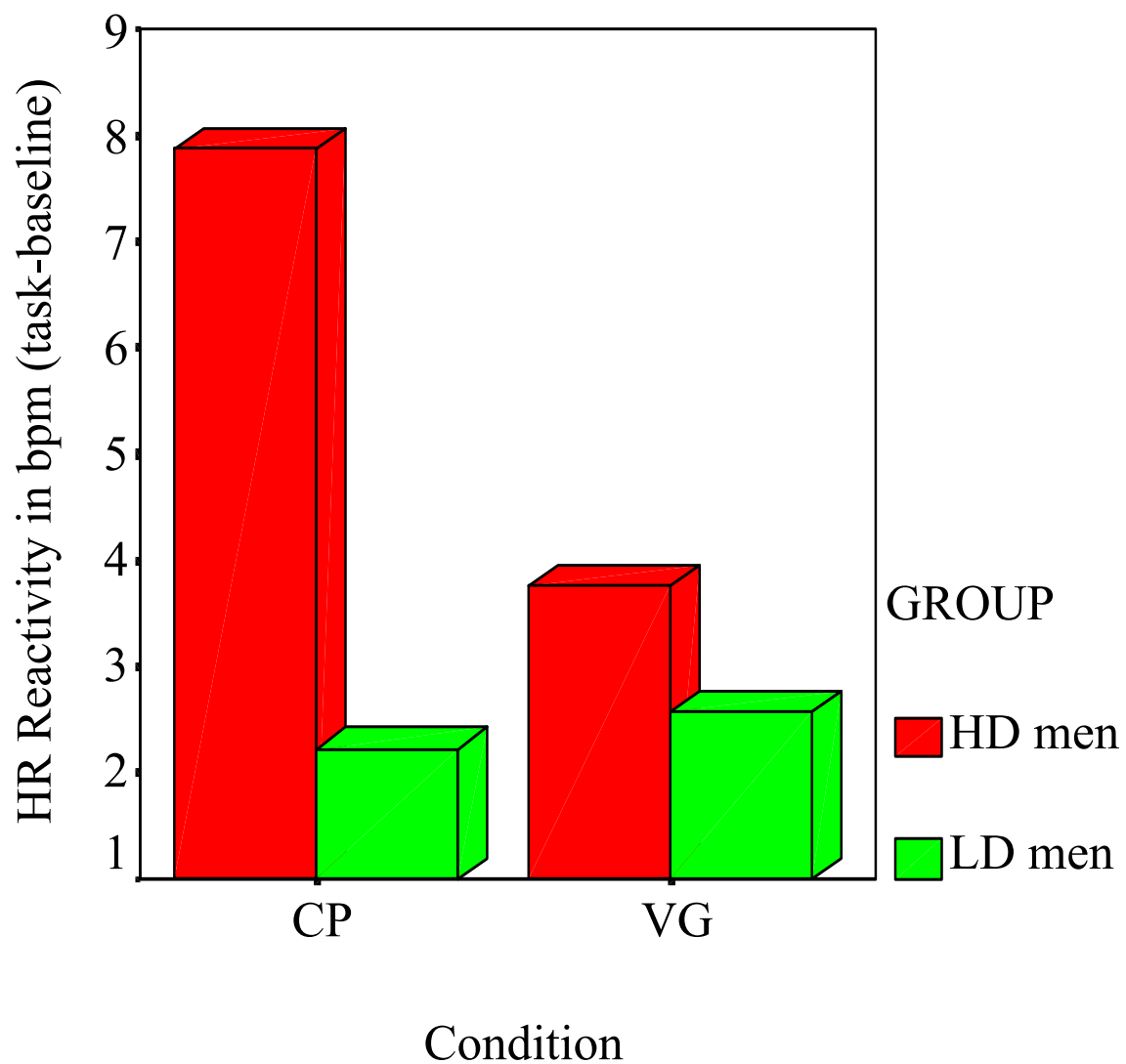


Figure A-4. Mean Heart Rate Reactivity to Cold Pressor and Video Game by Group for Men

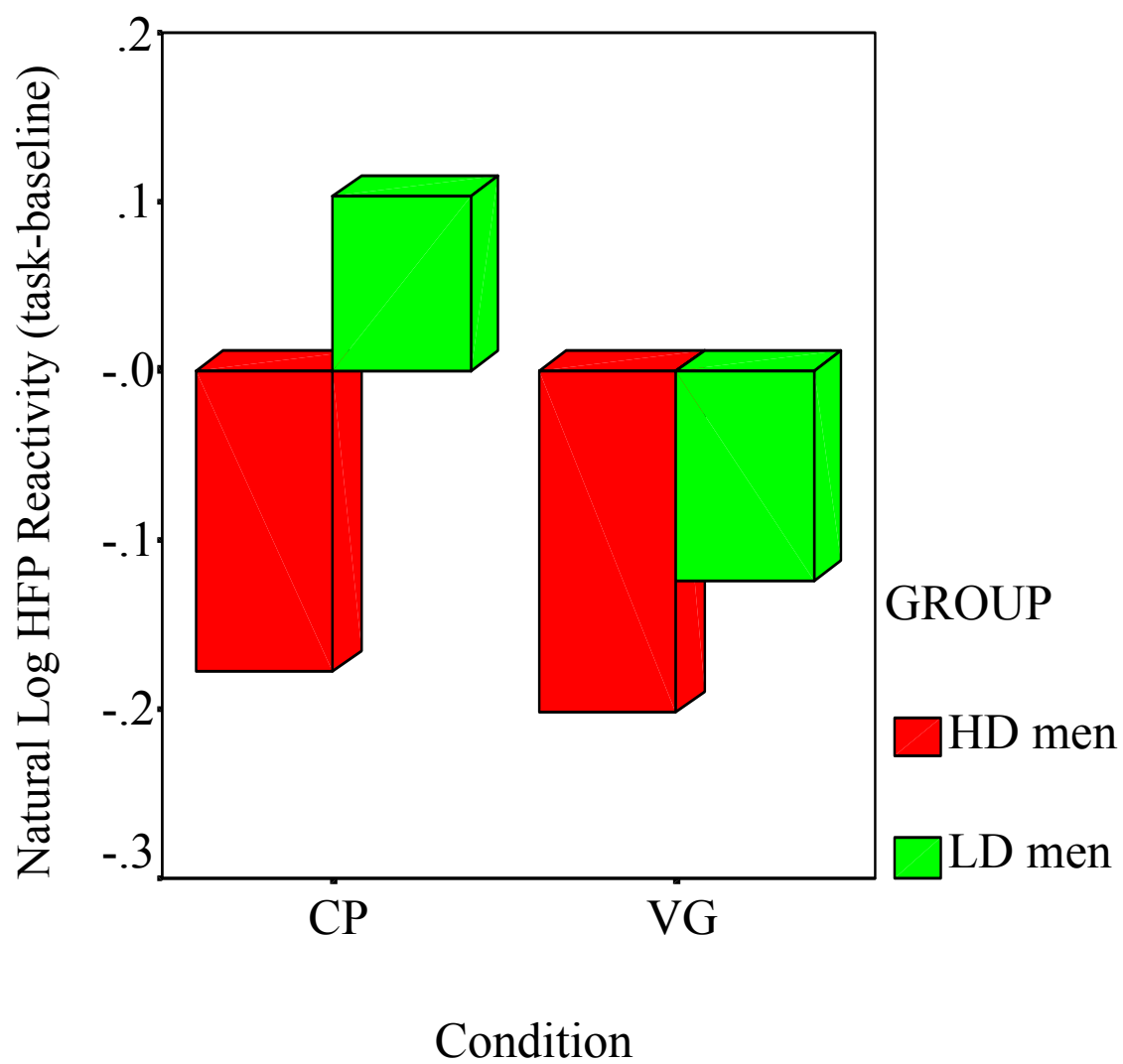


Figure A-5. Mean High Frequency Power Reactivity to Cold Pressor and Video Game by Group for Men

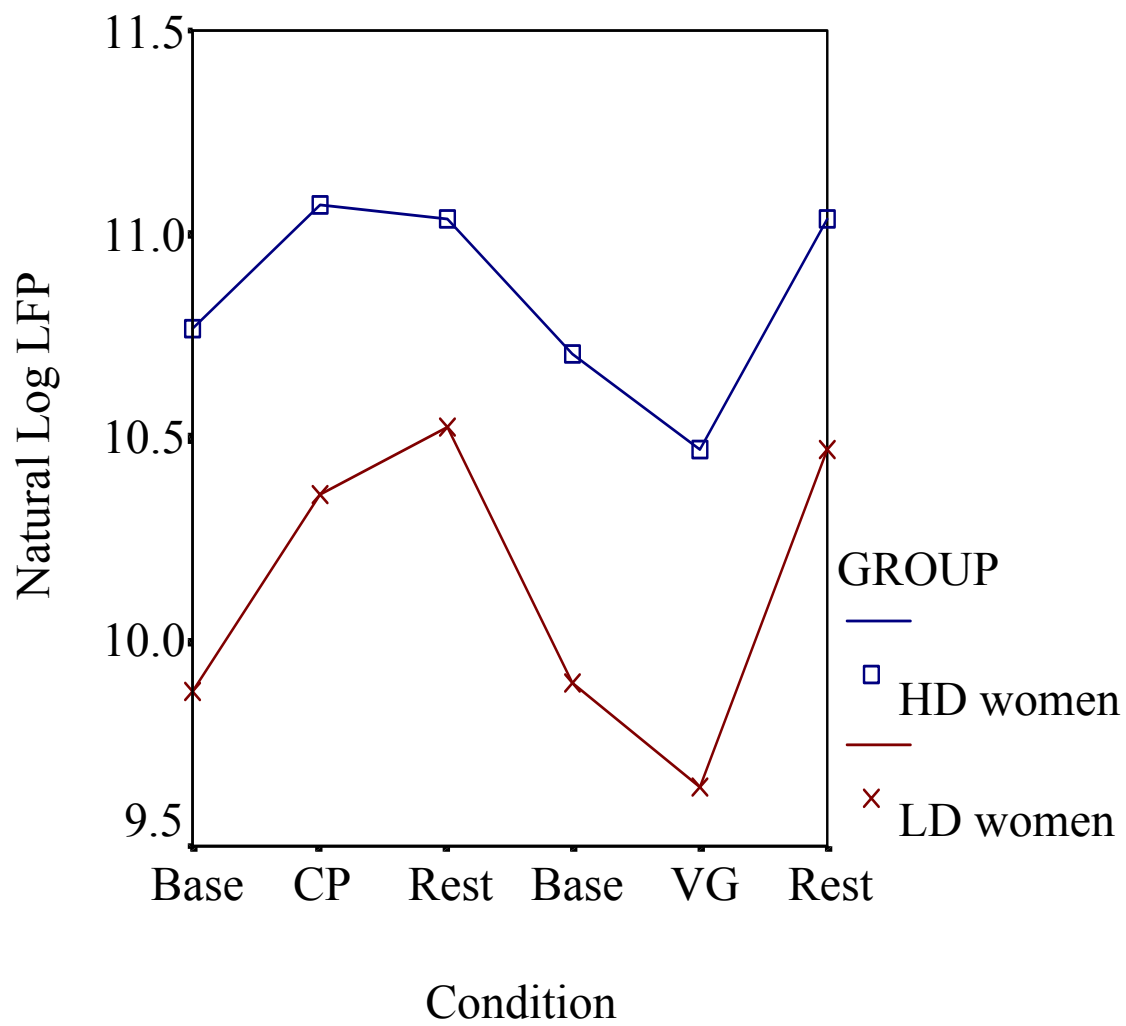


Figure A-6. Mean Low Frequency Power Across Conditions by Group for Women

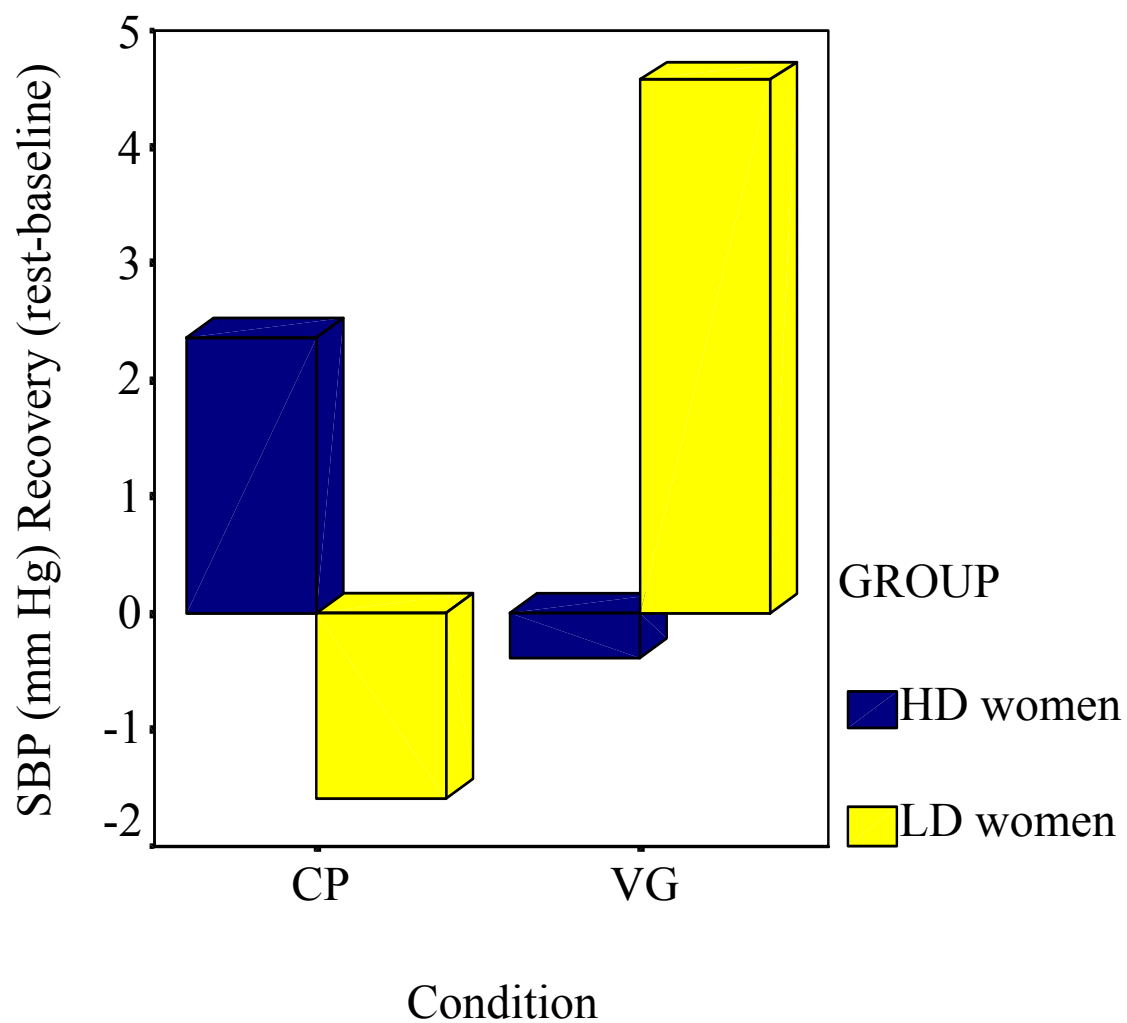


Figure A-7. Mean Systolic Blood Pressure Recovery to Cold Pressor and Video Game by Group for Women

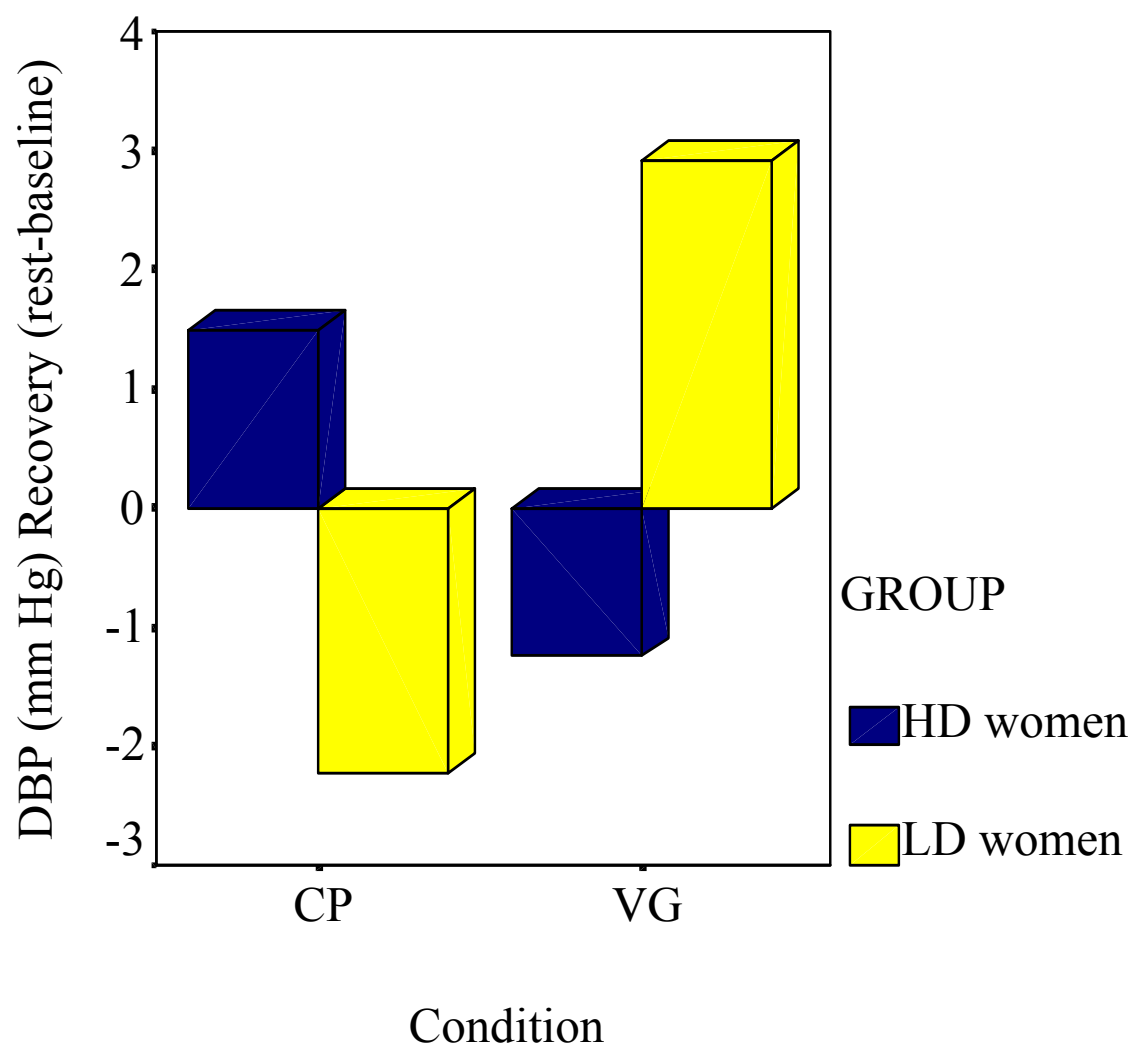


Figure A-8. Mean Diastolic Blood Pressure Recovery to Cold Pressor and Video Game by Group for Women

APPENDIX B
TABLES

	HD		HD Men		LD		LD Men	
	Women	n=16		n=15	Women	n=16		n=16
	M	SD	M	SD	M	SD	M	SD
MC	18.31	2.50	18.53	2.77	9.94	3.28	10.13	2.80
Chost	18.94	2.46	18.53	3.00	18.94	2.29	20.56	3.31
Age	19.31	1.01	19.20	0.56	19.81	1.22	19.44	2.22
BMI	22.01	2.87	23.84	4.46	22.04	2.49	25.59	4.14
Weight	131.31	19.15	168.80	34.73	130.75	14.55	180.38	37.15
Height	64.75	1.81	70.53	1.85	64.69	1.92	70.56	3.01
Caf/day	1.94	1.18	1.20	1.36	1.00	1.03	2.03	2.23
Alc/week	1.72	2.68	6.83	7.45	4.19	5.21	7.72	8.50

Table B-1. Means and Standard Deviations for Questionnaires by Group and Sex

	LD Men n=16		HD Men n=15		LD Women n=16		HD Women n=16	
	M	SD	M	SD	M	SD	M	SD
SBP baseline	142.20	19.49	135.62	17.40	128.90	18.85	127.44	11.36
SBP CP	152.55	23.23	151.48	14.04	141.99	15.31	142.44	13.13
SBP Rest	144.03	20.00	132.53	14.99	127.30	15.58	129.82	13.29
SBP baseline	140.00	17.82	132.98	18.34	127.40	17.73	129.78	11.86
SBP VG	151.59	19.00	140.11	16.88	134.52	21.14	133.24	11.94
SBP Rest	142.69	18.21	137.88	16.06	131.98	16.67	129.40	11.85
DBP baseline	82.61	15.00	76.78	11.39	73.42	12.14	72.00	7.57
DBP CP	90.01	15.73	89.06	11.32	82.62	9.60	82.85	9.59
DBP Rest	83.70	14.15	75.34	10.90	71.19	9.63	73.48	8.43
DBP baseline	80.17	11.66	76.87	13.97	71.64	11.12	73.51	7.56
DBP VG	88.19	13.45	80.87	12.22	77.36	14.03	76.08	8.52
DBP Rest	82.27	12.24	79.39	11.36	74.56	10.73	72.26	7.69
HR baseline	73.98	13.04	70.13	8.37	74.56	11.59	71.58	14.43
HR CP	76.20	13.00	78.00	10.74	77.91	10.30	76.63	12.87
HR Rest	74.21	12.36	69.74	8.24	75.41	10.76	72.95	12.97
HR baseline	73.81	14.32	69.84	7.83	74.34	11.25	71.89	13.45
HR VG	76.37	13.82	73.60	8.72	78.81	12.81	77.44	15.07
HR Rest	72.32	12.60	70.33	8.38	74.07	10.70	72.59	12.62

B-2. Means and Standard Deviations for Physiological Measures by Group and Sex

	LD Men n=16		HD Men n=15		LD Women n=16		HD Women n=16	
	M	SD	M	SD	M	SD	M	SD
SBP Reactivity to CP	10.35	15.39	15.86	10.92	13.09	10.33	15.00	10.70
SBP Recovery to CP	8.51	15.70	18.95	8.09	14.68	10.05	12.62	6.72
SBP Reactivity to VG	11.60	10.91	7.13	9.27	7.12	9.94	3.45	6.99
SBP Recovery to VG	8.91	9.79	2.23	6.89	2.55	7.26	3.84	5.68
DBP Reactivity to CP	7.40	11.04	12.28	8.68	9.21	7.40	10.85	8.79
DBP Recovery to CP	6.30	10.36	13.72	7.73	11.44	6.66	9.37	6.09
DBP Reactivity to VG	8.02	7.96	3.99	6.86	5.72	6.28	2.57	4.96
DBP Recovery to VG	5.92	6.97	1.48	5.08	2.81	5.95	3.82	4.32
HR Reactivity to CP	2.22	3.21	7.87	6.53	3.35	4.30	5.05	5.13
HR Recovery to CP	2.00	3.15	8.26	6.35	2.51	4.47	3.68	4.03
HR Reactivity to VG	2.57	5.97	3.76	3.78	4.48	5.66	5.55	5.17
HR Recovery to VG	4.05	6.34	3.27	4.76	4.75	7.36	4.84	5.41

B-3. Mean Reactivity and Recovery Scores by Group and Sex

APPENDIX C

COOK-MEDLEY SCALE

Directions: If a statement is true or mostly true, as pertaining to you, circle the letter T. If a statement is false or usually not true about you, circle the letter F. Try to give a response to every statement.

- | | |
|---|--------|
| 1. When I take a new job, I like to be tipped off on who should be gotten text to. | T F |
| 2. When someone does me wrong I feel I should pay him back if I can, just for the principle of the thing. | T F |
| 3. I prefer to pass by school friends, or people I know but have not seen for a long time, unless they speak to me first. | T F |
| 4. I have often had to take orders from someone who did not know as much as I did. | T F |
| 5. I think a great many people exaggerate their misfortunes in order to gain sympathy and help of others. | T F |
| 6. It takes a lot of argument to convince most people of the truth. | T F |
| 7. I think most people would lie to get ahead. | T F |
| 8. Someone has it in for me. | T F |
| 9. Most people are honest chiefly through the fear of getting caught. | T F |
| 10. Most people will use somewhat unfair means to gain profit or an advantage rather than to lose it. | T F |
| 11. I commonly wonder what hidden reason another person may have for doing something nice for me. | T F |

- | | | |
|--|---|---|
| 12. It makes me impatient to have people ask my advice or otherwise interrupt me when I am working on something important. | T | F |
| 13. I feel that I have often been punished without cause. | T | F |
| 14. I am against giving money to beggars. | T | F |
| 15. Some of my family have habits that bother and annoy me very much. | T | F |
| 16. My relatives are nearly all in sympathy with me. | T | F |
| 17. My way of doing things is apt to be misunderstood by others. | T | F |
| 18. I don't blame anyone for trying to grab everything he can get in this world. | T | F |
| 19. No one cares much what happens to you. | T | F |
| 20. I can be friendly with people who do things which I consider wrong. | T | F |
| 21. It is safer to trust nobody. | T | F |
| 22. I do not blame a person for taking advantage of someone who lays himself open to it. | T | F |
| 23. I have often felt that strangers were looking at me critically. | T | F |
| 24. Most people make friends because friends are likely to be useful to them. | T | F |
| 25. I am sure I am being talked about. | T | F |
| 26. I am likely not to speak to people until they speak to me. | T | F |
| 27. Most people inwardly dislike putting themselves out to help other people. | T | F |

- | | | |
|--|---|---|
| 28. I tend to be on guard with people who are somewhat more friendly than I had expected. | T | F |
| 29. I have sometimes stayed away from another person because I feared saying or doing something that I might regret afterwards. | T | F |
| 30. People often disappoint me. | T | F |
| 31. I like to keep people guessing what I'm going to do next. | T | F |
| 32. I frequently ask people for advice. | T | F |
| 33. I am not easily angered. | T | F |
| 34. I have often met people who were supposed to be experts who were no better than I. | T | F |
| 35. I would certainly enjoy beating a crook at his own game. | T | F |
| 36. It makes me think of failure when I hear of the success of someone I know well. | T | F |
| 37. I have at times had to be rough with people who were rude or annoying. | T | F |
| 38. People generally demand more respect for their own rights than they are willing to allow for others. | T | F |
| 39. There are certain people whom I dislike so much that I am inwardly pleased when they are catching it for something they have done. | T | F |
| 40. I am often inclined to go out of my way to win a point with someone who has opposed me. | T | F |
| 41. I am quite often not in on the gossip and talk of the group I belong to. | T | F |

42. The man who had the most to do with me when I was a child (such as my father, step-father, etc. . .) was very strict with me. T F
43. I have often found people jealous of my good ideas just because they had not thought of them first. T F
44. When a man is with a woman he is usually thinking about things related to sex. T F
45. I do not try to cover up my poor opinion or pity so that he/she won't know how I feel. T F
46. I have frequently worked under people who seem to have things arranged so that they get credit for good work but are able to pass off mistakes onto those under them. T F
47. I strongly defend my own opinions as a rule. T F
48. People can pretty easily change me even though I thought that my mind was already made up on a subject. T F
49. Sometimes I am sure that other people can tell what I am thinking. T F
50. A large number of people are guilty of bad sexual conduct. T F

APPENDIX D
MARLOWE-CROWNE SCALE

Listed below are a number of statements concerning personal attitudes and traits. Read each item and decide whether the statements are true or false as it pertains to you.

- | | | |
|---|---|--|
| T | F | 1. Before voting I thoroughly investigate the qualifications of all the candidates. |
| T | F | 2. I never hesitate to go out of my way to help someone in trouble. |
| T | F | 3. It is sometimes hard for me to go on with my work if I am not encouraged. |
| T | F | 4. I have never intensely disliked someone. |
| T | F | 5. On occasion I have had doubts about my ability to succeed in life. |
| T | F | 6. I sometimes feel resentful when I don't get my way. |
| T | F | 7. I am always careful about my manner of dress. |
| T | F | 8. My table manners at home are as good as when I eat out in a restaurant. |
| T | F | 9. If I could get into a movie without paying and be sure I was not seen, I would probably do it. |
| T | F | 10. On a few occasions, I have given up doing something because I thought too little of my ability. |
| T | F | 11. I like to gossip at times. |
| T | F | 12. There have been times when I felt like rebelling against people in authority even though I knew they were right. |
| T | F | 13. No matter who I'm talking to, I'm always a good listener. |
| T | F | 14. I can remember "playing sick" to get out of something. |
| T | F | 15. There have been occasions when I took advantage of someone. |

- | | | |
|---|---|--|
| T | F | 16. I'm always willing to admit it when I make a mistake. |
| T | F | 17. I always try to practice what I preach. |
| T | F | 18. I don't find it particularly difficult to get along with loud-mouthed, obnoxious people. |
| T | F | 19. I sometimes try to get even, rather than forgive and forget. |
| T | F | 20. When I don't know something I don't at all mind admitting it. |
| T | F | 21. I am always courteous, even to people who are disagreeable. |
| T | F | 22. At times I have really insisted on having things my own way. |
| T | F | 23. There have been occasions when I felt like smashing things. |
| T | F | 24. I would never think of letting someone else be punished for my wrongdoings. |
| T | F | 25. I never resent being asked to return a favor. |
| T | F | 26. I have never been irked when people expressed ideas very different from my own. |
| T | F | 27. I never make a long trip without checking the safety of my car. |
| T | F | 28. There have been times when I was quite jealous of the good fortune of others. |
| T | F | 29. I have almost never felt the urge to tell someone off. |
| T | F | 30. I am sometimes irritated by people who ask favors of me. |
| T | F | 31. I have never felt that I was punished without cause. |
| T | F | 32. I sometimes think when people have a misfortune they only got what they deserved. |
| T | F | 33. I have never deliberately said something that hurt someone's feelings. |

APPENDIX E

INFORMED CONSENT

VIRGINIA POLYTECHNIC INSTITUTE AND STATE UNIVERSITY

Informed Consent for Participation in Investigative Projects

Title of Project: Autonomic Characteristics of Personality.

Investigator: Elizabeth J. Vella, B.A.

I. The Purpose of this Project

The present study will be conducted to determine the cardiovascular patterning associated with personality. The investigator is interested in locating 64 participants, 32 men and 32 women, who rate high on two personality scales.

II. Procedures

Participation in this study consists in two phases. During phase one, participants will complete two personality scales, a task that should take no more than 15-20 minutes. Depending on scores obtained from the personality scales, participants from phase one may be invited by telephone to participate in phase two. Participation in phase two will last approximately 45 minutes. Each participant will be exposed to a race-car video game and the hand-cold pressor task (placing non-dominant hand wrist-deep in a bucket of ice water twice for 1.5 minutes) while continuous physiological measurements are assessed via electrodes placed on the torso and a blood pressure monitoring device. All experimental sessions will take place at the Laboratory for the Study of Human Thought and Action (LSHTA), located at 922 University City Blvd behind across from the U.S. Post Office and directly behind Macado's.

III. Risks

Participants are exposed to minimal risks in this study. The equipment for recording cardiovascular activity is noninvasive and relatively comfortable. However, the cold pressor task will provide moderate discomfort.

IV. Benefits of this Project

The benefits of the study are twofold. First, participants will be able to view their results and are likely to benefit from a greater knowledge of their physiological functioning (e.g., blood pressure). The second benefit is with respect to the scientific community. The conduction of this study is intended to provide useful information

regarding the hostility construct and how it may interact with defensiveness in predicting CV reactivity and recovery to laboratory stressors. No promise or guarantee of benefits has been made to encourage participation in this study.

V. Extent of Anonymity and Confidentiality

Participants will be assigned a code number appearing on their data sheets and in a corresponding computer file. Only research assistants and myself will have access to the coded data.

VI. Compensation

Participants may earn up to two extra credit points in a psychology course for participation in this study. If an instructor offers extra credit through research participation, he/she must also provide alternate ways for students to earn extra credit.

VII. Freedom to Withdraw

Participants are free to withdraw from this study at any time without penalty. Participants will still receive extra credit for participation irrespective to the decision to withdraw. Participants are free to refuse answering any questions or responding to experimental situations without any penalty whatsoever.

VIII. Approval of Research

This research project has been approved, as required, by the Institutional Review Board for Research Involving Human Subjects at Virginia Polytechnic Institute & State University, by the Department of Psychology.

IX. Participant's Responsibilities

I voluntarily agree to participate in this study. I have the following responsibilities:

1. Complete a health screening questionnaire.
2. Refrain from alcohol consumption for 24 hours prior to participation.
3. Refrain from strenuous exercise for 6 hours prior to participation.

X. Participant's Permission

I have read and understood the Informed Consent and conditions of this project. I have had all my questions answered. I hereby acknowledge the above and give my voluntary consent for participation in this project.

If I participate, I may withdraw at any time without penalty. I agree to abide by the rules of this project.

Signature

Date

Should I have any questions about this research or its conduct, I may contact:

Elizabeth J. Vella, B.A.

231-4428

Phone

Bruce H. Friedman, Ph.D.

231-9611

Phone

Dr. David Moore
Chair, IRB
CVM Phase II

231-4991

Phone

APPENDIX F
SUBJECT SCREENING QUESTIONNAIRE

Subject ID # _____

Age _____ Sex _____ Height _____ Weight _____ Handedness _____

Address _____

Phone _____ Social Security _____

Do you smoke? Yes _____ No _____

If yes, how many cigarettes do you smoke daily? _____

Have you smoked in the last 12 hours? _____

Average daily caffeine consumption (approximate number of cups of coffee, tea, or caffeinated sodas) _____

Have you consumed any caffeinated beverages in the last 12 hours? _____

When was the last time that you ate and what did you consume? _____

Average weekly alcohol consumption (approximate number of alcoholic beverages consumed weekly) _____

Have you consumed any alcoholic beverages in the last 12 hours? _____

Do you take any medication (prescription or over the counter) regularly? _____

If so, please describe the medication _____

Do you have any known medical problems that might affect your participation in this study? Examples: heart conditions, high or low blood pressure, bouts of dizziness or fainting spells, diabetes, asthma, or neurological disorders? If so, explain _____

If you exercise regularly, please indicate the type and average weekly frequency of the exercise: _____

Women: When was the 1st day of your last menstrual period? _____

VITA**Elizabeth J. Vella**

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 Blacksburg, VA 24060-0436. Phone: (540) 231-3630. Fax: (540) 231-3652
 E-mail: evella@vt.edu

Education

Virginia Polytechnic Institute & State University
 Department of Psychology
 Master of Science Degree

May 2003

Academic Appointments

Spring 2003

Graduate Teaching Assistant, Department of Psychology, Virginia Polytechnic Institute
 Courses: Nervous Systems & Behavior and Physiological Psychology

Fall 2002

Graduate Teaching Assistant, Department of Psychology, Virginia Polytechnic Institute
 Course: Research Methods

Summer 2001-2002

Graduate Research Stipend, Department of Psychology, Virginia Polytechnic Institute
 Fall 2000-Spring 2002

Graduate Teaching Assistant, Department of Psychology, Virginia Polytechnic Institute.
 Course: Introductory Psychology

Spring 1999

Volunteer Faculty, Department of Psychology, Sonoma State University
 Course: Neuroscience

Spring 1998

Teaching Assistant for advanced theories, methods, and issues in psychology.

Fall 1996

Statistics teaching intern for psychology majors

Research Interests: The usage of peripheral measures to investigate the psychophysiology of emotion and personality, with a particular emphasis on health and disease.

Publications and Presentations

Vella, E.J., & Friedman, B.H. (2003, March). *The autonomic characteristics of defensive hostility*. Poster session presented at the 61st annual meeting of the American Psychosomatic Society, Phoenix, AZ.

Vella, E.J., Friedman, B.H., Lozier, L., & Glaskey, G. (2003, March). *The autonomic correlates of defensive hostility*. Poster session presented at the 19th annual research symposium of the Graduate Student Association, Blacksburg, VA.

Vella, E.J., Irvin, M.D., Solle, J., Berendt, S., & Ramirez, E.E. (1999). The effects of music on mood and perception of a visual stimulus. *Psi Chi Journal of Undergraduate Research*, 4(3), 101-105.

Vella, E.J., Irvin, M.D., Solle, J., & Ramirez, E.E. (1998, April). *Effects of music on mood using MAACL-R on undergraduate students*. Poster session presented at the annual meeting of the Western Psychological Association, Albuquerque, NM.

Manuscript in Preparation

Vella, E.J., & Friedman, B.H. *The autonomic characteristics of defensive hostility*.

Honors and Awards

1997-98, Psi Chi Vice President, Sonoma State University

1997-98, Hewlett-Packard Scholarship, Sonoma State University

Professional Memberships

American Psychological Society

American Psychosomatic Society

Society of Behavioral Medicine

Society for Psychophysiological Research