INTRODUCTION

A substantial amount of research has attributed the psychological traits of anger and hostility as early risk factors of coronary heart disease (CHD) (see Miller, Smith, Turner, Guijaro, & Hallet, 1996 for a review). 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/angry individuals than encounters permitting anger expression (Brosschot & Thayer, 1998). 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 (e.g., Dembroski, MacDougal, Williams, Haney, & Blumenthal, 1985; Atchison & Condon, 1993).

Whereas many investigations suggest this combination of anger inhibition (often termed 'anger-in'; AI) paired with hostility to contribute to significant aspects of cardiovascular reactivity (CVR) to stressors likely to give rise to CHD over time (e.g., Helmers & Krantz, 1996), other studies have found that the combination of anger *expression* (often termed 'anger-out'; AO) and hostility to be particularly coronary pathogenic (e.g., Dembroski, MacDougall, Costa, & Grandits, 1989). Further, some findings suggest that anger expression alone is a better predictor of CHD progression than the combination of hostility and anger expression (e.g., McDermott, Ramsay, & Bray, 2001).

In view of the complexity of the relationship among these traits and CHD, replete with inconsistent findings, many investigators have focused their attention at

disentangling the various modifying influences in an effort to gain a clearer perspective on the mechanisms underlying this disease progression in hostile/angry individuals. One important development to this end is the discovery of trait by situation interactions, whereby hostile individuals exhibit coronary prone CVR to laboratory stressors mainly in the context of harassment or some form of interpersonal provocation (e.g., Suls & Wan, 1993). The basic premise behind such research investigations concerns the notion that in order to elicit significant coronary prone CVR to lab stressors, hostile individuals need to be exposed to some form of an anger-provoking situation.

Previous findings have indicated that hostile individuals do not exhibit significant CVR to standard lab stressors in the absence of harassment when compared to those scoring low on hostility (e.g., Suarez & Williams, 1989; Suarez et. al., 1998). Further, Vella and Friedman (2005) found non-significant differences in the context of cardiovascular (CV) recovery from these stressors as a function of hostility.

In addition to the potentially critical moderating influence of harassment in the relationships among anger, hostility, and CV recovery, assessments of the ability to evaluate the source of anger provocation may provide insight into another situational influence that modifies the recovery process. The inability to express anger following provocation among hostile individuals may attenuate CV recovery compared to those rating low on hostility (Brosschot & Thayer, 1998).

Another study reported that gender moderates anger expression following provocation and CV recovery, whereby men but not women recover faster when given the opportunity to express their anger (Lai & Linden, 1992). The focus of the current study examines the effects of dispositional anger on CV responses to lab stressors with

and without harassment in men. The design of the current study allows for the testing of main effects of anger as well as the interactive effects of this trait and situations that may help elucidate the predictors of CVR and recovery to lab stressors.

The theoretical basis for the present study concerns several salient issues. First, hostility has been characterized as a multifaceted construct (e.g., Smith, 1994). Accordingly, the relationship of hostility assessment with CVR is discussed below. Next, the influence of anger management style on CV activity will be described to validate the study of this construct in the context of anger out (AO) as opposed to hostility in the current investigation. Finally, a description of the relationships among autonomic nervous system (ANS) activity, anger, and CHD will provide a rationale for the dependent measures used in this study.

A couple of key methodological concerns are also offered. The issues of gender differences in this literature will be discussed to substantiate the use of only men in the current study. A closer examination of harassment in this literature is also important to support the use of such procedures. Finally, it is also essential to discuss issues related to CV reactivity and recovery, since they both may be critical in the disease processes related to the constructs of interest for this investigation. These issues are outlined below, followed by details of the present study.

Hostility Assessment

The Cook & Medley Hostility Scale (Ho) (1954) is perhaps the most widely used measure of hostility in the literature involving CVR to stressors. Further, the Ho scale has been found to provide a stronger association to the severity of CHD among patients undergoing coronary angiography than the association between Type A behavior

assessments and CHD in the same sample (Williams et al., 1980). Hostile individuals assessed through this scale may create a self-fulfilling prophecy by not only responding to everyday stressors with heightened CV reactivity, but also reinforcing negative interactions with other people via a cynical attitude, thereby increasing the probability of subsequent stressful interactions (Smith & Pope, 1990).

Numerous studies have established a relationship between the Ho scale and coronary dysfunction. Barefoot, Dahlstrom & Williams (1983) reported that physicians scoring high on the Ho scale in medical school were approximately six times more likely to be diagnosed with CHD 25 years later than physicians scoring low 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 18-30 yrs of age who rated high 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 low on the scale (Iribareen et al., 2000). Another study of 424 angiography patients revealed that 70% of those with Ho scores above 10 had clinically significant CHD (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 CVR to laboratory stress may be associated with increased risk of subsequent CV morbidity and/or mortality (e.g., Krantz & Manuck, 1984), a host of studies have investigated the potential relationship between the Ho scale and CVR 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, CVR 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 Ho scale was initially devised to identify instructors experiencing difficulty in establishing a rapport with their students (Cook & Medley, 1954), so it is bound to tap into areas outside the hostility construct and introduce noise in the relationship between 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 (Barefoot, Dodge, Perterson, 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 (Helmers, et. al., 1993), and CV responses to lab stressors (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.

Defensiveness. 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 CV patterning that ultimately contributes to the development of CHD (Jorgenson, Abdul-Karim, Kahan, & Frankowski, 1995; Helmers & Krantz, 1996).

The combination of trait hostility and defensiveness has been shown to provide a more accurate depiction of the hostility-CHD relationship than the Ho scale alone (e.g., Helmers et al., 1995). Researchers in the *defensive hostility* literature have tended to use the Marlowe-Crowne Social Desirability Scale (MC) to assess the tendency to inhibit anger. The MC Scale 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 recent study in this literature found that defensively hostile men and men rating low on both constructs exhibited significant HR reactivity to a cold pressor task in comparison to men who scored high on hostility but low on defensiveness (Vella & Friedman, 2005). Similar results were found from a study in which defensively hostile and low defensive, low hostile men exhibited increased CVR to a cold pressor task in comparison to men scoring high on hostility but low on defensiveness (Mente & Helmers, 1999).

The finding that defensively hostile men show increased CVR in comparison to those scoring high on hostility, but low on defensiveness, is congruent with the 'approach-avoidance' model of defensive hostility. However, the finding that those scoring low on both constructs display similar CV patterning to defensively hostile individuals is perplexing. One possibility is that low hostile, low defensive individuals are not used to encountering stressful events in daily life, but nonetheless tend to display enhanced CVR to laboratory stressors due to the novelty of the experience. Thus, lab CVR in these individuals may not reflect the typical daily scenario.

Another intriguing finding from Vella and Friedman (2005) concerns an interaction between hostility and defensiveness in the context of high frequency power (HFP), a measure of cardiac vagal activity. Men scoring low on both constructs showed significantly more HFP suppression to a cold pressor task and a video game in comparison to purely hostile men who scored high on hostility but low on defensiveness. The vagal patterning by men scoring low on these constructs is illustrative of flexible physiological responsiveness because they resumed a functional recovery of activity following stressor removal, whereas the vagal patterning of hostility and defensiveness was more rigid across tasks. The autonomic flexibility displayed by men scoring low on these constructs is reflective of adaptive organismic responding (Friedman & Thayer, 1998; Thayer & Friedman, 2002).

One potential problem in the defensive hostility literature concerns the notion that the MC scale assesses behaviors unrelated to the suppression of angry feelings. A more direct measure of anger disposition can be achieved through use of the Spielberger Anger Expression Scale (Spielberger et al., 1985). The experience and expression of anger

merits a closer assessment in the context of hostility and CV activity in an effort to better understand this area of investigation.

Anger

Spielberger et al (1985) defined anger as an emotional state that consists of feelings varying in intensity from minor irritation and annoyance to fury and rage. Variation in intensity of anger is referred to as 'state anger', whereas the frequency in which anger is experienced has been referred to as 'trait anger'. Feelings of anger can be either expressed or suppressed. Conscious suppression of anger has been distinguished by Spielberger as a construct known as 'Anger-In' (AI), reflecting the degree to which an individual typically inhibits the expression of anger, whereas the expression of anger is referred to as 'Anger-Out' (AO). Spielberger used this conceptualization to distinguish AI from the subtle psychodynamic unconscious process of anger repression. The characterization of AI and AO are intended to represent enduring processes that serve as personality traits, typifying individual tendencies regarding anger over time and reflecting the degree to which an individual engages in aggressive behavior when motivated by angry feelings.

The constructs of anger and hostility have often been treated synonymously in behavioral medicine research due to their overlapping qualities, particularly in the context of CVR. However, the constructs have important theoretical distinctions with empirical implications. Anger is oftentimes defined in terms of affect, ranging from minor annoyance to rage. Hostility has typically been conceptualized more broadly as including affect in addition to a strong cognitive component, reflecting negative attitudes toward others, such as distrust (Smith, 1994). Further, the emotions underlying hostility may be

more complex than anger, intertwined with cognitions such as cynicism, resentment, vengeance, and alienation.

Hostility, anger, and anger expression represent related aspects of what Speilberger called the AHA (Anger-Hostility-Aggression) Syndrome (Speilberger et al., 1985). These separable constructs (e.g., a person may feel angry toward another without cynically hostile cognitions or aggressive behavior) are nonetheless mutually and reciprocally reinforcing aspects of Speilberger's syndrome. A hostile attitude increases the probability of angry feelings, which in turn increases the probability of aggressive behavior. Further, this syndrome is not strictly unidirectional because aggressive behaviors can reinforce angry feelings and feed into cynically hostile cognitions.

Under conditions of harassment and social evaluation, the combination of hostility and AO has been found to produce the greatest BP reactivity (Burns & Katkin, 1993). However, in a study that did not involve harassment, subjects displaying a conflicting combination of anger expression style and hostility (e.g., high AO, low hostility) showed the most pronounced SBP responses to public speaking and mental arithmetic (Bongard, al'Absi, & Lovallo, 1998). Further, subjects showing these 'conflicted' combinations of anger expression and hostility exhibited higher HR reactivity to the tasks compared to the 'matching' combinations of being high or low on both constructs, respectively. These results suggest that in the absence of harassment, individuals with a 'matching' combination of anger and hostility (e.g., high AO, high hostility) are actually the least reactive in the context of HR and BP responses to lab tasks, thereby reflecting a non-conflicted emotional-behavioral style; in the context of rating high in both anger expression and hostility, subjects may require elicitation of

hostile cognitions (e.g., being treated unfairly by others) in order to observe significant increases on these CV parameters.

AO in and of itself may be associated with exaggerated HR and BP reactivity to lab stress, particularly during harassment or some form of anger induction (e.g., Siegman & Snow, 1997; Suchday & Larkin, 2001). Engebretson, Matthews, and Scheier (1989) found that AO men displayed larger systolic responses to harassment and no harassment conditions in comparison to AI men. Burns and Katkin (1993) found that men scoring high exclusively on AO showed the most BP responses to a reaction time task only when harassment was employed; however, their non-harassment condition included an element of social evaluation and so may not have served as a proper control.

Engebretson et al. (1989) proposed a 'matching hypothesis' by involving situational manipulations to test 'person X situation' interactions related to anger management style. Results of this study indicated that AO men exposed to an anger inhibition condition following harassment displayed attenuated recovery compared to such individuals exposed to an anger expression condition. The theoretical orientation behind this study suggests that situations tailored to personality are conducive to facilitated CV recovery from stress, whereas situations requiring anger inhibition for those prone to anger expression may be problematic and could conceivably contribute to disease processes.

To understand the relationship between anger and CV activity, it is helpful to introduce the potential role of ANS functioning. A general discussion of the ANS and the usefulness of heart rate variability (HRV) as a measure of cardiac vagal activity may

provide a coherent perspective on a mechanism under which CV activity mediates the anger-hostility/CHD link.

Autonomic Activity, Anger, and CHD

The ANS, a branch of the peripheral nervous system, is bifurcated into sympathetic and parasympathetic branches. HR 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 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 sino-atrial 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, Bosner, Kleiger, &

Conger, 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). 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 have established a relationship between low frequency power (LFP) and cardiac sympathetic influences across a variety of situations (Friedman, Thayer, & Tyrell, 1996; Snidman, Kagan, Riordan, & Shannon, 1995; Kamada et al., 1992), although there can be a substantial vagal influence on this frequency (e.g., Sloan et. al., 1996). The LF to HF power ratio (LF/HF) has been used as an assessment of sympathovagal balance, with higher values indicating greater relative sympathetic control (e.g., Cohen, Matar, Kaplan, & Kotler, 1999).

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 psychophysiologists 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, Gladstone, & Ehrenkranz, 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).

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.

To date, relatively few studies have investigated the relationship between spectral derived HRV and trait anger. However, there is some evidence to suggest a positive

relationship between LF power and the tendency to express anger in young men, whereas no such relationship was observed in young women (Ramaekers, Ector, Demyttenaere, Rubens, & Van de Werf, 1998). An inverse relationship has also been found between HF power and hostility in response to mental arithmetic stress involving harassment (Shapiro et al., 2000; Sloan et al., 2001).

In addition to assessments of LF power, a more pure assessment of sympathetic influences on CV activity may be provided by the use of 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 primarily sympathetic inotropic (cardiac contractility) assessment. Measures derived from ICG may include pre-ejection period (PEP), reflecting myocardial contractility issues that are primarily determined by β-adrenergic systolic time intervals. Since sympathetic chronotropic and inotropic activity are distinct (Uijtdehaage & Thayer, 2000), use of both LFP and PEP provide a well rounded assessment of cardiac sympathetic activity.

Gender

Cardiovascular reactivity to lab stressors may also vary as a function of gender. In general, men tend to score higher on the Ho scale (Sherwitz, Perkins, Chesney, & Hughes, 1991) and demonstrate more CVR to lab stressors than women (Guyll & Contrada, 1998).

However, men and women scoring high on the Ho scale have been found to display greater DBP and prolonged SBP post-task (i.e., weaker recovery) in response to an anagram task with harassment compared to their low hostile counterparts (Suarez &

Williams, 1990). Further, hostile men and women also tend to report more negative affect than low hostile subjects, illustrating the tendency for these individuals to experience anger and irritation more profoundly and frequently than others.

One significant gender difference emerged in a study assessing the influences of anger expression style, harassment, and opportunity to evaluate the experimenter following harassment on CVR and recovery (Lai & Linden, 1992). This study revealed that men exposed to the experimenter evaluation condition following mental arithmetic stress with harassment showed a more pronounced HR recovery than those not given such an opportunity. Interestingly, anger management style did not modify this effect in men and women did not show facilitated CV recovery as a function of exposure to the experimenter evaluation condition.

Many studies in the anger-hostility literature report a tendency for harassment or some form of interpersonal conflict to be necessary to contribute to significant differences in CVR. In the following section, this research area is discussed in order to highlight the theoretical rationale underlying harassment as a mediating force among these trait relationships to CHD.

Harassment

Williams et al (1985) proposed two qualitatively distinct hemodynamic response patterns to stressors based upon saliency of threat: 1) fight or flight responsivity involving increases in BP mediated by elevations in cardiac output (CO) that are likely to exist under social stressors with harassment and 2) vigilance responses characterized by rises in BP due to increased peripheral vasoconstriction that occur during environmental monitoring for threat.

Assessment of primary and secondary appraisal processes have shown challenge appraisal to be associated with enhanced effort and to lead to an increase in cardiac activity compensated for by a decrease in vascular resistance, whereas threat appraisal, associated with negative emotions, lead to an increase in vascular resistance (Tomaka, Blascovich, Kelsey, & Leitten, 1993). Consequently, adding negative emotions to a challenging task should exert more load on the CV system and may contribute to the risk for CV diseases.

Cardiovascular reactivity in situations of active coping are typically characterized by activation effects on HR, SBP, and PEP with smaller effects on DBP and vascular resistance (e.g., Obrist, 1981). The enhanced sympathetic activation during active coping has been suggested to be quite adaptive to the challenging character of lab tasks because it allows for an efficient and speedy distribution of metabolic resources throughout the body, which is necessary to improve task performance (Dienstbier, 1989). Compared with other emotions and neutral conditions, anger produces HR and SBP responses similar to those of fear, but the greatest responses have been obtained in DBP (e.g., Sinha, Lovallo, & Parsons, 1992). Effortful active coping and anger provocation produce different patterns of CVR and are related to different psychological constructs, with effortful active coping leading to increases in HR and SBP (e.g., Bongard, 1995) and anger provocation leading to increases in HR and DBP (e.g., Sinha et al, 1992).

Autonomic patterning has been found to vary as a function of trait hostility (Schuler & O'Brian, 1997) as well as exposure to different types of stressor tasks (Obrist, 1981). Active coping tasks such as reaction time and mental arithmetic require feedback for completion and tend to elicit β-adrenergic responsitivity, whereas passive tasks such

as the cold pressor require endurance for completion and tend to elicit α-adrenergic responsivity (Sherwood & Turner, 1992). However, studies using standard 'active' and 'passive' coping stressors to distinguish CVR among hostile subjects have generally found non-significant results, suggesting that it is necessary to elicit the construct-relevant affect of anger in hostile subjects to observe significant differences in CVR (see Suls & Wan, 1993 for a review). A recent study indicated that this active-passive distinction did not reliably predict CVR among subjects rating high and low in hostility and defensiveness (Vella & Friedman, 2005). This study revealed that defensive hostile subjects showed significantly higher HR reactivity to the cold pressor task in comparison to purely hostile subjects, but non-significant differences to a video game. Thus, it would seem that interpersonal harassment followed by the opportunity to express anger are more relevant to hostility studies than the more traditional 'active' and 'passive' classifications of lab tasks.

One investigation found that as harassment progressed and became more manifest during the course of stressor exposure, CVR in hostile subjects increased, whereas CVR for low hostile subjects remained relatively constant (Miller et al, 1998). These results provide support for the notion that CVR in hostile subjects is an interaction between personality and interpersonal conflict. Another investigation reported that in addition to significant increases in SBP during an anagram task with harassment and weak SBP recovery from this task, harassed hostile subjects showed greater negative affect ratings across a host of emotions compared to harassed control subjects who rate low on hostility (Suarez et al., 1998). The nature of these findings suggest a joint effect of hostility and emotional arousal, and more specifically *anger*, in triggering CVR to stressors involving

harassment. 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 provoke the pronounced CVR that may lead to coronary pathology (Smith, 1994).

In addition to reviewing reactivity to stressors involving harassment, it may be helpful to include recovery data to attain a comprehensive view on the relationship between physiological patterning and the anger-hostility complex. Recovery data may provide insights into a pathogenic process whereby hostile individuals maintain elevated physiological activity after stressor removal as a function of sustained elevations in anger.

Reactivity and Recovery

Hostile persons have been known to display larger and longer lasting BP responses to anger provoking situations (Fredrickson et al., 2000). Therefore, in addition to increasing CVR to lab stressors, hostility may also attenuate CV recovery. Slow CV recovery after exposure to frustrating events may be a critical factor in the hostility-CHD link that extends beyond the SNS hyperreactivity hypothesis (Brosschot & Thayer, 1998). One potential explanation for the slow CV recovery hypothesis is that hostile people may tend to inhibit their anger in a way that prolongs the SNS response. Further, both high SNS reactivity to frustration and slow CV recovery associated with anger inhibition may involve low vagal activity, suggesting an anger inhibition-vagal inhibition model (Brosschot & Thayer, 1998). Taking this potential situation into account, the psychometric assessment of anger management style along with the experimental

manipulation of anger expression may yield a more sensitive evaluation of the relationship among CVR parameters and harassment.

Opportunity to complete an experimenter evaluation form following harassment induced mental arithmetic stress has been found to facilitate HR recovery and a trend for DBP recovery in men but not women (Lai & Linden, 1992). One focus of the current study was to determine the relationship between CVR and recovery to a lab stressor with or without harassment in men from a young, non-clinical population. The inclusion of harassment and experimenter evaluation techniques in the current study permitted appraisal of whether such tactics were necessary to elicit significant differences in CVR and recovery among subjects scoring high or low on dispositional anger assessed through the subscale of AO.

The conflict between situational anger inhibition and AO may be associated with stressor-induced CVR, poor CV recovery from stress, and potentially stress related CHD. The inhibition of aggressive tendencies following lab stress is likely to be reflected in depressed parasympathetic activity and elevated sympathetic responsivity. This study adds to the CVR literature by providing analyses utilizing HRV as an indication of cardiac vagal tone. The assessment of recovery from the laboratory stressors was intended to highlight the importance of reduced vagal control as a pathogenic marker potentially linking hostility with CHD.

Design and Hypotheses

The present experiment was conducted to determine the CVR and recovery associated with dispositional anger in concert with the situational manipulations of harassment and evaluation. This relationship was examined by administering the AO

subscale of the Spielberger Anger Expression Inventory (Spielberger et al., 1985) as a personality assessment device. Each subject was exposed to a mental arithmetic task with or without harassment followed by a period when subjects were offered the opportunity to either evaluate the experimenter and study procedures (experimenter evaluation condition) or fill out a neutral questionnaire regarding coursework for the current semester (instructor evaluation condition), designed to mimic situations in which individuals are either permitted the opportunity to respond to the source of a provocation versus forced inhibition. Physiological measurements were assessed throughout this study via electrocardiography (ECG), impedance cardiography (ICG), and a BP monitoring device. The design for this study is a 2 X 2 X 3 (harassment X evaluation X condition) mixed design, with condition as the within subjects factor.

In the present experiment, a baseline period was followed by a mental arithmetic (MA) task, an experimenter evaluation/instructor evaluation task, and a recovery period (see Table 1). Change scores, task minus baseline for reactivity and post-stress minus baseline for recovery, were calculated for all physiological measures and compared between groups.

*Hypotheses.*1. In accord with previous findings, AO men were expected to show significant sympathetic α and β adrenergic reactivity to the MA task irrespective to harassment (eg., Engebretson et al., 1989; Ramaekers et al., 1998).

2. An interaction between evaluation condition and harassment was expected to reveal facilitated CV recovery from the MA task, whereby harassed subjects provided the opportunity to evaluate the experimenter show enhanced recovery compared to those in the instructor evaluation condition (Lai & Linden, 1992).

3. An interaction between AO and evaluation condition was expected to reveal attenuated BP and HFP recovery following MA with harassment in support of an anger inhibition-vagal inhibition model (Brosschot & Thayer, 1998) and the 'matching hypothesis' of CV recovery (Engebretson et al., 1989).

METHOD

Subjects

Subjects were 47 right-handed male undergraduate, non-smoking psychology students at Virginia Polytechnic Institute & State University. This sample size was adequate for a power of .71, based upon a previous investigation that employed similar methods (Burns, Evon, & Strain-Saloum, 1999). Non-smoking status was assessed via administration of a health screening questionnaire (see Appendix A). All subjects were instructed to abstain from caffeine and alcohol for 12 hours prior to the study and received extra credit in a psychology course for their participation.

Apparatus

Anger management style was assessed by usage of the 24 item Likert-type Anger Expression Scale, which taps into two independent dimensions of anger management: AI vs AO (Spielberger et al., 1985). Subjects responded to each item by indicating how often they experienced or expressed anger in a certain way based upon a four-point scale (1=almost never, 4=almost always). AI refers to how often angry feelings are experienced but not expressed, including the following items: "I withdraw from people" and "I tend to harbor grudges that I don't tell anyone about". AO refers to aggressive physical or verbal behavior when angered and includes the following items: "I say nasty things" and "I strike out at whatever infuriates me." Internal consistencies for the Anger

Expression Scale have ranged from 0.77 to 0.80 for males and females, respectively (Spielberger et al, 1985).

Vanilla Baseline. To ameliorate the possibility of previous activities or mood states affecting baseline measurements, a vanilla baseline condition has been standardized for comparison to physiological activity during lab tasks and was used in the current study (see Jennings, Kamarck, Stewart, Eddy, & Johnson, 1992). 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 and a previous investigation found it effective in serving this purpose (Vella & Friedman, 2005).

The electrocardiogram (ECG) and impedence cardiography (ICG) was 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 an IBS SD-700A monitor (Industrial & Biomedical Sensors Corp., Waltham, MA).

Dependent Variables

The measure derived from the ECG was HR, reflecting sympathetic β-adrenergic and parasympathetic influences (Saul, 1990). The ECG inter-beat interval (IBI) data were analyzed with a spectral analysis procedure (Matlab, v7.4, 2004). 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 (ms^2 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

All subjects signed an electronic version of the informed consent for the present study and completed the health screening information form and the Anger Expression Scale on-line. Upon arrival at the lab, each subject signed an additional copy of the informed consent and 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 dominant (right) arm.

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

(1) Vanilla Baseline (VB): The subject was instructed to sit 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) Mental Arithmetic (MA): The subject was instructed to engage in a serial subtraction task by counting backward out loud by 7's from 2000. This challenging task has been known to elicit sympathetic β-adrenergic activity and parasympathetic withdrawal (Obrist, 1981). All subjects were instructed to perform as accurately and as fast as possible during this task (see Appendix B). Performance was monitored for accuracy to ensure that subjects were engaged and trying to complete the task.

(3) Evaluation: In the experimental condition, subjects were given a questionnaire with items asking them to evaluate the experimenters and procedures in the current study (see Appendix C). A similar questionnaire was used in a previous investigation employing comparable procedures (Lai & Linden, 1992). In the control condition, subjects were given a questionnaire with items asking them to evaluate the psychology course for which they received extra credit through participation (see Appendix D).

(4) Recovery (RC): The subject was instructed to again sit quietly and relaxed. After completion of this RC period, recording equipment was removed and subjects were debriefed.

Harassment Manipulation. To test the effects of harassment on CVR, subjects performed the MA task under conditions involving either harassment or no harassment. Subjects were exposed to verbal harassment through tape-recorded statements, ostensibly coming from a research technician, played over an intercom. Three statements were used as harassing prods and were played at 45 second intervals irrespective to performance:

1)“You’re making too many mistakes, so try harder”; 2)“You’re still too slow and inaccurate, so focus”; and 3) “This can’t be the best you can do. You’re not trying hard enough”. The harassing statements were made by a man who used a stern, emphatic tone of voice. This method of MA harassment was first established by Hokanson and Shelter (1961) and has since been used in a variety of studies assessing anger relevant traits and CVR and recovery (e.g., Lai & Linden, 1992). Subjects in the non-harassment condition were permitted to complete the task without commentary.

To verify the efficacy of the 3 harassing prods as a means to induce anger, the Spielberger State Anger Scale (Spielberger et al., 1985) was administered before and after the MA task as a manipulation check. The Spielberger State Anger Scale contains 10 self report items that measure the respondent’s current feelings of anger and is a subscale of the State-Trait Anger Expression Inventory.

During debriefing procedures an attempt was made to gage the degree to which subjects believed in the deception and any relevant information was documented at that time. Specifically, subjects were asked during the debriefing period to describe the thoughts they experienced at the time of receiving feedback for their performance. If subjects mentioned thinking that the prods were prerecorded at this point or after the deception was revealed, such information was documented.

Statistical Analyses

For all analyses, harassment and evaluation scores were dummy coded. A single factor MANOVA on repeated measures for mean task scores was conducted to evaluate significant differences in CV activity as a function of harassment during the MA task.

Regression Analyses. The effects of AO and task manipulations on CV changes were evaluated with multiple regression analyses. Testing interactive models in this fashion permits determination of whether experimental manipulations moderate the relationship between traits and CV reactivity and/or recovery (see Baron & Kenny, 1986).

One model tested the significance of a two-way interaction with harassment and AO on reactivity difference scores. Analyses on CV recovery difference scores were conducted using a model that tested the significance of a three-way interaction, involving harassment, evaluation, AO. Predictor variables were entered into the regression models in a hierarchical step fashion. The reactivity and recovery models each consisted of the two or three 'main effect' terms and all possible interaction terms among the main effect variables. Interaction terms were computed by multiplying the relevant variables. In accord with previous studies and because baseline values are a strong predictor of reactivity and recovery levels, baseline values were entered into both models first as a covariate (eg., Burns & Katkin, 1993; Burns, 1995). The rationale behind this statistical method concerns the notion that controlling for baseline values in multiple regression analysis when using reactivity and recovery difference scores provides a more pure assessment of the relationship between predictor and criterion, beyond the variance accounted for by baseline values alone (see Cohen, Cohen, West, & Aiken, 2003).

RESULTS

Task Effects

A single factor MANOVA on harassment groups was conducted first to evaluate whether any significant differences were apparent at baseline. Harassment groups, formed by random assignment, did not differ significantly on any baseline CV measures.

The single factor MANOVA on repeated measures for mean task scores yielded a significant effect for task, $F(18,28) = 29.200$, $p < .001$, but a non-significant effect for harassment, $F(7,39) = .486$, p=ns. Univariate analyses showed significant task effects for all dependent variables with p's<.005. Table 2 displays means and standard deviations for all dependent variables as a function of task and harassment. The overall pattern of responding to the MA task reveals increases in HR, SBP, and DBP and decreases in PEP, LFP and HFP.

Manipulation Check

Subjects exposed to harassment did show higher post stressor state anger scores (M=16.875, SD=2.98) than those in the non-harassment condition (M=15.960, SD=1.80). However, an independent samples t-test on Spielberger State Anger scores revealed a non-significant effect for harassment, $t(45) = 1.272$, $p = .210$.

Reactivity Scores

To test the first hypothesis regarding CV reactivity as a function of AO, hierarchical multiple regression analyses were conducted for each dependent variable. Significant AO main effect terms were observed for PEP in the first model ($R = .475$; $R^2 = .226$; $\beta = 1.711$; $p = .007$) and LFP in the second model ($R = .672$; $R^2 = .452$; $\Delta R^2 = .073$; $\beta = .106$; $p = .016$). Further, a non-significant AO main effect term was observed for HFP in the second model ($R = .643$; $R^2 = .414$; $\Delta R^2 = .083$; $\beta = .0926$; $p = .066$). Tables 3-5 display the entire regression reactivity models for PEP, LFP, and HFP, respectively. Figure 1 displays PEP reactivity values by AO for subjects scoring one standard deviation above and below the mean on this scale (nine subjects per group), indicating that high AO men

showed decreased contractility to the MA task (M=7.82 ms, SD=16.53 ms), whereas low AO men showed increased contractility (M=-9.60 ms, SD=18.12 ms).

Figure 2 displays LFP reactivity values by AO for subjects scoring one standard deviation above and below the mean on this scale and paints a different picture than that portrayed with PEP. High AO men showed similar LFP levels relative to baseline with only a slight activity suppression in response to the MA task (M=-.0758, SD=.6158), whereas low AO men showed a more pronounced suppression (M=-.8682, SD=.7653).

Figure 3 displays HFP reactivity values by AO for subjects scoring one standard deviation above and below the mean on this scale, indicating a similar pattern as that exhibited for LFP reactivity, whereby high AO men show less vagal suppression (M=-.4028, SD=1.043) compared to low AO men (M=-1.078, SD=.7431). Figures 4-6 display mean task levels across conditions for subjects scoring one standard deviation above and below the mean on the AO scale in the context of PEP, LFP, and HFP, respectively.

The two-way interaction term involving harassment and AO was a significant predictor of LFP (p=.023) and HFP reactivity (p=.016) (see Tables 4-5). In order to interpret these interactions, a procedure was employed in which regression lines were constructed corresponding to reactivity at one standard deviation above the mean on the AO scale (High AO), at the mean (Med AO) and one standard deviation below the mean (Low AO) (see Cohen et al., 2003).

Figures 7-8 display LFP and HFP reactivity by harassment as a function of AO. Although low AO subjects tended to display more LFP suppression as evidence by the aforementioned main effect, this suppression appears to be driven appreciably by exposure to this task in the absence of harassment. The nature of the LFP interaction

revealed that non-harassed subjects who rate low on AO showed the most suppression of this variable to the MA task, whereas non-harassed subjects who rate high on AO showed the most of this measure relative to baseline. Harassment did not have as much of an impact on the range of LF reactivity as a function of AO, although the pattern of reactivity was reversed. Harassed high AO subjects showed more LFP suppression to the MA task compared to harassed low AO subjects. Interestingly, subjects showing an average level of AO did not vary in their LF reactivity as a function of harassment.

The pattern of findings portrayed in Figure 8 for HFP reactivity by harassment and AO are similar to the LFP reactivity just described, although more pronounced in the context of harassment. Vagal reactivity did not vary much as a function of harassment for subjects with average levels of AO. However, the situational manipulation of harassment did appear to exert an influence on vagal suppression for subjects rating low and high on AO. In the absence of harassment, low AO subjects showed the most vagal suppression to the MA task and high AO subjects showed the least suppression. This pattern was switched during harassment, whereby high AO subjects showed more vagal suppression to the MA task in comparison to low AO subjects. Thus, the vagal suppression displayed by low AO subjects in the non-significant effect for HFP reactivity appears to be driven more by those who were not harassed during the MA task. All other regression analyses on reactivity difference scores were non-significant.

Recovery Scores

To test the second and third hypotheses regarding interaction effects related to CV recovery, hierarchical multiple regression analyses were conducted for each dependent variable. A non-significant harassment main effect term was observed for PEP recovery

in the first model (R=.452; R^2=.204; β=-6.903; p=.065). Table 6 shows the full model for this regression. Figure 9 displays PEP recovery values by harassment for subjects scoring one standard deviation above and one standard deviation below the mean on the AO scale, indicating that harassed subjects maintained lower PEP values relative to baseline following the MA task (M=-3.556 ms, SD=15.347 ms) compared to those who were not harassed (M=-2.311 ms, SD=5.253 ms).

The AO X evaluation interaction terms were significant predictors of HFP (R=.462; R^2=.213; ΔR^2=.127; β=.114; p=.046) and DBP recovery (R=.564; R^2=.318; ΔR^2=.094; β=1.155; p=.048) for their respective second models. Tables 7-8 display these regression models. Figure 10 depicts the regression lines for low, medium, and high AO subjects corresponding to HFP recovery by evaluation type (instructor vs. experimenter). The nature of this interaction revealed that vagal recovery did not vary as a function of AO following instructor evaluation, but did following experimenter evaluation. Specifically, high AO subjects showed the most pronounced vagal recovery following experimenter evaluation, whereas low AO subjects showed the weakest vagal recovery following experimenter evaluation. Further, vagal recovery did not vary as a function of evaluation for subjects with an average level of AO.

Figure 11 displays the regression lines for low, medium, and high AO subjects corresponding to DBP recovery following either instructor or experimenter evaluation. The nature of this interaction revealed that high AO subjects showed the weakest DBP recovery following experimenter evaluation, but the most pronounced recovery following instructor evaluation. A stepwise pattern emerged in this interaction, whereby subjects with at moderate degree of AO also showed a slightly more facilitated recovery following

the instructor evaluation, but there was no distinction in recovery as a function of evaluation for low AO subjects.

DISCUSSION

The aim of this study was to determine the CVR and recovery associated with dispositional anger in combination with the situational manipulations of harassment and evaluation. Further, this study added to the extant literature through provision of spectral analysis and ICG assessments, thereby providing relatively pure assessments of sympathetic β-adrenergic and parasympathetic activity.

The single factor MANOVA on repeated measures for mean task scores revealed that the MA task alone is a potent stressor, producing significant elevations in HR, SBP and DBP and significant decreases in PEP, LFP and HFP. However, the combined null effects for the harassment manipulation from the single factor MANOVA and the manipulation check t-test indicate that the prods were ineffective at eliciting a more pronounced CV reactivity or a significant increase in state anger. One potential explanation for these null findings concern the degree to which the subjects believed the prods were being given spontaneously in accordance with their performance vs. being prerecorded. Based upon documentations made during debriefing, 11/24 (46%) of the subjects who where harassed during the MA task claimed thinking the prods were pre-recorded, whereas 13/24 (54%) of the subjects who were harassed claimed thinking the prods were genuine.

Results for reactivity regressions revealed main effects for AO in PEP, LFP, and a non-significant effect for HFP (p=.066). High AO men showed increases in PEP during the MA task, whereas low AO men showed decreases on this sympathetic β adrenergic

contractility measure. However, a different pattern emerged with respect to LFP reactivity, whereby low AO subjects displayed a more pronounced suppression on this measure compared to high AO subjects. The discrepancies between these two findings support the assertion that measures of chronotropic and inotropic activity assess distinct aspects of sympathetic β adrenergic responses (Uijtdehaage & Thayer, 2000). Further, LFP is an imperfect measure of sympathetic β adrenergic activity, which can include a substantial vagal influence (eg., Sloan et al., 1996). The main effects and interaction effects involving LFP and HFP do parallel one another and so might suggest some vagal involvement in the LFP measure.

Inspection of Figures 5-6 reveals that, in general, high AO subjects tended to exhibit a more rigid heart rate variability patterning compared to low AO subjects; the activity of low AO subjects in these figures could be considered as reflective of a more adaptive organismic responding (Thayer & Friedman, 2002). Alternatively, it could also be suggested that low AO subjects are more reactive to the MA task by exhibiting increased contractility and more vagal suppression. Although the Spielberger sub-scales of AO and AI are considered to be independent constructs (Spielberger et al., 1985), other studies have reported significant negative correlations between these measures (eg., Burns & Katkin, 1993). Therefore, a possible speculation might concern the notion that those not prone to outwardly expressing anger may display heightened CVR to daily stress.

Despite preliminary observations that 46% of harassed subjects did not believe in the deception, analyses involving reactivity as a function of harassment vs. no harassment revealed significant interactions with AO for LFP and HFP. The nature of these

interactions showed that high AO subjects displayed low LFP suppression (ie, comparatively higher LFP levels) paired with low vagal suppression in the absence of harassment, but an appreciably more pronounced dual suppression of these variables in response to the MA task with harassment. Although this particular pattern of reactivity was unexpected, a recent study did find dual vagal/LFP suppression to correspond to the experience of stress and anger in field assessments of 135 CHD patients (Bacon et al., 2004).

Conversely, low AO subjects exhibited more vagal and LFP suppression in the absence of harassment, and comparatively higher levels of these variables in response to harassment. The first hypothesis of this study predicted high AO men to show elevated sympathetic α and β adrenergic activity to the MA task irrespective to harassment. Although partial support for this hypothesis was found in that high AO men tended to show more LFP (ie., less suppression) to the MA task, the reactivity interaction effects calls this hypothesis into question. Rather, these findings suggest a more complex relationship between AO and reactivity, such that high AO men show somewhat elevated LF activity compared to low AO men exclusive to mental stress without provocation. Further, the range of LFP reactivity as a function of AO to the MA task with harassment was restricted compared to the pattern observed in the absence of harassment. This finding may suggest that sympathetic responsivity is more distinguished for the AO construct in stressors that do not involve harassment.

In addition to the reactivity interactions just described, a non-significant effect in PEP recovery was observed for harassment ($p=.065$). Harassed subjects maintained shorter PEPs (ie, sustained cardiac contractility) following the MA task relative to

baseline, whereas those who were not harassed displayed a slight elevation on this measure. This finding suggests that the harassment manipulation may have been effective at maintaining a degree of sympathetic β adrenergic activity following task completion.

The second hypothesis of this investigation concerned a situational interaction between harassment and evaluation for CV recovery, predicting facilitated recovery following experimenter evaluation for harassed subjects in accord with previous results (Lai & Linden, 1992). However, no such significant interaction was observed. The current findings suggest that AO levels interact with evaluation type to predict differential recovery. Person X situation interactions involving AO and evaluation revealed that subjects rating high in AO showed a comparatively attenuated DBP recovery, but increased HF power recovery, following experimenter evaluation.

The AO by evaluation interaction for HFP revealed that vagal recovery differed as a function of dispositional anger only following experimenter evaluation, whereby high AO subjects showed facilitated recovery compared to low AO subjects. From a vagal standpoint, this finding provides partial support for the 'matching hypothesis' of CV recovery (Engebretson et al., 1989), indicating that optimal vagal recovery may be predicted by dispositional anger. However, the situational manipulation of instructor evaluation did not influence vagal recovery as a function of AO. This situational manipulation was designed to mimic the circumstance of forced anger inhibition following provocation. The observation that recovery did not vary from exposure to this manipulation does not provide support for the anger-inhibition/vagal inhibition model of CV recovery (Brosschot & Thayer, 1998). However, the finding that low AO subjects did display the weakest vagal suppression to the MA task with harassment does provide some

support for this hypothesis (see Figure 8). It is possible that the mixture of being low on AO with provocation or high on AO without provocation predicts low vagal reactivity.

In contrast to the recovery interaction for HFP, the interaction between AO and evaluation for DBP did not provide support for the matching hypothesis that high AO subjects would show facilitated recovery following experimenter evaluation and attenuated recovery following instructor evaluation. Rather, the current findings show the opposite effect, whereby high AO subjects show facilitated recovery following instructor evaluation and attenuated recovery following experimenter evaluation; conversely, DBP recovery did not vary as a function of low AO. A possible explanation between the discrepancies between the HFP interaction and the DBP interaction for CV recovery might concern the notion that vagal responsivity is relatively fast acting (eg., Malliani et al., 1991) and so comparisons between these two measures in this instance may reflect the slower recovery processes underlying sympathetic α adrenergic activity.

Although the state anger scale administered following the MA task did not suggest significant increases in this mood state, the physiological responsivity to the task may suggest such an increase nonetheless. The Spielberger State Anger Scale may not have been an appropriate fit for this investigation, since each item requests ratings of rather dramatic displays of anger (eg., “I feel like kicking somebody”; “I feel like pounding somebody”). In the current study, increases on this scale following harassment tended to be on items regarding irritation and frustration rather than the more extreme items involving strong physical displays of anger. It may have been preferable to employ a less extreme assessment of state anger that is masked in the context of other mood assessments.

Limitations

There are many limitations to the current study that may at least partially explain some of the unexpected and/non-significant findings. First, it appears as if there may have been a 'ceiling effect' in the use of the MA task for this study. The MA task is a potent stressor that has been shown to account for rather large increases in HR, SBP, and DBP in the current study (see Table 2). It may have been difficult to assess significant differences as a function of harassment, given the potency of the MA task alone. Further, there is the issue of harassing prods administration that needs to be addressed. The use of prerecorded prods being administered via intercom may have seemed too artificial to be considered believable by an appreciable proportion of the harassed sample (ie., 46%). In future studies, additional steps will be taken to ensure the veracity of this manipulation by having the subjects meet the harasser at some point prior to the beginning of the task.

Finally, the evaluation condition was aimed at capturing the constructs of state anger inhibition versus expression. One problem with the completion of evaluation forms to this end may concern the ambiguity of the experimenter evaluation condition; the primary investigator was in the same room as the subjects throughout the conduction of the study and such a presence could feasibly have biased some of the comments made during evaluation in a way that facilitated recovery. An alternative to this type of evaluation may include a more socially germane method by involving some type of verbal discourse versus instructing the subject to sit quietly while the harasser is present in the room. Engebretson et al. (1989) did employ a more social form of evaluation in their study and this distinction may be formative in explaining the discrepancies with the current investigation.

Conclusions

The main findings of this study suggest that the interactive effects of dispositional anger and situational influences on CV reactivity and recovery are an important and complex area of investigation for behavioral medicine. Specifically, it was found that AO interacts with harassment to predict LFP and HFP reactivity to a stressful task. Further, trait anger may moderate the recovery process when considering the opportunity to evaluate the source of provocation.

Future studies will take necessary measures to ensure the verisimilitude of the situational manipulations. Further, women subjects will be included to test significant gender differences in the context of anger management style and the effects of evaluation therein. Previous studies have indicated that women are more prone to AI (eg., Houston & Vavak, 1991) and this tendency to be more associated with DBP reactivity in women than men (eg., Harralson, Suarez, & Lawler, 1997).

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Table 1: *2 X 2 X 3 (Harassment X Anger Release X Condition) Mixed Design*

Group	Baseline	MA	Recovery
Harassed/Anger Release			
Harassed/No Anger Release			
Non-harassed/Anger Release			
Non-harassed/No Anger Release			

Note. Condition is a within subjects variable. 11-12 subjects were examined in each group.

Table 2: *Means and Standard Deviations by Task and Harassment*

Dependent Variable	Baseline	MA	Evaluation	Recovery
Heart Rate (bpm)				
Harass	66.84 (*8.36*)	84.21 (*12.19*)	72.99 (*8.14*)	68.66 (*7.86*)
No Harass	71.12 (*10.12*)	87.02 (*10.87*)	75.24 (*9.38*)	72.03 (*8.51*)
SBP (mmHg)				
Harass	120.33 (*11.73*)	136.73 (*14.86*)	129.06 (*14.63*)	117.94 (*13.20*)
No Harass	119.41 (*10.16*)	133.00 (*11.74*)	125.21 (*8.98*)	116.13 (*10.53*)
DBP (mmHg)				
Harass	68.85 (*7.68*)	79.85 (*11.46*)	71.52 (*10.80*)	68.04 (*7.77*)
No Harass	68.50 (*10.26*)	80.50 (*10.83*)	70.00 (*12.04*)	68.09 (*9.79*)
PEP (ms)				
Harass	117.57 (*23.83*)	109.16 (*24.88*)	113.93 (*26.82*)	112.13 (*24.49*)
No Harass	113.53 (*27.77*)	107.41 (*28.44*)	117.71 (*28.30*)	114.67 (*24.84*)
LFP (Nlg)				
Harass	14.49 (*.8528*)	14.21 (*.6897*)	14.10 (*.6405*)	15.10 (*1.060*)
No Harass	14.27 (*.6074*)	14.03 (*.8051*)	13.97 (*.7398*)	15.18 (*.7886*)
HFP (Nlg)				
Harass	13.87 (*.7581*)	13.37 (*.8695*)	13.52 (*.6634*)	14.22 (*.9491*)
No Harass	13.79 (*.9234*)	13.15 (*.8597*)	13.18 (*.8536*)	14.15 (*.9174*)

Note: Standard Deviations in parentheses

Table 3: *Hierarchical Multiple Regression Analysis for PEP Reactivity*

Predictor	df1	df2	β	t	Sig	R	R^2	ΔR^2	Sig
Model 1	3	43				.475	.226	.226	.011
Baseline			-.160	-1.80	.08				
Harass			-4.28	-.931	.357				
Anger Out			1.71	2.823	.007				
Model 2	4	42				.496	.246	.02	.291
Baseline			-.151	-1.70	.098				
Harass			-4.51	-.982	.332				
Anger Out			2.479	2.683	.012				
AOXHarass			-1.31	-1.07	.291				

Table 4: *Hierarchical Multiple Regression Analyses for LFP Reactivity*

Predictor	df1	df2	β	t	Sig	R	R^2	ΔR^2	Sig
Model 1	3	43				.616	.380	.380	<.001
Baseline			-.727	-4.96	<.001				
Harass			.0647	.294	.770				
Anger Out			.0297	1.04	.306				
Model 2	1	42				.672	.452	.073	.023
Baseline			-.788	-5.56	<.001				
Harass			.0601	.287	.775				
Anger Out			.106	2.51	.016				
AO X Harass			-.132	-2.36	.023				

Table 5: *Hierarchical Multiple Regression Analyses for HFP Reactivity*

Predictor	df1	df2	β	t	Sig	R	R^2	ΔR^2	Sig
Model 1	3	43				.576	.331	.331	.001
Baseline			-.681	-4.56	<.001				
Harass			.192	.766	.448				
Anger Out			.014	.032	.975				
Model 2	1	42				.643	.414	.083	.019
Baseline			-.778	-5.29	<.001				
Harass			.180	.757	.453				
Anger Out			.0926	1.89	.066				
AO X Harass			-.160	-2.44	.019				

Table 6: *Hierarchical Multiple Regression Analyses for PEP Recovery*

Predictor	df1	df2	β	t	Sig	R	R^2	ΔR^2	Sig
Model 1	4	42				.452	.204	.204	.043
Baseline			-.156	-2.22	.032				
Harass			-6.90	-1.90	.065				
Evaluation			.760	.214	.831				
Anger Out			.634	1.323	.193				

Table 7: *Hierarchical Multiple Regression Analyses for HFP Recovery*

Predictor	df1	df2	β	t	Sig	R	R^2	ΔR^2	Sig
Model 1	4	42				.293	.086	.086	.424
Baseline			-.252	-1.95	.058				
Harass			.0012	.053	.958				
Evaluation			.0092	.435	.666				
Anger Out			.0006	.200	.842				
Model 2	3	39				.462	.213	.127	.115
Baseline			-.273	-2.07	.045				
Harass			-.006	-.224	.824				
Evaluation			.0066	.222	.826				
Anger Out			.0003	.074	.942				
Harass X Evaluation			.0079	.195	.847				
Anger Out X Harass			-.007	-1.23	.230				
Anger Out X Evaluation			.114	2.066	.046				

Table 8: *Hierarchical Multiple Regression Analyses for DBP Recovery*

Predictor	df1	df2	β	t	Sig	R	R^2	ΔR^2	Sig
Model 1	4	42				.474	.225	.225	.027
Baseline			-.389	-3.23	.002				
Harass			-.384	-.181	.858				
Evaluation			1.538	.739	.464				
Anger Out			.101	.353	.726				
Model 2	3	39				.564	.318	.094	.165
Baseline			-.345	-2.83	.007				
Harass			2.575	.869	.390				
Evaluation			4.863	1.629	.111				
Anger Out			-.316	-.660	.513				
Harass X Evaluation			-6.44	-1.51	.140				
Anger Out X Harass			.0036	.064	.950				
Anger Out X Evaluation			1.155	2.038	.048				

Figure 1

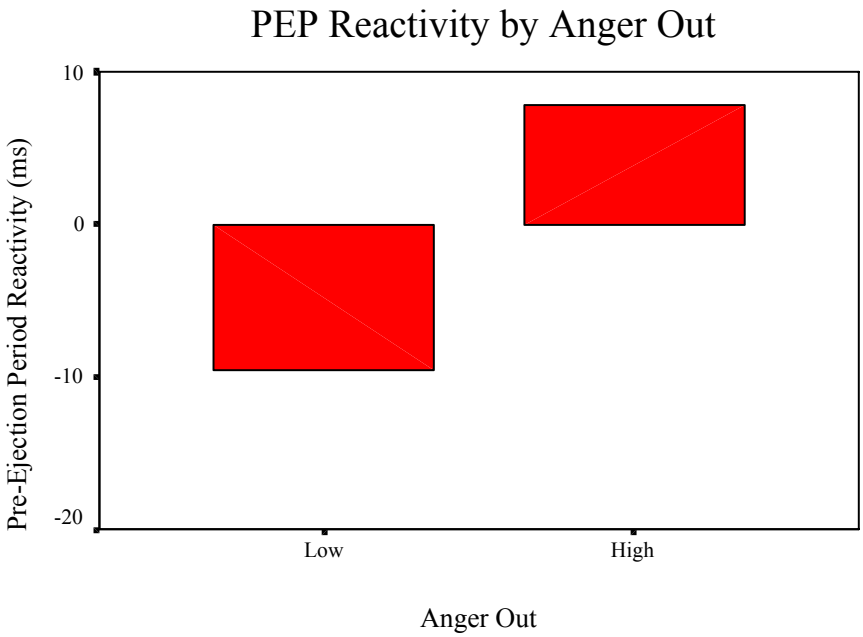

Figure 1: PEP Reactivity to MA Task by Anger Out

Figure 2

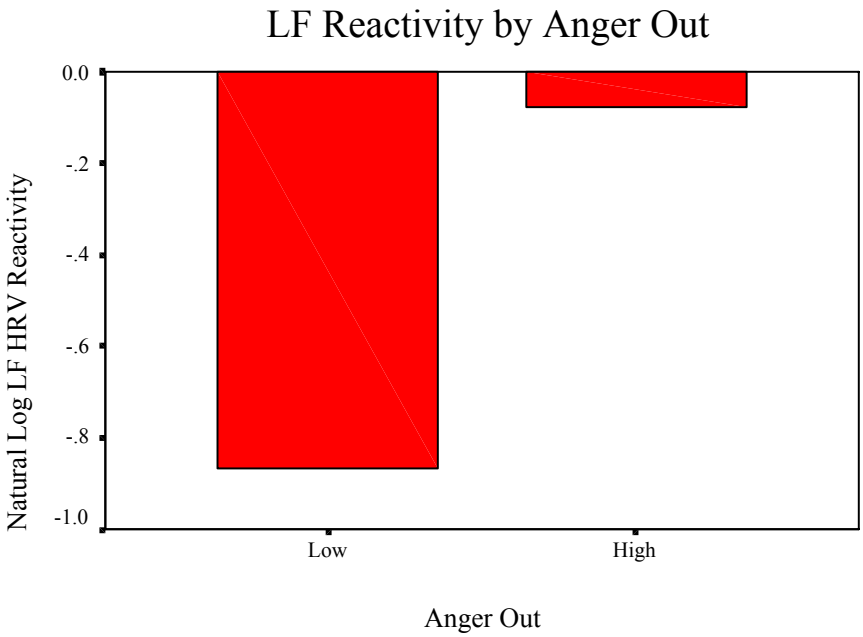

Figure 2: Natural Log LFP Reactivity to MA Task by Anger Out

Figure 3

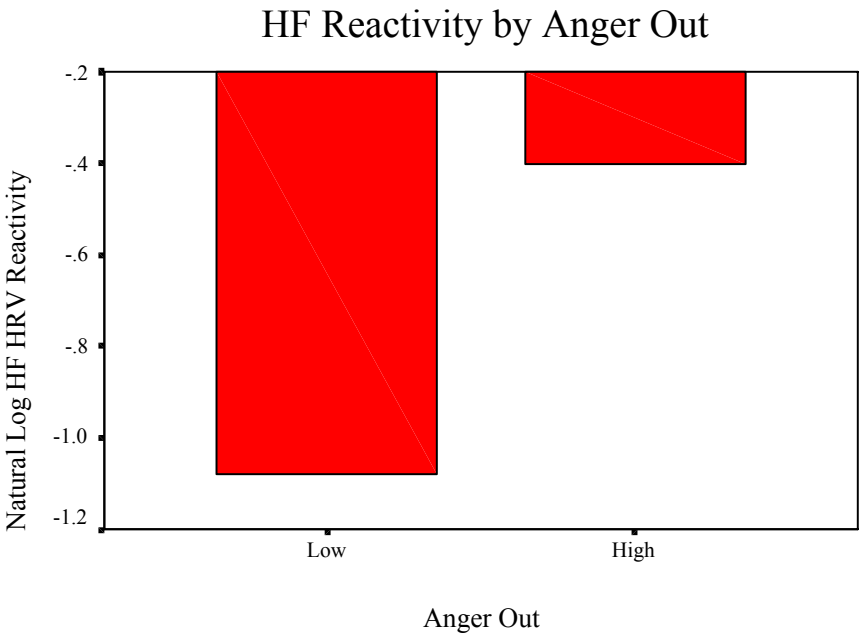

Figure 3: Natural Log HFP Reactivity to MA Task by Anger Out

Figure 4

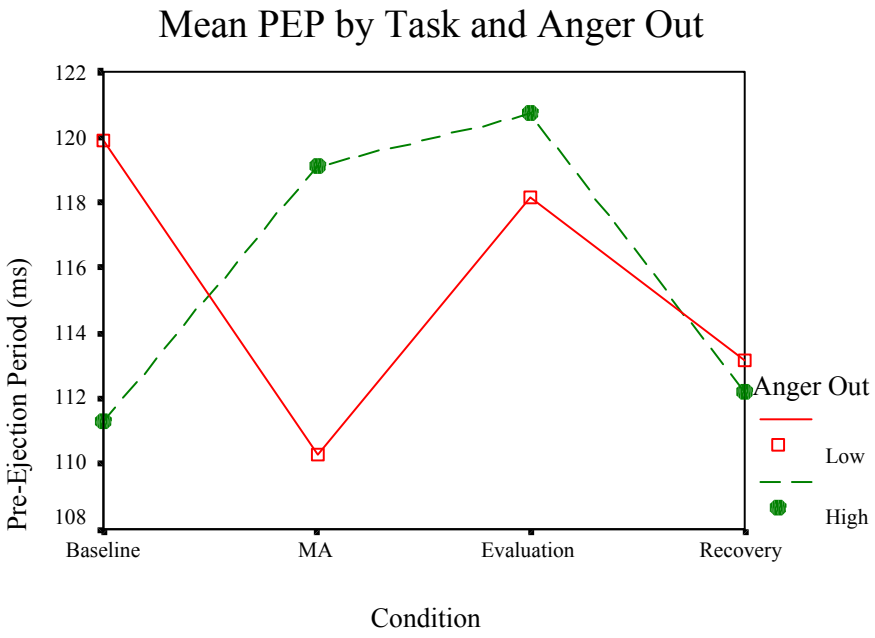

Figure 4: Mean PEP Activity by Task and Anger Out

Figure 5

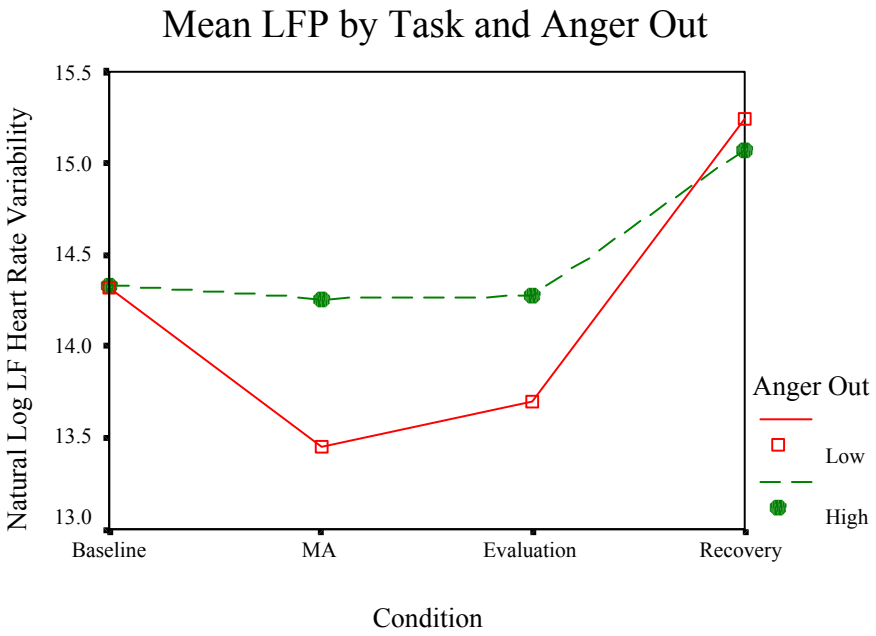

Figure 5: Mean Natural Log LFP Activity by Task and Anger Out

Figure 6

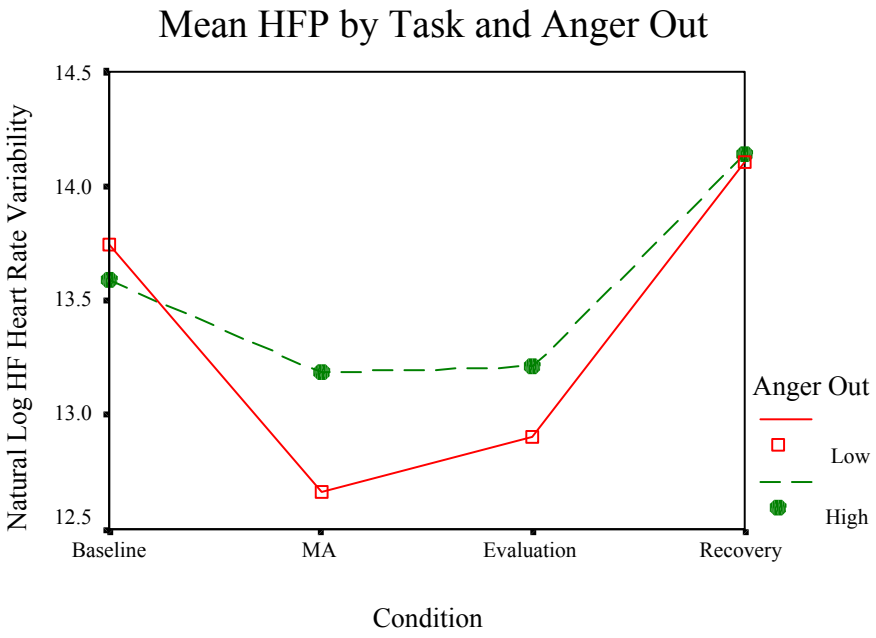

Figure 6: Mean Natural Log HFP Activity by Task and Anger Out

Figure 7

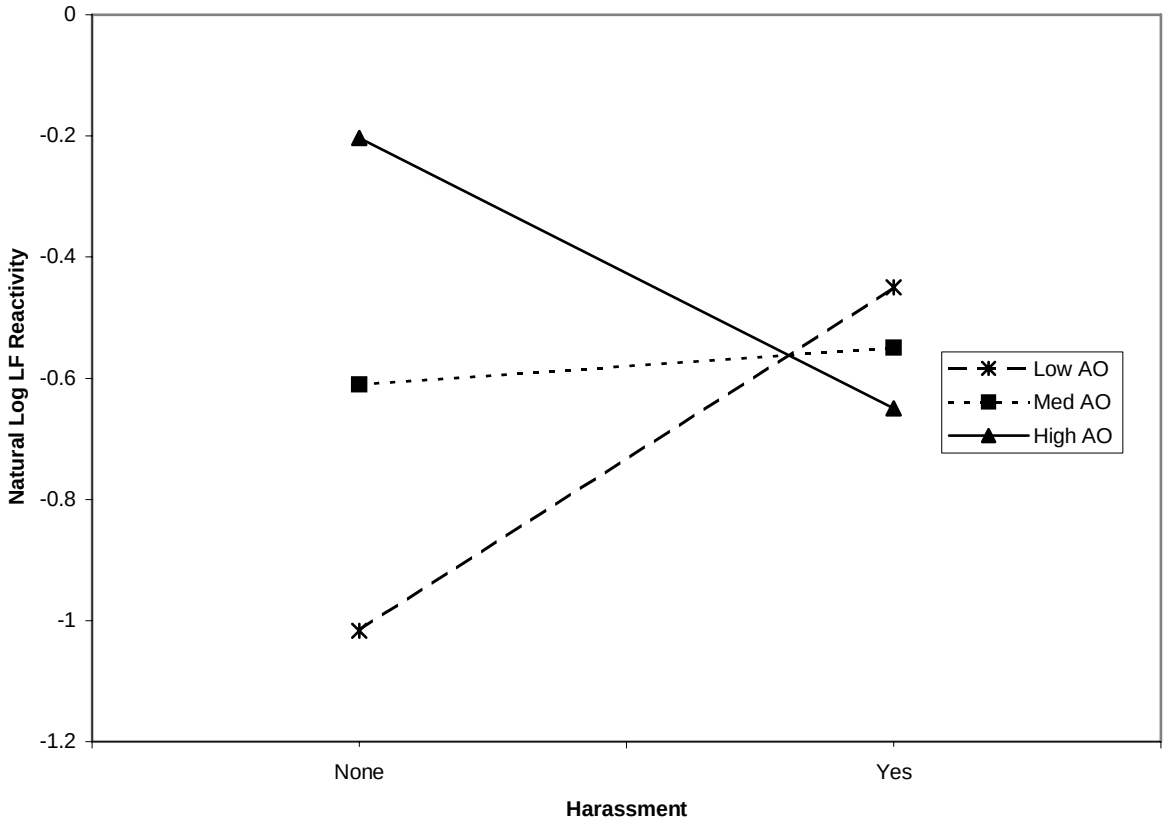

Figure 7: Natural Log LFP Reactivity by Harassment and Anger Out

Figure 8

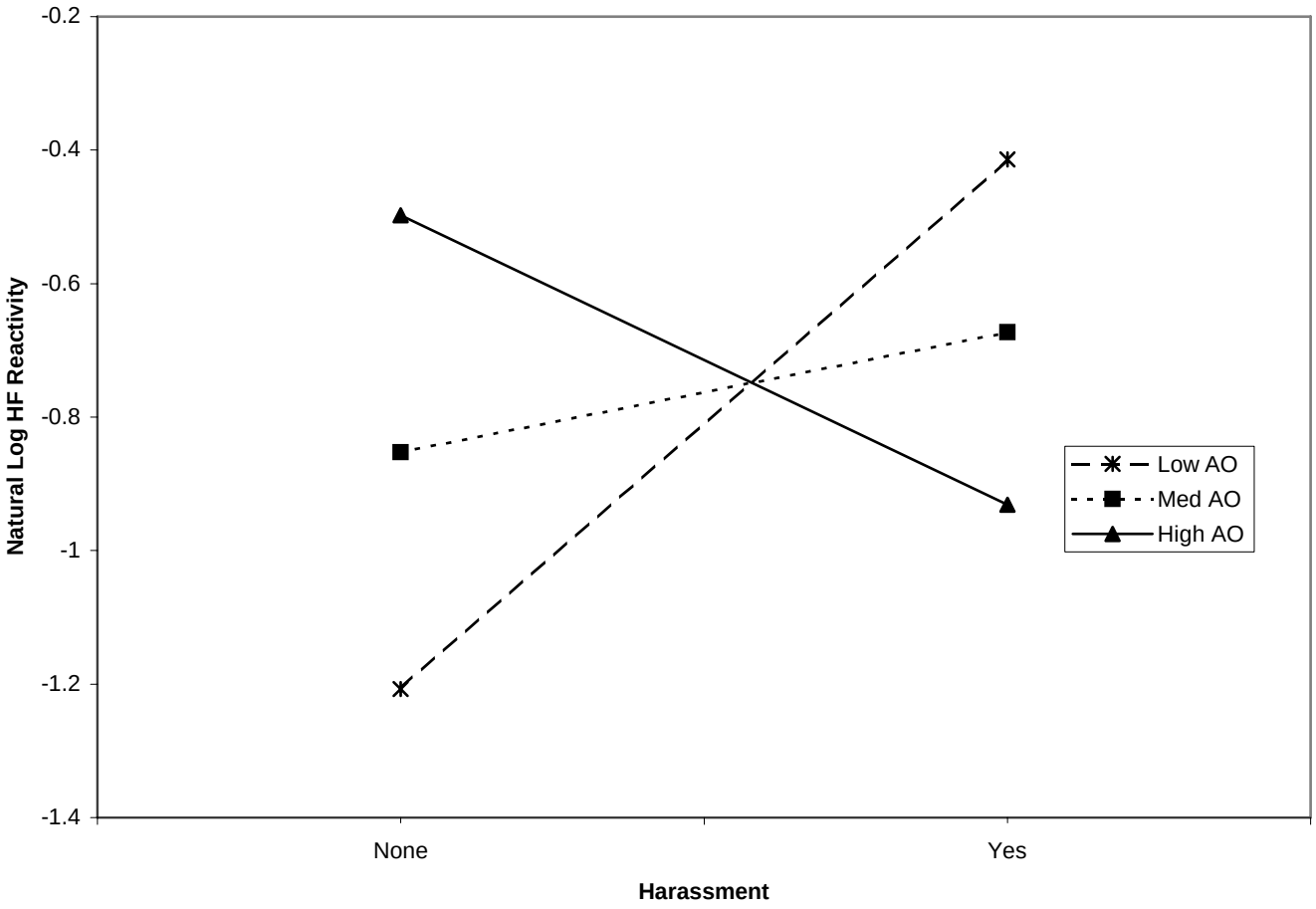

Figure 8: Natural Log HFP Reactivity by Harassment and Anger Out

Figure 9

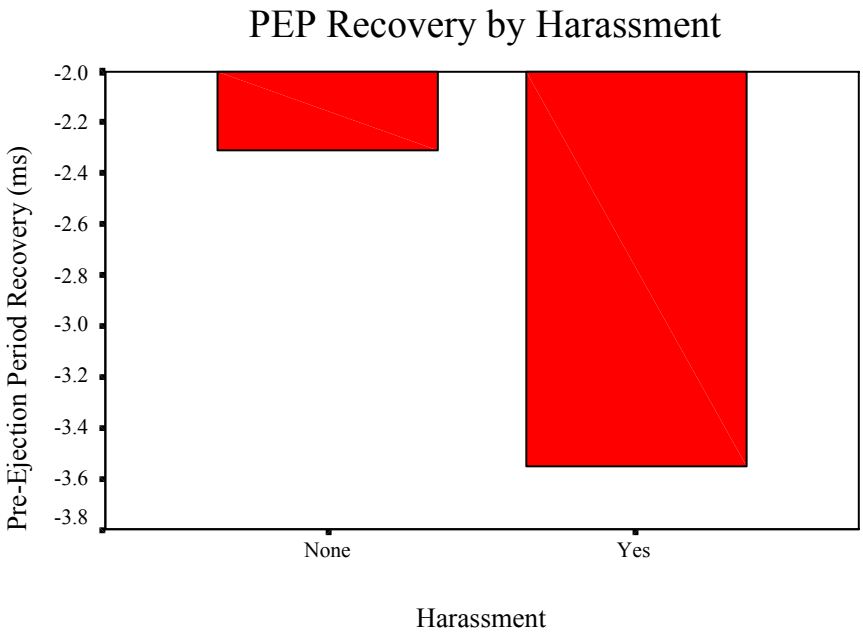

Figure 9: PEP Recovery by Harassment

Figure 10

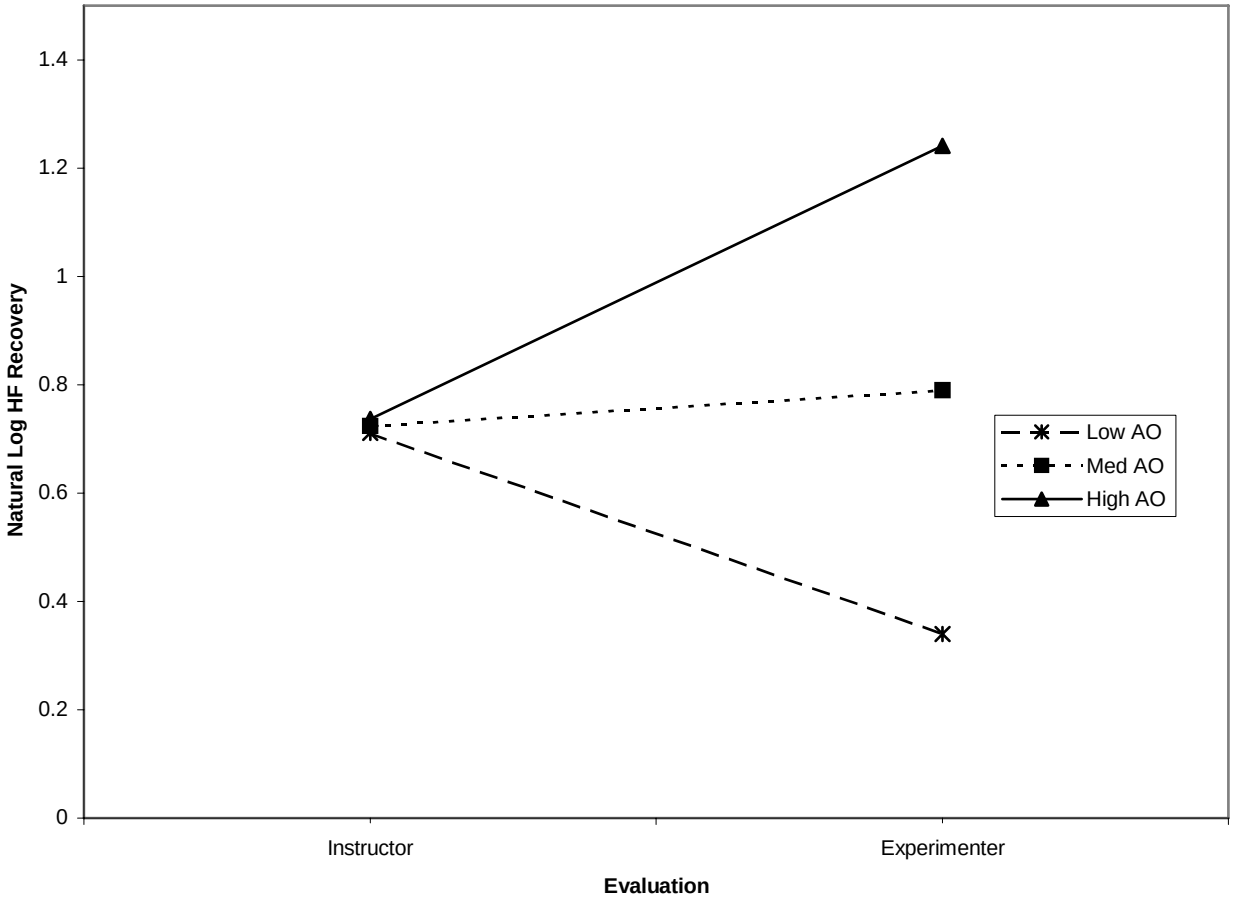

Figure 10: Natural Log HFP Recovery by Evaluation and Anger Out

Figure 11

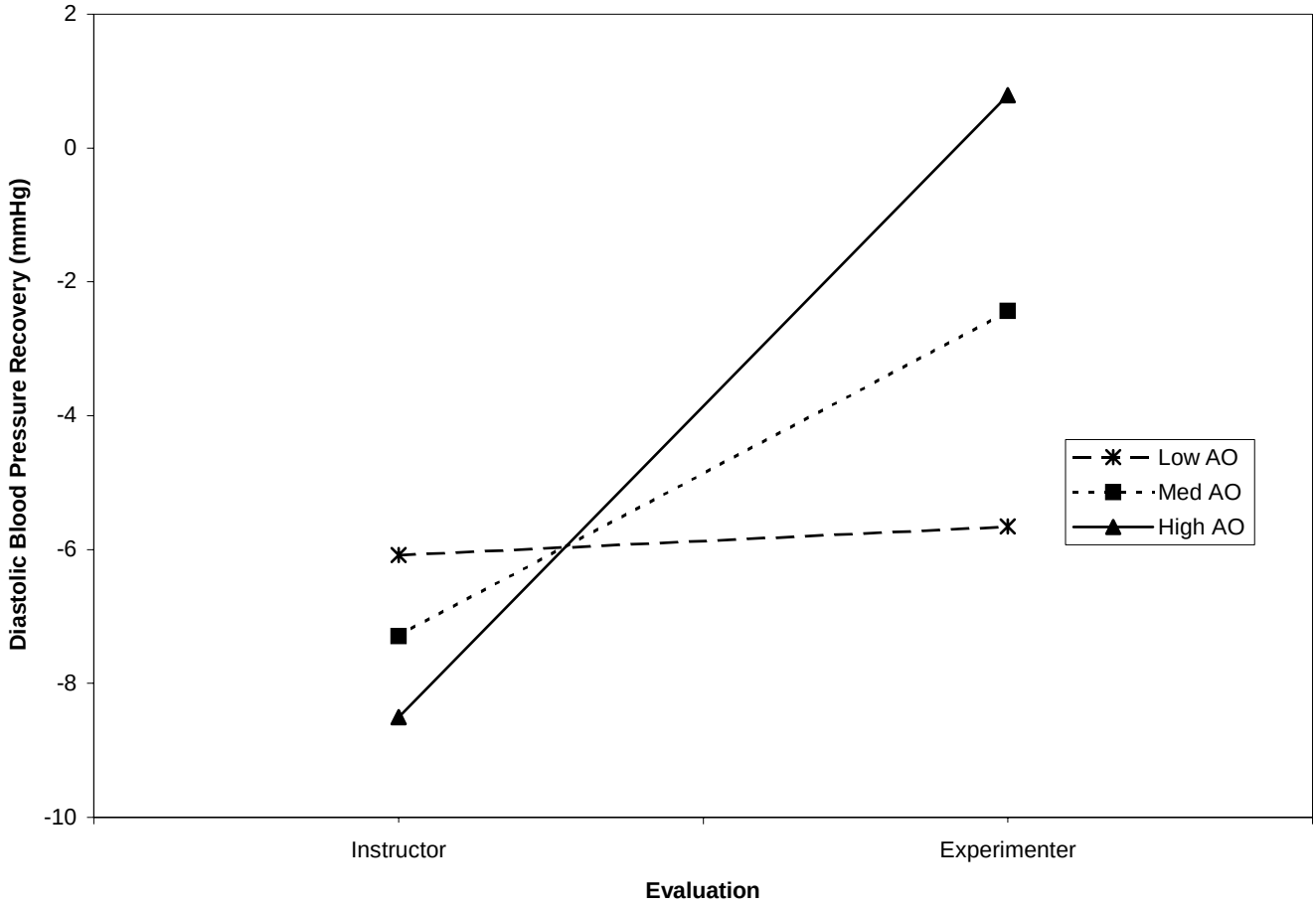

Figure 11: DBP Recovery by Evaluation and Anger Out

APPENDIX A
HEALTH SCREENING QUESTIONNAIRE

Age_____ Gender_____ Height_____ Weight_____ Handedness_____

Ethnicity (Circle one): American Indian African American Asian/Pacific Islander

Caucasian Hispanic Multi-Racial

Other (please specify):_____

Address_____

Phone_____

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_____

_____

_____

_____

Do you have a family history of hypertension? Yes_____ No_____

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_____

_____

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

APPENDIX B

Mental Arithmetic Protocol

For the next three minutes you will be asked to participate in a mental arithmetic task by counting backwards aloud from 2000 by 7. For example, you will begin by saying 2000, 1,993, 1,986, etc. Be as quick and accurate as possible. A research assistant in our control room will be monitoring your performance through this microphone and you may receive some feedback from him via intercom during this task. You can take this feedback into account, but be sure to continue counting backwards until instructed to stop. Do you have any questions? Okay, begin.

APPENDIX C
EXPERIMENT EVALUATION FORM

Your feedback is important to us! To improve the design of this study, please respond to each of the following items in an open and frank manner. Your responses are completely confidential. The information obtained may be used to evaluate the current research assistants.

Section A: Circle the number corresponding to your assessment of the experimenter (**1 = poor; 3 = fair; 5 = excellent**).

1. Competency:	1	2	3	4	5
2. Concern/Respect	1	2	3	4	5
3. Fairness	1	2	3	4	5
4. Success Communicating Procedures	1	2	3	4	5
5. Overall Experimenter Rating	1	2	3	4	5

Section B: Using the supplied list of adjectives, describe and explain the emotions you experienced as a subject of this study in the space provided below. Please indicate what you liked most and least about being participating in this experiment and explain why. You are welcome to use the back of this sheet or additional sheets if necessary.

APPENDIX D
ACADEMIC ASSESSMENT FORM

To provide us with an understanding of your current academic context when participating in this study, please respond to each of the following items by evaluating the psychology course for which you are receiving extra credit.

Section A: Circle the number corresponding to your assessment of the psychology instructor (**1 = poor; 3 = fair; 5 = excellent**).

1. Competency:	1	2	3	4	5
2. Concern/Respect for Students	1	2	3	4	5
3. Fairness in Grading	1	2	3	4	5
4. Success Communicating Subject	1	2	3	4	5
5. Overall Instructor Rating	1	2	3	4	5

Section B: Use the space below to describe the psychology course for which you are receiving extra credit. Please be sure to indicate the mode of performance evaluation for this course (i.e., paper, exam, or presentation), as well as your own distinction of difficulty (i.e., easy, average, or difficult). Feel free to use specific examples to describe the various assignments you have had to complete. You may use the back of this sheet or additional sheets if necessary.

APPENDIX E

VIRGINIA POLYTECHNIC INSTITUTE AND STATE UNIVERSITY

Informed Consent for Participation in Investigative Projects

Title of Project: Personality and Cardiovascular Reactivity to a Mental Arithmetic Task.
Investigator: Elizabeth J. Vella, M.S.

I. The Purpose of this Project

The present study will be conducted to determine the cardiovascular patterning associated with personality traits. The investigator is interested in locating 48 right-handed, non-smoking male participants.

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 20-30 minutes. Participation in phase two will last approximately 30 minutes. Each participant will be exposed to serial subtraction mental arithmetic task 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 Mind-Body Laboratory, located at 253 Williams Hall on the Virginia Tech main campus.

III. Risks

Participants are exposed to minimal risks in this study. The equipment for recording cardiovascular activity is noninvasive and relatively comfortable. However, the mental arithmetic task may be perceived as a bit challenging.

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 personality traits and how they interact with lab situations to predict cardiovascular patterning. 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:

_____	_____231-9174/evella@vt.edu_
Elizabeth J. Vella, M.S.	Phone/Email
_____	_____231-9611/bhfriedm@vt.edu
Bruce H. Friedman, Ph.D.	Phone
_____	_____231-4991/moored@vt.edu
Dr. David Moore Chair, IRB CVM Phase II	Phone

APPENDIX F
STATE ANGER INVENTORY

Instructions: A number of statements that people use to describe themselves are given below. Read each statement and then circle the number which indicates how you feel *right now*. There are no right or wrong answers. Do not spend too much time on any one statement. Mark the answer that *best* describes your *present feelings*.

1=Not at all; 2=Somewhat; 3=Moderately so; 4=Very much so

How I Feel Right Now

1. I am furious……	1	2	3	4
2. I feel irritated…….	1	2	3	4
3. I feel angry…….	1	2	3	4
4. I feel like yelling at somebody……....1		2	3	4
5. I feel like breaking things……1		2	3	4
6. I am mad……..1		2	3	4
7. I feel like banging on the table……1		2	3	4
8. I feel like hitting someone……...1		2	3	4
9. I feel like swearing……..1		2	3	4
10. I feel annoyed……1		2	3	4
11. I feel like kicking somebody……..1		2	3	4
12. I feel like cursing out loud……1		2	3	4
13. I feel like screaming…….1		2	3	4
14. I feel like pounding somebody…….1		2	3	4
15. I feel like shouting out loud……..1		2	3	4

APPENDIX G
ANGER OUT SUBSCALE

Instructions: Everyone feels angry or furious from time to time, but people differ in the ways that they react when they are angry. A number of statements are listed below which people use to describe their reactions when they feel *angry* or *furious*. Reach each statement and then **circle** the appropriate number to indicate how *often* you *generally* react or behave in the manner described when you are feeling angry or furious. There are no right or wrong answers. Do not spend too much time on any one statement.

1=Almost never; 2=Sometimes; 3=Often; 4=Almost always

How I Generally React or Behave When Angry or Furious…

1. I express my anger…….1	2	3	4
2. If someone annoys me, I'm apt to tell him or her how I feel 1	2	3	4
3. I lose my temper……1	2	3	4
4. I make sarcastic remarks to others…….1	2	3	4
5. I do things like slam doors…….1	2	3	4
6. I argue with others…….1	2	3	4
7. I strike out at whatever infuriates me……1	2	3	4
8. I say nasty things……...1	2	3	4

Elizabeth J. Vella
Department of Psychology, Virginia Polytechnic Institute & State University,
Blacksburg, VA 24061-0436. Phone: (540) 231-9174. Fax: (540) 231-3652
E-mail: evella@vt.edu

Education

Virginia Polytechnic Institute & State University, Blacksburg VA:
Ph.D. in Psychology (Psychological Sciences), 2005. Dissertation Title: *Anger expression, harassment and evaluation: Cardiovascular reactivity and recovery to mental stress*.
M.S. in Psychology (Psychological Sciences), 2003. Thesis Title: *Autonomic characteristics of defensive hostility: Reactivity and recovery to active and passive stressors.*

Sonoma State University, Rohnert Park, CA:
B.A. in Psychology, May 1998

Academic Employment

Virginia Polytechnic Institute & State University, Blacksburg, VA

Graduate Instructor, Department of Psychology:
- Introduction to Psychology, Summer 2003
- Psychology of Personality, Fall 2003
- Nervous Systems & Behavior, Spring 2004
- Developmental Psychology, Summer 2004
- Principles of Psychological Research, Fall 2004

Graduate Teaching Assistant, Department of Psychology
- Introduction to Psychology, Fall 2000-Spring 2002
- Principles of Psychological Research, Fall 2002
- Nervous Systems & Behavior/Physiological Psychology, Spring 2003

Graduate Research Stipend, Department of Psychology, Summer 2001 & 2002

Research Interests: The psychophysiology of emotion and personality, with an emphasis on measures of autonomic nervous system activity in relation to health and disease.

Publication

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.

Manuscript Submitted for Publication

Vella, E.J., & Friedman, B.H. *The autonomic characteristics of defensive hostility*: *Reactivity and recovery to active and passive stressors.*

Manuscript in Preparation

Friedman, B.H., & Vella, E.J. *Low-frequency ECG spectral power discriminates between active and passive coping tasks.*

Published abstract

Vella, E.J., Friedman, B.H., Lozier, L., & Laskey, G. (2003). Defensive hostility and cardiovascular reactivity to active and passive stressors in college-aged males. *Psychophysiology, 40*(Suppl. 1), S88.

Conference Presentations

Vella, E.J. & Friedman, B.H. (2004, March). *Defensive hostility and cardiovascular reactivity to active and passive stressors in male subjects*. Poster session presented at the 20th annual research symposium of the Graduate Student Association, Blacksburg, VA.

Vella, E.J., Friedman, B.H., Lozier, L., & Laskey, G. (2003, May). *Defensive hostility differentially predicts cardiovascular reactivity and recovery to active and passive stressors in male subjects.* Poster session presented at the 15th annual meeting of the American Psychological Society, Atlanta, GA.

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., & Laskey, 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., & 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.

Honors and Awards

2004: Graduate Student Association Research Award, 1st place for poster presented in category of Natural & Biological Sciences ($350), Virginia Polytechnic Institute & State University

1997-98: Psi Chi Vice President, Sonoma State University
1997-98: Hewlett-Packard Scholarship, Sonoma State University

Colloquium

The autonomic characteristics of defensive hostility: Reactivity and recovery to active and passive stressors. Department of Psychology, Virginia Polytechnic Institute & State University, Blacksburg, VA, September 2003.

Ad Hoc Reviewer

Biological Psychology
International Journal of Psychology

Professional Memberships

American Psychological Society
American Psychosomatic Society
Society of Behavioral Medicine
Society for Psychophysiological Research

References

Bruce H. Friedman, Ph.D.
Department of Psychology
221 Williams Hall
Virginia Polytechnic Institute & State University
Blacksburg, VA 24061-0436
(540) 231-9611
bhfriedm@vt.edu

David W. Harrison, Ph.D.
Department of Psychology
231 Williams Hall
Virginia Polytechnic Institute & State University
Blacksburg, VA 24061-0436
(540) 231-4422
dwh@vt.edu

Robin Panneton, Ph.D.
Department of Psychology
321 Williams Hall
Virginia Polytechnic Institute & State University
Blacksburg, VA 24061-0436
(540) 231-5938
panneton@vt.edu