THE INTERACTION OF SMOKING AND STRESS ON CARDIOVASCULAR REACTIVITY AS INFLUENCED BY HOSTILITY, ANGER, AND ANXIETY

by

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ABSTRACT

This study examined how smoking and stress interact to affect the reactivity of the cardiovascular system. Findings revealed that subjects who smoked first and who were then exposed to stress showed less cardiovascular reactivity (CVR) to stress compared to non-smoking subjects exposed only to stress. However, the combination of smoking and stress led to higher absolute levels and greater reactivity for all cardiovascular measures compared to stress alone.

The ability for trait hostility, state angry affect and state anxious-affect to predict CVR to smoking and stress was also examined. State anxious-affect was the only affect-related variable that predicted CVR across experimental phases for both smokers and non-smokers. None of the affect-related variables were able to predict CVR during smoking. Trait hostility and state anxious-affect predicted CVR to stress for smokers and non-smokers. Trait hostility was negatively associated with CVR to stress, while state anxious-affect was positively associated with CVR to stress. State angry affect did not predict CVR to stress after accounting for the effects due to trait hostility.

INTRODUCTION

Purpose of the Study

This study examines how smoking and stress interact to affect the reactivity of the cardiovascular system. The following questions are addressed:

1. How does prior exposure to cigarette smoking affect the reactivity of the cardiovascular system to stress, compared to the cardiovascular reactivity occurring in response to stress alone?
2. When smoking and stress are combined, do they lead to greater increases in cardiovascular reactivity compared to the effects of stress alone?

An additional purpose of this study is to determine if the cardiovascular reactivity occurring in response to smoking and stress can be explained by trait hostility, state angry affect, and state anxious-affect. The following questions are examined:

1. Is the cardiovascular reactivity to smoking affected by trait hostility, state angry affect, and state anxious-affect, or is the cardiovascular reactivity to smoking explained solely on the basis of smoking?
2. When stress occurs following smoking, can trait hostility, state angry affect, and state anxious-affect explain the cardiovascular reactivity to stress beyond the effects accounted for by smoking?
3. Do trait hostility, state angry affect, and state anxious-affect differentially predict the reactivity of the cardiovascular system to smoking and stress?

Organization of the Literature Review

The literature review begins by introducing cardiovascular reactivity (CVR) as the main outcome measure used in the study. In order to establish the importance of CVR as an outcome, the predictive validity of CVR in relationship to cardiovascular disease is reviewed. The pathophysiological processes responsible for the negative health effects of CVR are described. The impact of cigarette smoking on the reactivity of the cardiovascular system is examined, including studies analyzing the combined effects of cigarette smoking and stress on CVR.

The second half of the literature review focuses on evidence that trait hostility, state angry affect, and state anxious-affect account for some of the effects of stress on CVR. As a foundation for this logic, theories describing affect as the mediator between stress and CVR are reviewed. Evidence linking trait hostility, state angry affect, and state anxious-affect with CHD and CVR is reviewed.

Review of Literature

Cardiovascular Reactivity and the Psychophysiological Reactivity Model

The reactivity of the cardiovascular system is the subject of continued empirical interest (Linden, Gerin, & Davidson, 2003). Cardiovascular reactivity (CVR) is defined as the "magnitude or pattern of an individual's hemodynamic responses to behavioral stressors" (Treiber, Kamarck, Schneiderman, Sheffield, Kapuku, & Taylor, 2003, p. 46). The importance of CVR as an outcome stems from its prominence within the broader psychophysiological reactivity model, the dominant theory by which psychosocial variables are purported to cause physical disease outcomes. According to the

psychophysiological reactivity model, psychosocial variables directly cause activation of physiological processes. This activation is damaging when it is frequent, intense, and long lasting (Cohen & Rodriguez, 1995; Gerin & Pickering, 1995; Manuck, Marsland, Marsland, Kaplan, & Williams, 1995). Physiological reactivity is also considered harmful when there is slow recovery time from an aroused to a resting state (Glynn, Christenfeld, & Gerin, 2002; Mayne, 2001).

Association between Cardiovascular Reactivity and Cardiovascular Disease

Evidence is accumulating that the reactivity of the cardiovascular system in response to behavioral stressor is associated with negative physical health outcomes. There is a growing body of literature from both human and animal models linking cardiovascular reactivity with cardiovascular disease (CVD) (Manuck et al., 1995). In a review of prospective studies, Treiber et al. (2003) found that CVR is associated with numerous risk factors for CVD, including the development of hypertension, increased left ventricular hypertrophy, and the development of atherosclerosis in the carotid arteries. CVR predicts new coronary events in populations with essential hypertension and coronary heart disease (CHD). There is limited evidence linking CVR with clinical events of CHD in initially healthy samples.

Pathophysiological Mechanisms linking Cardiovascular Reactivity with Cardiovascular Disease

The associations between behavioral stressors and CHD are primarily mediated through activation of the autonomic nervous system and endocrine system (Manuck et al., 1995), via the sympathetic-adrenomedullary and pituitary-adrenocortical axes (Cacioppo,

1994; Manuck et al., 1995; Taylor, Repetti, & Seeman, 1997). CVR leads to the development of CVD by accelerating heart rate and blood pressure, causing increases in blood flow with enough force to damage the coronary endothelium and initiate coronary lesions (Knox, 2001). The specific processes by which CVR leads to CHD are thoroughly described (Knox, 2001; Sloan, Shapiro, Bagiella, Myers, & Gorman, 1999; Spence, Barnett, Manuck, & Jennings, 1996).

Effects of Cigarette Smoking on Cardiovascular Reactivity

The cardiovascular system is not only reactive to behavioral stressors; chemical stressors such as cigarette smoking also cause reactivity of the cardiovascular system. Cigarette smoking has excitatory effects on the heart and vascular system, increasing heart rate, blood pressure, cardiac output, and total systemic resistance (Dembroski & MacDougall, 1986; Hausberg, Kosch, & Barenbrock, 2002). Research clearly demonstrates that smoking causes acute increases in blood pressure (Pickering, 2001; Primatesta, Falaschetti, Gupta, Marmot, & Poulter, 2000). The activation of the cardiovascular system in response to smoking is one mechanism by which smoking exerts its pathogenic effects as a risk factor for CVD (Khot et al., 2003), and in the development of premature CHD (Keil et al., 1998). These negative health effects of smoking combined with the high rates of smoking (Wechsler, Rigotti, Gledhill-Hoyt, & Lee, 1998) justify continued research addressing the cardiovascular consequences of smoking (Girdler, Jamner, Jarvik, Soles, & Shapiro, 1997).

Inter-Relationship between Cigarette Smoking and Stress

There is a cyclical relationship between cigarette smoking and stress. Cigarette smoking is used as a method of coping with stress. Smokers report that smoking helps relieve feelings of stress (Parrott, 1999). Perceived stress is associated with more frequent urges to smoke and increased smoking behavior (Todd, 2004). Periods of nicotine abstinence are associated with increased stress levels in persons dependent on nicotine. It is important to understand the cumulative effects of smoking and stress on CVR because each of these stimuli either activate the sympathetic nervous system or mimic its effects (Girdler et al., 1997). Nicotine's exacerbation of CVR to stress is a potential mechanism whereby smoking increases risk for CHD.

Effects of Smoking and Stress on Cardiovascular Reactivity

Studies examining the effects of smoking and stress on the cardiovascular system found that the amount of CVR to smoking is similar to the amount of CVR to stress (MacDougall et al., 1983). When smoking and stress are combined, the cardiovascular consequences are not clear. Some researchers assert that the joint effects of smoking and stress on CVR are greater (Dembroski, MacDougall, Cardoza, Ireland, & Krug-Fite, 1985), or at least double in magnitude (MacDougall, Dembroski, Slaats, Herd, & Eliot, 1983), compared to the effects of either smoking or stress alone on CVR. However, other researchers conclude that smoking does not enhance or even lowers CVR to stress (MacDougall, Musante, Castillo, & Acevedo, 1988).

Relationship between Affective States / Traits and Cigarette Smoking

Just as there are relationships between cigarette smoking and stress, there are also associations between smoking and affective states and traits. Mood control is a primary reason for smoking (Parrott, 1994). Affective traits such as hostility are associated with an increased likelihood of being a cigarette smoker (Scherwitz et al., 1992; Siegler et al., 2003; Whiteman, Fowkes, Deary, & Lee, 1997). Because of the inter-relationships between smoking, stress, and affective variables, it is important to understand how they jointly impact CVR. Psychosocial variables such as personality traits are postulated to interact with risk factors such as smoking to impact disease (Eysenck, 1991; Knox, 2001). As a foundation for examining how affective variables influence CVR to stress and smoking, the remainder of the literature review addresses the role of these affective variables on CVR to stress.

Psychosocial Variables Affecting Cardiovascular Reactivity

Researchers are attempting to identify psychosocial variables that can explain the CVR occurring in response to behavioral stressors. With the increasing evidence for a link between psychological variables and cardiac mortality, there is a need to identify the psychophysiological mechanisms responsible for this association (Hughes & Stoney, 2000). Since the cardiovascular reactivity model is the prominent explanation for how psychological variables impact CHD, continued research on psychosocial variables that may moderate the effects of CVR on CHD is warranted (Treiber et al., 2003).

Affective Variables Mediating the Effects of Stress on Cardiovascular Reactivity

Affective traits and states figure prominently in the search for variables that mediate the effects of stress on CVR. This approach is consistent with theories postulating that negative emotions mediate the effects of psychological processes on disease outcomes (Feldman, Cohen, & Lepore, 1999). Stressors are postulated to cause negative affective states, which in turn alter physiological processes (Burns, 1995; Cohen, Tyrrell, & Smith, 1993; Herbert & Cohen, 1993a), increasing risk for disease (Cohen & Williamson, 1991; Herbert & Cohen, 1993b).

Associations between Affective Variables and Coronary Heart Disease

Hostility, anger, and anxiety received considerable attention in the search for affective variables explaining stress induced CVR. These affect-related variables are logical candidates, as each is associated with cardiovascular disease outcomes. Hostility is an established risk factor for CHD (Booth-Kewley & Friedman, 1987; Helmers, Posluszny & Krantz, 1994; Smith, 1992), independent of other risk factors (Miller, Smith, Turner, Guijarro, & Hallet 1996). Trait anger is associated with documented CHD in prospective studies (Kawachi, Sparrow, Spiro, Vokonas, & Weiss, 1996; Williams et al., 2000). Acute episodes of anger are associated with increased risk of premature CVD (Chang, Ford, Meoni, Wang, & Klag, 2002), ischemia (Gabbay et al., 1996), and the onset of myocardial infarction (Mittleman et al., 1995; Moller et al., 1999). Anxiety is associated with increased ischemic and arrhythmic complications following myocardial infarction (Moser & Dracup, 1996).

The associations between hostility, anger, and anxiety with CVR are now reviewed.

Associations between Affective Variables and Cardiovascular Reactivity

Trait Hostility and Cardiovascular Reactivity

Inconsistencies are reported in the literature regarding the relationship between trait hostility with CVR (Houston, 1994; Suls & Wan, 1993), and ambulatory blood pressure levels (Schum, Jorgensen, Verhaeghen, Sauro, & Thibodeau, 2003). Studies found increases in hostility associated with hyperreactivity, hyporeactivity, or null effects on the cardiovascular system. These inconsistent findings are explained by observations that hostility only predicts CVR when stressors are interpersonal in nature (Suarez, Harlan, Peoples, & Williams, 1993; Suarez & Williams, 1989; Suls & Wan, 1993). The mixed findings are also attributed to variations in the methods of assessing hostility (Suls & Wan, 1993), with Structured Interview based measures of hostility (SI; Dembroski, 1978) demonstrating the most consistent associations with CVR.

State Anger and Cardiovascular Reactivity

Anger is associated with increased CVR (Siegman, 1993; Suarez, Kuhn, Schanberg, Willams, & Zimmermann, 1998). Early psychophysiological studies linked anger with physiological arousal (Ax, 1955), with definitions of anger referring to this physiological arousal (Spielberger, Reheiser, & Sydeman, 1995).

Anxiety Tension and Cardiovascular Reactivity

Trait anxiety is associated with greater ambulatory BP levels (Raikkonen, Matthews, Flory, Owens, & Gump, 1999), as well as increased BP over time (Markowitz,

Matthews, Wing, Kuller, & Meilahn, 1991; Paterniti et al., 1999). Tension is associated with development of hypertension (Markowitz, Matthews, Kannel, Cobb, & D'Agostino, 1993).

Relative Contributions of Hostility and Anger on Cardiovascular Reactivity

Researchers questioned the relative contributions of trait hostility and state anger on cardiovascular outcomes. These inquiries centered on whether or not hostility is independently associated with cardiovascular outcomes or if the effects of hostility are due to experiences of anger (Siegman, 1994). There are conceptual distinctions between the terms that warrant the analysis of their separate effects.

Definition of trait hostility.

Hostility is defined as "a devaluation of the worth and motives of others, an expectation that others are likely sources of wrongdoing, a relational view of being in opposition toward others, and a desire to inflict harm or see others harmed" (Smith, 1994, p. 26). Hostility is generally described as a cognitive (Miller et al., 1996), or attitudinal trait (Eckhardt, Barbour, & Stuart, 1997; Eckhardt, Norlander, & Deffenbacher, 2004; Siegman, 1994; Smith & Ruiz, 2002; Spielberger et al., 1995). Hostility is not typically considered an emotion (Kubzansky & Kawachi, 2000).

Definition of state anger.

In contrast to the cognitive focus of the term hostility, anger is regarded as an emotional state (Spielberger et al., 1995), with emphasis on subjective feelings (Spielberger, Jacobs, Russell, & Crane, 1983). Anger is defined as "an unpleasant emotion of varying intensity from mild irritation to rage" (Miller et al., 1996, p. 322).

State anger is described as a "psychobiological state or condition consisting of subjective feelings that vary in intensity, from mild irritation or annoyance to intense fury and rage, with concomitant activation or arousal of the autonomic nervous system" (Spielberger et al., 1995, p. 55).

Anger is a logical candidate as a mediator between hostility and CVR, as this is consistent with trait-state concepts described in the personality literature. "The broader trait disposition is seen as underlying the types of states likely to be readily activated within the person" (Mischel & Shoda, 1998, p. 235). Individuals high in trait hostility (Eckhardt & Deffenbacher, 1995; Smith & Frohm, 1985), and trait anger (Spielberger et al., 1995), experience state anger more frequently and intensely.

Researchers addressing the role of anger and negative affect as mediating or moderating the effect of hostility on CVR reported inconsistent findings (Fredrickson et al., 2000; Raikkonen, Matthews, Flory, & Owens, 1999; Suarez, Kuhn, Schanberg, Williams, & Zimmermann, 1998; Suarez et al., 1993; Suarez & Williams, 1989, 1990). Kurylo and Gallant (2000) advocated for the continued study of hostility, negative mood, and reactivity. The present investigation examines the differential ability of trait hostility versus state angry affect to explain CVR to smoking and stress.

How this Study Expands upon Previous Work

The present investigation addresses the conflicting findings reported in the literature regarding the effects of smoking and stress on CVR. Specifically, this study attempts to clarify how CVR to stress is affected by prior cigarette smoking. This study

seeks to determine how smoking and stress combine to affect overall levels of CVR compared to smoking and stress alone.

This study attempts to improve upon previous work by addressing issues related to the characteristics of the comparison group, study sample size, order of the experimental manipulations, presentation of results, and influence of affective variables on the CVR occurring in response to smoking and stress. Each of these issues is addressed in turn.

Characteristics of the Comparison Group

Previous research examining the effects of smoking and stress on CVR did not use an adequate comparison group, as smokers served as subjects for even the non-smoking conditions. This did not allow for a true comparison of the cardiovascular effects of smoking and stress contrasted with the effects of stress alone independent of smoking status. In the current study, non-smokers are used as the comparison group.

Sample Size

The majority of prior studies used sample sizes of <20 per condition. The present study uses a much larger sample size, increasing the reliability of the effects reported.

Order of the Experimental Manipulations

In past work, researchers examined the cardiovascular effects of smoking after the smoking behavior ended, instead of the immediate cardiovascular consequences as smoking is occurring. The current study measures CVR at the time that smoking occurs. Previous studies often paired smoking with the presentation of the stressor or with

instructions to relax. The present study examines the effects of smoking alone on CVR, prior to presentation of the stressor.

Presentation of Results

In previous studies, the effects of smoking and stress on CVR were depicted jointly in the presentation of the results, so that the separate effects of smoking and stress on CVR could not be determined. This study examines more clearly the respective contributions of smoking and stress on CVR.

Influence of Affective Variables on Cardiovascular Reactivity to Smoking and Stress

The current study represents an extension of previous work by examining the influence of hostility, anger, and anxiety on CVR occurring in response to smoking and stress. While previous studies examined the influence of hostility on CVR to stress alone, the influence of hostility on CVR to smoking and stress was not examined.

Summary

This study seeks to understand the cardiovascular consequences of smoking and stress, alone and in combination with one another. An examination of the cardiovascular effects of smoking and stress is necessary because these stimuli often co-occur. People smoke more while under stress. People are likely to smoke and subsequently be exposed to stressful situations, in part due to the frequency of smoking behavior during the course of daily life. This investigation compares the cardiovascular responses of subjects who smoke and are exposed to stress to the cardiovascular responses of non-smoking subjects exposed only to stress. This study examines how exposure to cigarette smoking affects the CVR that occurs in response to stress.

This study represents an extension of previous research by examining if trait hostility, state anger, and state anxious-affect can account for any of the variability in CVR induced by smoking and stress. This research examines if the cardiovascular effects of smoking are so strong that trait hostility, state anger, and state anxious-affect account for no additional variance in CVR to smoking. Additionally, this study questions if trait hostility, state anger, and state anxious-affect predict CVR that occurs upon exposure to stress, even after accounting for the effects due to smoking. The present study seeks to examine the differential ability of trait hostility, state anger, and state anxious-affect to predict CVR to smoking and stress.

METHODS

Participants

Subjects were undergraduate students attending an introductory psychology course at a public university. Subjects were part of a larger intervention study targeting smoking cessation. Subjects were excluded from participation if they had diagnosed cardiovascular disease including hypertension or were taking medications with known effects on blood pressure, such as high blood pressure medication and antidepressants. A total of 227 subjects participated with an average age of 19.92 years (range from 17 to 38). Approximately 94% of subjects were between the ages of 18-24. The gender distribution included 117 males (ages 18-38, mean age 19.72), and 110 females (age range 17-36, mean age 20.13). The ethnic distribution of subjects included 71% Caucasian (N=160), 1.8% African American (N=4), 1.8% Native American (N=4), 11.5% Hispanic (N=26), and 10.6% Asian (N=24). Data for one subject was discarded due to failure to complete the experimental protocol. Subjects received ten dollars and credit in their introductory psychology course.

Procedure

The Human Subjects Committee at the University of Arizona approved all experimental procedures.

Subjects self-selected into either a smoking or nonsmoking experimental condition at the time that they signed up for the experiment. A total of 156 subjects participated in the smoking condition and 71 subjects participated in the non-smoking condition. Of the 156 subjects in the smoking condition, 48.7% were female (N=76), and

51.3% were male (N=80). Of the 71 subjects in the non-smoking condition, 47.9% were female (N=34), and 52.1% were male (N=37).

Upon arrival to the lab, subjects filled out an informed consent document. Subjects then completed a packet of questionnaires that took approximately one hour.

There were five different phases to the experiment, including baseline, smoking/sham smoking, video stress phase, Structured Interview (SI), and history/mental arithmetic stress phase. Blood pressure and heart rate readings were recorded using the subject's non-dominant arm, using the LifeSign automated vital sign monitor, model number 52SOP. During each of the five experimental phases, initial blood pressure cuff inflation began 15 seconds into the experimental phase and followed at 90-second intervals thereafter, yielding one reading every two minutes. Heart rate readings began 30 seconds into each experimental phase and followed at 30-second intervals. Subjects were seated throughout the experimental protocol.

During the baseline period, subjects were instructed to "Relax, as if you do not have any cares in the world." The baseline period lasted approximately 5 minutes.

Subjects who identified themselves as smokers engaged in paced smoking, lasting approximately five minutes. Subjects smoked a Marlboro Red filter cigarette. After the cigarette was lit, subjects were instructed by the experimenter to take an inhalation every 25 seconds until the cigarette was smoked within 1 cm of the filter. Blood pressure and heart rate readings followed the standardized protocol.

Subjects who identified themselves as nonsmokers engaged in 'sham' smoking, lasting five minutes. Subjects pretended to inhale from an unlit cigarette every 25

seconds as instructed by the experimenter. Blood pressure and heart rate readings followed the standardized protocol.

There were two different phases of the experiment that were each designed to elicit a state of mild stress, the video and history/mental arithmetic stress phases. These stressors included a low level of provocation, as the experimenter made comments indicating judgment of the subjects' performance and prompting the subjects to perform better. The order of the video and history/mental arithmetic stress phases was counterbalanced across subjects. During each of the two stress phases, blood pressure and heart rate readings followed the standardized protocol.

In the video stress phase, subjects performed a video-driving task under constant supervision by the experimenter. The video stress phase lasted four minutes. Subjects were instructed to drive at maximal speeds to make it difficult to perform the task without error. The experimenter constantly prompted subjects to perform the task more quickly and accurately.

In the history/mental arithmetic stress phase, subjects responded verbally to questions regarding American history interpolated with questions that required them to perform mental subtraction. The history/mental arithmetic stress phase lasted four minutes. Subjects were told that their performance would be compared to other participants and the experimenter behaved as though grading their responses. The experimenter frequently prompted subjects to respond quickly and accurately.

In between these two stress phases, subjects were administered the SI (Dembroski, 1978; Rosenman, 1978). The SI involves a series of questions to assess

attitudes and behavioral responses to various scenarios designed to elicit feelings related to job satisfaction, displays of anger, competitive drive, impatience, and irritation. The questions were asked in a business-like fashion with probes requiring the subjects to explain themselves. Responses to the SI were tape recorded per subject permission. Administering the SI took varying amounts of time for different subjects.

Following the last stress phase, subjects were disconnected from the blood pressure and heart rate monitors. Subjects completed a measure of state negative affect. Subjects were debriefed as to the purpose of the experiment and provided information on the physical health consequences of smoking. Subjects completed a brief true-false test regarding the health education information covered during the debriefing.

General Analysis Methods

All statistical analyses used two-tailed tests of significance with alpha levels of .05. All predictors were analyzed as continuous variables, with the exception of variables that were dichotomous by their nature (i.e. gender). Degrees of freedom for the different statistical tests varied due to missing data. For bivariate correlations, pairwise deletion was used to handle missing data. Levene's test for equality of variances was used and significant differences noted.

Measures

Subjects completed measures of demographics, health history, life-style, smoking behavior, and psychosocial constructs. The following description of measures is organized according their use as independent variables, dependent variables, and covariates.

Unless otherwise noted, measures used as independent variables and covariates had 4 response options ranging from 0 to 3, corresponding to the categories of "mostly false, somewhat false, somewhat true, and mostly true." Items worded in the opposite direction were reverse coded rather than subtracted from the total summary score. Items were summed to yield total scores, with higher scores reflecting more of the variables of interest.

Independent Variables

Subjects completed three measures of trait hostility including the Total Potential for Hostility (POHO; Dembroski, 1978; Dembroski et al., 1985), the Anger Content Scale from the MMPI-2 (Butcher, Graham, Williams, & Ben-Porath, 1990), and the Cook-Medley Hostility Scale (Ho; Cook & Medley, 1954). These measures of trait hostility are among the most commonly used in research examining CVR outcomes. For the descriptive statistics and bivariate analyses, scores for the POHO, Anger Content Scale, and Ho Scale were analyzed separately using their original metrics. For the regression analysis, scores for the POHO, Anger Content Scale, and Ho Scale were standardized and summed to create a single indicator of hostility. Each measure of hostility is described below:

Total Potential for Hostility

Total Potential for Hostility (Dembroski, 1978; Dembroski et al., 1985) reflects a relatively stable personality trait involving the tendency to respond to life events with anger, irritability, and resentment, leading to expressions of antagonism, disagreeableness, rudeness, etc. Total POHO is derived from responses to the Structured

Interview (SI; Dembroski, 1978; Rosenman, 1978). The Appendix includes the item content for the SI. Detailed descriptions of the scoring methods for the POHO are available (Dembroski, 1978; Dembroski & Costa, 1987; Dembroski & MacDougall, 1985).

Ted Dembroski trained two graduate level research assistants on scoring procedures for the SI. The research assistants jointly scored the SI using tape recordings of the interviews. Disagreements in score assignment between the research assistants were resolved by re-analysis of the interview, and/ or averaging of scores. Subjects' responses to the SI were scored for hostile content, intensity, and style using the following criteria:

1. Hostile content- Subject's responses referring to frequent and consistent reports of being angry, mad, or irritated.
2. Intensity- Subject's responses involving the repeated emphasis on negative affect including the use of modifiers such as obscene and emotionally laden words, harsh generalizations, a mean voice quality, and voice mannerisms including loudness.
3. Style- Subject's interaction with the interviewer, such as being argumentative, contemptuous, disdainful, sarcastic, uncooperative, challenging, bored, not interested, and using a hostile tone.

Score assignment was based on a five-point scale using the following anchors (Dembroski, 1978):

1 = No or very few statements with hostile content and no hostile voice stylistics.

3 = Some potentially hostile statements and some suggestion of hostility in voice stylistics.

5 = Frequent hostility expressed in content, form, and stylistics.

Subject's scores for hostile content, intensity, and style were integrated to determine the total POHO score.

Interrater reliability of Total POHO typically ranged between .70 and .85 (Dembroski & Costa, 1987). Total POHO scores of 3 and above predicted incidence of CHD (Dembroski, MacDougall, Costa, & Grandits, 1989).

Anger Content Scale from the MMPI-2

The Anger Content Scale was developed from the MMPI-2 using rational and statistical methods (Graham, 2000). The scale includes 16 items assessing tendencies towards hostile thoughts and expressions, including problems related to anger control, being physically abusive, etc. The Appendix includes the item from the Anger Content Scale.

In a normative sample, internal consistency for the Anger Content Scale was reported at .75, with a test-retest reliability of .84 (Butcher, et. al., 1990). Convergent validity of the Anger Content Scale was established in part by an examination of its intercorrelations with other anger scales (Schill & Wang, 2000). In terms of its predictive validity in relationship to cardiovascular disease outcomes, males with high scores on the Anger Content Scale had significantly greater relative risks for documented CHD and all coronary events (Kawachi et al., 1996).

Cook-Medley Hostility Scale

The Cook-Medley Hostility Scale (Cook & Medley, 1954) is regarded as a measure of cynical hostility (Smith & Frohm, 1985), based on the cognitive components of hostility (Barefoot & Lipkus, 1994), with other aspects of negative affectivity reflected in the scale (Han, Week, Calhoun, & Butcher, 2000).

The Ho scale was originally composed of fifty items from the MMPI. There is no agreed upon factor structure of the Ho (Contrada & Jussim, 1992). Numerous researchers attempted to clarify its' factor structure (Barefoot, Dodge, Peterson, Dahlstrom, & Williams, 1989; Contrada & Jussim, 1992; Costa, Zonderman, McCrae, & Williams, 1986; Greenglass & Julkunen, 1989; Han et al., 2000; Houston, Smith, & Cates, 1989).

Barefoot et al. (1989) analyzed the item content of the Ho guided by theory and the face validity of the items. They identified a 13-item cynicism factor representing a general negative view of mankind, as distinct from the experience of affect. Han et al. (2000) replicated this cynicism factor. The present investigation included the 13 item cynical factor identified by Barefoot et al. (1989). See Appendix for the item content.

The predictive validity of the cynicism factor identified by Barefoot et al. (1989) demonstrated mixed results. Barefoot et al. (1989) reported that the cynicism factor predicted all cause mortality in a linear, dose-response manner. Using this same cynicism factor, Siegman, Townsend, Blumenthal, Sorkin, and Civelek (1998) were not able to predict documented CHD when traditional risk factors were controlled.

State Angry Affect and State Anxious-Affect

Subjects completed a measure of state negative affect. This measure was subjected to a confirmatory factor analysis using EQS Version 5.1, which confirmed the presence of the two factors of angry and anxious-affect. The state angry affect measure included the sum of items angry, peeved, resentful, and grouchy. The state anxious-affect measure included the sum of items tense, anxious, relax (reverse coded), on edge, and nervous. For both state affect measures, subjects rated their feelings at the time of administration including the past half-hour of the experimental procedures. Response options ranged from 0 to 4, corresponding to the categories of "not at all, a little, moderately, quite a bit, or extremely," with higher scores representing more of the attribute.

Dependent Variables

Measures of diastolic blood pressure (DBP), systolic blood pressure (SBP), and heart rate (HR) served as dependent variables. For analysis purposes, this physiological data was organized in two formats: (a) Averages were calculated for each of DBP, SBP and HR for each experimental phase, (b) A composite measure was calculated for each experimental phase that summed the standardized averages of DBP, SBP, and HR.

For the descriptive statistics and bivariate analyses, the averages for DBP, SBP, and HR for each experimental phase were used. For the regression analysis, the composite measure was used.

The averages of DBP, SBP, and HR for each experimental phase were calculated according to the following methods: Averages were computed using all available data

for each subject during each experimental phase, with missing data simply excluded from the computation of the average of each experimental phase. For the baseline and smoking experimental phases, averages were computed using the first three DBP readings, the first three SBP readings, and the first eight HR readings. For the stress experimental phase, averages were computed for the video and math/history stress phases combined using the first three DBP readings, the first three SBP readings, and the first eight HR readings. This stress experimental phase average score represented an averaging of the type of stressor and an averaging of the order of the stressor and its proximity to smoking, (as the order of the video and math/history stressors following smoking were counterbalanced across subjects).

The composite measure was calculated according to the following methods: Averages of DBP, SBP and HR for each experimental phase were standardized and summed together so that a single CVR measure was generated for each experimental phase including baseline, smoking intervention, and stress.

Potential Confounds

Numerous demographic, health history, and life-style variables were assessed as potential confounds. These variables were defined as follows:

1. Gender
2. Ethnicity
3. Body mass index calculated using the formula= weight (kg)/height $(m)^2$.
4. History of high blood pressure rated separately for father and mother (dichotomous response options).

5. Use of medication affecting blood pressure, including antidepressants, diet pills, tranquilizers, and insulin (single item with dichotomous response options).

6. Use of birth control pills (dichotomous response options).

7. Caffeine /alcohol consumption and exercise practices during the past month. Response options ranged from 0 to 9, corresponding to "never, 1-3 per month, 1-2 per week, 3-4 per week, 5-6 per week, 1 per day, 2 per day, 3 per day, 4 per day, 5 or more per day." The Appendix includes the item content for the caffeine, alcohol, and exercise scales.

8. Chronic Sleep problems. The Appendix includes the item content for the sleep problems scale.

9. Symptom Checklist 90-Revised (SCL-90-R) (Derogatis, 1993), which included the subscales of Anxiety, Depression, Interpersonal, and Somatic Complaints. Subjects rated how distressed they were by the various symptoms during the past seven days, including the day of test administration. Response options ranged from 0 to 4, corresponding to the categories of "not at all, a little, moderately, quite a bit, or extremely," with higher scores indicating more of the attribute.

RESULTS

Preliminary Analyses

Effects due to Potential Confounds

The potential confounds were used in preliminary analyses to determine if they contributed significant variance in CVR. Bivariate correlations were calculated for each of the potential confounds and baseline physiological data. Out of these bivariate analyses, the only covariates that were occasionally predictors of baseline physiological data were gender, age, BMI, frequency of exercise, and alcohol use. Each of these variables was entered before the affect measures into preliminary regression equations separately for smokers and non-smokers, for each of the outcomes of DBP, SBP, and HR. Age was the only potential confound that infrequently predicted CVR. Gender was not predictive in any of the regression models. The variance that could be attributed to gender was likely accounted for by the baseline physiological measures included in the first step of these models. Overall, none of the potential confounds reliably accounted for any significant variance in CVR outcomes and so they were not included in the final regression analysis.

Effects due to Non-Random Assignment

Subjects were not randomly assigned to the smoking versus non-smoking conditions. The question arose as to whether or not CVR in response to smoking could be attributed to pre-existing differences between smokers and non-smokers rather than to the actual smoking behavior. Characteristics of smokers and non-smokers were

compared. Table 1 displays the means and standard deviations of the independent variables and potential confounds for the entire sample and by smoking status.

Smokers were slightly older and reported significantly more somatic complaints via the SCL-90- R ($t=-2.41$, $df=221$, $p<.017$), more caffeine consumption ($t=-4.94$, $df=223$, $p<.000$), more chronic sleep problems ($t=-3.15$, $df=219$, $p<.002$), and more hostile style on the SI ($t=2.94$, $df=140$, $p<.004$). Smokers were not significantly different on parental history of hypertension, use of birth control pills, use of medication affecting blood pressure, BMI, subscales on the SCL-90-R (anxiety, depression, interpersonal), alcohol consumption, frequency of exercise, trait hostility, state angry affect, or state anxious-affect.

While there were differences between the smokers and non-smokers on variables that could affect CVR, smoking has well known effects on CVR that are empirically established. The increased levels of CVR from baseline that occurred immediately following the start of smoking suggested that smoking was responsible for increases in CVR beyond any effects due to pre-existing differences between smokers and non-smokers.

Table 1

Means and Standard Deviations of Independent Variables and Confounds for Total Sample and by Smoking Status

Measure	Total (mean ± SD)	Smokers (mean ± SD)	Non-smokers (mean ± SD)
Age	19.92 ± 3.09	20.16 ± 3.49*	19.38 ± 1.86*
BMI	23.51 ± 4.73	23.57 ± 4.71	23.39 ± 4.80
Exercise	4.04 ± 3.07	3.92 ± 3.03	4.32 ± 3.15
Caffeine	5.83 ± 3.48	6.57 ± 3.44*	4.23 ± 3.01*
Alcohol	5.33 ± 4.44	6.11 ± 4.38	3.61 ± 4.12
Sleep problems	5.91 ± 4.86	6.60 ± 4.97*	4.43 ± 4.29*
SI Total POHO	2.76 ± .87	2.76 ± .84	2.76 ± .95
SI Content	2.84 ± .61	2.83 ± .63	2.88 ± .56
SI Intensity	1.31 ± .57	1.28 ± .51	1.41 ± .70
SI Style	1.46 ± .67	1.53 ± .69*	1.27 ± .52*
Anger Content	13.95 ± 8.12	14.42 ± 8.26	12.91 ± 7.74
Cook-Medley Ho	16.42 ± 6.43	16.38 ± 6.51	16.50 ± 6.29
State Angry Affect	1.06 ± 1.98	1.14 ± 2.23	.89 ± 1.29
State Anxious-Affect	8.67 ± 4.32	8.70 ± 4.33	8.61 ± 4.33
SCL-90-R Anxiety	5.07 ± 4.72	5.11 ± 4.71	5.00 ± 4.79
SCL-90-R Dep	8.49 ± 7.82	8.87 ± 7.95	8.00 ± 7.69
SCL-90-R Interp	5.36 ± 4.93	5.52 ± 5.01	5.00 ± 4.77
SCL- 90-R Somatic	4.71 ± 3.75	5.11 ± 3.84*	3.81 ± 3.41*

Note. BMI = Body Mass Index; SI = Structured Interview; POHO = Potential for Hostility; SCL-90-R = Symptom Checklist-90-Revised; Dep = Depression; Interp = Interpersonal.

*$p<.05$

Bivariate Analyses

The intercorrelations between the hostility and state affect variables are displayed in Table 2 for smokers and Table 3 for non-smokers.

Table 2

Intercorrelations of Hostility Measures and State Affect for Smokers

Variables	1	2	3	4	5	6	7	8
1. SI Total POHO	-							
2. SI Content	.58**	-						
3. SI Intensity	.54**	.30**	-					
4. SI Style	.54**	.06	.28**	-				
5. Anger Content	.37**	.34**	.24**	.09	-			
6. Ho Scale	.31**	.19*	.20*	.10	.49**	-		
7. Angry Affect	.08	.14	.02	.06	.21**	.22**	-	
8. Anxious-Affect	-.04	.04	-.18*	-.11	.15	.11	.34**	-

Note. SI = Structured Interview.

*p<.05. **p<.01.

Table 3

Intercorrelations of Hostility Measures and State Affect for Non-Smokers

Variables	1	2	3	4	5	6	7	8
1. SI Total POHO	-							
2. SI Content	.63**	-						
3. SI Intensity	.64**	.52**	-					
4. SI Style	.48**	.23	.50**	-				
5. Anger Content	.61**	.56**	.34**	.35**	-			
6. Ho Scale	.25	.33*	.06	.21	.53**	-		
7. Angry Affect	.41**	.40**	.32**	.36**	.41**	.25*	-	
8. Anxious-Affect	.02	.29*	.16	.02	.00	-.06	.45**	-

Note. SI = Structured Interview.

*$p<.05$. **$p<.01$.

Correlations between trait hostility and state affect revealed slightly different patterns for smokers and non-smokers. For non-smokers, state angry affect was significantly related to SI scores while for smokers, state angry affect was not related to SI scores.

For both smokers and non-smokers, there were low correlations between the hostility measures and state anxious-affect, suggesting differences between these constructs.

The intercorrelations for the mean physiological indices for each experimental phase are displayed in Table 4 for smokers and Table 5 for non-smokers.

Table 4

Intercorrelations of Physiological Indices for Smokers

Variables	1	2	3	4	5	6	7	8
1. Baseline DBP	--							
2. Smoking DBP	.71**							
3. Stress DBP	.56**	.71**						
4. Baseline SBP	.63**	.53**	.50**					
5. Smoking SBP	.59**	.76**	.63**	.79**				
6. Stress SBP	.45**	.57**	.79**	.72**	.76**			
7. Baseline HR	.29**	.10	.18*	.21**	.11	.17*		
8. Smoking HR	.21**	.31**	.27**	.14	.30**	.25**	.71**	
9. Stress HR	.07	.17*	.28**	.06	.18*	.31**	.65**	.80**

Note. DBP = Diastolic Blood Pressure (mm Hg); SBP = Systolic Blood Pressure (mm Hg); HR = Heart Rate (bpm).

*p<.05. **p<.01.

Table 5

Intercorrelations of Physiological Indices for Non-Smokers

Variables	1	2	3	4	5	6
1. Baseline DBP	--					
2. Stress DBP	.75**	--				
3. Baseline SBP	.73**	.69**	--			
4. Stress SBP	.62**	.86**	.82**	--		
5. Baseline HR	.01	.04	-.01	.06	--	
6. Stress HR	.13	.24*	.06	.27*	.67**	--

Note. DBP = Diastolic Blood Pressure (mm Hg); SBP = Systolic Blood Pressure (mm Hg); HR = Heart Rate (bpm).

*p<.05. **p<.01.

For both smokers and non-smokers, correlations between different blood pressure readings were higher than correlations between blood pressure and heart rate readings. Similarly, correlations between different heart rate readings were higher than correlations between blood pressure and heart rate readings. Correlations between blood pressure and heart rate readings were generally higher for smokers compared to non-smokers.

The absolute levels of physiological indices were compared for smokers and non-smokers using Independent Samples T-Tests (see Table 6).

Table 6

Means and Standard Deviations for Physiological Indices for Smokers and Non-Smokers

	Smokers (mean ± SD)	Non-Smokers (mean ± SD)	*t*	*df*	*p*
DBP (mm Hg)					
Baseline	74.08 ± 8.84	70.01 ± 8.92	3.20	225	.002
Smoke/Sham	82.05 ± 9.54	68.96 ± 9.02	9.75	225	.000
Stress	85.01 ± 9.16	78.44 ± 9.93	4.88	225	.000
SBP (mm Hg)					
Baseline	118.78 ± 11.74	114.56 ± 10.89	2.57	225	.011
Smoke/Sham	124.57 ± 11.58	112.62 ± 9.93	7.53	225	.000
Stress	130.30 ± 12.10	121.24 ± 11.96	5.25	225	.000
HR (bpm)					
Baseline	75.76 ± 10.59	73.52 ± 11.05	1.46	225	.147
Smoke/Sham	84.47 ± 12.11	74.37 ± 9.48	8.82	170*	.000
Stress	89.05 ± 13.28	77.90 ± 8.95	7.42	193*	.000

Note. *Equal variances not assumed.

Compared to non-smokers, smokers had significantly higher baseline levels of DBP (t=3.20, df=225, p=.002) and SBP (t=2.57, df=225, p=.011). There were no significant differences in baseline HR levels between smokers and non-smokers.

In terms of physiological responses to smoking versus pretending to smoke, smokers demonstrated significantly higher levels of DBP (t=9.75, df=225, p=.000), SBP (t=7.53, df=225, p=.000), and HR (t=8.82, df=170, p=.000), compared to non-smokers.

Compared to subjects who did not smoke and were exposed to stress, subjects who smoked and were exposed to stress evidenced significantly higher levels of DBP (t=4.88, df=225, p=.000), SBP (t=5.25, df=225, p=.000), and HR (t=7.42, df=193, p=.000).

Results from Table 6 are graphically represented in Figures 1, 2, and 3, displaying the absolute levels of physiological indices across the experimental phases.

Figure 1. Absolute Levels of Diastolic Blood Pressure across Experimental Phases for Smokers and Non-Smokers

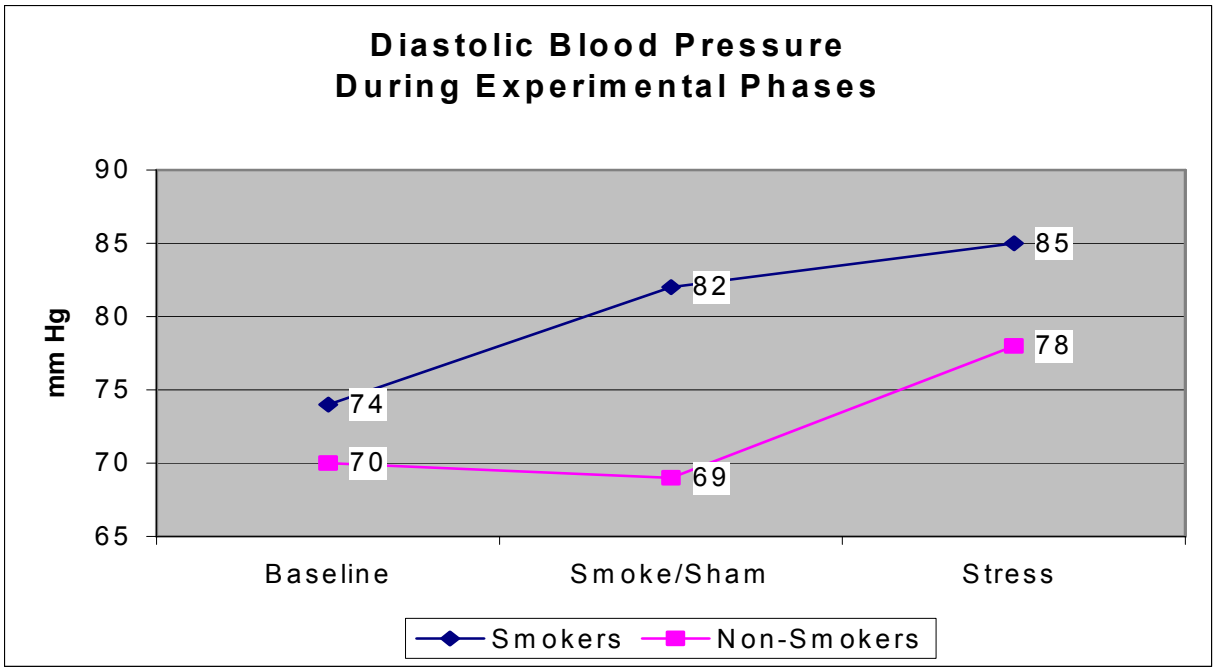


Figure 2. Absolute Levels of Systolic Blood Pressure across Experimental Phases for Smokers and Non-Smokers

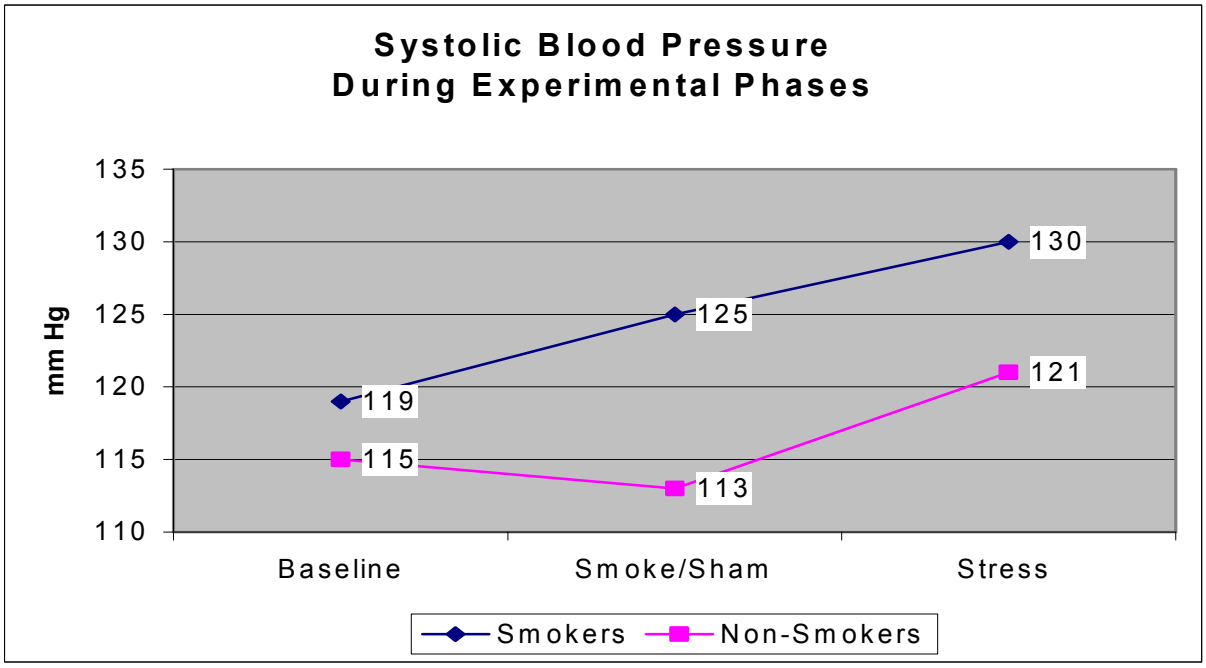
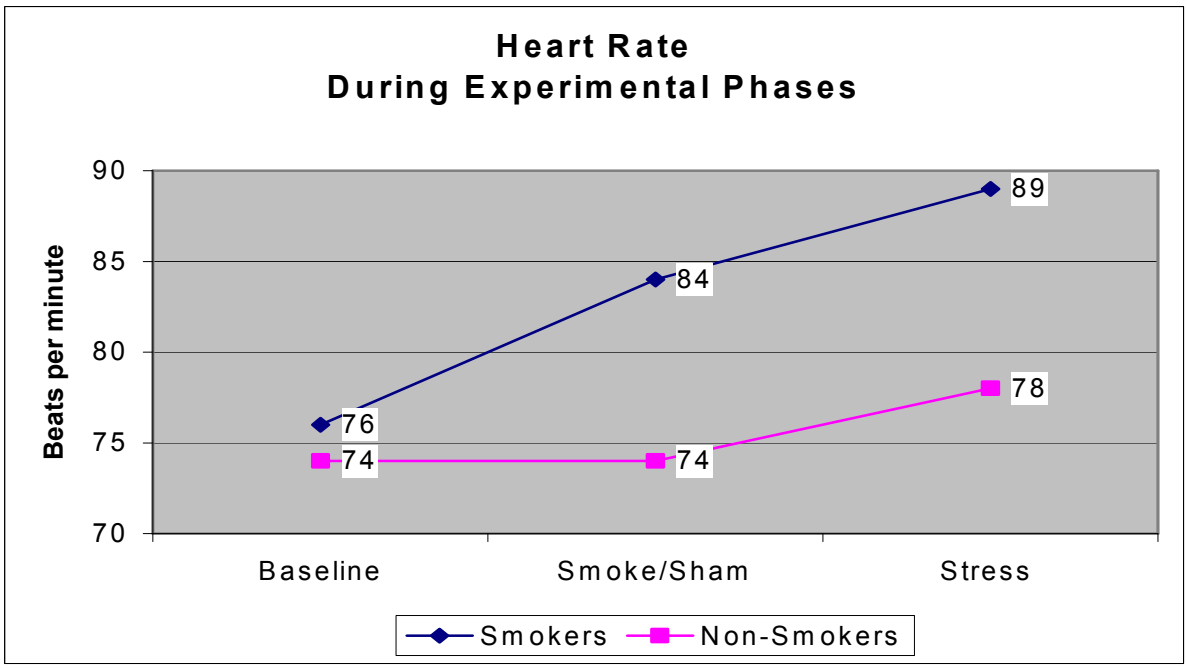


Figure 3. Absolute Levels of Heart Rate across Experimental Phases for Smokers and Non-Smokers



In order to account for baseline differences in physiological indices between smokers and non-smokers, cardiovascular reactivity was calculated by subtracting baseline levels from mean levels during the smoking and stress experimental phases. Comparisons in reactivity measures between smokers and non-smokers are displayed in Table 7.

Compared to subjects who pretended to smoke, subjects who smoked demonstrated significantly greater increases from baseline to smoking in DBP ($t=10.94$, $df=183$, $p=.000$), SBP ($t=9.00$, $df=191$, $p=.000$), and HR ($t=12.59$, $df=224$, $p=.000$). Cardiovascular reactivity evidenced by smokers in the present investigation was within range but less than previously reported (Ragueneau et al., 1999).

Cardiovascular reactivity was calculated from the time period of smoking or pretending to smoke until the stress experimental phase. For non-smokers, this basically represented a change from baseline to stress. Subjects who smoked first and then were exposed to stress demonstrated significantly smaller changes in DBP ($t=-6.45$, $df=225$, $p=.000$), SBP ($t=-2.62$, $df=225$, $p=.000$), and HR ($t=-1.78$, $df=225$, $p=.000$), compared to subjects who were exposed to stress without smoking.

When the overall amount of cardiovascular reactivity from baseline to stress was calculated, which included the time period of smoking, subjects who smoked and were exposed to stress demonstrated significantly greater increases in DBP ($t=2.19$, $df=225$, $p=.03$), SBP ($t=4.06$, $df=225$, $p=.000$), and HR ($t=6.89$, $df=165$, $p=.000$), compared to subjects who were exposed to stress without smoking.

Table 7

Comparison of Mean Physiological Reactivity between Smokers and Non-Smokers

	Smokers (mean ± SD)	Non-Smokers (mean ± SD)	*t*	*df*	*p*
DBP (mm Hg)					
Δ Baseline to Smoke/Sham	7.97 ± 7.05	-1.06 ± 5.07	10.94	183*	.000
Δ Smoke/Sham to Stress	2.96 ± 7.17	9.48 ± 6.80	-6.45	225	.000
Δ Baseline to Stress	10.93 ± 8.49	8.42 ± 6.74	2.19	225	.03
SBP (mm Hg)					
Δ Baseline to Smoke/Sham	5.79 ± 7.55	-1.95 ± 5.17	9.00	191*	.000
Δ Smoke/Sham to Stress	5.73± 8.15	8.62 ± 6.73	-2.62	225	.000
Δ Baseline to Stress	11.52 ± 8.88	6.67 ± 6.97	4.06	225	.000
HR (bmp)					
Δ Baseline to Smoke/Sham	11.72 ± 8.74	.86 ± 4.26	12.59	224*	.000
Δ Smoke/Sham to Stress	1.57 ± 8.11	3.54 ± 6.84	-1.78	225	.000
Δ Baseline to Stress	13.29 ± 10.32	4.39 ± 8.35	6.89	165*	.000

Note. Sham = sham smoking; SBP = systolic blood pressure; DBP = diastolic blood pressure; HR = heart rate; bpm = beats per minute. *Equal variances not assumed.

The correlations for physiological reactivity with trait hostility and state affect are displayed in Table 8 for smokers and Table 9 for non-smokers.

Table 8

Intercorrelations of Hostility and Affect with Change in Physiological Indices for Smokers

Variables	1	2	3	4	5	6	7	8
DBP Δ Base to Smoke	.04	.03	-.02	.00	-.15	-.14	-.26**	-.05
DBP Δ Smoke to Stress	-.17*	-.18*	-.05	-.02	-.08	-.09	.04	.05
DBP Δ Base to Stress	-.11	-.13	-.04	-.01	-.19*	-.19*	-.20*	.00
SBP Δ Base to Smoke	-.06	.05	-.08	-.07	-.11	-.17*	-.17	-.08
SBP Δ Smoke to Stress	-.09	-.16	-.07	.07	-.06	-.05	.01	.21**
SBP Δ Base to Stress	-.13	-.11	-.14	-.00	-.15	-.19*	-.14	.13
HR Δ Base to Smoke	-.08	-.04	-.02	-.10	-.15	-.16*	-.07	.03
HR Δ Smoke to Stress	-.17*	-.11	-.15	.02	-.10	-.04	-.02	.26**
HR Δ Base to Stress	-.19*	-.12	-.14	-.07	-.21**	-.17*	-.08	.23**

Note. 1.=SI Total POHO; 2.=SI Content; 3.=SI Intensity; 4.=SI Style; 5.=Anger Content; 6.= Ho Scale; 7.=Angry Affect; 8.=Anxious-Affect; DBP = Diastolic Blood Pressure (mm Hg); SBP = Systolic Blood Pressure (mm Hg); HR = Heart Rate (bpm).

*p<.05. **p<.01.

Table 9

Intercorrelations of Hostility and Affect with Change in Physiological Indices for Non-Smokers

Variables	1	2	3	4	5	6	7	8
DBP Δ Base to Stress	.10	.08	.02	.05	.03	-.04	-.06	.15
SBP Δ Base to Stress	-.13	-.05	-.05	-.19	-.03	-.14	-.02	.26*
HR Δ Base to Stress	-.13	-.04	-.06	-.24	-.11	.01	-.04	.09

Note. 1.=SI Total POHO; 2.=SI Content; 3.=SI Intensity; 4.=SI Style; 5.=Anger Content; 6.= Ho Scale; 7.=Angry Affect; 8.=Anxious-Affect; DBP = Diastolic Blood Pressure (mm Hg); SBP = Systolic Blood Pressure (mm Hg); HR = Heart Rate (bpm).

*$p<.05$.

For both smokers and non-smokers, the most striking finding was the relative consistency with which there were inverse associations between hostility / state angry affect and CVR. While only some of the correlations were significant, the negative correlations were regular across different measures of hostility / angry affect, different physiological indices, and across different experimental phases. In contrast to the inverse association between hostility / angry affect and CVR, state anxious-affect was generally positively associated with CVR.

Regression Analysis

General Analytical Procedure

A regression analysis was performed using SAS Release 8.2 for Windows. The analysis was conducted using a single regression equation to accommodate both smokers and non-smokers, analyzing outcomes for both the smoking and stress interventions.

For theoretical reasons, hostility was the most critical affect-related variable potentially related to CVR, with angry affect and anxious-affect following in importance. Smoking status was expected to have an effect on CVR beyond the effect of the affect-related variables. The smoking and stress interventions were expected to have effects beyond those of smoking status. Finally, interactions of variables could occur beyond the main effects of variables. Variables were entered into the regression equation in the following order:

1. Trait hostility measure
2. State angry affect
3. State anxious-affect
4. Smoking status
5. Smoking intervention
6. Stress intervention
7. Hostility measure, state angry affect, state anxious-affect and smoking status each interacting with the smoking and stress interventions

The smoking status variable was defined as the number of cigarettes currently smoked per day, in order to distinguish between smokers and non-smokers. The smoking

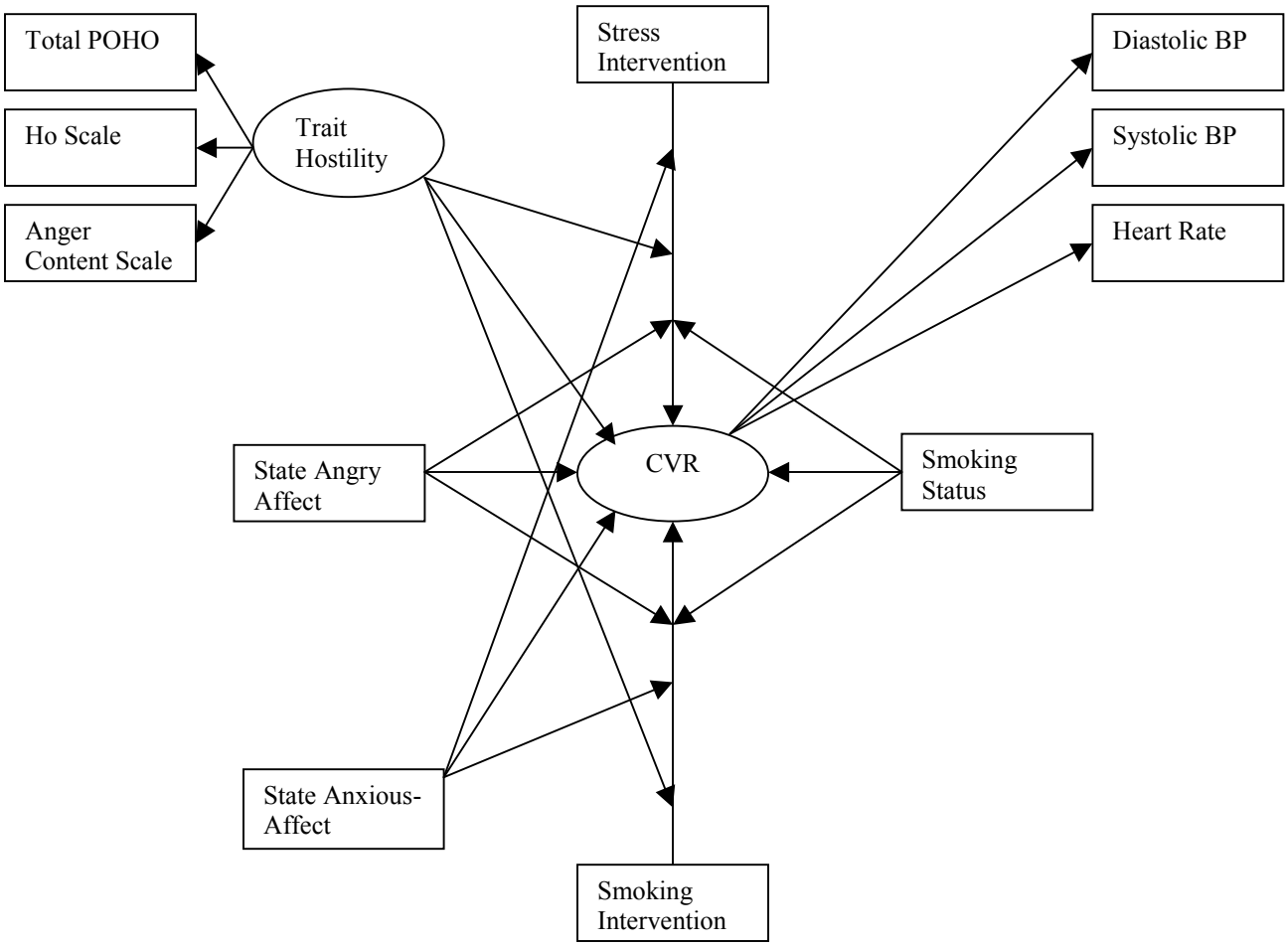
intervention variable represented CVR during the smoking / sham smoking phase of the experiment, regardless of actual smoking status. The stress intervention variable represented CVR during the stress phase of the experiment, regardless of smoking status. See Figure 4 for a diagram of the conceptual model used for analysis purposes.

Description of the Conceptual Model

The trait hostility measure and CVR represented the measurement components of the model. Trait hostility was defined as a composite of the three measured variables of Total POHO, the Ho Scale, and the Anger Content Scale. CVR was defined as a composite of the three measured variables of DBP, SBP, and HR. See the Methods section for a more thorough description of the procedures used for construction of these variables.

As displayed in the conceptual model, direct effects on CVR were calculated for trait hostility, state angry affect, state anxious-affect, smoking intervention, stress intervention, and smoking status. Interaction effects were calculated for trait hostility, state angry affect, state anxious-affect, and smoking status by the smoking and stress interventions.

Figure 4. Conceptual Model



The data set was organized in a multiple record format with subject code numbers included as part of all error terms. The proc glm model statement was used to determine the significance level for each variable after accounting for the effects of the preceding variables in the model (see Table 10). The independent variables found to be significant were run in two additional proc glms in order to calculate parameter estimates and to determine total variance accounted for by each model. One glm statement included the significant main effects, representing the relationship between the independent variables with the dependent variable across subjects during all conditions (see Table 11). Another glm statement included the significant interaction terms after taking into account the number of individual subjects. Results from this glm statement represented the effects of the independent variables with the dependent variable across subjects for a given experimental phase (see Table 12).

Regression Results

Table 10

Hierarchical Regression Analysis for Predicting Cardiovascular Reactivity

Variable	F Value	Pr > F
Hostility	.02	ns
Angry Affect	.41	ns
Anxious-Affect	9.02	<.01
Smoking Status	20.87	<.0001
Smoking Intervention	.31	ns
Stress Intervention	.01	ns
Hostility * Smoking Intervention	.01	ns
Hostility * Stress Intervention	8.15	<.01
Angry Affect * Smoking Intervention	3.18	ns
Angry Affect * Stress Intervention	1.84	ns
Anxious-Affect * Smoking Intervention	2.33	ns
Anxious-Affect * Stress Intervention	9.43	<.01
Smoking Status * Smoking Intervention	13.03	<.001
Smoking Status * Stress Intervention	5.28	<.05

Table 11

Regression Coefficients for Significant Main Effects

Variable	B	SE B
Anxious-Affect	.08	.02
Smoking Status	.76	.10
Total R^2 = .10; F Value = 35.44; p < .0001		

Table 12

Regression Coefficients for Significant Interaction Effects

Variable	B	SE B
Hostility * Stress Intervention	-.12	.05
Anxious-Affect * Stress Intervention	.03	.02
Smoking Status * Smoking Intervention	.22	.09
Smoking Status * Stress Intervention	.02	.11
Total R^2 = .83; F Value = 9.31; p < .0001		

Prediction of Cardiovascular Reactivity Across Experimental Phases

When trait hostility, state angry affect, and state anxious-affect were included as main effects in the model, they were attempting to predict CVR on average across the experimental phases of baseline, smoking intervention, and stress. Because these affect-related variables were entered first into the regression equation, they were attempting to account for CVR on average before partialling out the effects due to smoking status or

stress. State anxious-affect was only of these affect-related variables that significantly predicted average levels of CVR across experimental phases. The lack of a significant main effect for hostility across experimental phases is consistent with results in the literature indicating that hostility is not associated with baseline physiological levels. It is also quite likely that trait hostility and state angry affect were not strong enough predictors to account for CVR levels across experimental phases because of the influence of smoking status.

Aside from state anxious-affect, smoking status was the only other variable significantly related to CVR across experimental phases. When the effect due to smoking status was removed, the stress intervention did not significantly predict average levels of CVR across experimental phases. The lack of a significant effect of stress on CVR is likely the result of averaging across smokers and non-smokers. Smokers evidenced very little CVR to stress. With smokers composing nearly two-thirds of the sample, the main effect for the stress intervention across the entire subject population was diminished.

Prediction of Cardiovascular Reactivity During the Smoking Intervention

CVR during the smoking intervention phase represented an average level of CVR for both smokers and non-smokers. Smoking status was the only variable to predict CVR during the smoking intervention phase. Given that approximately two-thirds of the sample engaged in smoking, it is not surprising that CVR during the smoking intervention phase was predicted by smoking status. Possibly a better test of the influence of affective variables during smoking would be represented as a three-way interaction between the affective variable, smoking status, and the smoking intervention.

Prediction of Cardiovascular Reactivity During the Stress Intervention

CVR during the stress intervention represented an average level of CVR for all subjects regardless of smoking status. In the experimental protocol, exposure to stress followed the smoking intervention. Smoking status predicted CVR during the stress intervention. Results from descriptive statistics indicated that smokers showed significantly less CVR to stress compared to the non-smokers.

Trait hostility predicted CVR during the stress intervention across smokers and non-smokers. Hostility was inversely related to CVR, such that increases in hostility were associated with decreases in CVR to the stressor. State angry affect was not significantly related to CVR during the stress intervention after accounting for the effects due to trait hostility. State anxious-affect predicted CVR to stress, even after accounting for the effects of trait hostility and state anger. Increased levels of state anxious-affect were associated with increased levels of CVR during the stress intervention.

DISCUSSION

Effects of Cigarette Smoking and Stress on Cardiovascular Reactivity

This study examined the effects of smoking and stress on the reactivity of the cardiovascular system. Findings revealed that subjects who smoked first and who were then exposed to stress showed less CVR to stress compared to non-smoking subjects exposed only to stress. Despite the blunted CVR to stress that occurred following smoking, the combination of smoking and stress led to higher absolute levels and greater reactivity for all cardiovascular measures compared to stress alone.

When subjects were exposed to stress following either smoking or non-smoking, the non-smokers demonstrated significantly greater CVR to stress on all physiological indices compared to smokers. These results partially replicate those reported by MacDougall et al. (1988), where smokers demonstrated lower DBP responses to stress compared to non-smokers. However, in the MacDougall study, subjects who smoked showed the same amount of SBP and HR reactivity to stress as did the non-smokers. In the present investigation, the lowered CVR to stress for the smokers was evident for all cardiovascular measures.

How Does Smoking Moderate the Effects of Stress on Cardiovascular Reactivity?

Ceiling Effect on Cardiovascular Reactivity

Towards understanding the interaction effects of smoking and stress on the health of the cardiovascular system, it is important to explain why smoking moderated CVR to stress. One potential explanation for the lowered CVR to stress after smoking could be that there is a ceiling effect on normal CVR; smoking may affect CVR to such a degree

that there is not very much reactivity left upon exposure to stress following smoking. However, because subjects in the current investigation evidenced relatively small increases in CVR during smoking, it is difficult to imagine that ceiling effects on physiological reactivity blunted CVR to stress after smoking.

Cognitive / Emotional Attributions Affecting Physiological Arousal

Alternatively, there could be cognitive and emotion-related attribution processes that lessen the CVR response to stress after smoking. Smokers may attribute the physiological arousal associated with smoking in a positive context. Perhaps when they are exposed to stress, they are less likely to make negative attributions regarding any physiological arousal associated with the stress response. When the physiological arousal normally associated with stress is viewed within this positive attributional framework, perhaps it is diminished.

Smoking Reduces Negative Affect

Another hypothesis may be that smoking causes a reduction in negative affect and an increase in positive mood so that stressful events are perceived less negatively, evoking less physiological arousal. This is consistent with evidence linking positive mental states with healthier physiological functioning (Taylor, Lerner, Sherman, Sage, & McDowell, 2003).

In the present investigation, smoking did appear to alter either the experience and/or display of affect. This was apparent in a disconnection between smokers' responses to measures of trait hostility and state angry affect. After the smoking intervention, all subjects were administered the Structured Interview (SI), which is

designed to elicit hostile responses. For non-smokers, there were strong correlations between their hostile responses to the SI and their reports of state angry affect during the SI and other experimental phases. However, for smokers, there was no correlation between their hostile responses to the SI and state angry affect. It appears that smoking led subjects to respond to the measure of trait hostility in a manner that was not related to the amount of state angry affect that they reported experiencing. Unfortunately, the effects of smoking on the experience and reporting of negative affect are not well understood and remain an important area for future inquiry (Kassel, Stroud, & Paronis, 2003).

The Influence of Affective Variables on Cardiovascular Reactivity to Smoking and Stress

The secondary aim of this study was to examine the ability of trait hostility, state angry affect, and state anxious-affect to predict CVR to smoking and stress.

State anxious-affect was the only affect-related variable that predicted CVR across baseline, smoking/sham smoking, and stress experimental phases for both smokers and non-smokers. Increases in state anxious-affect were associated with increases in reactivity of the cardiovascular system. The effects of state anxious-affect on CVR appeared robust in that this affect variable was able to account for variance in CVR across experimental phases, despite the powerful influence of smoking on CVR and even after accounting for the effects of trait hostility and state angry affect.

None of the affect-related variables were able to predict CVR during smoking.

Trait hostility and state anxious-affect were able to predict CVR during stress for smokers and non-smokers. Trait hostility was negatively associated with CVR to stress,

while state anxious-affect was positively associated with CVR to stress. State angry affect did not predict CVR to stress after accounting for the effects due to trait hostility.

Inverse Association between Hostility / Anger and CVR

In the present investigation, there was a general trend for increases in trait hostility and state anger to be associated with decreases in CVR. The negative correlations between hostility / anger with CVR were evident for smokers and non-smokers across different measures of hostility, cardiovascular indices, and experimental phases.

The negative correlations between hostility and CVR replicate similar results reported in the literature. Inverse associations were also reported between other psychosocial variables such as stressful life events and CVR (Musante et al., 2000). Few researchers addressed the negative correlations between psychosocial variables and CVR. One explanation was that repeated exposure to stressors might attenuate the stress response (Eysenck, 1983 as cited in Musante et al., 2000). The negative correlation between hostility and CVR is important to explain, as it runs counter to the psychophysiological reactivity model that hostility exerts negative health effects via increased reactivity of the cardiovascular system.

Components of Hostility Differentially related to Cardiovascular Reactivity

One possible explanation for the inverse association between hostility and CVR is that various components of hostility are associated with different types of cardiovascular responses. Gottman (2001) provides evidence that different types of violent men vary in their styles of cardiovascular responding, including both hyper and hypo-reactive

response patterns. Gottman (2001) examined violent men during high-conflict interactions with spouses and found two different types of heart rate reactors. Type 1 men demonstrated decreased heart rate and increased vagal tone. Type 2 men demonstrated cardiovascular hyperreactivity. In describing the psychological correlates of these physiological reactivity patterns, the Type 1 men scored higher on measures of antisocial behavior compared to the Type 2 men.

Evidence from animal research indicates that different psychological processes result in distinctive physiological responses to stress. Mammals exposed to behavioral stress exhibit different cardio-respiratory and metabolic response patterns depending on their ability to activate appropriate coping actions. Two primary behavioral patterns exist, one that is considered activational (defense), while the other is considered inhibitional (vigilance). The activational response pattern is characterized by increased cardiac and SBP output. The inhibitional response is characterized by increased total peripheral resistance and DBP output (Brownley, Hurwitz, Schneiderman, 2000).

In consideration of these findings, it would be useful to determine how various components of hostility are associated with different levels and types of physiological reactivity. Researchers have examined the differential predictability of hostility measures in relationship to CVR and CVD. However, these studies were not guided by theoretical models postulating how different types of hostility should be related to distinctive cardiovascular response patterns.

State by Trait Interactions Influencing Cardiovascular Reactivity

Another explanation for the inverse association between hostility and CVR is that the direction of the relationship between hostility and CVR may depend on the interaction between hostility with other variables. The literature relating hostility to CVR has relied on the dispositional approach to personality that "focuses on the broad stable characteristics that differentiate individuals consistently, seeking evidence for the breadth and durability of these differences across diverse situations" (Mischel & Shoda, 1998, p. 231). The trait approach predicts a direct correspondence between dispositions and how they are expressed behaviorally (Mischel & Shoda, 1995). Unfortunately, the trait approach typically does not work well at predicting people's behavior across situations. "The average cross-situational coefficients are typically low but non-zero" (Mischel & Shoda, 1995, p. 247). This trend is apparent in the hostility-CVR literature, where the ability for trait hostility to predict CVR across situations is very low. However, there are expanded models of personality that analyze dispositions according to the social cognitive and affective processes within individuals and how these characteristics interact with situations (Mischel, 1999; Michel & Shoda, 1995; Mischel & Shoda, 1998).

The research on hostility and CVR is beginning to go beyond a simple trait approach in considering how hostility interacts with characteristics of the situation to predict CVR. Researchers found that the effect of hostility on CVR is influenced by the type of stress induction procedure (Christensen & Smith, 1993), such as its inter-personal characteristics (Lavoie, Miller, & Conway, 2001), and type of disclosure (Kurylo & Gallant, 2000). These interactional designs are in line with researchers' views advocating

for a multifactorial approach towards understanding the relationship between personality-behavior and essential hypertension (Jorgensen, Johnson, Kolodziej, & Schreer, 1996) and ambulatory blood pressure (Carels, Sherwood, & Blumenthal, 1998). These state by trait designs are consistent with psychophysiological models that describe autonomic responses to psychological stress as composed of two components: (a) individual response stereotypy- a trait approach to reactivity, and (b) situational stereotypy- whereby different types of stressors cause different physiological responses (Brownley et al., 2000).

Inaccurate Representations from Statistical Techniques Relying on Averages

The inverse association between hostility and CVR reported in the present investigation may not accurately reflect the relationship between hostility and CVR due to the use of statistical techniques that rely on averages instead of modeling change over time. This area of study would profit from the application of more advanced statistical techniques such as latent growth curve (LGC) analysis that examines change processes. In a recent study by Llabre, Spitzer, Siege, Saab, & Schneiderman (2004), hostility was not related to reactivity (the intercept), and was not associated with average change scores during recovery. However, LGC analysis demonstrated that hostility was associated with slower SBP recovery. These authors asserted that the recommendations for reactivity and recovery research proposed by Linden et al. (2003) cannot be achieved without the use of more advanced statistical methods such as LGC analysis.

Need to Expand Beyond the Psychophysiological Hyperreactivity Model

As researchers attempt to understand the physiological processes mediating the effects of psychological variables on CVD, it may be useful to expand beyond the simple hyperreactivity model. The focus of research has shifted from measures of acute reactivity to measures of recovery (Glynn et al., 2002; Mayne, 2001) and variability (Roman, Pickering, Schwartz, Pini & Devereux, 2001; Sloan et al., 1999). Research should consider the influence of psychological variables on other cardiovascular responses not necessarily associated with hyperreactivity, such as increased vascular resistance.

APPENDIX

Item Content of Measures

Structured Interview

1. May I ask your age, PLEASE?

2. What is your student classification?

3. Are you SATISFIED with your school-work thus far? (Why not?)

4. Do you feel that college carries HEAVY responsibility?
   Is there any time when you feel particularly RUSHED or under PRESSURE?
   When you are under PRESSURE does it bother you?

5. When you get ANGRY or UPSET, do people around you know it?
   How do you show it?
   In general, when you get angry do you hold it in or get it off your chest?
   Do you ever raise your voice and argue?
   Do you ever slam a door, throw something, or anything like that?
   Do you ever feel like doing that?
   About how many times a week do you get angry?
   Would you say that you get angry more or less often than your friends and acquaintances?

6. Have you ever played any competitive games with young children between the ages of six and eight? (like cards, checkers, or Monopoly)
   Did you ALWAYS allow them to WIN on PURPOSE?
   WHY? (WHY NOT?)

7. When you play games with people your own age, do you play for fun of it, or are you really in there to WIN?
   Suppose you are involved in a team sport and one of your partners is just going through the motions and not really trying his/her best. Would you get angry or irritated?
   Would you say anything to that person?

8. Do you feel there is COMPETITION in school? Do you enjoy this?
   Are you competitive in other areas, like sports or on the job?
   Do you get angry when you lose?

9. When you are in your automobile, and there is a car in your lane going FAR TOO SLOWLY for you, what do you do about it?

Would you get angry or irritated?
Would you MUTTER and COMPLAIN to yourself, flash your lights, tailgate the person, yell, honk the horn?
Would anyone riding with you know that you were angry or irritate?
How would they know?

10. If you make an appointment with someone, would you BE THERE on TIME?
    If you are kept waiting, do you RESENT it?
    Would you SAY anything about it? What? What if they are late all of the time?

11. If you see someone doing a job rather SLOWLY and you KNOW that you could do it much better and faster yourself, does it make you RESTLESS to watch?
    Would you be tempted to STEP IN AND DO IT yourself?
    Have you actually done that?

12. What IRRITATES you most about this college, or the students here? (work or the people with whom you work)
    Do you show your irritation?
    Do you see a lot of incompetence among the students and faculty? (your coworkers)

13. When you go out in the evening to a restaurant and you find 8 or 10 people waiting in line AHEAD OF YOU for a table, will you wait? (No? Say it's a good restaurant.)
    What will you do while you are waiting?
    Suppose after 20 minutes, the host or hostess comes back and says that there will be another 20 minute wait, what would you do?
    Would you say anything to him/ her?

14. How do you feel about waiting in lines, like at the BANK, SUPERMARKET?
    Suppose you are in line to purchase a small item, and the cashier is on the phone talking to a boyfriend or girlfriend, how would you feel?
    Would you say anything? Why? (Why not?)
    Would you ask to speak with the manager?
    What might you say?

15. Do you HURRY in doing most things?
    When you are in a hurry to get something done and somebody interrupts you, how do you react to that?

Cook-Medley Hostility - Cynicism Subscale

1. I think a great many people exaggerate their misfortunes in order to gain the sympathy and help of others.
2. Most people make friends because friends are likely to be useful to them.

3. Most people will use somewhat unfair means to gain profit or an advantage rather than to lose it.
4. People generally demand more respect for their own rights than they are willing to allow for others.
5. It is safer to trust nobody.
6. I think most people would lie to get ahead.
7. Most people are honest chiefly through fear of being caught.
8. Most people inwardly dislike putting themselves out to help other people.
9. No one cares much what happens to you.
10. I have often had to take orders from someone who did not know as much as I did.
11. It takes a lot of argument to convince most people of the truth.
12. A large number of people are guilty of bad sexual conduct.
13. I have often met people who were supposed to be experts who were no better than I.

Alcohol Consumption Scale

In the past month, how often did you:

1. Drink a 12 ounce can or bottle of beer?
2. Drink a 4 ounce glass of wine?
3. Drink 1-1.5 ounces of hard liquor?
4. Binge drink (6 or more beers, glasses of wine, or shots of hard liquor within an hour or so)?

Caffeine Consumption Scale

In the past month, how often did you:

1. Drink a 12 ounce caffeinated beverage (such as a coke or pepsi)?
2. Drink a cup of coffee or tea?

Exercise Scale

In the past month, how often did you:

1. Purposefully engage in jogging, swimming, or other aerobic-type exercises for at least 20 minutes?
2. Purposefully engage in basketball, tennis, handball, dance, brisk walking, or similar activities for at least 30 minutes?

Sleep Problems Scale

1. I have difficulty falling asleep.
2. Thoughts race through my mind and prevent me from getting to sleep.
3. I anticipate a problem with sleep several times a week.
4. I often wake up and have trouble going back to sleep.
5. I wake up earlier in the morning than I would like to.
6. I lie awake for half an hour or more before I fall asleep.
7. I often feel sad or depressed because I can't sleep.

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