PREDICTORS OF CATECHOLAMINE, HEART RATE AND BLOOD PRESSURE REACTIVITY DURING THE TYPE A STRUCTURED INTERVIEW: ANGER-MANAGEMENT STYLE

by

Barbara E. Silverman

A thesis
presented to the University of Waterloo
in fulfilment of the
thesis requirement for the degree of
Master of Science
in
Health Behaviour

Waterloo, Ontario, Canada, 1993

Author's Declaration

I hereby declare that I am the sole author of this thesis. This is a true copy of the thesis, including any required final revisions, as accepted by my examiners.

I understand that my thesis may be made electronically available to the public.

Barbara E. Silverman (known as Barbara McNeice-Stallard as of May 2003)

Department: Health Studies

Program: Health Behaviour

Degree: Master of Science in Health Behaviour

March 28, 2011

ABSTRACT

A subset of data was used from the Waterloo Longitudinal Reactivity Study (WLRS) to examine predictors of cardiovascular reactivity during the Type A Structured Interview (SI). Sex, age, total cholesterol, body mass index (BMI), activity level, smoking, drinking, family history of cardiovascular disease, anger-management styles (i.e., hostility, SI Components, and anger) and respective resting levels for the dependent measures were the predictor variables. Systolic blood pressure (SBP), diastolic blood pressure (DBP), heart rate (HR), norepinephrine, and epinephrine were the dependent measures. For this study, 111 males and 129 females completed the study. They were first or second year students from the University of Waterloo. The subjects' mean age was 20.44 years. In the WLRS, subjects were asked to come to the laboratory on several occasions. The first occasion was to obtain consent. The second occasion was to habituate subjects to the laboratory setting and the equipment. The third session was the test proper. The focus of this secondary data analysis was to examine the predictive property of anger-management styles to reactivity during one task in the test proper, the SI. Factor analyses were done on the psychometric scales. From this, three factors emerged: hostility, SI components, and anger. These variables, along with the remaining independent variables were entered in multiple regression equations. For norepinephrine, epinephrine, and DBP reactivity only the respective resting values were predictive of reactivity. The independent measures, sex and BMI were predictive of SBP reactivity. Predictors of HR reactivity were resting HR, anger (one of the three factors), and sex. The results of this study did not support the

contention that personality measures are predictive of catecholamine or blood pressure reactivity. HR reactivity, however, was predicted by personality and demographic measures. Although clinical applications are not possible, the study allows for the possibility that one day only psychometric scales will need to be completed in order to predict reactivity.

ACKNOWLEDGEMENTS

My thanks and gratitude are extended to Ken Prkachin whose experience in research and with cardiovascular reactivity have been immensely valuable to me.

I would also like to thank Dave Mills, and Jane Irvine for agreeing to be on my thesis committee. I hope the contents of this thesis will prove useful to their own research.

I would like to thank my family and friends for the motivation they gave me to succeed.

And I would like to thank my husband, Harris, for his love and support. I couldn't have done it without you.

TABLE OF CONTENTS

LIST OF TABLES

LIST OF FIGURES

INTRODUCTION

A number of contributing factors - physiological, psychological, and sociological - must be examined when assessing the causes of cardiovascular diseases (CVD). Many studies have examined the relationship between the traditional risk factors (e.g., smoking, cholesterol, hypertension) and development of CVD. However, these factors are believed to account for only half of all cases of CVD. The challenge, however, is to find a relationship that is indicative of future development of cardiovascular diseases. Is cardiovascular reactivity (i.e., change in cardiovascular responsiveness to a psychological or psychosocial or physiological challenge) part of that relationship?

The first step in addressing this question is to identify a relationship between independent and dependent variables that occur before CVD is clinically evident. This relationship would be strengthened if the independent variables were found to be predictors of CVD in prospective studies. Many studies have examined the relationship between anger-management styles, such as hostility and suppressed anger, and CVD, while fewer studies have examined anger-management styles and cardiovascular reactivity. Researchers have found these variables to be both strong and weak predictors of CVD which is indicative of inconsistent findings. These results suggest that anger-management style variables would be valuable in a predictive model. What would be a valuable dependent measure? Cardiovascular reactivity has been implicated in the development of CVD; however, reactivity is not as yet considered to be a risk factor. Cardiovascular reactivity could be a valuable predictive measure to use in subjects

without demonstrable CVD.

The overall purpose of this project was to examine the predictive role of anger-management styles in cardiovascular reactivity. In order to develop this concept, a number of different areas of the literature will be reviewed: the role of anger-management styles in CVD, stress reactivity as a potential mediating mechanism, catecholamine reactivity, blood pressure reactivity, and heart rate reactivity. Before proceeding, however, a few terms need to be clarified.

Cardiovascular Diseases

There are a number of different terms used in the literature that refer to cardiovascular diseases. The term coronary artery disease (CAD) refers to conditions that cause narrowing of the coronary arteries (i.e., atherosclerosis) so that blood flow to the heart is reduced. Atherosclerosis contributes to coronary heart disease (CHD); defined as damage to the heart muscle caused by insufficient blood supply from obstructed coronary arteries. Permanent damage to, or death of heart muscle is called a myocardial infarction (i.e., heart attack). When there is insufficient supply of blood to the brain a stroke can result. In general, cardiovascular disease is a broad term referring to disorders of the heart and the circulatory system. To date, the etiology and pathogenesis of CAD and CHD are not fully known (Williams & Barefoot, 1988).

A number of steps in the development of the atherosclerotic plaque have been theorized (Clarkson, Manuck, & Kaplan, 1986). There is a progressive stage of plaque formation: fatty streak, transitional plaque, advanced fibrolipid plaque, and complicated plaque with thrombus formation (Davies, & Woolf, 1993). The

fatty streak contains foam cells and intracellular lipid (Davies, & Woolf, 1993). The initiation of the plaque is thought to be caused by damage to the endothelial wall of the artery. Damage is thought to be caused by, for example, hemodynamic disruptions (e.g., turbulence, sheer stress), although the exact mechanisms are still unclear (Clark, et al., 1986). An immune/inflammatory response is thought to occur when the intima is damaged (Davies, & Woolf, 1993). Plasma low density lipoproteins (LDL) enter the subendothelial space (SES) and are oxidized. It is in the oxidized state that they are chemotactic for blood monocytes. The monocytes enter the SES and the oxidized LDL initiate an inflammatory response with the monocytes. The monocytes ingest the oxidized LDL which is toxic to the cell. Eventually the monocyte is filled with lipids and is known as a foam cell. Foam cell necrosis occurs as a result of the oxidized LDL cytotoxicity. When extracellular lipids are present, as a result of the foam cell necrosis, the fatty streak has progressed to a transitional plaque (Davies, & Woolf, 1993). The advanced fibrolipid plaque is the next stage of plaque progression. The plaque contains an extracellular lipid core, smooth muscle cells, and foam cells. The smooth muscle cells are formed as the result of growth factors released from the "platelets, endothelial cells, macrophages, and other smooth muscle cells" (Davies, & Woolf, 1993, p. S4). A layer of smooth muscle cells can be found on the lipid core of the luminal side. The plaque size is increased by the production of collagen. It should be noted that "most advance plaques are not angiographically visible" (Davies, & Woolf, 1993, p. S6). The final stage of plaque progression is known as complicated plaque with thrombus formation. Coronary-prone behaviour is thought

to involve physiological patterns which facilitate the development of atherosclerosis.

Historical Interest in "Coronary-Prone Behaviour"

For hundreds of years, people have been claiming a link between behaviours, emotions, and personality characteristics, and changes in cardiovascular function (Williams & Barefoot, 1988). Over the years, the names of three men have often been cited as suggesting this link: William Harvey, John Hunter, and Sir William Osler. The views held by these men tended to focus attention on psychological and behavioural factors as the causes of CHD (Williams & Barefoot, 1988). Associations, such as these, were not given much consideration over the years by researchers.

In contrast, a number of risk factors for CHD have been identified and have come to be known as "traditional" or "established" risk factors (e.g., smoking, cholesterol, hypertension). This moved the focus from psychological factors to more concrete, overt behaviours. However, these risk factors account for less than half of the new cases of CHD (Williams & Barefoot, 1988). Consequently, the question has become: What other factors contribute to new cases of the disease?

Friedman and Rosenman (1959) renewed interest in the psychological and behavioural focus of the early years. In doing so, they developed the concept of the Type A behaviour pattern (TABP) which describes individuals who are "... characterized by high levels of competitive achievement striving, incessant time-urgent behavior that lead to persistent acceleration of physical or mental activity, and high levels of free-floating hostility" (Williams & Barefoot, 1988, p. 190).

Coronary-prone behaviour can be viewed as a style of behaving and coping that leads to coronary disease (Booth-Kewley & Friedman, 1987). TABP is not synonymous with coronary-prone behaviour, rather TABP is thought to be one facet of it. Friedman (1989) views coronary-prone behaviour as, by definition, leading to CHD, whereas the effect of TABP on health is an empirical matter. That is, "only certain of the attributes associated with Type A are predictive of CHD development" (Booth-Kewley & Friedman, 1987, p. 344). Hence, TABP was initiated as a way to conceptualize the psycho-behavioural personality determinants of CHD.

Evolution of the Concept of Type A Behaviour

Friedman and Rosenman hypothesized that TABP was linked to the development of CHD, and evaluated their hypothesis by initiating the Western Collaborative Group Study (WCGS) (Rosenman, Brand, Jenkins, Friedman, Straus, & Wurm, 1975). Researchers recruited 3000 middle aged men who were free from clinical disease signs at intake. Subjects were required to undergo a Structured Interview (SI) which is a standardized interviewing technique that is designed to elicit a variety of behavioural "signs" in appropriately predisposed individuals (Chesney, Eagleton & Rosenman, 1981). In particular, it is constructed and performed in such a way as to provide an opportunity for the interviewee to display elements of so called "coronary-prone behaviour", such as competitiveness, energetic responding, and hostility. This is done through both the content of questions (e.g., "What about the people you work with makes you angry?") and the style of the interviewer, which is businesslike, brisk, and occasionally

challenging. Rosenman and Friedman developed the SI as a valid and reliable measure of TABP (Rosenman, 1978 cited in Rosenman, Swan, and Carmelli, 1988; Rosenman, Friedman, Straus, Wurm, Kositcheck, Hahn, & Werthessen, 1964; Williams & Barefoot, 1988).

The results of the WCGS identified half the men as exhibiting the TABP. At the 8.5 year follow-up, the Type A men were twice as likely as the Type B men (i.e., less hostile, less aggressive and less concerned with time than Type As) to have CHD manifestations. When traditional risk factors were controlled, using a multivariate statistical test, the effect of TABP remained constant (Rosenman, et al., 1975). Subsequent measures on this study population found that Type A men had increased coronary atherosclerosis (CAD) relative to Type B men, as measured either at autopsy or by coronary angiography (Rosenman, et al., 1975). The WCGS was an important landmark study of the relationship between TABP and CHD. It provided the first epidemiological evidence that TABP was associated with CHD (Booth-Kewley & Friedman, 1987).

Structured Interview as a Measurement Technique

Over the years, various coding systems have been applied to the SI. Initial scoring schemes utilized the following scores: Type A1, Type A2, Type B3, and Type B4 (Rosenman, et al., 1964). The Type A1 and Type A2 scores were given to subjects who displayed TABP mannerisms. Subjects who did not have Type A behaviours were classified as Type B. A slightly altered coding system for the SI was cited in Rosenman (1978; cited in Matthews, 1982). Individuals were classified as one of the following: "...A1, or fully developed Type A; X, or an equal

representation of Type A and Type B characteristics; Type B or the absence of Type A characteristics" (Matthews, 1982, p. 295). Over the years, questions in the SI have been altered or dropped as their ability to elicit Type A characteristics was assessed (Rosenman, Swan, & Carmelli, 1988).

As the need arose, different scoring systems were developed. In particular, the assessment of different sub-components of the Type A behaviour pattern has evolved. Chesney, Hecker, and Black (1988) developed a system for scoring each question of the interview. The end result of the scoring system was to assign each participant a score, from one to four, which described their degree of behaviours on fourteen sub-components: immediateness, Type A content, anger in, anger out, competitiveness, hostility, self-aggrandizement, exactingness, despondency, loudness of voice, syllabic emphasis, speaking rate, acceleration, and hard voice.

Dembroski and MacDougall (1983) also developed a scoring system for the SI (Matthews, Krantz, Dembroski, & MacDougall, 1982; cited in Dembroski & MacDougall, 1983). This system focused on seven sub-components of Type A behaviour: loud voice, explosive speech, rapid and accelerated speech, response latency, potential for hostility, anger in, and competition for control of the interview. Scores for each sub-component were given on a 5-point scale; the higher the score the more of the component the subject was exhibiting.

These components have been significantly related to CHD even when the Type A construct was not predictive of CHD, as will be discussed later in this paper.

Disenchantment with Type A Concept

In 1981, the results from the WCGS led the medical community (Review Panel on Coronary-Prone Behavior and Coronary Heart Disease, 1981) to conclude that the TABP is associated with CHD (Williams & Barefoot, 1988). More importantly, the group indicated that the TABP-CHD association was unrelated to, but of approximately the same magnitude, as the association between CHD and other risk factors, such as smoking. Shortly after this acknowledgement, negative findings, from prospective epidemiological studies and cross-sectional studies of coronary angiography patients, began to emerge which put into question the robustness of the TABP-CHD association (Dembroski, 1984; Dembroski and Costa, 1987; Williams & Barefoot, 1988). For example, Dembroski, MacDougall, Williams, Haney, & Blumenthal's (1985) large study found no relationship between TABP, as assessed by the SI, and severity of CAD. As of today, TABP is not considered a risk factor.

Miller, Turner, Tindale, Posavac, and Dugoni (1991) completed a meta-analysis of studies of the TABP and CHD association. The results of this study suggested three reasons for the trend toward null findings in the TABP-CHD association in recent years. The first reason relates to the fact that studies that reported most of the null findings recruited high risk or diseased subjects. These studies are prone to disease-based spectrum (DBS) bias. DBS refers to "the reduction in the correlation between TAB and CHD that occurs when researchers restrict the range of their sample to only high-risk subjects" (Miller, et al., 1991, p. 470). Restricting attention to high risk subjects has the effect of limiting the

range of scores on predictor and criteria variables. It is necessary to observe the full range of values on predictor and outcome variables in order to fully characterize their interrelationships. Therefore, in such circumstances any expression of the relation between predictor and outcome will be attenuated. The result is a partial picture which may not reflect the true relationship between CHD and TABP since the sample was high risk or diseased patients. For example, for argument's sake, if the true relationship were a parabola, but only the top right part of the parabola were seen in the relationship then the relationship would be thought to be a straight line and not a parabola. The second explanation for the trend toward null findings relates to the use of self-reported measures of TABP. Since 1981, many researchers have tested the relationship between CHD and TABP. Due to monetary reasons, a number of investigators have opted to use self-report measures of TABP instead of the SI. Self-report measures of coronary processes have questionable validity. Consequently, when used in this research there is a limited ability to predict CHD. The third reason proposed to explain null findings was the use of fatal myocardial infarction (MI) as a disease criterion. There are a number of reasons why the use of fatal MI as a disease criterion may be inappropriate. For one, the reliability of this criterion may be problematic because it depends on the use of death certificates. When death certificates are completed, a fatal MI may not necessarily be listed as the cause of death. Death may be registered as the result of some other cause. For another, the relationship between fatal MI and TABP may be mediated by age. In the review of Miller, et al. (1991) Type As were found to have fatal MIs at a younger age than Type Bs.

Sub-components of TABP may also be at the heart of the problem. The association of TABP with CAD in some studies, though small, suggests that there may be some specific underlying components of the TABP that are more strongly associated with CAD than are other components (Chesney, Hecker, and Black, 1988; Dembroski, 1984; Dembroski & Costa, 1987; Williams & Barefoot, 1988). The overall strength of the TABP-CHD association would, therefore, be decreased by the irrelevant components of the TABP. Based on this notion, it would be appropriate to assess the level of association between each of the components of the TABP and CHD.

As opposed to a meta-analysis, which Miller and colleagues (1991) conducted, Williams and Barefoot (1988) examined individual studies in a narrative review. They found both prospective and cross-sectional studies which failed to show the association between TABP - derived from the SI and self-reported questionnaires - and CHD. Prospective studies failed to predict new or recurrent CHD in samples that were at increased risk for CHD (Williams & Barefoot, 1988). This may be because confounds, such as hypertension, cigarette smoking, and hypercholesterolemia, masked the association of TABP and CHD. Insufficient statistical power might also result because it would be less likely that Type Bs would be in the sample. For example, in the Multiple Risk Factor Intervention Trial study (Shekelle, Hulley, Neaton, Billings, Borhani, Gerace, 1985) TABP was not associated with CHD in subjects that were initially free from CHD but who demonstrated multiple risk factors. On the other hand, Dembroski, MacDougall, Costa, & Granditis (1989) re-analyzed the same data and found the potential for

hostility, as measured in the SI, was positively associated with CHD incidence in younger men ($\leq$ 47 years).

A number of cross-sectional studies have used angiographic findings from patient samples to assess CAD severity and compare the findings to TABP distribution. Only one (Williams, Haney, Lee, Kong, Blumenthal, & Whalen, 1980) out of the ten studies showed a positive association between CAD severity and TABP (Williams & Barefoot, 1988).

Williams & Barefoot (1988) indicated that the negative angiographic findings may be due to confounds (e.g., age) which were not controlled in the analyses. In one angiographic study (Williams Barefoot, & Shekelle, 1986), the TABP, as assessed by the SI, was found to be related to CAD up to the age of 55. Beyond 55, the association reversed such that Type B people were found to have significantly more CAD. This relationship may reflect a survival effect. The authors hypothesized that the relationship reversed because Type A people develop the disease sooner and die, while Type B people take longer to develop the disease. Hence, a significant association between Type B behaviour and CHD may emerge later in life.

Krantz, Sanmarco, Selvester & Matthews (1979) examined psychological correlates of atherosclerosis progression in 67 men. Some subjects were recruited for secondary prevention of atherosclerosis while the rest were referred by their physicians to an ongoing cardiac rehabilitation program. All subjects had evidence of CHD, but they were currently asymptomatic. Testing occurred between 1974 and 1978, with the mean interval for testing being 17 months. The Rosenman

(1978; cited in Matthews, 1982) diagnostic interview, SI, was used to assess TABP. The results showed no relationship between global Type A and the progression of atherosclerosis.

The foregoing studies formed the basis for disenchantment with the global TABP. The evidence against a TABP-CHD construct link was strengthened by the varied methodologies. Not only did researchers who used cross-sectional designs find little or no TABP association with CHD development but researchers who used prospective designs found similar results. Combining the results of numerous studies, using meta-analysis, also indicated that the relationship, if anything, is weak. A review of these studies reveals that if the TABP construct has some value it may be in the components that comprise the TABP.

Anger-Management Characteristics as "Toxic Elements"

A number of researchers have studied sub-components of TABP such as anger and hostility. These components have been significantly related to CHD even when the TABP construct was not predictive of CHD.

Two studies have assessed the relationship between SI components and severity of CAD (Williams & Barefoot, 1988). Both studies re-analyzed SI data collected from previous studies. The results of one (MacDougall, Dembroski, Dimsdale, & Hackett, 1985) found a significant positive correlation between potential for hostility, anger-in and CAD. The second study (Dembroski, et al., 1985) found similar results. The positive association between potential for hostility, anger-in and CAD remained even after controlling for age, sex, and other risk factors (Williams & Barefoot, 1988). In these, the globally defined TABP was

not significantly related to CAD, while some components of the TABP were. This suggests that the component measures of TABP may be more consistently related to CAD and hence, are better measures of coronary-prone behaviour (Williams & Barefoot, 1988). In particular, the results implicate the potential for hostility and anger-in as the relevant "coronary-prone" behaviours (Booth-Kewley & Friedman, 1987; Williams & Barefoot, 1988).

Matthews, Glass, Rosenman and Bortner (1977) re-analyzed data from the Western Collaborative Group Study. They chose to examine data from 62 men who had developed clinical CHD and 124 matched controls. All subjects had undergone a 37-question interview at entry when they were free from CHD. Four and a half years later, the results from these interviews were compared for cases and controls. Not only were the participants classified as Type A or Type B, based on the results of the interview, but they were also coded on speech style and clinical judgements. Coding results were factor analyzed. Of the five factors found, only two were significantly correlated with Type A. These factors were termed competitive drive and impatience. T-tests were done between cases and controls on the various items in these two factors. Some of the items were significantly different between cases and controls and the mean value for the cases was higher than for the controls. Under the factor competitive drive, three items were rated significantly higher for cases than for controls: (1) explosive voice modulation, (2) potential for hostility, and (3) vigorous answers. Only one item, irritation at waiting in lines, was significantly different between the two groups in the factor impatience. These results alone indicate that impatience and competitive

drive are related to Type A behaviour. The authors went on to examine differences between group means for all other interview variables. Four other items were found to be different between the groups. One item of particular interest was anger directed outward. The researchers found that cases scored significantly higher on this item than the controls. Although this item loaded on both competitive drive and impatience, the item did not have a high enough loading on either to be included as part of that factor.

Hecker, Chesney, Black, & Frautschi (1988) also re-analyzed data from the Western Collaborative Group Study. They were interested in the relationship between CHD at the 8.5 year-follow-up period and component analysis of the SI at intake. In total, data from 3,154 male subjects were used. Only 250 males developed CHD; a matched-control group of 500 males who had not developed CHD was chosen for comparative analyses. All subjects had been free from CHD at intake. The results showed that hostility was a predictor of CHD incidence.

Results from these two studies reveal the merit in the SI component scoring system. In particular, the components hostility - and to a lesser extent, anger directed outward - have been at the forefront of predictors of CHD.

Thus far, only results from the SI have been considered in relation to predicting cardiovascular diseases. There is, however, another popular measure used to assess hostility: the Cook-Medley Hostility Scale (Cook & Medley, 1954).

Cook-Medley Hostility Scale

The Cook-Medley Hostility Scale (Cook & Medley, 1954) is a subset of questions from the Minnesota Multiphasic Personality Inventory (MMPI). Originally

developed to predict elements of the performance of teachers, more recent construct validation studies indicate that high scores on this scale reflect an interpersonal style characterized by cynicism, suspiciousness, anger, and proneness to become angry (Barefoot, Peterson, Dahlstrom, & Williams, 1989; Williams, 1984). The correlation between the hostility complex in the SI and Cook-Medley scale was significant in one study ($r = 0.37$, Dembroski et al., 1985); however, Engebretson and Matthews (1992) found no association between these two measures in their study of dimensions of hostility in men, women, and boys.

Positive correlations have been found between the hostility complex (Ho) in the Cook-Medley and the severity of CAD in both prospective and cross-sectional studies (Williams & Barefoot, 1988). One prospective study (Shekelle, Gale, Ostfeld, & Paul, 1983) followed 1877 healthy men from the Western Electric Study (Rosenman et al., 1964; Rosenman, Brand, Sholtz, & Friedman, 1976) who had completed the MMPI 20 years prior to analysis. The results showed that men with higher Ho scores were more likely to have a 10-year CHD event.

Barefoot, Dahlstrom, and Williams (1983) examined CHD events of 255 physicians 25 years after they had completed the MMPI. At the time of completing the questionnaires, the subjects were in a medical school clerkship program. The results indicated that subjects with a high Ho score were four-to-five times more likely to have had a CHD event than those with low Ho scores.

The results of the prospective studies show the predictive value of the Cook-Medley scale. Likewise, cross-sectional studies have shown the worth of this instrument. One cross-sectional study (Williams et al., 1980) recruited 424 male

and female patients. Subjects were required to complete both the Cook-Medley Scale (Cook and Medley, 1954) and the SI (Rosenman, 1978; cited in Williams, et al., 1980). The results revealed a positive relationship between TABP, as assessed by the SI, and severity of CAD and a positive relationship between hostility and degree of CAD. The authors contend that use of the results from SI together with the Cook-Medley Scale "... provide a more complete assessment of the likelihood of having a significant coronary occlusion on arteriographic examination than does either mode of characterization alone" (Williams, et al., 1980, p. 547). The authors also go on to question the over representation of Type As with high Ho scores in their sample. They still suggested their findings were valid because the results revealed differences in the likelihood of significant occlusion on arteriography between Type As with high Ho scores and non-Type As with low Ho scores.

These studies indicate that the Ho scale may predict coronary proneness. Of particular interest in the last study were the results from multivariate analyses. The significance of the relationship between Ho and CAD severity was increased when gender and TABP were controlled for. On the other hand, when gender and Ho scores were not controlled, the relationship between TABP and CAD severity was weakened (Williams & Barefoot, 1985). This implies that gender and Ho scores are influential in the predictive power of TABP and CAD.

On the other hand, three prospective studies (Hearn, Murray, & Luepker, 1989; Leon, Finn, Murray, & Bailey, 1988; McCranie, Watkins, Brandsma, & Sisson, 1986) found no association between hostility and CAD. McCranie, et al.

(1986) did a 25-year follow-up study of 478 physicians. High hostility scores on the MMPI did not predict incidence of coronary disease or total mortality. It is interesting, however, that in this study the medical students were required to complete the MMPI at their medical school admissions interview. It has been suggested (Williams, 1987) that the students would be inclined to answer the questions with socially appropriate responses to increase the probability of being accepted into medical school, and this may have affected the likelihood of finding a positive relationship.

Another 30-year-prospective study of 280 subjects found that MMPI hostility scores were not predictive of CHD (Leon, et al, 1988). However, a potential problem in this study was the age of the subjects, which ranged from 43 to 53 years. They were free from disease at intake in 1947, with a mean age of 45 years. By this age, many high risk men would likely already be showing signs of atherogenesis. Thus because these subjects had no signs or symptom of disease at intake, it is possible that the study was biased.

Using a larger sample (N = 1399), Hearn, et al. (1989) found the Hostility Scale did not predict CHD mortality or morbidity over a 33 year period. The subjects in this study completed the questionnaire as part of freshman orientation. This is also potentially a biased sample as the mean hostility score was higher than that obtained in other hostility studies (Hearn, et al., 1989).

Clearly, however, the results of studies of hostility-CHD relationship are inconsistent. In an attempt to provide a mathematical summary of findings in this field, Booth-Kewley and Friedman (1987) and Matthews (1988) performed meta-

analyses on studies that examined the association of Type A behaviour and Type A components and CHD. Booth-Kewley and Friedman (1987) examined the association between SI Type A, anger, and hostility and a variety of CHD endpoints. Results of the meta-analyses revealed all three measures - SI Type A, anger, and hostility - were related to CHD outcomes.

Matthews' (1988) meta-analyses was more up-to-date than Booth-Kewley's and Friedman's. In addition, Matthews used different decision criteria for including an article in the meta-analysis. In particular, she included only prospective studies. Cross-sectional studies were excluded because they are not able to discern cause and effect relationships, and they may be contaminated with spurious associations. Results of Matthews' study revealed a lower, but still significant relationship between SI Type A, hostility, and CHD. Thus meta-analytic studies are consistent with the conclusion that SI Type A and hostility are predictive of CHD. When both prospective and cross-sectional studies are combined, anger is also predictive of CHD.

These studies have indicated the value of the Cook-Medley and SI measures in predicting CAD, but how well do these behavioural measures predict cardiovascular reactivity?

Cardiovascular Reactivity

It has been suggested that a mechanism linking anger-management styles with CHD is cardiovascular reactivity (CVR) (Weidner, Friend, Ficarrotto, & Mendell, 1989; Williams & Barefoot, 1988; Williams, Barefoot, & Shekelle, 1986). Cardiovascular reactivity (CVR) refers to the change in cardiovascular

responsiveness to a psychological, psychosocial, or physiological challenge.

CVR has not been established as a risk factor for cardiovascular diseases. Heart rate reactivity, for example, is viewed as being "... only a marker or correlate of other pathogenic processes which more directly influence lesion development" (Manuck, Muldoon, Kaplan, Adams, & Polefrone, 1989, p. 219).

Animal Models of Reactivity

Several experiments have been done by Manuck's group to assess heart rate reactivity and atherosclerosis in male and female cynomolgus monkeys (Manuck, Kaplan & Clarkson, 1985; Manuck, Kaplan, & Clarkson, 1983 cited in Manuck, et al., 1989). Kaplan, Manuck, Clarkson, Lusso & Taub (1982; cited in Manuck, et al., 1989) measured heart rate reactivity of male monkeys during a 'monkey glove' procedure. During this procedure, the experimenter mimics movements normally done before an animal is captured and handled. The animals were separated into high and low reactivity groups based on their heart-rate response to this task. Severity of atherosclerosis was significantly different between the groups ($p < 0.04$): high reactors had more severe atherosclerosis than low reactors. The authors could not account for their results in terms of confounding influences of systolic blood pressure, diastolic blood pressure, total serum cholesterol, and high density lipoprotein concentration. The only other difference found between the groups was in their heart weights; high heart rate reactors had greater heart weights than low heart rate reactors ($p < 0.05$). Manuck, et al. (1989) suggested that high heart rate reactivity may be a factor promoting both coronary artery atherosclerosis and cardiac morphology.

In a similar study on female monkeys (Kaplan, Adams, Clarkson & Koritnik, 1984), high heart rate reactors had two times the amount of atherosclerosis of the low heart rate reactors. As with the male monkeys, atherosclerotic differences between the groups were not explained by systolic or diastolic blood pressure, total serum cholesterol, or high density lipoprotein. Similar to the results from the males, females who exhibited high heart rate reactivity had significantly higher heart weights than the low reactors.

Reactivity in Human Subjects

A number of studies have also been done relating measures of reactivity to the severity of CHD in humans. Cinciripini (1986b) reported a study that assessed heart rate reactivity in patients "... with advanced grades of ventricular arrhythmias during an interview concerning recently stressful areas of their lives". The study demonstrated that exposure to a stressful interview produced premature ventricular contractions and increased heart rate. These physiological reactions were not evident during physiological manipulations (e.g., postural adjustment), contrary to what would have been expected.

Keys, Taylor, Blackburn, Brozek, Anderson, and Somonson (1971) conducted a 23 year prospective study. Two hundred and seventy-nine men between the ages of 47 and 57 were free from clinical signs of CHD at the beginning of the study. CHD-free was assessed as a result of a 12-lead electrocardiogram, detailed medical history, and a chest pain questionnaire. The researchers found that diastolic blood pressure (DBP) reactivity greater than 20 mmHg, during the cold pressor (CP) task was predictive of CHD. It should be

noted that not all stressors produce the same sympathetic and parasympathetic response. For example, the CP task produces alpha-adrenergic responses while the reaction time task produces an increase in beta-adrenergic activity (Allen, Boquet, Shelley, 1991).

Krantz, Helmers, Bairey, Nebel, Hedges, and Rozanski (1991) examined reactivity in patients with different degrees of CAD. Specifically, three groups of patients were formed in which the level of ischemic (abnormal) cardiac wall motion responses to mental stressors were different. The results indicated that patients with the most severe ischemia problems (i.e., greatest wall motion abnormalities) had the highest systolic blood pressure reactivity to a mental stressor. Indeed, in this group, the effects of one particular mental stressor, public disclosure of a person failing, were comparable in magnitude to those of exercise.

Reactivity and Coronary-Prone Behaviour

Houston (1988) summarized a number of studies which had examined the relationship between cardiovascular reactivity and SI components of the TABP (Tables 1-3). As can be seen, these studies have had inconsistent findings. This inconsistency may reflect the substantial differences in gender and mental task used in various studies. These inconsistencies make comparison of the data obtained difficult, at best. At the very least, the results indicate that one component, potential for hostility, had more significant associations than the other components, as noted in Table 3.

Harbin (1989) conducted a meta-analysis of the literature on Type A/B - as assessed by the SI or the Jenkins Activity Survey - and CVR. The author was able

TYPE A COMPONENTS AND REACTIVITY:

Study	Gender	Task[a]	Latency	Rate	Loudness	Emphasis	Loud–Explosive (loudness and emphasis)
Dembroski et al., 1978	Male	(RT, pong, and anagrams)	0	+SBP**			0
				+DBP**			
Glass et al., 1983	Male	Modified Stroop	0	0			0
Allen et al., 1984	Male	RCR	−SBP**, +PTT**	0	0	0	
		RCQ	−SBP**, −DBP*	0	0	0	
		DB	0	0	0	0	
		CP	0	0	0	0	
		IH	0	0	0	0	
Diamond et al., 1984	Male	Pong					
		Competition	0′	0′			0′
		Frustration	0′	0′			0′
		Harassment	0′	0′			0′
MacDougall et al., 1981							
Study I	Female	CP	0′	0′			0′
		RT	0′	0′			0′
Study II	Female	SI	0′	0′			0′
		RT	0′	0′			0′
Anderson et al., 1986	Female	SI	0	+SBP**	+SBP**	+SBP**	
				+DBP**	+DBP**	+DBP**	
		MA	0	0	0	0	

[a]RT = reaction time; SI = Structured Interview; DB = digits backward; CP = cold pressor; MA = mental arithmetic; RCR = reading comprehension reading phase; RCQ = reading comprehension questioning phase; IH = isometric handgrip.

*$p < .10$

**$p < .05$.

Table 2
Cardiovascular Reactivity Studies

TYPE A COMPONENTS AND REACTIVITY:

Study	Subject Gender	Task[a]	Potential for Hostility	Verbal Competitiveness
Dembroski et al., 1978	Male	(RT, pong, and anagrams)	+SBP** +HR**	+SBP**
Glass et al., 1983	Male	Modified Stroop	−SBP* −DBP**	−DBP*
Allen et al., 1984	Male	RCR	0	0
		RCQ	0	+HR**
		DB	+HR**, −PTT*	+HR**
		CP	+SBP**, +DBP*	+SBP**
		IH	+SBP*, +DBP**	0
Diamond et al., 1984	Male	Pong		
		Competition	0	0′
		Frustration	0	0′
		Harassment	0	0′
MacDougall et al., 1981				
Study I	Female	CP	0	0′
		RT	+SBP** +HR**	0′
Study II	Female	SI	+SBP**	0′
		RT	−HR**	0′
Anderson et al., 1986	Female	SI	0	0
		MA	0	+DBP**

[a]RT = reaction time; SI = Structured Interview; DB = digits backward; CP = cold pressor; MA = mental arithmetic; RCR = reading comprehension reading phase; RCQ = reading comprehension questioning phase; IH = isometric handgrip.
*$p < .10$.
**$p < .05$.

Table 3
Cardiovascular Reactivity Studies

TYPE A COMPONENTS AND REACTIVITY:

Study	Subject Gender	Task[a]	Stylistic Vigor (loud–explosive and rapid and latency)	Hostility–Competition (hostility and verbal competitiveness)
Dembroski, MacDougall, Herd, & Shields, 1979	Male	CP—high–low challenge	+SBP**—high +HR** challenge	+HR**—high challenge +SBP**, +DBP**—low challenge
		RT—high–low challenge	+SBP**—high and low challenge	+SBP**—high and low challenge
Dembroski, MacDougall, & Lushene, 1979	Male	SI	+SBP** +DBP**	+SBP**

[a]RT = reaction time; SI = Structured Interview; CP = cold pressor.
*$p < .10$
**$p < .05$.

to give several points of insight into the Type A/B relationship: (1) in general, Type As were more reactive than Type Bs, (2) heart rate and systolic blood pressure reactivity in Type As was greater than in Type Bs, (3) diastolic blood pressure and norepinephrine reactivity did not consistently provide the A/B difference, (4) cortisol and epinephrine reactivity values were not different for As and Bs, (5) males' reactivity was a function of the Type A Behaviour Pattern, and (6) females' reactivity was not a function of Type A Behaviour Pattern. When the results were analyzed for studies that used the SI to measure Type A/B, the results indicated CVR (i.e., heart rate and blood pressure) was significantly different between Type A's versus Type B's.

Thus far, the focus of the literature summary has been to identify the historical evolution of TABP, the emergence of components of TABP, and the role of cardiovascular reactivity in this research area. Of the studies reported, none examined the relationship of predictors of cardiovascular reactivity measured during the structured interview. What power do behavioural measures play in the prediction of cardiovascular reactivity during the structured interview? This question will be addressed in the examination of catecholamine, blood pressure, and heart rate reactivity literature.

Catecholamines

The principal circulating catecholamines consist of three compounds: epinephrine, norepinephrine and, to a lesser extent, dopamine.

The adrenal medulla mainly secretes epinephrine - 80% epinephrine and 20% norepinephrine (Vander, Sherman, Luciano, 1980) - while norepinephrine is released mostly from post ganglionic cells of the sympathetic nerve endings (Axelrod & Reinsine, 1984; Frankenhaeuser, 1991; Goldstein & McDonald, 1986; Lake, Chernow, Feuerstein, Goldstein, & Ziegler, 1984). Dopamine is present in the adrenal medulla and the central nervous system, acting as a neurotransmitter (Bhagat, 1974). These hormones, and others, are released during stressful situations (Axelrod & Reisine, 1984; Lake, et al., 1984).

Norepinephrine is a neurotransmitter; which acts locally to affect the brain, heart, liver, adipose tissue, and blood vessels (Bhagat, 1974). When epinephrine is released, it acts as a hormone, targeting effector cells reached via the blood stream (Bhagat, 1974). Hormonal effects occur once they attach to adrenoceptors which occur in four types: alpha-1, alpha-2, beta-1, and beta-2 (Goldstein & McDonald, 1986). Alpha-1 receptors are predominately located in the post-synaptic areas near sympathetic nerve endings. Alpha-2 adrenoceptors can be located near the pre-synaptic and extra-synaptic areas. The heart has beta-1 receptors while the blood vessel walls predominately have beta-2 receptors.

Epinephrine secreted into the blood increases sympathetic functions of the body affecting the smooth and cardiac muscle (Vander, Sherman, Luciano, 1980). For example, in response to stress the adrenal chromaffin cells of the adrenal

medulla release epinephrine which causes increased heart rate, increased blood pressure, and the release of extra sugar from the liver for the muscles (Carmichael & Winkler, 1985). Increased rate and contractility of the heart occurs when norepinephrine is released (Vander, Sherman, Luciano, 1980). An increased blood pressure is caused by norepinephrine exerting a constricting effect on the blood vessels.

Table 4 describes a number of papers that have implicated catecholamine reactivity in the development of cardiovascular damage. Problems such as infarction, arrhythmias, and endothelial damage are, to mention a few, cardiovascular effects thought to be caused by exaggerated catecholamine secretion in response to challenges.

Type A and Type B Behaviour Patterns

Type A's and B's have been shown to exhibit differences in catecholamine reactivity to stressors. A landmark study showed a higher norepinephrine level in Type As than in Bs (Friedman, Byers, Diamant, & Rosenman, 1975). In this study, 15 participants were classified as Type A and 15 participants were classified as Type B by the Friedman and Rosenman (1971) SI assessment procedure. The participants were healthy male subjects with an average age of 48. A competitive task was used as the stressor. Catecholamine levels were measured before and during the task. Differences in norepinephrine levels were found between Type A's and B's with A's being more reactive than B's.

Table 4
Catecholamine Reactivity Implicated in Cardiovascular Damage

STUDY	SUGGESTED CARDIOVASCULAR EFFECTS
Glass, et al., 1980 & Goldstein & Rafjer, 1984	arterial damage, thrombus formation, cardiac arrhythmia
Dimsdale & Ziegler, 1991	cardiovascular pathophysiology
Ganguly, 1989	infarction and arrhythmias
Goldstein & Rafjer, 1984	myocardial ischemia, increase atherosclerotic narrowing, increase free fatty acid levels
Cohn, 1989	sympathetic nervous system effects on pathophysiology of cardiovascular diseases not fully understood, cardiovascular function can be injured after chronic stimulation of the nervous system
McKinney, et al., 1984	arterial damage, biochemical and cardiovascular changes linked to pathogenesis of cardiovascular disorders
Manuck, et al., 1989	atherosclerosis is initiated by behavioural stimuli which alter arterial flow and lead to endothelial injury
Markovitz & Matthews, 1991	increased platelet activity
Williams, 1989	endothelial injury leading to CAD

In another study, the response patterns of Type A's and Type B's were not consistent from task to task (Gellman, 1984). In particular, Type A's did not always have a higher response level than Type B's. For example, during exercise

at 80% of maximum HR, Type B's had higher norepinephrine responses than Type As. A small sample size (N = 12) may account for these unpredictable results.

More recently, Williams, Suarez, Kuhn, Zimmerman, & Schanberg (1991) found Type A's to exhibit chronic elevation of catecholamines. The reaction was also present in a naturalistic setting. These results point to a link between Type A and increased coronary risk, with chronic catecholamine response being the mediating factor. As shown in the summary of Tables 1-3, no studies have been done on catecholamine reactivity and Type A components of the SI. To this end, one purpose of the present study was to examine the relation of anger-management styles to neuroendocrine measures of cardiovascular reactivity.

Blood Pressure and Heart Rate

There is an abundance of studies in which blood pressure and heart rate reactivity have been assessed. Most, however, have not measured reactivity during the structured interview. Lake, et al. (1985) conducted a study to measure heart rate (HR) and blood pressure (BP) during the SI. The results from this study showed Type A's had higher BP increases than B's, during the SI.

BP and HR Reactivity and Development of Cardiovascular Damage

A number of studies have examined the relationship of BP and HR reactivity during various stressful tasks (e.g., cold water pressor, mental arithmetic) to cardiovascular damage. One of the earliest (Keys, et al., 1971), was a 23 year prospective study, described earlier. Results of the 23 year follow up revealed that resting systolic blood pressure (SBP), high serum cholesterol and DBP reactivity, during the CP test, were predictive of CHD death or infarction.

In a 45 year follow up study Wood, Sheps, Elveback, and Schirger (1984) examined reactivity during the CP task of 142 subjects initially tested as children. The authors noted that excessive response to the CP task was predictive of future hypertension. However, Coresh, Klage, Mead, Liang, and Whelton (1992) found no association between SBP reactivity to the CP task and the future development of cardiovascular disease in a sample of university students followed for 40 years. In another prospective study, Light, Dolan, Davis, and Sherwood (1992) utilized a reaction-time shock task to assess how predictive cardiovascular responses were of future BP. Subjects were from 29 to 36 years old at the 10 to 15 year follow up period. Initial testing was done while they were university students. The

results revealed high reactivity at the initial testing was predictive of higher BP levels 10 to 15 years later.

Cinciripini (1986a) noted a number of studies that revealed cardiovascular reactivity was related to future development of hypertension. He reported that

(1) average ambulatory BP, rather than casual BP, was predictive of future hypertension (Perloff, Sokolow, Cowan, 1983),

(2) BP during work, rather than at rest is more predictive of hypertension (Devereux, Pickering, Harshfield, Kleinert, Denby, Clark, Pregibon, Jason, Kleiner, Borer, & Laragh, 1983),

(3) BP reactivity of air traffic controllers was related to development of hypertension (Rose, Jenkins, Hurst, 1978 and Jenkins, Hurst, Rose, Anderson, Kreger, 1984; both cited in Cinciripini, 1986a), and

(4) borderline adolescent hypertensives' SBP reactivity to mental arithmetic was indicative of hypertension development five years later (Falkner, Onesti, and Hamstra, 1981; cited in Cinciripini, 1986a).

Falkner, Kushner, Onesti, and Angelakos (1981) also reported a study of borderline adolescent hypertensives and development of essential hypertension. Subjects who developed hypertension within 41 months differed from subjects who remained borderline hypertensive in having a family history of essential hypertension, higher resting HR and BP, and higher cardiovascular response (i.e. absolute scores) to a mental arithmetic test.

Stress has also been implicated in the etiology of hypertension (Chesney and

Black, 1986). People in a stressful, Western environment, have higher blood pressure than, for example, "22 populations living in small, cohesive, protected societies" (Chesney and Black, 1986, p. 572). Chronic exposure to stress in humans has been linked to hypertension as has chronic stress in animals been linked to pathogenesis of elevated blood pressure (Chesney and Black, 1986). Faulkner and Ragonesi (1986) examined the interaction of psychosocial and physiological factors on the cardiovascular system of young people and proposed that a "... stress neurogenic component" (Faulkner and Ragonesi, 1986, p. 779) was a mediator in the development of essential hypertension.

Rationale for Anger-Management Style Hypotheses

As noted earlier, as research progressed on the coronary prone behaviour pattern the focus shifted from a macro assessment of the Type A/B classification to a micro view of components of the Type A behaviour pattern such as anger and hostility. The same pattern has occurred with respect to blood pressure and heart rate reactivity studies. Although there have been negative findings, research is pointing toward suppressed anger (Cottington, Matthews, Talbott, & Kuller, 1986; Julius, Harburg, et al., 1986 cited in Ewart, 1991) or hostility as common personality characteristics in essential hypertensives (Ewart, 1991; Chesney and Black, 1986; Diamond, 1982). Tables 1-3 gave an overview of articles that examined reactivity and Type A components of the structured interview. Only five of the research teams used the structured interview as the challenging task while BP and HR reactivity measures were taken. Only three of these studies found significant relationships between SI components and blood pressure. Particular

interest should be paid to inconsistencies of the results; no two studies found similar relationships, between SI components and blood pressure, to be significant.

Hypotheses

None of the studies reviewed have reported on predictors of catecholamine reactivity during the structured interview. For this study, it was hypothesized that

1. Anger-in and potential for hostility, components of the SI coding system, would be better predictors of catecholamine reactivity during the SI than paper and pencil scales for determining TABP.

As Houston (1988) summarized, there have been a few studies that addressed the relationship of SI components and reactivity. The results of these studies, however, were inconsistent thus demonstrating the need for more studies in this area. It was further hypothesized that

2. One or more of the SI components would be predictive of BP reactivity while the subjective scales would not predict reactivity as well as the SI components, and
3. One or more of the SI components would be predictive of HR reactivity while the subjective scales would not predict reactivity as well as the SI components.

METHODS

Subjects

Data obtained for this study were obtained from a larger, ongoing, longitudinal study on stress and cardiovascular reactivity: the Waterloo Longitudinal Reactivity Study (WLRS). The students consented to be re-tested annually over a two year period. To be accepted into the study, each student had to meet the following inclusion criteria:

in his or her first or second year of university studies,
18-25 years of age,
in good health, and
in good academic standing.

Subjects were excluded from participation if they had a history of cardiovascular or related problems, if they had a chronic illness, or if they were currently taking medication which would jeopardize the reliability and validity of the results.

The present study made use of data from the first 240 subjects who completed the first testing session. Upon completion of the experimentation session that provided the present data, subjects were remunerated $20.00.

Apparatus

Blood samples were obtained from an intravenous catheter unit, inserted in the anti-cubital area of the arm. The catheter was connected to a PRN adaptor - a luer-lock adaptor (Deseret Medical Incorporated) with 0.3 ml fluid capacity. A 23 G 3/4", 3" tubing minicath, infusion set shorty (Deseret Medical Incorporated) was attached to the PRN adaptor. The minicath tubing was connected to a 3-way stopcock used to control the flow of blood. A 10 CC syringe was connected to

the stopcock. The interface used in the whole system contained 50 Units/litre of ammonium saline solution. Following centrifugation (5,000 revolutions per minute for 20 minutes) and separation of plasma, plasma epinephrine and norepinephrine levels were measured by high-pressure liquid chromatography (HPLC) using a Waters 712 WISP cooling system, Waters 740 data module, Waters Millipore operating pressures, and Waters M460 monitor. The procedure used to extract the catecholamines from the plasma is documented in Appendix C (Weicker, Feraudi, Hägele, Pluto, 1984).

The Structured Interview (SI) was recorded on a Sony, TCM 500 DEV tape recorder. The microphone was located 20 cm from the subject, level with his/her head. A videotape of the interview was also made using an Hitachi 5 head portable video cassette recorder and a JVC colour video camera.

Blood pressure and heart rate were obtained from a non-invasive ambulatory blood pressure monitor (SpaceLabs Ambulatory Monitor, 90202). The blood pressure cuff was placed on the arm opposite to that of the intravenous catheter.

Heart rate and skin temperature were measured using a Coulbourn Instruments physiograph. A photoplethysmograph was used to measure heart rate. The device was placed on the distal phalange of the middle finger. Skin temperature was recorded by a YSI reusable temperature probe. It was taped onto the distal phalange of the index finger.

During habituation and test sessions, subjects were seated upright in a reclining chair in the experimental room. Their catheterized arm was resting on a ledge hidden from their view. Obstructing the subjects' view was done to minimize

their reaction to drawing blood samples.

The experimenters observed the subjects through a one-way mirror. One experimenter entered the room periodically to administer questionnaires and draw blood samples. Blood pressure, heart rate, and skin temperature wires were run through a small hole in the wall behind the subjects' chair leading to the equipment in the next room.

Procedure

Initial Contact

To recruit participants, flyers were liberally placed across the University of Waterloo campus announcing the study and $20.00 remuneration. Frequently, flyers were checked to ensure visibility was maintained. At the beginning of each term, advertisements were placed in the students' newspaper, Imprint, requesting participants. Persons interested in obtaining more information about the study were asked to call the laboratory.

During the telephone conversation, more information was given about the study (e.g. time commitment, venipuncture) and a number of questions were asked to determine eligibility of the caller to participate according to the inclusion criteria. At the end of the conversation, they were thanked for their time and either asked to participate in the study or told they were not eligible to participate. Subjects accepted into the study were asked to come to the laboratory on up to four separate occasions to provide consent and a fasting blood sample (in the morning), for habituation and the test session proper. If possible, fasting blood samples were collected at the end of the consent or habituation sessions; however, if this was

not feasible a separate time was scheduled to obtain this blood sample. For the fasting session, subjects were required not to eat for 12 hours before the blood sample was taken. They were, however, encouraged to drink as much water as they wanted. Fasting samples were used to determine cholesterol levels.

Consent Session

Subjects were asked to come to the laboratory for one-half hour to read more about the study and to complete the following forms: consent to participate in the study, consent to allow videotaping, health status form, and a scale of their current emotional state (a modified Differential Emotions Scale [DES], Izard, 1971). At this point, subjects could still be rejected from the study if their current or past health would confound study results or if their resting blood pressure was greater than 140/90 after three blood pressure readings.

At the end of the consent session, the remaining appointments were scheduled. The optimum time frame was to schedule the habituation session one week later and the test session one week after that. Reminder sheets indicating appointment dates and times were given to the subjects. Appointment sheets also indicated restrictions to be followed before each session. Standard restrictions for each session were as follows:

Smoking	-no smoking for 3 hours before the session,
Eating	-no eating for 3 hours before the session,
Exercise	-no exercising for 24 hours before the session,
Alcohol	-no alcohol for 24 hours before the session, and
Caffeine	-no caffeine (chocolate, coffee, tea, pop) for 24 hours before.

Habituation Session

The purpose of this 1.5 hour session was to adapt subjects to the laboratory setting, researchers, experimental procedures, and venipuncture. Past research has shown subjects' reactivity to be higher at first exposure to a laboratory setting than in subsequent visits (Prkachin & Mills, 1988), while reactivity in subsequent visits is more stable.

At the beginning of the session, subjects were asked a number of questions to assess whether they followed the restrictions outlined for the session. Subjects who did not abide by the restrictions had their appointments re-scheduled and were reminded of the importance of following restrictions for future appointments.

If all restrictions were followed, the session began with insertion of the intravenous catheter system. Subjects were then seated upright in the reclining chair. The blood pressure cuff, photoplethysmograph, and thermistor were attached. Heart rate and skin temperature readouts were on a continual output to physiograph paper for later scoring.

Subjects were told which tasks they would be asked to perform during the session (see Figure 1). They were then given reading material and asked to relax for the first half-hour. This period was used to adapt subjects to the environment. The relaxation period began with a blood pressure reading. At the end of the period, a second blood pressure reading and a blood sample were taken. Each blood sample was added to a test tube that contained 100 uL of antioxidant and stored in ice. The anti-coagulant used was glutathione and EGTA. The procedure for mixing the solution is located in Appendix B. Samples were centrifuged and

plasma extracted and frozen for later analyses.

At the end of the relaxation period, subjects watched a twelve-minute videotape of a comedy routine. They were assured that the purpose of the task

Minutes	Task	Measure
0	Start Adaptation	bp, hr
30	End Adaptation	bp, hr, bl
0	Start Comedy Routine	
2		bp, hr
7		bp, hr
12	End Comedy	bp, hr, bl
0	Start Recovery	
3		bp, hr
6	End Recovery	bp, hr
0	Start Role Play	
2	End Role Play	bp, hr, bl
0	Start Recovery	
3		bp, hr
6	End Recovery	bp, hr
End of Session		

Legend: bp = blood pressure
hr = heart rate
bl = blood sample

Figure 1
Time Frame for Habituation Session

was simply to enjoy the video. During the video, blood pressures were taken at two, seven, and twelve minutes. A blood sample was also obtained at the twelve-minute mark. Participants were then asked to answer two questions regarding how they enjoyed the video. The next six minutes were a recovery period to allow subjects to relax. At the three-minute and six-minute mark, blood pressures were recorded.

The next task was a role play. Subjects were asked to imagine that the experimenter was their roommate. The roommate had just came home from writing a midterm that she thought she had failed. She was terribly upset. The object of the role-play was one of the experimenters, who acted according to a prepared protocol. Subjects were encouraged to ask any questions before the role play began. At the end of the two-minute task, blood pressure reading and blood sample were taken. Participants were then asked to fill in another questionnaire to indicate how they did during the role play. The session ended with another six-minute recovery period. Blood pressures were again taken at three- and six-minute marks.

At the end of the session, all equipment was disconnected from the subjects. They were asked to complete a package of questionnaires -to be identified in a few pages- at home and return the package during their next appointment, the test session, approximately one week later.

Test Session

The purpose of this two-hour session was to measure subjects' reactivity to psychologically, socially, and physiologically demanding tasks.

Again, the session began when the experimenters assessed if participants followed restrictions for the session (e.g. not smoking). Appointments were rescheduled for subjects who did not follow the restriction protocol.

As with the habituation session, the test session proceeded with insertion of the intravenous catheter system, and applying the blood pressure cuff, the photoplethysmograph, and the thermistor.

Subjects were then informed of the tasks they would be doing for the session: half-hour relaxation, Structured Interview, mental arithmetic, Stroop test, favourable impression test, and cold pressor (Figure 2). In the interest of simplicity, when physiological measures were taken will not be discussed since these are explained in Figure 2.

After the relaxation period, subjects participated in the Type A Structured Interview (SI). The SI is a standardized interviewing technique that is designed to elicit a variety of behavioural "signs" in appropriately predisposed individuals (Chesney, Eaglestone, & Rosenman 1980). In particular, it is constructed and performed in such a way as to provide an opportunity for the interviewee to display elements of "coronary-prone behaviour", such as competitiveness, energetic responding and hostility. This is done through both the content of questions (e.g. "What about the people you work with makes you angry?") and the style of the interviewer, which is businesslike, brisk, and occasionally challenging. The subjects' responses are monitored to identify inconsistencies or ambiguities. When these are found, the interviewer challenges the subjects to clarify their response.

Minutes	Task	Measure
0	Start Adaptation	bp, hr
30	End Adaptation	bp, hr, bl
0	Start SI	
2		bp, hr
7		bp, hr
12	End SI	bp, hr, bl
0	Start Recovery	
3		bp, hr
6	End Recovery	bp, hr
0	Start *	
2	End *	bp, hr, bl
0	Start Recovery	
3		bp, hr
6	End Recovery	bp, hr

End of Session

*=Stroop, or MA, or CP, or FI

NOTE: The marked area was repeated for each of the four tasks.

Legend: bp=blood pressure
hr=heart rate
bl=blood sample

Figure 2
Time Frame for Test Session

The order in which each subject completed the next four tasks was randomized. Each task lasted for two minutes. Before each task began, the experimenter would give instructions to the subject over an intercom. All instructions were read from a script.

The mental arithmetic task involved subjects repeatedly subtracting a two digit number from a four digit number (Williams, 1987). Both were odd numbers. A metronome was set to click every second in the background. Participants were told to give an answer on every second click of the metronome; in order to create a rushed feeling and increased task difficulty level. If an incorrect answer was given, the experimenter gave the correct answer and the subjects were expected to continue subtraction using this correct number. A final score consisted of the number answered correctly over the total number answered.

The Stroop test (MacLeod, 1991) consisted of a Bristol board filled with words organized into rows and columns. The words were colours: red, green, blue, purple, and brown. Each word was written in a coloured marker; however, the colour that the word was written in did not correspond to the written word (e.g. the word red was written in the colour purple). Subjects were asked to say the colour the word was written in and not the word. They were to do this as fast as they could for two minutes. Mistakes were not corrected. A score consisted of the number correctly answered over the total number answered.

For the favourable impression task (Borkovec, Stone, O'Brien, Kaloupek, 1974), the subjects were told that a person of the opposite sex would come into the room and sit down in the chair in front of them. The assistant had been trained not to talk to the subjects or to react to anything they said. The subjects' task was to impress the assistant. They were told they could do anything they wanted to try and make a favourable impression. The two minute task was video taped.

At the end of the favourable impression task, both subjects and assistants

were asked to complete a rating scale. Subjects were asked to rate how well they impressed this person; assistants were asked to rate how well the subjects had impressed them. Subjects were not allowed to see the assistants' ratings.

The cold pressor was the physical task used to elicit reactivity. Subjects were asked to immerse their arm, up to their elbow, in ice water. The water was circulating and maintained at a temperature between zero to one degree celsius. The maximum duration of the task was two minutes. If the subject could not endure the pain for this length of time, they were allowed to remove their arm.

All equipment was disconnected from the subject, at the end of the study. Subjects were paid $20.00.

Protocol Used for This Paper

The main purpose of the present study was to assess which measures of anger-management best predict catecholamine, blood pressure, and heart rate reactivity during the SI. To achieve this purpose, a subset of the data collected in the WLRS was used. Two procedures for assessing anger-management were used: psychometric scales and coding of the SI.

Instruments

Anger-Expression Scale

The Anger Expression (AX) Scale (Spielberger, Johnson, Russell, Crane, Jacobs, & Worden, 1986) is a device for measuring trait-anger-expression. It assesses two types of trait anger: anger-in and anger-out. Anger-in is defined as "... how often angry feelings are experienced but not expressed" (Spielberger, et al., 1986, p. 14). Anger-out, is defined as "... the extent that an individual

engages in aggressive behaviours when motivated by angry feelings" (Spielberger, et al, 1986, p. 14).

The AX scale consists of 24 items. Subjects are asked to indicate the frequency with which they demonstrate each stated action (e.g. I lose my temper) on a four-point scale: (1) almost never; (2) sometimes; (3) often; (4) almost always.

Table 5 provides the means and standard deviations of AX scores taken from a high school sample.

Table 5
Mean and Standard Deviation Scores for the Anger Expression scale (Spielberger, et al., 1986)

	TOTAL ANGER EXPRESSION		ANGER IN		ANGER OUT	
	M	F	M	F	M	F
Mean	46.30	48.05	18.92	18.04	16.64	16.75
Standard Deviation	9.07	8.33	5.93	5.28	4.18	4.38

State-Trait-Anger Scale

The State-Trait-Anger Scale (STAS) is comprised of two scales each with 15 items that measure state anger and trait anger (Spielberger, Jacobs, Russell, & Crane, 1983). State anger is defined as "... an emotional state or condition that consists of subjective feelings of tension, annoyance, irritation, fury and rage, with concomitant activation or arousal of the autonomic nervous system" (Spielberger,

et al., 1983, p. 166). Trait anger is defined as a characteristic of individuals who "...perceive a wide range of situations as anger-provoking (e.g. annoying, irritating, frustrating), and ... respond to such situations with elevations in state anger... [or who] ... experience more intense elevations in [state anger] whenever annoying or frustrating conditions [are] encountered" (Spielberger, et al., 1983, p. 167).

For the purposes of this study, only the trait-anger scale was used, since the object was not to assess momentary anger, but anger that occurs more regularly.

The trait-anger scale consists of ten items, derived from the Buss-Durkee Hostility Inventory (BDHI) (Buss & Durkee, 1957; cited in Spielberger, et al., 1983), other measures of anger and hostility, and new items. In response to each statement (e.g. "I am a hotheaded person"), subjects indicate how much they generally felt like the statement: (1) almost never; (2) sometimes; (3) often; (4) almost always. The internal consistency of the trait-anger scale is .87 (Spielberger, et al., 1983). Norms for college students on the trait-anger scale are presented in Table 6.

Table 6
Normative values for Trait-Anger Scale (Spielberger, et al., 1983)

SAMPLE	MEAN	STANDARD DEVIATION	ALPHA
Females	19.5	5.0	0.9
Males	19.2	4.9	0.8

Cook-Medley Hostility Scale

In 1954, Cook and Medley developed a 50 item sub-scale from the Minnesota Multiphasic Personality Inventory (MMPI) (Cook & Medley, 1954). The scales were initially constructed to test the rapport of teachers with pupils. However, the content of the scales is generic such that it may be valid to use them in any situation. The selection of items for the scale, in addition to studies of its construct validity, support the characterization of the scales as assessing a trait of "cynical hostility" (Smith & Frohm, 1985). There are several (Table 7) subscales within this scale that were developed by Barefoot et al. (1989): hostility, attributions, cynicism, hostility affect, aggressive responding, and social avoidance. Several studies have used this scale in investigation of cardiovascular parameters (Barefoot and Colleagues, 1983; Barefoot, et al., 1989; Helmer, Radland, & Syme, 1991; Krantz, Contrada, Hill, & Friedler, 1988; Scherwitz, Perkins, Chesney, & Hughes, 1991; Shekelle, et al., 1983; Weidner, et al., 1989; Williams, 1984).

Daily Hassles Scale

Kanner, Coyne, Schaefer, and Lazarus (1981) developed the Daily Hassles Scale to evaluate the health impact of hassles on subjects' lives. Hassles are defined as "... the irritating, frustrating, distressing demands that to some degree characterize everyday transactions with the environment" (Kanner et al., 1981, p. 3). Examples of Daily Hassles Scale would be losing ones keys, and financial and family concerns. The scale consists of 117 questions. In response to each question, subjects indicate if the hassle is an irritant and to what degree. Three scores are calculated from this scale. The first score, frequency, consists of the

Table 7
Values for Cook-Medley Subscales

VARIABLE	MALES (n=570)		FEMALES (n=645)	
	MEAN	STD	MEAN	STD
Cynicism	4.7	2.9	3.7	2.8
Social Avoidance	1.5	1.1	1.4	1.1
Other	2.5	1.5	2.1	1.4
Hostile Attribution	3.5	2.6	3.3	2.5
Hostile Affect	1.8	.3	1.9	1.3
Aggressive Responding	3.4	1.8	3.0	1.8
Sum: cynicism, hostile affect, aggressive responding	11.6	6.0	10.0	6.0

Taken from Scherwitz, Perkins, Chesney, and Hughes (1991).

number of times, that a hassle is endorsed. Cumulative severity, the second score, is the sum of the severity ratings - these ratings ranged from one to three. The third score, intensity is a measure of the cumulative severity divided by the frequency. Kanner et al. (1981) found a high correlation (r=0.95) between frequency and cumulative severity. Normative values are presented in Table 8, Table 15.

The reactivity measures of interest were plasma catecholamines (epinephrine and norepinephrine), systolic blood pressure, diastolic blood pressure, and heart rate. Reactivity was measured during the SI. Reactivity was defined as

Table 8
Normative values for Daily Hassles Scale

	FREQUENCY		INTENSITY	
	MEAN	STANDARD DEVIATION	MEAN	STANDARD DEVIATION
Males	22.4	16.9	1.43	0.27
Females	18.9	13.3	1.49	0.29

the difference between a measure during a task and a measure during a relaxed state. For the catecholamines, the baseline measure was taken at the end of the 30-minute relaxation session and the task response measure at the end of the 12-minute SI. For the other dependent measures, the baseline value was taken as the lowest of the habituation or test session 30-minute relaxation measures. The 7-minute measure during the SI was used as the task response measure because it was higher than the 12-minute task response measure. These measurement periods were chosen so as not to underestimate the magnitude of the cardiovascular response (Krantz & Manuck, 1984). A number of potential relevant covariates were also examined. These included measure of smoking, alcohol consumption, family history of cardiovascular problems, total cholesterol, body mass index, gender, resting blood pressure.

Coding the Structured Interview

The majority of the interviews were coded using audiotapes, however in instances where an audiotape recording was not available a videotape recording was used. When the videotape was used for scoring, the television screen was

always covered. This procedure eliminated possible visual biases which could influence the coders' scoring.

The coding procedure used was that developed by Dembroski and MacDougall (1983). The scoring system has seven components: loud voice, explosive speech, rapid & accelerated speech, response latency, potential for hostility, anger expression, and competition for control of the interview. In each case, less attention is paid to what the person says than to how they are saying

Table 9
SI components in the Multiple Risk Factor Intervention Trial (Dembroski, MacDougall, Costa, & Granditis, 1989)

SI	MEAN		STANDARD DEVIATION	
	Cases	Controls	Cases	Controls
Speech Style[1]	3.38	3.37	0.96	0.93
Verbal Competition	2.49	2.46	1.26	1.06
Anger-In	2.05	2.10	1.02	0.94
Potential for Hostility	2.77	2.58	1.12	1.15

[1] Speech Style was created as a combination of loudness, explosive voice, rapid and accelerated speech, and short latency answers.

it. For example, voice stylistics may be more expressive than what the person is saying.

Statistical Analyses

Statistical analyses involved calculating means and standard deviations, plotting normal curves, and conducting factor and regression analyses.

Factor analyses were done using the behavioural measures. Principal components factor analyses with varimax rotation were used to identify variables that loaded significantly on factors. Results of the analyses were used to decrease the number of variables that were entered into the regression model since the sample size was not sufficiently large to accommodate the large number of variables.

Stepwise regression analyses were used to assess which predictor variables (eg. smoking, family history) best estimated catecholamine, blood pressure, and heart rate reactivity (response variables). The stepwise procedure began with none of the predictor variables entered into the equation. At each step in the regression model, a variable was considered for entry as well as any variables already in the model are considered for removal, if appropriate. Only variables that added significantly to the model are entered into the equation. The criterion used for significance was the Probability of F-to-enter, set at $p < .05$.

RESULTS

Sample Description

In total, 111 females and 129 males completed year one testing. The mean age was 20.44 years old with a standard deviation of 1.64 years. The students were in their first or second year of university.

Measures of Anger-Management Style

Means, standard deviations and ranges of the anger-management style variables are found in Table 10.

Table 10
Means, Standard Deviations and Ranges for Structured Interview Variables.

Variables	Mean	Standard Deviation	Range
Voice (SI)	2.54	0.87	1 - 4
Expression (SI)	2.26	0.69	1 - 5
Latency (SI)	2.65	0.65	2 - 5
Speed (SI)	2.89	0.79	1 - 5
Hostility (SI)	2.57	1.08	1 - 5
Anger Expression (SI)	2.70	0.78	1 - 5
Competition (SI)	3.05	1.02	1 - 5

The results of the SI coding system show a pattern of giving midline codes, suggesting that the coders were avoiding the extreme scores (Table 10).

Inter-rater reliability on the SI coding was assessed by calculating Pearson

correlations between each coder's set of ratings. Three coders were used. One coder (BES) assessed all interviews; the other two coders (KR & JB) split 120 of the 240 interviews between them. Reliability was assessed between BES and KR and then BES and JB (Table 11 and Table 12). In all cases, reliability appeared to be adequate.

Table 11
Inter-rater reliability between BS & KR
(N=46)

SI	VO	EX	LA	SP	HO	AX	CO
VO	.76*						
EX		.77*					
LA			.69*				
SP				.81*			
HO					.91*		
AX						.86*	
CO							.91*

* $P < .001$

Legend: VO = Voice
EX = Expression
LA = Latency
SP = Speed
HO = Hostility
AX = Anger Expression
CO = Competition

Table 12
Inter-rater reliability between BS & JB
(N = 134)

SI	VO	EX	LA	SP	HO	AX	CO
VO	.74*						
EX		.55*					
LA			.66*				
SP				.63*			
HO					.91*		
AX						.88*	
CO							.87*

* $\underline{P} < .001$

Legend: VO = Voice
EX = Expression
LA = Latency
SP = Speed
HO = Hostility
AX = Anger Expression
CO = Competition

Table 13
Means, Standard Deviations and Ranges for Anger Expression Scale and Trait Anger Expression Scale

Variables	Mean	Standard Deviation	Range
Out (AX)	15.17	3.81	8 - 29
In (AX)	17.43	3.99	9 - 32
Control (AX)	23.22	4.56	13 - 32
Total (AX)	25.39	8.46	6 - 50
Trait Anger Scale	29.24	6.30	18 - 58

Values for the Anger Expression Scale (Table 13) were comparable to published norms. The scores indicate that subjects as a group were more likely to control their anger when confronted with an anger provoking situation (Control (AX) = 23.22). The Trait Anger Scale results are quite a bit higher than the normative values for males (Mean = 19.2) and females (Mean = 19.6). This would indicate that the present sample of university students exhibited more trait anger than the high school students did in the normative testing.

Results from the Cook-Medley Scale (Table 14) were higher than the values obtained in Scherwitz, et al. (1991). The subjects used in Scherwitz et al. (1991) were from the general public, while subjects in the present study were from university.

The Hassles Scale results are comparable to the normative values for that scale (Table 8, Table 15). On the whole, the results from this sample indicate that there is a large variation between subjects on the scores obtained, as indicated by the large standard deviations for Frequency and Cumulative Severity score. The large standard deviations may have been due to subjects who did not read the directions carefully. The directions indicated that a response was only to be circled if it was relevant to the person completing the scale. Some subjects, however, indicated every hassle was pertinent to them. This occasionally stretched credulity, especially when male subjects indicated that they were hassled by troubles of getting pregnant.

Table 16, Table 17, and Table 18 present the results from the remaining independent variables and the dependent variables.

Table 14
Means, Standard Deviations and Ranges for Cook-Medley Scale

Variables	Mean	Standard Deviation	Range
Cynical (CM)	5.63	2.57	1 - 13
Social Avoidance (CM)	1.82	0.87	1 - 4
Other (CM)	2.63	1.29	1 - 7
Hostile attribution (CM)	4.01	2.05	1 - 10
Hostile Affect (CM)	2.31	1.13	1 - 5
Aggressive responding (CM)	3.70	1.71	1 - 8
Hostile (sum of cynicism, hostile affect, aggressive responding) (CM)	12.04	3.96	3 - 24

Total Cholesterol values were within the normative range for this age group (140-270 mg/dL for 20-39 year old); however, the large standard deviation should be noted.

Body Mass Index (BMI) is a composite score of the subjects' height and weight (BMI = [(weight(lbs) * 0.454)/((height(inches) * 0.0254)Exponent 2)]). The mean BMI values are well below the values for obesity which are 27.2 for men and 26.9 for women (Turner, Sizer, Whitney, & Wilks, 1992) and are well within

Table 15
Means, Standard Deviations and Ranges for Hassles Scale

Variables	Mean	Standard Deviation (Kurtosis, Skewness)	Range
Frequency (Hasl) (log)	29.12	21.72 (.799, .021)	3 - 117
Cumulative Severity (Hasl)	46.55	33.36	3 - 190
Intensity (Hasl) (log)	1.59	0.35 (.002, .558)	1 - 2.67

normative values. The range of scores for BMI indicate that there were some under-weight and over-weight individuals.

Not many subjects in the study smoked or drank. Subjects were given one of two codes for smoking: 1 was given for subjects that smoked, and 0 was given to subjects that did not smoke. Only 10 subjects smoked which accounts for the very low mean and standard deviation. Due to the nature of this sample population, these results were anticipated.

Table 16

Means and Standard Deviations of Cholesterol level, BMI, Smoking, Drinking, and Activity Level.

Variable	Mean	Standard Deviation[1]	Range
Resting SBP (mmHg)	115.22	9.97 (.508, .624)	95 - 148
Resting DBP (mmHg)	70.00	7.02 (1.195, .657)	51 - 91
Resting HR (mmHg)	59.72	8.59 (-.194, .231)	39 - 86
Resting Norepinephrine (pg) (log)	199.66	32.10 (.565, -.088)	31.95 - 945.35
Resting Epinephrine (pg) (log)	35.33	52.47 (1.926, -.332)	.426 - 534.92
Total Cholesterol (mg/dL)	147.40	35.41 (.066, -.069)	73.79 - 282.48
BMI (log)	22.10	2.70 (1.561, .206)	13.53 - 33.01
Activity Level (Met-Minute) (log)	3459.29	3043.16 (.993, -.539)	136.5 - 27,514.55

	NO Percentage (n)	YES Percentage (n)
Smoking	95.92% (235)	4.08% (10)

	NO Percentage (n)	1-14 Percentage (n)	> 14 Percentage (n)
Drinking	37.96% (93)	56.74% (139)	5.31% (13)

[1] (kurtosis, skewness)

The variable drinking was coded similar to the smoking variable except three levels of coding were used: 0 was given if they did not drink in the past week (N=93), 1 was given if they had 1 to 14 drinks in the past week (N=139), and a code of 2 was given if they had consumed more than 14 drinks in the last week (N=13). This system was used because of evidence that more than 2 drinks per day is harmful to your health (Levenson, 1986).

The score for activity level represents a composite of how active the subjects were in the week before they completed the questionnaire. Subjects were asked to indicate, during the past week, how many times they did any of the following exercises, sports or recreational activities: walking, jogging or running, calisthenics, bicycling, vigorous dancing, skating, skiing (downhill and cross-country), racquet sports, team sports, golf, swimming, and other activities. They were also asked how much time they spent on the activity: 1-15 minutes, 16-30 minutes, 31-60 minutes, or more than 60 minutes. These scores were converted to the midpoint for each block of time: 7.5 minutes, 23.5 minutes, 45.5 minutes, and 63.5 minutes. The midpoint of 63.5 minutes for the more than 60 minutes of activity was calculated using a simple linear prediction equation (see Figure 3). Activity levels were equated by converting the energy expenditure for each activity into Metabolic Equivalents (METs) (see Table 17) . One Met is equivalent to 1.0 kcal/kg•hr. Conversions for each activity were calculated as follows: Met•Min•Freq = Mets • Midpoint for the time block • frequency in last week. The activity level for each subject was a summation of the Met•Min•Freq scores for all activities.

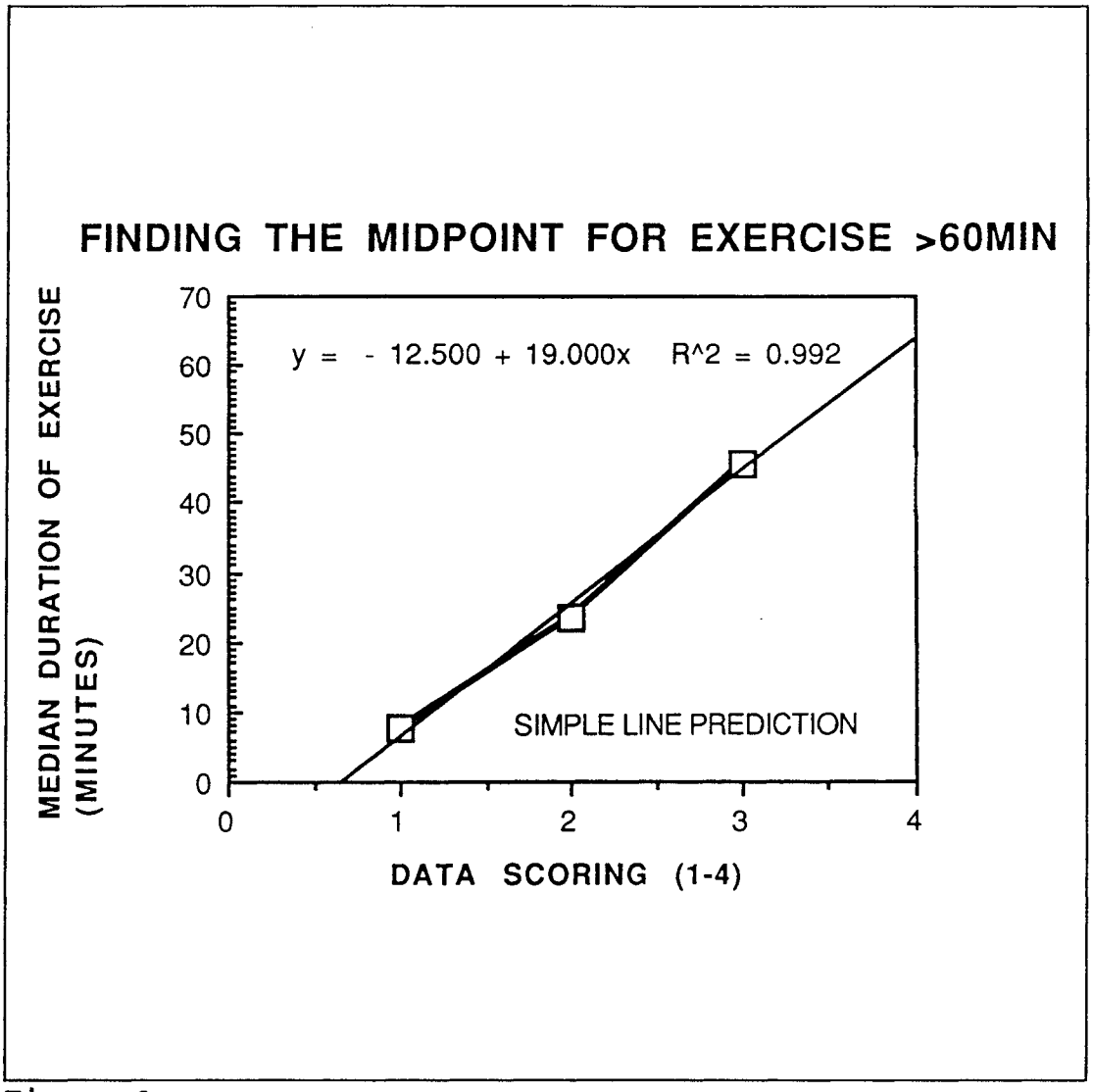

Figure 3
Linear Prediction of Exercise Midpoint

The parental history of cardiovascular problems was completed by the subjects' parents via a mailed questionnaire. The results indicate that none of the subjects' mothers had experienced a heart attack or stroke (Table 18).

In the interest of decreasing the number of variables that comprised the proposed model for regression analyses, the parental variables have been

Table 17
Metabolic Equivalents for Numerous Activities

The following values were found in numerous sources (American College of Sports Medicine, 1980; Hatfield & Krotee, 1984; McArdle, Katch, & Katch, 1981).

Walking	7.2
Jogging	18.95
Biking	5.5
Dancing	5.55
Skating	6.5
Skiing	7.75
Racquet Sports	8.5
Team Sports	6.98
Golf	5.1
Swimming	6.0
Calisthenics	[200 Kcal/hr - (RND(weight-154)/5)*10)]/weight
House Work	[185 Kcal/hr - (RND(weight-154)/5)*10)]/weight
Rowing	[900 Kcal/hr - (RND(weight-154)/5)*10)]/weight
Weight Lifting	11.1
Aerobics	7.5
Kendo	13.5
Foot Ball	7.9
Tai Chi	13.5
Roller Skating	6.5
Sit ups	5.5
Bowling	3.0
Self Defense	13.5
Karate	13.5
Push ups	5.5

compressed into one variable: family history. The variable was given a code of 0 if neither the mother or father had indicated the presence of high blood pressure or heart attack in their medical history. A value of 1 was assigned if either parent indicated either cardiovascular problem (N=54).

The results for the dependent measures indicate cardiovascular reactivity during the Structured Interview (Table 19). Reactivity was calculated as the difference between the resting value and the response value at the end of the SI.

Table 18
Frequency of Cardiovascular Problems experienced by the Parents.

Variable	Yes Percentage (n)	No Percentage (n)
High Blood Pressure (M)	9.1% (16)	90.9% (160)
High Cholesterol (M)	6.2% (11)	93.8% (165)
Heart Attack (M)	0.0% (0)	100.0% (176)
Stroke (M)	0.0% (0)	100.0% (176)
High Blood Pressure (F)	16.3% (28)	83.7% (144)
High Cholesterol (F)	15.7% (27)	84.3% (145)
Heart Attack (F)	5.8% (10)	94.2% (162)
Stroke (F)	0.6% (1)	99.4% (171)

Legend: M = Mother F = Father

Of concern, however, are the large standard deviations for norepinephrine, epinephrine, and diastolic blood pressure. These values are due to a few scores that were quite a bit higher than the rest. These scores were flagged in the regression analyses in case they were influential.

An analysis of variance, testing the null hypothesis that the mean reactivity score was zero was conducted on the dependent measures. Only epinephrine reactivity was not significantly different from zero indicating that reactivity did not occur with respect to this measure, but did on the others (Table 20).

Table 19
Mean, Standard Deviation and Range of the Dependent Measures. These values represent Reactivity Scores.

Variable	Mean	Standard Deviation (Kurtosis, Skewness)	Range
Norepinephrine (pg)	30.38	99.80 (8.613, -1.051)	(- 454.65) - 454.85
Epinephrine (pg) (log)	9.50	92.28 (14.872, -1.13)	(- 118.73) - 1042.40
Systolic Blood Pressure (mmHg)	14.42	7.83 (.212, .419)	(-7) - 36
Diastolic Blood Pressure (mmHg)	13.87	7.91 (1.03, .427)	(-3) - 49
Heart Rate (beats/minute)	11.58	7.18 (.392, .550)	(-7) - 35

Table 20
MANOVA results for dependent measures

VARIABLES	**F**	**SIGNIFICANCE** ($p <$)
Norepinephrine	17.36	.001
Epinephrine	0.13	.718
SBP	431.38	.001
DBP	351.61	.001
HR	311.09	.001

Factor Analyses

The psychometric measures were factor analyzed in order to identify a small number of factors which represent relationships between the psychometric measures. Component scores of the following scales were factor analyzed: Structured Interview (SI), Anger Expression (AX), Cook-Medley (CM), and Hassles (Hasl).

The sample was randomly split into two sub-samples in order to evaluate the comparability of the factor solutions.

In the first phase, variables that had low sampling adequacy in both sub-samples, as determined by the anti-Image covariance matrix, were removed from the analysis. These were Total (AX) from the Anger Expression Scale, Hostility (CM) from the Cook-Medley Scale, and all Hassle scale components. A principal component solution with rotation to simple structure via the varimax criterion was employed. Factors that had eigenvalues greater than one and passed the scree test were considered acceptable factors. Variables in these factors that loaded on the factor greater than or equal to 0.40 were included in that factor (Table 23).

Similar results were obtained for both samples indicating that factor analysis on the whole sample would be valid (Table 21, Table 22).

Table 21
Matrix of Factors (sub-sample 1)

Variable	Factor 1 Hostility	Factor 2 SI	Factor 3 Anger
Out (AX)	.07788	-.07385	.89999
In (AX)	.50905	.02341	-.07037
Control (AX)	-.12715	-.00242	-.63046
Trait Anger Scale	.44814	.00806	.13757
Cynical (CM)	.78965	.06165	.12569
Social Avoidance (CM)	.34878	-.10081	.00792
Other (CM)	.33528	-.29846	.23564
Hostile Attribution (CM)	.61684	-.07502	.19233
Hostile Affect (CM)	.43302	.08703	.45037
Aggressive Responding (CM)	.29585	.07218	.21467
Voice (SI)	-.05661	.48224	-.02171
Expression (SI)	-.02366	.74647	.03144
Speed (SI)	-.01613	.35113	.06777
Latency (SI)	.18554	.30314	.02653
Hostility (SI)	.18826	.53636	.03160
Anger Expression (SI)	.15445	-.26270	-.24161
Competition (SI)	-.04528	.50285	-.05194

Table 22
Factor Loadings (sub-sample 2)

Variable	Factor 1 Hostility	Factor 2 SI	Factor 3 Anger
Out (AX)	.36852	.13499	.75112
In (AX)	.14867	.13337	-.14805
Control (AX)	-.02050	-.03324	-.63931
Trait Anger Scale	.58022	.09528	.10034
Cynical (CM)	.72364	.02152	-.24220
Social Avoidance (CM)	.03410	-.15574	.03849
Other (CM)	.48389	.11175	.12456
Hostile Attribution (CM)	.66870	-.02903	.11090
Hostile Affect (CM)	.35654	.01073	.32985
Aggressive Responding (CM)	.48699	.03438	.30666
Voice (SI)	.01405	.65266	.20989
Expression (SI)	-.11430	.77044	.05655
Speed (SI)	-.07942	.66659	.09241
Latency (SI)	.18110	.56428	.04023
Hostility (SI)	.17399	.43975	.00161
Anger Expression (SI)	-.36297	-.23080	-.18865
Competition (SI)	.08248	.49716	-.14076

Table 23
Matrix of Factor loadings: Combined Analysis

Variable	Factor 1 Hostility	Factor 2 SI Components	Factor 3 Anger
Out (AX)	.18438	.04063	.88015
In (AX)	.43565	.07041	-.12552
Control (AX)	-.08832	-.03877	-.59761
Trait Anger Scale	.55041	.00502	.16838
Cynical (CM)	.74441	.09510	-.01142
Social Avoidance (CM)	.29494	-.12885	-.02366
Other (CM)	.44540	-.08545	.22405
Hostile Attribution (CM)	.59641	.00601	.17331
Hostility Affect (CM)	.53512	.03744	.33081
Aggressive Responding (CM)	.33407	.11120	.28084
Voice (SI)	-.06115	.58391	.08755
Expression (SI)	-.09029	.73558	.02182
Speed (SI)	-.10375	.53515	.07929
Latency (SI)	.09833	.50718	.03245
Hostility (SI)	.18240	.44905	.02758
Anger Expression (SI)	.00587	-.27826	-.25598
Competition (SI)	-.00125	.50078	-.06254

Three new variables were formed based on the results (Table 24):

Factor 1 (Hostility):	Anger In (AX) + Trait Anger Scale + Cynical (CM) + Other (CM) + Hostile Attribution (CM) + Hostile Affect (CM),
Factor 2 (SI Components):	Voice (SI) + Expression (SI) + Speed (SI) + Latency (SI) + Hostility (SI) + Competition (SI), and
Factor 3 (Anger):	Anger Out (AX) - Anger Controlled (AX).

Names for the factors were chosen by taking into account the variables that loaded heavily on that factor and what those variables represented. For example, the name hostility was chosen because a number of the variables were from the Cook-Medley Scale which measures hostility. In addition, the other two variables, Anger In (AX) and Trait Anger Scale can be viewed as part of hostile affect which is negative emotions expressed toward social relationships but that does not imply overt action. SI components was chosen for Factor 2 since all the variables in the factor are from the SI scoring. A general name of Anger was chosen for Factor 3 since both variables in the factor are from anger dimensions, however, they are capturing different aspects of this construct.

Multiple Regression

Each dependent measure was analyzed via stepwise multiple regression. The following independent variables were entered into the equations:
Sex, Age, Total Cholesterol, BMI, Activity Level, Smoking, Drinking, Family History,

Table 24
Mean, Standard Deviation and Range of the Factor Scores.

Variable	Mean	Standard Deviation (Kurtosis, Skewness)	Range
Factor 1 (Hostility)	63.24	12.11 (.872, .758)	41 - 109
Factor 2 (SI components)	15.97	3.18 (-.415, .105)	8 - 24
Factor 3 (Anger)	-8.05	7.36 (-.336, .471)	(-22) - 13

Factor 1 (Hostility), Factor 2 (SI Components), Factor 3 (Anger), and respective resting levels for the dependent measures.

Normality

Normal curves for all variables were plotted before the regression model was attempted. Several of the variables were log transformed to decrease the kurtosis and skewness of their distributions. Kurtosis is a measure of how closely the curve of the data fits a normal distribution. Skewness is a measure of how the variables are distributed within the curve. The more the variables are located in the middle of the curve, the closer skewness is to zero. If the variables fall more in one tail versus the other tail of the curve, the value for skewness increases, either negatively or positively depending on the tail.

In an ideal situation, the values for kurtosis and skewness would be zero. These values would be expected when measurements are normally distributed in

a population. Some of the variables have a high kurtosis (e.g. norepinephrine, epinephrine, diastolic blood pressure, and age), which at first glance may put the value of the variables into question; however, stringent subject selection criteria could explain these non-normal values. For example, the selection criteria for entry into the study was restricted to a certain age range. Based on these selection criteria, one would not expect a normal curve to be apparent because subjects had to be between 18 to 25 years of age.

Regression Equations

Norepinephrine and Epinephrine

For the catecholamines, only the respective resting values entered the multiple regression equations (Table 25). Explained variance in norepinephrine reactivity was 18.1% and 18.6% for epinephrine reactivity. Of the five independent variables, these two had high kurtosis and skewness.

Systolic BP

The model for systolic blood pressure incorporated the variables sex and BMI (Table 26). The model predicted 8.0% of the variance in systolic blood pressure during the Structured Interview. Increased BMI, when controlling for sex, predicted decreased SBP reactivity. The results for the gender variable revealed that, when controlling for BMI, females showed lower SBP reactivity.

The model to predict systolic blood pressure reactivity during the structured interview did not incorporate any of the anger-management style variables that were hypothesized as predictors of cardiovascular reactivity.

Table 25
Prediction of Catecholamine Reactivity During the SI

Model	R^2_{adj}	Multiple R	F	ß
Rest Nor --> Nor$_\Delta$	.181	.433	29.58*	
Rest Nor				-167.71
*p < .0001, df = 1, 128				
Rest Epi --> Epi$_\Delta$	.186	.439	26.08*	
Rest Epi				-.173
*p < .0001, df = 1, 109				

Table 26
Prediction of Systolic Blood Pressure Reactivity During the SI

Model	R^2_{adj}	Multiple R	F	ß
Sex + BMI --> SBP$_\Delta$	.080	.304	7.59*	
Sex (0 = female; 1 = male)				4.64
BMI (higher BMI is more obese)				-30.32
*p < .007 , df = 2, 149				

Diastolic BP

The results from the DBP reactivity model included only resting DBP. The regression model indicates that a higher resting DBP, was predictive of lower DBP reactivity, when all the variables were controlled for in the equation (Table 27).

Table 27
Prediction of DBP Reactivity During the SI

Model	R^2_{adj}	Multiple R	F	ß
Rest DBP --> DBP_Δ	.029	.189	5.55*	
Rest DBP				- 213
*p < .0198 , df = 1, 150				

Heart Rate Reactivity

The HR reactivity model included three independent variables as significant predictors: resting HR, anger (factor 3: Anger Out (AX) + (-Anger Controlled (AX)), and sex (Table 28).

Table 28
Prediction of Heart Rate Reactivity During the Structured Interview

Model	R^2_{adj}	Multiple R	F	ß
Rest HR + Anger (factor 3) + Sex --> HR_Δ	.108	.354	7.07*	
Rest HR Anger (Factor 3) Sex				-.237 -.168 -2.370
*p < .0002 , df = 3, 148				

The multiple R value was .35 and the explained variance was 10.77%. These results indicate that the higher the resting HR and anger (factor 3) value, the

lower the HR reactivity. In addition, males displayed lower HR change.

DISCUSSION

Generally speaking, the results of these analyses provide little support for the a priori expectations that measures of anger-management characteristics, particularly those derived from the SI, would be associated with enhanced cardiovascular reactivity during the SI.

It is important to consider that the hypotheses may not have been supported because the subjects were not reactive during the SI. However, the analysis of variance test for reactivity clearly demonstrated that the SI is provocative and does produce reactivity in all measures except epinephrine. Therefore, the overall lack of support for the hypotheses can't be attributed to lack of reactivity.

Catecholamine Reactivity

Catecholamine reactivity was predicted by resting levels of norepinephrine and epinephrine. Higher resting catecholamine levels were predictive of lower reactivity during the SI. This represents a ceiling effect insofar as the higher the resting level the lesser the reactivity scores will be because cardiovascular levels approach a maximum. Plasma blood samples were taken at the end of the 12-minute SI. Since the lag time between the stressor and the response of the catecholamines is few seconds (Dimsdale & Moss, 1980), the 12-minute plasma catecholamine levels should represent the effects of the stressor on the subjects.

The results did not support the hypothesis that anger-management styles would be predictive of catecholamine reactivity. Possible reasons for these results could be due to the sensitive nature of measuring catecholamine levels (Dimsdale & Ziegler, 1991). Obtaining catecholamines from plasma samples is tedious and

prone to problems. Consequently, there are a number of procedural steps that could decrease the level of catecholamines in the sample and the sensitivity of the testing procedure. For example, the more elapsed time after the end of a stressor the smaller the catecholamine concentration (Dimsdale & Ziegler, 1991). The half-life of norepinephrine is approximately 2.5 minutes and epinephrine is 1.2 minutes (Ward, Mefford, Parker, Chesney, Taylor, Keegan, & Barchas, 1983). It was not always possible, in this study, to obtain blood samples in a short period of time due to technical difficulties. Once the sample was obtained it was kept on ice, centrifuged, and plasma extracted and frozen. Due to the large sample size of this study, it was not possible to analyze the samples at the same time. Another procedural step that may have influenced reliability of catecholamine levels was that different laboratory assistants did the extractions (Dimsdale & Ziegler, 1991). Ideally, the same assistant should do catecholamine extraction to increase reliability of the results. These procedural problems may have jeopardized the likelihood of anger-management style variables or demographic variables entering the multiple regression equations due to the variability of the dependent measures being unrelated to anger-management style.

Although analysis of variance results indicated norepinephrine significantly increased during stress in this study, Floras, vann Jones, Hassan, and Osikowska (1986) cautioned on the usefulness of venous plasma norepinephrine in measuring sympathetic tone. The authors had 51 subjects perform mental arithmetic, static handgrip, and bicycle exercise. Since norepinephrine levels increased only during the bicycle task, the authors concluded that norepinephrine levels were not

consistently reflective of sympathetic tone. Further to this argument, the authors also found that norepinephrine levels were not related to arterial blood pressure when the subjects were engaged in the aforementioned tasks, or when they were seated or supine. If the usefulness of plasma norepinephrine levels were to be questioned, then the results obtained in this study would indicate that either (1) norepinephrine levels were not reliably measuring sympathetic tone and thus that predictive affects of anger-management styles, if indeed they do exist, could not have been detected or (2) norepinephrine levels were reliably measured and, therefore, in this study, anger-management styles were not predictive of norepinephrine reactivity. Unlike Floras and colleagues (1986), Ganguly (1989) found two studies that supported the interpretation of norepinephrine levels as being indicative of sympathetic tone (Goldstein, 1981; Goldstein, 1984). Once again in the research environment, there is both positive and negative support for a particular concept.

Friedman, Beyers, Diamant, and Rosenman (1975) found that epinephrine levels were unchanged between Type A's and Type B's in response to a challenge, while norepinephrine levels were different between the groups. The lack of epinephrine response in the present study could have been due to some of the technical difficulties listed above. A critical review of the literature on Type A behaviour and catecholamines (Glass & Contrada, 1984) showed a number of studies that found Type A/B differences. Results from some of the studies reviewed revealed that Type A's were more reactive than B's while other studies showed the opposite.

BP and HR Reactivity

Lower DBP reactivity was predicted by higher resting DBP. SBP and HR reactivity had a few more predictors than the other dependent measures. SBP reactivity was predicted by sex and BMI. Males were more reactive than females while low BMI was associated with high SBP reactivity. Similar to catecholamine reactivity, these results do not lend support to the predictive nature of anger-management styles in response to the SI.

Variables that predicted HR reactivity were resting HR, anger (factor score), and sex. A higher resting HR contributed to a lower HR reactivity measure when controlling for sex and anger (factor score). Interestingly, males had a lower HR reactivity than females, when controlling for the other variables. The anger score was a factor score comprised of variables from the Anger Expression scale (Anger Out + -(Anger Controlled)). Values for this variable were mostly negative indicating that subjects had more of a tendency to control anger than to express it. The multiple regression results revealed that the more subjects' tended to control their anger the higher their HR reactivity and vice versa.

Julius, Schneider, & Egan (1986) showed that anger-coping styles were predictive of mortality 20 years later. In particular, the results revealed that subjects who suppressed their anger were as 1.7 times the mortality risk of those who expressed their anger. Cottington, et al. (1986) found that suppressed anger interacting with job stress predicted hypertension. Thus, in past studies, suppressed anger has been related to mortality and hypertension, and in the present study, suppressed anger was predictive of HR reactivity.

Light et al. (1992) noted that reactivity was predictive of blood pressure patterns 10 to 15 years later. Although Light and colleagues (1992) did not incorporate anger-management style variables into the regression equations, they did include resting blood pressure, standard risk factors, and parental history of hypertension. Follow-up testing of subjects from the WLRS should provide valuable information on how reactivity was predictive of blood pressure patterns.

As mentioned earlier, Houston (1988) summarized a number of studies that examined the relationship between SI components and reactivity during the SI. Overall, the studies found that SBP, DBP, and HR were sometimes related to the SI components. The current study did not reveal this relationship in the multiple regression analyses possibly because the component scores from the SI were compressed into a factor score, while the studies that Houston examined compared reactivity measures to the components and not the factor score.

A number of researchers have investigated BMI as a risk factor (Curb & Marcus, 1991; Egan, Bassett, & Block, 1991; Pi-Sunyer, 1991). Pi-Sunyer (1991) did a review of the literature on health implications of obesity. He found that increased BMI was related to increased health complications (e.g., hypertension, and CVD). Egan, et al. (1991) conducted a study to assess differences in cardiovascular risk factors between younger and older men. The results showed that BMI (>25.5 kg/m^2) was related to a higher prevalence of hypertension in younger men and not older men. Curb and Marcus (1991) reported on the Honolulu Heart Program (HHP). The results of a 20 year follow-up study revealed that BMI was a predictor of CHD but not stroke.

There have also been two studies that examined the relationship between reactivity and BMI. Alderman, Ooi, Madhavan, and Cohen (1990) assessed the ability of blood pressure to predict MI. The sample consisted of 1737 patients who were diagnosed as hypertensive. The reactivity measure was the difference between the BP measurements taken from the doctor versus the nurse. After the initial assessment period, the patients were treated for their hypertension. The authors found that age, sex, and DBP reactivity at intake predicted MI within a 14-year follow-up period. BMI, however, was one of the independent measures that did not enter into the equation. Sallis, Patterson, McKenzie, Buono, Atkins, and Nader (1989) examined the stability of SBP reactivity to exercise. They had 63 children, mean age 3.9 years, complete a 40-meter run on a number of different days. SBP reactivity was stable both at one-week and at 6-months. Unrelated to SBP reactivity, however, was sex, BMI, and family history of CVD. These studies are thus inconsistent with the present study finding that BMI was predictive of SBP reactivity.

There are two new and important aspects of this study to consider. The first aspect was that there are few studies that examine predictors of reactivity during the SI, therefore this study adds to the literature in this area. Another is the result of the factor analysis. Three factors were formed from a number of psychometric scales (Anger Expression Scale, State Trait Anger Scale, SI components, and Cook-Medley Scale). At first the sample was partitioned into two randomly selected sub-samples. Similar factor analyses were done on both sub-samples. Since similar results were found with both sub-samples, the sub-samples

were aggregated. Three factor scores were formed from these analyses: hostility, SI components, and anger. The factor analyses allowed the identification of factors that simplified investigating the inter-relationships among a large number of variables. These factor scores imply that the factors anger, hostility, and SI components are underlying dimensions that explain the complex phenomena of anger-management styles. For example, hostility was composed of a number of sub-scales that measured aspects of hostility such as cynicism, hostile attribution, and suppressed anger, to mention a few. The number of variables that make-up this hostility factor score indicate the complexity of this anger-management style factor.

It is hard to decipher what anger-management style variable the SI component factor represents. If the original scoring of the SI is examined, it could be possible that the SI components factor is really some measure of the Global Behaviour Pattern scoring that was once used on the SI. From this perspective, it could be assumed that a high score in the SI components would be linked with a TABP tendency. It is not surprising, therefore, that this factor score did not enter into the regression equations, since a number of research studies, as listed previously, have noted inconsistencies in the predictive ability of the TABP. Anger, the other factor score, was a combination of anger out minus anger controlled. An examination of scores indicated that <u>all</u> subjects controlled their anger more than they expressed it. Controlling or suppressing anger has been related to hypertension and mortality, as stated previously. It is also important to note that this anger factor was predictive of reactivity in this study. Since reactivity may be

implicated in the development of CVD, it would seem that this forewarning could be used to prevent the onset of CVD by behavioural interventions directed at suppressed anger.

At first glance, doing a factor analysis may seem of little assistance for future research because it could be interpreted to mean that in all research all these scales should be completed. Another viewpoint, however, is that there is an overlap in the constructs that are being captured by these scales. This overlap indicates that it is time to develop another scale that is a combination of these scales, and thus will make the number of variables that comprise the factors more manageable.

Implications

One implication of this study is that since the SI was not predictive of reactivity, subjects may not need to undergo the SI in situations where predicting reactivity is the issue. Predictors of reactivity may be obtained more easily from psychometric scales. Certainly if similar results were found in other research studies, then future research could use BMI scores and results of psychometric scales to target individuals for intervention programs both with respect to decreasing BMI and reactivity.

As aforesaid, the SI component score may represent a Global Behavioral Pattern that would account for this variable not entering into the regression equations. Another possibility could be that the SI and the SI scoring system were not able to tease out the relationship between anger-management styles and reactivity during the SI. Still another possibility may be related to the methodology

used in this study. A better method for detecting this relationship would be to measure anger-management styles during the reactive measure. For example, BP and HR reactive measures were taken from the 7-minute point during the 12-minute SI while the SI component score was assessed as an overall score during the interview. A better method to capture this relationship would be to score the SI components 30 seconds before and 30 seconds after the BP and HR reactive measures were taken. This measurement sequence would increase the likelihood that the physiological measures were influenced by the behavioural measures and, thus the chance of finding this subtle relationship should be increased. One problem, still, with this measurement sequence is whether the peak reactivity response is being captured. Each person takes a different length of time to reach the maximum or peak response to a stressor (Dimsdale & Ziegler, 1991). Instead of discrete measurement of cardiovascular response, a continuous measure of the response rate would be better. This would allow for the detection of the peak cardiovascular response of each person to the task. Using these results, researchers would be able to locate the peak response and enter that information into the regression equation. This would, therefore, increase the likelihood of variables predicting peak reactivity.

Limitations

Limitations of this study are important when considering the results of this study. University of Waterloo students in their first or second year of study were asked to participate in the study. The sample is not representative of all young people between the ages of 18 to 25. This population tends to be healthier (e.g.

few smoked) and more educated than the general population. This sample, thus, limits the generalizability of the obtained results.

How would these results differ if a random sample of 18- to 25-year-olds were tested? A lot of the students who called as potential subjects decided not to enter the study for one or a combination of reasons, such as too much time involved and fear of needles. These were university students who were exposed to experimental protocols through both their university studies and as participants. How would people respond to this study without having any background knowledge on what to expect? The sample of participants taken from the population may not be representative because relatively few people would agree to participate in such a complex study that involved so much time, on different dates, with venipuncture and other physiological measures during most of the experiment. The people who would participate in the study would probably have a higher education level, a higher income, and be younger than the average person in the general public. The results from this biased sample would differ from an unbiased sample with respect to the following: lower reactivity levels and lower anger-management style values. In the interests of health promotion and disease prevention, it would be better to devise a less complicated protocol that would increase the probability of obtaining an unbiased sample from the general population.

Another limitation of this study is the lack of ability to determine which factor(s) are influencing cardiovascular reactivity. In this study, certain hypotheses were proposed and analyses conducted that limited the factors that effected

reactivity (e.g., anger-management styles, BMI). However, it is not possible to partition the amount of reactivity caused by each factor. For that matter, it is not possible to conclude that only the selected factors were involved in cardiovascular reactivity.

Subjects were asked to refrain from smoking, drinking, eating, and consuming caffeine, for different lengths of time, before the session. The implications of any withdrawal effects are not known. For example, it is not known what the effects of nicotine withdrawal on catecholamines are (Ward, et al., 1983). If subjects were adversely affected by withdrawal symptoms then their reactivity would be higher because they would be more irritable; however, this theory cannot be tested because methods were not employed to measure withdrawal symptoms.

Future Research Directions

Some of the independent measures were predictive of university students' cardiovascular reactivity during the SI. Further research, however, needs to be done in certain areas. It would be extremely beneficial to determine if, in all cases, both the SI and psychometric scales need to be used in research. Depending on the dependent measure, it may not be necessary to use both measures. Certainly, it would be less costly and time consuming if one psychometric scale could be developed that predicts cardiovascular reactivity and incidence of cardiovascular diseases.

The results from this study revealed that BMI was predictive of reactivity. More research needs to be done on this measure, in the area of reactivity, to

determine how reliable BMI is as a predictor of cardiovascular response.

Gender was also predictive of reactivity. The majority of studies that have been done in the area of cardiovascular diseases have examined male subjects. There need to be more researchers who explore the predictive nature of different variables on cardiovascular reactivity in both males and females.

In conclusion, more research needs to address the relationship between behaviour and cardiovascular reactivity during the SI. Research needs to address the following questions: (1) What are the predictors of reactivity?; (2) How closely do these predictors resemble predictors of cardiovascular diseases?; (3) What are the mechanisms (i.e., physiological and behavioural) that link reactivity to development of cardiovascular disease?; (4) Which anger-management style variables are better predictors of reactivity?; and (5) Are catecholamine measures of reactivity reliable and valid?

APPENDICES

Appendix A: Appendix of Terms

Potential for Hostility:

> Potential for hostility can be defined as "a stable predisposition to respond to a broad range of frustrating circumstances with varying degrees of anger, irritation, disgust, contempt, resentment, and the like, which may or may not be associated with overt behavior directed against the source of the frustration" (Dembroski, MacDougall, Williams, Haney, 1985, p. 230).

Anger-In:

> The anger-in component, on the other hand, is seen as an emotional-control state in which the subject does not want to or is not able to confront the aggravating situation (Williams & Barefoot, 1988).

Terms from Cook-Medley Hostility Scale (Barefoot et al., 1989)

Hostile Attributions:

> People who display hostile attributions feel that others' behaviour is intentionally harmful to themselves.

Cynicism:

> People who are cynical are negative toward others. They feel that others are unworthy, deceitful, and selfish.

Hostile Affect:

> Negative emotions toward social relationships is indicative of people with hostile affect. They feel anger, impatience and loathing. These people, however, may not overtly react on these feelings.

Aggressive Responding:

> People who are aggressive responders will use anger and aggression to deal with problems.

Social Avoidance:

> Signs of social avoidance are indirectly negative behaviours and avoidance of others.

Appendix B: Antioxidant

Antioxidant

0.6 gm Reduced Glutathione
0.9 gm EGTA
Add 10 ml distilled water
Adjust pH to 7.0
Store at 4 C

Appendix C: Plasma Extraction

Plasma Extraction

1. To a 1.5 mL microcentrifuge tube add 20 mg acid washed alumina
2. Add 400 uL 2.0 M Trish + 2% EDTA, pH 8.7 (Fresh Weekly)
3. Add 50 uL DHBA (500 pg)
4 Add 1.0 ml Plasma
5. Mix by inversion for 10 minutes (avoid foaming)
6. Wash with water 3 times (cap and invert all 3 times)
7. Add 100 uL 0.1 M PCA, Vortex, then centrifuge
8. Add 100 uL to HPLC tube
9. Inject 50 uL of the supernant

2.0 M, 2% EDTA, pH 8.7

1. Trish base 18.60 gm
2. Trish HCL 7.32 gm
3. EDTA.Na 2.00 gm
4. Bring to 100 ml with distilled water
5. pH to 8.7 One day prior to use
6. Check pH daily

Concentrated Internal Standard

1. 10 mg DHBA
2. Make to 10 mL with 0.1 M PCA

DHBA (500 pg): Working Internal Standard

1. To 1 mL 0.1 M PCA add 4 uL Concentrated Internal Standard, mix well
2. To 1 mL 0.1 M PCA add 5 uL of the above mixture, mix well This is your DHBA (500 pg)

REFERENCES

Alderman, M.H., Ooi, W.L., Madhavan, S., & Cohen, H. (1990). Blood pressure reactivity predicts myocardial infarction among treated hypertensive patients. Journal of Clinical Epidemiology, 43(9), 859-866.

Allen, M.T., Boquet, A.J., Shelley, K.S. (1991). Cluster analyses of cardiovascular responsivity to three laboratory stressors. Psychosomatic Medicine, 53, 272-288.

American College of Sports Medicine (1980). Guidelines for graded exercise testing and exercise prescription (second edition, pp. 16-19, 42-45). USA: Lea & Febiger.

Axelrod, J., & Reisine, T.D. (1984). Stress hormones: Their interaction and regulation. Science, 224(4), 452-459.

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

Barefoot, J.C., Peterson, B.L., Dahlstrom, G., & Williams, R.B. (1989). The Cook-Medley Hostility scale: Item content and ability to predict survival. Psychosomatic Medicine, 51, 46-57.

Bhagat, B.D. (1974). Role of Catecholamines in Cardiovascular Diseases: I. Hypertension. USA: Charles C. Thomas.

Booth-Kewley, S., & Friedman, H.S. (1987). Psychological predictors of heart disease: a quantitative review. Psychological Bulletin, 101(3), 343-362.

Borkovec, T.D., Stone, N.M., O'Brien, G.T., & Kaloupek, D.G. (1974). Evaluation

of a clinically relevant target behavior for analog outcome research. Behavior Therapy, 5, 503-513.

Carmichael, S.W., & Winkler, H. (1985). The adrenal chromaffin cell. Scientific America, 253, 40-49.

Chesney, M.A., & Black, G.W. (1986). Hypertension: Biobehavioral influences and their implications for treatment. In T.H. Schmidt, T.M. Dembroski, & G. Blumchen (Eds.), Biological and Psychological Factors in Cardiovascular Disease (pp. 568-583). USA: Springer.

Chesney, M.A., Eaglestone, J.R., & Rosenman, R. (1980). The Type A structured interview: A behavioural assessment in the rough. Journal of Behavioural Assessment, 2, 255-272.

Chesney, M.A., Hecker, M.H.L., & Black, G.W. (1988). Coronary-prone components of Type A behaviour in the WCGS: A new methodology. In B.K. Houston & C.R. Snyder (Eds.), Type A Behavior Pattern: Research, Theory, and Intervention (pp. 168-187). USA: John Wiley and Sons Inc.

Cinciripini, P.M. (1986a). Cognitive stress and cardiovascular reactivity. I. American Heart Journal, 112(5), 1044-1050.

Cinciripini, P.M. (1986b). Cognitive stress and cardiovascular reactivity. II. Relationship to atherosclerosis, arrhythmias, and cognitive control. American Heart Journal, 112(5), 1051-1065.

Clarkson, T.B., Manuck, S.B., & Kaplan, J.R. (1986). Potential role of cardiovascular reactivity in atherogenesis. In K.A. Matthews, S.M. Weiss, T. Detre (Eds.), Handbook of Stress, Reactivity, and Cardiovascular Disease

(pp. 35-47). New York: John Wiley & Sons.

Cohn, J.N. (1989). Sympathetic nervous system activity and the heart. American Journal of Hypertension, 2, 353S-356S.

Cook, W.W., & Medley, D.M. (1954). Proposed hostility and pharisaic-virtue scales for the MMPI. The Journal of Applied Psychology, 38(6), 414-418.

Corse, J., Klag, M.J., Mean, L.A., Liange, K., & Whelton, P.K. (1992). Vascular reactivity in young adults and cardiovascular disease: A prospective study. Hypertension, 19 [suppl II], II-218-II-223.

Cottington, E.M., Matthews, K.A., Talbott, E., & Kuller, L.H. (1986). Occupational stress, suppressed anger, and hypertension. Psychosomatic Medicine, 48(3/4), 249-260.

Curb, J.D., & Marcus, E.B. (1991). Body fat, coronary heart disease, and stroke in Japanese men. American Journal of Clinical Nutrition, 53, 1612S-1615S.

Davies, M.J., & Woolf, N. (1993). Atherosclerosis: What is it and why does it occur? British Heart Journal, 69(Supplement), S3-S11.

Dembroski, T.M. (1984). Stress and substance interaction effects on risk factors and reactivity. Behavioral Medicine Update, 6(3), 16-20.

Dembroski, T.M., & MacDougall, J.M. (1983). Behavioral and psychophysiological perspectives on coronary-prone behavior. In T.M. Dembroski, T.H. Schmidt & G. Blumchen (Eds.), Biobehavioral Bases of Coronary Heart Disease (pp. 106-129). Switzerland: Karger.

Dembroski, T.M., MacDougall, J.M., Costa, P.T., & Granditis, G.A. (1989).

Components of hostility as predictors of sudden death and myocardial infarction in the Multiple Risk Factor Intervention Trial. Psychosomatic Medicine, 51, 514-522.

Dembroski, T.M., MacDougall, J.M., Williams, R.B., Haney, L., & Blumenthal, W.A. (1985). Components of Type A, hostility, and anger-in: Relationship to angiographic findings. Psychosomatic Medicine, 47(3), 219-233.

Devereuex, R.B., Pickering, T.G., Harshfield, G.A., Kleinert, H.D., Denby, L., Clark, L., Pregibon, D., Jason, M., Kleiner, B., Borer, J.S., & Laragh, J.H. (1983). Left ventricular hypertrophy in patients with hypertension: Importance of blood pressure response to regularly occurring stress. Circulation, 68, 470.

Diamond, E.L. (1982). The role of anger and hostility in essential hypertension and coronary heart disease. Psychological Bulletin, 92(2), 410-433.

Dimsdale, J.E., & Moss, J. (1980). Short-term catecholamine response to psychological stress. Psychosomatic Medicine, 42, 493-497.

Dimsdale, J.E., & Ziegler, M.G. (1991). What do plasma and urinary measures of catecholamines tell us about human response to stressors? Circulation, 83(4), II36-II42.

Egan, B.M., Bassett, D.R., & Block, W.D. (1991). Comparative effects of overweight on cardiovascular risk in younger versus older men. The American Journal of Epidemiology, 67(4), 248-252.

Engebretson, T.O., & Matthews, K.A. (1992). Dimensions of hostility in men, women, and boys: Relationships to personality and cardiovascular responses to stress. Psychosomatic Medicine, 54, 311-323.

Ewart, C.K. (1991). Familial transmission of essential hypertension: Genes, environments, and chronic anger. Annals of Behavioral Medicine, 13(1), 40-47.

Falkner, B., Kushner, H., Onesti, G., & Angelakos, E.T. (1981). Cardiovascular characteristics in adolescents who develop essential hypertension. Hypertension, 3, 521-527.

Falkner, B., & Ragonesi, S. (1986). Psychosocial stress and reactivity as risk factors of cardiovascular disease. Journal of the American Academy of Child Psychiatry, 25(6), 779-784.

Floras, J., Jones, J.V., Hassan, O., Osikowska, B.A., Sever, P.S., & Sleight, P. (1986). Failure of plasma norepinephrine to consistently reflect sympathetic activity in humans. Hypertension, 8, 641-649.

Frankenhaeuser, M. (1991). Behavioral medicine: An international perspective. Annals of Behavioral Medicine, 13(4), 197-204.

Friedman, H.S. (1989). The role of emotional expression in coronary heart disease. In A.W. Siegman & T.M. Dembroski (Eds.), In Search of Coronary Prone Behavior: Beyond Type A (pp. 149-168). USA: Lawrence Erlbaum Associates Inc..

Friedman, M., Byers, S.O., Diamant, J., & Rosenman, R.H. (1975). Plasma catecholamine response of coronary-prone subjects (Type A) to a specific challenge. Metabolism, 24(2), 205-210.

Friedman, M., & Rosenman, R.H. (1959). Association of specific overt behaviour pattern with blood and cardiovascular findings. Journal of the American

Medical Association, 169(12), 1286-1296.

Friedman, M., & Rosenman, R.H. (1971). Type A behavior pattern: Its association with coronary heart disease. Annals of Clinical Research, 3, 300-312.

Ganguly, P.K. (1989). Catecholamines and cardiovascular disorders: Pathophysiologic considerations. American Heart Journal, 118 868-872.

Gellman, M.D. (1984). Cardiovascular and Catecholamines Responses to Behavioral Challenges. Dissertation Abstracts International, 45(8), 2728-B. (University of Miami, Coral Gables, Florida, USA.)

Glass, D.G., & Contrada, R.J. (1984). Type A behavior and catecholamines: A critical review. In M.G. Ziegler & C.R. Lake (Eds.), Norepinephrine (346-367). Baltimore, MD: Williams & Wilkins.

Glass, D.C., Krakoff, L.R., Contrada, R., Hilton, W.F., Kehoe, K., Mannucci, E.G., Collins, C., Snow, B., & Elting, E. (1980). Effect of harassment and competition upon cardiovascular and plasma catecholamine responses in type A and type B individuals. Psychophysiology, 17(5), 453-463.

Goldstein, D.S. (1981). Plasma norepinephrine as an indicator of sympathetic neural activity in clinical cardiology. American Journal of Cardiology, 48, 1147-1154.

Goldstein, D.S. (1984). Plasma catecholamines in clinical studies of cardiovascular diseases. Acta Physiol Scand, 527(Suppl), 39-41.

Goldstein, D.S., & McDonald, R.H. (1986). Biochemical indices of cardiovascular reactivity. In Matthews, K.A., Weiss, S.M., Detre, T. (Eds.). Handbook of Stress, Reactivity, and Cardiovascular Disease (pp. 187-203). New York:

John Wiley & Sons.

Goldstein, R.E., & Rafjer, S.I. (1984). Effects of catecholamines in clinical heart disease. In M.G. Ziegler & C.R. Lake (Eds.), Norepinephrine (327-345). Baltimore, MD: Williams & Wilkins.

Harbin, T.J. (1989). The relationship between the Type A behaviour pattern and physiological responsivity: A quantitative review. Psychophysiology, 26(1), 110-119.

Hatfield, F.C., & Krotee, M.L. (1984). Personalized weight training for fitness and athletics (second edition, pp. 140-141). USA: Kendall/Hunt Publishing Company.

Hearn, M.D., Murray, D.M., & Luepker, R.V. (1989). Hostility, coronary heart disease, and total mortality: A 33-year follow-up study of university students. Journal of Behavioral Medicine, 12(2), 105-121.

Hecker, M.H.L, Chesney, M.A., Black, G.W., & Frautschi, N. (1988). Coronary - prone behaviours in the Western Collaborative Group Study. Psychosomatic Medicine, 50, 153-164.

Helmer, D.C., Ragland, D.R., & Syme, S.L. (1991). Hostility and coronary artery disease. American Journal of Epidemiology, 133(2), 112-122.

Houston, B.K. (1988). Cardiovascular and neuroendocrine reactivity, global type A, and components of type A behavior. In B.K. Houston & C.R. Snyder (Eds.), Type A Behavior Pattern: Research, Theory, and Intervention (pp. 212-253). USA: John Wiley and Sons.

Julius, S., Schneider, R., & Egan, B. (1986). Suppressed anger in hypertension:

Facts and problems. In M.A. Chesney & R.H. Rosenman (Eds). Anger and Hostility in Cardiovascular and Behavioral Disorders (pp. 127-138). New York: Hemisphere Publishing Company.

Kaplan, J.R., Adams, M.R., Clarkson, T.B., & Koritnik, D.R. (1984). Psychosocial influences on female "protection" among cynomolgus macaques. Atherosclerosis, 53, 283-295.

Kanner, A.D., Coyne, J.C., Schaefer, C., & Lazarus, R.S. (1981). Comparison of two modes of stress measurement: daily hassles and uplifts versus major life events. Journal of Behavioral Medicine, 4(1), 1-39.

Keys, A., Taylor, H.L., Blackburn, H., Brozek, J., Anderson, J.T., & Simonson, E. (1971). Mortality and coronary heart disease among men studied for 23 years. Archives of Internal Medicine, 128(2), 201-214.

Krantz, D.S., Contrada, R.J., Hill, D.R., & Friedler, E. (1988). Environmental stress and biobehavioral antecedents of coronary heart disease. Journal of Consulting and Clinical Psychology, 56(3), 333-341.

Krantz, D.S., Helmers, K.F., Bairey, N., Nebel, L.E., Hedges, S.M., & Rozanski, A. (1991). Cardiovascular reactivity and mental stress-induced myocardial ischemia in patients with coronary artery disease. Psychosomatic Medicine, 53, 1-12.

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

Krantz, D.S., Sanmarco, M.I., Selvester & Matthews (1979). Psychological

correlates of progression of atherosclerosis in men. Psychosomatic Medicine, 41(6), 467-475.

Lake, C.R., Chernow, B., Feuerstein, G., Goldstein, D.S., & Ziegler, M.G. (1984). The sympathetic nervous system in man: Its evaluation and the measurement of plasma NE. In M.G. Ziegler & C.R. Lake (Eds.), Norepinephrine (pp. 1-26). Baltimore, MD: Williams & Wilkins.

Leon, G.R., Finn, S.E., Murray, D., & Bailey, J.M. (1988). Inability to predict cardiovascular disease from hostility scores or MMPI items related to type A behavior. Journal of Consulting and Clinical Psychology, 56(4), 597-600.

Levenson, R.W. (1986). Alcohol, reactivity, and the heart:Implications for coronary health and disease. In Matthews, K.A., Weiss, S.M., Detre, T., et al. (Eds.). Handbook of Stress, Reactivity, and Cardiovascular Disease, (pp. 345-363). New York: John Wiley & Sons.

Light, K.C., Dolan, C.A., Davis, M.R., & Sherwood, A. (1992). Cardiovascular responses to an active coping challenge as predictors of blood pressure patterns 10 to 15 years later. Psychosomatic Medicine, 54, 217-230.

Lown, B., DeSilva, R.A. (1978). Roles of psychologic stress and ANS changes in provocation of ventricular premature complexes. American Journal of Cardiology, 41, 979.

MacDougall, J.M., Dembroski, T.M., Dimsdale, J.E., & Hackett, T.P. (1985). Components of type A, hostility, and anger-in: further relationships to angiographic findings. Health Psychology, 4(2), 137-152.

MacLeod, C.M. (1991). Half a century of research on the Stroop effect: An

integrative review. Psychological Bulletin, 109(2), 163-203.

Manuck, S.B., Kaplan, J.R., & Clarkson, T.B. (1985). Stress-induced heart rate reactivity and atherosclerosis in female macaques. Psychosomatic Medicine, 47(1), 90.

Manuck, S.B., Muldoon, M.F., Kaplan, J.R., Adams, M.R., & Polefrone, J.M. (1989). Coronary artery atherosclerosis and cardiac response to stress in Cynomolgus monkeys. In A.W. Siegman & T.M. Dembroski (Eds.), In Search of Coronary-Prone Behavior: Beyond Type A (207-227). Hillsdale, NJ: Lawrence Erlbaum Associates Incorporated.

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

Matthews, K.A. (1982). Psychological perspectives on the type A behavior pattern. Psychological Bulletin, 91(2), 293-323.

Matthews, K.A. (1988). Coronary heart disease and Type A behaviors: Update on and alternative to the Booth-Kewley and Friedman (1987) quantitative review. Psychological Bulletin, 104(3), 373-380.

Matthews, K.A., Glass, D.C., Rosenman, R.H., & Bortner, R.W. (1977) Competitive drive, pattern A, and coronary heart disease: A further analysis of some data from the Western Collaborative Group Study. Journal of Chronic Diseases, 30, 489-498.

McArdle, W.D., Katch, F.I., & Katch, V.L. (1981). Exercise physiology: Energy, nutrition, and human performance (pp.486-493). USA: Lea & Febiger.

McCranie, E.W., Watkins, L.O., Brandsma, J.M., & Sisson, B.D. (1986). Hostility, coronary heart disease (CHD) incidence, and total mortality: Lack of association in a 25-year follow-up study of 478 physicians. Journal of Behavioural Medicine, 9(2), 119-125.

McKinney, M.E., Hofschire, P.J., Buell, J.C., & Eliot, R.S. (1984). Hemodynamic and biochemical responses to stress: The necessary link between type A behavior and cardiovascular disease. Behavioral Medicine Update, 6(4), 16-22.

Mehta, H. (1983). Platelets and prostaglandins in coronary artery disease. JAMA, 249, 2818-2823.

Miller, T.Q., Turner, C.W., Tindale, R.S., Posavac, E.J., & Dugoni, B.L. (1991). Reasons for the trend toward null findings in research on Type A behavior. Psychological Bulletin, 110(3), 469-485.

Perloff, D., Sokolow, M., Cowan, R. (1983). The prognostic value of ambulatory blood pressures. JAMA, 249, 2792-2798.

Pi-Sunyer, F.X. (1991). Health implications of obesity. American Journal of Clinical Nutrition, 53, 1595S-1603S.

Prkachin, K., & Mills, D. (1988). Longitudinal Study of Stress Reactivity. Heart and Stroke Foundation, Grant Proposal, University of Waterloo, Department of Health Studies.

Review Panel on Coronary-Prone Behavior and Coronary Heart Disease (1981). Coronary-prone behavior and coronary heart disease: A critical review. Circulation, 63(6), 1199-1215.

Rosenman, R.H. Brand, R.J., Jenkins, D., Friedman, M., Straus, R., & Wurm, M. (1975). Coronary heart disease in the Western Collaborative Group Study: Final follow-up experience 8.5 years. Journal of the American Medical Association, 233(8), 872-877.

Rosenman, R.H., Brand, R.J., Sholtz, R.I., & Friedman, M. (1976). Multivariate prediction of coronary heart disease during 8.5 year follow-up in the Western Collaborative Group Study. American Journal of Cardiology, 37, 903-910.

Rosenman, R.H., Friedman, M., Straus, R., Wurm, M., Kositcheck, R., Hahn, W., & Werthessen, N.T. (1964). A predictive study of coronary heart disease: The Western Collaborative Group Study. Journal of the American Medical Association, 189, 15-22.

Rosenman, R.H., Swan, G.E., & Carmelli, D. (1988). Definition, assessment and evolution of the type A behavior pattern. In B.K. Houston & C.R. Snyder (Eds.), Type A Behavior Pattern: Research, Theory, and Intervention (pp. 8-31). USA: John Wiley and Sons.

Sallis, J.F., Patterson, T.L., McKenzie, T.L., Buono, M.J., Atkins, C.J., & Nader, P.R. (1989). Stability of systolic blood pressure reactivity to exercise in young children. Developmental and Behavioral Paediatric, 10(1), 38-43.

Scherwitz, L., Perkins, L., Chesney, M., & Hughes, G. (1991). Cook-Medley Hostility Scale and Subsets: Relationship to demographic and psychosocial characteristics in young adults in the CARDIA study. Psychosomatic Medicine, 53, 36-49.

Shekelle, R.B., Gale, M., Ostfeld, A.M., & Paul, O. (1983). Hostility, risk of coronary heart disease, and mortality. Psychosomatic Medicine, 45(2), 109-114.

Shekelle, R.B., Hulley, S.B., Neaton, J.D., Billings, J.H., Borhani, N.O., Gerace, T.A. (1985). The MRFIT behavior pattern study. American Journal of Epidemiology, 122(4), 559-570.

Smith, T.W., & Frohm, K.D. (1985). What's so unhealthy about hostility? Construct validity and psychosocial correlates of the Cook and Medley HO Scale. Health Psychology, 4(6), 503-520.

Spielberger, C.D., Jacobs, G., Russell, S., & Crane (1983). Assessment of anger: the state-trait anger scale. In J.N. Butcher & C.D. Spielberger (Eds.), Advances in Personality Assessment (pp. 159-187; Vol. 2). Hillsdale, NJ:LEA.

Spielberger, C.D., Johnson, E.H., Russell, S.F., Crane, R.J., Jacobs, G.A., & Worden, T.J. (1986). The experience and expression of anger: Construction and validation of an anger expression scale. In M.A. Chesney & R.H. Rosenman (Eds). Anger and Hostility in Cardiovascular and Behavioral Disorders (pp. 5-30). New York: Hemisphere Publishing Company.

Turner, L.W., Sizer, F.S., Whitney, E.N., & Wilks, B.B. (1992). Life choices: health concepts and strategies. St. Paul, MN: West Publishing Company.

Ward, M.M., Mefford, I.N, Parker, S.D., Chesney, M.A., Taylor, B., Keegan, D.L., & Barchas, J.D. (1983). Epinephrine and norepinephrine responses in

continuously collected human plasma to a series of stressors. Psychosomatic Medicine, 45, 471-486.

Weidner, G., Friend, R., Ficarrotto, T.J., & Mendell, N.R. (1989). Hostility and cardiovascular reactivity to stress in women and men. Psychosomatic Medicine, 51, 36-45.

Weicker, H., Feraudi, M., Hägele, H., & Pluto, R. (1984). Electrochemical detection of catecholamines in urine and plasma after separation with HPLC. Clinica Chimica Acta, 141, 17-25.

Williams, R.B. (1984). Type A behavior and coronary heart disease: Something old, something new. Behavioral Medicine Update, 6(3), 29-35.

Williams, R.B. (1987). Psychological factors in coronary artery disease: Epidemiologic evidence. Circulation, 76(suppl I), I-117 - I-123.

Williams, R.B. (1989). Biological mechanisms mediating the relationship between behavior and coronary heart disease. In A.W. Siegman & T.M. Dembroski (Eds.), In Search of Coronary Prone Behaviour (pp. 195-205). USA: Lawrence Erlbaum Ass. Inc.

Williams, R.B., & Barefoot, J.C. (1988). Coronary-prone behaviour: The emerging role of the hostility complex. In B.K. Houston & C.R. Snyder (Eds.), Type A Behavior Pattern: Research, Theory, and Intervention (pp. 189-211). USA: John Wiley and Sons Inc.

Williams, R.B., Barefoot, J.C.& Shekelle, R.B. (1986). The health consequences of hostility. In M.A. Chesney & R.H. Rosenman (Eds). Anger and Hostility in Cardiovascular and Behavioral Disorders (pp. 173-185). New York:

Hemisphere Publishing Company.

Williams, R.B., Haney, T.L., Lee, K.L., Kong, Y.H., Blumenthal, J.A., & Whalen, R.E. (1980). Type A behavior, hostility, and coronary atherosclerosis. Psychosomatic Medicine, 42(6), 539-549.

Williams, R.B., Suarez, E.C., Kuhn, C.M., Zimmerman, E.A., & Schanberg, S.M. (1991). Biobehavioral basis of coronary-prone behavior in middle-aged men. Part I: Evidence for chronic SNS activation in Type As. Psychosomatic Medicine, 53, 517-527.

Wood, D.L., Sheps, S.G., Elveback, L.R., & Schirger, A. (1984). Cold pressor test as a predictor of hypertension. Hypertension, 6, 301-306.

Other References

Buss, A.H., & Durkee, A. (1957). An inventory for assessing different kinds of hostility. Journal of Consulting Psychology, 21, 343-349.

Falkner, B., Onesti, G., & Hamstra, B. (1981). Stress response characteristics of adolescents with high genetic risk for essential hypertension. A five year follow-up. Clinical and Experimental Hypertension, 3, 583.

Jenkins, C.D., Hurst, M.W., Rose, R., Anderson, M.C., & Kreger, B.E. (1984). Biomedical and psychosocial predictors of hypertension in air traffic controllers. In C.D. Spielberger, I.G. Saroson, & P.B. Refars (Eds). Stress and anxiety IX. New York: McGraw-Hill Book Co., Inc.

Julius, M., Harburg, E., Cottington, E.M., & Johnson, E.H. (1986). Anger-coping types, blood pressure, and all-cause mortality: a follow-up in Tecumseh,

Michigan (1971-1983). American Journal of Epidemiology, 124, 220-233.

Kaplan, J.R., Manuck, S.B., Clarkson, T.B., Lusso, F.M., & Taub D.B. (1982). Social status, environment, atherosclerosis in cynomolgus monkeys. Arteriosclerosis, 2, 359-368.

Manuck, S.B., Kaplan, J.R., & Clarkson, T.B. (1983). Social instability and coronary artery atherosclerosis in cynomolgus monkeys. Neuroscience & Behavioral Reviews, 7, 485-491.

Matthews, K.A., Krantz, D.S., Dembroski, T.M., & MacDougall, J.M. (1982). The unique and common variance in the structured interview and Jenkins activity survey measures of the Type A behaviour pattern. Journal of Personality and Social Psychology, 42, 303-313.

Rose, R.M., Jenkins, C.D., Hurst, M.W. (1978). Air traffic controller health change study (FAA-AM No. 78-79). Washington, D.C.: NTIS.

Rosenman, R.H. (1978). The interview method of assessment of the coronary prone behaviour pattern. In T.M. Dembroski, S.M. Weiss, J.L. Shields, S.G. Haynes, & M. Feinleib (Eds.), Coronary-Prone Behaviour (pp.). New York: Springer-Verlag.