ESTABLISHED AND EMERGING PERSONALITY CONSTRUCTS IN BEHAVIORAL CARDIOLOGY: ASSOCIATIONS WITH RESTING BLOOD PRESSURE AND CARDIOVASCULAR RESPONSES TO ACUTE STRESS

by

MARTINE E. HABRA

B.A., McGill University, 1996

A THESIS SUBMITTED IN PARTIAL FULFILMENT OF THE REQUIREMENTS FOR THE DEGREE OF MASTER OF ARTS

in

THE FACULTY OF GRADUATE STUDIES

Department of Psychology

We accept this thesis as conforming to the required standard

THE UNIVERSITY OF BRITISH COLUMBIA

September 2000

In presenting this thesis in partial fulfilment of the requirements for an advanced degree at the University of British Columbia, I agree that the Library shall make it freely available for reference and study. I further agree that permission for extensive copying of this thesis for scholarly purposes may be granted by the head of my department or by his or her representatives. It is understood that copying or publication of this thesis for financial gain shall not be allowed without my written permission.

Department of Psychology

The University of British Columbia
Vancouver, Canada

Date Sept 25/2000

ABSTRACT

Studies have revealed associations between established psychosocial factors and cardiovascular health, but the magnitude of effects remains small, and findings are fraught with inconsistencies. Recently, several putative psychological risk factors for cardiovascular disease, namely social support seeking, sociotropy, and Type-D personality, defensiveness and sex roles, have been proposed to further elucidate the relationship between personality and disease. The purpose of the present study was to examine how such emerging constructs relate to laboratory indices of cardiovascular health compared to more established psychosocial risk factors, i.e., hostility and social support.

173 undergraduates (87 female, 86 male) completed a stress protocol involving a mental arithmetic task (serial subtractions of seven) while receiving scripted harassing comments. Blood pressure and heart rate were measured both prior to and during the stressful task. The relationships between personality and both resting and reactivity levels were examined. Reactivity was defined as the amount of change between resting blood pressure and heart rate levels and levels reached during the task. Contrary to prediction, hostility was associated with lower resting blood pressure, while social support was unrelated to cardiovascular responses. Emerging personality constructs demonstrated consistent associations with physiology. Masculinity was associated with lower resting heart rate [$\beta = -.26$, $p < .01$]. Support seeking was associated with less SBP reactivity to the task ($r = -.15$, $p = .05$). Sociotropy was related to dampened reactivity to stress [$\beta = -.21$, $p < .05$ for DBP; $\beta = -.22$, $p < .05$ for HR]. Males who reported greater social inhibition, a dimension of the Type-D personality, showed greater reactivity to the lab stressor [$\beta = .41$, $p < .01$ for SBP; $\beta = .31$, $p < .05$ for DBP]. Impression management was also associated with resting blood pressure, but gender differences in the

pattern of associations were noted. Thus, the present results confirm the need to move away from global personality dimensions in order to explain better how personality can exert beneficial or deleterious effects on health. However, the direction of results was not always consistent with our predictions, indicating that these specific personality constructs would benefit from further refinement.

TABLE OF CONTENTS

LIST OF TABLES

LIST OF FIGURES

INTRODUCTION

Cardiovascular disease (CVD) is the top cause of mortality in North America, accounting for 36% of all deaths in Canada (Statistics Canada, 1997, as cited in Heart and Stroke Foundation of Canada, 1999). While biological and lifestyle risk factors such as smoking, obesity, dyslipidemia, hypertension, and diabetes have been identified (Heart & Stroke Foundation of Canada, 1999), they alone are not sufficient to predict the development of disease. Though behaviorally minded researchers have increasingly been turning to psychosocial factors such as personality and social support in order to explain some of the variance in CVD, their findings, to date, have been marked by inconsistencies and null findings. More recently however, researchers have moved from global personality dimensions to more specific personality trait and personality-by-situation interactions which might prove to be more potent in predicting the development of disease. The present paper will focus on several emerging putative psychosocial risk factors by discussing how they relate to the more established risk factors of hostility and perceived social support, as well as to indices of cardiovascular health in the laboratory.

Personality and Disease

Kop (1999) has suggested that psychological risk factors can be classified on the basis of their duration and temporal relationship to coronary events, ranging from acute to chronic (see Figure 1). Acute risk factors such as anger and mental activity can have immediate and potentially lethal effects on the cardiovascular system. Indeed, Krantz and colleagues have demonstrated in both laboratory and field studies that silent or asymptomatic ischemic events can be induced by emotional or mental stress (see Krantz, Kop, Santiago, & Gottdiener, 1996, for a review). In contrast, episodic and chronic risk factors are thought to slowly contribute to the pathogenic disease process over time. While time-limited episodic factors are related to

homeostatic imbalance and upset, chronic factors, including enduring personality traits or psychosocial factors such as hostility and social isolation, are more directly related to CVD severity (Kop, 1999). As can be noted from Figure 1, both chronic risk factors and background factors can lead to disease by contributing to exaggerated sympathetic arousal to stress (Krantz & Manuck, 1984). However, what is not as evident from Kop (1999)'s model is that personality can also have an impact on background factors by either promoting unhealthy behaviors (e.g. smoking, decreased help-seeking) or by creating an unhealthy social environment marked by adverse relationships and chronic interpersonal stress (Smith & Pope, 1990; Cohen, 1988). In turn, such background factors can also have an impact on the cardiovascular system by contributing to chronic sympathetic arousal (Smith & Pope, 1990). Enduring personality traits, then, can have multiple paths to disease. This paper, however, will focus on the more direct relationship between personality and cardiovascular disease via its impact on hyperresponsivity to stress.

Cardiovascular Reactivity and Disease

Hyperreactivity is hypothesized to contribute to the disease process of both hypertension and coronary artery disease (CAD). In the case of hypertension, reactivity is thought to lead to increased peripheral resistance, which over time, contributes to elevated blood pressure (Manuck, Kraprowicz, & Muldoon, 1990). Hyperreactivity could lead to CAD by causing injury to the endothelial lining of the arteries, thereby promoting the accumulation of plaque, which, over time, can lead to acute events such as thrombosis or ischemia (Manuck, 1995). While the link between reactivity and disease is still under investigation, evidence from both animal and human studies suggests a potential role in the pathogenesis of CVD. For example, highly reactive cynomolgus monkeys show more extensive artherosclerosis when fed a cholesterol-rich

diet than less reactive counterparts (Manuck, Kaplan, Adams, & Clarkson, 1989; Manuck, Kaplan, & Clarkson, 1983). Evidence linking reactivity and CVD in humans is only starting to emerge, as longitudinal studies are being published. While the evidence to date is limited, it suggests that heightened reactivity to stress in the laboratory is associated with greater blood pressure increases over time (Markovitz et al., 1998; Newman, McGarvey, & Steele, 1999; Parker et al., 1987; Light, Dolan, Davis, & Sherwood, 1992) as well as with the development of hypertension (Menkes et al., 1989). Hyperreactivity has also been proposed as the mechanism underlying the relationship between personality and disease. Using the reactivity model, researchers have sought to demonstrate consistent associations between both psychosocial vulnerabilities and heightened reactivity to stress, as well as between psychosocial buffers and dampened reactivity. However, as will be discussed below, this search has not always proved fruitful.

Personality and Cardiovascular Reactivity: Psychosocial Vulnerabilities

Established Risk Factors

Type-A behavior. The first major foray into the study of personality and CVD involved the Type-A or coronary-prone behavior pattern. Proposed by Friedman and Rosenman (1959), the Type-A behavior pattern consisted of a cluster of traits which would put the individual at risk for the development of CVD. These traits included a sense of time urgency, aggressiveness, hostility, and competitiveness. While initial prospective studies did provide evidence for a link between Type-A and cardiac outcome (e.g. Rosenman et al., 1975), later findings failed to show an association (see Booth-Kewley & H. Friedman, 1987 for a quantitative analysis). Studies linking the coronary-prone behavior pattern to physiological reactivity in the laboratory were similarly inconsistent in their findings (Myrtek, 1995).

Hostility. Recognizing that the coronary-prone pattern was comprised of a constellation of traits, researchers began to focus on individual Type-A components in order to determine whether certain aspects of the pattern were more "toxic" than others (Linden, 1987; Siegman, 1994a). In the early 80's, researchers started to focus on hostility as such a "toxic" agent. Hostility was found to be not only related to incidence of CVD, but to all-cause mortality as well (Barefoot, Dahlstrom, & Williams, 1983; Shekelle, Gale, Ostfeld, & Paul, 1983). Further, individual studies and quantitative analysis indicated that hostility had better predictive validity than the Type-A behavior pattern (Booth-Kewley & H. Friedman, 1987; Dembroski, MacDougall, Costa, & Grandits, 1989; Williams et al., 1980). Associations with reactivity have also been noted, and have thus provided additional evidence as to the underlying pathophysiology of hostility (e.g. Steptoe, Melville, & Ross, 1984; Suarez, Harlan, Peoples, & Williams, 1993). However, hostility has not always been consistently related to markers of cardiovascular health. Several studies have failed to find an association between hostility and CVD (e.g. Hearn, Murray, & Luepker, 1988; McCranie, Watkins, Brandsma, & Sisson, 1986), and acute reactivity (e.g. Felsten, 1995; Smith & Houston, 1987). Furthermore, meta-analyses suggest heterogeneity in the pattern of associations. For example, Suls and Wan (1993) found evidence of a negative association between hostility and acute reactivity. Jorgensen, Johnson, Kolodziej, and Schreer (1996) found that age moderated the relationship between negative affect (i.e. hostility, anxiety, depression) and blood pressure, such that negative affectivity and blood pressure were positively related in older individuals, but negatively related in younger individuals. Thus, the hostility literature is marked by the same inconsistencies that plague the Type-A literature. Consequently, researchers have begun to realize that hostility may not be a unidimensional construct, and that certain sub-components of hostility might show stronger

associations with disease outcome (Siegman, 1994a). Further, new constructs have been proposed to further elucidate the relationship between disease and negative affect such as hostility.

Emerging Risk Factors

Type-D personality. The Type-D or "distressed" personality construct was developed by Denollet in his investigation of coping styles which might be involved in the development of cardiovascular disease. Empirically identified through cluster analysis, Type-D individuals score highly on the negative affectivity and social inhibition personality dimensions (Denollet & De Potter, 1992). Negative affectivity is defined as the "tendency to experience negative emotions", including anger, depressed affect, and anxiety (Denollet, 1998, p. 209). Social inhibition, on the other hand, is defined as "the avoidance of potential dangers involved in social interaction such as disapproval or non-reward by others" (Denollet, 1998, p. 209). Thus, the "distressed" personality subtype is characterized by the joint tendency to experience negative emotions and to inhibit these emotions by avoiding social contact with others.

Denollet has repeatedly shown that the distressed personality is associated with CVD outcome. For example, Type-D carries a 6-fold increase in the likelihood of death from cardiac events two to five years post-MI in men (Denollet, Sys, & Brutsaert, 1995), a four-fold increase in mortality 6-10 years following a cardiac event (Denollet et al., 1996), and poorer outcome in post-MI patients with a decreased left ventricular ejection fraction, a condition with a particularly poor prognosis (Denollet & Brutsaert, 1997). In all of the above cited studies, Type-D was shown to be significantly associated with worse disease outcome, even after controlling for traditional risk factors – symptoms of depression, anger, and anxiety did not add to the predictive power of the Type-D construct. Finally, in a sample of CHD patients, Type-D personality was

found to be predictive of the development of cancer 6 to 10 years following the initial cardiac event. Denollet (1998) suggests that this indicates that the "distressed personality" construct can be viewed as a "non-specific risk factor for poor health outcomes in general" (p. 994).

While the studies cited above suggest a potentially causal link between Type-D and CVD, the specific pathway to disease has yet to be established. Denollet (1998) suggests that personality might be linked to disease outcome either directly through psychophysiological mechanisms or indirectly through psychosocial factors. There has been no published study relating the Type-D construct to cardiovascular reactivity as such a mechanism. Because Type-D individuals are more prone to experience negative emotions and are less likely to express such emotions, we can hypothesize that such individuals would show heightened arousal in response to acute stress.

Defensiveness. Other researchers have also examined whether unexpressed negative affect might have deleterious consequences for health. Krantz, Helmers, and colleagues, noting inconsistencies in the literature, have suggested that hostility coupled with defensiveness - the tendency to minimize or deny undesirable emotions or aspects about oneself - might be more toxic than hostility alone. Defensiveness can be considered a response style which can affect the accurate reporting of both psychological and somatic symptoms, leading to underestimation of symptoms (Linden, Paulhus, & Dobson, 1986). This has led many researchers to try to free self-report measures from socially desirable responding by applying a correction for defensiveness, such as the K-correction for MMPI-2 scores (Graham, 1993). While a review of this literature is beyond the scope of this paper, there is an indication that "correcting" for response styles does not necessarily improve the accuracy of self-report, and researchers have started to view social desirability as an individual difference trait on its own right (McCrae & Costa, 1983; Rutledge,

Linden, & Davies, in press). Factor analysis of the major tools used to measure response styles has indicated that items can be reduced to two independent factors: self-deception and impression management (Paulhus, 1984). Self-deception is defined as the unconscious tendency to deny or minimize one's faults or negative emotions, while impression management can be seen as a more deliberate attempt to present favorably in front of others (Paulhus, 1984). Most studies examining the relationship between defensiveness and health have used the Marlowe-Crowne scale (Crowne & Marlowe, 1964), which does not distinguish between these two dimensions; both self-deception and impression management are lumped together to create an index of socially desirable responding. Using this inventory and a hostility measure, Krantz and colleagues have examined the relationship between defensive hostility and cardiovascular health. In a series of studies, Helmers, Krantz, Merz, et al. (1995) found that defensive hostile individuals had more severe clinical manifestations of coronary artery disease (CAD), as evidenced by greater exercise-induced ischemia, greater frequency and duration of ischemic events during ambulatory (Holter) monitoring, and greater mental stress-induced ischemia in the laboratory. Further, Jamner, Shapiro, Goldstein, and Hug (1991) found that hostility and defensiveness interacted with work setting such that defensive hostile male paramedics showed higher ambulatory heart rate levels while actively involved in their work. However, when defining defensive hostility empirically, one artificially creates subgroups of individuals (e.g. defensive/non-hostile or non-defensive/non-hostile) which are conceptually poorly defined, or even illogical (Linden, 2000). For example, Helmers and Krantz (1996) found that both defensive hostile *and* non-defensive/non-hostile individuals had marginally greater resting diastolic blood pressure (DBP) levels. Similarly, Mente and Helmers (1999) found that the same two subgroups exhibited significantly greater DBP reactivity to a cold pressor task.

Researchers do not have ready answers to explain why non-hostile/non-defensive individuals should display equivalent blood pressure levels and reactions as the subgroup they have identified as most at risk.

Evidence suggests that defensiveness is a risk factor in its own right, regardless of a person's hostility status (McCrae, Costa, Dahlstrom, Barefoot, Siegler, & Williams, 1989; Rutledge, Linden, & Davies, in press). For example, defensiveness, globally defined, has been associated with higher resting blood pressure (Mann & James, 1998) and greater reactivity, both in healthy normals and patients with known CVD (Grossman, Watkins, Ristuccia, & Wilhelm, 1997; Jamner et al., 1991; Shapiro, Goldstein, & Jamner, 1995). Jorgensen et al. (1996), in their quantitative review of personality and blood pressure, found that defensiveness was a more robust predictor of high blood pressure than negative affect and anger/hostility (effect size $\underline{d}$ = .39). However, most of these studies have examined defensiveness using global measures such as the Marlowe-Crowne scale, which, as noted above, obscures differences between self-deception and impression management. Research from our laboratory has indicated that self-deception might be the more potent health risk, particularly in men. Self-deception has been found to be related to higher ambulatory DBP levels (Linden, Chambers, Lenz, & Maurice, 1993), to predict the incidence of hypertension after a 3-year follow-up (Rutledge & Linden, 2000), and to be associated with smaller treatment gains in a sample of males undergoing pharmacological treatment for ischemia (Rutledge, Linden, Davies, 1999). However, in women with a history of familial hypertension, impression management was found to be associated with slower physiological recovery following laboratory stress, suggesting that in women, this response style might have better prognostic value (Habra & Linden, 2000). On the basis of the

available evidence, we can hypothesize that response styles would be associated with indices of cardiovascular functioning, but that this relationship might be moderated by gender.

Sociotropy. Sociotropy has recently been proposed as a potential moderator of physiological reactivity to stress, especially for women (Ewart, Jorgensen, & Kolodner, 1998). Sociotropic cognition, a concept borrowed from the depression literature, was initially proposed by Beck to describe individuals who might be particularly vulnerable to the development of depression following interpersonal stress or loss (Beck, 1997). Sociotropic individuals are described as particularly vested in interpersonal relationships, and are dependent on them for a sense of esteem and approval (Persons, Miranda & Perloff, 1991). Such individuals are argued to have "heightened needs for acceptance, understanding, support, and guidance" (Coyne & Whiffen, 1995, p. 358). Sociotropy shares similarities with defensiveness in that it involves a strong desire to please. This desire, however, seems to center around a need to maintain interpersonal relationships in order to achieve a positive sense of self, whereas defensiveness seems to involve a more general concern with appearing to others in a positive light.

Ewart and colleagues have recently examined whether sociotropic thinking influences how one reacts to stress. Using a diathesis-stress paradigm, they have shown that sociotropic young women do react more, but only under specific conditions. For example, Ewart et al. (1998) have shown that adolescent women high in sociotropy show greater blood pressure change than low sociotropes in response to recall of a emotionally distressing event, but not during a mirror image tracing task. What this study suggests is that sociotropy seems to act as a vulnerability to stress, but only under conditions which have a strong interpersonal component. However, at this point, the finding needs to be replicated, and it remains to be seen whether the relationship between sociotropy and reactivity holds for men as well.

Personality and Cardiovascular Reactivity: Psychosocial Buffers

Established Risk Factors

Perceived social support. In contrast to the above, some psychosocial factors have been studied for their potential beneficial, rather than deleterious, effects on cardiovascular health. In this vein, social support, or lack thereof, has emerged as one of the strongest psychosocial predictors of physical health. Numerous large-scale epidemiological studies have indicated that a supportive environment is associated with decreased mortality from all causes (House, Robbins, & Metzner, 1982; Pennix et al., 1997; Yasuda et al., 1997) as well as from CVD (Orth-Gomér, Rosengren, & Wilhelmsen, 1993; Orth-Gomér & Unden, 1990). The relationship to cardiovascular disease morbidity is more tenuous, with some studies linking social support to survival following myocardial infarction (Berkman, Leo-Summers, & Horwitz, 1992) and to severity of coronary artery disease (CAD; Orth-Gomér et al., 1998), and others suggesting null findings (see Pennix, Kriegsman, van Eijk, Boeke, & Deeg, 1996, for a review).

While the above studies suggest an association between support and disease, meta-analysis reveals that the strength of the relationship is weak, with $r = -.07$ (Schwarzer & Leppin, 1991). Also, such studies do not shed any light on *how* support exerts its beneficial effects on health (Cohen, 1988). A further difficulty in understanding the potential mechanisms underlying social support's protective effects is the multi-dimensionality of the construct and the lack of an agreed upon definition of support (Barrera, 1986; Cohen, 1988). Reflecting this taxonomic confusion is the number of measures of support which have been developed. This decreases comparability across studies, and makes it difficult to identify which aspects of support are most beneficial. Finally, epidemiological studies, especially prospective designs, measure support at single time point, usually well before the onset of the disease process, which (a) makes it

difficult to ascertain how support affects health, and (b) ignores the dynamic nature of the support process, with sources of support increasing or eroding over time (Pennix et al., 1996).

Schwarzer & Leppin (1991) have suggested a useful way of classifying different aspects of social support. *Structural* aspects include characteristics of one's social network and the number and type of social ties within the network. Epidemiological studies have usually focused on structural aspects of support, including marital status, number of relatives or friends one sees on a regular basis, and affiliation to social groups such as church. *Cognitive* aspects of social support characterize support as "the cognitive appraisal of being reliably connected to others" (Barerra, 1986, p. 416). Thus cognitive aspects of social support involve a subjective evaluation of one's social environment as to the perceived availability and adequacy of one's social network. In turn, such an evaluation can influence the primary and secondary appraisal process (Lazarus & Folkman, 1984) and reduce or augment the perceived stressfulness of an event (Barrera, 1986). It has been suggested that perceived support should be viewed as an individual difference trait, as it has been shown to exhibit temporal stability and consistent associations with personality traits such as neuroticism and extraversion (Sarason, Sarason, & Shearin, 1986). Furthermore, perceived support seems to be relatively independent of enacted or received support (Barrera, 1986; Schwarzer & Leppin, 1991). Thus, "cognitive" support seems to be unrelated to actual aspects of one's social environment; rather, it seems to reflect a dispositional tendency to view one's world as supportive or not. Finally, *behavioral* aspects of support include mobilizing and receiving support from one's social network, as well as offering support to significant others (Barerra, 1986; Schwarzer & Leppin, 1991). Both behavioral and cognitive aspects of support can be further classified depending on the function they serve (Cohen, 1988). Such functions include emotional, affective, instrumental, or material support. Cohen and Wills

(1984) have suggested that support might be most beneficial when the type of support needed matches the support received. For example, material support such as a loan might not be helpful when what one needs is a shoulder to cry on.

Most of the research examining the relationship between support and cardiovascular reactivity to stress have studied the effects of having a supportive person present during a stressful task (e.g. Kamark, Manuck, & Jennings, 1990; Kors, Linden, & Gerin, 1997). While there have been some inconsistencies in results, probably due to differences in methodology, this line of research has indicated that a supportive confederate, friend, or partner tends to dampen physiological responses to stress (see Uchino, Kielcot-Glaser, & Glaser, 1996 for a review), suggesting one possible pathway through which support can offer protective health effects. However, it is unlikely that in a real-life situation individuals will always have a supportive friend present during acute stress. For example, rarely will one have a friend present when under pressure at work. In light of the fact that perceived support does not always correlate with enacted support (Sarason et al., 1986), a focus on the relationship between the perception of a supportive environment and physiology, regardless of whether or not one actually receives support, seems warranted.

Surprisingly, not many studies have explored the relationship between these two variables. Structural aspects of support - i.e., network size, social anchorage or number of social ties - have been inconsistently linked to blood pressure levels, with some studies finding that high support was related to lower resting BP (e.g. Bland, Krogh, Winkelstein, & Trevisian, 1991; Dressler, Dos Santos, & Viteri, 1986; Hanson, Isacsson, Janzon, Lindell, & Råstam, 1988), while others finding an association between support and *higher* BP (e.g. Dressler, Mata, Chavez, Viteri, & Gallagher, 1986). Perceived support has also been linked with both lower resting BP

in African Americans (Strogatz & James, 1986) and women (Uchino, Cacioppo, Malarkey, Glaser, Keicolt-Glaser, 1995), as well as lower ambulatory BP levels (Linden et al., 1993). Furthermore, Spitzer, Llabre, Ironson, Gellman, and Schneiderman (1992) found that ambulatory levels were lower in the company of family and friends than strangers. While the above studies suggest a relationship between a supportive environment and healthier blood pressure levels, the relationship of perceived support to stress reactivity has not been as consistent. Some studies have found that high levels of perceived social support are indeed associated with decreased reactivity to acute stress in the laboratory (Broadwell & Light, 1999; Lidderdale & Walsh, 1998), while contrary to prediction, some have found it to be associated with heightened reactivity (Roy, Steptoe, & Kirschbaum, 1998; Uchino, Kiecolt-Glaser, & Cacioppo, 1992). Thus while large population-based research has found support to exert a protective effect on health, laboratory studies have not been able to consistently explain the pathway to disease.

Emerging Risk Factors

Social support seeking. Perhaps one of the reasons that the results of reactivity and social support studies have been inconsistent is that researchers have focused on one aspect of the social support process - namely perceived support. Perhaps other related aspects of the process might reveal themselves to be important from a health perspective. Social support seeking has been a largely ignored individual difference trait which might offer some potential health benefits. For instance, recognizing that one needs help and taking steps to ensure that the help is received could be potentially life-saving under certain circumstances. Furthermore, inconsistencies in the perceived social support literature might be explained by individual differences in the perceived need for social support. In other words, whether an individual benefits from the buffering effects of social support might depend on whether or not they feel a

need for it. Thus, there might be a match-mismatch effect whereby low support-seeking individuals with low levels of perceived support might be as healthy as high support-seeking individuals with high levels of perceived support (Kors & Linden, 1999).

There has been very little research on the health-related aspects of support seeking. Support seeking has been associated with lower resting blood pressure levels and decreased reactivity to stress in women (Fontana, Pontari, & Nash, 1998). In their study of support and health in a Mexican sample, Dressler, Mata, Chavez, Viteri, and Gallagher (1986) found that the greater the number of persons an individual reported feeling comfortable asking for help, the lower the individual's resting blood pressure levels. Finally, in a test of the match-mismatch hypothesis outlined above, Kors and Linden (1999) investigated the impact of social support receipt in the laboratory in both high and low support seekers. While there was no evidence for the hypothesis, support seeking as a trait seemed to be associated with healthier resting BP, as well as dampened reactivity in men. Thus, on the basis of the available evidence, one can hypothesize that support seeking would be associated with lower resting and reactivity levels in the laboratory.

Gender roles. Gender and sex-role orientation differences might also affect the social support process. Women report perceiving, seeking, and receiving more social support than men (Ashton & Fuehrer, 1993; Burda, Vaux, & Schill, 1984; Butler, Giordano, & Neren, 1985). However, it may be that some of the variance between gender and support may be accounted for by a feminine sex-role orientation, perhaps to a lesser degree in men than in women (Butler et al., 1985). For example, Burda et al. (1984) found that feminine and androgynous individuals, regardless of gender, reported more social support than masculine and undifferentiated individuals. Ashton and Fuehrer (1993) found that gender-typed males were least likely to report

seeking emotional support compared to sex-typed females and androgynous males and females.

Two studies have examined how sex roles might influence the use of coping strategies under stress. Brooks, Morgan, and Scherer (1990) found that nontraditional females engaged in a greater amount of coping strategies in response to a stressful event, reporting more social support seeking than nontraditional males and traditional males and females. On the other hand, Korabik and Van Kampen (1995) found that men and women differed very little in terms of coping strategies used in response to a work-related stressor. Individuals high in femininity were more likely to use avoidance coping and support seeking as a coping strategy. Finally, as noted above, masculinity seems to buffer individuals from psychological distress following stressful life events (Nezu, Nezu, & Peterson, 1986). Interestingly, Roos and L. Cohen (1987) found that in their sample of undergraduates, the stress-buffering effects of social support were especially potent for individuals high in masculinity.

While gender-roles might influence who benefits from social support, they may also have a more direct impact on health as well. Bem (1981) initially proposed that androgyny might be associated with better mental health because it confers to individuals greater flexibility and adaptability in terms of the range of available behaviors. However, research has indicated that it is the masculinity component of androgyny which seems to mediate its positive relationship with mental health (e.g. Silten Bassoff & Glass, 1982). Some authors have suggested that masculinity might be associated with better psychological adjustment because of the emphasis North American society places on masculine traits such as independence and achievement (Shifren, Bauserman, & Carter, 1993).

While the masculinity model seems to hold for psychological health, the relationship between sex roles and physical health suggests a potentially different picture. For example,

Shifren et al. (1993) found that androgynous individuals reported better health practices than masculine, feminine, and undifferentiated individuals, as indicated by more regular stress management and less smoking and drinking. Masculine individuals reported maintaining a more regular exercise schedule. The authors suggest that the greater behavioural flexibility associated with androgyny might protect individuals from engaging in at risk behaviour associated with masculinity (e.g. drinking, smoking, less help-seeking), while conferring the health advantages associated with masculinity, such as exercise (Shifren et al., 1993).

There is very little research examining the relationship between cardiovascular health and sex-roles. Early research noted the overlap between the Type-A behaviour pattern and masculinity as a way to explain the differential incidence of cardiac events between men and women (e.g. Auten, Hull, & Hull, 1985; Baltis & Small, 1982; Blascovitch, Major, & Katkin, 1981). For example, hostility, competitiveness, dominance, independence, and aggressiveness are shared by both constructs. Help-seeking and asking for social support in times of need might be inconsistent with the male sex-role (e.g. admitting weakness, expression of emotion). Thus, sex-typed men might be at greater risk for CVD through impoverished social support networks and poor health care practices (Helgeson, 1995). In a study examining the relationship between some of the above mentioned variables, Helgeson (1990) found that socially undesirable aspects of masculinity predicted both Type A behaviour and myocardial infarct (MI) severity. In a follow-up study conducted 1 year post-MI, Helgeson (1991) found that masculinity predicted more severe chest pain, but not rehospitalization or perceived health status. Greater disclosure to one's spouse, however, significantly predicted all 3 cardiac outcomes, suggesting a beneficial effect.

A small number of studies have examined the effects of sex-roles on cardiovascular reactivity in the laboratory. Davis and Matthews (1996) found a mismatch effect of sex-role orientation and stressor. Individuals who went through a stress-protocol that was inconsistent with their sex-role orientation showed heightened reactivity. Lash, Eisler, and Schulman (1990) found that men scoring high on Masculine Gender Role Stress (MGRS), or the tendency to appraise situations which threaten the traditional male role as stressful, showed greater blood pressure reactivity and slower recovery to a cold pressor task emphasizing successful performance (high challenge). There were no differences between high and low MGRS participants in the low challenge task. Both these studies suggest that gender-roles can guide the way one processes information and appraises a situation, resulting in greater perceived stress in conditions that do not fit their perception.

Summary and Hypotheses

In summary, this brief review of the literature suggests that while more established psychosocial risk factors such as hostility and social support have a large research base and much heuristic value in guiding research, findings are fraught with inconsistencies. Researchers are now starting to recognize that these constructs are multi-dimensional in nature, and that more specific hypotheses as to what constitutes a vulnerability or a strength need to be proposed. In this vein, several new constructs building on previous research have been suggested in attempts to explain some of the inconsistencies in the literature. The purpose of the present paper is to:

(1) examine how these putative risk factors are related to physiology;

(2) determine whether these new constructs do a better job of accounting for some of the variance in explaining individual differences in resting blood pressure and reactivity to stress.

Specifically, it is predicted that:

(1) hostility will be associated with higher resting blood pressure and greater reactivity to stress;

(2) perceived social support will be associated with lower resting blood pressure and dampened reactivity to stress;

(3) while previous research is suggestive as to the directionality of the relationship between the emerging constructs and physiology, no specific predictions were made because at this point, the nature of the investigation into these variables is still exploratory.

METHOD

Participants

Participants were 182 University of British Columbia undergraduates who took part in the study for course credit. The data from 9 participants were excluded from analyses due to experimenter error or equipment failure. The final sample consisted of 173 participants (86 male, 87 female, average age 20.4). The ethnic composition of the sample was varied (41% North American/European; 35% Chinese; Other East Asian 14%; Indo-Pakistani 4%; Other 6%). Almost half of the sample (46%) had a familial history of hypertension. In terms of lifestyle factors, 9% of participants reported smoking and 64% consumed caffeine products on a regular basis. Finally, 24% of women reported using oral contraceptives.

Physiological Measures

Because we were interested in both cardiovascular and neuroendocrine reactivity to stress, heart rate (HR), systolic blood pressure (SBP), diastolic blood pressure (DBP) and salivary cortisol were monitored throughout the test session. SBP is a measure of the force exerted by blood on the artery walls when the heart is contracting, while DBP reflects the pressure exerted when the heart is as rest (Sarafino, 1994). Cardiovascular indices were

measured using a Dinamap 845 automated blood pressure monitor (Critikon Corporation, Tampa, Fl.). A standard occlusion cuff was placed on the participant's non-dominant arm, and participants were instructed not to move their arms when the cuff was inflated. Saliva samples were collected at set times during the test session by having participants expectorate into pre-labeled vials. Salivary cortisol analyses will be conducted at a later date.

Psychological Measures

Hostility. Hostility was assessed using two self-report inventories. The Cook-Medley Hostility Questionnaire (CMHQ) is a set of 50 true/false items derived from the MMPI initially used to discriminate empirically between teachers with good versus bad rapport with students (Cook & Medley, 1954). Individuals with high scores on the CMHQ seem to hold a cynical world view, regarding interpersonal relationships with bitterness and distrust (Smith & Pope, 1990). While high scores have been linked to poor cardiac outcome, three subsets of items have been identified as having greater predictive power than the total hostility score (Barefoot, Dodge, Peterson, et al., 1989). These subscales, Cynicism, Hostile Affect, and Aggressive Responding, for a total of 27 items, were administered in the current study. The CMHQ has good test-retest reliability ($r = .84$ over four years; Shekelle, Gale, Ostfeld, & Paul, 1983) and internal consistency (Cronbach's alpha = .82; Smith & Frohm, 1985).

The Hostile Attitude Scale (HAS; Arthur, Garfinkel, & Irvine, 1999), an 18-item measure on a 7-point scale, was also included in the present study. The HAS was designed to tap a cognitive set which reveals an enduring, cynical view of the world. The HAS has good psychometric properties (Chronbach $\alpha = .80$). In our sample, it was found to be strongly correlated with the CMHQ subscales (r ranging from .39 to .54). In order to reduce the potential

for variance overlap and multicollinearity for later regression analyses, a composite hostility score was derived by combining the CMHQ and HAS scores.

Social support. The Interpersonal Support Evaluation List (ISEL) consists of 4 subscales assessing the perception of different kinds of support within one's environment (Cohen, Mermelstein, Kamarck, & Hoberman, 1985). Each subscale is made up of 10 true/false items. The tangible subscale measures the perceived availability of instrumental or material aid; the self-esteem subscale taps the availability of people against whom one can make positive evaluations about oneself; the appraisal subscale measures the availability of emotional support; the belonging subscale assesses the availability of having friends/family to do activities with. The ISEL has been found to have good psychometric properties (Chronbach α ranging from .88-.90; test-retest reliability .87 over a 4-week period), and to be free from socially desirable responding biases (Cohen et al., 1985). Because the subscales are moderately to highly intercorrelated (r ranging from .40-.61),a total ISEL score was used in this study to tap a more general perception of a socially supportive environment.

Social support seeking. The Coping Strategy Indicator (CSI) is a 33-item measure assessing coping strategies (Amirkhan, 1990). It consists of 3 factor analytically derived subscales, viz. Problem Solving, Seeking Support, and Avoidance. Respondents are asked to think of a stressful event they experienced during the past 6 months, and then to answer questions on a 3-point scale indicating to what extent they engaged in particular coping behaviors during that time. For the purposes of the present study, only the Seeking Support subscale was used. The CSI has excellent psychometric properties, with internal consistency of .93 and test-retest reliability over 8 weeks of .86 for the Seeking Support subscale.

Presentation styles. Presentation styles were assessed with the Balanced Inventory of Desirable Responding (BIDR; Paulhus, 1991). The BIDR consists of 40 items, 20 tapping self-deception and 20 tapping impression management. Items include "I never regret my decisions" and "I always declare everything at customs" for the self-deception and impression management subscales respectively. Responses are on a 7-point scale, but only extreme scores are counted. Score can range from 0-20 for each subscale. Psychometric properties are good (Paulhus, 1991).

Sociotropy. As suggested by Ewart et al. (1998), the Need for Social Approval Scale from the National Institute of Mental Health (NIMH) collaborative study of depression was used (Imber et al., 1990). This measure was included to assess sociotropy, and not defensiveness, as the name of the scale would suggest. Derived through factor analysis, the measure consists of 11 items such as "If others dislike you, you cannot be happy" and "I am nothing if a person I love doesn't love me". Participants are requested to rate their agreement with each statement on a 7-point scale. Internal consistency for the scale is good ($\alpha = .82$).

Type-D personality. The DS24 (Denollet, 1997) is a 24-item measure answered on a 5-point Likert scale ranging from 0 (false) to 4 (true). The measure is made up of 2 subscales assessing the negative affectivity and social inhibition domains of the Type-D construct. Individuals are categorized as Type-D's if they fall above the median scores on both the negative affectivity and social inhibition subscales (29 for women, 31 for men). Psychometric properties are good (internal consistency = .76-.86; test-retest = .78-.87).

Sex-roles. The Bem Sex-Role Inventory (BSRI; Bem, 1974) consists of 60 personality characteristics (20 masculine, 20 feminine, and 20 filler) which participants rate on a 7-point scale reflecting the extent to which they believe the characteristic describes themselves. The measure yields a Masculinity score and a Femininity score which provide the basis for

classifying participants into one of 4 orientations: Masculine (hi masc/lo fem), Feminine (hi fem/lo masc), Androgynous (hi masc/hi fem), and Undifferentiated (lo masc/lo fem). The BSRI has good internal consistency, ranging from .75 and .87, and good test-retest reliability, ranging from .76 to .94 over a 4-week period. The BSRI was developed in the 70's by having judges evaluate how socially desirable it would be for men and women to possess certain personality characteristics in North American society. A recent study indicates that the items still reflect characteristics which are considered socially desirable in men and women today (Holt & Ellis, 1998).

Affect Measures

Self-report measures in the form of a visual analogue scale (VAS; 7.5 cm) were administered throughout the laboratory session, at the end of the baseline and task phases, and twice during the recovery phase (see below). Participants were asked to rate on the VAS the extent to which they felt happiness, anger, sadness, fear, disgust, and surprise at that moment in time.

Procedure

Participation consisted of 2 phases. During the first phase, participants came to the lab to pick up a questionnaire package which consisted of the psychological measures described above. At the time of their first visit, participants were given a tour of the lab in order to minimize reactivity to the lab environment during the test session. They were also instructed not to consume caffeine or nicotine for at least 2 hours prior to their test session, to eat a light meal at least 1 hour prior to their second visit, and to take care to not make their gums bleed during their dental hygiene routine.

During the second phase, participants completed a stress protocol which involved a math task (serial 7 subtraction) with harassment. Because the study involved deception, the experiment was always conducted by an experimenter and a confederate pair. The experimenter greeted the participants, led them into a quiet room where they sat alone for the remainder of the study, and gave information about the study and cardiovascular measurement. Resting blood pressure and heart rate were assessed during a 12-minute baseline phase (measurement at minutes 1, 2, 10, and 12). Prior to the stress task, participants were given instructions which emphasized speed and accuracy, told that the best performer would receive a $50 bonus prize, and that a "lab technician" would be monitoring their performance throughout the task. The harassment was delivered via intercom by the lab technician, a gender-matched confederate. The scripted negative feedback was similar to the protocol used by Earle, Linden, and Weinberg (1999) and was designed to induce anger and irritation during the task (e.g. "Look, you're always subtracting way too slow. You've got to do it much faster. Continue where you stopped"). A subset of participants acted as controls for an experimental manipulation during the recovery period (Anderson & Linden, 2000) and did not receive harassing feedback during the task. The task period lasted 13 minutes, and blood pressure and heart rate were measured 5 times during that time. At the end of the task period, participants, with the exception of the control participants described above, underwent 1 of 3 different experimental conditions which examined the impact of an interpersonal interaction on recovery from stress (Anderson & Linden, 2000). At the end of the test session, participants were fully debriefed about the nature of the study, and both the experimenter and the confederate were present to answer any questions that participants might have had.

RESULTS

Overview of Analyses

In order to first examine the strength and direction of the relationships among individual personality variables and physiology, bivariate correlation coefficient between personality and baseline and reactivity change scores were computed. Because initial resting levels have been shown to correlate with change scores, residualized change scores were used to control for individual differences in baseline levels and their impact on subsequent magnitude of change (Manuck, Kraprowicz, & Muldoon, 1990). Next, multiple regression was used to determine which of the psychological variables were the strongest predictors of physiology, after controlling for standard biological risk factors of age, familial history of blood pressure, caffeine and nicotine consumption.

Manipulation Check

Mean values for baseline, task, and raw change scores are presented in Table 1. In order to verify that the stress task was effective in producing physiological change, a series of 2 (gender: males, females) X 2 (time: baseline, task) between-within repeated measures ANOVA were run on each of the dependent variables. For all 3 outcome variables, results indicate that the task was associated with physiological arousal [SBP: $F(1,170) = 389.50$, $p < .001$; DBP: $F(1,170) = 895.73$, $p < .001$; and HR: $F(1,170) = 429.39$, $p < .001$]. For SBP, a significant main effect of gender also emerged, indicating males had higher resting and task levels than women [$F(1,170) = 45.33$, $p < .001$][1].

Personality Variables

Missing data (less than 1% of total scores) on the personality questionnaires were replaced with group means within each gender. This technique allows the full use of the sample

while minimizing loss of information, a conservative way of dealing with missing data as it reduces variability among scores (Tabachnick & Fidell, 1996). Mean values are presented in Table 2. Few gender differences emerged. Consistent with previous findings, men reported higher hostility levels [$t(171) = 3.83$, $p < .001$] while women reported higher levels of perceived support and support seeking [$t(171) = -2.60$, $p < .01$; $t(171) = -3.63$, $p < .001$ respectively] (Burda, Vaux, & Schill, 1984; Butler, Giordano, & Neren, 1985). Further, gender differences in sex-typing were gender-matched, with men reporting higher masculinity levels [$t(170) = 2.68$, $p < .01$] and women higher femininity levels [$t(171) = -2.24$, $p < .05$].

Intercorrelations among the personality variables are presented in Table 3. Perceived social support was found to be positively related to support seeking, masculinity and femininity. Further, hostility was found to be associated with higher levels of negative affectivity, social inhibition, and sociotropy, and with lower levels of impression management. Thus, bivariate correlations generally support the classification of related variables into the buffer and vulnerability groups.

Bivariate Correlations

Correlations with baseline physiology. Among the psychosocial vulnerability group, high hostility levels were found to be associated with lower DBP ($r = -.23$, $p < .01$), while high levels of impression management were associated with higher DBP levels ($r = .15$, $p < .05$). Among the support-related variables, masculinity was inversely associated with resting heart rate ($r = -.21$, $p < .01$). While both men and women showed the same pattern of association between physiology and hostility (DBP: $r = -.25$, $p < .05$ for men; SBP: $r = -.29$, $p < .01$, DBP: $r = -.27$, $p < .05$ for women), the relationship between heart rate and masculinity was only found in men (r

= -.30, $p < .01$), and the association between impression management and DBP was only found in women ($r = .28$, $p < .05$).

Correlations with reactivity. In the overall sample, few significant correlations emerged[2]. Support seeking and social inhibition[3] showed moderate relationships with SBP change ($r = -.15$, $p = .05$; $r = .16$, $p < .05$, respectively). However, when males and females were considered separately, gender differences in the pattern of associations were apparent. Males showed similar reactivity patterns as those in the overall sample [support seeking and SBP, $r = -.25$, $p < .05$; social inhibition and SBP, $r = .24$, $p < .05$). For women, sociotropy was found to be associated with less SBP change during the task ($r = -.21$, $p < .05$).

Regression Analyses[4]

In order to see whether any of the bivariate relationships still held after controlling for biological risk factors, a series of hierarchical multiple regression analyses were run on each of the dependent variables. For baseline SBP, DBP, and HR, biological variables were entered on the first step. Then, personality variables were entered on the second step. Finally, since gender differences in the pattern of correlations emerged, interaction terms among personality variables and gender, i.e. the cross-product of mean-deviated scores (Cohen & Cohen, 1983), were entered on the final step. To examine the relationship between personality and reactivity, biological variables were entered on the first step. Because a subset of participants did not receive harassment during the stress protocol, task was entered on the second step. Finally, personality variables and personality by gender interactions were entered on the third and fourth steps respectively. Significant interactions were followed-up in the manner described by Aitken and West (1991).

Regression predicting baseline levels. Among the buffering variables, only masculinity emerged as a significant predictor of resting heart rate [$\beta = -.26$, $p < .01$; see table 4]. Several of the variables from the negative affect group significantly predicted baseline levels. Consistent with associations observed at the bivariate level, regression analyses indicated that hostility was a predictor of both SBP and DBP resting levels, even after controlling for traditional risk factors [$\beta = -.19$, $p < .05$; $\beta = -.22$, $p < .05$ for SBP and DBP respectively; see Tables 5 and 6]. A significant interaction between impression management and gender emerged in the model predicting resting SBP. Follow-up analyses indicated that impression management was inversely related to SBP for men [$\beta = -.24$, $p < .05$], while no relationship was apparent for women. Finally, social inhibition significantly predicted higher resting HR levels [$\beta = .22$, $p < .05$]. However, this finding should be interpreted with caution given that the overall model predicting baseline HR was not significant.

Regression predicting reactivity. None of the stress-buffering variables significantly predicted reactivity. On the other hand, several of the negative affect variables were associated with reactivity. Sociotropy negatively predicted lower DBP and HR reactivity [$\beta = -.21$, $p < .05$; $\beta = -.22$, $p < .05$ for DBP and HR respectively; see Tables 8 and 9]. The Type-D dimensions interacted with gender in all 3 regression models. For SBP, both negative affectivity and social inhibition significantly interacted with gender (see Table 7). Follow-up analyses indicated that for males, social inhibition was associated with greater SBP reactivity [$\beta = .41$, $p < .01$], whereas the relationship was non-significant for women. Follow-up analyses for negative affectivity did not reveal any significant pattern of associations. For DBP, there was a significant interaction between social inhibition and gender (see Table 8), indicating that social inhibition was positively associated with reactivity for men [$\beta = .31$, $p < .05$], but not for women. Finally,

negative affectivity interacted with gender to predict HR reactivity (see Table 9), but follow-up analyses did not reveal any significant gender differences.

Personality and Self-Reported Affect

Correlations with baseline affect. The self-report VAS affect measures were administered as a validity check for the experimental manipulation. However, associations between affect and personality were examined on a post-hoc basis. Few significant correlations among the personality variables and self-reported affect emerged prior to the stressor, and most of them involved the psychosocial vulnerability variables. Individuals high in negative affectivity and social inhibition reported experiencing less happiness ($r = -.22$, $p < .01$ and $r = -.18$, $p < .05$ for negative affectivity and social inhibition respectively) and more disgust ($r = .16$, $p < .05$ for negative affectivity). High self-deception was associated with less self-reported fear ($r = -.16$, $p < .05$), while impression management was associated with less sadness ($r = .15$, $p < .05$). Finally, support seeking was related to less fear prior to the stress task ($r = -.16$, $p < .05$). Thus, Type-D dimensions were associated with more negative affect, defensiveness with less self-reported affect, and support seeking with a buffering effect of less fear.

Correlations with task affect. Again, most of the significant correlations between personality and self-reported affect at the end of the stress task involved the psychosocial vulnerability variables. Partial correlations controlling for the effects of the harassment indicated that social inhibition was associated with significantly more sadness ($r = .19$, $p < .05$) and disgust ($r = .15$, $p < .05$) during the task. Self-deception was related to less fear ($r = 0.27$, $p < .001$) and less disgust ($r = -.17$, $p < .05$). Sociotropy was also associated with more fear ($r = .19$, $p < .05$). Finally, individuals high in masculinity reported experiencing less fear during the task ($r = -.19$, $p < .05$).

DISCUSSION

To summarize, psychosocial factors were related to physiology in the present sample. Among the stress buffering variables, masculinity was found to be associated with lower resting heart rate, even after controlling for standard biological risk factors. Support seeking was related to lesser SBP change during the stress task, but the relationship was no longer significant in the regression analyses. Among the vulnerability variables, only the Type-D dimensions showed the expected pattern of associations of increased resting levels and change during the stressor. Hostility was found to be inversely related to resting DBP and SBP, while sociotropy was associated with less SBP and HR reactivity. Impression management showed a paradoxical pattern for men and women, with associations with higher resting DBP in women at the bivariate level, but with lower resting SBP in men in the regression analyses (see Table 10 for a visual summary of results).

Established vs. Emerging Traits: Any Winners?

One of the main objectives of the present study was to compare the predictive ability of newer personality constructs with that of the more established traits of hostility and social support. It was hypothesized that these putative personality risk factors might shed some light on the inconsistencies observed in the literature between the established traits and indices of cardiovascular disease. Results from this study indicate that the emerging psychosocial factors are significantly related to laboratory indices of cardiovascular health. Furthermore, results indicate that these newer constructs did a better job of accounting for the variance in both resting BP and cardiovascular activity during acute stress than their more established counterparts. However, the observed pattern of results was not always in the hypothesized direction,

suggesting that even these putative factors may benefit from further refinement and situational specificity.

Psychosocial vulnerability traits. Contrary to expectation, hostility was found to be inversely related to blood pressure levels, with high hostility associated with lower BP. The relationship was consistent across both SBP and DBP indices, arguing against random variation. Other studies have also found inverse relationship between hostility and resting BP (e.g., Brownley, Light, & Anderson, 1996; Davidson, Hall, & MacGregor, 1996; Durel, Carver, Spitzer et al., 1989; Shapiro et al., 1995). Suls, Wan, and Costa (1995), in their meta-analysis of the relationship between resting blood pressure and anger/hostility measures, found that the overall effect size between personality and BP was in the expected direction ($\underline{d}$ = .08 for SBP; $\underline{d}$ = .06 for DBP). However, results demonstrated considerable heterogeneity, with numerous studies fraught with methodological problems such as selection bias (e.g., studies of hypertensive patients) and unreliable BP assessment. However, these methodological issues cannot explain the present pattern of findings, given that sound methodology was used to assess BP and that participants were healthy college students. As such, the current results confirm the need to move away from hostility as a global construct and to seek more specific personality traits which might show more consistent associations with blood pressure.

Several of the putative constructs examined in the present study were related to physiology. Denollet's (1998) Type-D construct emerged as one of the most consistent predictors of both resting heart rate and DBP and SBP reactivity. However, the global Type-D construct, defined as the joint tendency towards negative affect and social inhibition (Denollet & De Potter, 1992) did not seem to have as much prognostic ability as its individual components. Specifically, social inhibition was associated with heightened reactivity to laboratory stress, but

only in men. Denollet has consistently found the Type-D construct to be a prognostic indicator of poor cardiac outcome, and his studies have included largely male samples. Thus, the present study suggests a potential pathophysiological role for the type-D construct in the development of disease, particularly in men.

Notably, both social inhibition and negative affectivity were moderately to highly associated with the perception of a socially unsupportive environment. Because lack of social support has been associated with disease morbidity and cardiac deaths (Berkman et al., 1992; Orth-Gomér et al., 1993; Orth-Gomér & Unden, 1990), low perceived support may partly confound the relationship between Type-D and cardiac outcome. Although the present study cannot address this issue, the fact that social inhibition was consistently related to physiology suggests that the Type-D construct might be causally related to the development of CVD through its relation to cardiovascular hyperreactivity.

Sociotropy was also significantly related to physiology, though the observed pattern of associations was not in the expected direction. Contrary to Ewart et al. (1998)'s finding that sociotropy, or dependence on interpersonal relationships for esteem and approval, is related to increased reactivity to stress in young women, the present results indicate that sociotropy was associated with dampened DBP and HR reactivity to stress in both men and women. Ewart et al. suggest that sociotropy acts as vulnerability to disease and distress through a stress-diathesis whereby situations with a strong interpersonal component may elicit strong physiological and affective responses, particularly in sociotropic individuals. Perhaps one of the reasons our study failed to replicate Ewart et al. (1998)'s finding is that the task used in the present study did not create a stressful enough interpersonal context to elicit similar cardiovascular responses. The current task was chosen to add an interpersonal dimension to the math task, as harassment has

been shown to evoke frustration, irritation, and anger among participants (Earle et al., 1999; Felsten, 1995; Suarez et al., 1993). However, because the harassment was delivered by a confederate, it may be that a negative interpersonal experience with a stranger is not enough to threaten the sociotropic individual. That is, sociotropic individuals might react most when they engage in a negative interaction with close others.

The fact remains, however, that regardless of the success of our manipulation in creating a "stressful" context, sociotropy was associated with *decreased* reactivity to stress. The reasons for such a relationship are unclear. Several researchers have found sociotropy to be related to neuroticism, or the tendency to experience negative affect (Cappeliez, 1993; Dunkley, Blankstein, & Flett, 1997; Gilbert & Reynolds, 1990; Moore & Blackburn, 1994; Zuroff, 1994). While there are inconsistencies in the literature, neuroticism has been associated with lower resting BP and cardiovascular reactivity (Davies, 1970; Siegman, 1994b). The possibility remains, then, that the relationship between sociotropy and dampened reactivity observed in the present study might be explained by a third variable, namely neuroticism. It should be noted that the 2 other variables which share some overlap with negative affect in the present study, hostility and Type-D's negative affectivity, also show inverse relationships with physiology, although the relationship with negative affectivity is non-significant. Thus, further research is needed to elucidate the role of trait neuroticism in explaining how sociotropy may affect one's reactions to stress.

Defensiveness also showed significant associations with physiology. Impression management, or the conscious tendency to present favorably in front of others (Paulhus, 1984), was found to be associated with higher resting DBP in women. While this finding should be interpreted with some degree of caution given that the relationship no longer remained

significant after controlling for traditional risk factors, it does provide some converging evidence that this response style might prove to be a gender-specific vulnerability to stress and disease (Habra & Linden, 2000). In contrast, regression analyses indicated that for men, impression management was associated with *lower* baseline SBP. This finding was somewhat surprising given that past studies had indicated that for men, self-deceptive strategies were particularly health-damaging (Linden et al. 1993; Rutledge & Linden, 2000; Rutledge et al., 1999). No other published study has documented a positive relationship between BP and impression management, indicating a need for replication and tentative interpretation. However, the fact that presentation styles were differentially associated with gender-roles, with self-deception strongly related to masculinity ($r = .47$) and impression management more weakly related to femininity ($r = .16$), suggests that men and women tend to engage in these defensive strategies in dissimilar ways. It might be that men and women experience differential success when they engage in impression management. Paulhus (1998) suggests that defensiveness should not be viewed as globally adaptive or maldaptive. Rather, he suggests looking for specific personality-by-situation interactions in explaining the effects of response styles on mental health. The present data suggest that gender should also be taken into consideration. It may be that women tend to use impression management in a relational or interpersonal context more often than men, which over the long run is perceived as being manipulative and alienates close others. Obviously, the above argument is conjecture at this point, but the current findings do provide some interesting possibilities for future research.

Psychosocial buffering traits. Perceived social support was unrelated to either baseline or reactivity blood pressure levels. The current findings do not therefore provide support that cognitive social support (Schwarzer & Leppin, 1991) influences on how one reacts

physiologically to stress, at least in the laboratory. This may suggest that under acute stress, support in the natural environment does not change one's appraisal of the perceived stressfulness of an event. It may be that the effects of support manifest themselves only once support has been mobilized, either behaviorally or cognitively. Thus, the presence of a supportive friend or confederate tends to dampen physiology (Kors, Linden, & Gerin, 1996), and perceived social support hastens the return to baseline following acute stress (Roy et al., 1998). However, the fact that the social support has been inconsistently linked to blood pressure suggests that researchers need to examine different variables to further elucidate how social support exerts beneficial effects.

Several support-related variables were examined in the present study, but few showed meaningful relationships with physiology. Masculinity was negatively associated with resting heart rate, even after controlling for traditional risk factors. Recall that masculinity has been associated with both positive psychological adjustment (Silten Bassoff & Glass, 1982) and increased risk for CVD (Helgeson, 1995). The present findings are more consistent with the masculinity model of psychological adjustment, with trait masculinity conferring stress-buffering effects. First, an association with lower resting heart rate is consistent with previous research indicating that individuals high in masculinity are more likely to keep a regular exercise schedule (Shifren et al., 1993). The emphasis on athleticism and physical strength associated with masculinity might be enough to counter the negative effects of engaging in risky health behaviors such as smoking and drinking alcohol, which are also associated with masculinity. Second, results from the present study suggest that masculinity is negatively associated with psychosocial vulnerability variables such as both dimensions of the Type-D personality and sociotropy. Finally, masculinity was positively correlated with high levels of perceived social

support. Thus, the picture emerging from the present data is that of a buffering personality trait associated with both positive mental and physical health.

Support seeking was also related to physiology, although the relationship no longer remained significant in the regression analyses. Support seeking was found to be associated with dampened SBP reactivity during the stress task, both in the overall sample and in males. In this respect, the present findings lend support to those of Fontana et al. (1998) and Kors and Linden (1999) in suggesting that the tendency to seek support in times of need might buffer the individual against the effects of stress. Furthermore, the relationship between support seeking and physiology seems to be stronger in men than in women, both in the current sample and in that of Kors and Linden (1999). Fontana et al. (1998) did not include males in their study. As noted above, women generally report higher levels of perceived support and support seeking (Ashton & Fuehrer, 1993; Burda, Vaux, & Schill, 1984; Butler, Giordano, & Neren, 1985). This gender difference was also noted in the current sample. In sum, men tend to seek less support than women, yet seem to benefit more from the coping strategy when they engage in it, at least at the physiological level. It may be that in men, engaging in support seeking is associated with a health-promoting approach to life, characterized by positive interpersonal relationships and psychological adjustment. In our sample, support seeking was associated with lower hostility, negative affectivity and social inhibition levels. Thus, the present findings suggest that seeking support as a way of coping with stress also has benefits at the physiological level, particularly for men.

Resting vs. Reactivity Levels: Implications for CVD

Interestingly, there was little overlap between baseline and reactivity levels in terms of associations with psychological variables. That is, personality was not consistently associated

with physiology across both resting and stress levels. What this means in terms of development of cardiovascular disease is unclear. Both resting blood pressure levels and cardiovascular reactivity have been linked to the future development of hypertension (Beckett, Rosner, Roche, & Guo, 1992; Menkes et al., 1989; Nelson, Ragland, & Syme, 1992) and CVD (Levy, Wilson, Anderson, & Castelli, 1990). The majority of participants in the present sample were young, healthy college students, who presumably in the early stages preceding the development of heart disease and hypertension. Without prospective data, it is difficult to say whether personality traits associated with resting BP are involved in the disease process differently than traits associated with reactivity. However, the present findings highlight the usefulness of examining both baseline and stress levels as indices of cardiovascular health, with each index yielding information about the pathophysiology of CVD.

Psychosocial Variables and Self-reported Affect

Another interesting pattern emerging from the present data is that the psychosocial buffers, in contrast to the vulnerability variables, showed few associations with physiology. Masculinity and support seeking were each related to a single index of cardiovascular health, whereas hostility, sociotropy, and social inhibition were associated with multiple markers of health. The reason for that discrepancy is again not clear. It may be that the vulnerability variables examined here are more closely related to affect than the buffer variables. Indeed, associations with self-reported affect both at rest and during the task suggest that individuals high in sociotropy, social inhibition, and negative affectivity do report experiencing more negative emotions. Among the psychosocial buffers, masculinity and support seeking were each associated with less fear, suggesting that each confers protective effects through a reduction in

negative affect. Interestingly, these were the two stress buffering variables which showed significant associations with physiology.

Summary and Conclusions

Several limitations of the present study should be noted. First, the sample consisted largely of young, healthy undergraduates. This limits generalizability of the current findings to a more at-risk population. In particular, the Type-D personality construct has only been examined in cardiac patients. This is the first study to examine the relationship of the construct to markers of cardiovascular disease in healthy individuals. While our results are generally supportive of a relationship between Type-D and CVD, social inhibition, and not the global Type-D, was found to be related to cardiovascular health. A reactivity study with cardiac patients seems warranted to examine whether disease status makes a difference in how Type-D affects physiology.

Second, the current findings are generally limited to how personality and physiology relate to stress experienced in the laboratory, which may not be representative of stress experienced during daily life. Ambulatory blood pressure monitoring would be a logical next step in determining whether the relationships observed in the current study still hold as individuals face daily hassles and stressors. However, the laboratory environment does provide the opportunity to more clearly refine our knowledge about personality and stress. For example, the fact that sociotropy was associated with dampened reactivity to the stress task clarifies the nature of what is perceived to be stressful. It not only sheds light on which aspects of the task are most "toxic" (i.e., is it the anger-induction, the interpersonal challenge, the need to preserve an amiable relationship with the experimenter), but it also allows us to make clearer statements as to which aspects of personality, under which conditions, are most harmful/helpful. Thus, lab

studies provide an essential first step in studying the relationship between personality and physiology.

To conclude, the present study set out to examine the relationship between established and emerging personality constructs. Noting that the established global traits of hostility and social support were often inconsistently associated with physiology, it was hypothesized that more specific traits might be better able to account for some of the variance in explaining resting and reactivity BP and HR. The present findings suggest that personality is indeed associated with how one reacts to stress. At the same time, however, they also point to a further refining of some of these emerging constructs, to better clarify how personality can confer deleterious or beneficial effects on health.

FOOTNOTES

[1] Initial analyses indicated that there were no differences in resting and reactivity levels between Asians and Caucasians. Consequently, ethnicity was dropped from all further analyses.

[2] Correlations with reactivity levels were initially examined separately for participants receiving the harassment and for those who did not. Because the direction of associations was similar in both groups, it was decided to use the entire sample in the analyses. However, the effects of task (harassment vs. no harassment) was controlled for statistically in the regression analyses.

[3] Initial analyses revealed that the global Type-D construct was unrelated to physiology. Consequently, all further analyses were conducted with the separate dimensions of social inhibition and negative affectivity.

[4] Residual scatterplots were examined to ensure that assumptions of linearity, homoscedasticity, and normality were met (Tabachnick & Fidell, 1996). None of the plots of the predicted and observed residual scores were found to violate the above assumptions. Mahalanobis and Cook's distances were used to identify potential outliers which might exert undue influence on the model. Cook's distance assesses the "combined impact of the *i*th case on all the estimated regression coefficients" (Neter, Wasserman, & Kutner, 1990, p. 403). Values greater than 1 were used as a cut-off to identify extreme scores (Tabachnick & Fidell, 1996). No extreme cases were found, indicating that none of the participants had disproportionate influence on the model. Mahalanobis distance identifies multivariate outliers, or cases which have an unusual combination of extreme scores. The Chi Square distribution with p (number of predictors) degrees of freedom was used to identify outliers. A conservative probability level was used ($p < .001$; Tabachnick & Fidell, 1996). Using this procedure, 3 cases were identified.

After checking that no data entry errors were made, cases were examined individually. Two of the cases were older participants who also had extreme scores on some of the personality measures [participant #104 was a 60 year-old with extremely high perceived social support and support seeking scores; participant #167 was a 52 year-old with low sociotropy and hostility scores]. The third outlier was a 22 year-old who reported high support seeking but unusually low masculinity. Thus, the decision was made to keep these outliers in the regression models, as they were considered part of the population of interest, even though some of their scores were extreme.

REFERENCES

Aiken, L. S. & West, S. G. (1991). Multiple Regression: Testing and Interpresting Interactions. Newbury Park, CA: Sage Publications.

Anderson, J. C. & Linden, W. (2000). Influence of Apology Strategies on Physiological Recovery from Harassment. Unpublished Master's thesis, University of British Columbia.

Amirkhan, J. H. (1994). Criterion validity of a coping measure. Journal of Personality Assessment, 62, 242-261.

Arthur, H. M., Garfinkel, P. E., & Irvine, J. (1999). Development and testing of a new hostility scale. Canadian Journal of Cardiology, 15, 539-544.

Ashton, W. A. & Fuehrer, A. (1993). Effects of gender and gender role identification of participant and type of social support resource on support seeking. Sex Roles, 28, 461-476.

Auten, P. D., Hull, D. B., & Hull, J. H. (1985). Sex role orientation and Type A behavior pattern. Psychology of Women Quarterly, 9, 288-290.

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, 59-63.

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

Barrera, M. Jr. (1986). Distinctions between social support concepts, measures, and models. American Journal of Community Psychology, 14, 413-445.

Batlis, N. & Small, A. (1982). Sex roles and Type A behavior. Journal of Clinical Psychology, 38, 315-316.

Beck, A. T. (1997). The past and future of cognitive therapy. Journal of Psychotherapy Practice and Research, 6, 276-284.

Beckett, L. A., Rosner, B., Roche, A. F., & Guo, S. (1992). Serial changes in blood pressure from adolescence into adulthood. American Journal of Epidemiology, 135, 1166-1177.

Bem, S. L. (1974). The measurement of psychological androgyny. Journal of Consulting and Clinical Psychology, 42, 155-162.

Bem, S. L. (1981). Bem Sex-Role Inventory: Professional manual. Palo Alto, CA: Consulting Psychological Press.

Berkman, L. F., Leo-Summers, L., & Horwitz, R. I. (1992). Emotional support and survival after myocardial infarction. Annals of Internal Medicine, 117, 1003-1009.

Bland, S. H., Krogh, V., Winkelstein, W., & Trevisan, M. (1991). Social network and blood pressure: A population study. Psychosomatic Medicine, 53, 598-607.

Blascovich, J., Major, B., & Katkin, E. S. (1981). Sex-role orientation and Type A behavior. Personality and Social Psychology Bulletin, 7, 600-604.

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

Broadwell, S. D. & Light, K. C. (1999). Family support and cardiovascular responses in married couples during conflict and other interactions. International Journal of Behavioral Medicine, 6, 40-63.

Brooks, P. R., Morgan, G. S., & Scherer, R. F. (1990). Sex role orientation and type of stressful situation: Effects on coping behaviors. Journal of Social Behavior and Personality, 5, 627-639.

Brownley, K. A., Light, K. C., & Anderson, N. B. (1996). Social support and hostility interact to influence clinic, work, and home blood pressure in Black and White men and women. Psychophysiology, 33, 434-445.

Burda, P. C. Jr., Vaux, A., & Schill, T. (1984). Social support resources: Variation across sex and sex-role. Personality and Social Psychology Bulletin, 10, 119-126.

Butler, T., Giordano, S., & Neren, S. (1985). Gender and sex-role attributes as predictors of utilization of natural support systems during personal stress events. Sex Roles, 13, 515-524.

Cappeliez, P. (1993). The relationship between Beck's concepts of sociotropy and autonomy and the NEO-Personality Inventory. British Journal of Clinical Psychology, 32, 78-80.

Cohen, J. & Cohen, P. (1983). Applied Multiple Regression/Correlation Analysis for the Behavioral Sciences. (2 ed.) Hillsdale, NJ: Lawrence Erlbaum Associates.

Cohen, S., Mermelstein, R., Kamark, T., & Hoberman, H. M. (1985). Measuring the functional components of social support. In I.G. Sarason & B. R. Sarason (Eds.), Social Support: Theory, Research, and Applications (pp. 73-94). Dordrecht, Netherlands: Martinus Nijhoff.

Cohen, S. & Wills, T. A. (1985). Stress, social support, and the buffering hypothesis. Psychological Bulletin, 98, 310-357.

Cohen, S. (1988). Psychosocial models of the role of social support in the etiology of physical disease. Health Psychology, 7, 269-297.

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

Coyne, J. C. & Whiffen, V. E. (1995). Issues in personality as diathesis for depression: The case of sociotropy-dependency and autonomy-self-criticism. Psychological Bulletin, 118, 358-378.

Crowne, D. & Marlowe, D. (1964). The Approval Motive: Studies in Evaluative Dependence. New York, NY: John Wiley & Sons.

Davidson, K. W., Hall, P., & MacGregor, M. (1996). Gender differences in the relation between interview-derived hostility scores and resting blood pressure. Journal of Behavioral Medicine, 19, 185-201.

Davies, M. (1970). Blood pressure and personality. Journal of Psychosomatic Research, 14, 89-104.

Davis, M. C. & Matthew, K. A. (1996). Do gender-relevant characteristics determine cardiovascular reactivity? Match versus mismatch of traits and situation. Journal of Personality and Social Psychology, 71, 527-535.

Dembroski, T. M., MacDougall, J. M., Costa, P. T. Jr., & Grandits, 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.

Denollet, J. & De Potter, B. (1992). Coping subtypes for men with coronary heart disease: Relationship to well-being, stress and Type A behavior. Psychological Medicine, 22, 667-684.

Denollet, J., Sys, S. U., & Brutsaert, D. L. (1995). Personality and mortality after myocardial infarction. Psychosomatic Medicine, 57, 582-591.

Denollet, J., Sys, S. U., Stroobant, N., Rombouts, H., Gillebert, T. C., & Brutsaert, D. L. (1996). Personality as independent predictor of long-term mortality in patients with coronary heart disease. Lancet, 347, 417-421.

Denollet, J. (1997). Personality, emotional distress and coronary heart disease. European Journal of Personality, 11, 343-357.

Denollet, J. (1998). Personality and coronary heart disease: The Type-D Scale-16 (DS16). Annals of Behavioral Medicine, 20, 209-215.

Denollet, J. & Brutsaert, D. L. (1997). Personality, disease severity, and the risk of long-term cardiac events in patients with a decreased ejection fraction after myocardial infarction. Circulation, 97, 167-173.

Denollet, J. (1998). Personality and risk of cancer in men with coronary heart disease. Psychological Medicine, 28, 991-995.

Dressler, W. W., Dos Santos, J. E., & Viteri, F. E. (1986). Blood pressure, ethnicity, and psychosocial resources. Psychosomatic Medicine, 48, 509-519.

Dressler, W. W., Mata, A., Chavez, A., Viteri, F. E., & Gallagher, P. (1986). Social support and arterial pressure in a central Mexican community. Psychosomatic Medicine, 48, 338-350.

Dunkley, D. M., Blankstein, K. R., & Flett, G. L. (1997). Specific cognitive-personality vulnerability styles in depression and the five-factor model of personality. Personality and Individual Differences, 23, 1041-1053.

Durel, L. A., Carver, C. S., Spitzer, S. B., Llabre, M. M., Weintraub, J. K., Saab, P. G., & Schneiderman, N. (1989). Associations of blood pressure with self-report measures of anger and hostility among Black and White men and women. Health Psychology, 8, 557-575.

Earle, T. E., Linden, W., & Weinberg, J. (1999). Differential effects of harassment on cardiovascular and salivary cortisol stress reactivity and recovery in women and men. Journal of Psychosomatic Research, 46, 125-141.

Ewart, C. K., Jorgensen, R. S., & Kolodner, K. B. (1998). Sociotropic cognition moderates blood pressure response to interpersonal stress in high-risk adolescent girls. International Journal of Psychophysiology, 28, 131-142.

Felsten, G. (1995). Cynical hostility influences anger, but not cardiovascular reactivity during competition with harassment. International Journal of Psychophysiology, 19, 223-231.

Fontana, A., Pontari, B., & Nash, D. (1998). Coping and parental history of cardiovascular disorders influence blood pressure in women tested during different phases of their menstrual cycle. Behavioral Medicine, 23, 179-188.

Friedman, H. S. & Rosenman, R. H. (1959). Association of specific overt behavior pattern with blood and cardiovascular findings. Journal of the American Medical Association, 169, 1286-1296.

Gilbert, P. & Reynolds, S. (1990). The relationship between the Eysenck Personality Questionnaire and Beck's concepts of sociotropy and autonomy. British Journal of Clinical Psychology, 29, 319-325.

Graham, J. R. (1993). MMPI-2: Assessing Personality and Psychopathology. (2nd ed.) New York, NY: Oxford University Press.

Grossman, P., Watkins, L. L., Ristuccia, H., & Wilhelm, F. H. (1997). Blood pressure responses to mental stress in emotionally defensive patients with stable coronary artery disease. American Journal of Cardiology, 80, 343-346.

Habra, M.E. & Linden W. (2000, March). Personality styles are related to blood pressure recovery from laboratory stress. Poster presented at the Meeting of the American Psychosomatic Society, Savannah, Georgia.

Hanson, B. S., Isacsson, S., Janzon, L., Lindell, S., & Rastam, L. (1988). Social anchorage and blood pressure in elderly men - a population study. Journal of Hypertension, 6, 503-510.

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, 105-121.

Heart and Stroke Foundation of Canada. (1999). The Changing Face of Heart Disease and Stroke in Canada. Ottawa, Canada: Author.

Helgeson, V. S. (1990). The role of masculinity in a prognostic predictor of heart attack severity. Sex Roles, 22, 755-774.

Helgeson, V. S. (1991). The effects of masculinity and social support on recovery from myocardial infarction. Psychosomatic Medicine, 53, 633.

Helgeson, V. S. (1995). Masculinity, men's roles, and coronary heart disease. In D.F. Sabo & D. F. Gordon (Eds.), Men's Health and Illness: Gender, Power, and the Body. (pp. 68-104). Thousand Oaks, CA: Sage Publications.

Helmers, K. F., Krantz, D. S., Bairey Merz, C. N., Klein, J., Kop, W. J., Gottdiener, J. S., & Rozanski, A. (1995). Defensive hostility: Relationship to multiple markers of cardiac ischemia in patients with coronary heart disease. Health Psychology, 14, 202-209.

Helmers, K. F. & Krantz, D. S. (1996). Defensive hostility, gender and cardiovascular level and responses to stress. Annals of Behavioral Medicine, 18, 246-254.

Holt, C. L. & Ellis, J. B. (1998). Assessing the current validity of the Bem Sex-Role Inventory. Sex Roles, 39, 929-941.

House, J. S., Robbins, C., & Metzner, H. L. (1982). The association of social relationships and activities with mortality: Prospective evidence from the Tecumeseh community health center. American Journal of Epidemiology, 116, 123-140.

Imber, S. I., Pilkonis, P. A., Sotsky, S. M., Elkin, I., Watkins, J. T., Collins, J. F., Shea, M. T., Leber, W. R., & Glass, D. R. (1990). Mode-specific effects among three treatments for depression. Journal of Consulting and Clinical Psychology, 58, 352-359.

Jamner, L. D., Shapiro, D., Goldstein, I. B., & Hug, R. (1991). Ambulatory blood pressure and heart rate in paramedics: Effects of cynical hostility and defensiveness. Psychosomatic Medicine, 53, 393-406.

Jorgensen, R. S., Johnson, B. T., Kolodziej, M. E., & Schreer, G. E. (1996). Elevated blood pressure and personality: A meta-analytic review. Psychological Bulletin, 120, 293-320.

Kamark, T., Manuck, S. B., & Jennings, R. (1990). Social support reduces cardiovascular reactivity to psychological challenge. Psychosomatic Medicine, 52, 42-58.

Kop, W. J. (1999). Chronic and acute psychological risk factors for clinical manifestation of coronary artery disease. Psychosomatic Medicine, 61, 476-487.

Korabik, K. & Van Kampen, J. (1995). Gender, social support, and coping with work stressors among managers. Journal of Social Behavior and Personality, 10, 135-148.

Kors, D. J. & Linden, W. (1999). Does social support reduce cardiovascular stress reactivity only if you want support: A test of a match/mismatch hypothesis. Unpublished doctoral dissertation, University of British Columbia, Vancouver.

Kors, D. J., Linden, W., & Gerin, W. (1997). Evaluation interferes with social support: Effects on cardiovascular stress reactivity in women. Journal of Social and Clinical Psychology, 16, 1-23.

Krantz, D. S., Kop, W. J., Santiago, H. T., & Gottdiener, J. S. (1996). Mental stress as a trigger of myocardial ischemia and infarction. Cardiology Clinics, 14, 271-287.

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.

Lash, S. J., Eisler, R. M., & Schulman, R. S. (1990). Cardiovascular reactivity to stress in men: Effects of masculine gender role stress appraisal and masculine performance challenge. Behavior Modification, 14, 3-20.

Lazarus, R. S. & Folkman, S. (1984). Stress, Appraisal, and Coping. New York, NY: Springer.

Levy, D., Wilson, W. F., Anderson, K. M., & Castelli, W. P. (1990). Stratifying the patient at risk from coronary disease: New insights from the Framingham Heart Study. American Heart Journal, 119, 712-717.

Lidderdale, J. M. & Walsh, J. J. (1998). The effects of social support on cardiovascular reactivity and perinatal outcome. Psychology and Health, 13, 1061-1070.

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

Linden, W., Paulhus, D. L., & Dobson, K. S. (1986). Effects of response styles on the report of psychological and somatic distress. Journal of Consulting and Clinical Psychology, 54, 309-313.

Linden, W. (1987). On the impending death of the Type A construct: or Is there a phoenix rising from the ashes? Canadian Journal of Behavioural Sciences, 19, 177-190.

Linden, W., Chambers, L., Maurice, J., & Lenz, J. W. (1993). Sex differences in social support, self-deception, hostility, and ambulatory cardiovascular activity. Health Psychology, 12, 376-380.

Linden, W. (2000). Hostility, defensiveness, and cardiovascular health. Unpublished manuscript, University of British Columbia.

Mann, S. J. & James, G. D. (1998). Defensiveness and essential hypertension. Journal of Psychosomatic Research, 45, 139-148.

Manuck, S. B., Kaplan, J. R., & Clarkson, T. B. (1983). Behaviorally induced heart rate reactivity and atherosclerosis in cynomolgus monkeys. Psychosomatic Medicine, 45, 95-108.

Manuck, S. B., Kaplan, J. R., Adams, M. R., & Clarkson, T. B. (1989). Behaviorally elicited heart rate reactivity and atherosclerosis in female cynomolgus monkeys (Macaca fascicularis). Psychological Medicine, 51, 306-318.

Manuck, S. B., Kasprowicz, A. L., & Muldoon, M. F. (1990). Behaviorally-evoked cardiovascular reactivity and hypertension: Conceptual issues and potential associations. Annals of Behavioral Medicine, 12, 17-29.

Manuck, S. B. (1995). Cardiovascular reactivity in cardiovascular disease: "Once more unto the breach". International Journal of Behavioral Medicine, 1, 4-31.

Markovitz, J. H., Raczynski, J. M., Wallace, D., Chettur, V., & Chesney, M. A. (1998). Cardiovascular reactivity to video game predicts subsequent blood pressure increases in young men: The CARDIA study. Psychosomatic Medicine, 60, 186-191.

McCrae, R. R. & Costa, P. T. Jr. (1983). Social desirability scales: More substance than style. Journal of Consulting and Clinical Psychology, 51, 882-888.

McCrae, R. R., Costa, P. T. Jr., Dahlstrom, W. G., Barefoot, J. C., Siegler, I. C., & Williams, R. B. (1989). A caution on the use of the MMPI K-correction in research on psychosomatic medicine. Psychosomatic Medicine, 51, 58-65.

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

Menkes, M. S., Matthew, K. A., Krantz, D. S., Lundberg, U., Mead, L. A., Qaqish, B., Liang, K., Thomas, C. B., & Pearson, T. A. (1989). Cardiovascular reactivity to the cold pressor test as a predictor of hypertension. Hypertension, 14, 524-530.

Mente, A. & Helmers, K. F. (1999). Defensive hostility and cardiovascular responses to stress in young men. Personality and Individual Differences, 27, 683-694.

Moore, R. G. & Blackburn, I. (1994). The relationship of sociotropy and autonomy to symptoms, cognition and personality in depressed patients. Journal of Affective Disorders, 32, 239-245.

Myrtek, M. (1995). Type A behavior pattern, personality factors, disease, and physiological reactivity: A meta-analytic update. Personality and Individual Differences, 18, 491-502.

Nelson, M. J., Ragland, D. R., & Syme, L. (1992). Longitudinal prediction of adult blood pressure from juvenile blood pressure levels. American Journal of Epidemiology, 136, 633-645.

Neter, J., Wasserman, W., & Kutner, M. H. (1990). Applied Linear Statistical Models. (3rd ed.) Burr Ridge, IL: Irwin.

Newman, J. D., McGarvey, S. T., & Steele, M. S. (1999). Longitudinal association of cardiovascular reactivity and blood pressure in Samoan adolescents. Psychosomatic Medicine, 61, 243-249.

Nezu, A. M., Nezu, C. M., & Peterson, M. A. (1986). Negative life stress, social support, and depressive symptoms: Sex roles as a moderator variable. Journal of Social Behavior and Personality, 1, 599-609.

Orth-Gomer, K. & Unden, A. (1990). Type A behavior, social support, and coronary risk: Interaction and significance for mortality in cardiac patients. Psychosomatic Medicine, 52, 59-72.

Orth-Gomer, K., Rosengren, A., & Wilhelmsen, L. (1993). Lack of social support and incidence of coronary heart disease in middle-aged Swedish men. Psychosomatic Medicine, 55, 37-43.

Orth-Gomer, K., Horsten, M., Wamala, S. P., Mittleman, M. A., Kirkeeide, R., Svane, B., & Schenck-Gustafsson, K. (1998). Social relations and extent and severity of coronary artery disease. European Heart Journal, 19, 1648-4656.

Parker, F. C., Croft, J. B., Cresanta, J. L., Freedman, D. S., Burke, G. L., Webber, L. S., & Berenson, G. S. (1987). The association between cardiovascular response tasks and future blood pressure levels in children: Bogalusa Heart Study. American Heart Journal, 113, 1174-1179.

Paulhus, D. L. (1984). Two-component models of socially desirable responding. Journal of Personality and Social Psychology, 46, 598-606.

Paulhus, D. L. (1991). BIDR Reference Manual for Version 6. Unpublished manuscript, University of British Columbia.

Paulhus, D. L. (1998). Interpersonal and intrapsychic adaptiveness of trait self-enhancement: A mixed blessing? Journal of Personality and Social Psychology, 74, 1197-1208.

Pennix, B. W. J. H., Kriegsman, D. M. W., van Eijk, J. T. M., Boeke, A. J. P., & Deeg, D. J. H. (1996). Differential effect of social support on the course of chronic disease: A criteria-based literature study. Families, Systems & Health, 14, 223-244.

Pennix, B. W. J. H., van Tilburg, T., Kriegsman, D. M. W., Deeg, D. J. H., Boeke, A. J. P., & van Eijk, J. T. M. (1997). Effects of social support and personal coping resources on mortality in older age: The longitudinal aging study of Amsterdam. American Journal of Epidemiology, 146, 510-519.

Persons, J. B., Miranda, J., & Perloff, J. M. (1991). Relationships between depressive symptoms and cognitive vulnerabilities of achievement and dependency. Cognitive Therapy and Research, 15, 221-235.

Roos, P. E. & Cohen, L. H. (1987). Sex roles and social support as moderators of life stress adjustment. Journal of Personality and Social Psychology, 52, 576-585.

Rosenman, R. H., Brand, R. J., Jenkins, C. D., Friedman, H. S., Strauss, R., & Wurm, M. (1975). Coronary heart disease in the Western Collaborative Group Study: Final follow-up experience of 8 1/2 years. Journal of the American Medical Association, 223, 872-877.

Roy, M. P., Steptoe, A., & Kirschbaum, C. (1998). Life events and social support as moderators of individual differences in cardiovascular and cortisol reactivity. Journal of Personality and Social Psychology, 75, 1273-1281.

Rutledge, T., Linden, W., & Davies, R. F. (1999). Psychological risk factors may moderate pharmacological treatment effects among ischemic heart disease patients. Psychological Medicine, 61, 834-841.

Rutledge, T. & Linden, W. (2000). Defensiveness status predicts 3-year incidence of hypertension. Journal of Hypertension, 18, 1-7.

Rutledge, T., Linden, W., & Davies, R. F. (in press). Psychological response styles and cardiovascular health: Confound or independent risk factor? Health Psychology.

Sarafino, E. P. (1994). Health Psychology: Biopsychosocial Interactions (2nd ed.). New York, NY: John Wiley & Sons.

Sarason, I. G., Sarason, B. R., & Shearin, E. N. (1986). Social support as an individual difference variable: Its stability, origins, and relational aspects. Journal of Personality and Social Psychology, 50, 845-855.

Schwarzer, R. & Leppin, A. (1991). Social support and health: A theoretical and empirical overview. Journal of Social and Personal Relationships, 8, 99-127.

Shapiro, D., Goldstein, I. B., & Jamner, L. D. (1995). Effects of anger/hostility, defensiveness, gender, and family history of hypertension on cardiovascular reactivity. Psychophysiology, 32, 425-435.

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

Shifren, K., Bauserman, R., & Carter, D. B. (1993). Gender role orientation and physical health: A study among young adults. Sex Roles, 29, 421-432.

Siegman, A. W. (1994a). From type A to hostility to anger: Reflections on the history of coronary-prone behavior. In A.W.Siegman & T. W. Smith (Eds.), Anger, Hostility, and the Heart (pp. 1-21). Hillsdale, NJ: Lawrence Erlbaum Associates.

Siegman, A. W. (1994b). Cardiovascular consequences of expressing and repressing anger. In A.W.Siegman & T. W. Smith (Eds.), Anger, Hostility, and the Heart (pp. 173-197). Hillsdale, NJ: Lawrence Erlbaum Associates.

Silten Bassoff, S. & Glass, G. V. (1982). The relationship between sex roles and mental health: A meta-analysis of twenty-six studies. The Counseling Psychologist, 10, 105-112.

Smith, M. A. & Houston, B. K. (1987). Hostility, anger expression, cardiovascular responsivity, and social support. Biological Psychology, 24, 39-48.

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, 503-520.

Smith, T. W. & Pope, M. K. (1990). Cynical hostility as a health risk: Current status and future directions. Journal of Social Behavior and Personality, 5, 77-88.

Spitzer, S. B., Llabre, M. M., Ironson, G. H., Gellman, M. D., & Schneiderman, N. (1992). The influence of social situations on ambulatory blood pressure. Psychosomatic Medicine, 54, 79-86.

Steptoe, A., Melville, D., & Ross, A. (1984). Behavioral response demands, cardiovascular reactivity, and essential hypertension. Psychosomatic Medicine, 46, 33-48.

Strogatz, D. S. & James, S. A. (1986). Social support and hypertension among Blacks and Whites in a rural, southern community. American Journal of Epidemiology, 124, 949-956.

Suarez, E. C., Harlan, E., Peoples, M. C., & Williams, R. B. (1993). Cardiovascular and emotional responses in women: The role of hostility and harassment. Health Psychology, 12, 459-468.

Suls, J. & Wan, C. K. (1993). The relationship between trait hostility and cardiovascular reactivity: A quantitative review and analysis. Psychophysiology, 30, 615-626.

Suls, J., Wan, C. K., & Costa, P. T. Jr. (1995). Relationship of trait anger to resting blood pressure: A meta-analysis. Health Psychology, 14, 444-456.

Tabachnick, B. G. & Fidell, L. S. (1996). Using Multivariate Statistics. (3rd ed.). New York, NY: Harper Collins College Publishers.

Uchino, B. N., Kiecolt-Glaser, J. K., & Cacioppo, J. T. (1992). Age-related changes in cardiovascular response as a function of a chronic stressor and social support. Journal of Personality and Social Psychology, 63, 839-846.

Uchino, B. N., Cacioppo, J. T., Malarkey, W., Glaser, R., & Kiecolt-Glaser, J. K. (1995). Appraisal support predicts age-related differences in cardiovascular function in women. Health Psychology, 14, 556-562.

Uchino, B. N., Cacioppo, J. T., & Kiecolt-Glaser, J. K. (1996). The relationship between social support and physiological processes: A review with emphasis on underlying mechanisms and implications for health. Psychological Bulletin, 119, 488-531.

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

Yasuda, N., Zimmerman, S. I., Hawkes, W., Fredman, L., Hebel, J. R., & Magaziner, J. (1997). Relation of social network characteristics to 5-year mortality among young-old versus old-old white women in an urban community. American Journal of Epidemiology, 145, 516-523.

Zuroff, D. C. (1994). Depressive personality styles and the five-factor model of personality. Journal of Personality Assessment, 63, 453-472.

Table 1

Mean Baseline, Task, and Change Values

Measurement	Males	Females	Overall
Baseline			
SBP	118.84 (7.89)	109.84 (7.28)	114.31 (8.81)
DBP	66.08 (7.97)	65.30 (5.80)	65.69 (6.95)
HR	69.03 (11.90)	68.92 (10.55)	68.97 (11.21)
Task			
SBP	132.12 (12.19)	123.17 (11.26)	127.60 (12.53)
DBP	79.30 (9.27)	77.76 (7.61)	78.52 (6.95)
HR	86.00 (15.00)	88.85 (16.07)	87.44 (15.57)
Raw Change			
SBP	16.31 (10.31)	15.78 (8.36)	16.04 (9.35)
DBP	14.65 (7.02)	13.49 (5.35)	14.06 (6.24)
HR	18.45 (11.44)	20.64 (13.14)	19.56 (12.34)

Note. Values enclosed in parentheses are standard deviations. SBP = systolic blood pressure; DBP = diastolic blood pressure; HR = heart rate.

Table 2

Mean Personality Scores

Variable	Overall	Males	Females	t[a]
Hostility	68.76 (15.55)	91.14 (15.84)	82.43 (14.05)	3.83***
Negative Affectivity	30.43 (8.57)	31.58 (9.38)	29.30 (7.56)	1.76
Social Inhibition	30.69 (8.90)	31.78 (9.47)	29.62 (8.21)	1.60
Self-deception	4.28 (2.59)	4.41 (2.71)	4.15 (2.47)	.65
Impression Management	5.09 (3.24)	4.84 (2.92)	5.34 (3.53)	-1.03
Sociotropy	38.63 (10.79)	39.55 (10.57)	37.71 (10.99)	1.13
Social support	32.72 (5.91)	31.56 (6.86)	33.86 (4.52)	-2.60**
Support Seeking	23.56 (5.77)	22.01 (5.70)	25.09 (5.46)	-3.63***
Masculinity	4.71 (.70)	4.85 (.73)	4.57 (.64)	2.68**
Femininity	4.79 (.57)	4.70 (.54)	4.89 (.59)	-2.24*

Note. Values enclosed in parentheses represent standard deviations.
[a] T-test reported test for gender differences.
*$p < .05$. **$p < .01$. ***$p < .001$.

Table 3

Intercorrelations Between Personality Variables for Males (n = 86) / Females (n = 87)

Variable	1	2	3	4	5	6	7	8	9	10
1. Hostility	-- --	.41**/ .25**	.33**/ .21	-.01/ -.09	-.34**/ -.15	.30**/ .33**	-.42**/ -.26*	-.08/ -.15	.00/ .03	-.02/ -.09
2. Negative Affectivity		-- --	.71**/ .40**	-.37**/ -.42**	-.25*/ .09	.54**/ .46**	-.69**/ -.42**	-.15/ -.21	-.50**/ -.30**	-.02/ -.21
3. Social Inhibition			-- --	-.42**/ -.19	-.16/ .03	.39**/ .05	-.66**/ -.30**	-.19/ -.39**	-.58**/ -.28**	-.03/ -.21*
4. Self-deception				-- --	.24*/ .22*	-.34**/ -.36**	.24*/ .15	.05/ .24*	.55**/ .37**	.09/ .15
5. Impression Management					-- --	-.27*/ .06	.23*/ -.20	.10/ .03	.06/ -.24*	.18/ .14
6. Sociotropy						-- --	-.38**/ -.36*	-.13/ -.13	-.35**/ -.27*	-.02/ .01
7. Social Support							-- --	.16/ .35**	.56**/ .27*	.10/ .21
8. Support Seeking								-- --	.18/ -.01	.32**/ .36**
9. Masculinity									-- --	.07/ -.01
10. Femininity										-- --

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

Table 4

Summary of Hierarchical Regression Analysis for Buffering Variables Predicting Baseline HR (N = 169)

Variable	B	SE B	β
Step 1			
Gender	-.36	1.72	-.02
Age	-.10	.16	-.05
Family History of Hypertension	3.61	1.72	.16*
Nicotine Intake	.52	2.96	.01
Caffeine Intake	3.56	1.84	.15
Step 2			
Gender	-2.22	1.84	-.10
Age	-.00	.16	-.02
Family History of Hypertension	2.84	1.72	.13
Nicotine Intake	-.11	2.98	-.00
Caffeine Intake	3.18	1.82	.14
Perceived Social Support	.20	.17	.11
Support Seeking	.26	.17	.13
Masculinity	-4.21	1.41	-.26**
Femininity	-2.19	1.65	-.11

Table 4 (continued)

Summary of Hierarchical Regression Analysis for Buffering Variables Predicting Baseline HR (N = 169)

Variable	B	SE B	β
Step 3			
Gender	-2.40	1.86	-.11
Age	-.00	.16	-.01
Family History of Hypertension	2.45	1.74	.11
Nicotine Intake	.26	3.02	.01
Caffeine Intake	2.65	1.86	.11
Perceived Social Support	.29	.18	.15
Support Seeking	.26	.17	.14
Masculinity	-4.18	1.43	-.26**
Femininity	-2.40	1.66	-.12
Perceived Support x Gender	.13	.36	.04
Support Seeking x Gender	.00	.33	-.01
Masculinity x Gender	3.53	2.88	.11
Femininity x Gender	2.18	3.21	.06

Note. R^2 = .045 for Step 1; ΔR^2 = .065 for Step 2 ($p < .05$); ΔR^2 = .017 for Step 3. HR = heart rate.
*$p < .05$. **$p < .01$.

Table 5

Summary of Hierarchical Regression Analysis for Vulnerability Variables Predicting Baseline SBP (N = 169)

Variable	B	SE B	β
Step 1			
Gender	-8.22	1.01	-.48***
Age	.29	.11	.18**
Family History of Hypertension	1.92	1.10	.11
Nicotine Intake	-3.49	1.89	-.12
Caffeine Intake	2.76	1.16	.16*
Step 2			
Gender	-9.13	1.14	-.54***
Age	.19	.11	.18
Family History of Hypertension	1.907	1.10	.11
Nicotine Intake	-3.00	1.92	-.10
Caffeine Intake	2.63	1.17	.15*
Hostility	-.10	.04	-.18*
Negative Affectivity	-.01	.09	-.01
Social Inhibition	.05	.08	.05
Self-deception	-.16	.25	-.05
Impression Management	-.18	.18	-.05

Table 5 (continued)

Summary of Hierarchical Regression Analysis for Vulnerability Variables Predicting Baseline SBP (N = 169)

Variable	B	SE B	β
Sociotropy	-.07	.06	-.09
Step 3			
Gender	-9.28	1.14	-.54***
Age	.22	.12	.14
Family History of Hypertension	1.71	1.11	.10
Nicotine Intake	-2.46	1.97	-.08
Caffeine Intake	2.32	1.17	.13*
Hostility	-.10	.04	-.19*
Negative Affectivity	-.06	.09	-.06
Social Inhibition	.07	.08	.07
Self-deception	-.15	.26	-.05
Impression Management	-.22	.19	-.08
Sociotropy	-.09	.06	-.12
Hostility x Gender	.02	.08	.01
Negative Affectivity x Gender	.01	.19	.00
Social Inhibition x Gender	-.25	.16	-.13
Self-deception x Gender	-.25	.51	-.04

Table 5 (continued)

Summary of Hierarchical Regression Analysis for Vulnerability Variables Predicting Baseline SBP (N = 169)

Variable	B	SE B	β
Impression Management x Gender	.82	.38	.16*
Sociotropy x Gender	.08	.12	.05

Note. R^2 = .336 for Step 1 ($p < .001$); ΔR^2 = .038 for Step 2; ΔR^2 = .031 for Step 3. SBP = systolic blood pressure.
*$p < .05$. **$p < .01$. ***$p < .001$.

Table 6

Summary of Hierarchical Regression Analysis for Vulnerability Variables Predicting Baseline DBP (N = 169)

Variable	B	SE B	β
Step 1			
Gender	-.21	1.01	-.02
Age	.34	.10	.26**
Family History of Hypertension	1.99	1.01	.14
Nicotine Intake	-3.09	1.74	-.13
Caffeine Intake	2.35	1.08	.16*
Step 2			
Gender	-.96	1.05	-.07
Age	.24	.11	.18*
Family History of Hypertension	2.26	1.01	.16*
Nicotine Intake	-2.72	1.77	-.12
Caffeine Intake	2.60	1.08	.18*
Hostility	-.10	.04	-.22*
Negative Affectivity	.08	.08	.10
Social Inhibition	.05	.07	.07
Self-deception	.03	.23	.01
Impression Management	.21	.17	.10

Table 6 (continued)

Summary of Hierarchical Regression Analysis for Vulnerability Variables Predicting Baseline DBP (N = 169)

Variable	B	SE B	β
Sociotropy	-.01	.06	-.02
Step 3			
Gender	-1.02	1.07	-.07
Age	.26	.11	.20*
Family History of Hypertension	2.14	1.04	.16*
Nicotine Intake	-2.27	1.84	-.10
Caffeine Intake	2.44	1.10	.17*
Hostility	-.10	.04	-.22*
Negative Affectivity	.07	.09	.09
Social Inhibition	.04	.08	.06
Self-deception	-.01	.24	-.01
Impression Management	.18	.18	.09
Sociotropy	-.02	.06	-.04
Hostility x Gender	.06	.08	.06
Negative Affectivity x Gender	-.13	.17	-.08
Social Inhibition x Gender	.03	.15	.02
Self-deception x Gender	-.18	.48	-.03

Table 6 (continued)

Summary of Hierarchical Regression Analysis for Vulnerability Variables Predicting Baseline DBP (N = 169)

Variable	B	SE B	β
Impression Management x Gender	.44	.35	.10
Sociotropy x Gender	-.02	.06	-.04

Note. R^2 = .138 for Step 1 ($p < .001$); ΔR^2 = .054 for Step 2; ΔR^2 = .012 for Step 3. DBP = diastolic blood pressure.
*$p < .05$. **$p < .01$.

Table 7

Summary of Hierarchical Regression Analysis for Vulnerability Variables Predicting SBP Reactivity (N = 168)

Variable	B	SE B	β
Step 1			
Gender	-.44	1.46	-.02
Age	.09	.14	.05
Family History of Hypertension	.39	1.47	.02
Nicotine Intake	-.527	2.52	-.17*
Caffeine Intake	.85	1.57	.04
Step 2			
Gender	-.45	1.43	-.02
Age	.15	.14	.09
Family History of Hypertension	..40	1.43	.02
Nicotine Intake	-4.60	2.47	-.14
Caffeine Intake	.66	1.54	.03
Task	4.79	1.67	.22**
Step 3			
Gender	-.09	1.50	-.01
Age	.11	.15	.06
Family History of Hypertension	.60	.144	.03
Nicotine Intake	-3.79	2.54	-.12

Table 7 (continued)

Summary of Hierarchical Regression Analysis for Vulnerability Variables Predicting SBP Reactivity (N = 168)

Variable	B	SE B	β
Caffeine Intake	.71	1.54	.04
Task	5.15	1.74	.24**
Hostility	.02	.06	.04
Negative Affectivity	.01	.12	.01
Social Inhibition	.18	.10	.17
Self-deception	-.07	.34	-.02
Impression Management	-.05	.24	-.02
Sociotropy	-.13	.08	-.15
Step 4			
Gender	.00	1.50	.00
Age	.14	.15	.08
Family History of Hypertension	.18	1.16	.01
Nicotine Intake	-3.83	2.60	-.12
Caffeine Intake	.62	1.54	.03
Task	5.55	1.74	.26**
Hostility	.03	.06	.06
Negative Affectivity	-.01	.12	-.01
Social Inhibition	.19	.11	.18

Table 7 (continued)

Summary of Hierarchical Regression Analysis for Vulnerability Variables Predicting SBP Reactivity (N = 168)

Variable	B	SE B	β
Self-deception	.01	.35	.00
Impression Management	-.05	.25	-.02
Sociotropy	-.13	.08	-.15
Hostility x Gender	.01	.11	.01
Negative Affectivity x Gender	.50	.25	.23*
Social Inhibition x Gender	-.48	.21	-.23*
Self-deception x Gender	.24	.68	.03
Impression Management x Gender	-.11	.50	-.02
Sociotropy x Gender	-.24	.16	-.14

Note. R^2 = .031 for Step 1; ΔR^2 = .047 for Step 2 ($p < .01$); ΔR^2 = .041 for Step 3; ΔR^2 = .045 for Step 4. SBP = systolic blood pressure.
*$p < .05$. **$p < .01$.

Table 8

Summary of Hierarchical Regression Analysis for Vulnerability Variables Predicting DBP Reactivity (N = 168)

Variable	B	SE B	β
Step 1			
Gender	-1.16	.96	-.09
Age	-.02	.09	-.01
Family History of Hypertension	.44	.97	.04
Nicotine Intake	-2.30	1.66	-.11
Caffeine Intake	1.39	1.03	.11
Step 2			
Gender	-1.17	.94	-.09
Age	.03	.09	.03
Family History of Hypertension	.45	.94	.04
Nicotine Intake	-1.80	1.62	-.09
Caffeine Intake	1.25	1.01	.10
Task	3.57	1.09	.25**
Step 3			
Gender	-1.44	.99	-.12
Age	-.03	.10	-.02
Family History of Hypertension	.53	.95	.04
Nicotine Intake	-.10	1.67	-.05

Table 8 (continued)

Summary of Hierarchical Regression Analysis for Vulnerability Variables Predicting DBP Reactivity (N = 168)

Variable	B	SE B	β
Caffeine Intake	1.25	1.01	.10
Task	4.11	1.14	.29***
Hostility	-.01	.04	-.02
Negative Affectivity	.00	.08	.00
Social Inhibition	.05	.07	.07
Self-deception	-.19	.22	-.08
Impression Management	.08	.16	.04
Sociotropy	-.11	.05	-.19*
Step 4			
Gender	-1.51	.99	-.12
Age	.00	.10	.00
Family History of Hypertension	.24	.96	.02
Nicotine Intake	-.59	1.72	-.03
Caffeine Intake	1.08	1.02	.08
Task	4.37	1.15	.31***
Hostility	.00	.04	-.01
Negative Affectivity	-.04	.08	-.05
Social Inhibition	.06	.07	.08

Table 8 (continued)

Summary of Hierarchical Regression Analysis for Vulnerability Variables Predicting DBP Reactivity (N = 168)

Variable	B	SE B	β
Self-deception	-.17	.23	-.07
Impression Management	.05	.17	.03
Sociotropy	-.12	.06	-.21*
Hostility x Gender	-.01	.07	-.01
Negative Affectivity x Gender	.16	.16	.11
Social Inhibition x Gender	-.31	.14	-.22*
Self-deception x Gender	-.05	.45	-.01
Impression Management x Gender	.32	.33	.08
Sociotropy x Gender	-.06	.11	-.05

Note. R^2 = .030 for Step 1; ΔR^2 = .060 for Step 2 ($p < .01$); ΔR^2 = .031 for Step 3; ΔR^2 = .039 for Step 4. DBP = diastolic blood pressure.
*$p < .05$. **$p < .01$. ***$p < .001$

Table 9

Summary of Hierarchical Regression Analysis for Vulnerability Variables Predicting HR Reactivity (N = 168)

Variable	B	SE B	β
Step 1			
Gender	2.38	1.90	.10
Age	-.33	.18	-.14
Family History of Hypertension	-1.20	1.90	-.05
Nicotine Intake	-3.76	3.27	-.09
Caffeine Intake	1.48	2.04	.06
Step 2			
Gender	2.37	1.85	.10
Age	-.25	.18	-.11
Family History of Hypertension	-1.19	1.86	-.05
Nicotine Intake	-2.83	3.20	-.07
Caffeine Intake	1.23	1.99	.05
Task	6.59	2.16	.23**
Step 3			
Gender	1.77	1.94	.07
Age	-.39	.19	-.17*
Family History of Hypertension	-1.03	1.86	-.04
Nicotine Intake	-1.19	3.28	-.03

Table 9 (continued)

Summary of Hierarchical Regression Analysis for Vulnerability Variables Predicting HR Reactivity (N = 168)

Variable	B	SE B	β
Caffeine Intake	1.13	1.99	.04
Task	7.80	2.24	.26**
Hostility	-.03	.07	.04
Negative Affectivity	.00	.15	.00
Social Inhibition	.13	.13	.10
Self-deception	-.29	.44	-.06
Impression Management	.01	.31	.00
Sociotropy	-.26	.11	-.23*
Step 4			
Gender	2.17	1.94	.09
Age	-.37	.20	-.16
Family History of Hypertension	-1.15	1.89	-.05
Nicotine Intake	-2.35	3.37	-.06
Caffeine Intake	1.23	2.00	.05
Task	7.93	2.25	.28**
Hostility	-.02	.07	-.02
Negative Affectivity	.02	.16	.01
Social Inhibition	.17	.14	.13

Table 9 (continued)

Summary of Hierarchical Regression Analysis for Vulnerability Variables Predicting HR Reactivity (N = 168)

Variable	B	SE B	β
Self-deception	-.11	.45	-.02
Impression Management	-.05	.25	-.02
Sociotropy	-.03	.32	-.01
Hostility x Gender	.06	.14	.04
Negative Affectivity x Gender	.77	.32	.27*
Social Inhibition x Gender	-.38	.28	-.14
Self-deception x Gender	.24	.88	.03
Impression Management x Gender	-.38	.64	-.05
Sociotropy x Gender	-.26	.21	-.12

Note. R^2 = .043 for Step 1; ΔR^2 = .052 for Step 2 ($p < .01$); ΔR^2 = .045 for Step 3; ΔR^2 = .040 for Step 4. HR = heart rate.
*$p < .05$. **$p < .01$.

Table 10:

Summary of Results

	Males		Females	
Variable	Baseline	Reactivity	Baseline	Reactivity
Psychosocial Vulnerability				
Hostility	-	0	-	0
Social Inhibition	0	+	0	0
Negative Affectivity	0	0	0	0
Self-deception	0	0	0	0
Impression Management	-	0	+	0
Sociotropy	0	-	0	-
Psychosocial Buffer				
Perceived Support	0	0	0	0
Support Seeking	0	-	0	0
Masculinity	-	0	-	0
Femininity	0	0	0	0

Note. + = positive association; - = negative association; 0 = no association.

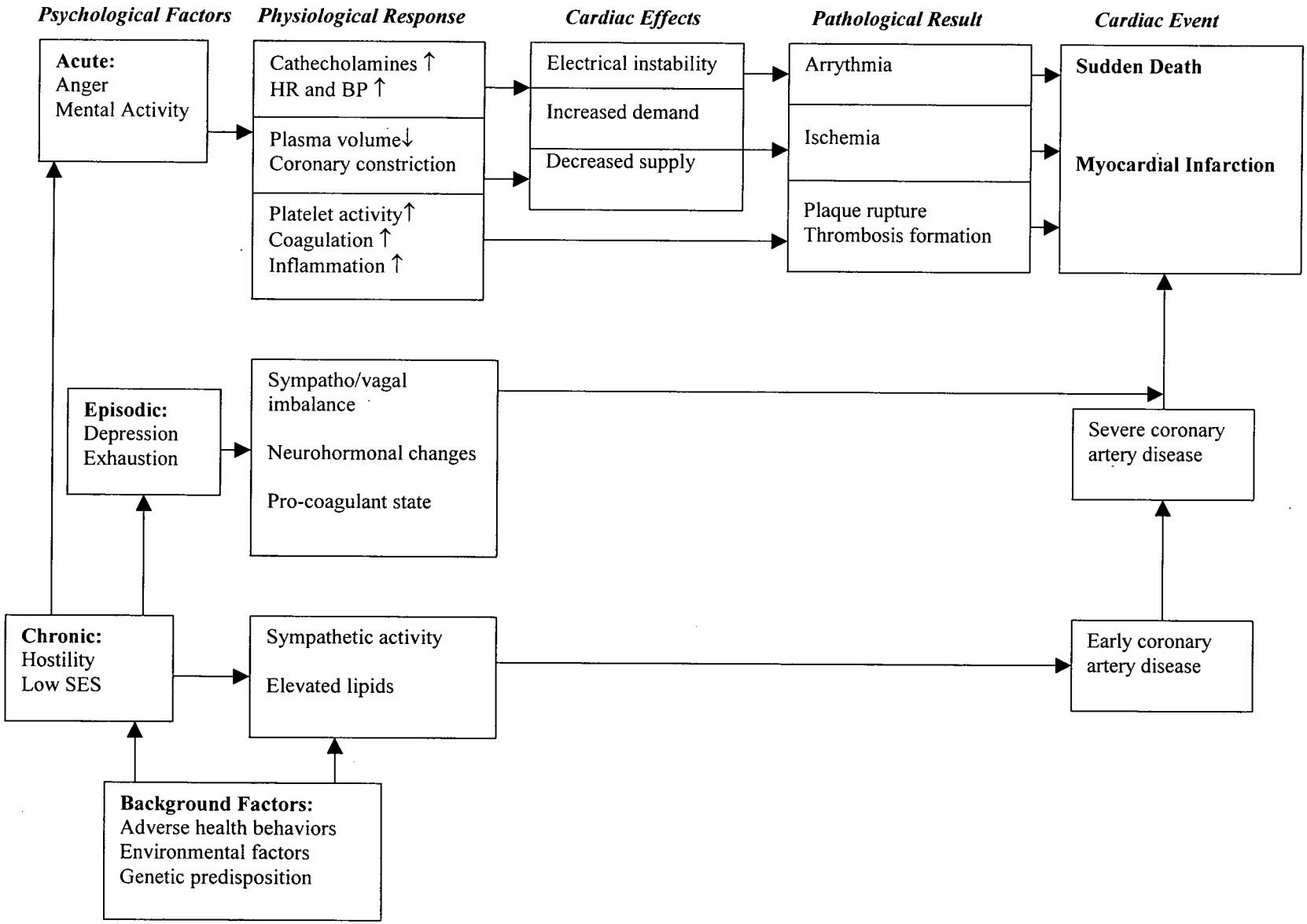

Figure 1. Pathophysiological model of the relationship between chronic, episodic, and acute psychological risk factors for coronary syndromes. From W. Kop, 1999, Psychosomatic Medicine, 61, p.477.