Defensive Hostility:
Its role in Cardiovascular Reactivity and Health-Risk Behaviours

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Abstract

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The present study examined the relationship between defensive hostility, cardiovascular responses to interpersonal stress and health-risk behaviours associated with CHD pathogenesis. Ninety-six healthy male university students, aged 18-30 years, were classified as either high or low hostile and high or low defensive by median split scores on the Cook Medley Hostility Scale and Marlowe Crowne Social Desirability Scale. They were then partitioned into four groups: Defensive Hostile (HiDef/HiHo), Low Defensive/High Hostile (LoDef/HiHo), High Defensive/Low Hostile (HiDef/LoHo) and Low Defensive/Low Hostile (LoDef/LoHo). For the examination of laboratory cardiovascular reactivity [systolic blood pressure (SBP), diastolic blood pressure (DBP), heart rate (HR) cardiac output (CO), and stroke volume (SV)], subjects were randomly assigned to either a harassment or non-harassment condition immediately before engaging in a 9-minute subtraction math-task. Negative emotional reactions to harassment were also assessed. For the analysis of health-risk behaviours, subjects completed a General Health Survey and monitored their intake of calories, saturated fat, total fat, cholesterol, alcohol, caffeine and cigarettes on one weekday and one weekend-day. The results offered no conclusive support for the hypotheses that the Defensive Hostile group would exhibit significantly greater cardiovascular reactivity under harassment and report increased engagement in health-risk behaviours relative to others. Instead, the LoDef/LoHo group showed the most elevated SBP reactivity under

harassment, although no strong conclusion can be made due to their limited sample size. The harassed LoDef/LoHo group also displayed greater SBP and SV responses while the harassed Defensive Hostile group displayed greater SBP reactivity relative to their own non-harassed counterparts. No group differences in negative emotional reactions to harassment were observed. The groups also did not differ in any of the health-risk behaviours although a positive association was found between hostility and cholesterol intake. A negative association between defensiveness and beer intake with trend results for general alcohol consumption were also obtained. These study findings suggest that further research is needed to examine the moderating influence of interpersonal stress, such as harassment, not only in the relationship between defensive hostility and cardiovascular reactivity but between low levels of both defensiveness and hostility and cardiovascular responses as well.

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For many years researchers have focused their attention on identifying risk factors for coronary heart disease (CHD), the leading cause of death in industrialized countries such as Canada (Heart and Stroke Foundation of Canada, 2001) and the United States (Anderson, 2002). CHD is defined as a group of related syndromes, such as myocardial ischemia and angina pectoris, which result from an inadequate supply of blood to the heart (Cotran, Kumar & Robbins, 1989). Although a number of traditional risk factors have been implicated, including hypertension, elevated serum cholesterol, smoking, obesity, and parental history of heart disease (Siegman, 1994), epidemiological data reveal that they account for no more than half of the new disease cases (Jenkins, 1978; Keys, 1970; Rosenman, 1983). The suggestion that additional risk factors may be involved has, subsequently, led researchers to explore the role of different psychosocial influences in CHD etiology. The well-known Type A Behaviour Pattern, as well as one of its components, hostility, have been the most widely studied. In light of some inconsistent research findings, however, several researchers have more recently examined the combined influence of hostility and defensiveness, referred to as defensive hostility, in the study of this life-threatening disease. It has been suggested that the addition of defensiveness may help explain conflicting results in the hostility literature by identifying a subset of individuals most at risk for disease development.

In the literature review to follow, this progression in interest from Type A behaviour to hostility to defensive hostility will be outlined. Studies examining the Type A-CHD and hostility-CHD relationships will be briefly reviewed. Conceptual and measurement issues with respect to the hostility construct will be presented as well. Two

proposed mechanisms linking hostility to CHD are heightened cardiovascular reactivity to stress and engagement in unhealthy lifestyle behaviours, such as smoking, alcohol and cholesterol consumption, to name a few. Research on the association between hostility and cardiovascular reactivity, as well as hostility and health-risk behaviours will subsequently follow. Finally, focus will then be given to the defensiveness construct and the studies relating defensive hostility to CHD and to cardiovascular reactivity in order to provide the rationale for the present study on the relationship between defensive hostility, cardiovascular reactivity to interpersonal stress and health-risk behaviours.

Type A Behaviour Pattern and Coronary Heart Disease

The Type A Behaviour Pattern (TABP) was first conceptualized by two cardiologists, Friedman and Rosenman, in the 1950s. They used this broad label to describe a group of attributes including intense ambition, competitiveness, hard-driving job involvement, impatience, time urgency, hostility and aggressiveness, that were hypothesized to play a role in the development of CHD (Friedman & Rosenman, 1974). The Type A person referred to anyone "aggressively involved in a chronic, incessant struggle to achieve more and more in less and less time, and if required to do so, against the opposing efforts of the other things or other persons" (Friedman & Rosenman, p. 37).

The first evidence that TABP was predictive of CHD emerged from findings of the Western Collaborative Group Study (WCGS), a large-scaled prospective study following over 3000 men for 8 1/2 years (Rosenman et al. 1964). At follow-up, Type A male subjects were twice as likely to manifest symptoms of CHD as Type B subjects, described as lacking Type A traits (Rosenman et al. 1964; Rosenman et al. 1975). These

findings were further strengthened by studies showing that TABP was associated with severity of angiographically-documented coronary artery disease (CAD) (Blumenthal, Williams, Kong, Schanberg & Thompson, 1978; Frank, Heller, Kornfeld, Sport & Weiss, 1978; Friedman, Rosenman, Strauss, Wurm & Kositchek, 1968; Williams et al. 1980).

As TABP was becoming recognized as a viable risk factor for CHD, the emergence of contradictory findings challenged the validity of the Type A-CHD relationship. Results from the Multiple Risk Factor Intervention Trial (MRFIT), a large-scale prospective study of over 3000 men, were particularly damaging. The MRFIT study was unable to replicate a positive association between TABP and CHD mortality at a 7-year follow-up (Shekelle et al. 1985). TABP also failed to predict severity of coronary occlusion in a number of angiographic studies (Arrowood, Uhrick, Gomillion, Popio, & Raft, 1982; Dembroski, MacDougall, Williams, Haney, & Blumenthal, 1985; Dimsdale, Hackett & Hunter, 1979; Krantz, Sanmarco, Selvester & Matthews, 1979; Scherwitz et al. 1983; Siegman, Feldstein, Tommaso, Ringel & Lating, 1987). Moreover, when the WCGS data were reanalysed using appropriate statistical controls, the original claims of a Type A behaviour-CHD mortality link were no longer substantiated (Ragland, Brand & Rosenman, 1987). Although Williams et al. (1988) found a significant association between TABP and CAD, the link was found only among younger patients and was not deemed very strong.

With the accumulation of discrepant findings, many researchers shifted their focus from the multidimensional construct of Type A behaviour to its separate components in an attempt to identify specific coronary-prone or "toxic" ones. Hostility was singled out among them.

Defining hostility

Before the potential influence of hostility in CHD etiology can be examined, conceptual issues must be addressed. An adequate definition and valid means to assess the construct of *hostility* are needed. Unfortunately, there has been confusion over its conceptualization, particularly in regard to interrelated constructs such as *anger* and *aggression*. These three terms have often been used interchangeably in the behavioural medicine literature (Helmers, Posluszny & Krantz, 1994).

Many researchers (Houston, 1994; T. Q. Miller, Smith, Turner, Guijarro & Hallet, 1996; Smith, 1992; 1994) have relied on distinctions made between affect, cognition and behaviour (Buss, 1961; Spielberger et al. 1985) to differentiate between anger, hostility and aggression. In accordance, a*nger* has been described in affective terms as an unpleasant emotion ranging in intensity from mild irritation to rage. As such, it can present as a transitory state or an enduring trait characterized by a proclivity to experience repeated and intense episodes of state anger (Smith, 1994). A*ggression* has been defined in terms of behavioural responses such as attacking, destructive or hurtful actions that can be expressed both verbally and physically (T. Q. Miller et al. 1996). *Hostility* differs from both anger and aggression in that it has been viewed as a multifaceted cognitive phenomenon consisting of enduring negative beliefs and attitudes toward others, such as *cynicism and mistrust*. *Cynicism* refers to the belief that selfishness rather than genuine concern motivates others and *mistrust* is the expectation that others are sources of mistreatment and harm (Smith, 1994). According to Smith, hostility is defined as an "enduring, general trait that connotes a devaluation of the worth and motives of others, an expectation that others are likely sources of wrongdoing, a

relational view of being in opposition toward others, and a desire to inflict harm or see others harmed" (p. 26).

Other prominent researchers have described hostility in broader terms and contend that it encompasses aspects of affect, cognition and behaviour (Barefoot & Lipkus, 1994; Barefoot, Dodge, Peterson, Dahlstrom & Williams, 1989). Chaplin (1982), for example, defined hostility as a tendency to feel anger towards others or the tendency to inflict harm on others, thus identifying both feelings and behaviours. Moreover, in his widely cited article, Barefoot (1992) noted that hostility has affective, cognitive and behavioural components, although any one component can occur in the absence of the other two.

Undoubtedly, confusion over definitions remains an issue as researchers continue to explore the role of hostility in different lines of research. Whether it is defined in cognitive terms, or more vaguely to include aspects of anger and aggressiveness, the suggestion that hostility, like TABP, is itself a complex, multidimensional construct, seems to have been largely accepted in the field (Barefoot & Lipkus, 1994; Brummett et al. 1998; Costa, Zonderman, McCrae & Williams, 1986; Siegman, 1994; Suls & Wan, 1993). Not surprisingly, such complexity is problematic when efforts are made to measure this construct to determine its impact on coronary disease processes.

Measuring hostility

The two most commonly used methods to assess hostility are the Cook Medley Hostility Scale (Ho Scale; Cook & Medley, 1954), a self-report questionnaire, and the Structured Interview (SI), developed by Friedman and Rosenman (1959). The Buss

Durkee Hostility Inventory (BDHI; Buss & Durkee, 1957), another self-report measure, has also been employed but to a lesser extent.

The Ho scale is a 50-item questionnaire derived from the Minnesota Multiphasic Personality Inventory (MMPI; Hathaway & McKinley, 1943). Several factor analyses of this scale have been performed. Two factors identified by Costa et al. (1986) are referred to as *cynical mistrust* and *paranoid alienation,* suggesting that the Ho scale measures cynicism, distrust, suspiciousness and resentment. Smith and Frohm (1985), in their study assessing construct validity, proposed that the Ho scale mainly taps suspiciousness, resentment, frequent anger, and cynical distrust of others rather than aggressive behaviour or general emotional distress. Barefoot et al. (1989) identified several content-derived subscales within the Ho scale including *Hostile Attributions, Cynicism, Hostile Affect, Aggressive Responding and Social Avoidance.* They also identified a composite hostility score (Chost) derived from three subscales (Hostile Affect, Cynicism and Aggressiveness Responding) and proposed that it may be a more sensitive measure than the full-scale Ho score in terms of associating hostility with CHD endpoints. This composite Ho score has sometimes replaced the full-scale Ho in research (Barefoot et al.; Brummett et al. 1998; Helmers et al. 1993; Suarez & Williams, 1989). Although there remains some disagreement, many in the field maintain that the Ho scale largely reflects attitudes of cynicism and mistrust (Barefoot & Lipkus, 1994; Smith, 1992), and is thus best described as a measure of *cynical hostility* (Smith & Frohm).

The SI was originally developed by Rosenman and Friedman to assess TABP. During this standardized assessment, trained interviewers question respondents in a confrontational way in order to elicit a response to challenge. Hostility ratings are then

made based on the respondent's behaviour. Dembroski, MacDougall, Costa and Grandit (1989) developed a Hostility Facet Scoring System for the SI that featured the *Potential for Hostility* construct. This widely used measure was defined as the relatively stable tendency to experience anger, irritability, and resentment in response to frustrating events and to react with rudeness, antagonism, and uncooperativeness (MacDougall, Dembroski, Dimsdale, & Hackett, 1985). To generate a *Potential for Hostility* score, separate ratings of hostile content, intensity and interaction style are used. Improved SI scoring systems have since been developed including the Component Scoring System (Chesney, Hecker, & Black, 1989) and the Interpersonal Hostility Assessment Technique (Barefoot, 1992) which yields a Hostile Behavior Index score. With this technique, more emphasis is placed on the respondent's manner of interpersonal interaction rather than on speech content. More focus is given to the subtle signs of hostility as well (Haney et al. 1996).

Although used in fewer studies, the Buss-Durkee Hostility Inventory (BDHI) (Buss & Durkee, 1957) may be particularly useful in the assessment of different hostility components and their relationship to CHD. This 75-item questionnaire measures global hostility and seven hostility subscales including *physical assault, indirect hostility, irritability, negativism, resentment, suspicion and verbal hostility* as well as one subscale of *guilt*. Factor analyses of the BDHI have yielded two factors. The first, mainly defined by the physical assault and verbal hostility subscales, reflects frustration-induced expressive aspects of hostility and has been labelled *Expressive Hostility*. The second factor, represented best by the resentment and suspicion subscales reflects the experience of anger and hostility and has been called *Experiential or Neurotic Hostility* (Bushman, Cooper, & Lemke, 1991; Musante, MacDougall, Dembroski, & Costa, 1989).

Having identified the two most widely used assessment methods, the Ho scale and the SI, it becomes clear that the description of cynical hostility differs somewhat from the operationalized definition of the SI-derived *Potential for Hostility* which emphasizes anger and antagonistic behaviours (Costa et al. 1986; Smith & Frohm, 1985). Indeed, correlational analyses of Ho scale and *Potential for Hostility* ratings have produced a significant but modest relationship ($r = .37$). It is also noteworthy that both expressive and neurotic aspects of hostility are reflected in the Ho scale and *Potential for Hostility* rating, albeit in varying degrees. Smith and Frohm reported that the Ho scale was more closely associated with experiential or cognitive aspects such as anger-proneness, resentment and suspiciousness than it was with expressive hostility. Conversely, Musante et al. (1989) revealed that *Potential for Hostility* scores correlated with some elements of neurotic hostility but largely reflected the expression of anger and hostility. These various analyses suggest that, despite some association, these two instruments may not tap the same hostility construct (Dembroski et al. 1985).

Hostility and CHD

Over the past decades, researchers have relied on these different methods to assess the relationship between hostility and CHD. Studies employing the Ho scale, SI-Interview and BDHI will be reviewed.

Ho scale studies. The Ho scale is easy to administer and has been used extensively in prospective as well as cross-sectional studies. Results from prospective research have demonstrated that Ho scores predicted increased risk and/or incidence of major coronary events (Barefoot, Dahlstrom, & Williams, 1983; Barefoot, Larsen, von

der Leith & Schroll, 1995; Everson et al. 1997; Shekelle, Gale, Ostfeld, & Paul, 1983) and total mortality (Barefoot et al. 1983; Barefoot et al. 1989; Barefoot et al. 1995; Everson et al. 1997; Shekelle et al. 1983). Three studies, however, failed to find significant positive hostility-CAD relationships (Hearn, Murray, & Luepker, 1989; Leon, Finn, Murray, & Bailey 1988; McCranie, Watkins, Brandsma & Sisson, 1986). Recently, Iribarren et al. (2000) reported that cynical hostility was positively related with coronary artery calcification, a marker of subclinical atherosclerosis, in their study of young adults. O'Malley, Jones, Feurerstein and Taylor (2000), however, found no such relationship when testing adult U.S. Army personnel.

In an early cross-sectional study, Williams et al. (1980) were the first to identify a significant association between Ho hostility scores and severity of angiographically-documented coronary atherosclerosis. Their positive finding, however, was not replicated in other angiographic research (Barefoot et al. 1994; Dembroski et al. 1985; Helmer, Ragland, & Syme, 1991). Meesters and Smulders (1994) conducted a case-control study and positively linked Ho hostility with myocardial infarction among younger male subjects only. Other studies assessing the relationship between Ho hostility and exercise-induced myocardial ischemia have produced significant (Helmers et al. 1993) as well as nonsignificant results (Malkin, Milgraum, Boyle, Townsend & Siegman, 1999). In addition, results from a case-controlled study of Vietnam vets revealed a small, but significant relationship between Ho hostility and peripheral artery disease (Joesoef, Wetterhall, DeStefano, Stroup & Fronek, 1989). Lastly, in a recent study of men and women referred for thallium stress tests, Siegman, Townsend, Civelek and Blumenthal (2000) noted that the relationship between Ho hostility and CHD was not

significant once statistical adjustments for socioeconomic status, hypertension and diabetes were made.

SI studies. The association between SI-derived ratings of hostility and CHD incidence has also been examined in a small number of prospective studies. Positive findings were reported in a reanalysis of the Multiple Risk Factor Intervention Trial (MRFIT) study (Dembroski, MacDougall, Costa, & Grandits, 1989) and in separate reanalyses of the Western Collaborative Group Study (WCGS) data (Hecker, Chesney, Black & Frautschi, 1988; Houston, Chesney, Black, Cates & Hecker, 1992; Matthews, Glass, Rosenman & Bortner, 1977). In another longitudinal study, Powell and Thoresen (1985) observed that SI-derived hostility predicted recurrence of cardiac events among men with previously diagnosed myocardial infarctions.

SI-hostility has also predicted severity of angiographically-documented coronary artery disease (CAD) in several cross-sectional studies (Arrowood et al. 1982; Dembroski et al. 1985; MacDougall et al. 1985). Within the past decade, Barefoot et al. (1994) found SI hostility to be positively linked with CAD among non-smoking, asymptomatic United States Airforce personnel undergoing coronary angiography. In another study, Haney et al. (1996) positively related a SI-derived Hostile Behaviour Index score to severity of coronary occlusion in male patients. Helmer, Ragland, and Syme (1991), however, failed to detect a significant association between hostility and severity of CAD when studying hospitalized men and women. The SI-derived Hostility Behaviour Index score was also found to be a significant independent risk factor for CHD in Siegman et al.'s (2000) thallium stress test study.

BDHI studies. Only a few studies have used the BDHI in their assessments of hostility and CHD. Siegman, Dembroski and Ringel (1987), for example, observed that expressive hostility was positively associated with severity of CAD in angiographic patients aged 60 years and younger, whereas neurotic hostility was inversely related to disease severity. No significant relationship was observed between the total hostility score and CAD severity. In Ranchor, Sanderman, Bouma, Buunk and van den Heuvel (1997)'s cross-sectional study, the link between the expression and experience of hostility and various health outcomes were investigated but no hostility components were found to be related to CHD.

Mechanism models

It is clear from the review of prospective and cross-sectional studies that there is some evidence of a positive association between hostility and CHD, although conflicting findings exist as well. Possible explanations for these mixed results will be addressed in a forthcoming section of the introduction. If, however, the assumption remains that a relationship between hostility and CHD exists, the question of mechanism then arises. Presently, the mechanisms underlying this connection are not yet fully understood although five etiological models have been proposed. The first two are of particular interest in the present study.

The most prominent model to date, the *psychophysiological reactivity model* (Williams, Barefoot, & Shekelle, 1985), postulates that heightened cardiovascular and neuroendocrine responses to stress mediate the relationship between hostility and CHD. Hostile individuals, unlike their non-hostile counterparts, are hypothesized to vigilantly

scan their environment for signs of mistreatment and experience frequent and intense anger episodes. These reactions to stress are believed to produce sympathetically-mediated increases in cardiovascular and neuroendocrine activity which, in turn, serve to initiate and accelerate the coronary atherosclerotic process and presentation of CHD symptoms (Krantz & Manuck, 1984). This theory is in keeping with the broader traditional psychological model of stress and disease (Lazarus & Folkman, 1984) which postulates that individuals who appraise their environment as threatening, react to these appraised threats with negative affect. This emotion, in turn, leads to physiological changes that can promote disease onset and progression.

The *health behaviour model* proposed by Leiker and Hailey (1988) offers an alternate view of an underlying mechanism. This theory suggests that hostile individuals may be at an increased risk for CHD because of poor health habits. That is, relative to less hostile people, they may be more prone to engage in unhealthy behaviours that confer a CHD risk (Houston & Vavak, 1991). Behaviours such as smoking, fat intake, alcohol consumption, caffeine use and physical inactivity have all been the focus of research. As noted by Siegler (1994), "each health behaviour could have multiple pathways to account for the association of the particular risk factors with CHD" (p.200).

Other mechanism theories include the *psychosocial vulnerability* (Smith & Frohm, 1985), *constitutional vulnerability* (Krantz & Durel, 1983) and *transactional models* (Smith & Pope, 1990). Whereas the *psychosocial vulnerability model* posits that hostility leads to disease through its association with unhealthy characteristics, such as low social support and high levels of interpersonal conflict, the *constitutional vulnerability model* views biological factors, such as a hyperreactive sympathetic nervous

system, as key. The *transactional model* integrates and builds upon both psychophysiological and psychosocial perspectives. It proposes that hostile individuals, through their thoughts and behaviours, create a more stressful world characterized by more interpersonal conflict and less social support. They, in essence, respond with greater reactivity to a hostile environment that they helped create. According to Smith (1992), these models are not necessarily mutually exclusive and, together, may help explain the moderating role hostility plays in the pathogenesis of CHD.

Hostility and cardiovascular reactivity

In order to accumulate some support for the *psychophysiological reactivity model* (Williams et al. 1985), evidence that hostile persons exhibit exaggerated stress-induced cardiovascular reactivity relative to their less hostile counterparts is needed. Cardiovascular reactivity refers to the changes from resting level in cardiovascular physiological parameters, such as blood pressure and heart rate, that occur in response to task conditions. Much research has been conducted to investigate the relationship between hostility and cardiovascular reactivity in the laboratory.

Ho scale studies. Cynical hostility, as measured by the Ho scale, has not been positively related to heightened cardiovascular responses in several studies employing laboratory stressors referred to as "traditional", such as mental arithmetic tasks, cold pressor, and the Stroop colour-word task (Carroll, Davey-Smith, Sheffield, Shipley & Marmot; 1997; Kamarck, Manuck, & Jennings, 1990; Sallis, Johnson, Trevorrow, Kaplan, & Melbourne, 1987; Smith & Houston, 1987; Williams, Suarez, Kuhn, Zimmermann & Schanberg, 1991). Interpersonal stressors differ from traditional

stressors in that they involve various types of interpersonal conflict, most notably, anger provocation. The suggestion that negative emotional reactions to stress mediate the relationship between hostility and cardiovascular reactivity has prompted some researchers to include stressors – capable of producing angry reactions – in their study designs. It is believed that these interpersonal stressors may play a key role in eliciting differential cardiovascular responses among high and low hostile persons.

Interestingly, many positive associations between Ho measures and cardiovascular reactivity have emerged with the inclusion of such interpersonal stressors (Christensen & Smith, 1993; Fichera & Andreassi, 2000; Hardy & Smith, 1988; Shapiro et al. 2000; Smith & Allred, 1989; Smith & Brown, 1991; Suarez, Kuhn, Schanberg, Williams, & Zimmermann, 1998; Suarez & Williams, 1989; Weidner, Friend, Ficarrotto, & Mendell, 1989). As an example, Hardy and Smith (1988) reported that high, relative to low, hostile subjects, exhibited greater diastolic blood pressure responses to a role-playing task involving high interpersonal conflict. No group differences in reactivity were observed during the low conflict discussion. Using a debate task, Smith and Allred (1989), showed that high hostile subjects exhibited greater blood pressure responses relative to low hostile subjects. In two other studies, Suarez and Williams (1989) and Suarez et al. (1998) found high levels of Ho hostility to be associated with elevated cardiovascular reactivity only when an anagram task was combined with harassment. Moreover, when assessing cardiovascular responses during a public speaking task, Fichera and Andreassi (2000) showed that high hostile men exhibited greater blood pressure when compared with low hostile men.

Not all findings have been consistent however (Allred & Smith, 1991; Felsten, 1995, 1996; Kamarck et al. 1990). Using a discussion task with harassment, Allred and Smith failed to show significant cardiovascular reactivity differences amongst high and low hostile subjects. Felsten (1995) also found no reactivity group differences when subjects high and low in cynical hostility were harassed during a video competition task. In another study, Felsten (1996) reported no link between cynical hostility and cardiovascular reactivity to a cognitive task regardless of whether subjects were harassed or not.

SI studies. Cardiovascular reactivity studies that employed the SI to assess hostility have also generated contradictory findings. When traditional laboratory stressors were used, positive associations (Allen, Lawler, Matthew, & Rakaczky, 1984; Dembroski, MacDougall, Shields, Petitto & Kushene, 1978; Engebretson & Matthews, 1992; Lundberg, Hedman, Melin & Frankenhaeuser, 1989; MacDougall, Dembroski & Krantz, 1981; McCann & Matthews, 1988) as well as negative associations (MacDougall et al. 1981; Glass, Lake, Contrada, Kehoe, & Erlanger, 1983) between hostility and elevated cardiovascular responses were observed.

Few studies using SI-derived hostility scores have involved interpersonal stress tasks. Of those that did, significant positive results were demonstrated by MacDougall et al. (1981) and Anderson et al. (1986) who linked the *Potential for Hostility* total score or subscale scores to reactivity during the confrontational Structured Interview itself. More recently, Fredrickson et al. (2000) observed greater blood pressure responses among hostile subjects during a re-lived anger memory task. Studies by Chesney, Ekman, Friesen, Black and Hecker (1990) and MacDougall et al., however, did not relate

Potential for Hostility to cardiovascular reactivity. Furthermore, Diamond et al. (1984), using stressors involving competition, frustration and harassment, reported no relationship between *Potential for Hostility* ratings and cardiovascular responses.

BDHI studies. There have also been investigations on the role of BDHI-defined expressive and neurotic hostility in elevated cardiovascular reactivity to stress. Siegman and Anderson (1990) reported no significant correlation between expressive hostility and cardiovascular reactivity to a serial subtraction task in male subjects. Neurotic hostility was negatively correlated with heart rate reactivity in their study. In a similar experiment, Siegman, Anderson, Herbst, Boyle, and Wilkinson (1992) combined the math task with harassment and found expressive hostility to be positively related with blood pressure responses whereas neurotic hostility was not. Other reactivity studies have produced some similar patterns of positive results for expressive hostility when interpersonal stressors, most often harassment, were included (Felsten & Leitten, 1993; Lawler, Harralson, Armstead & Schmied, 1993; S. B. Miller, Dolgoy, Friese & Sita, 1996; Suarez, Harlan, Peoples & Williams, 1993; Suarez & Williams, 1990).

In summary, studies on the relationship between hostility and cardiovascular reactivity have generated inconsistent findings, although there is evidence of more significant positive findings when interpersonal stressors are used. Many researchers (S. B. Miller et al. 1996; Siegman et al. 1992; Suarez & Williams, 1989, 1990; Suarez et al. 1993; Suarez, Kuhn et al. 1998) have provided valuable evidence that harassment may be particularly important in eliciting reactivity differences between hostility groups. According to Houston (1994), such findings underscore the importance of considering the

interaction of personality and situational variables in understanding possible mechanisms leading to CHD.

Hostility and health-risk behaviours

Just as the relationship between hostility and cardiovascular reactivity has been explored, so too has the connection between hostility and health-risk behaviours. Sedentary habits, excessive intake of cholesterol, saturated fats, and calories, daily cigarette smoking and excessive consumption of alcohol and caffeine are believed to tax the cardiovascular system, either directly or indirectly, and, therefore, be implicated in accelerating the CHD process (Dembroski & Costa, 1987; T. Q. Miller et al. 1996). Typically, these risk factors are statistically controlled in research aiming to identify hostility as an independent risk factor for CHD (Siegler, 1994).

Ho scale studies. Most researchers have employed the Ho Scale to examine whether cynical hostility correlated with different health-risk behaviours. In a study of undergraduate students, for example, Leiker and Hailey (1988) created high and low hostile groups and observed that high hostile subjects reported poorer overall health habits including less physical exercise and self-care and greater alcohol use, including drinking while driving. In another study of undergraduates, Houston and Vavak (1991) reported similar results regarding alcohol use and drinking and driving as well as greater body mass index values among high hostile subjects. No group differences in smoking, exercise or preference for high fat and salty foods were found, however. When testing adult subjects, Musante, Treiber, Davis, Strong and Levy (1992) reported positive associations between hostility, cholesterol intake and, against expectation, vigorous

physical activity. For men, associations were also positive for smoking and sugar intake and negative for calcium intake. In another study, Schonwetter and Janissse (1991) assessed behavioural differences among varying alcohol-consuming groups of male psychology students. Heavy drinkers were found to be more hostile than both light drinkers and abstainers. Furthermore, in a cross-sectional study of over 5000 young adults, Scherwitz et al. (1992) observed that Ho hostility was associated with tobacco and marijuana smoking, increased alcohol consumption and greater caloric intake. Null findings were observed for physical fitness, daily caffeine consumption and a measure of fat intake.

Data from large-scale prospective studies have also been examined. Shekelle et al. (1983) reported small but significant associations between cynical hostility, smoking and alcohol intake among middle-aged men at a 10-year follow-up. Barefoot et al. (1983), however, failed to find hostility predictive of physicians' smoking prevalence after 25 years. Similarly, follow-up studies by Hearn et al. (1989) and McCranie et al. (1986) also failed to relate Ho hostility to smoking. Analyses of prospective data from the University of North Carolina Alumni Heart Study (UNCAHS), revealed that persons with higher Ho scores in college consumed more caffeine, had larger body mass index values and were more likely to be smokers at a 20-year follow-up (Siegler, Peterson, Barefoot & Williams, 1992). Using the same study data, Lipkus, Barefoot, Williams and Siegler (1994) assessed the role of personality measures, including hostility, in smoking initiation and cessation at follow-up. High Ho scores were among the best predictors of smoking initiation whereas low Ho scores predicted successful smoking cessation. Persons who continued to smoke 20 years later were more hostile and sensation seeking

as well. Lane, Pieper, Barefoot, Williams & Siegler (1994) also examined UNCAHS data and revealed significant correlations between daily caffeine intake and Ho hostility. Moreover, in a different study, Everson et al. (1997) assessed a Ho subscale and showed that the most cynically hostile men reported greater alcohol intake and smoking and had higher BMI values at a 9-year follow-up.

SI and BDHI studies. A smaller number of laboratories have employed the SI and BDHI to investigate associations with health-risk behaviours. Whereas Dembroski et al. (1989) positively related SI-derived hostility to smoking, Dielman, Leech, Miller and Moss (1991) linked it to greater alcohol use, more frequent speeding, less sleep, and a lower frequency of breakfast eating. High hostile young men were more likely to be smokers as well. Barefoot et al. (1994) observed in their study that smokers, relative to nonsmokers, reported higher SI-derived hostility scores.

With respect to BDHI use, Lee, Mendes de Leon & Markides (1988) revealed moderate associations between BDHI-derived hostility, smoking and alcohol consumption among some of their younger subjects. Moreover, in a 11-year follow-up study of Mexican Americans, one of the BDHI subscales was used to predict subsequent heavy drinking, but not smoking or body mass index values (T. Q. Miller, Markides, Chiriboga & Ray, 1995). It is also worth noting that significant, positive associations between hostility and measures of smoking (Koskenvuo et al. 1988; Raikkonen & Kettikangas-Jarvinen, 1991), heavy alcohol use (Koskenvuo et al., Romanov et al. 1994; Raikkonen & Kettikangas-Jarvinen), and physical inactivity (Koskenvuo et al., Raikkonen & Kettikangas-Jarvinen) have also been demonstrated by researchers in Finland who employed their own hostility instruments.

In conclusion, many, though not all, of the aforementioned findings offer support for linking hostility to certain coronary health-risk behaviours. Whether hostility contributes to CHD through its association with these risk factors remains unknown.

Hostility and defensiveness

In the review of studies attempting to relate hostility with CHD, cardiovascular reactivity and health-risk behaviours, it is apparent that, despite many significant positive findings, there are enough inconsistencies to preclude one from drawing strong conclusions at the present time. Possible explanations for these conflicting results include ambiguous definitions, construct validity issues, gender-related issues and methodological weaknesses, to name a few (Helmers et al. 1994; Smith, 1992). It has also been speculated that inconsistencies may arise if assessment measures tap different hostility dimensions that do not confer the same disease risk (Suls & Wan, 1993). Moreover, the use of stressors that differ in their effectiveness to elicit cardiovascular reactivity group differences during laboratory testing may also have produced mixed results.

Because of conflicting findings, several researchers have advocated combining cynical hostility with other psychosocial variables to determine whether its predictive power would improve. Houston (1994), for example, wrote that "other individual differences may moderate relations between Ho scale scores and reactivity to situations high, as well as low, in interpersonal stress" (p.105). Many researchers further specified that the construct of defensiveness, in conjunction with hostility, might serve as a better predictor of CAD and cardiovascular reactivity than hostility alone. Referring to the

hostility-CHD and hostility-reactivity literatures, Helmers (1993), for example, noted that "positive, negative, or null findings may stem from the fact that the sampled populations differed on defensiveness" (p.1).

Interest in this construct seemed to arise from reports that high defensiveness, when combined with measures of low anxiety (referred to as repressive coping) and low anger, was associated with elevated cardiovascular responses to stressors in clinical and nonclinical populations (Asendorpf & Scherer, 1983; Emerson & Harrison, 1990; King, Taylor, Albright & Haskell, 1990; Miller, 1993; Warrenburg, Levine & Schwartz, 1989; Weinberger, Schwartz & Davidson, 1979). Defensiveness has also been positively associated with sustained elevated blood pressure, a risk factor for CHD, in some studies (Jorgensen, Johnson, Kolodziej & Schreer, 1996; Mann & James, 1998; Sommers-Flanagan & Greenberg, 1989).

There appear to be several aspects to the construct of defensiveness, as measured by the Marlowe Crowne Social Desirability Scale (MC scale; Crowne & Marlowe, 1960), a 33-item self-report questionnaire. According to Jamner, Shapiro, Goldstein and Hug (1991), defensiveness refers to a coping style characterized by an orientation away from threatening stimuli. There is also the suggestion from factor analyses of the MC scale that defensiveness involves unconscious self-deception, as well as the conscious deception of others or impression management (Paulhus, 1984). Jorgensen, Abdul-Karim, Kahan and Frankowski (1995) proposed that this defensive coping style reflects a need to maintain social approval and avoid interpersonal conflict and evaluative threats in order to protect self-esteem. In accordance, persons with high scores on the MC scale are

expected to underreport, deny or suppress negative emotions, distress, and undesirable aspects of themselves (Helmers & Krantz, 1996; Shapiro, Goldstein & Jamner, 1995).

The combination of defensiveness and cynical hostility has been referred to as *defensive hostility* in recent years (Helmers et al. 1995). Persons with a defensive hostile profile, as indicated by high scores on both the Ho and MC scales, tend to have a cynical and distrustful outlook about the world as well as a need to be perceived favourably by others. Given that the expression of hostility is generally regarded as socially inappropriate, defensive hostile individuals are hypothesized to suppress negative feelings and beliefs in an attempt to present positively to others. It has been proposed that this chronic conflict over harboring hostility but being unwilling to express it, may play a fundamental role in cardiovascular hyperreactivity to stress and subsequent CHD risk (Helmers et al. 1995; Jamner et al. 1991).

Defensive hostility studies. A limited number of researchers have combined measures of cynical hostility and defensiveness to assess their relationships with cardiovascular reactivity and CHD. The first study to introduce the construct of defensive hostility into the literature was conducted by Jamner et al. (1991). In this well-cited field research they assessed the relationship between hostility, defensiveness and ambulatory cardiovascular responses among 33 male paramedics in different work settings. Median split scores of the Ho and MC scales were used to identify subjects scoring high and low in hostility and high and low in defensiveness. Subjects were then partitioned into four groups: High Hostility/High Defensiveness, High Hostility/Low Defensiveness, Low Hostility/High Defensiveness and Low Hostility/Low Defensiveness. In a hospital setting characterized by high interpersonal conflict,

paramedics scoring high in both defensiveness and hostility showed greater ambulatory heart rate and diastolic blood pressure responses than those high in hostility but low in defensiveness. When this study was replicated with a larger sample size of 54 paramedics, a similar result emerged although the effect was weaker. High hostile/high defensive paramedics displayed marginally heightened diastolic blood pressure levels relative to high hostile/low defensive counterparts in the hospital as well as on-scene and ambulance settings (Shapiro, Jamner & Goldstein, 1993).

The same researchers reported conflicting results, however, when examining the influence of four traits – cynical hostility, anger-out, anxiety, and defensiveness – on ambulatory cardiovascular responses by race and gender. In this study, the sample consisted of 144 healthy young college students who wore ambulatory recorders for a 24-hour period. Among the results, a combination of high defensiveness and high anxiety, not high hostility, was linked with heightened waking diastolic blood pressure in black men (Shapiro, Goldstein & Jamner, 1996).

Laboratory paradigms have also been used to explore relationships between defensive hostility and cardiovascular responsivity. Jorgensen et al. (1995) employed a mental arithmetic task with harassment to assess cardiovascular responses among 74 male undergraduate students. Their analyses revealed significant correlations between defensiveness and both systolic blood pressure and heart rate reactivity in students with high Ho scores only.

In another study, Helmers and Krantz (1996) examined the cardiovascular responses of 67 healthy adults (33 men and 34 women) to two mental stressors, a mental arithmetic and interpersonal speech task. Subjects were classified into four groups

labeled: Defensive Hostile (High Ho/High MC), High Hostile (High Ho/Low MC), Defensive (Low Ho/High MC) and Low Hostile (Low Ho/Low MC). Relative to other male subjects, Defensive Hostile males displayed greater systolic blood pressure at baseline and maintained this difference during the two stress tasks. A trend toward greater diastolic blood pressure levels was also observed in Defensive Hostile and Low Hostile men, relative to High Hostile and Defensive men, during baseline and the math task. Interestingly, the Low Hostile men, more than others, displayed greater diastolic blood pressure responses during the speech task. Of note, no significant hostility by defensiveness interactions were found in the reactivity (change score) analyses.

Helmers (1993) described slightly different findings in her dissertation research that was based on approximately the same data. During baseline and the two tasks, both the Defensive Hostile and Low Hostile males demonstrated the highest systolic and diastolic blood pressure levels. The Defensive Hostile males' baseline systolic blood pressure responses were significantly greater than the Defensive and High Hostile males. For the Low Hostile males, baseline systolic blood pressure levels were significantly higher than the High Hostile males whereas diastolic blood pressure responses were significantly higher than both the Defensive and High Hostile males.

One of the main objectives of Larson's and Langer's (1997) research was to examine the relationship between defensive hostility and additional heart rate reactivity among 40 male undergraduates exposed to a mental arithmetic task with or without the threat of shock. Median splits of MC and composite Ho scores were used when partitioning subjects into four study groups. As hypothesized, the Defensive Hostile

group exhibited significantly greater reactivity to the stress tasks relative to the other groups.

Mente and Helmers (1999) used extreme Ho and MC scores to assign 46 male university students into Defensive Hostile, Nondefensive Hostile, Nonhostile Defensive and Nonhostile Nondefensive groups. Three stress conditions were employed including the cold pressor, anger recall, and public speech tasks. In response to the cold pressor task, Defensive Hostile and Nonhostile Nondefensive subjects displayed greater diastolic blood pressure reactivity relative to Nondefensive Hostile subjects. No group differences in reactivity to the mental stress tasks were observed. These results offered only limited support for the hypothesis that Defensive Hostile persons exhibit elevated cardiovascular responses to stress relative to all others.

In another study, Shapiro et al. (1995) researched the interactive effects of anger-out, hostility, defensiveness, gender, and family history of hypertension on the cardiovascular reactivity of 209 healthy college students. Stressors included a math task, handgrip, and cold pressor task. For men, increased heart rate reactivity to the math task in particular was associated, not with defensive hostility, but with combinations of high hostility/low defensiveness and high defensiveness/low hostility.

The only published research on the association between defensive hostility and CHD was conducted by Helmers et al. (1995), and more recently, by Jorgensen et al. (2001). Helmers et al. assessed the influence of defensive hostility on myocardial ischemia in 78 patients with CAD. Subjects were divided into four groups, much like the procedure used by Jamner et al. (1991), although the composite hostility (chost) score for the Cook Medley scale was used. Results showed that Defensive Hostile patients,

relative to all others, exhibited the most severe exercise thallium ischemia; the most frequent ischemia during ambulatory electrocardiographic recording; and the most severe ischemia during laboratory math and speech tasks. It was concluded that "defensive hostility was related to more functionally severe CAD and may therefore, predispose CAD patients to a more adverse prognosis" (p.202).

In Jorgensen et al.'s (2001) study, the sample consisted of 59 male veterans, at-risk for CAD, who were undergoing coronary angiography. Of the four groups partitioned by median split scores of Ho and MC scales, the Defensive Hostile group exhibited a significantly greater number of coronary arteries with at least 50% obstruction.

From this collection of research, it is evident that only some of the preliminary evidence thus far supports the hypothesis that defensive hostility, rather than hostility, may be a preferable construct in terms of identifying individuals with greater cardiovascular reactivity to stress and possibly a greater risk for CHD.

The hypothesis that defensive hostile individuals' hyperreactivity may result from their conflict over anger expression (Helmers et al. 1995) underscores the importance of employing interpersonal stress tasks aimed at inducing anger in these types of research studies. As indicated previously, several studies examining the association between hostility or hostility dimensions and cardiovascular responses used anger-provoking harassment and yielded positive results (S. B. Miller et al. 1996; Suarez & Williams, 1989, 1990; Suarez et al. 1993; Suarez, Kuhn et al. 1998). Of the defensive hostility laboratory studies conducted thus far, only Jorgensen et al. (1995) used a laboratory procedure with harassment although no control condition was included. There is clearly

a need for replication studies given that a standardized laboratory harassment protocol with controls has not yet been done. Moreover, the small number of conducted studies to date, and evidence of conflicting findings also highlight the need for further investigation into the role of defensive hostility in cardiovascular reactivity to interpersonal stress.

Objectives and hypotheses

The main goal in the present study was therefore to examine the interrelationships between hostility, defensiveness and cardiovascular responses to interpersonal stress in the laboratory. Like previous research (i.e., Helmers et al. 1995; Jamner et al. 1991; Mente & Helmers, 1999), the Ho scale and MC scale were employed to identify male subjects as high and low in hostility and defensiveness and to assign them to four groups. In this study, groups were referred to as Defensive Hostile (HiDef/HiHo), Low Defensive/High Hostile (LoDef/HiHo), High Defensive/Low Hostile (HiDef/LoHo), and Low Defensive/Low Hostile (LoDef/LoHo). (Of note, for the sake of clarity, these four labels will be used when referring to the present study groups as well as the groups in the aforementioned studies. Because the term Defensive Hostile has been used repeatedly in the literature, it will be replace High Defensive/High Hostile.)

To determine the moderating influence of harassment on the relationship between combined traits of hostility and defensiveness and cardiovascular reactivity, subjects were assigned to either a harassment or non-harassment condition. Only harassed subjects received anger-provoking statements during a math-task. Emotional responses to harassment and non-harassment conditions were also examined to ascertain whether

the harassment was effective in provoking angry reactions and to assess whether differential group responses in negative affect emerged.

Cardiovascular measures recorded during testing included heart rate, systolic blood pressure, diastolic blood pressure, cardiac output and stroke volume. Previous studies (Helmers & Krantz, 1996; Jamner et al. 1991; Jorgensen et al. 1995; Mente & Helmers, 1999; Shapiro et al. 1993, 1995) typically assessed only blood pressure and heart rate. One of the strengths of the current study is the inclusion of additional cardiovascular measures that may provide valuable data to improve one's understanding of the hemodynamic mechanisms mediating the relationship between hostility, defensiveness and cardiovascular reactivity to harassment.

As noted earlier, some evidence of a relationship between hostility and health-risk behaviours has been observed in the literature suggesting poor health habits as a possible mechanism linking hostility to CHD. If cynically hostile individuals are more prone to engage in unhealthy lifestyle behaviours, it is conceivable that defensive hostile persons – those who are presumed to be hostile but conflicted over anger expression – may do so as well. An additional aim of this research was therefore to examine the association between defensive hostility and health-risk behaviours, such as smoking, caloric intake, and consumption of fats, cholesterol, alcohol and caffeine. To the author's knowledge, no one has conducted a study to test an apriori hypothesis regarding a defensive hostility-heath-risk behaviour relationship. A small number of studies have collected limited health-related information in an attempt to control for standard risk factors. Among these, no significant group differences in smoking were detected by Helmers et al. (1995) or Jorgensen et al. (1995). Helmers et al. reported a negative association between the Ho

composite score and duration of exercise and Helmers and Krantz (1996) found no group differences in regular exercise.

In the present study, it was hypothesized that the Defensive Hostile group would exhibit significantly greater cardiovascular reactivity than the other three groups, particularly in the harassment condition. In light of some evidence suggesting a link between hostility and health-risk behaviours, there was interest in examining whether an association between defensive hostility and health-risk behaviours also existed. In accordance, it was speculated that the Defensive Hostile group would report increased engagement in unhealthy behaviours when compared to the others. To the author's knowledge, this is the first study to undertake an investigation of the relationship between defensive hostility and two possible pathways leading to coronary disease endpoints; namely, through enhanced cardiovascular reactivity to stress, and through increased engagement in health-risk behavours.

Method

Subjects

One hundred healthy, normotensive males between the ages of 18 and 30 years were recruited from the Concordia University and McGill University student populations to participate in the study. All of the students completed a Screening Health Questionnaire (see Appendix A). Those that reported any serious physical or psychological health problems and/or regularly used medication that affected blood pressure were not selected for the study.

Participating subjects completed the Cook Medley Hostility Scale (Ho Scale; Cook & Medley, 1954) and Marlowe Crowne Social Desirability Scale (MC scale; Crowne & Marlowe, 1964) (See Appendices B & C) in order to be identified as either high or low hostile and high or low defensive by median split scores (Ho median score = 23; MC median score = 16). High Hostile (HiHo) subjects had Ho scores ranging from 23 to 40 with a mean score of 29.57 ± .62. Low Hostile (LoHo) subjects had scores ranging from 4 to 22 with a mean score of 16.04 ± .67. High Defensive (HiDef) subjects' MC scores ranged from 16 to 29 with a mean score of 19.65 ± .47 and Low Defensive (LoDef) subjects' MC scores ranged from 6 to 15 with a mean score of 11.21 ± .41. Subjects were then classified into four groups: Defensive Hostile (HiDef/HiHo, $n = 18$); Low Defensive/High Hostile (LoDef/HiHo, $n = 31$); High Defensive/Low Hostile (HiDef/LoHo, $n = 30$), and Low Defensive/Low Hostile (LoDef/LoHo, $n = 17$). To ensure that the researchers conducting the study were blind to the subjects' group membership, classification was done after all subjects completed the study. For cardiovascular

reactivity testing, subjects were randomly assigned to either a harassment or non-harassment condition immediately prior to the session.

Three subjects were eliminated from the study because of incomplete questionnaire data. One additional subject was removed because of computer problems during cardiovascular reactivity recording. Data from 96 subjects were used in the final statistical analyses.

Physiological measures and apparatus

Systolic blood pressure (SBP) and diastolic blood pressure (DBP) measurements (in mmHg) were obtained at one minute intervals using an IBS Model SD-700A Automatic Blood Pressure Monitor and a blood pressure cuff placed on the subject's left thigh. All blood pressure values were corrected for distance between cuff and heart level according to the manufacturers' specifications. Heart rate (HR: in bpm), cardiac output (CO: in 1/min) and stroke volume (SV: in ml) were recorded non-invasively by way of impedance cardiography, requiring a Minnesota Impedance Cardiograph (Model 304B), the Cardiac Output Program (C.O.P) developed by Bio-Impedance Technology, Chapel Hill, North Carolina, and an IBM AT personal computer. A tetrapolar band-electrode configuration was used. Two inner recording electrode bands were placed around the base of the neck and the thorax over the tip of the xiphoid process. Two outer electrode bands were positioned at least a 3 centimetre (cm) distance from the inner electrode bands around the neck and thorax.

An electrocardiogram (ECG) signal was recorded independently using three spot electrodes. Two electrodes were placed on either side of the torso below the ribcage and

a ground electrode was positioned on the right hip bone. The ECG signal was filtered through a Coulbourn Instrument bypass filter before being routed to the Minnesota Impedance Cardiograph. Recordings of HR, CO, and SV were taken during the first 30 seconds of each minute and ensemble averaged by the C.O.P. system to obtain values for that minute.

Psychological measures

The Cook Medley Hostility Scale (Ho scale; Cook & Medley, 1954) and Marlowe Crowne Social Desirability Scale (MC scale; Crowne & Marlowe, 1964) were administered to measure hostility and defensiveness. The Ho measure has high internal consistency with a Cronbach alpha of .86 (Cook & Medley, 1954) and test-retest reliabilities of .85 (Barefoot et al. 1983) and .84 (Shekelle et al. 1983) after one and four years respectively. The MC scale has test-retest reliabilities of .89 (Crowne & Marlowe, 1960), .86 after a one-month period (Crino, Svobda, Rubenfeld, & White, 1983) and .75 over a 12-year period (McCrae & Costa, 1983). Cronbach alphas of .78 (Helmer & Krantz, 1996), .88 (Crowne & Marlowe, 1964), and .87 (Mente & Helmers, 1999) have indicated high internal consistency.

Additional measures were also administered to subjects. The Buss Durkee Hostility Inventory (BDHI; Buss & Durkee, 1953) was employed to assess expressive hostility and neurotic hostility. Test-retest reliability for the total score is .82 with individual subscale reliability ranging from .66 to .88 (Biaggo, Supplee & Curtis, 1981).

The Spielberger Trait Anger Inventory (Spielberger et al. 1985) was used to tap individual differences in trait anger, defined as the disposition to experience frequent feelings of anger. Cronbach alphas of .82 to .85 have indicated high internal consistency.

To assess anger expression, subjects completed the Spielberger Anger Expression Scale (Spielberger et al. 1985). This 41-item Likert scale yields a total score and two subscale score modes of expression, anger-in and anger-out. Anger-in refers to the tendency to suppress anger whereas anger-out is defined as the tendency to direct anger outwards toward others. Test-retest reliability has ranged from .58 to .75 over 8-10 week intervals (Spielberger et al). Cronbach alphas have ranged from .70 to .84 for total and subscale scores (Johnson, Spielberger, Worden & Jacobs, 1987). Good convergent and divergent validities have also been indicated (Spielberger et al.).

Subjects also completed the Spielberger Trait Anxiety Inventory (Spielberger et al. 1985) which assesses the general disposition to experience frequent feelings of anxiety. This 20-item scale has high internal consistency (Cronbach's alphas ranging from .86 to .92) and test-retest reliability (rs ranging from .73 to .86) over one hour to 104 day intervals (Engebretson & Matthews, 1992).

A State Affect Questionnaire (See Appendix D) was employed to determine the subjects' current state affect at two times (baseline and post-math-task) during the cardiovascular reactivity testing session. This measure consists of 9 affective terms including: *agreeable, happy, tense, anxious, discouraged, irritated, angry, depressed, and guilty.* Subjects rated their current affective state on a visual analogue scale. They marked a vertical stroke at a point along a 12-cm line with endpoints labeled *not at all* and *very.* Points on the line were measured and given numeric values for data analysis.

Health-risk behaviour measures

General Health Survey. A General Health Survey (See Appendix E) was administered to obtain self-reported health-risk behaviour patterns. Subjects responded to questions with Likert-type scales measuring: typical daily caffeineated beverage consumption, typical daily alcohol consumption, and typical pattern of beer, wine, and hard liquor consumption. Smoking status (current smoker, non-smoker or past-smoker), average daily cigarette totals, and total smoking years were also determined.

Food Diaries. Subjects were requested to keep a food diary for two days (one weekday and one weekend-day). This requirement involved weighing and/or measuring all food and beverages consumed and recording the information on Weekday and Weekend-Day Food Diary Forms (see Appendices F and G). A food scale, along with measuring cups and measuring spoons were provided. Subjects were also asked to record cigarette consumption (number per day), intake of caffeineated beverages (number of coffees, teas, and soft drinks per day) and alcohol consumption (number of standard drinks per day) for each day. They were informed that one standard drink was equivalent to 12 ounces of beer, 5 ounces of wine or 1 1/2 ounces of hard liquor.

Computerized math subtraction task

The mathematical subtraction task stressor (math-task) consisted of a Computerized Subtraction Version 1.21 computer program by Turner, Sherwood & Lutz, in conjunction with an IBM compatible PC computer and a Truemouse Model TX 300 computer mouse. The 9-minute math-task was divided into three 3-minute trials, and consisted of a series of mathematical subtraction equations presented with either correct

or incorrect solutions. During each trial, 180 equations were presented on the computer monitor, one at a time, for three seconds. Subjects used the right and left computer mouse buttons to indicate whether the solution on the screen was correct or incorrect. Auditory feedback, either a high- or low-pitched tone, informed the subjects whether they answered correctly or incorrectly. No tone was emitted if they failed to respond within the allotted time. The subtraction equations fluctuated in terms of level of difficulty, becoming easier or more difficult depending on each subject's performance. The math-task was designed so that each subject attained a 50 to 60 percent correct response rate.

Procedure

Prior to participating in the study, subjects met with Researcher A (female) to receive instructions and give written consent (see Appendix H). Subjects were informed that the purpose of the study was to investigate the effects of nutrition, stress and performance on cardiovascular reactivity. They were blind to the real intent of the study. Subjects were also informed that there were two study parts: (1) a take-home component that involved recording dietary information; and (2) a laboratory session for cardiovascular reactivity testing. They were also told that they were required to complete a battery of questionnaires. This research was approved by the Human Ethics Committee of Concordia University.

Take-home: Health-risk behaviour recording. As described earlier, on two different days (one weekday and one weekend-day), subjects weighed and/or measured all food, beverage (alcoholic and non-alcoholic) and recorded the information on forms

provided by the researchers. Cigarette intake for each day was also recorded by the subjects.

Laboratory: Cardiovascular reactivity testing. Following the take-home study component, subjects participated in the laboratory session to measure cardiovascular responses to a subtraction math-task in either a harassment or non-harassment condition. All subjects were asked to refrain from smoking and coffee drinking for four hours prior to testing.

At the beginning of the testing session, the subject was seated in a reclining chair and connected to the physiological recording apparatus by Researcher A (female). After the recording apparatus was calibrated, the subject rested for 13 minutes. Baseline cardiovascular measures were recorded during the last four minutes of this rest period. Researcher A then gave the subject a paper-and-pencil State Affect Questionnaire to complete in privacy. Following its completion, Researcher A started to explain the math-task instructions to the subject.

The harassment scenario used in the present study was a modified version of the procedure developed by Suarez and Williams (1989). For subjects in the harassment condition, Researcher B (male) entered the testing room while Researcher A was giving the math-task instructions (see Appendix I) to the subject to inform her that she had to answer a phone call. After Researcher A completed the instructions, she excused herself and exited to the adjacent room, leaving the door ajar so that the subject could overhear the conversation. In a loud voice, Researcher A pretended to engage in a telephone conversation. When she hung up the phone, she asked Researcher B to take over the testing for her because she had to leave. Researcher B voiced his opposition loudly and

stated angrily that he "would not be responsible for any problems!" Researcher A returned to the testing room and explained to the subject that Researcher B would be replacing her and then left the room. Researcher B, who pretended to be angry, entered the testing room to start the math-task for the subject (see Appendix J for a more detailed description). During the math-task, Researcher B delivered six harassment statements to the subject at predetermined times. Sample statements included: "Did you understand the instructions?" and "Can't you do better than this?" (see Appendix K). The subject's comments were ignored unless he wanted to stop the experiment.

For subjects in the non-harassment condition, Researcher B interrupted Researcher A while she was giving the math-task instructions to tell her that she had received a phone call. Once the instructions were explained, Researcher A left the testing room and then returned to the subject to explain that she must leave and that Researcher B would continue the testing for her. Researcher A exited the testing room and Researcher B entered to begin the math-task. In the non-harassment condition, Researcher B remained courteous and friendly throughout the 9-minute math-task (see Appendix L).

After the math-task was completed, Researcher B gave harassed and non-harassed subjects the State Affect Questionnaire to complete in privacy a second time, and then left the testing room. Data collection for the present study was stopped at this point although subjects remained in the laboratory to participate in another study protocol. Following a 20-minute rest period, all subjects were debriefed about the deception and the rationale of the study. At the end of the study, subjects completed a battery of questionnaires including the Ho Scale, MC Scale, BDHI, Spielberger Trait

Anger Inventory, Spielberger Anger Expression Scale, Spielberger Trait Anxiety Inventory and General Health Survey. They were paid $ 50 for their participation in the study.

Cardiovascular data reduction and analyses

Cardiovascular data recorded during the testing sessions were reduced in the following manner. For each cardiovascular measure, values collected during the last four minutes of baseline testing were averaged to obtain a mean baseline value. Similarly, all values obtained during the math-task were averaged across nine minutes yielding a mean math-task value. To facilitate stress analyses, baseline-stress change scores were calculated by subtracting mean baseline cardiovascular values from the corresponding mean math-task values. Baseline values served as covariates. These change scores were used in all stress analyses given the uncertainty regarding the validity of impedance-derived volume measures when absolute values are employed (Sherwood, Allen, & Fahrenberg, 1990). As well, univariate analyses were employed in keeping with the majority of research in this area which uses univariate, rather than multivariate, analyses (i.e., Helmers & Krantz, 1996: Larson & Langer, 1997; McCann & Matthews, 1988; Mente & Helmers, 1999; Polefrone & Manuck, 1988; Smith & Allred, 1989). Significant interactions were subsequently followed by analyses of simple main effects and, where necessary, analyses of simple simple main effects.

Health-risk behaviour data reduction and analyses

For every food and beverage item documented in the food diaries, values of

calories (kcal), saturated fat (g), total fat (g), cholesterol (mg), alcohol and caffeine (mg) were derived for the weekday and weekend-day. Sources used for these calculations included the: Dietexpert Computer Program (N-Squared Corporation, 1993), Table of Food Composition (Whitney & Rolfes, 1996) and Kraft Canada Grocery Products: Nutrition Composition (1996). In cases where the nutritional information was unavailable from these sources, the author travelled to different supermarkets to obtain the required data from the Nutrition Information labels on food products. All food and beverage items were then individually coded and entered into the computer using the Dietexpert Computer Program. Cigarette totals (number per day) were also calculated and entered into the computer program. To standardize nutritional values, sums of saturated fat, total fat, and alcohol were converted to grams per 1000 kcal. Total cholesterol and caffeine were converted to mg per 1000 kcal. A similar conversion was done by Musante et al. (1992) in their study. Weekday and weekend-day mean totals were used in separate univariate analyses.

Results

Subject characteristics

To assess whether subjects differed in age, and body mass index (BMI) as a function of hostility group and defensiveness group, 2 (HiHo/LoHo) x 2 (HiDef/LoDef) analyses of variance (ANOVAs) were conducted using mean age and BMI values. BMI refers to a height/weight ratio [weight (kg)/height (m^2)]. Chi-square tests were used to determine if the four study groups - Defensive Hostile, Low Defensive/High Hostile (LoDef/HiHo), High Defensive/Low Hostile (HiDef/LoHo) and Low Defensive/Low Hostile (LoDef/LoHo) – differed in marital status and race.

A significant hostility group main effect for age, $F(1, 92) = 4.50$, $p < .04$, was found indicating that the low hostile subjects were older than the high hostile subjects (23.0 years ± .41 vs. 21.8 years ± .40). A significant defensiveness group main effect for BMI, $F(1, 92) = 6.42$, $p < .01$, revealed that the high defensive subjects had a higher mean BMI value relative to the low defensive subjects (24.4 ± .47 vs. 22.7 ± .47). Because of these group differences, a decision was made to use age and BMI values as covariates in forthcoming univariate analyses but only when significant correlations between them and the dependent variables were found. No significant group differences were identified in the chi-squared tests for marital status or race. Means and standard errors of age and BMI for the study groups are presented in Table 1.

Trait psychological questionnaire analyses

To examine differences in trait anger, anger expression (anger-in and anger-out), trait anxiety, BDHI hostility, expressive hostility and neurotic hostility as a function of

Table 1

Means and Standard Errors of Age and Body Mass Index (BMI) for the Study Groups

	HiDef/HiHo	LoDef/HiHo	HiDef/LoHo	LoDef/LoHo
Age (yrs)	21.4 (0.7)	22.2 (0.5)	22.3 (0.5)	23.7 (0.7)
BMI [weight (kg)/height (m^2)]	24.4 (0.7)	23.4 (0.6)	24.4 (0.6)	21.9 (0.8)

hostility group and defensiveness group, a series of 2 (HiHo/LoHo) x 2 (HiDef/LoDef) ANOVAs were conducted using the subjects' questionnaire scores.

No interactions were observed for any questionnaire score. Significant hostility main effects were indicated for trait anger, $F(1, 92) = 10.51, p < .002$, anger-in, $F(1, 92) = 5.22, p < .03$, BDHI hostility, $F(1, 91) = 37.32, p < .000$, expressive hostility, $F(1, 91) = 6.70, p < .01$, neurotic hostility, $F(1, 91) = 50.83, p < .000$, and trait anxiety, $F(1, 92) = 9.20, p < .003$. Significant defensiveness main effects were revealed for trait anger $F(1, 92) = 10.17, p < .002$, anger-in, $F(1, 92) = 10.37, p < .002$, anger-out, $F(1, 92) = 4.12, p < .05$, BDHI hostility, $F(1, 91) = 7.70, p < .007$, and expressive hostility, $F(1, 91) = 5.67, p < .02$ and trait anxiety, $F(1, 92) = 11.76, p < .001$. Consistent with what might be expected, these results indicated that high hostile subjects reported greater trait anger, anger-in, BDHI hostility, expressive hostility, neurotic hostility and trait anxiety relative to low hostile subjects. In addition, high defensive subjects reported significantly less trait anger, anger-in, anger-out, BDHI hostility, expressive hostility and trait anxiety relative to low defensive subjects. Means and standard errors of trait psychological questionnaire scores for the four study groups are presented in Table 2.

Because psychological questionnaires were administered at the end of the study, it is conceivable that subjects exposed to the harassment condition may have responded differently to questions relative to non-harassed subjects. To address this potential problem, separate one-way ANOVAs (Harass/Non-Harass) were conducted on questionnaire scores to determine if there were differences as a function of harassment condition. Of most importance, no significant group differences were found for the Ho scale or MC scale measures. For the other questionnaires, no significant results presented

Table 2

Means and Standard Errors of Trait Psychological Questionnaire Scores for the Study Groups

	HiDef/HiHo	LoDef/HiHo	HiDef/LoHo	LoDef/LoHo
Trait Anger	29.2 (1.6)	35.9 (1.2)	26.5 (1.3)	29.1 (1.7)
Anger-In	16.4 (0.7)	17.8 (0.5)	14.5 (0.5)	16.9 (0.7)
Anger-Out	15.6 (0.9)	18.0 (0.7)	15.0 (0.7)	15.9 (0.9)
BDHI Hostility	33.7 (2.2)	39.6 (1.6)	22.5 (1.6)	27.2 (2.2)
Expressive Hostility	10.9 (1.0)	13.7 (0.7)	9.4 (0.7)	10.7 (1.0)
Neurotic Hostility	6.9 (0.7)	7.4 (0.5)	3.3 (0.5)	2.5 (0.7)
Trait Anxiety	40.9 (2.1)	46.3 (1.6)	33.9 (1.6)	44.6 (2.2)

with the exception of trait anxiety, $F(1, 94) = 5.81, p < .02$. Non-harassed subjects reported greater trait anxiety than harassed subjects. This is likely a spurious result. Means and standard errors of the trait psychological questionnaire scores by harassment condition are presented in Table 3.

To examine interrelationships between the trait psychological questionnaire measures, Pearson correlations were conducted between: (1) the Ho scale and other psychological measures; and (2) the MC Scale and other psychological measures. In general, trait psychological measures were positively correlated with the Ho scale and negatively correlated with the MC scale. A significant negative correlation was found between the Ho scale and MC scale, $r(96) = -.28, p < .006$. Correlations are presented in Table 4.

Cardiovascular reactivity analyses

Baseline analyses. The relationships between hostility group, defensiveness group and baseline cardiovascular values were assessed using 2 (HiHo/LoHo) x 2 (HiDef/LoDef) ANOVAs or ANCOVAs for the following five cardiovascular measures: systolic blood pressure (SBP), diastolic blood pressure (DBP), heart rate, (HR) cardiac output (CO) and stroke volume (SV). Age was used as a covariate in the SBP and SV analyses.

No significant interactions or main effects were observed for baseline values of HR or SBP. A marginally significant defensiveness group main effect for DBP was found, $F(1, 92) = 3.52, p <. 064$, suggesting that low, relative to high, defensive subjects had marginally higher resting DBP. For SV, a marginally significant defensiveness

Table 3

Means and Standard Errors of Trait Psychological Questionnaire Scores as a function of Harassment and Non-Harassment Conditions

	Harass	Non-Harass
Trait Anger	29.8 (1.2)	31.0 (1.1)
Anger-In	15.8 (0.4)	16.8 (0.5)
Anger-Out	15.7 (0.6)	16.7 (0.6)
BDHI Hostility	31.4 (1.8)	30.6 (1.5)
Expressive Hostility	11.5 (0.8)	13.7 (0.7)
Neurotic Hostility	5.1 (0.5)	5.2 (0.5)
Trait Anxiety	37.8 (1.4)	42.7 (1.5)

Table 4

Correlations between Trait Psychological Questionnaire Scores

	Ho Scale	MC Scale	Trait Anger	Anger-In	Anger-Out	BDHI Hostility	NH	EH	Trait Anxiety
Ho Scale									
MC Scale	-.28**								
Trait Anger	.49**	-.52**							
Anger-In	.37**	-.34**	.46**						
Anger-Out	.33**	-.33**	.67**	.26*					
BDHI Hostility	.66**	-.46**	.61**	.32**	.52**				
NH	.72**	-.21*	.46**	.29**	.25*	.79**			
EH	.39**	-.41**	.54**	.14	.61**	.73**	.37**		
Trait Anxiety	.44**	-.34**	.46**	.55**	.28**	.50**	.51**	.16	

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

(Note: NH: Neurotic Hostility; EH: Expressive Hostility)

group main effect $F(1, 91) = 3.51, p < .06$ and a significant two-way interaction, $F(1, 91) = 6.21, p < .02$, were observed. Similarly, for CO, a marginally significant defensiveness group main effect, $F(1, 92) = 3.49, p < .065$, and a significant two-way interaction, $F(1, 92) = 5.42, p < .02$, were found. Inspection of these interactions revealed that the LoDef/HiHo group and the HiDef/LoHo group displayed significantly heightened SV and CO responses in comparison to the Defensive Hostile group at baseline. These results must be treated cautiously given the concern over the validity of absolute values of volume-based impedance measures (Sherwood et al. 1990). Means and standard errors of baseline cardiovascular scores for the four study groups are presented in Table 5.

Stress analyses. To assess the relationships between hostility group, defensiveness group, harassment condition, and cardiovascular reactivity to stress, 2 (HiHo/LoHo) x 2 (HiDef/LoDef) x 2 (Harass/Non-Harass) ANCOVAs were conducted on stress-baseline change scores for each cardiovascular measure using corresponding baseline cardiovascular values as covariates. Means and standard errors of baseline-stress change scores for the four study groups by harassment condition are presented in Table 6.

For SBP, a significant harassment main effect, $F(1, 87) = 16.88, p < .000$, hostility group main effect, $F(1, 85) = 5.13, p < .03$, and a 3-way interaction, $F(1, 87) = 8.59, p < .004$, were found. Analysis of the 3-way interaction was followed up with analyses of simple main effects and simple simple main effects. Guided by the study hypothesis, between-group differences within the harassment condition were investigated. Differences between harassed and non-harassed conditions

Table 5

Means and Standard Errors of Baseline Cardiovascular Scores for the Study Groups

Cardiovascular Measures	HiDef/HiHo	LoDef/HiHo	HiDef/LoHo	LoDef/LoHo
SBP (mmHg) *	115.2 (2.1)	115.8 (1.6)	114.3 (1.6)	114.5 (2.2)
DBP (mmHg)	61.5 (2.2)	64.7 (1.6)	63.6 (1.7)	67.6 (2.2)
HR (bpm)	58.8 (2.4)	58.2 (1.9)	61.7 (1.9)	62.5 (2.5)
CO (l/min)	6.3 (0.4)	7.7 (0.3)	7.7 (0.3)	7.5 (0.4)
SV (ml) *	105.5 (6.8)	132.3 (5.2)	129.6 (5.2)	126.0 (7.1)

* Adjusted means with age used as covariate

Table 6

Adjusted Mean Cardiovascular Change-Scores and Standard Errors for the Study Groups as a function of Harassment and Non-Harassment Conditions (Baseline Cardiovascular Means used as Covariates)

		HiDef/HiHo	LoDef/HiHo	HiDef/LoHo	LoDef/LoHo
Cardiovascular Measures	Harassment Condition				
SBP (mmHg)	Harass	19.9 (2.7)	15.5 (2.3)	16.2 (2.2)	27.1 (3.4)
	Non-Harass	8.7 (2.7)	11.9 (1.9)	15.6 (2.1)	13.2 (2.5)
DBP (mmHg)	Harass	10.0 (2.0)	10.5 (1.6)	9.3 (1.6)	9.8 (2.4)
	Non-Harass	8.0 (2.0)	9.7 (1.4)	10.3 (1.5)	5.3 (1.8)
HR (bpm)	Harass	15.2 (2.9)	16.6 (2.4)	13.9 (2.3)	22.2 (3.5)
	Non-Harass	8.4 (2.9)	7.1 (2.0)	8.4 (2.2)	9.2 (2.6)
CO (l/min)	Harass	1.0 (0.5)	1.9 (0.4)	0.8 (0.4)	1.4 (0.5)
	Non-Harass	0.5 (0.5)	0.4 (0.3)	0.4 (0.3)	1.0 (0.4)
SV (ml)	Harass	-10.5 (3.3)	-7.0 (2.7)	-10.3 (2.6)	-14.3 (3.9)
	Non-Harass	-6.1 (3.2)	-6.9 (2.3)	-10.3 (2.4)	-0.9 (2.9)

of each group as well as between-group differences within the non-harassment condition were also assessed. Of note, no significant SBP response differences were revealed between the Defensive Hostile group and the other three groups under harassment. The results did shows that, among subjects in the harassment condition, the LoDef/LoHo group had significantly greater SBP responses than the LoDef/HiHo group, $F(1, 16) = 8.98, p < .009$, and HiDef/LoHo group, $F(1, 17) = 10.32, p < .005$. In terms of within-group differences, the harassed Defensive Hostile subjects, $F(1, 15) = 7.11, p < .02$, were significantly more reactive than the non-harassed Defensive Hostile subjects. Similarly, the harassed LoDef/LoHo subjects, $F(1, 14) = 7.36, p < .02$, were more reactive than their non-harassed counterparts. Furthermore, under the non-harassment condition, HiDef/LoHo subjects showed greater SBP change than the Defensive Hostile subjects, $F(1, 22) = 4.33, p < .05$ (See Figure 1).

For SV, a significant harassment condition main effect, $F(1, 87) = 4.63, p < .03$, and a 3-way interaction, $F(1, 87) = 4.53, p < .04$, were found. Further examination of this interaction revealed no significant SV reactivity differences among the groups under harassment. In other words, the harassed Defensive Hostile group did not display greater SV change relative to the other harassed groups. With respect to within-group differences, the results showed that the harassed LoDef/LoHo group displayed greater SV reactivity than the non-harassed LoDef/LoHo group, $F(1, 14) = 11.40, p < .005$. There was also evidence of a significant finding among the non-harassed subjects. More specifically, HiDef/LoHo subjects had greater SV change relative to the LoDef/LoHo group, $F(1, 24) = 4.57, p < .05$ (See Figure 2).

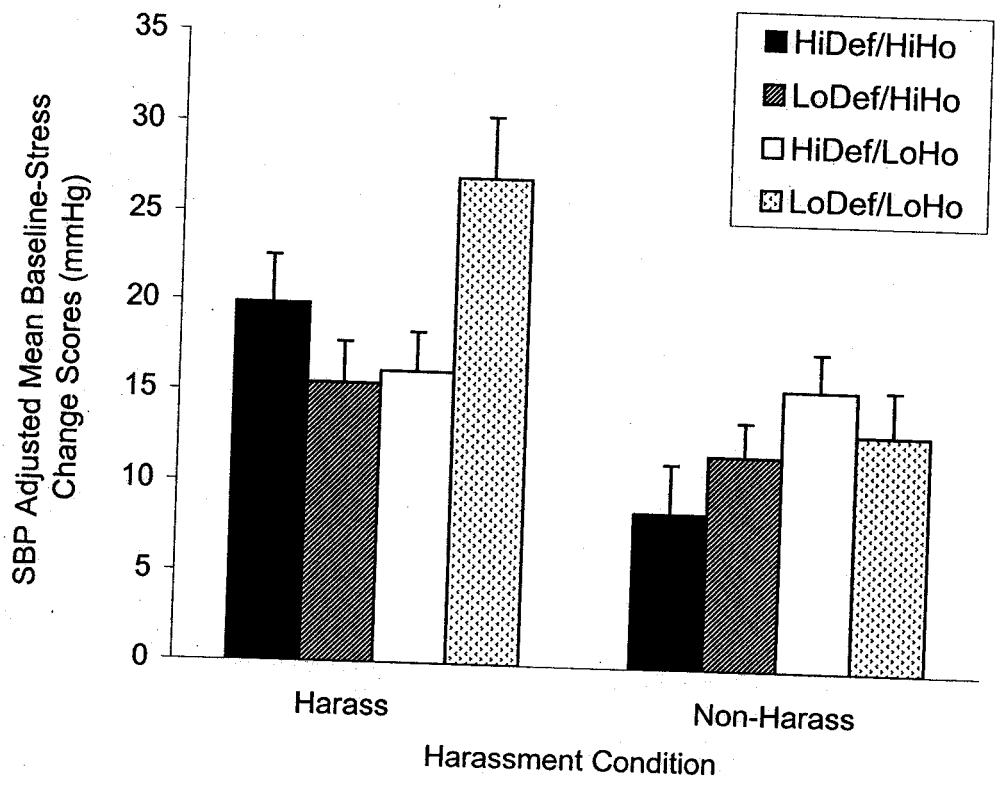

Figure 1. SBP adjusted mean baseline-stress change scores and standard errors for harassed and non-harassed study groups.

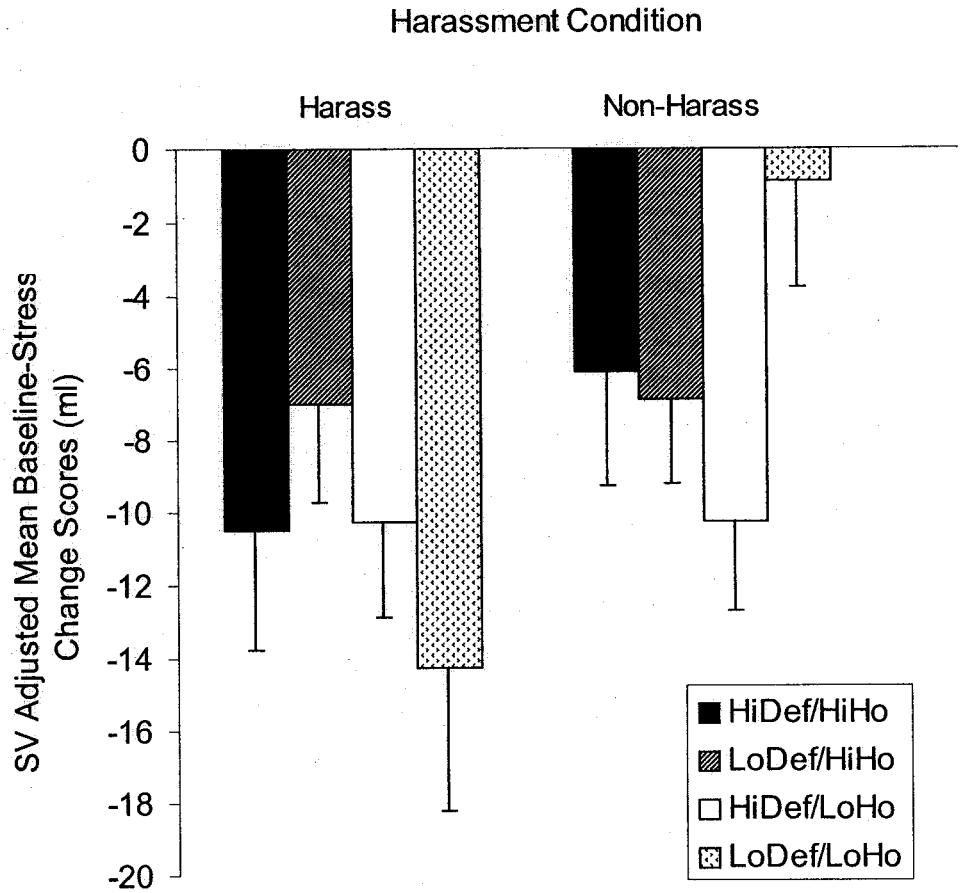

Figure 2. SV adjusted mean baseline-stress change scores and standard errors for harassed and non-harassed study groups.

In the HR-analysis, a significant harassment main effect, $F(1, 87) = 21.99, p < .000$ was found which indicated that harassed subjects displayed greater HR reactivity than non-harassed subjects. No other significant results for HR were observed.

A significant harassment condition main effect, $F(1, 87) = 5.01, p < .03$, was observed for CO. This finding revealed that harassed subjects exhibited significantly greater CO relative to their non-harassed counterparts. No other significant results for CO were found.

In the DBP analysis, no significant main effects or interactions were observed.

Affect analyses

Baseline affect analyses. To determine if there were differences by hostility group and defensiveness group on baseline (pre-math-task) self-reported affect scores, 2 (HiHo/LoHo) x 2 (HiDef/LoDef) ANOVAS were conducted for 9 affect terms: *agreeable, happy, tense, anxious, discouraged, irritated, angry, depressed and guilty.* Means and standard errors of baseline affect scores for the four study groups are presented in Table 7.

No interactions were found for any of the state affect terms. Significant defensiveness group main effects were revealed for *tense*, $F(1, 92) = 5.46, p < .02$, *anxious*, $F(1, 90) = 8.07, p < .006$, and *irritated*, $F(1, 92) = 7.52, p < .007$. A marginal defensiveness main effect was found for *guilty*, $F(1, 91) = 3.69, p < .058$. These findings suggest that prior to engaging in the math-task, high defensive subjects reported that they were significantly less tense, anxious, irritated and guilty relative to the low defensive subjects.

Table 7

Means and Standard Errors of Baseline Affect Scores for the Study Groups

Affects	HiDef/HiHo	LoDef/HiHo	HiDef/LoHo	LoDef/LoHo
Agreeable	8.4 (0.5)	7.3 (0.4)	8.9 (0.4)	8.3 (0.6)
Happy	7.5 (0.5)	6.4 (0.4)	7.3 (0.4)	6.8 (0.5)
Tense	2.7 (0.7)	3.4 (0.5)	2.3 (0.5)	4.6 (0.7)
Anxious	3.3 (0.7)	4.1 (0.5)	2.1 (0.5)	4.9 (0.7)
Discouraged	1.1 (0.4)	1.3 (0.3)	1.1 (0.3)	2.1 (0.4)
Irritated	1.6 (0.6)	3.0 (0.5)	1.0 (0.5)	2.8 (0.7)
Angry	0.7 (0.4)	1.3 (0.3)	0.7 (0.3)	1.2 (0.4)
Depressed	1.3 (0.5)	1.2 (0.4)	1.0 (0.4)	2.2 (0.5)
Guilty	0.8 (0.4)	1.4 (0.3)	0.7 (0.3)	1.5 (0.4)

Stress affect analyses

Two (HiHo/LoHo) x two (HiDef/LoDef) x two (Harass/Non-Harass) ANCOVAs were conducted to determine the effect of hostility group, defensiveness group and harassment condition on self-reported state affect scores following exposure to the math-task (post-task). Baseline-stress affect change scores were used for the 9 terms with baseline values serving as covariates. Means and standard errors of affect change-scores for the four study groups by harassment condition are presented in Table 8.

No significant interactions were revealed for any state affect terms. Instead, significant harassment condition, defensiveness group and hostility group main effects were observed. Significant harassment condition main effects were found for *angry*, $F(1, 85) = 14.90, p < .000$, *irritated,* $F(1, 87) = 7.34, p < .008$, *anxious,* $F(1, 85) = 7.92, p < .006$, *agreeable,* $F(1, 87) = 4.71, p < .03$, and *happy,* $F(1, 87) = 16.22, p < .000$. Validating the effect of the interpersonal conflict manipulation, these results suggested that subjects exposed to the anger-provoking harassment reported feeling significantly more angry, irritated, and anxious as well as less agreeable and happy when compared to their non-harassed counterparts. Significant defensiveness group main effects were also present for the terms *anxious,* $F(1, 85) = 5.34, p < .02$, revealing that high defensive subjects reported feeling less anxious following the math-task, regardless of harassment condition. In addition, significant hostility group main effects for *discouraged,* $F(1,87) = 4.32, p < .04$, *depressed,* $F(1, 87) = 4.61, p < .04$, and *guilty,* $F(1, 86) = 6.66, p < .01$, indicated that high hostile, relative to low hostile, subjects reported feeling more discouraged, depressed and guilty whether harassed or not.

Table 8

Adjusted Mean Affect Change-Scores and Standard Errors for the Study Groups as function of Harassment and Non-Harassment Conditions (Baseline Affect Scores used as Covariates)

		HiDef/HiHo	LoDef/HiHo	HiDef/LoHo	LoDef/LoHo
Affects	Harassment Condition				
Agreeable	Harass	-2.7 (0.9)	-3.4 (0.8)	-3.7 (0.7)	-3.7 (1.1)
	Non-Harass	-2.2 (0.9)	-3.1 (0.7)	-1.6 (0.7)	-1.6 (0.8)
Happy	Harass	-3.5 (0.8)	-2.8 (0.5)	-2.1 (0.6)	-2.3 (1.0)
	Non-Harass	0.1 (0.8)	-1.8 (0.6)	-0.2 (0.6)	-0.6 (0.7)
Tense	Harass	2.0 (1.0)	2.4 (0.8)	3.1 (0.8)	2.7 (1.2)
	Non-Harass	0.3 (1.0)	3.3 (0.7)	1.2 (0.7)	2.4 (0.9)
Anxious	Harass	1.1 (0.9)	2.2 (0.8)	2.0 (0.8)	3.3 (1.1)
	Non-Harass	-1.0 (0.9)	1.6 (0.7)	0.2 (0.7)	1.0 (0.9)
Discouraged	Harass	3.5 (1.1)	4.1 (0.9)	2.3 (0.9)	3.2 (1.3)
	Non-Harass	4.0 (1.1)	3.9 (0.8)	2.2 (0.8)	1.9 (1.0)

Table 8 continued

Affects	Harassment Condition	HiDef/HiHo	LoDef/HiHo	HiDef/LoHo	LoDef/LoHo
Irritated	Harass	4.2 (1.2)	3.6 (1.0)	3.4 (1.0)	5.6 (1.4)
	Non-Harass	2.0 (1.2)	3.2 (0.8)	0.7 (0.9)	2.8 (1.1)
Angry	Harass	4.5 (1.1)	3.5 (0.9)	3.8 (0.9)	3.8 (1.6)
	Non-Harass	2.3 (1.1)	1.1 (0.8)	0.4 (0.8)	0.5 (1.0)
Depressed	Harass	0.1 (0.8)	1.0 (0.7)	0.0 (0.6)	0.4 (1.0)
	Non-Harass	2.6 (0.8)	1.5 (0.6)	0.4 (0.6)	0.0 (0.8)
Guilty	Harass	1.1 (0.6)	0.7 (0.5)	0.6 (0.5)	-0.5 (0.7)
	Non-Harass	1.5 (0.6)	0.5 (0.4)	-0.2 (0.5)	-0.1 (0.5)

Health-risk behaviour analyses

General Health Survey analyses. In order to examine the relationships between hostility group, defensiveness group and self-ratings of heath-risk behaviour patterns, a series of 2 (HiHo/LoHo) x 2 (HiDef/LoDef) ANOVAs or ANCOVAs were conducted on select items of the General Health Survey. In addition, chi-squared tests were conducted to assess differences in smoking status (smoker, nonsmoker and past smoker) among the four study groups.

As noted previously, items of interest included: typical daily caffeineated beverage consumption; typical daily alcohol consumption; typical pattern of beer, wine, and hard liquor consumption; smoking status (current smoker, non-smoker or past-smoker); average daily cigarette totals; and total smoking years. Age was used as a covariate for the items typical daily alcohol consumption and wine consumption. Means and standard errors of the General Health Survey item scores for the four study groups are presented in Table 9.

In the General Health Survey analyses, a significant defensiveness group main effect was found for typical pattern of beer consumption, $F(1, 91) = 8.94, p < .004$ and a marginally significant defensiveness group main effect was present for typical daily alcohol consumption, $F(1, 91) = 3.67, p < .059$. These findings revealed that the high defensive subjects reported consuming less beer and marginally less alcohol when compared to their low defensive counterparts. No significant main effects or interactions were revealed for typical patterns of caffeineated beverage consumption, wine consumption or hard liquor consumption. In addition, chi-squared tests revealed no

Table 9

Means and Standard Errors of General Health Survey Item Scores for the Study Groups

	HiDef/HiHo	LoDef/HiHo	HiDef/LoHo	LoDef/LoHo
Caffeine (average day) (Item 6.)	1.3 (0.3)	1.2 (0.2)	1.1 (0.2)	0.9 (0.3)
Alcohol (typical day)* (Item 7.)	0.5 (0.1)	0.7 (0.1)	0.6 (0.1)	0.9 (0.1)
Beer intake pattern (Item 8.)	2.2 (0.4)	3.3 (0.3)	2.6 (0.3)	3.6 (0.4)
Wine intake pattern (Item 9.)	1.4 (0.2)	2.1 (0.2)	1.9 (0.2)	2.1 (0.2)
Hard liquor intake pattern (Item 10.)	1.4 (0.2)	1.7 (0.2)	1.7 (0.2)	1.7 (0.2)

* Adjusted mean with age used as covariate

significant group differences in smoking status (smoker, nonsmoker, past smoker). Because of the small number of smokers (n =12) and past smokers (n = 15) in the present sample, it was not possible to conduct further statistical analyses to compare group differences in average daily cigarette totals or total smoking years.

Take-home health-risk behaviour analyses

The relationships between hostility group, defensiveness group and subjects' mean weekday and weekend-day intake values for calories, saturated fat, total fat, cholesterol, alcohol, caffeine, and cigarettes were determined by 2 (HiHo/LoHo) x 2 (HiDef/LoDef) ANOVAs or ANCOVAs. An ANCOVA was conducted for weekend-day cholesterol values with age used as a covariate. As previously noted, values of saturated fat, total fat, cholesterol, alcohol and caffeine were standardized per 1000 calories. Means and standard errors of weekday and weekend-day intake scores for the four study groups are presented in Table 10.

No significant interaction or main effects were found for calories, saturated fat, total fat, alcohol, or cigarette intake. Hostility group main effects were demonstrated for weekday $F(1, 91) = 5.01, p < .03$, weekend-day $F(1, 89) = 5.95, p < .02$ cholesterol intake values. These findings indicated that high hostile subjects consumed higher levels of cholesterol on these two days relative to low hostile subjects.

Table 10

Means and Standard Errors of Weekday and Weekend-Day Intake Scores for the Study Groups (Standardized per 1000 kcal)

	HiDef/HiHo	LoDef/HiHo	HiDef/LoHo	LoDef/LoHo
Weekday				
Calories (kcal)	2886 (244)	2907 (181)	2863 (184)	2513(244)
Saturated Fat (g)	11.4 (1.2)	10.9 (0.9)	12.0 (0.9)	13.9 (0.1)
Total Fat (g)	34.3 (2.7)	33.2 (2.0)	34.0 (2.1)	35.3 (2.7)
Cholesterol (mg)	149 (23)	143 (17)	101 (17)	100 (22)
Alcohol (g)	1.6 (1.4)	3.6 (1.0)	1.8 (1.0)	2.4 (1.4)
Caffeine (mg)	20.9 (15.8)	56.6 (11.7)	43.6 (11.9)	56.7(15.8)
Cigarettes (# per day)	0.6 (1.1)	2.8 (0.8)	0.7 (0.8)	0.5 (1.1)
Weekend-Day				
Calories (kcal)	2530 (198)	2878 (151)	3132 (153)	2875 (204)
Saturated Fat (g)	11.0 (1.2)	13.1 (0.9)	12.3 (0.9)	11.2 (0.1)
Total Fat (g)	32.8 (2.5)	37.3 (1.9)	35.5 (1.9)	34.3 (2.5)
Cholesterol (mg)*	152 (21)	138 (16)	118 (16)	77 (22)
Alcohol (g)	0.0 (1.8)	3.8 (1.4)	4.3 (1.4)	3.6 (1.9)
Caffeine (mg)	49.3 (19.9)	78.7 (15.2)	42.7 (15.4)	52.9 (20.5)
Cigarettes (# per day)	0.3 (1.0)	2.7 (0.8)	0.7 (0.8)	1.3 (1.1)

* Adjusted mean with age used as covariate

Discussion

Of the several goals outlined in the present study, the primary objective was to examine the interrelationships between hostility, defensiveness, and cardiovascular reactivity to interpersonal stress among healthy male university students. There was also particular interest in determining whether harassment played a moderating role in this potential relationship between defensive hostility and cardiovascular response. Another study objective was to examine emotional reactivity among the four study groups to identify differential responses as well as to confirm whether the harassment was effective at eliciting anger as designed. A final goal of this research was to assess the relationship between defensiveness, hostility, and select health-risk behaviours. In accordance, it was hypothesized that the Defensive Hostile group would exhibit significantly greater cardiovascular reactivity than the other three groups, particularly under harassment. It was also speculated that the Defensive Hostile group would report increased engagement in health-risk behaviours when compared to the other subjects.

Overview of findings

Cardiovascular results from this study did not support the primary hypothesis. Against expectation, the harassed Defensive Hostile group did not show elevated cardiovascular reactivity relative to the other three harassed groups (please refer to Figures 1 and 2). In fact, their scores were statistically equivalent to those of the group on the other end of the scales (i.e., the LoDef/LoHo group). It was actually the LoDef/LoHo group that displayed the highest reactivity under harassment. More specifically, the LoDef/LoHo group had significantly greater reactivity under harassment

than did the LoDef/HiHo and HiDef/LoHo groups, but did not differ significantly from the Defensive Hostile group. The Defensive Hostile group did have numerically higher SBP reactivity than the LoDef/HiHo and HiDef/LoHo groups, and numerically lower SBP reactivity than the LoDef/LoHo group, but none of these differences were statistically significant. Because cell sizes for the harassed LoDef/LoHo and Defensive Hostile groups were small, no definitive conclusions of these findings can be made. Though interestingly, these results are consistent with the findings of some other researchers as will be discussed below.

Among the other notable findings was evidence that differential cardiovascular responses were observed between harassment and non-harassment conditions for two groups in particular. That is, only the harassed Defensive Hostile and LoDef/LoHo groups displayed significantly greater SBP reactivity than their non-harassed counterparts. The harassed LoDef/LoHo group also showed heightened SV responses. One unexpected finding was that differential group responses in SBP and SV reactivity were also observed in the non-harassment condition with no consistent pattern emerging. The HiDef/LoHo group had greater SBP than the Defensive Hostile group and greater SV than the LoDef/LoHo group.

In terms of emotional reactivity, surprisingly, no differential responses were found among the four study groups under harassment. Significant differences were observed, however, between harassed and non-harassed groups. All harassed subjects across groups reported more anger, irritation, anxiety and less agreeableness and happiness. In addition, high, relative to low hostile subjects felt more discouraged, depressed and guilty regardless of defensiveness scores or harassment condition, and,

high, not low, defensive subjects reported feeling less anxious irrespective of hostility scores or harassment condition.

Regarding the study hypothesis on health-risk behaviours, there was no indication that Defensive Hostile subjects reported increased engagement in the health–risk behaviours relative to any other subjects. In fact, no group differences were observed for any of the health-risk behaviours. Support was found, however, for a positive association between hostility and cholesterol intake and a negative association between defensiveness and beer intake. A negative association between defensiveness and general alcohol intake approached statistical significance. All of these findings will be discussed further in the sections to follow.

Defensive hostility: Cardiovascular and emotional reactivity findings

Cardiovascular reactivity findings. The main result showing no significant relationship between defensive hostility and cardiovascular reactivity conflicted with several of the research findings previously reviewed. The Defensive Hostile group in Jamner et al.'s (1991) field study of paramedics exhibited significantly greater HR and DBP increases when compared to the LoDef/HiHo group in hospital. In Shapiro et al's (1993) study, the Defensive Hostile paramedics displayed marginally greater DBP than the LoDef/HiHo paramedics in different settings. Among the laboratory studies, Larson and Langer (1997) found the Defensive Hostile group to be more reactive in additional HR reactivity than the other groups. Jorgensen et al. (1995) reported that, for high hostile subjects only, defensiveness scores correlated with SBP and HR responses to a mental arithmetic task with harassment. Moreover, Helmers and Krantz (1996) found higher

SBP during the tasks for the Defensive Hostile men but only when absolute value scores were used instead of change scores.

The present results were more consistent with the remaining defensive hostility studies, to the extent that they also offered no conclusive support for a defensive hostility – cardiovascular reactivity link. Results from these studies are, in fact, quite varied. Shapiro et al. (1996), for example, related exaggerated waking DBP among black males who were highly defensive and highly anxious. In their laboratory college study, heightened HR responses were found for HiDef/LoHo men and LoDef/HiHo men (Shapiro et al. 1995). Additional findings from Helmers & Krantz (1996) revealed that the LoDef/LoHo and Defensive Hostile males had greater DBP than the other two groups during the math task while the LoDef/LoHo men had the greatest DBP during the speech task. Helmers (1993) findings were slightly different. Both the LoDef/LoHo and Defensive Hostile males had the greatest SBP and DBP levels during the tasks. Furthermore, in Mente's and Helmers' (1999) study, both the LoDef/LoHo and Defensive Hostile groups had greater DBP reactivity during the cold pressor task when compared to the LoDef/HiHo group.

The reasons for conflicting results between this study and others, and in the literature as a whole, are unclear although some methodological explanations are worth considering. Like most of the previous studies, median splits were used to classify subjects into high and low hostile and high and low defensive groups in the present design. Cutoff scores for the Ho scale and MC scale were 23 and 16 respectively. These values were quite representative of cutoff scores used in other studies (ranging from 21 to 26 for the Ho scale and 13 to 18 for the MC scale). Nevertheless, these slight differences

may have played some role in discrepant findings. A more plausible explanation may be that a number of different stressors were used and there remains uncertainty over which are effective at differentiating cardiovascular responses between groups. As outlined in the literature review, these stressors included the cold pressor (Mente & Helmer, 1999; Shapiro et al. 1995), handgrip (Shapiro et al. 1995), anger recall task (Mente & Helmers); public speech task (Helmers, 1993; Helmers & Krantz, 1996; Mente & Helmers), mental arithmetic tasks with harassment (Jorgensen et al. 1995), mental arithmetic without harassment (Helmers; Helmers & Krantz; Shapiro et al. 1995) and mental arithmetic with and without the threat of shock (Larson & Langer, 1997). In light of Jorgensen's positive findings, there is some support for using an interpersonal stressor such as harassment. Some positive results, however, were also obtained without its use (i.e., Larson & Langer).

It is also evident that several studies had unique methodological designs that make direct comparisons between studies difficult. Mente's and Helmers' (1999) study was clearly different from the present and others, in that extreme cutoff scores were used rather than median split scores. In Helmers' & Krantz's (1996) and Helmers (1993) research, absolute values, rather than change scores (task minus baseline) were reported. From a psychophysiological perspective, there are clear problems with using absolute values, as they do not consider the subject's physiological starting point, and therefore are not reflective of a stress response. In another study, Larson and Langer (1997) were the only investigators to use the composite Ho scale rather than the full-scale Ho scale. They also did not report reactivity differences between their two stress conditions (math task and math task with shock) so their conclusions were based on the combined data

from two different stressors. In addition, while this study replicated several others by testing only male subjects (i.e., Jamner et al. 1991; Jorgensen et al. 1995; Larson & Langer; Mente & Helmers) Shapiro et al. (1993) assessed males and females without conducting separate analyses by gender. Their results may have thus been obscured since gender differences in the relationship between defensive hostility and cardiovascular reactivity have been reported by others (i.e., Helmers, 1993; Helmers & Krantz, 1996; Shapiro et al. 1995, 1996). Moreover, the present, as well as other laboratory studies clearly differed from the research by Jamner et al. and Shapiro et al. (1993) in that the latter assessed ambulatory reactivity in various natural settings.

Arguably, it is difficult to deduce which of these methodological differences may have accounted for discrepant results. Any of them, either alone or in combination, may have played some role.

Harassment and cardiovascular reactivity

The different methodologies and results described above highlight the need for implementing a standardized harassment protocol in this area of research. As outlined previously, laboratory harassment has played an important role in eliciting differential cardiovascular responses between high and low hostile groups (S. B., Miller et al. 1996, 1998; Suarez, Kuhn et al. 1998; Suarez & Williams, 1989, 1990). Its potential benefit seems to have also been acknowledged by some investigators of defensive hostility–cardiovascular reactivity associations. In attempting to account for null study results, for example, Mente and Helmers (1999) noted the absence of harassment and commented that "it may be necessary to harass subjects in order to elicit greater cardiovascular

responses in Defensive Hostile individuals" (p. 692). Moreover, Jorgensen et al. (1995), who employed a harassment stressor without a control condition questioned whether their harassing statements were sufficiently acrimonious when negative affect levels were not predicted by the Ho scale, MC scale, nor by their interaction.

In this study, the harassment stressor was indeed effective in heightening cardiovascular responses across groups. Higher levels in CO and HR were generally observed among harassed, relative to non-harassed, subjects. Interestingly, the harassment was also effective in elevating the cardiovascular responses of two particular groups, namely, the LoDef/LoHo and Defensive Hostile groups. Relative to their non-harassed counterparts, the harassed LoDef/LoHo group showed greater SBP and SV responses and the harassed Defensive Hostile group displayed a greater SBP response.

Before addressing further, the rationale for studying the combination of defensiveness and hostility rather than hostility alone is worth repeating. To reiterate, conflicting findings in the hostility-cardiovascular reactivity literature were suspected to arise because hostile subjects differed in their level of defensiveness and this construct had gone untapped. In order to resolve such disparities, there has been interest in determining whether a subset of high hostile subjects who are also defensive will consistently exhibit higher reactivity levels when exposed to interpersonal stress. As noted earlier, there was no clear support for this hypothesis in the present study (i.e., that under harassment, the Defensive Hostile group would be more reactive than the other groups). However, by examining the effects of harassment within each group, as described above, an interesting difference between the Defensive Hostile and LoDef/HiHo group emerged. The harassed Defensive Hostile group had greater SBP

reactivity than its non-harassed counterpart whereas the harassed LoDef/HiHo group did not. This response distinction could potentially have different health implications for these two groups given the proposed role of heightened cardiovascular reactivity to interpersonal stress in CHD pathogenesis (Williams et al. 1985). Although speculative, it may be that, if the Defensive Hostile group is exposed to stress on a regular basis and is reacting to it with exaggerated reactivity more often than the LoDef/HiHo group, then the Defensive Hostile group potentially may be at greater risk for CHD.

Another notable result emerged among the harassed groups. With respect to SBP reactivity levels, the harassed Defensive Hostile group could not be distinguished statistically from the group with the highest SBP reactivity, the LoDef/LoHo group. Interestingly, this was not the only study to observe such findings. The same two groups often displayed the highest and next-to-highest elevated cardiovascular responses, and often these elevations were not statistically different from one another (Helmers, 1993; Helmers & Krantz, 1996). The current study most closely replicated Mente's and Helmers' (1999) research in that cardiovascular reactivity change scores rather than absolute scores were reported. Thus, in light of these different study findings, there appears to be some suggestion that the combined personality constructs of high hostility and high defensiveness and conversely, low hostility and low defensiveness may both be associated with heightened cardiovascular reactivity to interpersonal stress.

At odds with the theoretical framework of this study, differential cardiovascular responses were also detected between groups in the non-harassment condition, even after controlling for baseline cardiovascular levels. Neither the Defensive Hostile group nor the LoDef/LoHo group, were the most reactive however. Instead, under non-harassment,

the HiDef/LoHo group showed greater SBP than the Defensive Hostile group, and greater SV than the LoDef/LoHo group. As no consistent pattern of responses emerged, these findings are difficult to explain. There seems to be some, albeit limited, evidence that subjects with high defensiveness and low hostility scores show greater cardiovascular responses than others without the use of an interpersonal stressor. Interestingly, these findings share some similarities with results from the repressive coping literature. Repressive copers, defined by high defensiveness scores on the MC scale and low trait anxiety scores on the Taylor Manifest Anxiety Scale (TMAS; Taylor, 1953) or short-formed version (Bendig, 1956) have shown exaggerated cardiovascular reactivity to different mental challenges (Asendorpf & Scherer 1983; King et al. 1990; Weinberger et al. 1979). Given that there is some uncertainty regarding the validity of this High Defensiveness/Low Hostility construct, as will be discussed later, the significance of this result involving the HiDef/LoHo non-harassed group is not clear.

Overall, these findings help to confirm that the harassment had an influential role in elevating cardiovascular responses in this study although it affected groups in unexpected ways.

Cardiovascular and emotional reactivity

An underlying assumption in this area of study is that an interpersonal stressor, such as harassment, impacts cardiovascular physiology through its influence on emotion (Lazarus & Folkman, 1984). Emotional arousal, in others words, is purported to mediate the relationship between personality constructs such as hostility and defensiveness and cardiovascular reactivity. (It is important to note, however, that this study was not

designed to test this specific hypothesis. It was also not possible to analyze correlations between cardiovascular change-scores and affect-change scores in each study cell because of limited sample size).

The harassed subjects in this study not only exhibited more pronounced cardiovascular responses (CO and HR) than non-harassed subjects but they also reported greater anger, irritation, anxiety, and less agreeableness and happiness. This was not surprising since the harassment protocol was specifically designed to elicit negative emotion. There was also evidence, however, of discrepancies between cardiovascular responses and emotional responses to stress. Specifically, under harassment, while group differences in cardiovascular reactivity were detected (even if not supportive of the study hypothesis) there were no group differences found for self-reported affect.

Researchers that have explored associations between cardiovascular and emotional reactions to stress in the hostility-cardiovascular reactivity literature have yielded mixed results. Suarez and his colleagues (Suarez, Kuhn et al. 1998; Suarez & Williams, 1989) have observed significant positive relationships between cardiovascular and affective responses for harassed high hostile groups only. In addition, Suarez and Williams (1990) documented that subjects with high, relative to low, expressive and neurotic hostility scores, had negative affects such as anger, irritation, upset and tension that were positively related with cardiovascular reactivity. Contradictory results obtained in one of the author's previous laboratory studies (S. B. Miller et al. 1996), however, showed that harassed high expressive hostile subjects displayed greater cardiovascular reactivity yet not greater negative affect when compared to the harassed low expressive hostile subjects. Moreover, Felsten (1995) observed that high hostile subjects reported

greater anger than low hostile subjects, independent of harassment, but did not exhibit greater cardiovascular reactivity responses. As noted by Felsten (1995) such findings "provide further evidence that cynical hostility, anger and cardiovascular reactivity are not simply nor consistently related"(p. 223).

Defensive hostility: Cardiovascular and emotional reactivity findings

Emotional reactivity findings. Part of the challenge in interpreting results from the current study in particular is understanding the role of defensiveness, along with hostility, in self-reported affect. In keeping with theory first proposed by Jamner et al. (1991), Defensive Hostile persons would presumably be angered by the harassment protocol and exhibit an exaggerated cardiovascular response relative to others. Given that such persons may also inhibit the expression of hostility because of self-deception and/or impression management concerns, they would be expected to report less negative affect than others under harassment. This did not occur in the present study. It is, of course, conceivable that this group was genuinely much more angered than the other three groups but masked or denied these feelings so that their lowered self-reported levels, when analyzed, were comparable to the others. Such an interpretation is not readily amenable to empirical evaluation however.

It is important to highlight, however, that of those researchers who also examined both cardiovascular and self-reported emotional responses in defensive hostile individuals, some yielded findings that were consistent with the present study. For example, hostility and defensiveness did not interact to predict affective reactions to the math task with harassment in Jorgenson et al.'s (1995) study. Mente and Helmers (1999)

found no significant group differences in affective responses, although this may have resulted, in part, because harassment was not used. Helmer and Krantz (1996) and Helmers (1993), in contrast, did detect differential responses in self-reported affect. Both Defensive Hostile and LoDef/LoHo male groups reported greater negative affect to the speech task than the two other groups.

One of the more perplexing findings to interpret in the current study is that the combination of hostility and defensiveness did not influence emotional reactivity in any way yet both personality constructs had independent effects on it. That is, high defensiveness was generally associated with lower levels of self-reported negative affect, and, conversely, high hostility was associated with higher levels of negative affect. It was also evident that high defensive subjects reported feeling less tense, anxious, irritated, and guilty and more agreeable than the low defensive subjects at baseline. Although the math task had not yet begun, the subjects may have felt some anticipatory anxiety and/or discomfort since they were wearing ECG recording apparatus and confined to the testing chair. The defensive subjects' responses may have reflected their intent to mask or deny these feelings. After the math-task, whether harassed or not, these high defensive subjects reported feeling less anxious than the low defensive subjects while the high, relative to low hostile subjects reported feeling more discouraged, depressed and guilty, although surprisingly, not more angry.

A helpful way to examine these affect findings is to view them together with the trait questionnaire results since similar patterns emerged. That is, as with the state affect results, no group differences were observed for any of the trait psychological questionnaire scores tapping trait anger, anger-in, anger-out, BDHI hostility, expressive

hostility, neurotic hostility and trait anxiety. It was also apparent that both the defensiveness and hostility constructs were independently related, not only with the state affect scores, but with the trait psychological scores as well.

It is surprising that the high hostile subjects, particularly those in the harassment condition, did not report greater state anger, although, as previously noted, this is not the only study to report such findings (Felsten, 1995; S. B. Miller et al. 1996). As might be expected, however, they did endorse higher scores on trait anger. Their report of greater discouragement, depression and guilt after the math task, regardless of harassment condition, might be explained by the fact that the Ho scale is more closely associated with neurotic aspects of hostility which include depression and guilt, than with expressive aspects (Smith & Frohm, 1985; Suarez & Williams, 1990). In support of this notion, the Ho scale was much more highly correlated with neurotic hostility than with expressive hostility in the present study.

Undoubtedly, there remain some puzzling findings in this examination of self-reported affect. Once the defensiveness construct was combined with hostility, the results seem to become more obscured and challenging to interpret.

Low Defensiveness/Low Hostility: Cardiovascular & emotional reactivity findings

Just as the cardiovascular and affective findings for the harassed Defensive Hostile group were unexpected, so too were those for the harassed LoDef/LoHo group. Before proceeding, it should be restated that interpretations were made cautiously since the sample size for the harassed LoDef/LoHo group was small. With the knowledge, however, that other researchers (Helmers, 1993; Helmers & Krantz, 1996; Mente &

Helmers, 1999) have also observed elevated cardiovascular responses for this LoDef/LoHo group, an argument can be made that further examination of this group is indeed justified.

Why did the LoDef/LoHo group evidence the highest SBP reactivity under harassment in this study? Although speculative, a hypothesis derived from the *transactional model of CHD* (Smith & Pope, 1990) may provide some insights. As suggested by this theory, high hostile individuals create a hostile environment characterized by high levels of interpersonal conflict and minimal social supports. If one were to infer that LoDef/LoHo persons, by contrast, would not engage in such behaviours, it follows that they likely experience less interpersonal stress on a daily basis. In so doing, they would likely be less accustomed to coping with such conflict. This proposition could potentially explain their exaggerated cardiovascular stress response when exposed to laboratory harassment. Although neither Helmers (1993) nor Mente and Helmers (1999) used the same stress task as in the present study, they posed similar arguments. Helmers for example, speculated that LoDef/LoHo subjects, who may be calm in most settings and create a life for themselves with minimal conflict, may have had heightened cardiovascular responses upon facing the novelty and challenge of the laboratory protocol.

As established earlier, the LoDef/LoHo group from this study was not unique in the way it responded in the laboratory since all harassed groups reported heightened negative affect on the State Affect Questionnaire. Although this measure did include several terms that effectively represented a wide range of emotional experiences, it might be prudent in future replication studies to add terms particularly suited to assess the merit

of the aforementioned hypothesis. Some possible choices are *surprised, vulnerable, loss of control, startled, confused, co-operative, bored, and ashamed.* For the same reason, it would be worthwhile to add measures that examine the subjects' social supports and self-esteem. There remains the challenge, however, of interpreting the validity of self-reported personal information when defensiveness is one of the constructs under study.

Nevertheless, in an attempt to acquire as much data as possible from this study in terms of the LoDef/LoHo group's emotional state during the math task, supplemental statistical tests comparing harassed and non-harassed members were conducted. The results did show that the harassed LoDef/LoHo group did indeed endorse more anger and irritation than its non-harassed counterpart. This emotional arousal, although not greater than the others harassed groups, may have played a role in heightening the SBP response for this group.

Arguably, there could be any number of unknown mechanisms, in lieu of, or in addition to, emotional arousal that may have influenced the LoDef/LoHo group's cardiovascular reactivity to stress. Level of task engagement might be considered. According to some researchers (Carroll et al. 1997; Piferi & Lawler, 2000; Suls & Wan, 1993) high hostile individuals, characterized as oppositional and mistrusting, may not exhibit the most elevated cardiovascular responses because they psychologically withdraw and become less engaged in the required task. This could potentially explain why the harassed LoDef/HiHo group from this study did not exhibit greater SBP or SV than its non-harassed counterpart. It may be that the LoDef/LoHo group exhibited greater cardiovascular responses, because they were, by contrast, more compliant and thus more fully engaged in the math-task relative to others.

Given that more attention, while understandable, has been directed at examining the adverse health implications of high levels of hostility and defensiveness in the literature, ironically, it becomes clear that less is understood about lower levels of this combined construct. Are the LoDef/LoHo subjects well-adjusted or vulnerable individuals who lack sufficient defenses to cope with interpersonal stress when exposed to it? It is not possible to rely on the data from the state and trait questionnaire measures since they do not differentiate this group from the others in any way. In terms of insights from others, Weinberger (1990) has noted that individuals scoring low in both defensiveness and anxiety emphasize their flexibility, vitality and enjoyment of interpersonal relationships in their own self-descriptions. Whether this description is applicable to the LoDef/LoHo subjects is not yet clear.

Implications and issues

The suggestion from this study and others (Helmers, 1993; Helmers & Krantz, 1996; Mente & Helmers, 1999) that a combination of low hostility and low defensiveness might be associated with greater cardiovascular reactivity, raises the question of whether this personality construct could be linked to elevated CHD risk. This notion contradicts current thinking. The *psychophysiological reactivity model of CHD* (Williams et al. 1985), for example, stipulates that the at-risk persons are highly hostile, hypervigilant to mistreatment, and frequently angry. According to the *transactional model of CHD* (Smith & Pope, 1990), at-risk hostile persons react with greater cardiovascular reactivity to a stressful environment that, through their thoughts and actions, they helped create.

Neither description seems germane to the LoDef/LoHo individuals who presumably do not experience interpersonal conflict, hostility, and anger on a chronic basis.

Alternatively, the question of whether some level of defensiveness and hostility might be protective to cardiovascular health can also be posed, particularly since both the LoDef/HiHo and HiDef/LoHo groups had the lowest cardiovascular reactivity (SBP and SV) levels under harassment in this study. In Piferi's and Lawler's (2000) study on women's cardiovascular reactivity, low hostile women were more reactive that the high hostile women who withdrew and disengaged from the task. It was postulated that high levels of hostility may therefore be protective by lessening women's reactivity to stress, but only in the short-term. In the long term, such avoidant coping and subsequent failure to address longstanding problems could result in enhanced levels of chronic stress and deleterious health effects. In another study, Levine et al. (1987) found that high deniers among male cardiac patients hospitalized for acute myocardial infarction, had shorter stays in intensive care and showed fewer signs of cardiac dysfunction. One year post discharge, however, the high deniers were more non-compliant with medical recommendations, showed poorer adaptation to their conditions and required longer rehospitalizations. These two study findings suggest that, although defensiveness may serve some adaptive function in the short-term, it, in turn, becomes maladaptive when used as a coping strategy for longer periods of time.

Defensiveness has also been associated with adverse health consequences in a number of research fields. It has, for example, been positively related to hypertension, a risk factor for CHD in some studies (Jorgensen et al. 1996; Mann & James, 1998; Sommers-Flanagan & Greenberg, 1989). Measures of defensiveness and repressive

coping have also been linked with the development and/or progression of malignancy (Dattore, Shontz & Coyne, 1980; Greer, Morris & Pettingale, 1979; Jensen 1987; Temoshok, 1987; Wirsching, Stierlin, Hoffman, Weber & Wirsching, 1982), tension and migraine headaches, Crohn's disease, ulcers, allergies, Derakshan & Eysenck, 1997; Schwartz, 1990; Weinberger 1990; Wickramasekera, 1995), greater salivary cortisol levels (Brown, Tomarken, Orth, Loosen & Davidson, 1996 lipid levels (Niaura, Herbert, McMahon & Sommerville, 1992), and plasma glucose levels (Jamner, Schwartz & Leigh, 1988). With respect to hostility, in addition to CHD, it has been linked to hypertension (Barefoot et al. 1983, Irvine, Garner & Craig, 1991; Siegler, et al. 1992), peripheral artery disease (Joesoef et al. 1989), and premature mortality from all causes (Barefoot et al. 1983; Barefoot et al. 1987; Shekelle et al. 1983).

The relationships between different measures of defensiveness and mental health have also been studied. In the psychoanalytic literature, defenses such as humour, sublimation and suppression are alleged to be adaptive by helping persons avoid self-criticisms, painful emotions and others' withdrawal of love (Horowitz, Markham, Stinson, Fridhandler & Ghannam, 1990). Measures of defensiveness have also been inversely related to lifetime prevalence of psychiatric disorders (Lane, Merikangas, Schwartz, Huang & Prusoff, 1990) and self-reported depression in cancer populations (Canning, Canning & Boyce, 1992; Phipps & Srivastava, 1997). Taylor and Brown (1988) have argued that "positive illusions" which seemingly have overlapping features with defensiveness, are essential for mental health. Well-adjusted persons using positive illusions to manage threats and negative feedback are reported to be happier, more caring and more productive than others who do not. This position has been criticized however

(i.e., Colvin & Block (1994). Conflicting reports linking repressive-defensiveness with lack of subjective well-being have also emerged (DeNeve & Cooper, 1998). Finally, Weisinger (1985) has argued that anger, rather than hostility, can serve positive functions by helping one to identify threats and conflicts as well as to potentiate feelings of control, and to immobilize energy to assert oneself and problem-solve.

Thus, in terms of these study findings, particularly with respect to physical health, there does not seem to be sufficient support linking defensiveness or hostility with protective benefits. On the contrary, there seems to be considerably more evidence linking these separate constructs to a variety of adverse health consequences.

Another important issue to contemplate is whether the Defensive Hostility construct is conceptually distinct from the HiDef/LoHo construct. In the repressive coping literature, high defensive/low anxious persons (the repressive copers), typically show disparities between their self-reported distress and objective behavioural and physiological indices of distress. Weinberger et al. (1979) remarked, for example, that they "deny having elevated levels of anxiety even though they often respond non-verbally as if they were highly anxious" (p. 369). These researchers, along with others, were able to distinguish repressive copers from low defensive/low anxious persons by changes in HR, SBP, sweat gland activity, muscle tension, reaction times and paralinguistic speech patterns (Asendorpf & Scherer, 1983; King et al. 1990; Weinberger et al.)

If the repressive copers are not genuinely low anxious, as postulated in these studies, their true level of anxiety is in question. By extending this line of reasoning to the current study of defensiveness and hostility, the same question can be posed with respect to the hostility levels for the HiDef/LoHo group. If subjects in this group are

more hostile than reported, are they characterologically akin to the Defensive Hostile group? To the author's knowledge, this issue has not yet been addressed in the literature. Furthermore, in many repressive coping studies, most notably, in Weinberger et al.'s (1979) study, a high defensive/high anxious group was omitted from the typology. Thus, whereas an attempt was made to differentiate the repressive copers from the low defensive/low anxious subjects in that study, there was no opportunity afforded to do the same with respect to high defensive/high anxious subjects.

It is also worth adding that, consistent with reports in the defensive hostility literature (Mente & Helmers, 1999; Schwartz, 1990; Shapiro et al. 1993) a moderate negative correlation between defensiveness and hostility was yielded in this study. Given this association, along with the questionable validity of the HiDef/LoHo construct, it is possible to speculate that subjects in this HiDef/LoHo group could be more heterogeneous than originally thought. That is, some might be more similar to Defensive Hostile subjects, although their hostility scores would not reflect it, whereas others could indeed be less hostile. Undoubtedly, if these two constructs – Defensive Hostility and HiDef/LoHo – are not conceptually distinct, any number of dubious results could emerge and the validity of all study findings would be in question.

One cautionary note, however, is that these conceptual issues are based on findings extrapolated from the repressive coping literature which pertains to high defensiveness and its interaction with low anxiety, not low hostility. Although positively correlated in this study, the association between hostility and anxiety is moderate ($r = .44$). They are indeed distinct constructs. Because it is not yet clear whether issues

pertaining to repressive coping do extend to defensive hostility, the conceptual issues raised above are speculative at present.

Defensiveness, hostility, and health-risk behaviours

While differential group responses in cardiovascular reactivity have been demonstrated in the current study, no differences between study groups emerged when their health-risk behaviours were analyzed. There was thus no support for the hypothesis linking defensive hostility to increased engagement in unhealthy lifestyle behaviours such as smoking, caloric intake, consumption of fats, cholesterol, alcohol and caffeine. Interestingly, there was empirical evidence that hostility was positively related with cholesterol intake and defensiveness was negatively associated with beer intake with trend findings for general alcohol consumption.

High hostility and cholesterol intake. According to the food diaries, high hostile subjects consumed significantly more cholesterol than the low hostile subjects on both self-monitoring days. This finding was consistent with the results by Musante et al. (1992) who used a structured questionnaire (Willett Food Frequency Questionnaire; Willett et al. 1985) to assess the frequency of specified food consumption over the preceding year. They too found a positive relationship between hostility and cholesterol intake for men. Other studies in this area of research did not assess dietary intake of cholesterol, but, instead, examined hostility and its relationship to dietary preferences as well as its relationship to serum lipid levels including total cholesterol and components, high-density lipoprotein (HDL) and low-density lipoprotein (LDL). Houston and Vavak (1991), who examined preferences for foods considered potentially deleterious to health,

found no differences between high and low hostile students in their preference for foods high in cholesterol and saturated fat. Moreover, of those studies investigating associations between hostility measures and serum cholesterol levels, results have been mixed. Significant positive results (Dujovne & Houston, 1991; Siegler et al. 1992; Suarez, Bates & Harralson, 1998; Weidner, Sexton, McLellarn, Connor & Matarazzo, 1987) as well as nonsignificant results have been observed (Richards, Hof & Alvarenga, 2000; Scherwitz et al. 1992; Shekelle et al. 1983; Suarez, Bates & Harralson, 1998) which help to show, as noted by Richards et al., that indeed the "the relationship between anger, hostility and unfavourable lipids is quite complex" (p. 394).

The positive hostility-cholesterol association obtained in this study offers some limited support for the *health behaviour model* (Leiker & Hailey, 1988) which postulates unhealthy lifestyle behaviours among high hostile persons as a mechanism underlying CHD pathogenesis.

Low defensiveness and alcohol consumption. The other interesting study finding emerged from questionnaire items in the General Health Survey. High, relative to low, defensive subjects reported that they consumed less beer on a typical day and showed a trend toward less alcohol consumption (beer, wine and spirits) in general. Given that these are the same subjects who consistently reported lower scores on the trait measures (with the exception of neurotic hostility) and less emotional distress before and after the stress task, it is highly possible that they may have under-reported or denied their beer/alcohol consumption, particularly if deemed problematic in their lives. If, however, these results do not represent an under-reporting bias or unconscious processes of

repression or denial, it is interesting to speculate whether high levels of defensiveness could play some protective function in terms of limiting alcohol use.

Viewing the same data from the alternate perspective, the findings show that low defensive subjects reportedly consumed more beer and marginally more alcohol than high defensive subjects. According to several studies, heavy, as opposed to moderate, alcohol consumption is related with increased risk of CHD (Klatsky, Friedman & Sieglaub, 1981; Shonwetter & Janisse, 1991). Proposed mechanisms underlying this relationship include alcohol-induced arrhymias, (Rubin 1980), alcoholic cardiomyopathy and its consequence, congestive heart failure (Orlando, Aronow, Cassidy & Prakash, 1976) and magnesium deficiency leading to coronary artery spasm and, in turn, possible sudden death due to ischemic heart disease (Turlapaty & Altura, 1980). (Of note, although the low defensive subjects showed a trend towards reporting more alcohol consumption on a typical day, there as no evidence of heavy alcohol consumption).

Health-risk behaviour summary. Based on these preliminary findings, there is no empirical evidence of a relationship between defensive hostility and increased engagement in any of the selected health-risk behaviours. No other combination of high and low defensiveness and hostility was linked with increased health-risk behaviours as well. Given that this study is purported to be the only one to date to investigate the associations between defensiveness, hostility and health risk behaviours, no definitive conclusions regarding these relationships should be made prematurely.

Study limitations and future directions

Before concluding, some methodological limitations in the present study should be acknowledged. Firstly, the sample size of 96 subjects was relatively small given that there were eight study cells. The harassed LoDef/LoHo group, in particular, was underrepresented. To circumvent this problem in future replication studies, it would be desirable to test more subjects and alter the protocol for classifying subjects into groups. In the present design, subjects were categorized after all subjects completed the study to ensure that the researchers conducting the study were blind to the subjects' group membership during laboratory testing. In the future, subjects could be pre-selected before participating in the study to ensure that appropriate subject quotas are met in each cell.

Although there is some loss in statistical power by using categorical (i.e., high/low groups) rather than continuous data, the decision to use the former was made in order to replicate the majority of previous studies that partitioned subjects into four study groups. Like most of these studies, median splits scores of the Ho scale and MC scales were used for classification. In order to obtain the most conceptually distinct groups in terms of high and low hostility and defensiveness scores, it would be preferable to use to tercile or quartile splits in the future.

One positive feature of the current study was that health-risk behaviour data was collected from two different sources; (1) through self-monitoring of food, beverage, alcohol and smoking consumption on two days; and (2) through a self-report questionnaire (General Health Survey) which tapped different health-risk behaviour patterns. An effort was made to collect data on both a sample weekday as well as a

weekend-day. Since it is not clear whether information collected on these two days reflects typical consumption patterns for subjects, it may be prudent to lengthen this monitoring phase in the future as well as seek verification from the subjects, although defensiveness may bias their responses. Moreover, it should also be noted that some of the health-risk behaviours under study (e.g., cholesterol and saturated fat intake) were not represented in the self-report General Health survey, thus limiting the amount of available data for these variables. It was also not possible to conduct all the statistical analyses as originally planned on smoking behaviour because of the small number of smokers and past smokers in the study sample. As a result, no conclusion about the relationship between defensive hostility and smoking behaviour can be derived from the present study.

As proposed earlier, one way to improve upon the current study would be to add affective terms to the State Affect Questionnaire along with additional measures (i.e., for social supports, self-esteem) to the questionnaire battery which subjects complete. It would be worthwhile to administer this battery prior to, rather than after, the laboratory testing to eliminate any biases that could result after the subjects have finished the experiment and have been debriefed about the deception.

The susceptibility of self-report measures to response biases, (i.e., a social desirability bias) is a well-recognized problem that challenges the validity of study outcomes. With respect to the present study, evidence of this defensive response style in self-reported affect was anticipated since the defensiveness construct was under study. Nevertheless, the subjects' defensiveness created many challenges when attempting to interpret this affect data, particularly when the true levels of hostility among the

HiDef/LoHo group were in question. Undoubtedly, the addition of some objective behavioural measures of affect to the present design would be helpful in terms of resolving some of the confusion.

An in-depth examination of different behavioural methods used to assess constructs such as hostility and defensiveness is beyond the scope of this dissertation. A few illustrations will be highlighted however. Facial expression has been found to distinguish hostile and nonhostile individuals in several studies (Brummett et al. 1998 Chesney, et al. 1990; Prkachin & Silverman, 2002; Rosenberg, Ekman & Blumenthal, 1998). Because facial expression is theorized to be linked to internal affective states (Ekman, 1977), researchers have examined it to make inferences about emotion and its regulation. The Facial Action Coding System (FACS) developed by (Ekman & Friesen, 1978) is a comprehensive technique used in studies employing the SI to assess behavioural manifestation of hostility. Trained coders record specific facial movements (i.e., muscular contractions) which, when combined, indicate emotional displays. Differences between defensive and nondefensive individuals have also been detected by various behavioural measures. According to Harrigan, Harrigan, Sale and Rosenthal (1996) audio cues taken from subjects' speech, speech errors, and prosodic features such as pitch, rhythm and amplitude are particularly salient for determining affective states as well as personality traits. Expressive behaviour was noted to reveal a defensive coping style.

The applicability of such observational techniques to the present study would have to be determined since the current protocol provides limited opportunity to assess visual or verbal cues during interpersonal conflict. Subjects received the harassment

statements from the researcher while engaging in the computer math-task and completed the State Affect Questionnaire in private immediately after it was completed. The researcher was also instructed to not engage the subject in conversation. In light of the limitations of self-report data, however, and the value of understanding emotional processes in cardiovascular reactivity to stress, these options should be strongly considered.

Finally, the generalizability of study findings based on a University student sample of males aged 18 to 30 years to the general population is debatable for a few reasons. Most importantly, gender differences have been identified in the relationship between defensive hostility and cardiovascular reactivity. In the study conducted by Helmers and Krantz (1996), LoDef/LoHo women displayed the lowest SBP and DBP responses during baseline and tasks whereas the Defensive Hostile, LoDef/HiHo, and HiDef/LoHo women did not exhibit differential cardiovascular responses. These results clearly differed from the results for men as previously reviewed. Gender differences in affect ratings had also observed among the four groups in their study. Another consideration is the impact that a student's lifestyle has on their health-risk behaviours. It could undoubtedly limit dietary choices as well as influence patterns of food, smoking, and alcohol consumption. In view of these limits to generalizability, there is indeed a need to replicate the present study with samples of women as well as men of different ages.

Conclusion

To the author's knowledge, the present study is the first to examine the interaction of defensiveness and hostility and its relationships to cardiovascular and emotional reactivity to interpersonal stress as well as health-risk behaviours. To summarize the major findings, there was no conclusive support for the hypotheses linking defensive hostility to the most exaggerated cardiovascular reactivity to interpersonal stress or to increased engagement in the selected health-risk behaviours. There was also no empirical evidence that defensive hostility was associated with lower self-reported state affect to harassment or with increased anger suppression, as predicted by theory (Jamner et al. 1991; Helmers et al. 1995). Surprisingly, a combination of low defensiveness and low hostility was instead associated with elevated SBP reactivity to harassment. Though consistent with some other studies, this conclusion remains tentative, given the small size of the harassed LoDef/LoHo group. No evidence linking this low defensiveness/low hostility construct to increased self-reported negative affect or increased engagement in health-risk behaviours was observed.

Interestingly, neither of the other two constructs consisting of low defensiveness/high hostility and high defensiveness/low hostility combinations were related to heightened cardiovascular reactivity to harassment. This finding for the low defensiveness/high hostility construct is particularly noteworthy for it conflicts with several reports from the hostility-cardiovascular reactivity literature which suggest that high levels of hostility are related to elevated cardiovascular reactivity to stress and thus possible CHD risk. There was, however, some limited support for the *health behavioural*

model (Leiker & Hailey, 1988) in this study since hostility was positively related with cholesterol intake.

The benefit of employing an interpersonal stressor, such as harassment, to assess interrelationships between defensiveness, hostility and cardiovascular reactivity was successfully demonstrated in this study, even though the moderating influence of harassment on cardiovascular reactivity was not exclusive to the Defensive Hostile group. The LoDef/LoHo group clearly reacted to the harassment with heightened reactivity; not only in contrast to other harassed groups but relative to its own non-harassed counterpart. Like the harassed LoDef/LoHo group, the harassed Defensive Hostile group also showed greater cardiovascular reactivity relative to its non-harassed counterpart. In light of these findings, there is sufficient support to argue that harassment should be used in future studies to differentiate cardiovascular responses among persons with different levels of hostility and defensiveness.

Since harassment–induced emotional arousal is hypothesized to mediate the relationship between personality constructs and cardiovascular reactivity, there was interest in examining differential group responses in self-reported affect. Results from the State Affect Questionnaire and various trait measures were, however, difficult to interpret because subjects differed in their level of defensiveness. Several discrepancies between cardiovascular and self-reported emotional reactivity findings were observed. Undoubtedly, these results highlight the challenge facing researchers who assess self-reported affect when studying the defensive hostility construct. For future studies, there clearly is a need to identify different means to tap the subjects' emotional responses in

addition to the use of self-report measures. As proposed earlier, observational methods may prove to be the most viable.

In light of these complex findings, there does seem to be some, albeit limited support that two personality constructs – consisting of low levels of both hostility and defensiveness and, conversely, high levels of hostility and defensiveness – may be associated with elevated cardiovascular reactivity to harassment. That such Defensive Hostile and LoDef/LoHo persons are at increased risk for CHD cannot be concluded on the basis of these findings. Given that this is the first study to explore the interactions between defensiveness, hostility and cardiovascular reactivity with the use of a standardized harassment protocol with control condition, additional research is needed to replicate these findings. It must also be established whether heightened cardiovascular reactivity to chronic stress is indeed a mechanism in the pathogenesis of CHD, as suggested by Williams et al. (1985). Whereas it has been hypothesized as one possible pathway to CHD for hostile persons, it is not clear whether this hypothesis extends to individuals who are both defensive and hostile. It certainly is not clear whether it applies to persons who score low in both constructs. If LoDef/LoHo persons do prove to exhibit heightened cardiovascular reactivity to interpersonal stress in future replication studies, there may be any number of unknown processes underlying their response.

Thus, based on the present findings, there appears to be sufficient support to argue that the defensiveness construct should again be combined with the hostility construct in future investigations of cardiovascular reactivity to interpersonal stress. The aim of these studies, however, should not only be to assess high levels of each construct, but to examine low levels of both hostility and defensiveness as well.

References

Allen, M. A., Lawler, K. A., Matthew, K. A., Rakaczky, C. J. (1984). Speech stylistic components of the structured interview and cardiovascular reactivity in college males. *Psychophysiology, 21*, 567.

Allred, K. D., & Smith, T. W. (1991). Social cognition in cynical hostility. *Cognitive Therapy and Research, 15*, 399-412.

Anderson, R. N. (2002). Deaths: Leading causes for 2000. *National Vital Statistics Reports, 40*, 1-82.

Anderson, N. B., & Williams, R. B., Lane, J., Haney, T., Simpson, S., & Houseworth, S. J. (1986). Type A behavior, family history of hypertension, and cardiovascular responsivity among black women. *Health Psychology, 5*, 393-406.

Arrowood, M., Uhrich, K., Gomillion, C., Popio, K., & Raft, D. 1982). New markers of coronary-prone behavior in a rural population. *Psychosomatic Medicine, 119*, 44.

Asendorpf, J. B., & Scherer, K. R. (1983). The discrepant repressor: Differentiation between low anxiety, high anxiety, and repression of anxiety by autonomic-facial-verbal patterns of behavior. *Journal of Personality and Social Psychology, 45*, 1334-1346.

Barefoot, J. C. (1992). Developments in the measurement of hostility. In H. S. Friedman (Ed.), *Hostility, coping and health* (pp. 13-31). Washington DC: American Psychological Association.

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., Dalhstrom, W. G., & Williams, R. B. (1989). The Cook-Medley Hostility Scale: Item content and ability to predict survival. *Psychosomatic Medicine, 51*, 46-57.

Barefoot, J. C., Larsen, S., von der Lieth, L., & Schroll, M. (1995). Hostility, incidence of acute myocardial infarction, and mortality in a sample of older Danish men and women. *American Journal of Epidemiology, 142*, 477-484.

Barefoot, J. C., & Lipkus, I. M. (1994). The assessment of anger and hostility. In A. W. Siegman & T. W. Smith (Eds.), *Anger, hostility and the heart* (pp. 43-66). Hillsdale, NJ: Lawrence Erlbaum Associates.

Barefoot, J.C., Patterson, J. C., Haney, T. L., Cayton, T. G., Hickman, J. R., & Williams, R. B. (1994). Hostility in asymptomatic men with angiographically confirmed coronary artery disease. *The American Journal of Cardiology, 74,* 439-442.

Bendig, A. W. (1956). The development of a short form of the Manifest Anxiety Scale. *Journal of Consulting Psychology, 20*, 384.

Biaggio, M. K., Supplee, K., & Curtis, N. (1981). Reliability and validity of four anger scales. *Journal of Personality Assessment, 45*, 639-648.

Blumenthal, J. A., Williams, R. B., & Kong, Y., Schanberg, S. M., & Thompson, L. W. (1978). Type-A behavior pattern and coronary atherosclerosis. *Circulation, 58,* 634-639.

Brown, L. L., Tomarken, A. J., Orth, D. N., Loosen, P. T., & Davidson, R. J. (1996). Individual differences in repressive-defensiveness predict basal salivary cortisol levels. *Journal of Personality and Social Psychology, 70,* 362-371.

Brummett, B. H., Maynard, K. E., Babyak, M. A., Haney, T. L., Siegler, I. C., Helms, M.

J., & Barefoot, J. C. (1998). Measures of hostility as predictors of facial affect during social interaction: Evidence for construct validity. *Annals of Behavioral Medicine, 20,* 168-173.

Bushman, B. J., Cooper, H. M., & Lemke, K. M. (1991). Meta-analysis of factor analyses: An illustration using the Buss-Durkee Hostility Inventory. *Personality and Social Psychology Bulletin, 17,* 344-349.

Buss, A. H. (1961). *The psychology of aggression.* New York: Wiley.

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

Canning, E. H., Canning, R. D., & Boyce, W. T. (1992). Depressive symptoms and adaptive style in children with cancer. *Journal of the American Academy of Child and Adolescent Psychiatry, 31,* 1120-1124.

Carroll, D., Davey-Smith, G., Sheffield, D., Shipley, M. J., & Marmot, M. G. (1997). The relationship between socioeconomic status, hostility and blood pressure reactions to mental stress in men: Data from the Whitehall II study. *Health Psychology, 16,* 131-136.

Chaplin, J. P. (1982). *Dictionary of psychology* (Rev. ed.). New York: Dell.

Chesney, M. A., Ekman, P., Friesen, W. V., Black, G. W., & Hecker, M. H. (1990). Type A behavior pattern: Facial behavior and speech components. *Psychosomatic Medicine, 53,* 307-319.

Chesney, M. A., Hecker, M., & Black, G. W. (1989). Coronary-prone components of Type A behavior in the WCGS: A new methodology. In B. K. Houston & C. R.

Snyder (Eds.), *Type A behaviour pattern: Research, theory and intervention* (pp. 168-188). New York: Wiley.

Christensen, A. J., & Smith, T. W. (1993). Cynical hostility and cardiovascular reactivity during self-disclosure. *Psychosomatic Medicine, 55,* 193-202.

Colvin, C. R., & Block, J. (1994). Do positive illusions foster mental health? An examination of the Taylor and Brown formulation. *Psychological Bulletin, 116,* 3-20.

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

Costa, P. T., Zonderman, A. B., McCrae, R. R., & Williams, R. B. (1986). Cynicism and paranoid alienation in the Cook and Medley Ho scale. *Psychosomatic Medicine, 48,* 283-285.

Cotran, R. S., Kumar, V., & Robbins, S. L. (1989). *Robbins Pathologic Basis of Disease* (4th ed.). Philadelphia: W. B. Saunders Company.

Crowne, D., & Marlowe, D. (1960). A new scale of social desirability independent of psychopathology. *Journal of Consulting Psychology, 24,* 349-354.

Crowne, D., & Marlowe, D. (1964). *The approval motive: Studies in evaluative dependence.* New York: John Wiley & Sons, Inc.

Dattore, P. J., Shontz, F. C., & Coyne, L. (1980). Premorbid personality differentiation of cancer and non-cancer groups: A test of the hypothesis of cancer proneness. *Journal of Clinical Psychology, 13,* 388-394.

Dembroski, T. M., & Costa, P. T. (1987). Coronary prone behavior: Components of the Type-A pattern and hostility. *Journal of Personality, 55,* 211-235.

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

Dembroski, T. M., MacDougall, J. M., Shields, J. L., Petito, J., & Lushene, R. (1978). Components of the Type A coronary-prone behavior pattern and cardiovascular responses to psychomotor performance challenge. *Journal of Behavioral Medicine, 1,* 159-176.

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

DeNeve, K. M., & Cooper, H. (1998). The happy personality: A meta-analysis of 137 personality traits and subjective well-being. *Psychological Bulletin, 124,* 197-229.

Derakshan, N., & Eysenck, M. W. (1997). Repression and repressors: Theoretical and experimental approaches. *European Psychologist, 2,* 235-246.

Dielman, T. E., Leech, S. L., Miller, M. V., & Moss, G. E. (1991*). Multivariate Experimental Clinical Research, 10,* 67-83.

Dimsdale, J. F., Hackett, T. P., & Hunter, A. M. (1979). Type A behavior and angiographic findings. *Journal of Psychosomatic Research, 23,* 273-276.

Dujovne, V. F., & Houston, B. K. (1991). Hostility-related variables and plasma lipid levels. *Journal of Behavioral Medicine, 14,* 555-565.

Ekman, P. (1977). Biological and cultural contributions to body and facial movement. In J. Blacking (Ed.), *The anthropology of the body* (pp. 39-84). London: Academic.

Ekman, P., & Friesen, W. V. (1978). *Manual for the Facial Action Coding System*. Palo Alto, CA: Consulting Psychologists Press.

Emerson, C. S., & Harrison, D. W. (1990). Anger and denial as predictors of cardiovascular reactivity in women. *Journal of Psychopathology and Behavioral Assessment, 12*, 271-283.

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

Everson, S. A., Kauhanen, J., Kaplan, G. A., Goldberg, D. E., Julkunen, J., Tuomilehto, J., & Salonen, J. T. (1997). Hostility and increased risk of mortality and acute myocardial infarction: The mediating role of behavioral risk factors. *American Journal of Epidemiology, 145*, 142-152.

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

Felsten, G. (1996). Cardiovascular reactivity during a cognitive task with anger provocation: partial support for a cynical hostility-anger-reactivity link. *Journal of Psychophysiology, 10*, 97-107.

Felsten, G., & Leitten, C. L. (1993). Expressive, but not neurotic hostility is related to cardiovascular reactivity during a hostile competitive task. *Personality and*

Individual Differences, 14, 805-813.

Fichera, L. V., & Andreassi, J. L. (2000). Cardiovascular reactivity during public speaking as a function of personality variables. *International Journal of Psychophysiology, 37,* 267-273.

Frank, K. A., Heller, S. S., Kornfeld, D. S., Sporn, A. A., & Weiss, M. B. (1978). Type A behaviour pattern and coronary angiographic findings. *Journal of American Medical Association, 240,* 761-763.

Fredrickson, B. L., Maynard, K. E., Helms, M. J., Haney, T. L., Siegler, I. C., & Barefoot, J. C. (2000). Hostility predicts magnitude and duration of blood pressure responses to anger. *Journal of Behavioral Medicine, 23*, 229-243.

Friedman, M., & Rosenman, R. (1959). Association of a specific overt behavior pattern with increases in blood cholesterol, blood clotting time, incidence of arcus senilis and clinical coronary artery disease. *Journal of American Medical Association, 169*, 1286-1296.

Friedman, M., & Rosenman, R. (1974). *Type A behavior and your heart.* New York: Knopf.

Friedman, M., Rosenman, R. H., Strauss, R., Wurm, M., & Kositchek, R. (1968). The relationship of behavior pattern to the state of the coronary vasculature. A study of 51 autopsy subjects. *American Journal of Medicine, 44,* 525-538.

Glass, D. C., Lake, C. R., Contrada, R. J., Kehoe, K., & Erlanger, L. R. (1983). Stability of individual differences in physiological responses to stress. *Health Psychology, 2,* 317-341.

Greer, S., Morris, T., & Pettingale, K. W. (1979). Psychological response to breast cancer: Effect on outcome. *Lancet, 2,* 785-787.

Haney, T. L., Maynard, K. E., & Houseworth, S. J. (1996). Interpersonal hostility assessment technique: Description and validation against the criterion of coronary artery disease. *Journal of Personality Assessment, 66,* 386-401.

Hardy, J. D., & Smith, T. W. (1988). Cynical hostility and vulnerability to disease: Social support, life stress, and physiological response to conflict. *Health Psychology, 7,* 447-459.

Harrigan, J. A., Harrigan, K. M., Sale, B. A., & Rosenthal, R. (1996). Detecting anxiety and defensiveness from visual and auditory cues. *Journal of Personality, 64,* 675-709.

Hathaway, S. H., & McKinley, J. C. (1943). *The Minnesota Multiphasic Personality Inventory.* Minneapolis, MN: The University of Minnesota Press.

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 (2001, September 22). *Heart disease: Risk factors – cholesterol.* Retrieved August 20, 2002 from http://www.heartandstroke.ca

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

Helmers, K. F. (1993). *Defensive hostility: Psychosocial correlates and associations with cardiovascular responses. Dissertation Abstracts International,* 54(9-B). (UMI No. 9333566)

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

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

Helmers, K. F., Krantz, D. S., Howell, R. H., Klein, J. K., Bairey, C. N., & Rozanski, A. (1993). Hostility and myocardial ischemia in coronary artery disease patients: Evaluation by gender and ischemic index. *Psychosomatic Medicine, 55*, 29-36.

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

Helmers, K. F., Posluszny, D. M., & Krantz, D. S. (1994). Association of hostility and coronary artery disease: A review of studies. In A. W. Siegman & T. W. Smith (Eds.), *Anger, hostility and the heart* (pp. 67-95). Hillsdale, NJ: Lawrence Erlbaum Associates.

Horowitz, M. J., Markham, H. C., Stinson, C. H., Fridhandler, B., & Ghannam, J. H. (1990). A classification theory of defense. In J. L. Singer (Ed.), *Repression and dissociation: Implications for personality theory, psychopathology, and health* (pp. 61-84). Chicago: University of Chicago Press.

Houston, B. K. (1994). Anger, hostility and psychophysiological reactivity. In A. W.

Siegman & T. W. Smith (Eds.), *Anger, hostility and the heart* (pp. 97-115). Hillsdale, NJ: Lawrence Erlbaum Associates.

Houston, B. K., Chesney, M. A., Black, G. W., Cates, D. S., & Hecker, M. H. L. (1992). Behavioral clusters and coronary heart disease risk. *Psychosomatic Medicine, 54,* 447-461.

Houston, B. K., & Vavak, C. R. (1991). Cynical hostility: Developmental factors, psychosocial correlates, and health behaviors. *Health Psychology, 10,* 9-17.

Irabarren, C., Sidney, S., Bild, D. E., Liu, K., Markovitz, J. H., Roseman, J. J., & Matthews, K. (2000). Association of hostility with coronary artery calcification in young adults. *Journal of American Medical Association, 283,* 2546-2551.

Irvine, J., Garner, D. M., & Craig, H. M. (1991). Prevalence of Type A behavior in untreated hypertensive individuals. *Hypertension, 18,* 72-78.

Jamner, L. D., Schwartz, G. E., & Leigh, H. (1988). Repressive and defensive coping predicts monocyte, eosinophil, and serum glucose levels: Support for the opioid peptide hypothesis of repressive coping. *Psychosomatic Medicine, 53,* 393-406.

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.

Jenkins, C. D. (1978). Behavioral risk factors in coronary artery disease. *Annual Review of Medicine, 29,* 543-562.

Jensen, M R. (1987). Psychobiological factors predicting the course of breast cancer. *Journal of Personality, 55,* 317-342.

Joesoef, M. R., Wetterhall, S. F., DeStefano, F., Stroup, N. E., Fronek, A. (1989). The

association of peripheral arterial disease with hostility in a young healthy veteran population. *Psychosomatic Medicine, 51,* 285-289.

Johnson, E. H., Spielberger, C. D., Worden, T. J., & Jacobs, G. A. (1987). Emotional and familial determinants of elevated blood pressure in black and white adolescent males. *Journal of Psychosomatic Research, 31*, 287-300.

Jorgensen, R. S., Abdul-Karim, K., Kahan, T. A., & Frankowski, J. J. (1995). Defensiveness, cynical hostility and cardiovascular reactivity: A moderator analysis. *Psychotherapy and Psychosomatics, 64,* 156-161.

Jorgensen, R. S., Frankowski, J. J., Lantinga, L. J., Phadke, K., Sprafkin, R. P., & Abdul-Karim, W. K. (2001). Defensive hostility and coronary heart disease: A preliminary investigation of male veterans. *Psychosomatic Medicine, 63,* 463-469.

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.

Kamarck, T. W., Manuck, S. B., & Jennings, J. R. (1990). Social support reduces cardiovascular reactivity to psychological challenge: a laboratory model. *Psychosomatic Medicine, 52*, 42-58.

Keys, A. (1970). Coronary heart disease in seven countries: XIII multiple variables. *Circulation, 41,* 138-144.

King, A. C., Taylor, C. B., Albright, C. A., & Haskell, W. L. (1990). The relationship between repressive and defensive coping styles and blood pressure responses in

healthy, middle-aged men and women. *Journal of Psychosomatic Research, 34*, 461-471.

Klatsky, A. L., Friedman, G. D., & Sieglaub, A. B. (1981). Alcohol and mortality: A ten year Kaiser-Permanente experience. *Annals of International Medicine, 95*, 139-145.

Koskenvuo, M., Kaprio, J., Rose, R., Kesaniemi, A., Sarna, S., Heikkila, K., & Langinvainio, H. (1988). Hostility as a risk factor for mortality and ischemic heart disease in men. *Psychosomatic Medicine, 50,* 330-340.

Krantz, D. S., & Durel, L. A. (1983). Psychobiological substrates of the Type A behavior pattern. *Health Psychology, 2,* 393-411.

Krantz, D. S., & Manuck, S. B. (1984). Acute psychophysiologic reactivity and risk of cardiovascular disease: a review and methodological critique. *Psychological Bulletin, 96,* 453-464.

Krantz, D. S., Sanmarco, M. I., Selvester, R. H., & Matthew, K. A. (1979). Psychological correlates of progression of atherosclerosis in men. *Psychosomatic Medicine, 41*, 467-476.

Lane, J. D., Pieper, C. F., Barefoot, J. C., Williams, R. B., & Siegler, I. C. (1994). Caffeine and cholesterol: Interactions with hostility. *Psychosomatic Medicine, 56,* 260-266.

Lane, R. D., Merikangas, K. R., Schwartz, G. E., Huang, S. S., & Prusoff, B. A. (1990). Inverse relationship between defensiveness and lifetime prevalence of psychiatric disorder. *American Journal of Psychiatry, 147*, 573-578.

Larson, M, R., & Langer, A. W. (1997). Defensive hostility and anger expression:

Relationship to additional heart rate reactivity during active coping. *Psychophysiology, 24,* 177-184.

Lawler, K. A., Harralson, T. L., Armstead, C. A., & Schmied, L. A. (1993). Gender and cardiovascular responses: What is the role of hostility? *Journal of Psychosomatic Research, 37,* 603-613.

Lazarus, R., & Folkman, S. (1984). *Stress, appraisal and coping.* New York: Springer.

Leiker, M., & Hailey, B. J. (1988). A link between hostility and disease: Poor health habits? *Behavioral Medicine, 19,* 129-133.

Levine , J., Warrenburg, S., Kerns, R., Schwartz, G., Delaney, R., Fontana, A., et al. (1987). The role of denial in recovery from coronary heart disease. *Psychosomatic Medicine, 49,* 109-117.

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

Lipkus, I. M., Barefoot, J. C., Williams, R. B., & Siegler, I. C. (1994). Personality measures as predictors of smoking initiation and cessation in the UNC Alumni Heart Study. *Health Psychology, 13,* 149-155.

Lundberg, U., Hedman, M., Melin, B., & Frankenhaeuser, M. (1989). Type A behavior in healthy males and females as related to physiological reactivity and blood lipids. *Psychosomatic Medicine, 51,* 113-122.

MacDougall, J. M., Dembroski, T. M., Dimsdale, J. E., & Hackett, T. P. (1985). Components of Type A, hostility, and anger-in: Further relationships to

angiographic findings. *Health Psychology, 4,* 137-152.

MacDougall, J. M., Dembroski, T. M., & Krantz, D. S. (1981). Effects of type of challenge on pressor and heart-rate responses in Type A and B women. *Psychophysiology, 18,* 1-9.

Malkin, A. R., Milgraum, M., Boyle, S., Townsend, S., & Siegman, A. W. (1999). Hostility, coronary heart disease, and ischemia: The role of socioeconomic status. *Annals New York Academy of Sciences, 896,* 455-457.

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

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

McCann, B. S., Matthews, K. A. (1988). Influences of potential for hostility, Type A behavior, and parental history of hypertension on adolescents' cardiovascular responses during stress. *Psychophysiology, 25,* 503-511.

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

Meesters, C. M. G., & Smulders, J. (1994). Hostility and myocardial infarction in men. *Journal of Psychosomatic Research, 38,* 727-734.

Mente, A. & Helmers, K. F. (1999). Defensive hostility and cardiovascular responses to

stress in young men. *Personality and Individual Differences, 27,* 683-694.

Miller, S. B. (1993). Cardiovascular reactivity in anger-defensive individuals: The influence of task demands. *Psychosomatic Medicine, 55,* 78-85.

Miller, S. B., Dolgoy, L., Friese, M. & Sita, A. (1996). Dimensions of hostility and cardiovascular response to interpersonal stress. *Journal of Psychosomatic Research, 41,* 81-95.

Miller, T. Q., Markides, K. S., Chiriboga, D. A., & Ray, L. A. (1995). A test of the psychosocial vulnerability and health behavior models of hostility: Results from an 11-year follow-up study of Mexican Americans. *Psychosomatic Medicine, 57,* 572-581.

Miller, T. Q., Smith, T. W., Turner, C. W., Guijarro, M. L., & Hallet, A. J. (1996). A meta-analytic review of research on hostility and physical health. *Psychological Bulletin, 119,* 322-348.

Musante, L., MacDougall, J. M., Dembroski, T. M., & Costa, P.T. (1989). Potential for hostility and dimensions of anger. *Health Psychology, 8,* 343-354.

Musante, L., Treiber, F. A., Davis, H., Strong, W. B., & Levy, M. (1992). Hostility: Relationship to lifestyle behaviors and physical risk factors. *Behavioral Medicine, 12,* 21-26.

Niaura, R., Herbert, P. N., McMahon, N., & Sommerville, L. (1992). Repressive coping and blood lipids in men and women. *Psychosomatic Medicine, 54,* 698-706.

O'Malley, P. O., Jones, D. L., Feuerstein, I. M., & Taylor, A. J. (2000). Lack of correlation between psychological factors and subclinical coronary artery disease. *The New England Journal of Medicine, 343,* 1298-1304.

Orlando, J., Aronow, W. S., Cassidy, J., & Prakash, R. (1976). Effects of ethanol on angina pectoris. *Annals of International Medicine, 84,* 652.

Paulhus, D. L. (1994). Two-component models of socially desirable responding. *Journal of Personality and Social Psychology, 46,* 5598-609.

Phipps, S., & Srivastava, D. K. (1997). Repressive adaptation in children with cancer. *Health Psychology, 16,* 521-528.

Piferi, R. L., & Lawler, K. A. (2000). Hostility and the cardiovascular reactivity of women during interpersonal confrontation. *Women and Health, 30,* 111-129.

Polefrone, J. M., & Manuck, S. B. (1988). Effects of menstrual phase and parental history of hypertension on cardiovascular response to cognitive challenge. *Psychosomatic Medicine, 50,* 23-23.

Powell, L. H., & Thoresen, C. E. (1985). Behavioral and physiologic determinants of long-term prognosis after myocardial infarction. *Journal of Chronic Diseases, 38,* 253-263.

Prkachin, K. M., & Silverman, B. E. (2002). Hostility and facial expression in young men and women: Is social regulation more important than negative affect? *Health Psychology, 21,* 33-39.

Ragland, D. R., Brand, R. J., & Rosenman, R. H. (1987). *Type A/B behavior pattern: CHD and non-CHD outcomes in the Western Collaborative Study Group.* Paper presented at annual meetings of the Society for Behavioral Medicine, Washington, DC.

Raikkonen, K., & Keltikangas-Jarvinen, L. (1991). Hostility and its association with behaviorally induced and somatic coronary risk indicators in Finnish adolescents

and young adults. *Social Science Medicine, 33*, 1171-1178.

Ranchor, A. V., Sanderman, R., Bouma, J., Buunk, B. P., van den Heuvel, W. J. A. (1997). An exploration of the relation between hostility and disease. *Journal of Behavioral Medicine, 20*, 223-240.

Richards, J. C., Hof, A., & Alvaregna, M. (2000). Serum lipids and their relationships with hostility and angry affect and behaviors in men. *Health Psychology, 19*, 393-398.

Romanov, K., Hatakka, M., Keskinen, E., Laaksonen, H., Kaprio, J., Rose, R. J., & Koskenvuo, M. (1994). Self-reported hostility and suicidal acts, accidents, and accidental deaths: A prospective study of 21,443 adults aged 25 to 29. *Psychosomatic Medicine, 56*, 328-336.

Rosenberg, E., Ekman, P., & Blumenthal, J. (1998). Facial expression and the affective component of cynical hostility in male coronary heart disease patients. *Health Psychology, 17,* 376-380.

Rosenman, R. H. (1983). Current status of risk factors and Type A behavior pattern in the pathogenesis of ischemic heart disease. In T. M. Dembroski, S. M. Weiss, J. L. Shields, S. G. Haynes & M. Feinlab (Eds.), *Coronary prone behavior*. New York: Springer-Verlag.

Rosenman, R. H., Brand, R. J., Jenkins, C. D., Friedman, M., Straus, 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.

Rosenman, R. H., Friedman, M., Straus, R., Wurm, M., Kositchek, R., Hahn, N., &

Wethesson, N. T. (1964). A predictive study of coronary heart disease: The Western Collaborative Group Study. *Journal of the American Medical Association, 189,* 15-22.

Rubin, E. (1980). Cardiovascular effects of alcohol. *Pharmacological Biochemical Behavior, 13,* 37-40.

Sallis, J. F., Johnson, C. C., Trevorrow, T. R., Kaplan, R. M., & Hovell, M. F. (1987). The relationship between cynical hostility and blood pressure reactivity. *Journal of Psychosomatic Research, 31,* 111-116.

Scherwitz, L., McKelvain, R., Laman, C., Patterson, J., Dutton, L., Yusim, S., Lester, J., Kraft, I., Rochelle,D., & Leachman, R. (1983). Type A behaviour, self-involvement, and coronary atherosclerosis. *Psychosomatic Medicine, 10,* 173-195.

Scherwitz, L. W., Perkins, L. L., Chesney, M. A., Hughes, G. H., Sidney, S., & Manolio, T. A. (1992). Hostility and health behaviors in young adults: The CARDIA study. *American Journal of Epidemiology, 136,* 136-145.

Schonwetter, D. J., & Janisse, M. P. (1991). Alcohol consumption, anger and hostility: A link to coronary heart disease. *Personality and Individual Differences, 12,* 1049-1955.

Schwartz, G. E. (1990). Psychobiology of repression and health: A systems approach. In J. L. Singer (Ed.), *Repression and dissociation: Implications for personality theory, psychopathology, and health* (pp. 405-434). Chicago: University of Chicago Press.

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.

Shapiro, D., Goldstein, I. B., & Jamner, L. D. (1996). Effects of cynical hostility, anger out, anxiety, and defensiveness on ambulatory blood pressure in black and white college students. *Psychosomatic Medicine, 58,* 354-364.

Shapiro, D., Jamner, L. D., & Goldstein, I. B. (1993). Ambulatory stress psychophysiology: The study of "compensatory and defensive counterforces" and conflict in a natural setting. *Psychosomatic Medicine, 55*, 309-323.

Shapiro, P. A., Sloan, R. P., Bagiella, E., Kuhl, J. P., Anjilvel, S., & Mann, J. J. (2000). Cerebral activation, hostility, and cardiovascular control during mental stress. *Journal of Psychosomatic Research, 48,* 485-491.

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

Shekelle, R. B., Hulley, S., Neaten, J., Billings, J., Borhani, N., Gerace, T., Jacobs, D., Lasser, N., Mittlemark, M., & Stamler, J. (1985). The MRFIT behavior pattern study: II. Type A behavior pattern and incidence of coronary heart disease. *American Journal of Epidemiology, 122*, 559-570.

Sherwood, A., Allen, M. T., & Fahrenberg, J. (1990). Methodological guidelines for impedance cardiography. *Psychophysiology, 27*, 1-23.

Siegler, I. C. (1994). Hostility and risk: Demographic and lifestyle variables. In A. W. Siegman & T. W. Smith (Eds.), *Anger, hostility and the heart* (pp. 199-237). Hillsdale, NJ: Lawrence Erlbaum Associates.

Siegler, I. C., Peterson, B. L., Barefoot, J. C., & Williams, R. B. (1992). Hostility during

late adolescence predicts coronary risk factors at mid-life. *American Journal of Epidemiology, 136*, 146-154.

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

Siegman, A. W., & Anderson, R. W. (1990). Dimensions of hostility and cardiovascular reactivity: The role of challenge. *Psychosomatic Medicine, 52,* 235-236.

Siegman, A. W., Anderson, R., Herbst, J., Boyle, S., & Wilkinson, B. (1992). Dimensions of anger-hostility and cardiovascular reactivity in provoked and angered men. *Journal of Behavioral Medicine, 15*, 257-272.

Siegman, A. W., Dembroski, T. M, & Ringel, N. (1987). Components of hostility and the severity of coronary artery disease. *Psychosomatic Medicine, 49*, 127-135.

Siegman, A. W., Feldstein, S., Tommaso, C., Ringel, N., & Lating, J. (1987). Expressive vocal behavior and the severity of coronary artery disease. *Psychosomatic Medicine, 49*, 545-561.

Siegman, A. W., Townsend, S. T., Civelek, A. C., & Blumenthal, R. S. (2000). Antagonistic behavior, dominance, hostility, and coronary heart disease. *Psychosomatic Medicine, 62,* 248-257.

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

Smith, T. W. (1992). Hostility and health: Current status of a psychosomatic hypothesis. *Health Psychology, 11*, 139-150.

Smith, T. W. (1994). Concepts and methods in the study of anger, hostility, and health.

In A. W. Siegman & T. W. Smith (Eds.), *Anger, hostility and the heart* (pp. 23-42). Hillsdale, NJ: Lawrence Erlbaum Associates.

Smith, T. W., & Allred, K. D. (1989). Blood pressure responses during social interaction in high and low cynically hostile males. *Journal of Behavioral Medicine, 12,* 135-143.

Smith, T. W., & Brown, P. C. (1991). Cynical hostility, attempts to exert social control, and cardiovascular reactivity in married couples. *Journal of Behavioral Medicine, 14,* 581-592.

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.

Sommers-Flanagan, J., & Greenberg, R. P. (1989). Psychosocial variables and hypertension: A new look at an old controversy. *Journal of Nervous Mental Disorders, 177,* 15-24.

Spielberger, C. D., Johnson, E. H., Russell, S. F., Crane, R. J., Jacobs, G. A., & Worden, T. J. (1985). The experience and expression of anger: Construction and validation of an anger expression scale. In M. A. Chesney, & R. H. Rosenman (Eds.), *Anger and hostility in cardiovascular and behavioral disorders* (pp. 5-29). Washington, DC: Hemisphere.

Suarez, E. C., Bates, M. P., & Harralson, T. L. (1998). The relation of hostility to lipids and lipoproteins in women: Evidence for the role of antagonistic hostility. *Annals of Behavioral Medicine, 20*, 59-63

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.

Suarez, E. C., Kuhn, C. M., Schanberg, S. M., Williams, R. B., & Zimmermann, E. A. (1998). Neuroendocrine, cardiovascular, and emotional responses of hostile men: The role of interpersonal challenge. *Psychosomatic Medicine, 60,* 78-88.

Suarez, E. C., & Williams, R. B. (1989). Situational determinants of cardiovascular and emotional reactivity in high and low hostile men. *Psychosomatic Medicine, 51,* 404-418.

Suarez, E. C., & Williams, R. B. (1990). The relationships between dimensions of hostility and cardiovascular reactivity as a function of task characteristics. *Psychosomatic Medicine, 52,* 558-570.

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

Taylor, J. A. (1953). A personality scale of manifest anxiety. *Journal of Abnormal and Social Psychology, 48*, 285-290.

Taylor, S. E., & Brown, J. D. (1988). Illusion and well-being: A social psychological perspective on mental health. *Psychological Bulletin, 103*, 193-210.

Temoshok, L., (1987). Personality, coping style, emotion and cancer: Toward an integrative model. *Cancer Surveys, 6,* 545-567.

Turlapaty, F. D. M. V., & Altura, B. M. (1980). Magnesium deficiency produces spasms of coronary arteries: Relationship to etiology of sudden death ischemic heart disease. *Science, 208,* 198-200.

Warrenburg, S., Levine, J., Schwartz, G. E., Fontana, A. F., Kerns, R. D., Delaney, R., & Mattson, R. (1989). Defensive coping and blood pressure reactivity in medical patients. *Journal of Behavioral Medicine, 12*, 407-424.

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

Weidner, G., Sexton, G., McLellarn, R., Connor, S. L., & Matarazzo, J. D. (1987). The role of Type A behavior and hostility in an elevation of plasma lipids in adult women and men. *Psychosomatic Medicine, 48,* 136-145.

Weinberger, D. A. (1990). The construct validity of the repressive coping style. In J. L. Singer (Ed.), *Repression and dissociation: Implications for personality theory, psychopathology, and health* (pp. 337-386). Chicago: University of Chicago Press.

Weinberger, D. A., Schwartz, G. E., & Davidson, R. J. (1979). Low-anxious, high-anxious, and repressive coping styles: Psychometric patterns and behavioral and physiological responses to stress. *Journal of Abnormal Psychology, 88*, 369-380.

Weisinger, H. D. (1985). *Dr. Weisinger's anger work-out book. New York: William Morrow and Company, Inc.*

Whitney, E. N., & Rolfes, S. R. (1996). *Table of food composition: Appendix H.* Understanding Nutrition (7th ed.). (pp. H1–H113). St. Paul, MN: West Publishing Company.

Wickramasekera, I. (1995). Somatization: Concepts, data and predictions from the high risk model of self-perception. *Journal of Nervous and Mental Disease, 183*, 15-23.

Willett, W. C., Sampson, L., Stampfer, M. J., Rosner, B., Bain, C., Witschi, J., Hennekens, C. H., & Speizer, F. E. (1985). Reproducibility and validity of a semiquantitative food frequency questionnaire. *American Journal of Epidemiology, 122*, 51-65.

Williams, R. B., Barefoot, J. C., Haney, T. L., Harrell, F. E., Blumenthal, J. A., Pryor, D. B., & Peterson, B. (1988). Type A behavior and angiographically documented coronary atherosclerosis in a sample of 2,289 patients. *Psychosomatic Medicine, 50,* 139-152.

Williams, R. B., Barefoot, J. C., & Shekelle, R. B. (1985). The health consequences of hostility. In M. A. Chesney, & R. H. Rosenman (Eds.), Anger and hostility in cardiovascular and behavioral disorders (pp. 173-185). Washington, DC: Hemisphere.

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

Williams, R. B., Suarez, E. C., Kuhn, C. M., Zimmerman, E. A., & Schanberg, S. M. (1991). Biobehavioral basis of coronary-prone behavior in middle-aged men. Part 1: Evidence for chronic SNS activation in Type As. *Psychosomatic Medicine, 53*, 517-527

Wirsching, M., Stierlin, H., Hoffman, F., Weber, G., & Wirsching, B. (1982). Psychological identification of breast cancer patients before biopsy. *Journal of Psychosomatic Research, 26*, 1-10.

Appendix A

Screening Health Questionnaire

Screening Health Questionnaire

Name: _____ Age: _____ Phone: _____

Major subject studied: _____
Minor subject studied: _____

Please answer all of the following questions carefully.

Have you had any medical or surgical problems during the last year? Yes ____ No ____
Please specify _____

Do you suffer from any chronic illnesses? Yes ____ No ____
Please specify _____

Have you ever had heart trouble of any kind? Yes ____ No ____
Please specify _____

Do you have diabetes? Yes ____ No ____

Have you ever had kidney trouble of any kind? Yes ____ No ____
Please specify _____

Do you suffer from epilepsy? Yes ____ No ____

Have you ever had liver trouble of any kind? Yes ____ No ____
Please specify _____

Do you have asthma? Yes ____ No ____

Do you now suffer from bronchitis or do you suffer from chronic bronchitis?
Yes ____ No ____

Have you ever had a fainting spell? Yes ____ No ____
If yes, please explain _____

Are you presently, or have you ever been treated for psychological or psychiatric reasons? Yes ____ No ____ If yes, please explain briefly _____
_____

Please list any medication that you are presently taking and the reason for taking it
_____

Please give the date (or approximate date) of your last medical check-up _____

Signature: _____ Date: _____

Appendix B

Cook Medley Hostility Scale

Subject #_____
Date _____

Cook Medley Hostility Scale

This questionnaire consists of numbered statements. Read each statement and decide whether it is true as applied to you or false as applied to you. If the statement is TRUE or MOSTLY TRUE, circle the (T). If it is FALSE or NOT USUALLY TRUE, circle the (F). Remember to give your own opinion of yourself. Do no leave any black spaces if you can avoid it.

(T) (F) 1. When I take a new job, I like to be tipped off on who should be gotten next to.

(T) (F) 2. When someone does me a wrong I feel I should pay him back if I can, just for the principle of the thing.

(T) (F) 3. I prefer to pass by school friends, or people I know but have not seen for a long time, unless the speak to me first.

(T) (F) 4. I have often had to take orders from someone who did not know as much as I did.

(T) (F) 5. I think a great many people exaggerate their misfortunes in order to gain the sympathy and help of others.

(T) (F) 6. It takes a lot of argument to convince most people of the truth.

(T) (F) 7. I think most people would lie to get ahead.

(T) (F) 8. Someone has it in for me.

(T) (F) 9. My relatives are nearly all in sympathy with me.

(T) (F) 10. Most people are honest chiefly through fear of being caught.

(T) (F) 11. Most people will use somewhat unfair means to gain profit or an advantage rather than to lose it.

(T) (F) 12. I commonly wonder what hidden reason another person may have for doing something nice for me.

(T) (F). 13. It makes me impatient to have people ask my advice or otherwise interrupt me when I am working on something important.

(T) (F). 14. I feel that I have often been punished without cause.

(T) (F) 15. I am against giving money to beggars.

(T) (F) 16. Some of my family has habits that bother me and annoy me very much.

(T) (F) 17. My way of doing things is apt to be misunderstood by others.

(T) (F) 18. I can be friendly with people who do things which I consider wrong.

(T) (F) 19. I don't blame anyone for trying to grab everything he can get in this world.

(T) (F) 20. No one cares much what happens to you.

(T) (F) 21. It is safer to trust nobody.

(T) (F) 22. I do not blame a person for taking advantage of someone who lays himself open for it.

(T) (F) 23. I have often felt that strangers were looking at me critically.

(T) (F) 24. Most people make friends because friends are likely to be useful to them.

(T) (F) 25. I am sure I am being talked about.

(T) (F) 26. I am not likely to speak to people until they speak to me.

(T) (F) 27. Most people inwardly dislike putting themselves out to help other people.

(T) (F) 28. I tend to be on guard with people who are somewhat more friendly than I had expected.

(T) (F) 29. People often disappoint me.

(T) (F) 30. I have often met people who are supposed to be experts who were no better than I.

(T) (F) 31. It makes me feel like a failure when I hear of the success of someone I know well.

(T) (F) 32. I am not easily angered

(T) (F) 33. People generally demand more respect for their own rights than they are willing to allow for others.

(T) (F) 34. I am quite often not in on the gossip and talk of the group I belong to.

(T) (F) 35. I have often found people jealous of my good ideas, just because they had not thought of them first.

(T) (F) 36. I have sometimes stayed away from another person because I feared doing or saying something that I might regret afterwards.

(T) (F) 37. I would certainly enjoy beating a crook at his own game.

(T) (F) 38. I have at times had to be rough with people who were rude or annoying.

(T) (F) 39. There are certain people whom I dislike so much that I am inwardly pleased when they are catching it for something they have done.

(T) (F) 40. I am often inclined to go out of my way to win a point with someone who has opposed me.

(T) (F) 41. The man who had most to do with me when I was a child (such as my father, stepfather, etc.) was very strict with me.

(T) (F) 42. I like to keep people guessing what I'm going to do next.

(T) (F) 43. When a man is with a woman he is usually thinking about things related to her sex.

(T) (F) 44. I do not try to cover up my poor opinion or pity of a person so that he won't know how I feel.

(T) (F) 45. I strongly defend my own opinions as a rule.

(T) (F) 46. I frequently ask people for advice.

(T) (F) 47. I have frequently worked under people who seem to have things arranged so that they get credit for good works but are able to pass off mistakes onto those under them.

(T) (F) 48. People can pretty easily change me even though I thought that my mind was already made up on a subject.

(T) (F) 49. Sometimes I am sure that other people can tell what I am thinking.

(T) (F) 50. A large number of people are guilty of bad sexual conduct.

Appendix C

Marlowe Crowne Social Desirability Scale

Subject #_____
Date _____

Marlowe Crowne Social Desirability Scale

Listed below are a number of statements concerning personal attitudes. Read each item and circle the statement TRUE (T) or FALSE (F) as it pertains to you personally.

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

18. I don't find it particularly difficult to get along with loud mouthed, obnoxious people. T / F

19. I sometimes try to get even, rather than forgive and forget. T / F

20. When I don't know something I don't at all mind admitting it. T / F

21. I am always courteous, even to people who are disagreeable. T / F

22. At times I have really insisted on having things my own way. T / F

23. There have been occasions when I felt like smashing things. T / F

24. I would never think of letting someone else be punished for my wrongdoing. T / F

25. I never resent being asked to return a favor. T / F

26. I have never been irked when people expressed ideas very different from my own. T / F

27. I never make a long trip without checking the safety of my car. (Answer even if you don't have a car) T / F

28. There have been times when I was quite jealous of the good fortune of others. T / F

29. I have almost never felt the urge to tell someone off. T / F

30. I am sometimes irritated by people who ask favors of me. T / F

31. I never felt that I was punished without cause. T / F

32. I sometimes think when people have misfortunes they only got what they deserved. T / F

33. I never deliberately said something that hurt someone's feelings. T / F

Appendix D

State Affect Questionnaire

Subject Number: _____ **1 2 3**

HOW ARE YOU FEELING RIGHT NOW?

Indicate on each of the scales below by marking a vertical stroke through the line at the appropriate point.

Not at all Agreeable _____ Very Agreeable

Not at all Happy _____ Very Happy

Not at all Tense _____ Very Tense

Not at all Anxious _____ Very Anxious

Not at all Discouraged _____ Very Discouraged

Not at all Irritated _____ Very Irritated

Not at all Angry _____ Very Angry

Not at all Depressed _____ Very Depressed

Not at all Guilty _____ Very Guilty

Appendix E

General Health Survey

GENERAL HEALTH SURVEY

Please answer the following questions to the best of your ability. All of the information will be kept confidential.

Name: _____ (please print)

Address: _____
Street City Province Postal Code

Telephone Number(s): _____ (home) _____ (work)

Date of Birth _____/_____/_____ Age: _____
Month Day Year

1. Sex: Male _____ Female _____

2. Marital Status:
   Single, never married _____
   Living with partner _____
   Married _____
   Separated _____
   Divorced _____
   Widowed _____

3. Living arrangements: Alone _____ Roommates(s) _____ Family _____

4. Race: White _____ Black _____ Asian _____ Hispanic _____
   Native _____ Other _____ (specify)

5. Weight: _____ (pounds) Height: _____ (inches)

6. How many cups of caffeineated beverages (coffee, tea or cola) do you typically consume in an average day?

   None _____ 1 cup _____ 2-3 _____ 4-5 _____ 6 or more cups _____

7. How much alcohol (beer, wine and/or hard liquor) do you consume in a typical day?

   None _____ Occasional drink _____ 1-2 drinks _____ 3-4 drinks _____

Please read the following 6 statements that describe different patterns of alcohol use:

1. I used to drink but I quit OR I never or almost never drink
2. I drink only on special occasions.
3. I drink about one drink a week.
4. I have about one drink a day.
5. I have several drinks on weekends but one a day or less on weekdays.
6. I have several drinks a day, every day.

8. Which of these 6 statements best describes your use of BEER? _____

9. Which of these 6 statements best describes your use of WINE? _____

10. Which of these 6 statements best describes your use of HARD LIQUOR? _____

11. Smoking Status: 1. Current Smoker _____
    2. Non-Smoker (never smoked) _____
    3. Past Smoker (smoked & quit) _____

12. If you are a current smoker, approximately how many cigarettes do you smoke in an average day? _____

13. What age did you start smoking cigarettes? _____

14. How many times have you tried to quit? _____

15. How many total years have you smoked, subtracting out years you may have quit in between? _____

16. If you are a past smoker, approximately how many cigarettes did you smoke in an average day?

17. What age did you start smoking cigarettes? _____

18. What age did you quit smoking? _____

19. How many total years did you smoke, subtracting out years you may have quit in between? _____

Date: _____

Appendix F

Take-Home Monitoring Instructions

Take-Home Monitoring Instructions

The food diary is to be completed on:

One (1) weekday (either Monday, Tuesday, Wednesday, Thursday or Friday)

And

One (1) weekend-day (Saturday or Sunday)

Your Food Diary Kit consists of:

- 1 Food Scale
- Measuring cups
- Measuring spoons

Please describe, measure and/or weight everything you eat and drink and record it on the provided paper. It is extremely important to provide as much detail as possible. If you eat mixed dishes (e.g., lasagna, sandwiches, hamburgers), please record all the ingredients and their amounts. If you know the amount of fat grams in the food, please record it.

Don't forget to document:

- Fats and oils
- Salad dressings, ketchup, mayonnaise, sauces
- Brand names (e.g., Campbells, Big Mac, Cheerios)
- Cooked or raw foods
- Skin or skinless meats

If you have any questions, feel free to call:

Lab: 848-2846 (ask for Lisa or Marianne)

Appendix G

Food Diaries: Weekday & Weekend-day

Food Diary
A Typical Weekday

Please weigh/measure all you eat and drink for the whole day and record it below

---

FOOD & DRINK LIST – WEEKDAY

_____	_____
_____	_____
_____	_____
_____	_____
_____	_____
_____	_____
_____	_____
_____	_____
_____	_____
_____	_____
_____	_____
_____	_____

How many cigarettes did you smoke today? _____ Brand Name: _____

How much alcohol did you drink today?

Beer _____ Brand Name _____
Hard Liquor (1½ oz) _____ Brand Name _____
Wine (5 oz. glass) Dry Red _____ Dry White _____
Sweet Red _____ Sweet White _____

How much caffeineated coffee/tea did you drink today?
cups of coffee _____
cups of tea _____
cups of herbal tea (no caffeine) _____

How much caffeineated soft drinks/cola did you drink today?
_____ (specify cans, bottles, etc.) Brand Name: _____

Food Diary
A Typical Weekend-Day

Please weigh/measure all you eat and drink for the whole day and record it below

---

FOOD & DRINK LIST – SATURDAY OR SUNDAY

_____	_____
_____	_____
_____	_____
_____	_____
_____	_____
_____	_____
_____	_____
_____	_____
_____	_____
_____	_____
_____	_____

How many cigarettes did you smoke today? _____ Brand Name: _____

How much alcohol did you drink today?

Beer _____ Brand Name _____
Hard Liquor (1½ oz) _____ Brand Name _____
Wine (5 oz. glass) Dry Red _____ Dry White _____
Sweet Red _____ Sweet White _____

How much caffeineated coffee/tea did you drink today?
cups of coffee _____
cups of tea _____
cups of herbal tea (no caffeine) _____

How much caffeineated soft drinks/cola did you drink today?
_____ (specify cans, bottles, etc) Brand Name: _____

Appendix H

Consent Form

Consent Form

RESEARCH STUDY CONDUCTED AT CONCORDIA UNIVERSITY, DEPARTMENT OF PSYCHOLOGY ON BEHALF OF DR. SYDNEY MILLER

We would like you to participate in a study investigating the effects of nutrition, stress and performance on cardiovascular reactivity. In particular the study requires you to:

- Keep a food diary for two days (one weekday and one weekend-day)
- Fill in the complete questionnaire package.

- In addition, you will be required to come to our lab for one session, lasting approximately 2 ½ hours. During the session you will engage in a task that involves making a decision on several mathematical solutions. Throughout the session we will obtain various physiological measures (e.g., heart rate, blood pressure, cardiac output, etc.). These physiological recordings are safe, painless and non-invasive and only require the placement of various transducers on the skin. During the lab-session changes such as increases in heart rate and blood pressure will occur. These increases, however, will only be temporary, returning to normal after the experiment and causing no adverse effects.

You will be paid $ 50.00 for your participation only if you have completed all the above mentioned requirements.

All information we obtain about you is completely confidential and will not be seen by anyone who is not a member of the research team. Ultimately, all data will be coded using subject numbers rather than names.

You are free to withdraw from the experiment at any time.

We ask you not to discuss the experiment with other individuals who are participating in the study.

Once you have carefully studied and understood this form, you may sign it in indication of your free consent and agreement to participate in the study.

NAME (PLEASE PRINT): _____

Signature: _____

Date: _____

Appendix I

Math-Task Instructions

Math-Task Instructions

You will be presented with a series of mathematical subtraction equations on the monitor. You must respond by pressing the right button down (with the C on it) if you think the answer on the screen is correct or by pressing the left button down (with the I on it) if you think the answer on the screen is incorrect.

If you are accurate in your response, that is, you say the answer on the screen is correct and it is, or you say it is incorrect and it is, you will hear a high-pitched tone. If you respond incorrectly, that is, if you say the answer on the screen is correct and it is incorrect, or if you say it is incorrect and it is correct, you will hear a low-pitched tone indicating that you have not responded accurately.

You have 3 seconds to respond to every equation. When the mathematical equation turns yellow, you have 1 second left to respond. If you are still unsure at this point - guess. A non-response is considered an incorrect response.

Appendix J

Harassment Preparation Scenario

Harassment Preparation Scenario

While Researcher A (female) is explaining the math-task instructions to the subject, the phone rings. Researcher B (male) enters the testing room to tell Researcher A that her supervisor is on the phone.

B: "Dr. Miller is on the phone."

A: "Just a minute please."

Researcher A completes the instructions, excuses herself and exists to the adjacent room, leaving the door ajar. In a loud voice Researcher A pretends to talk on the phone.

A: "Hello Dr. Miller. Right now? Well, I'm running a subject right now. Oh, okay, I'll ask if _____ (Researcher B) can take over for me. Okay, thanks, bye."

Researcher B pretends to be angry at Researcher A.

B: "Now what?!" (angrily)

A: Shhh! (pause) - that was Dr. Miller."

B: "And?"

A: "He wants to see me right away."

B: "Now? But you have a subject in there!"

A: "I know – but it sounds really important – would you mind taking over for me?"

B: "Look – I won't be responsible if your results screw up!"

A: "Don't worry – nothing will go wrong – everything is set up in there – just follow the instructions."

B: "I don't normally deal with the subjects – that's your job you know!"

A: "You know I wouldn't ask you if I didn't have to – everything will be fine – okay? (pause) Thanks, I'll be back as soon as I can."

Researcher A returns to the testing room and tells the subjects that she must leave and that another researcher will be taking her place. Researcher A then leaves the testing room and Researcher B soon enters it, pretending to be angry.

Appendix K

Harassment Statements

Harassment Statements

The 9-minute subtraction math-task consisted of three 3-minute trials. During each trial, two harassment statements were delivered, one at the beginning, and the other halfway through each 3-minute trial.

Trial 1

1. Did you understand the instructions?
2. The right button is correct, the left button is incorrect.

Trial 2

3. Could you try harder this time!
4. Can't you do better than this?

Trial 3

5. It isn't that hard you know!
6. I can do better than that!

Appendix L

Non-Harassment Preparation Scenario

Non-Harassment Preparation Scenario

While Researcher A (female) is explaining the math-task instructions to the subject, the phone rings. Researcher B (male) enters the testing room to tell Researcher A that her supervisor is on the phone.

B: "Dr. Miller is on the phone."

A: "Just a minute please."

Researcher A completes the instructions, excuses herself, and exits to the adjacent room. Researcher A soon returns and explains to the subject that her supervisors wants to see her and that another researcher will be taking her place. Researcher A then leaves the testing room. Researcher B soon enters the testing room and is friendly towards the subject throughout the math-task.