THE EFFECTS OF A HOSTILITY-REDUCTION TREATMENT ON HOSTILITY AND HEALTH MEASURES OF HIGH-HOSTILE STUDENTS AND CARDIAC PATIENTS: MATCHED-RANDOMIZED-CONTROLLED TRIALS.

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Yori (Yoram) Gidron

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This work is dedicated to my late father, Michael Gidron, for having struggled with courage against foreign hostility for the image of the state of Israel, until he succumbed to internal hostility, to my friend Udi Savitski, who lost his life, smiles and melody due to incidental hostility, to my mother Erika Gidron, for many more years of good health and humor, to my dear wife Laure-Anne Thieren Gidron and my dear son Aviv (Emile) Gidron, for many more years of loving and building together, and to the students and cardiac patients who took part in this study, for many more years of health.

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Abstract

Medical interventions for preventing coronary heart disease (CHD) typically target traditional risk factors (e g , cholesterol, blood-pressure or BP) As these risk factors partially predict CHD, medical interventions may only partially prevent CHD Hostility, the tendency to behave antagonistically, think cynically and feel anger, predicts CHD independently of traditional risk factors and better than the original "coronary-prone" Type-A behavioral pattern Hostility is cross-sectionally and prospectively significantly related to CHD outcomes (e.g myocardial infarction or MI) However no psychological treatment focusing on hostility alone has been tested with CHD patients Thus, the purposes of this research were to develop a brief cognitive-behavioral hostility-treatment focusing on antagonism, cynicism and anger and to test its efficacy at altering CHD-predictive hostility and CHD related outcomes Self-reported and observed hostility measures were employed in two single-blind matched-randomized-controlled trials In Study 1, 22 high-hostile healthy males were matched on age and hostility and then randomly assigned to the hostility-treatment (N = 11) or to an information control group (N = 11) After controlling for pre-treatment levels subjects group status accounted for an additional and significant 19% and 28% of the variance in change scores of self-reported and observed hostility, respectively Reactive-BP was not affected in the hypothesized manner Study 2 replicated and extended Study 1 by including a two month follow-up and CHD-related measures (e g , resting-BP quality of life), and by employing CHD patients Twenty-two high-hostile CHD males were matched on age and hostility and then randomly assigned to the hostility-treatment (N = 10) or to a control group (N = 12) After controlling for pre-treatment levels patients group status accounted for an additional and significant 20% of the variance in change scores of self-reported hostility at post-treatment, and 18% of the variance in change scores of observed hostility at follow-up At post-treatment only a significantly lower percentage of treatment patients (10%) were hypertensives than controls (50%) Patients' group status accounted for an additional and significant 28% and 16% of the variance in increased life-satisfaction and reduced depression scores, respectively, and this was maintained at follow-up Finally, reduction in hostility was significantly correlated with improvements in resting-BP life satisfaction and depression In conclusion, the hostility treatment repeatedly reduced self-reported and observed CHD-predictive hostility and positively affected resting-BP and quality of life. Evidence for causal relations between hostility and CHD-related measures support the etiological role of hostility in CHD However, the samples were small and many statistical tests were conducted. Future trials with larger samples and long-term outcomes (e g., MI) should test the treatment's preventative value. It is hypothesized that epinephrine may mediate the hostility-BP relation and that social support may mediate the hostility-quality of life relation

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To all of you I salute and say in a loud voice Toda

GENERAL INTRODUCTION

Coronary heart disease (CHD) is a major cause of morbidity and mortality in most western countries (Jenkins, 1988). Thus, it is critical to identify significant and independent CHD risk factors, and to develop interventions that target these factors for the prevention and treatment of CHD. Medical interventions for preventing CHD and several medical interventions for treating CHD typically target traditional risk factors (e.g., smoking, cholesterol, blood-pressure or BP). As these risk factors only partially predict CHD (Keys et al., 1972; Leon et al., 1988), medical interventions may be insufficient for preventing or treating CHD. Recognizing the limited impact of traditional risk factors, and observing recurrent psychological patterns in CHD patients motivated the search for psychological risk factors for the development of CHD. The role of emotional factors in health in general, and of anger and hostility in cardiac diseases in particular, has a long history (Siegman, 1994). The book of Ecclesiastics or Kohelet (11;10) tells us: "Remove anger from your heart and shift evil from your body", implying that already in biblical times, a causal relation was hypothesized to exist between anger and physical health.

Modern and systematic research into the role of psychological factors in CHD was first conducted by Friedman and Rosenman (1959) by identifying a pattern of behaviors they found to be common in their CHD patients, the Type-A Behavioral Pattern (TABP). However, after much work was done on the relation between the TABP and CHD, several problems began to emerge: a)

Negative findings resulted from well-designed studies (e.g., Shekelle et al., 1985); b) The prevalence of the TABP exceeded that of CHD (Dembroski & Costa, 1987) and c) Hostility, one of the components of the TABP, emerged as more strongly related to CHD than the TABP (e.g., Williams et al., 1980). These findings shifted the focus in behavioral cardiology to hostility and its impact on CHD. Indeed, the majority of evidence supports this relation (Smith, 1992).

However, despite these advances in understanding "coronary-prone behavior", there has been relatively little advance in modifying significant psychological risk factors for CHD (Deffenbacher, 1994). Although there have been a few successful clinical trials that modified the global TABP and reduced cardiac events (e.g., Friedman et al., 1986), to the best of my knowledge, no clinical trial has attempted to test the effects of modifying hostility alone on the health of CHD patients. Thus, despite epidemiological advances in theory, behavioral cardiology did not similarly progress in applied interventions. In addition to the importance of applying observed relations between risk factors and health to patients' well being, clinical trials provide a unique opportunity to test hypothesized **causal** relations between risk factors and health outcomes.

Therefore, the purpose of this thesis was threefold: a) To develop a theoretically and empirically derived brief treatment to reduce components of hostility that have been associated with CHD; b) To test the efficacy of this treatment at reducing hostility levels of healthy, but high-hostile, students, and

c) To test the efficacy of this treatment at reducing hostility levels and improving the short-term health status of high-hostile CHD patients. This thesis included two single-blind, matched-randomized-controlled clinical trials, for preliminarily testing the effects of this new hostility-treatment on hostility and CHD-related measures. Rather than conducting one large clinical trial, two, small-scale trials were conducted, since recruitment within this subject population was expected to be difficult, and since replication may be more important for demonstrating a treatment's efficacy than tests of statistical significance alone.

The first five chapters of this thesis are introductory chapters that provide the background for the two clinical trials. Chapter One examines the epidemiology and pathophysiology of CHD. Chapter Two reviews in detail the definition, assessment and epidemiology of hostility. Chapter Three, the basis of this thesis, critically reviews the majority of the empirical literature on the link between hostility and several CHD end-points. Chapter Four examines the theoretical models that attempt to explain the hostility-CHD link and provides empirical examples supporting these models. Finally, Chapter Five critically reviews and evaluates previous clinical trials on anger and hostility with healthy individuals, hypertensive patients, and relevant clinical trials with CHD patients. Chapter Five also outlines the rationale and content of the hostility-reduction treatment developed in this thesis. Chapter Six presents the methods and results of the clinical trial with high-hostile, non-CHD, students. Chapter Seven

presents the methods and results of the clinical trial with high-hostile CHD patients. Finally, Chapter Eight reviews the major findings, integrates both clinical trials, provides possible explanations for observed causal relations, outlines the limitations of this research, and provides future research directions arising from this thesis in this area of health psychology.

CHAPTER ONE

EPIDEMIOLOGY AND PATHOPHYSIOLOGY OF CHD

Introduction

Health Psychology is the field of psychology which applies knowledge from psychological research and practice to the understanding, prediction and treatment of health problems. Understanding the basic medical background of a health problem is a necessary step for applying psychological knowledge in the most relevant manner to that health problem, and for understanding and communicating with medical professionals. Providing a comprehensive background of the pathophysiology of myocardial infarction (MI) and unstable angina pectoris (UA), the two disease groups included in Study 2, is beyond the scope of this thesis. However, this chapter will provide the basic medical background concerning the epidemiology of coronary heart disease (CHD), etiology, diagnosis and treatment of MI and UA.

Epidemiology of CHD

Epidemiology is the field of medicine that seeks to identify the distribution and risk factors of health states in populations (Jenkins, 1988). Thus, epidemiological studies provide the incidence (rate of new cases during a given period) and prevalence (total rate of continuing or new cases in a given period) of a disease, together with identifying its underlying causes.

Distribution of CHD. Cardiovascular diseases (CVD), which include CHD as well as other forms of CVD (e.g., congestive heart failure), are the

leading cause of death in industrialized countries (Jenkins, 1988). The most prevalent CVD diagnosis is CHD, which includes MI and UA. Coronary heart disease constitutes a disease in the myocardium due to certain processes in the coronary arteries (discussed below). In the U.S. alone, CHD accounts for approximately half of the deaths related to CVD (Jenkins, 1988; Sokolow & McLlory, 1986). In the U.S. in 1978, 642,000 deaths were attributed to CHD alone. There have been declines in these numbers in many industrialized countries mainly due to advancements in treatment, while in East European countries (Bulgaria, Poland) there has been an increase in the incidence of CHD (World Health Organization, WHO, 1982). Despite this trend, 23% of world total mortality is from CVD, with higher percentages in developed countries (48%) than in developing countries (16%; WHO, 1983).

The majority of patients develop CHD due to coronary atherosclerosis (discussed below; Perkins, 1989); a third develop a form of angina pectoris, half develop a MI, and a fifth may suddenly die (Plotnick, 1985a). However, these diseases may overlap, and a patient with UA may have a MI or even die suddenly. Any form of CHD calls for special attention since sudden death may occur without any prodromal signs (Sokolow & McLlory, 1986).

Traditional CHD risk factors. Most information concerning the risk factors for developing CHD comes from prospective studies. Risk factors are more likely to be causal if they precede the disease, strongly predict it, are dose related, are consistent in their effect within and across populations, are

independent of other risk factors, are pathologically and theoretically plausible, and finally if their effects are supported by experimental studies (Kannel, 1983). Several large scale epidemiological studies have been conducted in order to identify the major CHD risk factors. The Multiple Risk Factor Intervention Trial (MRFIT; Kannel et al., 1986) screened 325,384 white middle-aged men who were free of CHD and followed them for six years. Hypertension, hypercholesterolemia and cigarette smoking were independent CHD risk factors across all age groups. These three are considered the major CHD risk factors (Perkins, 1989).

Hypertension causes arterial wall thickening and increased blood-flow velocity. Both processes cause endothelial hypoxia and injuries, the first steps in the atherosclerotic process (Perkins, 1989), which may lead to CHD. Hyperlipidemia exacerbates arterial injuries since low density lipoprotein (LDL) cholesterol induces smooth muscle cell proliferation from the inner to the outer arterial laminae, and thus, increases degree of arterial occlusion (as discussed below; Ross & Glomset, 1976). Cholesterol can penetrate injured arteries and increase arterial occlusions. Cigarette smoking may also lead to arterial wall injury by inducing arterial hypoxia, since carbon monoxide from cigarettes binds to plasma oxygen to reduce arterial-wall oxygen. Smoking is also associated with decreased coronary diameters (Perkins, 1989) which may initiate an ischemic event such as UA or MI.

Perkins (1989) demonstrated that certain interactions between the major

risk factors (e.g., hypertension and cholesterol) are more predictive of CHD than the additive effects of each risk factor in the absence of the other. Examining data of epidemiological studies (e.g., Kannel et al., 1986), Perkins (1989) showed that there were independent interaction effects between hypertension and cholesterol and between smoking and cholesterol in predicting CHD end-points. The observed CHD risk due to these interactions was at least twice the magnitude of the expected risk if the effects of the risk factors were additive. Most important, these interactions had a pathogenetic basis as well. Hypertension and smoking act alone in initiating arterial injuries, which then produce greater occlusions in the presence of penetrating cholesterol. Thus, hypertension and cholesterol, and smoking and cholesterol, may have synergistic effects on the pathogenesis of CHD (Perkins, 1989).

Rose (1982) showed that there is a 10-year delay or "incubation-period" between exposure to traditional CHD risk factors and CHD-related mortality. This was seen by observing that the correlation between base-line cholesterol, systolic blood-pressure (SBP) and diastolic blood-pressure (DBP) on the one hand, and CHD-related mortality on the other hand, grew steadily during 15 years of observation. However, this study did not follow the same individuals, but examined the predictive validity of a sample's base-line data and national CHD-deaths. Nevertheless, this study demonstrated the importance of duration of exposure to risk factors and manifestation of disease.

Age is also a CHD risk factor (Jenkins, 1988; Kannel et al., 1986). In

men, age is linearly related to CHD while in women this relation is weak prior to menopause and is strong thereafter (Jenkins, 1988). Age interacts with the major risk factors. The relative risk attributable to each of the three major risk factors (e.g., CHD-mortality in hypertensive versus normotensive people) decreases with age. However, the risk attributable to a major risk factor is highest among older people (e.g., CHD-mortality in older versus younger hypertensive people), mainly due to age-related health problems (Kannel et al., 1986). Age is positively associated with severity of atherosclerosis, as the formation of atheromas is a developmental process, and this may be one of the mechanisms by which age predicts CHD.

Other "traditional" risk factors include gender, sociodemographic status, alcohol-consumption, inactivity, diabetes and family history. The age-adjusted CHD-mortality rates for men are twice as high as for women. Men are at greater risk for CHD after controlling for the major risk factors, perhaps because men are higher on behaviors that are CHD-prone (e.g., Type-A; Jenkins, 1988). However, with approximately a 15-year age difference, women are at a similar risk for sudden-death and MI as are men. Until World War II, higher sociodemographic strata were at risk for CHD. Today the lower sociodemographic strata are at the highest risk possibly since hypertension, obesity and smoking are currently more prevalent among lower sociodemographic strata (Jenkins, 1988). A U-shaped relation between alcohol-consumption and CHD exists, as moderate drinking reduces the risk of CHD

while no alcohol and heavy drinking predict increased risk of CHD. Alcohol promotes deterioration of the myocardium and may also induce conductive disturbances which may lead to sudden death (Jenkins, 1988).

Physical activity has been shown to be inversely related to CHD (Peffenbarger, Wing & Hyde, 1978) independent of the major risk factors (Kannel, 1983). The protective effects of physical activity are uncertain and may be related to lipid-reduction. Diabetes is related to CHD, and this may result from occlusive processes or from direct myocardial damage (Kannel, 1983). Finally, genetic predisposition is a CHD risk factor. Kannel (1983) reported that in the Framingham Study, the occurrence of MI events of older brothers was significantly related to the occurrence of MI events of younger brothers, and this was independent of shared tendencies for hypertension, hypercholesterolemia and smoking. However, the separate contribution of genetic versus environmental factors is uncertain as families also share and experience similar (psychosocial) environments (Kannel, 1983).

These "traditional" risk factors may actually be a function of behavior (Jenkins, 1988). In particular, inactivity, smoking, alcohol-consumption, sociodemographic status and even gender differences may reflect life-style, primarily a **behavioral** issue. Thus, psychosocial factors may underlie or promote the effects of "medical" or "traditional" CHD risk factors. Chapter four will examine this issue in more detail.

The etiology of coronary-heart disease

Coronary heart disease includes a wide range of cardiac disorders that all have an underlying problem in the coronary arteries. The term CHD implies that the heart is diseased because of the coronaries, yet, the coronaries may not always present homogeneous problems. Diagnostically, CHD refers to the diseased heart and not to the diseased coronaries, which may be the underlying pathologic cause of CHD.

The term ischemic heart disease (IHD) can be used synonymously with CHD and both refer to the fact that the myocardium is diseased as a result of lack of blood. Coronary heart disease is a result of two aberrant conditions; an acute excess of myocardial demand for oxygen compared to available oxygen, or a an acute decrease in blood supply to the myocardium by the coronaries without increases in demand of oxygen (Sokolow & McLlory, 1986; Willerson & Buja, 1983). Increase in myocardial demand for oxygen may result from an increase in heart-rate. myocardial contractility or an increase in myocardial wall tension. Decreased coronary blood flow may result from an arterial stenotic process (arterial narrowing) due to an atherosclerotic process, a thrombus (blood-clot) overlying an atheroma, acute hypotension or coronary spasms. Atherosclerosis, coronary spasms and platelet aggregation and thrombosis, the major causes of reduced coronary blood-flow, will now be explained.

The atherosclerotic process. Atherosclerotic processes account for most cases of coronary artery disease (CAD; Perkins, 1989; Sokolow &

McLlory, 1986). Atherosclerosis is the development of localized lesions in the form of elevations of fatty fibre plaques (atheromas) situated within the intima and media (first and second arterial layers, respectively; Sokolow & McLlory, 1986). The atheromas include proliferation of smooth muscle-cells, deposition of plasma lipids, and accumulation of extracellular compounds (collagen, elastic fibers and polysaccharides). Complicated lesions, which include processes of internal hemorrhages, calcification, cell necrosis and superimposed thrombi, are associated with occlusive disease (Ross & Glomset, 1976).

Atherosclerotic lesions can be provoked by hypercholesterolemia, smoking and hypertension, and occur in the endothelium, the barrier between the blood and the intima. The lesion causes loss of endothelial cells and platelet aggregation near the lesion. Smooth muscle cells are triggered by these platelets and then migrate from the media to the exposed intima, multiply there and increase the lesion's thickness. This further increases degree of occlusion. Thus, platelet aggregation plays a central role in atherosclerosis (Ross & Glomset, 1976). Low density lipoprotein (LDL) cholesterol also induces arterial smooth muscle cell proliferation, thus, increasing the degree of occlusion. In the extracellular matrix of the atheroma, polisachandes bind LDL and both affect the process of coronary occlusion. Atherosclerotic lesions tend to be situated where arteries branch, and so where blood-compounds penetrate more easily into the arterial wall and cause lesions.

Atherosclerosis is a normal process which advances with age.

Atheromas do not normally impair myocardial blood-flow until occlusion exceeds 70% of intra-arterial diameter, which may precede an ischemic episode. Extent of atherosclerosis (degree, location and number of occlusions) predicts recurrent ischemic events (e.g., MI; De Belder et al., 1988). Location of occlusion is of major importance, since an occluded left anterior descending coronary artery may be a major cause of sudden-death, but an occluded right coronary artery may not be fatal (Sokolow & McIlory, 1986).

Coronary vasospasm. Despite the pathophysiological and prognostic importance of atherosclerosis in CHD, the impact of atherosclerosis has been challenged for to the following reasons: a) Only 10% of atherosclerotic patients are symptomatic; b) Atherosclerotic severity correlates poorly with CHD symptom severity; c) Approximately 10% of MI and angina patients have normal coronaries, d) Other processes also cause reduction in coronary blood-flow (Maseri et al., 1979). One such process is coronary spasms. Coronary spasms are sudden alterations in arterial smooth muscle tone associated with dramatic constriction of the coronaries, and reduced blood-flow. Maseri and his colleagues have suggested that coronary vasospasm may be a possible independent cause of CHD manifestations (ie., MI, UA and sudden death). In one study which examined eight patients with chest-pain at rest and ECG changes, all patients showed reduced myocardial blood supply concomitant to vasospasm during angina attacks (Maseri et al., 1978). The effects of vasospasm at rest on subsequent CHD-events seemed to be independent of

severity of atherosclerosis, as all patients subsequently had a MI, with little changes in CAD severity. In another study, Maseri et al. (1979) found no evidence of increases in heart-rate indicating an increase of myocardial demand for oxygen, that preceded ischemic attacks. However, vasospasm was observed during ischemic attacks, in patients with and without atherosclerosis. Since ischemic attacks were not provoked by unsatisfied increases in myocardial oxygen demand, since spasms occurred independent of atherosclerosis, and since vasospasm occurred during ischemic attacks, Maseri et al. (1979) concluded that coronary vasospasm may have an independent role in the pathogenesis of CHD.

While Maseri reported that 80-90% of their patients with UA have coronary spasms, others (e.g., Wigle, 1981) report that only 3% have spasms. This discrepancy may result from different studies recruiting patients with different cardiac profiles, and from many coronary spasms not being detected during angiography (Plotnick, 1985a). Spasms may result from an imbalance between platelet-released Thromboxane A2 (causing smooth-muscle contraction) and prostacyclin synthesized in the intima (causing smooth-muscle dilatation; Hirsh et al., 1981). Since Thromboxane A2 is released by aggregation of platelets, platelet aggregation may have a role in coronary spasms (Ouyang & Gerstenblith, 1985) in addition to its role in atherosclerosis. Other explanations include circadian or cyclical changes in coronary muscle tone, since anginal attacks at rest tend to occur in the early morning. Spasms,

or even small increases in arterial smooth-muscle tone narrowing the coronaries, may explain typical changes in pain patterns of UA patients (Plotnick, 1985b). Maseri et al. (1979) suggested that the different manifestations of CHD are a continuous spectrum of vasospastic myocardial ischemia, "progressing" from UA to MI and to sudden death.

Platelet aggregation and thrombosis. Platelets are blood-cells that participate in the process of blood-clotting. Platelets aggregate at injured sites, following any form of physical damage to blood-vessels. When platelets aggregate to a large extent, they may form thrombi, hard pieces of clotted-blood. Such thrombi can either become situated in a fixed place, or travel to a distal region (emboli). If the thrombus is large enough, it can reduce coronary blood-flow and cause a transient ischemia. Emboli rarely cause CHD events, but more often cause ischemia in cerebral regions or strokes (Sokolow & McLlory, 1986). However, static thrombi causing complete occlusion are thought to be a major cause of MI (De Wood et al., 1980). As mentioned above, platelet aggregation plays a role in atherogenesis (induces smooth-muscle cell proliferation) and in spasms (releases a vasoconstrictor). Thromboses most often form on atherosclerotic sites, and together may cause complete coronary obstruction. Platelets may also aggregate at an injured intima after a spasm. Thus, it is unclear whether platelet aggregation and its final stage of thrombosis are a cause of CHD independent of atherosclerosis and coronary spasms, whether they cause CHD via participating in

atherogenesis and spasms, or whether they follow an ischemic attack that induces coronary injuries (Plotnick, 1985b).

Unstable angina pectoris.

Etiology. Angina pectoris means "strangling shoulder" in Latin, and this encapsulates the difficulties of diagnosing angina pectoris in general and unstable angina (UA) in particular. However, UA provides an exceptional opportunity to understand and treat CHD. Angina pectoris is a sudden, acute attack of pain in the chest or neighboring areas such as the viscera, shoulders, arms and fingers. Stable angina or angina of effort is normally associated with exercise or effort. An angina becomes "unstable" when it increases in frequency, intensity or duration, and may no longer be attributed to effort, emotional exertion or weather changes alone (Sokolow & McIlory, 1986). Thus, UA is less predictable than stable angina.

Despite these differences, there has been large controversy over the definition and diagnosis of UA (Plotnick, 1985a). The two types of angina result from two generally independent pathological processes. Their mechanisms can be understood by examining how ischemia takes place. Ischemia occurs when coronary blood flow is insufficient to meet myocardial oxygen demands (Fuchs & Becker, 1982). In healthy individuals, there is a strong positive linear relation between coronary blood flow and myocardial oxygen demand, and an imbalance between the two is the key for understanding ischemia and CHD. Stable angina results from an increase in myocardial demand for oxygen (e.g.,

due to effort), which is not met mainly due to a fixed atherosclerotic occlusion (Fuchs & Becker, 1982). Individuals with atherosclerosis and stable angina may not experience an angina attack during rest as long as the occluded coronary can still supply resting levels of myocardial oxygen demands. However, beyond a certain level of activity (e.g., walking), the occluded coronary cannot supply the demanded oxygen and an ischemic attack will take place.

In UA, coronary blood flow is insufficient even to meet the levels of myocardial oxygen demand at rest. Coronary vasospasm has been suggested to be the main cause of UA (Fuchs & Becker, 1982; Maseri et al., 1979). Coronary vasospasm occurring at rest (without effort-induced increases in myocardial oxygen demand) reduces the level of coronary blood flow below resting levels of myocardial demands, and results in an angina attack at rest. In patients with atherosclerosis, vasospasm may occur near to or superimposed on atherosclerotic lesions. Thus, patients who initially had angina of effort because of fixed atherosclerotic CAD, may "progress" to developing UA at rest due to vasospasm. Therefore, UA is related to atherosclerotic CAD in most cases (about 66%), and the specific attacks at rest may be triggered by acute coronary vasospasm. However, in some cases (about 33%), individuals with normal coronaries may develop UA only as a result of coronary spasms (Maseri et al., 1979; Sokolow & McLlory, 1986).

Platelets aggregating in severely lesioned arterial regions may lead to

cyclical reductions in coronary blood-flow needed for resting levels of myocardial demands, potentially causing UA (Willerson & Buja, 1983). These cyclical reductions may result from platelets releasing Thromboxane, a vasoconstrictor (Willerson & Buja, 1983).

The neural mechanisms of chest-pain experienced during an ischemic attack are partly understood. Pain impulses are transmitted primarily via sympathetic nerve fibers originating near the coronary arteries, to the cardiac nerve, and then through the spinothalamic tract to the posterolateral and ventral nuclei of the thalamus. However, pain impulses from other somatic regions (e.g., skin, arms) and visceral regions (gallbladder) share the activation of the spinothalamic tract and final thalamic pathways. This common neuroanatomy may explain why cardiac chest-pain is often attributed or referred to other visceral or somatic regions (Foreman, Blair & Ammons, 1986; Fuchs & Becker, 1982). This referred nature of angina-like pain can often create difficulties in differentiating UA from other pains originating from gastrointestinal disorders, and requires other diagnostic criteria.

Diagnosis and classification. Unstable angina is diagnosed by the following criteria: 1). Chest-pain at rest, with or without angina of effort; 2). With objective evidence of myocardial ischemia by either ST-segment depression (in 66% of cases), elevation (in 33% of cases), T-wave changes, previous MI, thallium test revealing non-perfused myocardial regions, diastolic abnormality of heart muscle (increase in left-ventricular end-diastolic pressure),

systolic abnormality of heart muscle (wall motion abnormality), changes in heart-rate or BP, and angiographycally documented atherosclerotic CAD; 3). Without enzyme elevations indicative of a MI; (Plotnick, 1985a; Sokolow & McLlory, 1986). Plotnick suggested six subgroups of UA according to two main clinical features: Context of pain (effort, rest or both) and the time since onset of symptoms (recent - within the past four weeks, or previous - at least four weeks ago). This classification system allows to differentiate between angina patients and suggests underlying pathologies and treatments.

Prognosis. Since UA is normally seen as an intermediate syndrome between stable angina and MI, patients with chest-pain at rest are treated with greater caution, and are usually hospitalized for a few days for monitoring. Due to the increases in intensity, duration and frequency of chest-pain seen in UA, MI may easily develop in these patients. Between 3 to 60% of patients with UA are at risk for MI and 0 to 91% at risk for mortality. The main reason for the large range in risk is due to the controversy over the definition and diagnosis of UA. The main factors that affect the prognosis in UA are previous stable angina, recent MI, objective (ECG) evidence of ischemia, extensive CAD and poor left ventricular functioning (Plotnick, 1985a).

Treatment. The treatment of patients with UA depends on the underlying cause, and is divided into short- and long-term therapy. The short-term therapy focuses on terminating the chest-pain by administering intravenous morphine, and on terminating the ischemia by sublingual

Nitroglycerine. Nitroglycerine reduces myocardial oxygen demand, usually ending the ischemic episode (Plotnick, 1985c).

Long-term treatment follows from the diagnosed or assumed cause. If the underlying cause is related to atherosclerosis (i.e., the patient "progressed" from stable angina of effort to UA), then medications that reduce myocardial oxygen demand from the obstructed coronaries would be recommended. Beta-blockers such as propranolol have been shown to relieve pain symptoms and to reduce coronary events in UA patients (Mizgala et al., 1977).

If coronary spasms are thought to be the cause of the chest-pain at rest, then vasodilators are provided. Calcium antagonists (e.g., Nifedipine) can prevent coronary spasms and thus, reduce ischemic episodes. Long-acting nitrates and calcium antagonists can be given to UA patients with atherosclerosis or spasms, since these drugs reduce myocardial oxygen demand and prevent coronary vasospasm (Plotnick, 1985c). Finally, surgery is conducted only in patients who respond poorly to medication, and whose underlying CAD is severe. These include coronary artery bypass graft (CABG) surgery and angioplasty. These surgical procedures revascularize the myocardium, and have been shown to have a better effect on the functional status of UA patients than medical treatment (Sokolow & McLlory, 1986).

Myocardial infarction.

Etiology. A myocardial infarction (MI) is a state of death of myocardial cells (necrosis) resulting from prolonged insufficient blood flow to a myocardial

region. The pathophysiological causes of MI are similar to those of UA, however, they are more severe, and cause myocardial ischemia and necrosis rather than a transient ischemia alone. As with UA or any ischemic event, MI results from an imbalance between myocardial oxygen demand and coronary blood supply. For an infarct to develop, the ischemic imbalance normally lasts at least 20 minutes to an hour (Sokolow & McLlory, 1986; Willerson & Buja, 1983).

A severe and prolonged occlusion of an atherosclerotic occlusion may cause a MI. The causal role of platelet aggregation in the etiology of MI can be inferred from a study that showed that the antiplatelet agent acetyl salicylic acid (Aspirin) reduced 50% of non-fatal MIs in UA patients (Lewis et al., 1983). Thrombosis, formation of blood clots superimposed on atherosclerotic cites, may also lead to a MI (Sokolow & McLlory, 1986). Thromboses were found in 80% of acute MI patients within hours after the event (DE Wood et al., 1980).

Maseri et al. (1979) suggested that spasms resulting in complete and prolonged coronary occlusions may cause a MI. More so, the role of spasms and of platelet aggregation in the pathogenesis of MI are related. Spasms cause endothelial injury in the intima, which provokes platelet aggregation to the injured site. These platelets may then cause a severe thrombosis and total coronary obstruction in an atherosclerotic site. This total obstruction, if lasting 20 minutes, may then cause a MI. If not, the aggregating platelets that follow spasm-induced injuries, release Thromboxane A2 which causes more frequent

and perhaps longer spasms (Ouyang & Gerstenblith, 1985), and these may eventually cause a MI.

The site of an infarct in MI is commonly in the inner wall of the heart (subendocardium). This may result from a more severe reduction of coronary blood flow to the inner wall than to the outer wall (epicardium; Willerson & Buja, 1983). The site and degree of myocardial necrosis depends on the site and degree of the occluded coronary, the ability of collateral vessels to compensate for a malfunctioning coronary artery, and the presence of a previous infarct (Sokolow & McLlory, 1986). Myocardial cell damage is irreversible, and certain enzymes released during the infarct cause the "vertical border zone spread". This extension of the size of infarcts causes a further reduction in ventricular functioning (contractility) which may be fatal (Willerson & Buja, 1983).

Diagnosis and classification. According to Sokolow and McLlory (1986), the premonitory symptoms of MI include: 1) Chest-pain and/or pain radiating to the neck, left arm and fingers, and upper thoracic area; 2) Systemic manifestations of myocardial necrosis (fever, tachycardia, leukocytosis, 24-48 hours after onset of pain); 3) Cold sweat, weakness, apprehension; 4) Light-headedness (with symptoms of cerebral infarction) and hypotension; 5) Nausea and vomiting; 6) Pulmonary edema and left-ventricular failure (evidenced by shock, profound weakness and dyspnea).

Objective indices of MI include elevated serum enzymes which are indicative of myocardial necrosis, ECG changes indicative of ischemia and

radioisotope studies showing non-perfused areas in the myocardium. The myocardial-band (MB) isoenzyme of creatine phosphokinase (CPK), which is a myocardial specific enzyme, is found to be elevated within four hours after the MI. This enzyme will return to normal levels within 4-6 days. Other enzymes, such as lactate dehydrogenase, rise only 7-9 days post-MI, and are therefore suitable for confirming an "older" MI. There is a positive relation between enzyme level and myocardial cells necrosis (Sokolow & McLlory, 1986), thus enzyme elevation is a good index of severity of MI. The ECG indicates abnormal depolarization and repolarization of cardiac cells occurring after a MI. The most important ECG change is an irregularly large Q-wave (more than 30% of the following R-wave), and this is usually indicative of a transmural MI (involving the subendocardium and epicardium). A Non-transmural MI will usually yield only a change in the ST-T segments, in the form of ST-depression or elevation followed by an inverted T-wave a few hours or days later.

The major and widely accepted criteria for establishing a diagnosis of MI include: 1) Elevated myocardial enzymes (e.g., MB-CPK) and one of the following: typical chest-pain for at least 20 minutes or ECG changes (Q-waves or ST-segment depression or elevation; e.g., Shechter et al., 1990; Sokolow & McLlory, 1986).

There are several types of MI which differ in extent and location of the infarct. Each type is related to the precise coronary artery or arteries that failed to meet the necrosed region's demands for oxygen. Most types of MI involve

the left ventricle, which is larger than the right ventricle. A massive left ventricular MI may result from occlusion of the left main coronary artery. The site of infarct is commonly detected by the specific ECG leads that show ischemic changes, each lead representing a myocardial region.

Prognosis. Prognosis following a MI can be very poor since infarctions may extend (Willerson & Buja, 1983) and seriously reduce ventricular functioning, which in return reduces the heart's ability to supply blood to the body. There is a higher rate of mortality in the coronary care unit in MI patients (16%) than in UA patients (0%) and a higher rate of mortality in the hospital (outside the coronary care unit) in MI patients (5%) than in UA patients (1%; Krauss, Hutter & DeSanctis, 1972). After discharge, the rate of post-MI mortality is between 4-15% per year, and this depends on the severity of underlying CAD and left-ventricular functioning (Sokolow & McLlory, 1986). In mild MI, patients may not reexperience cardiac events after their pain has been relieved. However, in more severe MI the most common event is arrhythmias (irregular heart-beat), and the most fatal event is cardiogenic shock. The all-cause mortality rate is relatively high in the first month (30%), with most deaths occurring within the first 12 hours post-MI. Early reinfarcts occur in approximately 15% of patients. Among the important parameters that affect one-year survival after a MI are ejection-fraction below .40 (which reflects poor left-ventricular functioning), ventricular ectopy and functional status one month prior to the MI (The Multicenter Postinfarction Research Group, 1983).

Regarding life-readjustments, only one third of post-MI patients return to work, and this depends on whether they were employed before their attack, and on levels of depression (Sokolow & McLlory, 1986).

Treatment. The different treatment strategies can dramatically improve the prognosis of MI patients, and they depend on the underlying cause of the disease. Short-term treatment includes defibrillation for ventricular arrhythmia and ventricular fibrillation, morphine-like substances for pain-relief. sedatives for anxiety, treating and preventing cardiac failure and treating the infarcted myocardium (Sokolow & McLlory, 1986). Thrombolysis, the breaking of thromboses, has been conducted in hospital and has resulted with recanalization in 60% of patients within 90 minutes (Braunwald, 1985). Shechter et al. (1990) provided Magnesium Sulphate (an anticonvulsant) for 24 hours and showed significant reductions in mortality over a week compared to a control group. Many long-term clinical trials have successfully reduced reinfarction over different follow-up periods. For example, the Coronary Drug Project Research Group (1975) showed significant reductions in reinfarctions over five years in patients receiving Niacin (cholesterol-lowering drug) compared to a placebo control group. Timolol, a beta-blocker (reduces myocardial oxygen demand) has been shown to have positive effects on reinfarctions in The Norwegian Multicenter Study Group (1981). In contrast, a well designed randomized-controlled trial with Diltiazem, a calcium antagonist (vasodilator) failed to show any effects on reinfarction and mortality (The Multicenter

Diltiazem Post-infarction Trial Research Group, 1988). The decline seen in recent years in the incidence of CHD and mortality from CHD (Jenkins, 1988) may indeed reflect improvements in preventing and treating MI, UA and CHD in general.

CHAPTER TWO

HOSTILITY: DEFINITION, ASSESSMENT AND EPIDEMIOLOGY

Introduction

This chapter will define the construct of hostility in the particular context of coronary heart disease (CHD). A detailed description and analysis of the methods for assessing hostility will follow, together with an examination of the correlates of the major measures. Finally, this chapter will provide some information concerning the epidemiology and development of hostility.

Defining hostility

The field of coronary prone behavior aims at understanding which and how certain psychological parameters predict or are associated with CHD This task is concerned with the predictive validity of such parameters as well as their construct validity. To achieve construct validity, a clear definition and clarification of such parameters is essential (Smith, 1994) This clarification can improve prediction as well as prevention and treatment of CHD, since understanding the construct behind a predictive measure helps to identify its components that may be targeted in interventions.

According to the widely accepted trilogy of psychological experience, mental activities can be classified into three categories: Affect, cognition, and behavior (Hilgard, 1980). This classification scheme is useful in defining many psychological constructs since it is comprehensive enough for encompassing differing constructs such as hostility or depression. At the same time, this

classification allows one to emphasize one dimension (e.g., cognition), and allows one to examine the relations between the three dimensions. Thus, this framework will be used for reviewing several definitions and measures of hostility, as has been suggested by others (e.g., Barefoot, 1992).

Confusion and difficulties have surrounded the definition and conceptualization of related constructs such as hostility, anger and aggression (Smith, 1994). This confusion has had negative effects on promoting the understanding of these constructs, their assessment, and their impact on health. Anger is a primary emotional state of varying intensities from irritation to rage (Williams, Barefoot & Shekelle, 1985) linked to a real or imagined environmental stimulus or provocation, and is associated with enhanced sympathetic arousal. Hostility is seen as a broader construct, which according to some authors includes mainly a cognitive attitudinal component (Spielberger et al., 1985), but may include affective and behavioral components which will be defined below. Finally, aggression refers to overt attacking, destructive or harmful behavior (Smith, 1994). Hostility and aggression are related since aggression motivated by angry feelings or hostile attitudes is termed hostile aggression. Aggression that is not motivated by anger, but is directed toward removing an obstacle standing between the aggressor and a goal (i.e., blowing up an old building in order to build another one) is considered instrumental aggression (Spielberger, Jacobs, Russel & Crane, 1983).

Older definitions of hostility reveal the disagreement about this construct.

Saul (1976; cited in Diamond, 1982) employing a psychodynamic approach, viewed hostility as "a motivating force - a conscious or unconscious impulse, tendency, intent or reaction - aimed at injuring or destroying some object... hostility is usually accompanied by the feeling or emotion of anger" (p. 7). This definition includes affective and behavioral components, and also relates hostility to motivation. Furthermore, this definition views hostility as including highly destructive intentions and actions. According to Plutchik (1980; cited in Diamond, 1982), hostility includes anger, disgust, contempt and resentment, all affective components. These diverse definitions do not indicate whether hostility includes all three components of experience.

However, most of these definitions were not derived from or established for systematic scientific work (Barefoot, 1992). The definition that will be used in the present research is derived from work on the "coronary-prone" construct. Thus, the definition of hostility that is used in the field of coronary-prone behavior includes the components of hostility that predict or are related to CHD end-points. It is important to understand the difference between coronary-prone or CHD-predictive hostility and hostility in its broader scope. The broader term includes the more extreme aspects of hostile cognition (e.g., militant intentions) and extreme hostile behavior (violence, war, etc.) as well. These aspects are beyond the boundaries of hostility in the context of CHD, and are commonly not associated with CHD-predictive hostility (Barefoot, 1992).

Barefoot and colleagues conducted extensive research on the

assessment and predictive validity of the hostility construct in relation to CHD. According to Barefoot (1992) and Barefoot and Lipkus (1994), hostility consists of three components, cognitive, affective and behavioral. The cognitive component consists of negative beliefs about others (e.g., "many people are untrustworthy, undeserving and immoral"). These beliefs can lead to hostile attributions, a belief that others' antagonistic behavior is directed at the "self", and to cynicism, negative beliefs about human nature in general (e.g., "people are generally selfish"; Barefoot, 1992).

The affective component of hostility includes annoyance, anger, resentment, disgust and contempt. Thus, throughout this research, anger will be viewed as the emotional **component** of hostility, not as a parameter synonymous with hostility.

Finally, the behavioral component of hostility includes manifestations of antagonistic and disagreeable behavior such as verbal aggression, rudeness, argumentativeness, condescension, and less frequently, aggressive acts (e.g., slamming doors; Barefoot, 1992; Dembroski & Costa, 1987). Most antagonistic behavior is nonviolent, and it may be manifested in a large number of ways (Dembroski & Costa, 1987). As the more common forms of subtle antagonism (e.g., indirect challenge) appear to predict CHD end-points better than more extreme and infrequent forms of antagonism (e.g., direct aggression; Barefoot, 1992), the behavioral component of hostility does not emphasize extreme hostile behaviors.

To summarize Barefoot's (1992) definition, hostility is the trait-like tendency to think in a cynical and mistrusting manner and to attribute hostile intentions to others, to frequently feel annoyance or anger, and to behave in an antagonistic manner. This definition, and the criteria of being CHD-predictive, does not include the more violent or militant connotations of hostility. These extreme aspects of hostility are more relevant to international disputes or inter-group conflicts, and go beyond the magnitude and scope of our definition of hostility. This clarification has linguistic implications, since in some languages (e.g., French, Hebrew), hostility implies the extreme aspects more than the milder meanings referred to by CHD-predictive hostility.

Several authors have distinguished between the experience and expression of hostility and found them to be related to different personality dimensions (Costa, McCrae & Dembroski, 1989; Smith, 1994). The experience of hostility includes hostility's affective component of anger and hostility's cognitive components of cynicism, mistrust and hostile attributions. The expressive component of hostility refers to hostility's behavioral component of overt antagonism, such as rudeness, condescension, etc. While experienced hostility has been related to the personality domain of Agreeableness versus Antagonism **and** to the personality domain of Neuroticism, expressed hostility has been related to the former personality domain and not to the latter (Costa et al., 1989). Antagonism reflects an overt critical style, manipulativeness, opposition and rudeness (Costa et al., 1989). In contrast, Neuroticism

represents the tendency to experience general distress, physical and psychological discomfort, and several negative affective states such as anger and anxiety. In addition, individuals high on Neuroticism tend to be aware and over-attend to unpleasant psychological and somatic sensations (Watson & Pennebaker, 1989). These important distinctions between experienced versus expressed and between neurotic versus antagonistic hostility will be used in discussing the assessment and components of hostility throughout this thesis.

Relations between the components of hostility

From a theoretical perspective it is important to understand the relations between the three components of hostility, as this may reveal how hostile manifestations are engendered and maintained. While the cognitive, behavioral and affective components do not have to be all elevated among hostile individuals (Barefoot, 1992), empirically and across subjects, they are moderately correlated (Barefoot et al., 1989), and several investigators have postulated models linking them to each other. Chesney (1985) postulated that when facing a provocation, hostile cognitions ("My colleagues are not trustworthy") can elicit hostile behavior, affect, and physiological reactions (e.g., verbal confrontation, anger and increased blood-pressure, respectively). These responses of the individual may then affect the environment (colleagues act defensively) which affect again our reappraisal of the provocation ("My colleagues are against me"), which may sustain hostile thoughts, behavior and affect, and so on.

Powell (1992) postulated a similar transactional model for understanding how cognitions and behavior operate reciprocally with the environment. According to Powell (1992), hostile cognitions such as mistrust, emerge from a basic belief that justice will not prevail. To ensure justice for oneself, hostile people then adopt hostile attitudes and behavior ("The best defense is a good offense"). In addition, hostility is also expressed by facial expressions, tone of speech and verbal report of past annoying experiences. These behaviors then elicit hostile reactions from the environment, which serve as behavioral confirmations of hostile individuals' expectancies from others, which lead to more mistrust, and so forth. Burke (1985) showed that hostility was positively correlated with believing that justice will not prevail, but that study suffered from several methodological limitations (e.g., poorly defined measures, too many statistical tests).

Supporting Powell's (1992) transactional model, Snyder and Swann (1978) found that when subjects were told that they would be competing with a hostile opponent (i.e., hostile thought induction) they and their opponents exhibited greater hostility than if subjects were told their opponent was not hostile. Resulting from subjects' behavioral hostility, the reactive-hostility of the opponents served as behavioral confirmations for subjects' hostile expectations, since subjects then rated reputedly hostile opponents as more aggressive than opponents that were expected to be non-hostile. Thus, hostile cognitions (negative expectancies about others) yield hostile behavior in individuals holding

the hostile cognitions as well as in the individuals with whom they interact. However, Snyder and Swann (1978) did not assess individual levels of hostility, thus, it is unknown whether such transactions occur more often in hostile people than in non-hostile people. In summary, although the three components of hostility may not always co-occur (Barefoot, 1992), they may affect one another, with hostile cognitions potentially eliciting hostile affect and behavior (Chesney, 1985; Powell. 1992; Snyder & Swan, 1978).

Assessment of hostility

The systematic assessment of hostility includes self-report, observed and peer measurements. The relations between hostility scores using different measurement strategies can vary as a function of method variance and the components of hostility that are assessed by each measurement. Each measurement strategy has its psychometric strengths and weaknesses. This section will review the main measures of hostility used in the context of CHD.

Self-report measures of hostility

Self-report measures have been widely used since they are logistically easy to administer, are time and cost-effective, and do not require training of interviewers or raters. Since most constructs in hostility are subjective (i.e., angry emotions, mistrusting thoughts), using an introspective approach seems quite reasonable. However, self-report measures suffer from several methodological limitations (Barefoot & Lipkus, 1994). The first problem is related to their comprehensibility and format, and the extent to which

completing self-report measures depends on hypothetical situations. Hypothetically-based measures may also suffer from limited ecological validity. Second, not all subjects "know the answer", due to lack of self-awareness or use of defenses such as denial, particularly CHD patients (Rosenman, 1978). Third, not all respondents answer honestly. Situational factors (e.g., evaluative context; McCranie, Watkins, Brandsma & Sisson, 1986) and the general problem of social desirability may affect honesty. Finally, since self-reported anger and cynical hostile thoughts are related to the broader dimension of negative affectivity (NA) or Neuroticism, NA-related attentional and responding biases may prevail in these measures, and affect their construct and predictive validity (Watson & Pennebaker, 1989). This will be discussed in later sections.

The Ho Scale. The most widely used self-report hostility scale is the Cook and Medley (1954) Ho scale. It is derived from the Minnesota Multiphasic Personality Inventory (MMPI; Hathaway & McKinley, 1943), a personality inventory that has been administered to many American samples. Thus, a great deal of work on the predictive validity of the Ho scale has been done, as we shall see in Chapter Three. The Ho scale was originally developed in order to assess teachers' ability to establish rapport with their pupils. Two hundred and twelve Minnesota teachers who scored on the 92nd and 8th percentiles of the Minnesota Teacher Attitude Inventory (MTAI; Cook, Leeds & Callis, 1951) were administered the MMPI. The MTAI assesses teacher-pupil rapport. Among the 550 MMPI items, 250 were found to discriminate between the two

groups of teachers classified by the MTAI. Next, items that best correlated with MTAI categories (Empirical criterion) and whose content best reflected hostility (face validity) were chosen by five clinical psychologists, resulting in 50 hostility items (the Ho scale). The internal reliability of the Ho scale was high (.86), and it was significantly and negatively associated with MTAI rapport scores ($r = -.44$). Scores on the Ho scale and another subscale derived throughout this process, Pharisaic Virtue (adhering to morality) were quite strongly correlated ($r = .69$). Cook and Medley (1954) concluded that the Ho scale assesses the degree of dislike and mistrust of other people. A person scoring high on the Ho scale is one who "sees people as dishonest, unsocial, immoral, ugly, and mean, and believes they should be made to suffer for their sins. Hostility amounts to chronic hate and anger" (Cook & Medley, 1954). Thus, the Ho scale seems to assess primarily cognitive or attitudinal hostility, and to some extent hostile affect as well.

Several authors have criticized the Ho scale and its development (e.g., Barefoot, 1992). First, its items were selected according to empirical rather than theoretical criteria. Second, the criterion of identifying teachers with good versus bad teacher-student rapport with the MTAI may involve psychological parameters other than hostility, such as poor social skills and social isolation. This empirical criterion may not have covered all aspects of hostility such as behavioral hostility that is not fully assessed by the MTAI. Finally, the Ho scale suffers from the problems of all self-report measures (social desirability,

presentation biases, question difficulty; Barefoot, 1992).

Since hostility is assumed to be a chronic and enduring trait, establishing the test-retest reliability of hostility measures is central (Smith, 1994). Barefoot, Dahlstrom and Williams (1983) found that Ho scores over a one-year period were very stable among 42 students (r = .85). Shekelle, Gale, Ostfeld and Paul (1983) found that over a four-year period, Ho scores were also highly stable among 1653 men (r = .84). Thus, the Ho scale appears to assess a relatively stable psychological characteristic. This incredible stability indeed challenges therapeutic attempts to modify this psychological parameter.

In light of its questionable method of development, several studies have attempted to establish the construct validity of the Ho scale. Smith and Frohm (1985) tested the convergent and discriminant validity of the Ho scale. Ho scores were positively significantly and more strongly correlated with measures of trait-anger (r = .61) than with measures of trait-anxiety (r = 26) and depression (r = .38). This pattern remained intact even after partialling out the effects of social desirability that was significantly and negatively correlated with Ho scores (r = -.50). Furthermore, Ho scores were significantly and more strongly correlated with measures of resentment (r = .70) and suspicion (r = .69) than with measures of physical assault (r = .43), verbal aggression (r = .41) and indirect aggression (r = .35). Thus, the Ho scale does **not** primarily assess indices of negative affect such as anxiety and depression or behavioral hostility (discriminant validity), and it more strongly assesses cognitive and affective

hostility (convergent validity). Smith and Frohm (1985) concluded that the Ho scale measures **cynical hostility**, the tendency to experience anger, to be resentful and to mistrust others, but not necessarily to be overtly aggressive. In addition, the best psychological predictors of high versus low Ho scorers were trait-anger and low levels of hardiness (e.g., unadaptive cognitive appraisals, low resilience during stress). Thus, Ho scores reflect anger, mistrust and unadaptive cognitive appraisals.

Using an empirical approach, Costa, Zonderman, McCrae and Williams (1986) factor analyzed Ho scores of 1002 cardiac patients and found two replicable factors: Cynical Mistrust and Paranoid Alienation. Scores on both subscales were significantly correlated with each other (r = .54), and with MMPI-subscales indicative of psychopathology (e.g., Neuroticism, Psychoticism, Somatic Complaints, Inadequacy and Cynicism). Thus, in addition to assessing mistrusting attitudes, the Ho scale assesses neurotic aspects of personality, and this may limit its predictive validity (Costa et al., 1986). Despite these important findings and implications, this analysis was empirically- rather than theoretically-based. In addition, using the MMPI-based Cynicism subscale for validating the Ho-derived Cynical Mistrust factor may yield spurious correlations (r = .91) possibly due to item overlap (Sullivan & D'Eon, 1990).

Another study examined the correlates of the Ho scale (Greenglass & Julkunen, 1989) separately for males and females, since in most cases either only males were enrolled or results were collapsed across gender. Greenglass

and Julkunen (1989) found that Ho scores were more strongly correlated with measures of cynicism and paranoia than with measures of behavioral or expressed hostility. However, more important was the finding that Ho scores were more strongly correlated with Anger-Out in males than in females, and more strongly correlated with Anger-In in females than in males. Thus, the Ho scale measures different aspects of hostility in males and females, which have important implications for the scale's predictive validity. The Ho scale may be tapping primarily expressed or antagonistic hostility (Anger-Out) in males, and in females the Ho scale may be tapping primarily experienced or neurotic hostility (Anger-In; Musante et al., 1989). Thus, studies should not collapse results across gender, and should either include only males or females, or examine results for each gender separately.

Barefoot et al. (1989) attempted to examine the psychological domains assessed by the Ho scale using a **theoretically-based** approach. The items of the Ho scale were grouped by judges into six categories according to an a priori theoretical basis and the items' face validity: Cynicism (general negative thoughts of humankind), Hostile Attributions (the tendency to interpret others' behavior as purposefully harmful for the respondent), Hostile Affect (experience of anger in social contexts), Aggressive Responding (overt or indirect interpersonal aggression), Social Avoidance (withdrawing from interpersonal interactions), and an Other category (ambiguous items). Item classification was tested against subjects' scores on five personality dimensions (Neuroticism,

Extraversion, Openness, Conscientiousness and Agreeableness; Norman, 1963) and the Hostility subscale of the Neuroticism dimension (Costa & McCrae, 1985). Cynicism, Hostile Attribution, Hostile affect and Aggressive Responding were moderately to strongly intercorrelated. These four Ho subscales were all significantly correlated with the dimensions of Agreeableness (negatively) and Hostility (positively), supporting their convergent validity, but not with Extraversion, Openness or Conscientiousness, supporting their discriminant validity. Scores from the Cynicism and Hostile Affect subscales were significantly correlated with Neuroticism as well. Social Avoidance correlated with Agreeableness and Extraversion (both negatively) but not with Hostility, while items from the Other subscale correlated with Neuroticism alone (Barefoot et al., 1989). Thus, the Ho scale includes items that assess only Neuroticism. The Cynicism, Hostile Attribution, Hostile affect and Aggressive Responding Ho subscales appear to be better markers of a hostile personality, since they correlate with hostile and disagreeable personality dimensions. Costa and McCrae (1987) also stated that cynical hostility, the major trait assessed by the Ho scale (Smith & Frohm, 1985), is more closely related to the Antagonism domain of personality than to Neuroticism. Thus, these four Ho subscales should be assessed rather than the full Ho scale, since they are not reflections of Neuroticism alone, but do reflect Antagonism to a greater extent. This clarification of the Ho scale should have strong implications on its predictive validity (Watson & Pennebaker, 1989), as will be shown in Chapter

Three. Barefoot, Larsen, von der Leith and Schroll (in press) found that the test-retest reliability of an abbreviated Ho scale consisting of most items from these four Ho subscales was strong over a 10-year period ($r = .74$) in a Danish sample. Thus, the refined markers of hostility in the Ho scale are stable as well.

The BDHI. Another widely used measure of hostility is the Buss Durkee hostility Inventory (BDHI; Buss & Durkee, 1957). This scale was developed to differentiate between overt and covert hostility, as well as its behavioral, affective and cognitive components. Thus, the BDHI is more theoretically-based than the Ho scale. The BDHI consists of 66 items that include six subscales: Assault (physical violence), Indirect Hostility (malicious gossip, slamming doors), Irritability (low threshold for exploding with negative affect), Negativism (noncooperative behavior), Resentment (Jealousy, anger, hatred), Suspicion (distrust, hostile attributions) and Verbal Hostility (threats, shouting). The 66 items were selected from 105 initial ones, based on frequency of occurrence in a sample and internal consistency. Assault, Indirect Hostility, Negativism, Verbal Hostility (and Irritability) assess behavioral hostility, Suspicion assesses cognitive hostility, and Irritability and Resentment assess affective hostility. Buss and Durkee (1957) demonstrated a two-factor solution: An emotional-cognitive factor (Resentment, Suspicion) and a motor or behavioral factor (Assault, Indirect Hostility, Irritability, Verbal Hostility), in both men and women. Finally, the scale is mildly correlated with social desirability.

More recently, Siegman, Dembroski and Ringel (1987) found a similar two-factor solution for the BDHI: Neurotic hostility (the Resentment and Suspicion subscales) and Antagonistic hostility (the Assault, Indirect Hostility and Verbal Hostility subscales).

The suspiciousness subscale. A third and less frequently used self-report hostility scale is the Factor L, which is a subscale of a general personality inventory (Cattell's 16 Personality Factors, 16 P.F.; Cattell, Eber & Tatsouka, 1970). This 106-item questionnaire assesses suspiciousness versus trust, domains that are conceptually related to cynical mistrust assessed by the Ho scale (Smith & Frohm, 1985). Scores on the Factor L scale are significantly but mildly correlated with Ho scores (.26 to .37; Barefoot et al., 1987). These correlations are low, given the shared method variance between the two self-reported scales, and suggest that the Ho scale does not assess suspiciousness alone (Barefoot et al., 1989).

The Anger-Expression scale. The hostility scales discussed up to this point do not directly assess the important issue of the direction of hostility or anger expression (i.e., inward or towards others). The mode or direction of expressing hostility and anger has become an important topic for assessment since it was unclear whether suppressed anger (Anger-In) or expressed anger (Anger-Out) are equal risk factors for CAD and CHD. In addition, most measures of anger (e.g., State-Trait Anger Scale, STAS; Spielberger, et al., 1983) assessed the two forms of anger expression together, and it is

impossible to examine their consequences separately. To answer this need, the Anger-Expression Scale (AX, Spielberger et al., 1985) was developed. Anger-In was conceptualized as the frequency that angry feelings are experienced but not expressed, and Anger-Out as the frequency that an individual engages in aggressive behaviors when angry. The AX is a 20-item scale, originally developed to assess a unidimensional bipolar construct of anger expression (Anger-In to Anger-Out). However, results of correlational and principal component analyses revealed that the AX scale includes two independent subscales of Anger-In and Anger-Out, with eight items in each (the remaining four items asses Anger-Control). Sufficient internal reliability has been demonstrated (.81 to .84 for Anger-In; .73 to .75 for Anger-Out; Spielberger et al., 1985). Concurrent validity has been supported with AX subscale scores significantly predicted by classifications of individuals as anger-expressors or suppressors in hypothetical provocations. The Anger-In and Anger-Out subscales have been shown to be correlated with measures of trait/state anger and anxiety, and to be uncorrelated with measures of curiosity, supporting the AX scale's convergent and divergent validity, respectively (Spielberger et al., 1985).

Observed measures of hostility.

The most direct and "objective" way for assessing overt manifestations of hostility would be to observe individuals within a structured framework during challenging situations that mimic daily provocations. Observed measures allow

one to assess the **style** in which subjects express themselves, with and without reference to their speech-content. Focusing on style overcomes problems of subject comprehension, self-awareness, NA-related reporting biases and partially removes effects of social-desirability (related to content). Interviews also provide individual repertoires of hostility, compared to the restricted lists provided by questionnaires (Barefoot & Lipkus, 1994). For these reasons, observed hostility measures are thought to have better construct and predictive validity than self-report measures (Smith, 1992). However, individual repertoires are also a limitation, since different idiosyncratic responses are difficult to rate reliably across subjects. Thus, while observed measures may be more valid, their reliability may be lower than that of self-report measures. Interviews are logistically more complicated, expensive, and require rigorous administrator and coder training for achieving sufficient inter-rater reliability. The problem of social desirability still exists in observed measures, and can affect occurrences of expressed hostility, particularly toward the interviewer (Barefoot, 1992). Finally, interviews are a one-time sample of questions and responses, and other questions and situations may add important information about subjects' hostility.

The Structured Interview. Observed hostility measures originate from the Structured-Interview (SI; Rosenman, 1978), initially used to elicit and assess the "coronary-prone" Type-A-Behavioral Pattern (TABP). Rosenman (1978) reported the development and validation of the SI. The SI was developed to

elicit and assess the TABP, which includes impatience and time urgency, enhanced competitiveness, and aggressive drive and hostility (Rosenman, 1978). The assessment of these components depends upon the exhibition or report of these behaviors by the subject, and on the interviewer's and assessor's ability to elicit and observe these behaviors, respectively. The SI is a 12-minute brief videotaped interview, which includes questions regarding the way an individual responds to daily provocations (e.g., waiting for someone who is late), the manner and duration of anger expression, and the individuals' daily rhythm. The SI is a structured experimental situation for sampling certain behaviors. The questions are asked in a business-like manner, with the interviewer being task-oriented rather than empathic with the interviewee.

In its original format, the interviewer interrupted the interviewee, and directly challenged the interviewee, in order to elicit reactivity. However, Houston, Smith, O'Connor and Funk (1988) found that SI ratings of the TABP converged with self-report ratings, and Type-As had the expected higher stress-induced physiological reactivity than Type-Bs only if subjects were interviewed in a slow, non-disruptive manner. Thus, a non-provocative SI may yield more valid Type-A (and possibly hostility) ratings, that may be more predictive of CHD end-points (Houston, et al., 1988; Scherwitz, 1984).

Following findings that certain components of the TABP are more predictive of CHD than others (e.g., Dembroski et al., 1989), and acknowledging the multidimensionality of the TABP, Wright and Schmidt-Walker

(1990) developed the Augmented-Structured-Interview (ASI). The ASI included 19 additional questions, with seven questions specifically on Anger-In/Out. Significant changes were found between Anger-Out ratings based on original SI and additional SI questions, and this tended to happen with Anger-In questions. Thus, the new items were not redundant.

Potential for Hostility. The assessment of hostility from the SI began with dividing the global TABP rating into its components (Dembroski, 1978; Dembroski & MacDougall, 1983; Matthews et al., 1977). Component scoring was done to improve the detection of the toxic subcomponents in the TABP in relation to CHD. Dembroski and MacDougall (1983) suggested the term Potential for Hostility (PH), conceptually defined as the relatively stable tendency to **experience** anger, irritability and resentment in response to daily provocations, and/or to react with **expressions** of antagonism, rudeness, criticalness, argumentativeness and uncooperativeness (Dembroski, 1978; Dembroski & Costa, 1987).

Assessing PH is primarily a clinical judgement, and both overt stylistics and content are evaluated. As described below, the conceptualization and assessment of PH has undergone several modifications. The stylistics include manifestations of boredom, condescension, surliness and antagonism towards the interviewer. Content includes self-reports of expressions of antagonistic behavior, admissions of annoyance, anger and irritability in daily provocations, and harsh generalizations and emotionally laden words used to describe

intensity of hostile reactions (Dembroski & MacDougall, 1983, Dembroski & Costa, 1987). PH is rated with a five-point scale (1 = No statements with possible hostile content or structure, and no hostile voice stylistics; To 5 = Frequent hostility expressed in attitude or voice stylistics) Inter-rater reliability for PH ranges from .70 to .85, and test-retest reliability over six to 18 months was r = .55 (Dembroski & Costa, 1987).

Musante, MacDougall, Dembroski and Costa (1989) examined the construct validity of PH by testing its correlation patterns with 21 subscales derived from four measures of anger and hostility (e.g., BDHI; Buss & Durkee, 1957, Multidimensional Anger Inventory, MAI; Siegel, 1985). Scores on PH were unrelated to scores of attitudinal or cognitive hostility (e.g., Mistrust, Suspicion), but were related to measures of experience (e.g., Irritability) and expression (e.g., Verbal Expression) of hostility. Factor analyzing the 21 subscales yielded a three factor solution of experiential, expressive and attitudinal factors. Scores of PH were related to both expressive (r =.31) and experiential (r = .32) factors, but not to the attitudinal factor (r = -.14). Since the attitudinal factor mainly assessed mistrust, the main construct assessed by the Ho scale (Smith & Frohm, 1985), Ho and PH are complimentary measures that together assess all three dimensions of hostility Musante et al. (1989) suggested refining the measurement of PH and separating its experiential from its expressive components, so that their predictive validity could be examined separately.

Dembroski and Costa (1987) conceptually divided PH into its experiential (Hostile Content and Intensity) and expressive (Hostile-Style) facets. Hostile Content indicated the frequency of admissions of experiencing hostile affect in daily provocations. Hostile Intensity indicated the degree of hostile affect experienced in those provocations. Finally, Hostile-Style reflected degree of disagreeable and antagonistic behavior the interviewee directed towards the interviewer during the SI. All three components are assessed seperately and contribute to the assessment of global PH, with Hostile-Style being most important. Global PH, Hostile Content and Hostile Intensity were significantly associated with the Neuroticism **and** Agreeableness factors of personality. However, PH was significantly more correlated with Agreeableness than with Neuroticism ($r = -.87$ versus $.55$, $Z = 5.25$, $p < .01$ in the MRFIT sample; $r = -.81$ versus $.47$, $Z = 4.27$, $p < .01$ in the WCGS[1]). In contrast, Hostile-Style was associated **only** with the Agreeableness factor. However, SI-derived measures (e.g., PH) and subjects' personality measures were assessed by the same individual, possibly biasing this correlational pattern.

To overcome this latter problem, Costa et al. (1989) used trained SI-coders' observed ratings of hostility, and self-report scores for personality measures obtained from subjects and their peers. They showed that measures of experiential hostility (i.e., frequency, intensity and duration of anger) reflect

[1] Z tests on differences between correlations were performed by Y. Gidron and were not provided in the original paper by Dembroski and Costa (1987).

Neurotic personality, while measures of expressive hostility (i e , verbal aggression, overt anger expression) are inversely related to the personality factor of Agreeableness. Costa et al (1989) replicated the findings of Dembroski and Costa (1987) by showing that only the Hostile Style component of PH was exclusively related to Antagonism. Since the Ho and PH scales assess both Neurotic (experiential) and Antagonistic (expressive) hostility, this may limit their predictive validity in relation to CHD (Watson & Pennebaker, 1989). More refined observational hostility measures that assess only antagonistic hostility, such as SI-derived Hostile-Style, and more refined self-report measures such as the BDHI subscales of Assault, Indirect Hostility and Verbal Hostility, or Barefoot's Ho subscales of Cynicism, Hostile Attribution, Hostile Affect and Aggressive Responding, are more "pure" markers of antagonistic hostility, and thus, may be better predictors of CHD (Costa et al., 1989; Dembroski & Costa, 1987; Wastson & Pennebaker, 1989).

Finally, a recent study (Davidson & Hall, 1995) examined the construct validity of PH for male and female students separately. This study assumed that Barefoot et al.'s (1989) refined Ho and its subscales (Cynicism, Hostile Affect and Aggressive Responding) predominantly assesses antagonistic rather than neurotic hostility. Their first study showed that while PH was correlated with the Cynicism refined Ho subscale and total Ho scores in males, PH was unrelated to any Ho subscale or total Ho scores in females. In addition, while PH was unrelated to negative affect (NA) in males, it was related to NA in

females. In their second study, subjects and interviewers rated subjects' PH and NA. Observed PH was correlated with self-reported PH in males, however, observed PH was unrelated to self-reported or observed NA levels in males. In contrast, in females, observed PH was only related to NA measures. This study supported the construct and concurrent validity of PH in males but **not** in females, and suggests that PH measures antagonistic hostility in males and Neuroticism in females. Thus, PH may not predict CHD in women, since it is confounded by Neuroticism (Watson & Pennebaker, 1989), and the assessment of hostility needs to be improved in women. These findings converge with those of Greenglass and Julkunen (1989) who found that in males, Ho was correlated with Anger-Out, an index of antagonistic hostility, while in females, Ho was correlated with Anger-In, an index of neurotic hostility (Musante et al., 1989).

The component scoring system. Hecker, Chesney, Black & Frautschi (1988) developed the component scoring system (CSS) for assessing 12 SI components of the TABP. Specific operationalizations of how to code each component are provided, to increase inter-rater reliability. Hostility, one of the components, includes subjects' stylistic and content responses during the SI (Hecker et al., 1988) and is coded according to four weighted criteria: Evasiveness and cooperation of subject in responding, degree of hostility in tone, indirect challenging of the interviewer, and direct challenge of the interviewer (Barefoot & Lipkus, 1994). Hostility is a summed score from each

of the four criteria across 20 SI segments or questions. The inter-rater reliability for the hostility score is moderately-high (.64).

The Interpersonal Hostility Assessment Technique. Finally, the most recent SI measure of hostility is the Interpersonal Hostility Assessment Technique (IHAT; Barefoot, 1992; Haney et al., 1992). Since extreme manifestations of direct hostility towards the interviewer are rare, the IHAT provides a much more refined definition of subtle hostility directed at the interviewer. This increases the scale s predictive validity, since lack of variance in extreme hostile style may weaken associations with CHD. Like the CSS, the IHAT includes four (but unweighted) hostility components: Hostile withhold (evasive and uncooperative responding), Indirect challenge (not overt), Direct challenge (overt antagonism) and Irritation (hostile affect in voice). While the first three components strictly assess behavior, the latter also assesses hostile affect. As with Dembroski and Costa's (1987) system, the IHAT differentiates between content and expressed behavior. As with the CSS, the IHAT ratings are based on each question in the SI. Although the IHAT is time-consuming, its inter-rater reliability and predictive validity in relation to CAD are strong relative to other measures in other studies (Barefoot, 1992). Future studies may wish to compare the reliability and predictive validity of different measures (e.g., PH versus IHAT) using the same sample and outcome criterion. This issue will be detailed in Chapter Three.

Peer or spouse measures of hostility.

Observed techniques derive their ratings from a limited sample of subjects' behavior. By asking a peer or family member to rate subjects' hostility, ratings derived from more and longer observations are provided. However, the problems of peer's self-awareness, and the peer's awareness of the subject, and presentation biases still remain. It has been recommended to use peers that are well acquainted with the subject (e.g., spouses; Barefoot & Lipkus, 1994). Correlations between spouse and self-reports for overt hostility are expected to be stronger than for covert hostility (e.g., Cynicism; Barefoot & Lipkus, 1994). Spouses' ratings of subjects' anger and hostility have been shown to be significantly correlated with self-reports, and their predictive validity in relation to CHD is promising (Kneip et al., 1993).

The epidemiology and development of hostility

Epidemiology of hostility. Since hostility is a risk factor for CHD and mortality (e.g., Miller et al., in press; Smith, 1992), knowing the distribution of hostility in different populations and its sociodemographic correlates is central to detecting specific groups who are at the highest health risk. In addition, establishing standard norms of hostility levels may allow investigators to determine how representative their sample is in a given study. In a national survey in the U.S, Barefoot et al., (1991) examined the sociodemographic correlates of Ho scores and of Barefoot et al.'s (1989) refined Ho subscales. The sample included 1118 men and 1418 women from a wide range of socioeconomic status (SES). Age, gender, income, occupation, education and

race were significantly related to Ho scores. Age was related to full Ho scores and to Barefoot's Cynicism subscale in a curvilinear relation, with younger (18-29) and older subjects (60-90) having higher scores than middle aged men and women (30-59). Men scored higher than women in all age groups, and particularly on Barefoot's Aggressive-Responding and Cynicism subscales. Ho scores were inversely related to occupational status, in agreement with Shekelle et al. (1983) and inversely related to years of education. The only significant interaction was between income and race, indicating that income affected Ho scores only among non-whites. Age, gender, occupation, education, income, race and the income by race interaction accounted for 15.6% of the variance in Ho scores. Th[illegible], hostility, particularly Cynicism, may be higher among young and old, but not middle-aged people, and hostility may be higher in men, uneducated people, and low income non-whites. Finally, Barefoot et al. (1991) added that the effects of SES on health may be partly accounted for by the relation between SES and hostility.

In contrast to younger subjects' higher scores on cognitive hostility (the main dimension assessed by the Ho scale), Musante et al. (1989) found that younger subjects (mean age = 20) scored significantly lower on PH than middle-aged subjects (mean age = 40). Thus, middle-aged subjects score lower on cognitive hostility and higher on behavioral hostility (the main dimension assessed by PH).

Since Barefoot et al. (1991) and Musante et al. (1989) did not employ a

prospective design, it is difficult to know which environmental and personal changes account for the effects of age on hostility. Future research may validate the following hypotheses: Young adults may be more cynically hostile as a residue from adolescent antagonistic attitudes or "ideology" (e.g., opposing/mistrusting parental figures). In contrast, cynical thoughts in old-age people may reflect an enduring experience with untrustworthy people and institutions, and a defence against age-related vulnerability. The increase in behavioral hostility observed in middle-aged people may reflect the experience of more frequent and committed interpersonal challenges (e.g., marriage, work) than young adults experience

Developmental and etiological factors in hostility. Knowing the etiology of hostility may help to prevent its full manifestations and unhealthy consequences and may help treat hostile people. Erickson (1963) emphasized early "basic trust" as a precursor for healthy psychosocial development later in life. Thus, according to this view, general mistrust, a key factor in cynical hostility (Smith & Frohm, 1985) that reflects a degree of maladjustment, may result from hostile individuals' lack of basic trust during their childhood. Supporting Erickson's theoretically-based statements, Houston and Vavak (1991) found that subjects scoring high on the Ho scale reported having less genuine acceptance and more rejection by their parents, more interference in their childhood desires (e.g., less cognitive independence, more strict control) and more punitiveness (hostile and punitive control). Although based on

retrospective recollections, Houston and Vavak (1991) suggested that hostility may be a function of social learning. Thus, individuals may develop cynical and mistrusting attitudes (high Ho scores) after having been brought up in a family that exhibits mistrust, antagonism, rejection and intolerance.

Houston and Vavak (1991) also suggested two possible hereditary pathways for developing hostile attitudes. First, hostile individuals may inherit a disposition to view and respond to such parental behavior in ways (e.g., hostile attributions) that are instrumental for developing cynicism. Thus, individuals' inherited perception and responding styles to existing parental behavior are the source of cynicism. Second, hostile people inherit characteristics (e.g., low frustration tolerance) that elicit the parental behaviors mentioned above. Thus, hostile individuals may be the instigators of their parents' behavior during childhood. It is possible that, through a transactional mechanism (Smith, 1992), both processes elicit and maintain cynical hostility.

Smith, Pope, Sanders, Allred and O'Keefe (1988) found that high Ho scorers reported moderately more encouragement of personal achievement and less emphasis on moral/religious values in their families compared to low Ho scorers. Although based on retrospective data, these findings suggest that cynically hostile people may develop their cynicism because of encouragement of selfish views of others, and of personal rather than communal goals. As Smith et al. (1988) stated: "The family emphasis on achievement without conscience could conceivably contribute to the development of the cynical "dog

eat dog" world view".

Carmelli, Rosenman and Swan (1988) tested the heritability of Ho scores and its subscales of Cynicism and Paranoid Alienation (Costa et al., 1986) with Monozygotic and Dizygotic male twins. They found that only the Cynicism subscale had a clear genetic component: There were consistently larger intrapair correlations among Monozygotic than Dizygotic twins. Paranoid Alienation may be more affected by environmental factors. However, the reliability of these findings may be limited since they were based on the factor-analyzed Ho subscales of Costa et al. (1986).

Finally, Cates, Houston, Vavak, Crawford and Uttley (1993) tested in a more comprehensive manner the role of genetics in female twins' hostility using the BDHI and the Ho scale. Unlike Carmelli et al. (1988), there was no evidence of heritability for cynicism, suspiciousness or for physical assault. However, there was a clear genetic influence on women's trait-anger and indirect and verbal hostility. Differences in gender and measures of hostility may account for these discrepant findings. Since the genetic component did not exceed 50%, these findings suggest that hostility, particularly cynicism and suspiciousness, may be influenced to a greater extent by the environment than by genetic factors (Cates et al., 1993). This implies that cognitive hostility may be more open for therapeutic change. However, since half of the variance in hostility was unaccounted for by genetic factors, affective and behavioral hostility may be partly modifiable by therapy as well.

Summary

Hostility is a multidimensional construct which includes cognitive (cynicism, mistrust and hostile attributions), affective (anger, annoyance) and behavioral (antagonism) components. The two most widely used measures of hostility are the self-report Ho scale and measures derived from the SI (e.g., PH· Smith, 1992). Each of these measures has psychometric strengths and weaknesses, and each assesses different aspects of the hostility construct. While the Ho scale primarily measures cynical hostility (e.g., Smith & Frohm, 1985), it assesses anger and antagonism as well. Barefoot's refined Ho scale, which assesses hostility's three components, is a better marker of antagonistic hostility and is only mildly related to neurotic hostility when compared to the full Ho scale (Barefoot et al., 1989). The SI-derived measures of PH and Hostile Style have been recommended as good measures of Antagonism (Costa et al., 1989; Dembroski & Costa, 1987). While the construct validity of hostility measures is clear and empirically supported in males, it is different and remains unclear and understudied in females (Davidson & Hall, 1995; Greenglass & Julkunen, 1990). Hostility is associated with several demographic variables (e.g., age, gender; Barefoot et al., 1991), and this should be considered in clinical research. Finally, behavioral genetic studies imply that hostility, particularly its cognitive component, is primarily determined by the environment. Thus, therapeutic interventions may succeed in modifying hostility, and in spite of its incredible stability over time.

CHAPTER THREE

THE EMPIRICAL EVIDENCE FOR THE HOSTILITY-CHD RELATION

Introduction

This chapter will review the majority of published studies that examined the relation between hostility and several coronary heart disease (CHD) end-points. These studies were found in two recent reviews on this topic (Helmers, Posluszny & Krantz, 1994; Miller, Smith, Turner, Guijarro & Hallet, in press), from recent searches conducted with the Med-Line and Psychlit computer systems, and from references of obtained studies. These studies are central to this thesis since they provide the empirical basis and guidelines, and refine the theoretical rationale for developing a focused hostility-reduction intervention for CHD patients. The chapter will however, begin by briefly reviewing the Type-A-CHD relation and its limitations. We will then shift to studies on hostility and CHD.

The weight of each study will be assessed in light of its methodological strengths and weaknesses. The methodological ratings include poor, medium and good ratings. These were based on the following criteria: Subject selection (randomization, representativeness), assessment of hostility (reliability, validity), validity of outcomes (measurement-precision, validity), blindness of hostility raters/subjects to outcome status, and control for traditional risk factors. This rating system, though not comprehensive, was meant to provide some indication of each study's internal validity, and was devised according to

common criteria for evaluating the methodological merits of studies (e.g., Maher, 1993). Although some studies clearly suffer from methodological limitations, particularly older ones, they are presented in order to provide a comprehensive review of past and recent studies, and in order to see the development of solutions for methodological problems that prevail in this area of research.

The studies are divided according to the type of outcome assessed. a) Coronary artery disease (CAD), carotid and peripheral artery disease, b) Angina pectoris (AP); c) Cross-sectional CHD studies; d) Prospective CHD studies. The methodological strengths and weaknesses of each group of outcomes and designs will be discussed, and will be followed by a review of each of the studies in their chronological order. Finally, the chapter will be completed by integrating the findings.

The Type-A story

The search for psychosocial CHD risk factors in addition to "traditional" CHD risk factors (e.g., blood-pressure - BP, smoking, cholesterol) stems from several findings. First, traditional risk factors fail to account for all CHD cases. Keys et al. (1972) showed that approximately 50% of CHD cases occurring over a five-year period were unaccounted for by mens' age, relative weight, serum cholesterol, systolic-BP (SBP) and smoking. Other recent studies have shown that traditional risk factors accounted for only 6.25% of CHD cases over a 30-year follow-up (Leon et al., 1988). Most of the traditional risk factors are

actually behavioral in nature (i.e., smoking, obesity, alcohol-consumption, physical inactivity; Jenkins, 1988). Thus, elevations on traditional CHD risk factors may reflect poor health behaviors, which may result from psychological factors. Second, psychological factors have been thought to contribute to cardiac and health problems as early as biblical and Talmudic periods (Siegman, 1994). Some of these early accounts included factors such as anger.

Modern research in coronary-prone behavior originates from the systematic work of two cardiologists, M. Friedman and R. Rosenman. Their research stemmed from viewing the limited contribution of traditional risk factors, from observing a decline in CHD-prevalence with no concomitant alterations in other risk factors (e.g., eating, smoking), and from observing a cluster of behaviors common in their CHD patients.

Friedman and Rosenman (1959) compared 83 men who were categorized as group "A" with 83 men categorized as group "B" and 46 visually-impaired (blind) men who were similar to group B, but who showed manifestations of Neuroticism as well. Group A was labeled Pattern A and included people who exhibited a drive for achieving poorly defined goals, competitiveness, a strong desire for recognition, involvement in multiple tasks under deadlines, acceleration of physical and mental tasks, and extraordinary alertness. People labeled Pattern B lacked the behaviors seen in Pattern A people. This study found that Pattern A men had a significantly higher level of

cholesterol, a faster blood-clotting time and seven times higher prevalence of CHD than Pattern B men. In addition, none of the other factors, namely cholesterol, coagulation time, caloric intake, could explain the differences in CHD incidence between the groups. This was a first systematic attempt to link behavior to CHD. However, the reliability of labeling subjects was questionable as subjects were "diagnosed" and selected for the study by their colleagues and superiors. In addition, the interview for determining behavioral patterns was not standardized. Finally, several CHD cases were based only on self-reported history rather than objective indices of CHD (e.g., ECG, enzymes).

Further improvements on the conceptualization and measurement of Pattern A, which was termed the Type A Behavioral Pattern (TABP) resulted in a more precise definition: An "action-emotion complex, that can be observed in any person who is **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, 1974). Rather than depending on reports from questionnaires, a standardized Structured Interview (SI; Rosenman, 1978) for assessing the TABP was developed. This was done because CHD patients may not have much "psychological" insight for self-report, and since speech stylistics rather than content appeared to be the critical aspect of the TABP (Rosenman, 1978).

Rosenman et al. (1975) conducted the first prospective study that examined the relation between the TABP and CHD, using an early version of

the SI to assess the TABP. They followed 3154 men free of CHD at intake for an average of eight and a half years in the Western Collaborative Group Study (WCGS). Behavioral pattern (A or B) significantly predicted CHD incidence, fatal and non-fatal MI and sudden death, silent MI, and angina pectoris, and these findings were independent of other risk factors (e.g., SBP,DBP, cholesterol). This study was methodologically much more sound than that of Rosenman and Friedman (1959), and it demonstrated that Type-A was a significant and independent risk factor for developing CHD.

Further studies demonstrated an association between TABP and other cardiovascular end-points such as atherosclerosis. For example, Blumental, Williams, Kong, Schanberg and Thompson (1978) found that prevalence of SI-derived TABP was significantly higher than Type-B as severity of atherosclerosis increased, and this was independent of age, gender, BP, cholesterol and smoking. However, self-reported TABP (the Jenkins Activity Survey, JAS; Jenkins, Zyzanski & Rosenman, 1971) was not related to severity of atherosclerosis. The authors concluded that the SI-derived TABP may contribute to the etiology of CHD via the atherosclerotic process.

These important findings inspired two major conferences, sponsored by the American National Heart, Lung, and Blood Institute, and a final panel concluded that the "coronary-prone" TABP was an independent CHD risk factor, having the same order of magnitude as smoking, SBP or cholesterol (Review Panel on Coronary-Prone Behavior and Coronary Heart Disease, 1981).

However, the panel recommended that improvements be made in assessing the TABP, and that it would **not** be equated with the term "coronary-prone" since the latter assumes a causal relation between TABP and CHD.

Three major problems began to emerge with respect to the TABP. First, and of greatest importance was the failure of replication: Negative findings appeared from several well-designed studies. The Multiple Risk Factor Intervention Trial (MRFIT; Shekelle et al., 1985) found no prospective association between SI-derived TABP and CHD events over 7.1 years, in univariate or multivariate analyses adjusting for age, DBP, cholesterol, alcohol-consumption and education level. Dimsdale, Gilbert, Hutter, Hackett and Block (1981) found that men who showed **absence** of TABP (assessed with the JAS) were at greater risk for cardiac morbidity. However, their study included outcomes that may not reflect "hard" CHD (i.e., resuscitation, hospitalization for cardiac problems). Finally, in a methodologically sound and more recent study (Ragland & Brand, 1988), Type-A men with CHD were at a significantly **lower** risk of CHD-related mortality than their Type B counterparts, even after controlling for smoking, SBP, cholesterol, age during and type of first CHD event. Although risk factors for primary versus secondary events may differ, and this may explain the latter negative findings, the consistent emergence of negative and even opposite findings placed strong doubt on the the TABP-CHD relation. Thus, the review panel's conclusions (1981) may have been premature.

The second main problem with the Type-A construct was that the prevalence of the TABP in many samples was very high (70% - 90%; e.g., Dembroski & Costa, 1987; Shekelle et al., 1985). This prevalence renders the TABP as an unreliable CHD risk factor, since the prevalence of CHD is by far lower than that of TABP (Dembroski & Costa, 1987). This high prevalence of the TABP during an era where CHD-prevalence is declining (Jenkins, 1988) further reduces the chances for obtaining a reliable TABP-CHD relation.

The third problem with the Type-A construct, and of greatest importance to this thesis, was that several studies showed that **hostility**, one of the key components of the TABP (Friedman & Rosenman, 1974), was a better predictor of CHD end-points than the global TABP (e.g., Arrwood, Uhrich, Gomillion, Popio & Raft, 1982; Dembroski, MacDougall, Williams & Haney, 1985; MacDougall, Dembroski, Dimsdale & Hackett, 1985; Williams et al., 1980). Since TABP consists of several components (i.e. hostility, competitiveness, time-urgency, vocal stylistics; Friedman & Rosenman, 1974; Rosenman, 1978), a person may be designated as Type-A for being high on any one of these components. While the SI-derived TABP was heavily influenced by subjects' vocal stylistics (in addition to hostility; Dembroski & Costa, 1987), the JAS mainly assessed competitiveness, time-urgency and achievement (Jenkins et al., 1971), and neither consistently predicted CHD. If only one aspect of the TABP, hostility, is "toxic", the positive relations between hostility and CHD, and the negative or lack of relations between other components, such as

competitiveness or time-urgency, and CHD, will cancel each other, and so global TABP will not predict CHD. These findings were in line with recommendations of the Review Panel on Coronary-Prone Behavior and Coronary Heart Disease (1981) to examine the relations between the components of the TABP and CHD. Thus, the next generation of studies examined the components of the TABP, with particular focus on the assessment and predictive validity of the hostility complex (Williams, 1987).

Evidence for relations with coronary, carotid and peripheral artery disease

This category includes patients who develop coronary atherosclerosis, carotid atherosclerosis, and peripheral arterial diseases (atherosclerosis in the limbs). Studying these diseases is important not only for understanding their etiology, but also since coronary atherosclerosis is a precursor of CHD (Sokolow & McLlory, 1986). Thus, finding associations between hostility and atherosclerosis may be a mechanism by which hostility contributes to the development of CHD. Additionally, attempting to alter hostility in atherosclerotic, non-CHD patients, may actually prevent CHD.

However, several methodological biases exist in these studies, particularly angiographic ones. Two types of selection-biases exist. First, angiography studies may include primarily severe CAD cases. After patients undergo preliminary tests (e.g., stress-tests) to determine presence/absence of suspected CAD and CHD, high risk patients with severe test results and/or symptoms undergo the more expensive and risky angiography test, to precisely

detect presence and degree of CAD. Since most angiography subjects have confirmed CAD (approximately 92% of subjects) this results in a skewed distribution of CAD-presence (Pickering, 1986), which can reduce the magnitude of the associations between hostility (or other risk factors) and CAD-presence (Helmers et al., 1994; Pickering, 1986).

A second selection bias may stem from a number of patients who may be free of CAD, but, who are nevertheless referred for angiography due to persistent complaining of chest-pain. These patients are normally high on Neuroticism and on hostility measures that assess Neuroticism in addition to hostility (e.g., Barefoot's non-CHD- and mortality predictive Ho subscales; Barefoot et al., 1989; and experiential subscales of the BDHI; Siegman et al., 1987). The lack of CAD among these neurotically hostile patients may cancel out positive associations between antagonistic hostility and CAD in antagonistically hostile patients with CAD, and reduce the association between global hostility and CAD (Helmers et al., 1994; Siegman et al., 1987). However, these CAD-free patients should not be considered healthy controls since they exhibit high and chronic degrees of functional disability and health complaints (Ockene, Shay, Alpert, Weiner & Dalen, 1980). This constellation of symptoms and problems reflects high levels of Neuroticism and somatization (Costa & McRae, 1987; Watson & Pennebaker, 1989).

Additionally, angiography patients, who in many cases have advanced CAD or CHD, may be taking beta-blockers to reduce myocardial oxygen

demands, and thus to prevent ischemic episodes and chest-pain. These drugs can reduce hostility levels (Krantz et al., 1982), and this may seriously alter the reliability of hostility assessments, and alter the relation between hostility and CAD. On the other hand, angiography patients may have repeated and severe chest-pain which may elevate their levels of cynical mistrust and anger towards people in general and health professionals specifically. This may not allow one to determine whether hostility plays a role in CAD-development or results from CAD-symptoms.

As can be seen in Table 1, studies differ markedly in the criteria for determining clinically significant occlusions (e.g., 50% or 75% stenosis) or severity of CAD (e.g., number or location of occlusions, or both). These differences in rating CAD-severity may affect the magnitude of associations found in each study between psychological risk factors and CAD (Pickering, 1986). In relation to design issues, angiographic and other artery disease studies are cross-sectional designs, and this strongly limits any causal inferences between hostility and CAD. Finally Pickering challenges whether CAD is a valid marker of CHD since it is not a necessary condition for CHD mortality. Thus, other process variables, more transient ones, such as platelet aggregation, arrhythmia and ischemia, which lead to CHD events, should also be studied in relation to hostility. Table 1 presents the study characteristics of studies that examined the link between hostility and CAD, carotid atherosclerosis, and peripheral artery disease. Unless indicated differently, all

samples include American subjects. Below is a chronological review of each of the studies listed in Table 1.

Williams et al. (1980) found a threshold effect for hostility. Among patients scoring above 10 on the Ho scale, 70% had clinically significant atherosclerosis compared to only 48% of patients scoring 10 or less. After controlling for the effects of gender and TABP, hostility predicted atherosclerosis more strongly than did TABP after controlling for gender and Ho scores. Hostility had a stronger impact on CAD in the multivariate test than in the univariate test, while the opposite occurred with TABP. Thus, gender and TABP act as suppressor variables in the relation between hostility and CAD, while gender and hostility account for part of the relation between TABP and CAD. Items assessing cognitive hostility (i.e., their content implied that others are inconsiderate, immoral and selfish) and behavioral hostility (open antagonism toward others) were the critical Ho items distinguishing between those high and low in risk for CAD. This pioneering study had two main limitations. First, since patients who were very ill and patients with very minimal disease were excluded from the study, the results may be generalized to moderate levels of CAD alone. Second, the effects of traditional risk factors such as cholesterol and BP were not controlled for in the analyses.

Arrowood et al. (1982) examined a sample that was randomly selected from a rural population and admitted for angiography. Hostility, as assessed with the SI, was significantly correlated with CAD presence (26). However,

since data were only published as an abstract, gender of patients and procedure for diagnosing CAD were not provided, and it was unclear whether the psychological factors were associated with CAD after controlling for traditional risk factors.

Dembroski, MacDougall, Williams, Haney and Blumenthal (1985) used stratified random sampling with equal numbers of patients with different levels of CAD-severity. Both PH and Anger-In were significantly associated with CAD measures, even after controlling for age, sex, hypertension, smoking, hyperlipidemia and family history. There was an interaction between PH and Anger-In, where PH was positively associated with CAD only among patients who suppressed their anger. However, this study did not indicate whether there were any gender differences in the observed relations.

MacDougall et al. (1985) attempted to replicate the findings of Dembroski et al. (1985) in another population. While TABP was not associated with CAD severity ($r = -.02$), PH and Anger-In were ($r = .18$, $r = .28$, respectively). These relations remained significant after controlling for age, hypertension, smoking, family history and cholesterol. This time, PH and Anger-In did not interact in their effect on CAD as Dembroski et al. (1985) had found. Inspection of the results reveals that MacDougall et al. (1985) found a negative correlation between PH and Anger-In. Thus, conceptually and empirically it might be difficult to find individuals in their sample who were high on both factors. Therefore, these factors must have had additive effects on CAD, and the

scarcity of subjects elevated on both factors may have provided insufficient statistical power for detecting a synergistic interaction.

Tennant, Langeluddecke, Flucher and Wilby (1987) found that trait-anger was associated with severity of atherosclerosis, however, this was not maintained after controlling for effects of age. Suppression of anger was also unrelated to CAD, and these results did not change with different indices of CAD (e.g., number of arteries occluded more than 50% or 75%). However, the data from which CAD was rated originated from different medical centers, possibly with differences in the data provided. This may have affected the reliability and validity of their CAD ratings, which was also not indicated. Additionally, subjects with very severe CAD were excluded from the study (awaiting by-pass surgery), and this may have limited the range of CAD scores and reduced the ability to detect correlations between psychological measures and CAD. Finally, the authors stated that a proportion of their sample did not have CAD. Negative associations between anger and CAD for subjects high on Neuroticism without CAD may have canceled out the relation between anger and CAD among non-neurotic subjects with CAD (Siegman et al., 1987).

Siegman et al. (1987) tested the predictive power of neurotic hostility versus antagonistic hostility. The former included the Resentment and Suspicion subscales, and the latter included the Assault, Indirect Hostility and Verbal Hostility subscales of the BDHI. In patients at or below age 60, neurotic hostility was negatively related to CAD and antagonistic hostility was positively

and significantly associated with CAD, after controlling for gender and anxiety levels. Neither type of hostility was associated with CAD among patients above age 60. These results refine our understanding of the relation between hostility and CAD and suggest that antagonistic hostility may be the toxic component of hostility, while neurotic hostility is unrelated to CAD, and that age moderates the relation between hostility and CAD.

However, Helmer, Ragland and Syme (1991) did not find relations between observed or self-reported hostility and CAD, after controlling for nine traditional risk factors. The Ho scale was unrelated to CAD using three different cut-off points for the scale. Helmer et al. (1991) recommended using caution in deriving broad conclusions from individual studies in this area of research.

In contrast, one recent study was conducted with young CAD males (below 50 years; Haney et al. 1992; cited in Barefoot, 1992). This study found the strongest association ever documented between hostility (using the Interpersonal Hostility Assessment Technique; IHAT) and severity of CAD (e.g. $r = .59$). This remained intact after controlling for the effects of age, smoking, hyperlipidemia and hypertension, which were **not** significantly correlated with CAD in this small sample. Thus, the relation between IHAT-assessed hostility and CAD-severity appears **very** strong. Barefoot (1992) added that this relation mainly stemmed from the impact on CAD of Indirect Hostility, the type of hostility more frequently observed during the SI.

Angiography studies include patients with severe CHD symptoms, who may either have actual disease or complain of symptoms without CAD. As mentioned above, these selection biases may weaken relations between hostility and CAD. Having symptoms may also affect hostility levels by making one irritable and mistrusting of previous maltreatment. Thus, Barefoot et al. (1994) examined randomly selected **asymptomatic** men with preliminary evidence for CAD as detected by routine check-ups. Only observed IHAT-assessed hostility was significantly associated with CAD. However, hostility and smoking interacted such that hostility was positively associated with CAD presence only among non-smokers. Given this study's strong methodology, observed interpersonal hostility may have a reliable role in CAD pathogenesis. Self-reported Ho scores may have been unrelated to CAD because subjects had low scores (compared to published norms; Barefoot et al., 1991) while being evaluated for work status. This study also suggests that interactions between hostility and other risk factors (e.g., smoking) need to be considered in predicting and understanding the relation between hostility and CAD.

Several studies examined the role of hostility in other vascular diseases. Stevens, Turner, Rodewalt and Talbot (1984) found a curvilinear relation, with higher hostility scores among moderate levels of carotid atherosclerosis than among non-diseased and severely diseased subjects. Hostility levels were significantly higher in the moderate than the non-diseased group only in men, but not in women. However, hostility was assessed with a simple three-item

measure, degree of carotid-atherosclerosis was not precisely measured, the small sample size may have obscured effects within each gender, and additional traditional risk factors were not considered.

A recent study by Julkunen, Salonen, Kaplan, Chesney and Salonen (1994) examined the relation between hostility and two-year change in intima-media thickness (progression of carotid atherosclerosis; PCA). Disease status was assessed with greater precision than by Stevens et al. (1984). After controlling for several risk factors (e.g., age, smoking, LDL-cholesterol, base-line intima-media thickness), the additive combination of Cynical Distrust and Anger-Control contributed to the largest extent in predicting PCA. Subjects high on both Cynical Distrust and Anger-Control showed a two-fold accelerated PCA compared to subjects low on both measures. Thus, cognitive hostility and the way people cope with angry feelings are important predictors of atherosclerotic progression, and may need to be focussed upon in patients with early stages of carotid atherosclerosis. However, psychological assessments were conducted one year after assessment of base-line carotid-atherosclerosis, and knowledge of the latter may have affected hostility levels.

Two studies examined the role of hostility in peripheral artery disease (PAD), manifested by periodical pain, tension and weakness in the legs resulting from atherosclerosis or valvular problems in the limbs. Joesoef, Wettterhall, DeStefano, Stroup and Fronek (1989) found that prevalence of PAD was significantly related to Ho scores. The PAD odds ratios tended to

significantly increase with increased Ho quartile scores, and this remained intact after controlling for age, race, smoking, CHD family history, diabetes and LDL/HDL cholesterol ratio. The hostility-PAD odds-ratios were similar in magnitude to those of traditional risk factors and PAD. However, it was unclear whether physicians were blind to subjects' Ho scores, and the reliability of the assessment of PAD was not indicated.

Finally, Deary, Fowkers, Donnan and Housley (1994) examined symptomatic PAD, asymptomatic PAD, and control subjects in an Edinburgh randomly selected population study. While Type-A was significantly but inversely related to severity of PAD, the odds ratio of having the most severe form of PAD compared to no disease was 1.41 with an increase of one SD in self-reported hostile acts in men, and this was independent of age and smoking. The odds ratio of having major asymptomatic PAD compared to no disease was 1.39 with increases in self-reported cognitive hostility, independent of age and sex. Thus, hostility is a risk factor for PAD in men, and this relation does not appear to be the consequence of having symptoms. However, the most severe form of PAD was assessed with a self-report measure, unlike the other categories of PAD-severity.

These studies do suggest a consistent relation (85% of studies) between hostility and coronary, carotid and peripheral atherosclerosis. Hostility may have general effects on the vascular system beyond the coronary arteries, and this relates to the important issue in behavioral medicine concerning generality

versus specificity of effects of risk factors. Pickering's strong arguments about the methodological limitations of angiographic studies suggest that one should focus more on **degree** of CAD rather than CAD presence/absence, that one should examine the relations between hostility and other CHD markers, and finally, that one should use prospective designs.

Table 1

Studies linking hostility with coronary, carotid and peripheral artery disease.

Authors	Subject characteristics	Hostility assessment	Outcome	Results	Methodology
Arwood et al (1982)	74 CAD subjects randomly selected	SI-derived measure	≥ 75% occlusion	+	Medium
Barefoot et al (1994)	25 CAD & 25 control asymptomatic men	SI-derived IHAT & Ho	> 50% occlusion	+	Good
Deary et al (1994)	1592 men & women from Edinburgh	Hostile acts & Cognitive hostility	4-graded PAD severity	+	Good
Dembroski et al (1985)	131 patients 98 men, 33 women randomly selected	SI-derived PH & anger-In, & Ho	# of occlusions > 75% & coronary index	+	Good
Haney et al (1992)	98 young men	SI-derived IHAT	6-level CAD index	+	Good
Helmer et al (1991)	158 subjects, 118 men, 40 women	SI derived hostility & Ho	≥ 75% occlusion & mean CAD score	-	Good

Authors	Subject characteristics	Hostility assessment	Outcome	Results	Methodology
Joesoef et al (1989)	4,462 young male veterans	Ho scale	Doppler ultrasonography	+	Medium
Julkunen et al (1994).	119 Finnish men	Ho, Anger-In & Control & Irritability	Change in carotid atherosclerosis	+	Good
MacDougall et al. (1985)	125 CAD males	SI-derived PH & Anger-In	# of occlusions ≥ 50%	+	Good
Siegman et al. (1987)	72 subjects 51 men, 21 women	BDHI	# of occlusions > 50% & CAD index	+	Good
Stevens et al (1984)	44 subjects 21 men 23 women	3-item scale	3-grade stenosis in carotid artery	+	Medium
Tennant et al (1987)	519 Australian subjects, 78% men	Trait-anger & anger-suppression	Several CAD indices	-	Medium
Williams et al. (1980)	424 CAD subjects	Ho scale	Presence of ≥ 75% occlusion	+	Good

Evidence for relations with angina pectoris

The pathophysiology of unstable angina (UA) has been detailed in Chapter 1. Angina pectoris (AP) and UA are forms of ischemic heart disease that have not resulted in significant myocardial necrosis as in MI. However patients with AP or UA develop debilitating chest and abdominal pains leading to high levels of functional disability (Ockene et al., 1980), and are at high risk for MI and sudden death (Plotnick, 1985a). Thus, detecting psychological risk factors for developing UA is important for treating these patients in an attempt to prevent further progression to MI or coronary death.

We must distinguish between angina that is cardiac in its origin, with an underlying cause of myocardial ischemia, and angina with no detectable cardiac basis or pseudoangina. Costa (1987) reviewed several studies on pseudoangina, and showed that it can be predicted by different measures of Neuroticism. Accordingly, self-reported chest-pain is correlated strongly with patients' Neuroticism, and self-reported and physician-rated chest-pain are not correlated with CAD (Costa, 1987). Patients high on Neuroticism were not at greater risk of death from MI, and even had significantly less severe CAD. Costa (1987) concluded that Neuroticism is a risk factor only for pseudoangina. Such patients continuously manifest functional disability despite knowing that their chest-pain is not cardiac (Ockene et al., 1980). Finally, patients seem to be diagnosed more according to their symptom reporting, in other words their Neuroticism levels, rather than according to objective tests of CAD. Thus,

Neuroticism has a role in the etiology of pseudoangina, and this may happen in the following manner. Individuals high on Neuroticism may attend more to unpleasant somatic sensations (attention bias), perceive them as more threatening (perceptual bias), and then report more somatic complaints (report bias) than individuals low on Neuroticism (Costa, 1987; Watson & Pennebaker, 1989). Neurotic patients' reported symptoms need to be considered with more caution than those of non-neurotic patients, and objective tests need to be given more weight for the former than for the latter. In this section, UA or AP are referred to as CHD when an underlying cardiac basis is confirmed (via angiography, stress-tests or previous MI), although not all studies allow this distinction. Table 2 presents the main characteristics and findings of these studies, and a chronological review of these studies follows.

Jenkins, Stanton, Klein, Savageau and Harken (1983) examined men awaiting coronary-artery-by-pass-graft (CABG) surgery with underlying CAD. Subjects were asked about their experience of chest-pain following different levels of effort, at rest/sleep (UA), when upset and angry (emotional angina), and after a heavy meal. Forty-four percent of the patients reported pain when upset or angry. Hostility (assessed with the Profile of Mood States, POMS; McNair, Lorr & Droppleman, 1971) was significantly associated with emotional angina, and hostility was more strongly associated with angina at rest than age or smoking. Since resting angina may be related to reduced blood-supply caused by spasms (Maseri et al., 1979), hostility may be related to coronary

spasms. However, patient vere asked about the chest-pain they had experienced during 30 to 90 days prior to the study, and recall biases may have reduced the reliability of this assessment.

One prospective study examined the relation between hostility and AP (Barefoot, Dahlstrom & Williams, 1983). Subjects who scored above the median on the Ho scale (i.e., 13) were significantly nearly six times at greater risk for having non-fatal CHD than those scoring at or below the median. The CHD cases included AP and MI. However, it was unclear whether Ho scores predicted AP alone, the number of CHD cases (11) may have precluded specific end-point analyses, and the validity of these self-diagnoses was not confirmed. In addition, it was unclear whether AP included underlying CAD in all cases. Nevertheless, this study suggests that hostility may be a risk factor for subsequent AP.

Smith, Follick and Korr (1984) found that while age, gender and CAD-severity were unrelated or marginally related to angina, trait-anger was significantly related to angina frequency ($r = .55$) and to angina's interference with activities ($r = .53$). However, it was unclear whether all patients had underlying CAD, and CAD-severity was not controlled for in the relation between trait-anger and angina outcomes.

One case-control study conducted in Spain examined AP and healthy control subjects (Bernardo, De Flores, Valdes, Mestre & Fernandez, 1987). Two independent cardiologists verified patients' AP. In both males and

females, AP patients were higher than controls on all BDHI hostility subscales after matching on age. However, the precise criteria for evaluating AP were not provided, and additional risk factors (e.g., cholesterol, BP) were not controlled for. That Neuroticism was also higher in AP patients suggests that not all patients may have had an underlying cardiac disease (Costa, 1987).

Dembroski et al. (1985) found that SI-derived PH was significantly associated with presence of AP ($r = .27$) among angiographic patients. This remained significant after controlling for subjects' cholesterol levels, sex, age, smoking and hypertension status. Anger-In derived from the SI was not associated with AP.

Hallstrom, Lapidus, Bengtsson and Edstrom (1986) did not find any prospective relation between Aggression or assertiveness and AP in a 12-year follow-up study. Other psychosocial parameters (Neuroticism, mental strain and depression) were predictive of AP. However, the psychometric properties of their measure (the Cesarec-Marke's Personality Schedule, CMPS; Cesarec & Marke, 1968) were unclear.

A Finnish study (Koskenvuo et al., 1988) showed that hostility (assessed with three items about irritability, ease of anger, and argumentativeness) was significantly related to prevalence of AP, after controlling for the effects of age. However, it was unclear whether AP included only verified cardiac cases.

Finally, Mendes de Leon (1992) wished to examine how anger measures are related to the diagnosis of CHD in a low socioeconomic status (SES) group,

a relatively understudied, though high risk, group (Jenkins, 1988). All UA patients had underlying cardiac disease (verified with angiographic or ECG tests). No differences were found between UA and control groups on any anger measures. Cases with previous MI were then removed, since previous disease may alter behavior, and thus possibly suppress or alter the relation between behavior and CHD. Dembroski and Costa (1987) also speculated that cardiac patients become higher on Anger-In, following physicians' recommendations to avoid upsetting reactions. After this deletion of cases, mean Anger-Out scores were significantly higher in the UA group than in the controls. These differences remained intact after controlling for age, marital status, education, smoking, hypertension and chronicity of clinical condition. Trait-Anger and Anger-In, which reflect experienced hostility (Costa et al., 1989) were not different among UA and control patients. Mendes de Leon (1992) confirmed previous findings (e.g., Siegman et al., 1987) and concluded that expressed or antagonistic hostility (Anger-Out) is the "coronary-prone" aspect of hostility, rather than experienced or neurotic hostility (Costa et al., 1989). Since these effects emerged after removing cases with previous MI and after controlling for disease chronicity, Anger-Out may be a precursor rather than a consequence of UA. However, it was unclear whether all patients in the UA group suffered from UA or from stable AP. Despite this limitation, this important and well designed study examined the correlates of UA in a specific SES group, and provided relatively sound support to the link between hostility and UA.

In summary, based on these studies, hostility, anger and its antagonistic mode of expression, namely Anger-Out, appear to be consistently (86% of studies) associated with AP and UA. The main limitation of these studies is that most did not verify presence of underlying cardiac disease, and no study verified whether patients had UA or AP. Since these diagnostic categories are difficult to determine reliably (Plotnick, 1985a), associating hostility with graded and more precise indices of CHD, such as transient ischemia, may yield more compelling evidence for the hostility-CHD relation

Taole 2

Studies linking hostility with angina pectoris and unstable angina

Authors	Subject characteristics	Hostility assessment	Outcome	Results	Methodology
Barefoot et al (1983)	255 physicians	Ho scale	AP (& MI) after 25 years	+	Medium
Bernardo et al (1987)	89 men & 19 Spanish women with AP 157 male & 95 female controls	BDHI	Presence of AP	+	Poor
Dembroski et al (1985)	131 subjects, 98 men 33 women	SI derived PH & Anger In	Presence of AP	+	Good
Hallstrom et al (1986)	795 age-stratified Swedish women	Aggression & neurotic assertiveness	AP		Good
Jenkins et al (1983).	204 men with CAD awaiting surgery	POMS Hostility	Pain at rest & when angry	+	Medium

Authors	Subject characteristics	Hostility assessment	Outcome	Results	Methodology
Koskenvuo et al. (1988).	3750 Finnish men	3-item hostility scale	AP after 3 years	+	Medium
Mendes de Leon (1992).	26 UA & 26 orthopedic controls all male	Anger-In/Out (AX) & Trait-anger	UA-presence	+	Good
Smith et al. (1984).	50 subjects, (37 AP) 40 men, 10 women	Trait-Anger	Pain frequency & interference	+	Poor

Evidence for a relation between hostility and transient ischemia

Pickering (1986) suggested examining associations between behavior and indices of ischemia with non-invasive tests such as thallium-scans. Due to the lower risk associated with thallium scans, this approach includes patients with a greater range of disease severity than angiography samples, thus increasing the chances for detecting associations between behavior and ischemia. In addition, thallium-scans yield more precise measures of CHD than a diagnosis of AP. Excluding patients with previous CHD symptoms from these samples reduces the possibility that hostility results from CHD. Mild levels of myocardial ischemia, normally associated with AP or UA, can be signs of a previous or possible upcoming MI. Thus, examining the correlates of myocardial ischemia can be a way to evaluate a patients' risk of MI (Landenheim et al. 1986).

Kneip et al. (1993) attempted to find the most CHD-prone components of hostility, by using the Multidimensional Anger Inventory (MAI; Siegel, 1986) with self- and spouse-ratings of patients' anger and hostility. Results revealed that ischemic cases did not differ from normals on any self-report MAI subscale. However, ischemic cases were rated by their spouses significantly higher on Hostile Outlook (cognitive hostility) and Anger-In than were controls. After controlling for traditional risk factors (e.g., age, gender, smoking, hypertension), only spouse-rated Hostile-Outlook accounted for an additional and significant 2% of the variance in ischemic status. Removing subjects with symptomatic MI

revealed that scores on all spouse-rated MAI subscales were significantly higher in ischemic cases than in controls. Thus, hostility may be a precursor of ischemia. These findings were similar for males and females. However, the clinical significance of the small effect sizes of hostility may be limited. Despite this limitation, this study revealed that spouse-rated hostility may be more important than self-reports for predicting CHD-risk.

Helmers et al. (1993) assessed hostility with the full Ho scale and with Barefoot al.'s (1989) refined Ho scale. In males below age 60, Barefoot's refined Ho scale accounted for 31% of the variance in number of reversible ischemic segments, and in females there was a similar trend. The full and refined Ho scores significantly predicted total minutes of ischemia per 24 hours (on ECG) in women. Barefoot's refined Ho scores accounted significantly for 51% of the variance in women's number of ischemic events per hour. Finally, hostility accounted for an additional and significant amount of variance in total minutes of ischemia and in maximal ST-segment depression after controlling for gender and number of reversible ischemic segments. These impressive effect-sizes in the relations between hostility and daily ischemic events may partly explain the link between hostility and CHD. This study also reveals how different measures of hostility have different predictive validity, with Barefoot's refined Ho being a better CHD-predictor than the Multi-Dimensional Anger Inventory mentioned above (Kneip et al., 1993).

Burg, Jain, Soufer, Kerns and Zaret (1993) examined the role of anger

and hostility in silent ischemia. During mental stressors, half of the subjects exhibited left-ventricular dysfunction (LVD), but did not differ from those without LVD on traditional risk factors (e.g., age, hypertension). However, subjects with LVD scored significantly higher on Hostile Affect, Aggressive-Responding, and Trait-Anger and lower on Anger-Control than subjects without LVD. In a multivariate test, controlling for severity of disease, Aggressive-Responding and Trait-Anger independently and significantly differentiated between the two groups. Since elevations in heart-rate and BP during stressors were similar in both groups, suggesting a similar increase in oxygen demand, insufficient oxygen supply related to a hostile/angry profile may have yielded the group differences in ventricular functioning. However determining LVD was based on an absolute decrease in ejection-fraction, rather than considering subjects' base-line values. Despite this limitation, this study suggests that during daily stressors, men with CAD, who frequently experience and antagonistically express anger, may be at greater risk for transient silent ischemia possibly induced by spasms (reduced myocardial blood-supply).

Most recently, Helmers et al. (1995) examined the combined effects of hostility and defensiveness (suppression, assessed with the Marlow-Crowne social Desirability scale; Crowne & Marlow, 1964) in transient ischemia. In Study 1, Barefoot's refined Ho scale, but not defensiveness, significantly predicted number of reversible ischemic segments after a stress-test. Controlling for gender and the main effects, the interaction of hostility x

defensiveness accounted for an additional and significant 7.3% of the variance in reversible ischemic segments. In study 2, only hostility significantly predicted number of minutes of daily ischemia. Controlling for gender and the main effects, the interaction of hostility x defensiveness added additional and significant variance to duration and frequency of daily ischemic episodes. Finally, study 3 showed that following two mental stressors, the interaction of hostility x defensiveness had a significant effect on abnormal wall-motions reflecting severity of ischemia. In all three studies, subjects who were both hostile and defensive showed the most severe ischemia compared to those low on one or both measures. However, this study suffered from a few small limitations. First, measuring defensiveness, conceptually an unconscious process, via self-report may be invalid. Second, median-splitting hostility and defensiveness scores, two normally-distributed measures, is statistically incorrect. Third, severity of CAD was not controlled for in all analyses. Finally, subjects in Study 3 were debriefed prior to psychological assessment. Despite these limitations, this research found in three contexts and measures of ischemia that the tendency to experience and repress hostile thoughts was associated with more severe ischemia.

Finally, I shall report upon one important study that examined the effects of inducing anger, rather than the trait of anger, on myocardial ischemia in CAD and healthy subjects (Ironson et al., 1992). An anger-inducing stressor (subjects reported in detail about a recent anger-provoking event) was

compared with two general psychological stressors and with physical exercise. Among CAD patients, the anger-recall task elicited significantly the greatest reduction in ejection-fraction (-5%) than the other psychological and physical tasks. This reduction was significantly larger in CAD patients than in controls. Significantly more CAD patients showed a clinically meaningful reduction in ejection-fraction ($\geq$ 7%) during anger-induction than during other psychological tasks, and compared with controls. However, all change scores were not residualized with respect to base-line ejection-fraction levels (Keppel & Zedek, 1989). Despite this limitation, this important study showed that a stressor specifically related to anger/hostility yielded larger ischemia than more general psychological stressors, supporting the importance of anger and hostility as **the** toxic component of stress-reactions. Second, since the dependent variable was change scores in myocardial functioning following experimentally-induced anger, this study suggests that provocations and reactions related to anger/hostility may be **causally** related to myocardial functioning and ischemia, a theoretically and clinically significant finding.

In summary, these studies provide compelling evidence for the role of hostility in daily and stress-induced ischemia. The different components of hostility (cognitive, affective, behavioral) are involved in this relation and may interact with other personality traits (e.g., suppression; Helmers et al., 1995). Kneip et al. (1993) revealed the importance of spouse ratings of patients' hostility in predicting patients' transient ischemia. Finally, the study by Ironson

et al. (1992) provides important evidence which suggests that specifically anger-hostility, rather than stress-reactions in general, may be **the** psychological toxic parameter causally related to myocardial ischemia and myocardial dysfunction. However, transient ischemia is not a static state of CHD, and the ultimate evidence for the hostility-CHD relation should be derived from studies on CHD as an outcome. The next two, final sections of this chapter review the cross-sectional and prospective relations between hostility and CHD, respectively.

Table 3

Studies linking hostility with transient ischemia.

Authors	Subject characteristics	Hostility assessment	Outcome	Results	Methodology
Burg et al. (1993).	30 CAD males	Barefoot's refined Ho, Anger-In/Out	Reduced ejection-fraction	+	Good
Helmers et al. (1993)	80 CAD subjects, 63 men 17 women	Ho & Barefoot's refined Ho	# of reversible ischemic segments & ST depression	+	Good
Helmers et al. (1995)	79 CAD patients, 39 CAD patients & 30 CAD patients	Barefoot's refined Ho & Marlow-Crowne Social Desirability	# of reversible ischemic segments, ECG & wall-motion abnormality	+	Medium
Kneip et al (1993).	185 cardiac patients (112 men, 73 women)	Multidimensional Anger Inventory	reversible & fixed ischemia	+	Good

Evidence for a cross-sectional relation between hostility and CHD

The pathophysiology of MI has been examined in Chapter 1. MI or sudden death constitute the most harmful outcomes in CHD, in terms of disability, morbidity and mortality. Although finding cross-sectional associations between hostility and incidence of MI and sudden death cannot inform us on the mechanism of this relation, identifying the psychological correlates is of greatest importance for pursuing further, experimental or prospective studies on these relations. Additionally, several cross-sectional and prospective studies on CHD do not include AP in their outcomes, and focus on MI and sudden death alone. The diagnosis of the latter two outcomes is more reliable and definite than that of AP. This makes any observed hostility-CHD link more valid, since patients without objectively-based CHD (i.e., alive or without myocardial necrosis), who may be high on Neuroticism (Watson & Pennebaker, 1989), are excluded from samples. However, the main limitation of cross-sectional designs is in the limited inference they allow. Causality may not be inferred from these studies, even if groups are carefully chosen and alternative hypotheses (i.e., traditional risk factors) are fully considered. Thus, the possibility that hostility may result from CHD in cross-sectional studies always remains. Table 4 presents the main study characteristics and findings of cross-sectional studies on CHD, and their chronological presentation follows.

One of the earliest controlled studies that examined the personality correlates of CHD was done by Miles, Waldfogel, Barrabee and Cobb (1954).

Significantly more MI cases coped inappropriately with anger (using repression or aggression) than controls. Significantly higher suspiciousness scores were found for MI than for control subjects. However, interviewers were not blind to subjects' group status, and assessments took place at a mean of 5.6 years post-MI. Thus, these differences may have resulted from having a MI. In addition, noticeable differences were found between the groups on SES measures, which were not considered in the analyses. Finally, combining repressors and aggressors for statistical purposes may be conceptually incorrect as these are different modes of coping with provocation.

Wardwell, Bahnson and Caron (1963) examined whether there are personality variables that are specific to MI patients, by comparing post-MI and severely ill (non-CHD) controls. This method also attempted to control for the effects of illness on recalled pre-morbid hostility levels. Results showed that 3.5 times more MI cases than controls reported that they experience anger and anxiety while disagreeing with their boss. Six times more MI patients than controls said they keep anger to themselves and then blow up, but twice more controls than MI cases confirmed that people are cynical and selfish. In a subsample of 24 pairs matched on age, professional status, religion and ethnic background, MI cases scored significantly higher than controls on tension-reaction to anger. Thus, anger-suppression and argumentativeness (but not suspiciousness) may be specific characteristics of MI patients. However, the reliability and validity of these psychological measures were unknown, the

investigators performed too many and inadequate statistical tests, and they where not blind to subjects' group status.

Miller (1965) compared MI patients with healthy controls, matched on age, sex, education and IQ, on three hostility measures (Hostility Outward, Hostility Inward, and Ambivalent Hostility derived from a three-part interview). Age interacted with group status, with MI patients scoring significantly higher on Hostility Outward and Ambivalent Hostility than controls, but only among subjects younger than 55 years. However, MI cases scored higher than controls on Hostility Inward, across ages. Since these measures reflected mainly self-criticalness and worthlessness, Miller interpreted these results as reflecting the consequence of MI (e.g., reduced self-esteem).

One rarely cited study (Jenkins, 1966) compared the components of the TABP among men who had silent MI with non-CHD controls matched on age and occupation status, and non-CHD controls additionally matched on global TABP. Silent MI cases reported significantly higher levels of manifest hostility, but not higher scores on other Type-A components, than the first control group. However, lipid levels, which were related to hostility and were higher among silent MI cases, were not controlled for. Despite this limitation, this study provided early empirical evidence that hostility may be the only toxic TABP component in relation to CHD.

Bengtsson, Hallstrom and Tibblin (1973) assesse ost-MI Swedish women. Among 11 traits derived from the Cesarec-Marke's Personality

Schedule (CMPS; Cesarec & Marke, 1968), MI cases scored significantly higher on aggression alone compared with controls. Of five derived factors, MI cases scored higher than controls only on Neurotic Self-Assertiveness, which included aggression. However, the psychometric properties of these traits were unknown and analyses did not control for other risk factors. But, this study does suggest that behavioral hostility can distinguish Swedish women with and without CHD.

Theorell (1973) assessed post-MI survivors, who hadn't had significant illnesses two years prior to MI. While a significantly higher percentage of MI cases reported hostile feelings when slowed down by a slow car compared with controls, the groups did not differ on time-urgency. However, the reliability of the assessments was unknown, and traditional risk factors were not considered.

Croog, Koslowski and Levine (1976) compared post-MI patients with their wives, as a control group. Subjects did not differ from "controls" on questions regarding "Gets angry easily" and "Critical of others". However, the reliability of their assessment is questionable, spouses of coronary patients do not constitute an independent valid control group, and traditional risk factors were not considered. Thus, similarities on psychological factors related to marriage may have contributed to the lack of difference in hostility.

Theorell, DeFaire, Schalling, Adamson and Askevold (1979) compared twins whose members had mild or severe CHD on the Buss Aggression Inventory (Buss, 1961). Severe and mild CHD partners did not differ on any

hostility subscale. However, since CHD had been chronic, hostility levels may have been altered due to the duration of the disease. It was also unclear how mild/severe CHD was determined, and since all twins grew up together, genetic and environmental similarities may have masked potential differences in hostility between mild and severe CHD partners.

Dembroski, MacDougall and Lushene (1979) found that post-MI patients scored significantly higher on SI-derived hostility (i.e., stylistic and content-based) than matched (non-CHD) ill controls. The hostility difference remained even when comparing only Type-A cases and controls. Thus, the global TABP *may not be sufficient for distinguishing CHD from other illnesses, while hostility* may be a specific characteristic of CHD patients.

van Dijl (1982) used a 9-item factor-analyzed aggressiveness/hostility scale, and found in three case-control samples that MI males scored significantly higher on aggression/hostility than age- and education-matched healthy male controls. However, information on the psychometric properties of their scale was insufficient, and levels of other risk factors (e.g., smoking, BP, cholesterol) were not controlled for. Despite these limitations, replication of their findings in three samples, a rare design-feature in behavioral medicine, made the results more compelling.

Dembroski et al. (1985) also found that SI-derived levels of PH and Anger-In were positively and significantly related to number of previous clinically documented MI events ($r = .30$, $r = .23$, respectively). These effects were

significant even after controlling for significant risk factors (age and smoking), and were based on psychometrically sound measures of hostility.

A Canadian study (Wielgosz et al., 1988) found that males with MI scored significantly higher on SI-derived hostility, and male and female MI cases scored significantly higher on suppressed hostility than controls In a multivariate analysis, controlling for traditional risk factors, only suppressed hostility remained a significant discriminator between cases and controls However, an average of seven months had passed since the MI, and this may have affected recall of hostile reactions prior to MI

A Swedish study (Liechtenstein, Pedersen, Plomin, De Faire & McClearn, 1989) found that the Cynicism and Paranoid alienation subscales of the Ho scale were related to self-reported CHD in women, but not in men In both genders, lack of assertiveness was unrelated to self-reported CHD In a multivariate analysis, hostility was significantly related to CHD across genders The effect-size of the hostility-CHD relation was small since hostility, time-pressure and Neuroticism together accounted for only 2.8% of the variance in CHD Since a twin sample was employed, within-twin correlations on hostility scores (Carmelli et al., 1988), may have reduced the associations between hostility and CHD when one twin member was healthy Additionally, CHD was assessed with self-reports, and included AP. Finally, risk factors (e.g., smoking, BP) were not considered.

Fontana et al. (1989) attempted to reduce the effects of CHD symptoms

in cross-sectional studies on hostility levels by excluding cases with current symptoms or those who were on beta-blockers. Significantly more CHD subjects scored high on Cynical Mistrust than controls. However, risk factors other than age and SES were not considered, and the CHD group was not homogeneous. Despite these limitations, this study suggests that cynical hostility may be a risk factor for CHD.

Mendes de Leon (1992) found that levels of Anger-Out were significantly higher among MI cases than non-CHD ill controls. This remained intact when excluding subjects with previous MIs. Finally, Anger-Out significantly predicted subjects' group status, after controlling for age, marital status, education, smoking, hypertension and disease chronicity, with and without excluding previous MI cases. Anger-In was not associated with MI-presence in any analysis. Although the control group did not consist of a homogeneous group of patients, this study suggests that antagonistic hostility is a risk factor for MI and not for other diseases (mainly orthopedic).

Meesters and Smulders (1994) tested a Dutch sample of MI cases and randomly selected non-CHD neighborhood male controls. Cases did not differ significantly from controls on Ho scores. However, following previous studies (e.g., Siegman et al., 1987), Ho scores were significantly higher among MI patients than controls only for men below age 50, and this difference was independent of the effects of smoking and hypertension. Their findings corroborate other studies (i.e., Dembroski et al., 1989; Siegman et al., 1987) to

suggest that the relation between hostility and CHD is age dependent. The finding that hostility's effects on CHD attenuate with age is in line with the attenuation of effects of BP, smoking and cholesterol on CHD (Kannel et al., 1986), and this may be attributed to survival effects. Biologically vulnerable high-hostile subjects may not survive to be included in older samples, and those who do survive may be biologically hardier. Thus, older samples may include high-hostile but biologically hardy subjects who are at lower risk of death than non-hostile non-hardy subjects. This may explain the absence of a relation between hostility (or other risk factors) and CHD in older samples (Williams et al., 1988).

Finally, while most studies examined the effects of trait hostility/anger on CHD, an original study (Mittleman et al., 1995) tested the effects of exposure to anger-episodes as a state and onset of MI. Four days after MI, patients were asked about their anger-episodes and state-anger two hours prior to the MI, as well as those occurring 26 hours prior to MI and their annual anger-episodes The 26-hour and annual episodes served as self-controls. Subjects were considered "exposed" if they reported being at least very angry on an Onset of Anger Scale. This method partially controls for self-reported biases. The relative risk of having a MI was doubled for subjects experiencing an anger episode two hours prior to the MI. Thirty six patients reported an anger-episode only two hours prior to the MI compared with nine patients who reported episodes only 26 hours before the MI. During the two hours prior to MI, state-

anger was significantly higher than 26 hours prior to the MI. These results interacted with gender and use of aspirin: Males had a lower risk of MI than females, and aspirin users had a lower risk for MI than non-users, when experiencing an anger-episode two hours prior to MI. As expected (Krantz et al., 1982), taking beta blockers tended to reduce the risk as well. Mittleman et al. (1995) suggested that aspirin may reduce platelet aggregation associated with intense emotions. However, several patients were interviewed two weeks post-MI, possibly eliciting serious memory biases, and patients' own "theory" on the relation between anger and MI might have increased the reports of anger-episodes two hours prior to the MI. Finally, the Onset Anger Scale confounded hostile affect with behavior. Despite these limitations, this study suggests that transient hostile affect (anger) and/or behavioral hostile states associated with environmental provocations are among the triggers of a MI The interesting concept of psychological triggers of MI should be explored further as well as the relation between the predisposition to experience transient hostile states and an exaggerated physiological arousal in daily provocations which may precipitate MI.

In summary, 87% of cross-sectional studies reviewed here showed a positive association between hostility and CHD. Some studies attempted to show this relation without the effects of chronic CHD on hostility levels by excluding cases with previous MI (e.g., Fontana et al., 1989; Mendes de Leon, 1992). In addition, some studies showed that hostility is associated specifically

with CHD but not with other illnesses (e.g., Dembroski et al., 1979, Mendes de Leon, 1992). However, the main limitation of cross-sectional studies is that they are retrospective Even excluding cases with previous MI cannot remove the possibility that hostility results from current CHD status. In addition, recall biases ("how hostile or angry was I **before** the MI"; e.g., Mittleman et al., 1995) and biases related to "search for meaning" (subjects may overestimate their levels on a risk factor as a reflection of cause-attribution to that factor) limit the internal validity of cross-sectional studies (Meesters & Smulders, 1994) The age-dependent relation between hostility and CHD in cross-sectional studies may also reflect a bias resulting from younger MI victims being more emotionally affected and angered by the new adjustments and limitations that follow a MI than older victims (Meesters & Smulders, 1994). Positive findings from prospective studies on initially healthy individuals help to infer causality, and provide more compelling support to the hostility-CHD link. These studies will be reviewed next.

Table 4

Cross-sectional studies linking hostility with CHD

Authors	Subject characteristics	Hostility assessment	Outcome	Results	Methodology
Bengtsson et al. (1973)	42 MIs & 68 age-matched healthy Swedish women	Aggression (CMPS)	MI-presence	+	Medium
Croog et al (1976)	283 MIs & their wives	Psychiatric interview Anger & Criticalness	MI presence	-	Poor
Dembroski et al. (1979)	31 MIs & 33 matched non-CHD males	SI-derived hostile style & content	MI-presence	+	Good
Dembroski et al (1985)	131 CAD patients 98 men, 33 women	SI derived PH & Anger-In	# of previous MIs	+	Good
Jenkins, (1966)	25 silent MIs, 2 x 25 matched male controls	SI derived hostility	Silent MI	+	Good

Authors	Subject characteristics	Hostility assessment	Outcome	Results	Methodology
Fontana et al. (1989)	23 MIs & CAD, 41 non-CHD males	Cynical Mistrust factor (Ho scale)	CHD-presence	+	Medium
Liechtenstein et al. (1989).	1881 Swedish subjects, 1104 females, 777 men	Cynicism & Paranoia from the Ho scale	CHD-presence (MI & AP)	+	Poor
Meesters et at. (1994).	81 MIs & 168 Dutch controls	Ho scale	MI-presence	+	Good
Mendes de Leon (1992).	31 MIs & 26 orthopedic males	Anger-In/Out (AX)	MI-presence	+	Good
Miller (1965).	43 MIs & 34 healthy matched controls	Interview-based: Outward/Inward Hostility	MI-presence	+	Medium
Milles et al. (1954).	46 MIs & 49 healthy controls, all men	Psychiatric interview & Suspicion (16 P F)	MI Presence	+	Poor

Authors	Subject characteristics	Hostility assessment	Outcome	Results	Methodology
Mittleman et al. (1995)	1623 post MIs, 1122 males, 501 females	Onset Anger Scale & State-anger	MI	+	Good
Theorell, (1973)	62 MIs & 109 non-CHD matched Swedish males	Hostility when slowed by a car (self-report)	MI Presence	+	Medium
Theorell et al. (1979).	30 male MZ & DZ Swedish twins	Buss Aggression Inventory	Severe versus mild CHD	-	Poor
van Dijl (1982)	102, 98 & 63 MI & matched Dutch males	9-item factor-analyzed scale	MI-presence	+	Medium
Wardwell et al (1963)	32 MIs & 32 age matched controls with other illness	Interview & written responses about coping with anger	MI presence	+	Poor
Wielgosz et al (1988).	100 MIs & 100 healthy Canadians	SI-derived hostility & suppressed hostility	MI-presence	+	Good

Evidence for the prospective relation between hostility and CHD

As has been stated, prospective studies that follow initially healthy subjects rule out the effects of previous CHD on base-line hostility levels, thus increasing the validity of inferring that hostility may be a risk factor for CHD. Prospective designs allow one to examine the predictive validity of hostility in relation to CHD. The main limitation of studying the prospective risk factors of MI or sudden death is their low incidence-rates compared with other CHD outcomes (e.g., AP). This requires conducting large scale and/or long-term longitudinal studies in order to observe a sufficient number of CHD events that provide statistical power for demonstrating significant relations between hostility and MI or sudden death. Additionally, patients may develop UA during the follow-up period and may undergo preventative revascularization procedures (e.g., coronary artery by-pass graft surgery, angioplasty), and can this prevent them from having a MI. If these patients include hostile subjects, this may weaken the expected relations between hostility and subsequent infarctions or mortality (Helmers et al., 1993). Finally, as with cross-sectional studies, some prospective studies include AP in the outcome of CHD, in addition to MI and sudden death. Again, since not all AP cases are cardiac, this may reduce the validity of the outcome measure and affect the hostility-CHD relation (Costa, 1987). Table 5 presents the study characteristics and findings of prospective studies, and their chronological presentation follows.

Theorell, Lind and Floderus (1975) followed Swedish men for 12-15

months. Two self-report items asked subjects to rate their hostility when faced with a slow person, and when being held up in queues (the latter item is currently coded in the PH rating). Both items significantly predicted all-cause mortality, and hostility in queues significantly predicted cardiac deaths and non-fatal MIs, independent of the effects of age. However, no psychometric data were provided for the hostility measures, and the many statistical tests performed (276!) may have resulted in type-1 errors.

Matthews, Glass, Rosenman and Bortner (1977) wished to determine which components of the TABP were CHD-predictive in the prospective Western Collaborative Group Study (WCGS). The predictive validity of five SI-derived factors and their items was examined. While several TABP factors were not CHD-predictive (e.g., Past Achievements), two were (Competitive-Drive and Impatience). The most CHD-predictive items included in both factors were all related to hostility. Thus, Irritation at waiting in lines, PH, Anger-Out and frequent experience of anger were significantly higher in men who developed CHD than in age- and working place- matched controls. However, the reliability of the specific components was unclear, traditional risk factors were not considered and CHD included a few AP cases. Despite these limitations, this study showed that not all the components of the TABP are CHD-predictive (e.g., Past-Achievements), and that in the factors that were CHD-predictive, the toxic elements were hostility-related.

Haynes, Feinleib and Kannel (1980) examined men and women in the

Framingham Heart Study. Men reporting lower levels of Anger-Out were at significantly greater risk for developing CHD, independent of age, SBP, cholesterol, smoking, TABP and number of job-promotions, but only among white-collar men. Among working women (but not housewives), lower levels of Anger-Discuss were significantly associated with CHD, independent of traditional risk factors and TABP. Thus, the TABP does not account for the relation between suppressed hostility and CHD. However, many psychosocial predictors were included, and many were assessed with few items, which may limit their reliability. In addition, it was unclear whether all AP cases included in CHD were cardiac. This is one of the few prospective studies suggesting a positive relation between suppressed hostility and CHD.

Barefoot, Dahlstrom and Williams (1983) found that the total CHD incidence was significantly lower (.9 per 1000 person-years of follow-up) for physicians scoring at or below the median Ho score (13) compared to those scoring above the median (4.5 person-years per 1000). Among subjects who were alive at follow-up, physicians scoring above the median were nearly six times at greater risk for CHD than those scoring at or below the median. These threshold effects remained significant after controlling for presence of hypertension. Hostility also predicted all-cause mortality which included other causes of death (e.g., cancer), suggesting that hostility affects the ability to survive illnesses other than CHD. However, recall biases may have been present since physicians reported their own health during the follow-up years,

and the validity of these self-reports was not indicated. These results are relatively stronger than those obtained from other prospective studies that used the Ho scale (Miller et al., in press), and even a few misclassifications in CHD-status resulting from inaccurate self-reports may have yielded these relations.

Shekelle, Gale, Ostfeld and Paul (1983) found in the Western Electric Study a significant association between Ho scores and 10-year CHD incidence, with the highest rate occurring for men in the middle (3rd) quintile of Ho scores (i.e., a curvilinear relation). This remained significant after controlling for age, SBP, cholesterol, smoking and alcohol-consumption, but only when dichotomizing the scores (i.e., Ho $\leq$ 10 versus Ho > 10; Williams et al., 1980). Hostility univariately predicted 20-year mortality-incidence due to CHD, malignant neoplasms, all other causes, and total mortality, with the latter two remaining significant after controlling for traditional risk factors. Hostility predicted all-cause mortality in a linear manner. Shekelle et al. (1983) concluded that since hostility predicted death due to causes other than CHD, hostility may be related to a factor that has broad effects on survival such as social support. This will be discussed in Chapter 4.

Powell and Thoresen (1985) examined the relation between hostility and CHD-**progression** (recurrent non-fatal MI or cardiac death) in the Recurrent Coronary Prevention Project. Ten interview based measures related to hostility (e.g., Hostility and Anger-Out) univariately predicted recurrent CHD. Arousal while driving behind a slow car and interview-related emotional intensity

significantly predicted recurrent CHD after controlling for whether patients received Type-A treatment. Interview-related emotional intensity (which partly resembles SI-derived Hostile-Style; Dembroski & Costa, 1987) predicted recurrent CHD independent of the severity of the previous MI. This is one of the few studies that examined the relation between hostility and recurrent-CHD, and its results have important implications for secondary prevention of CHD.

A Swedish group (Hallstrom et al., 1986) found that Aggression was negatively related to women's ischemia as measured with ECG, with women in the lowest aggression quintile being more than 8 times at risk for ischemia than women in the highest aggression quintile. Similarly, (Neurotic) assertiveness was negatively related to MI. These effects were independent of age, social status, physical activity, obesity and triglycerides. Aggression and assertiveness were assessed with the CMPS (Cesarec & Marke, 1968). Thus, in Swedish women, low levels of aggression and low levels of assertiveness may be independent CHD risk factors. These findings contrast with those showing that expressed hostility or aggression is positively related to CHD in men (e.g., Dembroski et al., 1989) and a cross-sectional study in Swedish women (Bengtsson et al., 1973). The psychometric properties of the CMPS in the study by Hallstrom et al. (1986) were not indicated, and this may partly explain the discrepancies between the studies.

McCranie, Watkins, Brandsma and Sisson (1986) found no relation between Ho scores and CHD incidence, non-fatal CHD, mortality from other

causes or all-cause mortality among physicians. Traditional risk factors (e.g., hypertension, obesity, smoking) were not related to Ho scores. However, the hostility ratings may have been invalid since subjects completed the Ho scale during an evaluation for entering into medicine school. The substantially lower mean and median Ho scores compared with other studies with a positive hostility-CHD relation (Barefoot et al., 1983; Shekelle et al., 1983) suggest that social desirability may have reduced Ho scores. McCranie et al. (1986) did find that Ho scores were strongly correlated with MMPI K-scores indicative of defensiveness ($r = -.74$). Although the Ho by K interaction was not significant for CHD-incidence or all-cause mortality, the measure of defensiveness employed may be invalid, and controlling for its effects may have not fully removed the effects of defensiveness on Ho scores. Finally, the validity of self-diagnosing CHD by the physicians was unclear.

Julius, Harburg, Cottington and Johnson (1986) found that subjects who suppressed their anger or would not manifest it when provoked by their spouse were at 2.4 and 1.7 times the risk for dying, respectively, than those who did not suppress or would manifest anger. These effects were independent of age, sex, smoking, relative weight, bronchitis, education, CHD status at base-line and pulmonary functioning. Anger-suppression interacted significantly with SBP such that hypertensive anger-suppressors were five times at risk of dying than non-suppressor hypertensives. However, specifically CHD-related mortality was not tested due to small numbers of deaths. This study showed that the manner

in which men and women report they cope with anger when provoked is an independent and significant predictor of mortality. In addition, this study suggested that certain traditional risk factors (i.e., SBP) may interact with hostility-related measures in predicting death, as has been shown in a study with CAD patients (Barefoot et al., 1994).

Barefoot et al. (1987) found that a suspiciousness, jealousy and irritability scale (Factor L, derived from the 16 Personality Factor scale, 16 P.F; Cattel et al., 1970) significantly predicted death, independent of age, sex, functional health, cholesterol, smoking and the interaction of smoking by age. However, the vital status of nearly 6% of the sample was not known at follow-up. In addition, Factor L scores were obtained over four assessments during follow-up, and situational factors may have affected the scores' stability and the scale's predictive validity. Despite these limitations, this study extended previous studies to another measure of hostile attitudes, and supported the relation between hostility and mortality in an older age group.

Koskenvuo et al. (1988) found in a twin sample that high-hostile subjects were at significantly greater risk for all-cause naturally-occurring deaths (relative risk, RR = 2.88) and for cardiovascular deaths (RR = 2.72) compared with low-hostile men, after controlling for age. Among initially healthy subjects, hostility did not predict ischemic heart disease (IHD). Among subjects with initial hypertension and IHD, hostility predicted subsequent IHD after controlling for age, smoking, heavy drinking, obesity, snoring and dyspnea. However, the use

of a sample of twins may have reduced measurement variability and thus, reduced hostility's predictive power. In addition, the short follow-up period (three years) may have revealed few new ischemic cases, thus, reducing the power for detecting effects among initially healthy subjects. Finally, adding AP in the outcome may have included non-cardiac cases, possibly obscuring the hostility-IHD relation (Costa, 1987). Despite these limitations, this study suggested that hostility may be a risk factor for recurrent-CHD, confirming the findings of Powell and Thoresen (1985).

Leon, Finn, Murray and Bailey (1988) showed that Ho scores did not differentiate among subjects who developed a MI, hypertension and congestive heart failure, and all other men. These results did not change when controlling for traditional risk factors (i.e., cholesterol, DBP,SBP, smoking, age, height and weight). Finally, Ho scores did not differentiate between fatal and non-fatal CHD cases. A power analysis revealed adequate number of subjects for detecting Ho-CHD effects had they occurred as in previous studies. These findings may have resulted from subjects' relatively high initial age (Mean = 45) which may weaken the relation between hostility (and other risk factors) and CHD (Kannel et al., 1986; Williams et al., 1988). The low mean Ho in this sample (10) may reflect social desirability or a narrow range of scores, thus, statistically reducing the chances of finding significant Ho-CHD relations.

Hecker, Chesney, Black and Frautschi (1988) used a carefully developed measurement system for coding the SI and found that SI-derived hostility

differentiated between men who developed CHD and age- and work-place-matched controls. Hostility was the only predictor of CHD when all other Type-A components were considered together, and hostility predicted CHD independent of global TABP and of major risk factors. Unlike other findings (e.g., Siegman et al., 1987), hostility did not interact with age in predicting CHD. However, CHD diagnosis included AP as well. Hecker et al. (1988) concluded that hostility plays "a leading role" in TABP in predicting CHD.

One of the theoretically and methodologically most compelling studies that viewed hostility as a multidimensional construct was that of Dembroski, MacDougall, Costa and Grandits (1989). PH was divided into Hostile Content and Intensity (frequency and degree/emphasis of annoyance in daily provocations, respectively) and Hostile-Style (antagonistic behavior directed at the interviewer). Dichotomized PH and Hostile-Style differentiated between males who did and did not develop CHD in the Multiple Risk Factor Intervention Trial (MRFIT; RR = 1.7 and 1.5, respectively). After controlling for major risk factors, only PH remained a significant CHD predictor (RR = 1.5). The global TABP and its other components did not predict CHD. Unlike Hecker et al. (1988), hostility interacted with age; Only among men at or below age 47 (the sample's median age), both PH and Hostile-Style significantly predicted CHD, with the latter relation remaining significant after controlling for traditional risk factors (RR = 1.4). However, this study's generalizeability may be limited since MRFIT subjects were initially elevated on major risk factors. On the other hand,

finding significant hostility-CHD relations despite the potential restricted range in disease-status that might occur in high-risk subjects (disease-spectrum bias; Miller et al., in press), makes these findings more impressive. Thus, this study suggests that hostility, particularly its behavioral component of interpersonal Hostile-Style or Antagonism, is the only significant and independent CHD risk factor in the TABP among high-risk men (Dembroski et al., 1989).

Another important study examined the relation between the theoretically-derived components of the Ho scale and 29-year all-cause mortality (Barefoot et al., 1989). The full Ho scale showed a significant and linear relation with all-cause mortality (of which half were related to cardiac diseases), and this was independent of age. The empirically-derived Ho subscales of Cynicism and Paranoid Alienation (Costa et al., 1986) did not predict death better than the full Ho scale. However, the theoretically-derived subscales of Cynicism, Hostile Affect and Aggressive-Responding separately and together (Barefoot's refined Ho) significantly predicted mortality. Barefoot's refined Ho had a 50% and 29% **larger** X^2 than that of the full Ho scale and age, respectively. Subjects scoring 1 SD above the mean on Barefoot's refined Ho were 5.54 times at risk of dying from all causes than those scoring 1 SD below the mean. Theoretically, these findings were coherent, since Cynicism, Hostile Affect and Aggressive-Responding mainly assess Antagonistic hostility, and reflect the three aspects of psychological experience (thought, feeling and behavior, respectively; Barefoot et al., 1989). A recent meta-analysis suggests that the Ho scale (and

Barefoot's Ho) may be a particularly good predictor of all-cause mortality (Miller et al., in press).

These three subscales may be the most toxic components of the Ho scale. Negative findings between the full Ho scale and CHD (e.g , Leon et al., 1988; McCranie et al., 1986) may have resulted from subjects scoring high on the Ho items that assess Neuroticism, a factor unrelated to objective health (Costa & McCrae, 1987).

Hearn, Murray and Luepker (1989) failed to show an association between full Ho scores and non-fatal CHD. CHD-related mortality, mortality from other causes or total mortality in a more representative sample than previous studies. The results did not change after controlling for age, hypertension and smoking. The Ho scale did not differentiate between cases who died from CHD or any cause and controls randomly selected from the remaining sample. These results did not change using Ho quantiles or cut-off points previously employed (Barefoot et al., 1983; Shekelle et al , 1983; Williams et al., 1980) Finally, Barefoot's refined Ho did not predict survival Although Hearn et al. (1989) conducted several carefully designed tests, their cases included patients without definite CHD, and this may have weakened the Ho-CHD relation. Hearn et al. (1989) added that subjects' early age at intake (19 years) may have not considered developmental changes that may alter the stability and predictive validity of Ho scores. Finally, Hearn et al. (1989) added that they and others may have been testing the predictive validity of the Ho

scale, not of the hostility **construct** (unlike SI-derived measures).

Carmelli et al. (1991) found that SI-derived behavioral hostility (as rated by Hecker et al., 1988) significantly predicted 27-year CHD-mortality. Hostile subjects above age 48 showed the poorest survival. Hostility significantly predicted CHD and all-cause mortality only among subjects older than age 48, while hostility predicted mortality from cancer only among subjects younger than age 49. These results were independent of age, SBP, cholesterol, body mass index, education, smoking and TABP (the latter did not predict any outcome). These results do not support those of Dembroski et al. (1989) who found that hostility predicted CHD only among men below age 48. The study by Carmelli et al. (1991) differs from Dembroski et al. (1989) in outcomes (CHD-mortality versus fatal and non-fatal CHD, respectively), in sampling procedures (total WCGS sample versus randomly selected cases and controls from the MRFIT, respectively), and in the assessment of hostility. However, both studies clearly demonstrated that SI-derived hostility is an independent CHD-predictor.

Almada et al. (1991) followed men from the Western Electric Study for 25 years, and assessed Neuroticism and Cynicism from the MMPI. Cynicism was strongly correlated with the Ho scale (.93). Cynicism significantly predicted mortality from CHD and from all causes, even after controlling for age, SBP, smoking, cholesterol, alcohol-consumption and Neuroticism. Cynicism also predicted death from cancer, particularly lung cancer, after controlling for age alone. Neuroticism significantly predicted death from other causes and from all

causes, after controlling for traditional risk factors, however, Cynicism was responsible for these effects However, Almada et al (1991) pointed out that errors in self-reported alcohol and cigarette use, parameters that predicted several outcomes, may have been errors related to Cynicism (e g., cynical people fearing investigators will misuse information about them). They suggested that the effects of these two risk factors may have been incompletely controlled for Thus, investigators may consider assessing traditional risk-factors via more objective methods, particularly when testing the additional effects of cynical hostility Finally, the investigators added that controlling for base-line levels of traditional risk factors does not consider changes during follow-up (e.g., smoking resumption) typically not assessed and possibly related to hostility

One of the few prospective studies conducted in women was done with subjects from the Framingham Study (Eaker, Pinsky & Castelli, 1992) In a 20-year follow-up Anger-In, Anger-Out and Anger-Discuss (talking about anger with a friend/relative) did not predict incidence of MI or coronary death. The main psychosocial parameters predictive of CHD were tension and lack of vacations. Eaker et al. (1992) termed these variables "coronary-prone situation" rather than coronary-prone personality. However, anger items were based on one-item self-report questions, and their reliability is questionable.

Houston, Chesney, Black, Cates and Hecker (1992) divided participants according to their manifested patterns or clusters of SI-derived components in

the TABP. Two behavioral clusters, one including hostility and explosive speech, the other including competitiveness-dominance, were positively related to CHD incidence. Two other clusters, lack of hostility and explosive speech, dejection, were negatively related to CHD. There results remained significant after controlling for DBP, smoking and cholesterol, with the hostility and explosiveness cluster and the lack of hostility clusters showing trends. Finally, after adding hostility as a covariate, the competitiveness-dominance cluster still predicted CHD. This study suggests that in addition to hostility, competitiveness-dominance, which reflects verbal competitiveness, is a CHD-predictive component of the TABP. However, CHD included AP cases, some of whom may have not had CHD. In addition, the contribution of hostility alone was unclear, since it was assessed within behavioral clusters. This study suggests examining behavioral profiles rather than discrete parameters, and that a behavioral profile characterized mainly by hostility, is CHD-predictive.

Maruta et al. (1993) found that the Ho scale was only univariately predictive of CHD events, CHD-related mortality and all-cause mortality. However, after controlling for the effects of age, sex, hypertension and relative weight, Ho scores tended to predict all-cause mortality alone. Thus, the effects of hostility (as assessed with the Ho scale) on CHD may not be independent of traditional risk factors. However, gender differences were observed in hostility, and are commonly found in CHD as well (Jenkins, 1988). Thus, the investigators should have examined the Ho-CHD relation in males and females

separately. Additionally, CHD events included AF cases. Finally, the vital status of 57% of the subjects was unclear.

Barefoot (1993) criticized this study in the following ways: 1) The interaction of Age x Hostility should have been tested in light of previous studies (e.g., Dembroski et al., 1989); 2) Since subjects were actually self-referred patients, their initial health status should have been controlled for. Finally, Barefoot (1993) added that although the Ho scale may not be the most valid hostility measure, previous positive Ho-CHD findings "attest to the robustness of the phenomenon".

Finally, the most recent and perhaps the most methodologically stringent study was conducted in Denmark (Barefoot, Larsen, von der Leith & Schroll, in press). Hostility was assessed with an Abbreviated Cook Medley (ACM) scale which included items from Barefoot's refined Ho scale and items from the Hostile Attribution subscale (Barefoot et al., 1989). The ACM did not predict MI after controlling for and sex, but did after adding controls for SBP, triglycerides, smoking, and physical activity at work and during leisure (RR = 1.53), and after removing subjects with ischemia at base-line (RR = 1.56). The ACM significantly predicted all-cause mortality after controlling for age, sex, SBP, triglycerides, smoking, pulmonary functioning and base-line ischemia (RR = 1.36). All relations were linear, and did not differ for men and women Finally, the Cynicism subscale tended to predict MI and mortality, and Hostile Affect and Hostile Attributions significantly predicted both outcomes. Unlike

Barefoot et al. (1989), the Aggressive-Responding subscale, which reflects antagonistic but not neurotic hostility, did not predict either outcome. The investigators hypothesized that in a later age and in more reserved cultures cynicism and anger play more important roles in CHD, while in younger American samples, behavioral hostility may be relatively more toxic (Barefoot et al., 1989; Dembroski et al., 1989). This study extended previous prospective associations between self-reported hostility and CHD to males and females, to older age groups in a European culture, and controlled for several risk factors.

In summary, 82% of prospective studies showed evidence for the hostility-CHD link, using different hostility measures, different CHD end-points and subjects from different cultures. All three negative findings were conducted with the full Ho scale, which includes items that assess only Neuroticism (Barefoot et al., 1989). This may reduce the predictive validity of the full Ho scale (Costa & McRae, 1987; Watson & Pennebaker, 1989). Since these studies followed initially healthy subjects, this sample of positive findings support the notion that hostility is a risk factor of CHD, rather than its consequence (Smith, 1992).

Table 5

Prospective studies linking hostility with CHD and mortality

Authors	Subject characteristics	Hostility assessment	Outcome	Results	Metnodology
Almada et al (1991)	1871 middle aged men	MMPI derived Cynicism	Death from CHD, CVD, cancer & all causes over 25 years	+	Good
Barefoot et at (1983)	255 male physicians	Ho scale	CHD (MI & UA) & death over 25 years	+	Good
Barefoot et al (1987)	500 older subjects, 260 men, 240 women	Suspicion Factor (16 P F)	Deaths from all causes over 15 years	+	Good
Barefoot et al (1989)	118 lawyers	Barefoot's refined Ho	Death from all causes over 29 years	+	Good
Barefoot et al (in press)	409 men, 321 women all Danish	Similar to Barefoot s refined Ho	MI & death from all causes over 27 years	+	Good
Carmelli et al (1991)	3058 men	SI derived behavioral hostility	Death from CHD, cancer & all causes over 27 years	+	Good

Authors	Subject characteristics	Hostility assessment	Outcome	Results	Methodology
Dembroski et al. (1989).	192 CHD versus 384 matched healthy men	PH, Style, Content & Intensity (SI)	MI & coronary death over 7.1 years	+	Good
Eaker et al. (1992).	749 women	Anger-In/Out & Discuss	MI & coronary death over 20 years	-	Good
Hearn et al. (1989).	1313 men	Ho & Barefoot's refined Ho	CHD, fatal CHD & death from all causes over 33 years	-	Good
Hallstrom et al. (1986).	795 age-stratified women	Aggression & Neurotic Assertiveness (CMPS)	MI & coronary ECG	+	Good
Haynes et al. (1980).	1674 subjects, 725 men, 949 women,	Anger-In/Out & Discuss.	Total CHD, MI & AP over 8 years	+	Good
Hecker et al. (1988).	250 CHD & 500 matched control men	SI-derived hostility	CHD (MI AP & coronary death over 8.5 years	+	Good

Authors	Subject characteristics	Hostility assessment	Outcome	Results	Methodology
Houston et al (1992)	250 CHD & 500 matched control men	SI-derived behavioral clusters	CHD (MI, AP & coronary death) over 8 5 years	+	Good
Julius et al (1986)	696 subjects 324 men 372 women	Anger In/Out with spouse	Death from all causes over 12 years	+	Good
Koskenvuo et al (1988)	3750 Finnish men	Irritability, anger argumentativeness	IHD (MI & AP), all deaths over 3 years	+	Medium
Leon et al (1988)	280 men randomly selected from a stratified sample	Ho scale	MI, CHF or hypertension over 30 years		Good
Maruta et al (1993)	620 patients from a private clinic 254 men 366 women	Ho scale	CHD events fatal CHD & death from all causes over 20 years	+	Medium
McCranie et al (1986)	478 physicians 97% males	Ho scale during admission exams	CHD, fatal CHD & all deaths over 25 years		Medium

Authors	Subject characteristics	Hostility assessment	Outcome	Results	Methodology
Matthews et al (1977).	62 MIs & 124 matched controls	SI-derived PH & Irritation in lines	MI, silent MI & AP, over 4.5 years	+	Medium
Powell et al (1985)	44 CHD & 74 male controls	SI-derived hostility, Anger-Out	Recurrent MIs or CHD death over 2 years	+	Good
Shekelle et al (1983)	1877 men	Ho scale	MI, fatal CHD & all deaths over 10 & 20 years	+	Good
Theorell et al (1975).	6579 Swedish males	2 Self-report items	MI & CHD deaths over 12-15 months	+	Medium

Integration of studies

What do these studies tell us? In their comprehensive review, Miller et al. (in press) concluded that hostility is an independent CHD risk factor The studies reviewed here provide compelling empirical evidence for the relation between hostility and CHD. Examining studies across all end-points (which includes a few studies twice), measures and methodological ratings, 55 out of 64 or 86% showed a positive relation between hostility and CHD. Of the nine negative studies, seven assessed hostility with self-report measures, and only one used the SI. Similarly, 87% of studies rated "good" on methodology (39 studies) found a positive hostility-CHD relation. The study by Ironson et al. (1992) found that experiences and reactions of anger and hostility may be **causally** and more strongly related to myocardial dysfunction than are general stressful situations and stress-reactions. Powell and Thoresen (1985), Koskenvuo et al. (1988) and Julkunen et al. (1994) suggest that hostility is predictive of CHD-progression as well. Barefoot et al. (in press) suggest that hostility predicts CHD in males and females, in older subjects and in a non-American culture. Finally, some studies (e.g., Dembroski et al., 1979, Mendes De Leon, 1992) suggest that hostility is a risk factor specifically for CHD and not for other (e.g., orthopedic) illnesses. Thus, it is safe to conclude that hostility is a reliable risk factor for CHD, and in several cases this holds across genders, age groups, and cultures (American, Canadian, Danish, Dutch, Finnish, Spanish and Swedish), independent of traditional risk factors (Miller et

al., in press, Smith, 1992).

Some studies began to identify which components of the multidimensional construct of hostility are more "coronary prone" than others. Haney et al. (1992), Dembroski et al. (1989) and Siegman et al. (1987) suggest that behavioral interpersonal hostility (SI-derived Hostile-Style, Indirect Hostility or the BDHI-Expressed hostility factor), which reflects the personality dimension of Antagonism, may be the most toxic component of hostility in relation to CHD Studies using Barefoot's refined Ho (Barefoot et al. 1989, Burg et al., 1993; Helmers et al., 1993) or the Abbreviated Cook & Medley scale (Barefoot et al., in press) have shown that, in addition to Aggressive Responding, the Ho subscales of Cynicism, Hostile Attribution and Hostile Affect more powerfully predict CHD than the remaining Ho subscales. All these toxic hostility measures assess Antagonism (e.g., Hostile-Style), and are only mildly or unrelated to Neuroticism (e.g., Barefoot's Ho; Barefoot et al., 1989; Costa et al., 1989; Dembroski & Costa, 1987). This pattern of correlations with Antagonism and Neuroticism is what makes these measures more CHD-predictive than others (Costa et al., 1989; Watson & Pennebaker, 1989). Future epidemiological studies may wish to include both self-reported (e.g., refined Ho) and observed (e g. PH) measures to assess hostility in a comprehensive manner (Costa et al., 1989).

However, the relations between hostility facets and Antagonism or Neuroticism should be considered with caution since these personality variables

are broader constructs than their corresponding facets in hostility (Costa et al., 1989). Hostile affect or anger and hostile cognition or cynicism which are CHD-predictive, reflect Antagonism and only certain aspects of Neuroticism (Almada et al., 1991; Barefoot et al., 1989; Barefoot et al., in press; Costa et al., 1989) Thus, the three components of hostility, cognitive (cynical mistrust and hostile attributions), affective (anger) and behavioral (interpersonal antagonism), may **all** be CHD-predictive.

While some studies suggest that anger-suppression or Anger-In may be unhealthy (e.g., Dembroski et al., 1985; Julius et al , 1986), others suggest that anger-expression or Anger-Out is the unhealthy mode of coping with hostile thoughts or feelings (e.g., Matthews et al., 1977; Siegman et al., 1987). Siegman (1993) reviewed several studies showing that, while the **expression** of anger/hostility is positively related to cardiovascular reactivity, CAD and CHD, the **experience** of anger/hostility is unrelated or negatively related to these outcomes (e.g., Siegman et al., 1987).

One solution to the conflicting results was proposed by Dembroski and Costa (1987) and is related to the problem of causes versus consequences of diseases. Since ratings of PH are heavily affected by Anger-Out that is reported and/or expressed during the SI, the two are expected to be positively correlated. In contrast, these investigators showed that, while PH and Anger-In were negatively correlated in healthy people ($r = -.48$), they showed a tendency to be positively correlated in MI patients ($r = .13$, ns). Dembroski and Costa

(1987) suggested that for CHD patients, symptoms may become associated with Anger-Out. Following recommendations from physicians, and attempting to reduce their symptoms, CHD patients who were initially high on Anger-Out prior to their illness become high on Anger-In. Thus, Anger-In may be a **consequence** of CHD rather than its precursor. Accordingly, expressed hostility or Anger-Out assessed in initially healthy people is expected to predict CHD (e.g., Hostile Style; Dembroski et al., 1989), but Anger-In is expected to correlate with CAD and CHD in cross-sectional studies with subjects that already have CHD (e.g., Dembroski et al., 1985). Future studies should examine whether initially healthy people who are high on Anger-Out become high on Anger-In when ill. In addition, suppressed anger has been implicated in the development of immune-system based diseases (e.g., cancer; Jensen, 1987). Thus, part of the relations found between Anger-In and all-cause mortality (e.g., Julius et al., 1986) may include cancer-related rather than cardiac-related deaths (Siegman, 1993).

Several hostility measures may assess both Anger-In and Anger-Out (e.g., the complete BDHI scale). Anger-Out is more strongly related to Antagonism and expressed hostility, and Anger-In is mainly related to Neuroticism and experienced hostility (Musante et al., 1989). Thus, hostility measures that measure both modes of anger-expression assess Antagonism and Neuroticism, and this may weaken their relationship with CHD (Watson & Pennebaker, 1989). Several solutions for this problem exist: a) To separate

hostility measures into neurotic and antagonistic components (e.g., the Experienced versus the Expressed factors in the BDHI, respectively; Siegman et al., 1987); b) To control for Neuroticism before examining the effects of hostility on CHD (Almada et al., 1991, Siegman, 1993)

Hostility is related to mortality from cancer and all-cause mortality (e.g., Almada et al., 1991, Carmelli et al., 1991, Shekelle et al., 1983). This raises the important but neglected question in behavioral medicine of specificity versus generality of the effects of risk factors. Hostility, particularly that assessed with the full Ho scale, may have broader effects on health via other psychological parameters (e g., social support, stressful events), which predict other diseases (e.g., cancer; Jensen, 1987). This will be discussed in the next chapter

Several studies showed that hostility plays a stronger role in CHD in younger than in older adults (e g., Dembroski et al , 1989, Siegman et al , 1987), while others found hostility to predict CHD in older samples as well (Barefoot et al , in press) Williams et al (1988) has argued that some of those subjects who are at risk (e g., elevated hostility) are excluded from follow-up studies on initially healthy people as they may die or already have CHD at screening. Initially older high-hostile but healthy subjects, who remain in the sample, may be biologically hardier than their at-risk hostile counterparts who were excluded, since the former survived the "natural selection process", and thus, may not be at further risk for CHD These surviving biologically hardy but hostile persons may even be at less risk for CHD than less hardy people who

are not hostile, who may develop CHD later. These factors together may weaken prospective relations between hostility (or other risk factors) and CHD in initially older subjects. The potential reduced CHD-risk of high-hostile and hardy older subjects may cancel the potential increased CHD-risk of high-hostile young subjects. Thus, age should be considered as an important moderating variable in the relation between hostility and CHD.

Finally, these findings provide the empirical basis and guidelines for psychological interventions in CHD (Dembroski et al., 1989; Miller et al., in press). Based on the studies reviewed above, one may conclude that psychological interventions for reducing the risk of CHD or its progression should focus on modifying the significant and independent CHD-risk factor of hostility, and on its behavioral antagonistic component (Dembroski et al., 1989; Siegman et al., 1987). Attempting to reduce levels of cynicism and anger, and not pure neurotic hostility facets (e.g., Social Avoidance from the full Ho scale) is of therapeutic importance as well, since these hostility facets are conceptually and empirically related to antagonistic hostility (Barefoot et al., 1989; Chesney, 1985) and they are CHD-predictive as well (e.g., Almada et al., 1991; Barefoot et al., in press). Thus, this review forms the empirical basis for the development of a hostility-reduction treatment, one which is outlined in Chapter Five, and is the focus of this thesis.

CHAPTER FOUR

MODELS LINKING HOSTILITY WITH CHD

Introduction

This chapter reviews the models that suggest mechanisms by which hostility may cause CHD. After each model is presented, its strengths and weaknesses are discussed, followed by studies that have examined each model. The chapter will end with an attempt to integrate the different models in a meaningful manner, and relate them to hostility-modification as well.

Smith (1992; 1994) reviewed five models that attempt to explain the link between hostility and CHD: The psychophysiological reactivity model, the psychosocial vulnerability model, the transactional model, the health behavior model, and the constitutional vulnerability model. Each model suggests different paths for associating hostility with CHD. Some models link hostility explicitly with CHD (e.g., the psychophysiological reactivity model) and some models link hostility with CHD and disease in general (e.g., the psychosocial vulnerability model). Thus, the models differ with respect to their hypothesized mediating processes and their outcome specificity.

The psychophysiological reactivity model

This is the most prominent and widely tested model of the relation between hostility and CHD. It takes a reductionist approach and assumes that hostility, a psychological parameter, should be reduced to physiological processes (e.g., increased blood-pressure or BP, enhanced epinephrine) to be

related to CHD, a physical outcome. The basic premise of the psychophysiological reactivity model is that: a) Hostility is related to several indices of physiological hyper-reactivity; and b) Chronic repetition of hyper-reactivity may develop into or precipitate cardiac events (Smith, 1992; Williams, Barefoot & Shekelle, 1985). Speculating about the connection between the components of hostility, Chesney (1985) suggested that hostile cognitions (e.g., cynicism) mediate the effects of stressful events on behavioral (antagonism), emotional (anger), cognitive (hostile attributions) and physiological (increased BP) reactions. Although not always explicitly mentioned, this cognitive mediation is implied in the psychophysiological reactivity model.

Dembroski (1978) used a systems theory approach which allows one to explore the markers of several subsystems in a hypothesized model linking coronary-prone behavior with CHD. According to this model: a) Psychological risk factors (e.g., TABP, hostility) may affect or be affected by traditional risk-factors (e.g., cholesterol) and lead to CHD; b) Certain appraisals (e.g., cynicism) may follow environmental stimuli (e.g., provocations), which may affect or be affected by psychological factors (e.g., antagonistic behavior), and/or physiological factors (e.g., enhanced BP) and lead to CHD. The fact that this model specifies multicomponents with bidirectional relations makes it flexible and open to empirical validation.

One of the first biological links between hostility and CHD was established by Williams et al. (1985) who proposed two possible pathways by

which hostility may lead to CHD: a) Hostility may reflect higher levels of vigilance for environmental hazards, which is associated with higher secretion of testosterone, which may enhance atherogenesis; b) Hostility is associated with more frequent and higher levels of anger (hostile affect), which has been associated with higher levels of cortisol. Cortisol enhances the cardiovascular effects of norepinephrine, i.e., endothelial injury, which leads to atherogenesis. Smith and Frohm (1985) support the psychological segment of these pathways, since hostility is associated with both vigilance/suspicion and anger. Several studies cited below support the biological segments of the pathways in Williams et al.'s (1985) model as well as Dembroski's (1978) multicomponent model.

In a review of the literature, Houston (1994) showed that different hostility components and measures (e.g., Ho scale, Trait-Anger, PH) have been related to different manifestations of enhanced physiological reactivity (e.g., reactive diastolic blood pressure - DBP, reactive systolic blood pressure - SBP, reactive heart-rate - HR). Houston (1994) articulates several assumptions that exist in this research area. First, psychological factors need to be present as traits to have a recurring effect on cardiovascular reactivity and on CHD-progression. Second, traits interact with situational variables in affecting physiological reactivity. Thus, personality, individual differences, biological constitution and situational variables need to be considered when testing the stress-hostility-reactivity link. Third, the experimental situation mimicking this link has to "allow" the hypothesized trait (e.g., antagonism) to be manifested,

and thus, affect reactivity. Finally, Houston (1994) attempts to explain the mixed results reported previously by suggesting a third variable: Degree of engagement of subjects in a stressful task. The extent to which experimental stressors engage subjects' resources predicts the physiological consequences of their hostility. Thus, when subjects are not stressed interpersonally, their resources are unaffected and hostility may not be associated with enhanced cardiovascular reactivity (e.g., Suarez & Williams, 1989; discussed below). When subjects are stressed interpersonally to a moderate degree, hostile subjects may resort to or manifest their antagonism by disengaging from the task, thus, possibly exhibiting lower reactivity levels. However, when subjects are strongly stressed interpersonally, high hostile subjects cannot disengage from the situation and exhibit the expected higher reactivity than non-hostile subjects. The following studies will exemplify these points and so potentially show support for the hostility-cardiovascular reactivity-CHD model.

One of the first studies that examined the physiological correlates of hostility compared 33 MI cases with 31 non-CHD controls on physiological reactivity measures during the SI and a subsequent history quiz (Dembroski et al., 1979). Across all subjects, Potential for Hostility (PH) ratings were positively and significantly correlated with increases in SBP during the history quiz relative to base-line levels ($r = .36$). This supported the first segment in the psychophysiological reactivity model by linking hostility with reactivity. Additionally, DBP levels of MI patients increased significantly more during the SI

than in the control group. This supported the second segment of the psychophysiological reactivity model by linking reactivity with CHD, a relatively neglected aspect of the model.

Suarez and Williams (1989) manipulated the type of psychological stressor (i.e., the situation) inflicted upon subjects. High Ho subjects were compared with low Ho subjects on cardiovascular reactivity, after having been randomly assigned either to a stressor with or without harassment. High hostile subjects who were **also** harassed, showed the greatest increases in DBP and forearm blood-flow compared with high hostile non-harassed and low hostile harassed and non-harassed subjects. Hostile harassed subjects reported the highest levels of state-anger. Additionally, hostile subjects that reported being angered during the stressor had higher reactivity levels than hostile subjects who were not angered, while state-anger had no effect on reactivity among non-hostile subjects. The effects of the harassment on reactivity were diminished after considering subjects' state-affect. This study had one limitation: Harassed subjects received differential (stress-related) treatment prior to measuring base-line physiological measures. Despite this limitation, this study showed how traits (hostility), type of stressor (harassment) and emotional state (anger) are all important determinants of cardiovascular reactivity. Thus, hostile people may experience increased reactivity only when a harassing event makes them angry. This supports Houston's (1994) notion that situations which elicit an affect (anger) that is congruent with the hypothesized underlying trait

(hostility) increase reactivity.

In a further analysis of the later study, Suarez and Williams (1990) tested the relations between different dimensions of hostility, task characteristics and reactivity. A factor analysis of several trait measures (e.g., AX, BDHI) revealed two hostility factors: Antagonistic hostility (reflected by Anger-Out, low Agreeableness and expressive hostility) and Neurotic hostility (reflected by Anger-In, Neuroticism and experienced hostility). A significant interaction of Antagonistic hostility x task type x period revealed that subjects high on Antagonistic hostility showed the highest reactive-SBP and reactive forearm blood-flow only under a harassed condition. A significant interaction of Neurotic hostility x task type x period revealed that subjects high on Neurotic hostility showed the highest reactive-forearm blood-flow only under a harassed condition. However, the effect of Neurotic hostility was weaker than that observed for Antagonistic hostility. Finally, only among high-hostile subjects, either Antagonistic or Neurotic, were there positive and significant correlations between self-reported negative affective states (anger, irritation, upset and tension) and cardiovascular reactivity. These findings supported and extended those of Suarez and Williams (1989) and showed that type of hostility, type of stressor and experienced affective states during the stressor, are all important in determining cardiovascular reactivity. These results support studies suggesting that antagonistic hostility is the toxic type of hostility in relation to CHD (Dembroski et al., 1989; Siegman et al., 1987).

Smith and Brown (1991) extended these findings to social interactions within the marital context. They asked members of couples either to simply discuss or to try to influence their spouse's opinion on a topic. While wives' Ho scores did not predict their reactivity levels, husbands' Ho scores significantly and positively predicted their increased HR levels across conversation types. Hostility interacted with conversation condition in predicting SBP Ho scores were positively correlated with change in SBP during the influencing condition and negatively during the discussing condition The latter finding may have resulted from hostile men disengaging from the mild stressor (simple discussion), resulting in low levels of reactivity (Houston, 1994) Finally, and more fascinating was that husbands' Ho scores significantly and positively predicted wives' changes in SBP This study replicates and extends the findings of Suarez and Williams (1989) by showing that hostility and reactivity are associated only in interpersonally stressful situations that involve attempts to control or influence others Finally, men's hostility may have negative effects on their wives' physiological parameters during conflicting situations, a finding with potential health implications

The studies reviewed so far included hemodynamic measures and laboratory paradigms. Pope and Smith (1991) extended these findings to cortisol levels obtained during daily activities. Cortisol is important since it is a hormone that has been experimentally shown to enhance coronary and cerebral atherosclerosis induced by cholesterol in animals (Rosenfeld, Marmorston,

Sobel & White, 1960). Pope and Smith (1991) found that men with high Ho scores had significantly higher levels of cortisol during daytime than men with low Ho scores. However, these differences were not observed during waking or evening urine analyses. High Ho men experienced three times higher increases in daily cortisol levels compared with low Ho men. Although the groups did not differ in other potential confounding variables (e.g., age, weight, waking time), differences in type/amount of daily activities were not controlled for. To the extent that degree of stress encountered during the day would have accounted for these differences, these findings may have supported the psychosocial vulnerability model as well.

Another study (Schonwetter, Dion, Ready, Dyck & Gerrard, 1991) examined the relations between SI-derived TABP, Ho-assessed hostility and levels of thromboxane (a vasoconstrictor and platelet aggregator), prostacyclin (a vasodilator and platelet inhibitor) and bleeding time. Following a mild vascular injury, hostile Type-A subjects had the highest levels of thromboxane B_2, a metabolite of thromboxane, compared with hostile Type-B and all other non-hostile subjects. Following brief physical exercise, hostile subjects had higher levels of thromboxane B_2 than non-hostile subjects. Furthermore, bleeding time was shorter for hostile Type-A subjects than for hostile Type-B subjects. No effects were found after a psychological stressor (color naming stroop test), nor were prostacyclin levels related to any psychological trait. Thus, hostility (and the TABP) may be related to vasoconstriction and spasms,

a possible mechanism by which hostility may be related to ischemia (Helmers et al., 1993) and to UA at rest (Masen et al., 1979; Mendos De Leon, 1992). Finally, as hostility and TABP are related to bleeding time and to a platelet-aggregator, they may affect thrombosis, a key factor in MI (De Wood et al., 1980).

Suarez, Williams, Kuhn, Zimmerman and Schanberg (1991) showed that Ho scores significantly interacted with total cholesterol levels in predicting hormonal reactivity levels (change in epinephrine) to an arithmetic task. Among high-hostile subjects, cholesterol and change in epinephrine were positively correlated ($r = .59$), while among low-hostile subjects, cholesterol and change in epinephrine were unrelated. Similarly, in high-hostile subjects, cholesterol was positively correlated with changes in HR ($r = .25$) and was negatively correlated with changes in HR among low-hostile subjects ($r = -.43$). Thus, hostility may moderate the effects of another CHD-risk factor, cholesterol, on cardiovascular and neurohormonal reactivity.

These outcomes are important since a faster HR may increase myocardial demand for oxygen, which can result in ischemia, and increased epinephrine may result in a higher cardiac output, which increases levels of BP (Julius, Schneider & Egan, 1985). According to Williams (1994), the positive association found in hostile men between cholesterol and reactivity has important implications for CAD- and CHD-progression. Higher cholesterol and catecholamine levels (e.g., norepinephrine) have been shown to result in an

altered activation pattern of macrophage, or cells in the arterial walls This altered pattern results in a reduction in arterial mobility and increased release of growth factors by the macrophage, two key processes in atherosclerosis (Ross & Glomset, 1976) Thus, hostility may enhance the atherogenetic properties of cholesterol, norepinephrine and epinephrine

Some experimental studies examined hostility's role in reactive-ischemia For example, Ironson et al (1992) found that stressors related to anger/hostility resulted in greater ventricular dysfunction (reflecting ischemia) than general anxiety-related stressors Thus, stressors that induce anger/hostility may **cause** more severe ischemia, which may lead to CHD However, this study did not address individual differences in trait-hostility and their effects on ischemia

All the previous studies examined hostility and physiological reactivity Hostility may also be related to elevated **resting**-BP. For example, Mann (1977) showed that antagonistic hostility (acting out hostility) was significantly higher in hypertensive than normotensive subjects, but only in **non-neurotic** subjects Thus, antagonistic hostility which is not accompanied by Neuroticism is more strongly related to hypertension. This study suggests that hostility may be related to CHD via its relation with elevated resting-BP, a CHD risk factor (Kannel et al., 1986).

The latter findings with resting-BP are important in light of a recent study (Carroll, Smith, Sheffield, Shipley & Marmot, 1995) that found that reactive-SBP and reactive-DBP did not add a meaningful amount of variance (1%) to the

prediction of resting-BP over 4.9 years, after initial resting-BP was considered. The best predictors of resting-BP at follow-up were base-line resting-BP and subjects' age. These findings cast doubt on the importance of reactive-BP, and suggest that resting-BP may be a more reliable predictor of subsequent hypertension.

Finally, experimental studies conducted with animals provide compelling evidence for the relation between psychological parameters and CAD. Manuck, Kaplan and Clarkson (1985) reviewed three studies conducted on cynomolgus monkeys, that tested the assumption that behavioral factors interact with social or environmental ones in affecting atherogenesis. In one study, moderate hypercholesterolemic male monkeys were either grouped in a stable or an interrupted environment (the social condition variable). Animals were then observed and categorized as dominant or subordinate (the behavioral or social status condition). Whereas social condition and social status did not have any main effects on atherogenesis after 22 months, their interaction did, independent of total and HDL-cholesterol. Dominant monkeys in the unstable environment had significantly more severe atherosclerosis than dominant monkeys in the stable environment and more than subordinate ones in the unstable environment. A second experiment showed that monkeys who responded to stress (capture threat) with higher HR, developed more severe atherosclerosis than those responding with lower reactive-HR. This important finding links reactivity to CAD. Finally, the third experiment found that

behavioral factors (i.e., social stress) provoked atherogenesis even in normocholesterolemic monkeys, suggesting why certain individuals with normal or even hypocholesterol develop CAD.

The psychosocial vulnerability model

This model states that a) Hostility is related to an unhealthy psychosocial profile (e.g., low social support, frequent stressful events); B) This unhealthy psychosocial profile predicts CHD (Smith, 1992; 1994). This model suffers from two main limitations. First, it does not specify how the unhealthy psychosocial correlates of hostility are related to CHD. The psychophysiological reactivity model may play a role in this model as well (Smith, 1992) Second, the specificity of this model is not strong, as an unhealthy psychosocial profile (e.g., chronic stress) predicts other diseases such as cancer (e.g., Jensen, 1987). Thus, this model may not explain why hostility is a CHD, as apposed to a health, risk factor. On the other hand, it may explain previous prospective associations between hostility and other diseases such as cancer and all-cause mortality (e.g., Shekelle et al., 1983). The following studies provide evidence for and elaborate upon this model.

Smith and Frohm (1985) found that high Ho scorers reported more negative life events, more frequent and severe daily hassles, and less amount and satisfaction from social support than low Ho scorers. The best social discriminators between high and low Ho scorers were subjects' satisfaction with and number of social ties, and more severe and more frequent hassles.

However, this study used self-reported measures alone, and shared method variance may partly account for these results. In addition, negative affect or Neuroticism, which is related to hostility and daily hassles (Barefoot et al., 1989; Watson & Pennebaker, 1989) may account for these relations. Thus, reporting an unhealthy psychosocial profile may reflect a reporting bias related to Neuroticism rather than to hostility. If Neuroticism plays a role in the relation between hostility and negative life events, this would weaken the relation between hostility and objective outcomes such as CHD (Watson & Pennebaker, 1989).

In addition, the bias of reporting more hassles may result from hostile people having more negative schemas about others and about their life. Allred and Smith (1991) found that high Ho males recalled more hostile adjectives describing a person from a hostile interaction than did low Ho males after the same type of interaction. Additionally, high Ho males rated their partners, whether interacting with them in a hostile or neutral manner, as more hostile than did low Ho males. Thus, hostile people may report more hassles since they **perceive** interpersonal interactions more negatively. Thus, hostility may be related to a perceptual bias of others and of daily interactions.

Supporting this perceptual bias, and extending it to SI-defined hostility, Hall and Davidson (in press) found that SI-derived Hostile-Style was positively correlated with subjects' ratings of the aggression of the SI-interviewer. This was despite the fact that the interviewer's aggression, as rated by a third

person, was unrelated to subjects' Hostile-Style. Thus, interviewers did not react or alter their behavior towards more hostile subjects, but the latter perceived this to occur. This supports a perceptual bias concerning others' behavior among hostile individuals.

Smith, Pope, Sanders, Allred and O'keefe (1988) conducted an extensive study of the psychosocial profile of hostility in several social domains. They found that hostility was associated with more frequent and severe daily hassles, more negative life events and less amount and satisfaction with social support. These findings remained intact even after controlling for anxiety, thus, they may not be due to biases related to Neuroticism. Hostile subjects reported more conflicts, and less support and less constructive emotional-expression in their original family than low hostile subjects. In the marital domain, husbands' hostility was positively associated with self-reported marital conflicts, and negatively associated with marital satisfaction and with receiving positive regard from their spouses. In wives, hostility tended to be correlated only with marital conflicts. Finally, hostility was positively associated with job-related interpersonal stress and not task-related stress, and negatively related with job-satisfaction. Smith et al. (1988) concluded that hostility is associated with a poor psychosocial profile across domains (general, family, marital and work). Thus, this study strongly supports the first segment of the psychosocial vulnerability model.

Despite the subjectivity of reporting an unhealthy psychosocial profile, the

latter has been related to CAD and CHD. Ruberman, Weinblatt, Godlberg and Chaudhary (1984) found that stress (crises and adverse reactions to them) and social isolation (lack of social support and communication about illness) significantly predicted all-cause and CHD-related mortality 3 years after MI. The combination of stress and social isolation put men at 4.5 and 5.6 times the risk of all-cause mortality and CHD-related mortality, respectively, compared with men who had neither psychosocial limitations. These effects were independent of other prognostic variables (e.g., age, myocardial function). However, the assessment of stress confounded stressor with reactions, and social isolation included difficulties with communicating concerns about the MI. Despite these limitations, this study supports the second segment of the psychosocial vulnerability model which postulates a link between an unhealthy psychosocial profile and CHD.

The second segment has been supported in several other studies. For example, Seeman and Syme (1987) demonstrated that instrumental social support (i.e., frequency of receiving help for rides, household tasks and financial aid) and the feeling of being loved were significantly and negatively related to degree of atherosclerosis. These relations were independent of several variables including age, gender, smoking, cholesterol, TABP and hostility (assessed with the Ho scale). Finally, Helgeson (1991) found that post-MI patients who believed they had a confidant were readmitted less to hospital and had less chest-pain than those not holding this belief. Taken together, lack of

social support and increased stressful events, a poor psychosocial profile, are related to CAD and CHD-outcomes.

The transactional model

This model is an extension of the psychosocial vulnerability model. It arose as a result of the psychosocial vulnerability model's inability to explain why hostile people have an unhealthy psychosocial profile. Do adverse stressful events just "happen" to hostile people or are they also **created** by them? The transactional model (Smith, 1992) states several stages: a) Hostile people mistrust others, anticipate negative events, and behave antagonistically; b) This creates both more antagonism in the people with whom they interact and more interpersonal conflict; c) This results in increased interpersonal stress and reduced social support, an unhealthy psychosocial profile; d) This reinforces hostile people's views (e.g., mistrust); e) This psychosocial profile is associated with CHD.

Support for this model comes from several indirect findings. Fontana et al. (1989) found that scores on the Ho subscale of Cynical Mistrust (Costa et al., 1986) were positively correlated with levels of self-worth, perceived injustice and revenge. Thus, cognitively hostile individuals think highly about themselves, and see others as sources of evil who deserve punishment. This mode of thinking can easily elicit interpersonal conflicts.

Smith, Sanders and Alexander (1990) examined the correlates of hostility within the marital context, a potentially salient stressor for eliciting the hostile

interpersonal style that is central to the hostility construct (Barefoot, 1992). Married couples were observed for their hostile behavior while discussing high and low conflict topics selected by each couple. High Ho men reported more anger, and blamed their wives for the conflict and attributed to them greater intentionality than low Ho men. These findings were not found for women. Analysis of couples as a unit indicated that it was sufficient that one member of the couple was hostile for the couple as a unit to exhibit more hostility during the high conflict topic, and for the hostility of the other non-hostile partner to increase as well. Thus, high-hostile people become more angry during conflicting interactions, and elicit greater hostility in non-hostile people, supporting several of the segments of the transactional model.

Finally, indirect support for this model comes from a unique study conducted by Snyder and Swann (1978). Subjects (perceivers) were told that their opponent target in a reaction-time game was either hostile or non-hostile, according to his alleged "personality". Perceivers who were told their opponent was hostile initiated greater hostility toward that opponent (delivered a higher range of noises) than perceivers who were told their opponent was non-hostile. Opponents then returned higher noise levels when playing against perceivers who perceived them to be more hostile. Finally, perceivers induced to think their opponent was hostile rated him as more aggressive after the game than perceivers induced to think their opponent was non-hostile. Thus, the hostile expectations perceivers held about their opponent increased their hostile

behavior toward that opponent, which increased their opponents' hostile behavior in return. The latter served as **behavioral confirmation** for perceivers' expectations and hostile attributions, which led them to rate their opponents as more aggressive. This important study showed how hostile perceptions can create a hostile reality, supporting the transactional model. However, individual-differences in trait-hostility were not assessed, and this would have provided a more direct test of the transactional model.

Although more difficult to experimentally validate, the transactional model may be a more realistic account of the complexity of human interactions. Dembroski's (1978) model related to these bidirectional complex processes as well. Through behavioral confirmation, the transactional model also helps to explain the endurance of the hostility trait: Hostile people, who expect others to be hostile, create hostility in their interpersonal interactions, which reinforces and maintains their hostility.

The health behavior model

This model assumes a pathological path that differs from the previous models. According to the health behavior model: a) Hostility is associated with poor health habits (e.g., smoking, alcohol-consumption, little physical exercise); b) These poor health behaviors predict CHD. The importance of this model with respect to health psychology is that several "traditional" CHD risk factors (e.g., smoking, inactivity) may be manifestations of psychological parameters (Jenkins, 1988), one of which may be hostility. Thus, hostility may

underlie several traditional CHD risk factors. The main limitation of this model is in its specificity, since several of the unhealthy health habits (e.g., smoking) are linked to illnesses other than CHD, such as cancer. The following studies support and add important information to this model.

Leiker and Hailey (1988) found that high Ho scorers reported poorer overall health habits than low Ho scorers. Specifically, high Ho scores were associated with lower levels of physical fitness/exercise, and self-care (adequate amount of sleep, dental care), and with intoxicated driving (driving under the influence of alcohol or drugs). Among subjects reporting poor health habits, 62% scored above the median on the Ho scale. However, this study used only self-report measures, and the predictive validity of their health-habits measure in relation to CHD is unknown. Despite these limitations, Leiker and Hailey (1988) offered possible explanations for the link between hostility and poor health behavior. First, mistrust and suspicion may lead hostile people to reject recommendations of others (e.g., family) and of scientific knowledge (e.g., from health professionals) that living a healthier life style prevents CHD. Second, hostile people may also be unwilling to improve their health since this may prolong their life in a "hostile world". However, this study did not test these underlying cognitions.

Houston and Vavak (1991) found that high Ho scores were associated with a tendency to drink more alcohol, to drive after drinking alcohol and to have a higher relative body mass. However, cigarette smoking and preference

for unhealthy foods were not associated with hostility.

Several studies examining the prospective relation between hostility and CHD found that SI-derived and self-reported hostility assessed at base-line were positively associated with base-line levels of smoking and/or alcohol-consumption (Dembroski et al., 1989; Koskenvuo et al., 1988; Shekelle et al., 1983). Shekelle et al. (1983) also found that smoking (and hostility) was associated with all-cause mortality, supporting the second segment of the health-behavior model.

The studies reviewed above showed correlations between hostility and CHD risk factors in a cross-sectional design. However, developmental changes and life circumstances may affect the nature of these associations. High-hostile people, who smoke or lack physical activity, may reduce their smoking or increase their activity as a result of social pressure or other reasons. Thus, it is important to test the link between hostility and health behavior prospectively as well. Siegler, Peterson, Barefoot and Williams (1992) found that Ho scores assessed in 19 year-old men and women significantly and positively predicted caffeine levels, body mass index, ratio of total cholesterol to HDL-cholesterol and smoking 21-23 years later. These relations were independent of the effects of age and sex. Most correlations were observed cross-sectionally again at follow-up. Although their correlations were small, prospective and constant associations between hostility and poor health-behavior may have a large impact on health later in life. The only limitation of this study was that the

effects of base-line levels of health-behaviors (e.g., base-line smoking) should have been considered when evaluating risk factors in adulthood.

The previous studies examined the correlates of hostility in healthy individuals. However, hostility may lead to poor health-behavior among diseased people as well. Lee et al. (1992) found that hostility levels tended to be higher for subjects who withdrew from an antihypertensive trial than for those who remained in the trial. Additionally, subjects who forgot to take their medication at least once during the past week had significantly higher levels of hostility than those not forgetting to take their medication. However, the measure of hostility was confounded with distress, thus, it is unclear to what extent these results reflect biases related to Neuroticism or hostility. Despite this limitation, this study suggests that when ill, hostile people may be at greater health risks due to low medical adherence (to antihypertensive regimes in particular). The behavior observed by Lee et al. (1992) may further increase risk for hypertension and CHD as well (Kannel et al., 1986).

Constitutional vulnerability model

This model suggests the following stages: a) An underlying, biological constitution predisposes certain people to have a hostile profile; b) The underlying constitution also leads to CHD. The psychophysiological reactivity model suggests that processes inherent in hostility (e.g., cognitive cynical appraisals) are necessary for interpersonally stressful events to elicit physiological reactivity (e.g., Chesney, 1985; Houston, 1994). In contrast,

Krantz and Durel (1983), to whom the constitutional vulnerability model may be originally attributed, suggest that the TABP and its components (e.g., hostility) **reflect** an existing constitutional characteristic of increased physiological reactivity, underlying the hostility complex. Thus, hostility is the psychological **manifestation** of an underlying enhanced physiological reactivity

Krantz and Durel (1983) reviewed several studies which together show that increased cardiovascular reactivity in Type-A individuals does not necessarily require the cognitive mediation inherent in the TABP or in hostility. First, Type-A individuals under general anaesthesia (without effects of appraisal) have greater BP-reactivity than Type-B individuals. Second, beta-blocking drugs (e.g., propranolol), which attenuate sympathetic reactivity, also reduce Type-A and hostility levels (e.g., Krantz et al., 1982; Schmieder, Friedrich, Neus and Ruddel, 1982). According to the constitutional vulnerability model of Krantz and Durel (1983), a basic tendency of increased peripheral reactivity (e.g., higher SBP, HR) results in a basic cognitive interpretation of "fight-flight", which in turn yields emotional (e.g., anger), behavioral (e.g., Anger-Out), and cognitive reactions (e.g., hostile attributions). These feed back into the initial cognitive interpretation of the physiological reactivity and exacerbate it. Such individuals may have a genetically (familial) or environmentally-determined (early conditioning) tendency to experience increased physiological reactivity in certain situations (Krantz & Durel, 1983), overtly manifested by hostility. Interestingly, Krantz et al. (1982) found that only the stylistic

components of TABP (such as PH which is heavily influenced by Hostile-Style) were lower in patients taking beta-blockers, and that the content components of TABP were unchanged. Thus, PH and Hostile-Style may indeed be more intrinsic manifestations of physiological reactivity, unlike more cognitive content aspects (e.g., Hostile-Content) derived from the SI.

Williams (1994) suggested a constitutional deficit underlying the hostility complex. His theory is a meta theory which integrates the correlates of hostility outlined by the models mentioned above. As the previous models have shown hostile people have a psychophysiological profile including a behavioral /transactional interpersonal aspect (antagonism; Barefoot, 1992; Smith, 1992), a physiological reactivity aspect (heightened BP and HR mediated by the sympathetic nervous system; e.g., Suarez & Williams, 1989), and poor health-behavior (higher caloric-intake, alcohol-consumption and more smoking; e.g., Houston & Vavak, 1991). Williams (1994) shows that each of these aspects, the behavioral, the physiological reactivity and the poor health behavior, can be directly or indirectly related to **depletion of brain serotonin** levels. For example, Roy, Adinoff and Linnoila (1988) found that scores on a self-report measure of "Urge to act out hostility" (or antagonism) were significantly and negatively correlated with levels of a metabolite of serotonin ($r = -.53$) among 17 normal volunteers.

Thus, the complex profile associated with hostility as suggested by the transactional model, the psychophysiological reactivity model and the health-

behavior model, may actually result from an underlying biological marker of the hostility syndrome: Decreased serotonin levels. However, not all aspects of this model are supported nor are they detailed. As Williams (1994) suggests, further empirical support for this meta-model may have fascinating assessment and therapeutic implications. Finding precise ranges of serotonin depletion that are related to the hostility complex and to CHD may be a more reliable assessment of "coronary-prone" risk than assessing hostility. Second, clinical trials may test whether increasing serotonin levels in hostile CHD patients reduces hostility and recurrent CHD events. Finally, future psychotherapies aimed at hostility-reduction may wish to test whether their possible effects on CHD are mediated via increasing serotonin levels, as this model would predict.

Integration of models

Smith (1992; 1994) and Dembroski's (1978) model suggest that the hostility-CHD models reviewed above are not mutually exclusive, but can be complementary to each other or interrelated. Thus, experiencing and/or creating more interpersonal stress, as postulated by the psychosocial vulnerability and transactional models may result in more physiological reactivity as postulated by the physiological reactivity model. The unhealthy life style postulated by the health-behavior model (e.g., alcohol-consumption) may isolate hostile individuals and reduce their social support, as postulated by the psychosocial vulnerability and transactional models. The findings of Suarez et al. (1991) that hostility may mediate the relations between cholesterol and

epinephrine, links the health-behavior model (e.g., increased caloric intake) and the physiological reactivity model (enhanced sympathetic activation). Finally, the constitutional deficit of serotonin depletion suggested by Williams (1994) ties together several segments of the other models, and points to their possible interdependence and underlying cause.

Future studies should test more clearly the segments of each of these models, and how each segment is related to one another. Furthermore, prospective studies should examine each model's explanatory power in the hostility-CHD relation by assessing hostility, its correlates as suggested by each model, and CHD-development over a long follow-up period (Smith, 1992). Such a study could administer hostility measures (e.g., PH, Barefoot's refined Ho scale), and measure physiological reactivity (e.g., stress-induced SBP), daily hassles and social support, health-behavior (e.g., adherence to doctor's advice), traditional risk factors (e.g., resting-SBP), and CHD-incidence (e.g., MI). Such a study could then test the impact of hostility on CHD-development after controlling for each model's components. If hostility predicts CHD after comprehensively controlling for a certain model's components but not after controlling for another model's components, then the latter model may play a crucial role in the hostility-CHD link, and the former model may only partly explain this link. Finally, interesting interactions between hostility and components of various models (e.g., daily hassles) may increase the prediction of CHD compared with consideration of main effects alone (Houston, 1994;

Perkins, 1989; Smith, 1992).

Advances in predicting CHD and in understanding the mechanisms by which hostility may cause CHD are critical for the development of therapeutic interventions. Hostility-modification may be conducted with certain individuals who are at risk for CHD according to certain models (e.g., those high on hostility and physiological reactivity). Finally, treatments may target certain life style parameters associated with hostility (e.g., drinking alcohol, creating daily hassles) in addition to hostility-reduction. Such interventions may be more effective as well as provide a test of the validity of these models.

CHAPTER FIVE

HOSTILITY-MODIFICATION: PREVIOUS RELEVANT STUDIES AND RATIONALE OF PROPOSED NEW TREATMENT

Introduction

Despite the progress in the understanding of coronary-prone behavior, the clinical application of this knowledge has been limited. Furthermore, despite the growing acceptance of hostility as the toxic component of the TABP (Williams, 1987), few clinical trials have focussed directly on hostility or anger alone (Deffenbacher, 1994). This chapter will first review guidelines and strategies suggested by two leading investigators regarding the modification of hostility/anger. The chapter will then provide a critical review of relevant clinical trials that have been conducted with healthy subjects, hypertensive patients and cardiac patients. The methodology of these trials will be evaluated with a 26-item check-list (Gardner, Machin & CampBell, 1989) which considers design features, conduct of trial, analysis and presentation of intervention data. Finally, based on the conceptualization of CHD-predictive hostility, empirical data, previous trials and clinical literature, this chapter will end with an outline of the rationale and content of the proposed hostility-reduction treatment.

General guidelines

Deffenbacher (1994) reviewed several anger-hostility treatments and provided general guidelines for successful treatment with high-hostile or angered individuals: Group therapy is warranted, sessions must last at least

75-90 minutes, rehearsal of material after each meeting is central, and therapists must employ an accepting yet, problem-confronting approach. Deffenbacher (1994) identified the following recurring therapeutic elements that promote therapeutic success. First, therapeutic changes are enhanced by personal awareness of thoughts, feelings and behavior by using self-monitoring and assessment. These techniques yield early sensitization to cognitive processes and hostility-related cues. Second, hostile reactions can be cut short by response disruption skills such as "time-outs" or disengagement, reaction delay, thought-stopping, self-talk and distraction. Third, the affective and physiological components of hostility (e.g., muscular tension, increased BP, HR) can be effectively altered by relaxation skills (deep breathing and progressive muscular relaxation). Fourth, seven types of common cognitive errors need to be restructured: Incorrect estimation of aversive probabilities ("things will **always** go wrong"), dictator-like thinking/expectations ("**everyone** must know what I think"), catastrophizing ("This is the **end** of the world"), overgeneralization ("**All** ____ are ____"), obscene thoughts, dichotomous thinking ("Things are either good or bad") and misattribution ("You surely did this to **hurt me**"). Modifying these distorted cognitions can be done via enhanced self-awareness, learning the relation between cognitive and affective and behavioral responses, suggesting alternative thoughts, and reinforcing and rehearsing new realistic cognitions. Fifth, affective and physiological reactions can be reduced by humor. Humor induces an incompatible affective state (i.e.,

laughter rather than anger), and ridicules and induces cognitive distance from one's distorted cognitions. Sixth, hostile reactions to provocations can be effectively altered by problem-focused coping (looking at provocations as problems calling for solutions). Finally, antagonism and suppressed reactions can be reduced by alternative interpersonal behaviors such as assertiveness. As we shall see below, all hostility-reduction treatments include several of these elements and skills, and which ones are included depends on what dimension of hostility they target (e.g., relaxation for anger and reactivity, cognitive-restructuring for maladaptive hostile thoughts, assertiveness training for explosive and suppressed hostility). However, most of Deffenbacher's work (reviewed below) focuses on anger, rather than on the broader construct of hostility, as the main therapeutic target, and does not link it to CHD.

In contrast, Williams and Williams (1993) developed a hostility-reduction treatment in the context of CHD. Their treatment attempts to reduce all three facets of hostility Antagonism, cynicism and anger. Their program lasts 10 hours, provided in 10 sessions or five double sessions, and it may be given to cardiac patients or to healthy but hostile people and their partners. The first hour includes self-rating of hostility and introductions (aim of treatment, participants' goals and past-week's angering events). The second hour sensitizes participants to what angers them. Participants mention the things that annoy them the most, and learn why and how to monitor their own hostility at home. Hour three reviews participants' hostility logs and the health

consequences of hostility. In the fourth hour, participants learn how to analyze events by using the Hostility Road Map. This map helps to construct a mental representation of the sequences of events from provocation to reaction and includes the following questions: Is the matter worth my attention? Am I justified? Do I have an effective response?. Additionally, participants learn in hour four a hostility-reducing skill: Reasoning with oneself to cut reactions short. Hour five teaches graded assertiveness training: Simple assertions, describing others' misbehavior, showing one's empathy, reminding others of their commitments, expressing one's feelings, and stating the consequences. Hour five also discusses how to deal with hostile people (using acceptance and assertiveness). Hour six covers several deflection skills (thought-stopping, distraction, meditation). Hours seven and eight focus on improving interpersonal relations and reducing interpersonal antagonism by practicing listening, trust, empathy, tolerance and having a confidant. Hour nine focuses on humor and supporting networks (e.g., religion) for reducing cynicism. Finally, the 10th hour teaches one more skill: Pretending today is one's last day on earth, and summarizes the program.

The hostility-reducing skills suggested by Williams and Williams (1993) may be grouped according to the dimension of hostility they target. Antagonistic hostility may be reduced with assertiveness (moderating Anger-In and Anger-Out), listening, empathy and tolerance. Cynicism can be reduced with thought stopping, practicing trust, having a confidant and humor. Affective

hostility can be reduced with distraction and meditation. Reasoning with oneself and pretending today is your last may be appropriate for all dimensions However, this treatment has not yet been tested in a randomized controlled study, thus, its efficacy is unknown Study 1 of this thesis (Chapter 6) attempts to fill this gap, using the hostility treatment outlined below, which derived much of its content from the treatment developed by Williams and Williams (1993).

Previous hostility/anger modification trials with healthy subjects

Most of the controlled trials on hostility-reduction have been conducted with physically healthy participants These trials allow one to examine the efficacy of such treatments without CHD affecting outcome. Thus, before testing the effects of one variable (change in hostility) on another (change in CHD-related outcomes), we need to test whether it is possible to produce change in the first variable The trials are reviewed below according to their chronological order within a contextual framework. Table 6 presents each trial's sample, treatment and therapeutic targets, results and methodological rating (Gardner et al., 1989), and each study is briefly reviewed below

Hearn and Evans (1972) showed that systematic desensitization applied to imagined provocations reduced anger levels during imagined scenes, and reduced anger from stimuli not practiced during therapy, compared with no changes reported in a no-treatment control group. These results were maintained at a six-month follow-up (Evans & Hearn, 1973). Thus, relaxation was successful in reducing long-term arousal and anger. However, no objective

measure of physiological arousal was used, and subjects were not randomly assigned to groups.

Raymond Novaco was among the first to test the application of stress-inoculation training (exposure to stressful stimuli together while practicing learned counteractive responses) to anger and hostility in several populations. Novaco (1976a) assigned subjects with anger-control problems to one of four treatments: Cognitive self-instruction, relaxation, cognitive self-instruction and relaxation, and an attention-control group. A linear trend analysis indicated that subjects treated with the combined therapy reported the largest changes in experiencing anger and showed the largest changes in DBP and SBP during provocations, followed by cognitive self-instruction alone, followed by relaxation alone, followed by controls. However, since randomization was not used, and several issues were not considered in this study's brief report (e.g., background variables), biases may have existed in these results. Nevertheless, Novaco's treatment was the basis for future clinical trials in anger management.

Schlichter and Horan (1981) tested the effects of complete individualized stress-inoculation (gradual application of cognitive-restructuring and relaxation to six personally anger-provoking events), partial stress-inoculation (relaxation alone) or a no-treatment control group with juvenile delinquents. Only complete stress-inoculation therapy reduced observed verbal aggression during role-playing. Observed daily behavior in the institution was unchanged; The researchers speculated that this was partly due to negative modeling by

institution workers Thus, society's reactions (e g , school, family) may affect therapeutic outcome in hostility-modification.

Moon and Eisler (1983) found that cognitive stress-inoculation, social skills training and problem-solving training all reduced the number of angering events and observed aggression compared with an attention-control group. While cognitive stress-inoculation appeared to reduce cognitive, affective and physiological aspects of anger (reactive-SBP), it failed to improve assertion in this study In contrast, social skills training appeared to reduce all anger components and to improve assertion as well. The social skills training and problem-solving training groups significantly improved their anger-expression towards an "optimal" level Moon and Eisler (1983) concluded that improving interpersonal skills may alter hostile cognitions, and yield an overall benefit as well. This result also suggests that interpersonal communication skills are important in hostility-modification. However, observed hostility measures were not administered by raters who were blind to subjects' group-status Despite this limitation, this comprehensive trial used observed, self-report and physiological measures

Hazaleus and Deffenbacher (1986) tested whether cognitive and relaxation treatments are equally effective at reducing anger. At post-treatment and at a four-week follow-up, both groups reported lower levels of general anger, anger-arousal and less verbal antagonism than a no-treatment control group. Subjects receiving cognitive therapy reported more constructive coping

with provocations and less anxiety than controls. After one-year, both treatments still yielded lower anger levels, and subjects receiving cognitive therapy still reported lower levels of anxiety than controls. Despite the lack of observed behavioral measures, this study suggested that cognitive-restructuring and relaxation are equally effective at long-term reduction of anger, and that the effects of cognitive therapy may generalize to anxiety-reduction as well.

Deffenbacher, Story, Stark, Hogg and Brandon (1987) compared a combined cognitive-relaxation therapy (relaxation and cognitive-restructuring), a social-skills training group (practicing listening, feedback-provision and assertiveness) and a no-treatment control group. Both therapies yielded significantly lower general anger levels, Anger-In and Anger-Out than controls immediately after treatment and five weeks later. Both groups reported greater use of constructive coping with provocation at the five-week follow-up, and less general and situation-specific anger, and less anxiety and fewer physical symptoms related to anger than controls after one year (Deffenbacher, 1988). Although objective behavioral and physiological ratings were not used, this study showed that both cognitive-relaxation and social-skills training programs are equally effective at reducing short- and long-term levels of anger.

Deffenbacher, McNamara, Stark and Sabadell (1990) compared a cognitive-behavioral treatment, an anger-related process-oriented group (emotional disclosure and group problem-solving) typically provided in university mental health services, and a no-treatment control group. Both treatments

yielded lower levels of trait-anger, Anger-In, anger-arousal, dysfunctional coping with provocations and anxiety at a five-week follow-up when compared to controls, and some effects were maintained after 15 months. The treatments did not differ from each other on outcomes. Additionally, more than 50% of subjects in both treatments but only 11% of controls reported clinically meaningful reductions in trait-anger. This study showed that a general process-oriented treatment is as effective at reducing self-reported anger as a treatment specifically designed for anger-modification.

Nakano (1990) extended the use of relaxation training to Japanese men. Both hostility-related anxiety management training (relaxation for tension induced by hostile imagery) and operant self-control procedures (behavioral changes in speed/impatience guided by situational cues) were effective at reducing overall Type-A levels as well as competitiveness and impatience compared to wait-list controls. However, the therapy groups targeted different components of the TABP, hostility was not assessed despite being targeted by the anxiety management treatment, and randomization was not employed.

The work conducted by Deffenbacher and colleagues provides valuable information on reducing affective hostility or anger, antagonism and hostile cognitions. However, none of the trials reviewed up to this point conceptualized or related their treatment to CHD, nor did they provide an educational component linking hostility with CHD for treating high- anger or -hostile subjects. The following three trials addressed this issue.

Thurman (1985a; 1985b) used several conceptual and methodological improvements regarding these and other issues. University faculty high on Type-A were randomly assigned to either a cognitive-behavior modification (CBM), CBM plus assertiveness-training (CBM + AT) or a single-session minimal-treatment control group. Thurman (1985a; 1985b) attempted to **focus** on the CHD-prone aspects of the TABP, which included hostility, competitiveness and impatience (prior to emergence of hostility as the most toxic component). This trial also included assessments by subjects' spouses/friends. At post-treatment, both CBM and CBM + AT were equal and more effective at reducing global Type-A, hostility and irrational beliefs than the minimal-treatment control group. Type-A levels and irrational beliefs were still reduced to a greater extent in both treatments after three, six and 12 months, and this was supported by ratings from subjects' spouses/friends at each follow-up. After 12 months, only subjects receiving CBM reported lower levels of hostility than controls. Thus, this study showed that cognitive and behavioral treatments can reduce self- and spouse-rated levels of Type-A and hostility, and that these reductions are durable. Thurman (1985b) concluded that the self-help nature of these treatments makes subjects become independent of the therapist and helps to maintain therapeutic gains.

Roskies et al. (1986) found that a cognitive-behavioral stress-management program for Canadian Type-A managers yielded significantly greater reductions in SI-derived Type-A ratings including hostility (PH) than

aerobic and weight-training controls. However, no differences were observed between the groups on reactivity measures (e.g., reactive-SBP). The cognitive-behavioral program taught subjects to make their perceptions of and responses to different stressors more flexible, and also included self-monitoring skills, comprehensive application of coping skills (e.g., restructuring, communication-skills), pleasure planning and relapse-prevention. Their treatment emphasized homework as the main source of behavioral change. This study provided important information on reducing hostility and important methodological issues.

Finally, Eysenck and Grossarth-Maticek (1991) conducted a series of important studies which tested the efficacy of their particular treatment, creative novation behavior therapy (described below) in several formats (e.g., individual, group and bibliotherapy). Findings from the group therapy study will be presented since it is most relevant to the treatment proposed below. This study is important for three reasons. First, it is the only clinical trial which tested the effects of modifying psychological risk factors on primary prevention of CHD and mortality. Second, the treatment was planned to focus on TABP and particularly on hostility. Third, subjects (who were high on stress, smoking or cholesterol) were matched on traditional risk factors (age, sex, smoking) and personality type (CHD-prone and cancer-prone personality) prior to randomization to creative novation behavior therapy or to a control group. This methodological procedure made the groups more equal before treatment, and provided a more sensitive design. Creative novation behavior therapy was

designed to change strong tendencies for dependent interpersonal relationships, which result either in hostility (CHD-prone) or in emotional inhibition and helplessness (cancer-prone), to autonomous self-regulation (i.e., self-initiated ability to modify one's behavior according to consideration of its consequences and employing appropriate emotional expression and stress-responses). This treatment uses relaxation techniques, imagery, thought stopping and social skills training. Subjects in the treatment-group had a significantly lower CHD mortality rate (4.2%) than controls (15.4%), and the former tended to have a lower CHD-incidence (12.3%) than the latter (19.5%). However, results were not provided separately for CHD- and cancer-prone subjects, and change in personality was not assessed (i.e., a manipulation-check). Despite these limitations, this study demonstrated that behavior modification provided to CHD- and cancer-prone subjects can prevent CHD-mortality and cancer.

In summary, these trials conducted on healthy but high-anger/hostility/Type-A individuals provide strong support for the efficacy of psychological treatments to modify such parameters. These findings were found in both males and females, and were shown in several cultures (e.g., American, Canadian, Japanese). More so, several studies showed that these effects are maintained several months after treatment has ended (e.g., Deffenbacher, 1988; Evans & Hearn, 1973; Hazaleus & Deffenbacher, 1986). While several studies (e.g., Deffenbacher et al., 1987; Deffenbacher et al., 1990) suggest that the specific hostility-reduction strategies used yield similar

effects on anger and hostility measures, one study showed that interpersonal behavioral skills yielded the most comprehensive changes (Moon & Eisler, 1983) The latter study was also unique in assessing "optimal" anger-expression, thus addressing the fact that both Anger-In and Anger-Out may be unhealthy responses (Julius et al., 1986; Mendes De Leon, 1992).

However, none of these studies designed their treatment to solely and explicitly modify CHD-predictive hostility. While the more focused trials (e.g., Deffenbacher et al., 1987; Moon & Eisler, 1983) aimed at reducing general anger and hostility, the treatments were not conceptualized nor related to subjects as targeting factors related specifically to CHD. In contrast, the three trials that did relate their treatment to CHD (Eysenk & Grossarth-Maticek, 1991, Roskies et al., 1986; Thurman, 1985a) did not focus on hostility alone. Finally, the study by Eysenk and Grossarth-Maticek (1991) showed that psychotherapy had a strong preventative effect on CHD-mortality A few studies suffered from serious methodological limitations (e.g., non-randomized groups) and most studies did not employ observed measures of hostility (e.g., SI-derived PH).

Table 6
Study characteristics, results and methodological evaluation of hostility/anger modification trials with healthy subjects

Author	Sample	Treatment & Targets	Results	Follow Up	Method
Deffenbacher et al (1987), & Deffenbacher, (1988)	49 students 26 males, 23 females with Anger problems	8 weekly sessions of cognitive relaxation or social skills or no treatment control for reducing anger, antagonism and arousal	Short-term reduction in general anger, Anger-In/Out & long term reduction in general anger, anxiety & arousal compared to controls	Immediately after, 5 weeks & 12 months post treatment	35[a]
Deffenbacher et al (1990)	48 students, 26 males 22 females with anger problems	8 weekly sessions of either cognitive behavioral, process-group or no treatment control for reducing anger & arousal	Less trait-anger, Anger In anger arousal, dysfunctional coping & anxiety 5 weeks post treatment, & partial maintenance at 15 months	Immediately after, 5 weeks & 15-months post treatment	36
Eysenk et al (1991)	500 healthy men & women high on stress smoking or cholesterol, matched on risk-factors	6-15 weeks of creative-novation behavior therapy (autonomy training & appropriate emotional expression) for preventing CHD and cancer	Significantly less CHD and cancer mortality and trend to lower CHD incidence in therapy than in control group	7 years	30
Hazaleus et al (1986)	60 students, 31 males, 29 females, with anger-problems	6 weekly sessions of cognitive restructuring, relaxation, or no treatment control for reducing anger & arousal	Both groups reported less anger, arousal & antagonism, & cognitive group yielded lower anxiety than controls, maintained at one year	Immediately after, 4 weeks & one year post treatment	38
Hearn et al (1972) & Evans et al (1973)	34 female nursing students, matched on aggression	15 sessions of systematic desensitization applied to imagined provocations for reducing anger	Lower levels of anger in imagined scenes and in treatment & non treatment related stimuli	Immediately & 6 months post treatment	27

Author	Sample	Treatment & Targets	Results	Follow-Up	Method
Moon et al (1983)	40 male students scoring > mean of Anger inventory	5-weeks of either cognitive stress inoculation, social skills training, problem-solving training or control, for anger, antagonism & reactivity	Fewer angering events, less observed aggression & more assertion, more "optimal" anger expression & lower reactive-SBP in experimental than control groups	1 week post-treatment	33
Nakano (1990)	18 male Japanese managers matched on Type-A level	4 w[illegible]ly sessions of either anxiety management for reducing tension from anger/hostility, operant self-control for impatience & competitiveness or wait-list control	Both treatments reduced self-reported global Type-A and impatience and competitiveness levels better than controls	Immediately post-treatment	29
Novaco (1976a)	34 males & females with anger-control problems	Cognitive self-instructi[illegible] & relaxation, each alone, or an attention-control, for reducing anger, antagonism and BP	Combined treatment > self-instruction > relaxation > attention-control in anger- and BP-reduction	Immediately post-treatment	22
Roskies et al (1986)	107 Canadian Type A & physiologically reactive male managers	10 weeks of cognitive-behavioral, aerobic training or weight-training for reducing Type-A & physiological reactivity	Cognitive-behavioral treatment yielded greater reductions in SI-derived Type-A & hostility than other groups, no effects on reactivity	Immediately post-treatment	37
Schlichter et al (1981)	27 male delinquents with anger-problems	10 individual sessions for 5 weeks of complete or partial stress-inoculation or no-treatment control for reducing anger & aggression	Lower reported anger & aggression in both treatments, & less observed verbal-aggression in full stress-inoculation than in controls	2 weeks post-treatment	31
Thurman (1985a,b)	39 Type A university faculty 30 males, 9 females	8 weekly sessions of cognitive-behavior modification or cognitive behavior & assertiveness-training or 1-session control for reducing impatience, competitiveness & hostility	Long-term reductions in global Type A & irrational believes, supported by spouse/friends' ratings, & greater reductions of hostility in cognitive-behavior modification alone	Immediate & 3, 6, & 12 months post-treatment	40

* These metnodological ratings were based on the Check-list for statistical review of papers on clinical trials for the British Medical Journal (Gardner et al , 1989) Scores range from 1 to 45

Previous hostility/anger modification trials with hypertensive patients

The following trials are important for several reasons. First, they provide evidence for the feasibility of reducing hostility and its components among patient populations, rather than physically healthy populations. Second, and of theoretical and clinical importance, they provide evidence for the effects of hostility-modification on resting-BP, a major CHD risk factor and disease outcome (Jenkins, 1988). Third, they provide valuable methodological information for conducting clinical trials with patient-populations (e.g., recruitment criteria, outcome measures). The subject characteristics, results and methodological evaluation of these trials appear in Table 7, and are reviewed below.

Peled-Ney, Silverberg and Rosenfeld (1984) found that a six-month group therapy aimed at increasing emotional awareness (including anger) and at reducing anger-suppression reduced resting-SBP and resting-DBP throughout and after treatment, and to a greater extent than a no-treatment control group. Blood-pressure reductions were maintained three and 12 months after treatment. Finally, none of the subjects in group therapy who were followed after one year had mild hypertension (i.e., SBP/DBP $\geq$ 160/95 mm Hg). An interesting feature of their treatment was that group-competition was directed at enhancing therapeutic changes (e.g., emotional awareness and expression). However, randomization was not used, the treatment was unstructured, and few patients were followed after one year. Additionally,

change in anger-expression and medication-adherence were not assessed as possible mediators of therapeutic success.

Using a better methodology, Achmon, Granek, Golomb and Hart (1989) compared the effects of a cognitive group therapy (stress-inoculation and assertiveness), biofeedback (heart-rate control) and a no-treatment control group among Israeli hypertensives. Both treatment groups yielded significant reductions in SBP and DBP after treatment and at one and six-month follow-ups, while the control group did not. After treatment, the biofeedback treatment yielded significantly greater reductions in resting-BP than the cognitive therapy, which was superior to the control group. At one and six month follow-ups, biofeedback remained superior to cognitive therapy with respect to SBP alone. The cognitive therapy yielded significantly greater reductions in general anger and greater increases in levels of Anger-Out than biofeedback. This study provides important evidence for the feasibility and efficacy of anger-reduction among hypertensive subjects in a non-American culture. However, increasing Anger-Out may result in greater antagonistic hostility, a CHD-predictor (Dembroski et al., 1989; Matthews et al., 1977), and should not be equated with healthier or optimal anger-expression (Moon & Eisler, 1983). Achmon et al. (1989) concluded that BP was changed via each treatment's hypothesized path (i.e., anger-reduction in psychotherapy, HR-control in biofeedback). However correlations between change in resting-BP and anger-reduction or HR-control were not computed to test these claims more directly.

Davison, Williams, Nazemi, Bice and DeQuattro (1991) showed that a combined treatment of medical information for hypertension and relaxation-training reduced SBP, pulse-rate and "on-line" hostile thoughts significantly more than medical information alone. Group status interacted with subjects' norepinephrine levels such that reductions in BP were highest among subjects receiving the combined treatment, and who were high on base-line norepinephrine. Finally, and of greatest importance, Davison et al. (1991) were the first study to show that reductions in hostile thoughts were significantly and positively correlated with reductions in pulse-rate and resting-BP. These associations were highest among subjects with initially high norepinephrine levels. These findings suggest that enhanced sympathetic reactivity (as measured by norepinephrine) may mediate the relation between hostility and resting-BP. Although randomization was not used and the combined treatment did **not** target hostility, the results suggest a causal relation between hostility and resting-BP.

Hagga et al. (1994) improved on the previous design by employing randomization, equating number of sessions between experimental and control groups, and utilized a more theory-driven treatment approach. They found that a combined treatment of progressive-muscle relaxation and medical information yielded significantly greater reductions in reactive-SBP, reactive-DBP (trend), "on line" hostile thoughts and observed Anger-Out (trend) than a medical information control group. No differences were found between the groups in

relation to self-report hostility measures (e.g., Ho scale, Anger-Out). The authors implied that the self-report measures unaffected by the treatment are contaminated by Neuroticism, a personality dimension that may not be easily modified by relaxation therapy. However, it was more important that this trial altered hostility aspects that are relatively unrelated to Neuroticism (i.e., "on line" hostile thoughts, observed Anger-Out) since these are more predictive of CHD (Watson & Pennebaker, 1989). Hagga et al (1994) hypothesized that relaxation would be particularly effective at reducing the physiological consequences of TABP (reactivity). However, this was not confirmed, as the effects of relaxation appeared to "spread" and affect observed cognitive and behavioral hostility dimensions as well.

Taken together, these trials suggest that cognitive-behavioral modification aimed at anger-reduction (Achmon et al., 1989) and relaxation training aimed at reducing cardiovascular reactivity (Hagga et al., 1994) are effective at reducing hostile affect, observed antagonism, hostile cognitions and resting and reactive-BP. Additionally, Davison et al.'s (1991) findings suggest that modification of hostile thoughts may be causally related to decrease in resting-BP. However, the latter conclusion should be taken with caution since randomization was not employed in that study and their treatment was not designed to reduce hostility. Thus, a randomized-controlled trial with an experimental treatment focusing on hostility-modification would provide a more rigorous and direct test of the possible causal relation between hostility and BP.

Table 7
Study characteristics, results and methodological-evaluation of hostility/anger modification trials with hypertensive patients

Author	Sample	Treatment & Targets	Results	Follow-Up	Method
Achmon et al (1989)	77 Israeli hypertensives, 49 males, 28 females	17 weekly sessions of cognitive and assertiveness training for anger-reduction, or biofeedback for HR-control, or no-treatment control	Both treatments but not controls showed BP-reduction maintained after 6 months Cognitive treatment showed greater anger-reduction but less BP-reduction than biofeedback	Immediate & 1 & 6-months post-treatment	36[a]
Davison et al (1991)	58 males with borderline hypertension	7 weeks of medical information for reducing hypertension (Hygiene) or hygiene + 7 weeks of relaxation-training	Combined treatment reduced more hostile thoughts, SBP & pulse-rate than Hygiene alone, & levels of norepinephrine moderated results	Immediately post-treatment	27
Hagga et al (1994)	43 male borderline Type-A Hypertensives	7 weeks of medical information for reducing hypertension + relaxation or 7 weeks of information alone	Combined treatment reduced SBP & & hostile thoughts more than medical information alone	Immediately post-treatment	32
Peled-Ney et al (1984)	70 Israeli hypertensives, 32 males, 38 females	6 months of weekly group therapy for increasing awareness & expression of anger and negative feelings or no treatment control	D/SBP were reduced only in group therapy & to a greater extent than in controls during & after therapy, with maintenance after 3 & 12 months	Immediately & 3 & 12-months post-treatment	26

[a] These methodological ratings were based on the Check-list for statistical review of papers on clinical trials for the British Medical Journal (Gardner et al , 1989) Scores range from 1 to 45

Previous relevant psychotherapeutic trials with cardiac patients

Psychotherapeutic clinical trials conducted with cardiac patients have either targeted the psychological sequelae of CHD (e.g., depression, life-dissatisfaction) or the hypothesized psychological risk factors for CHD (e.g., the TABP). Whereas the former attempt to improve patients' prognosis by facilitating their rehabilitation after cardiac events (e.g., Rahe et al., 1979), the latter attempt to improve patients' prognosis by reducing their risk for recurrences via risk-factor modification. Since the proposed new treatment focusses on modifying hostility, a CHD risk factor (Smith, 1992), most of the trials reviewed below are trials which targeted psychological CHD risk-factors, particularly those related to hostility (e.g., the TABP) or assessed its modification. The subject characteristics, results and methodological evaluation of these trials appear in Table 8.

Stern, Gorman and Kaslow (1983) randomly assigned post-MI patients who had a low work-capacity and/or scored high on anxiety or depression to either an exercise therapy (rhythmic movements), a psychotherapy (education on risk factors, and Type-A and hostility modification) or a usual treatment control group. Exercise patients increased their working capacity significantly more than the other two groups, and reduced their anxiety, fatigue, depression and dependency more than controls. Psychotherapy patients showed short- and long-term reductions on depression and increased their friendliness significantly more than both other groups, and reduced their interpersonal

friction more than controls. Finally, no group differences were found on mortality rates, sexual functioning or return to work. However, the statistical presentation of tests mixed comparisons of change scores with comparisons of follow-up scores between groups. Additionally, although hostility was targeted in the psychotherapy group (in addition to the TABP), it was not directly measured, thus, it is unknown whether hostility levels were altered.

Friedman et al. (1986) conducted the most theoretically and methodologically sound clinical trial in the field of coronary-prone behavior (the Recurrent Coronary Prevention Project; RCPP). They showed that a combined cardiac counseling and Type-A modification treatment yielded significantly lower recurrence rates (non-fatal MI and cardiac death) than a cardiac counseling control group. Among subjects who reduced their Type-A behavior in the combined group, there were significantly fewer MI cases (6.6%) than among controls who did not alter their TABP (17.2%). Since no other risk factors were altered at the same time, this important finding suggested a **causal** relation between the TABP and CHD. However, it is possible that change in hostility, the toxic component of TABP (Williams, 1987), was responsible for this causal relation. The psychological component of their treatment was a multidimensional one, aimed at altering the global TABP. Mendes De Leon, Powell and Kaplan (1991) examined the specific psychosocial parameters altered by the RCPP. They found that after controlling for base-line levels and recurrent MI events during treatment, observed hostility and self reported anger

and depression were among the specific psychological parameters altered to a greater extent by the combined treatment than by the control treatment. Finally, they demonstrated that a dose-response relation between number of sessions attended and change in psychological measures was found only in the psychotherapy group. Thus, type and amount of therapeutic contact were important factors in promoting change. The main limitation of the RCPP is that patients underwent behavior modification for 4.5 years. Such a long-lasting treatment may not be practical for all cardiac patients, and results in high expenses as well. Despite this limitation, this study provided strong evidence for the role of psychological interventions in secondary prevention of CHD

Razin, Swencionis and Zohman (1986) pilot tested the effects of a cardiac stress management training focussing on stress-reduction and anger. The treatment was offered to patients and their spouses. Subjects also viewed the SI and concomitant elevations in cardiovascular reactivity, a therapeutic feature unique to this study. Their treatment significantly reduced reactive-SBP, SI-derived PH, and self-reported situational anger, depression, anxiety and somatization. However, this study had many methodological problems: No control group, results included data of healthy spouses, and therapy groups included too many subjects (8-12).

Ornish et al. (1990) tested whether CAD patients can modify and sustain a comprehensive change in their life style (vegetarian diet, stress-management and social support, physical exercise and smoking cessation), and what effects

these modifications had on degree of atherosclerosis. Although this study did not target or assess hostility modification, the treatment included stress-management and communication skills, both previously shown to reduce hostility (Deffenbacher et al., 1987). The experimental group yielded greater reductions in to. - and LDL-cholesterol, weight, and duration of chest-pain, and greater adherence to life style changes than a usual-care control group. Most important, the experimental group yielded reductions in degree of coronary artery stenosis compared with progression in the control group. Considering only clinically significant stenoses (> 50%), the experimental group **regressed** on average from 61.1% to 55 8% stenosis and the controls progressed from 61.7% to 64.4% stenosis, and these changes were significantly different from each other. In the experimental group, 82% of subjects showed a regression in atherosclerosis compared to 42% in the control group Finally, degree of adherence was strongly related to change in stenosis, suggesting a **causal** relation between adherence to life style change and atherosclerosis. However, it is impossible to know the relative effect of the components of this multidimensional treatment, and base-line differences in risk factors (e.g., HDL-cholesterol) were not controlled for in the analyses. Despite these limitations, this important study suggested that life style changes, which are essentially a behavioral change (Jenkins, 1988) and which included aspects that can reduce hostility (relaxation, communication skills), may stop and even reverse CAD.

Burell et al. (1994) replicated some of the findings of Friedman et al

(1986) with Swedish post-MI men. Patients receiving a combination of Type-A modification and cardiac counseling showed significantly greater reductions on observed global TABP and hostility, TABP as rated by their spouses, and lipid levels, and increased their walking capacity more than patients receiving cardiac counseling alone. Cardiac recurrences tended to be fewer in the combined group one year post-treatment. This study used a shorter, and thus, more feasible, therapy compared with Friedman et al. (1986) and used spouse ratings as well. In addition, Burell et al. (1994) aimed at reducing hostility and time-urgency levels within the TABP.

A very recent pilot study wished to examine the feasibility of stress-management (SM) with post-MI patients and patients awaiting coronary-artery-by-pass-grafting (CABG; Turner, Linden, van der Wal & Schamberger, 1995). Stress-management included modification of Type-A behavior, expression of hostility and anger, self-talk, improvements in communication, use of humor and relaxation. This study found that patients receiving SM and exercise-rehabilitation (ER) showed strong changes in self-reported distress, small changes in self-reported hostility (Ho scale) and strong changes in reactive SBP. Controls receiving only ER changed on resting-DBP, but worsened on triglycerides and HDL-cholesterol. Although the SM program more specifically targeted hostility, it included Type-A modification as well. Large participant drop-outs did not enable use of inferential statistics, and more valid self-report and observed hostility measures (i.e., Barefoot's refined Ho; SI-derived PH)

could have been used.

These trials suggest that psychological interventions have statistically as well as clinically meaningful effects on CHD morbidity and mortality (e.g., Friedman et al., 1986; Ornish et al., 1990). Although none of these trials targeted hostility alone, changes in psychosocial outcomes (anxiety, hostility, depression) and health outcomes (reinfarct, lipid levels) were observed. Given that psychotherapy yielded statistically significant effects with sample sizes smaller than those typically used in medication trials, psychotherapy may be more effective than medication therapy for CHD patients.

Support for this "bold" suggestion comes from a recent study by Davidson, Gidron and Chaplin (1995). They compared statistically significant (i.e., "p" levels) and clinically significant effects (i.e., number of MIs or deaths prevented by experimental group/100 patients) of psychotherapeutic, medication and cardiac-rehabilitation (mixed) treatments. The psychotherapeutic treatments were all eight published trials with reinfarct and/or mortality as a dependent variable. Medication and mixed treatments were randomly selected from larger meta-analytic reports of respective clinical trials. As expected, a higher percentage of medication trials (21%) yielded highly statistically significant effects (at least $p < .01$), compared to psychotherapy (8%) and mixed (0%) trials. Medication trials included on average, 19 times more subjects than psychotherapeutic trials. In sharp contrast, psychotherapeutic trials prevented actual MI and deaths 10 times and three times more,

respectively, than did medication trials. Mixed and medication trials yielded similarly small clinically significant effects (Davidson, et al., 1995). Thus, psychological trials that focus on significant psychological CHD risk factors alone (i.e., hostility) may yield even greater clinically and statistically significant effects (Dembroski & Costa, 1987; Williams et al., 1980). The last section of this chapter develops the rationale for the proposed focussed hostility-reduction treatment tested in this thesis.

Table 8
Study characteristics, results and methodological-evaluation of relevant trials with CHD patients

Author	Sample	Treatment & Targets	Results	Follow-Up	Method
Burell et al (1994)	49 Swedish post-MI men with moderate cardiac impairment	35 sessions of cardiac counseling & Type-A modification or 6 sessions of cardiac counseling for reducing the TABP, CHD risk factors & recurrence of MI	Combined treatment reduced more observed TABP & hostility, spouse-rated TABP, lipids, & increased walking more than control group	1 & 12 months post-treatment	38[a]
Friedman et al (1986)	862 post-MI patients, 791 males, 71 females	34 sessions of cardiac counseling or 84 sessions of cardiac counseling & Type-A modification for reducing TABP & cardiac recurrence	Combined treatment yielded fewer recurrences, greater reductions of TABP, hostility, anger & depression than did the control group	4 5 years	38
Ornish et al (1990)	41 CAD patients, 36 males, 5 females	Comprehensive life style change treatment over 1 year including diet, physical exercise, stress-management & smoking-cessation	Experimental treatment yielded more reductions in cholesterol, weight, chest-pain & showed **regression** in coronary stenosis compared with CAD-progression in control group	15 months	34
Razin et al. (1986)	34 CHD & healthy spouses	10 weekly sessions of stress & anger management for reducing TABP, its components & HR & BP-reactivity	Significant changes in reactive-SBP, SI derived PH, & self-reported anger, depression & anxiety	Immediately post-treatment	28
Stern et al (1983)	103 post-MI, low on work-capacity &/or anxious or depressed	12 weeks of exercise, psychotherapy or usual-treatment control, for increasing work capacity & reducing anxiety & depression	Exercise increased work-capacity & reduced anxiety Psychotherapy reduced depression & interpersonal friction	3, 6 & 12-months post treatment	34
Turner et al (1995)	24 post-MI or or CABG patients, 19 males, 5 females	8 weeks of Exercise rehabilitation (ER) or ER + stress-management (SM) for reducing distress hostility, cortisol, cholesterol, catacholamines	ER + SM strongly changed distress & reactive-SBP & mildly changed hostility ER alone changed resting-DBP & worsened on reactive-DBP, HDL cholesterol & triglycerides	1 month post-treatment	34

[a] These methodological ratings were based on the Check-list for statistical review of papers on clinical trials for the British Medical Journal (Gardner et al , 1989) Scores range from 1 to 45

New proposed hostility-reduction treatment

The last section of this chapter describes the rationale for the new proposed hostility-reduction treatment, its therapeutic approach, technical considerations and content. A therapeutic manual is being revised for separate publication, and will contain this information in greater detail. The tests of the efficacy of this treatment in healthy but high-hostile young males and in high-hostile CHD patients will be presented in Chapters 6 and 7, respectively.

Rationale for the proposed new hostility-reduction treatment. The proposed hostility-reduction treatment was derived from the definition of CHD-predictive hostility, from the relations known to exist between its components, from the theoretical and empirical literature linking hostility with CHD, and from previous clinical trials and literature. Thus, the treatment is theoretically and empirically driven, and was developed to fulfill the unanswered need to focus on hostility-modification alone with CHD patients (Chesney, 1985, Dembroski & Costa, 1987; Williams et al., 1980).

Hostility is defined as the stable tendency to behave antagonistically, think cynically and attribute hostility to others, and to feel anger across situations (Barefoot, 1992; Barefoot & Lipkus, 1994). The three dimensions of hostility are theoretically related, since hostile cognitions are thought to underlie or elicit hostile behaviors and emotions (Chesney, 1985; Powell, 1992). Empirically, scores on these three dimensions (behavioral, cognitive, emotional) are moderately but significantly correlated (Barefoot et al., 1989). Conceptually,

modifying one hostility dimension may affect the others. Thus, each hostility component should be assessed separately and focused upon in treatment.

Based on empirical findings, investigators in the field of coronary-prone behavior have theorized that hostility is the most CHD-predictive component of the TABP (Dembroski & Costa, 1987; Williams, 1987). This theorizing answered several major problems with the Type-A-CHD relation as well (i.e., studies showing no relation between Type-A and CHD, and over-representation of Type-A across samples). The review of empirical findings in Chapter 3 showed that in approximately 86% of studies hostility **is** related cross-sectionally or prospectively to different CHD end-points (i.e., CAD, transient ischemia, angina pectoris, MI and CHD-mortality) in several cultures, and in most cases, more strongly related to CHD than the global TABP (e.g., Barefoot et al., 1983; Barefoot et al., 1989; Barefoot et al., in press; Dembroski et al., 1985; Dembroski et al., 1989; Haynes et al., 1980; Koskenvuo et al., 1988; Mendes De Leon, 1992; Powell & Thoresen, 1985; Williams et al. 1980). In several studies hostility predicts CHD independent of traditional risk factors (e.g., smoking, cholesterol, BP; Dembroski et al., 1989; Hecker et al., 1988; Shekelle et al., 1983). Two studies have found that hostility predicts progression of atherosclerosis and CHD (Julkunen et al., 1994; Powell & Thoresen, 1985). Additionally, provocations that specifically elicit anger/hostility result in greater myocardial dysfunction reflecting ischemia than provocations that elicit general distress (Ironson et al., 1992). Episodes of experiences of

anger and expressed hostility may even temporally precede a MI (Mittleman et al., 1995). Finally, some studies suggest that antagonistic or behavioral hostility, hostility not confounded by Neuroticism (Costa et al. 1989), is the most CHD-predictive component of hostility (Dembroski et al., 1989; Siegman, 1993; Siegman et al., 1987). Although the evidence is weaker for hostility's cognitive (cynicism) and affective dimensions (anger), other studies suggest that these components predict CHD and mortality as well (Almada et al., 1991; Barefoot et al., 1989; Barefoot et al., in press; Haynes et al., 1980). Cynicism and anger are important facets of hostility since they do not completely overlap with the personality dimension of Neuroticism, and both are correlated with the personality dimension of Agreeableness versus Antagonism (Barefoot et al., 1989; Costa et al., 1989). Therefore, all three components of hostility, that is, antagonistic behavior, cynical thoughts and angry feelings, were conceptualized in this research as CHD-predictive hostility.

Thus, CHD-predictive hostility is the toxic component of the TABP (Dembroski & Costa, 1987; Dembroski et al., 1989, Williams, 1987). Therefore, hostility should be focused upon in psychotherapeutic interventions with CHD patients, rather than the global TABP (Chesney, 1985; Dembroski et al., 1989; Miller et al., in press). However, none of the treatments described above attempted to target CHD-predictive hostility alone in CHD patients, using a randomized-controlled design. Based on the empirical findings mentioned above and reviewed in Chapters 2 and 3, this new treatment focused on

modifying all three dimensions of CHD-predictive hostility: Antagonistic behavior (e.g., rudeness, argumentativenss), cognitive hostility (e.g., cynicism, mistrust, hostile-attributions) and hostile affect (e.g., anger). All three dimensions follow the conceptualization of CHD-predictive hostility, and all are theoretically and empirically related to each other and to CHD.

Therapeutic approach and strategies. A cognitive-behavioral treatment was chosen as the most suitable treatment for altering hostility. This approach has been used in previous studies on anger-modification with healthy (e.g., Deffenbacher et al., 1987) and hypertensive subjects (e.g., Achmon et al 1989). Modifying hostile cognitions was seen as a central step, since cynicism and hostile attributions are believed to underlie hostile behavior and angry feelings (Chesney, 1985). Modifying hostile misconceptions via cognitive therapy was expected to have a strong impact on individuals' ability to cope with environmental and personal challenges and provocations (Raimy, 1985). Modifying hostile behavior was seen as a central step since the behavioral component of hostility may be the most toxic element of CHD-predictive hostility (Dembroski et al., 1989; Siegman et al., 1987). In addition, modifying interpersonal behavioral hostility via behavior-modification was seen as a basic means for ameliorating a problem which is primarily of an interpersonal nature (Barefoot et al., 1989).

According to the multimodal treatment theory (Lazarus, 1986), treatments should be matched to the psychological dimension they wish to modify. Thus,

for example, behavior therapy should be used to reduce antagonistic hostility, and cognitive therapy should be used to restructure hostile cognitions. However, since hostile individuals may present with any combination or profile of hostility's three dimensions (Barefoot, 1992), since its dimensions are related (Barefoot et al., 1989; Chesney, 1985), and since all three dimensions were considered CHD-predictive, the new hostility-reduction treatment adopted a **multicomponent** approach. This approach was in line with the matching approach of the multimodal theory (Lazarus, 1986), and considered the fact that individuals in group-therapy may have different hostility profiles. Such a multicomponent approach guaranteed that all types of individual profiles would be targeted. Therefore, reducing hostility's behavioral modality, antagonism, was done with a behavioral approach (e.g., communication skills), reducing cynicism and hostile attributions was done with cognitive skills (e.g., altering self-talk), and reducing anger was done with relaxation and problem-focused coping.

The clinical literature and trials discussed above concerning modifying anger, hostility and the TABP provided the specific clinical strategies for modifying the CHD-predictive components of hostility (e.g., Achmon et al., 1989; Deffenbacher, 1994; Deffenbacher et al., 1987; Moon & Eisler, 1983; Novaco, 1976b; Roskies, 1987; Roskies et al., 1986; Thurman, 1985a; Redford B. Williams, Private communications, May, 1993; Williams & Williams, 1993). The main novelty of the proposed treatment is in the combination and

application of previously used skills to the modification of CHD-predictive hostility.

Throughout the entire program, therapists use a didactic and coaching approach towards participants (Roskies, 1987). Therapists employ an accepting, yet, problem-confronting approach with participants (Deffenbacher, 1994; Roskies, 1987). In other words, participants are always accepted in general, however, their specific hostile behaviors or misconceptions are dealt with. The therapists need to be educated about the connection between hostility and CHD, the contents of the sessions, and the skills to be used. Therapists transmit this knowledge, and occasionally provide examples of problems with hostility and daily provocations from their own lives. Thus, the therapists are seen as experts on hostility-modification, but not as people who are free of experiencing hostility or daily provocations. After reviewing the home assignments of each previous session, therapists present a new topic, usually by modeling a problem or a new coping skill. Participants are then asked to identify the problem or skill, and practice it after therapists outline its basic stages. Corrective feedback and group support are provided to participants for trying out new skills (Roskies, 1987). One additional important therapeutic approach adopted during treatment was the "One step below" strategy. This assumes that hostile people feel better and are more cooperative if they are given the feeling that their opinion about the intervention is needed and that they have some control over the program. Thus, therapists

repeatedly stress the value of participants feedback about the intervention and which skills work best, since "this is a newly tested treatment". In addition, at the start of each group, participants who are perceived by the therapists as more cooperative and psychologically oriented are "employed" as models for answering questions and for role-playing. This enhances other members' motivation to cooperate and practice therapeutic skills, and helps channeling group competition to therapeutic gains (Peled-Ney et al., 1984). Finally, hostility-related humor is used throughout the program, as a means for reducing affective hostility, changing misconceptions, and reducing discomfort surrounding certain topics (Deffenbacher, 1994; Dworkin & Efran, 1967; Roskies, 1987).

Technical considerations in the new proposed treatment. Several technical and procedural issues were planned and were based on logical reasoning and previous empirical findings. First, duration of treatment: The treatment consists of eight weekly sessions of 90 minutes each. This number of sessions followed the estimated amount of time and breakdown of sessions according to their contents. This number has been used in previous studies (e.g., Deffenbacher et al., 1987; Thurman, 1985a), and has been shown to yield change in (self-reported) psychological measures in 50% of patients undergoing psychotherapy (Howard, Kopta, Krause & Orlinski, 1986). The duration of 90 minutes/session is in line with Deffenbacher's general guidelines (1994). Second, group therapy: This was done for reducing the resistance to treatment and perceived

threat commonly experienced by patients in individual therapy. Group therapy also provides an interpersonal context for modifying hostility, which is primarily an interpersonal problem (Barefoot et al., 1989). In group therapy, hostile people can learn about their hostility and how to alter it by viewing others (vicarious learning), and they can learn to accept their hostility by knowing that others have similar difficulties. Finally, group therapy is more time- and cost-effective than individual therapy, hence, it may be more feasible in medical settings for the treatment of CHD. Third, number of participants/group is limited to 6-8, as in other studies (e.g., Stern et al., 1983; Turner et al., 1995). This number guarantees a meaningful group interaction and practicing of skills by all participants, and sufficient attention devoted to each participant. Fourth, two therapists lead all sessions. This provides a means for modeling interpersonal daily provocations, hostile reactions and non-hostile solutions. This also allows one to model solving actual disagreements and different perspectives that emerge between people (therapists). The segments within a session are introduced alternatively by the therapists, to share the therapeutic work, and to reduce boredom.

The content of the new hostility-reduction treatment. The new treatment is highly structured, with each session fully detailed in the manual, and each segment conducted with precise timing. This was done to standardize the treatment as much as possible across groups and for future research. Table 9 presents the content, learned skills and home-assignments of each session.

The program includes five sections: 1. Introduction (session 1); 2. Behavioral hostility (sessions 2-3); 3. Cognitive hostility (sessions 4-5); 4. Affective hostility (sessions 6-7); 5. Summary and relapse prevention (session 8).

The behavioral section precedes the cognitive and affective sections for the following reasons: a) It was thought that coronary patients, who do not commonly have much insight into their thoughts or feelings (Rosenman, 1978), would find it easier to begin monitoring and altering the more concrete dimension of behavior; b) Since hostility is primarily an interpersonal problem (Barefoot et al., 1989), beginning with monitoring and altering overt interpersonal hostility (i.e., behavior) seemed most important; c) Behavioral hostility (antagonism) may be the most toxic element of hostility in relation to CHD (e.g., Dembroski et al., 1989); and d) In line with cognitive dissonance theories, modifying hostile behavior is expected to be followed by altered hostile cognitions and attitudes (e.g., Aronson & Mills, 1959). Alteration of affective hostility (anger) is done after five weeks of group therapy, since by this stage in treatment, participants are expected to establish sufficient rapport with other participants and with the therapists in order to disclose and change their angry feeling.

Session 1 is an introductory session and an attempt to increase participants' interest and motivation to participate. After group "rules" are mentioned (e.g., not interrupting a person who is speaking, group confidentiality) and participants introduce each other, participants are asked to

view and rate the differences between an actor exhibiting high and low hostility from the SI (Razin et al., 1986). Session 1 then provides a comprehensive educational component of the health consequences of hostility with particular emphasis on CAD and CHD. After participants write down their expectations from the treatment, they learn deep-breathing skills, and Progressive Muscular Relaxation (Craske et al , 1992), and learn how to monitor their own general reactions to stress Session 2 and each subsequent session begin with reviewing home-assignments Participants learn how to monitor antagonistic hostility (e.g , aggression, rudeness), and the counteractive skills of smiling, listening and tolerance Session 3 teaches participants the Hostility Road Map (Williams & Williams, 1993), as a means of conceptualizing the order of events and reactions around provocations Participants then learn how to use assertiveness training (Williams & Williams, 1993) as a third option and as means of moderating both explosive and suppressive hostility (Anger-Out Anger-In, respectively Roskies, 1987, Siegman, 1993) These are taught in gradually increasing intensity, from mild assertions (making a simple assertive statement) to more demanding behavior (stating the consequences). In session 4, participants learn how to monitor hostile cognitions (hostile attributions, mistrust and cynicism), and learn the connection between hostile thoughts, feelings and behaviors in the context of provocations (Chesney, 1985). In session 5, participants learn to use the counteractive skills of thought-stopping, changing negative and hostility-producing self-talk statements, and practice

sharing control and one's feelings (Novaco, 1976b; Williams & Williams, 1993). Although sharing one's feelings with others may increase social support as well, this skill was used to provide participants with a reality test of their mistrusting beliefs (e.g., "Was I hurt after confiding in my spouse?"), and as a way to regain trust in close people. In addition, believing that one has a confidant, thus believing that one can trust another close person, has been shown to predict fewer rehospitalizations and less chest-pain after a MI (Helgeson, 1991). Session 6 teaches participants about the causes, functions and identification of anger and its physiological signs (e.g., Ben-Zur & Breznitz, 1991; Novaco, 1976b). Session 7 teaches the counteractive skills of distraction, relaxation and remaining problem-focused (Novaco, 1976b). Finally, session 8 reviews the entire program and participants learn relapse-prevention skills. The session ends with participants providing their feedback about the program (e.g., most useful and useless skills), and with therapists providing general feedback.

An essential part of the treatment is the home-assignments, which are written logs and actual practicing of skills (Deffenbacher, 1994). This provides participants the opportunity to review the material from previous session/s, and to monitor their hostile behavior, cognitions and emotions, and use of hostility-reducing skills "in vivo" (Roskies, 1987). Home assignment is seen as an additional "self-administered" treatment, and as an essential path for inducing enduring changes in hostility. Participants receive handouts summarizing each session, and a log for entering daily their experience of the hostility dimension

targeted in the last session, and the use of counteractive skills. At the beginning of each session, subjects receive corrective feedback for reporting their experiences and use of skills from the previous week (Roskies, 1987).

Table 9

Outline of sessions[a]

Session number and content	Exercises	Homework
Section 1: INTRODUCTION		
1. Familiarization and motivation: Detecting hostility in others (actors), learning hostility's health consequences, stating expected goals, learning Progressive Muscular Relaxation, how to do homework.	Examine actor's reactions; State your goals; practice relaxation	Monitor own reactions to stress & relaxation
Section 2: BEHAVIORAL HOSTILITY		
2. Check homework. Antagonistic behavior: Condescension, aggression, rudeness. Skills: Smiling, listening, tolerance.	State own reactions; practice listening & tolerance	Monitor Ho antagonism & skills
3. Check homework. Suppression & explosive-reducing skills: Assertion. Williams & Williams' (1993) Hostility Road Map.	Act gradual use of assertion skills	Rate Anger-In/Out; use of skills
Section 3: COGNITIVE HOSTILITY		
4. Check homework. Hostile thoughts: Mistrust, hostile attributions, cynicism. Chesney's (1985) model.	Learn Anger Model; Write trust-list	Monitor cognitive Ho
5. Check homework. Mistrust-reducing skills: Sharing feelings & control, alter provoking self-talk, and use thought-stopping.	Practice sharing, changing self-talk & thought-stopping	Monitor cognitive Ho & skills
Section 4: EMOTIONAL HOSTILITY		
6. Check homework. Hostile affect: Anger's origins, identifying our anger & its signs.	Anger-list; imagine & detect physical signs	Monitor anger's frequency' duration & intensity
7. Check homework. Anger-reducing skills: Distraction, relaxation and remaining problem-focused.	Practice remaining problem-focused	Monitor anger & skills
Section 5: SUMMARY		
8. Check homework. Summary and relapse-prevention: Summarize treatment. Skills: "Pretend it's your last day", self reinforcement & combine skills.	Imagine relapse; participants provide feedback	

[a] Ho refers to hostility/hostile.

CHAPTER SIX

STUDY 1: THE EFFECTS OF THE HOSTILITY-REDUCTION TREATMENT ON CHD-PREDICTIVE HOSTILITY LEVELS OF HIGH-HOSTILE HEALTHY STUDENTS

Introduction

Hostility is the tendency to behave antagonistically, think cynically and feel anger (Barefoot, 1992). Hostility has become the main focus of attention in recent research in behavioral cardiology, since it has emerged as the most toxic component of the Type-A Behavioral Pattern (TABP) in relation to several coronary heart disease (CHD) end-points (e.g., Dembroski et al., 1989; Williams et al., 1980). As reviewed in Chapter Three, hostility has been shown to be related to daily ischemic episodes (e.g., Helmers et al., 1993) and to induce greater ischemia than general distress (Ironson et al., 1992). Hostility is significantly related to coronary artery disease (CAD) in cross-sectional designs (e.g., Dembroski et al., 1985, MacDougall et al., 1985; Williams et al., 1980) and to CHD-incidence, progression and mortality in prospective studies (e.g., Barefoot et al., 1983; Barefoot et al., 1989; Barefoot et al., in press; Carmelli et al., 1991; Dembroski et al., 1989; Hecker et al., 1988; Powell & Thoresen, 1985). Finally, hostility predicts CHD, after controlling for traditional CHD risk factors such as age, smoking, cholesterol level and blood pressure (BP; e.g., Dembroski et al., 1989; Hecker et al., 1988; Shekelle et al., 1983).

Several studies have failed to support these associations (e.g., Hearn et

al., 1989; Leon et al., 1988; McCranie et al., 1986). However, from an assessment perspective, these studies did not assess hostility with the most valid measures. All three negative studies used the full Ho scale, which may be less predictive of CHD as it contains items that assess both Neuroticism (i.e., psychological distress) and hostility (Barefoot et al., 1989). As explained in Chapter 2, measures that assess Neuroticism have been shown to be unrelated to objective health outcomes such as CHD (Watson & Pennebaker, 1989). Additionally, limitations related to the assessment context (i.e., completing the Ho scale as part of a medical school entry exam; McCranie et al., 1986) may explain one of the findings.

Several studies have began to identify dimensions within the hostility construct and subscales within hostility measures that predict CHD better then others. Using the Cook and Medley (1954) Ho scale, Barefoot et al. (1989) showed that the summed score of three conceptually-derived subscales, Aggressive-Responding, Cynicism and Hostile Affect, was a better predictor of mortality than was the overall Ho score. Helmers et al. (1993) confirmed these results with respect to ischemia, and a similarly refined Ho scale, which additionally included Ho items assessing Hostile Attributions, was recently found to predict CHD-incidence for both males and females in a prospective Danish population study (Barefoot et al., in press). However, Hearn et al. (1989) did not find a prospective association between Barefoot's refined Ho scale and CHD over a 33-year follow-up. This may have been due to selection of

relatively young subjects at base-line (mean age = 18.9 years), where developmental changes may have made the assessment of hostility unstable. Additionally, CHD included indefinite cases, without diagnostic tests, possibly reducing the validity of the outcome measure. Barefoot's refined Ho scale includes items that assess mainly the Antagonism dimension of personality, and only minimally assess the Neuroticism dimension of personality (Barefoot et al., 1989), and this increases its construct validity (Barefoot, 1992) and predictive validity (Watson & Pennebaker, 1989).

Siegman et al. (1987) derived from the Buss-Durkee Hostility Inventory (BDHI; Buss & Durkee, 1957) an expressive or antagonistic hostility factor (including the Physical Assault, Verbal Hostility and Indirect Hostility subscales) and an experiential or neurotic hostility factor (including the Suspicion and Resentment subscales). While expressive hostility was significantly and positively related to CAD-severity, experienced hostility was significantly and negatively related to CAD-severity (in men younger than age 60). This demonstrates again the need to separate aspects of antagonistic hostility from those of neurotic hostility (Siegman, 1993).

Using the Structured Interview (SI; Rosenman, 1978), Dembroski and Costa (1987) divided the global Potential for Hostility (PH) rating into Hostile-Style (i.e., expressed interpersonal antagonism), Hostile Content and Hostile Intensity (i.e., frequency and degree/emphasis of experienced hostile affect, respectively). Dembroski et al. (1989) showed that total PH ratings and

particularly Hostile-Style significantly predicted CHD incidence.

Finally, Anger-Out, another measure of antagonistic behavior, and related to the mode of anger-expression (i.e., exploding versus suppressing) may also be important. Self-reported Anger-Out, assessed by the Anger-expression scale (AX: Spielberger et al., 1985) has been significantly associated with unstable angina and myocardial infarction (Mendes De Leon, 1992) and SI-derived Anger-Out has been significantly and prospectively associated with CHD-incidence (Matthews et al., 1977).

Thus, hostility measures that assess behavioral hostility, and that are related more strongly to Antagonism than to Neuroticism, appear to have greater predictive validity in relation to CHD. While behavioral hostility reflects the personality dimension of Antagonism alone, both cognitive hostility (cynicism, hostile attributions) and affective hostility (anger) reflect Antagonism **and** Neuroticism (Barefoot et al., 1989; Costa et al., 1989). Despite this, cynicism and anger have also been found to be significantly related to CHD and mortality (e.g., Almada et al., 1991; Barefoot et al., 1989; Barefoot et al., in press; Kneip et al., 1993). Furthermore, cynicism, anger and antagonistic behavior are conceptually related (e.g., Chesney, 1985), and scores on all three hostility dimensions have been shown to be empirically related to each other (Barefoot et al., 1989). Thus, the hostility components of antagonism, cynicism and anger were regarded in this research as the CHD-predictive components of hostility, with antagonistic behavior potentially being the most toxic of the three

(Siegman, 1993). Following these theoretical and empirical findings, the next logical step in understanding the relation between hostility and CHD, and in reducing risk of CHD would be to **target** CHD-predictive hostility in an intervention (Chesney, 1985; Dembroski & Costa, 1987; Miller et al., in press).

As outlined in Chapter Five, several studies have attempted to modify anger and hostility with different therapeutic approaches (e.g., Deffenbacher et al., 1987; Hazaleus & Deffenbacher, 1986; Moon & Eisler, 1983). However, these studies suffered from methodological limitations. Deffenbacher et al. (1987) and Hazels and Deffenbacher (1986) did not employ objective measures of hostile behavior, and Moon and Eisler (1983) used observational measures that have not been related to CHD. Both self-reported (e.g., Barefoot's Ho) and observed (e.g., PH) measures of CHD-predictive hostility should be used (Costa et al., 1989). In addition, these treatments were not conceptualized nor designed to target "coronary-prone" psychological risk factors, and did not comprehensively educate their subjects about the hostility-CHD relation.

Other studies with healthy subjects (e.g., Eysenck & Grossarth-Maticek, 1991; Roskies et al., 1986; Thurman, 1985a) that targeted coronary-prone constructs, did not focus on CHD-predictive hostility alone. Roskies et al. (1986) and Thurman (1985a) targeted the TABP. Although Eysenck and Grossarth-Maticek (1991) aimed at modifying the TABP with particular emphasis on hostility, they aimed at modifying cancer-prone behaviors (i.e., helplessness) as well, and change in personality was not assessed.

Finally, Williams and Williams (1993) developed a hostility-reduction intervention which targeted and was conceptualized to alter CHD-predictive hostility. Although this treatment provides important information on hostility-modification, it has never been systematically tested with a randomized-controlled design.

Since hostility is the most CHD-predictive component of the TABP (Dembroski et al., 1989; Williams et al., 1980; Williams, 1987), focussing upon hostility and its CHD-predictive components as therapeutic targets rather than the overall TABP may be more effective in preventing CHD in coronary-prone individuals, and this has been advocated by others (e.g., Chesney, 1985; Dembroski & Costa, 1987). This may also allow one tc examine which of the components of CHD-predictive hostility are modifiable.

Thus, the purpose of Study 1 was to systematically test the efficacy of the proposed hostility-reduction intervention outlined in Chapter Five at reducing CHD-predictive hostility levels. In addition, this study tested the efficacy of the hostility-reduction treatment at reducing reactive-BP, since cardiovascular reactivity has been suggested to link hostility with CHD (Williams et al., 1985). By testing the treatment first on healthy, but, high-hostile students, it was possible to examine the effects of the intervention on hostility without any confounds imposed by CHD (e.g., limited adherence due to illness). Finally, this study wished to improve the methodology of previous clinical trials by employing the following steps: a) A matched-randomized-controlled design

(e.g., Eysenck & Grossarth-Maticek, 1991); b) Reliable and valid measures of self-reported and observed CHD-predictive hostility (Costa et al., 1989); c) All measures were administered and rated by experimenters who were blind to subjects' group status (single-blind) to reduce any potential biases (e.g., demand characteristics).

It was hypothesized in general that subjects receiving the experimental hostility-reduction treatment would show greater reductions on hostility and reactive-BP than subjects assigned to an information-control group. Specifically, scores on Barefoot's refined Ho scale, SI-derived PH and Anger-Out, and levels of reactive-SBP and reactive-DBP were expected to be reduced to a larger extent by the experimental hostility-reduction treatment than by the information-control group.

Method

Subjects. One hundred and twenty-eight male university students and employees at Dalhousie University, Halifax, Nova Scotia, Canada, were assessed for demographic and health status, hostility and personality. As detailed below, only data regarding background variables (e.g., gender, cardiac health) and hostility levels were considered for inclusion into this trial.

Inclusion criteria for entry into the clinical trial. a) Males only (as hostility has been shown to be related to CHD primarily in males; Smith, 1992); b) Scoring 3 or above on ratings of PH **and/or** a score of 18 or higher on the Ho scale; c) Without reported CHD-related illnesses (e.g., hypertension; heart-

disease); d) Without reported psychiatric disorders (e.g., depression, personality disorders). A PH cut-off point of 3 was chosen as its descriptive ratings implied an antagonistic style (i.e., "two or three hostile statements and some hostility in voice"), and this cut-off point has been prospectively related to CHD (Dembroski et al., 1989). The Ho cut-off point of 18 was chosen as it was the mean Ho score of a student-sample in our lab (Davidson & Hall, 1995), and as it was greater than a cut-off point previously associated with CAD (i.e., 10; Williams et al., 1980). These cut-off points were chosen to guarantee that the trial's entire sample will be relatively high on hostility from a statistical perspective as well as a clinical perspective related to CHD. Using one of the two cut-offs guaranteed that a sufficient number of candidates will be initially identified for the trial. This also guaranteed a heterogeneous sample in relation to hostility, as subjects may be high on cynical hostility (the main construct assessed with the Ho scale; Smith & Frohm, 1985) and/or high on antagonistic hostility (the main construct assessed with PH; Musante et al., 1989).

After screening for hostility, reported psychiatric disorders and CHD-related diseases, 71 high-hostile males were invited to participate in a new stress-management study. Twenty-two subjects agreed to participate. According to power analyses performed prior to the study, this number was sufficient to detect a medium effect size of approximately 18 to 22% change (i.e., a difference of four points on the Ho scale or a difference of .5 to 1 point on PH) with a power of .80. Forty-nine could not be located, or refused to take

part in the study, in most cases due to time constraints related to academic course load. Unfortunately, the precise number of candidates not located or not agreeing to participate in the trial and the reasons they provided were not recorded. No significant differences were found for either Ho scores of participating ($\underline{M}$ = 23.1) and non-participating subjects ($\underline{M}$ = 22.8; t(65) = .15 p > .05) or for PH scores of participating ($\underline{M}$ = 2.9) or non-participating subjects ($\underline{M}$ = 3.0; t(62) = -.89, p > .05). Thus, although only 31% of tnal candidates were enrolled in the study, they are representative of the non-participating candidates with respect to hostility levels.

Measures

Background variables. The following background data were of interest in this study: Subjects' age (in years), education (in years) and physical activity (in Kilocalories/week). These parameters have been related to CHD (Jenkins, 1988), and were seen as basic CHD risk-factors for ensuring equality between groups. Physical activity was assessed with a four-item self report measure, The Physical Activity Index (Paffenbarger, Wing & Hyde, 1978). This measure asks subjects to indicate the number of flights climbed, blocks walked, and amount of light and strenuous sports engaged in, during the past week. The items are weighed, such that number of flights are multiplied by 28, blocks by 56, light sports by 300 and strenuous sports by 600. The predictive validity of this index has been supported by demonstrating that it is prospectively related to the occurrence of myocardial infarctions (Paffenbarger et al., 1978).

Observed hostility. Observed ratings of hostility were assessed with the Augmented Structured Interview (ASI; Wright and Schmidt-Walker, 1990). This 12-15 minute, videotaped interview was administered by a person trained in eliciting Type A behaviors and hostile reactions in subjects by altering the pace of questions asked during the SI. The male interviewer asked the questions in a business-like, task-oriented manner, without engaging in confrontation with the subject. A non-confrontational SI has been shown to yield Type-A ratings that have greater construct and predictive validity than a confrontational SI (Houston et al., 1988). One male coder, who was well informed about coronary-prone behavior and its assessment, rated all screening and post-treatment interviews for total-PH. The inter-rater reliability and predictive validity of this measure in relation to CHD have been previously demonstrated (e.g., Dembroski et al., 1989). For Study 1, inter-rater reliability for PH was established by the ratings of six coders who coded 60 of the screening interviews (30 males, 30 females) conducted for selecting trial candidates (as part of another study on the effects of coder-gender on reliability; MacGregor & Davidson, 1994). Using these coders as items yielded an internal consistency reliability coefficient of Alpha = .79 for PH, indicating moderately high inter-rater reliability.

The overt behavioral manifestations of hostility may be the most toxic component of hostility in relation to CHD progression (e.g., Siegman, 1993). Thus, it was important to include a reliable measure of expressed hostility.

Coders used the Anger-Expression scale (AX, Spielberger et al., 1985) to assess subjects' expressed anger (Anger-Out) as reported and expressed during the SI. Although the AX is usually used as a self-report measure (e.g., Mendes De Leon, 1992), in the current study it was used as an observational measure, such that coders observed the SI and then completed the AX for that subject. This was done in order to achieve greater reliability with an eight-item measure of Anger-Out than that obtained from single-item observational measures of anger-expression previously used (e.g., Matthews et al., 1977) and to increase sensitivity for detecting possible therapeutic changes. One female coder, informed about the conceptualization of anger-expression, rated all interviews for Anger-Out. Anger-Out was operationalized as relatively extreme manifestations of expressed hostility (e.g., slamming doors, making nasty remarks), and was coded mainly according to what subjects reported during the SI. The item internal consistency reliability level for Anger-Out in this study was sufficient (Cronbach's Alpha = .83). Inter-rater reliability in this study was established by a second coder who rated all interviews of subjects participating in the trial at pre-treatment. Inter-rater reliability as measured by a correlation coefficient was high (.83) for SI-derived Anger-Out. Ratings of SI-derived PH and Anger-Out were positively and significantly correlated, $r(21) = .40$, $p < .05$, supporting the construct validity of this new assessment of Anger-Out.

The two coders who assessed PH and SI-derived Anger-Out at pre- and post-treatment were blind to subjects' group status throughout the entire

protocol, and were unaware of subjects' self-reported hostility scores.

Self-reported hostility. Self-reported hostility was assessed with the Cook and Medley (1954) Ho scale. This scale consists of 50 statements to which subjects respond either true or false. Although initially developed to distinguish among teachers with good versus poor rapport with students, the construct validity of this scale as a measure of hostility has been supported (Pope et al., 1990; Smith & Frohm, 1985). The Ho scale has been shown to have high test-retest reliability over one and four years and has been shown to predict CHD and mortality (e.g., Barefoot et al., 1983, Shekelle et al., 19 However, the main focus of attention was placed upon subjects' scores on Barefoot's refined Ho scale, since the conceptualization of CHD-predictive hostility and the proposed treatment are in line with this refined scale and since it is more predictive of CHD and mortality than is the full Ho scale (e.g., Barefoot et al., 1989). Barefoot's refined Ho scale includes the total score from 27 items comprising the Aggressive-Responding, Cynicism and Hostile Affect subscales (Barefoot et al., 1989). In the present study, the internal consistency reliability of Barefoot's refined Ho scale at pre-treatment was moderately high (Cronbach's Alpha = .79).

Cardiovascular reactivity. This measure included ratings of reactive-SBP and reactive-DBP in the context of the SI. Since the SI does not only inquire about daily provocations, but, mimics them as well, it may be a suitable tool for measuring cardiovascular reactivity related to challenges (Chesney,

1985). Blood pressure was measured with an Oscillometric Spacelabs 90207 ambulatory monitor (Redmond, WA), positioned on subjects' non-dominant arm. Subjects had their resting-BP monitored manually four times prior to the SI, at 2, 4, and 4 minute intervals, respectively. The mean of the last two readings formed the resting-SBP and resting-DBP scores, as has been done in previous studies (e g., Dembroski et al., 1979). Three more BP readings were taken automatically, one at minute six during the SI (assumed peak reactivity), one at the end of the SI (minute 12) and a final one six minutes after the SI (recovery period) Cardiovascular reactivity was defined as the percentage of change from resting-BP to that measured at assumed peak reactivity (e.g., 100 x [SBP at minute 6 - mean resting SBP / mean resting SBP], Roskies et al., 1986). Although absolute difference scores may reflect more meaningfully the impact of a psychological stressor (the SI), the percentage of change was chosen to facilitate comparisons across time within and between groups. Additionally, absolute changes in BP (e.g., 10 mm Hg) may be clinically more meaningful for measuring resting-BP thar for measuring transient reactive-BP, as deviations from certain resting levels are associated with disease states such as hypertension. The predictive validity of the measure of reactive-BP in the context of the SI has been supported by showing that post-MI subjects exhibited significantly higher reactivity levels than non-CHD controls during the SI (Dembroski et al., 1979). In the current study, subjects' reactive-SBP and reactive-DBP were significantly and positively correlated with each other, r(22) =

.44, $p < .05$, supporting the construct validity of the reactivity measures.

Hostility-modification treatment. Chapter Five explained in detail the rationale and content of the proposed hostility-reduction treatment. Briefly, a cognitive-behavioral intervention was developed for modifying individuals' CHD-predictive hostility levels of antagonistic behavior, cynical cognitions and angry feelings. Based on theoretical and empirical findings relating these components to CHD (e.g., Almada et al., 1991; Barefoot et al., 1989; Dembroski & Costa, 1987; Dembroski et al., 1989), the treatment attempted to modify all three components of hostility, with particular emphasis on antagonistic behavior (Siegman, 1993). The clinical techniques for modifying these hostility dimensions were based on previous clinical literature on anger and Type-A modification (e.g., Deffenbacher et al., 1987; Novaco, 1976b; Roskies, 1987; Williams & Williams, 1993).

The intervention consisted of eight, 90-minute, weekly meetings, and daily monitoring of hostility and use of skills. The treatment was divided into five sections: An introduction (session 1), monitoring and altering antagonistic hostility (sessions 2-3); monitoring and altering cynical hostility (sessions 4-5); monitoring and altering angry feelings (sessions 6-7); and summary and relapse prevention (session 8). The sessions were highly structured in order to maintain experimental control as much as possible. The content of modeling and role-playing used during sessions was appropriate to students' age and daily life (e.g., disputes between room-mates; waiting for a tardy friend).

Information-control group. Control subjects received one group session about the CHD risks of hostility (e.g., hostility is related to smoking and drinking, CAD and CHD) and basic skills for modifying hostility (e.g., relaxation, listening, assertiveness). This group served as an information-control condition, attempting to control for several non-specific therapeutic effects (e.g., attention to a problem, receiving information, group-contact and meeting therapists; O'Leary & Borkovec, 1978; Thurman, 1985a). Providing this information to "at-risk" subjects (i.e., healthy, but hostile individuals) was also important for ethical reasons.

Both treatment and control sessions were conducted by a female resident in clinical psychology (Mrs. Maureen Sullivan) and a male Ph D student in experimental health psychology (Yori Gidron).

Procedure.

After screening for hostility and background measures, 71 healthy high-hostile students were contacted again and were asked whether they were interested in participating in a new and focused stress-management program. They were told that they were contacted according to their data at screening. The 22 subjects, who were located and agreed to participate, were matched into 11 pairs according to their age and hostility levels. In order to create therapy groups that were homogeneous with respect to maturity level, participants were first grouped according to age, i.e., below and above age 25, with constraints of scheduling. Subjects were then matched on hostility levels.

Subjects were first matched on PH scores and then on Ho scores, since the construct and predictive validity of PH in relation to CHD are better than that of the Ho scale (e.g , Smith, 1992). Subjects in each pair were then assigned to either the hostility-reduction treatment (experimental group) or to the information-control group using a randomization table. This matching and randomization procedure was employed to overcome possible differences between groups after randomization and prior to treatment. In addition, this procedure, which has been used by others (Eysenck & Grossarth-Maticek, 1991) may increase the trial's statistical power and sensitivity for detecting potentially significant differences between the experimental and control groups at post-treatment, particularly given the stable nature of hostility (Shekelle et al., 1983) and the anticipated small sample size. Finally, there were two intervention groups in the experimental condition, one having five subjects and the other, six. The 11 controls were seen in groups ranging from two to five subjects (one control was seen alone). At the first session, all subjects provided their written informed consent (See Appendix A).

Subjects' hostility levels were assessed pre-treatment and after eight weeks, at post-treatment. At post-treatment, all questionnaires and the SI were readministered by an experimenter who was blind to the subjects' group status in an attempt to reduce demand characteristics. Subjects were instructed not to reveal their group status to the interviewer during the reassessments. Subjects received $15 for undergoing screening and $10 for reassessment. Subjects in

the experimental group received an additional $40 for the experimental sessions. Finally, subjects were sent letters summarizing the study's purpose and results. The results of one control subject at post-treatment were not available as he did not return for reassessment due to an illness of his father.

Data analyses.

Between-groups t-tests were performed on all base-line measures to ensure that there were no group differences prior to treatment To test the treatment's efficacy, a gains-analysis was performed, in which the dependent variable of change scores (e.g., pre-treatment PH minus post-treatment PH) was regressed first on subjects' pre-treatment levels and then on their group status (dummy coded as Control = 0; Treatment = 1). This enabled determination of the variance in change scores (e.g., reduced hostility) accounted for by group status, after accounting for the effects of base-line levels on change scores (Keppel & Zedeck, 1989; Manuck, Kasprowicz & Muldoon, 1990). Rather than employing the more commonly used repeated-measures analysis of variance (ANOVA), a gains-analysis was used for the following reasons: a) A gains-analysis may be more appropriate for clinical trials because it tests more directly whether therapeutic change (i.e., improvement) is a function of or is predicted by subjects' therapeutic group, compared with a repeated-measures ANOVA that examines means at each assessment; b) Unlike an ANOVA, which provides the statistical significance of differences between groups on mean scores, the gains-analysis provides

researchers with an estimate of the effect size of the relation between group status and change (i.e., percentage of variance accounted for; Cohen & Cohen, 1983) in addition to statistical significance; c) A gains-analysis is a specific form of hierarchical multiple regression, and as such, is more flexible and tolerant to unequal sample-sizes that may exist between experimental conditions, compared with an ANOVA which requires complex adjustments for such cases. Finally, some attempts were made to test the clinical significance of the findings by relating the results of the current study to those of previous epidemiological studies. As hostility-reduction was the main aim of this study, tests concerning hostility measures were seen as primary tests, while those concerning reactive-BP were seen as secondary tests.

Results

Statistical assumptions.

Since the main analysis in this study was a gains-analysis, a type of hierarchical multiple regression, the following statistical assumptions and issues were tested: Univariate normality, ratio of subjects to independent variables, lack of singularity and multicollinearity, lack of multivariate outliers, and multivariate normality, linearity and homoscedasticity of residuals (Tabachnick & Fidell, 1989). Univariate normality was tested on all pre-treatment measures. Lack of singularity and multicollinearity were tested on the relations between predictor variables. The remaining assumptions were tested on the five main gains-analyses (i.e., predicting change in Barefoot's Ho, change in SI-derived

PH and Anger-Out, and change in reactive-SBP and reactive DBP).

Univariate normality. This assumption was tested on subjects' pre-treatment scores alone, since changes in the distribution of post-treatment scores in the experimental group (e.g., skewed in one direction) may reflect systematic change which is not expected to occur in the control group. Across groups, the distributions of subjects' pre-treatment scores on age, education, physical activity, SI derived PH and Anger-Out, the full Ho scale, Barefoot's refined Ho scale, and reactive-SBP and -DBP were not significantly different from a normal distribution. Thus, this assumption was fully met.

Ratio of subjects to variables. Examining each gains-analysis separately, there were between 20 to 22 subjects per each pair of independent variables or predictors (e.g., Pre-treatment PH and group-status). This yielded a ratio of at least 10 subjects per each independent variable. However, given that there were five main gains-analyses, the ratio was 3.3 subjects per independent variable. While the first ratio is sufficient for a multiple regression, the latter falls short of the minimal requirement of five subjects per variable recommended by some investigators (Tabachnick & Fidell, 1989). This may reduce the power of the analyses. Thus, this assumption was not fully met. Despite this limitation, according to a prior power analysis, the number of subjects in the trial was sufficient for detecting reduction in hostility.

Lack of singularity and multicollinearity. None of the correlations between any of the predictors (i.e., Pre-treatment PH, Anger-Out, Barefoot's

Ho, reactive-SBP, reactive-DBP and group status) were at or above the value of .85. Thus, this assumption was fully met.

Lack of multivariate outliers. The SPSS Regression command (SPSS Inc., 1985) produces a list of the 10 most deviant cases in a multiple regression from the index of Mahalanobis (Tabachnick & Fidell, 1989). This index is the difference between each subject's mean scores on all variables in a regression and a centroid that reflects scores of all other subjects on all variables in that regression. The significance of the index is tested with a critical Chi square statistic with degrees of freedom equal to the number of predictors (two in the gains-analyses of this research; $X^2(2) = 9.02$). None of the five regressions revealed any significant outlier. Thus, this assumption was fully met.

Multivariate normality, linearity and homoscedasticity of residuals. These assumptions are tested by plotting the relations between the predicted scores (Y') and the residuals (Y - Y'). The assumption of normality was met in all five main gains-analyses. However, the assumption of linearity was not met in one analysis. In predicting change scores of Barefoot's Ho, low and high levels of predicted change scores were associated with negative residuals, while medium levels of predicted change scores were associated with positive residuals. The assumption of homoscedasticity of residuals was not met in one analysis. In predicting change scores of reactive-DBP, residuals were greater in low and high levels of the predicted change scores, while residuals were small in medium levels of predicted change scores. These violations do not

invalidate the analyses, but, they do weaken them (Tabachnick & Fidell, 1989). Thus, this three-part assumption was partially met.

Therefore, four of the five assumptions were met. The one that was not met, ratio of subjects per variables, suggests that the power of the gains-analyses may not be sufficient. However, most of the statistical assumptions were met, and the analyses may be seen as valid (Tabachnick & Fidell, 1989).

Equality of groups at pre-treatment.

Table 10 depicts mean scores of both experimental and control groups on all background, hostility and BP data. At pre-treatment, the only significant difference observed between groups was with respect to resting-SBP, with experimental subjects having higher resting-SBP ($\underline{M}$ = 135.7 mm Hg) than controls ($\underline{M}$ = 126.0 mm Hg; $t(20) = 2.91$, $p < .01$). There were no other ignificant differences between the groups on any pre-treatment measure. Since resting-SBP was not among the major outcomes in a sample of healthy individuals, and to avoid further loss of statistical power, adjustments on this measure were made only with respect to testing the effects of group status on a related outcome, reactive-SBP. Thus, the matching and randomization procedures were generally successful.

Table 10
Background, hostility and BP data of experimental and control groups at pre- and post-treatment.

Measure	Experimental Group				Control group			
	Pre-treatment N = 11		Post-treatment N = 11		Pre-treatment N = 11		Post-treatment N = 10[a]	
	Mean	SD	Mean	SD	Mean	SD	Mean	SD
Background								
Age (years)	25.2	8.0			24.9	6.8		
Education (years)	14.7	1.5			15.1	2.0		
Physical activity (Kilocalories/week)	5544.0	4214.5			5814.9	5471.5		
Hostility								
Potential for Hostility	3.0	.8	3.0	.9	2.7	.8	3.0	.9
Anger-Out	13.8	4.5	11.8	2.9	14.6	5.1	15.7	5.4
Full Ho scale	22.9	7.7	20.5	9.1	23.4	6.9	24.5	6.0
Barefoot's Ho scale	14.1	5.3	11.9	5.1	14.1	4.7	15.9	3.0
BP								
Resting-SBP (mm Hg)	135.7[b]	5.8	126.9	8.2	126.0[b]	9.5	127.4	12.0
Resting-DBP (mm Hg)	77.4	9.0	73.6	8.1	74.0	8.2	74.5	8.8
Reactive-SBP (%)	6.6	5.2	8.8	4.7	5.9	7.4	3.6	5.1
Reactive-DBP (%)	9.3	8.3	6.9	6.6	6.6	8.6	5.9	10.7

[a] One control subject did not return for reassessment. Certain variables include only valid or available data (i.e., BP, Anger-Out). [b] Pre-treatment resting-SBP was significantly higher in the experimental group than in the control group.

Efficacy of intervention in relation to hostility.

Tables 11-13 present gains-analyses for SI-derived Anger-Out, Barefoot's refined Ho scores, and SI-derived PH, respectively. Positive signs in the beta-coefficients of group-status indicate greater decreases in hostility for subjects in the experimental treatment compared to controls.

Table 11
Gains-analysis: Multiple regression analysis regressing change in SI-derived Anger-Out scores on pre-treatment levels and group status (Treatment/Control).

Step	Predictor	R^2	R^2 change	F change	Beta	Significance
1)	Pre-treatment SI-derived Anger-Out	.14		4.37*	.42	.03
2)	Group Status	42	.28	8.93**	.54	01

N = 21. * p < .05 ** p < .01

Table 12
Gains-analysis: Multiple regression analysis regressing change in Barefoot's refined Ho scores on pre-treatment scores and group status (Treatment/Control).

Step	Predictor	R^2	R^2 change	F change	Beta	Significance
1)	Pre-treatment Barefoot's Ho	.28		9.03**	55	01
2)	Group Status	.47	.19	5.96*	43	03

N = 20. * p < .05; ** p < .01.

Table 13
Gains-analysis: Multiple regression analysis regressing change in SI-derived Potential for Hostility (PH) scores on pre-treatment scores and group status (Treatment/Control).

Step	Predictor	R^2	R^2 change	F change	Beta	Significance
1)	Pre-treatment PH	.17		3.78	.39	.09
2)	Group Status	.18	.01	.23	.10	.64

N = 21.

As hypothesized, both SI-derived Anger-Out and Barefoot's refined Ho scores were reduced significantly more in the experimental treatment-group than in the information-control group. The gains-analyses revealed that after controlling for pre-treatment levels, subjects' group status accounted for an additional and significant 28% of the variance in change on SI-derived Anger-Out scores ($F(1,18) = 8.93$, $p < .01$), and an additional and significant 19% of the variance in change on Barefoot's refined Ho scores ($F(1,17) = 5.96$, $p < .05$). Inspection of the means in Table 10 reveals that experimental subjects' Anger-Out scores decreased on average from 13.8 to 11.8, whereas the control subjects' scores increased slightly from 14.6 to 15.7. Similarly, experimental subjects' scores on Barefoot's refined Ho scale decreased on average from 14.1 to 11.9, whereas control subjects' scores increased from 14.1 to 15.9. However, group status did not account for a significant percentage of variance in change scores of SI-derived PH, $F(1,18) = .23$, $p > .05$, after controlling for pre-treatment levels. Finally, subjects' group status did not account for an additional and significant percentage of variance in change scores of full Ho

scores, (only 4%; $F(1,17) = .83$, $p > .05$).

The clinical significance of the positive findings was then tested. Several studies have shown that hostility predicts CHD-incidence and all-cause mortality (e.g., Barefoot et al., 1989; Dembroski et al., 1989; Shekelle et al., 1983). Although these were correlational studies, having been prospective and conducted on initially healthy individuals, they imply a causal relation between hostility and CHD and mortality. **Assuming** that such a relation is valid, the hypothetical reduction in premature mortality given the reduction in hostility reported in the current study was tested by relating the findings from this study to a previous epidemiological study with a similar sample (Barefoot et al., 1989). In both studies, 25 year-old students were recruited. The information provided by Barefoot et al. (1989) was used to estimate the reduced risk of all-cause mortality potentially conferred by the hostility-reduction in the current study. After calculating the beta-weight of the refined Ho scale in Barefoot et al (1989) associated with pre-mature mortality, I calculated the risk of mortality associated with change in Barefoot's refined Ho scores in each of the groups in the current study. This calculation was done as following. First, the applied formula for relative risk (RR) is $RR = e^{(\text{beta} \times \text{relative change in hostility})}$ (Kleinbaum, 1992). Second, a change of 2 SD or 9.06 on Barefoot's Ho was associated with an RR of 5.54 in Barefoot et al. (1989). However, that study did not provide the beta coefficient of Barefoot's Ho. Third, to find the value of the beta coefficient, I performed a Ln function on both sides of the equation mentioned above, with

the given numbers, and this yielded a beta coefficient of .189 for Barefoot's refined Ho. Fourth, substituting the former reduction in hostility (9.06) with that found in the current study relative to controls (4 points on Barefoot's Ho scale), I found RR = $e^{(.189 \times 4.00)}$ = 2.13. Thus, experimental subjects may be 2.13 times less likely to die prematurely, or are at only .47 times the risk of mortality as controls, given their relative reported reduction on Barefoot's Ho scale in the current study, and assuming that hostility causes mortality.

Efficacy of intervention in relation to reactive-BP.

With respect to modifying reactive-BP, no effect was found for change in reactive-DBP, $F(1,18) = .00$, $p > .05$, after controlling for pre-treatment levels (see Table 14). Regarding reactive-SBP, subjects' group status accounted for an additional and significant 13% of the variance in change in reactive-SBP, after controlling for pre-treatment reactive-SBP and pre-treatment resting-SBP scores, $F(1,17) = 6.19$, $p < .05$ (see Table 15). Resting-SBP was also entered as groups differed significantly on this measure at pre-treatment. However, the effect of group status on reactive-SBP was not as hypothesized; As shown in Table 15, the sign of the beta weight of group status was negative, indicating greater reduction in reactive-SBP in the control group than in the experimental group. The means in Table 10 confirmed this finding. However, subjects in the experimental group did not significantly worsen on reactive-SBP ($t(10) = -1.46$, $p < .09$; paired t-test), nor did controls significantly improve on reactive-SBP ($t(9) = .89$, $p > .10$; paired t-test).

Table 14
Gains-analysis: Multiple regression analysis regressing change in reactive-DBP scores on pre-treatment scores and group status (Treatment/Control).

Step	Predictor	R^2	R^2 change	F change	Beta	Significance
1)	Pre-treatment reactive-DBP	.38		9.12**	.62	.00
2)	Group Status	.38	.00	.00	-.01	.94

N = 21. ** p < .01.

Table 15
Gains-analysis: Multiple regression analysis regressing change in reactive-SBP scores on pre-treatment scores, resting-SBP and group status (Treatment/ Control).

Step	Predictor	R^2	R^2 change	F change	Beta	Significance
1)	Pre-treatment reactive-SBP & Resting-SBP	.51		12.14***	.76	.00
					.16	.42
2)	Group Status	.64	.13	6.19*	-.44	.03

N = 21. * p < .05; *** p < .001

Discussion

Study 1 presented the results of a preliminary matched-randomized-controlled trial which tested the efficacy of the proposed hostility-reduction treatment with healthy high-hostile students. The sample employed in Study 1 permitted a test of the intervention without having confounds related to CHD. One hypothesis, hostility-reduction, was almost fully supported, while the hypothesis regarding reactive-BP was not supported.

The results suggest that the proposed treatment can be administered to a population of high-hostile students. Supporting the main hypothesis, the

results showed that the treatment may alter specific pathogenic components of hostility. The gains-analyses revealed that reductions in observed and self-reported hostility (SI-derived Anger-Out and Barefoot's refined Ho scores, respectively) were significantly larger in the experimental treatment-group than in the information-control group. As the Cynicism subscale in Barefoot's Ho includes more items than the other subscales, reductions on Barefoot's Ho may reflect primarily reduced cognitive hostility. Additionally, the treatment may reduce behavioral hostility, the main component assessed with SI-Anger-Out.

These reductions were statistically and **clinically** significant, since extrapolating from Barefoot et al. (1989) suggested that treatment-subjects may be potentially 2.13 times less likely to die prematurely of all causes than controls. However, the latter finding must be taken with caution since mortality was **not** assessed in the present study, and since this extrapolation assumes a causal relation between hostility and mortality. As the hostility components that were altered predict CHD and mortality (Barefoot et al., 1989; Helmers et al., 1993; Matthews et al., 1977; Mendes De Leon, 1992), this treatment may reduce CHD-predictive and mortality-predictive hostility levels.

However, contrary to what was expected, the treatment did not reduce observed levels of PH. Compared with the negative results in the current study, Roskies et al. (1986) did demonstrate a significant reduction in PH scores following a Type-A modification program. Roskies et al. (1986) had a larger sample and the mean ages of their groups were higher than in the

current study. Musante et al. (1989) found that PH was significantly higher in older samples than in younger ones. Indeed, base-line levels of PH were descriptively higher in Roskies et al.'s (1986) sample, which may have provided a larger range of scores for having therapeutic effects and for detecting them compared to the current study. These age-related differences in base-line PH may partly account for the discrepancy between the studies with respect to PH. Additionally, the negative result regarding SI-derived PH may be due to the fact that this measure is a single-item rating. Anger-Out, also derived from the SI, but assessed with eight items from the AX scale (Spielberger et al., 1985), may have been more reliable and sensitive than PH for detecting greater reductions in the treatment-group.

The treatment failed to affect reactive-BP in the hypothesized manner. Two problems exist with the reactive-BP results. First, the intervention did not affect reactive-SBP and reactive-DBP in the same direction. Supporting the hypotheses, reductions in reactive-DBP were descriptively but not significantly greater in the treatment-group than in the control group, but unexpectedly, reductions in reactive-SBP were significantly greater in the control group than in the treatment-group. DBP and SBP reflect related but different stages in the cardiac cycle. While DBP reflects minimal BP during ventricular dilatation, SBP reflects maximal BP during ventricular contractions (Steptoe & Johnston, 1991). This may explain how reactive-SBP and reactive-DBP may be affected differently by the same intervention.

Second, the effect with reactive-SBP was unexpected. Although not statistically significant, reactive-SBP was descriptively reduced in the control group and descriptively increased in the treatment-group. The following explanations may partly explain these tendencies. Controls may have tended to reduce their reactive-SBP due to familiarity with the SI and its challenging context (Roskies et al., 1986). However, for treatment-subjects, learning for two months about the relations between hostility and BP and CHD may have induced great expectations of reductions in their hostility and BP. These expectations may have caused increased anxiety and tension during the SI, resulting in transient hyper-reactivity (Suarez & Williams., 1990) that masked any familiarity effects possibly experienced by control subjects.

Finally, it is possible that the reactivity findings are generally unreliable as there was great variability in each assessment particularly for controls (as seen in the standard deviations). This variability may have resulted from random transient changes in reactivity, that reduced its reliability. One previous trial on Type-A modification failed to alter reactivity as well, and questioned the sensitivity and relevance of SI-derived reactivity measures for evaluating the effectiveness of psychological interventions (Roskies et al., 1986). Similarly, Turner et al. (1995) found that while their control group appeared to have improved on resting-DBP, it worsened on reactive-DBP Future trials may wish to test the efficacy of their interventions for reducing reactivity using both interpersonal challenges (e.g., public speech tasks) and asocial challenges

(e.g., mental arithmetic tasks). This may provide a wider range of situations for testing the feasibility of reducing cardiovascular reactivity. Finally, although an exceptional finding, Carroll et al. (1995) recently found that adding reactive-BP to initial resting-BP did not improve the prediction of long-term resting-BP, placing some doubt on the meaningfulness of reactive-BP measures.

Anger-Out scores were modified by the hostility intervention. These reductions were smaller than those reported by Deffenbacher et al. (1987) However, unlike the latter study, in the current study Anger-Out was assessed from the SI by a coder who was blind to subjects' group status. This may have partially removed biases from the results, biases that may have occurred in the study by Deffenbacher et al. (1987). Anger-Out has been shown to be related to the Antagonism dimension of personality and to be unrelated to Neuroticism (Costa et al., 1989), and antagonistic hostility was a major target of the intervention. Parameters not contaminated with Neuroticism are better predictors of CHD and mortality (Watson & Pennebaker, 1989) Thus, altering Anger-Out may be more important for preventing CHD and mortality than altering experiential components of hostility that are contaminated by Neuroticism (e.g., experienced anger; Costa et al., 1989; Dembroski et al., 1989; Musante et al., 1989).

The fact that overall Ho scores were not significantly altered, but that the more specific "toxic" components assessed with Barefoot's refined Ho scale **were** altered, validates the claim that the treatment targets the most CHD-

predictive components of hostility (i.e., antagonism, cynicism and anger). Although previous clinical trials employed similar clinical skills (e.g., relaxation, assertiveness-training, cognitive-restructuring) and found reductions in hostility and anger measures (e.g., Achmon et al., 1989; Deffenbacher et al., 1987; Roskies et al., 1986), this study was designed to target and reliably assess CHD-predictive hostility dimensions alone. Additionally, the relevance of hostility to CHD was emphasized to subjects in a comprehensive educational component and throughout treatment.

Changes in both observed and self-reported hostility (SI-derived Anger-Out and Barefoot's refined Ho) suggest that the proposed treatment may indeed modify hostility. Thus, although hostility is a highly stable characteristic (e.g., Shekelle et al., 1983), CHD-predictive hostility may be modified by a brief, but, intense and focussed, eight-week cognitive-behavioral treatment.

Study 1 had several limitations. First, the results are based on a small sample size, which may have prevented the detection of other effects (e.g., reductions in reactive-DBP). The low enrollment rate (31%) limits the generalizations that may be made from the results of this study to high-hostile students and reveals the difficulty of conducting research with hostile individuals. However, the fact that statistically significant effects were found with a small sample size suggests that the observed effects are strong.

A second limitation is that several negative results were found (i.e., SI-derived PH, reactive-BP). As mentioned above, limitations related to

measurement-reliability may underlie in part these negative results. Additionally, due to the relative scarcity of high PH levels in young samples (Musnate et al., 1989), a larger or an older sample may have revealed therapeutic effects in relation to PH as well, as in the study by Roskies et al. (1986). Third, the hostility components that were altered (Barefoot's refined Ho, and SI-derived Anger-Out) were based primarily on subjects' **reported** hostility. These measures, unlike SI-derived PH and particularly Hostile Style (Dembroski & Costa, 1987) were not derived from observed overt antagonistic behavior exhibited by subjects during the SI. Altering mainly reported hostility may reflect a demand characteristics bias. Although the measures revealing reductions in hostility predict CHD and mortality, corroborating reports of reductions from other sources (e.g., family members) would render these findings more compelling. Fourth, it is unknown whether the observed effects are stable over time as no follow-up was conducted. And finally, it is unknown whether the proposed treatment affects other psychological and health outcomes. Endurance and breadth are important requirements for declaring an intervention effective (Kazdin & Wilson, 1978).

Despite these limitations, the feasibility of administering this treatment to high-hostile subjects, and preliminary evidence for its ability to alter specifically CHD-predictive hostility assessed by several measures, are encouraging. Study 2 addresses several of the limitations in Study 1 and extends its findings to CHD patients.

CHAPTER SEVEN

STUDY 2: THE EFFECTS OF THE HOSTILITY-REDUCTION TREATMENT ON HOSTILITY AND HEALTH MEASURES OF CHD PATIENTS

Introduction

Most medical interventions for preventing CHD, and several medical interventions for treating CHD typically target "traditional" risk factors such as cholesterol, smoking and blood-pressure (BP; e g., The Coronary Drug Project Research Group, 1975). Other medical interventions target certain precipitating pathological processes such as platelet-aggregation (e.g., Lewis et al., 1983) Since traditional risk factors account for between 6.25% to approximately 50% of the variance in CHD (Keys et al., 1972; Leon et al., 1988), targeting them alone may be insufficient for complete secondary prevention. Thus, other significant psychological risk factors, which predict CHD independent of traditional risk factors, should be targeted as well, to increase the effects of current medical interventions.

As reviewed in Chapter Three, hostility is more strongly associated with CHD end-points than is the original "coronary-prone" Type-A Behavioral Pattern (TABP; Dembroski et al , 1985, Dembroski et al., 1989; Williams et al., 1980) Hostility has been associated with transient daily ischemia (e.g., Helmers et al , 1993), with unstable angina (UA; Mendes De Leon, 1992), and hostile-provocations induce greater myocardial dysfunction than do general stressors (Ironson et al., 1992). In cross-sectional studies, hostility is significantly related

to coronary artery disease (CAD; Dembroski et al., 1985; Siegman et al., 1987; Williams et al., 1980), and hostility is prospectively and significantly related to CHD incidence, CHD-progression and mortality (e.g., Barefoot et al., 1989; Barefoot et al., in press; Dembroski et al., 1989; Powell & Thoresen, 1985), independent of other risk factors (e.g., Barefoot et al., in press; Dembroski et al., 1989; Hecker et al., 1988). The few negative prospective studies which found no association between hostility and CHD (e.g., Hearn et al., 1989; Leon et al., 1988; McCranie et al., 1986) used measures of hostility that have limited validity or suffered from serious measurement biases (McCranie et al., 1986). Thus, hostility (particularly derived from the SI) is an independent and significant CHD risk factor (Miller et al., in press; Smith, 1992).

Several psychological clinical trials attempted to improve the rehabilitation of CHD patients by altering the psychological sequelae of CHD (e.g., distress, social isolation, depression; Ibrahim et al., 1974; Rahe, Ward, & Hayes, 1979). Other trials attempted to improve patients' prognosis by targeting significant psychological CHD risk factors such as the global TABP (e.g., Friedman et al., 1986; Turner, et al., 1995), but did not focus solely on hostility. Although some of these treatments did reduce hostility (Mendes De Leon et al., 1991), focussing solely on the more toxic dimension of CHD-predictive hostility should increase therapeutic efficacy (Chesney, 1985; Dembroski & Costa, 1987), and allow the hypothesized causal relation between hostility and CHD (Smith, 1992) to be tested. Adding such a hostility-treatment

to current medical interventions may increase their efficacy in relation to CHD.

Study 1 in this thesis showed that a brief hostility-reduction treatment reduced self-reported (Barefoot's Ho) and observed (SI-derived Anger-Out) CHD- and mortality-predictive hostility levels in healthy, high-hostile males. However, no follow-up was conducted to determine the durability of the treatment, and no additional health and psychological measures were used to determine the breadth of the treatment's efficacy, two important criteria for evaluating interventions (Kazdin & Wilson, 1978). Furthermore, based on Study 1 alone, it is unknown whether the proposed hostility-reduction treatment is feasible and effective with CHD patients. Finally, Study 1 did not employ hostility measures from sources other than the subjects (e.g., family) to increase the reliability of self-reported hostility measures (Barefoot & Lipkus, 1994).

Therefore, Study 2 was designed to replicate and extend the findings of Study 1 and improve its methodology. Specifically, the purposes of study 2 were as following: a) To replicate the findings regarding hostility-reduction from Study 1 with CHD patients, (the main objective of Study 2); b) To test the the effects of the treatment on other CHD risk factors and outcomes (e.g., hypertension, depression, quality of life; Frasure-Smith, Lesperance & Talajic, 1993; Kannel et al., 1986; Kaplan, 1988); c) To test the endurance of therapeutic effects by including reassessments at post-treatment and two months later; and d) To reduce demand-characteristics and presentation-

biases by including hostility measures obtained from patients' spouses in addition to patients' self-reports.

It was hypothesized in general that patients assigned to the hostility-reduction treatment group would show greater reductions in CHD-predictive hostility measures and greater improvements on health measures compared to patients assigned to an information-control group. Specifically, patients assigned to the cognitive-behavioral hostility-reduction treatment group were expected to show greater reductions on SI-derived Anger-Out, Potential for Hostility, Hostile Style, self- and spouse-reported Barefoot's Ho, resting-SBP and -DBP, impaired quality of life and depression than patients assigned to the information-control group. These effects were expected to be maintained at a two-month follow-up. A single-blind, matched-randomized-controlled design and reliable and valid measures were employed to test these hypotheses

Method

Subjects.

Forty-nine CHD patients participated in a screening stage and were assessed for psychological and health background. The majority of patients were recruited from one of two medical centers (The Camp-Hill Medical Centre, or The Victoria General Hospital, Halifax, Nova Scotia, Canada). A few patients were recruited by advertisements in the media.

Inclusion criteria for entry into the clinical trial. a) Patients with a medically documented diagnosis of either myocardial infarction (MI) or unstable

angina (UA) pectoris within the past six months. Myocardial infarction was based on evidence of elevated CPK-MB enzyme and, typical chest-pain or ECG changes (Shechter et al., 1990; Sokolow & McLlory, 1986). The diagnosis of UA had to include chest-pain at rest or pain that had progressed in frequency, duration or intensity, and one of the following indices of cardiac disease: Diagnostic ST-segment changes in ECG, previous MI, or previous angiographically documented CAD (Plotnick, 1985a; Sokolow & McLlory, 1986); b) Only males were included as hostility has been shown to be related to CHD primarily in males (Smith, 1992), and as there are gender differences in hostility and in CHD (Barefoot et al., .991; Davidson & Hall, 1995; Jenkins, 1988) which could obscure the results; c) Patients between 35 to 60 years of age, as hostility may be more strongly related to CHD under age 60 (e.g., Helmers et al., 1993; Siegman et al., 1987); d) Patients who scored 15 and above on SI-derived Anger-Out and/or 13 and above on Barefoot's refined Ho scale. The first cut-off point has been previously associated with UA (Mendes De Leon, 1992). The second cut-off point was chosen as it represented a mean score of a CAD sample (John Barefoot, private communication, August, 1994), and since Barefoot's Ho has been shown to be related to CHD and mortality (Barefoot et al., 1989; Helmers et al., 1993). These cut-off points were chosen to guarantee that the trial's sample will be relatively high on hostility from statistical and clinical perspectives related to CHD. As in Study 1, using one of the two cut-offs guaranteed that a sufficient number of candidates would be

initially identified for the trial; e) Patients with reported chronic psychiatric disorders (e.g., clinical depression, psychosis) were excluded to avoid confounding with hostility-reduction.

Of the 49 screened patients, two were excluded since they did not fulfil the diagnosis of either MI or UA (they were recruited through advertisements). Two additional high-hostile patients were excluded since they were unsuitable for short-term cognitive therapy (Safran, Segal, Valis, Shaw & Samstag, 1993; Yalom, 1985; see Procedure below). Thirty-five patients scoring at or above the cut-offs of one of the two hostility measures were then asked to take part in a new stress-management study. Eight patients refused to participate (work-related constraints (4); distance from home (1); domestic problems (1); moving away (1); not interested (1)) and one patient was not located. Three patients withdrew at an early stage from the treatment-group and study due to personal or medical problems (death of a parent (1); medical and psychological problems beyond treatment's scope (1); severe medication problems (1)), and one control patient moved away. The remaining 22 patients agreed to take part in the study (10 with MI, 12 with UA). Of the 35 high-hostile patients, participants (22) and non-participants (13) did not differ significantly on SI-derived Anger-Out ($\underline{M}$ = 19.6 versus 17.5, $t(33) = 1.44$, $p > .05$) or on Barefoot's refined Ho scale ($\underline{M}$ = 13.7 versus 12.7, $t(32) = .66$, $p > .05$), respectively. Thus, with respect to hostility levels, participating patients were representative of non-participating patients. The final number of participating patients (22) was deemed sufficient

according to a power analysis conducted prior to the study (Cohen & Cohen, 1983), with a power of .80 for accounting for 20% of the variance in change scores of SI-derived Anger-Out and 15% of the variance in change scores of Barefoot's Ho by group status. These power analyses were based on the findings of Study 1, and assumed greater difficulty with reducing hostility of CHD patients, due to their age and confounds related to CHD.

Instruments.

Demographic and medical background. The following background data were collected for each participant: Patients' age (years), education (years), documented CHD event (UA or MI, based on the above criteria and obtained from medical records), one of three forms of revascularization (thrombolytic therapy, angioplasty or coronary artery bypass graft - CABG - surgery), cigarette-smoking (cigarettes/day * years of smoking), resting-SBP (mm Hg; see below) and whether or not medicated with beta-blockers. Data on age, education, resting-SBP and smoking were collected as CHD risk factors (Jenkins, 1988; Kannel et al., 1986). Revascularization was recorded due to its prognostic value (GISSI, 1987). Finally, the percentages of patients taking beta-blockers in each experimental condition were compared, as such drugs may reduce levels of hostility (Krantz et al., 1982). Due to the small sample size, group differences in use of other types of medication were not tested.

Observed hostility. Observed ratings of behavioral hostility were assessed with the Augmented Structured Interview (ASI; Wright & Schmidt-

Walker, 1990). As in Study 1, this 12-15 minute, videotaped interview was administered in a non-confrontational manner to yield more valid ratings (Houston et al., 1988). A male interviewer rated each interview for Potential for Hostility (PH), Hostile-Style and Anger-Out. PH and Hostile-Style were assessed with 1-5 rating scales, according to previous studies (Dembroski & Costa, 1987; Dembroski et al., 1989). Whereas PH reflects both reported hostility and overt antagonism exhibited by the subject during the SI, Hostile-Style reflects overt antagonism directed by the subject towards the interviewer alone. Inter-rater reliability in the present study on a subset of 19 interviews at screening was high for PH ($r = .81$), and moderate for Hostile-Style ($r = .60$). Ratings of participants in the clinical trial on PH and Hostile-Style at pre-treatment were moderately-strongly correlated ($r = .72$), indicating that PH is heavily affected by subject's overt antagonism manifested during the SI.

As in Study 1, Anger-Out was assessed from the SI as well. Coders observed the SI and then completed the Anger-Expression Scale (AX; Spielberger et al., 1985) for that subject, to achieve greater reliability than that obtained from single-item observational measures of Anger-Out previously used (e.g., Matthews et al., 1977) and to increase sensitivity for detecting possible therapeutic changes. Inter-rater reliability in the present study for SI-derived Anger-Out was moderate ($r = .65$). The internal consistency reliability level for the eight-item rating of Anger-Out in the present study was sufficient (Cronbach's Alpha = .87).

One rater, who was blind to patients' group status and was unaware of their self-reported hostility scores, assessed PH, Hostile Style and Anger-Out at pre-, post-treatment and follow-up. The interview scores of one patient were omitted from all analyses (i.e., hostility, reactive-BP), since his first and second interviews lasted approximately 19 minutes and his third interview lasted 32 minutes. Thus, his interviews were seen as not comparable to other patients' standardized 12-minute interviews.

Self-reported hostility. Self-reported hostility was assessed with Barefoot's 27-item refined Ho scale comprising of the Aggressive-Responding, Cynicism and Hostile Affect subscales (Barefoot et al., 1989). As in Study 1, the internal consistency reliability of this scale was sufficient (Cronbach's Alpha = .81).

Spouse ratings of patients' hostility. Patients' hostility was also rated by their spouses or significant others. These measures included Anger-Out with the AX (Spielberger et al., 1985), and Barefoot's refined Ho scale (Barefoot et al., 1989), phrased accordingly. Initially, these spouse-ratings were intended to be used as outcome measures, however, approximately half of the spouses did not complete the scales. Thus, it was impossible to compare the experimental conditions reliably, and these ratings served to validate patients' self-reported measures, as has been previously suggested (Barefoot & Lipkus, 1994). Spouse-ratings of patients' refined Ho were moderately to strongly correlated with patients' self-reported refined Ho scores ($r = .76$, $p < .01$) and

spouse-ratings of patients' Anger-Out were moderately to strongly correlated with the coder's ratings of SI-derived Anger-Out ($r = .71$, $p < .01$). These correlations provide strong support for the validity of self-reported refined Ho scores and SI-derived Anger-Out.

Cardiovascular measures Patients' resting- and reactive-SBP and -DBP were measured before and during the SI, respectively. These measures were obtained with an Oscillometric Spacelabs 90207 ambulatory monitor (Redmond, WA) placed on patients' non-dominant arm. The procedure was identical to that performed in Study 1. The last two base-line readings were averaged to obtain the resting-BP measures (Dembroski et al., 1979). It has been recommended that the validity of ambulatory measures be established on the basis of comparison with manual ones (Steptoe & Johnston, 1991), and this was important particularly as resting-BP constituted the only objective health outcome in the current study. Thus, resting-BP levels of eight individuals were also assessed by a physician with a sphygmomanometer. The physician was blind to ambulatory measures. A high correlation was found between the two BP measures for resting-SBP ($r = .94$) and for resting-DBP ($r = .78$). Thus, the resting-BP scores obtained with the ambulatory monitor were valid.

Quality of life. Perceived quality of life was assessed with the Quality of Life Questionnaire for Severe Heart Failure (QLQ-SHF; Wiklund, Lindvall, Swedberg & Zupkis, 1987). This 23-item scale includes Somatic Symptoms, Life Satisfaction, Emotions and Physical Activity subscales. Its' selected items

resulted from previous research with MI and heart-failure patients, a cardiologist's judgement of item-relevancy, patients' judgements of frequency of complaints, and from empirical criteria (factor loadings). In the current study, the internal consistency reliability of the entire scale was high (Cronbach's Alpha = .87), and moderate to high for its subscales (Somatic Symptoms: .71; Life Satisfaction: .77; Emotions: .82; and Physical Activity: .59). Using previous categorizations of quality of life measures (Kaplan, 1988), this scale is a psychometrically-based disease-specific measure, focussing primarily on health status, but assessing general quality of life as well. Disease-specific measures of quality of life may be preferable over generic measures since the former are more responsive to treatment-induced changes and are more relevant to patients than are the latter (Guyatt, Bombardier & Tugwell, 1986; Wiklund & Karlberg, 1991). The assessment and improvement of quality of life in clinical trials with CHD patients is essential since CHD adversely affects patients' psychosocial adjustment and functioning (Kaplan, 1988; Wiklund, Sanne, Vedin & Wilhelmsson, 1984).

As the QLQ-SHF included items related to depression, a significant predictor of post-MI mortality (Frasure-Smith et al., 1993) a depression index was formed from this questionnaire. Thus, a separate depression measure was not added to reduce the number of questionnaires patients had to complete. Items from the QLQ-SHF were selected according to an empirical criterion and their face validity. Thus, all items whose scores correlated at or above .60 with

scores on the "feel depressed" item were then examined for their face validity. Two items reflecting anxiety were excluded, and the depression index finally included five items ("feel depressed", "feel pessimistic", "satisfied with your personal life" (reversed), "feel generally dissatisfied" and "difficulty in deciding what to do"). The depression index's internal reliability was high (Cronbach's Alpha = .88), supporting its reliability.

Hostility-modification treatment. The cognitive-behavioral hostility-reduction intervention outlined in Chapter Five and employed with students in Study 1 was used in Study 2 as well. The intervention followed the same format and content, with the following exceptions: a) Age-appropriate modeling and examples were used for patients (e.g., facing daily provocations at work and with patients' spouses); b) Patients handed in their weekly monitoring logs of hostility and use of skills, in order to increase adherence to treatment and therapeutic change (Davison et al., 1991); c) Following patients' requests, and in order to increase patients' acceptance of the role of hostility in CHD, two published manuscripts were provided and explained to patients (Barefoot et al., 1983; Williams, 1987).

Control group. As in Study 1, patients assigned to the information-control group received one session about the CHD risks of hostility and basic skills for modifying it. This group served as an information-control condition (O'Leary & Borkovec, 1978), and was seen as important for ethical reasons as well. Controls were also provided the opportunity to contact one of the

therapists, should they have any questions or concerns (none of them did so). For ethical reasons, and only after analyses of the results revealed several positive effects for the hostility-reduction treatment, controls were offered the opportunity to participate in the hostility-reduction treatment after the research was completed. Both treatment and control sessions were provided by a female resident in clinical psychology (Mrs. Maureen Sullivan) and a male Ph.D student in experimental health psychology (Yori Gidron), under the supervision of a registered clinical psychologist (Dr. Karina Davidson).

Procedure.

After screening for hostility levels, and prior to randomization, high-hostile CHD patients underwent a semi-structured assessment with one of the therapists to assess suitability for short-term cognitive-behavioral group therapy. This procedure was aimed at assessing patients' understanding of their illness, motivation and appropriateness for therapy, potential for alliance with others and interpersonal appropriateness (Safran et al., 1993; Yalom, 1985)[2]. This additional screening was used to increase the matching between the proposed treatment and patients, and to increase therapeutic efficacy.

After screening, patients were matched on a prognostic variable (age) and on hostility. It was initially planned to match patients on stress-test scores,

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[2] This additional screening procedure was conducted only with the second half of the clinical trial (14 patients in total), prior to randomization. It was added since a few patients in the first half of the trial were unsuitable for group therapy, one of whom withdrew from the trial voluntarily.

however, most scores were negative by the time patients had been discharged from hospital. An alternative prognostic matching variable, namely, ejection fraction, was also unsuitable as it was not uniformly measured across the different medical centers involved in this study. Age was chosen as the prognostic matching variable because age is related to severity of underlying CAD (Sokolow & McLlory, 1986) and to CHD (Jenkins, 1988), and since age moderates the relation between hostility and CHD (e.g., Dembroski et al. 1989; Siegman et al., 1987). Thus, age was seen as a simple prognostic matching variable relevant specifically to this study. Finally, eleven pairs of high-hostile CHD male patients were matched according to age and hostility levels. The hostility matching variable was SI-derived Anger-Out, a predictor of CHD (Matthews et al., 1977), and a hostility parameter that had been reduced in Study 1. A conceptually similar matching procedure was done in Study 1 and by Eysenck and Grossarth-Marticek (1991), to increase trials' sensitivity.

Patients in each matched pair were then randomly assigned to either the experimental hostility-reduction treatment (treatment-group; N = 10) or to the information-control group (n = 12) using a randomization table. Group sizes were unequal due to attrition of patients from the trial, as mentioned in the Subjects section above. Finally, there were two hostility therapy groups, one with four and another with six patients. Controls were mostly seen in groups ranging from three to four patients. All patients provided their written informed consent to participate in this study (see Appendix B). Both treatment and

control groups met at the Cardiac Prevention Research Centre at the Camp-Hill Medical Centre, Halifax, Nova Scotia. During assessments at pre- and post-treatment, a physician in a nearby room was available in case of a cardiac arrest. Patients received partial compensation for travel ($15 at screening, $10 after the control session, $50 after the eight hostility-reduction sessions, and an additional $10 for each reassessment). At post-treatment and at the two-month follow-up, all instruments were administered by an interviewer who was blind to patients' group status. Patients were instructed not to reveal their group status to the interviewer during the reassessments.

Data analyses.

To test equality of groups at pre-treatment, between-groups t-tests were performed on all continuous measures and chi-square tests on dichotomous variables (i.e., type of CHD-event, revascularization, beta-blockers). Correlations between scores on pre-treatment measures of major variables (e.g., hostility, resting-BP) were examined to determine their interrelationships and to compare these relations with those expected from past research. As in Study 1, the main analysis for determining the efficacy of the intervention was a multiple-regression gains-analysis, using the Regression command (SPSS Inc., 1985). This test determined the variance in change scores (i.e., improvement) accounted for by group status, after accounting for the effects of pre-treatment levels. Thus, each dependent variable (e.g., pre- minus post-treatment PH; post- minus pre-treatment Life-Satisfaction) was regressed first on patients' pre-

treatment levels (e.g., Pre-treatment PH; pre-treatment Life-Satisfaction) and then on patients' group status (dummy coded as Control = 0; Treatment = 1). In cases where the gains-analysis did not yield significant effects but the means descriptively changed in the hypothesized directions, additional exploratory change analyses were conducted (e.g., Rahe et al , 1979). These tests compared the percentages of patients who changed on specific measures (e.g., resting-DBP) to a clinically-meaningful level (e.g., < 95 mm Hg) in each group, with a Chi-square test. Such tests have been advocated by others for evaluating treatments' efficacy (Kazdin & Wilson, 1978). As in Study 1, some attempts were made to relate the findings of the current study to previous epidemiological studies in relation to mortality and CHD-prevention. These tests will be explained in their context. Since this was a first trial testing the efficacy of the hostility-treatment with CHD patients, an effect-size analysis was performed (Cohen, 1977), which revealed the **magnitude** of change in different measurement-categories resulting from the treatment and control groups. These tests reveal the strengths and weaknesses of the intervention, and may guide future trials (Turner et al., 1995). In these tests, the difference between pre- and post-treatment means (in the hypothesized direction) was divided by the pre-treatment standard deviation of each measure within each group, yielding a d-Coefficient (Cohen, 1977). A d-Coefficient of .2 indicates small change, .5 indicates moderate change, and > .8 a large change (Turner et al., 1995). Finally, evidence for causal relations between variables of interest (e.g.,

hostility and resting-BP) was revealed by examining the correlations between change scores of measures that were significantly altered by the treatment.

Results

Statistical assumptions.

The main statistical analysis for testing the intervention's efficacy was a gains-analysis, a type of hierarchical multiple-regression. Thus, the following assumptions were tested: Univariate normality, ratio of patients to independent variables, lack of singularity and multicollinearity, lack of multivariate outliers, and multivariate normality, linearity and homoscedasticity of residuals (Tabachnick & Fidell, 1989). Univariate normality was tested on all pre-treatment measures. Lack of singularity and multicollinearity were tested on the relations between predictor variables. The remaining assumptions were tested on the eight main gains-analyses of post-treatment data (i.e., predicting change in Barefoot's Ho, SI-derived PH, Anger-Out and Hostile Style, resting-SBP and resting-DBP, and change in overall quality of life and the depression-index scores). Assumptions were not tested on the change scores of follow-up data as follow-up was primarily a test of the endurance of the intervention's efficacy.

Univariate normality. As in Study 1, this assumption was tested on patients' pre-treatment scores alone. Across groups, patients' pre-treatment scores on age, education, cigarette smoking, resting-SBP and DBP, SI-derived PH, Anger-Out and Hostile-Style, Barefoot's refined Ho scale, overall quality of life scores and the depression-index scores were normally distributed.

However, two of the 14 scores: Type of event and having a revascularization were not normally distributed. Of course the scores of these two variables are not expected to be normally distributed as they are dichotomous variables. Thus, this assumption was fully met.

Ratio of patients to variables. Examining each gains-analysis separately, there were 20 to 22 patients per each pair of predictors (e.g., Pre-treatment PH and group-status). This yielded a ratio of at least 10 patients per each independent variable. However, since eight main gains-analyses were performed, there was a ratio of 2.2 patients per independent variable. Whereas the first ratio is sufficient for a multiple regression, the latter falls short of the minimal requirement of five patients per variable (Tabachnick & Fidell, 1989) This violation reduces the power of the analyses. Thus, this assumption was not fully met. However, according to prior power analyses, the number of patients in the trial was sufficient for detecting reduction in hostility.

Lack of singularity and multicollinearity. None of the correlations between predictors (i.e., Pre-treatment PH, Anger-Out, Hostile-Style, Barefoot's Ho, resting-SBP and -DBP, quality of life, depression and group status) was at or above the value of .85. Thus, this assumption was fully met.

Lack of multivariate outliers. Calculating the index of Mahalanobis (SPSS Inc., 1985) and a critical Chi square statistic ($X^2(2) = 9.02$) revealed that there were no significant outliers on any of the eight main gains-analyses. Thus, this assumption was fully met.

Multivariate normality, linearity and homoscedasticity of residuals. The assumption of multivariate normality was met in all but one gains-analysis. There were more positive than negative residuals in predicting change in Hostile-Style. The assumption of multivariate linearity was met in four gains-analyses In predicting change scores of PH, low and high levels of predicted change scores were associated with negative residuals, while medium levels of predicted change scores were associated with positive residuals In predicting change scores of Hostile Style, low and medium levels of predicted scores were associated with positive residuals, while high predicted scores were associated with positive and negative residuals. In predicting change in resting-SBP and -DBP, low and high levels of predicted change scores were associated with positive residuals, while medium levels of predicted change scores were associated with negative residuals.

The assumption of multivariate homoscedasticity was not met in most analyses. In predicting change in PH, residuals were small in medium in levels of predicted scores while residuals were large in low and nigh predicted scores In predicting change in Hostile-Style, residuals increased with levels of predicted scores. In predicting change in resting-DBP, residuals were large in low and high levels of predicted scores, while residuals were small in medium level of predicted scores Finally, in predicting change in overall quality of life and depression, residuals decreased with increases in predicted scores. Thus, these assumptions were not met. These violations do not invalidate the gains-

analyses, however, they do weaken them (Tabachnick & Fidell, 1989).

Therefore, three assumptions were met. The violation of ratio of patients per variables and of the three-part assumption of multivariate normality, linearity and homoscedasticity of residuals, suggests that the power of the gains-analyses may not be sufficient. However, the analyses may be seen as valid (Tabachnick & Fidell, 1989). In comparison to Study 1 with high-hostile students, more assumptions were violated with high-hostile CHD patients These violations may result from the fact that the CHD sample was more heterogeneous than the student sample in variables such as education and health status, possibly yielding greater variability in patients' responses to therapy, when compared with students' responses. These violations decrease the ability to find statistically significant intervention effects, and finding such effects would suggest that the intervention has a strong effect

Equality of groups at pre-treatment

Table 16 presents the demographic and medical background of the treatment and control groups at pre-treatment No significant differences were found between the groups on any pre-treatment measure. However, descriptively more controls underwent revascularization (75%) than did patients in the treatment group (50%), and descriptively fewer controls were receiving beta-blockers (33%) than treatment patients (60%). Table 17 presents the mean scores of patients in both groups on hostility, BP and quality of life at pre-treatment, post-treatment and two months later. Again, there were no

significant differences between the groups on any pre-treatment measure

Thus, the matching and randomization procedures were successful.

Table 16
Pre-treatment demographic and medical background of patients in treatment and control groups.

Measure	Hostility-reduction Treatment group N = 10		Information-Control group N = 12	
	Mean	SD	Mean	SD
Demographic				
Age	54.4	6.8	54.3	5.8
Education (years)	11.4	3.9	12.5	2.7
Medical				
MI	.4		.5	
UA	.6		.5	
Revasc.[a]	.5		.7	
Resting-SBP (mm Hg)	145.9	22.9	148.0	20.9
Beta-blockers	.6		.3	
Smoking (Cigarettes/day x Years)	498.0	466.8	330.2	356.3

No statistically significant differences were observed between treatment and control groups on any pre-treatment measure. [a] Revasc. = Ratio of patients undergoing one of three forms of revascularization (thrombolysis, angioplasty, CABG).

Table 17

Pre-treatment, post-treatment and follow-up scores of patients in treatment and control groups.

Measure	Hostility-reduction Treatment group (N = 10)						Information-Control group (N = 12)					
	PRE*		POST		FOLLOW-UP (2-month)		PRE*		POST		FOLLOW-UP (2-month)	
	Mean	SD	Mean	SD	Mean	SD	Mean	SD	Mean	SD	Mean	SD
Hostility												
Barefoot's Ho'	13.9	5.3	11.0	6.2	10.3	6.3	13.8	4.9	14.2	5.4	13.7	6.0
Anger-Out	19.8	5.3	17.0	4.5	17.8	4.5	19.2	3.6	17.1	3.9	19.0	3.9
Potential for Hostility	3.7	.9	3.3	1.0	3.0	.9	3.7	.9	3.9	1.0	3.8	.8
Hostile-Style	1.9	.9	1.6	.7	1.6	.9	1.7	.9	2.0	.8	1.7	.6
BP												
Resting-SBP	145.9	22.9	136.8	16.7	138.0	19.6	148.0	20.0	153.0	24.5	139.7	12.3
Resting-DBP	90.8	7.7	85.6	10.2	91.2	12.5	88.9	15.7	93.3	12.5	91.3	9.9
Quality of Life												
Life Satisfaction	4.0	.8	4.8	.6	4.7	.6	4.2	.7	3.6	1.1	4.1	.9
Physical Activity	4.8	.6	4.8	.9	4.7	1.0	4.5	.6	4.4	.9	4.4	1.1
Somatic Symptoms	4.8	.6	4.8	.9	5.0	.8	4.6	.8	4.4	1.0	4.7	1.0
Emotions	4.7	.8	4.9	.7	5.1	.5	4.5	.9	4.6	.8	4.7	.9
Depression	4.5	.9	5.1	.6	5.2	.5	4.7	.9	4.2	1.1	4.7	.8

* No statistically significant differences were observed between treatment and control groups on any pre-treatment measure. † Barefoot's Ho: Barefoot et al.'s (1989) refined Ho scale. Higher scores indicate better quality of life (e.g., more life satisfaction, less depression).

Correlations between measures.

To provide information on the construct validity of the measures and whether they are meaningfully inter-related, a bivariate correlation matrix was constructed (see Table 18). These correlations must be taken with caution as tney are based on a small sample size, and reflect many tests of statistical significance. Keeping this caution in mind, however, a certain coherent picture does emerge. Hostility, as assessed by the SI (PH and Anger-Out) and by spouses (spouse-rated Barefoot's Ho) is positively correlated with resting-BP. Hostility, as rated by spouses, is positively correlated with patients' self-reported hostility and with that assessed by coders (SI-derived Anger-Out). Quality of life is negatively correlated with patients' resting-DBP and with patients' hostility (spouse-rated Barefoot's Ho). Finally, the strongest correlations appear to be consistently those involving spouse-rated hostility.

Table 18
Correlations between scores on selected pre-treatment measures.

	SBP	DBP	Anger-Out	PH	Barefoot's Ho	Spouse-Anger-Out	Spouse-Barefoot's Ho	Quality of Life
SBP	-----	.47*	.24	.41*	.09	.21	.49*	-.13
DBP		-----	.50*	.33	.36	.41	.56*	-.44*
Anger-Out			-----	.35	.22	.71**	.52*	-.19
PH				-----	.0	.56*	.18	-.04
Barefoot's Ho					-----	.14	.76**	-.36
Spouse-Anger-Out						-----	.33	-.02
Spouse-Barefoot's Ho							-----	-.46*

* p < .05; ** p < .01 (all 1-tailed). SBP - Resting systolic blood-pressure; DBP - Resting diastolic blood-pressure; PH - Potential for Hostility; Barefoot's Ho - Barefoot et al.'s (1989) refined Ho; Spouse Anger-Out - Spouse-rated Anger-Out (AX, Spielberger et al., 1985); Spouse Barefoot's Ho = Spouse-rated Barefoot et al.'s (1989) refined Ho; Quality of life = QLQ-SHF (Wiklund et al., 1987).

Efficacy of intervention: Hostility-modification.

Self-reported hostility. Tables 19-20 present results of gains-analyses regressing change scores of hostility measures at post-treatment and follow-up, respectively, on pre-treatment levels and patients' group status. At post-treatment, patients' group status accounted for an additional and significant 20% of the variance in change scores of Barefoot's refined Ho, after controlling for pre-treatment levels ($F(1,18) = 4.42$, $p < .05$). Examining the means in Table 17 and the sign of the beta coefficient reveals that the reduction was greater in the treatment than in the control group. At follow-up, patients' group status accounted for an additional and marginally significant 14% of the variance in change scores of Barefoot's refined Ho, after controlling for pre-treatment levels ($F(1,18) = 2.93$, $p = .10$).

Table 18
Gains-analysis: Multiple-regression analysis regressing change in Barefoot's Ho scores at post-treatment on pre-treatment levels and group status (treatment/control).

Step	Predictor	R^2	R^2 change	F change	Beta	Significance
1)	Pre-treatment Barefoot's Ho	.00		.00	.01	95
2)	Group status	.20	.20	4.42*	.44	.05

N = 21; * $p < .05$.

Table 20
Gains-analysis: Multiple-regression analysis regressing change in Barefoot's Ho scores at follow-up on pre-treatment levels and group status (treatment/control)

Step	Predictor	R^2	R^2 change	F change	Beta	Significance
1)	Pre-treatment Barefoot's Ho	00		00	03	89
2)	Group status	14	.14	2 93	37	10

N = 21

Observed nostility. At post-treatment (see Table 21), patients' group status accounted for an additional but not significant 9% of the variance in change scores of SI-derived PH, after controlling for pre-treatment levels ($F(1,18) = 2.28$, $p > .05$). However, at follow-up, (see Table 22) patients' group status accounted for an additional and significant 18% of the variance in change scores of SI-derived PH, after controlling for pre-treatment levels ($F(1,18) = 5.88$, $p < .05$). Again, based on the means in Table 17 and the sign of the beta coefficient, reductions were larger in the treatment than in the control group.

Table 21
Gains-analysis. Multiple-regression analysis regressing change in SI-derived Potential for Hostility (PH) scores at post-treatment on pre-treatment levels and group status (treatment/control)

Step	Predictor	R^2	R^2 change	F change	Beta	Significance
1)	Pre-treatment PH	14		3 26	37	09
2)	Group status	.23	.09	2.28	.31	.15

N = 21.

Table 22
Gains-analysis: Multiple-regression analysis regressing change in SI-derived Potential for Hostility (PH) scores at follow-up on pre-treatment levels and group status (treatment/control)

Step	Predictor	R^2	R^2 change	F change	Beta	Significance
1)	Pre-treatment PH	25		7 81*	50	01
2)	Group status	43	.18	5 88*	43	.03

N = 21, * p < 05.

As shown in Tables 23 and 25, no significant effects for group status were found at post-treatment with respect to SI-derived Hostile-Style ($F(1,18) = 2.22$, $p > .05$) or with respect to SI-derived Anger-Out ($F(1,18) = .09$, $p > .05$), respectively. These negative results were found again at follow-up ($F(1,18) = .82$, $p > .05$ for SI-derived Hostile-Style; $F(1,18) = 1.19$, $p > .05$ for SI-derived Anger-Out, see Tables 24 and 26, respectively).

Table 23
Gains-analysis: Multiple-regression analysis regressing change in SI-derived Hostile-Style scores at post-treatment on pre-treatment levels and group status (treatment/control)

Step	Predictor	R^2	R^2 change	F change	Beta	Significance
1)	Pre-treatment Hostile-Style	36		11.25**	.58	00
2)	Group status	43	.07	2.22	26	.15

N = 21. ** p < .01

Table 24
Gains-analysis Multiple-regression analysis regressing change in SI-derived Hostile-Style scores at follow-up on pre-treatment levels and group status (treatment/control)

Step	Predictor	R^2	R^2 change	F change	Beta	Significance
1)	Pre-treatment Hostile-Style	38		11 51**	61	00
2)	Group status	41	03	82	16	38

N = 21 ** p < .01

Table 25
Gains-analysis: Multiple-regression analysis regressing change in SI-derived Anger-Out scores at post-treatment on pre-treatment levels and group status (treatment/control)

Step	Predictor	R^2	R^2 change	F change	Beta	Significance
1)	Pre-treatment Anger-Out	23		5 35*	47	04
2)	Group status	23	00	09	06	76

N = 21 * p < .05.

Table 26
Gains-analysis: Multiple-regression analysis regressing change in SI-derived Anger-Out scores at follow-up on pre-treatment levels and group status (treatment/control)

Step	Predictor	R^2	R^2 change	F change	Beta	Significance
1)	Pre-treatment Anger-Out	23		5 75*	47	03
2)	Group status	28	05	1 19	22	29

N = 21. * p < 05.

As in Study 1, the clinical significance of these findings was tested The mean reported reductions in Barefoot's Ho scale across both reassessments (i.e., post-treatment and follow-up, see Table 17) in the treatment-group (M = 3.25) were contrasted with those in the control group (M = -.15). Using the beta-coefficient for Barefoot's Ho scale obtained in Study 1 (beta = .189; calculated from Barefcot et al., 1989), and the difference between the groups

on the reported changes in Barefoot's Ho (difference = 3.4), yielded a reduction in risk ratio (RR) of 1.90. Thus, assuming that hostility is causally related to mortality, CHD patients participating in the proposed hostility-reduction treatment may be potentially 1.90 times at a reduced risk of mortality, or at only .53 times the risk of mortality as controls.

Second, the mean observed reductions in SI-derived PH across both reassessments (Table 17) in the treatment group ($\underline{M}$ = .55) were contrasted with those in the control group ($\underline{M}$ = -.15). The data from Dembroski et al. (1989) for the RR of SI-derived PH < 2 versus $\geq$ 3 yields a beta coefficient for PH of .525 (I assumed the investigators used a change of 1 unit in PH to calculate the RR). The reduction in RR for CHD potentially rendered by the difference in changes in observed PH between the treatment and control groups (difference = .70) is 1.44. Thus, under the assumption that hostility is causally related to CHD, CHD patients participating in the proposed hostility-reduction treatment may be potentially 1.44 times at a reduced risk for CHD, or at only .69 times the risk of CHD as controls.

Efficacy of intervention: BP-reduction.

Although analyses were conducted on reactive-BP as well, only findings relating to resting-BP are presented for the following post-hoc reasons: a) Although exceptional, a recent study found that reactive-BP did not add meaningful variance (only 1%) to the prediction of subsequent resting-BP levels beyond the variance accounted for by base-line resting-BP levels (Carroll et al.,

1995); b) As Study 2 concerns CHD patients, a clinical sample, it is clinically more meaningful to focus the attention on reducing resting-BP, a measure of hypertension and a CHD risk-factor (Kannel et al., 1986), than to focus on reactive-BP; c) Study 1 revealed that the proposed hostility-treatment did not reduce reactive-BP; d) No effects were observed for group status in relation to reactive-BP in Study 2 as well; e) Previous interventions have failed to alter this physiological parameter as well (e.g., Roskies et al., 1986); f) Due to the large variability within groups in the standard deviations of reactive-BP, the reliability of reactive-BP as measured in this research is questionable; g) Reducing the number of findings presented will clarify the overall findings from this research.

At post-treatment (Tables 27-28), patients' group status accounted for an additional and marginally significant 11% and 9% of the variance in change scores of resting-SBP ($F(1,18) = 2.75$, $p = .11$) and of resting-DBP ($F(1,18) = 2.81$, $p = .11$), respectively, after controlling for pre-treatment levels. At the two-month follow-up (tables not shown) these marginally significant effects for group status were not replicated ($F(1,18) = .01$, $p > .05$ for resting-SBP; $F(1,18) = .03$ $p > .05$, for resting-DBP).

Table 27
Gains-analysis: Multiple-regression analysis regressing change in resting-SBP at post-treatment on pre-treatment levels and group status (treatment/control).

Step	Predictor	R^2	R^2 change	F change	Beta	Significance
1)	Pre-treatment resting-SBP	.17		4 25	43	05
2)	Group status	.28	11	2 75	33	.11

N = 21.

Table 28
Gains-analysis: Multiple-regression analysis regressing change in resting-DBP scores at post-treatment on pre-treatment levels and group status (treatment/control).

Step	Predictor	R^2	R^2 change	F change	Beta	Significance
1)	Pre-treatment resting-DBP	.31		9 39**	.53	.01
2)	Group status	.40	09	2 81	31	11

N = 21. ** $p < .01$.

The gains-analyses of change in resting-SBP and -DBP at post-treatment and the means presented in Table 17 suggest that insufficient statistical power may have precluded statistically significant effects for group status. Thus, additional tests were performed to examine whether these reductions were clinically significant. These tests focused on the percentages of patients' who changed in the hypothesized manner, a test advocated by Kazdin and Wilson (1978) for testing treatments' efficacy. Considering all patients' mean age (M = 54.4 years), a cut-off of DBP $\geq$ 95 mm Hg was used to assign hypertensive status to patients (Sokolow & McLlory, 1986). This cut-off, which reflects mild hypertension, has been shown to be predictive of CHD (Rosenman et al., 1975). The percentages of normotensive patients (i.e., resting ' < 95 mm

Hg) were not significantly different at pre-treatment in the treatment group (56%) and control group (67%). However, at post-treatment, there was a significantly higher percentage of normotensive patients in the treatment-group (90%) than in the control group (50%; $X^2(1) = 4.02$, $p < .05$). A similar trend was observed when using the resting-SBP cut-off of $\geq$ 160 mm Hg. Whereas at pre-treatment, the percentages of normotensive treatment patients (89%) and controls (83%) were not different, at post-treatment, 90% of treatment patients compared with 58% of controls showed normotensive levels of resting-SBP ($X^2(1) = 2.76$, $p < .10$).

These results could not be explained by changes in patients' medication or weight occurring up to reassessments. At post-treatment, none of the treatment-patients and 36% of controls reported changes in their medication, $X^2(1) = 2.89$, $p > .05$). At post-treatment, 20% of treatment patients and 50% of controls reported changes in their weight. $X^2(1) = 2.12$, $p > .05$). At follow-up, 20% of treatment-patients and 17% of controls reported changes in their medication, $X^2(1) = .04$, $p > .05$. Finally, at follow-up, 30% of treatment patients and 42% of controls reported changes in weight, $X^2(1) = .32$, $p > .05$. Thus, the groups did not differ on medication or weight changes up to reassessments.

Efficacy of intervention: Improvement in quality of life and depression.

Quality of life. As the measure of quality of life includes separate subscales, gains-analyses were performed on each subscale. To reduce the number of tables, only tables concerning the Life Satisfaction subscale are

presented. Table 29 presents results of a gains-analysis regressing change scores in Life-Satisfaction at post-treatment on pre-treatment levels and patients' group status At post-treatment, patients' group status accounted for an additional and significant 28% of the variance in increases on the Life Satisfaction subscale scores, after controlling for pre-treatment levels ($F(1,18) = 12.46$, $p < .01$). These effects were maintained at follow-up (see Table 30), with group status accounting for an additional and significant 22% of the variance in change scores of Life Satisfaction, after controlling for pre-treatment levels ($F(1,18) = 8.46$, $p < 01$). The means in Table 17 and the beta coefficients indicated that patients in the treatment group reported greater increases in Life-Satisfaction than controls

However, no effects were obtained at post-treatment for group status in relation to the subscales of Somatic Symptoms ($F(1,18) = .56$, $p > .05$), Physical Activity ($F(1,18) = .75$, $p > .05$) or Emotions ($F(1,18) = 1.54$, $p > .05$). These negative findings did not change at the two-month follow-up ($F(1.18 = 70$, $p > .05$, for Somatic Symptoms; $F(1,18) = 20$, $p > .05$, for Physical Activity; $F(1,18) = 1.84$, $p > .05$, for Emotions).

Table 29
Gains-analysis: Multiple-regression analysis regressing change in Life-Satisfaction scores at post-treatment on pre-treatment levels and group status (treatment/control).

Step	Predictor	R^2	R^2 change	F change	Beta	Significance
1)	Pre-treatment Life Satisfaction	.32		14.54**	-.52	00
2)	Group status	.6[illegible]	28	12.46**	.53	.00

N = 21; ** $p < .01$.

Table 30
Gains-analysis: Multiple-regression analysis regressing change in Life-Satisfaction scores at follow-up on pre-treatment levels and group status (treatment/control)

Step	Predictor	R^2	R^2 change	F change	Beta	Significance
1)	Pre-treatment Life-Satisfaction	31		11 92**	- 52	00
2)	Group status	53	22	8 46**	46	.01

N = 21; ** p < .01.

Depression index. As seen in Table 31, at post-treatment, patients' group status accounted for an additional and significant 16% of the variance in reduction of depression, after controlling for pre-treatment levels ($F(1,18) = 6.68$, $p < .05$). This was maintained at follow-up (see Table 32), with patients' group status accounting for an additional and significant 13% of the variance in reduction of depression, after controlling for pre-treatment levels ($F(1,18) = 7.22$, $p < .05$). The means in Table 17 and the beta coefficients indicated that there were greater reductions in depression in the treatment-group than in the control group at post-treatment and at follow-up.

Table 31
Gains-analysis Multiple-regression analysis regressing change in depression-index scores at post-treatment on pre-treatment levels and group status (treatment/control)

Step	Predictor	R^2	R^2 change	F change	Beta	Significance
1)	Pre-treatment depression	40		16.67**	-.60	00
2)	Group status	56	16	6.68*	40	02

N = 21, * p < 05, * p < 01

Table 32
Gains-analysis: Multiple-regression analysis regressing change in depression-index scores at follow-up on pre-treatment levels and group status (treatment/control)

Step	Predictor	R^2	R^2 change	F change	Beta	Significance
1)	Pre-treatment depression	.54		30.00***	- 72	.00
2)	Group status	67	13	7.22*	35	02

N = 21, * p < 05, *** p < .001

Effect-size analyses.

Table 33 presents the d-Coefficients (Cohen, 1977) of change in each measurement-category and across all measurement-categories within each group across both reassessments (i.e., post-treatment and two-month follow-up) These coefficients were calculated with the data in Table 17. Using the criteria of Turner et al. (1995), hostility (i.e., SI-derived PH, Anger-Out, Hostile-Style and Barefoot's Ho) was moderately reduced in the treatment, and unchanged in the control group. Resting-BP (i.e., resting-SBP and resting-DBP) and quality of life (i.e., Life-Satisfaction, Physical Activity, Somatic Symptoms and Emotions) were each mildly to moderately changed in the treatment, and unchanged in the control group. Finally, depression scores were moderately to strongly reduced by the treatment, and were mildly worsened for controls. Overall, across all measurement-categories and reassessments, the hostility-treatment had a moderate effect size (d = .46) while the control group had a weak negative effect size (d = -.11).

Table 33
Effect-size d-Coefficients of treatment and control groups in each measurement-category across reassessments

Measurement category	d - Coefficient*	
	Treatment Group	Control Group
Hostility	.50	- 02
Resting-BP	35	- 06
Quality of life	33	- 16
Depression	68	- 20
Total	46	- 11

* d = (mean pre-treatment minus post-treatment scores)/SD of pre-treatment score (Cohen 1977).

Evidence for causal relations between hostility and health measures.

To determine the possible presence and magnitude of a causal relation between hostility and health outcomes, correlations were tested between change-scores of hostility measures that revealed significant effects for group status and change-scores in health outcomes that were altered by the intervention. Thus, correlations were tested between changes in Barefoot's Ho and changes in resting-BP, Life-Satisfaction and depression at post-treatment, and between changes in Barefoot's Ho and changes in Life-Satisfaction and depression at follow-up. Additionally, correlations were tested between changes in SI-derived PH levels and change in Life-Satisfaction and depression at follow-up. At post-treatment, reductions in Barefoot's refined Ho scores were significantly and positively correlated with reductions in resting-DBP ($r = .47$, $p < .05$). At follow-up, reductions in Barefoot's refined Ho scores were

significantly and positively correlated with improvements in Life-Satisfaction scores ($r = .62$, $p < .01$), and with reduced depression ($r = .53$, $p < .01$). Finally, at follow-up, reductions in SI-derived PH were positively but not significantly correlated with increases in Life-Satisfaction scores ($r = .30$, $p > .05$) or with decreased depression ($r = .26$, $p > .05$).

Discussion

This study to the best of my knowledge is the first randomized-controlled trial testing the efficacy of a hostility-reduction treatment at altering CHD-related hostility and health measures with CHD patients. The findings support the hypotheses that the treatment can reduce CHD-predictive hostility levels and positively affect CHD-related health outcomes as well. Furthermore, certain effects were maintained after two months (e.g., Barefoot's Ho, life-satisfaction and depression). These findings replicate and extend those observed in Study 1 with high-hostile students. The effect-size analysis revealed that while the information-control group yielded no changes across all measurement-categories, the hostility-treatment had a moderate overall effect-size.

The correlations observed between scores on pre-treatment measures suggest that male CHD patients with greater hostility have higher resting-BP and lower scores on overall quality of life. Additionally, patients with higher resting-DBP have lower scores on quality of life. This pattern of correlations makes sense clinically, and supports the construct validity of the measures used in this study. Patients' hostility, as rated by self-report and by the SI-

coder, was moderately to strongly correlated with corresponding spouse-ratings. Furthermore, the best predictor of health outcomes (i.e., resting-BP and quality of life) was spouse-rated hostility. This confirms previous studies that found that patients' hostility as rated by their spouses was a better predictor of ischemia than self-reported hostility (Kneip et al., 1993). Unlike previous studies which confirmed their positive treatment-effects with spouse/friends' assessments (Burell et al., 1994; Thurman, 1985a), such tests could not be conducted reliably in the current study, as half of the spouses did not complete the scales. Future hostility trials should expand efforts to involve spouses in the assessment of patients (Barefoot & Lipkus, 1994), in light of the strong concurrent and predictive validity these assessments had in the current study.

That both self-reported (Barefoot's Ho) and observed (SI-derived PH) levels of hostility (rated by an observer blind to group status) were reduced significantly more by the hostility-reduction treatment than by the information-control group suggests that the proposed treatment indeed reduces CHD-predictive hostility. This conclusion is supported by the effect-size analysis that revealed a moderate effect-size across all hostility measures for the treatment-group alone. At post-treatment, only self-reported hostility was altered, while at follow-up, reductions in observed behavioral hostility (PH) increased and reached statistical significance. This may result from one of two reasons. First, patients may perceive their hostility to be reduced before it is objectively detected to be reduced. Second, since the majority of items in Barefoot's Ho

reflect cynicism, hostility's cognitive component (Barefoot, 1992; Barefoot et al , 1989), reductions on this scale may reflect primarily reduced cognitive hostility Thus, it may be that changes in cognitive hostility (assessed with Barefoot's Ho) precede changes in behavioral hostility (PH). Such a process follows models of hostility which place cognition as underlying hostile behavior and affect (Chesney, 1985; Powell, 1992).

Contrary to what was hypothesized, the proposed treatment did not reduce levels of SI-derived Anger-Out or Hostile-Style Unlike these results, levels of SI-derived Anger-Out were reduced in Study 1 with high-hostile students. Inspection of Table 17 and the results of the gains-analyses suggests that insufficient statistical power may have precluded the achievement of statistical significance for Hostile-Style at post-treatment and for Anger-Out at follow-up, as both were reduced by the intervention, but not sufficiently to attain statistical significance. Additionally the inter-rater reliability of both Anger-Out and Hostile Style was not high in Study 2 (.65 and 60, respectively), suggesting that measurement-error may have masked potential group effects.

The reductions in hostility were on measures that have been shown to predict CHD, CHD-progression and mortality (Barefoot et al., 1989, Dembroski et al., 1989; Helmers et al., 1993, Powell & Thoresen, 1985). Reducing levels of SI-derived PH, primarily a measure of Antagonism (Musante et al., 1989), suggests that hostility's most toxic component of antagonistic behavior may have been altered by the treatment (Dembroski et al., 1989, Siegman, 1993).

Extrapolating the results to previous epidemiological studies (Barefoot et al., 1989; Dembroski et al., 1989), and assuming that hostility is causally related to CHD and mortality, CHD patients participating in the hostility-treatment are hypothetically 1.44 times at a reduced risk for CHD and 1.90 times at a reduced risk for premature mortality than controls. Thus, this treatment may have preventative value in relation to CHD-progression. The reductions in depression (discussed below) may add to this preventative value as depression is an independent predictor of mortality after MI (Frasure-Smith et al., 199?).

These findings support those of previous clinical trials (Burell et al., 1994; Mendes De Leon et al., 1991) that showed reductions on observed hostility measures with CHD patients. The findings in Study 2 with respect to SI-derived PH are comparable to those of Roskies et al. (1986), who treated healthy Type-A managers. Thus, Type-A treatment may yield similar reductions in hostility as a hostility-treatment alone. However, the more important question would be to compare the two treatments in relation to CHD-prevention. Turner et al. (1995) did not demonstrate reductions in hostility as assessed with the full Ho scale. Unlike all these studies, the present study focussed on reducing CHD-predictive hostility alone rather than modifying the global TABP. As hostility may be the most "toxic" component of the TABP (Dembroski et at., 1989; Williams, 1987), the proposed treatment may be more effective at improving the prognosis of CHD patients than Type-A modification (Chesney, 1985; Dembroski & Costa, 1987). Testing the effects of the proposed hostility-treatment on long-term

outcomes (e.g., reinfarct) would be the next step in validating this claim.

At post-treatment, a significantly greater percentage of patients in the treatment-group (90%) had normotensive levels of resting-DBP than controls (50%), and a similar trend was observed with resting-SBP. Although based on retrospective and subjective reports, the treatment and control groups did not differ significantly on changes in medication or weight occurring until reassessments. Thus, one may infer with greater validity that the experimental manipulation yielded the reductions in BP. The reductions in resting-DBP are statistically as well as **clinically** significant, since this cut-off point reflects a cut-off point of hypertension (e.g., Peled-Ney et al., 1984), and since it has been related prospectively to CHD-incidence (Rosenman et al., 1975). The actual reductions in resting-DBP (5.6%) are smaller than those found by Achmon et al. (1989) following an anger-modification group (11%). Their sample consisted of patients with essential hypertension without CHD. Employing a more homogeneous sample than in the current study may have increased the effects of their treatment. In the current study, some patients may have had essential hypertension (prior to CHD) while others' hypertension may have been related to their CHD, making the response of the treatment-group more heterogeneous

Significantly greater increases were found in the treatment group with respect to life-satisfaction, and this effect was maintained at follow-up. This result contrasts with those of a previous study where brief psychotherapy did not improve life-satisfaction for post-MI patients (Rahe et al., 1979). Improving

life-satisfaction, a measure of quality of life, is central to the treatment of CHD patients, who show high levels of psychosocial maladjustment after a CHD event (Kaplan, 1988; Wiklund et al., 1984). Additionally, significant reductions on the depression index were reported, and these findings were maintained at follow-up as well. These findings may have rehabilitative importance as Frasure-Smith et al. (1993) found that 16% of post-MI patients met criteria of major depression, and Wiklund et al. (1984) found that high rates of depression remained one year after MI compared to non-CHD controls. As mentioned above, the reductions found in the current study may also have prognostic value as depression has been shown to be a significant and independent predictor of post-MI mortality (Frasure-Smith et al., 1993). Stern et al. (1983) also found greater reductions in depression in a group of post-MI patients who were given Type-A modification than a control group. However, Burell et al. (1994) and Rahe et al. (1979) did not achieve reduction in depression levels. Although the depression index used in the current study was internally reliable and the effect was replicable, this finding should be taken with caution as a standardized depression scale was not used.

Significant and positive correlations were observed between changes in hostility and resting-DBP, life-satisfaction and depression. As these correlations were obtained between **change-scores** derived from a randomized-controlled-trial, and since patients did not differ on these measures at pre-treatment (Table 17), these results suggest a **causal** relation between hostility (assessed with

Barefoot's Ho) and resting-BP, Life-Satisfaction, and depression. These findings replicate those first shown by Davison et al. (1991) in relation to resting-BP. However, the latter study did not attempt to systematically modify hostility nor did it employ randomization. Thus, the current study represents a more rigorous demonstration of a dose-response relation between hostility and resting-BP. These results are important for understanding the etiological role of hostility in CHD, as hostility is related to resting-BP (e.g., Mann, 1977), and resting-BP is a CHD risk factor (Kannel et al., 1986). Possible explanations of these causal relations (e.g., epinephrine-reduction, increased adherence to taking medication) will be offered in the last chapter.

This study had several methodological limitations. First, the small sample size limited the trial's statistical power for detecting additional potential effects (e.g., SI-derived Hostile-Style). However, given that statistically significant effects were observed in a small sample, and given the results of the effect-size analysis, the effects of the treatment are relatively strong. Second, long-term CHD outcomes (e.g., recurrent MI) were not included, and these represent more "hard" outcomes. Third, BP-reduction was not maintained at follow-up. An additional "booster session" after treatment may be required to maintain certain effects (Turner et al., 1995). Finally, many statistical tests were conducted. However, most of the results had a uniform and predicted pattern, and preventing type-I errors seemed less important than preventing type-II errors (i.e., not failing to find/report effects) in a first trial whose results

could direct future trials This issue will be discussed in further detail in the next chapter.

In summary, preliminary support was provided for the proposed treatment's efficacy in reducing hostility, resting-BP, life-dissatisfaction and depression, all risk factors and outcomes related to CHD and mortality (Barefoot et al., 1989; Dembroski et al., 1989; Frasure-Smith et al., 1993, Kanniel et al., 1986; Wiklund et al., 1984) Thus, this treatment may have preventative and rehabilitative effects. In addition, causal relations between hostility and CHD-related health-measures were suggested. These findings provide further support to the growing evidence for the role of hostility in the etiology and treatment of CHD (Dembroski & Costa, 1987; Miller et al., in press, Smith, 1992, Williams, 1987).

CHAPTER EIGHT

GENERAL DISCUSSION

The final chapter of this thesis will summarize its purposes and major findings. The two clinical trials will be compared and integrated, and possible explanations for the causal relations found in Study 2 will follow. This chapter will conclude with a detailed description of the limitations of this research, suggestions for future research directions and, final conclusions.

Purposes of research

This research had three main purposes: 1) To develop a brief and focussed hostility-reduction intervention; 2) To tests the effects of this intervention on high-hostile healthy students (Study 1); and 3) To test the effects of the intervention on hostility and CHD-related health measures of high-hostile CHD patients (Study 2). Study 2 also allowed the examination of hypothesized causal relations between hostility and CHD-related health measures (e.g., resting-BP, quality of life).

The development of the hostility-reduction treatment was based on: 1) Current theory in coronary-prone behavior (Chesney, 1985; Dembroski & Costa, 1987; Smith, 1992; Siegman, 1993; Williams, 1987); 2) The conceptualization and definition of CHD-predictive hostility (e.g., Barefoot, 1992; Chesney, 1985); 3) Empirical findings that demonstrate the hostility-CHD link (e.g., Almada et al., 1991; Barefoot et al., 1989; Barefoot et al., in press; Dembroski et al. 1989; Ironson et al., 1992; Powell & Thoresen, 1985; Williams et al., 1980), and 4)

Previous related clinical trials and literature (e.g., Deffenbacher et al., 1987; Novaco, 1976b; Roskies, 1987; Williams & Williams, 1993).

This thesis's second and third purposes, to test the effects of the intervention on healthy and cardiac samples, were seen as an opportunity to replicate and extend findings from one type of subjects (students in Study 1) to another (CHD patients in Study 2). Replication is rarely done in behavioral medicine, yet, it was seen as a more compelling manner of providing evidence for an intervention's efficacy than conducting one clinical trial with a larger sample. Replication allows one not to rely too much on tests of statistical significance, but, rather to examine the reproducibility of results. The latter is perhaps a more realistic test of "chance findings" than the former. Study 1 allowed me to test the treatment's effects without having the complications of CHD confound the findings. Study 2 allowed me to test the treatment's efficacy in the context of CHD, with the aim of altering CHD-related health problems (e.g., hypertension; depression) in addition to altering hostility, the main purpose of both studies.

The main findings of this research

Before reviewing the main findings, it is important to consider a few criteria that have been suggested for evaluating the findings and effectiveness of psychotherapies. Detection of statistically significant effects provides researchers one way for inferring effectiveness of treatments beyond the effects of chance. Among several criteria, Kazdin and Wilson (1978) suggested to add

the following ones: The endurance of effects (i.e., their maintenance across time), the breadth of effects (i.e., changing parameters targeted and not directly targeted by the treatment), proportion of improved patients (i.e. proportion of patients who gained from treatment, rather than emphasis on mean group changes) and importance of change (i.e., where the effects clinically significant). These criteria will be referred to throughout the following sections

Study 1 demonstrated that reductions in self-reported hostility (Barefoot's refined Ho) and observed hostility (SI-derived Anger-Out) were significantly greater for high-hostile students assigned to the hostility-reduction treatment than for those assigned to a single-session information-control group. As these effects were obtained from different sources (i.e., students and SI-coders), Study 1 suggests that the treatment can reliably reduce levels of CHD-predictive hostility. However, levels of SI-derived PH were not reduced. Psychometric limitations of a one-item scale (i.e., insufficient sensitivity) may have accounted for the null effects with SI derived PH. Additionally, the treatment may have not altered hostility that is **manifested during** the SI, but did alter hostility levels that are primarily based on self-report (i.e , Barefoot's refined Ho and SI-derived Anger-Out). Study 1 also failed to alter reactive-BP, and subjects assigned to the hostility-treatment exhibited significantly greater increases (rather than decreases) in reactive-SBP than controls. Psychometric limitations (i.e., large within-group variability) and insufficient statistical power may explain the lack of effects with reactive-DBP. In addition, familiarity with

the SI may have reduced reactive-SBP in controls, whereas tension induced by exaggerated expectancies after the intervention may have increased reactive-SBP in treatment subjects (Suarez & Williams, 1990). Nevertheless, Study 1 showed that it is feasible to administer the treatment to a sample of high-hostile students, and that the treatment can reduce CHD-predictive and mortality-predictive hostility levels.

Finally, 69% of high-hostile candidates for the student trial did not take part in Study 1. Whereas this reduces the study's generalizeability, it may reflect the uncooperative and mistrusting nature of this population (Barefoot, 1992; Dembroski & Costa, 1987) or may be an example of the non-adherence of hostile individuals to health and self-care behavior (e.g., Leiker & Haily, 1988). Future interventions with healthy high-hostile "coronary-prone" individuals should consider this issue when planning a clinical trial.

Study 2 replicated and extended the findings of Study 1 to CHD patients. Study 2 showed that self-reported hostility (Barefoot's refined Ho) and observed hostility (SI-derived PH) were reduced significantly more by the hostility-treatment than by the information-control group. Reductions in Barefoot's Ho scores tended to be maintained after two-months, meeting the criterion of endurance. However, reductions in PH were large enough to reach statistical significance only at follow-up. Study 2 also showed that a larger proportion of subjects assigned to the hostility-treatment had normotensive resting-DBP at post-treatment compared with controls, and this tended to occur with resting-

SBP as well. Thus, the treatment had statistically and clinically significant effects at post-treatment. These effects met the criteria of proportion of improved patients and the importance of therapeutic effects. However, BP-reduction disappeared at follow-up. Termination of treatment may have caused this effect to disappear, suggesting that high-hostile CHD patients may require constant "doses" of hostility-reduction treatment or booster-sessions for maintaining therapeutic gains of BP-reduction (e.g., Turner et al., 1995).

Study 2 also found that the best predictor of patients' resting-BP at pre-treatment was their hostility as assessed by their spouses (Barefoot's Ho). This is in line with a previous study on ischemia (Kneip et al., 1993), and highlights the importance of using spouse-ratings for the assessment of hostility (Barefoot & Lipkus, 1994). Future trials should encourage more spouses to assess patients' hostility before and after treatment, in light of its prognostic value.

Study 2 showed that the hostility-reduction treatment increased patients' life-satisfaction and reduced their levels of depression. These effects were maintained at follow-up. These results met the criteria of breadth and endurance of effects of treatments. Since life-dissatisfaction and depression are common outcomes in CHD, and since depression has prognostic value (Frasure-Smith et al., 1993; Wiklund et al., 1984), their reduction may have rehabilitative as well as preventative value. Finally, correlations were observed between change scores in hostility and health-outcomes: Reductions in Barefoot's Ho were positively and significantly correlated with reductions in

resting-DBP and depression scores, and with increases in life-satisfaction.

Study 2 failed to reduce SI-derived Anger-Out and Hostile-Style. Nevertheless, the means on these measures changed in the treatment group in the hypothesized manner. Possible measurement-error (as reflected by the moderate inter-rater reliabilities of these measures), insufficient statistical power resulting from the small sample size, and the heterogenous response to therapy (as seen in the statistical assumptions) may have caused these null findings. In addition, no effects were found with respect to other scales of quality of life (i.e., Somatic Symptoms, Emotions and Physical Activity). However, the fact that scores of the Life-Satisfaction subscale changed while other self-report quality of life subscales did not argues against a demand-characteristics bias in Study 2, as this observed effect is so specific.

Taken together, the findings of Study 2 suggest the five following conclusions. First, hostility was indeed reduced by the hostility-treatment. As the hostility measures that were altered predict CHD and mortality (Barefoot et al., 1989; Dembroski et al., 1989; Helmers et al., 1993; Matthews et al., 1977; Mendes De Leon, 1992), the treatment reduces CHD-predictive and mortality-predictive hostility levels. These findings were found despite the incredible stability that hostility measures show (Barefoot et al., 1983; Barefoot et al. in press; Shekelle et al., 1983). Second, the hostility-treatment had positive effects on health outcomes that predict CHD such as resting-BP (Kannel et al., 1986), depression (Frasure-Smith et al., 1993) and outcomes that result from

CHD such as life-dissatisfaction (Kaplan, 1988, Wiklund et al., 198[illegible]). This suggests that the treatment may positively affect patients' prognosis and rehabilitation. Third, as significant correlations were obtained between **changes** in hostility and CHD-related outcomes from a randomized-controlled trial, after having shown no group differences on these measures at pre-treatment, hostility may be **causally** related to resting-DBP, life-satisfaction and depression. These findings provide further support for the hypothesized causal role hostility has in CHD (Smith, 1992). It is also possible that short-term changes in health status (i.e., BP) and possible long-term changes not assessed in the current study (e g., longevity) may be accounted for by the observed reductions in depression, since depression is an independent predictor of mortality after MI (Frasure-Smith et al., 1993). Fourth, under the assumption that hostility is causally related to CHD and mortality, the findings of studies 1 and 2 regarding hostility-reduction were extrapolated to previous epidemiological studies (i.e., Barefoot et al., 1989; Dembroski et al., 1989). The results of this extrapolation suggest that subjects undergoing this intervention are **hypothetically** 1.44 times at a reduced risk of CHD and between 1.90 to 2.13 times at a reduced risk of premature mortality than subjects assigned to the control group. While these findings must be taken with considerable caution, as CHD and mortality were not assessed as outcomes, and as the above assumed causal relations have not been confirmed, they suggest that the treatment's effects may be **clinically** significant as well. Finally, fifth, the

effect-size analysis suggests that the treatment has a moderate overall effect.

These findings suggest that the hostility-treatment showed endurance, breadth, and a greater proportion of treatment-patients showed clinically significant benefits (showed normotensive BP levels) than controls. All these achievements suggest that the treatment **is** effective according to the criteria of Kazdin and Wilson (1978).

The fact that the hostility-treatment had a "vertical" impact by affecting psychological parameters (hostility, life-satisfaction, depression) and physical parameters (resting-BP), confirms findings of previous psychological trials (e.g., Burell et al., 1994). This "vertical" effect may explain why psychological treatments may be clinically more effective than medication treatments at secondary prevention of CHD (Davidson et al., 1995). Whereas medication may have very specific effects (e.g., inhibition of platelets induced by aspirin), psychotherapy may affect several significant psychological and traditional CHD risk factors, which together yield a greater cumulative effect.

Integrating and comparing studies 1 and 2

As has been mentioned throughout the thesis, Study 2 was a replication and extension of Study 1. The common finding from both studies is that the hostility-reduction intervention reduced CHD-predictive and mortality-predictive hostility levels. The fact that hostility-reduction was observed in two samples that differ in age and health status, suggests that this effect holds across clinically different samples. Thus, replicability and generalizeability have been

demonstrated by the two trials. Replication may be a more compelling way to test a treatment's efficacy than **inferring** replicability from tests of statistical significance (i.e., that an effect is not due to chance). Furthermore, Study 2 was not merely an identical replication of Study 1, but a "constructive" replication, since it extended our knowledge of the intervention's efficacy to a cardiac sample and to other outcomes.

Study 2 includes several methodological improvements that extend the findings of Study 1. By adding a two-month follow-up, the endurance of certain effects was demonstrated (i.e., self-reported hostility, life-satisfaction and depression). By adding CHD-related health-outcomes, the breadth of the interventions' efficacy has been shown.

Comparing the reductions in hostility, Study 1 may have succeeded in altering mainly reported hostility (i.e., Barefoot's Ho and SI-derived Anger-Out), as even SI-derived Anger-Out is primarily based on subjects' reports during the SI. In contrast, Study 2 may have reduced reported **and** observed hostility (Barefoot's Ho and SI-derived PH). There is evidence from Study 2 to validate both reductions. Two findings in Study 2 support the validity of self-reported reductions in hostility: The significant and strong correlation between patients' and spouses' scores on Barefoot's Ho at pre-treatment ($r = .76$), and the significant correlation between change in self-reported Barefoot's Ho scores and reduced resting-DBP ($r = .47$), an objective measure.

Reductions in PH constitute a more objective reduction of hostility than

reductions in SI-derived Anger-Out for several reasons. First, unlike SI-derived Anger-Out, ratings of PH are influenced by subjects' reports **and** actual antagonism exhibited during the SI (Dembroski & Costa, 1987). Second, the treatment may have reduced these overt manifestations as well, which are assessed with SI-derived Hostile-Style. Although not statistically significant, there were descriptively and consistently larger reductions in SI-derived Hostile Style in the experimental group than in the control group (see Table 17). Third, ratings of PH and Hostile Style were moderately to strongly correlated ($r = .72$). As reductions in SI-derived PH reflect more objective changes in behavior than do reductions in SI-derived Anger-Out, the results of Study 2 may be less open to demand characteristics biases than are those of Study 1: Subjects may be able to modify **what** they report, but it may be more difficult to modify **how** they report their hostility. Had Study 2 yielded significant reductions in Hostile Style as well, this argument would have received further support.

Changes in observed behavioral hostility (PH) reached statistical significance in Study 2 only at follow-up, while changes in self-reported hostility (Barefoot's Ho) were already seen at post-treatment. Changes in self-reported hostility mainly reflect reduction in cognitive or cynical hostility, as Barefoot's Ho includes mainly items assessing the Cynicism subscale, whose scores are highly correlated with total Barefoot's Ho scores (in Study 2: $r = .90$). It is possible that only after a certain time has passed, and after cognitive hostility has been changed, that overt manifestations of behavioral hostility assessed by

PH are reduced and become observable to coders. These reductions in PH might have been detected in Study 1 had there been a follow-up as in Study 2. It is possible that patients needed more time to rehearse the material learned in treatment for behavioral changes to be sufficiently manifested

In accordance with cognitive theories, alteration of hostile cognitions (Barefoot's Ho) may precede alteration of hostile behavior (SI-derived PH; Chesney, 1985; Powell, 1992). However, these findings may only be a consequence of lack of statistical power, as PH scores were descriptively reduced by the hostility-treatment even at post-treatment in Study 2. In addition, the possibility that cognitive changes may have preceded behavioral ones goes against the order hypothesized to take place when developing the treatment. It was expected that hostile behavior, a more concrete dimension, would be modified before enduring hostile cognitions were modified, in line with cognitive dissonance theories (e.g., Aronson & Mills, 1959). Future, large-scale trials can resolve this issue more definitively by assessing the three dimensions of hostility separately at each reassessment with the three subscales of Barefoot's Ho or with the BDHI subscales (Buss & Durkee, 1957).

Finally, the enrollment rate was more successful in Study 2 (63%) than in Study 1 (31%). CHD patients may have had more motivation to participate in such a clinical trial after having had a MI or an UA, compared with young and healthy students. For most students the hostility-CHD relation was a theoretical relation, while CHD patients could comprehend this relation more directly via

their own experiences. These differences should be considered in future research, and means for increasing subjects' enrollment should be used. Students may be encouraged to participate in such a trial by providing them with a portion of their course-credits. Advertising the importance of hostility in CHD in the media may motivate healthy, but at risk candidates to participate in future trials. Controls should be offered in advance the option of a complete treatment, after completion of the entire trial.

Possible mediators underlying the relations between hostility and health-outcomes

This section provides possible explanations for the observed relations between hostility and resting-BP and quality of life measures. These explanations are based on converging evidence from previous studies and should be validated in future research, as the hypothesized mediators were not assessed in the current studies.

Hostility and resting-BP. The observed correlation between reductions in hostility (Barefoot's Ho) and resting-DBP replicate those first reported by Davison et al. (1991). As with Barefoot's Ho, the measure of hostility used by Davison et al. (1991), the articulated thoughts during simulated situations (ATSS; Davison Robins & Johnson, 1983) also predominantly assessed cognitive hostility. Thus, both studies suggest that change in cognitive hostility is associated with change in resting-BP. As mentioned earlier, the correlation observed in the current study represents more rigorous, direct and systematic

evidence for the causal relation between hostility and resting-BP.

How is this relation possible? Previous studies have shown that hostility, particularly antagonistic hostility, is related to resting-BP (Mann, 1977) However, which physiological parameter/s is/are common to hostility and resting-BP? Based on evidence from previous studies, I would like to suggest that epinephrine and norepinephrine are the link between hostility and resting-BP, and this may be inferred from several findings First, Davison et al. (1991) found that among subjects initially high on epinephrine, hostility-reduction was positively correlated with reduced-BP, whereas this correlation was not present among subjects initially low on epinephrine. Thus, epinephrine mediates the relation between change in hostility and change in resting-BP. Second, drugs which block beta-adrenergic receptors (e.g., propranolol), and thus block the effects of epinephrine, have been shown to reduce hostility as well (Krantz et al., 1982; Schmieder et al., 1982). Third, in a recent experiment, newlywed couples, who were coded as high-hostile during a marital conflict, reacted with greater elevations of epinephrine and norepinephrine than their low-hostile counterparts (Malarkey, Kiecolt-Glaser, Pearl & Glaser, 1994). Thus, elevated hostility is related to elevated epinephrine and norepinephrine. Finally, elevations in epinephrine can increase cardiac output and elevations in norepinephrine can increase vascular resistance, two main factors which elevate resting-BP (Julius et al., 1985). Therefore, hostility may enhance the sympathetic nervous system as indexed by elevations in epinephrine and

norepinephrine, which in turn cause elevations in resting-BP. This hypothesized chain of events is in line with the psychophysiological reactivity model of hostility and CHD (e.g., Williams et al., 1985).

The constitutional vulnerability model of hostility and CHD (e.g., Krantz & Durel, 1983) may claim that hostility results from elevated BP that results from a constitutional enhanced sympathetic nervous system. An experimental study may resolve this issue of directionality.

Finally, hostility may be causally related to BP-reduction via increasing patients' adherence to medication-taking (Lee et al., 1992). Although not all patients were on antihypertensive medication, several types of medications may indirectly reduce BP (e.g., beta-blockers) by reducing cardiac output and/or vascular resistance. Future trials may wish to reliably assess adherence to treatment as a possible mediator between hostility and health outcomes, confirming to the health-behavior model (Leiker & Hailey, 1988).

Hostility, life-satisfaction and depression. The following discussion will consider the observed correlations found between hostility-reduction and increases in life-satisfaction and decreases in depression together, as life-satisfaction and depression reflect similar psychological constructs that were assessed in the current study by the same measure (QLQ-SHF, Wiklund et al., 1987).

How is hostility-reduction related to increased life-satisfaction and reduced depression? One possibility, that I wish to reject, is that since these

parameters were all assessed with self-report measures, these relat' ns may reflect biases related to Neuroticism (e.g., negative thoughts and feelings and a pessimistic view of life) Although Neuroticism was not assessed, Barefoot's Ho is mildly related to Neuroticism (Barefoot et al , 1989; Davidson & Hall, 1995) This reduces the possibility that Neuroticism mediated these relations

The second possibility that I wish to suggest, is that increased social support may mediate these relations in the following way First, hostility has been previously associated with depression and lack of social support (Smith & Frohm, 1985) Second, hostile attributions have been shown to be related to greater hostile behavior in subjects holding such beliefs, and to elicit greater hostile behavior in people interacting with these subjects The environment's hostile reaction then serves as behavioral confirmation for the first subject's hostile attributions, who then reacts with more hostile thoughts and behaviors (Snyder & Swann, 1978) The hostility-reduction treatment may have broken this cycle, a cycle central to the transactional model of hostility and CHD (Smith, 1992). Thus, subjects who underwent treatment for reducing their hostility reported lower levels of cognitive hostility (Barefoot's Ho) and were rated as less behaviorally hostile (SI-derived PH) The cognitive changes may have altered patients' perceptions of their environment from negative to positive ones (or at least neutral), and the behavioral changes may have actually elicited more supportive and less hostile reactions from patients' intimates (e g , family, friends). Perceptions of positive environmental responses and their

actual elicitation may have resulted in patients perceiving increased social support. Perceptions of increased social support may have then increased patients' life satisfaction (Linn & McGranaham, 1980) and reduced their depression (e.g., Warheit, 1979). This hypothesized chain of events is in line with the psychosocial vulnerability and transactional models linking hostility with CHD (Smith, 1992). Including spouse measures of patients' social support and familial conflicts may help to elucidate this model as well.

Evaluation, limitations and criticism

In order to evaluate the two trials with more objective means, the checklist for statistical review of papers on clinical trials for the British Medical Journal (Gardner et al., 1989) was employed. This scale was used to evaluate the clinical trials in Chapter Five. Study 1, the student trial, was rated 39 on this 1-45 scale or achieved 87% of the maximal rating. The only study reviewed in Chapter Five with healthy individuals that achieved a higher score was that of Thurman (1985a). Study 2, the CHD trial, was rated 41 on this scale or achieved 91% of the maximal rating. None of the clinical trials reviewed in Chapter Five obtained this score. These ratings are comparable to the mean methodological rating of eight medical trials, randomly selected from meta-analyses of eight types of drug treatments for post-MI patients (Davidson et al., 1995). However, Studies 1 and 2 included several limitations not tested by this rating system, which are discussed below.

First, both trials included small samples. This made it difficult to detect

significant effects for BP in Study 1 and Study 2 (with the gains-analysis), and for SI-derived Hostile-Style in Study 2. In addition, the small number of subjects, particularly the low enrollment rate in Study 1 (31%) reduces one's ability to generalize the findings to other high-hostile healthy young men or to other CHD male patients. However, both sample sizes were sufficient, according to power analyses conducted prior to the research, for detecting significant effects with respect to hostility-reduction, the main aim of both studies. In this regard, power analyses for detecting other effects (e.g., BP-reduction) should have been conducted prior to the study. Despite this limitation, the fact that statistically significant effects were found with small sample-sizes suggests that the effect-sizes were relatively **strong**. This conclusion is supported by the effect-size (as opposed to power) analysis conducted in Study 2, particularly for hostility and depression.

Second, and related to the issue of sample size, was that too many statistical tests were conducted considering the sample sizes. This seriously opened the possibility for Type-1 errors, i.e., incorrectly rejecting null hypotheses (Keppel & Zedeck, 1989). The following four points, however, restrict this error. First, the tests were planned and were theoretically based. Second, tests of group equality, tests of construct validity at pre-treatment, tests of the intervention's efficacy and tests of causality are conceptually different tests, and may not be seen as additive. Some of these tests (e.g., group equality and construct validation) were conducted only to test the internal

validity of the study and to support its' conclusions. Tests of the efficacy of the intervention examined the main question, and are additive. Third, the **pattern** of all the results, particularly in Study 2, is consistent with the predictions and is theoretically coherent (i.e., most means changed in the hypothesized manner, variables were correlated as expected). This reduces the chances for Type-1 errors. Fourth, preventing type-2 errors (i.e., failing to find/report significant results) may be more important for a first trial than preventing type-1 errors (failing to accept null hypotheses), since new findings guide future. large-scale trials Thus, corrections for multiple tests (e.g., Bon Feronni) were not performed to avoid further loss of statistical power and loss of important information.

The third limitation of this research is that neither study examined long-term outcomes (prevention of MI or death). However, such tests were not planned for initial research on the preliminary effects of hostility-modification. The main aim of this research was to test whether CHD-predictive hostility levels can be reduced in high-hostile healthy and CHD men by the proposed treatment. The second aim was to test the immediate health consequences of hostility-reduction (i.e. reducing resting-BP, depression and improving quality of life) in CHD patients. Large-scale studies are expensive and testing outcomes such as reinfarct require long follow-ups. Finding the drawbacks and potential of a new treatment is more appropriate to do with a small-scale study. Thus, Studies 1 and 2 can be viewed as pilots for a large-scale multi-centre trial on

hostility-modification and CHD

The fourth limitation is that the effects of interactions of group status with certain third variables such as CHD-severity or age on outcomes was not tested. Friedman et al (1986) found that Type-A treatment was successful at reducing cardiac morbidity only among post-MI patients who had a mild MI Similarly, tests of therapeutic-efficacy were not performed separately for MI and UA patients. Such tests could not be done with the small sample sizes in the current research, and are needed in future trials Nevertheless, the treatment and control groups did not differ at pre-treatment on these third variables (e g., age, CHD-event, revascularization procedures)

Fifth, in absolute terms, more compensation was provided in both trials to treatment subjects than to controls, and this may have affected the results. However, considering the number of sessions and subjects' expenses (travel, time), compensation was actually lower for treatment-subjects than for controls This may have helped in eliciting greater change in hostile attitudes and behavior. Using cognitive dissonance theory and the concept of effort justification, providing less money together with requiring more effort from treatment-subjects may have actually increased therapeutic efficacy (Axsom & Cooper, 1984). Thus, attempting to avoid cognitive dissonance between a hostile attitude towards the program on the one hand, and the behavior of attending the sessions on the other hand, subjects may have altered their attitudes towards the program and altered their hostile attitudes in general.

Furthermore, a dissonance between the efforts that treatment-subjects had to make (i.e., attending eight sessions, daily monitoring of hostility and use of skills) on the one hand, and noticing no change in their hostility on the other hand, may have caused subjects to justify their efforts by altering their hostility.

Sixth, the focus of the treatment may have been too specific, and other, significant psychological CHD risk factors not derived from the TABP such as depression (Frasure-Smith et al., 1993) or social isolation (Ruberman et al., 1984) need to be considered (Chesney, 1985). However, as a first clinical trial on modifying hostility, the most toxic component of the TABP (e.g., Dembroski et al., 1989), it was important to isolate the hostility complex and examine the feasibility of modifying this stable psychological parameter. Furthermore, the treatment, although not focusing on depression, did reduce levels of depression. Nevertheless, from a theoretical, empirical, clinical and ethical point of view, future trials may wish to develop treatments which target factors such as depression and social isolation, in addition to hostility (Chesney, 1985). Such trials may compare the preventative value of a hostility-treatment with those of treatments aimed at reducing depression, social isolation, and a treatment targeting all three factors in CHD patients.

Seventh, control subjects did not receive the same number of sessions as did treatment subjects. Thus, receiving more attention may have accounted in part for the observed effects (O'Leary & Borkovec, 1978). However, as the study by Mendes De Leon et al. (1991) suggests, **type** of therapeutic contact

may be the critical aspect in the effects of amount of therapeutic contact on behavioral change. In their study, positive correlations between number of sessions attended and behavioral change were found only for subjects undergoing psychological treatment (Type-A counseling and cardiac counseling) but not for controls (cardiac counseling alone) Thus, it is possible that only treatment-subjects benefitted from having more therapeutic contact in the current study, due to the type and content of this contact The aim of the control group used in the current study was to remove the effects of information, expectancies for improvement, contact with a group and with therapists, and provision of information to at-risk patients However, clearly, these were not fully controlled for by the single-session information control group For ethical reasons, controls were offered the opportunity to participate in the hostility-reduction treatment at the end of the research.

Eighth, it may have been conceptually and methodologically more compelling to have included a third group for which there would be an attempt to modify the overall TABP Had this group yielded weaker effects on hostility and CHD-related outcomes than the hostility-treatment, this would have been a strong support for the claim that hostility is the toxic component of the TABP (Dembroski & Costa, 1987; Williams 1987). This was not done as this trial attempted first to isolate and focus on hostility alone, and examine whether it is possible to modify it. Additionally, as other TABP components may not be CHD-predictive (e.g., achievement; Dembroski et al., 1989), it may be unethical

to provide such a treatment to CHD patients.

Ninth, although a matching procedure was incorporated in the design of both studies, comparisons were not performed within matched pairs of subjects assigned to the different experimental conditions. Subject attrition and problems occurring at random (i.e., one student assigned to the control group inadvertently came to the treatment-group) altered a few matched pairs, and this did not permit comparisons within pairs. Furthermore, the matching was mainly a methodological procedure (Eysenk & Grossarth-Maticek, 1991) aimed at ensuring group-equality prior to commencement of treatment, for enhancing the trials' sensitivity to detect potential treatment-effects.

Finally, the effects of the hostility-treatment may have been weakened due to recruiting subjects/patients who were high on cynical and/or antagonistic hostility. Providing a treatment for reducing cynicism to cynical subjects, and a separate treatment for reducing antagonism to antagonistic subjects may have yielded stronger therapeutic effects. However, to avoid loss of further statistical power via sample-reduction, subjects/patients with different hostility profiles were treated and tested together. This matched the multicomponent treatment that was developed in this thesis. Since hostile people may present with different combinations of the three components of hostility (Barefoot, 1992), a multcomponent treatment is more suitable for heterogenous samples. Future trials may wish to compare the effects of providing multimodal treatments with single-modality treatments that are matched to the profile of their subjects.

Future research directions

Based on the effect-size analysis (Cohen, 1977), the proposed hostility-treatment had an overall moderate effect-size across measures and reassessments. Future trials using this treatment may profit by measuring hostility and depression. For resting-BP and perhaps other objective outcomes (e.g., reinfarct), larger samples will be required for the treatment's observed mild to moderate effect to reach statistical significance.

The first two studies that would be recommended to follow this research are a replication of Study 1 with larger samples of healthy subjects and long-term outcomes to test the primary prevention efficacy of the treatment, and a replication of Study 2 with larger samples of CHD patients and long-term outcomes to test the secondary prevention efficacy of the treatment. These trials could examine effects that were tested in the current research with greater statistical power, and examine "hard" CHD outcomes such as MI and cardiac death (e.g., Burell et al., 1994; Friedman et al., 1986). Such trials would need to involve several medical centers, to guarantee a sufficient number of patients. A secondary prevention multi-centre trial on hostility-modification and CHD is planned to be conducted in Canada and Israel.

Future trials should measure levels of epinephrine and norepinephrine, to examine their hypothesized role in the hostility-BP link, and to see if these neurohormones are reduced by the proposed treatment. These measurements would enhance our understanding of the underlying biological mechanisms by

which hostility may cause CHD, as suggested by the psychophysiological reactivity model (e.g., Williams et al., 1985).

Additionally, future trials should assess patients' daily hassles, frequency of interpersonal conflicts and social support, to examine whether they are modified by the intervention, and whether they mediate the link between hostility and CHD, as suggested by the psychosocial vulnerability and transactional models (Smith, 1992).

An additive or component design study should examine the effects of the three components of the proposed treatment (monitoring and altering behavioral, cognitive and affective hostility, respectively) on reducing hostility. All three dimensions of hostility should be assessed regardless of whether all three are targeted in the intervention. This would inform us whether altering one dimension of hostility is necessary and/or sufficient for altering the other two. Cognitive theories would suggest that hostile cognitions would be the main factor to modify (Chesney, 1985; Powell, 1992). However, this needs to be tested empirically. Such component designs could also examine which skills in the program are essential (e.g., assertiveness training, relaxation), rather than which hostility dimension must be targeted.

The proposed hostility intervention could be applied to other domains where hostility is a risk factor such as car accidents (Donnovan & Marlatt, 1982). Such a study is planned to take place in Israel, where the mortality from car accidents is among the highest in the world.

Finally, although Chapter Two clearly stated that CHD-predictive hostility is a more limited construct than the broader one attributed to international or inter-group conflicts, these findings may have implications for such conflicts. Hostile attributions and mistrust, anger and aggression (e.g., war, local combat) exist between nations in conflict (e.g., the Middle East, the former Yugoslavia). Alteration of mistrust via sharing control, substitution of problem-focused negotiations for anger, and substitution of tolerance and listening for aggressive acts may not harm, and at best, may actually help minimize these long-lasting conflicts. This is a personal wish more than a fact.

Conclusions

Overall, the results of Studies 1 and 2 provide preliminary evidence for the efficacy of the proposed hostility-reduction intervention at reducing CHD-predictive and mortality-predictive hostility levels. These results were replicated in samples which differ in age and health status.

Study 2 extended these findings, and suggested that certain effects are maintained after two months (self-reported hostility, quality of life), and that the hostility-treatment positively affects CHD-related health outcomes (i.e., resting-BP, life-dissatisfaction and depression). Finally, Study 2 provided preliminary evidence for a **causal** relation between hostility and CHD-related variables (i.e., resting-DBP, life-satisfaction and depression).

These findings support the central role hostility is hypothesized to have in CHD (Dembroski & Costa, 1987; Miller et al., in press; Smith, 1992; Williams,

1987). The replication and extension of therapeutic effects and the evidence for causal relations all point to reliable and positive effects of this treatment. These findings together with the matched-randomized-controlled design employed allow one to conclude that the proposed treatment reduces CHD-predictive hostility and has short-term positive physical and mental health consequences.

However, given the small sample size, the many statistical tests performed, and the type of outcomes used, this research should be replicated with larger samples and with long-term outcomes (e.g., reinfarct), to test the preventative value of this treatment. Should the results of such future studies be positive, the causal role of hostility in CHD will be supported, and behavioral medicine may have an additional therapeutic tool for CHD-prevention and treatment, the ultimate goal of applied clinical research.

(Appendix A)
DEPARTMENT OF PSYCHOLOGY, DALHOUSIE UNIVERSITY.
MODIFYING REACTIONS TO STRESS AND PHYSICAL HEALTH

CONSENT FORM (Student form)

We are conducting a study on the effects of modifying reactions to stressful situations on physical health measures.

We would like you to participate in our study, in which you will be asked either to attend 8 group meetings (experimental condition) or to attend one meeting (control condition), and to come to a final assessment in eight more weeks. If you are assigned to attend the 8 meetings, you will learn how to change reactions you may have in stressful situations. After some meetings, you will also be asked to monitor at home your own behavior, using a diary once a day. The meetings will be closed, and only researchers involved in the study will be allowed to join them. Meetings will last one and a half hours, once a week, for eight weeks. The meetings will take place in the department of psychology at Dalhousie University. The time of meetings will be scheduled to match the availability of all participants. Finally, you will be asked to participate in a final assessment, similar to the first study. These assessments involve completing questionnaires and a short video-taped interview on how you deal with daily stressful situations. During the interview, your blood-pressure and heart-rate will also be measured.

You will receive $40 for participating in the treatment group if you are assigned to the experimental condition. You will receive additional $10 for undergoing the final assessment, regardless of the group to which you were assigned.

Participation in this study is entirely voluntary, and you may withdraw from this study at any time and for any reason. There are no known risks associated with this study, but in any case of discomfort, you will be notified about the appropriate resources available to you. You may learn how to alter unhealthy reactions you may have to stress by participating in this study. In addition, your participation will help us understand more about improving physical health by modifying psychological reactions.

The information obtained (e.g., questionnaires, video-tapes, heart-rate and blood-pressure measures) will be kept in a confidential manner. Results from the study may be published in the scientific literature, but no names or identifying information will be used, and your privacy will be protected at all times. Scores on the measures will be provided to you upon your request.

If you have any questions regarding the study, you may phone the researchers:

Yori Gidron, 479-3753 (home), 494-1448 (work).
Dr. Karina Davidson, 494-6915 (work).

I have read the consent form and agree to participate in this study.

Name: _____ Signature: _____

Date: _____ Phone number: _____

Appendix B

CARDIAC PREVENTION RESEARCH CENTER
CAMP HILL MEDICAL CENTER
CONSENT FORM (CHD form)

THE EFFECTS OF REDUCING REACTIONS TO STRESS ON THE HEALTH MEASURES OF CARDIAC PATIENTS

INTRODUCTION

We invite you to take part in a research study at the Camp Hill Medical Center. It is important that you read and understand several general principles that apply to all who take part in our study: a) taking part in the study is entirely voluntary. Whether you participate or not, the quality of medical care provided to you will be the same; b) personal benefit may not result from taking part in the study, but knowledge may be gained that will benefit others; c) you may withdraw from the study at any time without loss of any benefits to which you are otherwise entitled. This study is described below. The description includes information about the risks to you, as well as any inconvenience or discomfort which you may experience. You are urged to discuss any questions you have about this study with the staff members who explain it to you.

NATURE OF THE STUDY

Certain reactions people have to daily stress have been shown to put them at risk for Coronary Heart Disease. However, treatments for modifying these reactions have not been tested on individuals with coronary heart disease, and may improve their health status. This study tests the effects of a such a treatment on modifying such reactions and on measures of health status such as chest-pain. Patients will be randomly assigned to either a stress management treatment and usual medical care (experimental condition) or to minimal stress-management treatment and usual medical care (control condition).

CONDITIONS OF YOUR INVOLVEMENT

This study includes men, who are between the ages of 35-60 years, who have had a heart-attack or have been diagnosed with unstable angina in the last 6 months, and whose physical and mental health otherwise permits them to participate (e.g., no debilitating physical disability, no known psychiatric disorders).

SCREENING FOR YOUR PARTICIPATION

It will be necessary to confirm that you had a medically documented heart-attack or unstable angina and to verify your medical condition, by viewing your medical record. Your scores on certain measures from the first screening study served as selection criteria for asking you to participate in this study.

THE EFFECTS OF REDUCING REACTIONS TO STRESS ON THE HEALTH MEASURES OF CARDIAC PATIENTS

PROCEDURES OF THE STUDY

This study will compare two treatments conditions. If you are assigned to the experimental condition, you will be asked to attend eight group meetings and you will learn how to change certain reactions you may have to stress. You will be asked to monitor at home once a day your own behavior. The meetings will last one and a half hours, once a week, for two months. Meetings will take place at the Cardiac Prevention Research Center (CPRC), on the 9th flour of the Camp Hill Medical Center. If you are assigned to the control condition, you will be asked to attend only one meeting about risks of stress-reactions.

In order to test the effects of the treatments, you will be asked to undergo assessments in another two months, and three and six months later. The assessments involve completing questionnaires and a short video-taped interview on how you deal with daily stressful situations. During the interview, your blood-pressure and heart-rate will also be measured.

Additional measures of readmission to hospital, chest-pain and quality of life will be obtained from either your medical record or your own reports to examine your health after the treatment. Your spouse will also be asked to complete questionnaires that are concerned with the way you deal with stress and with your physical health.

RISKS AND DISCOMFORTS

There are no major hazards or risks in this study. However, during the meetings or the follow-up interviews you may experience brief and slight tension, as they primarily deal with how you cope with daily stress. This discomfort is not more than that experienced by most individuals on a daily basis. This study has been tested on university students, and no adverse effects were identified. Should you feel adversely affected by any part of this study, counselling will be offered to you by the primary investigator without charge.

ALTERNATIVE TREATMENTS:

As mentioned above, you will be receiving your usual medical care regardless of which experimental condition you are assigned to. This study will test whether the proposed treatment can increase the effects of usual medical treatment.

THE EFFECTS OF REDUCING REACTIONS TO STRESS ON THE HEALTH MEASURES OF CARDIAC PATIENTS

PAYMENT

If you are assigned to attend the eight meetings or the single meeting, you will receive partial compensation for your travel costs. Finally, all patients will receive partial compensation for their travel costs for attending each of the three follow-up assessments.

OTHER PERTINENT INFORMATION

1. Confidentiality: you will not be identified as a study participant in any reports of this research. Your questionnaires and recorded interviews will be kept in a locked file cabinet. They will only be available to the staff involved in this study.

2. Questions or problems: if any questions arise with regard to the study, please contact: Karina Davidson, Ph.D at 494-6915 or Yori Gidron at 494-1448 at the Department of Psychology, Dalhousie University.

3. You will be advised of any new information which may affect your decision to remain in this study.

4. Consent document: we suggest that you retain a copy of this document for your later reference and personal records.

Complete Item Below

I have read the explanation about this study and have been given the opportunity to discuss it and to ask questions. I hereby consent to take part in this study.

_____ _____

Signature of Patient Date Signed

_____ _____

Signature of Investigator Date Signed

_____ _____

Signature of Witness Date Signed

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