Depressive Style and Lifestyle Changes:
Assessment, Association, and Prediction in the Lifestyle Heart Trial

by

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Acknowledgements

This thesis literally could not have been written without the advice, support, and understanding of many people. First of all, I'd like to thank Larry Scherwitz, who as one of the principal investigators of the Lifestyle Heart Trial had enough interest and faith in me to offer me the opportunity to use the LHT data. I am also deeply indebted to Stephen Sparler of the Preventive Medicine Research Institute, who generously gave of his time to help me clear one logistical hurdle after another.

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Finally, I owe an immense debt of gratitude to three very special people in my life. Deanna, you have suffered immensely through the last three years while I have greedily profited from your humor, your understanding, and your love, not to mention your labor on this project. Thank you for everything. And Mom and Dad, I cannot thank you enough for the last 29 years. Everything I accomplish, including this thesis, is a reflection on you. I wish you could be here to see this, Dad, because I know you would be proud.

Ken McClain
Berkeley, California
Chapter 1. Introduction

Overview

Cardiovascular diseases are the leading cause of mortality in the United States, accounting for nearly one million deaths in 1989 (Gunby, 1992). The American Heart Association estimates that 69 million people in the U.S. — roughly one quarter of the population — currently suffers from one or more cardiovascular diseases.

Coronary heart disease (CHD), the most prevalent of the cardiovascular diseases, affects 11 million Americans (Lavie, et al., 1991). CHD, also called ischemic heart disease or coronary artery disease, is caused by narrowing of the coronary arteries encircling the heart. In 1992, as many as 1.5 million people will suffer a myocardial infarction because of CHD, and a third of them will die (Gunby, 1992). Many more will experience the other manifestations of CHD: angina pectoris, sudden cardiac death, and/or chronic ischemia of the heart.

CHD has a multifactorial etiology. The “standard” risk factors include age over 30, male gender (until age 75), hypertension, hyperlipidemia, low HDL, smoking, diabetes mellitus, lack of exercise, obesity, and family history (Chandrasoma and Taylor, 1991). Except for age, gender, and family history, all of these risk factors have some behavioral component. Much research has found that lifestyle changes — less fat intake, smoking cessation, exercise, and weight loss — can modify the standard risk factors and reduce the attendant risk of various CHD endpoints (e.g., Hjermann, et al., 1981; MRFIT Research Group, 1982).
Coronary-prone behavior

While most epidemiological research has demonstrated a clear relationship between the standard risk factors and CHD outcomes, most new cases of CHD cannot be predicted by these risk factors (Jenkins, 1971a; Rosenman, 1983). This finding has lent statistical weight to the notion that behavior — not simply as it affects other risk factors, but in and of itself, i.e., coronary-prone behavior — can account for at least some of the remaining cases.

"Coronary-prone behavior" is perhaps the most general term used to describe the various personality types, behavior patterns, coping styles, psychosocial factors, and modes of psychological expression posited to increase CHD risk. By many names, coronary-prone behavior has a long and controversial history. Sir William Osler (1892), one of the fathers of modern American medicine, considered the coronary-prone individual "not the delicate, neurotic person, but the robust, the vigorous in mind and body, the keen and ambitious man, the indicator of whose engine is always at full speed ahead" (quoted in Dembroski and Czajkowski, 1989, p. 23).

Friedman and Rosenman (1959), two cardiologys, ushered coronary-prone behavior into its scientific age by describing a behavior pattern they claimed was associated with cholesterol level, coronary artery disease, blood clotting time, and arcus senilis. This behavior pattern, which soon became widely known as "Type A," was described as resulting from the interaction of personality attributes, environment, and the perception of external stressors. This interaction produced an aggressive, hostile, and time-urgent style of living, and Type A individuals were described "to be in a chronic struggle to achieve poorly defined goals or to obtain an excessive number of things from their environment and to be in habitual conflict with others and with time"
(Rosenman, et al., 1988). Friedman later stressed that Type A behavior was not an emotional complex but a medical disorder, with signs and symptoms (Friedman and Powell, 1984; Friedman, 1988).

Early in its history, the hypothesized association of Type A with CHD received support from a number of retrospective studies. It then gained considerably more credibility when a large, 8.5-year prospective investigation, the Western Collaborative Group Study (WCGS; Rosenman, et al., 1975), found that Type A persons had almost twice the risk of developing CHD than did non-Type A persons.

Cross-sectional angiographic studies provided yet more evidence that Type A behavior was a risk factor for CHD (e.g., Zyzanski, et al., 1976; Blumenthal, et al., 1978; Frank, et al., 1978). By 1981, the evidence in favor of the Type A construct was sufficiently compelling that a review panel convened by the National Heart, Lung, and Blood Institute declared Type A behavior a greater risk factor for CHD in employed, middle-aged Americans than any of the standard risk factors (Review Panel, 1981). This was the first time a psychosocial risk factor had enjoyed such a distinction, and represented perhaps the apex of scientific support for the notion of Type A behavior as coronary-prone. Some authors had even taken to using the terms “Type A” and “coronary-prone” interchangeably (e.g., Jenkins, 1971b, 1978), which of course begged the question of what behaviors truly did increase the risk of CHD.

Since that time, however, the Type A ship has been foundering in murky shoals. On the one hand, intervention studies have generally shown that modifying Type A behavior decreases the risk of subsequent CHD events. In the Recurrent Coronary Prevention Project (RCPP), for example, reduction of Type A behavior by group counseling was associated with a decreased risk
of fatal and nonfatal cardiac events (Friedman, et al., 1986). (See Nunes, et al. [1987] for a meta-analysis of Type A intervention studies.)

On the other hand, the large MRFIT study failed to show a relationship between Type A behavior and CHD incidence (Shekelle, et al., 1985), and a number of studies using high-risk subjects (e.g., Case, et al., 1985) or angiographic outcome measures (e.g., Dembroski, et al., 1979) have failed to find an association between Type A behavior and disease, leading participants at the 1987 meeting of the Psychosomatic Society to question the validity of the Type A concept altogether (Miller, et al., 1991). And while Friedman (1988) and Scherwitz (1988) have criticized MRFIT and the study by Case, et al. (1985) for inadequately assessing Type A behavior, even Rosenman and Friedman’s WCGS database yielded negative findings in an analysis using 22-year follow-up data. In that analysis, Ragland and Brand (1988) found that CHD mortality after an initial CHD event was unexpectedly lower for Type A patients than for Type B patients.

Amidst the controversy, Dimsdale (1988) has tried to affirm that "something is going on in terms of the relation between personality and heart disease" (p. 112). And to apprehend that "something," researchers have generally followed one of two strategies: isolating certain components of the Type A behavior pattern, or looking at potential coronary-prone behaviors outside the Type A complex. Among the components of Type A behavior — speed and impatience, job involvement, time urgency, hard-driving competitiveness, anger, aggression, and hostility — hostility in particular has emerged as a promising factor (Williams, 1987). Chesney, et al. (1988), for example, found that hostility served as the best discriminator between WCGS cases and controls.
Non-Type A behaviors that have been studied include principally anxiety, extraversion, and depression, though none of these has received the attention that Type A and its components have. In a meta-analysis of studies of psychosocial variables and CHD endpoints (Booth-Kewley and Friedman, 1987), however, depression was found to have associations of comparable magnitude to that of the best Type A measure. The authors of the meta-analysis concluded that the picture of the coronary-prone personality emerging from their review seemed

... to be one of a person with one or more negative emotions: perhaps someone who is depressed, aggressively competitive, easily frustrated, anxious, angry, or some combination... In sum, it appears that the concept of the coronary-prone personality and its associated research should be broadened to encompass certain psychological attributes such as depression, in addition to those associated with Type A behavior (p. 358).

Research objectives

The present study undertakes to examine what role depression plays in a program of comprehensive lifestyle changes designed to retard or reverse the process of coronary atherosclerosis. To do so it makes use of the work of Dr. Dean Ornish and his colleagues on the Lifestyle Heart Trial (Ornish, et al., 1990) and the research of Dr. Jerry R. Pattillo into the predictive ability of depressive behavior scores on recurrent cardiac mortality in the RCPP (Pattillo, 1990).

The specific objectives of this study will be to assess:

1. what effect the Lifestyle Heart Trial intervention has on “depressive style,” a construct developed by Pattillo (1990); and

2. what relationship baseline depressive style has to the adoption of lifestyle changes both within a structured program and outside of it.
Outline of thesis

Chapter 2 discusses what is known about coronary heart disease and atherosclerosis in more detail. Chapter 3 describes the methods and results of the Lifestyle Heart Trial, which provided the data for the present study. Chapter 4 reviews the literature concerning depression and lifestyle factors, as well as the study by Pattillo (1990). Chapter 5 presents the rationale for the present study and enumerates the research questions, and Chapter 6 details the methods used to answer them. Chapter 7 provides the results of the investigation, and Chapter 8 discusses their implications. Finally, Chapter 9 anticipates the logical extension of this study by reviewing the literature concerning depression, atherosclerosis, and CHD.
Chapter 2. Coronary heart disease and atherosclerosis

Coronary heart disease (CHD) is a problem of enormous import, causing more deaths than any other disease in most developed countries. In the U.S., it accounts for 25-30% of all mortality, or over 500,000 deaths a year (Chandrasoma and Taylor, 1991). In 1992, as many as 1.5 million people will suffer a myocardial infarction because of CHD, and a third of them will die (Gunby, 1992). Many more will experience the other manifestations of CHD: angina pectoris (chest pain), sudden cardiac death, arrhythmias, cardiac failure, and/or chronic ischemia of the heart.

Pathophysiology

About 98% of CHD is due to atherosclerosis, a process involving the deposition of fat in arterial walls and the consequent narrowing (stenosis) and hardening (sclerosis) of arteries (Chandrasoma and Taylor, 1991). Atherosclerosis of the coronary arteries encircling the heart, also known as coronary artery disease, causes CHD. In the cerebral circulation, it is responsible for most cases of stroke, which accounts for approximately 200,000 deaths per year in the U.S. (Chandrasoma and Taylor, 1991).

Atherosclerosis develops insidiously and mostly asymptptomatically over many years, beginning in early adulthood. Hard, brittle fibrofatty plaques (atheromas) develop in the inner lining of arteries, slowly choking off blood flow and perhaps occluding its passage entirely, thus depriving tissue of its oxygen supply. The atheromas also weaken the arteries and decrease their elasticity, rendering them more susceptible to damage and less
able to accommodate demands for increased blood flow. Acute events may complicate the chronic course of the disease; ulceration or hemorrhage of the atheromas create clots that lodge at the same site or farther downstream where the passageways are smaller; arterial spasm may decrease blood flow through an already compromised passageway.

Anatomy

The major arteries supplying the heart are the left main, left anterior descending, left circumflex, and right coronary (Figure 2.1). Variations in the branching pattern of the arteries are common, and arterial diameter differs

![Heart Diagram](image-url)

*Figure 2.1. Anterior view of the heart and the coronary arteries. (From Philo, et al., 1985.)*

from person to person as well. For example, in about 50% of cases, the right coronary artery is dominant, crossing to the left side and supplying the left ventricular wall and the interventricular septum. In about 20% of cases, the left main artery is dominant, and in the remaining 30%, neither artery is
dominant (Moore, 1985). The normal variation in anatomy has implications for the interpretation of angiographic data.

**Angiography**

Coronary angiography, or arteriography, has had a dramatic impact in both medical practice and research, enabling the number and severity of coronary stenoses to be quantified. The technique requires a long, thin catheter to be advanced through the femoral or brachial artery into the ascending aorta, where a radiopaque dye can be selectively injected into a coronary artery. An X-ray motion picture, or cineangiogram, then records the dye's transit through the artery and its branches. Because the procedure has significant risks, potential research subjects have generally been limited to patients who require angiography for clinical reasons. Thus the subjects are usually considered high-risk; i.e., they are suspected if not known to have coronary artery disease.

A greater than 50% reduction of the arterial diameter, corresponding to an approximately 75% reduction in cross-sectional area, is thought to be hemodynamically significant, allowing adequate blood flow while a subject is at rest but not during exertion or emotional stress. Lesions greater than 75% diameter stenosis are definitely significant, producing a 95% reduction in cross-sectional area.

Different protocols exist for quantifying the degree of pathology, using degree of stenosis as well as number of affected segments of the arterial tree as indices. Subjectivity on the part of the reader is often addressed by having multiple independent readers or by using computer-assisted measurements.

Angiography has a number of advantages over clinical criteria in studying CHD or atherosclerosis. First of all, atherosclerosis is often present
long before any clinical manifestations of CHD, and thus misclassification bias is avoided. Second, angiography provides a continuous, precisely localized, and fairly objective measure of disease, whereas CHD manifestations are fairly crude measures of pathology. Third, if done serially, angiograms provide a way of assessing treatment efficacy or the effect over time of risk factors.

Disadvantages of angiography as a research tool in observational epidemiological studies are well described by Pearson (1984), and include the risks of the procedure, selection bias, and reduced variation in the distribution of risk factors and disease among the subjects due to their high-risk status. Research using angiography has also sometimes contradicted epidemiological findings. Hypertension, for example, has been shown to have little or no association with disease in at least 10 angiographic studies, although it has a strong and consistent association in most epidemiologic studies (Pearson, 1984).

Risk factors

CHD has a number of risk factors (Table 2.1), though these do not fully account for its incidence, as discussed above. Chief among them are age, male gender (until age 75), hypertension, hyperlipidemia or hypercholesterolemia, and cigarette smoking. What has made CHD an ideal subject for research into

<table>
<thead>
<tr>
<th>Table 2.1. Risk factors for atherosclerosis and CHD</th>
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<tbody>
<tr>
<td>age (increases after age 30)</td>
</tr>
<tr>
<td>sex (males &gt; females until age 75)</td>
</tr>
<tr>
<td>genetics</td>
</tr>
<tr>
<td>hypertension</td>
</tr>
<tr>
<td>hyperlipidemia</td>
</tr>
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</table>

(From Chandrasoma & Taylor, 1991.)
psychosocial influences on disease is not only its societal importance but also the significant role of lifestyle factors in its pathogenesis. Aside from age, sex, and a small number of genetic factors, all the risk factors for atherosclerosis and CHD have a behavioral or psychological component. Some of the risk factors, such as cigarette smoking, leading a sedentary lifestyle, and the controversial "coronary-prone" behaviors, are behaviors in and of themselves. Other risk factors, including hyperlipidemia, hypertension, obesity, and to some extent diabetes, are influenced by behaviors having to do with diet, exercise, and ways of interacting with the environment. One's lifestyle, then, is very much at issue.

To underscore how important lifestyle factors can be, both hypertension and hyperlipidemia increase the risk of atherosclerosis five-fold, and smoking one pack of cigarettes a day increases the risk three-fold (Chandrasoma and Taylor, 1991). Making lifestyle changes can thus have a significant impact on one's risk of developing heart disease.

The challenge for those who design and implement health interventions is to identify what lifestyle changes to make, how to make them, and how to sustain them. This was the challenge undertaken by a group of researchers in the Lifestyle Heart Trial, to be described next.
Chapter 3. The Lifestyle Heart Trial

The Lifestyle Heart Trial (LHT; Ornish, et al., 1990) is a controlled, randomized clinical trial that has attempted to determine whether comprehensive lifestyle changes alone can retard or reverse coronary atherosclerosis. The components of the lifestyle intervention include a low-fat vegetarian diet, an exercise program, stress management training, smoking cessation, and social support. The LHT was begun in January, 1986, and subjects were enrolled in the study over the next three years. Though originally planned as a one-year intervention, the trial was extended three years because of its promising results.

Subjects

The Lifestyle Heart Trial subjects were recruited between January, 1986, and October, 1988, from patients undergoing coronary angiography for clinical reasons at two San Francisco hospitals. Patients were excluded if they had had a recent myocardial infarction, had other life-threatening illnesses, were scheduled for coronary artery bypass surgery or angioplasty, had ever received the thrombolytic agents streptokinase or t-PA, or were taking lipid-lowering drugs. If patients had undergone bypass surgery or angioplasty, they needed at least one measurable lesion in a non-dilated or non-bypassed coronary artery (Scherwitz, et al., 1991).

After their angiograms, 96 patients were determined to be eligible. Fifty-three were randomly invited to be in the experimental group, of whom 28 (53%) volunteered, and the remaining 43 were invited to join the control group, of whom 20 (47%) volunteered. The baseline Videotaped Standardized Interview for Type A behavior (VSI; Friedman and Powell,
1984) and other tests were administered thereafter. Six experimental and one control subject did not have follow-up angiographic data, leaving 22 in the experimental group and 19 in the control group who were available for studies of lesion changes. The baseline demographic characteristics of the subjects are shown in Table 3.1. There were no significant differences between the experimental and control groups in these variables or in diet and lifestyle characteristics, functional status, cardiac history, risk factors, or disease severity (Ornish, et al., 1990).

<table>
<thead>
<tr>
<th>Table 3.1. Baseline demographic characteristics of experimental and control groups</th>
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<tbody>
<tr>
<td>Mean (SD)</td>
</tr>
<tr>
<td>Experimental group (n = 22)</td>
</tr>
<tr>
<td>Control group (n = 19)</td>
</tr>
<tr>
<td>Male/female</td>
</tr>
<tr>
<td>Age (yr)</td>
</tr>
<tr>
<td>Weight (kg)</td>
</tr>
<tr>
<td>Body mass index (kg/m²)</td>
</tr>
<tr>
<td>Education (yr)</td>
</tr>
</tbody>
</table>

(From Ornish, et al., 1990.)

Lifestyle intervention

In several small groups, the experimental group participants and their spouses or partners were introduced to the program in a week-long retreat at a resort hotel. Daily lectures and slide programs covered such topics as nutrition; the effects of smoking, stress, diet, and exercise on the heart; and cooking and shopping. The participants also received three hours per day of stress management training, one hour per day of aerobic exercise at individually specified levels of exertion, and one hour per day of group discussion facilitated by a clinical psychologist. A cook prepared all meals. Table 3.2 summarizes the lifestyle changes of the intervention.
Table 3.2. Components of the Lifestyle Heart Trial

<table>
<thead>
<tr>
<th>Diet</th>
<th>Exercise</th>
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<td>- vegetarian and mostly non-dairy</td>
<td>- at least half an hour per day and</td>
</tr>
<tr>
<td>- less than 10% calories from fat</td>
<td>three hours a week</td>
</tr>
<tr>
<td>- &lt;5 mg/day cholesterol</td>
<td></td>
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**Stress management**

Total of at least one hour per day of:
- stretching (hatha yoga), 20 minutes
- progressive deep relaxation, 15 mins
- breathing techniques, 5 minutes
- meditation, 15 minutes
- directed or receptive visualization, 5 minutes

**Group support**

Discussions facilitated by a clinical psychologist, one hour twice a week:
- strategies for maintaining adherence to the program
- communication skills (listening, expressing feelings)

**Smoking cessation**

(From Ornish, 1990.)

**Diet**

The prescribed diet was wholly vegetarian and non-dairy save for egg whites and up to one cup per day of nonfat yogurt or milk. Foods high in saturated fats were also discouraged. This achieved a diet of less than 10% calories from fat and almost no cholesterol. Salt was restricted for hypertensive participants and alcohol was prohibited for anyone with a history of alcoholism, but otherwise salt, alcohol, and sugar were allowed in moderate amounts. Caffeine and other stimulants were not allowed. Within these limitations, participants could eat as much as they wished.

A cook provided up to 14 take-home lunches and dinners each week for those who requested them. During the first year these take-home meals accounted for approximately half of the total calories consumed.

**Exercise**

Participants were asked to exercise at least half an hour per day and three hours per week. They were given individual guidelines about exercise levels.
Stress management

Participants were also asked to spend at least an hour per day practicing stress reduction techniques. These consisted of hatha yoga (a form of stretching exercises) for 20 minutes, progressive deep relaxation for 15 minutes, breathing techniques for five minutes, meditation for 15 minutes, and imagery for five minutes. The purpose of these techniques was to develop a sense of relaxation, concentration, and awareness of internal states (Scherwitz, et al., 1991). A professionally produced audiocassette of these techniques was given to each participant.

Group meetings

Every Tuesday and Thursday the experimental group participants met for four hours, with one hour each allotted for exercise, stress management, dinner, and a group discussion facilitated by a clinical psychologist. Dinner was catered on Tuesdays and provided by the participants themselves on Thursdays. In the group sessions, participants discussed strategies for maintaining adherence to the program and learned and practiced communication skills such as listening and expressing their feelings.

Smoking cessation

A final component of the intervention was smoking cessation. Only one experimental group participant smoked at the beginning of the study, however, and she agreed to stop.

Results

Adherence

Adherence to the program was assessed for diet, exercise, stress management, and smoking. A overall adherence scale from zero to one was
constructed, with zero representing virtually total noncompliance and 1.00 representing a diet of 10% fat and 5 mg/day cholesterol, three hours of exercise and seven hours of stress management per week, and no smoking.\textsuperscript{1} Thus scores greater than one indicated that program recommendations were exceeded. During the first year, most of the experimental group participants met or exceeded the minimum recommendations for all four assessed components (diet, exercise, stress management, and smoking), while the usual care group made more moderate changes in line with conventional medical recommendations. Average total adherence rose from 0.55 to 1.22 in the experimental group and from 0.56 to 0.62 in the control group, a highly significant between-group difference (Table 3.3).

**Table 3.3.** Total adherence scores. Twenty experimental subjects had scores greater than 1.00, 14 had scores greater than 1.20, and seven had scores greater than 1.40.

<table>
<thead>
<tr>
<th>Total adherence scores</th>
<th>Baseline</th>
<th>Follow-up</th>
<th>Change</th>
</tr>
</thead>
<tbody>
<tr>
<td>Experimental group (n=20-22)</td>
<td>0.55</td>
<td>1.22</td>
<td>+0.67</td>
</tr>
<tr>
<td>Control group (n=17-19)</td>
<td>0.56</td>
<td>0.62</td>
<td>+0.06</td>
</tr>
</tbody>
</table>

(From Ornish, et al., 1990, and Scherwitz, et al., 1991.)

The average weight of the experimental-group participants decreased over 20 pounds, from 200 to 178 pounds, while the average weight of the control-group participants increased slightly, from 177 to 180 pounds.

**Coronary atherosclerosis**

At baseline there were no significant differences in coronary artery disease severity. After excluding 33 lesions that were 100% occluded, the researchers counted 105 lesions in the experimental group, with a mean percentage diameter stenosis of 40.0%, and 90 lesions in the control group

\textsuperscript{1} See Methods for the formula used to calculate total adherence.
(mean stenosis 42.7%). Follow-up angiography showed that the experimental group's average percentage diameter stenosis had decreased while the average stenosis of the control group had increased (Table 3.4). This was the first demonstration that regression of coronary atherosclerosis could be achieved by lifestyle changes alone and without the use of cholesterol-lowering drugs or surgery.

Table 3.4. Baseline, follow-up, and change in average percentage diameter stenosis of 195 detectable coronary artery lesions. Eighteen of the 22 patients in the experimental group showed average changes in the direction of regression of atherosclerosis, while 10 of the 19 patients in the control group showed average changes in the direction of progression.

<table>
<thead>
<tr>
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<th>Average % diameter stenosis (SD)</th>
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<tbody>
<tr>
<td></td>
<td>Baseline</td>
</tr>
<tr>
<td>Experimental group</td>
<td></td>
</tr>
<tr>
<td>(n = 22)</td>
<td>40.0 (16.9)%</td>
</tr>
<tr>
<td>Control group</td>
<td></td>
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<tr>
<td>(n = 19)</td>
<td>42.7 (15.5)%</td>
</tr>
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</table>

(Adapted from Ornish, et al., 1990.)

Adherence and changes in atherosclerosis were strongly related in a "dose-response" manner. That is, those who made the greatest changes showed the biggest improvement, suggesting that the relationship was causal (Ornish, et al., 1990). This relationship was observed for both the experimental group alone and for the whole study group.

Angina

In addition to showing objective evidence of health improvement, participants in the experimental group also reported decreases in chest pain frequency, duration, and severity (Table 3.5). Those in the control group, on

Table 3.5. Change in chest pain (angina pectoris) frequency, duration, and severity over one year. Intergroup differences were significant.

<table>
<thead>
<tr>
<th></th>
<th>Change in chest pain</th>
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<tbody>
<tr>
<td></td>
<td>Frequency</td>
</tr>
<tr>
<td>Experimental group</td>
<td></td>
</tr>
<tr>
<td>(n = 20)</td>
<td>-91%</td>
</tr>
<tr>
<td>Control group</td>
<td></td>
</tr>
<tr>
<td>(n = 17)</td>
<td>+165%</td>
</tr>
</tbody>
</table>

(Adapted from Ornish, et al., 1990.)
the other hand, reported large increases in the same measures.

Scherwitz, et al. (1991) investigated whether baseline psychosocial status could predict adherence and found that measures of social support, sense of coherence, anxiety, and depression (the latter two obtained from subscales on a 30-item questionnaire) were more predictive of the degree of adherence in the control group than in the experimental group. This suggests that the intervention itself accounted for adherence and the changes in atherosclerosis.
Chapter 4:  Review of the literature

Overview

Clinicians have long noted the effect that emotions seem to have on the body in general and the heart in particular. The 17th-century English physician William Harvey, who was the first to demonstrate the circulation of the blood, believed that “every affection of the mind that is attended with either pain or pleasure, hope or fear, is the cause of an agitation whose influence extends to the heart” (quoted in Dembroski and Czajkowski, 1989, p. 22).

Research findings from diverse areas have tended to corroborate Harvey’s conviction. In one of the larger studies of the general health and well-being of depressed people, for example, Wells, et al. (1989) examined 11,242 outpatients in three U.S. cities, assessing several kinds of functioning, perceived current health, and bodily pain. Patients with either current depressive disorder or depressive symptoms in the absence of disorder tended to have poorer functioning and well-being than did patients with no chronic medical conditions. In addition, the level of functioning and well-being in these patients was comparable with or worse than those of patients with major chronic conditions such as diabetes, advanced coronary artery disease, and arthritis. It is not clear, of course, whether depression preceded or followed a decline in functioning and well-being.

In another large undertaking, Murphy, et al. (1987) found that depression at baseline, with or without anxiety, was associated with a significantly increased risk of mortality during a 16-year follow-up period of a general population sample, controlling for age, sex, and the presence of self-
reported physical disorder at baseline (standardized mortality ratio, 1.6). In addition, there was a significant association between affective disorder (depression and/or anxiety) and death due to cardiovascular disease (SMR, 1.5).

Another study found an SMR of 2.6 among 62 elderly depressed patients followed for one year (Rabins, et al., 1985). Although the numbers were small, six of the eight deceased patients had been diagnosed with cardiovascular disease while only three of 54 patients alive at follow-up had such a diagnosis ($p < 0.001$).

Coupled with the prevalence of CHD, such findings create an imperative to examine the multifaceted relationships of depression, lifestyle, atherosclerosis, and CHD.

![Diagram](image)

* Lifestyle: diet, exercise, smoking, stress management
** CHD manifestations: angina, MI, sudden cardiac death, coronary bypass, angioplasty

**Figure 4.1.** Conceivable relationships among depression, lifestyle, coronary atherosclerosis, and CHD manifestations
The interaction of depression, lifestyle changes, atherosclerosis, and CHD

One can conceive of multiple interactions, causal relationships, and modifying influences among depression, lifestyle changes, atherosclerosis, and coronary heart disease. Figure 4.1 diagrams the conceivable two-variable relationships, leaving aside for the moment the role of other risk factors, both known and unknown. Note that all the relationships except that between coronary atherosclerosis and CHD events can plausibly be bidirectional. Note also that more complex relationships, such as three-way interactions, may exist but are not depicted.

It is often assumed that the risk factors for CHD act solely by initiating and promoting the development of atherosclerosis. But risk factors, including possibly depression, may differentially affect the development of coronary atherosclerosis and the appearance of CHD manifestations (Figure 4.2). One study, for example, found that for different degrees of coronary atherosclerosis, the prevalence of prior MI was associated with degree of cigarette smoking (Hartz, et al., 1981). While smoking has been implicated in the pathogenesis of atherosclerosis, this finding suggests that it also has precipitating effects in causing MI. It is conceivable that other risk factors operate similarly. Note in Figure 4.2 that the combination of risk factors

![Figure 4.2. Pathogenesis of coronary atherosclerosis and CHD manifestations or indicators](image-url)
contributing to the development of atherosclerosis may differ from the combination contributing to the appearance of CHD manifestations, and different risk factors may apply to each manifestation of CHD.

Studies of the relationships between depression and lifestyle will be briefly reviewed here, as will the study by Pattillo (1990), the conceptual predecessor to the current endeavor. This chapter also includes a discussion of mechanisms linking depression, lifestyle changes, and CHD. In anticipation of future research, a more thorough review of the richer literature regarding depression, atherosclerosis, and CHD manifestations is presented in Chapter 9.

Studies of depression and lifestyle

A number of studies have examined the relationship between depression and matters of lifestyle such as exercise, smoking, stress management, and diet. Findings relating exercise and depression have been mixed and often leave open the question of causality. Some of the best evidence that a relationship exists comes from treatment programs for depressed persons that have incorporated exercise. Roth and Holmes (1987), for example, found that aerobic exercise was more effective than relaxation training or no treatment in reducing depression in college students who had experienced a high number of negative life events during the preceding year. In a nondepressed population sample, Camacho, et al. (1991) found that low levels of activity at baseline placed subjects at significantly greater risk of depression nine years later, and the risk diminished somewhat in people who increased their activity levels.

A link between smoking and depression has been demonstrated in a number of contexts (for a review, see Anda, et al., 1990). Glassman, et al.
(1990) found that individuals in the general population who had experienced major depressive disorder were more likely to have a history of regular smoking than those who had never experienced major depression. They also found that smokers with major depression were less successful in quitting than those without it. In another study, Covey, et al. (1990) attempted to uncover the reasons depressed persons were less successful in smoking cessation programs. They found that in the first week of a behaviorally oriented intervention, the frequency and intensity of depressed mood were higher among subjects with a history of depression, and dysphoric symptoms were associated with a poorer treatment outcome.

The relationship of stress to depression has been remarked upon by Selye (1976), the father of stress research. He considered depression to be one of the many self-observable signs of stress, particularly the unpleasant variety he called distress. Supporting this view, Phelan, et al. (1991) found that both chronic occupational and domestic stress were related to major depressive disorder and current depressive symptoms in over 1500 married professional and managerial employees of a major corporation. In depressed patients, stressful life events and long-term difficulties have been associated with depression severity, treatment response, and relapse (Reno and Halaris, 1990).

The relationship between depression and diet is less well characterized. Certainly weight change and psychological status have been shown to covary, both in clinically depressed populations (e.g., Stunkard, et al., 1990) and in obese subjects attempting to lose weight. Few studies, however, have investigated the effect that diet composition has on depression. A low-carbohydrate diet in nondepressed female athletes was associated with significantly greater self-reported depression scores than were medium- and high-carbohydrate diets (Keith, et al., 1991). In other studies, subjects suffering
from premenstrual syndrome (Wurtman, et al., 1989) or from seasonal affective disorder (Wurtman and Wurtman, 1989) were found to crave and significantly increase their consumption of carbohydrates. Eating a carbohydrate-rich, protein-poor meal prior to menses was then associated with improved scores on the Hamilton depression scale for the subjects with premenstrual syndrome. A relationship between fat intake and depression has not been found, though there is intriguing speculation that linoleic acid, an Ω-6 polyunsaturated fatty acid, is a promoter of depression while fish oil, an Ω-3 fatty acid, is a prophylactic against depression due to the effects these compounds have on macrophages (Smith, 1991).

How depression and lifestyle relate to CHD

To be tenable, a theory relating depression to atherosclerosis or CHD manifestations must not only be conceptually appealing but also biologically plausible. Speculation as to plausible mechanisms of depression’s effect on the cardiovascular system abounds, but two types of mechanisms in particular have received support: (1) influences on other, mostly behavioral, risk factors; and (2) psychophysiological processes set in motion by the biochemical changes in depressed states. The possibility that CHD causes depression should not be ignored, but this discussion confines itself to the ways in which depression may play a causal role in disease.

Influences on other risk factors

As discussed earlier in this chapter, depression has been associated with such standard risk factors as smoking, lack of exercise, and obesity. An indirect relationship between depression and old age has also been observed, perhaps through the mediation of social isolation, poor health, or other
factors (Epstein, 1988). Depression may also act to reduce patient motivation to adhere to treatment regimens in recovering from MI (Blumenthal, et al., 1982).

Depression may interact with any of these risk factors, then, in their association with CHD, and analyses should take this possibility into account. As mentioned above, Kaplan and his colleagues found that the relationship between smoking and carotid atherosclerosis by a factor of three depending on the level of depression (Bower, 1992). Since depressed subjects are more likely to smoke and less likely to quit (Glassman, et al., 1990; Anda, et al., 1990), the relationship between depression and smoking is especially important.

Depression may also confound the observed relationship (or dilute a true relationship) between risk factors and CHD, and Pattillo (1990) has speculated that some of the contradictory results in the Type A literature may be due to a failure to account and control for depression and other psychosocial factors.

**Psychophysiological processes**

Depression initiates at least three physiological pathways to cardiovascular disease, all of which may operate concurrently, by increasing (1) serum lipid levels, (2) serum cortisol levels, and (3) sympathetic nervous system activity.

The first pathway involves hyperlipidemia, an established risk factor. Several studies have shown that free fatty acid and cholesterol levels are elevated in depression (Mueller, et al., 1970; Rahe, et al., 1971), either because of catecholamine-induced lipolysis or decreased glucose utilization, both of which have also been reported in depression. Some authors have also
implicated the hyperlipidemic effect of steroids such as cortisol, which are secreted in response to stress (Mueller, et al., 1970).

In addition to raising blood lipid levels, cortisol, an adrenocortical hormone, increases blood pressure and peripheral resistance of the blood vessels and causes vasoconstriction. Elevated levels of cortisol have been associated with angiographically demonstrated early atherosclerosis (Troxler, et al., 1977). Cortisol levels are often elevated in depression, and hypercortisolism is considered a crude biological marker of severe depression (Reus, 1988). It is not clear, however, whether the elevations in cortisol produced by depression are of sufficient magnitude and duration to have adverse cardiovascular effects.

In the third and best-studied pathway, also called the hyperreactivity or hyperresponsivity hypothesis, sympathetic nervous system arousal leads to release of the catecholamines epinephrine and norepinephrine. High levels of catecholamines have a number of deleterious effects on the cardiovascular system, as discussed by Price (1982) and Fielding (1991). Within the coronary arteries, they may cause endothelial damage, thus promoting the development of atherosclerosis, and they effect vasoconstriction, which may compromise blood flow and precipitate ischemic events. In very high levels, catecholamines may also be toxic to the myocardium, increasing the risk of arrhythmias and sudden death. Blood pressure, heart rate, and myocardial oxygen demand all rise under the influence of catecholamines, and platelet aggregation and “sludging” of red blood cells are enhanced as well, increasing the risk of clot formation.

While sympathetic activation has been more extensively studied in Type A subjects, alterations in norepinephrine systems in the brain have long been implicated in depression (Reus, 1988), and peripheral norepinephrine
has also been shown to be elevated in subjects with major depressive disorder (Esler, et al., 1982). Some researchers have suggested that increased sympathetic tone causes the elevated heart rate and decreased heart rate variability that have been observed in depressed patients with CAD (Carney, et al., 1988). Decreased heart rate variability can reflect either increased sympathetic or decreased parasympathetic tone, either of which may predispose the patient to potentially fatal arrhythmias (Dalack and Roose, 1990).

Pattillo (1990): Depressive behavior and cardiac death

Pattillo investigated whether a "Depressive Behavior Pattern" could predict cardiac death in a case-control study of patients drawn from the Recurrent Coronary Prevention Project (RCPP). The RCPP randomized 862 non-smoking, non-diabetic post-MI patients under 65 into an experimental group that received cardiac counseling plus Type A behavioral counseling, and a control group that received cardiac counseling only. After 4.5 years, experimental subjects showed a statistically significant decrease in Type A behavior and had a 44% lower rate of nonfatal MI and cardiac death (Friedman, et al., 1984).

The Type A counseling taught participants to manage stress and excessive emotional reactions, to use techniques of progressive muscle relaxation, and to establish new values and goals. As Friedman and Booth-Kewley (1987) noted, such counseling may have affected depression in addition to Type A behavior.

Pattillo developed a novel method of assessing depressive symptoms from Videotaped Structured Interviews for Type A behavior that were administered at the beginning of the RCPP. The Depressive Behavior Pattern
was meant to encompass a range of cognitive, affective, and behavioral manifestations of depression and not merely the more extreme signs and symptoms that would warrant a psychiatric diagnosis of major depressive disorder. The mean Baseline Depressive Behavior Pattern score of the 60 deceased cases was 133.2 (SD 52.0), while that of the 60 matched survivor controls was 74.2 (SD 33.7), a highly significant difference ($t = -7.37, p < .0001$). When scores from both cases and controls were compared to the overall median score, 70% of those who died had depressive scores above the median.

Using Type A videotaped structured interviews (VSIs) appealed to Pattillo for two reasons. One, VSIs are widely used and offer an abundant resource for researchers. And two, a clinical interview-based assessment follows in the “rich tradition of clinical assessment and diagnosis in the medical and psychological sciences” (p. 5). Making use of an interview avoided one of the major methodological problems of previous studies of depression and CHD — the “looking good” and “faking bad” biases imposed by self-report questionnaires. Another advantage of Pattillo’s methodology was the continuous measure of depressive symptomatology provided by his rating scale. This prevented the loss of information entailed by more formal diagnostic categories or dichotomies.

Summary

To summarize, depression has been associated with poor health habits such as smoking and lack of exercise, with carbohydrate consumption, and with stressful events and situations. All of these associations have been observed both in clinically depressed populations and in general population samples.
A number of mechanisms linking depression and CHD have been postulated, including interactions with standard risk factors, elevations in serum lipid levels, increases in cortisol secretion, and sympathetic nervous system arousal, and all have some scientific support. This is clearly an area with plenty of room for investigation.

Pattillo (1990) found that depressive behavior was predictive of cardiac death in a sample of post-MI patients. The present study extends his work to subjects with angiographically documented coronary atherosclerosis who participated in the Lifestyle Heart Trial. The study’s rationale, research questions, and research hypotheses are presented in the next chapter.
Chapter 5. Rationale, questions, and hypotheses

Rationale for the current study

To test the hypothesis that depression, lifestyle, and CHD are related in some way, one would like to be able to conduct a large, prospective study of initially healthy individuals using a sensitive, simple instrument to assess the degree of depression; an accurate and reliable measure of lifestyle practices; and a sensitive, simple, and safe technique to directly assess the degree of CHD. These individuals would be reassessed at periodic intervals over a long follow-up period to see what temporal relationship, if any, emerged between the variables.

In practice, of course, instruments for simply, reliably, and safely assessing depression and CHD have yet to be perfected. Self-report measures of depression, while simple to administer, may suffer from the same problems associated with self-report measures of Type A, which the Booth-Kewley and Friedman (1987) meta-analysis showed to be less predictive of outcome than interview-based measures. Clinical diagnoses of depression have the advantage of the diagnostician’s experience and expertise, but typically provide only a dichotomous measure of depression (i.e., present vs. absent). Aware of these methodological difficulties, Pattillo (1990) endeavored to combine only the most appealing features of self-report scales and clinical diagnoses, creating an interview-based rating scale which yielded a continuous measure of depressive symptomatology.

The assessment of diet, exercise, stress management practices, and smoking, whether it be by questionnaire or by personal interview, requires a cooperative, well-instructed subject. The method of assessment must be
accurate and complete without being so taxing to subjects as to discourage them from finishing it. The Lifestyle Heart Trial researchers were able to achieve these goals using a questionnaire that assessed participation in exercise and stress management activities, a three-day diet history and food frequency questionnaire, and, for subjects who had quit smoking for the study, random blood samples drawn for a nicotine metabolite.

In assessing severity of CHD, researchers have until relatively recently only been able to use manifestations — angina, MI, cardiac death, EKG changes — as indices of the underlying disease process. In some cases, even more indirect measures have been used, such as the incidence of angioplasty or coronary artery bypass grafting. These measures fail to quantify the pathological basis of CHD, atherosclerosis. Angina, for example, is a completely subjective manifestation, and several studies have demonstrated a negative correlation between degree of angina and extent of coronary atherosclerosis (e.g., Jenkins, et al., 1983; Elias, et al., 1982). And since consideration of surgical intervention is influenced by anginal complaints, using angioplasty or coronary artery bypass as an outcome measure may pose a subject selection bias.

With the advent of angiography, however, researchers can directly, sufficiently reliably, and objectively quantify the degree of atherosclerosis in the coronary arteries. While the use of angiography in research raises several methodological controversies (see Pearson, 1984; Pickering, 1984), the coronary arteriogram is generally considered the “gold standard” for evaluating coronary anatomy. Quantitative angiography can provide a continuous measure of atherosclerosis, accounting for number, location, and severity of occlusions. And serial angiograms can demonstrate even small changes in coronary artery disease in a relatively short time, permitting much
smaller sample sizes and shorter study periods than research using CHD manifestations as an outcome variable.

As discussed in the previous chapter, the progression from healthy coronary arteries to atherosclerotic arteries to CHD manifestations depends on a number of different risk factors, all of which may have different degrees of importance during the progression. While further research will also be needed to clarify the second step of the sequence, i.e., the interplay of risk factors and atherosclerosis in producing CHD manifestations, the objective here is to investigate the first step of the sequence, by examining the relationship between depression and lifestyle changes. Clearly, a prospective study using an interview-based continuous measure of depression on subjects undergoing comprehensive lifestyle modification is well suited to this objective. Further research will bring in the coronary atherosclerosis data obtained by serial quantitative angiography and assess their relationship to depression. The present study sets the stage for this research.

Research questions and hypotheses

This study addresses the following two research questions:

(1) What effect does the Lifestyle Heart Trial intervention have on depressive style?

The Lifestyle Heart Trial achieved a substantial and long-term change in behavior, and its subjects experienced a modest but statistically significant regression of coronary atherosclerotic lesions over 1.25 years, on average. It is hypothesized that the LHT intervention led to a decrease in depressive style and that the decrease may have influenced the lifestyle changes in some way to cause a regression in coronary lesions. Thus depressive style scores in the
experimental group should decrease from baseline to follow-up but remain unchanged or become worse in the control group.

(2) What is the relationship of baseline depressive style to one-year lifestyle adherence in the experimental group, the control group, and in both groups combined?

It is hypothesized that depressive style at baseline will be associated with the degree of adherence in both groups. Thus those who are less depressed at baseline will display a greater degree of adherence during the course of the study.

The next chapter describes the methods used to test these hypotheses.
Chapter 6. Methods

Design

This is a study using baseline and follow-up data from the first phase of the Lifestyle Heart Trial (LHT), a controlled, randomized clinical trial begun in 1986. The mean duration between baseline and follow-up Videotaped Structured Interviews was 15 months (SD 3.3), but ranged from 10 to 26 months. The data include measures of lifestyle practices (diet, exercise, stress reduction, and smoking) as well as videotaped interviews from which depressive style scores are obtained. (Other data are described later in this chapter.)

The study reconstructs a prospective course of events with existent data, making it possible to determine whether the appearance of one variable precedes another in time.

Subjects

The subjects of this study will be the 42 subjects who participated in the first phase of the Lifestyle Heart Trial and for whom relevant data are available and who consented to participate in the research. The recruitment and characteristics of the Lifestyle Heart Trial subjects are described in the Chapter 3.

Of the 48 subjects in the first phase of the LHT, one declined to participate in the present study, one had a VSI that was inaudible, and five had only a baseline or follow-up VSI. The latter five subjects were considered ineligible for the study, but due to a recordkeeping error, the VSI of one of them was rated and is included in the analyses using baseline data. For the
analyses using only depressive style, there were thus 42 subjects: 24 experimental and 18 control. Because some of these subjects had adherence data missing, analyses of depressive style and adherence included 19 experimental and 17 control subjects.

Among the 42 subjects, there was one woman in the experimental group and four in the control group. One experimental subject and one control subject were Hispanic, one control subject was black, and the rest were white.

The Videotaped Structured Interview (VSI)

All of the VSIs were performed by the same interviewer (Nancy Fleischmann) and ranged from 12 to 32 minutes in duration (mean 20.6 minutes, SD 4.3 minutes). The baseline VSIs were administered after all other tests, including diagnostic coronary angiograms, had been performed, and after they had agreed to participate in either the experimental or control group. The experimental subjects had thus committed themselves to making the required lifestyle changes by the time they were interviewed.

Appendix A lists the questions asked in the VSI. Friedman and Powell (1984) believe the VSI to be the most sensitive instrument yet developed for Type A behavior because it takes into account newly discovered indicators of Type A behavior and because it assesses both symptoms (subjective, self-reported manifestations) and signs (objective, observed manifestations) of the "medical disorder" of Type A behavior. Signs of Type A behavior include such psychomotor features as abnormal speech rhythms, grimaces, clenched fists, etc., and only observation, preferably in a standardized context, allows their assessment.
Conceptualization of depression: The construct of "depressive style"

The label "depression" implies a simple dichotomy between those who have it and those who do not, the distinction being made at an arbitrary degree of symptomatology. For many reasons, however, a more precise index of individual symptomatology is desirable for research purposes. In order to elucidate different types and degrees of depression, the assessment of depression should take into account the profile of symptoms as well as the intensity of their expression.

The present study uses the concept of "depressive style" to achieve these goals. Depressive style describes a broader range of experience than does "depression," including "feelings, moods, attitudes, cognitions, speech stylistics, facial expressions, and postures associated with depression" (Pattillo, 1990, p. 10). It may be considered a quality that everybody possesses to some degree, along a continuum from very low to very high, and only the upper end of this continuum might represent a state more formally diagnosed as depression or "major depressive disorder."

Assessment of depressive style: The Pattillo Depressive Style Scale

If, as Friedman and Powell (1984) and others believe, an interview provides a better assessment of personality than a pen-and-paper self-report checklist, one would ideally conduct a "Depressive Style Structured Interview" to rate depressive style. The costs of training interviewers and of conducting interviews often dissuade researchers from using interview-based instruments, however. To realize the benefit of such an instrument without all of its costs, Pattillo (1990) developed a rating scale that allowed an assessment of depressive style to be made in other structured interviews, such
as the VSI for Type A behavior. This rating scale has great practical value because of the great number of such interviews already extant and available for study. It was further developed into the Pattillo Depressive Style Scale (PDSS) used in the current study (see Appendix B).

As a rating scale applied to interviews conducted for unrelated purposes, the PDSS requires a certain conceptual orientation. Since PDSS raters are not at liberty to personally elicit features of depressive style from subjects, they must fully exploit their powers of observation, interpretation, and inference (as well as their training in the use of the PDSS). Based on Pattillo’s findings with an earlier version of the PDSS, it is hypothesized that the observable expression of depressive style and non-depressive style characteristics is at the very least a valid proxy for the actual presence or absence of those characteristics in the subjects’ natural setting. Since these characteristics are postulated to be directly related to lifestyle and CHD events, PDSS scores would thus be indirectly related. (Whether PDSS scores are themselves directly related to lifestyle and CHD events is a possibility to be explored in future research.)

The PDSS is thus designed to rate self-reported and observable characteristics of depressive style in whatever standardized context they are manifested, such as the VSI. If a behavior is not manifested during the VSI, the relevant item receives the lowest possible rating (0), even though the behavior might be manifest in another situation or if a different question were asked.

Development of the PDSS

The original rating scale designed by Pattillo (1990) drew from the Hamilton Rating Scale for Depression (Hamilton, 1969), the Beck Depression
Inventory (Beck, et al., 1961), the MMPI-Depression (D) scale, and the DSM-IIIR. Pattillo also used expert consultation and clinical judgment based on the content of the VSI to refine the instrument (personal communication, 1992). After testing 95 items, 32 were selected to make up the final scale, and they were grouped into five categories (depressed mood, low self-worth, worries/complaints, depressed speech, and sad/depressed facial).

The current scale builds on the original one, with four major modifications. First, Pattillo and I decided to expand the scale to include as many conceivable features of depressive style as we thought practical. By doing so we hoped to avoid missing any potential features of depressive style, with the knowledge that unreliable or redundant items could be identified and excluded from analysis and later versions of the scale. During several discussions and with references to the literature, we enumerated as many features of depressive style as possible. After testing these items on VSIs from the RCPP, we eventually settled on 38 depressive style items with 10 global and two overall ratings as well, all with non-depressive counterparts (described below). The 38 features hypothesized to characterize depressive style were organized into eight categories, both to facilitate use of the scale and to lay out a first approximation of the potential dimensions of depressive style. These categories were not created by any statistical method; they are conceptually rather than empirically derived and the items within may or may not correlate in practice.

Second, we added a converse scale to assess healthy, non-depressive or “positive” style, composed of approximate counterparts to each depressive style item. Again, we hoped to gain as much information as possible from the videotaped interviews, and we believed that “positive” personality features and behaviors might factor into an analysis of DS and CHD.
Therefore, there are actually two separate scales within the PDSS: a depressive style and a positive style scale. Raters are instructed to consider these two scales independent and to not let a high depressive style score, for example, necessitate a low positive style score.

Third, we changed the scoring system. Originally, items were rated as 1 (mild), 2 (moderate), or 3 (severe) each time they appeared, until a frequency "ceiling" was reached. (The ceiling — 10 for some items and five for others — prevented overweighting of items that occurred almost constantly during the interview and allowed the rater to attend to other behaviors.) This system, while predictive of outcome in Pattillo's (1990) study, seemed unwieldy and difficult to learn. The present instrument uses five-point scales that allow raters to account for both frequency and degree of expression. Zero is considered the anchor point, indicating non-manifestation or absence of expression of a given behavior, with four being a high number of manifestations, very high expression, and/or a very strong impression of a particular item.

Fourth, for each of the eight categories of items, we created global ratings. (The last two categories, "Speech & facial expression" and "Demeanor & engagement," each yield two global ratings.) These global ratings allow the rater to weight and integrate the items within a category as he or she sees fit. We wanted to see how these subjective "gestalt" ratings would compare with more mathematical reductions of the data. In addition, we created two "overall impression" items at the end of the scale. The first is an overall depressive style/positive style item, for rating the subject's appearance, verbal content, and behavior over the entire interview. The second is a prediction of the subject's outcome, with the outcome of interest specified by the study.
design. The outcome of interest that was specified for this study was change in coronary atherosclerosis during one year of intervention.

Utility of the PDSS in other settings

As explained above, the scale in its present version was designed to be as sensitive as possible to the expression of depressive style and its converse. Some of the verbal content and observable behaviors rated in the PDSS are in fact frequently not observed during the VSI because of the nature of its questions. In another type of interview, however, the same verbal content or behaviors may be manifest quite clearly. Thus the comprehensiveness of the scale will make it useful for rating depressive style in other clinical or research settings. It will then facilitate a comparison of results from different studies, including those with different disease endpoints, such as cancer, arthritis, peptic ulcers, or AIDS.

Training

The drawback to the comprehensiveness of the revised scale, of course, is that it may be difficult to use and obtain interrater agreement unless the items are well defined and the raters well trained. Thus Pattillo and I undertook to write a manual of philosophical guidelines and scoring instructions that we agreed upon during a long period of testing different versions of the scale on VSIIs from the Recurrent Coronary Prevention Project. A copy of the manual is appended (Appendix C).

We each spent approximately 20 hours developing a working model of the scale, 10 hours refining the scale, and 10 hours training ourselves using VSIIs from the RCPP until we felt comfortable proceeding with the study. After I had viewed all the interviews, we met again for several hours to review ratings we had conducted on the same subjects and to discuss sources
of agreement and disagreement. With the manual, we anticipate that
training a new rater will take 10-20 hours.

Procedure for conducting ratings

The 83 interviews were rated over a period of three weeks. Each
interview was viewed once. Watching and scoring the interviews took from
20 to 40 minutes apiece.

During the course of the interview, for each of the 38 depressive and 38
positive items, running scores were kept as items were manifested, taking
into account in each instance the guidelines from the manual and the
intensity of the manifestation. At the end of the interview the items that had
accumulated scores usually received the highest score marked, unless there
was a compelling reason to reduce the score. Remaining items were either
not manifested during the interview and thus scored as 0, or required an
integration of data over the entire interview. Within each category of items
on the front side of the rating scale, the negative items were all scored before
beginning the scoring of the positive items. For Category A, "Mood," for
example, items 1 and 2 were rated before items 51 and 52. This reduced the
likelihood that scores on one side of the scale would influence those on the
other side.

After all the items on the front of the scale were completed, the global
ratings, #39-48 and #89-98, were scored, one category at a time. The rater used
the respective item ratings to decide upon a global rating, but did not rely
solely on them. This facilitated an intuitive weighting process whereby some
items figured more prominently than others in helping to determine the
global rating.
Finally, the overall impression ratings, #49-50 and #99-100, were made. The rater again made use of both item and global ratings to score these variables. For the "Prediction of outcome" variables (#50 and #100), all available information from the subject's interview was used — including, for example, mention of risk factors, medical history, and family history of CHD — in addition to the depressive style rating.

As much as possible, dates, assignment to treatment or control group, and whether the interview was baseline or follow-up were masked from the rater. Each interview began with the interviewer announcing the date, so the tapes were cued past this information. References during the interview to date (or current events), group assignment, or baseline vs. follow-up status were not deleted. While this was done because of practical constraints, references to group assignment or interview sequence could be expected to be more frequent among the experimental subjects and during the follow-up interview and thus deleting them would not completely remove this potential source of bias.

Dates were occasionally disclosed either by subjects' references to current events or by comments such as "my heart attack was three years ago, in 1985." However, subjects were enrolled in the Lifestyle Heart Trial over several years and thus the date of an interview does not indicate whether it is being conducted at baseline or one-year follow-up. In addition, the rater was not aware of when baseline interviews were begun in that study.

To examine the effect that knowledge of group assignment or interview sequence might have had, the rater made a judgment of his awareness of these variables. He either had no awareness of these variables, a mild suspicion that proved correct, a strong suspicion that proved correct, a mild suspicion that proved incorrect, or a strong suspicion that proved
incorrect. The results for group assignment are given in Table 6.2. Eleven experimental subjects and 12 control subjects engendered no awareness of their group assignment. Three experimental and one control subject engendered a mild suspicion of their group assignment during their follow-up interview that proved correct, one experimental subject engendered a mild suspicion during the baseline interview that proved correct, and one experimental subject engendered a mild suspicion during both interviews that proved correct. The final experimental subject engendered a strong suspicion during the baseline interview and a mild suspicion during the follow-up interview, both proving correct. A total of five control subjects engendered a mild or strong suspicion during the baseline interview or during both interviews that proved incorrect.

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<td>Key: 0:</td>
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<td>mild suspicion of group assignment that proves correct</td>
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<td>2:</td>
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</table>

Table 6.3 shows the primary rater’s judgment of interview sequence. By engendering suspicion of their group assignment, most of the experimental subjects also indicated that the interview was a follow-up one, since they probably would not have begun participating in the program at baseline. In some cases, though, the sequence of interviews was disclosed without revealing group assignment, as when a subject spoke of an
impending retirement during the first interview and of being fully retired during the second interview. Eleven experimental and 12 control subjects gave no indication of interview sequence during either interview. Three experimental subjects' follow-up interviews engendered a mild suspicion of the correct interview sequence, and seven experimental subjects' and two control subjects' follow-up interviews engendered a strong suspicion. One control subject's baseline interview engendered a strong suspicion of the correct interview sequence. Three control subjects gave the incorrect impression regarding interview sequence during their baseline interview or during both interviews.

Table 6.3. Primary rater's awareness of interview sequence (baseline vs. follow-up) due to information disclosed in interview

<table>
<thead>
<tr>
<th>Experimental Control</th>
<th>Baseline, follow-up (see key for explanation)</th>
</tr>
</thead>
<tbody>
<tr>
<td></td>
<td>0.0</td>
</tr>
<tr>
<td>11</td>
<td>3</td>
</tr>
<tr>
<td>12</td>
<td>—</td>
</tr>
</tbody>
</table>

Key:
0: no awareness of interview sequence
1: mild suspicion of interview sequence that proves correct
2: strong suspicion or interview sequence that proves correct
3: mild suspicion of interview sequence that proves incorrect
4: strong suspicion of interview sequence that proves incorrect

With regard to the first research hypothesis, that the lifestyle intervention would be associated with a decrease in depressive style scores, there is a risk of bias incurred by knowing (or suspecting) the group affiliation of a subject, particularly when the interview sequence is also known (or suspected). In fact, the seven experimental subjects who aroused a strong suspicion of their true group assignment during their second interview were the same seven who engendered a strong suspicion of their true interview
sequence. It is conceivable that they received lower depressive style ratings than they deserved because of experimenter bias or expectation.

Adherence and other data

All other data were collected as part of the Lifestyle Heart Trial. These include demographic information, risk factor data such as smoking status, lipid values, and blood pressure; baseline measures of disease severity; and adherence scores. Adherence was computed for four elements: aerobic exercise, diet, stress reduction practices, and smoking.\(^1\) For each element, a formula was devised such that zero reflected no adherence to the program recommendations and one reflected 100% adherence, i.e., a diet of 10% fat and 5 mg/day cholesterol, three hours of exercise and seven hours of stress management per week, and no smoking. Scores greater than one were possible.

The overall adherence score averaged the scores for each element:

\[
\frac{t + (u/35 + v/420)/2 + (x/3 + y/180)/2 + z}{4}
\]

where \(t\) is the smoking compliance (0 or 1), \(u\) is the number of stress reduction sessions per week, \(v\) is the number of minutes of stress reduction per week, \(x\) is the number of exercise sessions per week, \(y\) is the number of minutes of exercise per week, and \(z\) is the dietary compliance, which took into account dietary fat and cholesterol content.

\(^1\) The term “adherence” is applied to both the experimental and control group subjects, even though the latter were not asked to adhere to the LHT intervention. Adherence may be considered a measure of the degree to which the subjects practiced certain healthy behaviors.
Analysis

The PDSS has a total of 100 variables: 38 negative and 38 positive individual items, 10 negative and 10 positive global ratings, and two negative and two positive overall impressions. While the multitude of variables encourages raters to attend fastidiously to the behavior, verbal content, and appearance of the subjects, it poses a problem of excess for the statistician. A rational desire for economy dictated reducing the data to four negative scores and four positive scores. These were #49, “Pessimistic, depressive;” #50 “Deterioration or death;” a sum of #1-38, the individual items; a sum of #39-48, the global ratings; and the positive counterparts to these.

The four negative scores offer four views of the same data — the interview — and should be expected to contain some redundancy, as should the four positive scores. Correlation analyses were thus performed among the negative scores and the positive scores. In addition, the negative and positive subtotals for categories A-H were compared with the respective global ratings. (Categories G and H each contain items for two global ratings; thus there are eight categories but 10 global ratings.) This analysis indicated at a finer level how much agreement there was between the items and the globals.

While the eight summary scores offered the easiest reduction of the data, some caution must be exercised in interpreting them. The arithmetic sum of individual ratings weights all items equally, even though some are undoubtedly more “pathogenic” than others, and subjects with different profiles of individual item ratings may have the same summary scores but very different degrees or types of depressive style. Thus the summary scores may obscure meaningful differences in depressive style as well as the details of relationships with other variables.
From a practical standpoint, however, because the sample size in the current study is small, summary scores may be the only way to reach significance in associations. Also, because of the great number of possible item-by-item associations, some would be expected to be significant by chance alone (a Type I error).

Baseline differences between the experimental and control groups with respect to demographic information, risk factors, and lifestyle behaviors were assessed (by the LHT researchers) using conventional two-tailed $t$ tests.

For the continuous depressive and positive style measures (the item sums and global sums), between-group differences were assessed using one-way analyses of variance. For the ordered categorical data (the overall impressions), Fisher's exact probability test was used.

The relationship of baseline depressive style to overall adherence was assessed using single-variable linear regression. Each of the four depressive style measures was considered a single independent variable and adherence was the dependent variable.
Chapter 7. Results

Baseline depressive style and positive style

Correlations among the measures

A high degree of agreement was found among the four measures of depressive style and among the four measures of positive style at baseline (Figures 7.1 and 7.2). The correlations ranged from .65 to .94 among the depressive measures and from .59 to .95 among the positive measures. For both the depressive and the positive measures, the prediction of outcome variable had the lowest correlation coefficients, while the correlations between the item totals and global totals were the highest. This finding could

Figure 7.1. Intercorrelations among the four depressive style measures. Correlation coefficients are included in the lower triangle (the upper triangle is a transposition of the lower one).
have been expected given that the rater based his prediction of outcome on all available information, not just depressive style, and that the global ratings were based largely on the item ratings. The correlations between the depressive/positive style variable and the respective item and global totals were in the range of .81 to .87.

![Correlation Coefficients Diagram](image)

**Figure 7.2.** Intercorrelations among the four positive style measures. Correlation coefficients are included in the lower triangle (the upper triangle is a transposition of the lower one).

Correlation coefficients were also computed between each global rating and the sum of the respective category's items. For example, global rating #39, "Mood," was analyzed with the sum of items #1 and #2 in category A, "Mood." For the depressive side of the scale, these coefficients ranged from .62 to .92, averaging .84. For the positive scale, they ranged from .63 to .93, averaging .83.
From the above data it appears that the item total and the global total are virtually equivalent and interchangeable measures, that style is nearly equivalent to these two measures, and that prediction of outcome is a related but distinct measure that reflects the consideration of other information in the interview.

Between-group differences

Contrary to expectations, baseline differences between the experimental and control groups were significant \((p < .05)\) for two of the four depressive-style measures, "Total items" and "Total globals," and the difference in "Depressive style" was nearly significant (Table 7.1). None of the positive

<table>
<thead>
<tr>
<th></th>
<th>Mean (SD)</th>
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<tbody>
<tr>
<td></td>
<td>Experimental group (n=23-24)</td>
<td>Control group (n = 18)</td>
<td>(F)</td>
</tr>
<tr>
<td>Total individual negative items (#1-38)</td>
<td>20.04 (8.40)</td>
<td>27.11 (12.48)</td>
<td>4.81</td>
</tr>
<tr>
<td>Total global negative ratings (#39-48)</td>
<td>7.58 (3.20)</td>
<td>10.33 (4.31)</td>
<td>5.64</td>
</tr>
<tr>
<td>Depressive, pessimistic style (#49)</td>
<td>1.25 (.61)</td>
<td>1.78 (.88)</td>
<td>—</td>
</tr>
<tr>
<td>Prediction of negative outcome (#50)</td>
<td>1.42 (.93)</td>
<td>2.00 (.77)</td>
<td>—</td>
</tr>
<tr>
<td>Total individual positive items (#51-88)</td>
<td>37.29 (8.81)</td>
<td>33.33 (8.42)</td>
<td>2.16</td>
</tr>
<tr>
<td>Total global positive ratings (#89-98)</td>
<td>14.75 (4.20)</td>
<td>12.61 (4.27)</td>
<td>2.63</td>
</tr>
<tr>
<td>Optimistic, vibrant style (#99)</td>
<td>1.5 (.88)</td>
<td>1.17 (.92)</td>
<td>—</td>
</tr>
<tr>
<td>Prediction of positive outcome (#100)</td>
<td>1.33 (.70)</td>
<td>1.00 (.77)</td>
<td>—</td>
</tr>
</tbody>
</table>

* \(P\) values for continuous measures ("Total items" and "Total global ratings") obtained with one-way ANOVA. \(P\) values for ordered categorical data ("Style" and "Prediction of outcome") obtained with Fisher's exact test.

measures showed significant differences, although the positive item and global totals tended to be higher in the experimental group.
The distribution of scores for two of the depressive measures, "Depressive style" and "Total global ratings," are shown in Figure 7.3. These data suggest that the between-group differences were due to the distributions of each entire group rather than to a few outliers.

**Figure 7.3.** Histograms for baseline Depressive style (top) and baseline Negative global total (bottom), according to group. The entire distribution of control group scores is shifted to the right of the distribution of experimental group scores, and there are no outliers.

Net scores — positive minus depressive — were not calculated, but it is apparent from Table 7.1 that the between-group differences would be large. “Net total items” scores, for example, would be 17.25 for the experimental group and 6.22 for the control group. “Net prediction of outcome” would be .09 for the experimental group and -1.00 for the control group.
Changes in depressive and positive style: The effect of the intervention

Both groups had lower mean depressive style scores at follow-up than at baseline, with the exception of “Prediction of negative outcome” in the control group (Table 7.2). The magnitude of change was relatively small in both groups, approximately 10-20% of the initial scores in most cases. While the control group’s scores remained higher than the experimental group’s,

<table>
<thead>
<tr>
<th></th>
<th>Mean follow-up (SD)</th>
<th>Mean change from baseline (SD)</th>
<th>F</th>
<th>p*</th>
</tr>
</thead>
<tbody>
<tr>
<td></td>
<td>Experimental group (n=22-23)</td>
<td>Control group (n = 18)</td>
<td></td>
<td></td>
</tr>
<tr>
<td>Total individual negative items (#1-38)</td>
<td>18.39 (7.90)</td>
<td>23.94 (9.96)</td>
<td>3.97</td>
<td>.053</td>
</tr>
<tr>
<td></td>
<td>-2.13 (10.4)</td>
<td>-3.17 (10.5)</td>
<td>.10</td>
<td>.754</td>
</tr>
<tr>
<td>Total global negative ratings (#39-48)</td>
<td>7.13 (4.01)</td>
<td>9.44 (4.06)</td>
<td>3.32</td>
<td>.076</td>
</tr>
<tr>
<td></td>
<td>-70 (4.64)</td>
<td>-89 (3.56)</td>
<td>.02</td>
<td>.885</td>
</tr>
<tr>
<td>Depressive, pessimistic style (#49)</td>
<td>1.13 (.87)</td>
<td>1.44 (.70)</td>
<td>—</td>
<td>.247</td>
</tr>
<tr>
<td></td>
<td>-.17 (.78)</td>
<td>-.33 (.69)</td>
<td>—</td>
<td>.050</td>
</tr>
<tr>
<td>Prediction of negative outcome (#50)</td>
<td>1.26 (1.05)</td>
<td>2.06 (1.00)</td>
<td>—</td>
<td>.021</td>
</tr>
<tr>
<td></td>
<td>-.17 (.98)</td>
<td>.06 (.64)</td>
<td>—</td>
<td>.201</td>
</tr>
<tr>
<td>Total individual positive items (#51-88)</td>
<td>35.90 (8.48)</td>
<td>33.56 (8.47)</td>
<td>.76</td>
<td>.388</td>
</tr>
<tr>
<td></td>
<td>-96 (8.25)</td>
<td>.22 (7.13)</td>
<td>.23</td>
<td>.637</td>
</tr>
<tr>
<td>Total global positive ratings (#89-98)</td>
<td>14.09 (4.43)</td>
<td>13.06 (3.90)</td>
<td>.61</td>
<td>.441</td>
</tr>
<tr>
<td></td>
<td>-.48 (4.03)</td>
<td>.44 (4.05)</td>
<td>.53</td>
<td>.472</td>
</tr>
<tr>
<td>Optimistic, vibrant style (#99)</td>
<td>1.26 (.92)</td>
<td>1.22 (.81)</td>
<td>—</td>
<td>.930</td>
</tr>
<tr>
<td></td>
<td>-.22 (.67)</td>
<td>.06 (1.16)</td>
<td>—</td>
<td>.020</td>
</tr>
<tr>
<td>Prediction of positive outcome (#100)</td>
<td>1.39 (.99)</td>
<td>1.00 (.84)</td>
<td>—</td>
<td>.465</td>
</tr>
<tr>
<td></td>
<td>.09 (1.08)</td>
<td>0.0 (.77)</td>
<td>—</td>
<td>.667</td>
</tr>
</tbody>
</table>

* P values for continuous measures (“Total items” and “Total global ratings”) obtained with one-way ANOVA. P values for ordered categorical data (“Style” and “Prediction of outcome”) obtained with Fisher’s exact test.

they showed a greater decrease in three of the four measures. The fact that the only measure to contradict this trend was “Prediction of negative outcome” further suggests that it is distinct from the other three. Only the
change in "Depressive style" and the follow-up "Prediction of negative outcome" showed statistically significant differences between the two groups.

Of the four positive measures at follow-up, the experimental group's mean scores had decreased for three and the control group's scores had increased for the same three. The exception was again the "Prediction of outcome" variable. The magnitude of change in all cases but one was less than 10% of the initial score. Only the change in "Optimistic style" was significantly different in the two groups, with the control group's scores increasing and the experimental group's scores decreasing.

The correlation among the change scores was lower than it was among the baseline scores, reflecting the added variance of the follow-up measurements. For the depressive measures, the coefficients ranged from .35 to .85 (mean .57), and for the positive measures, from .50 to .88 (mean .66). Again, the lowest correlations involved the prediction of outcome variable and the highest were between the item total and global total measures.

**Depressive style and adherence**

The ability of baseline depressive style to predict adherence was assessed using simple linear regression analysis. None of the four measures was a significant predictor in either the experimental or the control group (Table 7.3). The regression coefficient is expressed in units of adherence per unit of negative item total. Thus, in the experimental group, the best estimate of the relationship between adherence and negative item total showed adherence to decrease .007 units per unit increase in negative item total. This translates into a decrease of approximately .2 adherence units over
Table 7.3. Linear regression analysis using single baseline depressive style measures as independent variables and overall adherence as the dependent variable

<table>
<thead>
<tr>
<th></th>
<th>Depressive style</th>
<th>Prediction of negative outcome</th>
<th>Total negative items</th>
<th>Total negative global ratings</th>
</tr>
</thead>
<tbody>
<tr>
<td></td>
<td>regress. coeff.</td>
<td>p</td>
<td>regress. coeff.</td>
<td>p</td>
</tr>
<tr>
<td></td>
<td>(SE)</td>
<td></td>
<td>(SE)</td>
<td></td>
</tr>
<tr>
<td>Experimental group (n = 19)</td>
<td>-.010 (.091)</td>
<td>.92</td>
<td>.020 (.055)</td>
<td>.72</td>
</tr>
<tr>
<td>Control group (n = 17)</td>
<td>-.035 (.084)</td>
<td>.69</td>
<td>-.116 (.091)</td>
<td>.22</td>
</tr>
<tr>
<td>Combined (n = 36)</td>
<td>-.133 (.087)</td>
<td>.14</td>
<td>-.106 (.073)</td>
<td>.15</td>
</tr>
</tbody>
</table>

the range of approximately 30 units of difference in the negative item totals, as Figure 7.4 shows. While this is the best estimate of the relationship, there is a good deal of uncertainty in the data. The confidence interval (± 2 SE) for the relationship between negative item total and adherence in the control group, for example, ranges from -.010 to .002.

The within-group analyses are important because, while it appears that significance was almost attained in the combined-group analyses of depressive style and adherence, this apparent relationship was induced by the confounding effect of the intervention. That is, the experimental group subjects exhibited both significantly higher adherence and, by two measures, significantly lower depressive style than did the control group subjects, but within the experimental group, there was little relationship between depressive style and adherence. Figure 7.4 provides an example of this pattern.
Figure 7.4. Comparison of relationship between baseline negative item total and overall adherence in three groups: experimental, control, and combined. Note that adherence is uniformly high in the treatment group, and that negative item totals tend to be higher in the control group, thus inducing a regression line in the combined analysis whose slope is greater than those in the within-group analyses.
Chapter 8. Discussion

Summary of the present study

This study examined the effect that a comprehensive program of lifestyle changes had on depressive style and the relationship of depressive style to lifestyle behaviors. Based on a review of the literature, clinical judgment, and earlier work by Pattillo (1990), it was hypothesized that depression would be a key psychological variable in the equation between lifestyle changes and atherosclerotic heart disease.

A depressive style scale with a positive counterpart was developed to rate observable and verbal behavior of subjects in a videotaped structured interview for Type A behavior. The purpose of the positive scale was primarily exploratory, and its data were reported alongside those of the depressive style scale. Four measures of both depressive style and positive style were extracted from the data for analysis. The four measures were highly correlated with each other on both sides of the scale, especially the two sum measures. One measure, “Prediction of negative/positive outcome,” had lower correlations with the other three measures, probably because it took into account factors other than the hypothesized features of depressive or positive style.

The results of the study failed to support the hypotheses that 1) depressive style would decrease in the experimental group more than in the control group, and 2) baseline depressive style would be predictive of the degree of lifestyle adherence in both the experimental and the control group. These results are discussed below.
Baseline differences in depressive and positive style

The research design, objectives, and questions of this study were predicated on an assumption that did not hold, namely, that there would be no significant between-group differences in depressive style at baseline. Instead, the experimental group had a significantly lower mean score on the “Total negative items” and “Total negative globals” measures and a marginally significantly lower “Depressive style” score. The differences on the positive side of the scale were smaller and nonsignificant, but on all four measures the experimental group scored higher.

One can appeal to methodological explanations or speculate as to possible reasons for a true difference in these measures at baseline.

Methodological explanations

This study tested a new rating scale and a trained but relatively inexperienced rater. Although Pattillo’s original rating scale was found to have predictive validity of recurrent coronary death, the current PDSS has not been validated in any way. (Pattillo has rated a sample of the VSIs used in this study to provide an estimate of interrater reliability, but the results are not yet available.) One could argue that any findings obtained by this study would be suspect because of the potential for measurement error. If this were the case, however, associations would tend to be diluted rather than enhanced. The differences at baseline were quite remarkable given the relatively small group sizes, and it seems unlikely that measurement error would have created a distribution of scores that was divided along group lines.

Another possible explanation for these findings is experimenter bias. It is conceivable that the experimental subjects tended to disclose their group
status during the interview and thus influenced their ratings. The disclosure could have been overt enough to have been duly noted by the rater, or just subtle enough to plant unconscious bias in the rater’s mind. Experimenter bias would be most likely if both group assignment and interview sequence were known or suspected, as it was for 10 experimental subjects during their second interview. Only three subjects, however, were suspected to be experimental group members during their baseline interview, and thus it seems unlikely that the between-group differences at baseline would be affected much if the depressive scores of these three subjects were lower than they should have been. Also, the control group showed a greater mean decrease in three of the depressive style measures, whereas experimenter bias would have promoted a greater decrease in the experimental group.

Other explanations

If the baseline differences are real, what could account for them? After all, there were no significant differences in demographic characteristics, risk factor status (except for higher HDL cholesterol and apolipoprotein A1 in the control group), subjective and objective measures of disease severity, the relevant lifestyle behaviors, and various psychosocial measures (Scherwitz, et al., 1991).

Given the recruitment procedure of the Lifestyle Heart Trial, however, it is conceivable that the experimental and control subjects differed in several key ways. After completing their initial angiograms but before being told of the study, potential subjects were randomly assigned to one of the two groups and invited to participate. While the volunteer rate was nearly equal in the two groups, those who agreed to join the experimental group were agreeing to commit substantial time and effort in making fairly radical long-term
changes in their lifestyle. The subjects who volunteered for the control group, on the other hand, were not required to change their behavior or to devote a great deal of time in participating. The experimental subjects may thus have been more motivated to change, more optimistic, and less depressed; they may have had lifestyles more conducive to the demands of the intervention (e.g., time to exercise and practice stress management techniques, a work schedule that allowed time off for the week-long retreat, etc.); and they may have had more supportive social networks. Factors such as these would tend to give the subjects lower depressive and higher positive scores.

It is also conceivable that the subjects who volunteered for the control group tended to do so because they were depressed, lonely, seeking attention, or anxious about their health. These motivations might then be manifested in the VSI and contribute to higher depressive and lower positive scores. Subjects of similar psychological makeup in the pool of patients recruited for the experimental group would have had to decline to participate for there to be a skewed representation; this might have happened if, for example, they tended to be less willing to meet the demands of that group.

Since the PDSS includes items that directly or indirectly measure the lifestyle behaviors targeted by the LHT intervention, the control group may have rated higher on those items because they generally exercised less, did not talk about practicing stress management, and did not refer to the social support engendered by regular group meetings. There are relatively few of these items, however. Future analyses can nevertheless test this possibility by identifying the items or groups of items that accounted for most of the difference between the two groups. If they are items such as "Lack of or decrease in exercise," one can control for their effects.
Another possible explanation is that the baseline measures are not really baseline measures at all, because the subjects are already influenced by knowledge of the program and the enthusiasm and involvement of the researchers. This exposure to the intervention could have affected some of the cognitive and self-report items on the PDSS and thus rendered the initial depressive style assessment an early outcome measure.

Changes in depressive and positive style: The effect of the intervention

Perhaps more surprising than the baseline differences was the fact that these differences were maintained at follow-up, and the only significant differences between the two groups in the degree of change they underwent were contrary to expectations: the control group showed a greater decrease in the "Depressive style" measure and a greater increase in the "Optimistic style" one. The control group improved more in all the change measures except for the predictions of outcome, in fact. The improvement was just enough to make the differences in negative item and global totals marginally nonsignificant. The prediction of negative outcome measure, however, became a significant differentiator between the two groups.

Either the experimental group failed to improve as much as expected, or the control group improved more than expected, or both. It is possible that the intervention, particularly the group meetings, affected the psychology of the experimental subjects such that they introspected more and became more self-aware. They may then have verbalized more of their introspections during the follow-up interview, possibly incurring higher scores on some depressive style items. While they still showed an overall decrease in depressive style, the decrease might have been greater if they had been as
"blissfully ignorant" as before. This argument requires a good deal of conjecture, however.

The control group may have improved by virtue of the Hawthorne effect, i.e., the attention they received, even as control subjects, was sufficient to elevate their mood, self-esteem, and outlook, especially during the time of testing. The control subjects underwent the same four-day battery of tests that the experimental subjects did at baseline and follow-up, including a well-received, all-expenses paid trip to Houston for a PET scan. While the Hawthorne effect might be expected to occur at baseline in addition to follow-up, the psychological benefits of the control subjects' participation might have accumulated over the study period.

A final interpretation is that the immediate psychological impact of the angiographic procedure or the diagnosis of coronary artery disease prompted a depressive reaction in both groups of subjects at baseline, but that after one year most of them had come to terms with the knowledge of their disease and were less depressed, irrespective of their group assignment. This is one of the drawbacks of studies using patients undergoing coronary angiography — it is difficult to rule out the possibility that the procedure or the necessity for it changes a person's behavior and psychology at least transiently. This methodological flaw, if it can be called that, does not explain the between-group differences, however.

Depressive style and adherence

None of the four baseline depressive style measures showed a significant ability to predict overall adherence within each group. This is surprising given that exercise, stress reduction, and reductions in angina have been associated with decreases in depression in previous studies. These
findings are somewhat consistent, however, with those of Scherwitz, et al. (1991), who also found no significant correlations between baseline psychosocial factors and overall adherence in the experimental group. Scherwitz and his colleagues did find several significant correlations in the control group, however. They speculated that psychosocial factors played some role in making lifestyle changes outside of a structured program such as the LHT, but that the LHT intervention was powerful enough to achieve high levels of adherence regardless of individual psychological makeup.

In the current study, depressive style measures were no more predictive of adherence in the control group than in the experimental group, with the possible exception of “Prediction of negative outcome,” which had regression coefficients of -.116 in the control group ($p = .22$) and .020 in the experimental group ($p = .72$). While the specified “negative outcome” was an increase in coronary atherosclerosis, Ornish, et al. (1990) showed that changes in atherosclerosis were associated with adherence, so the rater’s prediction should have pertained indirectly to degree of adherence as well.

Implications for further research

Depressive style and the PDSS

Our understanding of depression, especially as it relates to diseases such as CHD, is still limited. Although everyone has an idea of what is meant by depression, it can and has been defined in many ways. “Depressive style” is a term coined to acknowledge the ambiguity of definition by suggesting that everyone displays some degree of depressive style if not outright depression.

The Pattillo Depressive Style Scale was intended to be a kind of dragnet for capturing any and all suspected features of depressive style, releasing them
only when they have been found innocent of significant meaning. In the present study, four measures of depressive style were used, primarily because they offered a gross reduction of the data and were simple to obtain. Further research will have to identify redundant items, clarify the relative weights of items, and perhaps develop a streamlined scale that includes only the most important cluster of items. The simultaneous examination of positive style was only just begun in this study, and further research should determine whether and how it can be combined with depressive style to achieve a more thorough characterization of individual psychology and behavior.

Lifestyle interventions

Several implications for future lifestyle interventions, as well as caveats for interpreting findings from the Lifestyle Heart Trial, have emerged from this study. First of all, subject recruitment should minimize the possibility of self-selection bias. That is, researchers should make certain that subjects who volunteer for an experimental group are as equivalent as possible to subjects who volunteer for a control group. One way to at least test for this equivalence would be to administer VSIs (or other interviews) to all consenting patients before they undergo coronary angiography. Then, after the VSIs have been conducted, the subjects can be randomly invited to participate in one group or the other. Obviously this will require more VSIs to be conducted, but the information gained from subjects who decline to participate in each group can then be compared with the information from those who volunteer to participate.

This strategy would also limit the possibility of some baseline tests reflecting an early treatment effect. If, as in the LHT, baseline interviews are administered after subjects are introduced to the program, it is difficult to
remove the influence the intervention has already had on the subjects' hopes for the future.

**Depressive style and coronary atherosclerosis**

It will eventually be of interest to examine the relationship between depressive style and changes in coronary lesions and the ability of depressive style and adherence in combination to predict changes in atherosclerosis. For this purpose it may be fortunate that depressive style is not significantly related to adherence, as its effect independent of adherence may be more readily assessed. Further work must refine the rating scale, evaluate the role of positive style, and identify ways to extract the most meaningful information from the scale in relatively few variables. At that point the information gained from this study can be applied to an investigation involving coronary atherosclerosis. In anticipation of this investigation to come, the final chapter reviews the literature concerning depression, atherosclerosis, and CHD.
Chapter 9: Depression, atherosclerosis, and CHD: An anticipatory review of the literature

A review of the literature concerning depression, atherosclerosis, and CHD is included in this thesis to justify the effort of the present study and to whet the reader's appetite for the research that will follow it.

Studies of depression and atherosclerosis

Few studies to date have applied coronary angiography to investigating depression and atherosclerosis, and most of these have been cross-sectional surveys. In one of the earliest such studies, Zyzanski, et al. (1976) found that subjects with greater obstruction of four major coronary arteries scored significantly higher on a depression scale derived from the MMPI (this study will be discussed further below). In a recent report, Kaplan and colleagues failed to find a direct relationship between depression and carotid atherosclerosis in a large sample of Finnish men, but showed that the link between atherosclerosis and two of its risk factors, cigarette smoking and fibrinogen, a blood clotting factor, differed by depression status (Bower, 1992). Depressed smokers had three times as much atherosclerosis as nondepressed smokers, controlling for years of smoking and cigarettes per day, and depressed subjects with high levels of fibrinogen had four times as much atherosclerosis as nondepressed subjects with the same levels of fibrinogen.

As atherosclerosis is the presumed basis for CHD manifestations, more research is clearly needed in this area. In the meantime, one can evaluate the more abundant literature regarding depression and CHD manifestations and speculate as to the role of atherosclerosis.
Studies of depression and CHD events

Consistent with Figure 4.1, many avenues of investigation lie before the researcher wishing to study depression and CHD. That depression occurs in response to a serious physical insult such as myocardial infarction is widely accepted and documented (see Hackett, 1985; Fielding, 1991). But depression can also occur prior to serious illness, in which case it may be implicated in its etiology, or depression may have a reciprocal relationship with illness and play a role in recovery from it.

This section, with one exception (Mendes de Leon, 1991), reviews only studies using some measure of depression as an independent variable and some measure of CHD or atherosclerosis as a dependent or outcome variable. Two recent papers provide a more inclusive review of the literature concerning depression and CHD, and the reader is referred to them for other studies (Fielding, 1991; Dalack and Roose, 1990).

Meta-analytic reviews are discussed first, followed by summaries of the more important studies contained within those reviews and results from significant studies carried out more recently.

Meta-analytic reviews

As with the more abundant Type A literature, interpreting the findings in depression-CHD studies is beset with complications: some studies are prospective but most are cross-sectional, depression is defined in different ways and assessed with different instruments, a variety of CHD endpoints are used, and methodological quality varies greatly.

Thankfully, statistical techniques of meta-analysis have provided a way to see the forest for the trees. Meta-analysis summarizes the results of any
number of independent studies in a single variable, the combined effect size. This value, sometimes provided by the mean of the product-moment correlation coefficients obtained from each study, describes the strength of the relation between the variables of interest — in this case, depression and CHD — across all the studies. While the technique is not immune to bias and problems of interpretation, it "seems particularly appropriate for ... coronary-prone personality literature, in which a lot of research has been conducted but ... is in so much theoretical disarray" (Booth-Kewley & Friedman, 1987, p. 345).

Booth-Kewley & Friedman (1987) searched the literature from 1945 to 1984 for studies that used certain personality traits as an independent variable and some manifestation of CHD or atherosclerosis as a dependent variable. To be included in the meta-analysis the study had to have quantitative data and enough information to allow calculation of effect size and significance level. The personality variables included Type A and its components as measured by a number of instruments, speed & impatience, time urgency, job involvement, competitiveness/hard driving/aggressiveness, anger, hostility, aggression, depression, extraversion, anxiety, and several combinations of these. Fifteen studies were found in which both depression and CHD were studied.

The results regarding depression surprised even the authors (Table 9.1). The combined effect size for the association between depression and all disease outcomes (i.e., CHD and atherosclerosis) in 13 independent samples

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1 See discussion below and in Booth-Kewley & Friedman (1987), Matthews (1988), and Friedman & Booth-Kewley (1988).
2 Four prospective studies were added to the sample during 1985 and 1986.
was .205 ($p < .000001$). Only SI-assessed Type A (.221) had a higher combined effect size among the personality variables under study.

<table>
<thead>
<tr>
<th>Outcome variable</th>
<th>No. of published reports</th>
<th>No. of independent effect sizes</th>
<th>Combined effect size ($r$)</th>
<th>Combined $z$</th>
<th>$p$</th>
<th>Fail-safe $N^*$</th>
</tr>
</thead>
<tbody>
<tr>
<td>All disease outcomes**</td>
<td>15</td>
<td>13</td>
<td>.205</td>
<td>6.16</td>
<td>&lt;.000001</td>
<td>170</td>
</tr>
<tr>
<td>All CHD outcomes</td>
<td>13</td>
<td>11</td>
<td>.225</td>
<td>6.44</td>
<td>&lt;.000001</td>
<td>158</td>
</tr>
<tr>
<td>Myocardial infarction</td>
<td>9</td>
<td>7</td>
<td>.259</td>
<td>5.24</td>
<td>&lt;.000001</td>
<td>65</td>
</tr>
<tr>
<td>Angina</td>
<td>4</td>
<td>3</td>
<td>.165</td>
<td>4.29</td>
<td>.000009</td>
<td>18</td>
</tr>
<tr>
<td>Atherosclerosis</td>
<td>3</td>
<td>3</td>
<td>.146</td>
<td>1.55</td>
<td>.0610</td>
<td>—</td>
</tr>
</tbody>
</table>

(Adapted from Booth-Kewley and Friedman, 1987.)

* An “estimate of the number of unpublished, nonsignificant studies that would have to exist for the obtained probability value to be rendered nonsignificant” (Booth-Kewley and Friedman, 1987, p. 348).

** I.e., CHD and atherosclerosis.

For depression and all CHD outcomes (MI, angina, cardiac death, and/or electrocardiographic abnormalities), the combined effect size in 11 independent samples was .225 ($p < .000001$), higher than for Type A behavior, hostility, or any other personality variable. When the outcome was myocardial infarction specifically, the combined effect size in seven independent samples was .259 ($p < .000001$), again the largest $r$ obtained. The combined effect size between depression and angina was .165 ($p = .000009$), the second largest $r$, but this was based on just three samples.

For the final outcome variable studied, atherosclerosis, the combined effect size for depression was .146, which did not reach statistical significance ($p = .0610$). Three other personality variables had higher effect sizes. Although this finding was based on only three independent samples, it raises an interesting question: Since the effect size for atherosclerosis was lower than for MI (.259) and angina (.165), might depression contribute to CHD
events (e.g., MI and angina) more than it contributes to the pathological basis of those events, i.e., atherosclerosis?

Table 9.2. Relationship between depression and all disease outcomes (CHD and atherosclerosis) in prospective, cross-sectional, pre-1977, and 1977-on studies

<table>
<thead>
<tr>
<th>Study type</th>
<th>No. of published reports</th>
<th>No. of independent effect sizes</th>
<th>Combined effect size (r)</th>
<th>Combined z</th>
<th>p</th>
<th>Fail-safe N</th>
</tr>
</thead>
<tbody>
<tr>
<td>Prospective studies</td>
<td>6</td>
<td>3</td>
<td>.168</td>
<td>3.77</td>
<td>.00008</td>
<td>13</td>
</tr>
<tr>
<td>Cross-sectional studies</td>
<td>11</td>
<td>11</td>
<td>.204</td>
<td>4.49</td>
<td>&lt;.0000001</td>
<td>112</td>
</tr>
<tr>
<td>Pre-1977 studies</td>
<td>12</td>
<td>10</td>
<td>.187</td>
<td>5.03</td>
<td>&lt;.0000003</td>
<td>84</td>
</tr>
<tr>
<td>1977-on studies</td>
<td>3</td>
<td>3</td>
<td>.279</td>
<td>4.17</td>
<td>.00002</td>
<td>17</td>
</tr>
</tbody>
</table>

(Adapted from Booth-Kewley and Friedman, 1987.)

Booth-Kewley & Friedman (1987) also performed analyses of prospective vs. cross-sectional studies and pre-1977 vs. 1977-on studies, though only for all disease outcomes (Table 9.2). As was true for nearly all the personality variables, there were more cross-sectional (11) than prospective (six) studies of depression. Unlike most of the other variables, however, depression had a combined effect size nearly as high in prospective (.168) as in cross-sectional studies (.204). Only hostility (.135 prospective and .166 cross-sectional) and anxiety (.136 and .122, respectively) showed a similar degree of agreement. For comparison’s sake, SI-assessed Type A behavior, consistently the best Type A predictor of outcomes in the meta-analysis, had a combined effect size of .238 in 14 cross-sectional studies but only .062 in seven prospective studies. The authors caution that conclusions from the small number of prospective studies must be limited, however.

Comparison of the pre-1977 and 1977-on studies is also interesting. Booth-Kewley and Friedman conducted this comparative analysis to quantify the differences over time in CHD research, particularly regarding Type A behavior. As suspected, the apparent relationship between Type A behavior
and CHD has weakened. For SI-assessed Type A behavior, the combined
effect size in pre-1977 studies was .271, while that in 1977-on studies was .174

g in both cases). (For a discussion of possible reasons for this
phenomenon, see Booth-Kewley and Friedman [1987] and Miller, et al. [1991].)

The trend was just the opposite for depression, however. Whereas the
combined effect size in 10 independent samples was .187 in pre-1977 studies of
depression, in three 1977-on studies, it was .279 (p = .00002). Only the variable
"competitiveness/hard driving/aggressiveness" showed a similar trend.

Matthews (1988) reanalyzed Booth-Kewley and Friedman's (1987) data,
using different decision rules and incorporating the results of more recently
published studies. Most significantly, she chose to exclude cross-sectional
studies because of their "methodological and interpretative difficulties"
(p. 373). Among the difficulties she cited were prevalence-incidence bias,
which describes the underrepresentation of certain subgroups of subjects,
unmasking or detection signal bias, and diagnostic suspicion bias.

Matthews also decided to compare Booth-Kewley and Friedman's
method of weighting each study equally with a method that weights for
number of participants in each study. The results of her meta-analysis of
depression and CHD are shown in Table 9.3. Just three prospective studies of
depression and CHD met her criteria, and only one of these was significant at

<table>
<thead>
<tr>
<th>Variable</th>
<th>No. of independent significance tests</th>
<th>No.</th>
<th>Combined z (unweighted)</th>
<th>p</th>
<th>Combined z (weighted)</th>
<th>p</th>
</tr>
</thead>
<tbody>
<tr>
<td>Depression</td>
<td>3</td>
<td>1</td>
<td>2.36</td>
<td>.009</td>
<td>.29</td>
<td>.386</td>
</tr>
</tbody>
</table>

(From Matthews, 1988.)
the .05 level. Using Booth-Kewley and Friedman's method of weighting, depression was a significant predictor of CHD incidence ($p = .009$) in these three studies. When weighting for number of participants, however, depression was not a significant predictor ($p = .386$).

Friedman and Booth-Kewley (1988), however, responded that Matthews' method overweighted large studies since $Z$ already takes $N$ into account. Friedman and Booth-Kewley also believed that if prevalence-incidence bias were operating, it would tend to weaken the observed association of two variables, thus underestimating their true association.

Though the statistical bases for the two meta-analyses can be debated, there is agreement that very few prospective studies of depression as a risk factor for CHD have been performed and that future research should investigate the relationship further. A selected sample of the studies that have already done so are reviewed next.

Individual studies

Lebovits, et al. (1967) administered the MMPI to 1990 men aged 40-55 who were free of clinical CHD. Within four years, 106 men had developed CHD, as manifested by a fatal event ($n = 15$), a non-fatal MI ($n = 27$), or angina ($n = 51$; 13 CHD patients had to be excluded from the study). No statistically significant differences in MMPI scale scores were found between the deceased subjects and the survivors with angina. However, whereas just one of the 27 MI survivors, or 4%, had initial MMPI-Depression scores ≥ 2 SD above the mean, five of the 15 deceased subjects, or 33%, had elevated scores.

Bakker and Levenson (1967) administered the MMPI to 256 Caucasian men with arteriosclerotic heart disease. The subjects as a whole showed a high elevation of the "neurotic triad" of depression, hypochondriasis, and
hysteria scales. Approximately half of the subjects reported anginal symptoms, and there were no significant differences between these men and the symptom-free subjects. A number of the initially symptom-free subjects developed angina during the subsequent six months. These men (n = 6) were compared with those who remained symptom-free (n = 43) on MMPI scales. The average T-scores on the MMPI-D scale, which has a mean of 50 and SD of 10 in normative populations, were considerably higher for those who developed angina: T > 70 vs. T < 65. It is not clear, however, why approximately 80 subjects initially free of angina were not included in the analysis, and the relatively small sample sizes preclude conclusions.

In a retrospective study of white males who had suffered a myocardial infarction, Bruhn, et al. (1969) compared the MMPI scores of three groups of subjects: 30 “survivors” of one or more MI’s prior to the study period, 17 “nonsurvivors” who had survived one or more MI’s prior to entry but experienced a fatal MI during the study period (26.4 months after entry, on average), and 30 matched controls who were free of clinical CHD. The only significant difference in baseline MMPI scores of the survivors and nonsurvivors was in the depression scale. The mean T-score of the nonsurvivors was 71.4; that of the survivors, 61.8 (t = 2.10, p < .05).

In the same study, Bruhn, et al. also administered the Welsh depression subscale of the MMPI to survivors and controls bimonthly for 1.5 years in order to better assess short-term changes in depression. The survivors were found to have significantly higher mean scores (Z = 3.18, p < .0007). The survivors were also found to have significantly higher D scores at the end of the study period than they did upon entry approximately 53 months earlier, while the controls’ scores showed a downward trend. Whereas the survivors’ initial D scores approximated those of the controls,
their final D scores were closer to the initial scores of the nonsurvivors. It is important to note that both the survivors and nonsurvivors had already suffered at least one MI, and that the authors did not control for disease severity.

In one of the first studies using angiographic data, Zyzanski, et al. (1976) administered several self-report scales to 94 men one day prior to diagnostic coronary catheterization. Scores on a short depression scale extracted from the MMPI were found to covary with the number of coronary vessels with ≥ 50% luminal obstruction. Subjects with two to four vessels affected (n = 58) had a mean T score of 57.6 while those with zero or one vessel affected (n = 36) had a mean T score of 51.9 (F = 12.25, p = .001). A positive correlation was also found between depression scores and angina intensity (F = 5.00, p = .01). Of the psychological variables examined — Type A behavior, anxiety, hypochondriasis, hysteria, and depression — only depression showed statistically significant relationships to both the pathological basis of CHD, atherosclerosis, and the clinical effect of CHD, angina. The authors found that the observed relationship between depression and vessel obstruction could not be explained by degree of angina pain, age, or prior experience of MI.

Friedman and Booth-Kewley (1987) conducted a case-control study of 50 men aged 40-68 who had suffered an MI and 50 matched, healthy controls. Using a scale from the Hopkins Symptom Checklist, depression scores were found to be significantly correlated with health status (r = .23, p < .05). Depression and anxiety scores were highly correlated with each other as well (r = .68). Multiple regression analyses of the personality variables measured showed that maximum predictability was obtained by the combination of anxiety and SI Type A, R = .37, and by the combination of depression and SI Type A, R = .31.
Carney, et al. (1988) conducted a one-year prospective study of 52 patients, approximately three-fourths of whom were male, with significant CAD (defined angiographically as ≥ 50% stenosis in one or more major coronary artery or branch). At baseline, before catheterization, nine (17%) met DSM-III criteria for major depressive disorder (MDD). MDD was not significantly related to age, clinical indices of disease, or severity of stenosis, but smoking was significantly more prevalent in the depressed patients (89% vs. 53%, *p* < .05). During the one-year follow-up period, seven (78%) of the depressed patients and 15 (35%) of the non-depressed patients experienced at least one major cardiac event: MI, cardiac death, coronary bypass surgery, or angioplasty. The presence of MDD had the strongest univariate correlation (*r* = 0.40) with the occurrence of these cardiac events of all the variables analyzed, including severity of CAD. The predictive value of MDD was independent of extent of coronary disease, smoking status, and left ventricular ejection fraction (an important prognostic indicator).

Because the study by Carney, et al. used a dichotomous measure of depression, a dose-response relationship could not be demonstrated. Also, assessment of depression was only made at baseline, so there was no information concerning the course of depression in either group of patients. And because all the subjects had some clinical indication of CHD prior to their entry into the study, it is impossible to say whether the depression or the CHD came first. However, as the authors concluded, the findings suggest that MDD is an important independent risk factor for major cardiac events following diagnostic coronary angiography.

A large prospective study by Barefoot, et al. (1989) found that MMPI-D scores failed to predict survival of 1,467 patients with ≥ 75% diameter narrowing of at least one coronary artery over follow-up periods of up to 9.2
years. While the null finding is important because of the large sample size, the authors focused on Type A data and did not conduct or report further analysis of depression data. Such analysis could have included assessment of the the ability of MMPI-D scores to predict other cardiac events, specifically nonfatal MI, coronary artery bypass surgery, and angioplasty. In fact, more than half of the patients underwent bypass surgery a median of 0.1 years after their initial angiogram and enrollment into the study and were excluded from further follow-up.

Mendes de Leon, et al. (1991) examined the behavioral and psychological variables that changed in Recurrent Coronary Prevention Project (RCPP) participants. The RCPP, as discussed in Chapter 4, randomized post-MI patients under the age of 65 into an experimental group that received cardiac counseling plus Type A behavioral counseling, and a control group that received cardiac counseling only. After 4.5 years, experimental subjects showed a statistically significant decrease in Type A behavior and had a 44% lower rate of nonfatal MI and cardiac death (Friedman, et al., 1984).

Mendes de Leon, et al. found that depression, as assessed by six items on self-report questionnaires, decreased significantly more in the experimental group than in the control group ($F = 13.10, p = .001$), and the decrease was unrelated to the number of counseling sessions attended. The authors speculated that the reduction in depression may have been an important ingredient in the treatment effect. Other variables were also affected by the treatment, however, and it would be difficult if not impossible to determine the effect that the change in depression, independent of other factors, had on outcome.
Summary of findings

Research on depression and atherosclerosis has been sparse, but several studies have found some relationship or an influence on other risk factors, and the combined effect size in Booth-Kewley and Friedman’s (1987) meta-analysis was .126 \((p < .0610)\), although this result was based on only three studies. The literature regarding CHD manifestations and depression has been nicely summarized by the same meta-analysis, which found that depression had the highest association with CHD of all the psychosocial variables studied \((r = .225, p < .0000001)\).

Of the eight individual studies of depression and CHD reviewed above, four used cohort designs, three used case-control designs, and two used cross-sectional surveys.\(^3\) Most of the subjects in these studies were male. Six studies used the MMPI-D (depression) scale or a derivative of it, two used short self-report measures, and one used a psychiatric interview to make a DSM-III diagnosis. The outcome variables included angina, nonfatal MI, fatal MI or other cardiac death, coronary bypass surgery, angioplasty, and coronary atherosclerosis. Although these studies applied different designs, different measures of depression, and different outcome variables to the investigation of depression and CHD, they nevertheless found associations fairly consistently. As Booth-Kewley and Friedman (1987) remarked, “Increased research attention should be focused on depression as a component of the coronary-prone personality” (p. 356).

\(^3\) One study (Bakker and Levenson, 1967) used both a cross-sectional survey and a cohort design. The study by Mendes de Leon, et al. (1991) was unique among the articles reviewed in that it assessed depression as an outcome variable in response to the RCPP intervention.
References


Appendix A: Videotaped Structured Interview

Questions asked during the Videotaped Structured Interview (from Friedman and Powell, 1984):

I. Questions for diagnosis and assessment of sense of time urgency

A. By elicitation of biographic and self-appraisal data

1. Does your wife ever tell you take things easier or slow down?
2. Do you think that you are a hard-driving, no-nonsense sort of achiever or do you believe that you tend to do things in a rather unhurried manner?
3. Do you walk fast? Do you eat fast? After you have finished eating do you like to sit and dawdle at the table, or do you like to leave and do something else?
4. If there are five or ten persons waiting to eat at a restaurant, would you wait? At the movie theater?
5. Do you frequently find yourself both listening to someone and also thinking about another subject? Do you find yourself doing this as your wife talks? What does she say when she discovers that you are doing this?
6. Do you frequently just daydream? What do you daydream about or why don’t you daydream?
7. Do you sometimes find yourself timing how long it takes you to do certain things like jogging, walking, auto trips, household chores, and so on?
8. When you are in the bathroom, do you sometimes do two things at once, such as reading trade or professional journals or washing your teeth as you shower?

B. By observation of psychomotor manifestations

1. Mr. Jones, what is your occupation or profession?
2. Would your 20-year-old self have been proud or disappointed in what you have accomplished at this time?
3. Do you feel that most of the important parts of your life are now behind you?
4. The following three questions are structured and delivered by the interviewer in a way that tempts the interviewee to finish the question/sentence. The interviewer proposes the questions, but begins to stutter before he finishes.
   Question 1. Most working people usually arise before 8:30 a.m. on weekdays, that is on Mondays, Tuesdays, Wednesdays, Thursdays, and Fridays, but may sleep longer on Saturdays and
Sundays. Now in your case, during the weekdays, at what time do you, uh, uh...

Question 2. The interviewer asks the interviewee about his hobbies. After the latter lists his hobbies, the interviewer asks: “Have you ever taken a snapshot of your family or friends?” The interviewee usually responds affirmatively. “Well, after you have taken the snapshot of your family member or friend, the films, did you develop them yourself or did you, uh, uh...”

Question 3. The interviewer first asks what kind of car the interviewee drives. Then: “Most persons in our city, particularly those people who have to go up and down a lot of hills during their driving, have their brakes checked or inspected or adjusted about every six months. Now in your own case, with respect to the brakes of your own car, during the last six months, have you had them, uh, uh...”

5. Do you like to go shopping with your wife? Why or why not?

II. Questions for diagnosis and assessment of free-floating hostility

A. By elicitation of biographic and self-assessment data

1. Do you admire and have as much respect and faith in doctors as your father and mother probably had? (Why not?)

2. Do you have children or nephews and nieces? When they were younger and you played games like checkers, dominoes, or cards, did you always let them win? Why?

3. All of us have insecurities about certain things. What are your insecurities about?

4. When you play a game with your contemporaries, do you play to win or do you play for the fun of it?

5. What do you do if a car ahead of you in your lane is going too slowly? Have you ever sworn at such drivers? Has your wife ever told you to “cool it”? How does she put it?

B. By observation of psychomotor manifestations

1. What sorts of events, activities, or actions tend to irritate you?

2. Do you feel that most of the important parts of your life are now behind you?

3. What would you do if you and your wife were at a fine restaurant and she was becoming ill from the cigar smoke reaching her from a man sitting at the table next to yours? Suppose you asked him to stop smoking and he said, “Drop dead, mister”? Suppose there was no other free table.

4. What would you do if you were driving your car at 80 miles an hour on a busy highway to get your sick wife to the hospital and suddenly you spotted in your rearview mirror the red light of a highway patrol officer obviously signaling you turn off the
highway? What would you do if this patrolman should reply, "I
don't give a damn about getting your wife quickly to the hospital,
I'm giving you a ticket right now and I'll give you another if you try
to speed again"?

5. I think you are absolutely wrong in your view about . . . Your
answer doesn't make much sense! (This is another attempt to elicit
anger or irritation.)
# Appendix B: The Pattillo Depressive Style Scale

<table>
<thead>
<tr>
<th>Randomized #:</th>
<th>Subject ID:</th>
<th>Rating date:</th>
<th>Time:</th>
<th>VCR Counter:</th>
<th>Rate:</th>
</tr>
</thead>
<tbody>
<tr>
<td>Order on tape:</td>
<td></td>
<td></td>
<td></td>
<td></td>
<td></td>
</tr>
</tbody>
</table>

## PATILLO DEPRESSIVE STYLE SCALE

### A. Mood

<table>
<thead>
<tr>
<th></th>
<th></th>
<th>Score</th>
</tr>
</thead>
<tbody>
<tr>
<td>1.</td>
<td>Self-reported depression</td>
<td>4 3 2 1 0 — 0 1 2 3 4</td>
</tr>
<tr>
<td></td>
<td>Self-reported happiness</td>
<td>51.</td>
</tr>
<tr>
<td>2.</td>
<td>Expressed sadness</td>
<td>4 3 2 1 0 — 0 1 2 3 4</td>
</tr>
<tr>
<td></td>
<td>Expressed joy</td>
<td>52.</td>
</tr>
</tbody>
</table>

### B. Interest & pleasure

<table>
<thead>
<tr>
<th></th>
<th></th>
<th>Score</th>
</tr>
</thead>
<tbody>
<tr>
<td>3.</td>
<td>Lack/loss of interest, pleasure</td>
<td>4 3 2 1 0 — 0 1 2 3 4</td>
</tr>
<tr>
<td></td>
<td>Same or greater interest, pleasure</td>
<td>53.</td>
</tr>
<tr>
<td>4.</td>
<td>Reduced activity level</td>
<td>4 3 2 1 0 — 0 1 2 3 4</td>
</tr>
<tr>
<td></td>
<td>Same or increased activity level</td>
<td>54.</td>
</tr>
</tbody>
</table>

### C. Self-appraisal

<table>
<thead>
<tr>
<th></th>
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<tbody>
<tr>
<td>5.</td>
<td>Hopelessness</td>
<td>4 3 2 1 0 — 0 1 2 3 4</td>
</tr>
<tr>
<td></td>
<td>Hopefulness</td>
<td>55.</td>
</tr>
<tr>
<td>6.</td>
<td>Helplessness</td>
<td>4 3 2 1 0 — 0 1 2 3 4</td>
</tr>
<tr>
<td></td>
<td>Strong self-efficacy</td>
<td>56.</td>
</tr>
<tr>
<td>7.</td>
<td>Disappointment, regrets</td>
<td>4 3 2 1 0 — 0 1 2 3 4</td>
</tr>
<tr>
<td></td>
<td>Pride, satisfaction, consentment</td>
<td>57.</td>
</tr>
<tr>
<td>8.</td>
<td>Talks of negative past events</td>
<td>4 3 2 1 0 — 0 1 2 3 4</td>
</tr>
<tr>
<td></td>
<td>Talks of positive past events</td>
<td>58.</td>
</tr>
<tr>
<td>9.</td>
<td>Self-criticism, self-blame, guilt</td>
<td>4 3 2 1 0 — 0 1 2 3 4</td>
</tr>
<tr>
<td></td>
<td>Positive self-regard, acceptance</td>
<td>59.</td>
</tr>
<tr>
<td>10.</td>
<td>Feels self-pity; victimized</td>
<td>4 3 2 1 0 — 0 1 2 3 4</td>
</tr>
<tr>
<td></td>
<td>Feels fortunate</td>
<td>60.</td>
</tr>
<tr>
<td>11.</td>
<td>Health worries/complaints</td>
<td>4 3 2 1 0 — 0 1 2 3 4</td>
</tr>
<tr>
<td></td>
<td>Positive health statements</td>
<td>61.</td>
</tr>
<tr>
<td>12.</td>
<td>Death-related statements</td>
<td>4 3 2 1 0 — 0 1 2 3 4</td>
</tr>
<tr>
<td></td>
<td>Life-affirming statements</td>
<td>62.</td>
</tr>
</tbody>
</table>

### D. Outlook

<table>
<thead>
<tr>
<th></th>
<th></th>
<th>Score</th>
</tr>
</thead>
<tbody>
<tr>
<td>13.</td>
<td>Externally oriented pessimism</td>
<td>4 3 2 1 0 — 0 1 2 3 4</td>
</tr>
<tr>
<td></td>
<td>Externally oriented optimism</td>
<td>63.</td>
</tr>
<tr>
<td>14.</td>
<td>Criticism of others</td>
<td>4 3 2 1 0 — 0 1 2 3 4</td>
</tr>
<tr>
<td></td>
<td>Praise of others</td>
<td>64.</td>
</tr>
<tr>
<td>15.</td>
<td>Work-related worries/complaints</td>
<td>4 3 2 1 0 — 0 1 2 3 4</td>
</tr>
<tr>
<td></td>
<td>Positive work-related statements</td>
<td>65.</td>
</tr>
<tr>
<td>16.</td>
<td>Family worries/complaints</td>
<td>4 3 2 1 0 — 0 1 2 3 4</td>
</tr>
<tr>
<td></td>
<td>Positive family statements</td>
<td>66.</td>
</tr>
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### E. Somatic symptoms

<table>
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<tr>
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<tbody>
<tr>
<td>17.</td>
<td>Cardiovascular complaints</td>
<td>4 3 2 1 0 — 0 1 2 3 4</td>
</tr>
<tr>
<td></td>
<td>Good cardiovascular functioning</td>
<td>67.</td>
</tr>
<tr>
<td>18.</td>
<td>CHD risk factors</td>
<td>4 3 2 1 0 — 0 1 2 3 4</td>
</tr>
<tr>
<td></td>
<td>CHD protective factors</td>
<td>68.</td>
</tr>
<tr>
<td>19.</td>
<td>Appetite disturbances</td>
<td>4 3 2 1 0 — 0 1 2 3 4</td>
</tr>
<tr>
<td></td>
<td>Good appetite</td>
<td>69.</td>
</tr>
<tr>
<td>20.</td>
<td>Sleep disturbances</td>
<td>4 3 2 1 0 — 0 1 2 3 4</td>
</tr>
<tr>
<td></td>
<td>Good sleep</td>
<td>70.</td>
</tr>
<tr>
<td>21.</td>
<td>GI disturbances</td>
<td>4 3 2 1 0 — 0 1 2 3 4</td>
</tr>
<tr>
<td></td>
<td>Good GI functioning</td>
<td>71.</td>
</tr>
<tr>
<td>22.</td>
<td>Lack of or decrease in exercise</td>
<td>4 3 2 1 0 — 0 1 2 3 4</td>
</tr>
<tr>
<td></td>
<td>Exercise</td>
<td>72.</td>
</tr>
<tr>
<td>23.</td>
<td>Other somatic complaints</td>
<td>4 3 2 1 0 — 0 1 2 3 4</td>
</tr>
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<td></td>
<td>Other positive somatic signs</td>
<td>73.</td>
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### F. Social support

<table>
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<tr>
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<tbody>
<tr>
<td>24.</td>
<td>Feels unloved, unappreciated</td>
<td>4 3 2 1 0 — 0 1 2 3 4</td>
</tr>
<tr>
<td></td>
<td>Feels loved, appreciated</td>
<td>74.</td>
</tr>
<tr>
<td>25.</td>
<td>Feels isolated, lonely</td>
<td>4 3 2 1 0 — 0 1 2 3 4</td>
</tr>
<tr>
<td></td>
<td>Feels connected, supported</td>
<td>75.</td>
</tr>
<tr>
<td>26.</td>
<td>Loss of spouse, family, job</td>
<td>4 3 2 1 0 — 0 1 2 3 4</td>
</tr>
<tr>
<td></td>
<td>Security in family, job</td>
<td>76.</td>
</tr>
<tr>
<td>27.</td>
<td>Other interpersonal stressors</td>
<td>4 3 2 1 0 — 0 1 2 3 4</td>
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<td>Other interpersonal support</td>
<td>77.</td>
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### G. Speech & facial expression

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<tr>
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<th>Score</th>
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</thead>
<tbody>
<tr>
<td>28.</td>
<td>Depressed, sad speech</td>
<td>4 3 2 1 0 — 0 1 2 3 4</td>
</tr>
<tr>
<td></td>
<td>Animated, joyful speech</td>
<td>78.</td>
</tr>
<tr>
<td>29.</td>
<td>Monotonic (flat) speech</td>
<td>4 3 2 1 0 — 0 1 2 3 4</td>
</tr>
<tr>
<td></td>
<td>Full range of speech</td>
<td>79.</td>
</tr>
<tr>
<td>30.</td>
<td>Low volume</td>
<td>4 3 2 1 0 — 0 1 2 3 4</td>
</tr>
<tr>
<td></td>
<td>Good volume</td>
<td>80.</td>
</tr>
<tr>
<td>31.</td>
<td>Sad, tired expression</td>
<td>4 3 2 1 0 — 0 1 2 3 4</td>
</tr>
<tr>
<td></td>
<td>Energetic, interested expression</td>
<td>81.</td>
</tr>
<tr>
<td>32.</td>
<td>Little range of facial expression</td>
<td>4 3 2 1 0 — 0 1 2 3 4</td>
</tr>
<tr>
<td></td>
<td>Good range of facial expression</td>
<td>82.</td>
</tr>
<tr>
<td>33.</td>
<td>Eyes downcast, head down</td>
<td>4 3 2 1 0 — 0 1 2 3 4</td>
</tr>
<tr>
<td></td>
<td>Good eye contact</td>
<td>83.</td>
</tr>
<tr>
<td>34.</td>
<td>Mouth downturned</td>
<td>4 3 2 1 0 — 0 1 2 3 4</td>
</tr>
<tr>
<td></td>
<td>Smiling, pleasant</td>
<td>84.</td>
</tr>
</tbody>
</table>

### H. Demeanor & engagement

<table>
<thead>
<tr>
<th></th>
<th></th>
<th>Score</th>
</tr>
</thead>
<tbody>
<tr>
<td>35.</td>
<td>Tense, impatient, hostile</td>
<td>4 3 2 1 0 — 0 1 2 3 4</td>
</tr>
<tr>
<td></td>
<td>Relaxed, pleasant, at ease</td>
<td>85.</td>
</tr>
<tr>
<td>36.</td>
<td>Fidgety, restless</td>
<td>4 3 2 1 0 — 0 1 2 3 4</td>
</tr>
<tr>
<td></td>
<td>Calm</td>
<td>86.</td>
</tr>
<tr>
<td>37.</td>
<td>Poor concentration; forgetful</td>
<td>4 3 2 1 0 — 0 1 2 3 4</td>
</tr>
<tr>
<td></td>
<td>Aware, alert</td>
<td>87.</td>
</tr>
<tr>
<td>38.</td>
<td>Apathetic, withdrawn</td>
<td>4 3 2 1 0 — 0 1 2 3 4</td>
</tr>
<tr>
<td></td>
<td>Engaged, energetic</td>
<td>88.</td>
</tr>
</tbody>
</table>
I. Global ratings

Mood

39. Depressed 4 3 2 1 0 — 0 1 2 3 4 Happy, satisfied 89. ____

Interest & pleasure

40. Low or decrease 4 3 2 1 0 — 0 1 2 3 4 High or increase 90. ____

Self-appraisal

41. Negative 4 3 2 1 0 — 0 1 2 3 4 Positive 91. ____

Outlook

42. Criticism, complaints, worries 4 3 2 1 0 — 0 1 2 3 4 Positive statements 92. ____

Somatic symptoms

43. Negative 4 3 2 1 0 — 0 1 2 3 4 Positive 93. ____

Social support

44. Stress, loss, insecurity, isolation 4 3 2 1 0 — 0 1 2 3 4 Connected, supported, secure 94. ____

Speech

45. Sad, monotonic 4 3 2 1 0 — 0 1 2 3 4 Animated; full range 95. ____

Facial expression

46. Sad, flat 4 3 2 1 0 — 0 1 2 3 4 Engaged, interested; full range 96. ____

Demeanor

47. Tense, hostile 4 3 2 1 0 — 0 1 2 3 4 Relaxed, easygoing, pleasant 97. ____

Engagement

48. Apathetic, withdrawn 4 3 2 1 0 — 0 1 2 3 4 Engaged, interactive 98. ____

J. Overall impression

Depressive style

49. Pessimistic, depressive 4 3 2 1 0 — 0 1 2 3 4 Optimistic, vibrant 99. ____

Prediction of outcome

50. Deterioration or death 4 3 2 1 0 — 0 1 2 3 4 Improvement or survival 100. ____
Appendix C. PDSS Manual of Instruction

General guidelines for rating

0: Non-manifestation or absence of expression
1: At least one manifestation of slight expression (may be very subtle, hinted at, or implied)
2: One or more manifestations of mild to moderate expression
3: Several manifestations of moderate expression or one manifestation of high expression (making a strong impression on the rater)
4: High number of manifestations and/or very high expression (making a very strong impression on the rater)

The two sides of the PDSS should be considered separate scales, and ratings on one side should not influence ratings on the other. Therefore, a subject may have high scores on both sides of a particular item.

It is helpful to think of each item as having a "control lever" that the rater can slide back and forth from 0 to 4. For each item this lever starts at 0, the anchor point, and when a certain behavior is observed, the lever can be moved up to a higher number. The rater can keep track of the lever’s location with a dot or a slash through a number on the scale. In general, levers should be not be moved down by new information, but there may be exceptions if a rater later thinks he or she misinterpreted a remark or gave too much weight to it. When the interview is over, the rater can circle the numbers that the levers rest on, and not have to rely on a more subjective process of remembering and integrating information. Some items, of course, will require some judgment after the interview is over, particularly those on the back side of the rating.

When making the final ratings, raters should do all the negative items within a category before doing the positive items in that category to minimize the influence the two sides have on each other.

It is easiest and most time-efficient to view the tapes just once. If all raters prefer, however, the tapes may be viewed twice or more. In any case, the number of viewings should be the same for all the tapes, and there should be a systematic way of viewing and rating. For example, if there will be two viewings, raters could concentrate on verbal content during the first viewing, and on observation of behavior during the second viewing.
Mood:

1. Ratings should be based entirely on the content of the subject’s speech and not a more subjective appraisal or inference. That is, the subject must say, “I feel/am depressed” (or down, down in the dumps, blue, in the doldrums, under the weather, sad, etc.). In general don’t allow the presumed etiology of the depression to affect the rating. Subjects who talk about feeling depressed after an MI should be rated the same as subjects who were depressed before the MI, other things being equal. When subjects speak of feeling depressed in the past, try to determine the present import of the depression. If a person was severely depressed after the death of his son 15 years ago and appears to be affected by the memory of his son and/or the depression, a 3 may be warranted. On the other hand, if someone speaks of a depression 15 years ago as though it has no affect on him currently (“Oh yeah, I got really depressed once when I was about 45 years old, but I saw a psychiatrist and after a few months or so, I was back to normal, and I’ve never felt that way since”), 1 may be the highest reasonable rating.

1: Currently or recently mildly depressed; or past episode(s) of depression
2: Currently or recently somewhat depressed; or past episode(s) of depression
3: Currently or recently moderately depressed, and/or severe depression in past
4: Currently or recently severely depressed, with or without past depression

2. Expressed sadness: Some inference is allowed to evaluate whether a subject experiences sadness in any context, specific or general. A subject may explicitly complain of being sad, which would also warrant a rating in (1), or he may express sadness more subtly. For example, he may say he loves to do something but cannot do it anymore (due to his health, age, retirement, etc.). A statement such as this is often tinged by some sadness, regret, or disappointment, earning a rating here and in (7) as well. Or he may bring up the subject of a close friend’s death, and from his facial expression, tone of voice, and speech content, the rater can infer that he is expressing sadness.

51. Self-reported happiness: Again, the content of the subject’s speech is the basis for the rating, as in (1). A general sense of happiness, such as in the statement, “I feel really good,” deserves a high rating, but subjects may also report happiness in many specific contexts, e.g., “I’m very happy with my progress since the heart attack,” “Work is going pretty well for me now,” “I’m
enjoying my family life a great deal." When subjects speak of happiness experienced in the past, use the guidelines for (1) above. Statements such as "I love to do X" should only be rated in (52) and (53) unless the subject is giving it as a reason for his or her happiness.

52. Expressed joy: Guidelines for (2) apply. "Enjoyment" of something, such as friends, hobbies, work, or life in general, is rated here. Someone may speak enthusiastically about his children, for example, leading to the inference that he enjoys spending time with them and is expressing that joy.

Interest & pleasure:

3. Lack/loss of interest, pleasure: This applies to the interest and pleasure people take in relationships, activities, jobs, avocations, hobbies, etc. Both the absolute amount and the decrease in amount are to be rated here. Thus the rater must make a subjective appraisal of the normal amount of interest and pleasure a healthy person would have and compare the subject to that standard. In response to the question "Do you have any hobbies?," answers such as "I'm really not interested in doing anything right now" are rateable. A decreased level of activity (rated in [4]) is often due to decreased interest or pleasure, but this should not be assumed. If subjects are clearly interested in an activity but can't participate in it because of their health, age, retirement, etc., they should not be rated here. Similarly, if they speak of a lack of energy, it is important to understand whether this is due to a lack of interest or due to physical complaints such as shortness of breath or chest pain, which would be rated in (11), (17), and perhaps (22).

0: No loss and normal or greater amount of interest and pleasure
1: Slight decrease in or slightly lower than normal amount of interest and pleasure
2: Mild to moderate decrease or moderately lower than normal amount
3: Marked decrease or profoundly lower than normal amount
4: Profound decrease or complete lack of interest and pleasure

4. Reduced activity level: This item rates the decrease in activity level more than the absolute amount of activity, and ignores the reason for it. "I used to bowl quite a bit, but I don't do that so much anymore on account of my health," or "I like to hunt, fish, and camp but haven't done any of that in a long time," would be rateable here.
53. Same or greater interest, pleasure: Rate both the absolute amount and the increase in amount, if any. If the subject is interested in activities and derives pleasure from them but gives no indication of how much, rate 2. If the subject's interest in activities has increased but is still less than that of most people, rate 1. Similarly, if the subject's interest in activities has decreased but is still greater than most people's, rate 3. Subjects may speak of an interest in or devotion to hobbies, travel, work, children, and other activities or people. If a subject talks of being very active, e.g., walking three miles a day, the rater may consider this activity to be a manifestation of interest and/or pleasure in this activity unless the subject states otherwise.

0: No interest & pleasure, and/or significantly less than before
1: Little interest & pleasure, and/or somewhat less than before
2: About normal amount, and/or same as before
3: More than normal, and/or more than before
4: Much more than normal, and/or much more than before

54. Same or increased activity level: The guidelines for (53) apply to this item as well.

Self-appraisal: Generally these are items which apply to the subjects' appraisals of themselves and not of their external environment or other people (which are to be rated under "Outlook").

5. Hopelessness: A global despair, generally future-oriented, as in "I probably won't live long enough to see my daughter graduate from college." If a subject answers "yes" to the question, "Do you feel the most important parts of your life are behind you?" rate 1 (or more, depending on the elaboration).

6. Helplessness: A sense that one cannot change things, that circumstances beyond one's control or other people (e.g., boss, wife) determine one's fate. This may be manifest when subjects speak of not being able to do something because of their health or doctor's orders or in such statements of resignation as "you just can't win." Whereas hopelessness is a despair about the future, helplessness is experienced in the present and is based on past experiences.

7. Disappointment, regrets:

8. Talks of negative past events: These must involve the subject but not necessarily be caused by him or her. (An event can be considered to involve a person if it had some emotional impact.
on that person.) In response to the question, “Some people are glad they had a heart attack. Are you?” subjects will of course speak of their MI. If they answer with just a few words, without expressing significant emotion or elaborating, they should not be rated. If, however, they appear to be emotionally affected by the memory of the MI, or if they talk at greater length about the MI or its consequences, they should be rated. More spontaneous, voluntary mention of negative past events should of course be rated as well. The rater should account for the significance as well as number of the events mentioned by the subject. A vague allusion to negative past events should be rated, but not as high as more specific, explicit reference to them.

9. Self-criticism, self-blame, guilt: Self-doubt and self-derogatory statements can be rated here. These include subtle statements of self-criticism, such as “I tend to work too hard,” or “I probably used to get too worked up about things before my heart attack,” or of self-doubt, such as “I sometimes wonder what people think of me,” or “I don’t think I’m really as good at X as people think.” Statements of guilt usually refer to doing somebody wrong, letting someone down, or doing something which adversely affected somebody else.

10. Feels self-pity: May be manifest when a subject talks about a family history of CHD or a feeling of not being appreciated (which may also be rated in [24]). This often overlaps with hopelessness (5) and hopelessness (6).

11. Health worries/complaints: This may overlap with (E.) Somatic symptoms.

12. Death-related statements: The assumption here is that if someone’s death is important enough to mention during an interview it has had an emotional impact on the subject. Any mention of a person’s death rates at least a 1. A family member’s or friend’s death or references to one’s own death automatically rates at least a 2. Statements referring to one’s own death are often disguised: “I didn’t know if I was going to make it when I had my first heart attack,” “I want to make sure that my family is taken care of should anything happen to me,” “The doctor told me I wouldn’t live six months if I didn’t stop smoking.”

55. Hopefulness: A conviction that the future holds a positive outcome, not simply the fragile hope that a negative outcome will not occur. For example, a robust, cheery subject may say, with a great deal of enthusiasm and conviction, ‘I’m planning
on retiring next year and then traveling around the world with my wife.” Another subject may be more tentative in saying, “I’m thinking about buying an RV, but I don’t know if I’ll be around long enough to enjoy it. I hope I will be, but I just don’t know.” The second subject may earn a rating of 1 for his “hoping,” but the first subject might earn from 2 to 4, depending on the rest of the interview, for his conviction that the future will come to pass as he wants it to. Responses to the question “Do you think the most important parts of your life are behind you?” often provide rateable content.

55. Strong self-efficacy: A belief that one is capable of achieving a particular goal or performing at a certain level, and that one has a certain measure of control over his or her destiny. A person may have a perception of high self-efficacy in one situation and not in another, but subjects should be given credit for each situation in which they perceive themselves to be self-efficacious and not rated for global self-efficacy. More weight can be placed on some perceptions of self-efficacy; doing well in a chosen profession probably means more to a person than being able to tie shoelaces well. Some subjects may have a perception of high self-efficacy that is not really accurate, and the rater should try not to be influenced by faulty perceptions. This item may overlap with (59).

57. Pride, satisfaction, contentment: “Pride” has several meanings. The pride that is also called conceit should not be rated here, nor should the pride that prevents someone from asking for help. In this context pride means “a reasonable delight in one’s position, achievements, possessions, etc.” (Webster’s). Some judgment is required, as claiming to be proud does not necessarily mean one is proud. An affirmative answer to the question, “Would your 20-year-old self be proud of what you’ve accomplished?” rates 1 (or more, depending on the elaboration).

58. Talks of positive past events: See guidelines for (8). Subjects often say they reminisce about positive past events when asked, “Do you frequently daydream?” In general, the more specifically and concretely they talk about positive past events, the higher the rating.

59. Positive self-regard, self-acceptance: This should be construed as a positive attitude, not simply an avoidance of self-criticism. It can be expressed in the context of family (“I’ve been a good father”), work (“I take a lot of pride in my work and do a good job”), or life in general (“I’m proud of what I’ve achieved”; this
is often offered in response to question, “Would your 20-year-old self be proud of what you’ve accomplished?). Self-forgiveness may also be rated here. When subjects are asked if their behavior might have caused their heart attack, for example, a rateable answer would be “Probably working too hard all those years. But I was supporting my family and nobody knew then what they know now about Type A.”

60. Feels fortunate: For example, a subject may feel fortunate to have survived an MI, to have been diagnosed with CAD before suffering an MI, to be alive, to have a supportive spouse, to have good doctors, etc.

61. Positive health statements: Includes information rated in (E.).

62. Life-affirming statements: Consider these to be the opposite of “death-related statements” and not simply any statements that reflect an enjoyment of life. Rateable examples: “I feel young,” “I plan on living a long time,” “My father died of a heart attack when he was 40, but if I stick with this program I think I’ll live till I’m 100,” “When I get better, I’m going to do X.”

Outlook: Items in this category attempt to capture subjects’ views of their environment, not of themselves per se. Comments about other people, a job, the economy, world affairs, etc. offer rateable material. Be careful not to rate self-appraisal content here.

13. Externally oriented pessimism: This should be distinguished from “Hopelessness” and “Helplessness” above, which refer to the subjects’ perceptions of themselves. Externally oriented pessimism refers to other people, a subject’s employer, the economy, institutions such as government or the health care system, and natural forces such as the weather. Essentially all worries and complaints other than those involving family or work are rated here.

14. Criticism of others: There is often rateable content in response to the question, “Do you have as much respect for and faith in doctors as your parents did?” This item applies to criticism of people in general (e.g., “A lot of people just don’t know what they’re doing”) or of specific people in specific situations (e.g., “There’s one teller at my bank who is really slow”). Criticism of people at work or of family members is rated here as well as in (15) or (16).
15. Work-related worries/complaints: Includes criticism of fellow employees and of supervisors, complaints about working conditions or pay, worries about job security, and anything else relating to a person's job. A person who is retired may talk about past worries, complaints, and criticisms, and these are rateable.

16. Family worries/complaints: Includes criticism of family members, concerns about their welfare if the subject should die, concerns about children, financial concerns affecting the family, etc.

63. Externally oriented optimism: See guidelines for (13).

64. Praise of others: See guidelines for (14).

65. Positive work-related statements:

66. Positive family statements:

Somatic symptoms: For symptoms that occurred in the past, rate one lower than if they were occurring currently.

17. Cardiovascular complaints: Angina (chest pain), shortness of breath, fatigue, inability to exercise because of cardiovascular functioning. This item should be restricted to manifestations (symptoms) of cardiovascular disease, not the presence of the disease or of risk factors (rateable in 19).

1: Rare angina; slight decrease in activity due to fatigue or shortness of breath
2: Occasional angina; moderate decrease in activity due to fatigue or SOB
3: Moderate or frequent angina; significant decrease in activity due to fatigue or SOB
4: Severe or frequent angina; no activity due to fatigue or SOB

18. CHD risk factors: Mention of the so-called “standard” risk factors: atherosclerosis, hypertension, high cholesterol or lipids, smoking, diabetes mellitus, family history of CHD, obesity, low HDL, high-fat diet, and lack of exercise. Psychological risk factors such as Type A behavior, stress, depression, anxiety, etc. are not to be rated. It is the subject’s mention of risk factors as risk factors that is sought here; mention of a risk factor outside of the context of CHD is not sufficient. For example, a subject may talk of enjoying rich French cuisine without any reference to its effect on his health, or it may be clear from what he is saying that he does not exercise, but he does not bring up the subject of exercise. Also, if the subject is obese, do not rate for obesity unless he or
she mentions it. Since prior MI, angioplasty, and coronary bypass surgery indicate that the subject has coronary artery disease, rate mention of these events here, too.

1. 1-2 risk factors
2. 2-3 risk factors, or prior MI, angioplasty, or coronary bypass surgery
3. 3-4 risk factors, or more than one prior MI, angioplasty, or bypass surgery
4. 4 or more risk factors or multiple MIs, angioplasties, or bypass surgeries

19. Appetite disturbances: Irregular or decreased appetite.

20. Sleep disturbances: Trouble falling asleep, early morning awakening, insomnia, frequent waking, sleeping too much, abnormal sleep pattern, etc.

1. Isolated occurrence or in one situation (e.g., the eve of a major operation)
2. Occasional
3. Frequent
4. Constant

21. GI disturbances: Diarrhea, constipation, vomiting, “upset stomach,” heartburn, ulcers, etc.

22. Lack of or decrease in exercise:

1. statements such as “not as much as I should” or “not as much as I’d like to.”
4. inability to exercise for whatever reason

23. Other somatic complaints: Anything that can alter mental or emotional well-being. Examples: cancer, other illnesses, migraine headaches, back pain, arthritis, sexual dysfunction, hearing loss, etc. Does not include CHD symptoms (#19).

1. 1-2
2. 2-3
3. 3-4
4. 4 or more

67. Good cardiovascular functioning: As evidenced by report of increased exercise tolerance, less angina, or being told by physician that cardiovascular status is good or improved.

68. CHD protective factors: Mention of the absence or decrease of CHD risk factors.

69. Good appetite: Normal, “hearty” appetite.
70. Good sleep: Restful, relaxing, normal amount.
   3: sleeping "well," "real well"

71. Good GI functioning: Rarely mentioned.

72. Exercise: Consider what would be "normal" for a person of the
    subject's age.
    
    1: occasionally; "a little"
    2: routinely; mild
    3: routinely; moderate
    4: routinely; very active

73. Other positive somatic signs: Rarely reported, but include
    positive changes not included in 69-73, such as good eyesight,
    good muscle strength, or improvement in the types of
    complaints mentioned in 24.

Social support:

24. Feels unloved, unappreciated: If a subject's wife, for example,
    wants him to be something else, rate for a feeling of being
    unappreciated. Similarly, if a subject talks about doing work that
    is unrecognized by his employer or unappreciated by a customer,
    rate here. Watch for a sense of self-pity as well, rated in 10.

25. Feels isolated, lonely: A feeling of isolation from or lack of
    family, friends, coworkers, or other sources of support.

26. Loss of spouse, family, job: Fear or threat of loss is also rateable,
    but not as much as actual loss. A recent retirement may be
    rateable if the transition has been difficult or if the person misses
    his job. A more remote retirement should not be rated unless
    the person still misses his job and does not seem to have
    something else to look forward to.

27. Other interpersonal stressors: For example, loss of friends
    (through death, disputes, or change in location), moving to a
    new community, leaving a church or community group.

74. Feels loved, appreciated:

75. Feels connected, supported: If a subject mentions family
    members or current marriage or significant other, rate at least 1.
Rate higher if the subject talks about the supportiveness of family members or a strong, happy marriage or relationship.

76. Security in family, job: A sense of stability and of confidence that a marriage or job will always be there.

77. Other interpersonal support: For example, friends, community groups, social clubs, religious groups, support groups.

Speech & facial expression: Whereas A through F require the rater's integration of observation, subject's self-report, and inference, G and H are based only on the rater's observation and interpretation. Although the "anchor point" is still 0, most of the behaviors are observable, particularly on the right side of the scale, and ratings may be consistently higher in these categories than in the previous ones. 2-3 should be considered normal for the right side of G-H, 2 being low normal and 3 being high normal.

It may be difficult to see how a subject can have both a negative characteristic and its opposing positive one on some of these items, especially 29/79, 30/80, and 31/81. But speech and facial expression, as well as demeanor and engagement, can vary throughout the interview, often in relation to the content of the speech. Thus someone who displays a full range of affect, including periods of sad facial expression, might receive a high rating on both poles of item 31 ("Little range of expression"/"Good range of expression"). The rater should take into account both the degree and the duration of behavior. For example, a short duration of mild flat affect might rate 1, but a short duration of profoundly flat affect might rate a 3.

When a person's speech or appearance is consonant with the content of his speech, it is said to be mood-congruent, and the rater should observe speech and facial characteristics carefully when the content of the subject's conversation has an affective component.

28. Depressed, sad speech

29. Monotonic (flat) speech: Depressed speech can have real feeling associated with it, so depressed speech is not necessarily flat or blunted.

30. Low volume

31. Sad, tired expression: Noticeable in eyes and/or mouth.
32. Little range of facial expression

33. Eyes downcast, head down: This is a subtle characteristic that requires close observation and differentiation from normal eye movements.

0: Good eye contact throughout interview
1: Occasional downward glances that seem mood-congruent
4: Little eye contact; subject's head is down and gaze is directed toward floor during most of interview

34. Mouth downturned

78. Animated, joyful speech

79. Full range of speech

80. Good volume

81. Good range of facial expression

82. Energetic, interested expression

83. Good eye contact

84. Smiling, pleasant: Engaged, appropriate smiling, not forced or nervous grimacing.

Demeanor & engagement: See general guidelines for "Speech & facial expression" above.

35. Tense, impatient, hostile: Some subjects may belie an unwillingness to participate in the interview or answer questions curtly or tersely. They may also interrupt the interviewer frequently in impatience.

36. Fidgety, restless: Nervous movements of the hands, crossing and uncrossing of the legs, shifting position in the chair, etc.

37. Poor concentration; forgetful

38. Apathetic, withdrawn: Subject gives laconic answers and does not attempt to engage the interviewer in conversation. Often accompanied by flat affect and diminished range of speech modulation.
85. Relaxed, pleasant, at ease: Subjects convey the impression that they enjoy talking with the interviewer.

86. Calm

87. Aware, alert

88. Engaged, energetic: At the extreme, the subject is sitting forward in the chair, answering questions enthusiastically, engaging the interviewer in the conversation. This state is typically accompanied by good eye contact and a full range of affect and speech.

Global ratings: These are "gestalt" ratings that are guided by the individual item ratings (1-38) but not determined by them in any strict mathematical way. In effect, this is the rater's chance to assign different weights to the items within a category to come up with a single score for that category. However, it bears repeating that the individual items have been grouped into their particular categories more by inductive reasoning than by deductive reasoning and are heuristic tools rather than empirically determined factors of depressive style.

The keywords at either end of the scales are only reminders of the entire constellation of behavioral indicators of the particular category and should not be construed as the sole features of the category.

Category G (Speech & facial expression) has been separated into its two components in 45/95 and 46/96, and category H (Demeanor & engagement) has been separated into its two components in 49/97 and 48/98. The items included in each global rating are as follows:

45/95 (speech): items 28-30/78-80
46/96 (facial expression): items 31-34/81-84
47/97 (demeanor): items 35-36/85-86
48/98 (engagement): items 37-38/87-88

Ratings of 2-3 should be considered normal for 95-98, as they were in the corresponding items of 78 through 88. If unsure whether to rate 2 or 3, rate 2.

Overall impression: For these last two ratings, the rater should again bear in mind that the two sides of the scale are distinct from one another. That is, the "depressive style" rating for #49 should be the rater's overall impression of the subject's depressive style, untempered by whatever positive, non-depressive behaviors the subject displays. Similarly, the "optimistic, vibrant" rating for
#99 should be the rater’s overall impression of the subject’s positive features only. Number 49 is thus the rater’s estimate of the extent of depressive style, however it is manifested.

For item #50/#100, the outcome of interest — deterioration vs. improvement or death vs. survival — must be specified. In a study with a short follow-up and a means of quantifying the outcome variable, deterioration vs. improvement may be appropriate. With a long follow-up period or a study that employs death from cardiac causes as an outcome variable, it will be easier to use death vs. survival.

In rating #50 and #100, the rater should use all available information and not just the depressive style and optimistic style scores. A subject who has low depressive style but a strong family history of CHD may thus receive a higher rating on #50 than a subject who has low depressive style and no family history of CHD.

The rating should reflect the rater’s judgment of the percent risk of the specified outcome, not the degree of the outcome.

0: No risk of the specified outcome
1: 0-25% risk
2: 25-50% risk
3: 50-75% risk
4: 75-100% risk