THE EFFECTS OF WEIGHT LOSS ON PLASMA CHOLESTEROL AND LIPOPROTEINS AMONG FEMALE PARTICIPANTS IN A RESIDENTIAL WELLNESS PROGRAM

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COMPLETED RESEARCH IN HEALTH, PHYSICAL EDUCATION, RECREATION, AND DANCE
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This retrospective study investigated the relationships among adiposity, cardiovascular fitness, age, initial cholesterol and lipoprotein levels, menopausal status, weight loss, and changes in cholesterol and lipoprotein levels in adult female participants (n = 56) who attended a 13-day residential program. Matched t tests revealed significant ($p = \leq .05$) mean decreases from pretest to posttest in total plasma cholesterol (TPC), low-density lipoprotein (LDL), and weight. Point-biserial correlations showed no statistically significant correlations between menopausal status and changes in TPC, HDL, and/or LDL levels, but the relationship between cardiovascular fitness and decreases in weight was significant ($p = \leq .05$). Pearson product moment correlations indicated a statistically significant ($p = \leq .05$) relationship between weight loss and changes in TPC. Pearson correlations found statistically significant ($p = \leq .05$) correlations between adiposity (measured as body fat percentage) and weight loss and between age and changes in TPC and LDL levels. Finally, Pearson correlations found significant ($p = \leq .05$) relationships between initial TPC levels and decreases in TPC; between initial HDL levels and decreases in

HDL; and between initial LDL levels and decreases in TPC and LDL. For the Pearson correlations, the greater the initial levels of these components, the greater their decrease from pretest to posttest.

TABLE OF CONTENTS

ACKNOWLEDGEMENTS iii

ABSTRACT iv

LIST OF TABLES ix

Chapter

I. INTRODUCTION 1

Statement of the Problem 2
Purpose of the Study 2
Hypotheses 2
Definition of Terms 3
Limitations of the Study 4
Delimitations of the Study. 4
Assumptions 5
Background and Significance 5

II. REVIEW OF THE LITERATURE. 8

Epiemiology of Coronary Heart Disease (CHD) in Women. 9
Total Plasma Cholesterol 10
High Density Lipoproteins (HDL). 12
Low Density Lipoproteins (LDL) 13
Adiposity 15
Adiposity and Coronary Heart Disease (CHD) 15
Obesity and Cardiovascular Risk in Women 17
Adiposity and Total Plasma Cholesterol (TPC) 19
Adiposity and Lipoproteins 20
Weight Loss 21
Weight Loss and CHD 21
Weight Loss and TPC 22
Weight Loss and Lipoproteins 25
Calorie Restriction for Weight Loss 28

Chapter Page

Physical Activity 30
Physical Activity and CHD 30
Physical Activity and CHD in Women 32
Physical Activity and Cholesterol 33
Exercise and Lipoproteins 35
Physical Activity and HDL in Women 38
Relationship of Age to Cholesterol and Lipoproteins . . . 40
Menopause, Cholesterol, and Lipoproteins 42
Summary 44

III. METHODOLOGY. 47

Population and Sample 47
Instrumentation 48
Procedures 49
Data Collection 50
Treatment of the Data 50

IV. ANALYSES OF THE DATA 52

Description of the Subjects 52
Statistical Analysis of the Data 57

V. SUMMARY, FINDINGS, DISCUSSION, CONCLUSIONS, AND RECOMMENDATIONS 64

Summary of the Study 64
Findings of the Study. 65
Discussion of the Findings 66
Conclusions. 71
Recommendations 72

REFERENCES 75

APPENDICES. 93

Appendix A: Medical History Questionnaire 93

Appendix B: Blood Analysis Form 115

Appendix C: Treadmill Stress Test Record Sheet 117

Appendix D: Body Composition Analysis Form 119

Chapter Page

Appendix E: In-Residence Program 13-Day Schedule. 121

Appendix F: Informed Consent Form. 126

LIST OF TABLES

Table		Page
1.	Age, Height, and Adiposity of Subjects	53
2.	Distribution of Subjects' Menopausal Status	54
3.	Subjects' Weight and TPC, HDL, and LDL Levels	55
4.	Decreases in Subjects' Weight, TPC, HDL, and LDL Levels	56
5.	Subjects' Cardiovascular Fitness	57
6.	Results of Matched t Tests for Changes in TPC, HDL, LDL, and Weight Means	58
7.	Point-Biserial Correlations Among Menopausal Status, Cardiovascular Fitness and Changes in Weight, TPC, HDL, and LDL Levels	60
8.	Pearson Correlations Among Weight Loss and Changes in TPC, HDL, and LDL Levels	60
9.	Pearson Correlations Among Adiposity and Age and Changes in Weight, TPC, HDL, and LDL Levels	61
10.	Pearson Correlations Among Initial Levels of TPC, HDL, and LDL, and Changes in Weight, TPC, HDL, and LDL Levels	63

CHAPTER I

INTRODUCTION

In 1986, Weinstein, Coxson, and Williams predicted that, despite the recent decline in coronary heart disease (CHD) mortality rates, CHD would continue as the leading cause of death in the United States for the next two decades. It is well established that an elevated plasma cholesterol level is a major risk factor for CHD. Results from several clinical trials indicate that therapeutic reduction of high risk plasma cholesterol levels is followed by a reduction in CHD risk, and that reducing plasma cholesterol levels can reduce both morbidity and mortality from CHD. Cholesterol is transported in the plasma by low-density lipoproteins (LDL) and high-density lipoproteins (HDL). High levels of LDL are associated with increased risk of CHD while high levels of HDL are associated with decreased risk of CHD (Grundy, 1986).

Studies examining the effects of weight loss on changes in plasma lipid levels in women have had inconsistent results. Some studies have shown an increase in total plasma cholesterol (TPC) with weight loss (Carmena, Ascaso, Tebar, & Soriano, 1984; Larosa, Fry, Muesing, & Rosing, 1980) and LDL with weight loss (Friedman et al., 1982; Larosa et al., 1980), while others have shown a decrease (Hagan, Upton, Wong, & Whittam, 1986; Zimmerman et al., 1984). In relation to weight loss, HDL levels reportedly either have increased (Carmena et al., 1984; Sorbis, Petersson, & Nilsson-Ehle, 1981), remained the same (Hagan et al., 1986; Zimmerman et al., 1984), or decreased (Larosa et al,

1980; Thompson, Jeffery, Wing, & Wood, 1979). Questions remain regarding the influence of possible confounding variables, such as age, initial lipid and weight values, menopausal status, amount of weight lost, and cardiovascular fitness levels, on the relationships between weight loss and plasma cholesterol and lipoproteins.

Statement of the Problem

The problem examined in this study was to describe the relationships and differences among variables related to plasma cholesterol as reported in preexisting data collected for the 13-day In-Residence Program (IRP) at the Aerobics Center in Dallas, Texas.

Purpose of the Study

The purpose of this descriptive, retrospective study was to determine the relationships among adiposity, cardiovascular fitness levels, age, initial cholesterol and lipoprotein levels, menopausal status, weight loss, and changes in cholesterol and lipoprotein levels in adult female participants in a 13-day residential wellness program. In addition, the study was to determine whether there were differences in the TPC, HDL, and LDL levels as a result of participation in the IRP.

Hypotheses

The following null hypotheses were tested at the .05 level of significance:

1. There are no significant differences between pretest and posttest measures of TPC, HDL, and LDL.

2. There is no significant relationship between menopausal status and changes in levels of TPC, HDL, and/or LDL.

3. There is no significant relationship between short-term weight loss and changes in levels of TPC, HDL, and/or LDL.

4. There is no significant relationship between adiposity and changes in TPC, HDL, LDL, and/or weight.

5. There is no significant relationship between levels of cardiovascular fitness and changes in TPC, HDL, LDL, and/or weight.

6. There is no significant relationship between age and changes in TPC, HDL, LDL, and/or weight.

7. There is no significant relationship between initial lipid levels and changes in TPC, HDL, LDL, and/or weight.

Definition of Terms

For the purpose of this study, the following terms were defined:

1. Cardiovascular Fitness. Fitness categories as measured by the Modified Balke Treadmill Protocol based upon treadmill time and age group: very poor, poor, fair, good, excellent, and superior (Balke & Ware, 1959).

2. Short-Term Weight Loss. Number of pounds lost during the 11 days between the pretest and posttest measurements.

3. Adiposity. The percent body fat as determined by hydrostatic weighing and the mean value calculated from the formulas of Brozek et al. (1957).

4. Menopausal status. Subjects were premenopausal unless it had been 12 months or more since their last menstruation at the time of data collection, in which case they were considered menopausal, or no longer menstruating.

5. Nonsmoker. A person who had not smoked for at least 12 months prior to the collection of data and who did not smoke during the 13 days of the program.

6. Cooper Clinic. A medical clinic located in Dallas, Texas, where, since 1970, more than 35,000 patients have been given a comprehensive medical evaluation, individual counseling, and specific recommendations for attaining and maintaining good health.

7. In-Residence Program. A program that emphasizes weight loss, physical activity; stress management; diet, nutrition, and exercise education; behavior modification; preventive and therapeutic medicine; smoking cessation; and total well-being in a supportive live-in environment. All programs include both individual supervision and counseling, and supervised group exercise classes (Van Itallie & Hadley, 1988).

Limitations of the Study

The limitations identified for this study included:

1. The accuracy of the data collection.
2. The reliability of abstracting data from the subjects' medical charts.

Delimitations of the Study

The delimitations of this study included:

1. Predominantly white females aged 30 to 69 years, from a higher than average socioeconomic stratum, who attended the IRP from 1986 through 1990.

2. Nonsmokers who did not use medications which were known to affect cholesterol.

3. Subjects who followed similar diet and exercise regimes during their 13-day stay at the IRP.

Assumptions

The following conditions were assumed:

1. The participants had adhered to the diet and exercise regimens recommended during the IRP.

2. The medical history forms had been completed accurately by the participants during the medical evaluation.

Background and Significance

Although several studies have examined the effect of weight loss on TPC, HDL, and LDL in women, the results have been inconsistent. The inconsistent results may be due in part to the lack of control of several variables. One of the variables that is not controlled in the reporting of the results is the duration of the study and the subsequent follow-up period. Studies report findings of interventions of one week (Zimmerman et al., 1984) to 19 months (Carmena et al., 1984), with no follow-up periods after weight stabilization (Brownell & Stunkard, 1981; Hagan et al., 1986; Larosa et al., 1980) or follow-up periods after one month (Thompson et al., 1979) to 6 months (Stevenson, Darga,

Spafford, Ahmad, & Lucas, 1988). Only one published study was located that investigated the effects of one and two weeks of weight loss on TPC, HDL, and LDL in women (Zimmerman et al., 1984), and that study did not control for physical exercise or menopausal status. In addition, it examined only seven women, all of whom were obese.

Adiposity is another variable that has not been controlled in the reporting of the results of the effect of weight loss on TPC, HDL, and LDL levels. The studies that have been conducted on women show an initial group average weight from 155 pounds (Follick, Abrams, Smith, Henderson, & Herbert, 1984) to 440 pounds (Sorbis et al., 1981). The weight of 155 pounds was described as moderately obese while the weight of 440 pounds was described as grossly obese by the authors of these studies. No studies have compared the effect of weight loss on cholesterol and lipoproteins in women who are at acceptable weights or only slightly overweight with the effect of weight loss in women who are obese.

The physical activity of the subjects also may have moderated the reported effects of weight loss on cholesterol and lipoproteins in women. In some studies (Larosa et al., 1980; Stevenson et al., 1988; Zimmerman et al., 1984), exercise was not mentioned. In other studies (Brownell & Stunkard, 1981; Carmena et al., 1984; Thompson et al., 1979), exercise levels were kept constant during each study, but those levels were variable among the subjects and were not examined for their possible relationship to the outcome. In one study (Hagan et al., 1986), the subjects were sedentary at the beginning of the study, and were broken into groups that either remained sedentary or began a

program of regular aerobic exercise. Cardiovascular fitness levels were not examined as a possible confounding variable in the effect of short-term weight loss on the cholesterol and lipoproteins of women in any of the existing studies.

Two other possible confounding variables that have not been focused upon in the current literature are the relationships among age, menopausal status, and changes in weight, TPC, HDL, and LDL. Studies have included women from a wide range of ages. For example, Carmena et al. (1984) included females from 14 to 66 years of age. No study has examined the relationships of age with changes in weight, TPC, HDL, and LDL. Only one study (Zimmerman et al., 1984) has discussed the menopausal status of the subjects. This study mentioned that all of the subjects were premenopausal. None of the studies examined have compared the results of premenopausal and menopausal women.

It is important to study the relationships among short-term weight loss, TPC, HDL, and LDL to increase scientific knowledge and understanding concerning the reduction of cholesterol as a possible mediator of increased risk of mortality. This study also would provide researchers with specific information concerning the relationships among weight loss; adiposity; cardiovascular fitness levels; menopausal status; age; and changes in TPC, HDL, and LDL levels.

CHAPTER II

REVIEW OF THE LITERATURE

The review of literature in this study is presented in eight sections. The first section presents a general review of the epidemiology of heart disease in women. This review is followed by a section on TPC, HDL, and LDL, which is fundamental to understanding the basic underlying concept of CHD risk and mortality. The next two sections discuss the relationships of adiposity and weight loss with CHD, TPC, and lipoproteins. The fifth section reviews calorie restriction for weight loss. The sixth section examines the role that physical activity plays in CHD, serum cholesterol, and lipoprotein levels. The final two sections review the relationships of age and menopause with CHD, TPC, HDL, and LDL.

The term, risk factor, is used generally by epidemiologists to describe any attribute of an individual that is associated empirically with a future adverse health outcome (Sherwin, 1982). One important use of risk factors is to identify individuals at risk of early death from disease. This is the application utilized by insurance companies which are concerned not that obesity is a cause of early death, but that obese individuals die at an earlier age than nonobese individuals. Another use of risk factors is to suggest causal factors in certain diseases and to suggest approaches to the prevention of these diseases. Observational studies by which risk factors are identified do not demonstrate causal relationships, and generally are followed by direct and controlled

experimentation to demonstrate that manipulation of a factor does indeed alter the risk of disease (Sherwin, 1982).

Epidemiology of Coronary Heart Disease (CHD) in Women

CHD is the leading cause of death in women as it is in men. Approximately 500,000 women die of cardiovascular disease each year in the United States. Half of these deaths are due to CHD, surpassing the total number of deaths from all cancers combined (Miller, 1990). Despite the impact of CHD on the health and well-being of women, very few population-based studies of CHD have included women and none has focused exclusively on women. Neither have many clinical trials had sufficient numbers of female participants to evaluate treatments for CHD in women effectively. One of the reasons for the lack of information about CHD in females is that CHD only rarely occurs in premenopausal women. It commonly is thought that women lose this advantage after menopause because thereafter the rates of CHD in women and men become similar. Since more women live to be older than do men, even though women develop CHD an average of 7 to 8 years later than do men, more women than men actually are dying from CHD (Bush, 1990; Castelli, 1988).

Although CHD is relatively rare in premenopausal women, it still is responsible for more morbidity and mortality at this developmental stage than are other causes such as maternal death. The death rate from CHD was 21 times that from maternal causes in 1981 (Vital Statistics, 1981). Another

perspective of CHD risk in females is the evidence documenting atherosclerosis as a chronic, progressive disease that begins in childhood (Newman, Freedman, & Voors, 1986). Therefore, although clinical manifestations of CHD generally may not become apparent in women until after menopause, the process of atherosclerosis is begun in early life. Thus, early intervention for risk factors in young women of reproductive age is important to the prevention of later CHD.

Total Plasma Cholesterol (TPC)

Several risk factors have been identified as strongly associated with CHD. One of these is TPC. Cholesterol is essential to humans as it is the structural component of cellular membranes and a precursor of hormones and other important molecules. It has been estimated that only about 7% of all cholesterol in the body circulates in the plasma, while over 93% is found in cell membranes (Gordon & Cooper, 1988). The human body acquires cholesterol from the consumption of animal products and from synthesis in the liver and other tissues (Gordon & Rifkind, 1989).

The positive association between TPC and cardiovascular morbidity and mortality has been one of the most consistent and well-established of all areas of epidemiology. The relationship has been found in different demographic and ethnic groups; geographic areas; study designs (cross-sectional, case-control, family, ecologic, and cohort); and, in cohort studies, for both short and long intervals between cholesterol measurement and disease occurrence (Wallace & Anderson, 1987).

Various published studies have assessed the relationship between TPC and CHD in women (Aronow, 1990; Barrett-Connor, Khaw, & Wingard, 1987; Bengtsson, 1973; Brunner, Weisbort, & Meshulam, 1987; Bush, Criqui, & Cowan, 1987; Higgins, Keller, & Ostrander, 1987; Kannel, 1987; Keil, Gazes, & Loadbolt, 1987; Lipid Research Clinic, 1984; Stampfer, Colditz, & Willett, 1987; Tyroler, Heyden, & Bartel, 1971). These studies agree that women with concentrations of total cholesterol over 260 mg/dl have a two- to three-fold increased risk of CHD compared to the risk in women with concentrations less than 200 mg/dl. It has been demonstrated that, with every 1% increase in total cholesterol, there is a 2% increase in the incidence of CHD in both younger and older women (Lipid Research Clinic, 1984). These data support the recent trend toward intervention at cholesterol levels greater than 200 mg/dl, rather than at greater than 240 mg/dl as previously recommended (Kafrissen, 1990). Grundy (1986) identified the ideal cholesterol range for the whole population as within the range of 130 to 190 mg/dl. If most Americans could achieve this range, the rate of CHD would be reduced by 30% to 50%.

Cholesterol is transported in the body by three major carriers: very-low-density lipoprotein (VLDL), which also carries triglycerides; LDL, which is the primary supplier of cholesterol; and HDL, which is thought to remove excess cholesterol from cells for excretion by the liver. The significance of these lipoproteins in terms of the risk of CHD varies with the molecule and the sex of the patient. However, the most predictive factor for CHD risk seems to be HDL (Kafrissen, 1990).

High Density Lipoproteins (HDL)

HDL is secreted primarily by the liver and intestine. It is believed that HDL picks up cholesterol from the peripheral tissues and returns it to the liver to be utilized by the body. An inverse relationship between CHD risk and plasma HDL has been confirmed (Gordon & Cooper, 1988). Plasma levels of HDL first were correlated with the risk of coronary disease in the early 1950's and were confirmed in the 1960s (McCunney, 1987). Since that time, there have been a large number of cross-sectional, case-control, and cohort studies which have confirmed the relationship (Wallace & Anderson, 1987). Since 1977, HDL has been considered the most powerful lipid parameter for predicting the development of CHD in people of all ages, and depressed levels of HDL have been shown to be an independent risk factor (Castelli, Doyle, & Gordon, 1977; Gordon et al., 1977; Mjos, Thelle, & Forde, 1977). Only the Framingham Heart Study (cited in Kannel, 1987), the Lipid Research Clinics' Follow-Up Study (cited in Bush et al., 1987; cited in Jacobs et al., 1990), and the Donolo-Tel Aviv Study (cited in Brunner et al., 1987) have evaluated the relationship of HDL on CHD risk specifically in women. In the two American studies, the associations between HDL and CHD risk were very similar: In both reports, a 10 mg/dl difference in HDL levels was associated with a 40% to 50% difference in the risk of CHD. Females with HDL levels of 60 mg/dl had CHD rates of 40% to 50% lower than females with HDL levels of 50 mg/dl.

In the Lipid Research Clinics' Follow-Up Study (cited in Jacobs et al., 1990), the HDL cholesterol level was found to be related more closely to cardiovascular disease than was LDL cholesterol. The conclusion of this study

was that HDL cholesterol is related inversely to both coronary heart disease and other cardiovascular disease mortality. Thus, this finding confirms both the importance and the independence of HDL cholesterol in the epidemiology of CHD in women.

In the study from Tel Aviv (Brunner et al., 1987), the adverse effects of high concentrations of total cholesterol were neutralized essentially by high levels of HDL. Women with high levels of both HDL and TPC had age-adjusted rates of CHD that were no different than the rates in females with lower cholesterol levels. It also was found that females with low concentrations of HDL cholesterol were at an increased risk of CHD, no matter what their levels of total cholesterol.

Because women have a mean HDL of 55 mg/dl as compared to 45 mg/dl for men, proportionately more of their TPC is composed of a protective or nonatherogenic cholesterol. Thus, it takes more LDL and TPC to reflect a more atherogenic profile (Miller, 1990). However, neither our current understanding of lipid metabolism nor these epidemiologic studies can assure that low levels of HDL cholesterol are a causative rather than a coincidental factor in CHD, or that intervention would be beneficial (Gordon & Rifkind, 1989).

Low Density Lipoproteins (LDL)

Most cholesterol in the blood is contained within the LDL subfraction. LDL usually constitutes approximately two thirds of TPC; therefore, there is a high correlation ($r = 0.8$) between TPC and LDL (Rossouw & Rifkind, 1990). Since most cholesterol in plasma is transported in LDL, this lipoprotein accounts for

most of the linkage between the plasma cholesterol level and CHD (Grundy, 1986).

In normal people, 70-75% of LDL is cleared from the plasma by specific LDL receptors located on cell surfaces. The liver is the major site for receptor-mediated LDL clearance, but peripheral tissues also have the capacity for LDL uptake. The remaining plasma LDL is removed by nonreceptor mechanisms in a variety of tissues. LDL particles are small and can penetrate the arterial wall readily (Gordon & Cooper, 1988).

Many lines of evidence lead to the conclusion that LDL is a causal factor in the onset of CHD. These range from animal studies, pathology studies, inborn errors of metabolism, clinical observations, and the existence of plausible biologic mechanisms, to the vast body of epidemiologic evidence. Likewise, the clinical trials of cholesterol lowering, together with regression studies in animals and angiographic studies in humans, provide evidence that the progress of atherosclerosis can be halted (Rossouw & Rifkind, 1990). The Lipid Research Clinic's Coronary Primary Prevention Trial (1984), using dietary modifications and cholestyramine to treat hypercholesterolemia, demonstrated that it is possible to protect against CHD by decreasing elevated plasma LDL levels.

Few studies concerning lowering LDL for the prevention of CHD have included women, and in no case were studies located in which females were analyzed separately (Carlson & Rosenhammer, 1988; Group of Physicians, 1971; Research Committee, 1971). Because it has not been addressed specifically, the benefit to women of intervention to lower LDL levels has not been demonstrated yet (Crouse, 1989).

Adiposity

Adipose tissue is a normal component of the human body: It serves the important function of storing energy as fat that can be used in response to metabolic demands. Obesity is an excess of body fat frequently resulting in a significant impairment of health. The excess fat is the result of increased fat cell size and, in people with extreme obesity, increased fat cell numbers (National Institutes of Health [NIH], 1985).

The proportion of overweight adults in the United States has been increasing steadily during the past several decades, especially among women (Harlan, Landis, Flegal, Davis, & Miller, 1988). In 1985, approximately one in five of the 34 million adult Americans were obese (NIH, 1985). Because the amount of body fat is a continuous variable, NIH panelists agreed that obesity, or body weight of 20% or more above desirable body weight, constitutes an established health hazard. Significant health risks at lower levels of obesity can present hazards especially in the presence of diabetes, hypertension, heart disease, or their associated risk factors.

Adiposity and Coronary Heart Disease (CHD)

The effect of adiposity on longevity has been of interest for several years. The interest came initially from life insurance companies in the process of constructing actuarial tables (Simopoulos & Van Itallie, 1984). Together, the Build and Blood Pressure Study (1959) and the Build Study (1979) have provided information on weight and mortality for over 8 million people in the United States, and have been the basis for the development of the 1959

Metropolitan Life Desirable Weight Tables (Metropolitan Life, 1959) and the 1983 Metropolitan Height and Weight Tables (Metropolitan Life, 1983). Mortality risk was found to be lowest for the groups with weights 5-15% below and 5-15% above the average weight for this insured population, with mortality increasing for those in the lowest and highest weight categories. The resulting relationship was thus U- or J- shaped (Manson, Stampfer, Hennekens, & Willett, 1987). In the 1979 Build Study, average weights were higher in the study population than in the 1959 study population; therefore, the lowest mortality rates in 1979 occurred among subjects weighing more than those in the 1959 study. As a result, the 1983 Height and Weight Tables list desirable weights that are from 0-13% higher than the 1959 tables (Metropolitan Life, 1983). This increase in what is considered desirable weight has stirred controversy and stimulated criticism of the methodology used to relate body weight to longevity (Harrison, 1985; Knapp, 1983; Simopoulos & Van Itallie, 1984). In both Build Studies (1959; 1979), the relative mortality risk from all causes for women was considerably lower than for men. In the 1979 study, women who were 45% overweight had a mortality risk of 109%.

The influence of obesity on the risk of CHD remains controversial despite a well-established association between adiposity and unfavorable coronary risk-factor status (Manson et al., 1990). Some investigators have questioned the relationship between relative weight and coronary heart disease (Andres, 1982; Bradley, 1982; Epstein, 1967; Keys, 1979; Wilcosky, Hyde, Anderson, Bangdiwala, & Duncan, 1990). On the other hand, many studies have shown that the incidence of CHD is greater in heavier people (Chapman, Coulson,

Clark, & Borun, 1971; Dyer, Stamler, & Berkson, 1975; Heyman et al., 1971; Paffenbarger, Laughlin, & Gima, 1970; Paul et al., 1963; Petitti, Wingerd, Pellegrin, & Ramcharan, 1979; Rabkin, Mathewon, & Hsu, 1977; Robertson et al., 1977). Few suggest, however, that obesity makes an additional contribution to risk once the levels of coexisting risk factors are taken into account (Chapman et al., 1971; Rabkin et al., 1977; Robertson et al., 1977). Obesity is associated with elevated blood pressure, blood lipids, and blood glucose; and changes in body weight are coincident with changes in these risk factors for disease (Hubert, Feinleib, McNamara, & Castelli, 1983).

Obesity and Cardiovascular Risk in Women

There are few studies concerning the influence of obesity on cardiovascular risk in women since most prospective studies of obesity and coronary disease have included only men. Noppa, Bengtsson, Wedel, and Wilhelmsen (1980) followed 1,463 middle-aged women in Sweden for 10 years. They found only a weak, nonsignificant association between obesity and CHD; but there were only 15 cases of myocardial infarction and 55 cases of angina, and the analyses were not controlled for cigarette smoking.

In the Framingham Study (cited in Hubert et al., 1983), 2,818 females, 28 to 62 years of age, were followed for a 26-year period, and a strong positive association was found between relative weight and the incidence of CHD. For each pound above ideal weight gained over the first 26 years of the Framingham Study, the CHD death rate increased by 2%. The logistic coefficients were fairly strong and consistent throughout the study but statistical

significance was not achieved until nearly 14 years of follow-up study had been conducted. Therefore, early analyses, based on shorter periods of observation, suggested that there was not independent relationship between adiposity and coronary risk in Framingham women.

In a large-scale study by the American Cancer Society (cited in Lew, 1985), self-reported weight was associated positively with mortality from CHD, approximately doubling the risk in females more than 40% above the average weight. Control for cigarette smoking increased these mortality ratios further. Nonfatal coronary events were not included in the analyses.

Tuomilehto, Salonen, and Marti (1987) studied 4,120 women aged 30 to 59 years, and found a statistically nonsignificant association between adiposity and the rate of myocardial infarctions. Only 52 females in the total group, however, had experienced these events in the 7 years of follow-up study.

Examination of the Nurses' Health Study cohort (cited in Manson et al., 1990) found a strong positive association between obesity and the risk of CHD in women. Adjustment for cigarette smoking, which correlated inversely with relative weight and directly with coronary risk, increased the magnitude of the association. After adjustment for age and smoking, the risk of both nonfatal myocardial infarction and fatal coronary disease among women in the heaviest weight category (30% or more above ideal weight) was more than three times higher than that in the leanest group (less than 95% of desirable weight). A significantly increased rate of CHD also was found for mildly to moderately overweight women, among whom the rate was increased by 80%. In the overall

study population, 40% of the coronary events were attributable to adiposity. In obese women, as much as 70% of the CHD was attributable to adiposity.

Adiposity and Total Plasma Cholesterol (TPC)

Several studies have found a relationship between adiposity and serum cholesterol levels (Jacobsen & Thelle, 1987; Kannel, Gordon, & Castelli, 1979; Leclerc, Bouchard, Talbot, Gauvin, & Allard, 1983; Matter, Weltman, & Stamford, 1980; Montoye, Epstein, & Kjelsberg, 1966). Obesity is associated with increased rates of cholesterol synthesis that appear to occur in the liver or intestine (Nestel, Schreibman, & Ahrens, 1973; Schreibmen & Dell, 1975).

Waxler and Craig (1964) found that obese women had slightly elevated serum cholesterol levels until 49 years of age, as compared with normal-weight women. After age 49, the levels of both obese and nonobese women were similar.

According to Van Itallie (1985), the relative risk of hypercholesterolemia (concentration of 250 mg/dl or higher) for overweight Americans aged 20 to 75 years was 1.5 that of those who were not overweight. Among overweight Americans aged 20 to 45 years, the relative risk of hypercholesterolemia was 2.1 times that of nonoverweight Americans in the same age group. Among Americans in the 45 to 75 age range, overweight did not affect the risk for hypercholesterolemia.

Adiposity and Lipoproteins

Plasma lipid levels are often abnormal in obese people. Obesity is inversely related to HDL levels and directly related to LDL, VLDL, and triglyeride levels (Dietz, 1989; Garrison et al., 1978; Garrison et al., 1980; Thelle, Shaper, & Whitehead, 1983). Foster et al. (1987) studied 286 women entering a weight control program. The results concurred with those of previous studies concerning the correlation between body weight and HDL levels, but there was no significant correlation with LDL levels. In this study, LDL levels were predicted by age only.

The Tromso Heart Study (cited in Jacobsen & Thelle, 1987) examined 7,257 women in a cross-sectional study in Tromso, Norway. Adiposity was correlated negatively with HDL concentration in this study. Another study in Finnmark County, Norway, found a strong and almost linear negative association between relative body weight and HDL (Forde, Thelle, Arnesen, & Mjos, 1986). This study included 6,768 healthy women aged 20 to 53, and used an analysis of covariance.

Another study (Owens, Matthews, Wing, & Kuller, 1990) examined 541 healthy premenopausal women and used multivariate analyses to find that HDL levels were highly negatively related to adiposity. The concentration of HDL was lower in overweight women. The authors commented that the observed relationship between adiposity and lipoprotein patterns may be mediated partly through triglyceride concentrations. As triglyceride levels increase as a result of weight gain, the HDL concentration decreases.

A study of 86 healthy premenopausal obese Arab women (Emara, Saadah, Hassan, Moussa, & Hourani, 1989) found a significant correlation between adiposity and HDL levels. Compared with age-matched nonobese controls, obese women had significantly lower HDL levels.

In another study (Connor, Connor, Sexton, Calvin, & Bacon, 1982) that examined 233 families (n = 742), TPC levels correlated with age, body mass index, triceps skinfold measurements, and body weight in women. For both men and women, HDL levels correlated inversely with body weight, other measures of adiposity, and plasma triglyceride levels.

Weight Loss

Weight loss is a commonly prescribed therapy for CHD and for hypercholesterolemia. Several studies have examined the effects of weight loss on the risk initially posed by excessive weight.

Weight Loss and CHD

Weight reduction lessens most risk factors for CHD. Ashely and Kannel (1974) used the multiple logistic model to estimate the risk of CHD in relationship to weight loss. This risk was compared most concisely by the relative odds ratio. With ratios corresponding to 10% reductions in relative weight in females, approximately 20% reduction in coronary incidence could be anticipated. For each 10% increase in relative weight, approximately 30% increase in incidence could be anticipated. The effect was slightly less

pronounced for women and for older people than for men and for younger people.

In the Framingham Heart Study population (cited in Hubert et al., 1983), a change in weight after the young adult years was an independent predictor of CHD. At any level of initial weight at 25 years of age, weight increase was positively and significantly associated with CHD risk in women. These results illustrated the detrimental effects of weight gain and the benefits of weight reduction in obesity.

The National Institutes of Health Consensus Development Panel (1985) stated that weight reduction may be lifesaving for patients with extreme obesity, which is defined as weight that is twice the desirable weight. Weight reduction also is highly desirable for people with lesser degrees of obesity. In view of the excess mortality and morbidity associated with obesity, weight reduction is recommended to people with excess body weight of 20% or more above desirable weight. Additionally, weight reduction is likely to be helpful specifically in the reduction of CHD, although the benefits may not be as clear as in the reduction of all-cause mortality.

Weight Loss and TPC

The effect of weight loss on TPC has been investigated extensively. The results have not been entirely consistent, but the majority of studies demonstrate a decrease in TPC with weight loss (Brownell & Stunkard, 1981; Contaldo et al., 1980; Davis, Anderson, Ginsburg, & Goldenberg, 1985; Dudleston & Bennion, 1970; Follick et al., 1984; Friedman et al., 1982;

Galbraith, Connor, & Stone, 1966; Gonen, Halverson, & Schonfeld, 1983; Mann, 1974; Olefsky, Reaven, & Farquhar, 1974; Ribeiro, Hartley, Sherwood, & Herd, 1984; Rucker, Goldenberg, Varco, & Buchwald, 1981; Sopko et al., 1985; Sorbis et al., 1981; Wechsler, Hutt, Wenzel, Clor, & Ditschuneit, 1981). TPC also decreases significantly after weight has stabilized following weight loss (Davis et al., 1985; Follick et al., 1984; Gonen et al., 1983; Stevenson et al., 1988; Wechsler et al., 1981). A meta-analytic review of several studies (Tran, Weltman, Glass, & Mood, 1983) showed that decreases in body weight were correlated significantly with decreases in cholesterol. However, the studies included in the review were not examined in relation to weight gain, weight loss, and weight stable groups. These differences may explain the different results that some studies have found.

In the study by Larosa et al. (1980), TPC levels increased somewhat for both sexes after weight loss, although women experienced more striking increases than men. This increase also was seen in studies by Rickman, Mitchell, Dingman, and Dalin (1974) and Carmena et al. (1984). Other studies have reported that TPC levels have remained relatively unchanged after weight loss from baseline values (Eckel & Yost, 1989; Hagan et al., 1986; Lewis et al., 1976; Rabkin, Boyko, & Streja, 1981; Streja, Boyko, & Rabkin, 1980; Thompson et al., 1979; Wolf & Grundy, 1983; Zimmerman et al., 1984). One reason for this finding may be that some studies included a follow-up period after initial weight loss. These studies found that TPC decreased during weight loss but returned to baseline levels after weight stabilization.

The research by Wolf and Grundy (1983) followed women through three phases. Phase I was a preweight-loss period in which cholesterol was monitored for baseline values. Phase II was the weight-loss phase which lasted varying lengths of time until the patient attained 110% of ideal weight. Phase III was a maintenance phase lasting 4 to 5 weeks. At the beginning of Phase II, TPC commonly rose. Thereafter, TPC usually decreased until it reached a low point near the middle of Period II. In most patients, levels then began to rise near the end of caloric restriction; although, for the group as a whole, the levels were significantly lower in the last month of caloric restriction than at the beginning of the study. With a return to weight maintenance at the lower weight, the TPC levels rose still higher; and for the group of patients, the values for Period III were not significantly different from Period I (219 ± 9 vs. 227 ± 7 mg/dl, respectively).

This same pattern was seen in several other studies that included a follow-up period after weight loss. Zimmerman et al. (1984) examined women who had preweight-loss mean TPC values of 173.2 ± 5 mg/dl. These values went down to 157.3 ± 8.8 during weight loss, but returned to 169.0 ± 11.2 during the follow-up. Thompson et al. (1979) studied females who had a preweight-loss mean TPC of 209.9 ± 36.3 mg/dl. This value was reduced to 200.0 ± 22.8 mg/dl during weight loss, only to rise again to 208.3 mg/dl during the follow-up period. Hagan et al. (1986) had similar results when the females in this study had a preweight-loss mean TPC of 198 ± 28 mg/dl reduced to 181 ± 34 mg/dl during weight loss, only to rise again to 189 ± 40 mg/dl during follow-up. This

rise in TPC levels was not accompanied by a weight gain during the follow-up period.

In order to lower risk of CHD, weight loss needs to be only moderate. It is not mandatory to reach ideal weight in order to receive the benefits of weight reduction. Weight loss of 10 to 20 kilograms has been found to normalize blood lipids in overweight and obese subjects even though they remained at weights well above their ideal weight. Stamler (1980) found that with an average of 12 kilogram weight loss, serum cholesterol levels fell 26 mg/dl, a 10% decrease. Relative weight decreased only 6% with body weight still 22% above ideal, a level considered overweight. Olefsky et al. (1974) found similar results in women. Following a 11 kilogram weight loss, these subjects had a 21% decrease in serum cholesterol. Improvements in these cholesterol levels were similar among the subjects regardless of their initial or final weight.

Weight Loss and Lipoproteins

In most studies of females, the change in LDL levels during weight loss and stabilization paralleled that of TPC. Weight loss in the absence of exercise, produced by caloric restrictions from diets of 1,000 to 1,600 kilocalories per day, has been found either to decrease LDL levels (Brownell & Stunkard, 1981; Stevenson et al., 1988; Wechsler et al., 1981; Zimmerman et al., 1984), or to produce no changes (Eckel & Yost, 1989; Wolf & Grundy, 1983).

Larosa et al. (1980) studied 10 women who lost an average of 6.81 kg during a 12-week weight loss program. He found that LDL levels rose from an

average of 119 ± 10 mg/dl to 158 ± 19 mg/dl. This increase in LDL paralleled the increase in total cholesterol during this study.

Friedman et al. (1982) studied 15 morbidly obese women before and after achieving a stable and reduced weight by diet and/or gastric stapling procedure. During active weight loss, TPC and LDL levels did not change. After weight stabilization, however, mean TPC levels remained unchanged but mean LDL levels declined from 145 ± 23 to 135 ± 30 mg/dl.

Eckel and Yost (1989) studied 14 obese (27% above ideal weight) women who had a 11.7% weight reduction. Immediately after weight loss, LDL and TPC levels fell. The longer the dieting period, the less the decrement in cholesterol and lipoproteins. During the maintenance phase of the study, there was an increase in both TPC and LDL levels back to baseline values. This return of LDL levels to baseline amounts has been reported in several other studies (Follick et al., 1984; Weisweiler, 1987; Zimmerman et al., 1984).

Studies of the effects of weight reduction on HDL levels have yielded controversial results. This is due partly to the lack of controlled dietary regimens and the heterogeneity of the subjects studied. Some of the investigations were conducted on subjects with morbid obesity, which may constitute a unique subset of patients incomparable to the more common simple obesity (Zimmerman et al., 1984). The majority of studies demonstrate an increase in HDL levels in females as a result of weight loss (Carmena et al., 1984; Contaldo et al., 1980; Eckel & Yost, 1989; Follick et al., 1984; Friedman et al., 1982; Gonen et al., 1983; Lewis et al., 1976; Schwartz & Brunzell, 1981; Sorbis et al., 1981; Stevenson et al., 1988; Streja et al., 1980; Wolf & Grundy, 1983;

Zimmerman et al., 1984). However, few studies show this increase before the weight has stabilized following weight loss (Carmena et al., 1984; Sorbis et al., 1981; Wolf & Grundy, 1983). Carmena et al. (1984) investigated the effects of a 1200 kilocalorie per day diet on body weight and plasma lipids in 40 obese female subjects. This diet was instituted for 9 months, and the average body weight loss was 14.8 kg. The increase in HDL was significant and occurred before weight stabilization. Using a 4-week fast, Docubu and Dupont (1980) found that, after a mean weight loss of 18 kg, HDL increased significantly in women from a mean of 36 to 49 mg/dl. These elevations correlated with weight reduction, and no significant changes were found in other lipid fractions. Sorbis et al. (1981) examined eight grossly obese females before and after a mean weight reduction of 4 to 22 kg. Blood samples were taken at the end of the first week and after 5 weeks on a restricted diet. Then the restricted diet was modified and, after 2 to 3 weeks of weight maintenance, blood samples were taken again. Total cholesterol levels were not altered, but there was a mean 20% increase in HDL concentrations during the period of caloric restriction. The increase in HDL levels was related to the weight reduction rather than to caloric restriction, since HDL concentrations remained elevated when the patients were put on the weight-maintaining diet.

The studies that reported a reduction in HDL as a result of weight loss looked at the values during weight loss and did not include a follow-up period (Brownell & Stunkard, 1981; Larosa et al., 1980). Brownell & Stunkard (1981) studied 62 obese women who participated in a 16-week weight reduction

program. At the end of the weight loss, the women had a reduction in HDL concentration of 3.5% from 48.9 $\pm$ 1.5 mg/dl to 46.1 $\pm$.8 mg/dl.

Only two studies showed no change in HDL levels in females as a result of weight loss. The study by Thompson et al. (1979) found that the HDL levels went down during weight loss and then returned to baseline levels during the follow-up period. Hagan et al. (1986) studied women who had an HDL average of 49 $\pm$ 9 mg/dl that decreased to 43 $\pm$ 7 mg/dl during weight loss, which the authors stated was an HDL level that remained constant. This level was maintained after 8 weeks of follow-up, with an HDL average of 44 $\pm$ 8 mg/dl.

Weight gain or an increase in adiposity may result in a decrease in HDL levels. Presumably, the apoproteins of HDL have been shunted into the transport of triglyceride in VLDL. Therefore, HDL levels will be lower when triglyceride levels rise with weight gain (Connor et al., 1982).

Calorie Restriction for Weight Loss

Some degree of caloric restriction is incorporated into almost all weight loss programs and attempts (Dwyer, 1980). The diets used range from those of 1,200 to 1,800 kilocalories per day to those drastically restricting calories to 400 to 1,000 per day or containing no calories at all, as in the case of fasting or starvation. Some restrict carbohydrate partially or completely to promote very rapid weight loss (Dwyer, 1980; Stern, 1983). The nutritional adequacy of these diets is often very poor.

The Food and Nutrition Board (1980) stated that it is difficult to achieve nutritional adequacy in diets that are less than 1,800 to 2,000 kilocalories.

Nutritional analysis of 20 different popular weight loss diets found levels of thiamin, vitamins B_6 and B_{12}, iron, and zinc to be less than 70 to 80% of the recommended daily allowance (RDA) with kilocalorie intakes ranging from 600 to 2,000 per day. No studies were located that have looked at actual nutrient intake of dieters. However, in nationwide surveys of women's diets, intakes of calcium, iron, magnesium, zinc, vitamin B_6, and folacin also have been found to be less than 70 to 80% of the RDA (Kurinji, Klebanoff, & Graubard, 1986; Murphy & Calloway, 1986; Peterkin, 1986). With more than 50% of women in the United States dieting, the low nutrient levels in weight loss diets very well may be responsible for the low intakes of several of these same nutrients in general population surveys.

In spite of the wide variety of weight loss diets and the wide variety of dieters, there is only one goal: reaching ideal or desirable weight for height. The failure of reaching that goal and being able to maintain it for any length of time may lie in both the technique and the goal (Berchtold & Van Itallie, 1985; Stunkard & McLaren-Hume, 1959; Wing & Jeffery, 1979). One problem inherent in going on a diet is that sooner or later one goes off the diet (Stunkard, 1984). Old habits usually are resumed and so is old weight. Dieting is often ineffective because of this phenomenon, if for no other reason. In addition, dieting itself may predispose a person physiologically for the regain of weight once the caloric restriction is ended. This may be due to the lowered calorie needs that result from weight loss and/or a biological response to semistarvation.

Physical Activity

According to the President's Council on Physical Fitness and Sports (1971), fitness is defined as the ability to complete daily tasks with vigor and alertness, without unnecessary fatigue, and with enough energy to enjoy leisure-time pursuits and to meet emergencies. Most investigators agree that physical fitness encompasses a number of factors, including cardiovascular endurance, body composition, flexibility, and strength. Cardiovascular endurance is the ability to deliver sufficient oxygen to exercising skeletal muscles and is the most important aspect of physical fitness. It is measured most often by calculating maximal oxygen uptake (VO_2 max) on an exercise treadmill or bicycle ergometer. VO_2 max is an indication of maximal cardiovascular function when pulmonary function is normal (McCunney, 1987).

Physical Activity and CHD

Many epidemiological studies have examined the role of cardiovascular endurance in the prevention of CHD (McCunney, 1987). Although the precise role of habitual physical activity as a protector from CHD still has to be determined conclusively, the results of over 40 studies support the inference that physical activity is related inversely and causally to the incidence of CHD (Gordon & Cooper, 1988). Consistent favorable effects of physical activity have been reported for police officers, fire fighters, civil servants, and longshoremen. In one of the most thoroughly studied groups, which was composed of Harvard alumni who entered college between 1916 and 1950, the physically active individuals had markedly lower mortality rates (Paffenbarger, Hyde, & Wing,

1986). The risk of death became progressively lower as the physical activity level increased from an energy expenditure of less than 500 kilocalories to more than 3,500 kilocalories per week. Increases in energy expenditure beyond 2,000 kilocalories per week (the amount of energy expended in walking or running 20 miles) did not appreciably affect the risk of CHD.

Berlin and Colditz (1990) used meta-analysis techniques to extract data from the recent published studies addressing the relationship of physical activity and CHD. The results of the meta-analysis found an association between the lack of physical activity and increased risk of CHD. This association is stronger when the highly active group in a study is compared with a sedentary group, rather than when compared with a group that has a moderate activity level. This pattern of association supports a dose-response relationship between physical activity and protection from CHD. This relationship supports the argument by Paffenbarger, Hyde, and Jung (1984) that the explanation for a lack of association between increased activity and decreased CHD risk in some studies is the relatively low activity level in the more active group. The lack of apparent difference between activity groups could stem also from measurement error that is largely relative to among-person variability in physical activity. However, even perfect measurement of activity levels that are close together could yield no association between the lack of activity and CHD risk. The data of the meta-analysis indicated that the protective effect of physical activity was due to prevention of the occurrence of major cardiovascular events, rather than to the reduction of the severity of events that do occur.

The precise means by which aerobic exercise training aids in reducing CHD risk has been speculated. According to Gordon and Cooper (1988), several theories have been postulated to account for the apparent protective effect of aerobic exercise conditioning against CHD. These include reduced myocardial oxygen demand and increased myocardial oxygen supply, decreased platelet aggregation, increased plasma fibrinolytic activity, reduced adiposity, improved carbohydrate metabolism, reduced blood pressure, reduced anxiety and stress levels, and a reduced likelihood of ventricular arrhythmias. Another frequently cited possibility is a favorable effect of aerobic exercise training on lipoprotein metabolism (Gordon & Cooper, 1988).

Physical Activity and CHD in Women

The studies on physical activity and CHD in women are conflicting, with approximately 50% showing no advantage in the active group (Powell, Thompson, Caspersen, & Kendrick, 1987). Many of the physical activity questionnaires used in epidemiologic studies were developed and validated primarily on men. Many women may undertake considerable activity in child care and household activities. If previous questionnaires misclassify more women than men on physical activity, studies on sedentary habits and disease in women would be more likely to show no association (Blair et al., 1989).

In Finland, Salonen, Puska, and Tuomilento (1983) found significant relationships between physical activity, either at work or at leisure, and acute myocardial infarction and all-cause mortality. Similar findings were reported by Lapidus and Bengtsson (1986) in their 12-year prospective study of women.

Females with lower levels of leisure time activity in that study had significantly higher incidence rates of myocardial infarction and electrocardiographic evidence of ischemic heart disease, when compared with women with higher levels of leisure time activity. The Framingham Study (cited in Kannel & Sorlie, 1979) found significant univariate relationships between women's activity levels and all-cause mortality and mortality due to CHD. When statistical adjustment was made for age, however, the relationships no longer were statistically significant. Additionally, persons with CHD may benefit from physical training (Miller, Thelle, Forde, & Mjos, 1977; Oberman & Kouchoukos, 1978; Rechnitzer, Pickard, & Paivio, 1972).

A study on the relationship of physical fitness (as opposed to physical activity) in women and CHD mortality (Blair et al., 1989) demonstrated an inverse relationship. Physical fitness was measured by a maximal treadmill exercise test among 3,120 women, and the results demonstrated that higher levels of physical fitness appear to delay CHD mortality.

Physical Activity and Cholesterol

A meta-analysis of studies examining the effects of exercise on blood lipids and lipoproteins (Tran et al., 1983) examined 66 training studies involving the measurement of TPC changes over time. These studies were conducted over a 26-year period and represented 2,925 subjects (2,086 experimental and 839 control). Across all types of subjects, treatments, sources, and research designs, the average exercising subject was found to have a reduction in TPC of 10 mg/dl. None of the changes for the control groups were significant.

Higher initial levels of TPC resulted in greater decreases in post-exercise TPC ($r = 0.48$). The correlation between the number of hours spent exercising and the changes in TPC was such that an increase in the number of hours of exercise was associated with greater decreases in total cholesterol. The lower training intensities (when measured as a percent of maximum heart rate) were associated with more beneficial changes in TPC. This relationship was valid only at intensities equal to or greater than 60% of maximum heart rate.

The effects of a 10-week supervised aerobic exercise training program on cholesterol levels was assessed in 9 women (Hill et al., 1989). The subjects were sedentary and of normal body weight. The subjects were placed on a progressive exercise program until they were exercising for 1 hour, 4 times a week. The results were controlled for the effect of smoking, change in dietary intake, and alcohol. TPC levels were related positively to the amount of body weight, body fat, and fat-free mass. The changes in TPC that occurred with exercise were greatest in subjects with higher initial levels of body weight and body fat-free mass. Mean TPC was reduced from 187 ± 8 mg/dl to 175 ± 8 mg/dl.

The relationship between self-reported physical activity and levels of TPC was examined in a group of 541 premenopausal women by Owens et al. (1990). Physical activity was assessed using the Paffenbarger Activity Questionnaire, and women were classified according to quartile of weekly energy expenditure into groups of 0-500, 501-999, 1,000-1,999, and 2,000 kilocalories or greater. The results showed that the more active women had

lower TPC levels. Only the women who reported 2,000 kilocalories expended in exercise per week had significantly lower TPC.

Brownell, Bachorik, and Ayerle (1982) studied 37 overweight women (12.2% above desirable weight) over a 10-week period. Thirty-minute sessions were held three times weekly and the exercise was progressive until, by the fourth week, the subjects were exercising at approximately 70% of maximal heart rate for 15 to 20 minutes at each session. The women showed significant decreases of 3.9% in TPC levels ($p < 0.06$).

The failure of physical activity to have a significant independent effect on TPC levels in some studies may be due partially to the the differential changes in the concentration of cholesterol transported as part of the various lipoproteins. For example, an increase in the cholesterol content of HDL may be nearly matched by a decrease in the cholesterol content of other lipoproteins such that no significant change occurs in TPC levels (Haskell, 1985).

Exercise and Lipoproteins

Tran et al. (1983) conducted a meta-analysis of studies on the effects of exercise on lipoproteins. The results of 66 training studies looking at the changes over time and representing 2,986 experimental and 839 control subjects were analyzed. Across all types of subjects, treatments, sources, and research designs, the average exercising subject was found to have a LDL reduction of 5.1 mg/dl ($p < 0.05$). There was no significant correlation with initial levels of LDL, total amount of exercise as indicated by the total time spent exercising, or age with this finding. The larger decreases in LDL levels were

associated with lower intensities as long as the intensities were equal to or greater than 60% of maximum heart rate.

Several mechanisms have been postulated to explain exercise training-related reductions in LDL levels. One theory is the reduced hepatic syntheses of VLDL, which is a precursor of LDL. Another theory postulates an impaired conversion of VLDL remnants to LDL. Another possible explanation is the enhanced LDL receptor activity with increased fractional uptake of LDL by hepatic or peripheral cells. Currently, the data to support any of these postulates are scarce (Gordon & Cooper, 1988).

Cross-sectional comparisons have failed to consistently document lower LDL levels in endurance-trained individuals compared to sedentary controls, even though they are reduced generally by vigorous aerobic exercise. Longitudinal studies evaluating the influence of aerobic exercise training on LDL levels have yielded conflicting results, with the observed effect generally being only a minor reduction (Haskell, 1985). When LDL has been lower in the endurance athletes, the difference generally has been 7 to 12% and has occurred most frequently in very lean runners. Plasma LDL concentrations of speed and power-trained persons are similar or somewhat lower than sedentary controls. In the general population, habitual physical activity status or exercise capacity has not been related to plasma LDL concentration (Kusela, Vouticainen, Kukkonn, & Rauramaa, 1980; Lehtonen & Viikari, 1978; Schwane & Cundiff, 1979). The reasons for this conflict may be due to the duration and intensity of the exercise training and/or to baseline LDL levels (Gordon & Cooper, 1988).

Many studies demonstrate a relationship between physical activity and HDL. Some studies show an increase in HDL levels with a regular program of exercise (Altekruse & Wilmore, 1973; Erkelens, Albers, & Hazzard, 1979; Huttunen, Lansimies & Vouitainen, 1979; Kiens, Jorgensen & Lewis, 1980; Lopez, Vial, Balart, & Arroyave, 1974; Streja & Mymin, 1979; Williams, Wood, & Haskell, 1982). Other studies found no significant increase in HDL levels with exercise (Jennings, Nelson & Nestel, 1986; Pauly, Palmer & Wright, 1982; Shephard, Youldon & Cox, 1980).

Cross-sectional studies have examined the relationships between various fitness levels and HDL. Among 50 runners (500 miles per year), HDL levels were 20% greater (Adner & Castelli, 1980); among distance runners (35 miles per week), 20 mg/dl greater (Wood, Haskell, & Stern, 1977); and among Finnish cross-country skiers (15 miles per week), 25% greater (Lehtonen & Viikari, 1978) than those of sedentary controls. Norwegian cross-country skiers (Enger, Herbjornsen, & Erikssen, 1977), and elite long-distance runners (Martin, Haskell, & Wood, 1977) were found to have HDL levels 70% and 14% greater than those of sedentary controls, respectively.

In one of the larger cross-sectional studies (Hartung, Foreyt, Mitchell, Vlasek, & Gotto, 1980) HDL levels were compared among 85 joggers (3 miles per week), 59 marathoners (40 miles per week), and 74 sedentary subjects. Values for the sedentary group were 43.3 mg/dl, and for the joggers 58.0 mg/dl (34% greater). The marathoners had HDL levels 12% greater than those of joggers.

Physical Activity and HDL in Women

Few studies on the effect of physical activity and HDL have been done on women. Gibbons, Blair, and Cooper (1983) evaluated more than 1,700 healthy women, and found that fitness (measured by duration on a maximal exercise test) was independently associated with HDL. Morgan et al. (1986) compared female endurance runners with female weight trainers and sedentary female controls, and found that the mean HDL concentration was significantly higher in the endurance runners than in both the weight lifters and controls. There was no significant difference in mean HDL between the weight lifters and the controls. The association of fitness and high levels of HDL was strong in a study by Sallis, Patterson, Buono and Nader (1988). Forde et al. (1986) studied physical activity by self-report among 6,768 healthy women aged 20 to 53, and found a modest effect on HDL by leisure-time activity. Reaven, McPhillips, Barrett-Connor, and Criqui (1990) studied 1,273 women aged 50 to 89, and found that strenuous exercisers had significantly higher age-adjusted HDL levels than nonstrenuous exercisers. Adjusting for differences in cigarette smoking, alcohol, or obesity did not alter these results. Physical activity was measured by self-report.

The relationships of physical activity to HDL levels were examined in 255 menopausal women using self-report measures (Cauley et al., 1986). Physical activity was related significantly to HDL in these menopausal women.

A study using a population-based sample of middle-aged women reported that those who engaged in strenuous physical activity had higher HDL levels relative to their less active counterparts (Folsom et al., 1985). Another study

using a population-based sample, examined the relationship between self-reported physical activity and HDL (Owens et al., 1990). The sample included 541 premenopausal women. The results showed that the more active the women, the higher the HDL cholesterol levels. When the analysis was repeated controlling for the effect of education and body mass index, the results of the statistical test for linear trend remained significant. Women reporting activity levels of 1,000 kilocalories per week had higher HDL levels compared with women reporting lower levels of activity. Meilahn et al. (1988) found that activity level in a community sample of middle-aged women was not an independent determinant of lipoproteins after controlling for adiposity and cigarette smoking.

Several studies showed insignificant increases in HDL as a result of physical activity. Sixteen middle-aged, nonobese, nonsmoking, asymptomatic women participated in unsupervised aerobic activities 3 days per week for 6 months, and HDL levels rose 1.9 mg/dl, which was nonsignificant (Ballantyne et al., 1978). Hill et al. (1989) examined 9 females after a 10-week supervised aerobic exercise training program. The subjects were of normal body weight and were premenopausal. The rise in HDL levels was 3 mg/dl, which was not significant. Another study that had a nonsignificant rise in HDL was by Lewis et al. (1976). This study examined 22 obese women during a 17-week program that consisted of 2 days per week of walking and jogging at 80% maximum heart rate, plus 2 days per week of calisthenics. At the end of the study, the rise in HDL was 4.7 mg/dl, which was nonsignificant. Even though some studies have failed to document a concomitant elevation of plasma HDL levels with an

increase in physical activity, results generally show an increase (usually by 5 to 15%) in plasma HDL with training (Gordon & Cooper, 1988).

The apparent beneficial effect of physical activity on HDL may be mediated through alterations in lipoprotein metabolism. HDL has been shown to be related directly to lipoprotein lipase activity, which is increased by chronic training (Nikkila, Taskinen, & Kekki, 1978). Part of HDL is derived from the catabolism of VLDL by the activity of lipoprotein lipase (Tall & Small, 1978).

The amount of exercise required to increase HDL concentrations appears to be related to the baseline status of the subjects. According to Haskell (1985), in very sedentary cardiac patients, the expenditure of 200 to 300 kilocalories per session, three or more times per week, has been reported to increase HDL levels. For healthy but inactive persons more exercise generally seems necessary to produce HDL changes. The lower threshold appears to be moderate-intensity, endurance-type exercise requiring an energy expenditure of at least 1,000 kilocalories per week, with greater changes occuring as energy expenditure increases to 4,500 kilocalories per week (Haskell, 1985).

Relationship of Age to Cholesterol and Lipoproteins

Hypercholesterolemia and low HDL levels are risk factors for CHD in young, middle-aged, and elderly women. In the Framingham study (cited in Kannel, Anderson, & Christiansen, 1989), TPC levels correlated with CHD incidence in elderly women. In a study by Aronow (1990), a serum TPC of 200 and 250 mg/dl correlated with CHD prevalence in elderly women. Multivariate

analysis showed that TPC was a risk factor for new coronary events in elderly women, in women with prior CHD, and in women with no prior CHD.

Several studies have documented a rise in TPC and LDL levels with age in women. Connor et al. (1982) studied 243 females aged 16 to 69. They found the rise in plasma cholesterol levels with age was 1.68 mg/dl per year. For every decade, the plasma cholesterol was 16.8 mg/dl higher in females from the young adult years through the middle years of life. In another study by Taylor et al. (1981), 218 females were studied and it was found the TPC concentrations increased with age, with the mean serum cholesterol level in women aged 50 to 60 years exceeding that of men ($p < 0.02$). These results are similar to those in a study by Lewis et al. (1974). Larosa (1990) found that LDL levels in women rose with age.

Blacket, Woodhill, Leelarthaepin, and Palmer (1975) suggested that the rise in plasma lipids with age is related to increased body weight as adipose tissue, which is so characteristic of Western populations. There is no significant age trend for the plasma lipids and lipoproteins with more physical activities, less adiposity, and a diet that is habitually low in cholesterol and total and saturated fat among contrasting, less affluent populations. The Tarahumara Indians, who eat a nutritionally adequate diet that contains less than 100 mg per day of cholesterol and about 12% of the total calories as fat, do not exhibit this trend in elevated TPC and LDL levels with age. The Japanese, the Highland New Guineans, the Bantu, and other populations also have a different trend than the Westernized populations of the world (Connor et al., 1982).

Low HDL levels correlate with CHD in the elderly. In the Framingham study, low HDL levels correlated with CHD in elderly women (cited in Kannel et al., 1989). Aronow (1990) found that HDL levels below 35 mg/dl correlated with new coronary events in elderly women based on univariate analysis. Multivariate analysis showed that HDL levels correlated with CHD in elderly women with and without prior CHD.

In a study of 3,365 females by Green et al. (1985), HDL levels remained stable up to 34 years of age among participants not taking hormones. Only in the age group 35 to 39 was there evidence of a decline which continued until 59 years of age. Thereafter, there was slight increase in HDL levels that appeared to stabilize.

The serum HDL levels did not change with age in a study by Taylor et al. (1981). This study examined 218 females under 65 years of age. Other studies have shown either no change in serum HDL cholesterol levels with increasing age (Lewis et al., 1974; Williams, Robinson, & Bailey, 1979), or a slight decrease with age in females (Gordon et al., 1977).

Menopause, Cholesterol, and Lipoproteins

The role of menopause in the development of CHD is controversial. Several studies have shown an association between female castration or precocious menopause and an increased risk of CHD (Bengtsson, Rybo, & Westerberg, 1973; Colditz et al., 1982; Gordon, Kannel, Hjortland & McNamara, 1978; Parrish, Carr, Hall, & King, 1967; Rosenberg et al., 1981; Svanberg, 1982). However, the influence of natural menopause on the risk of

CHD is not clear. Two of the most important prospective studies on menopause have reported conflicting results. In the Framingham Study (cited in Gordon et al., 1978), naturally menopausal women as well as hysterectomized or oophorectomized women had higher incidence rates of CHD than premenopausal women of the same age. In the Nurses' Health Study (cited in Colditz et al., 1987), natural menopause was not associated significantly with an increased risk of CHD, in contrast to the relationship to bilateral oophorectomy. Thus, menopausal effects on the atherogenic process, which were attributed classically to the decline in ovarian estrogen production, remain uncertain.

Another approach to examining whether menopause may result in a higher risk of CHD involves exploring the relationships between menopausal status and CHD risk factors. There is some evidence that menopausal women have higher cholesterol levels than premenopausal women (Baird, Tyroler, Heiss, Chambless, & Hames, 1985; Bengtsson & Lindquist, 1979; Hjortland, McNamara, & Kannel, 1976; Kannel, Hjortland, McNamara, & Gordon, 1976; Linquist, 1982; 1976; Shibata, Haga, Suyama, Kumagai, & Seino, 1987; Shibata, Matsuzaki, & Hatano, 1979; Weiss, 1972).

Recent prospective data suggest that lipoprotein changes begin in perimenopause, or the period just before full menopause (Meilahn, Kuller, & Matthews, 1989). During 3 years of observation, all women in a prospective study, both the perimenopausal and those who became menopausal, demonstrated a mean increase in LDL of 10.1 mg/dl. These changes were greater, however, in women who became menopausal. HDL levels fell slightly,

primarily in menopausal women. These data controlled for smoking, alcohol intake, and weight. These perimenopausal and menopausal lipid changes are consistent with the gradual reduction in estrogen which occurs throughout the perimenopausal years (Korenmen, Sherman, & Korenman, 1978).

Recent preliminary data suggest that, while premenopausal women may have less dense, less atherogenic LDL particles than do men, levels of smaller, denser LDL particles increase after menopause. These denser particles are correlated with triglyceride levels and decreased HDL levels, and are atherogenic (Bengtsson, Rybo, & Westerberg, 1973).

Summary

CHD is the leading cause of death in women. Although fewer studies have been conducted on women than have been conducted on men, levels of TPC, HDL, and LDL have an association with risk of CHD in both sexes.

On the other hand, adiposity as a risk factor for CHD is controversial, especially since few studies have been conducted on women. The effect of body composition on CHD risk may stem from its effect on cholesterol and lipoproteins. There is evidence that obese women have elevated TPC levels and lower levels of HDL than normal weight women.

It generally is thought that weight reduction decreases most risk factors for CHD, but the results of studies are controversial. The majority of studies have demonstrated decreases in TPC and LDL levels during weight loss, but several studies have found that TPC and LDL levels did not decrease significantly, or they increased during weight loss. Other studies have shown that TPC and LDL

levels decreased during weight loss, but returned to baseline levels during weight stabilization. HDL levels were increased as a result of weight loss in the majority of studies. The increase in HDL generally occurred after weight stabilization.

Physical activity is inversely and causally related to the incidence of CHD, but studies on physical activity and CHD in women have not had consistent results. Physical activity has been shown to elevate HDL levels in many studies, and this may be the basis for the results in several studies demonstrating no decrease in TPC as a result of physical activity. Since TPC is composed of both HDL and LDL, if the HDL levels increase and the LDL levels decrease, the net effect on TPC is reduced.

Increasing age and menopause seem to have a negative effect on cholesterol and lipoprotein levels. With increasing age, TPC and LDL levels tend to increase and HDL levels decrease. It is not clear, however, if this phenomenon is due to age itself or is a result of increased body fat that occurs with increasing age. The effect of menopause on TPC, HDL, and LDL levels has not been studied thoroughly. Studies reported to date give some evidence that menopausal women have higher cholesterol levels than premenopausal women.

Since the effects of weight loss on TPC, HDL, and LDL levels are not consistent, further research is warranted. Many variables can change cholesterol and lipoprotein levels directly or indirectly by affecting weight loss. Furthermore, it is not known how quickly the cholesterol and lipoprotein levels can be affected by changes in these variables. Therefore, the relationships of

short-term weight loss and changes in TPC, LDL, and HDL should be researched further.

CHAPTER III

METHODOLOGY

The methodology of this descriptive study is discussed in relation to its population, procedures used to sample the population, instruments used to measure the variables, procedures used to collect the data, and descriptive and statistical techniques that were used to treat the data. The data collected for analysis in this study were retrospective data.

Population and Sample

The subjects for this study were drawn from the 111 females who had participated in the 13-day In-Residence Program (IRP) at the Aerobics Center in Dallas, Texas from 1986 through 1990. The women in this study included all of those who were not delimited by smoking, cholesterol-affecting medications, incompletion of the program, or age. These participants were mostly self-referred and came from across the United States to the IRP to receive a fitness evaluation, a health examination, and preventive medical advice. These women were well-educated, Caucasian (98.9%), and from middle and upper socioeconomic strata.

The subjects had a pretest medical evaluation on the first day, and a posttest medical evaluation on the twelfth day in the IRP. Exclusionary criteria for sample selection included smoking during or within 12 months immediately before program participation and/or consuming

medications known to affect levels of TPC, HDL, and/or LDL levels during the program.

Instrumentation

Serum cholesterol and lipoprotein fractions were measured using venous blood drawn in the supine position from an antecubital vein after 12 to 14 hours of fasting. TPC was measured using a Technicon Autoanalyzer (Technicon, Tarrytown, NY). HDL concentration was measured using an RA 1000 analyzer (Technicon, Tarrytown, NY) after very low-density lipoprotein and LDL were precipitated with sodium phosphotungstate in the presence of magnesium chloride. LDL was calculated according to the equation of Friedewald, Levy, and Fredrickson (1972). Cholesterol and lipoprotein measurements were performed by a Centers for Disease Control (Atlanta, Georgia) standardized laboratory.

Maximal graded treadmill testing was performed using the Balke and Ware (1959) protocol. Treadmill test time from this procedure is highly correlated (r = .92) with measured maximal uptake (Pollock et al., 1976). Fitness categories measured by the Modified Balke Treadmill Protocol were based upon treadmill time and age group, and were classified as very poor, poor, fair, good, excellent, and superior (Balke & Ware, 1959).

Body density was determined by hydrostatic weighing with 100 ml added to the residual volume to allow for air trapped in the gastrointestinal tract. Residual volume was calculated from height and age according to the equations of Goldman and Becklake (1959). Percent body fat was determined as the mean value calculated from the formulas of Brozek et al. (1957). Fat-free

weight was calculated by subtracting fat-tissue weight from total body weight. Studies using cadavers have reported the densities of the various body components and the proportion each represents relative to the entire body mass components (Forbes & Hursh, 1963; Martin, Drinkwater, Clarys, & Ross, 1981; Mitchell, Hamilton, Steggerda, & Bean, 1945; Widdowson, McCance, & Spray, 1951).

Procedures

During the first day of the 13-day IRP, the subjects were given a comprehensive medical examination which included a medical history questionnaire (see Appendix A), blood analysis (see Appendix B), maximal graded treadmill stress test (see Appendix C), and psychological profile. This medical examination also included hydrostatic weighing (see Appendix D), which was used to determine the participants' percent body fat. At the end of the examination, subjects had a consultation with a physician to review the findings. At this time the physician suggested additional follow-up tests if necessary.

On the second day of the program, the participants began the intervention (see Appendix E). The subjects started each day with a one-half mile walk at 7 a.m. At 8 a.m., breakfast was served. The IRP diet consisted of three meals a day totaling 1,000 kilocalories. The diet was high in complex carbohydrates and fiber, and low in fat, cholesterol, and sodium (15% protein, 20% fat, and 65% carbohydrate). At 9 a.m., the first lecture of the day was presented concerning a health-related topic. After the morning lecture, the subjects had

an exercise session. The guidelines for these sessions were determined by the treadmill test results, a physician's recommendation, and the exercise history of each participant. Exercise sessions included such aerobic activities as walking, stationary cycling, swimming, and rowing. After exercise, the participants were encouraged to use the time before the 1 p.m. lunch to relax, take care of personal business or appointments, or take a lesson in a health-related activity such as biofeedback, swimming, or weight training. After lunch, there was another presentation, and then a third exercise session was followed by the third lecture of the day. After dinner, which followed this third presentation, participants were encouraged to take a evening walk on their own or use the whirlpool, sauna, or have a massage.

On the twelfth day of the program, the subjects began the day with a second blood test, and height, weight, and circumference measurements. The rest of that day followed the same schedule as above.

Data Collection

Permission was obtained from Dr. Roy Vartabedian, Executive Director of the IRP, to use the previously collected data for this research study. Consent forms had been signed by each subject just prior to testing (see Appendix F).

The results of the complete medical evaluation were kept in each subject's medical file. Pertinent information for this study was extracted, and the abstracted data were entered into the computer and subjected to statistical analysis.

Treatment of the Data

Analyses of the data included descriptive statistics. The matched *t* test was used to examine the differences in the pretest and posttest measures of TPC, HDL, and LDL. Parametric correlations were used to examine the relationships among the variables and to determine variance accounted for by the independent variables.

CHAPTER IV

ANALYSES OF THE DATA

A descriptive, retrospective study was conducted to determine the relationships among adiposity, cardiovascular fitness levels, age, initial cholesterol levels, menopausal status, weight loss, and changes in TPC, HDL, and LDL levels in adult female participants in a 13-day residential wellness program. The study also determined whether there were differences in TPC, HDL, and LDL levels as a result of participation in the IRP. The data obtained were analyzed using matched *t* tests, point-biserial correlations, and Pearson product moment correlations. The assumptions of linearity, homoscedasticity, normality, and lack of outliers were checked and passed. These tests were conducted to insure the assumptions were met when appropriate. In this chapter, the findings are reported in both narrative and tabular presentations.

Description of the Subjects

The 56 subjects in this study were selected from the females who had attended the 13-day IRP from 1986 through 1990 and had participated in pre- and posttesting. These women were nonsmokers and were not taking medications that are known to affect cholesterol levels, including beta blockers, diuretics, and insulin, during the time between pretest and posttest measures. There were nine women who were being treated with estrogen replacement therapy, five women who were using oral contraceptives, and nine women

who were using thyroid medications. Comparisons of changes in weight, TPC, HDL, and LDL levels found no significant differences between groups using these medications and those not using these medications. Therefore, the data for the women who were using these medications were included with the data for subjects who were not using these medications.

Table 1 presents descriptive data concerning the subjects' age (years), height (inches), and adiposity (percentage of body fat). The women ranged in age from 30 to 68 years. The height of the subjects ranged from 55.25 inches to 70 inches. The adiposity of these women ranged from 18.75% to 44% body fat.

Table 1

Age, Height, and Adiposity of Subjects

Variables	M	Range	SD
Age	45.69	38.00	9.80
Height	64.27	14.75	2.61
Adiposity	32.08	25.25	5.99

Table 2 describes the menopausal status of the 56 subjects. More than half (62.5%) of the subjects were premenopausal.

Table 2

Distribution of Subjects' Menopausal Status

Menopausal Status	Frequency	Percent
Premenopausal	35	62.5
Menopausal	21	37.5
Total	56	100.0

Table 3 describes the pretest and posttest values of weight and TPC, HDL, and LDL levels. All four of these variables decreased during the 11 days between the pre- and posttesting. The subjects' weight ranged from 102 to 274 pounds at the time of pretest measurement, and from 102 to 265 pounds at the time of posttest measurement. The subjects' mean pretest TPC levels ranged from 129 to 320 mg/dl, and mean posttest measurements ranged from 58 to 302 mg/dl. The mean pretest levels of HDL ranged from 33 to 94 mg/dl, and the mean HDL posttest levels ranged from 33 to 92 mg/dl. The subjects' mean pretest LDL levels ranged from 61 to 240 mg/dl, and mean posttest levels ranged from 60 to 217 mg/dl. The pretest standard deviations (SD) indicated that the mean pretest levels for TPC, HDL, and LDL were more dispersed than the mean posttest levels.

Table 3

Subjects' Weight and TPC, HDL, and LDL Levels

Variable	M	Range	SD
Weight			
Pretest	155.56	172.00	35.36
Posttest	151.85	163.00	35.70
TPC			
Pretest	218.39	191.00	44.12
Posttest	190.54	244.00	40.95
HDL			
Pretest	60.93	61.00	14.82
Posttest	59.36	59.00	11.96
LDL			
Pretest	141.95	179.00	40.27
Posttest	119.55	157.00	33.73

Table 4 describes the change between the mean pretest and posttest levels for TPC (mg/dl), HDL (mg/dl), LDL (mg/dl), and weight (pounds). This table reports the mean level decrease in the 11 days between the pretest and the posttest for these female subjects. The decrease in the mean levels of LDL (22.39 mg/dl) paralleled the decrease in the mean levels of TPC (27.85 mg/dl), while the decrease in the mean HDL levels was much smaller (1.57 mg/dl). The

change in TPC levels from pretest to posttest measurements ranged from an increase of 54 mg/dl to a decrease of 132 mg/dl. The change in HDL ranged from an increase of 21 mg/dl to a decrease of 30 mg/dl. The change in LDL levels ranged from an increase of 46 mg/dl to a decrease of 95 mg/dl. The change in weight ranged from a gain of 1.5 pounds to a loss of 10 pounds.

Table 4

Decreases in Subjects' Weight, TPC, HDL, and LDL Levels

Variable	M	Range	SD
Weight	3.71	11.50	2.36
TPC	27.85	186.00	30.49
HDL	1.57	51.00	10.38
LDL	22.39	141.00	26.56

The number and percentage of subjects at each of the six levels of cardiovascular (CV) fitness (Balke & Ware, 1959), which corresponded to treadmill test scores taken during pretest measurements, are reported in Table 5. This table shows that more than half of the subjects (58.1%) were in the combined fair and good categories. The table also indicates that 18.21% of

the subjects were in the combined very poor and poor categories, and that 23.70% were in the combined excellent and superior categories.

Table 5

Subjects' Cardiovascular Fitness

Fitness Level	Frequency	Percent
Very Poor	6	10.90
Poor	4	7.30
Fair	13	23.60
Good	19	34.50
Excellent	10	18.20
Superior	3	5.50
Total	55	100.00

Note. n did not equal 56 because one subject did not take a treadmill test.

Statistical Analyses of the Data

The data were analyzed using the BMDP statistical software (PC90 version). The matched t test was used to determine if there were significant differences between pretest and posttest measures of TPC, HDL, and LDL. According to Tuckman (1978), a t test allows the researcher to compare two

means to determine the probability that the difference between the means is a real difference rather than a chance difference.

Table 6 summarized the results of matched t tests to analyze the differences between the mean pretest and posttest levels of TPC, HDL, LDL, and weight. The t values for the declines in mean TPC, LDL, and weight from pretest to posttest measures were significant at the .05 level. The t value for the decline in HDL levels from the pre- to posttest measures was not significant at the .05 level.

Table 6

Results of Matched t Tests for Changes in TPC, HDL, LDL, and Weight Means

Dependent Variable	t	p
TPC	-6.84	<.0001
HDL	-1.13	.26
LDL	-6.31	<.0001
Weight Loss	11.98	<.0001

Null hypotheses 2 through 7 were analyzed using correlational techniques. According to Tuckman (1978), the parametric correlation is used to deal with two interval variables, each of which is normally distributed. A correlation is an indication of the predictability of one variable given the other.

Table 7 shows the relationships between menopausal status, and changes in weight, TPC, HDL, and LDL. Using the point-biserial correlation technique, no significant correlations were found between menopausal status and weight loss or decreases in the levels of TPC, HDL, and LDL. The point-biserial correlation was selected because, in each test, one of the variables (weight, TPC, HDL, or LDL) was continuous while the other (premenopausal or menopausal status) was a true dichotomy.

Table 7 also summarizes the correlations among baseline cardiovascular (CV) fitness levels, weight loss, and decreases in levels of TPC, HDL, and LDL. The point-biserial correlation technique was selected for the analysis because weight, TPC, HDL, and LDL are continuous variables while the six CV fitness categories are dichotomous variables. For the subjects in this study, there was a significant inverse relationship between baseline levels of CV fitness and decreases in weight from pretest to posttest. The greater the CV fitness level, the less the weight lost from pretest to posttest. Changes in levels of TPC, HDL, and LDL were not related significantly to baseline CV fitness levels.

The Pearson product moment correlation was used to examine the relationships between changes in weight, TPC, HDL, and LDL. Table 8 shows one significant correlation: The significant inverse relationship between weight loss and changes in TPC levels from pretest to posttest. The more pounds lost, the greater the reduction in levels of TPC. The Pearson product moment correlation was chosen because both weight loss and changes in levels of TPC, HDL, and LDL are continuous variables. There was no significant correlation between weight loss and changes in HDL or LDL levels.

Table 7

Point-Biserial Correlations Among Menopausal Status, Cardiovascular Fitness, and Changes in Weight, TPC, HDL, and LDL Levels

Dependent Variable	Menopause (r)	CV Fitness (r)
Weight Loss	0.1211	0.2717 *
TPC	-0.0561	-0.0482
HDL	-0.0861	0.0377
LDL	-0.0851	-0.0015

* significant at .05 level

Table 8

Pearson Correlations Among Weight Loss and Changes in TPC, HDL, and LDL

Dependent Variable	Weight Loss (r)
TPC	-0.2852 *
HDL	-0.0697
LDL	-0.1946

* significant at .05 level

The Pearson product moment correlation was used to analyze the relationships between adiposity and changes in the weight, TPC, HDL, and LDL. Table 9 shows that the only variable found to be related significantly to baseline body fat percentage was weight loss. Females in this study with a higher initial percentage of body fat had a greater decrease in weight from pretest to posttest as compared to women who had a lower percentage of body fat at baseline. Changes in levels of TPC, HDL, and LDL were not related significantly to adiposity.

Table 9

Pearson Correlations Among Adiposity and Age and Changes in Weight, TPC, HDL, and LDL Levels

Dependent Variable	Adiposity (r)	Age (r)
Weight Loss	-0.3712 *	0.1480
TPC	-0.1037	-0.2750 *
HDL	0.0682	-0.0062
LDL	-0.2242	0.2690 *

* significant at .05 level

The Pearson product moment correlation technique was selected to examine the relationships among age, weight loss, and changes in the levels of

TPC, HDL, and LDL. This technique was selected because all of the variables were continuous. Table 9 indicates that age was significantly, inversely related to decreases in TPC and LDL levels. In this study, older females as compared with younger females had greater decreases in the levels of TPC and LDL between pretest and posttest. There were no significant correlations between age and weight loss or changes in HDL levels.

Table 10 illustrates the correlation among initial levels of TPC and changes in weight, TPC, HDL, and LDL levels. The Pearson product moment correlation technique was selected again because all of the variables were continuous. There was a significant inverse relationship between initial TPC levels and decreases in levels of TPC and LDL. The women in this study who had higher initial levels of TPC had greater decreases in posttest TPC and LDL levels than did women with lower initial levels of TPC. Initial levels of TPC were not correlated significantly with changes in weight or HDL levels.

There was a significant inverse relationship between initial levels of HDL and changes in HDL levels from pretest to posttest for the women in this study. Subjects with higher initial levels of HDL had greater decreases in HDL levels from pre- to posttest as compared to those in this study who had lower initial levels of HDL. Initial levels of HDL were not correlated significantly with changes in weight, TPC, or LDL levels.

Table 10 also shows correlations between initial levels of LDL and changes in weight, TPC, HDL, and LDL levels. There was a significant inverse relationship between initial levels of LDL and changes in levels of TPC and LDL levels. Females in this study with higher baseline levels of LDL had greater

reductions in levels of TPC and LDL from pretest to posttest as compared to females who had lower baseline LDL levels. Correlations were not significant between initial levels of LDL and changes in weight or HDL levels.

Table 10

Pearson Correlations Among Initial Levels of TPC, HDL, and LDL, and Changes in Weight, TPC, HDL, and LDL Levels

Dependent Variable	Initial TPC (r)	Initial HDL (r)	Initial LDL (r)
Weight Loss	0.1346	0.0734	0.1324
TPC	-0.4342 *	-0.0805	-0.3658 *
HDL	-0.1525	-0.6065 *	0.0949
LDL	-0.4632	0.0885	-0.5550 *

* significant at .05 level

CHAPTER V

SUMMARY, FINDINGS, DISCUSSION, CONCLUSIONS, AND RECOMMENDATIONS

This chapter is presented in five sections. The first section presents a summary of the study, which includes information concerning the purpose of the study, a description of the subjects, and methods of data collection and analysis. The second section is a summary of the findings, and the third section is a discussion of these findings, relating them to previous research. The fourth section, the conclusion of the study, restates each of the seven hypotheses and states whether or not each was rejected. The last section contains a list of recommendations for future research.

Summary of the Study

The purpose of this study was to determine the relationships among adiposity, cardiovascular fitness levels, age, initial levels of cholesterol and lipoproteins, menopausal status, weight loss, and changes in levels of cholesterol and lipoproteins in adult female participants in a 13-day residential wellness program (IRP). In addition, the study was to determine whether there were differences in the TPC, HDL, and LDL levels as a result of participation in the IRP.

The subjects for this study were 56 females who had participated in the 13-day IRP at the Aerobics Center in Dallas, Texas, from 1986 through 1990.

These women ranged in age from 30 to 68, were nonsmokers, and were not taking medications during the time between pretest and posttest which are known to affect levels of cholesterol.

Data for this retrospective study were secured by examining the subjects' medical information which was recorded during their stay in the IRP at the Aerobics Center. These data were analyzed using matched t tests, point-biserial correlations, and Pearson product moment correlations.

Findings of the Study

Statistical analyses of the data resulted in the following findings:

1. There are significant differences between pretest and posttest measures of TPC, HDL, LDL, and weight in this study.

2. No significant relationships were found in this study between menopausal status and changes in TPC, HDL, LDL, or weight.

3. There is a significant relationship between weight loss from pre- to posttest and changes in TPC. The greater the weight loss by the subjects, the greater the decrease in TPC.

4. There is a significant relationship in this study between baseline adiposity and weight loss. The women in this study with greater body fat percentage had greater weight loss from pretest to posttest when compared to women with smaller body fat percentage.

5. There is a significant relationship between cardiovascular fitness (as measured by fitness category) and weight loss. The more aerobically fit subject,

as compared with the less aerobically fit subject, had less weight loss from pretest to posttest measurement.

6. There is a significant relationship between age and changes in TPC levels and between age and changes in LDL from pretest to posttest in this study. Older women as compared to younger women had greater decreases in TPC and LDL levels from pretest to posttest measurement.

7. There is a significant relationship between initial levels of TPC and changes in TPC and LDL levels in this study. The females with greater initial levels of TPC had greater decreases in TPC and LDL levels from pretest to posttest when compared to females with smaller initial levels of TPC.

8. There is a significant relationship between initial levels of LDL and changes in TPC and LDL levels in this study. The females with greater initial levels of LDL had greater decreases in TPC and LDL levels from pretest to posttest when compared to females with smaller initial levels of LDL.

9. There is a significant relationship between initial levels of HDL and changes in HDL levels in this study. The females with greater initial levels of HDL had greater decreases in HDL levels from pretest to posttest when compared to females with smaller initial levels of HDL.

Discussion of the Findings

The females in this study had a significant decrease in levels of TPC, LDL, and weight levels during a 13-day IRP. Furthermore, statistical analysis found a significant correlation between weight loss and the decrease in TPC. These results parallel those of a meta-analytic review (Tran et al., 1983) that showed

that decreases in body weight were correlated significantly with decreases in cholesterol. The uniqueness of the IRP study, however, revolved around the short period between pre- and posttesting. The current published research on this topic has involved longer intervention periods from 4 weeks (Barnard et al., 1982) to 19 months (Carmena et al., 1984). Zimmerman et al. (1984), studied seven women for 8 weeks, but retested weekly so that values were known after one and two weeks of intervention. Zimmerman's study also found a significant decrease in TPC levels after one week of intervention that continued through the second week. A similar pattern developed for the decrease in LDL levels. Zimmerman found these declines in levels of cholesterol and LDL levels concomitant with weight loss. The study of the IRP subjects found a significant relationship between weight loss and changes in TPC levels, but did not find a significant relationship between weight loss and changes in LDL levels from pretest to posttest.

Is the decrease in TPC and LDL the result of weight loss alone or is it the result of caloric restriction, some other undetermined factor, or a combination of factors? The answer to this question is not clear. Since the follow-up measures in many of the published studies resulted in TPC and LDL levels returning to near baseline levels even though weight loss was maintained (Hagan et al., 1986; Thompson et al., 1979; Wolfe & Grundy, 1983; Zimmerman et al., 1984), it might seem as if the reduction in TPC and LDL was due to the process of caloric restriction and losing weight instead of the lower weight itself. On the other hand, other studies have found that TPC levels which decreased significantly with weight loss had stabilized during maintenance of weight loss (Davis et al.,

1985; Follick et al., 1984; Gonen et al., 1983; Stevenson et al., 1988; Wechsler et al., 1981). Perhaps, other variables are responsible for whether or not changes in cholesterol and lipoprotein levels are maintained.

In this study of IRP subjects, age was correlated significantly with the decrease in levels of TPC and LDL. The relationship between increasing age and decreases in TPC and LDL levels is interesting since there was no significant correlation between age and weight loss or adiposity. Several other studies have documented a rise in baseline TPC and LDL levels with increasing age in women (Connor et al., 1982; Larosa, 1990; Lewis et al., 1974; Taylor et al., 1981). Since initial levels of cholesterol rise with age, and since higher initial levels of cholesterol result in greater decreases in TPC and LDL, it may be that the older females in this IRP study had greater declines in cholesterol levels because they had greater initial cholesterol levels.

In this IRP study, age had no significant correlation with the decrease in HDL levels. This finding agreed with results from Williams et al. (1979) and Lewis et al. (1974). The only significant correlation with changes in HDL in the IRP study was the initial level of HDL. In this study, the women with higher initial levels of HDL had greater decreases in HDL from pretest to posttest.

The changes in the HDL levels were very inconsistent in this IRP study. There were almost equal numbers of subjects who had increased in HDL levels (28 females) and subjects who had decreased HDL levels (25 females). Three subjects had no change in HDL levels. These inconsistencies may explain the lack of correlations with changes in HDL. This dichotomous pattern of change

also may explain why the mean change in HDL levels from pretest to posttest was only 1.57 mg/dl.

The only significant relationships with weight loss were initial adiposity and cardiovascular fitness levels. The greater the subjects' level of CV fitness in the IRP study, the less their weight loss was from pretest to posttest. Cardiovascular fitness did not, however, significantly correlate to changes in levels of TPC, HDL, and LDL or to initial levels of TPC, HDL, and LDL in these subjects. Cross-sectional comparisons have failed to document consistently lower LDL levels in highly fit women when they have been compared to sedentary controls (Kusela et al., 1980; Lehtonen & Viikari, 1978; Schwane & Cundiff, 1979), even though it has been demonstrated that sedentary subjects have had decreased TPC and LDL levels as a result of consistent aerobic exercise (Tran et al., 1983). Decreases in TPC and LDL levels may be a result of an increase in activity and not the result of a certain amount of activity. Therefore, when the amount of activity remains constant, even though it is at a much higher level, the TPC and LDL levels may return to baseline.

Initial HDL levels and changes in these levels do not seem to follow the same pattern that TPC and LDL levels follow with respect to exercise. In this IRP study, the level of cardiovascular fitness was not associated with either the initial amount or the change in the level of HDL. This is contrary to previous studies, which have found that cardiovascularly fit females have higher HDL levels than controls (Gibbons et al., 1983; Morgan et al, 1986; Sallis et al., 1988). Even though some studies have failed to demonstrate an elevation of HDL levels with an increase in physical activity, previous study results generally

show an increase of 5-15% in HDL with aerobic training (Gordon & Cooper, 1988). One of the possible reasons the IRP study did not show a significant correlation between fitness levels and changes in HDL levels from pretest to posttest was the short period of time from pretest to posttest. The results of studies of training that lasted several months showed an increase in HDL levels. It seems likely, according to the literature (Gordon & Cooper, 1988), that a threshold for the duration of training and intensity of training must be exceeded in order to cause positive changes in HDL levels. For example, one study revealed that HDL levels did not begin to change until the subjects had been running more than 10 miles per week for at least 9 months (Williams et al., 1982).

The lack of significant relationship between weight loss and changes in HDL levels in the IRP study also may have been caused by the slow rate of change with intervention. Although the majority of studies reveal an increase in HDL levels in females as a result of weight loss (Carmena, 1984; Contaldo et al., 1980; Eckel & Yost, 1989; Follick et al., 1984; Friedman et al., 1982; Gonen et al., 1983; Lewis et al., 1976; Schwartz & Brunzell, 1981; Sorbis et al., 1981; Stevenson et al., 1988; Streja et al., 1980; Wolf & Grundy, 1983; Zimmerman et al., 1984), most studies found this increase only after weight had stabilized following weight loss.

There were no significant correlations between menopausal status and changes in weight or TPC, HDL, and LDL levels in the IRP study. This is an interesting finding in light of the fact that there was a correlation between menopausal status and initial levels of TPC and HDL. In the IRP study,

menopausal females had higher baseline levels for TPC and HDL than premenopausal women. This finding agrees with other studies which found that menopausal women have higher cholesterol levels than premenopausal women (Baird et al., 1985; Bengtsson & Lindquist, 1979; Hjortland et al., 1976; Kannel et al., 1976; Linquist, 1982; Shibata, et al., 1987; Shibata et al., 1979; Weiss, 1972). In the IRP study, there was no significant relationship between menopause and higher baseline levels of LDL; therefore the higher initial levels of TPC are due to the higher initial levels for HDL.

Conclusions

The following null hypotheses were tested at the .05 level of significance and within the scope of this study the following conclusions were drawn:

Hypothesis 1. There are no significant differences between pretest and posttest measures of TPC, HDL, and LDL. Based on the results of the matched t tests, this null hypothesis is REJECTED.

Hypothesis 2. There is no significant relationship between menopausal status and changes in levels of TPC, HDL, and/or LDL. Based on the results of the point-biserial correlations, this null hypothesis is NOT REJECTED.

Hypothesis 3. There is no significant relationship between short-term weight loss and changes in levels of TPC, HDL, and/or LDL. Based on the results of the Pearson product moment correlations, this null hypothesis is REJECTED.

Hypothesis 4. There is no significant relationship between adiposity and changes in TPC, HDL, LDL, and/or weight. Based on the results of the Pearson product moment correlations, this null hypothesis is REJECTED.

Hypothesis 5. There is no significant relationship between levels of cardiovascular fitness and changes in TPC, HDL, LDL, and/or weight. Based on the results of the point-biserial correlations, this null hypothesis is REJECTED.

Hypothesis 6. There is no significant relationship between age and changes in TPC, HDL, LDL, and/or weight. Based on the results of the Pearson product moment correlations, this null hypothesis is REJECTED.

Hypothesis 7. There is no significant relationship between initial lipid levels and changes in TPC, HDL, LDL and/or weight. Based on the results of the Pearson moment correlations, this null hypothesis is REJECTED.

Recommendations

Based on the results of this study, the following recommendations are offered for future studies:

1. Replicate the study using larger samples to reduce sampling error.
2. Replicate the study with a longer intervention period and a follow-up period after weight loss to see what changes may occur over a longer time of weight loss and during weight stabilization.
3. Replicate the study including men as well as women so that gender comparisons may be made.

4. Replicate the study using only sedentary subjects divided into two groups for comparisons of outcome: One group would remain sedentary during the intervention, and the other group would begin an aerobic exercise program.

5. Perform a dietary analysis of the subjects before participation in the IRP to establish baseline consumption of fats, carbohydrates, protein, and calories. Monitor food consumption in the program and analyze the relationships between the changes in diet and the changes in levels of TPC, HDL, LDL, and weight.

6. Replicate the study with the same dietary composition and kilocalorie consumption, but divide the subjects into two groups for comparisons. One group would be composed of those who did not lose weight from pretest to posttest, and the other group would be composed of those who did lose weight from pretest to posttest.

7. Replicate the study to analyze hip-waist ratios in subjects to determine relationships between centralized adiposity and initial levels of TPC, HDL, LDL, and weight. Additionally, examine the correlations between hip-waist ratios and changes in TPC, HDL, and LDL levels.

8. Replicate the study to compare the differences in outcomes between smokers and nonsmokers.

9. Replicate the study including initial levels of triglycerides and glucose and changes in levels of triglycerides and glucose to examine the relationships among triglycerides, glucose, and initial levels and changes in TPC, levels of HDL, and LDL.

10. Replicate the study divding the subjects into two matched groups for comparision. One group would complete the IRP as designed. The second group would add to the current design relaxation training that would be conducted twice daily.

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Appendix A

Medical History Questionnaire

MEDICAL HISTORY QUESTIONNAIRE: RETURN VISIT

Name: _____

Date of Examination: _____

Date of *Previous* Examination: _____

Please print your responses.

We encourage you to complete this form *prior* to your visit. If you are unclear about dates, family illnesses, or other information included on the previous questionnaire, you may refer to your last Cooper Clinic report to refresh your memory concerning your previous responses.

I. GENERAL INFORMATION

NAME:

_____ Dr.
_____ Rev.
_____ Mr.
_____ Mrs.
_____ Ms.
_____ Miss
_____ Other

_____ Age: _____
(Last) (First) (Middle)

_____
(Nickname or named used) (Maiden Name, if applicable)

ADDRESS: _____
(Number and Street) (City and State)

_____ ( )
(Country) (Zip Code) (Home Phone Number)

_____
(Soc. Sec. Account Number) (Birthday month-day—year) (Mother's Maiden Name)

PERSONAL PHYSICIAN: _____
(Last Name) (First)

_____ ( )
(Number and Street) (Physician's Phone Number)

_____
(City) (State) (Zip Code)

Do you want a copy of your report and all other documents relating to this medical examination sent to your personal physician? ☐ Yes ☐ No

If yes, give permission by signing your name. _____

Do you wish to authorize the loan of x-ray films to your personal physician or other consultant whom you may designate? ☐ Yes ☐ No

If yes, give permission by signing your name. _____

CURRENT OCCUPATION: Are you currently employed? ☐ Yes ☐ No

Name of Business or Employer: _____

Type of Business: _____

Your position, title or type of work: _____ ( )
(Business Phone Number)

How long have you been at your present job? _____

Complete Office Address: _____

_____

_____

_____

BILLING AND INSURANCE INFORMATION

PATIENT'S NAME _____ DATE _____

INSTRUCTIONS

If you are responsible for your charges, go to section marked SELF.

If your company is responsible for your charges, go to section marked COMPANY.

NOTE: Charges for any procedures which we perform at your request, which your company does not cover, will be your responsibility.

SELF

MAILING ADDRESS FOR STATEMENT: ☐ HOME ☐ OFFICE

Patients are responsible for prompt payment of charges. If you plan to file for insurance for reimbursement to yourself, please indicate:

☐ Insurance form required (number of copies needed _____).

☐ Participation in Type B Medicare.
Please provide your Health Insurance Claim Number as it appears on your Health Insurance Card if you are a participant in Medicare. Is Medicare your Primary ____ or Secondary ____ Coverage.

___ ___ ___ ___ ___ ___ ___ ___ ___ ___ ___

A standard insurance form will be mailed to you. You will need to fill in the name of the insurance company, your policy number, and sign a release form. You should then forward the completed form to your insurance company. If you need any assistance, please contact our bookkeeping department.

COMPANY

You will receive the original medical report. If a copy of this report and other documents relating to this medical examination are to be forwarded to your company, you MUST sign the authorization below. This copy will only be sent to an individual. Please indicate the name and address below.

I authorize the Cooper Clinic to send a copy of my medical report to the following individual:

NAME: _____ COMPANY NAME: _____

ADDRESS: _____

_____

SIGNED: _____

MAILING ADDRESS FOR STATEMENT:

Same as above. Other: _____

_____

_____

IF YOU NEED ANY HELP IN COMPLETING THIS PORTION, PLEASE ASK OUR RECEPTIONIST AT THE TIME OF YOUR VISIT.

I. GENERAL INFORMATION (CONT.)

REASON FOR VISIT:

Please check the appropriate box(es):

- ☐ Comprehensive Medical Evaluation
- ☐ Evaluation of Previously-Diagnosed Heart Disease
- ☐ Evaluation of Heart Disease Risk
- ☐ Determination of Present Level of Cardiovascular Fitness
- ☐ Recommendations for Exercise Program
- ☐ Recommendations for Nutritional Program
- ☐ Recommendations for Weight Loss Program
- ☐ Referred by Personal Physician
- ☐ Company Benefit
- ☐ Company Requirement
- ☐ Other _____

OTHER HEALTH DATA:

1. How many days of work did you lose due to illness in the past year? _____
2. How many times did you see a physician for medical reasons last year? _____
3. When was your last visit to a physician? (Approximate date) _____

   What was the reason for that visit? _____
4. When was your last visit to a dentist? _____
5. Please indicate someone outside your immediate family who will always know your address: (For our longitudinal research project)

   Name: _____

   Address: _____

   _____
6. Name, Address, and Phone Number of person to be notified in case of emergency:

   Name: _____ Relationship: _____

   Address: _____

   _____

   Phone Number: _____
7. How did you learn about the Cooper Clinic? _____

II. PERSONAL PROFILE

Sex: ☐ Male ☐ Female

Race: ☐ White ☐ Black ☐ Hispanic ☐ Asian ☐ Other (specify _____)

Place of Birth: _____

A. Marital History:

1. Are you now or have you ever been married? ☐ Yes ☐ No
   If yes, how many times have you been married? _____

2. Current marital status:
   - ☐ Single
   - ☐ Married
     If yes, how long? _____
   - ☐ Divorced
   - ☐ Widowed

3. Number of children? _____

B. Education: *(Circle highest level attained).*

Grade:	7	8	9	10	11	12		Degree	Field	College/Univ.
College:				1	2	3	4	BACHELOR	_____	_____	_____
								MASTERS	_____	_____	_____
Post Graduate:				1	2	3	4	DOCTORATE	_____	_____	_____

C. Military: Are you now or have you in the past served in the Armed Forces?
☐ Yes ☐ No

If yes, give branch and dates: _____

D. Present Household *(Check all that apply).*

- ☐ Apartment
- ☐ City
- ☐ House
- ☐ Suburbs
- ☐ Other _____
- ☐ Country

Does anyone live with you?

- ☐ Live alone
- ☐ Spouse
- ☐ Children
- ☐ Parents
- ☐ In-Laws
- ☐ Other

E. Present Occupation: What is your present work situation *(Check all that apply.)*

- ☐ Employed Full-time
- ☐ Employed Part-time
- ☐ Semi-Retired
- ☐ Fully-Retired
- ☐ Self-Employed
- ☐ Unemployed
- ☐ Housewife
- ☐ Student
- ☐ Other _____

If you are employed, please indicate the following:
Name of business or employer: _____

III. CURRENT MEDICAL STATUS

A. **PRESENT MEDICAL PROBLEMS:** Please list any known *significant* medical problems that you have *at present*:

PROBLEM	DATE OF ONSET	

IV. REVIEW OF SYSTEMS

Please indicate whether you have ever had a *significant* problem with any of the symptoms or conditions listed below.

	Yes	No	Don't know	If yes, when or onset?	Is this still a problem?	
GENERAL						
1. Unexplained weight loss	☐	☐	☐			
2. Chronic fatigue	☐	☐	☐			
3. Change in appetite	☐	☐	☐			
4. Night sweats	☐	☐	☐			
5. Fever or chills	☐	☐	☐			
6. Any type of cancer	☐	☐	☐			
7. Sleep disorder	☐	☐	☐			
HEART/VASCULAR						
8. Chest pain or pressure	☐	☐	☐			
9. Chest pain with exertion	☐	☐	☐			
10. Heart attack	☐	☐	☐			
11. Rapid or irregular heartbeats	☐	☐	☐			
12. Fainting or lightheadedness	☐	☐	☐			
13. High blood pressure	☐	☐	☐			
14. Rheumatic fever	☐	☐	☐			
15. Calf pain with exercise	☐	☐	☐			
16. Varicose veins	☐	☐	☐			
17. Phlebitis	☐	☐	☐			
18. Stroke	☐	☐	☐			
19. High blood cholesterol	☐	☐	☐			
20. High blood triglycerides	☐	☐	☐			
EYES						
21. Decrease in vision Date of last eye exam _____	☐	☐	☐			
22. Double vision	☐	☐	☐			
23. Glaucoma	☐	☐	☐			
24. Color blindness	☐	☐	☐			
25. Cataracts	☐	☐	☐			
26. Serious injury to eye	☐	☐	☐			
EAR-NOSE-THROAT						
27. Hearing loss	☐	☐	☐			
28. Prolonged exposure to loud noise	☐	☐	☐			
29. Ringing in ears	☐	☐	☐			
30. Chronic ear infection	☐	☐	☐			
31. Ruptured eardrum	☐	☐	☐			
32. Sinus infection	☐	☐	☐			
33. Vertigo	☐	☐	☐			
34. Vocal cord polyp	☐	☐	☐			
ENDOCRINE						
35. Thyroid disease	☐	☐	☐			
36. High blood sugar	☐	☐	☐			
37. Diabetes	☐	☐	☐			

IV. REVIEW OF SYSTEMS (CONT.)

	Yes	No	Don't know	If yes, when or onset?	Is this still a problem?	
PULMONARY						
38. Chronic cough or phlegm	☐	☐	☐			
39. Wheezing	☐	☐	☐			
40. Asthma	☐	☐	☐			
41. Tuberculosis	☐	☐	☐			
42. Bronchitis	☐	☐	☐			
43. Pneumonia	☐	☐	☐			
44. Emphysema	☐	☐	☐			
45. Coughed up blood	☐	☐	☐			
46. Unexplained shortness of breath	☐	☐	☐			
—while sleeping	☐	☐	☐			
—while sitting	☐	☐	☐			
—with physical activity	☐	☐	☐			
GASTROINTESTINAL						
47. Fatty food intolerance	☐	☐	☐			
48. Ulcer disease	☐	☐	☐			
49. Frequent heartburn	☐	☐	☐			
50. Vomited blood	☐	☐	☐			
51. Gallbladder trouble	☐	☐	☐			
52. Abdominal pain	☐	☐	☐			
53. Jaundice, hepatitis or cirrhosis	☐	☐	☐			
54. Frequent diarrhea	☐	☐	☐			
55. Diarrhea caused by milk (lactose intolerance)	☐	☐	☐			
56. Blood in stools	☐	☐	☐			
57. Tarry black stools	☐	☐	☐			
58. Hemorrhoids	☐	☐	☐			
59. Colon polyps	☐	☐	☐			
60. Chronic constipation	☐	☐	☐			
GENITOURINARY						
61. Veneral Disease	☐	☐	☐			
—syphilis	☐	☐	☐			
—gonorrhea	☐	☐	☐			
—herpes	☐	☐	☐			
62. Sexual problems	☐	☐	☐			
63. Decreased sex drive	☐	☐	☐			
64. Impotency	☐	☐	☐			
65. AIDS	☐	☐	☐			
66. Blood in urine	☐	☐	☐			
67. Burning or pain during urination	☐	☐	☐			
68. Kidney/bladder infection	☐	☐	☐			
69. Difficulty urinating (starting or stopping)	☐	☐	☐			
70. Prostate trouble	☐	☐	☐			
71. Awakening at night to urinate	☐	☐	☐			
72. Kidney stones	☐	☐	☐			

IV. REVIEW OF SYSTEMS (CONT.)

	Yes	No	Don't know	If yes, when or onset?	Is this still a problem?	
BONE AND JOINT						
73. Chronic joint or muscle pain	☐	☐	☐			
74. Low back pain	☐	☐	☐			
75. Swollen/stiff joints	☐	☐	☐			
76. Arthritis	☐	☐	☐			
77. Gout	☐	☐	☐			
NEUROPSYCHIATRIC						
78. Loss of consciousness	☐	☐	☐			
79. Vertigo	☐	☐	☐			
80. Seizures or epilepsy	☐	☐	☐			
81. Frequent headaches	☐	☐	☐			
82. Treatment for nervous disorder	☐	☐	☐			
83. Numbness or tingling of arms, legs or face	☐	☐	☐			
84. Difficulty sleeping	☐	☐	☐			
85. Depression	☐	☐	☐			
86. Anxiety	☐	☐	☐			
87. Thoughts of suicide	☐	☐	☐			
88. Nervous breakdown	☐	☐	☐			
89. Psychiatric of psychological counseling	☐	☐	☐			
HEMATOLOGY						
90. Anemia	☐	☐	☐			
91. Blood clotting deficiency	☐	☐	☐			
92. Enlarged or swollen lymph nodes	☐	☐	☐			
93. Previous blood transfusion	☐	☐	☐			
DERMATOLOGY						
94. Skin rash	☐	☐	☐			
95. Skin cancer	☐	☐	☐			
96. Shingles (herpes zoster)	☐	☐	☐			
97. Skin sores that won't heal	☐	☐	☐			
98. Unusual moles	☐	☐	☐			
99. Mouth sores that won't heal	☐	☐	☐			
100. Other skin problems	☐	☐	☐			

ALLERGIES AND IMMUNIZATIONS

	Yes	No	Don't know
101. Do you have any allergy problems?	☐	☐	☐
102. Do you have hay fever symptoms?	☐	☐	☐
103. Do you have food allergies?	☐	☐	☐
104. When was your last tetanus shot? _____			
105. Do you have an annual flu vaccine?	☐	☐	☐
106. Have you had a pneumonia vaccine (Pneumovax)?	☐	☐	☐
107. Have you had a polio immunization series?	☐	☐	☐
108. Have you had recent immunizations?	☐	☐	☐
109. Have you had a tuberculosis skin test (PPD or Tine)?	☐	☐	☐
If yes, was it negative?	☐	☐	☐
Date of test? _____			

IV. REVIEW OF SYSTEMS (CONT.)

CURRENT MEDICATIONS: *(Include oral contraceptives, over-the-counter medications, vitamins, diet supplements, etc.)*

MEDICATION	DOSAGE	DOSES PER DAY	FOR WHAT?	WHEN STARTED?

DRUG ALLERGIES: Are you allergic to any medication? ☐ No ☐ Yes

If so, list medication and reaction to it.

MEDICATION	TYPE OF ALLERGIC REACTION	YEAR

GYNECOLOGICAL HISTORY
WOMEN ONLY:

1. When was your last menstrual period? _____
2. When was your last pelvic examination? _____
   Was the pelvic examination abnormal? ☐ Yes ☐ No
   Was the Pap Smear abnormal? ☐ Yes ☐ No
3. Are (or were) your menstrual periods abnormal? ☐ Yes ☐ No
4. Do you have urine loss when you cough, sneeze or laugh? ☐ Yes ☐ No
5. Have you had a hysterectomy? ☐ Yes ☐ No
6. Are you currently using a form of birth control? ☐ Yes ☐ No
   If yes, what kind? _____ _____
7. Number of pregnancies? _____
8. Number of live births? _____
9. Year of last pregnancy? _____
10. When was your last breast examination by a physician? _____
11. Do you examine your breasts for lumps each month? ☐ Yes ☐ No
12. Are you aware of any breast lumps? ☐ Yes ☐ No
13. Do you have any nipple discharge or bleeding? ☐ Yes ☐ No
14. Have you ever had breast x-rays (mammography) performed? ☐ No ☐ Yes
    If yes, date _____ ☐ Yes ☐ No
    Was it abnormal? ☐ Yes ☐ No
15. Have you ever had a breast biopsy? ☐ Yes ☐ No
16. Have you had any other breast surgery?
    Type? _____

V. PAST MEDICAL HISTORY

A. SIGNIFICANT PAST ILLNESSES: Please list any other significant illnesses you had as a child or adult.

ILLNESS	YEAR(S)	

B. PAST SURGERY: Please list in chronological order any surgeries you have had. Include hospital *and* out-patient surgery.

TYPE OF SURGERY	YEAR	

C. INJURIES: Please list any significant injuries you have had.

TYPE OF INJURY	YEAR	

D. RADIATION TREATMENT: Please list any radiation treatment that you have received to your head, neck, skin or elsewhere. *(Do not include diagnostic studies.)*

AREA TREATED	YEAR	REASON FOR TREATMENT

E. DIAGNOSTIC STUDIES: Check which of the following diagnostic studies you have had in the past.

	TEST	YEAR
☐	ECG (Electrocardiogram)	
☐	Treadmill Stress Test	
☐	Ultrasound examination of the heart (Echocardiogram)	
☐	Heart catheterization (Dye test of heart vessels)	
☐	X-ray exam of stomach ("Upper GI Series")	
☐	X-ray exam of large intestine ("Barium Enema")	
☐	Proctoscopy or sigmoidoscopy (Examination of the lowest portion of the colon and rectum with a rigid tube)	
☐	Colonoscopy (Examination of the colon with a long flexible tube)	

VI. FAMILY MEDICAL HISTORY

PARENTS	AGE IF ALIVE	OR	AGE AT DEATH	SIGNIFICANT HEALTH PROBLEMS	IF DECEASED, CAUSE OF DEATH
FATHER					
MOTHER					

BROTHERS/SISTERS SEX	AGE IF ALIVE	OR	AGE AT DEATH	SIGNIFICANT HEALTH PROBLEMS	IF DECEASED, CAUSE OF DEATH

SPOUSE: NAME _____ AGE _____ HEALTH _____

CHILDREN SEX	AGE IF ALIVE	OR	AGE AT DEATH	SIGNIFICANT HEALTH PROBLEMS	IF DECEASED, CAUSE OF DEATH

FAMILY ILLNESSES: Have your parents, grandparents, sisters or brothers, aunts or uncles, or your children developed any of the following? *Exclude* cousins, relatives by marriage or adoption, and half relatives. *(Please check appropriate boxes.)*

	FAMILY RELATION
☐ Heart attacks, coronary bypass, angioplasty or angina under age 50 *(circle problem)*	
☐ Heart attacks, coronary bypass, angioplasty or angina age 50-65 *(circle problem)*	
☐ Strokes under age 50	
☐ Strokes age 50-65	
☐ Other heart disease	
☐ High blood pressure	
☐ Sudden unexplained death	
☐ High cholesterol or triglycerides	
☐ Diabetes	
☐ Thyroid disease	
☐ Osteoporosis	
☐ Obesity	
☐ Colon polyps	
☐ Lung Cancer	
☐ Colon Cancer	
☐ Breast Cancer	
☐ Other Cancer	

Please indicate any death or serious illness, of immediate family members in the past year: _____

VII. PERSONAL HABITS

A. TOBACCO:

1. Do you currently use tobacco? ☐ Yes ☐ No
   (If not, go to question 2.)
   a. If you smoke cigarettes now, how many per day? _____
      What year did you start? 19_____
   b. If you smoke cigars now, how many per day? _____
      What year did you start? 19_____
   c. If you smoke a pipe now, how many pipefuls per day? _____
      What year did you start? 19_____
   d. If you use "smokeless" tobacco now, how often? _____
      What year did you start? 19_____

2. Have you used any of the following in the past, but do not use them now?
   ☐ Yes ☐ No
   (If not, go to the next section.)

a. Cigarettes	How many per day? _____	
	What year did you start?	19_____
	What year did you Stop?	19_____
b. Cigars	How many per day? _____	
	What year did you start?	19_____
	What year did you stop?	19_____
c. Pipe	How many per day? _____	
	What year did you start?	19_____
	What year did you stop?	19_____
d. "Smokeless" Tobacco	How many times per day? _____	
	What year did you start?	19_____
	What year did you stop?	19_____

3. Do you live with people who smoke? ☐ Yes ☐ No

4. Did your parents smoke when you were growing up? Father ☐ Yes ☐ No
   Mother ☐ Yes ☐ No

B. ALCOHOL:

1. Do you drink alcoholic beverages? ☐ Yes ☐ No
   If yes, how many drinks *per week*?
   Beer (12 oz.) _____
   Wine (5 oz. glass) _____
   Hard Liquor (1.5 oz.) _____

2. Do you now have or have you ever had problems with excessive alcohol use? ☐ Yes ☐ No
3. If you drink alcoholic beverages ....
   a. Have you ever felt you ought to cut down on your drinking? ☐ Yes ☐ No
   b. Have people annoyed you by criticizing your drinking? ☐ Yes ☐ No
   c. Have you ever felt bad or guilty about your drinking? ☐ Yes ☐ No
   d. Have you ever had a drink first thing in the morning to steady your nerves or to get rid of a hangover? ☐ Yes ☐ No
   e. Has your drinking ever affected your job or ability to work ☐ Yes ☐ No
   f. Have you ever been arrested for driving while intoxicated or under the influence of alcohol? ☐ Yes ☐ No

VII. PERSONAL HABITS (CONT.)

C. WEIGHT:

1. What is your current weight? _____ pounds
2. What do you consider a good weight for yourself? _____ pounds
3. What was your highest weight after age 18 (excluding pregnancy)? _____ pounds
   At what age? _____
4. What was your lowest weight after age 18? _____ pounds
   At what age? _____
5. What was your weight at age 21? _____ pounds
6. Weight loss history: How many times *in your life* would you estimate you have lost the number of pounds shown below?

	5 lbs.	10 lbs.	20 lbs.	30 lbs.	50 lbs.	80 lbs.	100 lbs.
Number of Times							

D. DIET:

1. Some people have to watch what they eat all the time to control their weight, others eat all they want and their weight is fine, and others have to eat more than they want to keep their weight up. What is your case?

☐ 1	☐ 2	☐ 3	☐ 4	☐ 5
Eat Much Less Than I Want	Eat Somewhat Less Than I Want	Eat Just What I Want	Eat Somewhat More Than I Want	Eat Much More Than I Want

2. How often are you dieting (eating less than you would like)?

☐ 1	☐ 2	☐ 3	☐ 4	☐ 5
Never	Rarely	Sometimes	Often	Always

3. Are you currently on any diet or dietary restriction?
   ☐ Yes ☐ No
   If yes, check the appropriate description.

   ☐ Low Fat
   ☐ Low Cholesterol
   ☐ Low Sodium (salt)
   ☐ Low Calorie (wt. reduction)
   ☐ High Fiber
   ☐ Other *(Specify)*: _____

   Who (if anyone) supervises or sponsors the program? _____
   How long have you been following the diet? _____

VII. PERSONAL HABITS (CONT.)

E. MEALS:

1. In an average week, how many meals do you eat? _____

2. How many of those meals include the following?

_____ Poultry or fish (fried)
_____ Beef
_____ Pork (include bacon & ham)
_____ Luncheon meat (include hot dogs)
_____ Cheese (include pizza)
_____ Fried foods (include chips)
_____ Pie, cake, ice cream, or cookies
_____ Eggs
(Number of eggs per week = _____)
_____ Butter

_____ Poultry or fish (baked or broiled)
_____ Fruit
_____ Vegetables
_____ Low-fat yogurt
_____ Ice milk or sherbet
_____ Grains (bread, rice, pasta, corn)
_____ Legumes (beans, lentils, etc.)
_____ Breakfast cereal
(Specify Types: _____)
_____
_____

3. In an average week, how many "snacks" do you eat? _____
Circle those that you eat most frequently:

chips peanuts pretzels candy bars candy ice cream
cookies popcorn fruit

F. Beverages: How many of each of the following do you consume in an average week?

Water (glass) ..... _____

Coffee: (cups) Regular ..... _____
Decaffeinated ..... _____

Tea: (cups) Regular ..... _____
Decaffeinated or Herbal ..... _____

Soft Drinks (12 oz.) ..... _____
With Caffeine ..... _____
With Sugar ..... _____

Whole Milk (glass) ..... _____
Low-Fat (2%) Milk (glasses) ..... _____
Skim (½-1%) Milk (glasses) ..... _____

VIII. EXERCISE

A. AEROBIC ACTIVITIES:

1. Are you currently involved in a routine of regular exercise *(moderate continuous exertion for at least 15-20 minutes duration at least 3 days a week?)* ☐ Yes ☐ No

2. How long have you been exercising regularly? _____Yrs. _____ Mos. _____ Wks.

3. For the last three months, which of the following activities have you performed regularly? (Please check **YES** for all that apply and **NO** if you do not perform the activity; provide an estimate of the amount of activity for all marked **YES**. Please be as complete as possible.)

Walking
☐ Yes
☐ No
How many workouts per week? _____
How many miles (or fractions) per workout? _____
Average duration of workout? _____ (minutes)
Average time per mile? _____

Jogging or Running
(outdoors or on track)
☐ Yes
☐ No
How many workouts per week? _____
How many miles per workout? _____
Average duration of workout? _____ (minutes)
Average time per mile? _____

Treadmill
(walking or running)
☐ Yes
☐ No
How many workouts per week? _____
Average duration of workout? _____ (minutes)
Speed? _____ Grade? _____ % Heart Rate? _____

Bicycling
(outdoors)
☐ Yes
☐ No
How many workouts per week? _____
How many miles per workout? _____
Average duration of workout? _____ (minutes)
Average time per mile? _____

Stationary Cycling
☐ Yes
☐ No
Type of stationary cycle? _____
How many workouts per week? _____
Average duration of workout? _____ (minutes)
Heart rate during exercise? _____

Swimming Laps
☐ Yes
☐ No
How many workouts per week? _____
How many miles per workout? _____
(100 yds. = 1/16 mi.)
Average duration of workout? _____ (minutes)
How many months per year? _____

Aerobic Dance
or
Floor Exercises
☐ Yes
☐ No
How many workouts per week? _____
Average duration of workout? _____ (minutes)
Heart rate during exercise? _____

Vigorous Racquet Sports
(e.g. Racquetball, Singles Tennis)
☐ Yes
☐ No
How many workouts per week? _____
Average duration of workout? _____ (minutes)

Other Vigorous Sports
Or Exercise
(e.g. Basketball or Soccer) Please specify:
_____
☐ Yes
☐ No
How many workouts per week? _____
Average duration of workout? _____ (minutes)

4. Do you follow the Aerobics points exercise program? ☐ Yes ☐ No
   If yes, about, how many Aerobics points do you earn per week? _____
   How many Aerobics points did you earn last week? _____

5. What time of day do you usually exercise? _____

VIII. EXERCISE HISTORY (CONT.)

6. How do you rate the physical activity that you are now getting compared to others in your same age and sex? Think about both your leisure and work activities. *(Please check your response.)*

- ☐ A. EXTREMELY INACTIVE
- ☐ B. INACTIVE
- ☐ C. SOMEWHAT INACTIVE
- ☐ D. ABOUT AVERAGE
- ☐ E. SOMEWHAT ACTIVE
- ☐ F. ACTIVE
- ☐ G. EXTREMELY ACTIVE

7. Compared to a year ago, how much regular exercise do you currently get?

- ☐ A. MUCH LESS
- ☐ B. SOMEWHAT LESS
- ☐ C. ABOUT THE SAME
- ☐ D. SOMEWHAT MORE
- ☐ E. MUCH MORE

8. Have you continuously followed your program?

☐ No Approximately how many times have you stopped for at least six months? _____
What is the longest period that you were continuously active? _____
What is the longest period that you were not on any program? _____
Since you started an exercise program, how many total years have you been regularly active? _____

☐ Yes

9. What exercise equipment, if any, do you own? *(Check those that apply)*

- ☐ Running Shoes
- ☐ Stationary Cycle
- ☐ Bicycle
- ☐ Rowing Machine
- ☐ Treadmill
- ☐ Cross Country Ski Simulator
- ☐ Other *(Specify)* _____

10. To what exercise facilities do you have easy access? *(Check those that apply)*

- ☐ Fitness Club
- ☐ Jogging Path
- ☐ Bicycle Path
- ☐ Aerobic Exercise Class
- ☐ Swimming Lap Pool
- ☐ Suitable Area For Walking

11. If you are not exercising regularly, what exercise activities might be of most interest to you? *(List in order of decreasing preference.)*

a. _____

b. _____

c. _____

B. MUSCLE STRENGTHENING ACTIVITIES

1. Are you currently involved in a muscle strengthening program? ☐ Yes ☐ No

If yes, what type? *(Check those that apply)*

- ☐ Calisthenics
- ☐ Free Weights
- ☐ Weight Training Machines
- ☐ Other: *(Specify)* _____

How many days per week do you do these exercises? _____
Average duration of workout? _____
How long have you been involved in this routine? _____

EXERCISE HISTORY (CONT.)

C. FLEXIBILITY ACTIVITIES

1. Are you currently involved in exercises to maintain or improve your joint flexibility? ☐ Yes ☐ No

   If yes, what type?

   ☐ Stretching
   ☐ Calisthenics
   ☐ Exercise Class

   How many days per week? _____
   Average duration of exercise? _____
   How long have you been involved in this routine? _____

2. Can you touch your toes without bending your knees? ☐ Yes ☐ No

D. EXERCISE SAFETY

1. Do you warm up prior to exercise? ☐ Yes ☐ No
2. Do you cool down slowly after exercise? ☐ Yes ☐ No
3. Do you know how to take your pulse? ☐ Yes ☐ No
4. Do you monitor your heart rate when exercising? ☐ Yes ☐ No
5. If you bicycle, do you wear a protective helmet? ☐ Yes ☐ No
6. If you exercise outdoors at night, do you use reflective gear or a light? ☐ Yes ☐ No

IX. STRESS AND EMOTIONAL FACTORS

1. How stressful do you consider your home life to be?

☐ Low ☐ Moderate ☐ High

2. How stressful do you consider your occupation to be?

☐ Low ☐ Moderate ☐ High

3. How would you classify yourself on the following tension and anxiety scale?

☐ 1	☐ 2	☐ 3	☐ 4	☐ 5
No Tension Very Relaxed	Slight Tension	Moderate Tension	High Tension	Very Tense "High-Strung"

4. What is your greatest source of worry or concern *at present?*

☐ Marriage ☐ Family ☐ Job ☐ Finances ☐ Health ☐ Other

5. How well do you feel you manage your stress?

☐ Not well most of the time
☐ Fairly well most of the time
☐ Very well most of the time

6. Do stress and tension in your life seem to cause you to have any of the following symptoms? *(Check all that apply)*

☐ General irritability or impatience
☐ Headache
☐ Abdominal discomfort
☐ Sleeplessness
☐ Other *(Specify)*

7. How often do you use medications, alcohol, or other substances to help you relieve stress and relax?

☐ Frequently (several times a week)
☐ Occasionaly (once or twice a week)
☐ Seldom (once or twice a month)
☐ never

8. Please rate your general emotional outlook on life on the following scale:

☐ 1	☐ 2	☐ 3	☐ 4	☐ 5
Often very Depressed	Generally Sad	Happy & Sad Equal Amount	Generally Happy	Usually Very Happy And Optimistic

9. How do you rate overall health?

☐ 1	☐ 2	☐ 3	☐ 4
Poor	Fair	Good	Excellent

10. How do you spend your leisure time?

_____

_____

_____

X. LIFESTYLE RISK EVALUATION

HOME

	Yes	No
1. Do you live in a dwelling without a smoke alarm?	☐	☐
2. Do you live in a dwelling without a fire extinguisher?	☐	☐
3. Do any household members use alcohol to excess or use illicit drugs?	☐	☐

AUTO

4. Do you drive a sports car or a subcompact car?	☐	☐
5. Do you ever drive or ride in a car without using seat belts?	☐	☐
If yes, what percent of the time without seat belts? _____		
6. Does your commute to work involve freeway traffic?	☐	☐
7. Does anger occasionally affect your driving?	☐	☐
8. Do you ever pick up hitchhikers?	☐	☐
9. Have you received any speeding tickets or warnings in the past year?	☐	☐
10. Do you ever drive after drinking alcohol?	☐	☐

LIFESTYLE

11. Do you have any hobbies that involve high risk such as race cars, motorcycles, ATV's, small planes, parachuting, or scuba diving?	☐	☐
12. Do you attend happy hour more than once per week?	☐	☐
13. Do you use any "recreational" drugs?	☐	☐

XI. CURRENT LEVELS OF SATISFACTION

Please indicate your level of satisfaction in each of the following areas by checking the appropriate box. Then indicate whether you intend to make any changes in those areas during the next 12 months.

	Generally satisfied	Generally dissatisfied	Intend to make changes
1. My diet	☐	☐	☐
2. My weight	☐	☐	☐
3. My physical condition and stamina	☐	☐	☐
4. My use of cigarettes	☐	☐	☐
5. My use of alcohol or recreational drugs	☐	☐	☐
6. My blood pressure	☐	☐	☐
7. My handling of tension and stress	☐	☐	☐
8. My job	☐	☐	☐
9. My family life	☐	☐	☐
10. My general health and lifestyle	☐	☐	☐

DISCLOSURE AND CONSENT
MEDICAL AND SURGICAL PROCEDURES

TO THE PATIENT: You have the right, as a patient, to be informed about your condition and the risks and hazards involved in the recommended surgical, medical, or diagnostic procedure to be used. You may then make the decision whether or not to undergo the procedure. This disclosure is not meant to scare or alarm you; it is simply an effort to make you better informed so you may give or withhold your consent to the procedure.

CONSENT

I voluntarily consent and authorize Dr. _____, as my Cooper Clinic physician, and such technical assistants and other health care providers as he may deem necessary, to administer an exercise stress test.

Just as there may be risks and hazards in continuing any present condition without treatment, there may also be risks and hazards related to the performance of this procedure. I realize that common to many surgical, medical, and diagnostic procedures is the potential for infection, blood clots in veins and lungs, hemorrhage, allergic reaction, and even death. In addition, I realize that the following risks and hazards may also occur in connection with this particular procedure: disorders of heart rhythm, fall in blood pressure, heart attack.

For the purpose of aiding medical research, I permit the Institute for Aerobics Research and the Cooper Clinic to accumulate and analyze data relating to my evaluation and to contact me for follow-up information regarding my health status in the future.

I have been given an opportunity to ask questions about the procedure and the risks and hazards involved, and I believe that I have sufficient information to give this informed consent. I certify this form is clear to me, that I have read it or have had it read to me, and that I understand its contents.

SIGNATURE: _____
PATIENT OR LEGALLY RESPONSIBLE PERSON

DATE: _____ **TIME:** _____

WITNESS: _____

Are you an Activity Center Member? YES ☐
NO ☐

(NOTE TO TECHNICIAN: IF YES IS CHECKED, YOU MUST COMPLETE THE AAC MEMBERSHIP MEDICAL FORM AND SEND TO AAC BUSINESS OFFICE.)

Appendix B

Blood Analysis Form

COOPER CLINIC
12200 PRESTON ROAD
DALLAS, TX 75230

PATIENT NAME:
AGE: 51 Y
SEX: F
PHYSICIAN: COOPER
SAMPLE DRAWN: 01/21/91

COMMENT: SMAC II

LAB No:
PATIENT ID:

TRAY: 13
CUP: 1
DIL: 1

FLAG	RESULT	UNITS	TEST	DESIRABLE RANGE.	
H	236	MG/DL	CHOLESTEROL	130.	200.
H	95	MG/DL	HDL CHOLESTEROL	55.	85.
	128	mg/dL	LDL CHOLESTEROL	65.	130.
	13	mg/dL	VLDL CHOLESTEROL	0.	23.
	2.5	.	CHOL:HDL RATIO	0.0	3.6
	66	MG/DL	TRIG 8-D	40.	115.
	88	MG/DL	GLUCOSE	67.	110.
	4.7	mg/dL	URIC ACID	2.4	8.2
	5.3	mmol/L	POTASSIUM	3.5	5.3
	141	mmol/L	SODIUM	137.	145.
	8	U/L	GGT	8.	44.
	10.5	mg/dL	CALCIUM	8.8	11.1
	3.8	mg/dL	PHOSPHORUS	2.2	4.5
	75	U/L	ALP (ALK PHOS)	41.	133.
	12	mg/dL	UREA NITROGEN	7.	19.
	0.9	mg/dL	CREATININE	0.8	1.4
	7.9	g/dL	TOTAL PROTEIN	6.2	
	4.3	g/dL	ALBUMIN	3.4	
	0.6	mg/dL	TOTAL BILIRUBIN	0.2	
	17	U/L	AST (SGOT)	6.	38.
	127	U/L	LD (LDH-L)	98.	230.
	58	U/L	CK (CPK)	0.	225.

Appendix C

Treadmill Stress Test Record Sheet

Previous Test: Time ☐☐ ☐☐ MAX HR ☐☐☐

COOPER CLINIC

Exercise Test

MIN	HR.	BLOOD PRESSURE	COMMENTS
1	☐☐☐	☐☐☐ / ☐☐☐	
2	☐☐☐	☐☐☐ / ☐☐☐	
3	☐☐☐	☐☐☐ / ☐☐☐	
4	☐☐☐	☐☐☐ / ☐☐☐	
5	☐☐☐	☐☐☐ / ☐☐☐	
6	☐☐☐	☐☐☐ / ☐☐☐	
7	☐☐☐	☐☐☐ / ☐☐☐	
8	☐☐☐	☐☐☐ / ☐☐☐	
9	☐☐☐	☐☐☐ / ☐☐☐	
10	☐☐☐	☐☐☐ / ☐☐☐	
11	☐☐☐	☐☐☐ / ☐☐☐	
12	☐☐☐	☐☐☐ / ☐☐☐	
13	☐☐☐	☐☐☐ / ☐☐☐	
14	☐☐☐	☐☐☐ / ☐☐☐	
15	☐☐☐	☐☐☐ / ☐☐☐	
16	☐☐☐	☐☐☐ / ☐☐☐	
17	☐☐☐	☐☐☐ / ☐☐☐	
18	☐☐☐	☐☐☐ / ☐☐☐	
19	☐☐☐	☐☐☐ / ☐☐☐	
20	☐☐☐	☐☐☐ / ☐☐☐	
21	☐☐☐	☐☐☐ / ☐☐☐	
22	☐☐☐	☐☐☐ / ☐☐☐	
23	☐☐☐	☐☐☐ / ☐☐☐	
24	☐☐☐	☐☐☐ / ☐☐☐	
25	☐☐☐	☐☐☐ / ☐☐☐	
26	☐☐☐	☐☐☐ / ☐☐☐	
27	☐☐☐	☐☐☐ / ☐☐☐	
28	☐☐☐	☐☐☐ / ☐☐☐	
29	☐☐☐	☐☐☐ / ☐☐☐	
30	☐☐☐	☐☐☐ / ☐☐☐	
31	☐☐☐	☐☐☐ / ☐☐☐	
32	☐☐☐	☐☐☐ / ☐☐☐	
33	☐☐☐	☐☐☐ / ☐☐☐	
34	☐☐☐	☐☐☐ / ☐☐☐	
35	☐☐☐	☐☐☐ / ☐☐☐	
36	☐☐☐	☐☐☐ / ☐☐☐	

Age ☐☐☐ Sex ☐ Ht. _____ Wt. _____

R-HR ☐☐☐ PMHR ☐☐☐ 85% PMHR ☐☐☐

BLOOD PRESSURE

	Right Arm	Left Arm
Supine	☐☐☐ / ☐☐☐	☐☐☐ / ☐☐☐
Sitting	☐☐☐ / ☐☐☐	☐☐☐ / ☐☐☐
Standing	☐☐☐ / ☐☐☐	☐☐☐ / ☐☐☐

☐ Asymptomatic
☐ C Pcne
☐ CHD

☐ Max
☐ Submax

Test Protocol
☐ Balke
☐ Modified Bike
☐ Other _____

Recovery
☐ Walk down 5 min.
☐ Lie down 7 min.
☐ Other

Max. HR ☐☐☐
Max BP ☐☐☐ ☐☐☐
Duration ☐☐ ☐☐

Impressions
☐ Normal
☐ Equivocal
☐ Abnormal
☐ Inconclusive

RECOVERY

MIN	HR.	BLOOD PRESSURE	COMMENTS
1	☐☐☐	☐☐☐ / ☐☐☐	
2	☐☐☐	☐☐☐ / ☐☐☐	
3	☐☐☐	☐☐☐ / ☐☐☐	
4	☐☐☐	☐☐☐ / ☐☐☐	
5	☐☐☐	☐☐☐ / ☐☐☐	
6	☐☐☐	☐☐☐ / ☐☐☐	
7	☐☐☐	☐☐☐ / ☐☐☐	
8	☐☐☐	☐☐☐ / ☐☐☐	
9	☐☐☐	☐☐☐ / ☐☐☐	
10	☐☐☐	☐☐☐ / ☐☐☐	
11	☐☐☐	☐☐☐ / ☐☐☐	
12	☐☐☐	☐☐☐ / ☐☐☐	
13	☐☐☐	☐☐☐ / ☐☐☐	
14	☐☐☐	☐☐☐ / ☐☐☐	
15	☐☐☐	☐☐☐ / ☐☐☐	

COMMENTS:

Reason for Stopping (check one reason)

☐ Leg weakness or fatigue
☐ Volitional exhaustion or general fatigue
☐ Dyspnea
☐ Lightheadness, Dizziness
☐ Hypertension
☐ Subject's desire to stop
☐ Chest pain
☐ ECG changes (specify) _____
☐ Hypotension
☐ Failure of Monitoring equip.
☐ Physician's discretion
☐ Other (specify) _____

CC FORM H

Date _____ Name _____ Pt. No. ☐☐☐☐☐☐

Appendix D

Body Composition Analysis Form

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~~~~~~~~~~~~~~~~~~~~~~~~~~~~~~~~~~~~~~~~~~~~~~~~~~~~~~~~~~~~~~~~~~~~~~~~~~~~~~~~
               B O D Y   C O M P O S I T I O N   A N A L Y S I S
Patient Name                                          # 0123567
          Sex = F      Age =          Height =          Weight = 130.00
~~~~~~~~~~~~~~ ACCEPTABLE BODY FAT 18 %  IDEAL BODY FAT 16 %     ~~~~~~~~~~~~~~~
WATER METHOD

   H2O Temp = 34.0    C&B Weight =   7.10    Total Weight =   8.60    Suit = N
   Residual Volume .    1.70   Density ..............    1.05    % Fat  20.54
   Fat Pounds ......   26.70   Lean Pounds ..........  103.30
   Acceptable Weight  125.98   Acceptable Lbs to Lose    4.02
   Goal Weight .....  122.98   Goal Pounds to Lose ..    7.02

~~~~~~~~~~~~~~~~~~~~~~~~~~~~~~~~~~~~~~~~~~~~~~~~~~~~~~~~~~~~~~~~~~~~~~~~~~~~~~~~
SKINFOLD METHOD
   Chest = 12.00        Axilla = 12.00       Triceps = 14.00      Back = 12.00
   Abdomen = 20.00      Hip = 17.00          Thigh = 18.00        Waist =    .00
   Gluteal =    .00
                               Density ..............    1.05    % Fat  20.70
    Fat Pounds ......   26.91  Lean Pounds ..........  103.09
    Acceptable Weight  125.72  Acceptable Lbs to Lose    4.28
    Goal Weight .....  122.73  Goal Pounds to Lose ..    7.27

~~~~~~~~~~~~~~~~~~~~~~~~~~~~~~~~~~~~~~~~         Average of methods    20.62 %
```

Appendix E

In-Residence Program 13-Day Schedule

13-DAY AEROBICS PROGRAM FOR TOTAL WELL-BEING

Sample Schedule

All classes will meet in the **Carter Room** (CR) except where otherwise noted.

Sunday

3:00 - 10:00 PM	Arrival and Registration ..... Guest Lodge (GL)

Monday

7:00 - 3:00 PM	Physical Assessments and **Exams** ..... Cooper Clinic (CC)
1:00 - 4:00 PM	LUNCH (for those with completed exams) ..... Center Table (CT)
4:00 - 4:30 PM	**Program Orientation** ..... Ava Bursau, M.S.
4:30 - 5:30 PM	**"Wellness, Fitness and Total Well-Being"** ..... Roy E. Vartabedian, D.H.Sc.
5:30 - 6:00 PM	Group Picture/Individual Schedule
6:15 - 7:00 PM	DINNER ..... Cookery (C)
7:30 - 9:30 PM	Evening Walk/Whirlpool/Sauna/Massage

Tuesday

7:00 - 8:00 AM	Morning Walk ..... (CR)
8:00 - 8:30 AM	BREAKFAST ..... (C)
8:30 - 9:00 AM	**"Fitness is Good Business"** ..... Kenneth H. Cooper, M.D.
9:00 - 10:30 AM	**"Exercise Facts and Prescription"** ..... Syd Teague, M.Ed.
10:30- 12:00 PM	Activity Center Tour and Equipment Demo ..... Activity Center (AC)
12:00 - 1:00 PM	Individual Schedule
1:00 - 2:00 PM	LUNCH ..... (C)
2:00 - 3:00 PM	**"Low Back Care"** ..... Amy Jones, M.Ed.
3:00 - 3:30 PM	Exercise Review ..... (CR)
3:30 - 4:00 PM	Exercise Session ..... (AC)
4:00 - 5:00 PM	Individual Schedule
5:00 - 6:00 PM	**"Nutrition: The Facts"** ..... Patty Kirk, R.D.
6:00 - 7:00 PM	DINNER ..... (C)
7:30 - 9:30 PM	Evening Walk/Whirlpool/Sauna/Massage

Wednesday

7:00 - 8:00 AM	Morning Walk/Stretch ..... (AC)
8:00 - 9:00 AM	BREAKFAST ..... (C)
9:00 - 10:15 AM	**"Maintaining Behavior Change"** ..... Pam Walker, Ph.D.
10:15- 10:30 AM	Relaxation Technique
10:30- 11:30 AM	Exercise Session ..... (AC)
11:30- 12:00 PM	Individual Schedule
12:00- 1:00 PM	**"Osteoporosis"** ..... Sydney Bonnick, M.D.
1:00 - 2:00 PM	LUNCH ..... (C)
2:00 - 3:00 PM	Equipment Demonstration ..... (CR)
3:00 - 4:00 PM	Exercise Session ..... (AC)
4:00 - 5:00 PM	Individual Schedule
5:00 - 6:00 PM	**"Coronary Disease and Risk Factors"** ..... Boyd Lyles, M.D.
6:00 - 7:00 PM	DINNER ..... (C)
7:30 - 9:30 PM	Evening Walk/Whirlpool/Sauna/Massage

Thursday

7:00 - 8:00 AM	Morning Walk/Stretch.....(AC)
8:00 - 9:00 AM	BREAKFAST.....(C)
9:00 - 10:30 AM	**"Concepts and Effects of Stress"**.....Stephen Harvill
10:30 -11:00 AM	Exercise Review.....(CR)
11:00 -12:00 PM	Exercise Session.....(AC)
12:00 - 1:00 PM	Individual Schedule
1:00 - 2:00 PM	LUNCH
2:00 - 3:00 PM	Equipment Demonstration.....(CR)
3:00 - 4:00 PM	Individual Schedule
4:00 - 5:00 PM	**"Fats & Cholesterol"**.....Georgia Kostas, M.P.H., R.D.
5:00 - 7:00 PM	COOKING SCHOOL and DINNER.....Kathleen Duran,R.D.(C)
7:30 - 9:30 PM	Evening Walk/Whirlpool/Sauna/Massage

Friday

6:45 - 8:30 AM	BREAKFAST..... Le Peep Restaurant
9:00 - 9:30 AM	**"You Can Do It"**.....Suity Cureton
9:30 - 10:30 AM	**"Health Consequences of Obesity"**.....Jean P. Wisner, R.D.
10:30 -11:30 AM	Exercise Session.....(AC)
11:30 -12:00 PM	Individual Schedule
12:00 - 1:00 PM	**"Aerobic Walking"**..... Casey Meyers
1:00 - 2:00 PM	LUNCH.....(C)
2:00 - 3:00 PM	**Walking Clinic**.....(AC)
3:00 - 6:00 PM	Individual Schedule
3:30 - 4:30 PM	**"Goal Setting"** (7-day only).....Syd Teague, M.Ed.
6:15 - 7:00 PM	DINNER.....(C)
7:30 - 9:30 PM	Evening Walk/Whirlpool/Sauna/Massage

Saturday

7:45 - 8:00 AM	Weigh-In.....(CC)
8:00 - 9:00 AM	BREAKFAST.....(C)
9:00 - 10:00 AM	**"Strength and Muscular Conditioning"**.....Eric Samaniego, M.S.
10:00 -11:30 AM	Exercise Session or "Low Moves" exercise class (10:15 AM).....(AC)
11:30 -12:00 PM	Individual Schedule
12:00 - 1:00 PM	LUNCH.....(C)
1:00 - 4:30 PM	Afternoon Activity.....Galleria
4:30 - 6:00 PM	**"Healthy Dining"**..... Kathleen Duran, R.D. Video: "Low Fat Dining"
6:00 - 8:00 PM	DINNER..... Bay Street Restaurant
8:00 - 9:30 PM	Evening Walk/Whirlpool/Sauna/Massage

Sunday

8:00 - 9:00 AM	BREAKFAST.....(C)
9:00 - 10:00 AM	Outdoor Morning Walk (on your own)
10:00- 11:00 AM	**Breadmaking Class**..... Kathleen Duran, R.D. (C)
11:00- 12:00 PM	Individual Schedule
12:00- 2:00 PM	LUNCH..... Jasmine Restaurant
2:00 - 3:30 PM	Individual Schedule
3:30 - 4:00 PM	Introduction to Aerobic Dancing.....(AC)
4:00 - 5:30 PM	"Low Moves" (4:00 PM) or Bench Aerobics (5:15 PM).....(AC)
6:00 - 7:00 PM	DINNER.....(C)
7:30 - 9:30 PM	Evening Walk/Jacuzzi (Guest Lodge)

Monday

7:00 - 8:00 AM	Morning Walk/Stretch.....(AC)
8:00 - 9:00 AM	BREAKFAST.....(C)
9:00 - 10:30 AM	**"Coronary Prone Behavior"**.....Pam Walker, Ph.D.
10:30 - 11:00 AM	Exercise Review.....(AC)
11:00 - 12:00 PM	Exercise Session.....(AC)
12:00 - 1:00 PM	Individual Schedule
1:00 - 2:00 PM	LUNCH.....(C)
2:00 - 2:30 PM	**Aerobic Points/E-Log**.....Molly Burns, B.S.
2:30 - 4:30 PM	Weight Training Demo and Exercise Session.....(AC)
4:30 - 5:00 PM	Individual Schedule
5:00 - 6:00 PM	**Cooking Video**.....Cookery
6:15 - 7:00 PM	DINNER.....(C)
7:30 - 9:30 PM	Evening Walk/Whirlpool/Sauna/Massage

Tuesday

7:00 - 8:00 AM	Morning Walk/Stretch (on your own)
8:00 - 9:00 AM	BREAKFAST.....(C)
9:00 - 10:00 AM	**"Success Tips"**.....Syd Teague, M.Ed.
10:00 - 10:15 AM	Relaxation Technique.....(CR)
10:30 - 11:30 AM	Exercise Session.....(AC)
11:30 - 12:00 PM	Individual Schedule
12:00 - 1:00 PM	**"Understanding High Blood Pressure"**.....John Duncan, Ph.D.
1:00 - 2:00 PM	LUNCH.....(C)
2:00 - 5:00 PM	**Group Outing**.....White Rock Lake
5:00 - 6:00 PM	**"Oral & Dental Health"**.....Jim Gallman, D.D.S.
6:15 - 7:00 PM	DINNER.....(C)
7:30 - 9:30 PM	Evening Walk/Whirlpool/Sauna/Massage

Wednesday

7:00 - 8:00 AM	Morning Walk/Stretch (on your own)
8:00 - 9:00 AM	BREAKFAST.....(C)
9:00 - 10:30 AM	**"Shopping Smart"**.....Kathleen Duran, R.D.
10:30 - 11:00 AM	Exercise Review.....(CR)
11:00 - 12:00 PM	Exercise Session.....(AC)
12:00 - 1:00 PM	**"Fitness on the Road"**.....John Poteet, Ph.D.
1:00 - 2:00 PM	LUNCH.....(C)
2:00 - 4:00 PM	**Afternoon Group Activity**.....Molly Burns, B.S.
4:00 - 6:00 PM	Individual Schedule
6:15 - 7:00 PM	DINNER.....(C)
7:30 - 8:00 PM	Evening Walk/Whirlpool/Sauna/Massage

Thursday

7:00 - 8:00 AM	Morning Walk/Stretch (on your own)
8:00 - 9:00 AM	BREAKFAST.....(C)
9:00 - 10:30 AM	**"Nutripoints"**.....Roy Vartabedian, D.H.Sc.
10:30 - 11:30 AM	Exercise Session.....(AC)
11:30 - 12:00 PM	Individual Schedule
12:00 - 12:30 PM	LUNCH.....(C)
1:00 - 5:00 PM	OMNI Theater/Museum of Science and Natural History.....Ft. Worth
6:00 - 7:00 PM	**DINNER**.....Rodolfo's Restaurant
7:30 - 9:30 PM	Evening Walk/Whirlpool/Sauna/Massage

Friday

6:30 - 7:00 AM	Blood Test, Weigh-In & Circumference Measurements..... (CC)
7:00 - 8:00 AM	Morning Walk
8:00 - 9:00 AM	BREAKFAST..... (C)
9:00 - 10:30 AM	**"Goal Setting"**.....**Syd Teague, M.Ed.**
10:30 - 11:30 AM	Exercise Session.....(AC)
11:30 - 1:00 PM	Individual Schedule
1:00 - 2:00 PM	LUNCH..... (C)
2:30 - 3:30 PM	**Calisthenics/Dynaband Workout**.....**Molly Burns, B.S.**
3:30 - 5:00 PM	Individual Schedule
5:00 - 6:00 PM	**Graduation/Film: Psychology of Winning**.....(CR)
6:15 - 7:00 PM	DINNER..... (C)
7:30 - 9:30 PM	Evening Walk/Whirlpool/Sauna/Massage

Saturday

7:00 - 9:00 AM	BREAKFAST..... (CT)
9:00 - 1:00 PM	Exercise Session and Individual Schedule
1:00 - 2:00 PM	Check Out..... **Guest Lodge**

Appendix F

Informed Consent Form

INFORMED CONSENT AND AUTHORIZATION FOR IN-RESIDENCE PROGRAM PARTICIPATION AND IMPERSONAL RELEASE OF MEDICAL/HEALTH RECORDS FOR SCIENTIFIC INVESTIGATION

I, the undersigned, hereby voluntarily give my informed consent and authorization to the Aerobics Center for me to engage in a series of health and medical evaluations and to participate in a lifestyle modification/health enhancement ("wellness") program.

I understand that the wellness program in which I will participate will be led by trained health promotion specialists including exercise leaders, health educators, and nutritionists. There is very little risk associated with the nutrition and stress management aspects of the program. There may be some slight risk associated with the exercise program, including muscle soreness or injury; there is a chance that some cardiovascular problem could develop, and in very rare instances a "heart attack" may occur. I will be responsible for following the instructors recommendations regarding safety procedures during the program, which will minimize these risks. Excessive exercise in hot humid conditions can lead to heat injury such as heat exhaustion or heat stroke. This danger can be reduced by altering my exercise program during hot and humid weather, by exercising in climate controlled environments, by drinking plenty of water, and by recognizing the early signs of heat injury.

These risks are minimized by careful medical screening prior to entering the program and through observations by trained exercise leaders. Exercise leaders are trained in first aid and emergency care, and such assistance will be rendered in the event of an emergency. If further diagnostic or therapeutic care is needed, I understand that it is my personal financial responsibility.

I also hereby voluntarily give consent and authorization to inclusion of data concerning my health and fitness status, which are obtained by personnel of the Aerobics Center, in a research data bank which will be used to investigate the relationships between various aspects of lifestyle and health (especially risk of heart disease). These data are derived from questionnaires, medical examinations, and lab testing. Included are medical history, family history of heart disease, smoking history, body composition, blood pressure, blood, diet, psychosocial, demographic, and physical activity data.

I understand that these data used for scientific research will receive only impersonal statistical treatment with my right of privacy protected. None of my data will be revealed in individualized form to another person without my prior written consent. Further, I recognize that I can discontinue participation at any time without penalty of any kind.

Further, I have read the foregoing carefully and I understand its content. Any questions which may have occurred to me concerning this informed consent have been answered to my satisfaction.

Finally, I release and discharge the Aerobics Center, its divisions, officers, agents, staff, faculty, physicians, technicians, and any others connected therewith from all claims and/or damages whatsoever that I or my representatives may have arising from, or incident to this program.

NAME:_____

ADDRESS:_____

_____

SIGNATURE:_____

DATE:_____

WITNESS:_____