# A COMPARISON OF CHANGE IN SELECTED - <br> PHYSIOLOGICAL VARIABLES BETWEEN <br> PARTICIPANTS AND NONPARTICIPANTS <br> OF HEALTHY LIFESTYLE CLASSES 

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## CHAPTER I

## INTRODUCTION

At the turn of the century, medical care was much less expensive, but also much less effective. Doctors could diagnose and explain maladies, but were often helpless to treat them. Medical intervention became more far-reaching and appropriate once antibiotics and vaccines were developed in the mid-twentieth century [Thomas,1983]. Treatment of existing disease, using new techniques, drugs, and technology, became the primary health care goal. However, such progressive methods carry their own financial burden.

By 1984, health care costs for the nation exceeded $\$ 390$ billion, more than $11 \%$ of the gross national product, or more than one billion dollars a day [Sagan, 1987]. In 1988, corporate executives estimated health benefit costs for their employees were rising 14 to 20 percent annually [Teitelman,1988]. Obviously, the American government, private industry, and taxpayers cannot continue to support this rate of growth in medical expenses. The question is, what is the most effective way to help reduce these health care costs?

Reducing incidence of the most prevalent diseases in this country: heart disease, cancer, and stroke, could be a key factor in lowering medical expenses. According to Healthy People: The Surgeon General's Report on Health Promotion and Disease Prevention [USDHEW, 1979], 75\% of all deaths in the United States are due to these three degenerative diseases, with heart disease leading as the primary cause of mortality in America today [Taylor, Ureda, and Denham, 1982].

The leading causes of death have become known as "diseases of lifestyle" or even "diseases of choice" [Parsons, 1987], for numerous studies have linked cancer, stroke, and heart disease to daily activities common to many Americans [Hubert, et al,1983; Center for Health Promotion and Education,1987; Kolata and Marx,1976; USDHHS,1983]. Habitual cigarette smoking, patterns of physical activity or inactivity, dietary practices, and responses to stress are instances of lifestyle components.

These habits and practices tend to influence certain physiological characteristics of individuals, such as body weight, total serum cholesterol, and blood pressure. These "health status indices" are measurable traits influenced by changes in behaviors [USDHHS,1983]. Both the behavioral patterns and physiological characteristics have been termed "risk indicators" or "risk factors", having
consequences for future health-related risk [Goetz, et al,1987].

Can these physiological risk factors, then, be reduced by modifying an individual's lifestyle patterns? And, if so, what is the best method to produce lifestyle change?

Healthy People: The Surgeon General's Report on Health Promotion and Disease Prevention reported that the health gains of the twentieth century resulted mostly from development and implementation of preventive measures, including "changes in personal lifestyle" [USDHEW, 1979]. This report set specific health objectives for the nation to reach by 1990 , and a follow-up report, Promoting Health/Preventing Disease, set forth "implementation plans" to meet these objectives [USDHHS, 1983]. The plans were aimed at improving health status, reducing risk factors, and increasing public and professional awareness of major health problems. The main thrust was on providing education and information through electronic and print media, consumer education programs and guides, brochures, and health fairs.

Information on modification of risk factors is of little use if the target population does not act on this knowledge. Will people who attend health education/health promotion programs make a change in their lives based on increased awareness and improved information alone, or are
other methods required? Behavioral modification, scare tactics, competition, and incentive/reward systems have all been used in attempts to affect lifestyle change, in conjunction with educational efforts [Klesges,et al,1987; Meyer, et al,1980; Farquhar,et al,1977; Simons-Morton, et al,1987; Martin and Dubbert,1984]. The search continues for the technique that optimally combines efficacy, effectiveness, low cost, limited time, and long-lasting effects.

OSU Wellness Program

Beginning in mid-1987, the Oklahoma State University Wellness Program began a series of "healthy lifestyle" classes for OSU faculty/staff, which attempted to promote lifestyle modification through improved nutrition and weight reduction, proper exercise, cholesterol reduction, smoking cessation, and stress management [Cole,et al,1986].

The approach was strictly educational in scope, presenting information to participants on potential risks and consequences of "unhealthy" behaviors and on alternative behaviors which have been associated with decreased incidence of disease. Goals of the presentations were to help participants understand and act on this information.

Were these goals achieved? Physiological variables
were measured before and after the educational session and used as indices of behavior change. Changes in systolic and diastolic blood pressure (SBP and DBP), body weight (WT), and total serum cholesterol (TC) from pretest to posttest were compared in participants and nonparticipants of the healthy lifestyle classes.

Statement of the Problem

The purpose of this study is to determine whether there was a significant change, from pretest to posttest, in the physiological variables of systalic blood pressure, diastolic blood pressure, total serum cholesterol, and body weight in those persons who attended at least one two-hour session of healthy lifestyle classes compared to those who did not attend such classes.

## Hypotheses

The following hypotheses will be tested at the 0.050 level of significance:

1. There will be no significant differences between class participants and nonparticipants in variation of systolic blood pressure from pretest to posttest.
2. There will be no significant differences between class participants and nonparticipants in variation of diastolic blood pressure from pretest to posttest.
3. There will be no significant differences between class participants and nonparticipants in variation of body weight from pretest to posttest.
4. There will be no significant differences between class participants and nonparticipants in variation of total serum cholesterol between pretest and posttest.

## Delimitations of the Study

1. The study was delimited to Oklahoma State University faculty members and professional, clerical, and technical personnel (hereafter referred to as faculty/staff) who voluntarily underwent two separate health risk assessments (pretest and posttest).
2. It was further delimited to those faculty/staff who did not receive exercise tolerance testing with its concomitant individualized counseling sessions on risk factor modification.

## Limitations

1. Pretest and posttest attendance was strictly voluntary, leading to attrition of nearly one-fourth of those who attended the pretest. Thus, the sample was not randomly selected from the population.
2. Class attendance was also strictly voluntary, making random assignment impracticable. It is unknown whether
class participants were those at high risk for
cardiovascular disease.
3. Elapsed time between pretest and posttest varied among subjects.
4. Most of those at highest risk due to $T C>239 \mathrm{mg} / \mathrm{dL}$ and/or $\mathrm{DBP}>89 \mathrm{~mm} \mathrm{Hg}$, who might be expected to change more than those at less risk, were removed from the study because they, were referred for, and received, exercise tolerance tests under the guidelines of the OSU Wellness Program.

## Assumptions

The following assumptions were made:

1. All subjects who attended the pretest and posttest followed instructions on abstaining from food, caffeine, and vigorous exercise prior to serum cholesterol testing. 2. Body weight measurements reflected fat weight changes and not changes in lean muscle tissue as the recommended exercise was aerobic in nature.
2. All members of the study were equally as likely to be exposed to electronic or print media information dealing with cardiac risk factors between the pretest and the posttest.

## Conceptual

Arteriosclerosig. Arterial disease that leads to thickening and hardening of arteries of any size [USDHHS, 1983].

Atherosclerosis. A specific form of arteriosclerosis in which lipid, connective tissue fibers, and various blood components accumulate in the intima of large elastic arteries and medium-sized muscular arteries [USDHHS,1983].

Cardiovascular disease. All diseases affecting the cardiovascular system including coronary heart disease, atherosclerosis, high blood pressure, stroke, rheumatic fever, and rheumatic heart disease [Thomas, 1975].

Cholesterol. A sterol widely distributed in animal tissues, that is both produced by the body and ingested by eating certain foods of animal origin [Christian, J.L. and Greger, J.[.,1988].

Coronary heart disease. Atherosclerotic lesions affecting one or more of the coronary arteries [Thomas, 1975].

Fatty acid. Units attached to glycerol in glycerides, which give different glycerides their characteristics. Fatty acids differ from each other most significantly in chain length and degree of saturation
[Christian, J.L. and Greger, J.L., 1988].
Health education. Any combination of learning experiences designed to predispose, enable, and reinforce voluntary adaptations of behavior conducive to well-being [Green, L.W. and Lewis, F.M.,1986].

Health promotion. Any combination of health education and related organizational, economic, and environmental supports for behavior conducive to well-being [Green, L.W. and Lewis, F.M., 1986].

Lifestyle. Complex patterns of behavior that are shaped by socialization and demands of the cultural and social environment [Yoder, et al,1985].

Polyunsaturated fats (fatty acid). A fat that is generally liquid at room temperature; it has two or more double bonds between carbon atoms [Christian, J.L. and Greger, J.L.].

Risk factor. Life habits or body characteristics that may increase a person's chances of developing a serious illness [Reed, 1984].

Saturated fat (fatty acid). A fat that is a solid at room temperature; it has no double bonds between carbon atoms [Christian, J.L. and Greger, J.L.].

Stress. A perceived negative reaction to psychosocial and environmental stimuli [Schwartz,1980].

## Functional

Hypercholesterolemia. High blood cholesterol levels that include borderline-high blood cholesterol (200 to 239 $\mathrm{mg} / \mathrm{dL}$ ) and high blood cholesterol ( $\geq 240 \mathrm{mg} / \mathrm{dL}$ ).

Hypertension. High blood pressure levels that include borderline-high blood pressure ( $\geq 140 / 90 \mathrm{mmHg}$ ) and high blood pressure ( $>160 / 95 \mathrm{mmHg}$ ).
Description of Instruments

Sphygmomanometer. An instrument for measuring the force of the arterial blood pressure.

Vision (tm) Automated Blood Analyzer. A fullyautomated spectrophotometric blood analyzer used to measure different components of the blood.

## CHAPTER II

## A SELECTED REVIEW OF THE LITERATURE

This study looks at the relationship between attendance at healthy lifestyle classes and observed changes in the physiological characteristics of systolic blood pressure (SBP), diastolic blood pressure (DBP), body weight (WT), and total serum cholesterol level (TC). Healthy lifestyle classes attempted to increase participants' awareness of risk factors for cardiovascular disease (CVD), especially cigarette smoking, hypertension, total cholesterol, obesity, physical inactivity, and stress, and to present detailed knowledge of ways to reduce these risk factors through changing behaviors common to high risk lifestyles.

Results of several studies [Belloc,1973; Belloc and Breslow, 1972] demonstrated that the individual health practices of cigarette smoking, maintenance of desirable weight, alcohol intake, hours of sleep, regularity of meals, and physical activity were each inversely-related to ageadjusted mortality rates, independent of income level and physical health status. Wiley and Camacho [Wiley and Camacho,1980] further reported that certain aspects of lifestyle, including smoking, physical exercise, weight, alcohol intake, and hours of sleep, were associated with
future health status. Such associations between lifestyle or daily habits and health and mortality provide a basis for the wellness approach to health: individual practices can frequently influence one's future health status.

Since the late $1960^{\prime}$ s, U.S. mortality rates from coronary heart disease (CHD) have decreased nearly $30 \%$ with a corresponding decline in incidence of first major coronary events. An editorial in the New England Journal of Medicine [Stamler, 1985] attributes this decline to improvements in lifestyle and related risk factors. Lifestyle improvements included greater degree of blood pressure control, substantially lower intake of foods high in cholesterol and saturated fats, and reduction in proportion of cigarette smokers in the adult American population.

The Surgeon General's 1979 report [USDHEW, 1979] divided health risks into the "major risk categories" of inherited biological, environmental, and behavioral risks. While heredity may increase risks for certain diseases through predisposition or susceptibility, this report states that major risk factors for common chronic diseases are environmental and behavioral and therefore amenable to change.

A number of studies attempt to assess the impact of interventions on health behaviors or health indicators. Some interventions are specific to one risk factor, while
others are multifactorial.

## Specific Risk Factor Intervention

## Cigarette Smoking

Cigarette smoking is considered the most important of the three major risk factors. Although its association with CVD is similar to hypertension or hyperlipidemia, the number of Americans who smoke, 55 million in 1983 , is considerably larger than those with the other two major risk factors [USDHHS, 1983].

Cigarette smoking has been strongly associated with CVD morbidity and mortality since the Surgeon General's report in 1964. As reported in The Health Consequences of Gmoking: Cardiovascular Disease [USDHHS, 1983], numerous studies concur. Smoker's death rates from CHD are 70\% greater than those of nonsmokers.

A dose-response relationship exists, in that higher numbers of cigarettes per day, total years of smoking, and degree of inhalation are positively associated with increased CVD risk, while CVD risk is inversely related to age of initiation of smoking [USDHFS, 1983].

The mechanism by which cigarette smoking produces CVD is not yet well-understood. Smoking does seem to lead to development of atherosclerotic lesions and to clinical
manifestations of atherosclerotic vascular disease. Nicotine and carbon monoxide in cigarette smoke are associated with the production of an imbalance between myocardial oxygen supply and demand, enhanced platelet aggregation, and lowered threshold for ventricular fibrillation. Lesions in coronary arteries are consistently greater in heavy smokers than in nonsmokers, possibly due to damage to arterial walls from smoke particulates and tissue hypoxia [USDHHS,1983].

Smoking cessation results in diminished CHD mortality. Those who quit smoking reduce their relative risk for death from CHD by almost half compared with those who continue to smoke. Epidemiological studies show that former cigarette smokers reduce CHD death risk to the level of nonsmokers over a 10 to 15 year period after stopping smoking [USDHHS,1983].

Cigarette smoking is a learned behavior which can be changed. Education is needed to increase Americans' awareness of the problem, as evidenced by 1979 and 1980 studies which showed that 40 to $50 \%$ of the American public were not aware of the link between smoking and heart disease [USDHHS, 1983].

Worksite interventions to promote smoking cessation have included competitions among work groups and relapse
prevention training. A study of smokers in North Dakota and Oregon reported a significantly higher cessation rate in worksites competing to quit over those with no competition. At six-month follow-up, however, recidivism had abolished this difference. Relapse prevention techniques were not seen to be effective in reducing smoking after the program concluded [Klesges, et al,1987].

Methods designed to reduce or eliminate smoking behavior were compared in a summary of 89 studies. Aversive conditioning success ranged from $22 \%$ to $67 \%$ for abstention but had high drop out rates. Smoking reduction rates were high, however. Drug therapy data were scanty, but results indicated very low success ( $8 \%$ to $17 \%$ ) of abstention. Hypnosis seemed to give very good abstention results, ranging from $15 \%$ to $88 \%$, but treatment populations were small and high dropout rates were experienced. Behavior modification showed $17 \%$ to $70 \%$ success in reducing smoking behaviors.

Education and group support reported abstention success rates from $14 \%$ to $78 \%$ up to one year, and smoking reduction rates from $16 \%$ to $81 \%$. This technique included programs such as the five-day plans with health education supplemented by discussion groups and supportive patient interaction [Hunt and Bespalec,1973].

## Hypertension

Blood pressures greater than 140/90 are considered abnormal [USDHEW, 1979], and blood pressures consistently over 160/95 are considered hypertensive. About 35 million Americans have pressures above 160/95.

High blood pressure is one of the best predictors of CVD [Kolata and Marx, 1976]. The Framingham study [Castelli,et al,1986] showed that men aged 45 to 64 with blood pressures above 160/95 had 2 to 3 times the CHD incidence rate of those with pressures below 140/90. Strokes are at least three times as frequent in the hypertensive group [Castelli,et al,1986]. Individuals with borderline hypertension (>140/90 but (160/95) also experienced "substantially increased incidences" of CVD compared to those with normal blood pressures. Other studies provide evidence that CVD risk increases as blood pressure rises, even for blood pressures within the normal range [Kolata and Marx,1976].

High blood pressure does not produce symptoms unless it is severe. It does require the heart to work harder in order to pump blood against higher pressures, which may gradually lead to congestive heart failure. Hypertension may lead to atherosclerotic placque developed by damaging arterial walls either mechanically or through hypoxia.

Ninety percent of incidents of hypertension are termed "essential", whose cause is unknown. It is known that adrenal gland stimulation or sympathetic nervous system norepinephrine release help increase blood volume and constrict arterioles, both of which lead to higher blood pressures [Marx,1976].

Less than one-third of hypertensive persons in the U.S. have their condition under adequate control. Drugs are an effective technique to control high blood pressure, but problems with side effects may limit their use in many [USDHEW,1979]. Weight reduction and dietary modification that includes decreased salt intake is frequently part of blood pressure therapy [Marx,1976]. According to Gibbons, in adult white women who were well educated and in the middle to upper socioeconomic strata, the evidence that exercise lowers blood pressure independently of changes in weight and salt intake is not overwhelming, especially in normotensive persons [Gibbons,et al,1983]. Aerobic exercise may, however, reduce blood pressure in moderate hypertensives, perhaps by reducing sympathetic nervous system activity and catecholamine levels. In addition, the degree of physical fitness in both men and women is strongly associated with subsequent incidence of hypertension [Blair,et al,1984].

Evidence exists that visceral vasoconstriction and
muscular vasodilation in response to psychosocial stimuli (stress) leads to heightened blood pressure. Higher resting heart rates and cardiac outputs from sympathetic nervous system response to stress are associated with transient increases in systolic blood pressure as well [Blair,et al,1984]. It is not proven whether chronic stress can elevate blood pressures over the long-term. Exposure to chronic noise, crowding, or stressful conditioning in animals has repeatedly produced permanent or prolonged elevations of blood pressure in animals, however [Apte, 1980]. Stressful wartime conditions in soldiers has also been associated with chronic hypertension [USDHEW, 1979].

Interventions that work to reduce blood pressure in the hypertensive population have been associated with decreased CVD morbidity and mortality. A study among black urban poor individuals in 1975 demonstrated a hypertension-related mortality rate $53.2 \%$ less after 5 years among those receiving health education interventions than among a control group who received only standard medical care [Morisky,1985]. Interventions included a counseling session, a home visit to encourage family support, and invitations to small-group sessions. Family support appeared to be the single most important factor affecting weight control and compliance with blood pressure treatment.

A review of occupational hypertension control programs to detect and control blood pressure showed that screening and health education alone were not effective in improving blood pressure control in workers. Programs that were effective had elements of high frequency and continuity of contact between patient and health care provider, greater intensity for each contact, and active patient participation in self-monitoring and goal-setting for behavioral change. Social support both at home and at the worksite was also important in maintaining blood pressure control [Alderman, et al,1980].

## Hypercholesterolemia

High total serum cholesterol levels are considered one of the three major risk factors for CHD. Borderline cholesterol levels are those between 200 and $240 \mathrm{mg} / \mathrm{dL}$, and levels above or equal to $240 \mathrm{mg} / \mathrm{dL}$ are termed hypercholesterolemia. Approximately 15 to $20 \%$ of American adults have hypercholesterolemia [Grundy, 1986].

The Framingham study identified high TC levels as being related to the development of coronary heart disease in both men and women 49 years old or older [Castelli, et al, 1986]. The Seven Countries Study also showed a good correlation between TC and age-adjusted 5-year incidence rates of CHD [Oliver, 1981].

Other lipid levels, particularly high density lipoprotein cholesterol levels, have also been strongly associated with cardiac disease risk [Castelli,et al,1986], but will not be addressed here because they were not measured by the OSU study due to the cost of such widespread screening.

Data from major studies such as Framingham, the Pooling Project, and an Israeli study all suggest that CHD rates are relatively constant for cholesterol levels up to 200 or 220 $\mathrm{mg} / \mathrm{dL}$, but above this "threshold" range, the risk for CHD increases as cholesterol concentrations rise [Grundy,1986]. MRFIT data also suggested that at higher concentrations of TC, "CHD mortality was compounded" [MRFIT Research Group, 1982]. Results from that study observed that TC levels of 250 doubled the CHD risk compared to $T C$ levels of 200, and TC levels of $300 \mathrm{mg} / \mathrm{dL}$ doubled the risk once more.

Such results indicate to some researchers that the severity of coronary atherosclerosis rises as TC levels rise. Once approximately $60 \%$ of the arterial surface is covered by atherosclerotic placque, however, a "critical level" is reached which substantially increases CHD risk even more rapidly [Grundy,1986].

This lipid-heart hypothesis is still controversial, however, in that the relationship between total serum cholesterol and heart disease does not hold well for women
and those over 65 [Smith and Kummerow, 1987].
The Lipid Research Clinics Coronary Primary Prevention Trial (LRC-CPPT) and others indicate that lowering TC can reduce the incidence and mortality rates of CHD [Lipid Research Clinics Program, 1984]. The LRC-CPPT modified TC levels with diet and drugs, and found that, on average, a reduction of TC by $1 \%$ was associated with a $2 \%$ reduction in CHD incidence. The "diet-heart hypothesis" [Queen, 1987], assumes that TC can be lowered either through lifestyle changes including a low-fat, low-cholesterol diet, or through these lifestyle changes in combination with a cholesterol-lowering drug.

There is evidence that decreasing $T C$ by $50 \mathrm{mg} / \mathrm{dL}$ should cut CHD risk in half, if the initial cholesterol level was $250 \mathrm{mg} / \mathrm{dL}$ or more. If the majority of Americans reduce their TC below $200 \mathrm{mg} / \mathrm{dL}$, the overall CHD rate should diminish by 30 to 50\% [Grundy,1986].

It is thought that hypercholesterolemia raises the membrane cholesterol content of endothelial cells, producing cell damage and allowing low-density lipoprotein cholesterol to accumulate in the intima of arteries. Smooth muscle growth is stimulated, and smooth muscle cells along with accumulated macrophages and cholesterol ester become foam cells to produce an atherosclerotic lesion. Calcium may eventually accumulate to produce hardening of this portion
of the artery. Such lesions tend to produce stenosis of the involved artery and/or thrombus formation [Smith and Kummerow,1987; USDHHS,1983].

High cholesterol levels in the blood develop in response to an interaction of habitual diet and genetic influence [Oliver,1987]. Perhaps 40 to $45 \%$ of any individual TC level is genetically determined [Queen, 1987]. Total cholesterol levels and cholesterol metabolism involve a complex balance of dietary and endogenous cholesterol absorption, synthesis, catabolism, and excretion. Only approximately $10 \%$ of serum cholesterol is derived from dietary cholesterol intake. High excess cholesterol intake, however, may overwhelm the balance system and result in increased serum cholesterol levels [Oliver, 1987].

Dietary fat, specifically the ratio of polyunsaturated to saturated fatty acids (P:S ratio), has "profound effects" on serum cholesterol levels in humans [McNamara, 1987]. The Seven Countries Study showed that the percent of energy derived from saturated fatty acids was highly correlated with TC and with CHD incidence [Oliver, 1987].

A number of trials indicate that substantial changes in TC and lipoproteins can be obtained by changing the fatty acid, dietary cholesterol, and dietary fiber content of the diet [Kromhout and Arntzenius, 1987]. The Leiden Intervention Trial, for example, obtained serum lipid
lowering solely by dietary measures involving a P:S ratio of 2 and very low dietary cholesterol intake. Results also suggested a reduction in the rate of coronary lesion growth. Dietary manipulation reportedly can lower TC by 10 to $15 \%$ [Smith and Kummerow, 1987].

Reduction of cholesterol-rich diets in animals are associated with regression of atherosclerotic lesion size, especially the size of the necrotic lipid-rich core and the extracellular lipid in the fibrous cap. A few limited studies have confirmed this in humans as well [Oliver,1987].

Several classes of drugs have been shown to effectively reduce TC (bile-acid sequestrants and cholesterol-metabolism inhibitors, for example) and reduce mortality rates from CHD [Grundy,1986]. The problem of short term side effects and possible long term side effects, as well as high cost, makes drug therapy a less-than-ideal solution [Grundy, 1986]. Lifestyle modification through diet appears to be effective in reducing CHD risk from $T C$, but with the attendant problems of motivating lifestyle change.

The nationwide Cholesterol Education Campaign was kicked off in 1986, focusing on reduction of high total serum cholesterol. Its emphasis is on mass screening and referral of the high-risk individual to appropriate therapy [National Cholesterol Education Program, 1986].

There are few worksite or community-wide programs which
focus solely on cholesterol reduction. Most cholesterolreduction programs take place in the context of multifactorial attempts to change lifestyle, specifically combinations of proper diet and aerobic exercise.

## Obesity

In 1979, the Surgeon General's Report stated that 35\% of women and $5 \%$ of men between the ages of 45 and 64 with incomes below the poverty level, as well as $29 \%$ of women and $13 \%$ of men with incomes above this level, were considered obese [USDHEW,1979].

Twenty-six years of observation in the Framingham study indicated that in both men and women, the degree of obesity was a significant independent predictor of CVD incidence, especially among women [Hubert,et al,1983]. Furthermore, obesity was independently related to CVD, not solely in association with other risk factors. Premature onset of CVD, especially in men, was predisposed by obesity. For men under 50 years of age, the heaviest group experienced twice the risk of CHD compared with the leanest group. Recent studies have shown that the distribution of fat deposits may be a better predictor of $C V D$ and death than the degree of adiposity, however [McNamara,1987].

The unique association of obesity to CVD risk may be due to plasma fibrinogen changes in obese people, which can
influence the course of ischemic heart disease.
Additionally, excess body weight heightens the cardiac work load and intravascular volume, and can alter glucose and lipid metabolism [Hubert,et al,1983; Messerli,1986].

Obesity tends to increase other risk factors as well. Excess body weight is a well-documented correlate of hypertension [Apte,1980; Stamler,1978]. Overweight adults exhibit high blood pressure 50 to $300 \%$ more often than those of normal weight or underweight. A number of studies summarized by Apte found that weight reduction was associated with decreases in blood pressure.

Obesity is associated with an increase in cholesterol synthesis as well, independent of the presence of hyperlipidemia. Body weight is directly related to the mass of the total cholesterol pool in the body, and perhaps increases TC in genetically-susceptible individuals [McNamara,1987]. McNamara reports evidence that total calories ingested and body weight have "the largest single effect" on cholesterol synthesis and metabolism, in addition to an increased incidence of hyperlipidemia. In his view, the most effective intervention to reduce hyperlipidemia is weight reduction and maintenance of an ideal body weight, even more so than reducing cholesterol or saturated fat intake or other dietary changes.

Excess weight is frequently associated with increased
incidence of diabetes mellitus. Diabetics have more incidence and severity of atherosclerosis, twice as many heart attacks, and about twice as many strokes as nondiabetics of the same age [USDHEW,1979].

Weight change was positively and significantly associated with CVD risk in both sexes, according to the Framingham study [Castelli,et al,1986]. The duration of obesity was also important in risk, so weight loss may reduce the incidence of CVD.

Weight loss interventions overall have had little longterm success. The problems of attrition and motivation may seem overwhelming in this aspect of lifestyle modification. Combinations of low-calorie diets, exercise and medication are unsuccessful in the majority of cases. Behavioral treatment has shown somewhat greater success in changing eating habits, with an emphasis on environmental/behavioral causes of overweight rather than heredity. Behavior modification frequently involves self-monitoring, stimulus control, and contingency management techniques [Foreyt,et al,1980; USDHEW, 1979]. Some behavior modification treatments have shown losses to be maintained at least one year after formal treatment ceased.

Many worksite interventions use a multifactorial health promotion approach that includes weight control and exercise. Organizations such as ARCO, General Foods, and

Metropolitan Life rely on weight control education sessions, from a few hours to six month participation. They have generally noted weight loss at the end of the program but long-term weight maintenance was unsuccessful. Greater weight loss was maintained after one year when programs used incentives (Campbell Soup Co., Dupont Co., Union Carbide Corp.); group competition (John Hancock Life Insurance Co., Lockheed); and environmental changes (Johnson \& Johnson, Mattel Toys) [Glanz and Orr,1986]. A project to "Lighten Up" the community as part of the Pawtucket Heart Health Program used incentives, self-help kits, mass media, and worksite support, and reported an average weight loss of only three and a half pounds over a five week period [Nelson, et al,1987]. Weight control is highly sought after but among the most difficult to achieve of all risk factor intervention goals.

## Physical Inactivity

Numerous studies address the role of physical fitness in limiting CVD mortality. Paffenbarger's well-known studies found the risk of a first heart attack to be inversely related to energy expenditure (adult exercise) independent of other influences on heart attack risk. Low risk was related to physical activity involving at least $2000 \mathrm{kcal} / \mathrm{week}$. Men with lower levels of energy expenditure
had 50 to $70 \%$ higher rates of myocardial infarction.
Subjects of the studies were Harvard alumni, who presumably worked at mainly sedentary jobs and had higher socioeconomic levels [Paffenbarger, Wing, and Hyde, 1978; Paffenbarger, et al,1984].

Longshoremen, however, with much higher levels of energy expenditure on the job, showed a difference in ageadjusted coronary death rates between high activity and medium/low activity categories. Paffenbarger concluded that repeated bursts of high energy output give a protective effect against CHD mortality independent of the factors of cigarette smoking, high SBP, weight, diagnosed heart disease, and glucose metabolism. These workers had many different characteristics from the Harvard alumni, however [Paffenbarger and Hale, 1975].

A 1986 report covering nearly 17,000 Harvard alumni followed for 12 to 16 years found that death rates from all causes but primarily due to cardiovascular or respiratory causes declined steadily as energy expended on exercise or sports increased, independent of hypertension, smoking, high body weight or weight gains, or family history of premature death [Paffenbarger, et al,1986].

A study of leisure time physical activity in over 16,000 male, executive-level civil servants 40 to 64 years old found that physically active men had a CHD incidence
over the course of eight years approximately half the rate of those who did not participate in vigorous exercise. This finding was independent of other CHD risk factors. This study also pointed out the importance of intermittent peaks of high intensity energy output as opposed to simply high totals of physical activity [Morris,et al,1980].

In 1987 the Morbidity and Mortality Weekly Report reviewed the literature on physical activity associations with CHD. Of 47 comparisons selected under stringent guidelines, 32 (68\%) showed a statistically significant inverse relationship between physical activity and CHD. The association increased as the quality of the study methods and measures improved. The association was consistent for more than two-thirds of the studies. The strength of the association between physical activity and CHD was of similar magnitude as that for risk factors of high SBP, TC, and smoking (as reported in the Coronary Pooling Project). Even so, the prevalence of physical inactivity is more widespread in the U.S. (59\% of Americans do not exercise regularly 3 or more times/week for at least 20 minutes) than the prevalence of high blood pressure (36\%), high TC (25 to 40\%), and smoking (30\%). This study concluded that physical inactivity was therefore a greater risk factor for CHD [Center for Health Promotion and Education, 1987].

A Cooper Clinic study of 3,000 men of average age 44.5
years showed a direct inverse relationship between levels of cardiorespiratory fitness and variables related to high CHD risk. Physical fitness was related to the CHD risk factors of high TC, blood pressure, and triglycerides independently of age, weight, and percent bodyfat. There was a substantial difference between people with high versus low levels of fitness [Cooper, et al,1976].

The mechanism of regular vigorous physical activity in reduced CVD incidence and mortality is unexplained. It has been hypothesized that cardiovascular conditioning prevents or diminishes ventricular ectopic activity leading to ventricular fibrillation and death. The training effect seems to improve the efficiency of cardiac action, increase cardiac output, slow the heart, and regularize the rhythm [Paffenbarger and Hale, 1975; Haskell and Blair, 1980]. High exercise levels may increase fibrinolytic activity, reducing coronary thrombosis deaths. Increased collateral circulation or greater luminal area of coronary arteries may result from prolonged high energy output. In addition, heavy energy expenditures help reduce the influence of highrisk factors of CHD including hypertension, hyperlipidemia, tachycardia, and obesity [Stromme, 1984].

Unlike other risk factor interventions, evaluations of programs that address physical inactivity usually focus on various effects of reducing inactivity rather than on the
extent or amount of activity produced by the intervention. That is, exercise efficacy is more likely to be determined by measuring weight loss,' blood pressure reduction, or diminished stress rather than measuring total amount of exercise performed or added.

Interventions are common both within communities and the workplace in attempts to increase physical activity and thus reduce coronary risk. Exercise programs at KimberlyClark Corp. involve medical testing, treadmill testing, and exercise prescriptions [Dedmon, 1979]. Sentry Corp. offers recreational activity and fitness programs to employees and their families, as well as employee screening and stress testing [Cook,Walden, and Johnson, 1979]. Johnson \& Johnson's Live for Life program reports that $40 \%$ of all employees participate in exercise programs, which are just a portion of the total health promotion program [Wilbur,1983]. It is estimated that at least 300 firms in the U.S. have employee fitness programs of some kind [Martin,1978].

Southern New England Telephone's Reach Out For Health program aims at motivating employees by providing information and teaching skills. Health screenings aided awareness, counseling improved participants' knowledge, and health education and fitness courses provided behavior modification to maintain behavior change. The program includes incentives, promotional activities, self-help
tools, and environmental changes [Kaplan and BurchMinakan,1986]. Data on results of the program were not provided.

Studies in Minneapolis and among faculty and staff at several northeastern universities involved sedentary men 45 to 59 years old who had high blood pressure and high TC levels. These subjects participated in supervised exercise programs for one hour three times/week for about 18 months. Results showed statistically significant differences between randomly-chosen exercise and control groups in work performance and attitudes, weight reduction, more positive feelings about health, greater ability to deal with stress and tension, and greater physical activity in their lifestyles [Heinzelmann and Bagley, 1970].

A study on sedentary manager-executives with high CHI risk factors in Finland involved 18 months of vigorous, submaximal preventive exercise for three sessions/week. There was a $72 \%$ participation rate throughout the program, but this dropped sharply at the end of the program. This is one of the few studies reporting participation rate in a long-term training program [Oja, et al,1974].

A 14 -week study reported in 1982 screened and evaluated subjects for fitness, then prescribed an aerobic exercise program at least three days/week. No significant differences were found for weight, bodyfat percentage, or
resting DBP, but significant differences were found in resting heart rate, resting $\mathrm{SBP}, \mathrm{TC}$ and total triglycerides, and VO2 max. A unique finding was that none of the variables with the exception of $V O 2$ max varied significantly in response to frequency of exercise sessions attended per week [Pauly, et al, 1982].

A ten-week aerobic exercise program for educators highlighted exercise and fitness, providing subjects with only one supervised exercise session per week along with weekly educational sessions. Physical fitness increased, suggesting that all exercise need not be done in a highly supervised and structured setting [Blair,et al, 1984].

Characteristics of successful adult fitness programs seem to be: the promotion of cardiovascular health, the aerobic nature of the exercise, frequency of at least three times/week, duration of 20 to 30 minutes/session, and an intensity that increases metabolism at least $50 \%$ of maximum (increasing heart rate at least $60 \%$ of maximum).

Information from past programs indicates that only $20 \%$ of the target population will enroll, and within six months, about $50 \%$ of enrollees will have dropped out [Pate and Blair,1983].

## Stress

Stress may result from inadequate coping responses to
major life changes or when a person's needs, values, and expectations are not met [Levi,1979; Shields,1983]. An individual's responses to stress appear to be related to CHD incidence and mortality [JSDHEW,1979; Schwartz,1980; Shields,1984; Weiman,1983].

This relationship is curvilinear, with disease/risk more frequent in those who perceive themselves as underutilized or overworked. A Swedish study stated that $42 \%$ of all academically-trained professionals report distress at work [Weiman,1983].

Mechanisms linking stress levels and CVD may be biochemical, in that raising stress levels appears to increase blood levels of catecholamines, mediated through the sympatho-adrenomedullary system. Continued catecholamine production may result in permanent pathogenic structural changes in predisposed individuals. Animal (including primate) studies indicate heart rate and blood pressure may be sustained at high levels in response to $a$ stressful stimulus, eventually leading to continuous high cardiac output, visceral vasoconstriction, and muscular vasodilation [Levi,1979; Schwartz,1980]. Selye found that stress may provoke degenerative myocardial changes, apparently as a result of neuroendocrine reactions [USDHEW, 1979].

Individual responses to stress are moderated by
cognitive, personality, and behavioral characteristics of the individual, which may be hereditary or learned. These factors interact with environmental characteristics both at work and at home [Schwartz, 1980].

It has been suggested that a behavioral approach to modifying stress responses may in turn influence biochemical and physical responses. Behavioral approaches include improving interpersonal communication and eliciting "relaxation responses" [Schwartz, 1980]. In addition, stress reduction can promote other healthy behaviors, such as increasing physical activity, improving the diet, and reducing cigarette smoking [Apte,1980].

## Interactions of Risk Factors

Each risk factor acts in concert with and upon other risk factors to influence the onset of CVD. For instance, Grundy reports that as levels of TC increase, the age at onset of CVD decreases. Adding one other risk factor, such as smoking, further reduces that individual's onset age. The cumulative effect of three risk factors in the same individual leads to high risk of premature CVD manifestations and possibly death [Grundy, 1986]. A study by Paffenbarger indicated that the presence of low physical activity, cigarette smoking, or a history of hypertension was accompanied by a $50 \%$ increase in risk, and the presence
of two of these characteristics nearly tripled the risk [Paffenbarger,Wing, and Hyde,1978].

Not only do high risk behaviors and characteristics multiply risk, but the presence of one high risk behavior may actually lead to other risky behaviors or traits. For instance, the carbon monoxide from cigarette smoke may raise total cholesterol levels, and nicotine has been associated with increases in blood pressure [USDHHS,1983]. Although cigarette smoking is inversely related to obesity, it is associated to some extent with physical activity levels. In several studies, the least active men had higher smoking rates. In smoking cessation programs, exercise may serve as a substitute for smoking [Gibbons,1983; Leon,et al,1987].

Reduced participation in physical activity is also associated with obesity [Pender and Pender, 1986], and obesity has been shown to be a factor in high BP and high TC levels [Hubert,et al,1983]. Exercise is associated with reduced SBP levels but only with reduced DBP if the individual is hypertensive. Exercise may be slightly associated with weight loss, though the relationship is not consistent. Total cholesterol levels have been seen to reduce in correlation with physical activity [Pauly, et al,1982].

Physical activity has frequently shown a relationship with reduction of anxiety and improved self-concept measures
[Pauly, et al,1982]. Responses to stress may include increased cigarette smoking or weight gain, to which the body may react with higher blood pressure levels [Apte, 1980]. Stress may also diminish the individual's ability to participate in healthy behaviors such as regular exercise and reduced smoking [Schwartz,1980].
[See Table 1].

## Multifactorial Risk Factor Interventions

Since risk factors interact with and add to other risk factors, it makes sense to address them as a group. The OSU healthy lifestyle classes (smoking cessation, weight control and nutrition, cholesterol reduction, proper exercise, and stress management) spoke to each "unhealthy" behavior by providing information for the participant to change the behavior individually and in conjunction with other healthy behaviors.

This multiple risk factor approach does bring up the question of whether attempting multiple behavior changes simultaneously allows reinforcement of risk factor reduction or, conversely, decreases the ability of the participant to respond to and effectively change any behaviors [Fihn, 1987].

The approach may be necessary, however. A combined reduction of both risk factors of high BP and high TC was found to be required to achieve any substantial favorable

TABLE I
INTERACTIONS OF CVD RISK FACTORS

| Risk Factor | Smoking | BP | TC | Obesity | Exercise | Stress |
| :---: | :---: | :---: | :---: | :---: | :---: | :---: |
| Smoking | xxx | D | D | I | I | D |
| BP | D | xxx | 0 | D | I | D |
| TC | D | 0 | xxx | D | I | D |
| Obesity | I | D | D | xxx | I | 0 |
| Exercise | I | I | I | I | xxx | I |
| Stress | D | D | D | 0 | I | xxx |

$D=$ direct relationship
I = inverse relationship
$0=$ no demonstrated relationship
[Based on information from Apte,1980; Gibbons, et al, 1983;
Hubert,et al,1983; Leon,et al,1987; Pauly, et al,1982;
Pender and Pender,1986; Schwartz,1980; USDHHS,1983].
effect on CVD and CHD morbidity and mortality. Antihypertensive treatment was found to be ineffective in people who smoked and had high TC [Paffenbarger, et al, 1987].

Large-scale multifactorial studies have been widely reported. The Multiple Risk Factor Intervention Trial (MRFIT) combined cholesterol reduction education, a smoking cessation program, and stepped-care drug therapy for hypertension in over 12,000 middle-aged men, and found a substantial reduction of risk factors. Risk reduction and rates of CHD mortality and nonfatal acute heart attack were not significantly different from controls, however [MRFIT Research Group,1982].

The North Karelia project in Finland demonstrated significant reduction in SBP and DBP but nonsignificant reductions in smoking rates and TC levels in men and women in response to community-wide health education attempts. Mass media messages, public meetings, and environmental changes were used [Salonen, et al,1981].

The Stanford Heart Disease Prevention Project attempted to modify smoking, exercise, and dietary behavior through mass media and/or face-to-face individual instruction to produce awareness of risk factors and to provide knowledge and skills necessary to accomplish the recommended behavior changes. Project results showed that media plus personal instruction induced immediate and reasonably stable risk
reduction while media-only instruction took longer to achieve its greatest effect, which was only partially maintained after treatment. The authors concluded that knowledge about CVD risk factors and related behavior can be a prerequisite for risk reduction [Meyer, et al, 1980;

Farquahar, et al,1977].
A program for South Carolina state employees used a comprehensive approach that targeted exercise, nutrition, stress, safety, tobacco, and alcohol. Programs in lifestyle education were supplemented with films, health risk appraisals, posters, newsletters, special exercise events, and policy changes related to smoking. Results indicated success in achieving smoking reduction, increased exercise participation, and a few positive dietary habit changes. No significant changes were seen in stress levels, but job satisfaction was improved [Kronenfeld,et al,1987].

A number of smaller multifactorial risk reduction trials have been reported, frequently involving university faculty/staff. A small sample of 41 university faculty and administrators received an exercise class; progressive muscle relaxation instruction, and lifestyle educational literature. In comparison to 9 control subjects, participants showed greater improvements in weight loss, exercise participation, cholesterol intake, self-esteem, and tolerance for work overload [Horowitz,et al; 1985].

Tischler tested the effects of providing an exercise program and nutrition and stress management education on a group of 24 faculty/staff, with 16 subjects as controls. Significant improvements were seen only in physical measurements such as flexibility and cardiovascular endurance, but the experimental group did report increased self-esteem [Tischler,1984].

More and more corporations are providing employees with structured programs that include health risk screening and comprehensive risk reduction education, based on the assumption that healthier employees are more satisfied and productive. Although the success of such programs on specific risk factors seems to be variable, company executives have enthusiastically embraced the concept and acted upon it. Control Data Corporation's "Staywell Program", Johnson \& Johnson's "Live for Life" program, and Tenneco's "Lifecycles" project all emphasize lifestyle and behavior change to impact health, job performance, and quality of life [Sloan,1987; Glanz and Orr,1986].

## Factors Affecting Health Behavior Change

Nearly all health promotion programs that attempt to provoke behavior change have problems with subject motivation, attrition, and maintenance of desired behaviors [Sloan, 1987].

One factor in motivation to attempt lifestyle change seems to be locus of control of the individual. Those people with a more internal locus of control perceive $a$ greater relationship between their own behavior and its effects, and seem to perceive higher personal responsibility for potential CHD development [Sechrist,1979].

Another study focused on attitudes relating to health behaviors found that individuals who were more likely to act on their own to maintain their health and promote wellness tended to value such health behaviors, to emphasize lifestyle as a determinant of health, and to have a positive rating of their own health [Yoder, et al,1985].

Intentions, attitudes, and subjective norms are elements which may predispose one to engage in healthy behaviors, according to the "theory of reasoned action" [Pender and Pender,1986]. Healthy behaviors are much more likely to occur if consequences are believed to be desirable (attitudes) and if those people the individual considers to be important to him/her see these healthy behaviors as worthwhile (subjective norms).

Studies by Salzer and Sejwacz, however, failed to find significant correlations between stated intentions to lose weight and actual weight loss, or between attitudes toward weight loss and actual weight loss behavior, leading to a surmise that weight loss behavior may be more related to
other factors [Pender and Pender,1986]. Those at or close to recommended weight were more likely to have intentions to exercise during the following week than those underweight or overweight.

Cholesterol reduction as seen by the theory of reasoned action could be explained by the concept of subjective norms. That is, great emphasis on the value of and. strategies for cholesterol reduction are presented daily, almost hourly, in the mass media, and people have responded to this impetus, as seen by the great demand for oat bran and its perceived cholesterol-lowering properties.

Dishman has found evidence that attitudes (perceived consequences) may be useful in explaining intentions to exercise regularly, to eat nutritiously for weight control. and to manage stress [Pender and Pender,1986]. Subjective norms seemed to play a significant part only in intentions to control weight and exercise regularly, and an individual's perceived health status was a principle determinant of weight control intention.

On the other hand, subjective norms, demonstrated by social support from family or others important to an individual, may promote continued, regular exercise. This support may be verbal or through group or family fitness activities.

Motivation to begin or continue a healthy activity is
one barrier to individual participation. Once the behavior is undertaken, problems of compliance or adherence to the behavioral regimen must be addressed. Behavioral influences on adherence may include the individual's health status, weight, perceived consequences of the behavior, and social support by significant others. Group sociability and removal of inconveniences may also play a part [Parsons, 1987].

Dishman found that environmental factors such as the setting, exercise leader, and interpersonal relationships may exert only minor influence on adherence, while body weight, percent body fat, and degree of self-motivation were more important. Attainment of personal goals was more likely to predict adherence. Those who were self-referred, discontinued smoking, had spousal support of behavior change, or were involved in active leisure pursuits were more likely to adhere to an exercise program. Lower occupational status tended to predict dropouts as well [Pender and Pender, 1986].

This information seems to reinforce the concept of interaction of CVD risk factors, in that each influences the development of other risk factors.

Adherence rates for voluntary exercise programs typically are 40 to 65 percent during the first year, with similar rates for a variety of therapeutic regimens (smoking
cessation, dietary modification, etc.). Typically there is a rapid and substantial loss of participants ( 30 to 50 percent) in the first one to six months, with a fairly stable plateau for the next 12 to 15 months. One study showed a high 77 percent compliance after 24 months with a whitecollar managerial population, however [Oldridge, 1982]. After 48 months, the dropout rate generally stands at 45 to 80 percent [Martin and Dubbert, 1982].

Education, occupation, and socioeconomic level have been seen to affect health behaviors and response to health promotion /health education [Sagan, 1987]. College education and socioeconomic class show a strong inverse relationship with cigarette smoking incidence. The percentage of persons who are ex-smokers is very high among the college-educated. Trends between 1977 and 1983 show that for lowest income and education groups, cigarette smoking is increasing. Stressful life events, obesity, and physical inactivity appear to be significantly associated with socioeconomic class as well.

Literacy and health are consistently and more strongly related than health and income. Education may increase health by increasing knowledge of health, health behaviors, and hazards of unhealthy behaviors; by providing better coping strategies and early recognition of stressful life events; and by promoting higher self-esteem [Sagan, 1987].

## CHAPTER III

## METHODS AND PROCEDURES

The purpose of the study was to determine whether OSU faculty/staff who attended healthy lifestyle classes changed significantly on the variables of systolic blood pressure, diastolic blood pressure, total serum cholesterol, and body weight from pretest to posttest compared to faculty/staff who did not attend such classes.

Both pretest and posttest were voluntarily attended by 265 faculty/staff. High risk individuals (DBP $\geq 90 \mathrm{mmHg}, T C$ $\geq 240 \mathrm{mg} / \mathrm{dL}$ ) were removed from this study, leaving 140 lowmoderate risk subjects who did not attend classes and 58 low-moderate risk subjects who participated in at least one class session.

A total of five topics were presented in separate fourweek periods. Topics included smoking cessation, weight control/nutrition, cholesterol reduction, proper exercise, and stress management. Each topic was presented in sessions lasting two hours one night a week for a total of eight hours per topic. Faculty and staff could attend one or all sessions under each topic.

Subjects were pretested with a health risk appraisal and counseled afterwards for approximately 10 to 15 minutes
during the spring and summer of 1987. Classes were offered in the summer and fall of 1987 , and a second health risk appraisal (posttest screening) was offered in the spring and summer of 1988.

Selection of Subjects

All Oklahoma State University faculty and staff received a letter via campus mail inviting their participation in the health risk appraisal as part of the OSU Wellness Program Pilot Project. Of 600 volunteers, 400 were chosen for a stratified sample to include faculty, administrative and professional personnel ( $A$ and $P$ ), and classified staff (clerical and technical). An equal number of $A$ and $P$ and classified subjects were chosen, with faculty accounting for double that of any of the other strata. After stratification, subjects were picked at random from the list of volunteers. The number of subjects was limited for financial reasons.

For the initial testing, 393 of the 400 selected were tested. Of these, 285 returned when invited for a second screening approximately one year later. All 400 were invited to attend healthy lifestyle classes, and 87 did so. Of these 87, 29 individuals received treadmill testing and personal counseling in addition to attending classes and so were removed from the study, while 58 remained in the study.

One hundred forty people who received both screenings did not participate in healthy lifestyle classes.

Descriptive Data

Both Group I (nonparticipants) and Group II (participants) were approximately evenly divided as to gender, although Group II held a slightly larger proportion of males (58.6\%). [See Table II ].

Mean age of Group I was 44.21 years, while for Group II it was 45.4 years. Ages ranged from 21 to 66 years, with a normal distribution among subjects. The highest percentage of subjects were in the 40 to 49 year old bracket [See Table III ].

Forty subjects (20.2\%) attended only one class, while 18 subjects ( $9.1 \%$ ) attended two or more. The two categories were combined for statistical analysis. Nonparticipants made up $70.7 \%$ of subjects ( 140 individuals) [See Table IV ].

Baseline measurements indicated a mean SBP of 120.8 $\mathrm{mmHg}, \mathrm{SD}=13.5$ for Group I , and $125.1 \mathrm{mmHg}, \mathrm{SD}=14.7$ for Group II. Overall mean for both groups was 122 mmHg , with a range between 92 and 168 mmHg [See Table $V$ ].

Baseline mean $D B P$ was $78.2 \mathrm{mmHg}, \mathrm{SD}=8.4$ for Group $I$, with a mean of $80.4 \mathrm{mmHg}, \mathrm{SD}=9.3$ for Group II. Overall mean for both groups was 78 mmHg , with a range of 58 to 106

TABLE II
COMPARISON OF GENDER FREQUENCY
IN GROUPS I AND II

| Gender | $f$ | $\%$ |
| :---: | :---: | :---: |
| Group I |  |  |
| male | 73 | 52.2 |
| female | $\mathrm{n}=140$ | 47.8 |
|  |  |  |
| Group II | 34 | 58.6 |
| male | 24 | 41.4 |
| female | $n=58$ |  |
|  |  |  |



TABLE IV
CLASS ATTENDANCE FREQUENCY

| Class Attendance | $f$ | $\%$ |
| :---: | :---: | :---: |
| 0 Classes | 140 | 70.7 |
| 1 Class | 40 | 20.2 |
| 2 or more | 18 | 9.1 |

$n=198$
mmHg [See Table $V$ ].
Mean TC at baseline was $206.3 \mathrm{mg} / \mathrm{dL}, \mathrm{SD}=33.5$ for Group I, and $203.2 \mathrm{mg} / \mathrm{dL}, \mathrm{SD}=35.2$ for Group II. The overall mean for both groups at baseline was $205 \mathrm{mg} / \mathrm{dL}$, with a range of 111 to $345 \mathrm{mg} / \mathrm{dL}$ [See Table V ].

Baseline WT measurements showed a mean of $161.3 \mathrm{lbs}, \mathrm{SD}$ $=30.3$, for Group $I$, with a mean of $164.3 \mathrm{mg} / \mathrm{dL}, \mathrm{SD}=27.5$, for Group II. Overall mean weight for both groups was 162 lbs. with a range between 98 and 283 lbs. [See Table V].

## Testing Procedures

Subjects entered the laboratory area, paid a fee of \$3.75 for the health risk appraisal (HRA), and signed an informed consent form. Total cholesterol was tested by the fingerstick method [Curtius and Roth,1978] using a Vision (tm) blood analyzer. While waiting for blood results, the subjects proceeded to various stations to receive measurements of blood pressure, height and weight, and percent body fat, and then filled out a 19-item cardiac risk questionnaire. All data were entered into a computer program devised at the OSU Health and Fitness Center, and subjects received a two-page printout of results, including a calculated risk score.

Each subject was then individually counseled in a location removed as much as possible from the general
testing area. Counseling lasted 10 to 15 minutes and included information on risk factors and methods of reducing risks through diet, exercise, smoking cessation, or stress management. Counseling sessions covered these topics succinctly due to limited time.

The second HRA was carried out in a similar fashion seven to twelve months later, with the exception that no cardiac risk questionnaire was completed. Counseling was minimal, involving only comparison of risk factor measures from pretest to posttest and further brief recommendations for change if needed.

Classes were presented on the topics of smoking cessation, nutrition/weight control, cholesterol reduction, proper exercise, and stress management, each presented separately over four weeks for two hours per week. Participation was voluntary, in response to flyers distributed by campus mail. Each class cost the participant five to ten dollars.

## Clinical Laboratory Procedures

Blood pressure was measured with the subject in the sitting position, using a sphygomomanometer with arm cuff. Diastolic blood pressure was determined as the fifth Korotkoff sound [Pinckney and Pinckney, 1982].

Body weight was determined on a medical scale with the
subject fully clothed but with shoes removed.
Total serum cholesterol measurement required a fingerstick [Curtius and Roth,1978] with a sterile microlance lancet. After wiping away the first drop of blood, blood was drawn into the capillary tube and placed in a Vision (tm) cholesterol reagent kit. The kit was then put in the Vision (tm) automated blood analyzer which determined the cholesterol concentration by spectrophotometric means and provided a printout of results.

## Apparatus and Equipment

Measurements of blood pressure were performed with a PyMaH Trimline (tm) mercury sphygmomanometer (Somerville, New Jersey), brachial pressure cuff, and stethoscope.

Body weight was determined using Detecto-Medic (tm) body scales (Detecto Scales, Inc., Brooklyn, New York).

Total serum cholesterol was measured with Abbott Laboratories Vision (tm) automated blood analyzer (Abbott Laboratories, Dept. 99F, North Chicago, Illinois, 80064).

Statistical Treatment

Data were analyzed using independent t-tests for each variable [Polit and Hunger, 1978]. Comparisons between groups were analyzed pre-treatment and again post-treatment to determine whether there were significant differences at
baseline and over time. SBP gain scores were subjected to analysis of variance as well. The level of significance was set at 0.050 .

## CHAPTER IV

## RESULTS AND DISCUSSION

The purpose of the study was to determine whether OSU faculty/staff who attended healthy lifestyle classes changed significantly on the variables of systolic blood pressure, diastolic blood pressure, total serum cholesterol, and weight from pretest to posttest compared to faculty/staff who did not attend such classes.

Health risk appraisal and class attendance were strictly voluntary so there was neither random selection nor random assignment.

## Results

Results of Systolic Blood

## Pressure Analysis

The results of systolic blood pressure analysis are given in Table VI. Although there was a slight but significant difference in SBP between groups at baseline, this difference was insignificant at posttest. There was no difference after treatment between groups in systolic blood pressure levels at the 0.050 level of significance.

## Results of Diastolic Blood

## Pressure Analysis

The results of diastolic blood pressure analysis are given in Table VI. There was no difference after treatment between groups in diastolic blood pressure levels at the 0.050 level of significance.

## Results of Total Serum

## Cholesterol Analysis

The results of total serum cholesterol analysis are given in Table VI. There was no difference after treatment between groups in total serum cholesterol levels at the 0.050 level of significance.

## Results of Body Weight Analysis

The results of body weight analysis are given in Table VI. There was no difference after treatment between groups in body weight measurements at the 0.050 level of significance.

TABLE V

## COMPARISON OF PRETEST DATA BETWEEN GROUPS I AND II

| Pre SBP | n | X | SD | SE | t | $p$ |  |
| :---: | :---: | :---: | :---: | :---: | :---: | :---: | :---: |
| Group I | 140 | 120.80 | 13.53 | 1.14 |  |  |  |
| Group II | 58 | 125.09 | 14.74 | 1.94 |  |  |  |
|  | 198 |  |  |  | -1.97 | 0.050 | * |


| Pre DBP | n | X | SD | SE | t | D |
| :---: | :---: | :---: | :---: | :---: | :---: | :---: |
| Group I | 140 | 78.19 | 8.45 | 0.72 |  |  |
| Group II | 58 | 80.41 | 9.32 | 1.22 |  |  |
|  | 198 |  |  |  | -1.64 | 0.10 |


| Pre TC | n |  | X | SD | SE | $t$ | D |
| :--- | ---: | ---: | ---: | :--- | :--- | :--- | :--- |
| Group I | 140 | 206.32 | 33.50 | 2.83 |  |  |  |
| Group II | $-\frac{57}{98}$ | 203.18 | 35.16 | 4.66 |  |  |  |
|  |  |  |  |  |  | 0.59 | 0.56 |


| Pre WT | n | X | SD | SE | t | p |
| :--- | ---: | ---: | ---: | ---: | ---: | ---: |
| Group I | 140 | 161.28 | 30.34 | 2.56 |  |  |
| Group II | 1688 | 164.30 | 27.50 | 3.61 |  |  |
|  |  | 198 |  |  |  | -0.65 |

* significant at 0.050 level

TABLE VI
COMPARISON OF POSTTEST DATA
BETWEEN GROUPS I AND II

| Post SBP | n | X | SD | SE | $t$ | p |
| :---: | :---: | :---: | :---: | :---: | :---: | :---: |
| Group I | 138 | 122.07 | 12.50 | 1.06 |  |  |
| Group II | 58 | 124.57 | 14.10 | 1.85 |  |  |
|  | 196 |  |  |  | -1.23 | 0.22 |


| Post_DBP | n | X | SD | SE | t | $p$ |
| :---: | :---: | :---: | :---: | :---: | :---: | :---: |
| Group I | 138 | 79.80 | 8.04 | 0.68 |  |  |
| Group II | 58 | 80.29 | 8.08 | 1.06 |  |  |
|  | 196 |  |  |  | -0.39 | 0.69 |


| Post TC | n | X | SD | SE | $t$ | $D$ |
| :--- | ---: | ---: | ---: | ---: | ---: | ---: |
| Group I | 139 | 193.57 | 29.98 | 2.54 |  |  |
| Group II | $-\frac{5}{9} \frac{8}{7}$ | 190.56 | 27.97 | 3.67 |  |  |
|  |  |  |  |  |  | -0.30 |


| Post WT | n | X | SD | SE | $t$ | p |
| :---: | :---: | :---: | :---: | :---: | :---: | :---: |
| Group I | 138 | 162.39 | 30.76 | 2.63 |  |  |
| Group II | $\underline{58}$ | 163.79 | 27.68 | 3.63 |  |  |
|  | 196 |  |  |  | 1.00 | 0.32 |

TABLE VII
COMPARISON OF SYSTOLIC BLOOD PRESSURE GAIN BETWEEN GROUPS I AND II

| SBP Gain | n | X | SD | SE | t | p |
| :---: | :---: | :---: | :---: | :---: | :---: | :---: |
| $\begin{aligned} & \text { Group I } \\ & \text { Group II } \end{aligned}$ | 138 | 1.30 | 10.88 | 0.93 |  |  |
|  | 58 | -0.52 | 13.11 | 1.72 |  |  |
|  | 196 |  |  |  | 1.00 | 0.32 |

## Discussion

Heart disease, stroke, and cancer account for $75 \%$ of U.S. deaths annually [USDHEW, 1979], yet seem to be influenced by individual lifestyle, including the practices of cigarette smoking, eating high-cholesterol or high-fat diets, physical inactivity, and inadequate responses to stress. With the recent advent and burgeoning growth of the wellness movement, many more people have come to believe that habitual behaviors under personal control can affect one's health status to a large extent. Communities and corporations have instigated wellness programs to provide knowledge of risk factors for disease and to promote healthy behaviors. Oklahoma State University's Wellness Program used a health risk appraisal to screen faculty and staff for risk factors for cardiovascular disease and offered voluntary "healthy lifestyle" classes to provide in-depth information on risk factors and methods of behavior change specific to the risk factor.

The health status indices of DBP, TC, and WT at baseline were similar in Group I, who chose not to participate in healthy lifestyle classes, and Group II, who did attend at least one of the class sessions. After class attendance, again no significant differences in these health indices were noted between Group I and Group II.

Because SBP level was significantly higher in Group II
at baseline ( $p=0.050$ ), SBP gain was compared between groups and found to be insignificant ( $p=0.318$ ) at the 0.050 level of significance [See Table VII ].

Total cholesterol levels did seem to decrease substantially but equally in both groups, demonstrating insignificant differences between groups on posttest ( $\mathrm{p}=0.221$ ).

This lack of significant difference on the posttest between comparable pretest groups indicates that no significant change occurred in the group that attended one or more classes as compared to the group that did not attend any classes. Apparently, then, no real change associated with class attendance was noted in blood pressures, total cholesterol, or weight. SBP, DBP, TC, and WT were used as indicators of behavior change for the behaviors of cigarette smoking, weight control and cholesterol reduction, exercise, and stress management. Thus, class attendance did not seem to be associated with behavior change for these behaviors.

Lack of change associated with class attendance can be explained in several ways. One explanation is that the classes were not effective for those who attended.

Information was presented by lecture by highly-trained wellness staff, although some behavior modification techniques were presented in the smoking cessation class. The lecture method, though it tends to be traditional and
well-received in a university setting, is seen to be less effective in prompting behavior change because it is limited in the key learning principles of motivation, transfer of learning, feedback, and reinforcement [Stone, 1982]. This method is used often, however, to reach large numbers of people in a short time at low cost with limited staff.

A number of subjects who did attend classes may have attended only one two-hour class session. This amount of instruction may be insufficient to motivate or to provide adequate information to promote health behavior change.

Some educational research indicates that attempting several behavior changes at once limits program effectiveness, but since the behaviors interact to produce a healthy lifestyle, it was deemed necessary in this situation. This concept could have been tested by determining variations in those who attended two or more classes as compared to those who attended only one, but sample sizes for comparison were not closely equivalent.

Another explanation of results could be that the classes presented information that was already known or available to many nonparticipants, who therefore took appropriate steps to change behavior on their own. Information similar to that presented in classes has been widespread in recent newspapers, magazines, television programs and books, especially on the subjects of
cholesterol, smoking, and exercise. Such media influence before and during the study is an unavoidable source of contamination of the study.

In fact, the population in general has been changing lifestyles to include more exercise, less smoking, and an improved diet since the late 1960's, and CHD risk has gone down as a result [Stamler,1985; Pell and Fayerweather, 1985], indicating that the mass media has been at least somewhat influential.

Cholesterol levels showed a similar decline in both groups which may be due to recent media or wellness program emphasis on this one risk factor. Normal day-to-day variation or variations in automated blood analyzer readings could account for a decline of such magnitude also [Record and Record,1988].

Nonparticipants received information on risk factors and general methods to reduce risk in the health risk appraisal and in the following counseling mini-session. Such information could have been sufficient to inspire cholesterol-reduction behaviors in all participants. Nonparticipants are likely also to have received information in daily conversation with class participants in some cases and from newsletters sent out to all faculty and staff.

Mean values of systolic blood pressure, diastolic blood pressure, and body weight changed slightly pretest to
posttest, which could represent actual insignificant changes, but are more likely to represent regression towards the mean.

Motivation can play a part in bringing about behavior change, but it is hard to measure. Adherence to a behavioral regimen (such as exercise or smoking reduction), however, can be measured more easily, although it was not measured in this study. Typical adherence declines rapidly at first, then more slowly over three months to a year [Oldridge, 1982]. Variations in time between treatment (class attendance) and posttest measurements ranged from immediately post-treatment to as much as six to eight months post-treatment. For the longer time periods, recidivism would be expected to be prevalent among subjects, particularly as there was no feedback or reinforcement postprogram except for occasional newsletters.

Perhaps most important in looking at the results is the fact that almost all of those OSU faculty/staff at very high risk for cardiovascular disease were removed from the study because they were provided with intensive counseling and treadmill stress testing, contaminating the effect of class attendance in providing knowledge of risk factors. Approximately half the class attendees fell in this category, and the class may have been more influential for them. These people may have perceived the risks as more
serious and immediate than subjects of this study, and therefore could have been motivated to change more utilizing the additional information they received from the classes.

Lack of control of influential factors such as spousal support, current health status, ethnic heritage, and intervening medical treatment could have reduced the ability of the investigator to determine real differences associated solely with class attendance.

## CHAPTER V

SUMMARY, FINDINGS, CONCLUSIONS, AND RECOMMENDATIONS

## Summary

This study was undertaken to determine whether attendance at healthy lifestyle classes by OSU faculty/staff would be associated with significant changes of SBP, DBP, $T C$, and $W T$ in comparison to those faculty/staff who did not attend such classes. Initial screening results showed similar values for $\mathrm{DBP}, \mathrm{TC}$, and WT; however, class participants had significantly higher baseline SBP. On subsequent screening post-treatment, the two groups of participants and nonparticipants again showed very similar values, including the values for SBP.

This study was conducted using a quasi-experimental research design. Data analysis was performed using independent t-tests for each of the variables of SBP, DBP, TC, and WT.

## Findings

Based on the hypotheses stated and the limits of this study, the following results were seen:

1. There were no significant differences between class participants and nonparticipants in systolic blood pressure variation from pretest to posttest.
2. There were no significant differences between class participants and nonparticipants in diastolic blood pressure variation from pretest to posttest.
3. There were no significant differences between class participants and nonparticipants in total serum cholesterol variation from pretest to posttest.
4. There were no significant differences between class participants and nonparticipants in body weight variation from pretest to posttest.

## Conclusions

Studies of lifestyle behaviors and risk factor changes are important in reducing incidence and mortality of cardiovascular disease and coronary heart disease. This study concluded that informational lecture classes have little influence in modifying behaviors that result in changes in the health indicators of systolic or diastolic blood pressure, total serum cholesterol, or body weight.

Suggestions for further study include controlling such confounding variables as support by spouse, health status, previous experience with exercise or dietary regimens, treatment by medical professionals or medications taken in
the interim between tests, environmental influences, or length of time between the treatment and the posttest. Control over any number of these variables could help reduce contamination of the study by other factors which might influence physiological or behavior change.

Future studies should report exactly which classes and how many sessions were attended by subjects to determine the effects or interaction of effects of multiple versus single class attendance.

Pretest measurements of CVD risk knowledge and attitudes toward behavior change are suggested to determine whether class participants initially had less knowledge or more positive attitudes than nonparticipants, and thus would be expected to receive greater benefit from classes.

## Recommendations

Behavior change is highly influenced by the learning principles of motivation, feedback, and reinforcement. Information alone may not be enough to motivate one to change behaviors ingrained over a lifetime. Providing reinforcement at the worksite by program follow-ups and contacts would be recommended to determine whether this would improve compliance with necessary behavior changes.

A recommended addition to this study would be a second
screening of the treadmill population, who received much educational information through intensive personal contact. Comparing changes in treadmill participants who attended class to treadmill participants who did not attend class could give an indication of class effectiveness when CVD risk was more likely to be perceived as serious or potentially life-threatening.

Additional research in this area would be important in determining the most effective educational and motivational methods of changing behaviors that produce health risk.

## A SELECTED BIBLIOGRAPHY

1. Alderman, M., Green, L.W., and Flynn, B.S.
"Hypertension Control Programs in Occupational Settings." Public Health Reports. March/April 1980. 95 (2): 158-163.
2. Apte, J.K. "The Relationship Between Blood Pressure Levels, Diet, Weight Perception, Salt Preference and Psychosocial Stress." Ph.D. Dissertation. University of North Carolina at Chapel Hill. University Microfilms, Inc. 1980.
3. Belloc, N.B. "Relationship of Health Practices and Mortality." Preventive Medicine. 1973. 2: 67-81.
4. Belloc, N.B. and Breslow, L. "Relationship of Physical Health Status and Health Practices." Preventive Medicine. 1972. 1: 409-421.
5. Blair, S.N., et al. "Health Promotion for Educators: Impact on Health Behaviors, Satisfaction, and General Well-Being." American Journal of Eublic Health. Feb.1984. 74 (2): 147-149.
6. Blair, S.N., et al. "Physical Fitness and Incidence of Hypertension in Healthy Normotensive Men and Women." Journal of the American Medical Association. July 27,1984. 252 (4): 487-490.
7. Castelli, W.P., et al. "Incidence of Coronary Heart Disease and Lipoprotein Cholesterol Levels: the Framingham Study." Journal of the American Medical Association. Nov. 28,1986. 256 (20): 2835-2838.
8. Center for Health Promotion and Education, Center for Disease Control. "Protective Effect of Physical Activity on Coronary Heart Disease." Mortality and Morbidity Weekly Report. July 10, 1987. 36 (26): 426-430.
9. Christian, J.L. and Greger, J.L. Nutrition for Living, 2nd ed. Menlo Park, CA: The Benjamin/Cummings Publishing Company, Inc. 1988.
10. Cole, G.E., Duncan, D.J., and Friedman, G.M. "A Systems Perspective for Hospital-Based Health Promotion." Optimal Health. Nov./Dec. 1986. pp. 24-27.
11. Cook, R.J., Walden, R.T., and Johnson, D.D. "Employee Health and Fitness Program at the Sentry Corporation." Health Education. July/Aug. 1979. pp. 4-6.
12. Cooper, K.H., et al. "Physical Fitness Levels vs Selected Coronary Risk Factors." Journal of the American Medical Association. July 12, 1976. 236 (2): 166-169.
13. Curtius, H.C. and Roth, M. Clinical Biochemistry: Principles and Methods. New York: Walter de Gruyter, 1978.
14. Dedmon, R. "Health Management Program at KimberlyClark Corporation." Health Education. July/Aug. 1979. p. 7.
15. Farquhar, J.W., et al. "Community Education for Cardiovascular Health." The Lancet. June 4, 1977. pp. 1192-1194.
16. Fihn, S.D. "A Prudent Approach to Control of Cholesterol Levels." Journal of the American Medical Association. Nov. 6,1987. 258 (17): 2416-2418.
17. Foreyt, J.P., Scott, L.W., and Gotto, A.M. "Weight Control and Nutrition Education Programs in Occupational Settings." Public Health Reports. March/April, 1980. 95 (2): 127-136.
18. Gibbons, L.W., et al. "Association Between Coronary Heart Disease Risk Factors and Physical Fitness in Healthy Adult Women." Circulation. May, 1983. 67 (5): 977-982.
19. Glanz, K. and Orr, R. Worksite Nutrition: A DecisionMaker's Guide. Chicago, IL: American Dietetic Association. 1986.
20. Goetz, A.A., Duff, J.F., and Bernstein, J.E. "Health Risk Appraisal: The Estimation of Risk." Public Health Reports. Mar./Apr., 1980. 95 (2):119126.
21. Green, L.W. and Lewis, F.M. Measurement and Evaluation in Health Education and Health Promotion. Palo Alto, CA: Mayfield Publishing Company. 1986.
22. Grundy, S.M. "Cholesterol and Coronary Heart Disease: A New Era." Journal of the American Medical Association. Nov. 28,1986. 256 (20): 2849-2860.
23. Haskell, W.L. and Blair, S. N. "The Physical Activity Component of Health Promotion in Occupational Settings." Public Health Reports. Mar./Apr., 1980. 95 (2): 109-118.
24. Heinzelmann, F. and Bagley, R.W. "Response to Physical Activity Programs and their Effects on Health Behavior." Public Health Reports. October, 1970. 85 (10): 905-911.
25. Horowitz, S.M., et al. "Wellness Intervention Effects on Lifestyle, Attitudes, and Stress." Presented at Annual Meeting of American Educational Research Association. April, 1985.
26. Hubert, H.B., et al. "Obesity as an Independent Risk Factor for Cardiovascular Disease." Circulation. May, 1983. 67 (5).
27. Hunt, W.A. and Bespalec, D.A. "An Evaluation of Current Methods of Modifying Smoking Behavior." Journal of Clinical Psychology. Oct. 1973. 30 (4): 431-438.
28. Kaplan, L.H. and Burch-Minakan, L. "Reach Out for Health: A Corporation's Approach to Health Promotion." The American Journal of Qccupational Therapy. Nov., 1986. 40 (11): 777-780.
29. Klesges, R.C., et al. "Competition and Relapse Prevention Training in Worksite Smoking Modification." Health Education Research. 1987. 2 (1): 5-14.
30. Kolata, G.B. and Marx, J.L. "Epidemiology of Heart Disease: Searches for Causes." Science. Oct. 29, 1976. 194: 509-512.
31. Kromhout, D. and Arntzenius, A.C. "Use of Diet to Modify Serum Cholesterol, Lipoproteins, and Progression of Coronary Atherosclerosis" in Nutrition and Heart Disease: Volume I. Ed. Watson, R.R. Boca Raton, FL: CRC Press, Inc. 1987. pp. 93-106.
32. Kronenfeld, J.J., et al. "Evaluating Health Promotion: A Longitudinal Quasi-Experimental Design." Health Education Quarterly. Summer, 1987. 14 (2): 123-139.
33. Leon, A.S., et al. "Leisure-Time Physical Activity Levels and Risk of Coronary Heart Disease and Death: The Multiple Risk Factor Intervention Trial." Journal of the American Medical Association. Nov. 6, 1987. 258 (17): 2388-2395.
34. Levi, L. "Psychosocial Factors in Preventive Medicine." In Healthy People: The Surgeon General's Report on Health Promotion and Disease Prevention: Background Papers. Washington, D.C.: U.S. Government Printing Office, USDHEW. 1979.
35. Lipid Research Clinics Program. "The Lipid Research Clinics Coronary Primary Prevention Trial Results. I." Journal of the American Medical Association. Jan. 20, 1984. 251 (3): 351-364.
36. Martin, J.E. "Corporate Health: A Result of Employee Fitness." The Physician and Sportsmedicine. March, 1978. 6(3): 135-137.
37. Martin, J.E. and Dubbert, P.M. "Behavioral Management Strategies for Improving Health and Fitness." Journal of Cardiac Rehabilitation. 1984. 4: 200-208.
38. Martin, J.E. and Dubbert, P.M. "Exercise Applications and Promotion in Behavioral Medicine: Current Status and Future Directions." Journal of Consulting and Clinical Psychology. 1982. 50 (6): 1004-1017.
39. Marx, J.L. "Hypertension: A Complex Disease with Complex Causes." Science. Nov. 19, 1976. 194: 821-825.
40. McNamara, D.J. "Nutrition and Cholesterol

Metabolism." in Nutrition and Heart Disease:
Volume I. Ed. Watson, R.R. Boca Raton, FL: CRC Press, Inc. 1987. pp. 29-44.
41. Messerli, F.H. "Cardiopathy of Obesity -- A Not-SoVictorian Disease." The New England Journal of Medicine. Feb. 6, 1986. 314 (6): 378-379.
42. Meyer, A.J., et al. "Skills Training in a Cardiovascular Health Education Campaign." Journal of Consulting and Clinical Fsychology. 1980. 48 (2): 129-142.
43. Morisky, D.E. "Hypertension Among the Urban Poor." Health Education Focal Points. 1985. No. 1. U.S. Department of Health and Human Services.
44. Morris, J.N., et al. "Vigorous Exercise in LeisureTime: Protection Against Coronary Heart Disease." The Lancet. Dec. 6, 1980. pp. 1207-1210.
45. MRFIT Research Group. "Multiple Risk Factor Intervention Trial: Risk Factor Changes and Mortality Results." Journal of the American Medical Association. Sept. 24, 1982. 248 (12): 1465-1477.
46. National Cholesterol Education Program. "Detection, Evaluation, and Treatment of High Blood Cholesterol in Adults." National Heart, Lung, and Blood Institute. USDHHS. 1986.
47. Nelson, D.J., et al. "A Campaign Strategy for Weight Loss at Worksites." Health Education Research. 1987. 2 (1): 27-31.
48. Oja, P., et al. "Feasibility of an 18 Months' Physical Training Program for Middle-Aged Men and its Effect on Physical Fitness." American Journal of Public Health. May, 1974. 64 (5): 459-464.
49. Oldridge, N.B. "Compliance and Exercise in Primary and Secondary Prevention of Coronary Heart Disease: A Review." Preventive Medicine. 1982.
11: 56-70.
50. Oliver, M.F. "Diet and Coronary Heart Disease." British Medical Bulletin. Jan., 1981. 37 (1): 49-58.
51. Paffenbarger, R.S., et al. "A Natural History of Athleticism and Cardiovascular Health." Journal of the American Medical Association. July 27, 1984. 252 (4): 491-495.
52. Paffenbarger, R.S., et al. "Physical Activity, AllCause Mortality, and Longevity of College Alumni." New England Journal of Medicine. March 6, 1986. 314 (10): 605-613.
53. Paffenbarger, R.S. and Hale, W.E. "Work Activity and Coronary Heart Mortality." The New England Journal of Medicine. March 13, 1975. 292 (11): 545-550.
54. Paffenbarger, R.S., Wing, A.L., and Hyde, R.T. "Physical Activity as an Index of Heart Attack Risk in College Alumni." American Journal of Epidemiology. Sept., 1978. 108 (3): 161-175.
55. Parsons, T.W. "Faculty/Staff Wellness Programs. and Trends Among Midwestern Universities." NIRSA Journal. Spring, 1987. pp. 28-32.
56. Pate, R.R. and Blair, S.N. "Physical Fitness Programming for Health Promotion at the Worksite." Preventive Medicine. 1983. 12: 632-643.
57. Pauly, J.T., et al. "The Effect of a 14 -Week Employee Fitness Program on Selected Physiological and Psychological Parameters." Journal of Qccupational Medicine. June, 1982. 24 (8): 457-463.
58. Pell, S. and Fayerweather, W.E. "Trends in the Incidence of Myocardial Infarction and in Associated Mortality and Morbidity in a Large Employed Population, 1957-1983." The New England Journal of Medicine. April 18, 1985. 312 (16): 1005-1011.
59. Pender, N.J. and Pender, A.R. "Attitudes, Subjective Norms, and Intentions to Engage in Health Behaviors." Nursing Research. Jan./Feb. 1986. 35 (1): 15-18.
60. Pinckney, C. and Pinckney, E.R. Patient's Guide to Medical Tests. New York, NY: Facts on File Publications. 1982.
61. Polit, D. and Hungler, B. Nursing Research: Principles and Methods. Philadelphia: J.B. Lippincott Company. 1978.
62. Queen, H.L. "Diet as a Potent Modifier of Cardiovascular Risk: Has Something Been Overlooked?" in Nutrition and Heart Disease: Volume I. Ed. Watson, R.R. Boca Raton, FL: CRC Press, Inc. 1987. pp. 11-20.
63. Record, N.B. and Record, S.S. "Combined Screening: Blood Pressure and Cholesterol." Reply to letter in Journal of the American Medical Association. Jan. $22 / 29,1988$. 259 (4).
64. Reed, R.W. "Is Education the Key to Lower Health Care Costs?" Personnel Journal. Jan. 1984. pp. 40-46.
65. Sagan, L.A. The Health of Nations. New York: Basic Books, Inc. 1987.
66. Salonen, J.T., et al. "Changes in Smoking, Serum Cholesterol, and Blood Pressure Levels During a Community-Based Cardiovascular Disease Prevention Program -- the North Karelia Project." American Journal of Epidemiology. 1981. 114 (1): 81-94.
67. Schwartz, G.E. "Stress Management in Occupational Settings." Public Health Reports. Mar./Apr. 1980. 95 (2): 99-108.
68. Sechrist, W. "How Do People Respond to Health Messages?" Health Education. July/Aug. 1979. pp. 32-33.
69. Shields, S.L. "Cardiovascular Health, Stress, and Cardiac Risk Among Women in Higher Education as Compared to Women in Law and Medical Professions." in Wellness Promotion Strategies. Ed. Opatz, J.P. Dubuque, IA: Kendall/Hunt Publishing Company. 1984.
70. Simons-Morton, B.G., et al. "Effectiveness and Cost Effectiveness of Persuasive Communications and Incentives in Increasing Safety Belt Use." Health Education Quarterly. Summer, 1987. 14 (2): 167-169.
71. Sloan, R.P. "Workplace Health Promotion: A Commentary on the Evolution of a Paradigm." Health Education Quarterly. 14 (2): 181-194.
72. Smith, T.L. and Kummerow, F.A. "The Role of Oxidized Lipids in Heart Disease and Aging" in Nutrition and Heart Disease: Volume I. Ed. Watson, R.R. Boca Raton, FL: CRC Press, Inc. 1987. pp. 45-64.
73. Stamler, J. "Coronary Heart Disease: Doing the Right Things." The New England Journal of Medicine. April 18, 1985. 312 (16): 1053-1055.
74. Stamler, R., et al. "Weight and Blood Pressure." Journal of the American Medical Association. Oct. 6, 1978. 240 (15).
75. Stone, T. Understanding Personnel Management. Chicago: The Dryden Press. 1982.
76. Stromme, S.B. "Physical Activity and Health: Part I." Journal of Cardiac Rehabilitation. 1984. 4: 316-326.
77. Taylor, R.B., Ureda, J.R., and Denham, J.W. Health Promotion: Principles and Clinical Applications. Norwalk, CT: Appleton-Century-Crofts. 1982.
78. Teitelman, R. "Snake Pit." Financial World. Jan. 12, 1988. pp.14-15.
79. Thomas, C.L. Taber's Cyclopedic Medical Dictionary. Philadelphia: F.A. Davis Company. 1975.
80. Thomas, L. The Youngest Science: Notes of a MedicineWatcher. New York: Viking Press. 1983.
81. Tischler, J.W. "A Wellness Program for University Faculty and Staff." Presented at Annual Convention of AAHPERD. March, 1984.
82. U.S. Department of Health, Education, and Welfare, Public Health Service. Healthy Feople: The Surgeon General's Report on Health Fromotion and Disease Prevention. Washington, D.C.: U.S. Government Printing Office. 1979.
83. U.S. Department of Health and Human Services, Public Health Service. "Promoting Health/Preventing Disease." Public Health Reports. Sept./Oct. 1983 suppl., 98.
84. U.S. Department of Health and Human Services, Public Health Service. The Health Consequences of Smoking: Cardiovascular Disease. Washington D.C.: U.S. Government Printing Office. 1983.
85. U.S. Department of Health and Human Services. "Report of the Health Education - Risk Reduction Conference: Anaheim, CA, Oct. 27-30, 1981." Atlanta, GA: Center for Disease Control. Jan. 19, 1983.
86. Weiman, C.G. "A Study of Occupational Stressors and the Incidence of Disease/Risk." in Reducing Occupational Stress. National Institute for Occupational Safety and Health. USDHEW. April, 1978.
87. Wilbur, C.S. "The Johnson \& Johnson Program." Ereventive Medicine. 1983. 12: 672-681.
88. Wiley, J.A. and Camacho, T.C. "Life-Style and Future Health: Evidence from the Alameda County Study." Preventive Medicine. 1980. 9: 1-21.
89. Yoder, L.E., et al. "The Association Between Health Care Behavior and Attitudes." Health Values: Achieving High Level Wellness. July/Aug. 1985. 9 (4).

APPENDIX

## CONTENTS OF HEALTHY LIFESTYLE CLASSES

Smoking Cessation:

> Session 1 - Analyzing Your Present Behavior Session 2 - The Effects of Smoking Session 3 - Methods of Quitting Session 4 - The Best Way to Quit and Stay Quit

Cholesterol Reduction:
Session 1 - Dietary Guidelines, Calories, and Exchanges
Session 2 - Exercise and Bioenergetics
Session 3 - Cholesterol, Fats Session 4 - Behavior Change and Motivation

Weight Control/Nutrition:

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Session 1 - Eating Right - Oklahoma Style
Session 2 - Obesity in America
Session 3 - Omega Three Fatty Acids
Session 4 - Osteoporosis
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Proper Exercise:

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Session 1 - Principles of Exercise
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Session 2 - Walking, Running, and Shoes
Session 3 - Water Aerobics
Session 4 - Weight Training for Adults

Stress Management:
Session 1 - Stress: What Is It/ Symptoms
Session 2 - Causes of Stress; Where Does It All Lead Session 3 - Remedies; Can You Cure It? Session 4 - Remedies; Where To Go From Here


## CONGRATULATIONS!!

You have been selected for the Faculty/Staff Wellness Program (pilot project). We will begin immediately with the wellness screening test (appointments only please). The hours for wellness screening are as follows:

Mon.-Wed.-Fri.: 7:30-8:30 atm. (call $\times 7260$ to schedule appointment) Tues.-Thurs.: 8:00-10:00 atm.

All tests will be done in 120 Calvin Center (Health and Fitness Center lab). The test requires 12 hours of fasting; nothing except water for 12 hours prior to your blood cholesterol test. The blood will be taken from a finger stick (relatively painless). You will also have a blood pressure check, skinfold body fat analysis and cardiac risk questionnaire (approximately 20 questions). The entire process should take 20-30 minutes, excluding results counseling (which may be done then or scheduled later).

Cost for the wellness screen is $\$ 15.00$ but you pay $\$ 3.75$ (checks payable to Health and Fitness Center or cash graciously accepted).

Once we have completed the screening, we will determine if further testing is needed. If physician supervision is required, the cost will fall under the insurance program and not wellness.

We will accomplish the screening this semester, with most fitness evaluations being conducted this summer or early fall semester. Most classes and activity courses will be conducted in the Fall. The pilot program will conclude around Christmas.

If you have any questions or to schedule your screening appointment, call Karen at $\times 7260$.


## OKLAHOMA STATE UNIVERSITY <br> HEALTH AMD FITNESS CENTER

INFORMED CONSENT FORM

## EXPLANATION OF TESTS

The fitness evaluation and cardiac risk profile you are about to undergo is part of the Oklahoma State University Faculty/Staff Wellness Program. The evaluation includes height and weight analysis, skinfold fat test, resting blood pressure, selected blood variables analyzed from the fingerstick method or from a venous sample, and a cardiac risk profile.

It will be determined, prior to testing, that this evaluation is appropriate and safe for you. All testing will be conducted by trained personnel and procedures will be explained to your satisfaction at the outset.

## POSSIBLE RISKS

The potential risks associated with venipuncture are: (1) Venipuncture may cause some pain or discomfort. The exact amount, if any, will be dependent upon individual preconceptions and pain threshold levels. (2) Possible hematoma (bruising) at the venipuncture site following the procedure. The occurrence or non-occurrence will be dependent upon bleeding/coagulation times and adherence to instructions pertaining to holding a cotton ball against the venipuncture site, with pressure, for five minutes following extraction of the needle. (3) Slight risk of infection. Any break in the integrity of the skin is associated with a small degree of risk of infection. However, if directions are followed the risk is very small.

The potential risks associated with the fingerstick method of obtaining a blood sample are essentially the same as for venipuncture.

## POTENTIAL BENEFITS

The potential benefits of the program are as follows: (1) The results of testing will serve to give the subject an in-depth view of his current cardiac risk and could possibly alert the subject to potential health problems. (2) The potential benefits of a comprehensive wellness program are to aid the subject in achieving an optimal state of wellness.

## CONSENT BY SUBJECT

The information which is obtained will be treated as privileged and confidential and will not be released or revealed to anyone without your express written consent. Information will, however, be treated in an aggregate manner to provide group information.

I have read the foregoing, l understand it, and any questions which may have occurred to me have been answered to my satisfaction. I understand I may withdraw from this test at any time without penalty.

Date $\qquad$
Subject's Signature $\qquad$
Witness $\qquad$

GEALTH AND FITNESS CENTER HELLNESS SCREENING

$\qquad$
$\qquad$

VITA<br>Diane. Kay Lowe<br>Candidate for the Degree of<br>Master of Science

## Thesis: A COMPARISON OF CHANGE IN SELECTED PHYSIOLOGICAL VARIABLES BETWEEN PARTICIPANTS AND NONPARTICIPANTS OF HEALTHY LIFESTYLE CLASSES

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