RISK OF CORONARY HEART DISEASE, DIETARY FAT MODIFICATION, STAGES OF CHANGE, AND SELF-EFFICACY IN SURGICAL AND NATURAL POSTMENOPAUSAL WOMEN

A DISSERTATION

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BY

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March 31, 1998
Date

To the Associate Vice President for Research
and Dean of the Graduate School:

I am submitting herewith a dissertation written by Nancy D. Schwab entitled "Risk of Coronary Heart Disease, Dietary Fat Modification, Stages of Change, and Self-Efficacy in Surgical and Natural Postmenopausal Women." I have examined the final copy of this dissertation for form and content and recommend that it be accepted in partial fulfillment of the requirements for the degree of Doctor of Philosophy, with a major in Nursing.

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We have read this dissertation and
recommend its acceptance:

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ABSTRACT

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The purpose of this nonexperimental two-by-two design study was to determine whether a difference exists in postmenopausal women between those who have undergone surgical menopause and those who have experienced natural menopause and between those who are taking hormone replacement therapy (HRT) and those who are not taking HRT on the variables of risk of coronary heart disease (CHD), dietary fat modification, stages of change, and self-efficacy. The framework for the study was Bandura's self-efficacy theory and Prochaska and DiClemente's transtheoretical model of change.

Seventy-seven subjects were recruited from an internal medicine physician's practice at one large metropolitan multispecialty outpatient clinic. Instruments were the Demographic Data Form, Anderson et al.'s Risk of Coronary

Heart Disease Table, Kristal et al.'s Food Habits Questionnaire, Curry et al.'s Stage of Change to Dietary Fat Reduction, and Sallis et al.'s Self-Efficacy for Health-Related Diet Behaviors. The independent variables--type of menopause and hormone status--had two levels. The four dependent variables were: CHD risk, dietary fat modification, stages of change, and self-efficacy.

Two-way ANOVAs were used to determine differences between the two independent variables on the dependent variables. Findings indicated that those women who experienced natural menopause and were not on HRT were at greater risk for CHD than those women who underwent surgical menopause, the majority of whom were on HRT. The majority of women who experienced surgical menopause were on HRT. In addition, those women with natural menopause had a greater risk of developing CHD over time than their counterparts. These significant findings indicated that women who experience natural menopause should receive education on the risks and benefits of HRT. Hopefully, this education will enhance their ability to wisely choose hormone replacement therapy.

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INTRODUCTION

Coronary heart disease (CHD) is the number one cause of death in the United States for both men and women. It is responsible for approximately one half of all deaths in men and women. Women develop CHD 10 years later than men, and they develop myocardial infarctions 20 years later than men (American Heart Association, AHA, 1994; Douglas, 1993; Flavell, 1994). Even though dramatic declines in mortality have occurred over the past two decades, the decline for women has been less than for men. The rationale for this difference in coronary morbidity and mortality between men and women is multifactorial and unclear, but some research shows the difference to be related to a female's better tolerance of coronary risk factors due to protective hormonal and metabolic functions (Douglas, 1993; Herd, Farmer, & Snabes, 1995).

Epidemiological data have demonstrated that men and women with few CHD risk factors are less likely to suffer a cardiac event. Additionally, reducing CHD risk factors prevents death and disability from CHD (AHA, 1994; Dawber, Kannel, & Lyell, 1963).

One of the controllable risk factors in both men and women is that of dietary intake of products known to increase blood cholesterol. Of all Americans, 25% have cholesterol levels above 240 mg/dl (Schell, 1990). For every 1% reduction in cholesterol, there can be a 2% reduction in the risk of CHD (Schrott, 1988). Healthy People 2000 (U.S. Department of Health & Human Services, DHHS, 1990) reflects a desire to reduce Americans' blood cholesterol levels to an average of no more than 200 mg/dl, which is a 6% overall decrease.

During menopause women experience an increase in total cholesterol, low density lipoproteins (LDL), and triglycerides. Their high density lipoproteins (HDL) are lowered by the loss of ovarian function (Herd et al., 1995). Twenty-seven percent of all women and 50% of women aged 55-74 years are candidates for dietary intervention. Researchers have shown that these women respond positively to dietary intervention (Kris-Etherton & Krummel, 1993). Additional research on hormone replacement therapy (HRT) (estrogen and progestins) has demonstrated that postmenopausal women can lower their incidence of CHD by 50% (Stevenson, Crook, Godsland, Collins, & Whitehead, 1994).

The primary users of HRT are women who have their ovaries removed before the age of 50. The most recent data

on hormone use were obtained from the North American Menopause Society. Their Gallup Poll in 1993 revealed that of 833 women randomly selected, 34% were on estrogen or estrogen and progestogen replacement and that 58% had never used hormones. Of these 833 women, 34% (n = 280) were in the climacteric, 20% (n = 166) were premenopausal and 43% (n = 360) were postmenopausal (Voda, 1994). HRT lowers LDL cholesterol and raises HDL cholesterol in postmenopausal women (Herd et al., 1995). However, the National Cholesterol Education Program recommends that dietary modification be combined with HRT in females over the age of 55 (DHHS, 1993).

Most current research on HRT and menopause in women has been done epidemiologically, and results are varied and conflicting. In general, HRT does lower the risk of coronary heart disease, but recent Framingham Study data suggest that there is an increase in the risk of coronary heart disease. The differences in those postmenopausal women on HRT and those not on HRT and their coronary risk factors were addressed. The relationship of a low fat diet to cholesterol levels and other coronary heart disease risk factors in those postmenopausal women who have experienced surgical and natural menopause also were measured. The majority of postmenopausal women using HRT are candidates

for dietary modification. Surgical intervention with a hysterectomy exposes these postmenopausal women to the choice of HRT sooner than postmenopausal women who have experienced natural menopause.

Hysterectomy is one of the most frequently performed major surgical procedures in the United States, even though only half of the population is at risk for the procedure. About 50% of women within 5 years of their hysterectomy develop ovarian failure and require HRT. With an oophorectomy (bilateral or unilateral), an abrupt fall in estradiol occurs, so these women often have severe menopausal symptoms. The women who undergo these procedures are 10-20 years younger than the women who experience natural menopause and are therefore exposed earlier to the need for HRT and its concomitant benefits for the reduction of CHD (Griffin et al., 1993; Voda, 1994; Youngkin & Davis, 1994). Therefore, those women who select HRT are preventing the apparent onset of heart disease at an earlier age and are thus more health promoting.

Women who undergo a natural menopause usually develop symptoms and an increased risk for CHD more gradually over a number of years. Therefore, they are less likely to choose HRT and benefit from the hormones over a long period of time (Utain, 1993; Voda, 1994).

Modifying eating habits requires a change in behavior. Long-term behavioral habits are often difficult to change because these habits have been developed over time and are resistant to modification. Knowledge of risk behaviors alone is not sufficient to change behavior (Damrosch, 1991; Prochaska, 1991).

Research documents that change processes employing stages of change are essential to effect health behavior change (Damrosch, 1991; DiClemente & Prochaska, 1985; Feury, 1990; Marcus et al., 1992; Marcus & Simkin, 1992; Prochaska & DiClemente, 1992b). Langeludecke (1986) reviewed a number of research studies in which individual behavior intervention strategies were identified as the key to having a lasting effect on lowering CHD. Current nursing literature related to health behavior change was focused more on interventions such as client education and client compliance. Few researchers reported using the stages of change to bring about a change in health behavior.

The traditional conceptualization of behavior change is thought to be a dichotomous event in which discrete categories exist (Prochaska, 1991). Another way to review behavior change is as a process in which individuals move through discrete stages in a dynamic, cyclical fashion to achieve a maintained alteration or initiation of a behavior.

Individuals are thought to progress through these stages at varying rates, with some subjects stalling at certain stages and others relapsing back to earlier stages (Prochaska & DiClemente, 1992b).

In addition to the stages of change, the construct of self-efficacy has been shown to be valuable in exploring successful changes in behavior. Self-efficacy is defined as the perception that an individual can successfully accomplish the behavior or task (Bandura, 1977b, 1987). There have been a few researchers (DiClemente, 1981, 1986; DiClemente, Prochaska, & Gibertini, 1985; Marcus, Selby, Niaura, & Rossi, 1992) who linked the theory of self-efficacy (Bandura, 1977b) and the stages of behavior change for smoking (Prochaska & DiClemente, 1992b). The results of these studies have shown high self-efficacy influencing the action and maintenance stages of change.

Behavior change can be a challenging and rewarding experience, but it requires commitment and incentive. Since readiness to change varies in a dynamic, cyclical fashion and proceeds through stages (Prochaska & DiClemente, 1992b), the more successful an individual is at making behavior changes (high self-efficacy) the more likely the individual will be to continue to pursue new and additional healthy behaviors. Individuals who have adopted health promotion

and disease prevention activities will be able to change additional behaviors with less effort (Bandura, 1977b).

The study included two groups of postmenopausal women: (1) those who have undergone surgical menopause, and (2) those who have experienced natural menopause. The issue of HRT also was explored in this study. Therefore, it was postulated that findings would demonstrate at which age menopausal women are most health promoting (Lichtman & Papera, 1990; Youngkin & Davis, 1994).

Problem of Study

The purpose of the study was to determine whether a difference exists in postmenopausal women, the type of menopause (surgical and natural), and their hormone status on the variables of risk of coronary heart disease, dietary fat modification, stages of change, and self-efficacy for health-related diet behavior. Application of self-efficacy theory (Bandura, 1977b) to Prochaska and DiClemente's (1992b) transtheoretical model of change was incorporated in defining the stages of change and determining the influence of the construct of self-efficacy for health-related dietary behavior change on these stages. Risk of coronary heart disease, dietary fat modification, stages of change and self-efficacy were assessed in application to these postmenopausal women. The variables of type of menopause

surgical and natural) and hormone status in postmenopausal women were studied in an attempt to determine if those women who have undergone surgical menopause were more focused on health promotion strategies than their natural menopause counterparts.

Rationale for Study

According to Healthy People 2000 (DHHS, 1990), the 1980s brought a major decline in death rates among Americans from heart disease. Much of this progress in decreasing mortality mirrors reductions in risk factors. It was estimated that 190,000 lives were saved through reduction in serum cholesterols (Goldman, 1990). However, heart disease remains the leading cause of mortality in men over 45 years and in women over 65 years (Pyorola, DeBaker, Graham, Poole-Wilson, & Wood, 1994).

The national focus on the promotion of health and prevention of disease has lent impetus for nurses to study strategies that are necessary to bring about changes in health behavior aimed at promoting health. In the areas of CHD, there have been many studies on disease prevention and health promotion, but no studies were found which combined the variables of surgical and natural menopause, risk of coronary heart disease, dietary fat modification, stages of change and self-efficacy.

Postmenopausal women who were selected for this study were those who had undergone surgical menopause and those who had experienced natural menopause. Their hormone status was also ascertained. The literature cited that most HRT users have had surgical menopause. Thus, women who have undergone surgical menopause use HRT earlier and are more aware of the beneficial effects of HRT on risk of CHD (Voda, 1994). These women should have adopted more health-promoting behaviors compared to their counterparts who experienced natural menopause. In addition, nurses are interested in the screening and identification of at-risk populations, development of interventions to screen this population, and developing prevention strategies for them (Lichtman & Papera, 1990; Voda, 1994; Youngkin & Davis, 1994).

Postmenopausal women were selected for this study because alterations in risk of CHD is often overlooked in these women. As Douglas (1993) and Flavell (1994) stated, health care professionals recognize that many forms of heart disease are affected by gender and that hormonal and metabolic differences between men and women have an impact on the progression of coronary heart disease.

It is imperative that cardiac risk factors and lipid profiles be evaluated in this population in addition to hormonal status in order to predict the risk of developing

cardiac disease. HRT research in menopause is currently epidemiologic. Results from some studies supported the premise that HRT lowers serum cholesterol and others did not (Stampfer & Colditz, 1991; Stampfer et al., 1991; Postmenopausal Estrogen/Progestin Interventions (PEPI) Trial, 1995; Wilson, Anderson, Harris, Kassel, & Estelli, 1994). Thus, it is important that the focus of the study was to investigate HRT along with dietary modification to determine if a difference exists between the two groups of postmenopausal women, those who had undergone surgical menopause and those who had experienced natural menopause.

Cholesterol has been documented through research as a major contributor to CHD morbidity and mortality. The loss of economic productivity in 1980 attendant to illness and early death from CHD equaled nearly 18% of the gross national produce (GNP). The cost of CHD has put a major burden on U.S. health care expenditures ($50-$100 billion per year), and it is projected that the cost of CHD will continue to rise due to the increase in the size of the aging population (Stason, 1990). Menopausal women often experience more complications, have poorer response to therapy, need a longer time for recovery, and return to work less often than men. This nation cannot afford to care for these women with CHD in acute care settings when their

health problems can be affected by risk reduction and prevention strategies (DHHS, 1990, 1993; Douglas, 1993). Calculating the odds ratios for women with angina pectoris and disability, women 55 to 69 years of age experienced disability at the onset of angina pectoris (odds ratio 5.87; 95% confidence interval (CI) = 3.30%, 10.43%). Therefore, angina pectoris proved to be a better predictor of disability than myocardial infarction or coronary insufficiency (Pinsky, Jette, Branch, Kannel, & Feinleib, 1990).

No data were found on the association between CHD risk and health care costs. However, in two recent articles utilizing Framingham Heart Study data, Anderson, Wilson, Odell, and Kannel (1991) and Pyorola et al. (1994) reported a regression model predicting the average level of risk for CHD, which is associated with Medicare claims cost that are 19% higher (95% CI = 10%, 29%) than those for persons with no elevated risk. This CHD risk equation was developed for use by clinicians in predicting the development of CHD in individuals free of disease. This association is equivalent to an average of $9.3 billion per year in Medicaid expenses. In addition, a total blood cholesterol level of 260 mg/dl is associated with 6% (95% CI = 3%, 9%) higher Medicare costs, compared with a total blood cholesterol of 180 mg/dl.

Postmenopausal women (surgical and natural) were compared on the variables of risk of coronary heart disease, dietary fat modification, stages of change, and self-efficacy to determine whether the women who have undergone surgical menopause have adopted more health promotion strategies related to lowering cholesterol than their natural menopause counterparts. If this difference exists, health care costs may be reduced. For every 1% reduction in cholesterol, there is a 2% reduction in the risk of CHD (Schrott, 1988). This fact documents that lowering cholesterol levels should lower CHD and health care costs.

Utilizing a stage approach of behavior change recognizes an individual's place on a continuum of change and allows specific interventions to be defined and implemented to move the individual along the stages toward permanent change. Nurses need to assess the usefulness of a stage model to effect changes in people's health behavior. Staging individuals and then defining interventions which promote and document health behavior change can contribute to cost-effective health care.

Theoretical Framework

Bandura's self-efficacy theory (1977b) and Prochaska and DiClemente's transtheoretical model (1992b) provided the framework guiding this study. The second assumption of

self-efficacy theory (Bandura, 1977b), states that "one major source of human learning is response consequences or learning by doing" (p. 35). Assumption five of the transtheoretical model (Prochaska & DiClemente, 1992b) states "there are five basic stages of change: precontemplation, contemplation, preparation, action, and maintenance" (p. 303). These two statements were the basis of the theoretical approach to this study.

Self-Efficacy Theory

Bandura's self-efficacy theory (1977b), which arose from social learning theory, concerns the effects of self-referent thought on psychosocial functioning. The basic assumption of Bandura's self-efficacy theory is that psychological procedures, whatever their form, alter expectations of self-efficacy. Bandura's model for self-efficacy consists of a person engaging in a behavior that will lead to a consequent outcome (Figure 1). In this model, two important expectations are depicted, efficacy expectations and outcome expectations. "An outcome expectation is defined as a person's estimate that a given behavior will lead to certain outcomes. An efficacy expectation is the belief that one can successfully execute the behavior required to produce the outcomes" (Bandura, 1977b, p. 79).

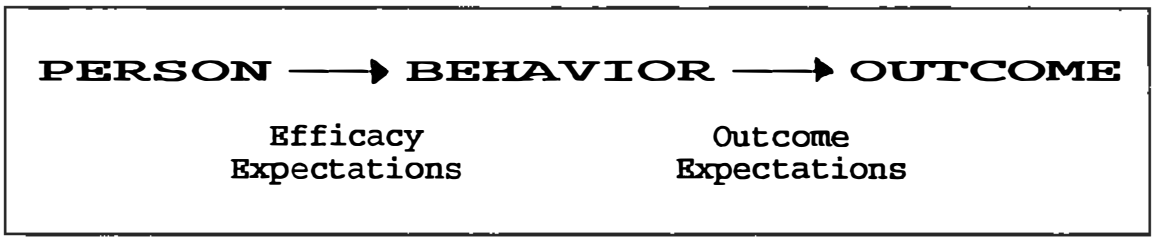


Figure 1. Bandura's (1977b) Self-Efficacy Theory

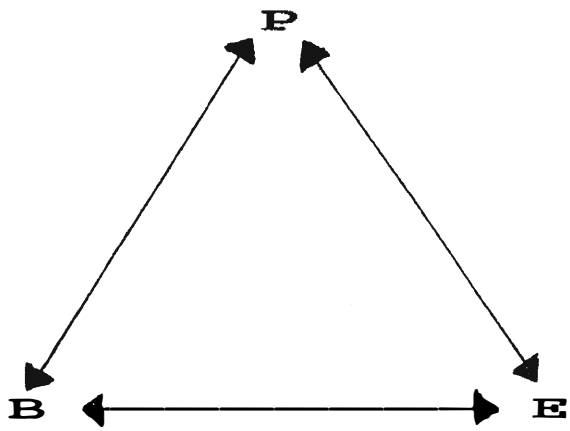
Bandura (1977b) emphasized that outcome and efficacy expectations differ because people may believe a specific course of action will lead to certain outcomes, but question the capability to perform those actions. Thus, it is peoples' perception that most heavily influences their behaviors. The strength of peoples' beliefs in their own ability to deal with difficult situations determines whether they will even attempt to cope with these situations. Therefore, efficacy expectations are the most significant aspect of Bandura's theory and correlate with the construct of self-efficacy.

Interventions that have an impact on self-efficacy expectations can assist individuals with risk-reducing behavioral choices. Persistence can be crucial in maintaining a desired behavioral change in individuals who chose a behavior such as modifying the diet to lower fat intake (Jenkins, 1988).

The theory also proposes an interactional process called reciprocal determinism (Figure 2) as its base. This process emphasizes internal and external influences as well as the environment interdependently contributing to human behavior. It is through efficacy expectations, cognitive processing, and self-direction that a resulting reciprocal interaction between the individual's internal personal factors, behavior, and the environment occurs. Bandura (1977b) described the internal personal factors, with which the person is continually interacting, as beliefs, thoughts, preferences, expectations, and self-perceptions. The environment provides efficacy information to the person through interactions and observations. The internal personal and environmental factors may occur as a result of personal experiences or through observations of events or people (Bandura, 1977b). In addition, the environment continually provides information through response consequences, which determine an individual's probability of success or failure at attaining a behavior.

Transtheoretical Model

The transtheoretical model, based upon various psychotherapy theories, was first developed by Prochaska (1979, 1984) as 10 processes of change. The model was



KEY:

B = Behavior
P = Personal Factors
E = Environmental Factors

Figure 2. Reciprocal Interaction Model (Bandura, 1977a, p. 345)

refined by Prochaska and DiClemente (1992b) to include stages and levels of change.

There are three basic elements of the transtheoretical model: processes of change, stages of change, and levels of change (Prochaska & DiClemente, 1992b). The transtheoretical model of behavior change is depicted in Figure 3.

Although the research was guided by the transtheoretical model, it was focused upon the second element, stages of change (Figure 4). The word individual is used to define each stage of change in Figure 4; Prochaska and DiClemente (1992b) identified these as the stages of change and not processes.

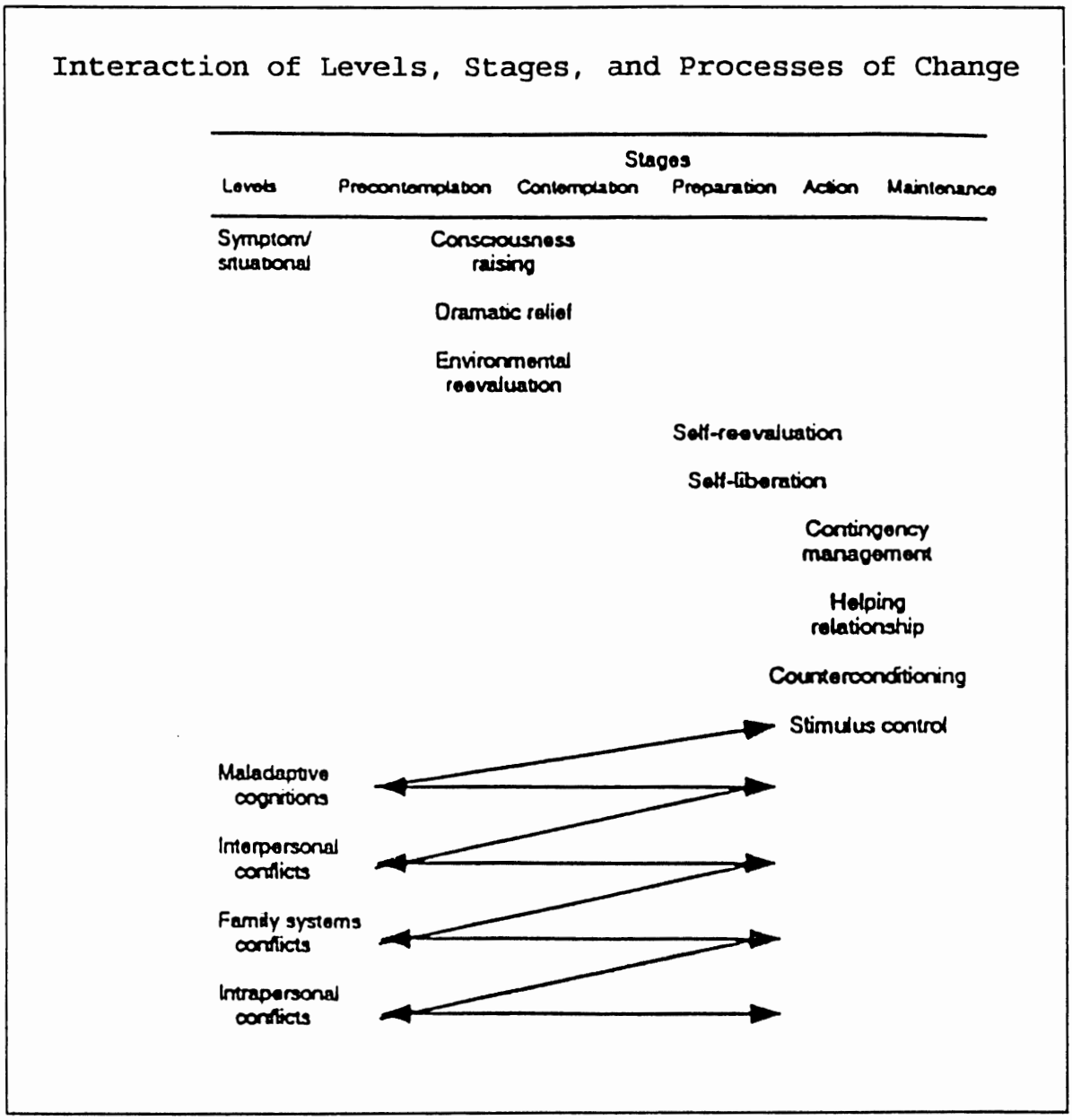


Figure 3. Transtheoretical Model (Prochaska & DiClemente, 1992b, p. 308)

STAGE	DEFINITION
Precontemplation	Individuals who do not perform the behavior and have no intention of performing the behavior.
Contemplation	Individuals who do not perform the behavior, but intend to perform it.
Preparation	Individuals who perform the behavior, but not regularly.
Action	Individuals who currently began to perform the behavior regularly.
Maintenance	Individuals who perform the behavior regularly over a long period of time.

Figure 4. Stages of Change (Prochaska & DiClemente, 1992b)

Three stages of change (preparation, action, and maintenance) were integrated with the efficacy expectation portion of self-efficacy theory (Bandura, 1977b) (Figure 5). The stages define change as an intentional process with movement through them, and they have been defined in respect to studies on various outpatient populations (DiClemente & Hughes, 1985; McConnaughy, DiClemente, Prochaska, & Velicer, 1989; McConnaughy, Prochaska, & Velicer, 1983).

Two foci have been represented by the stages of change: (1) a period of time, and (2) a set of tasks for behavior

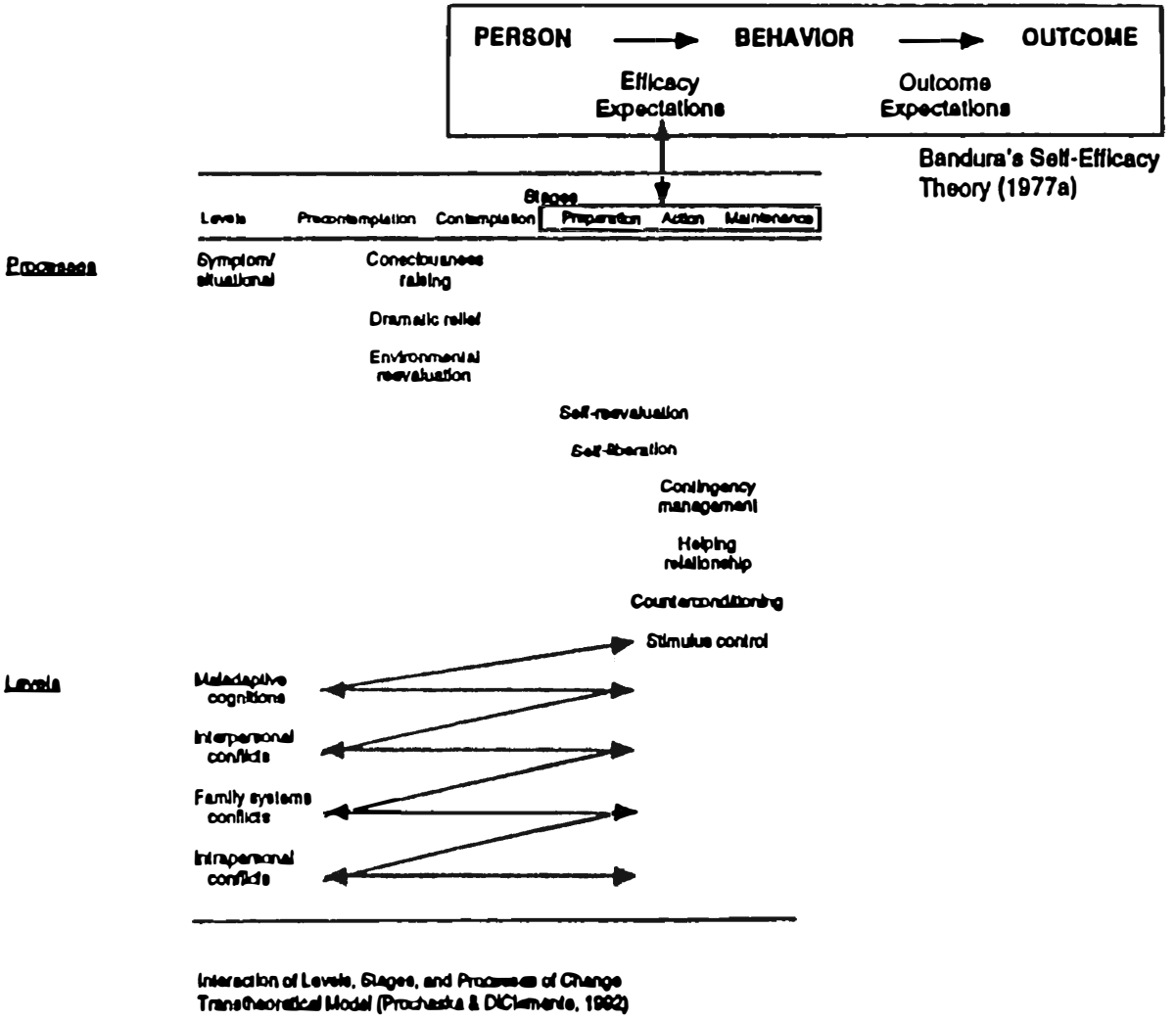


Figure 5. Interface of Bandura's (1977b) Self-Efficacy Theory and the Transtheoretical Model (Prochaska & DiClemente, 1992b, p. 308)

change. Both are needed for movement to the next stage. The time spent in each stage may vary, but the tasks for behavior change in each stage must be accomplished before the individual moves to the next stage. The stages of change represent the temporal, motivational, and constancy aspects of change (DiClemente & Prochaska, 1985).

The transtheoretical model and its relationship to self-efficacy theory are discussed initially. Then the interface of the model and the theory are related through one assumption from the model and one assumption from the theory. The construct of self-efficacy (Bandura, 1977a, 1977b) has been shown to successfully influence behavior change and affect the stages of change (Prochaska & DiClemente, 1992b).

Bandura's (1977b) model for self-efficacy consists of a person engaging in a behavior that will lead to a consequent outcome. Research has been undertaken using the construct of self-efficacy and has demonstrated its positive influence on behavior change (Bandura, Adams, Hardy, & Howels, 1980; Bernier & Avard, 1986; Champliss & Murray, 1979; Condiotte & Lichtenstein, 1981; Feltz & Weiss, 1982; Marcus & Owen, 1992; O'Leary, 1985).

The interface between the two models and the focus of this study occurs at the point of intersection between

efficacy expectations and their influence on the preparation, action, and maintenance stages of change. Efficacy expectations determine the amount of effort people will expend and length of time they will persist in the face of obstacles and aversive experiences to complete tasks. The stronger the efficacy expectation, the more persistent the efforts. Those who consistently perform activities that are subjectively threatening will gain experiences that further reinforce their sense of efficacy thereby eliminating their fears and defensive behaviors (Bandura, 1977b). DiClemente (1981, 1986; DiClemente & Prochaska, 1985) has shown a relationship between self-efficacy effecting behavior change through its relationship with the stages of change. Therefore, it is not only important to define the client's stage of change, but also the level of self-efficacy in order to have greater insight into an individual's process of behavior change.

However, self-efficacy theory and the transtheoretical model have not been used in nursing studies. They lend themselves to nursing research, however, because behavior change is supported and encouraged by nurses. The goal of nursing is the promotion of health among individuals. Many nursing conceptual frameworks identify the need for definition of mechanisms that underlie human health

processes, but these models often lack specific operational mechanisms to accomplish this definition. Self-efficacy and the transtheoretical model provide an operational framework for the study that was designed to test differences and relationships in two groups of postmenopausal women related to self-efficacy and stages of change.

Integration of Self-Efficacy Theory and the Transtheoretical Model

The intersection between these two models and the focus of this study is between efficacy expectations, which are derived from response consequences, and their influence on the preparation, action, and maintenance stages of change. Processes which are influential in effecting health promoting behaviors at the point of intersection of this theory (Bandura, 1977b) and model (Prochaska & DiClemente, 1992b) are motivation and modeling. Motivation in the form of external and vicarious reinforcement through present and past life experiences may influence these behaviors resulting in valuable benefits, no appreciable effects, or aversion of future problems. Modeling provides visual stimuli that may facilitate performance of a behavior in the future (Bandura, 1977b).

Response consequences are largely cognitive. They result in actions that have either a positive or a negative

effect on the individual. Successful actions are eventually selected, and ineffectual actions may be discarded after experiences with changing behavior. This concept correlates with the notion that efficacy expectations vary on the dimensions of magnitude, strength, and generality (Bandura, 1977b). Response consequences impart information (cognitive process), act as motivators (through positive and negative responses), and strengthen an individual's responses through repetition of the correct response.

Prochaska and DiClemente's (1992b) preparation and action stages are defined as being ready to change in the near future and acquiring valuable lessons from past change attempts and failures. To take action, goals are set and prioritized. In addition, an action plan is developed. The action stage has both a cognitive and affective component. The affective component is greater during this stage and is defined as the belief that one's own efforts play a critical role in succeeding in the face of difficult situations. Individuals learn to cope with coercive forces that are a part of life and may cause relapse. Autonomy is also important at this stage. The process of change, called self-liberation by Bandura (1977b), is dominant during these stages. Bandura's process of change is defined as an individual's choice and commitment to change the problem

behavior. Self-liberation includes the belief that one can change. This process is based on a sense of self-efficacy (Prochaska & DiClemente, 1992b) particularly efficacy expectations (Bandura, 1977b).

Despite the numerous citations in the health promotion and disease prevention literature, few investigations have addressed the issue of self-efficacy and the stages of change as essential to decrease mortality and morbidity from cholesterol related CHD in menopausal women. In addition, no studies were found addressing this issue with women who experience surgical versus natural menopause.

Health promotion and disease prevention have been touted as means of controlling the spiraling cost of health care. The nurse as a health care provider can play a vital role in assisting individuals to engage in behaviors that may modify certain risk factors.

Assumptions

The assumptions for the study were based on Bandura's (1977b) theory of self-efficacy and Prochaska and DiClemente's (1992b) transtheoretical model. The assumptions underlying this study were:

1. One major source of human learning is response consequences or learning by doing (Bandura, 1977b).

2. The motivational process is influenced by (1) external reinforcement, (2) vicarious reinforcement, and/or (3) self-reinforcement (Bandura, 1977b).
3. Exposure to a model may produce different effects on learning (Bandura, 1977b).
4. There are five basic stages of change: precontemplation, contemplation, preparation, action, and maintenance (Prochaska & DiClemente, 1992b).
5. Stages of change reflect the temporal and motivational aspects of change with gradual movement through the stages (Prochaska & DiClemente, 1992b).

Research Questions

The research questions for the study were:

1. Is there a difference between postmenopausal women who have undergone surgical menopause and those who have experienced natural menopause with regard to risk of coronary heart disease, dietary fat modification, stages of change, and self-efficacy for health-related diet behaviors?
2. Is there a difference between hormone replacement therapy (HRT) and no HRT in postmenopausal women with regard to risk of coronary heart disease, dietary fat modification, stages of change, and self-efficacy for health related diet behaviors?

3. Is there an interaction between the use of HRT and the type of menopause, natural or surgical, with regard to risk of coronary heart disease, dietary fat modification, stages of change, and self-efficacy for health-related diet behaviors?

Definition of Terms

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

1. Dietary fat modification: selecting and maintaining a low-fat diet by measuring an individual's dietary patterns through categorizing the available foods eaten daily and weekly by their frequency of use. The term was operationally defined as the score on the Food Habits Questionnaire (Kristal, Shattuck, & Henry, 1990).
2. Hormone replacement therapy (HRT): use of synthetic or natural estrogen alone or with a progestin to alleviate the physical and emotional symptoms of menopause. The HRT regimes may consist of unopposed estrogen in women without a uterus, or combined estrogen and progestin in women with a uterus (Speroff, Glass, & Kase, 1995). HRT was operationally defined as a response of yes to question 5h on the Demographic Data Form.
3. Postmenopause: used to describe the stage when ovulation ceases entirely and the cessation of menses

occurs. Postmenopause is characterized by the complete termination of ovarian activity and signs of estrogen decline. During postmenopause, the majority of estrogen production occurs via the conversion of estrogen from adrenal androstenedione which causes numerous changes to specific target organs (Lichtman & Papera, 1990; Speroff et al., 1995). For the purposes of this study, two types of postmenopause were defined:

a. Surgical postmenopause: includes removal of the uterus alone or in combination with removal of one or both ovaries during a surgical procedure (Voda, 1994), which leads to immediate cessation of menstruation. This term was operationally defined by answering question 5e on the Demographic Data Form.

b. Natural postmenopause: Postmenopause begins one year after the cessation of menstruation without surgical intervention. This term was operationally defined by answering question 5e on the Demographic Data Form.

4. Risk of coronary heart disease: based on The Framingham Heart Study, this term is widely used to describe those characteristics found in healthy individuals that have been found in observational, especially epidemiological,

studies to be related to the subsequent occurrence of coronary heart disease. The term risk factor includes modifiable lifestyles, biochemical, and physiological characteristics, as well as nonmodifiable personal characteristics such as age, sex, and family history of early onset coronary heart disease. The term was operationally defined as the score on the Risk of Coronary Heart Disease Table (Pyorola et al., 1994).

5. Self-efficacy: "the certainty with which a person feels he or she can accomplish a specific task" (Bandura, 1977b, p. 194). This term was operationally defined as the scores on the Self-Efficacy for Eating Behaviors Scale of the Self-Efficacy for Health-Related Diet Behavior Questionnaire (Sallis, Pinski, Grossman, Patterson, & Nader, 1988).
6. Stages of change: "an intentional process with movement through five stages of change: precontemplation, contemplation, preparation, action, maintenance" (Prochaska & DiClemente, 1992b, p. 303). This term was operationally defined as the score on the Stage of Change to Dietary Fat Reduction Questionnaire (Curry, Kristal, & Bowen, 1992).

Limitations

A convenience sample of postmenopausal women who had undergone surgical menopause or who had experienced natural menopause was selected from a large multispecialty clinic in Houston. Thus no random sampling was used. In addition, it was not possible to control whether the women were on hormone replacement therapy (HRT) or not and what type of HRT they had been prescribed.

The hormonal factor data and menopause status were collected on the Demographic Data Form and were used to describe the sample. These data also were used to compare hormone replacement status of those postmenopausal women who were or were not on HRT with the four dependent variables: risk of coronary heart disease, dietary fat modification, stages of change, and self-efficacy for health-related diet behavior change. Therefore, the findings of the study cannot be generalizable beyond the sample studied because there was no random sampling (purposive sampling) and the investigator was not able to control for the types of women selected for the study. The weaknesses inherent in purposive sampling can be overcome by using knowledge, expertise, and care in the selection of the sample, conservative interpretation of the results, and replication of the study with different samples (Kerlinger, 1986). The

advantage of this type of research lies in the practicality and low cost in obtaining the sample. The data for the study were collected at one point in time for the purpose of determining differences in the two groups (postmenopausal women who had undergone surgical and those who had experienced natural menopause, including their hormone status).

Summary

Self-efficacy theory (Bandura, 1977b) and the transtheoretical model (Prochaska & DiClemente, 1992b) provided the framework to determine the differences in two groups of postmenopausal women (surgical and natural menopause). The variables to be studied were: risk of coronary heart disease, dietary fat modification, stages of change, and self-efficacy.

CHAPTER 2

REVIEW OF THE LITERATURE

Health promotion and disease prevention have been the focus of improving Americans' health for almost 20 years. Defining risk factors and lifestyle habits for diseases, such as coronary heart disease (CHD), and developing prevention strategies aimed at limiting the occurrence of this disease have been the thrust of current research in women's health.

Interest in women's health and issues related to women has undergone a recent upsurge. Currently, women experience a longer life span than men and develop CHD later. This disease is the leading cause of death and disability in women over the age of 40. Health care professionals have a duty to establish a scientific knowledge base on women's health issues, to provide health care to women based on appropriate biological and medical indications, and to facilitate the adoption and continuance of behaviors that emphasize a healthy lifestyle.

Current literature relevant to the study on factors which influence women and their CHD health promotion practices was reviewed and categorized into the following

related sections: women and CHD; women and CHD risk factors including diabetes mellitus, hypertension, smoking, and cholesterol and menopause; hormone replacement therapy; prevention practices for menopausal women with risk of CHD; and behavior change. Included in the behavior change section is a historical overview of self-efficacy theory (Bandura, 1979a) and the transtheoretical model (Prochaska & DiClemente, 1992) with relevant research pertinent to the study as well as current research linking the two models. The purpose of this literature review was to join these various areas together and provide a basis for this study so that results may be measured.

Women and CHD

The life span of women has increased and so has the incidence of CHD. More than 500,000 women die of CHD each year. Twice as many women die of cardiovascular disease than of cancer, and the number of women who die of CHD is 50% from any medical practice (American Heart Association, AHA, 1996; Castelli, 1988; Flavell, 1994). With each decade of life, the rate of death from CHD increases threefold to fivefold. By the ages of 75 to 84, the death rate for women is 2,590 per 100,000 population (Eaker et al., 1993).

Cardiovascular diseases are also the leading cause of disability in women. Estimates of the percentage of women

with CHD who are disabled varied from 36% (age 55-64) to 55% (age 75 and older) (Eaker et al., 1993).

Cost is another area in which the impact of CHD can be determined to affect society. In 1997, the cost of cardiovascular disease was estimated to be $259.1 billion. CHD alone is estimated to cost $167.2 billion (AHA, 1996). Women incur more than half (58%) of the year health care cost related to CHD, although their death rate from CHD ranges from one fourth to almost equal that of men, depending on the age group (Eaker et al., 1993). A significant rise in the amount of the gross national product (GNP) allocated to medical services (5% in 1960 compared to an estimated 12% in 1990) was expected. The reason for this increase in GNP is related to the rapidly growing elderly population (Kern, 1990; U.S. Department of Health and Human Services, DHHS, 1990). By the year 2000, 38% of all women in the U.S. is expected to be 45 years old or older. By the year 2015, this proportion will increase to 45%. With this increase in a population of older women, CHD is most likely to be even a greater health problem. While the overall death rate from CHD is declining, the number of women dying from CHD continues to grow every year (Eaker et al., 1993).

Prevention is critical because 40% of all coronary events in women are fatal and 67% of all sudden deaths in

women occur in those without a history of CHD. The risk factors for cardiovascular disease in women are known, but much less is known about the modification of these risk factors and its effect on the reduction of risk in women.

Women and CHD Risk Factors

Among the risk factors that are thought to influence the progression of CHD are the unmodifiable ones--family history, age, racial/ethnic background, and the modifiable ones--diabetes, hypertension, smoking, menopause, cholesterol, obesity, sedentary lifestyle, and psychosocial factors. The risk factors that are pertinent for the study are diabetes mellitus, hypertension, smoking, menopause, and cholesterol. Each of these risk factors is described in relation to women.

Diabetes Mellitus

In the U.S., 7,800,000 persons have diabetes mellitus (DM), including 3.6 million males and 4.2 million females. The mortality data in 1993 for this chronic controllable disease was 23,430 male deaths (43.5% of total deaths from diabetes) and 30,464 female deaths (56.5% of total deaths from diabetes). More than 80% of the people with DM die of some form of cardiac or peripheral vascular disease, which

makes this disease an important cardiac risk factor (AHA, 1996)

From 20 years of prospective Framingham Heart Study data, according to Kannel and McGee (1979), the levels of cardiovascular risk factors were found to be higher in diabetic than in nondiabetic men and women. The adjusted incidence/1,000 of specified cardiovascular disease was: (a) CHD--DM men 24.8; non-DM men 14.9; DM women 17.8; non-DM women 6.9; and (b) CV death--DM men 17.4; non-DM men 8.5; DM women 17.0; non-DM women 3.6. In addition, the incidence of cardiovascular mortality was greater in women (risk factor adjusted--women 3.3, men 1.7).

The prevalence data for diabetes increased with age in both sexes in the Framingham cohort. The overall prevalence for women was 6.2% and for men, 7.8%. Among diabetic women the incidence of cardiovascular disease was almost three times that among nondiabetic women. In addition, the impact of diabetes (17.2 of the age adjusted annual rate) on CHD exceeded that of smoking (12.1 of the age adjusted annual rate) on CHD (Kannel & McGee, 1979).

Among those with diabetes, the female advantage over the male with regards to cardiovascular mortality is lost. With adjustment for differences in other associated cardiovascular risk factors, the impact on women was greater

for CHD (risk factor adjusted men 1.7; women 2.1). The reason for this difference is not clear. In the diabetics, the high density lipoprotein (HDL) cholesterol values were consistently lower than in those without diabetes in both sexes (143 women DM, $\underline{t}$ = 1.44; 1,215 women non-DM, $\underline{t}$ = 0.44, $p < .01$). Diabetic individuals also had a higher ECG-LVH documentation and higher levels of low-density lipoprotein than non-DM women. There was no significant difference in LDL levels in 143 DM versus 1,215 non-DM women (Douglas, 1993; Kannel & McGee, 1979).

Similar to the Framingham Study, findings from the Nurses' Health Study (Colditz et al., 1987) showed a three- to sevenfold increase in CHD events in DM women. The association between DM and the relative risk (RR) of CHD had a greater than sixfold increase in risk for both nonfatal and fatal CHD in age adjusted analysis (381 non-DM women, RR = 1.0; 68 DM women, RR = 6.7). Total cardiovascular mortality was markedly elevated among women with DM (203 non-DM women, RR = 1.0; 35 DM women, RR = 6.3). The age adjusted rate of CHD rose sharply in the presence of smoking, hypertension, and hypercholesterolemia. The results indicated a total of 13.8% of coronary events were attributed to diabetes (Munson, Colditz, & Stampfer, 1991).

Perhaps the association between DM and CHD is evidence that control of blood sugars should be expanded to include modification of all metabolic risk factors to achieve a maximum risk reduction. Therefore, the modification of one risk factor is likely to effect other risk factors, which in turn will cause an overall improvement of the other factors (Flavell, 1994; Kannel, 1987).

Hypertension

Hypertension (HTN) is both a disease and a risk factor for CHD. Currently it is estimated that 50 million Americans age 6 and older have high blood pressure. Men are at greater risk for HTN than women until age 55. From age 55 to 74, the risks for HTN in men and women are about equal. After that age, women's risk rises above men. The prevalence of HTN increases with age. In fact, 27% of women aged 18 to 74 years have blood pressures higher than 140/90. When those women are compared with those in the 55 to 64 age bracket, the prevalence increases to 53%. In the over-65 group of women, 67.5% of women will have hypertension (Gorodeski & Utian, 1994).

Blacks develop hypertension at an earlier age than whites. As a result, blacks have a 1.5 greater rate of CHD. The 1993 death rates were 6.5 for white males, 30.0 for

black males, 4.8 for white females, and 22.6 for black females (AHA, 1996).

The great risk for hypertension in the U.S. population at varying ages and in various ethnic groups resulted in the development of a federally funded program (U.S. DHEW, 1973) and prevalence studies as well as community/nationwide risk factor identification studies. In addition, programs to begin the modification and reduction of this risk factor and to define the relationship between HTN and atherosclerosis were initiated.

In 1973, the federally sponsored National High Blood Pressure Education Program (U.S. DHEW, 1973) was responsible for increasing health care professionals' awareness of the detection, evaluation, and treatment of HTN. It was through this program and expanded awareness that three studies, the National Health and Nutrition Examination Survey I (NHNES I, Cornoni-Huntley, LaCroix, & Havlik, 1989), the Community Hypertension Evaluation Clinic (CHEC) Program (Stamler, Stamler, & Riedlinger, 1976), and the Hypertension Detection and Follow-up Program (HDFP), were undertaken. The purpose of all three studies was to investigate the approaches to developing cooperative community action to understand the importance of HTN and the need for treatment as well as the morbidity and mortality associated with this condition.

The Framingham Study (n = 5,127) (Kannel, Gordon, & Schwartz, 1971), which reported 14 years of follow-up data, demonstrated that HTN is a powerful independent predictor of CHD and mortality in both sexes. Kannel et al. determined that systolic blood pressure was a better measure for CHD than diastolic blood pressure, especially in older age groups and in women. As determined by discriminate analysis, systolic blood pressure was the second (to age) most powerful predictor of CHD (coefficients, age = 0.63, systolic BP = 0.56).

There is a paucity of literature on the relationship between hypertension and CHD in black women. Tryoler et al. (1971) included black women in their sample. Findings indicated that neither elevated systolic nor diastolic blood pressure increased the risk of CHD for black women. However, the results demonstrated that the incidence rates of CHD in individuals with HTN were 2.5 times higher for white women, 1.8 times higher for white men, and 4.4 times higher for black men, which were sizable and reflected the changes in systolic blood pressure. The results for diastolic blood pressure were the same for white men and women and black men (2.1 increase).

In trying to determine a reason for women's elevated blood pressure in the later years, some investigators looked

at menopause as a factor. The earliest study done by Taylor, Corcoran, and Page (1947) on menopausal hypertension measured the blood pressure of 200 postmenopausal women (21 = natural menopause; 179 = surgical menopause) and 2,860 industrial and factory workers. Results showed no greater frequency of hypertension. Of the 200 menopausal women who participated in this study, 13% had HTN as compared to the 2,860 industrial and factory workers, who had 14.7%.

Weiss (1972) studied 899 women from age 40 to 51 to determine differences in diastolic and systolic blood pressures between premenopausal and postmenopausal women. Weiss found that postmenopausal women (natural and surgical) had significantly higher levels of diastolic blood pressure than premenopausal women (linear $X^2 = 6.0$, $p = 02$). Means and standard deviations of diastolic blood pressure of both pre- and postmenopausal women were reported in four age groups: 40-42, premenopausal $M = 78.2$ ($SD = 10.3$) and postmenopausal $M = 81.8$ ($SD = 10.6$); 43-45, premenopausal $M = 80.6$ ($SD = 11.7$) and postmenopausal $M = 83.5$ ($SD = 15.7$); 46-48, premenopausal $M = 82.0$ ($SD = 11.4$) and postmenopausal $M = 81.1$ ($SD = 12.1$); and 49-51, premenopausal $M = 81.7$ ($SD = 7.4$) and postmenopausal $M = 83.8$ ($SD = 12.5$). No significant difference was found between systolic blood pressures in these women.

The Framingham Study (Hjortland, McNamara, & Kannel, 1976) showed no relationship between a change in menopausal (natural and surgical) status and changes in systolic or diastolic blood pressures (menopausal *n* = 480, control *n* = 3,117). In another study, Matthews, Kuller, Wing, Meilahn, and Plantinga (1989) also determined there was no change in blood pressure associated with natural menopausal status in 541 women.

It is evident that the influence of menopause on blood pressure is controversial. However, high blood pressure in women was most often previously detected, treated, and adequately controlled.

Smoking

During the 1970s, it was clearly established that smoking is an independent and important risk factor for developing CHD. Smoking is the single most important preventable risk factor for CHD in women. In 1990, about 417,000 Americans died of smoking related illnesses, which cost the U.S. about $50 billion dollars annually in medical care. Smoking is responsible for 50% of the coronary events before the age of 55, which results in a two- to sixfold increase in risk of CHD compared to nonsmokers. In the last decade, smoking has decreased by a rate of 0.3% per year;

however, smoking initiation has increased by 1% per year. Of all women in the U.S., 29% smoke; it is anticipated that in the next 1000 years, women will exceed men in smoking (AHA, 1996; Douglas, 1993; Gorodeski & Utian, 1994).

Several mechanisms are known to cause adverse events in an individual who smokes. These events have various negative effects on the heart and blood vessels. Nicotine is the primary agent that causes an increase in plasma levels of free fatty acids and low density lipoproteins (LDLs), increased platelet aggregation, decreased levels of high density lipoprotein (HDL), and an antiestrogen effect (Flavell, 1994; Gorodeski & Utian, 1994).

Two important studies, the Washington County, Maryland Study (Bush & Comstock, 1983) and the Nurses' Health Study (Willett et al., 1987), lend support to smoking as an important preventable independent risk factor for CHD in women. The Washington County, Maryland study (Bush & Comstock, 1983) had 23,572 subjects. Findings showed that smokers had the higher mortality from CHD when compared to nonsmokers, except in the age group of 65-74 years. The 25-64 years age group, particularly those who smoked more than 20 cigarettes per day, had the highest mean incidence of CHD: 44 smokers, 89.3; 231 nonsmokers, 38.3. In relation

to CHD mortality, the highest relative risks were found in two groups who smoked more than 20 cigarettes per day: (1) younger women (25-44 years) had a relative risk (RR) = 2.5 compared to 1.5 for nonsmokers, and (2) middle age women (45-64 years) had RR = 2.0 compared to -0.05 for nonsmokers. No significant risk was found among the women aged 65-74 years. In addition, the death rates and relative risks (RRs) for CHD mortality increased as the number of cigarettes increased in each age group.

The Nurses' Health Study (Willett et al., 1987) is a large prospective cohort study of 119,404 women. Data revealed that the age adjusted relative risk for CHD in smokers increased with the number of cigarettes smoked. In addition, nonsmokers and ex-smokers had similar rates of fatal CHD: nonsmokers, RR 1.0; ex-smokers, RR 1.5. In other words, the RR rate of ex-smokers dropped almost a whole point lower than the lowest smoker rate (1/4 day, RR 2.4). However, in contrast to findings from the Washington County, Maryland (Bush & Comstock, 1983) study, the RR for CHD was somewhat higher in older women than it was among younger women.

Findings from both the above studies lend support to the fact that cigarette smoking is a important cause of CHD

in men and women. However, should women choose to stop smoking, their risk will become almost equal to those who have never smoked.

Cholesterol and Menopause

The Framingham Heart Study examined the effects of cholesterol on CHD in men and women over the last 40 years. Findings from this study have clearly demonstrated that the higher the level of serum cholesterol, the higher the rate of CHD. Every 1% rise in serum cholesterol and LDL levels increases the risk of CHD by 2% in women. In addition, a 1% decrease in HDL increases the risk of CHD by 2% to 4.7% (Castelli, 1988; Castelli et al., 1986; Gorodeski & Utian, 1994)

As reported by Castelli (1988), only 6 of 1,600 premenopausal women in the Framingham Heart Study had died of CHD. However, after menopause, the incidence rates for CHD rapidly rose to those in men. Women developed CHD at the same rate as men, but approximately 6-10 years later (Castelli, 1988). Findings also showed that around the age of menopause (age 50) the LDL levels became higher in women and the HDL changed very little and often remained higher in women (Barrett-Connor, 1994). Currently, before the age of 45, means for total blood cholesterol of women vary from 185

to 207 mg/dl, but between the ages of 45 and 65, the means increase to between 217 and 237 mg/dl (AHA, 1996). This change in serum cholesterol levels may be related to menopause.

From the United States' Health Examination Survey, 1961-1962 (Weiss, 1972), the first study, 897 women ages 40-51 were selected to participate in a study on the relationship between cholesterol and menopause. This age range was selected so both pre- and postmenopausal women would be included. In addition, the type of menopause (surgical or natural) was included. Findings showed that premenopausal women (n = 428) and postmenopausal women (n = 469) had significantly different cholesterol levels (X^2 (10, N = 897) = 15.3, p < .001). In addition, women who had natural menopause (n = 297) showed no significant difference (X^2 (10, N = 469) = 2.7, NS) from their counterparts who had surgical menopause (n = 172).

Matthews et al. (1989) reported that 541 healthy, initially premenopausal women (ages 42-50) were followed for 2.5 years to evaluate the changes in their lipid levels. Of the women, 69 spontaneously had their menstrual cessation for 12 months and 32 had stopped natural menses and received hormone-replacement therapy for 12 months. Controls for

this study included an equal number of age-matched premenopausal controls. Measurements for the study variables occurred premenstrually (baseline), at the time of menstrual cessation, and for 12 months (follow-up). Natural menopause resulted in changes in LDL and HDL cholesterol levels. The total cholesterol change was not significant from premenopausal to menopausal women. The LDL cholesterol increased twofold from baseline to follow-up visit in the women who experienced natural menopause (0.31 menopausal, 0.14 premenopausal; $p = .04$). The HDL cholesterol level significantly declined from the premenopausal to the menopausal examination (-0.09 menopausal; ± 0.02, $p = .01$). The premenopausal women had no change in the HDL.

The purpose of the third study by Hjortland, McNamara, and Kannel (1976), part of the Framingham Heart Study, was to examine the role of menopause on the atherogenic process of 1,686 women, ages 40 to 51. In comparison to the two previous studies, the Framingham Heart Study periodically examined a large cohort of women longitudinally (nine examination sessions), which facilitated detection of the changes associated with menopause. Of the 1,686 women in this study, 1,119 experienced menopause: 817 had natural menopause and 297 had surgical menopause which included 183

with bilateral oophorectomy, 49 with unilateral oophorectomy, and 65 with hysterectomy alone. The remaining women who had other causes for menopause were eliminated from the study. Only total cholesterol was examined.

Data from this study were analyzed from three examination periods: (a) before menopause, (b) menopause; and (c) following menopause. Findings revealed that women undergoing natural menopause or surgical menopause with bilateral oophorectomy had significantly elevated total cholesterol levels compared to their controls. Cholesterol levels for women who had natural menopause (n = 377) and their controls (n = 2,522) premenopausal were 233.41 and 224.63 (t = 8.78); menopausal, 245.70 and 230.46 (t = 15.24); and postmenopausal, 252.67 and 235.87 (t = 16.8). Cholesterol levels for women who had surgical menopause with bilateral oophorectomy (n = 100) and their controls (n = 2,522) premenopausal were 216.52 and 218.31 (t = -1.79, NS); menopausal, 234.14 and 223.51 (t = 10.63); and postmenopausal, 246.34 and 230.87 (t = 15.47). The women who had surgical menopause without bilateral oophorectomy did not experience a significant difference in total cholesterol levels (Hjortland et al., 1976).

The fourth study, the Healthy Women Study (Everson et al., 1995) is an ongoing longitudinal study of biological and psychological changes experienced by 541 initially menopausal women who are going through menopause. There are four groups of women in this study: Bilateral oophorectomy (BSO)/HRT users, BSO/non-HRT users, hysterectomy only, and premenopausal controls. The relationship of lipoproteins in the four groups was examined in one section of the study.

Analysis of covariance (ANCOVA) revealed that the four groups had significant overall group effects for total HDL and LDL ($p < .05$). The women who had BSO/non-HRT ($n = 7$) had a significantly lower adjusted HDL ($F = 0.26$). The group who had BSO/HRT ($n = 12$) had significantly higher HDL and lower LDL at follow-up (HDL $F = 0.26$, LDL $F = 0.43$) compared to premenopausal controls. The women who had only a hysterectomy did not differ from the premenopausal women in adjusted levels of HDL or LDL (Everson et al., 1995).

Among the BSO cases ($n = 19$), comparisons of mean lipid values at follow-up between HRT and non-HRT groups showed that non-HRT users had significantly lower levels of HDL (both HRT and non-HRT = 57 mg/dl HDL; follow-up HRT = 66 mg/dl HDL, non-HRT = 50 mg/dl HDL). The difference between the HRT and non-HRT groups was not significant for LDL

cholesterol. Total serum cholesterols were higher for the groups of HRT users and the non-HRT users than for the other groups, but the differences were not significant. The differences appeared to be related to the increased HDL and lower LDL cholesterols. Thus, findings from this study demonstrated that a sudden decline in ovarian hormones related to the BSO had a particular adverse effect on HDL cholesterol and that HRT improved the lipid profile after BSO (Everson et al., 1995).

Findings regarding women in the Healthy Women Study who had natural menopause were previously reported by Matthews, Wing, Kuller, Meilahn, and Plantinga (1994). Women who had natural menopause without HRT (non-HRT) showed a definitive increase in total serum cholesterol ($\underline{M}$ = 17.8 mg/dl) and LDL cholesterol ($\underline{M}$ = 17.8 mg/dl) during this time.

Women with both surgical and natural menopause experience alterations in their lipid profiles although the changes are more dramatic with surgery. These alterations in the lipid profile may contribute to the increase in CHD (Castelli, 1988; Stampfer, Colditz, & Willett, 1990) as has been demonstrated by many observational studies.

Data from the previously cited studies indicated that changes in total, LDL, and HDL cholesterols made women more

prone to CHD after menopause. These findings brought light to the fact that at different time periods in their life, women also experienced an increased risk for CHD as men did.

The Lipid Research Clinics Program's (1984) Coronary Prevention Trial, a multicenter, randomized, double-blind study of 3,806 men was designed to evaluate the efficacy of cholesterol lowering in reducing the risk of CHD. The two groups--a treatment group that received cholestyramine and a low fat diet and a control group that received a placebo and a low fat diet--were followed for 7.4 years. The treatment group experienced a reduction of 8.5% in average total plasma cholesterol and 12.6% in LDL cholesterol compared to the placebo group. The cholestyramine group also experienced a 9% decrease in cholesterol levels which led to a 19% reduction in CHD. Although this study was not designed to assess directly whether cholesterol lowering by diet prevents CHD, the findings supported the view that it would be beneficial in preventing CHD.

The Lipid Research Clinics Program's (1984) study was the impetus for the development of a nationwide program by the U.S. Department of Health and Human Services (DHHS, 1989), National Heart, Lung and Blood Institute called the National Cholesterol Education Program (NCEP). Currently an

estimated 97.2 million American adults (52.1% of total adult population) have blood cholesterol levels of 200 mg/dl and higher (AHA, 1996).

Four years later, A Second Report of the Expert Panel on Detection, Evaluation and Treatment of High Cholesterol in Adults (DHHS, 1993) was published. This report provided updated guidelines on the management of hypercholesterolemia and included particular guidelines for women. Initial treatment for the primary prevention of high cholesterol levels begins with dietary management. Those individuals who have previous CHD and high LDL cholesterols are to be considered for drug and diet therapy (DHHS, 1993).

The literature on the relationship between diet, serum cholesterol, and CHD is enormous. Two authors, Stone (1993) and Woodard and Limacher (1993) reviewed the major epidemiological investigations, international randomized studies on diet and heart disease and those on diet and angiography. Included in Stone's review was experimental animal works. In conclusion, there was data to adequately document the relationship between diet and the prevention and treatment of CHD.

Critics of the diet and CHD relationship argue that a definitive, randomized, blinded dietary intervention trial

of serum cholesterol reduction by diet and its effect on CHD had not been conducted. However, the Women's Health Initiative is currently in the data collection stage and intends to include a large-scale, long-term dietary intervention. However, these data will not be available until 2004 (Stone, 1993; Woodard & Limacher, 1993).

Currently, data from the Framingham Heart Study has been published (Millen et al., 1996) on diet and plasma lipids (total cholesterol, LDL and HDL) in women ages 22-79 years (*n* = 695 premenopausal, *n* = 727 postmenopausal). The premenopausal women's plasma lipids were: total cholesterol *M* = 194.5 (*SD* = 36.1); LDL *M* = 118.6 (*SD* = 31.8); and HDL *M* = 57.3 (*SD* = 13.9). Plasma lipids for the postmenopausal women were: total cholesterol *M* = 229.2 (*SD* = 40.2); LDL *M* = 118.6 (*SD* = 31.8); and HDL *M* = 57.3 (*SD* = 13.9).

The multivariate regression models indicated a direct association between saturated fat intake and plasma total cholesterol levels in both groups of women. Findings indicated that premenopausal women who consume 5% more saturated fat have a total serum cholesterol that is about 2.7 mg/dl higher and a total LDL level that is approximately 2.0 mg/dl higher (total cholesterol ß = 0.549, *p* < .04; total LDL ß = 0.414, *p* < .05), whereas postmenopausal women

have total serum cholesterol and total LDL levels that are approximately 3.6 mg/dl higher (total cholesterol ß = 0.722, $p < .05$; total LDL ß = 0.727, $p < .01$). Results showed that serum total cholesterol and LDL levels were consistently related to saturated fat intake in pre- and postmenopausal women (Millen et al., 1996).

Also using a multivariate regression model, results for HDL cholesterol reflected that fiber was directly related to HDL in postmenopausal women (ß = 0.248, $p < .001$) and unrelated to HDL in premenopausal women. Carbohydrates were inversely associated with HDL in both groups (premenopausal ß = -0.203, $p < .001$; postmenopausal ß = -0.138, $p < .001$). In the literature, studies done on women and the above variables were limited (Millen et al., 1996).

The foregoing current data lends support to increasing public awareness of nutrients that will alter blood lipids. Changing those dietary practices that are associated with increasing LDL and total cholesterols are most important in lowering CHD risk. Therefore, the focus should be placed on primary prevention and following the NCEP guidelines so the U.S. can have a healthier population.

Hormone Replacement Therapy

The protective effect of hormone replacement therapy (HRT) on lowering CHD risk, CHD, and CHD mortality was purported in four reviews dealing primarily with epidemiological literature (Barrett-Connor & Bush, 1991; Colditz et al., 1987; Grodstein & Stampfer, 1995; Stampfer & Colditz, 1991; Stampfer & Grodstein, 1994) and one dealing with meta-analysis (Grady et al., 1992) which included controlled clinical trials. The majority of the studies were done on estrogen alone replacement therapy due to its popularity in the 1970s and 1980s in treating menopause. Recently, estrogen and progesterone therapy has become popular in women who have a uterus because of the increased incidence in endometrial cancer in these women with estrogen alone.

The general consensus of all these studies was that they strongly supported the view that postmenopausal HRT can substantially reduce the risk for CHD. The consistency of findings is primarily in the prospective cohort studies. A summary relative risk reported from those studies was 0.5 (95% CI 0.43-0.56), which indicates that there is a greater than or equal to 50% reduction of CHD risk experienced by HRT users.

Hormone replacement therapy in the form of estrogen only has been shown to reduce serum LDL cholesterol and increase serum HDL cholesterol in a dose dependent fashion. Estrogen alone decreases LDL about 10%-15% and increased HDL about 10%-15% (Grady et al., 1992). A 1 mg/dl increase in HDL is associated with an approximately 3% decrease in CHD risk and a 1 mg/dl decrease in LDL is associated with about a 2% decrease in risk. Therefore, the changes in lipoproteins caused by estrogens could lead to a relatively large decrease in CHD risk (Grodstein & Stampfer, 1995).

Estrogens and progestins are used primarily with women who still have their uterus. These hormones tend to raise LDL and lower HDL levels and may detract from the beneficial effects of estrogens on lipid profiles (Grodstein & Stampfer, 1995). The Postmenopausal Estrogen/Progestin Intervention Trial is investigating the effect of lipid levels on estrogen alone and various estrogen and progesterone regimens on postmenopausal women (Writing Group, 1995).

Currently, no large clinical trials of estrogen and progesterone use and CHD have been completed, but several have been initiated. The Women's Health Initiative is a primary prevention trial of estrogen alone and estrogen and

progesterone use among approximately 63,000 women. Data from this study will not be available until 2004 (Stone, 1993; Woodard & Limacher, 1993).

Three recent studies were designed to examine the effects of estrogen alone and estrogen and progesterone on lipids in postmenopausal women utilizing various study designs. Two of the studies were longitudinal cohort studies (Folsom et al., 1996; Grodstein et al., 1996), and the third was a double-blind, randomized placebo controlled clinical trial (Writing Group, 1996).

The Atherosclerosis Risk in Communities Study (Folsom et al., 1996) examined about 4,000 postmenopausal women over a 6-year period to determine how starting or stopping postmenopausal HRT would affect serum lipids. Results of this study demonstrated that women who started using estrogen along (10 mg/dl, n = 149) or estrogen plus progesterone (9.8 mg/dl, n = 74) had approximately equivalent decreases in LDL cholesterol ($p > .25$). The women who stopped taking estrogen alone (n = 114) had a significant increase of 5.5 mg/dl in LDL cholesterol. Only 21 women stopped taking estrogen and progesterone, and there was minimal change (0.3 mg/dl) in their LDL cholesterol. Therefore, there was a significant decrease in the LDL

cholesterol between those who started HRT and those who stopped HRT (p = .05). The women who changed hormone type had a mean change of 1.2 mg/dl for estrogen to estrogen and progesterone group and a mean change of -0.8 mg/dl for the estrogen and progesterone to estrogen group, which was not significant.

Findings from the Atherosclerosis Risk in Communities Study (Folsom et al., 1996) showed increases in HDL cholesterol were larger (5.8 mg/dl, $p < .01$) in the estrogen alone group (n = 149) versus the estrogen and progesterone group (n = 74; 1.2 mg/dl, $p < .01$). Discontinuing the estrogen therapy (n = 114) was associated with a significant decrease in HDL cholesterol (3.3 mg/dl, $p < .05$). Therefore, starting and stopping HRT resulted in significant opposite changes in HDL cholesterol ($p > .25$) (Folsom et al., 1996).

Results of the Atherosclerosis Risk in Communities Study (Folsom et al., 1996) indicated that estrogen alone therapy and estrogen and progesterone therapy may have the same affect on LDL and HDL cholesterols. Additional studies presented expand on this result.

The Nurses Health Study (Grodstein et al., 1996) was designed to examine the relationship between postmenopausal

HRT (estrogen and progesterone) and CHD. There were more than 30 epidemiological studies which indicated that postmenopausal women who use estrogen alone are at lower risk of CHD. However, there is a paucity of data on the use of combined HRT (estrogen and progesterone) on CHD.

Results of this study were based on 16 years of follow-up data on 59,337 postmenopausal women. Estrogen alone and estrogen and progesterone data were combined since the results were similar (Grodstein et al., 1996).

The multivariate adjusted relative risk of major CHD was 0.60 for current hormone users (n = 98), 0.85 for past users (n = 195), and 1.0 for never users (n = 452). Among current users, a longer period of use was not associated with any apparent trend toward further reduction in the risk of CHD (data not available in this study). An analysis of subgroups of women was performed in relation to the affects of HRT on CHD. In the less than 50 year group, the current users (n = 4) had a multivariate adjusted RR = 0.18 compared to RR = 1.0 of the never users (n = 22). In the 50-59 years group, current users (n = 61) had a multivariate RR = 0.71 versus RR = 1.0 in the never users (n = 272). In the 60-71 years group, the current users (n = 33) had a multivariate

RR = 0.66 compared to RR = 1.0 in the never users (n = 158) (Grodstein et al., 1996).

The Nurses Health Study (Grodstein et al., 1996) demonstrated that the risk of CHD was decreased considerably among current users of either estrogen alone or estrogen and progesterone. However, a randomized controlled placebo trial is still needed to document these changes in an experimental fashion.

The Postmenopausal Estrogen/Progestin Interventions (PEPI) Trial (Writing Group, 1996) is a 3-year multicenter, randomized, double-blind, placebo-controlled trial. It was developed to assess differences between placebo, unopposed estrogen, each of three different estrogen/progestin regimes (estrogen and cyclic medroxyprogesterone acetate, MPA; estrogen and consecutive MPA in a lower dose; and estrogen and cyclic micronized progesterone, MP) and selected heart disease risk factors in healthy postmenopausal women. Relative to LDL and HDL cholesterol, all four active treatment regimens were associated with a significantly greater (Bonferroni $p < .001$) rise in mean HDL cholesterol levels than placebo: placebo (n = 174), -1.2 mg/dl; estrogen alone (n = 175), 5.6 mg/dl; estrogen and MPA cyclic (n = 174), 1.6 mg/dl; estrogen and MPA consecutive

(n = 174), 1.2 mg/dl; and estrogen and MP cyclic (n = 178), 4.1 mg/dl. The average increases in HDL were similar in women assigned to estrogen alone or estrogen with MP. These women had a significantly greater HDL than the other two groups (Bonferroni p < .004).

Women who had hysterectomies and were taking unopposed estrogen (n = 54) had HDL levels that were on the average 5.5 mg/dl greater at the end of the study than women who were taking estrogen along without hysterectomy (n = 114). There was a total of 49 women with a uterus in the estrogen only group that developed endometrial cancer (n = 1), endometrial hyperplasia (n = 41), and had a hysterectomy (n = 7). These women withdrew from the study. In addition, women with a hysterectomy who took unopposed estrogen had a significantly greater average increase in the HDL levels than the estrogen and MP group (p = .10) (Writing Group, 1996)

Changes in the LDL levels decreased significantly (p < .001) from the placebo group (-4.1 mg/dl) compared to all the active treatment regimens (estrogen, -14.5 mg/dl; estrogen and MPA cyclic, -17.7 mg/dl; estrogen and MPA consecutive, -16.5 mg/dl; and estrogen and MP, -14.8 mg/dl) and in women with or without a hysterectomy. The largest

decrease in LDL occurred in the estrogen and MPA cyclic group (Writing Group, 1996).

The PEPI Trial (Writing Group, 1996) provided the most evidence to date that unopposed estrogen has a more favorable effect on HDL than estrogen given with progestin. In women, epidemiologic evidence has shown that HDL is more closely related to CHD than is LDL. In these studies, a 4 to 5 mg/dl change was associated with a 20% to 25% decrease in CHD (Castelli, 1988). However, no clinical trial were found that show increasing HDL reduced CHD risk in women.

One problem with the observational studies that have been done on estrogen replacement therapy (ERT) and CHD is that many of the women who are ERT users have the profile of being better educated, having higher HDLs, enjoying leisure activities, drinking alcohol more, having lower weights, and lower systolic and diastolic blood pressures than non-users of ERT (Matthews, Kuller, Wing, Meilahn, & Plantinga, 1996). Therefore, it is important to conduct randomized clinical trials to estimate the direct benefit of postmenopausal ERT for protecting women from CHD.

Prevention Practices for Menopausal Women with Risk of CHD

CHD is the single largest killer of American women. CHD often develops from risk factors which may be modifiable. One of the modifiable risk factors is high blood cholesterol, which can be controlled by limiting a diet risk in saturated fats. Risks from CHD rise progressively as the serum cholesterol rises above 200 mg/dl. Although there has been a reduction in deaths related to CHD, the overall mortality remains high and the cost to treat the disease ($259.1 billion in 1997) is exorbitant (AHA, 1996). Modification of risk factors by changing behavior must be the goal if mortality and health care costs are to decrease (Kern, 1990; Stason, 1990; DHEW, 1990).

The public's attention has been focused on lowering cholesterol levels in all age groups. The 1990 Objectives for the Nation (DHHS, 1986) was written as a mid-decade review of the health objectives set in 1980. One of the objectives stated that the mean serum cholesterol levels in the adult population (18-74 years of age) should be at or below 200 mg/dl by 1990. Results of this report revealed that the mean serum cholesterol levels for adult men and women was lowered from 223 mg/dl to 211 mg/dl for men and

215 mg/dl for women. From these data, one may discern that the public has become more aware of their eating habits and modified their diets by decreasing saturated fats and lowering serum cholesterol, but the public has not yet effectively met the goal of the 1990 objectives. The emphasis on lowering cholesterol in one's diet must continue so cholesterol levels continue to decline and the goal for the nation is attained. However, regardless of the emphasis placed on changing eating habits, modification of any lifestyle, or habits thereof, can occur only as the result of changes in behavior(s). The task of behavior change lies with the individuals, but individuals can be influenced to make the changes needed to engage in preparing and eating more healthy diets. To reach this goal, health care professionals need to know and understand how to become change agents to effect behavior change in their interactions with individuals.

Behavior Change

Currently, the U.S. is focused on improving the health of its people through health promotion and disease prevention. Both Healthy People and the 1990 Objectives (DHHS, 1986, 1990) provide guidelines to accomplish the task of prevention. Behavior change is the core concept which

makes one responsible for the adoption of a healthy lifestyle. Self-efficacy and the transtheoretical model have both held an influential role in guiding healthy behavior. However, little research has been focused on the way these two models work together to enhance the behavior change process.

To better understand how the models conjoin, literature relevant to social cognitive theory (self-efficacy) (Bandura, 1986) and the transtheoretical model (Prochaska & DiClemente, 1992), along with empirical evidence of self-efficacy and the transtheoretical model, is presented. The research that has been done using both, with emphasis on exploring the interrelationship of self-efficacy and the transtheoretical model and incorporating this knowledge as it relates to women and CHD, is described.

Theoretical Conceptualization of Self-Efficacy

Social cognitive theory (Bandura, 1977a, 1986) purports that there is a role for self-referent thought in psychosocial functioning. Together behavior, cognitive and physiological factors, and environmental influences all operate as interacting determinants of one another.

According to Bandura (1977a), "people process, weigh, and integrate diverse sources of information concerning

their capability, and they regulate their choice behavior and effort expenditure accordingly" (p. 212). Behavioral change occurs through altering one's self-efficacy expectations. These expectations are: (a) a person's ability to believe that he or she is or is not capable of performing a behavior or set of behaviors in question, (b) the amount of effort expended in accomplishing the behavior, and (c) the amount of persistence in the face of obstacles and aversive experiences.

As Bandura (1977a) pointed out, efficacy expectancies vary on the dimensions of magnitude, strength, and generality. Each of these dimensions has important implications for performance. Magnitude may affect an individual's performance of a specific task. When tasks are ordered according to difficulty, individuals vary in their performance of the easier, moderately difficult, or the most difficult tasks. Generality also affects efficacy expectations. Some situations only create narrow mastery expectations, whereas other situations apply broadly to experiences beyond the treatment situation. Strength is also important when addressing expectancies. Weak expectations are easily extinguished by unpleasant experiences, while individuals who possess strong

expectations of personal mastery will persevere in their coping efforts despite negative experiences. It is important to assess these dimensions in order to determine their effect on the way a person deals with threatening tasks.

Efficacy expectations are based on four major sources of information. The first is called performance accomplishments, which provide the most dependable source of efficacy expectations. One's own experiences are used to overcome a difficult or feared task, which results in increasing self-efficacy. The second method is vicarious experience (modeling). This source of information is accomplished through observing others perform threatening activities without adverse consequences. Verbal persuasion is the third source of efficacy expectations and is easy and readily available. It is through this source that people are led, through suggestion, into believing they can cope successfully with what has overwhelmed them in the past. However, in the face of distressing threats and a long history of failure in coping with them, suggestion-induced successful expectations will be rapidly extinguished by adverse experiences. Fourth, a person's emotional arousal influences efficacy expectations in threatening situations.

High physiological arousal usually debilitates performance. Therefore, a person may tend to expect failure when feeling anxious or fearful (Bandura, 1977a).

Empirical Evidence for Self-Efficacy

Self-efficacy was first tested by using desensitization, which had intervening effects on self-efficacy expectations, to change the behavior of severe snake phobics. The initial studies conducted by Bandura and his colleagues (Bandura & Adams, 1977; Bandura, Adams, & Beyer, 1977; Bandura, Adams, Hardy, & Howells, 1980; Bandura, Reese, & Adams, 1982) were concentrated on reducing emotional arousal and avoidance behavior of phobic subjects. Thus, the result would be a change in self-efficacy expectations.

Bandura and Adams (1977) investigated the change in self-efficacy expectations on snake phobics (n = 10) by utilizing systematic desensitization and participant modeling. Initially, the systematic desensitization treatment was given to each subject. The subjects' avoidance behavior, fear arousal, and self-efficacy for feared encounters were measured before and after treatment. The results showed that systematic desensitization enhances self-efficacy and approach behavior toward similar and

dissimilar threats. In addition, results demonstrated that self-efficacy was a high accurate predictor of approach behavior (84% congruence).

The subjects (n = 8), who only received partial improvement of their symptoms, were administered participant modeling after the formal experiment was completed. The results of this supplementary treatment were marked changes in all aspects of self-efficacy, performances, fear arousal, and coping with reptiles and other animals. Results also indicated that self-efficacy expectations were highly reliable predictors of approach behavior toward similar (97%) and dissimilar (62%) threats. Bandura and Adams (1977) concluded that the results support the notion that psychological influences alter defensive behavior by enhancing self-efficacy.

An experimental three-group study (Bandura et al., 1977) was designed to test the theory that psychological procedures (direct mastery, e.g., participant modeling) and vicarious experience achieved behavior changes by altering self-efficacy. The adult snake phobics received either performance mastery experiences (group 1), vicarious experiences (group 2), or no treatment (group 3). The level, strength, and generality of efficacy expectations as

well as approach behaviors were measured before and after treatment. The researchers predicted that the treatment based on participant modeling would produce higher stronger and more generalized expectations than would vicarious experience alone. In addition, it was hypothesizes that self-efficacy would vary according to performance level.

Results demonstrated that self-efficacy was an accurate predictor of individual task performance. It did not matter whether the changes were produced through performance accomplishments (89%) or vicarious experience (86%). In addition, data showed that self-efficacy was greater in subjects who experienced less psychological distress while performing the behavior (Bandura et al., 1977).

Following Bandura's early works on the relationship between self-efficacy expectations and changes in phobic behavior, other investigators have shown that there is a general relationship between self-efficacy expectations and behavior change. To exemplify this relationship, increases in self-efficacy expectations have been found to accompany self-protective skills (Ozer & Bandura, 1990), smoking maintenance after childbirth (Quinn, Mullen, & Ershoff, 1991), pain tolerance in childbirth (Manning & Wright, 1983), and coping after an abortion, (Major et al., 1990).

Findings from additional studies have indicated that a decrease in self-efficacy expectations result in a sustained decline in perceived health for the elderly (Rodin & McAvay, 1992) and an increase in smoking relapse postpartum (McBride, Pirie, & Curry, 1992). Seeman, Rodin, and Albert (1993) tested the hypothesis that stronger self-efficacy beliefs are associated with better cognitive performance in older ages. Seeman et al. found that instrumental efficacy beliefs had no significant associations with tests of cognitive ability and interpersonal efficacy beliefs showed no significant associations for men or women. In an epilepsy self-management program (DiIorio, Faherty, & Manteuffel, 1994), a definitive relationship was found between self-efficacy and self-management. In addition, regression analysis revealed that self-efficacy made important contributions to the variance in self-management. Thus, it has been shown that self-efficacy is operationally related to a large number of behaviors.

With the emphasis on health promotion and disease prevention, results of a number of investigations showed that self-efficacy expectations are positively and significantly associated with health behaviors (O'Leary, 1985; Strecher, DeVellis, Becker, & Rosenstock, 1986). The

types of health behaviors involved in the research aspects of self-efficacy are smoking cessation (Coelho, 1984; Condiotte & Lichtenstein, 1981; DiClemente, 1981; McIntyre, Lichtenstein, & Mermelstein, 1983), weight control (Edell, Edington, Herd, O'Brien, & Witkin, 1987; Shannon, Bagby, Wang, & Trenkner, 1990; Weinberg, Hughes, Critelli, England, & Jackson, 1984); exercise (Ewart et al., 1986; Kaplan & Atkins, 1984; McAuley & Jacobson, 1991; Taylor, Bandura, Ewart, Miller, & Debusk, 1985; Vidmar & Rubinson, 1994), use of contraception in college women (Heinrich, 1993), and use of condoms in adolescents and college age men and women (Jemmott & Jemmott, 1992; Jemmott, Jemmott, Spears, Hewitt, & Cruz-Collins, 1992; Joffee & Radius, 1993; O'Leary, Goodhart, Jemmott, & Boccher-Lattimore, 1992). However, no current studies on dietary modification and self-efficacy were found.

Findings of the previously reviewed studies lend support to Bandura's assertion that efficacy expectations reflect a person's perceived, rather than actual, capabilities. It is important to remember that these perceptions and not one's true abilities most often influence behavior.

Theoretical Conceptualization of the Transtheoretical Model

The transtheoretical model (Prochaska & DiClemente, 1982, 1984, 1986, 1992a,b) grew out of Prochaska's (1979, 1984) initial journey through the various systems of psychotherapy, seeking the commonalities among them. Ten processes of change were discovered in this journey and were found to summarize the theories of psychotherapy. Application of this model to research situations that required behavior change took place, which resulted in the development of The Transtheoretical Approach: Crossing the Traditional Boundaries of Therapy (Prochaska & DiClemente, 1984).

First, the elements of this model, processes of change, stages of change, and levels of change (of interest in this study), are defined and described. Empirical evidence which supports the stages of change as they apply to the current study is presented.

Processes of Change

The first element of this model is the 10 processes of change (Prochaska & DiClemente, 1982, 1984, 1986, 1992a,b; Prochaska, DiClemente, & Norcross, 1992). The processes are listed and defined: (1) consciousness raising--efforts by

the individual to seek new information and to gain understanding and feedback about the problem behavior; (2) counterconditioning--substitution of alternative behaviors for the problem behavior; (3) dramatic relief--affective aspects of change, often involving intense emotional experiences related to the problem behavior; (4) helping relationships--trusting, accepting, and utilizing the support of caring others during attempts to change the problem behavior; (5) reinforcement management--changing the contingencies that control or maintain the problem behavior; (6) self-liberation--the individual's choice and commitment to change the problem behavior, including the belief that one can change; (7) self-reevaluation--emotional and cognitive reappraisal of values by the individual with respect to the problem; (8) social liberation--awareness, availability, and acceptance by the individual of alternative, problem-free lifestyles in society; (9) environmental reevaluation--consideration and assessment by the individual of how the problem affects the physical and social environment; and (10) stimulus control--control of situations and other causes which trigger the problem behavior.

These processes represent a middle level of abstraction between the basic theoretical assumptions of the model and the techniques proposed by the model to facilitate behavior change. Therefore, a process of change represents a type of activity and event that creates a successful modification of a problem behavior. The activity and event results in a modification of an individual's thinking, which leads to behavior change (DiClemente et al., 1991; Prochaska & DiClemente, 1982, 1984, 1986, 1992a,b; Prochaska et al., 1992).

The processes are used as intervention strategies to move clients through the stages of change. The processes vary in how they are used during the different stages of change, with experiential processes peaking in the contemplation stage and behavioral processes peaking in the preparation, action, and maintenance stages (DiClemente et al., 1991; Prochaska & DiClemente, 1982, 1984, 1986, 1992a,b; Prochaska et al., 1992).

Stages of Change

The five stages of change (Prochaska & DiClemente, 1982, 1984, 1986, 1992a,b; Prochaska et al., 1992) comprise the second element of the model. These stages are listed and defined: (1) precontemplation--individuals who do not

perform the behavior and have no intention of performing the behavior; (2) contemplation--individuals who do not perform the behavior, but intend to perform it; (3) preparation--individuals who perform the behavior, but not regularly; (4) action--individuals who currently began the behavior regularly; and (5) maintenance--individuals who perform the behavior regularly over a long period of time. These stages represent distinct, but related, periods of time that are marked by different types of activities. The processes of change are used to move individuals through the stages of change and are linked to specific stages of change. The current study is focused on the interrelationship of the stages of change and self-efficacy.

Levels of Change

The third element of the transtheoretical model (Prochaska & DiClemente, 1984, 1986, 1992a,b) addresses the levels of change. These levels of change are a hierarchical organization of five distinct, but interrelated, levels of psychological problems which are addressed in psychotherapy by psychotherapists. Implementation of the levels of change are beyond the scope nursing practice. The levels include: (1) symptoms-situational problems; (2) maladaptive cognitions; (3) current interpersonal conflicts; (4) family/

systems conflicts; and (5) intrapersonal conflicts. The psychotherapist who utilizes the transtheoretical model intervenes at level 1 or 2, because these are the most conscious levels. Interventions at the first two levels will impact the deeper levels and eventually lead to longer term therapy (Prochaska & DiClemente, 1984, 1986, 1992a,b).

Empirical Evidence for Stages of Change

The transtheoretical model has as its primary research focus a self-initiated and professionally facilitated smoking cessation (DiClemente & Prochaska, 1982, 1985; Prochaska & DiClemente, 1983, 1984; DiClemente, Velicer, Ginpil, & Norcross, 1985; DiClemente et al., 1991; Prochaska, Velicer, DiClemente, Guadagnoli, & Rossi, 1991). This addictive behavior and others were chosen because of their complexity and difficulty to treat. Certain treatment methods consistently demonstrated successful outcomes, but often individuals cycled through the stages of change in varying ways more than once to reach maintenance. The stages of change emerged during an empirical investigation of the processes used by individuals to change their behavior.

DiClemente and Prochaska (1982) compared the processes of change used by individuals who quit smoking on their own

(n = 29) and those who quit smoking using one of two commercial smoking cessation programs (aversive stimuli n = 18; behavior management (n = 16). The stages of change used by these subjects were described by the subjects during a pilot study of feasibility. There were three stages of change: decision to change, active change, and maintenance.

As subjects completed the Change Process Questionnaire, they were asked to identify in which stage the experience was the most helpful. This study uses a retrospective design. A contingency table was constructed from the frequencies in order to examine the relationship between the 10 change processes and the stages of change. Results showed a significant difference (X^2 (18, N = 63) = 324.45, p < .001). Findings showed that a relationship existed between the processes and stages of change. Therefore, both the processes and stages were individual and related constructs of the transtheoretical model (DiClemente & Prochaska, 1982).

In addition, there was a significant difference between verbal and behavioral processes (X^2 (10, N = 63) = 71.14, p < .001). Thus, the verbal processes tended to be used more frequently in the decision to change stage and the behavioral processes were employed more frequently in the

active change and maintenance stages (DiClemente & Prochaska, 1982).

Self-changers (n = 872) participated in Prochaska and DiClemente's (1983) second study. A cross-sectional design was used to study subjects who were in one of the following five stages of change: precontemplation, contemplation, action, maintenance, and relapse. Subjects were staged using one of five statements. For the precontemplation stage, the subjects (n = 108) had no intention of quitting smoking in the next year. Subjects in the contemplation stage (n = 187) reported they were seriously thinking about quitting smoking in the next year. Those subjects in the action stage (n = 134) had quit smoking on their own within 6 months of entering the study. The maintainers (n = 247) had quite smoking for at least 6 months. Subjects who relapsed (n = 196) failed within the last year in their attempt to quit smoking.

Multivariate analysis of variance (MANOVA) for these data was significant (F (1,40) = 11.199, $p < .001$). This result indicated that the stages and processes of change are different and exist individually. The types of processes used and their frequency of usage in each stage were also

described, resulting in a cyclical process of change (Prochaska & DiClemente, 1983).

In a 2-year longitudinal study, DiClemente and Prochaska (1985) explored coping activity in the self-change of smoking. A total of 872 smokers and ex-smokers were assigned to one of five groups according to their smoking stage of change. The criteria used to assign these subjects was described by Prochaska and DiClemente in their 1983 study. The number of subjects for each group stayed the same as the previous study: 108 precontemplators, 187 contemplators, 134 action, 247 maintainers, and 196 relapsers. Subjects completed an extensive questionnaire at the initiation of the study and at 5-6 month intervals over the next 2 years.

A MANCOVA was calculated comparing all change process scores across the five groups. The MANCOVA was significant (F (1,40) = 11.19, p < .001) which indicated that there were significant differences on these processes for the different stages of change (DiClemente & Prochaska, 1985).

Another part of the above mentioned investigation was focused on which subjects would remain in the same groups over time and which would change groups. A discriminant function analysis was performed for four of the five groups

(omitting the maintenance group because very few subjects changed groups) to predict changes in group membership at 6-month follow-up. This statistical analysis showed that individuals changed stages and utilized specific processes to make the particular stage change. Thus, findings supported the theoretical notion that change occurs in a cycle with smokers entering and exiting the cycle at different points in time and spending varying lengths of time in the separate stages.

Prochaska and DiClemente (1985) asked whether these stages and processes are only applicable to smoking or whether they are relevant to other problems related to substance abuse. Out of this question grew a study designed to examine smoking (n = 605), weight control (n = 402), and psychological distress (n = 320). The instruments were revised by adding two additional processes: substance use and interpersonal systems control. These 12 processes were then viewed across three different problems. The study took place over 2 years with questionnaire completions every 6 months. The stages of change (contemplation, action, or maintenance) were also defined using Prochaska and DiClemente's (1983, 1985) categorical classification.

Data analyses were focused on processes to move individuals through the stages of change. There was no

statistical analysis comparing particular processes to the relevance for certain stages. However, because the processes were described according to the stages, it can be inferred that the stages of change construct is supported (Prochaska & DiClemente, 1985).

DiClemente et al. (1991) examined 1,466 subjects who were still smoking. According to Prochaska and DiClemente (1983, 1985), the subjects were divided into four groups according to the three stages of change: precontemplation (PC, $\underline{n}$ = 166), contemplators (C, $\underline{n}$ = 794), and preparation (PA, $\underline{n}$ = 506). Comparisons among smokers in the precontemplation, contemplation, and preparation stages were analyzed for process, outcome differences on smoking history variables, and prospective cessation activities.

The results at one month showed point prevalence abstinence from smoking rates as follows: PC = 1.8%, C = 4.8%, and PA = 11.9%. This one-month posttest data clearly supports the stage of change classification of smokers. At 6 months the point prevalence abstinence from smoking rates were as follows: PC = 6.0%, C = 9.1%, and PA = 16.2%. These results once again support the stage of change classification of smokers (DiClemente et al., 1991).

Findings from DiClemente et al.'s (1991) study also demonstrated that a preparation stage exists between the

contemplation and action stages. This preparation stage was evident in earlier research, but was ignored due to misinterpretation of the analyzed data. Currently there are five stages of change.

In 1991, Prochaska, Velicer, and DiClemente investigated patterns of change occurring in a combined cross-sectional and longitudinal analysis of 14 variables. This study took place over 2 years and had five 6-month follow-up periods in which data were collected. The 544 subjects who completed the five follow-up periods were self-changers for smoking. Using categorical classification (Prochaska & DiClemente, 1983, 1985), the subjects were divided into five groups: precontemplators (n = 122), contemplators (n = 206), those in action (n = 143), maintainers (n = 231), and relapsers (n = 255).

Data analysis was completed using a dynamic typology (descriptive methodology), which allows subjects to be groups according to their patterns of change. Fourteen profile groups that had adequate sample size for interpretation resulted. The results then generated graphic illustrations of a central tenet of the transtheoretical model of change--that particular processes of change are emphasized during particular stages of change (Prochaska et al., 1991).

Findings from the previously reviewed studies provided support for Prochaska and DiClemente's assertions that individuals change behavior according to the stages which they are in, utilize various processes of change to make these stage movements, and progress through the stages in cyclical manner--at times going back and forth between the stages of change. Furthermore, there is empirical evidence that the stages and processes of change are important in initiating and maintaining changes in behavior.

More recent research on the stages of change supported a division of subtypes within each of the stages. These subtypes can be used to further categorize subjects into their stages and also to develop interventions for movement through the stages (Velicer, Hughes, Fava, & Prochaska, 1995). An expert system, which is a computerized intervention, has been created for the stages of change model. It has been used in an intervention study to determine its ability to move individuals through the stages of change (Velicer et al., 1993; Prochaska, DiClemente, Velicer, & Rossi, 1993). A large three-group (n = 4,144, n = 9,534, n = 4,785) study was undertaken to document the existence of the stages of change construct (Fava, Velicer, & Prochaska, 1995; Velicer et al., 1995). Two constructs of the model--stages of change and decisional balance--have

been investigated in an integrated study across 12 problem behaviors (Prochaska et al., 1994). The results of this study showed clear commonalities across the 12 areas, including both the internal structure of the measures and the pattern of changes in decisional balance across the stages.

In addition to the work of Prochaska and DiClemente on the transtheoretical model, other investigators have utilized the stages of change to study exercise behavior (Marcus et al., 1992; Marcus & Simkin, 1993), adopting a diet high in fruits and vegetables (Laforge, Greene, & Prochaska, 1994), and assessing contraceptive use for the prevention of pregnancy, sexually transmitted diseases, and acquired immunodeficiency syndrome (Grimley, Riley, Bellis, & Prochaska, 1993). No studies were found on women and heart disease in which this model was utilized.

Empirical Evidence for Stages of Change and Self-Efficacy

Bandura (1977, 1986) assumed that self-efficacy can serve as the core construct in a more comprehensive model of change and serve as a key variable used to predict progress from one stage to the next. Self-referent thought is an important mediator between knowledge and action. Perception of self-efficacy can influence motivations and behaviors.

Bandura theorized that successful change is due to significant increases in self-efficacy. Self-efficacy represents the level of confidence that individuals have as they cope with different situations in the face of obstacles and either control or abstain from their problem behavior. The levels of self-efficacy increase across the stages of change. This assumption is empirically described below (DiClemente, 1986; DiClemente, Fairhurst, & Piotrowski, in press; Prochaska & DiClemente, 1984, 1985).

In 1981, DiClemente studied smoking cessation maintenance by evaluating 63 subjects who had quit smoking (either through a program or on their own) on the variable of self-efficacy. Subjects were initially interviewed and consented to a follow-up telephone interview 4 to 6 months later. At the 5-month follow-up, two-thirds of all subjects remained successful quitters. In analyzing the 5-month data, differences existed on self-efficacy scores between the individuals who were successful in quitting and the recidivists. Successful maintainers had significantly higher self-efficacy scores (F (1,61) = 8.7, p < .005). This finding supported that efficacy expectations appeared highly related to the ability to maintain smoking cessation.

DiClemente, Prochaska, and Gibertini (1985) examined how self-efficacy is related to the stages of change in

smoking cessation by 954 subjects. Subjects had to be currently smoking, have smoked in the past, not be involved in any formal treatment program, and be willing to follow-up over a 2-year period. Categorical classifications were used to assign the subjects to one of five groups according to their stage of change (Prochaska & DiClemente, 1983). Groups included 122 precontemplators, 204 contemplators, 143 in action, 231 maintainers, and 254 relapsers.

DiClemente et al. (1985) found that self-efficacy scores demonstrated predictive utility for contemplators and subjects in the action stage. An analysis of variance (ANOVA) comparison of self-efficacy mean scores at initial assessment for subjects (n = 172) who remained contemplators and those who changed status was significant (F (3,167) = 282, $p < .05$). Using the ANOVA for the 118 subjects in the action stage, self-efficacy means significantly differed (F (2,114) = 2.95, p = .05) for subjects who remained in action, maintained, or relapsed. Group differences for self-efficacy proved to be higher in the action and maintenance stages of change. Thus, self-efficacy emerged as a relevant variable for subjects in the contemplation, action, and maintenance stages for smoking behavior change.

Additional studies previously described also support the assumption that self-efficacy is higher in the later

stages of smoking behavior change. Prochaska, Crimi, Lapsanski, Martel, and Reid (1982) studied individuals in the relapse and maintenance stage of smoking cessation. They found substantial differences on the self-efficacy measure with the 38 maintainers scoring high, with *M* = 91.1 (*SD* = 19.8) and the 24 relapsers scoring low, with *M* = 38.5 (*SD* = 22.6). Using the ANOVA, the two groups differed significantly (*F* (1,69) = 92.9, *p* < .001) on the variable of self-efficacy.

Prochaska and DiClemente (1984) examined individuals across the five stages of change for smoking cessation. Self-efficacy means (*SD*s were not available) were found to be the highest in the 247 maintainers with *M* = 127, followed by 114 subjects in action with *M* = 114, while the lowest scores were among the 196 relapsers with *M* = 66, 187 contemplators with *M* = 65, and 108 precontemplators with *M* = 58. Also, among the maintainers, self-efficacy did not decrease until 18 months after quitting.

DiClemente and Prochaska (1985) examined the connections between stress, coping, and smoking cessation across five stages of change. The self-efficacy means for the groups were 60.5 (*SD* = 23) for 108 precontemplators, 61.1 (*SD* = 20) for 187 contemplators, 116.9 (*SD* = 28) for 134 subjects in action, 131.1 (*SD* = 32) for 247 maintainers,

and 68.8 (*SD* = 23) for 196 relapsers. Using an ANOVA, significant differences were found between the groups (*F* = 253, *p* < .001). These findings supported the previously mentioned assumption.

The precontemplation, contemplation, and preparation stages were investigated on the process of smoking cessation by DiClemente et al. (1991). Stage comparisons were performed using standardized scores with multiple ANOVAs, regression, and post hoc procedures for the measure of self-efficacy to smoke. The *F* values were not available in the article. The 506 preparation subjects with *M* = 53.8 (*SD* = 10.1) had a significantly higher level of self-efficacy to stop or maintain nonsmoking. The 794 contemplators with *M* = 48.8 (*SD* = 9.3) were significantly different from both the 166 precontemplators with *M* = 44.4 (*SD* = 9.0) and the preparation subjects on the variable of self-efficacy.

Prochaska et al. (1991) investigated patterns of change and developed a dynamic typology for smoking cessation. Self-efficacy increased linearly across the stages of change. It was in the maintenance stage that self-efficacy became greater than the other stages.

There were seven studies with findings supporting the assumption that self-efficacy is a construct that increases

across the stages of change. Therefore, it is important to evaluate this variable with the stages of change in order to fully determine long-term success for quitting and predicting relapse within the first 5 months of quitting smoking. The more effective the individual saw himself or herself in dealing with the internal and external pressure to smoke, the more likely the individual was able to resist relapse (Prochaska & DiClemente, 1982).

Additional studies conducted by various investigators supported health promotion behaviors, self-efficacy, and the stages of change. Three of the studies explored exercise, self-efficacy, and stages of change (Marcus, Eaton, Rossi, & Harlow, in press; Marcus, Pinto, Simkin, Audrain, & Taylor, 1994; Marcus, Selby, Niaura, & Rossi, 1992). Two studies were designed to investigate contraceptive use, self-efficacy, and stages of change (Galavotti et al., 1995; Grimley, Prochaska, Velicer, & Prochaska, 1995). On study was designed to examine weight control, self-efficacy, and stages of change (Prochaska, Norcross, Fowler, Follick, & Abrams, 1992). However, no studies were found that were designed to investigate coronary heart disease risk, dietary fat modification, stages of change, and self-efficacy in postmenopausal women.

Summary

Cardiac risk factors--particularly cholesterol, menopause, and hormone replacement therapy--and coronary heart disease in women, dietary fat reduction as the mainstay of treatment for elevated cholesterol levels, and the influence of menopause and HRT on cholesterol levels were presented as risk reduction and health promotion behaviors. Bandura's (1977) self-efficacy theory was shown to influence the stages of change construct from the transtheoretical model (Prochaska & DiClemente, 1984, 1986, 1992). Both constructs have empirical evidence that supports their use in health promotion behavior change. Therefore, using self-efficacy theory and the transtheoretical model as a framework to investigate the influence of eating foods low in saturated fat on the cardiac risk profiles of postmenopausal women was the focus of this study.

CHAPTER 3

PROCEDURE FOR COLLECTION AND TREATMENT OF DATA

A nonexperimental two-by-two group design (Campbell & Stanley, 1963) was used to examine the main effects of the interaction of the type of menopause and hormone replacement therapy (HRT) on the dependent variables: (1) risk of coronary heart disease (CHD), (2) dietary fat modification, (3) stages of change, and (4) self-efficacy for health-related diet change. These variables were evaluated at a single point in time to determine significant differences between groups on health promotion behaviors.

Nonexperimental studies provide a means for researchers to explore differences between variables. The results of the nonexperimental design are the generation of hypotheses that will be tested in subsequent studies. This design facilitated linkages between the identification and description of phenomena and hypothesis-testing studies (Campbell & Stanley, 1963). Data were collected by administering four questionnaires to two samples of postmenopausal women: (1) those who had undergone surgical menopause, and (2) those who had experienced natural menopause.

Since exploratory designs are meant to be hypothesis-generating instead of confirming, the researcher does not exert great control. However, internal validity (Campbell & Stanley, 1963) must still be considered. Internal validity relates to the accuracy of the relationships between the variables that are studied.

The threat to internal validity that was relevant to this study was instrumentation. Instrumentation error may interfere with internal validity in this study due to subjects' performance when completing the instrument. By allowing the subjects to set the pace of the telephone interviews and stop if they become tired, an attempt was made to prevent fatigue (Campbell & Stanley, 1963).

Setting

There was one setting for the study: a multispecialty outpatient diagnostic clinic in the greater Houston area. In the clinic setting, the prospective subjects from one physician's practice were sent a letter describing the study and a consent form. The signed consent was sent back to the investigator, and the subject was contacted by telephone. Any questions or concerns about the study were answered over the telephone. If they consented, subjects were requested to participate in a telephone interview utilizing four questionnaires. If an interview appointment was requested

by the subjects, the questionnaires was completed with the investigator present in an available office with a desk. Otherwise, all the questionnaires were completed by a telephone interview.

Population and Sample

The population of the study consisted of postmenopausal women who had undergone surgical menopause and those who had experienced natural menopause. In addition, hormone status of both groups was determined. These women were selected from a large multispecialty clinic in the greater Houston area.

The age range (45-74 years of age) for the subjects was determined using the average age at which menopause may begin (Speroff, Glass, & Kase, 1995) and the Framingham study data (Anderson, Wilson, Odell, & Kannel, 1991) which stated that in women over the age of 74, the issue of aging and the development of atherosclerosis will impact the coronary heart disease risk table. This issue was not factored into the score of this instrument at this time.

Recruitment strategies for the clinic participants were done through personal interviews and letters. The ethnicity of the clinic population was primarily Caucasian and the socioeconomic status was middle to upper middle class. The rationale for using the clinic setting was that it

represents the general working population and therefore their usual health promotion strategies.

A total of 2,500 consent forms with information about the study were mailed to women between the ages of 45 and 74 years in the participating physician's practice. Of these 2,500 consent forms, 139 consent forms were signed and returned to the investigator. Of the 139 potential subjects, 33 did not participate in the study for the following reasons: incorrect address for 20, age beyond inclusion criteria for 1, 3 declined to participate by telephone, and 9 did not respond to the phone call.

A total of 106 subjects responded to the phone call interview. However, 17 of these women were still premenopausal and 22 were perimenopausal. Therefore, 77 subjects who met the criteria for the study were left.

The total study sample was 77 postmenopausal (surgical and natural) women who met the criteria to participate in the study. Criteria for admission to the study were as follows:

1. Postmenopausal women (surgical and natural) between the ages of 45-74.
2. Ability to read, write, and understand spoken English.
3. Lipid profile within the last year.
4. No preexisting coronary heart disease or stroke.

The study included two groups of postmenopausal women, 46 who had undergone surgical menopause and 31 who had experienced natural menopause, for a total of 77 menopausal women. This sample size was determined by the use of power .80, effect size .50 and alpha level .05 as stated in Cohen's (1988) ANOVA table. A power analysis was compiled after the data were collected.

Protection of Human Subjects

Permission to conduct the study was obtained from the Human Subjects Review Committee of Texas Women's University and the agency where the research was conducted (Appendix A). A letter and written description of the study (Appendix B) were given to each potential subject. The subjects' written informed consent (Appendix C) to participate in the study was obtained, and the investigator assured the subjects that participation in the study was of a voluntary nature.

The risks of the study were explained to each potential subject before completing the questionnaires. No physical or psychological risks had been identified. Subjects were told that completion of the questionnaire data required 20 to 30 minutes of their time. The subjects were informed that they could withdraw from the study at any time without untoward consequences.

Several benefits could be derived from the study. The benefits included: (1) providing preliminary information on women with surgical and natural menopause regarding CHD risk, (2) evaluating the eating habits of the population sampled and readying them for a health promotion program, and (3) determining the effects of self-efficacy and stages of change on postmenopausal women.

Confidentiality of information was maintained by coding the instruments. Aggregate data only were reported in any written reports or publications of the study data.

Instruments

The objective of the study was to explain the difference between the two groups of postmenopausal women, surgical and natural, and their hormone status on the variables of risk of coronary heart disease, dietary fat modification, stages of change, and self-efficacy. In addition, analysis was done on specific demographic variables which impact the study variables. Administration of the questionnaires took 20 to 30 minutes.

Five instruments were used for the study: the Demographic Data Form (Appendix D), Risk of Coronary Heart Disease Table (Appendix E), the Food Habits Questionnaire (Appendix F), Stage of Change to Dietary Fat Reduction (SCDFM) (Appendix G), and Self-Efficacy for Health-Related

Diet Behaviors (SEHRDB) Questionnaire (Appendix H). Permission to use and reproduce the questionnaires was obtained from their authors (Appendix I).

Demographic Data Form

The Demographic Data Form (Appendix D) was developed by the investigator. This form includes the following variables: date of birth, sex, marital status, ethnicity, years of education, lipid profile, and hormonal data (menopausal signs and symptoms, surgical and natural menopause, last menses, hormone replacement therapy, and hormonal medications).

The demographic variables were selected for a variety of reasons that were pertinent for the study. Each variable and its rationale in relation to the study is described. In addition, the demographic variables were used to describe the sample.

The demographic variables of age and hormonal factors were selected for the study because they define whether the woman is actually postmenopausal, had surgical or natural menopause, and therefore met the criteria to participate in the study. In addition, the variables of age and hormonal factors define the time in which a woman was no longer protected against CHD and had entered menopause (Douglas, 1993; Flavell, 1994).

Marital status was asked to describe the sample. Ethnicity and education level were also used for descriptive purposes.

The traditional cardiac risk factors as stated in The Risk of Coronary Heart Disease Table (Anderson et al., 1991)--age, sex, HDL cholesterol, total cholesterol, hypertension, cigarette smoking, diabetes mellitus, and electrocardiogram-LVH--were also described as they affect a woman's CHD risk (Douglas, 1993). They were measured by the score on the Risk of Coronary Heart Disease Table (Appendix D) and are described below (Douglas, 1993).

High total blood cholesterol levels (>200 mg/dl) after menopause affect a females CHD risk more than in men. Estrogen increases HDL cholesterol and lowers the total cholesterol. At the time of menopause, HDL cholesterol decreases and total cholesterol increases along with LDL cholesterol and triglycerides, which increase a woman's risk for CHD. Premature menopause, occurring before age 35, was shown to increase the risk of myocardial infarction two- to threefold, and premature oophorectomy (prior to age 35) increased the risk sevenfold. The reason for this increase in risk is that total cholesterol rises at an accelerated rate in women who have premature menopause (Douglas, 1993). Hypertension (systolic >160 mm/hg and/or diastolic blood

pressure >90 mm/hg) increases the risk of CHD fourfold in women as compared to threefold in men. This does appear to be a significant increase between men and women (Douglas, 1993; Flavell, 1994). Smoking decreases the levels of HDL cholesterol, which is cardioprotective thus increasing the risk of CHD. The effects of smoking on the heart are reversible when smoking is stopped, but the rate of reversibility in women has not been studied (Douglas, 1993). Diabetes mellitus raises females' CHD risk equal to a male of the same age. The relationship of family history of CHD risk for women is currently being studied (Douglas, 1993; Flavell, 1994).

Risk of Coronary Heart Disease Table

The Risk of Coronary Heart Disease Table (RCHDT) (Appendix E) was developed from the Framingham Heart Study data (Anderson et al., 1991). It measures an estimate of patients's 5- and 10-year coronary heart disease (CHD) risk. Other CHD risk equations were developed from early Framingham data (1950 to mid-1960s). The data base used for the development of this table was from the baseline examination (1968-1975) of all members of the original Framingham cohort, who are all over 50. The advantages of this CHD risk table over previous ones are: (1) the data base was larger and more recent; (2) there is more data for

individuals over the age of 60 years; and (3) the influence of HDL cholesterol has been added.

To provide current estimates of CHD risk over a large age range, data from the original cohort have been combined with data from the second-generation study population, the Framingham Offspring Cohort. Twelve years of follow-up have been completed. These two groups together span the ages of 12-82 years; however, only persons 30-74 years old were included in this study. Those 75 years or older were excluded because of possible differences in risk factors in this older group and its potentially large influence in the algorithm determination. From the experience of this group during the 12 year period, estimates of CHD risk have been produced that reflect the approximate combined impacts of: (1) total cholesterol, (2) HDL cholesterol, (3) systolic blood pressure, (4) cigarette smoking, (5) diabetes mellitus, and (6) left ventricular hypertrophy (electrocardiogram, ECG-LVH).

The members of the original and offspring cohorts who met three basic criteria were included in the study. Requirements for inclusion were: (1) age 30-74 years at the time of the baseline examination; (2) measurements available for systolic and diastolic blood pressure, cigarette smoking status, total and HDL cholesterol and diagnosis (yes or no)

of diabetes and ECG-LVH (when information on diabetes or LVH was not available, diagnoses were presumed to be negative); and (3) free from cardiovascular disease (stroke, transient ischemia, CHD-angina pectoris, unstable angina, myocardial infarction, and sudden death, congestive heart failure and intermittent claudication) until time of risk factor measurement. The definitions of risk factors and end points are those considered standard in the Framingham study (U.S. Department of Health, Education & Welfare, DHEW, 1976).

A parametric regression model was used for risk estimation. This model provides a simple formula for estimating probabilities of disease given risk factor levels. It has the additional advantage of allowing computation for variable durations of follow-up. These mathematical calculations (Anderson et al., 1991) are based on the Framingham experience for individuals free of cardiovascular disease at baseline measurement. The point scoring technique allows estimation of an individual's 5- and 10-year risks of CHD.

Certain numbers of points are acquired from the risk factor prediction chart on the seven variables previously mentioned. These points are added together for number of total points which is compared to a risk and point total table. The risk and point total table provides the

individuals' 5- and 10-year risk of developing CHD (Anderson et al., 1991).

It has been repeatedly demonstrated that the Framingham risk model is effective in predicting heart disease in other large well white adult population samples: Western Collaborative Group Study (Brand, Rosenman, Sholtz, & Friedman, 1976), National Cohort Study (Leaverton et al., 1987), Multiple Risk Factor Intervention Trials ("Multiple Risk Factor," 1977; Multiple Risk Factor Research Group, 1982). There are no reliability and validity data available on this instrument or the previous risk factor equations developed.

Food Habits Questionnaire

The Food Habits Questionnaire (FHQ) (Appendix F) was designed to measure dietary behavior related to selecting low-fat diets. The conceptual focus of this instrument is on adopting and maintaining a diet low in fat. Traditional measures of nutrition intake, which are often lengthy, susceptible to bias, and costly, do not address health behaviors. The conceptual model for this instrument is based on anthropological research of Bennett (1942), who described dietary patterns by categorizing available foods by their frequency of use: foods used daily, foods used weekly, are the core diet. This model emphasizes:

(a) incorporation of new foods into the non-core diet (expansion/variation), with (b) incorporation of preferred foods into the core diet (expansion and stabilization), and (c) selection of foods in the core diet through rhythmic patterns of inclusion, exclusion, and replacement.

Kristal, Shattuck, and Henry (1990) proposed four dimensions or concepts of dietary behavior that can be used to describe this process: (a) avoiding high-fat foods (exclusion); (b) altering commonly available foods to make them lower in fat (modification); (c) using new specially formulated, lower-fat foods (substitution); and (d) using preparation techniques or food ingredients that replace the common higher-fat alternatives (replacement). The five factors (modify meat, avoid fat as a seasoning, replace a fat serving, substitute a fat serving, and fruit or vegetable) in the Food Habits Questionnaire (Kristal et al., 1990) were derived from these dimensions/concepts.

Three nutritionists developed 86 items for the questionnaire that addressed the four dimensions: exclusion, modification, substitution, and replacement. An expert panel selected 28 items for inclusion in the test instrument; 22 of the items were used in the final questionnaire. Responses to the items were on a 4-level

scale (usually or always, often, sometimes, and rarely/never) (Kristal et al., 1990).

Validity was determined by factor analysis using principal components analysis with prior common estimates set to squared multiple correlations and varimax rotation. The resulting factor structure was used to define the final dietary behavior constructs. The constructs were calculated as the sum of the item scores divided by the number of items. Therefore, all the scores varied from 1 to 4 (Kristal et al., 1990).

Internal consistency and test-retest reliability were calculated for each construct (Kristal et al., 1990). Of the 28 items on the diet behavior questionnaire, only 3 had test-retest reliabilities under .50. According to Waltz, Strickland, and Lenz (1984), a test-retest score of 1.0 indicates complete agreement and 0 indicates lack of agreement. The scale test-retest reliabilities varied from .60 to .90 with an average of .87. The scales were also internally consistent. The internal consistency average was .76. A high degree of internal consistency is 1.0 (Waltz et al., 1984). The factors corresponded well to the dimensions of exclusion, modification, substitution, and replacement. However, exclusion was split between avoidance of meat and

fat as a seasoning. Factor analysis data were not available in Kristal et al.'s article.

Although some of these factors were significantly intercorrelated, they were also more correlated with percentage of calories from fat than with one another. A multiple regression model predicted that the average percentage of calories from fat from all five subscales (modify meat, avoid fat as a seasoning, replace; general foods, substitute, replace; fruits and vegetables) remained statistically significant ($p<.05$), with the exception of meat avoidance ($p<.08$). This statistical significance suggested that the scales do measure different dimensions of dietary behavior related to selecting low-fat diets (Kristal et al., 1990). Kristal et al. did not determine the relationship between the overall score and the statistical significance in the regression model. They calculated the total score in relation to the possible range of scores (1-4) and the percentage of fat.

The Food Habits Questionnaire was correlated with food records and food frequency questionnaires which assured validity (Kristal et al., 1990). Since more than one measure of dietary intake was used, reliability and validity were increased (Nunnally, 1978).

A 22-item Food Habits Questionnaire (FHQ) was administered in this study (Appendix F). Scoring the FHQ is accomplished by completing the Fat Factor Score Sheet (Appendix F), which is composed of five fat factors. These factors are: (1) modify meat; (2) avoid fat as a seasoning; (3) replace; general foods; (4) substitute; and (5) replace; fruit and vegetables.

The question numbers for each fat factor are listed on the score sheet and correspond to the 22-item questionnaire. For each question, the number circled in the response column on the score sheet is recorded. Only one of the items in questions 1, 2, and 11 was scored. The score for each fat factor was calculated by summing the responses and dividing that score by the number of items completed. The foods that are not eaten were not counted. The final score was the sum of the five fat factor scores divided by 5.

Kristal et al. (1990) determined that low scores on the five fat factors correlated with low fat intake. The lower the score, the lower the fat intake. A score of 2.5 or less predicts fat intake at 30% or less.

Stage of Change to Dietary Fat Reduction (SCDFR)

The Stage of Change to Dietary Fat Reduction (SCDFR) (Appendix G), a questionnaire based on the stage model of behavior change to dietary fat reduction (SMBC), is a

staging algorithm (Curry, Kristal, & Bowen, 1992). This questionnaire is a paper and pencil test that measures the person's stage of change (Prochaska & DiClemente, 1992b) related to the amount of fat reduced in the diet. The questionnaire is scored using a 6-question algorithm. The questions are asked in order, and the answer "yes" or "no" to each question guides the participant through the algorithm that determines the individual's stage of change. The score from this instrument is ordinal data. According to Kerlinger (1986), ordinal measures may be treated as interval measurements, but it is imperative to determine whether there is a gross inequality of the intervals. However, most psychological and educational scales approximate interval equality fairly well (Kerlinger, 1986).

The questions to assess the stage of dietary fat reduction were adapted from questions for assessing stage of change for smoking cessation (DiClemente & Prochaska, 1985; DiClemente et al., 1991). The first question divides those individuals into the action or maintenance stages of change. Those who answered "yes" were then asked whether they were currently limiting the amount of fat in their diet and, if so, for how long. The maintenance stage of dietary fat reduction was defined as limiting one's dietary fat for longer than 6 months; the action stage was defined as

limiting one's dietary fat for 6 months or less. The questions for those individuals who were not in the action or maintenance stages referred to changes an individual could make to decrease the amount of fat in their diet.

The contemplation stage was defined in terms of recent (within the past month) consideration of changes that the person could make to reduce dietary fat. Confidence in making changes to reduce fat during the next month defined the decision stage (Curry et al., 1992).

Assessing the stage of dietary fat reduction posed a conceptual challenge. The validity of self-reported stage of fat reduction depends on whether people know if they are eating a high fat diet and if they are making changes which can reduce dietary fat. Therefore, the Food Habit Questionnaire (Kristal et al., 1990) will be used to determine the participant's percentage of fat intake.

Spearman correlation coefficients and one-way analysis of variance were used to assess the univariate associations of stage of dietary change with dietary intake and demographics for the SCDFR. Multiple regression was used to examine the relationship of stage of change with percent calories from fat controlled for the confounding variables (e.g., age, education, and indicators of health status) (Curry et al., 1992).

Curry et al. (1992) showed that the stage of dietary change remained a highly significant ($p<.001$) predictor of percent calories from fat in multiple regression models. Reliability was not determined in the original study (per telephone conversation December 6, 1994 with A. Kristal) utilizing this instrument. It is not possible to calculate Cronbach's alpha because of the stepwise relationship of the questions upon each other (per telephone conversation December 6, 1994 with A. Kristal).

Self-Efficacy for Health-Related Diet Behaviors (SEHRDB) Questionnaire

The Self-Efficacy for Health-Related Diet Behaviors (SEHRDB) Questionnaire (Appendix H) (Sallis, Pinski, Grossman, Patterson, & Nader, 1988) is a 61-item Likert-type scale that measures five factors: resisting relapse, reducing calories, reducing salt, reducing fat, and behavioral skills. Only 34 of the items, including three of the factors (resisting relapse, reducing fat, and behavioral skills), were used in the study. The questionnaire was used to record how subjects felt about making dietary changes.

The investigator's pilot study reliability for the 34 questions used from the original 61 was $\alpha = .865$ ($n = 10$), which is comparable to the alpha coefficients the authors of the questionnaire calculated. The subscale reliabilities

for the investigator's pilot study (n = 10) were .899 (resisting relapse), .601 (reducing fat), and .937 (behavior skills). The reliabilities for the resisting relapse and behavior skills are consistent with the calculations of Sallis et al. (1988). The alpha coefficient for the reducing fat subscale is within the .6 recommended by Nunnally (1978), but may reflect the small sample size of the pilot study. These reliabilities were calculated for the purpose of the study.

Ratings of self-efficacy were obtained using a 5-point Likert-type probability scale, varying in 1-point intervals from 0 to 4 depending on the degree of the subject's perceptions to change dietary behaviors. The scores for each subscale were summed and averaged and yielded a composite score for the individual's self-efficacy. The subscales' weight was not addressed in the article by Sallis et al. (1988). The score is interval level data.

Reliability for the SEHRDB questionnaire (Sallis et al., 1988) was determined by Cronbach's alpha coefficients varying from α = .85 to α = .93 (n = 171). The reliabilities for each subscale are: resisting relapse .93; reducing calories .90; reducing salt .86; reducing fat .85; and behavioral skills .86. These high alpha values

reflect that the questionnaire is measuring the attribute of self-efficacy for health-related diet change (Waltz et al., 1984).

Construct- and criterion-related validity for the self-efficacy scales were assessed through development of a food frequency questionnaire which facilitated quantification of dietary habits. Subjects rated their usual frequency of consumption of 39 food items.

A registered dietician categorized each item as either "heart healthy" or "not heart healthy." These two categories were based on amount of saturated fat and sodium. A "not heart healthy/heart healthy" dietary index was created. The scores varied from 0.08 to 10.50 ($\underline{M}$ = 1.0, $\underline{SD}$ = 1.03) with higher scores indicting a diet higher in fat. The scale is significantly correlated with a 24-hour diet recall interview measure of saturated fat and sodium intake and has been shown to be responsive to dietary change interventions (Sallis et al., 1988). The results of the correlations are unpublished data from The University of California, San Diego, as stated in the article by Sallis et al (1988).

Factor analysis was used to determine the factor structure of each scale using principal-components analysis with varimax rotation. The principle components of the

self-efficacy for eating behavior scale consisted of five factors accounting for 44% of the variance. All of the factors had Eigenvalues of >2.0 (Sallis et al., 1988).

Data Collection

The protocol for conducting the study involved administration of the four questionnaires and the Demographic Data Form to the subjects during a telephone interview. The subjects were recruited through a letter. A description of the study, consent form, and stamped, return envelope were included with the letter. Permission to participate in the study was indicated by returning the signed consent form to the investigator. In addition, other eligible subjects were approached during their clinic appointments and invited to participate in the study. They were given the letter, a description of the study, and a consent form to be signed at that time. The subjects then answered the questionnaires by telephone interview. Cholesterol levels taken within the last year and other demographic data (Appendix D) were obtained from all subjects' medical records.

The Demographic Data Form was read to the subjects first. Upon completion of the Demographic Data Form, the subjects were read the Food Habits Questionnaire (FHQ). Following completion of the FHQ, the subjects were read the

Stages of Change to Dietary Fat Reduction (SCDFM) questionnaire. After the SCDFM was completed, the last questionnaire, the Self-Efficacy for Health-Related Diet Behaviors (SEHRDB), was read to the subjects. Coronary heart disease risk information was collected from their charts and then calculated using The Coronary Heart Disease Risk Table. The risk score was given to the subjects along with an explanation.

Pilot Study

The investigator's pilot study, using a quasi-experimental nonequivalent control group pretest-posttest design, was carried out using a self-efficacy theory-based dietary intervention. Due to difficulty in obtaining the sample size, the logistics of administering the questionnaires, and the intervention at the specified intervals in a corporate setting, it was determined that a change in design was necessary. The current study with an expermental, two-by-two group design was the outgrowth of the pilot study with the addition of another theoretical framework and three questionnaires. Statistical analyses were not completed because of the small sample size.

Treatment of the Data

Data analysis for the study was done to determine differences between the two groups (surgical and natural menopause) of postmenopausal women. Demographic data were reported by frequencies and percentages for nominal level data and frequencies, percentages, means, and standard deviations for interval level data.

The three research questions were analyzed using the two-way analysis of variance (ANOVA) as a statistical test. The two independent variables were (1) surgical and natural menopause, and (2) HRT and no HRT. The dependent variables were risk of coronary heart disease, dietary fat modification, stages of change, and self-efficacy (Munro & Page, 1994; Roscoe, 1975).

A two-way ANOVA is used with more than one independent variable, which allows the investigator to look for differences in dependent variables according to the main effect of the independent variable, as well as the interaction of the independent variables. Testing for interaction allows the researcher to determine whether or not a variable varies depending on the groups or conditions in which it is applied. Testing of the interaction gives information regarding whether or not effects are altered by a combination of factors. This technique allows one to

investigate differences among groups of subjects in relation to an outcome measure (Munro & Page, 1994; Roscoe, 1975). Tables and graphs were used to report the findings.

Summary

Women have been identified in the literature as having coronary heart disease at an older age than men. Many variables reflect these gender differences. Elevated cholesterol levels have been associated with coronary heart disease in both men and women. It is, therefore, important for a combined approach of lowering fat in one's diet and hormone replacement to facilitate a decrease in cholesterol levels for women and thus a decrease in the incidence of coronary heart disease risk.

The theoretical framework for this study utilized the stages of change and self-efficacy which define one's readiness to change. Further defined was the relationship of self-efficacy to a woman's belief that she can comply with a low-fat diet and modify her risk for heart disease during her menopausal years.

This study determined if a motivated health promotion group of postmenopausal women (who had undergone surgical menopause) and a normally motivated health promotion group of postmenopausal women (who had experienced natural menopause) demonstrated any differences between the groups

on the study variables. These variables were: risk of coronary heart disease, dietary modification, stage of change, self-efficacy for health-related diet change, and selected demographic data. The difference between these two groups of postmenopausal women on the stated variables has not been addressed in the literature.

CHAPTER 4

ANALYSIS OF DATA

This nonexperimental descriptive study was conducted using a two-by-two group design. The purpose was to investigate whether the type of menopause and presence or absence of hormone replacement therapy (HRT) in postmenopausal women will affect the variables of risk of coronary heart disease (CHD), dietary fat modification, stages of change, and self-efficacy. Analysis of the data was done by using the Statistical Package for the Social Sciences (SPSS, Norusis, 1990). Demographic data and scores are presented utilizing descriptive statistics. A two-way analysis of variance (ANOVA) was done to analyze the effect of the type of menopause and the presence or absence of hormones on risk of CHD, dietary fat modification, stages of change, and self-efficacy.

Description of Sample

A total of 2,500 consent forms with information about the study were mailed to women between the ages of 45 to 74 years in the selected physician's practice. Of these 2,500 consent forms, 139 (5.6%) forms were signed and returned to

the investigator. Of the 139 potential subjects, 33 (24%) did not participate in the study for the following reasons: 20 (61%) were returned with wrong address, 1 (3%) was 83 years old, 3 (9%) declined to participate by telephone, and 9 (27%) did not respond to the telephone calls. Of the remaining 106 (76%) potential subjects who responded to the telephone interviews, 17 (16%) of these women were still premenopausal and 11 (11%) were perimenopausal. Therefore, 77 (73%) subjects were left who met the criteria for the study. The 77 subjects, surgical or natural postmenopausal women, who comprised the sample for this study used a private health maintenance organization for their health care in a large urban southwestern area.

Of the 77 subjects, 46 (60%) women had undergone surgical menopause and 31 (40%) women had experienced natural menopause. Ages of the total sample varied from 47 to 74 years (M = 59.96 years, SD = 7.61). Ages of the 46 women who had undergone surgical menopause varied from 47 to 74 years, with a mean age of 60 (SD = 7.5) years. Of the surgical menopause women, 8 (17.4%) had one ovary, 16 (34.8%) had both ovaries, 19 (41.3%) had no ovaries, and 1 (2.2%) subject had 1½ ovaries. Two (4.3%) surgical menopause women did not identify number of ovaries. The age range for the 31 women who had experienced natural menopause was 49 to

72 years, with a mean age of 61 (SD = 8) years. Of these women, 30 (96.8%) had both ovaries and 1 (3.2%) had one ovary.

The marital status of the total sample was 41 (53.2%) married, 18 (23.4%) divorced/separated, 7 (9.1%) never married, and 11 (14.3%) widowed. The ethnic mix of the sample was 58 (75.3%) Caucasians, 2 (2.6%) Hispanics, 16 (20.8%) African-Americans, and 1 (1.3%) Asian.

The educational level was ascertained by asking the respondents to report the highest grade or year of school they completed. Of the total sample, two subjects (2.6%) completed elementary school, which was the lowest level attained. Two subjects (2.6%) completed grades 9-12, 16 (20.8%) graduated from high school, 9 (11.7%) completed junior college, 19 (24.7%) completed some college, 7 (9%) graduated from college, and 22 (28.6%) completed graduate school. This sample represented a highly educated group of individuals.

All 77 subjects were without a menstrual period for at least one year. Twenty-three (30%) subjects experienced menopausal symptoms, while 53 (69%) had no symptoms. One subject (1%) did not respond.

Because all of the subjects were postmenopausal, their cholesterol levels should be elevated (American Heart

Association [AHA], 1996; Barrett-Connor, 1994; Castelli, 1988; Everson, Matthews, Guzick, Wing, & Kuller, 1995; Hjortland, McNamara, & Kannel, 1976; Matthews, Wing, Kuller, Meilahn, & Plantinga, 1994; Stampfer, Colditz, & Willett, 1990; Weiss, 1972). The treatment for elevated cholesterol levels is either HRT or cholesterol lowering medication. Therefore, it was important to determine which medication the subjects were taking because the U.S. Department of Health and Human Services' (USDHHS) (1993) National Cholesterol Education Program (NCEP) recommends HRT first before beginning cholesterol lowering medication.

The subjects were asked if they took any cholesterol lowering medication. The purpose of this question was to determine whether the subjects' cholesterol levels were affected by diet alone or diet and cholesterol lowering medication. The numbers of subjects on cholesterol lowering medication alone and those on cholesterol lowering medication and HRT are depicted in Figure 6.

Questions to determine whether the subjects were on hormone replacement therapy (HRT) or not and, if so, how long they were on HRT were asked. The rationale for these questions was that HRT influences cholesterol levels. It also was important to determine whether the subjects were on estrogen alone or in combination with progesterone. Both

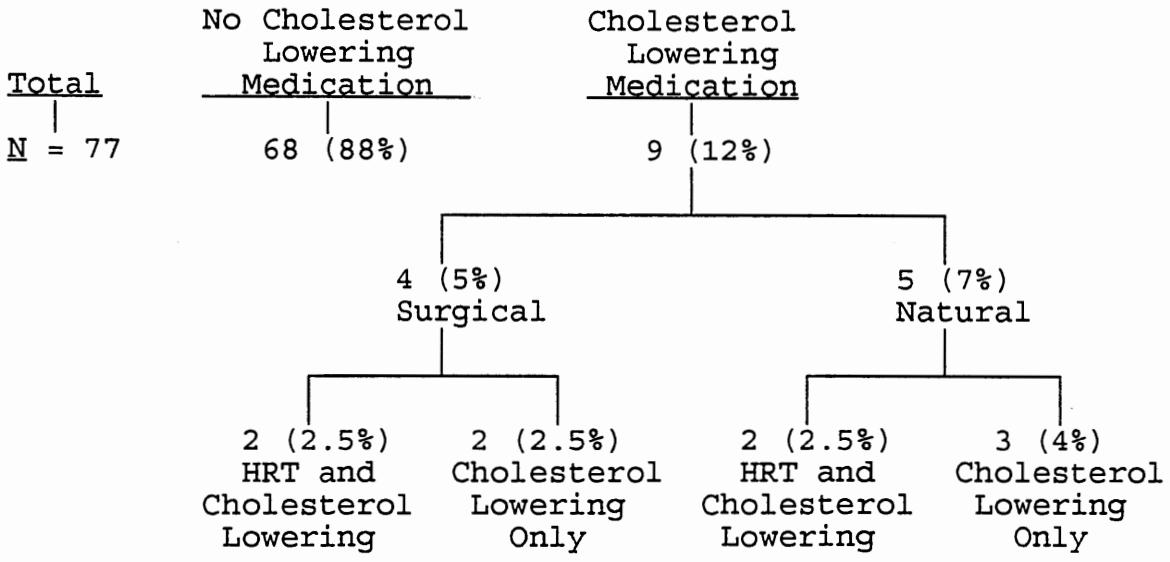


Figure 6. Total Number of Subjects and Cholesterol Lowering Medication

estrogen alone and estrogen and progesterone influence changes in cholesterol levels and its components differently. In addition, HRT, a diet low in saturated fat, and cholesterol lowering medication have all been shown to affect cholesterol levels, thus influencing coronary heart disease risk.

Of the 77 subjects, 45 (58.4%) were on HRT, while 31 (40.3%) were not; one (3.2%) subject did not respond. The length of time the 45 women were on HRT varied from 1 to 43 years (Table 1). Of the 45 subjects on HRT, 30 (66.7%) subjects were on estrogen alone, while 15 (33.3%) were on estrogen and progesterone.

Table 1

Frequency Distribution and Percentages for Length of Time on HRT and Type of HRT Reported by Surgical and Natural Postmenopausal Women (N = 77)

Variable	Surgical		Natural		Total	
	n	%	n	%	n	%
Years on HRT						
1-5	7	15.2	7	22.6	13	16.8
6-10	6	13.0	2	6.5	9	11.7
11-15	8	17.3	1	3.2	9	11.7
16-20	4	8.7	0	0.0	4	5.2
21-25	4	8.7	0	0.0	4	5.2
26-30	3	6.5	0	0.0	3	3.9
31-35	0	0.0	0	0.0	0	0.0
36-43	2	4.3	1	3.2	3	3.9
Total HRT	34	73.8	11	32.5	45	58.4
No Response	0	0.0	1	3.2	1	1.3
No HRT	12	26.0	19	61.3	31	40.3
Total	46	100.0	31	100.0	77	100.0
Type of HRT						
Estrogen Only	30	65.2	0	0.0	30	38.9
Estrogen/ Progestin	3	6.5	12	38.7	15	19.5
No Response	1	2.3	0	0.0	1	1.3
No HRT	12	26.0	19	61.3	31	40.3
Total	46	100.0	31	100.0	77	100.0

Note: One subject in the natural menopause group did not respond to number of years on HRT, while one subject in the surgical menopause group did not respond to type of HRT.

Of the 46 subjects having had surgical menopause, 34 (73.8%) subjects were on HRT and 12 (26%) were not on HRT. Of the 34 subjects on HRT, 30 (65.2%) of them were on estrogen replacement alone and 3 (6.5%) subjects were on an estrogen and progesterone preparation; one (2.3%) subject did report the type of HRT.

The majority (n = 19; 61.3%) of the 31 women who had experienced natural menopause were not on HRT. Estrogen and progesterone was the choice of the 12 (38.7%) subjects who were on HRT. No one with a uterus was on estrogen alone. Certain risk factors have been identified as influential in the development of CHD (Anderson, Wilson, Odel, & Kannel, 1991). The risk factors of total cholesterol, low density lipoprotein (LDL) cholesterol, high density lipoprotein (HDL) cholesterol, blood pressure, smoking status, diabetes, and left ventricular hypertrophy (LVH) were investigated as they relate to postmenopausal women and their risk for CHD.

The 77 subjects in this sample of postmenopausal women did not meet the 1990 objective as set forth by the DHHS: a total cholesterol of at or below 200 mg/dl. The majority of the subjects fell in the borderline-high-risk category (200-239 mg/dl) (Table 2). For the 77 subjects, the total cholesterol levels varied from 148 to 282, with a mean total cholesterol of 219.9 mg/dl (SD = 29.7). The highest and

lowest LDL cholesterol levels for the total sample varied from 234 to 77, with a mean LDL of 138.7 mg/dl (SD = 30.3). For the total sample, the highest and lowest HDL cholesterol levels varied from 92 to 30, and the mean HDL was 54.6 mg/dl (SD = 14.9).

Table 2

Frequency Distribution and Percentages of Total Cholesterol, LDL Cholesterol, and HDL Cholesterol Using NCEP Criteria for Surgical and Natural Menopause Women (N = 77)

Variable	Surgical n	Surgical %	Natural n	Natural %	Total n	Total %
Total Cholesterol						
< 200 mg/dl	9	19.6	6	19.4	15	19.5
200-239 mg/dl	22	47.8	18	58.1	40	51.9
≥ 240 mg/dl	11	23.9	7	22.5	18	23.4
Missing	4	8.7	0	0.0	4	5.2
Total	46	100.0	31	100.0	77	100.0
Mean	220.6		219.0		219.9	
SD	29.3		30.7		29.7	
LDL Cholesterol						
< 130 mg/dl	12	26.1	7	22.6	19	24.7
130-159 mg/dl	11	23.9	13	41.9	24	31.2
≥ 160 mg/dl	8	17.4	5	16.1	13	16.9
Missing	15	32.6	6	19.4	21	27.2
Total	46	100.0	31	100.0	77	100.0
Mean	138.3		139.2		138.7	
SD	35.4		23.2		30.3	
HDL Cholesterol						
< 35 mg/dl	1	2.2	2	6.5	3	3.9
≥ 35 mg/dl	30	65.2	25	80.6	55	71.4
Missing	15	32.6	4	12.9	19	24.7
Total	46	100.0	31	100.0	77	100.0
Mean	59.8		48.7		54.6	
SD	15.5		11.9		14.9	

For the 46 women in the surgical postmenopausal group, the total cholesterol levels varied from 157 to 282, with a mean total cholesterol level of 220.6 (SD = 29.3) which was almost equal to the total cholesterol levels of the 31 women in the natural postmenopausal group. The natural postmenopausal group's total cholesterol levels varied from 198 to 273, with a mean cholesterol level of 219.0 (SD = 30.7) (Table 2).

The LDL cholesterol levels for the 46 women in the surgical postmenopausal group varied from 77 to 234, and the mean was 138.3 (SD = 35.4), which was again similar to the natural postmenopausal group. The 31 women in the natural postmenopausal group had LDL cholesterol levels which varied from 121 to 188, with a mean LDL of 139.2 (SD = 23.2). Both the means for the total cholesterol levels and LDL levels are in the borderline-high-risk category (Table 2).

The HDL cholesterol levels of the 46 women in the surgical postmenopausal group were from 34 to 92, with a mean HDL of 59.8 (SD = 15.5), which was higher than the mean HDL level for the natural postmenopausal group. For the 31 women in the natural postmenopausal group, the HDL cholesterol levels varied from 30 to 80, and the mean was 48.7 (SD = 11.9) (Table 2). Thus, both groups of women would have some protection against heart disease (less than 35

mg/dl of HDL cholesterol is considered not protected against CHD). The surgical postmenopausal group which has a higher mean HDL score (59.8, surgical postmenopausal group vs. 48.7, natural postmenopausal group) would have more protection than the natural postmenopausal group (USDHHS, 1993).

It is important to review the cholesterol levels (total, LDL, and HDL) of the 46 women who were on HRT and the 31 who were not on HRT (Table 3). The women on HRT had almost a 10 mg/dl lower mean total cholesterol level than the women not on HRT (HRT, *M* = 216.4, *SD* 31.2; no-HRT, *M* = 225.1, *SD* = 26.9). However, most of the women in both groups (HRT, *n* = 19, 41.3%; non-HRT, *n* = 21, 67.7%) were in the borderline-high-risk category (200-239 mg/dl). In addition, 14 (30.4%) women in the HRT group were in the high-risk category, compared to 6 (19.4%) women who did not take HRT (Table 3).

For LDL cholesterol (surgical, *M* = 138, *SD* = 35.4; natural, *M* = 139.2, *SD* = 23.2), the mean was about 10 mg/dl lower in the HRT group. A little over half of the women in both the HRT (56.4%) and the no-HRT (54.9%) groups were in the desirable LDL range and the borderline-at-risk LDL range. However, there were about 15% more women from the

Table 3

Frequency Distribution and Percentages of Total Cholesterol, LDL Cholesterol, and HDL Cholesterol Using NCEP Criteria for Women on HRT and Women not on HRT (N = 77)

Variable	HRT n	HRT %	No-HRT n	No-HRT %
Total Cholesterol				
< 200 mg/dl	12	26.1	3	9.7
200-239 mg/dl	19	41.3	21	67.7
≥ 240 mg/dl	14	30.4	6	19.4
Missing	1	2.2	1	3.2
Total	46	100.0	31	100.0
Mean	216.4		225.1	
SD	31.2		26.9	
LDL Cholesterol				
< 130 mg/dl	12	26.1	7	22.6
130-159 mg/dl	14	30.4	10	32.2
≥ 160 mg/dl	5	10.9	8	25.8
Missing	15	32.6	6	19.4
Total	46	100.0	31	100.0
Mean	133.7		144.8	
SD	32.6		26.6	
HDL Cholesterol				
< 35 mg/dl	0	0.0	3	9.7
≥ 35 mg/dl	32	69.6	23	74.2
Missing	14	30.4	5	16.1
Total	46	100.0	31	100.0
Mean	62.3		45.3	
SD	14.6		8.8	

no-HRT group in the high-risk LDL range than women in the HRT group (Table 3).

HDL cholesterol has a protective effect against the development of CHD. Those individuals with an HDL

cholesterol level (surgical, M = 59.8, SD = 15.5; natural, M = 48.7, SD = 11.9) of less than 35 mg/dl are at higher risk for developing CHD. In both the HRT and no-HRT groups, about 70% of the subjects (HRT--n = 32, 69.6%; non-HRT--n = 23, 74.2%) had HDL cholesterol levels greater than 35 (Table 3), which puts both groups at lower risk for developing CHD (USDHHS, 1993).

Cholesterol is one of the three most important risk factors in the development of CHD. Hypertension also remains influential as one of the three most important risk factors (Castelli, 1988; Castelli et al., 1986; Gorodeski & Utian, 1994). The average systolic blood pressure reading for the 77 subjects was 127 (SD = 14.07), and the average diastolic blood pressure was 79 (SD = 8.54) (Table 4). The highest and lowest blood pressure readings were 160/90 and 90/60, respectively, with systolic readings varying from 90 to 160 and diastolic readings varying from 60 to 100. For the surgical postmenopausal group, the average systolic and diastolic readings were 128.7 (SD = 15.4) and 79.5 (SD = 8.9), respectively. The women who had natural menopause had a systolic average of 124.6 (SD = 13.5) and a diastolic average of 78.5 (SD = 8.12). Thus, the entire sample of women reflected a normotensive group.

Table 4

Frequency Distribution and Percentages of Systolic and Diastolic Blood Pressure (BP) for Surgical and Natural Menopause Women (N = 77)

Variable	Surgical n	Surgical %	Natural n	Natural %	Total n	Total %
Systolic BP						
90-100 mmHg	2	4.3	2	6.5	4	5.2
101-110 mmHg	4	8.7	5	16.1	9	11.7
111-120 mmHg	13	28.3	6	19.4	19	24.6
121-130 mmHg	10	21.8	10	32.2	20	26.0
131-140 mmHg	8	17.3	7	22.6	15	19.5
141-150 mmHg	6	13.1	0	0.0	6	7.8
> 150 mmHg	3	6.5	1	3.2	4	5.2
Total	46	100.0	31	100.0	77	100.0
Mean	128.7		124.6		127.0	
SD	15.4		13.5		14.7	
Diastolic BP						
60-70 mmHg	12	26.1	9	29.0	21	27.3
71-80 mmHg	16	34.8	14	45.2	30	38.9
81-90 mmHg	16	34.8	7	22.6	23	29.9
> 90 mmHg	2	4.3	1	3.2	3	3.9
Total	46	100.0	31	100.0	77	100.0
Mean	79.5		78.5		79.1	
SD	8.9		8.1		8.5	

Of the 77 postmenopausal women, the majority (n = 63; 81.8%) of women were non-smokers; only 14 (13.2%) subjects smoked. There were 9 (11.7%) diabetics and 68 (88.3%) non-diabetics. No subjects had left ventricular hypertrophy on their electrocardiogram.

In summary, the demographic data indicated the ages of the surgical and natural postmenopausal women who

participated in this study varied from 47 to 74 years. The majority of the sample was married, Caucasian, and highly educated. Most of the subjects who had surgical menopause still had both ovaries. Only a few subjects (n = 9, 11.68%) were on cholesterol lowering medication. Typically, more women with surgical menopause were on HRT and taking estrogen only than the women with natural menopause. The women with natural menopause who were undergoing HRT were, for the most part, taking estrogen and progestin. The mean total cholesterol levels for the subjects in each group were in the 200-239 mg/dl range which is a moderate elevation. This mean total cholesterol level is above the 200 mg/dl goal for individuals in the U.S. determined by the 1990 Objectives (USDHHS, 1986). The mean blood pressure for women in this study also was within the normal range. More than three-quarters of the sample did not smoke (n = 63, 81.8%) and were not diabetics (n = 68, 88.3%).

Findings

There were two independent variables and four dependent variables in this study of 77 postmenopausal women. The independent variables, type of menopause and HRT status, were measured from the demographic data and findings have

been described. Each of the four dependent variables, CHD risk, dietary fat modification, stages of change, and self-efficacy, were measured independently with four instruments.

Coronary heart disease risk was measured by the Risk of Coronary Heart Disease Table (RCHDT; Appendix E) (Anderson et al., 1991). This table is based on the original Framingham Heart Study data, the second-generation study population, and the Framingham Offspring Cohort.

The total RCHDT points for this sample of 77 postmenopausal women varied from 2 to 23 (M = 10.6, SD = 4.7) (Appendix J, Table A). According to Anderson et al. (1991), the lowest number of points attained on the instrument is 1 and the greatest number of points is 32. The lower the score, the lower the risk for developing CHD. The average number of points attained is 16.5.

The RCHDT measures a woman's risk of developing CHD. The mean score on the RCHDT for women in the total sample was 10.6 (SD = 4.7). The mean score for the surgical group was 9.6 (SD = 4.1) and the natural group was 12.1 (SD = 5.1) (Appendix J, Table A).

The majority of the women had a low risk of developing CHD in the next 5 years (M = 3%, SD = .02). The women who had experienced natural menopause had a slightly higher risk

of CHD in 5 years (M = 3.7, SD = .028) than the women with surgical menopause (M = 2.5%, SD = .019). However, the risk was still considered low for both groups. The 10-year risk for developing CHD for these postmenopausal women was higher (M = 6.9, SD = .046) than the 5-year risk. The women in the natural group had a slightly higher risk of CHD in the 10-year risk group (M = 8.4%, SD = .056) than the surgical group (M = 6%, SD = .035) (Appendix J, Table B).

The mean total points on the RCHDT for the postmenopausal women on HRT was 8.7 (SD = 3.4) and those not on HRT was 12.8 (SD = 5.0). The mean score for women in the HRT group was 8.7 (SD = 3.4) and for the no HRT group, it was 12.8 (SD 5.0) (Appendix J, Table C). For the 5-year risk of developing CHD, the women who were not on HRT had a slightly higher CHD risk (M = 4%, SD = .03) than the women on HRT (M = 2%, SD = .01). The women in the natural menopause group had a higher 10-year risk (M = 9.5%, SD = .06) than the women in the surgical group (M = 5%, SD = .02) (Appendix J, Table D).

In summary, the total sample is at low risk for developing CHD (average CHD risk for the instrument is 5.5% for 5-year and 12.5% for 10-year). The time-sensitive risk

prediction demonstrated a higher risk for developing CHD over time.

The Food Habits Questionnaire (FHQ) (Appendix F) (Kristal, Shattuck, & Henry, 1990) was used to measure dietary behavior related to selecting low-fat diets. The lower the score, the lower the fat intake. A score of 2.5 or less predicts fat intake at 30% or less.

The lowest FHQ score for the 77 subjects in this study was 1.2 and the highest FHQ score was 3.48 (M = 2.55, SD = .46) (Appendix J, Table E) which indicated that the sample was at the 30% level on their fat intake. The 46 (60%) women who experienced surgical menopause had a mean score of 2.6 (SD = .45) on the FHQ, which corresponds closely to the 2.5 required to be at 30% fat intake. The 31 (40%) women who experienced natural menopause had a mean score of 2.5 (SD = .48), which correlates with the 30% fat intake level of the FHQ. Therefore, both groups had a fat intake of only 30%, which indicates a low fat diet.

The 45 (58%) women who were on HRT had a minimum FHQ score of 1.20 and a maximum score of 3.45, with a mean score of 2.55 (SD = .48) (Appendix J, Table F). The 32 (42%) women in the no HRT group had a minimum FHQ score of 1.73 and a maximum score of 3.48, with a mean score of 2.54 (SD = .45). Both groups had mean scores that correlated with a 30% fat

intake level, which indicates they were compliant with a low fat diet.

According to Kristal et al. (1990), test-retest reliability was calculated for each construct on the FHQ. Of the 28 items on the diet behavior questionnaire, only 3 had test-retest reliabilities under 0.50. As pointed out by Waltz, Strickland, and Lenz (1984), a test-retest score of 1.0 indicates a complete agreement and 0 indicates lack of agreement. On Kristal et al.'s scale, test-retest reliabilities varied from .60 to .90, with an average of .87. Reliability for this study using Cronbach's alpha was not done because the questionnaire had a yes or no question before the Likert-type scale item and test-retest was not done because the subjects only completed the instrument once.

Stages of change were measured by administering the Stage of Change to Dietary Fat Reduction (SCDFR) (Appendix G) (Curry, Kristal, & Bowen, 1992). This questionnaire was based on the stage model of behavior change to dietary fat reduction. Each stage was given a number: precontemplation = 5, contemplation = 4, decision = 3, action = 2, and maintenance = 1. The lowest score is associated with a longer-term behavior change. Reliability was not determined in the original study or in this study. Test-retest

reliability was not performed in this study because only one point in time was evaluated to obtain the data for this descriptive study. It is not possible to calculate Cronbach's alpha because of the stepwise relationship of the questions upon each other (per telephone conversation on December 6, 1994 with A. Kristal).

The 77 subjects in this study had a mean SCDFR score of 1.53 (SD = 1.18) which indicated they were in the maintenance stage of change. For the total subjects, the highest number of responses (n = 59, 76.6%) on the five questions of the SCDFR was in maintenance (Appendix J, Table G). Responses of the remaining subjects (n = 18, 23.4%), in descending order, were in the stages of action (n = 9, 11.7%), precontemplation (n = 6, 7.8%), contemplation (n = 2, 2.6%), and decision (n = 1, 1.3%).

The 46 (60%) women in the surgical postmenopausal group had a mean SCDFR score of 1.41 (SD = 1.09) which identified that they were also in the maintenance stage of change. Of the women in the surgical postmenopausal group, 38 (82.6%) subjects scored highest in maintenance (Appendix J, Table G). Scores of the remaining 8 (17.4%) women in the surgical postmenopausal group, in descending order, were in the action (n = 4, 8.7%), precontemplation (n = 3, 6.5%), and

contemplation (n = 1, 2.2%) stages of change. None were in the decision making stage.

The 31 (40%) women in the natural postmenopausal group had a mean SCDFR score of 1.71 (SD = 1.30) which indicated that they too were in the maintenance stage of change. A total of 21 (67.8%) natural menopausal women scored highest in maintenance as well (Appendix J, Table G). Scores of the remaining 10 (32.2%) women in the natural postmenopausal group, in descending order, were in action (n = 5, 16.1%), precontemplation (n = 3, 9.7%), contemplation (n = 1, 3.2%), and decision making (n = 1, 3.2%) stages of change.

Of the 77 subjects, 45 (58%) were on HRT. For the postmenopausal women on HRT, the mean SCDFR score was 1.50 (SD = 1.21) which indicated this group of women was in the maintenance stage of change. Of the women in the HRT group, 37 (82.2%) subjects scored highest in maintenance (Appendix J, Table H). Scores of the remaining 8 (17.8%) women in the HRT group, in descending order, were in the precontemplation (n = 4, 8.9%), action (n = 3, 6.7%), and contemplation (n = 1, 2.2%) stages of change. None were in the decision making stage.

Of the total sample (n = 77) of postmenopausal women, 32 (42%) women had no HRT. For these women with no HRT, the mean SCDFR score was 1.59 (SD = 1.13) which also identified

this group of women as being in the maintenance stage of change. Of the women in the no HRT group, 22 (68.8%) subjects scored highest in maintenance (Appendix J, Table H). Scores of the remaining 10 (31.2%) women in the no HRT group, in descending order, were in the action (n = 6, 18.8%), precontemplation (n = 2, 6.3%), contemplation (n = 1, 3.1%), and decision making (n = 1, 3.1%) stages of change.

Overall, the results of the SCDFR analysis indicated that the majority of the postmenopausal women in the total sample for the type of menopause (surgical and natural) and for HRT status (HRT and no HRT) were following a low fat diet regularly for more than one year. These results were validated by findings of the FHQ: an average FHQ score of 2.55 (SD = .46) which indicated subjects were following a diet of about 30% fat.

The Self-Efficacy for Health-Related Diet Behaviors (SEHRDB) Questionnaire (Appendix H) (Sallis, Pinski, Grossman, Patterson, & Nader, 1988) was used to measure how positive subjects were about making dietary changes. Reliability for the entire SEHRDB calculated for this study yielded α = .897 (N = 77). Scores of the total sample (N = 77) for the entire questionnaire indicated the highest individual score was 3.97 of a highest possible score of 4.0

and the lowest score was .76 of a lowest possible score of 0. The mean score for the questionnaire was 2.0 (SD not reported). The mean score for the total sample of 77 postmenopausal women was 2.6 (SD = .63). This mean score reflected a moderate level of self-efficacy for the entire sample (Appendix J, Table I).

For the 46 (60%) women in the surgical postmenopausal group, SEHRDB scores varied from 1.35 to 3.97 (Appendix J, Table I). The mean SEHRDB score for the surgical postmenopausal group was 2.59 (SD = .61), which reflected a moderate level of self-efficacy.

For the 31 (40%) women in the natural postmenopausal group, scores on the SEHRDB varied from 0.76 to 3.76 (Appendix J, Table I). The mean SEHRDB score for the natural menopausal group was 2.62 (SD = .65) which also reflected a moderate level of self-efficacy.

A total of 45 (58%) of the 77 subjects were on HRT. Scores on the SEHRDB for this HRT group varied from 1.35 to 3.97 (Appendix J, Table J). A mean SEHRDB score of 2.66 (SD = .64) was obtained from the scores of the HRT group of women. This mean score reflected that self-efficacy was at a moderate level for the HRT group.

The no HRT group of postmenopausal women included 32 (42%) of the total 77 subjects. The SEHRDB scores for the

no HRT group varied from 0.76 to 3.82 (Appendix J, Table J). The mean SEHRDB score for the no HRT group was 2.51 (SD = .60) which indicated that the no HRT group was at a moderate level of self-efficacy.

All data were collected during 15-20 minute telephone interviews for the 77 subjects using the demographic sheet and the four questionnaires. The two-way analysis of variance (ANOVA) was used to analyze the three research questions. These questions explored the differences between postmenopausal women and their hormone replacement status (HRT) and type of menopause on the variables of risk of coronary heart disease, dietary fat modification, stages of change, and self-efficacy for health-related diet behaviors. In addition, the interaction between the type of menopause and the use of HRT with regard to the risk of coronary heart disease, dietary fat modification, stages of change, and self-efficacy for health-related diet behaviors in postmenopausal women was examined.

Research Question 1

The first research question of this study was: Is there a difference between postmenopausal women who have undergone surgical menopause and those who have experienced natural menopause with regard to risk of coronary heart disease, dietary fat modification, stages of change, and

self-efficacy for health-related diet behaviors? A summary ANOVA table for each of the four dependent variables is included with each variable. A table of means and standard deviations for the four dependent variables is also included with each variable.

Coronary Heart Disease (CHD)

There was no significant difference in the risk of CHD between the surgical and natural postmenopausal women (Table 5). The CHD risk mean for the total group was 10.65 (SD = 4.65). There was a 2.48 difference in the mean scores for CHD risk in the surgical and natural postmenopausal groups. The natural postmenopausal group had a higher mean CHD risk (12.13, SD = 5.08) than the surgical postmenopausal group (9.65, SD = 4.09). The means and standard deviations for CHD risk were calculated for all groups (Table 6).

5-Year Risk of CHD

There was no significant difference in the 5-year risk for CHD between women who underwent surgical menopause and those who experienced natural menopause (Table 7). The 5-year risk of CHD total mean score was .03 (SD = .023). The surgical and natural postmenopausal group had almost equal means--.03 (SD = .018) and .04 (SD = .028), respectively.

Table 5

Summary ANOVA Table for CHD Risk for Surgical and Natural and HRT and No HRT Postmenopausal Women (N = 77)

Source	df	SS	MS	F	p
CHD Risk					
Main Effects	2	391.370	195.680	11.44	.000*
Type of Menopause	1	12.080	12.080	.71	.403
HRT/No HRT	1	277.750	277.750	16.24	.000*
2-Way Interactions					
Type of Menopause/ HRT and No HRT	1	3.700	3.700	.22	.643

*$p < .001$

Table 6

Summary of Means and Standard Deviations of CHD Risk for Postmenopausal Women by Surgical and Natural and by HRT and No HRT Groups (N = 77)

	HRT			No HRT			Total		
Group	n	M	SD	n	M	SD	n	M	SD
Surgical	34	8.68	3.50	12	12.42	4.52	46	9.65	4.09
Natural	11	9.09	2.98	20	13.80	5.28	31	12.13	5.08
Total	45	8.78	3.36	32	13.28	4.98	77	10.64	4.65

The means and standard deviations for 5-year risk of CHD were calculated for all groups (Table 8).

Table 7

Summary ANOVA Table for 5-Year CHD Risk for Surgical and Natural and HRT and No HRT Postmenopausal Women (N = 77)

Source	df	SS	MS	F	p
5-Year Risk of CHD					
Main Effects	2	.008	.004	9.10	.000*
Type of Menopause	1	.000	.000	.66	.420
HRT/No HRT	1	.006	.006	12.65	.001*
2-Way Interactions					
Type of Menopause/ HRT and No HRT	1	.000	.000	.63	.432

*$p < .001$

Table 8

Summary of Means and Standard Deviations of 5-Year CHD Risk for Postmenopausal Women by Surgical and Natural and by HRT and No HRT Groups (N = 77)

	HRT			No HRT			Total		
Group	n	M	SD	n	M	SD	n	M	SD
Surgical	34	.02	.01	12	.04	.03	46	.03	.02
Natural	11	.02	.01	20	.05	.03	31	.04	.03
Total	45	.02	.01	32	.04	.03	77	.03	.02

10-Year Risk of CHD

No significant difference was found in the 10-year CHD risk between the surgical and natural postmenopausal women (Table 9). The 10-year risk of CHD had a total mean of .07 (*SD* = .05). For type of menopause, the natural postmenopausal group had a higher mean--.08 (*SD* = .06) than the surgical postmenopausal group--.06 (*SD* = .03). The means and standard deviations for 10-year risk of CHD were calculated for all groups (Table 10).

Table 9

Summary ANOVA Table for 10-Year CHD Risk for Surgical and Natural and HRT and No HRT Postmenopausal Women (N = 77)

Source	*df*	*SS*	*MS*	*F*	*p*
10-Year Risk of CHD					
Main Effects	2	.038	.019	11.49	.000*
Type of Menopause	1	.001	.001	.77	.383
HRT/No HRT	1	.027	.027	16.15	.000*
2-Way Interactions					
Type of Menopause/ HRT and No HRT	1	.001	.001	.91	.345

*$p < .001$

Table 10

Summary of Means and Standard Deviations of 10-Year CHD Risk for Postmenopausal Women by Surgical and Natural and by HRT and No HRT Groups (N = 77)

Group	HRT			No HRT			Total		
	n	M	SD	n	M	SD	n	M	SD
Surgical	34	.05	.02	12	.08	.05	46	.06	.03
Natural	11	.05	.02	20	.10	.06	31	.08	.06
Total	45	.05	.02	32	.10	.06	77	.07	.05

Dietary Fat Modification

There were no significant differences in dietary fat modification between the women who had surgical menopause and those who experienced natural menopause (Table 11). The dietary fat modification total mean score was 2.55 (SD = .46). The surgical and natural postmenopausal groups had similar mean scores--2.60 (SD = .45) and 2.47 (SD = .48), respectively. The means and standard deviations for dietary fat modification were computed for all groups (Table 12).

Stages of Change

There were no significant differences in the stage of change between the surgical and natural postmenopausal women (Table 13). The total mean score for the stages of change was 1.53 (SD = 1.18). The natural postmenopausal group had

Table 11

Summary ANOVA Table for Dietary Fat Modification for Surgical and Natural and HRT and No HRT Postmenopausal Women (N = 77)

Source	df	SS	MS	F	p
Dietary Fat Modification					
Main Effects	2	.367	.184	.863	.426
Type of Menopause	1	.364	.364	1.710	.195
HRT/No HRT	1	.362	.032	.152	.697
2-Way Interactions					
Type of Menopause/ HRT and No HRT	1	.310	.310	1.458	.231

Table 12

Summary of Means and Standard Deviations for Dietary Fat Modification for Postmenopausal Women by Surgical and Natural and by HRT and No HRT Groups (N = 77)

	HRT			No HRT			Total		
Group	n	M	SD	n	M	SD	n	M	SD
Surgical	34	2.60	.44	12	2.50	.49	46	2.60	.45
Natural	11	2.30	.54	20	2.50	.43	31	2.50	.48
Total	45	2.55	.48	32	2.54	.45	77	2.55	.46

a higher mean--1.71 (SD = 1.3) than the surgical postmenopausal group--1.41 (SD = 1.1). The means and

standard deviations for stages of change were calculated for all groups (Table 14).

Table 13

Summary ANOVA Table for Stages of Change for Surgical and Natural Postmenopausal Women (N = 77)

Source	df	SS	MS	F	p
Stages of Change					
Main Effects	2	1.630	.815	.575	.565
Type of Menopause	1	1.430	1.430	1.005	.319
HRT/No HRT	1	.001	.001	.001	.975
2-Way Interactions					
Type of Menopause/ HRT and No HRT	1	.004	.004	.003	.957

Table 14

Summary of Means and Standard Deviations for Stage of Change to Dietary Fat Reduction Scores for Postmenopausal Women by Surgical and Natural and by HRT and No HRT Groups (N = 77)

	HRT			No HRT			Total		
Group	n	M	SD	n	M	SD	n	M	SD
Surgical	34	1.41	1.08	12	1.42	1.16	46	1.41	1.09
Natural	11	1.73	1.62	20	1.70	1.13	31	1.71	1.30
Total	45	1.49	1.22	32	1.59	1.13	77	1.53	1.18

The SCDFR questionnaire was based on the stage model of behavior change to dietary fat reduction. Each stage was given a number: precontemplation = 5, contemplation = 4, decision making = 3, action = 2, and maintenance = 1. The lowest change is associated with a longer-term change. The means for the total group and the two groups (surgical and natural) reflect the subjects in both groups are in the maintenance stage of change.

Self-Efficacy

There were no significant differences in self-efficacy for the postmenopausal women who had surgical or natural menopause (Table 15). The total mean for self-efficacy was 2.60 (SD = .63). The mean scores for the surgical--2.59 (SD = .61) and natural--2.62 (SD = .65) postmenopausal groups had a difference of only 0.03. The means and standard deviations for stages of change were calculated for the groups (Table 16).

Research Question 2

The second research question was: Is there a difference between hormone replacement therapy (HRT) and no HRT in postmenopausal women with regard to risk of coronary heart disease, dietary fat modification, stages of change, and self-efficacy for health related diet behaviors?

Table 15

Summary ANOVA Table for Self-Efficacy for Postmenopausal Women by Surgical and Natural and by HRT and No HRT Groups (N = 77)

Source	df	SS	MS	F	p
Self-Efficacy					
Main Effects	2	.624	.312	.79	.46
Type of Menopause	1	.182	.182	.46	.49
HRT/No HRT	1	.605	.605	1.53	.22
2-Way Interactions					
Type of Menopause/ HRT and No HRT	1	.287	.287	.74	.38

Table 16

Summary of Means and Standard Deviations for Self-Efficacy for Postmenopausal Women by Surgical and Natural and by HRT and No HRT Groups (N = 77)

	HRT			No HRT			Total		
Group	n	M	SD	n	M	SD	n	M	SD
Surgical	34	2.61	.64	12	2.43	.56	46	2.59	.61
Natural	11	2.84	.66	20	2.50	.64	31	2.61	.65
Total	45	2.66	.64	32	2.51	.60	77	2.60	.63

Two-way ANOVAs were used to determine the differences that existed between the postmenopausal women and HRT and no-HRT

status and the risk of CHD, dietary fat modification, stages of change, and self-efficacy.

CHD Risk

There was a significant difference in CHD risk between the postmenopausal women who had HRT and those who did not take HRT ($F(1, 76) = 16.24$, $p = .000$) (see previous Tables 5 and 6). The HRT group of postmenopausal women had a lower mean CHD risk score (8.78, $SD = 3.4$), which put these women at 2.7% CHD risk, than the postmenopausal women who were not taking HRT (13.28, $SD = 5.0$), which put these women at 4.2% CHD risk.

5-Year Risk for CHD

Hormone status showed a significant difference in 5-year CHD risk ($F(1, 76) = 12.65$, $p = .001$) (see previous Tables 7 and 8) between those women not on HRT and those women on HRT. The group of postmenopausal women who did not use HRT had a greater mean 5-year risk of 4% for developing CHD (0.4, $SD = .03$) when compared with the postmenopausal women (2% 5-year risk) who used HRT (0.2, $SD = .01$).

10-Year Risk for CHD

There was a significant difference for the postmenopausal women and their hormone status ($F(1, 76) = 16.15$, $p = .000$) (see previous Tables 9 and 10). The

postmenopausal women who were not on HRT had a 10% greater mean (M = .10, SD = .06) 10-year CHD risk than those postmenopausal women (5% 10-year risk) who were taking HRT (M = .05, SD = .02).

Dietary Fat Modification

There was no significant difference in dietary fat modification for the postmenopausal women who were on HRT and those who had no HRT (see previous Tables 11 and 12). The hormonal status means for dietary fat were almost equal: 2.55 (SD = .48) for HRT and 2.54 (SD = .45) for no HRT. Both of these groups were eating slightly greater than 30% dietary fat.

Stages of Change

No significant differences in stages of change were found between the women who were on HRT and those who had no HRT (see previous Tables 13 and 14). The postmenopausal women who were not on HRT had a higher mean (1.59, SD = 1.1) on the stages of change dependent variable than those postmenopausal women who were on HRT (M = 1.49, SD = 1.2). Both of these groups were in the maintenance stage of change, which is greater than one year of eating a low fat diet.

Self-Efficacy

No significant differences were found in self-efficacy between the postmenopausal women who used HRT and those who did not use HRT (see previous Tables 15 and 16). The postmenopausal women in the HRT group of had a mean self-efficacy of 2.66 (SD = .64), and the no HRT group had a mean self-efficacy of 2.51 (SD = .60). This level of self-efficacy was moderate.

Research Question 3

The third research question was: Is there an interaction between the use of HRT and the type of menopause, natural or surgical, with regard to risk of coronary heart disease, dietary fat modification, stages of change, and self-efficacy for health-related diet behaviors? Two-way ANOVAs were used to determine if an interaction occurred between the two independent variables, type of menopause and hormone status, and the four dependent variables, CHD risk, dietary fat modification, stages of change, and self-efficacy. The summary ANOVA tables and tables of means and standard deviations are depicted under Research Question 1.

There was no significant interaction of the two independent variables, type of menopause and hormone status when evaluating risk of CHD (Tables 5 and 6), dietary fat

modification (Tables 11 and 12), stages of change (Tables 13 and 14), and self-efficacy (Tables 15 and 16). In addition, both the 5-year (Tables 7 and 8) and 10-year (Tables 9 and 10) risk of CHD showed no significant interaction for the two independent variables as well.

Summary of Findings

This chapter contained the results of the descriptive statistical analyses which described the sample of 77 postmenopausal women. Results of the instruments measuring the dependent variables were reported. In addition, findings related to the three research questions were presented.

CHD Risk

Coronary heart disease risk showed significant differences with HRT status. The postmenopausal women who took HRT had a lower score than the postmenopausal women who did not take HRT. Thus, the postmenopausal women who took HRT had a lower risk for CHD. In addition, the 5-year and 10-year risk for CHD demonstrated significant differences, so a greater risk for the postmenopausal women not on HRT would be evident at these time points as well. In fact, at the 10-year risk point, the mean doubled from .05 (*SD* = .02) for HRT users to .10 (*SD* = .06) for no HRT users.

There were no significant differences for the type of menopause and CHD risk, 5-year risk, and 10-year risk. Thus, the women with surgical and natural menopause were similar on these three dependent variables. In addition, no significant interaction was shown for type of menopause and hormone replacement status and the dependent variables of CHD risk, 5-year risk, and 10-year risk.

Dietary Fat Modification

There were no significant differences for the type of menopause or HRT status and dietary fat modification. No interaction was found between the independent variables of type of menopause and HRT status on the dependent variable of dietary fat modification.

Stages of Change

No significant differences were demonstrated for the independent variables of type of menopause and HRT status and the dependent variable of stages of change. Thus, both of the groups for both independent variables were similar on the variable of stages of change. When looking at the means for both types of menopause (surgical--M = 1.41, SD = 1.1; natural--M = 1.71, SD = 1.3) and HRT status (HRT--M = 1.49, SD = 1.2; no-HRT--M = 1.59, SD = 1.3) on the variable of stages of change, each of the groups is in the maintenance

stage of change. This stage of change is described by Curry, Kristal, and Bowen (1992) as maintaining a low fat diet for more than one year. In addition, no significant interaction occurred between type of menopause, HRT status, and the stages of change.

Self-Efficacy

There were no significant differences between the type of menopause and HRT status in postmenopausal women and the dependent variable of self-efficacy. The means for the two independent variables (surgical menopause--*M* = 2.59, *SD* = .61; natural menopause--*M* = 2.62, *SD* = .65; HRT--*M* = 2.66, *SD* = .64; no HRT--*M* = 2.51, *SD* = .60) were almost equal. Thus, according to Sallis et al. (1988), this finding showed that all the groups for both independent variables fell in the moderate level of self-efficacy category. Additionally, there was no significant interaction between the type of menopause, hormone status, and self-efficacy.

CHAPTER 5

SUMMARY OF THE STUDY

The purpose of this study was to determine whether a difference existed in postmenopausal women who were on hormone replacement therapy (HRT), those who were not on HRT, those who had undergone surgical menopause, or had experienced natural menopause on the variables of coronary heart disease (CHD) risk, dietary fat modification, stages of change, and self-efficacy for health-related diet behavior. Combining self-efficacy theory (Bandura, 1977b) and Prochaska and DiClemente's (1992b) transtheoretical model of change, it was possible to determine the stages of change and self-efficacy levels of the postmenopausal women to see which group was more focused on health promotion and disease prevention behaviors. A summary of the study is provided along with a discussion of the findings as compared and contrasted with relevant literature. In addition, conclusions and implications are presented. Recommendations for further research are offered.

Summary

The health promotion behavior of adopting a low fat diet and CHD risk in postmenopausal women with surgical and natural menopause was examined in this study. This model proposed that self-efficacy changes as an individual's stage of change evolves. Findings from research have shown that the level of self-efficacy increases as the stage of change moves from precontemplation to contemplation to decision to action and to maintenance. The highest level of self-efficacy is associated with the maintenance stage of change (DiClemente, 1981; DiClemente & Prochaska, 1985; DiClemente, Prochaska, & Gibertini, 1985; DiClemente et al., 1991; Prochaska, 1984; Prochaska, Crimi, Lapsanski, Martel, & Reid, 1982; Prochaska & DiClemente, 1992b).

A nonexperimental two-by-two group design was used to examine the main effects and the interaction of the type of menopause and hormone replacement therapy (HRT) on the dependent variables: (1) risk of coronary heart disease (CHD), (2) dietary fat modification, (3) stages of change, and (4) self-efficacy for health-related diet change. These variables were evaluated at a single point in time to determine significant differences between groups on health promotion behaviors. The data from this evaluation were used to address the three research questions of this study.

The two independent variables in this study, surgical or natural menopause and HRT or no HRT, were measured from the demographic data. Each of the four dependent variables, CHD risk, dietary fat modification, stages of change, and self-efficacy, were measured independently with the four instruments: Risk of Coronary Heart Disease Table (RCHDT) (Anderson et al., 1991); Food Habits Questionnaire (FHQ) (Kristal, Shattuck, & Henry, 1990); Stage of Change to Dietary Fat Reduction (SCDFR) (Curry, Kristal, & Bowen, 1992); and Self-Efficacy for Health-Related Diet Behaviors (SEHRDB) Questionnaire (Sallis, Pinski, Grossman, Patterson, & Nader, 1988). These instruments were administered via 15-20 minute telephone interviews.

The sample included 77 surgical or natural postmenopausal women from ages 47 to 74 years (M = 60.5) who used a private health maintenance organization for their health care in a large urban southwestern area. Of the 77 subjects, 46 (60%) women had undergone surgical menopause and 31 (40%) women had experienced natural menopause. The majority of the sample was married, Caucasian, and highly educated. Most of the subjects who had surgical menopause had both ovaries removed. Only a few subjects were on cholesterol lowering medication. Typically more women with surgical menopause were on HRT and taking estrogen only than

the women with natural menopause. The latter group with natural menopause and on HRT were, for the most part, taking estrogen and progestin.

Discussion of Findings

The demographic characteristics of the sample reflected the usual profile of an HRT user, which according to Matthews, Kuller, Wing, Meilahn, and Plantinga (1996) includes higher education, higher high density lipoproteins (HDL), and lower systolic and diastolic blood pressures than non-HRT users. It is therefore important to conduct randomized trials including all socioeconomic groups of women to estimate the direct benefit of postmenopausal HRT for protecting all women against CHD.

The literature (AHA, 1996; Castelli, 1988; Flavelle, 1994) supported the fact that as women age and become menopausal, their death rate from CHD increases threefold to fivefold. In this study, the researcher utilized The Risk of Coronary Heart Disease Table (RCHDT) (Anderson, Wilson, Odell, & Kannel, 1991) to examine this risk in women according to type of menopause and hormone replacement status.

The average total score for the Risk of Coronary Heart Disease Table (RCHDT) is 16.5 (Anderson et al., 1991). The surgical and natural postmenopausal groups in this study had

mean scores (9.6 and 12.1, respectively) which were well below the average for the instrument. The HRT and no-HRT groups also had mean scores (8.7 and 12.8, respectively) which were well below the average for this instrument.

Thirteen percent was the average score for the 5-year risk of acquiring CHD. The surgical postmenopausal group had an average risk of 2.5%. The natural postmenopausal group had an average risk of 3.7%.

For the 10-year risk of developing CHD, the average score was 23%. In this study, the surgical postmenopausal group had an average of 6% and the natural postmenopausal group had an average of 8.4% 10-year risk for developing CHD. The HRT postmenopausal group had an average of 5% and the no-HRT postmenopausal group had an average of 19% 10-year risk for developing CHD.

The one finding in these data that seems to stand out is the 19% 10-year risk for the no-HRT postmenopausal group to develop CHD. This group seems to be at highest risk for developing CHD.

The results of the Food Habits Questionnaire (FHQ) showed that for the type of menopause (surgical--$\underline{M}$ = 2.6, natural--$\underline{M}$ = 2.5) and for the hormone status (HRT--$\underline{M}$ = 2.55, no-HRT--$\underline{M}$ = 2.54) postmenopausal groups were eating a diet with only 30% fat content. This percentage is considered a

low fat diet and corresponds to the AHA (1996) low fat diet which is used to treat hypercholesterolemia. In addition, findings from the Stage of Change to Dietary Fat Reduction (SCDFR) questionnaire showed that the majority of all subjects were in the maintenance (score = 1, but < 2) stage of change (surgical $\underline{M}$ = 1.41, natural $\underline{M}$ = 1.71, HRT $\underline{M}$ = 1.50, no-HRT $\underline{M}$ = 1.59). The maintenance stage of change is defined as maintaining a low fat diet for more than one year. Since data from both of the instruments correlated, it can be determined that the subjects in this study ate a low fat diet for greater than one year.

The majority of postmenopausal women in this study had a moderate level of self-efficacy for health related diet behaviors and were in the maintenance stage of change for adopting a low fat diet. This finding is well supported in the literature by numerous researchers (DiClemente, 1981; DiClemente et al., 1985; DiClemente et al., 1991; DiClemente & Prochaska, 1985; Prochaska, 1991; Prochaska et al., 1982).

Research Question 1

The first research question proposed that there was a difference between the two groups of postmenopausal women (surgical and natural) with regard to risk of CHD, dietary fat modification, stages of change, and self-efficacy for health-related diet behaviors. Interval level data were

low fat diet and corresponds to the AHA (1996) low fat diet which is used to treat hypercholesterolemia. In addition, findings from the Stage of Change to Dietary Fat Reduction (SCDFR) questionnaire showed that the majority of all subjects were in the maintenance (score = 1, but < 2) stage of change (surgical $\underline{M}$ = 1.41, natural $\underline{M}$ = 1.71, HRT $\underline{M}$ = 1.50, no-HRT $\underline{M}$ = 1.59). The maintenance stage of change is defined as maintaining a low fat diet for more than one year. Since data from both of the instruments correlated, it can be determined that the subjects in this study ate a low fat diet for greater than one year.

The majority of postmenopausal women in this study had a moderate level of self-efficacy for health related diet behaviors and were in the maintenance stage of change for adopting a low fat diet. This finding is well supported in the literature by numerous researchers (DiClemente, 1981; DiClemente et al., 1985; DiClemente et al., 1991; DiClemente & Prochaska, 1985; Prochaska, 1991; Prochaska et al., 1982).

Research Question 1

The first research question proposed that there was a difference between the two groups of postmenopausal women (surgical and natural) with regard to risk of CHD, dietary fat modification, stages of change, and self-efficacy for health-related diet behaviors. Interval level data were

obtained. The two-way analysis of variance (ANOVA) was used to analyze the results. The total CHD risk scores indicated there was no significant difference between the two groups on this variable ($F(1, 76) = 5.57$, $p = .21$). There was no significant difference between the variables of 5-year risk, 10-year risk, dietary fat modification, stages of change, and self-efficacy for health-related diet behavior change. The women in both groups ate less than 30% dietary fat, the majority were in the maintenance stage of change and had moderate self-efficacy. Data comparing the surgical and natural groups on the variable of CHD risk does not support the research question.

The literature is conflicting when determining the difference in the type of menopause and CHD. The Framingham Study data (Gordon, Kannel, Hjortland, & McNamara, 1978) demonstrated that there a similar CHD risk between women who had surgical and natural menopause. On the other hand, the Nurses Health Study data (Colditz, Willett, Stampfer, Rosner, Speizer, & Hennekens, 1987) showed a significant increase in the risk of CHD among women who had surgical menopause over those who had natural menopause. HRT was not an issue in these two studies because it was not widely prescribed when the studies were done. There was no

literature found comparing the type of menopause, dietary fat, stages of change, or self-efficacy.

Research Question 2

The second research question examined whether there was a difference between HRT and no-HRT in postmenopausal women with regard to risk of CHD, dietary fat modification, stages of change, and self-efficacy for health related diet behaviors. Results of the two-way ANOVAs showed a significant difference between women who were on no-HRT and those on HRT ($F(1, 76) = 16.24$, $p = .000$ for risk of CHD; $F(1, 76) = 12.65$, $p = .001$ for 5-year risk; $F(1, 76) = 16.5$, $p = .000$ for 10-year risk) (Tables 5-10). Those women who were not on HRT had a significantly higher risk for CHD than did those women who were on HRT. This part of the research question was supported. The other three dependent variables demonstrated no significant differences between HRT and no-HRT women. In addition, the 5- and 10-year probabilities of developing CHD were significantly greater in the women who did not take HRT.

The epidemiologic studies (Barrett-Connor & Bush, 1991; Grady et al., 1992; Grodstein & Stampfer, 1995; Grodstein et al., 1996; Stampfer & Colditz, 1991; Stampfer & Grodstein, 1994) done showed a CHD protective effect in women who took estrogen. Most of the studies showed a 50% reduction in risk

of CHD in those women who used estrogen. The pooled relative risk for developing CHD in current estrogen users was 0.50 (95% confidence interval, 0.45-0.59).

The results of these epidemiological studies are supported by the significant difference found by this study on the variable of HRT status and risk of CHD including the 5-year and 10-year risk for developing CHD. Because of the strength of the statistical analysis performed in this study, the differences found between the variables of CHD risk and hormone status are more firmly established than the epidemiological associations found in the previously mentioned studies.

There are no studies reported that examined HRT prospectively to evaluate the effects of HRT. However, findings from this study show that over time (10-year CHD risk) that women not on HRT will have a greater CHD risk. It is recommended that a double-blind, randomized prospective clinical trial be undertaken to more firmly establish a relationship between CHD and HRT use. Currently, the Women's Health Initiative is underway. Data are expected to be published in 2007. In addition, there were no studies found in the literature evaluating hormone status and dietary fat, stages of change, and self-efficacy.

Research Question 3

The third research question proposed an interaction between the use of HRT and the type of menopause, natural or surgical, with regard to risk of CHD, dietary fat modification, stages of change, and self-efficacy for health-related diet behaviors. Using a two-way ANOVA, no significant interaction was found between these variables. Thus, the research question was not supported.

The literature supports the finding of this research question which indicated there was no significant interaction between the variables of type of menopause and hormone status. Stampfer and Colditz (1991) and Stampfer and Grodstein (1994) cited in their epidemiological reviews of research studies on HRT and CHD that no differences between the type of menopause and HRT status were found.

Conclusions

The following conclusions have been drawn from the findings of this study. These conclusions relate to postmenopausal women, the CHD risk, and their health promotion behaviors. The conclusions also may facilitate the development of health promotion programs for this particular group of women.

1. The demographic characteristics of the postmenopausal women in this sample reflect a similar profile to that of HRT users in the literature.
2. The results of this study showed women on HRT had a lower risk of developing CHD than women not on HRT. The likelihood for developing CHD risk was also greater at the projected 5-year and 10-year time periods for women who were not on HRT.
3. All of the participants in this study showed a low CHD risk and a low 5- and 10-year probability of developing CHD.
4. The Food Habits Questionnaire scores reflected that the participants were eating a diet of less than or equal to 30% fat and were in the maintenance stage of change for adopting a diet low in saturated fat, which reflects a healthy lifestyle.
5. The postmenopausal women who participated in this study for the most part had moderate levels of self-efficacy and were in the maintenance stage of change for a low fat diet. This finding reflects DiClemente and Prochaska's findings of self-efficacy and stages of change.

Implication

Based on the findings of this study of postmenopausal women, the following implication is offered. Women do not need information about following a low fat diet, but do need to be educated on the risks and benefits of HRT. This education should be reinforced over a 5-10 year period. In this study both the surgical group and the natural group ate a diet of 30% or less fat and were maintaining this type of diet for one or more years. The education, therefore, needed to be focused on HRT since the majority of women who experienced natural menopause (61%) were not on HRT versus the 72% of women in the surgical group who were on HRT.

Recommendations for Further Study

The following recommendations for further study are made to promote the health of postmenopausal women.

1. This study should be replicated in a multiethnic group who obtain their care from a public clinic because most women in postmenopausal studies are Caucasian, healthy, highly educated, and on HRT.
2. This study should be replicated in the same population after instruments are developed to measure stage of change and self-efficacy related to knowledge of menopause and HRT.

3. The subjects of this study were acquired from one internist's practice. To ensure generalizability of the findings, more than one internist's or obstetrician/gynecologist's practice should be used to obtain subjects. Comparing the patients from the two different physicians' practices might also yield interesting results.
4. Use of the telephone format to collect data should be continued to make this process expedient and efficient. It would be easy to use if more than one contact was required.

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APPENDIX A

AGENCY APPROVALS

TEXAS WOMAN'S UNIVERSITY
COLLEGE OF NURSING
1130 M.D. ANDERSON BLVD.
HOUSTON, TEXAS 77030-2897

AGENCY PERMISSION FOR CONDUCTING STUDY*

THE Helen Gerhold Institutional Review Board

GRANTS TO Nancy Schwab

a student enrolled in a program of nursing leading to a Ph.D. in nursing at Texas Woman's University, the privilege of its facilities in order to study the following problem:

Risk of Coronary Heart Disease, Dietary Fat Modification, Stages of Change and Self-efficacy in Women with Surgical and Natural Menopause (Post Menopausal)

The conditions mutually agreed upon are as follows:

1. The agency (may) (may not) be identified in the final report.
2. The names of consultative or administrative personnel in the agency (may) (may not) be identified in the final report.
3. The agency (wants) (does not want) a conference with the student when the report is completed.
4. The agency is (willing) (unwilling) to allow the completed report to be circulated through interlibrary loan.
5. Other _____

Date: 10/4/95

Signature of Agency Personnel

Nancy Schwab
Signature of Student

Signature of Faculty Advisor

*Fill out and sign three copies to be distributed as follows: Original-Student; First copy - agency; Second copy - TWU College of Nursing.

DR:lt
1/13/92

Kelsey-Seybold Clinic

Corporate Administration
1709 Dryden
Houston, Texas 77030
(713) 797-1551

November 7, 1995

Nancy Schwab
Kelsey-Seybold - Medical Center
Medical Tower, 17th Floor
1709 Dryden
Houston, Texas 77030

Dear Ms. Schwab:

Please accept this letter as official notification that I have reviewed and approved your study titled "Risk of Coronary Heart Disease, Dietary Fat Modification, Stages or Change, and Self Efficacy in Women with Surgical and Natural Menopause," via the expedited review process. This study has been approved for one year.

At the end of one year or at termination of the study we will send you a Human Subjects Annual Review or Final Report form to fill out. This is necessary to comply with our regulations to review all studies at least annually and at the conclusion of the study. Failure to complete this form will result in **automatic** suspension of this approval.

If you have any questions please call the Human Subjects Committee Coordinator Cynthia Sustaita, at 791-6983. Completed forms or inquiries should be addressed to her at, St. Luke's Medical Tower, 17th Floor.

Sincerely,

F. J. Garcia-Torres, MD

Francisco Garcia-Torres, M.D.
Chairman, Human Subjects Committee

APPENDIX B

LETTER AND DESCRIPTION OF THE STUDY

Kelsey-Seybold Clinic

Medical Center Clinic
Medical Towers
1709 Dryden
Houston, Texas 77030
(713) 797-1551

Date

Patient Name
Street Address
City, State, Zip

Kelsey #

Dear XXXXXXXXX:

I am taking part in a research study to determine how menopause affects patients' health and well-being. Patient participation consists of answering questions over the telephone according to a written survey. You would qualify for the study because you are between the ages of 45-74 years, have no current cardiovascular disease, and are able to read, write, and understand spoken English.

I have enclosed a description of the study, an Informed Consent form, and a return envelope. If you are willing to participate in the survey, please read and sign the Informed Consent and mail it back in the postage paid envelope. You will then be called by Nancy Schwab, RN, CS, MSN for a 20-minute telephone interview.

Please note that you may change your mind and withdraw from the study at any time. You may chose not to answer certain questions on the interview.

You should not feel any obligation to participate in the study. Be assured, your choice about whether to participate in the survey will not affect your care as my patient in any way.

Sincerely,

Marilyn Rice, MD
Kelsey Seybold Clinic

DESCRIPTION OF THE STUDY

You are being asked to participate in a voluntary study of factors that may influence how menopause affects patients' health and well-being. This study will be conducted by Nancy Schwab, R.N., M.S.N., and Marilyn Rice, M.D., your primary care physician. I am a nurse practitioner at Kelsey Seybold Clinic and a doctoral nursing student at Texas Woman's University-Houston Center.

You are eligible to participate in my study. If you agree to participate, you will be asked to answer a series of questions on the telephone. Completing these questions will take approximately 20-30 minutes. In addition, your cholesterol levels, and other demographic data will be taken from your most recent physical examination and recorded.

You are free to refuse to participate in the study or to withdraw at any time during the study without any penalty or loss of benefits. The questionnaires with your answers will not have your name on them. The information collected is confidential. It will only be seen by me and will be destroyed at the end of the study.

You may feel some anxiety, fatigue, or have questions while participating in the phone survey. To accommodate for these feelings or needs, the questions will be answered at your pace. If you have any questions about your health, the study, or concerns as you participate in the study, I will be happy to answer them.

I am not conducting any similar study at this time in which you could participate. If you are willing to participate, please sign the enclosed consent form and return it to me in the enclosed pre-stamped envelope. I will then contact you by telephone and administer the survey. If you have any questions about the study, please feel free to contact me at 713/791-7895.

APPENDIX C

CONSENT FORM FOR SUBJECTS

Kelsey-Seybold Clinic

Medical Center Clinic
Medical Towers
1709 Dryden
Houston, Texas 77030
(713) 797-1551

Code _____

CONSENT FORM FOR SUBJECTS

Kelsey-Seybold Clinic is participating in a study to try to find out how menopause affects patients' health and well-being. This study will be a phone survey. If you agree, Nancy Schwab will ask you to answer questions in four survey forms designed to learn how your menopause affects your health promotion practices. In addition, information from your medical record will be obtained to supplement the fifth form. The information will include your most recent blood pressure and cholesterol levels.

YOUR MEDICAL CARE WILL NOT BE CHANGED IN ANY WAY DUE TO THIS STUDY.

The surveys should take between 20-30 minutes to complete.

There is no obligation to participate in this study. If you do decide to participate, please answer each question the best you can. Remember, there are no right or wrong answers. You are free to refuse to answer any particular question and you are free to withdraw your consent and stop at any time without any effect on your medical care or other services.

There is no direct benefit to you from participation in this study. However, your participation will help increase the knowledge about menopause and its impact on women's lives and health promotion practices.

The risks to the study include fatigue and possible loss of confidentiality. If you feel tired, fatigued, or anxious while answering the questions, appropriate measures will be taken to make you comfortable.

Your confidentiality as a study participant will be protected. The information you give us will not be disclosed to anyone in any way that would reveal your identity. All of the study staff with access to medical information and records are bound by confidentiality from disclosing any personal information about you or your participation in this study to unauthorized persons. These records will be kept confidential to the extent permitted by law. It is possible that quality monitors for the clinic may review some of your records. They may need to verify that it was appropriate for us to ask you to participate in the study.

There is no additional cost to you for participating in this study.

CONSENT FORM FOR SUBJECTS page 2

If you have any questions concerning your rights as a study participant, at any time, please speak with:

Your personal physician, Marilyn Rice, M.D., at Kelsey-Seybold Clinic, or

The Kelsey-Seybold Clinic Human Subjects Committee staff, Cynthia Sustaita at 713/791-6989, or

The Office of Research and Grants Administration at Texas Woman's University at 817/696-3375.

If you have any questions or concerns about specific information about the study, please speak with Nancy Schwab at 713/791-7895.

I HAVE READ THE INFORMATION ABOVE, MY QUESTIONS HAVE BEEN ANSWERED, AND I AGREE TO PARTICIPATE IN THIS STUDY.

Note: You may withdraw from the study by verbal request even after signing this form.

_____ _____

Patient Signature Date

_____

Patient Name (Please Print)

_____ _____

Witness' Signature Date

Kelsey Seybold # _____

Phone Number _____

APPENDIX D

DEMOGRAPHIC DATA FORM

Code _____

DEMOGRAPHIC DATA FORM

1. Date of Birth _____

2. Marital Status: Married _____ Never Married _____
   Divorce/Separated _____ Widowed_____

3. Ethnicity: White _____ Black _____
   Hispanic _____ Asian _____
   Other _____

4. What is the highest grade or year of school you completed?
   Grades 1-8 (elementary) _____
   Grades 9-12 (some high school) _____
   Grade 12 or GED (high school graduate) _____
   Junior College _____
   College 1-3 years (some college) _____
   College 4 years (college graduate) _____
   Graduate or professional school _____

5. Hormonal factors
   a. When was your last menses (period)?
      D/M/Y _____
   b. Have you been without a period for one year?
      Yes _____ No _____
   c. How many menstrual periods have you had in the last year? _____
   d. Are you experiencing any symptoms of menopause (hot flashes, night sweats)?
      Yes _____ No _____
   e. How have you experienced menopause?
      Surgical _____ At what age _____
      Natural _____ At what age _____
   f. Do you have: One ovary ____ Both ovaries ____ No ovaries____

g. Are you on cholesterol lowering medication?
Yes _____ No _____
If so, what medication(s) are you taking?
_____
_____

h. Are you on hormone replacement therapy (HRT)?
Yes _____ No _____
If so, how long have you been on HRT?
Y/M _____
If so, are you on estrogen alone? Yes ____ No_____
Estrogen/progestin? Yes____ No_____
If so, what medications are you taking:
_____
_____

DATA FROM MEDICAL RECORD AND DEMOGRAPHIC DATA

Cholesterol levels

Month/Year of bloodwork_____

Total cholesterol _____ TG _____

LDL _____

HDL _____

CV Risk _____

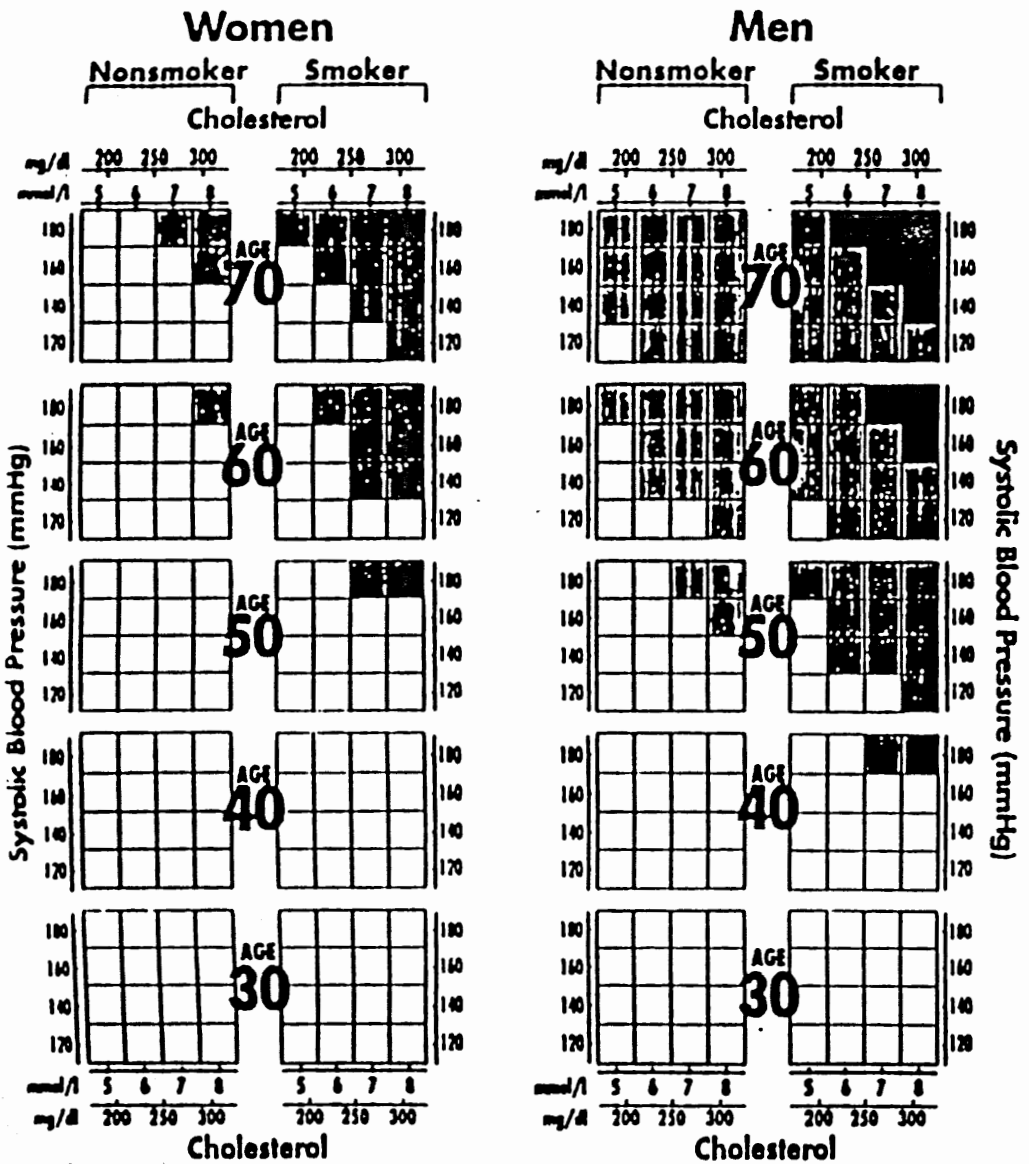
Date/Time of blood pressure _____

Blood Pressure reading_____

APPENDIX E

RISK OF CORONARY HEART DISEASE TABLE (ANDERSON, WILSON, ODELL, & KANNEL, 1991)

RISK OF CORONARY HEART DISEASE



HOW TO USE THE RISK TABLES

1. To determine a person's absolute 10-year risk of a coronary event (heart attack), identify the table relating to the person's sex, smoking status, and age.
2. Within the table, find the cell nearest to the person's systolic blood pressure (mmHg) and cholesterol
3. Compare cell colour with key and read the risk level.
4. The effect of lifetime exposure to risk factors can be assessed by following the table upwards with increasing age.
5. **Notice — For patients with coronary heart disease, the level of risk should be increased by at least one category.**
   People with family history of coronary event at an early age, diabetes, or a family history of hyperlipidaemia are also at increased risk.

RISK LEVEL
Percent chance of coronary event in 10 years

Very high	>40%
High	20–40%
Moderate	10–20%
Mild	5–10%
Low	<5%

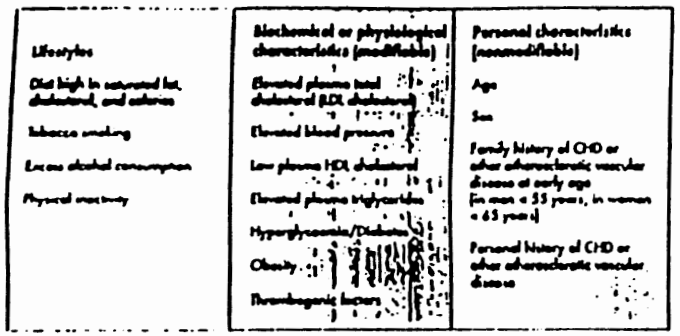
Based on a risk function derived from the Framingham Study, Anderson KM et al. An updated coronary risk profile. A statement for health professionals. Circulation 83:356-362, 1991

The table assumes the HDL cholesterol to be 1.0 mmol/l (39 mg/dl) in men and 1.1 mmol/l (43 mg/dl) in women. People with lower levels and/or with triglycerides above 2.3 mmol/l (200 mg/dl) are at higher risk

EUROPEAN SOCIETY OF CARDIOLOGY
EUROPEAN ATHEROSCLEROSIS SOCIETY
EUROPEAN SOCIETY OF HYPERTENSION

Prevention of Coronary Heart Disease in Clinical Practice
Recommendations to be published in October 1994 in the *European Heart Journal* and *Atherosclerosis*.

Lifestyles and Characteristics Associated with Increased Risk of Future Coronary Heart Disease



Priorities of Coronary Heart Disease Prevention in Clinical Practice

1 **Patients with established CHD** or other atherosclerotic vascular disease

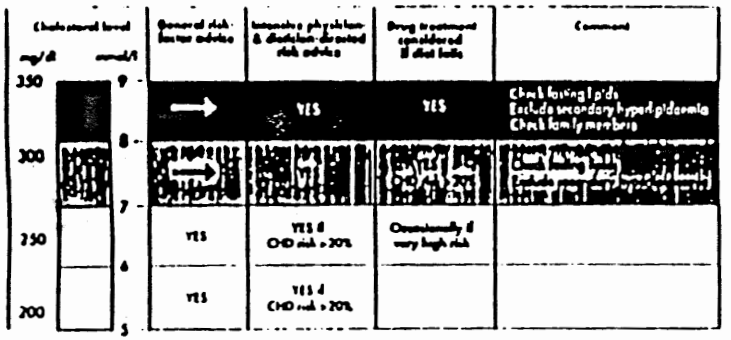
2 **Asymptomatic subjects with particularly high risk**
(subjects with severe hypercholesterolemia or other form of dyslipidaemia, diabetes, or hypertension; subjects with a cluster of several risk factors)

3 **Close relatives** of
- patients with early-onset CHD or other atherosclerotic vascular disease
- asymptomatic subjects with particularly high risk

4 **Other individuals met** in connection with ordinary clinical practice

Guide to Lipid Management

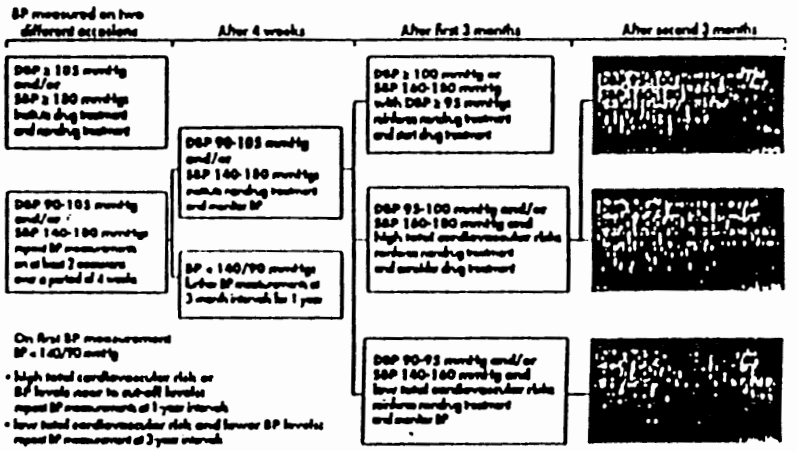
Total cardiovascular risk should be assessed first and the major components of risk identified. If 10 year CHD risk exceeds 20% or will exceed 20% if projected to age 60, more intensive advice for all risk factors will be required. Clinical vascular disease will increase the risk to more than 20% for most and to more than 40% for many.



- Diet is the cornerstone of management
- General risk factor advice implies avoidance of tobacco, weight control, less than 30 percent of dietary calories as fat (of which less than a third are from saturated fat), control of hypertension, and frequent leisure exercise
- Management decisions should not be based on a single cholesterol measurement. Laboratory variation may be 0.5 mmol/l (20 mg/dl) or more
- Raised triglycerides signal the need for fasting lipid estimations (HDL cholesterol may be low). Hypertriglyceridaemia often responds to weight and alcohol control.
- The benefits or otherwise of drug treatment in women and in the elderly are unknown.

Guide to Blood Pressure Management

Total cardiovascular risk should be assessed first and the major components of risk identified. If 10 year CHD risk exceeds 20% or will exceed 20% if projected to age 60, more intensive advice for all risk factors will be required. Clinical vascular disease will increase the risk to more than 20% for most and to more than 40% for many.



Coronary Heart Disease Risk Factor Prediction Chart

American Heart Association.

1. Find Points For Each Risk Factor

Age (If Female)				Age (If Male)				HDL-Cholesterol		Total-Cholesterol		Systolic Blood Pressure			
Age	Pts	Age	Pts.	Age	Pts.	Age	Pts.	HDL-C	Pts.	Total-C	Pts.	SBP	Pts.	Other	Pts.
30	-12	47–48	5	30	-2	57–59	13	25–26	7	139–151	-3	98–104	-2	Cigarettes	4
31	-11	49–50	6	31	-1	60–61	14	27–29	6	152–166	-2	105–112	-1	Diabetic-male	3
32	-9	51–52	7	32–33	0	62–64	15	30–32	5	167–182	-1	113–120	0	Diabetic-female	6
33	-8	53–55	8	34	1	65–67	16	33–35	4	183–199	0	121–129	1	ECG-LVH	9
34	-8	56–60	9	35–36	2	68–70	17	36–38	3	200–219	1	130–139	2		
35	-5	61–67	10	37–38	3	71–73	18	39–42	2	220–239	2	140–149	3	0 pts for each NO	
36	-4	68–74	11	39	4	74	19	43–46	1	240–262	3	150–160	4		
37	-3			40–41	5			47–50	0	263–288	4	161–172	5		
38	-2			42–43	6			51–55	-1	289–315	5	173–185	6		
39	-1			44–45	7			56–60	-2	316–330	6				
40	0			46–47	8			61–66	-3						
41	1			48–49	9			67–73	-4						
42–43	2			50–51	10			74–80	-5						
44	3			52–54	11			81–87	-6						
45–46	4			55–56	12			88–96	-7						

2. Sum Points For All Risk Factors

____ (Age) + ____ (HDL-C) + ____ (Total-C) + ____ (SBP) + ____ (Smoker) + ____ (Diabetes) + ____ (ECG-LVH) = ____ (Point Total)

NOTE: *Minus Points Subtract From Total.*

3. Look Up Risk Corresponding To Point Total

Probability			Probability			Probability			Probability		
Pts	5 Yr.	10 Yr.	Pts.	5 Yr.	10 Yr.	Pts.	5 Yr.	10 Yr.	Pts.	5 Yr.	10 Yr.
≤ 1	< 1%	< 2%	10	2%	6%	19	6%	16%	28	19%	33%
2	1%	2%	11	3%	6%	20	8%	16%	29	20%	36%
3	1%	2%	12	3%	7%	21	9%	19%	30	22%	38%
4	1%	2%	13	3%	8%	22	11%	21%	31	24%	40%
5	1%	3%	14	4%	9%	23	12%	23%	32	25%	42%
6	1%	3%	15	5%	10%	24	13%	25%			
7	1%	4%	16	5%	12%	25	14%	27%			
8	2%	4%	17	6%	13%	26	15%	29%			
9	2%	5%	18	7%	14%	27	17%	31%			

4. Compare To Average 10 Year Risk

Probability		
Age	Women	Men
30–34	< 1%	3%
35–39	< 1%	5%
40–44	2%	6%
45–49	5%	10%
50–54	8%	14%
55–59	12%	16%
60–64	13%	21%
65–69	9%	30%
70–74	12%	24%

These charts were prepared with the help of William B. Kannel, M.D., Professor of Medicine and Public Health and Ralph D'Agostino, Ph.D., Head, Department of Mathematics, both at Boston University; Keaven Anderson, Ph.D., Statistician, NHLBI, Framingham Study; Daniel McGee, Ph.D., Associate Professor, University of Arizona.

Framingham Heart Study

© 1990, American Heart Association

APPENDIX F

FOOD HABITS QUESTIONNAIRE AND SCORE SHEET

(KRISTAL, SHATTUCK, & HENRY, 1990)

FOOD HABITS QUESTIONNAIRE

Directions: These questions are about the way you ate over the past 3 months. Please circle your response and check either yes or no" in the blank space.

MEAT, FISH AND MAIN DISHES

IN THE **PAST 3 MONTHS:**

			Usually or Always	Often	Sometimes	Rarely or Never
1. Did you eat fish?						
NO	YES					
___	___ →	How often did you:				
		a.have it broiled	1	2	3	4
↓	*answer*	or baked?[1†]				
	both	b.Have it fried?[1*†]	1	2	3	4
2. Did you eat chicken?						
NO	YES					
___	___ →	How often did you:				
		a.have it broiled				
↓	*answer*	or baked?[1*†]	1	2	3	4
	all	b.have it fried? [1*†]	1	2	3	4
	three	c.take off the				
		skin?[1]	1	2	3	4
3. Did you eat spaghetti or noodles?						
NO	YES					
___	___ →	How often did you use				
		a. meatless tomato				
↓		sauce?[3]	1	2	3	4
4. Did you eat red meat?						
NO	YES					
___	___ →	How often did you:				
		a.eat only small				
↓	*answer*	portions?[1]	1	2	3	4
	both	b.trim all the				
		visible fat?[1]	1	2	3	4

		Usually or Always	Often	Sometimes	Rarely or Never
5.	How often did you have a vegetarian dinner?[3,6]	1	2	3	4
6.	Did you eat fish, chicken or red meat? NO YES ___ ___ → How often did you eat fish or chicken ↓ Instead of red meat?[3]	1	2	3	4

MILK AND CHEESE

IN THE **PAST 3 MONTHS...**

7.	Did you drink milk or use milk on cereal? NO YES ___ ___ How often was it very low fat (1%) or non-fat skim milk?[4] ↓	1	2	3	4
8.	Did you eat cheese? (include sandwiches or in cooking) NO YES ___ ___→ How often was it specially-made, low fat (diet) cheese?[4] ↓	1	2	3	4
9.	Did you eat frozen desserts (ice cream, sherbet, etc.)? NO YES ___ ___→ How often did you choose frozen yogurt, sherbet,or non-fat ice cream instead of regular ice cream?[4] ↓	1	2	3	4

FRUITS, VEGETABLES AND SALADS

IN THE **PAST 3 MONTHS...**

			Usually or Always	Often	Sometimes	Rarely or Never
10.	Did you eat cooked vegetables? NO YES ___ ___ → ↓	How often did you put butter or margarine on vegetables?[2*]	1	2	3	4
11.	Did you eat potatoes? NO YES ___ ___ → ↓ *answer both*	How often were they: a.fried (french fries, hash browns?[2*†]	1	2	3	4
		b.boiled or baked?[2†]	1	2	3	4
12.	Did you eat boiled or baked potatoes? NO YES ___ ___ → ↓ *answer both*	How often did you: a.eat potatoes without butter, margarine, or sour cream?[2]	1	2	3	4
		b.eat the skin?[7]	1	2	3	4
13.	Did you eat green salads? NO YES ___ ___ → ↓ *answer both*	How often did you: a.use no dressing?[2]	1	2	3	4
		b.use low-calorie, diet dressing?[4]	1	2	3	4
14.	How often did you eat beans, peas, or lentils as a vegetable or main course?[6]		1	2	3	4

		Usually or Always	Often	Sometimes	Rarely or Never
15.	How often did you eat at least two vegetables (not green salad) at dinner?[7]	1	2	3	4
16.	How often did you eat a vegetable (not green salad) at lunch?[7]	1	2	3	4
17.	Did you eat dessert? NO YES ___ ___→ How often did you eat only fruit for ↓ dessert?[5,7]	1	2	3	4
18.	Did you eat snacks? NO YES ___ ___→ How often did you: a.eat raw vegetables ↓ *answer* for snacks?[5,7]	1	2	3	4
	both b.eat fresh fruit for snacks?[7]	1	2	3	4

BREADS, ROLLS, MUFFINS, AND CEREALS

IN THE **PAST 3 MONTHS...**

19.	Did you eat bread, rolls, or muffins? NO YES ___ ___→ How often did you: a.eat breads, rolls,or ↓ *answer* muffins without butter				
	both or margarine?[2]	1	2	3	4
	b.eat breads, rolls, or muffins made from whole grains (whole wheat, rye, pumper-nickel)?[8]	1	2	3	4

			Usually or Always	Often	Sometimes	Rarely or Never
20.	Did you eat breakfast? NO YES					
___	___→	How often did you: a.eat cereal for				
↓	*answer*	breakfast?[8]	1	2	3	4
	both	b.eat fruit at breakfast?[7]	1	2	3	4

FOOD PREPARATION

IN THE **PAST 3 MONTHS...**

21.	Did you cook or prepare meals? NO YES					
___	___→	How often did you: a.add bran to cas-				
↓	*answer*	seroles or cereals?[9]	1	2	3	4
	both	b.use Pam® or other non-stick spray instead of oil, margarine,or butter?[4]	1	2	3	4
22.	Did you use mayonnaise? NO YES					
___	___→	How often did you use diet, low-calorie				
↓		mayonnaise instead of regular mayon- naise? [4]	1	2	3	4

THANK YOU.

CODE_____

Fat Factor Score Sheet

Question	Response	
Fat Factor 1 (modify meat)		
1a†	____	
2a†	____	
2c	____	
4a	____	
4b	____	
Total	____	÷ number answered = Factor 1 score _____
Fat Factor 2 (avoid fat as a seasoning)		
10*	____	
11b†	____	
12a	____	
13a	____	
19a	____	
Total	____	÷ number answered = Factor 2 score _____
Fat Factor 3 (replace, general foods)		
3	____	
5	____	
6	____	
Total	____	÷ number answered = Factor 3 score _____
Fat Factor 4 (substitute)		
7	____	
8	____	
9	____	
13b	____	
21b	____	
22	____	
Total	____	÷ number answered - Factor 4 score _____
Fat Factor 5 (replace, fruit/vegetable)		
17	____	
18a	____	
Total	____	÷ number answered = Factor 5 score _____

Summary score $\frac{\Sigma \text{ Fat Factors}}{5}$ = _____

* Reverse scoring: 1=4; 2=3; 3=2; 4=1

† Score only one item in each set for questions 1, 2, and 11. The score sheet lists 1a, 2a, and 11b as choices to avoid the need for reverse scoring. The lower the score, the lower the fat intake. A score of 2.5 or less predicts fat intake at 30% or less.

APPENDIX G

STAGES OF CHANGE TO DIETARY FAT REDUCTION (SCDFR) QUESTIONNAIRE AND ALGORITHM (CURRY, KRISTAL, & BOWEN, 1992)

Code _____

STAGES OF CHANGE TO DIETARY FAT REDUCTION (SCDFR) QUESTIONNAIRE

Directions: Please complete this set of questions by circling yes or no appropriately.

1. Have you ever changed your eating habits to decease the amount of fat in your diet?

Yes 1
No 2 (Skip to #2)

1a. If YES, are you currently limiting the amount of fat in your diet?

Yes 1
No 2 (Skip to #2)

1b. If YES, how long have you been limiting the amount of fat in your diet?

Less than 30 days 1
Less than 1-6 months 2
Less than 7-12 months 3
Over 1 Year 4

2. In the past month, have you thought about changes you could make to decrease the amount of fat in your diet?

Yes 1
No 2

2a. If YES, how confident are you that you will make some of these changes during the next month?

Very confident 1
Somewhat confident 2
Mildly confident 3
Not at all confident 4

Curry A., Kristal, A., & Bowen, D. (1992)

Code_____

STAGES OF CHANGE TO DIETARY FAT REDUCTION (SCDFR) QUESTIONNAIRE

Staging Algorithm

Stage	Questions	Answers
Precontemplation	1 or 1A	No
	2	No
Contemplation	1 or 1A	No
	2	Yes
	2A	Mildly or not at all
		confident
Decision	1 or 1A	No
	2	Yes
	2A	Somewhat or very confident
Action	1 or 1A	Yes
	1B	6 months or less
Maintenance	1 or 1A	Yes
	1B	7 months or more

Curry A., Kristal, A., & Bowen, D. (1992)

APPENDIX H

SELF-EFFICACY FOR HEALTH-RELATED DIET BEHAVIORS (SEHRDB) QUESTIONNAIRE (SALLIS, PINSKI, PATTERSON, & NADER, 1988)

Code _____

SELF-EFFICACY FOR HEALTH RELATED DIET BEHAVIORS (SEHRDB) QUESTIONNAIRE

Using the following scale, please circle the number which comes closest to how sure you feel about your ability to change your eating behaviors. Each item is followed by five choices.

(0) Completely Unsure	(1) Moderately Unsure	(2) Moderately Sure	(3) More Sure	(4) Completely Sure

	Statement	Circle Choice				
1.	Stick to low-fat foods when you feel depressed, bored, or tense.	0	1	2	3	4
2.	Stick to low-fat foods when there is high-fat food available at a party.	0	1	2	3	4
3.	Stick to low-fat foods when dining with friends or coworkers.	0	1	2	3	4
4.	Stick to low-fat foods when the only snack close by is available from a vending machine.	0	1	2	3	4
5.	Stick to low-fat foods when you are alone and there is no one to watch you.	0	1	2	3	4
6.	Stick to low-fat foods when you feel too lazy to prepare something healthy.	0	1	2	3	4
7.	Stick to low-fat foods when you have guests staying in your home.	0	1	2	3	4
8.	Stick to low-fat foods when someone offers you a high-fat food at a party.	0	1	2	3	4
9.	Stick to low-fat foods when someone eats a high-fat food right in front of you.	0	1	2	3	4
10.	Stick to low-fat foods when you must eat in a hurry.	0	1	2	3	4
11.	Eat fruits instead of cookies, candy, cake, and ice cream for desserts.	0	1	2	3	4
12.	Eat fruits instead of cookies, candy, cake, and ice cream for snacks.	0	1	2	3	4
13.	Stick to low-fat foods while traveling.	0	1	2	3	4

(0) Completely Unsure	(1) Moderately Unsure	(2) Moderately Sure	(3) More Sure	(4) Completely Sure

Statement					Circle Choice				
14.	Stick to low-fat foods while you are drinking alcohol.				0	1	2	3	4
15.	Avoid junk food that other family members have brought into your home.				0	1	2	3	4
16.	Eat carrots, celery, and raw vegetables instead of dips, crackers, and potato chips.				0	1	2	3	4
17.	Avoid eating fast food for lunch.				0	1	2	3	4
18.	Eat meatless (vegetarian) entrees for dinner.				0	1	2	3	4
19.	Substitute low- or non-fat milk for whole milk at breakfast.				0	1	2	3	4
20.	Cut down on gravies and cream sauces.				0	1	2	3	4
21.	Eat poultry and fish instead of red meat for dinner.				0	1	2	3	4
22.	Avoid ordering red meat at a restaurant.				0	1	2	3	4
23.	Eat at restaurants that offer a greater variety of low-fat dishes.				0	1	2	3	4
24.	Eat cooked cereals.				0	1	2	3	4
25.	Substitute foods like beans, peas, lentils, potatoes, corn, rice, bread for the meat in your diet.				0	1	2	3	4
26.	Eat poultry without skin.				0	1	2	3	4
27.	Bake, broil, barbecue, or steam food instead of frying.				0	1	2	3	4
28.	Read labels for fat content.				0	1	2	3	4
29.	Go to the grocery store on a full stomach.				0	1	2	3	4
30.	Serve low-fat foods to dinner or party guests in your home.				0	1	2	3	4
31.	Post a weekly menu plan on your kitchen bulletin board.				0	1	2	3	4
32.	Keep a food diary for one week if you begin to slip in your food program.				0	1	2	3	4
33.	Say encouraging things to yourself if you begin to slip in your food program.				0	1	2	3	4
34.	Keep problematic high-fat foods out of sight if possible.				0	1	2	3	4

Total Score _____

APPENDIX I

PERMISSIONS TO USE AND REPRODUCE INSTRUMENTS

Consent To Use Instrument

Nancy Schwab R.N.C, M.S.N. a Ph.D student at Texas Woman's University in Houston, Texas has permission to use the Self-efficacy for Health-related Diet Behaviors in her doctoral dissertation titled Differences in Self-efficacy among Adults with Borderline Hypercholesterolemia.

Nancy will share the raw data and reliability data she acquires from her study with the original authors.

James Sallis
Signature

4/11/91
Date

Philip R. Nader, MD

4-10-91

Ms. Schwab,

You are welcome to use all or part of this instrument. Any modification you make should be described in reports. Please share your final research report.

I have enclosed an abbreviated version of the scale for your inspection.

Consent To Use Instruments

Nancy Schwab R.N.C., M.S.N. a PH.D student at Texas Woman's University in Houston, Texas has permission to use the Rapid Assessment of Dietary Intake of Fat, Fiber, and Saturated Fat and Staging Question for Dietary Fat Reduction in her doctoral dissertation.

Nancy will share the raw data and relability data she acquires from her study with the original authors.

Signature

6-19-91
Date

1/6/95 phone consent given to use the 2 instruments:

1) Food Habits Questionnaire (1990)
(Kristal, A., Shattuck, A + Henry, H)

2) Stages of change in adopting Healthy diets: Cleary, K., Patterson, R., Kristal, A., DiClemente, C., Hermenegen, J. Lowan, L., McLerran, D. (1994)

N Schwab

APPENDIX J

SUMMARY TABLES OF DATA COLLECTION RESULTS BY INSTRUMENT, TYPE OF MENOPAUSE, AND HORMONE STATUS

Table A

Frequency Distribution and Percentages of Total Points with 5-Year and 10-Year Probabilities for Risk of CHD Table (RCHDT) for Surgical and Natural Postmenopausal Women (N = 77)

Scores	Surgical		Natural		Total	
	n	%	n	%	n	%
2	2	4.3	0	0.0	2	2.6
3	1	2.2	0	0.0	1	1.3
4	0	0.0	1	3.2	1	1.3
5	4	8.7	2	6.5	6	7.8
6	3	6.5	0	0.0	3	3.8
7	3	6.5	1	3.2	4	5.2
8	4	8.7	5	16.1	9	11.7
9	10	21.8	2	6.5	12	15.6
10	2	4.3	3	9.7	5	6.5
11	2	4.3	0	0.0	2	2.6
12	5	11.0	5	16.1	10	13.0
13	3	6.5	3	9.7	6	7.8
14	1	2.2	1	3.2	2	2.6
15	3	6.5	1	3.2	4	5.2
16	0	0.0	1	3.2	1	1.3
17	2	4.3	0	0.0	2	2.6
18	0	0.0	1	3.2	1	1.3
20	0	0.0	2	6.5	2	2.6
21	0	0.0	2	6.5	2	2.6
22	1	2.2	0	0.0	1	1.3
23	0	0.0	1	3.2	1	1.3
Total	46	100.0	31	100.0	77	100.0
Mean	9.6		12.1		10.6	
SD	4.1		5.1		4.7	

Note: The total number of questions was 7.

Table B

Frequency Distribution and Percentages of 5-Year and 10-Year Risk for CHD Table (RCHDT) for Surgical and Natural Postmenopausal Women (N = 77)

Percent of Risk	Surgical		Natural		Total	
	n	%	n	%	n	%
5-Year Risk						
1%	13	28.3	4	12.9	17	22.1
2%	16	34.8	10	32.3	26	33.8
3%	10	21.7	8	25.8	18	23.4
4%	4	8.7	2	6.5	6	7.8
5%	0	0.0	1	3.2	1	1.3
6%	2	4.3	0	0.0	2	2.6
7%	0	0.0	1	3.2	1	1.3
8%	0	0.0	2	6.5	2	2.6
9%	0	0.0	2	6.5	2	2.6
11%	1	2.2	0	0.0	1	1.3
12%	0	0.0	1	3.2	1	1.3
Total	46	100.0	31	100.0	77	100.0
Mean ± SD	2.5% ± .019		3.7% ± .028		3% ± .02	
10-Year Risk						
2%	3	6.5	1	3.2	4	5.2
3%	7	15.2	2	6.5	9	11.7
4%	7	15.2	6	19.4	13	16.9
5%	10	21.7	2	6.5	12	15.6
6%	4	8.7	3	9.7	7	9.1
7%	5	10.9	5	16.1	10	13.0
8%	3	6.5	3	9.7	6	7.8
9%	1	2.2	1	3.2	2	2.6
10%	3	6.5	1	3.2	4	5.2
12%	0	0.0	1	3.2	1	1.3
13%	2	4.3	0	0.0	2	2.6
14%	0	0.0	1	3.2	1	1.3
18%	0	0.0	2	6.5	2	2.6
19%	0	0.0	2	6.5	2	2.6
21%	1	2.2	0	0.0	1	1.3
23%	0	0.0	1	3.2	1	1.3
Total	46	100.0	31	100.0	77	100.0
Mean ± SD	6% ± .035		8.4% ± .056		6.9% ± .046	

Table C

Frequency Distribution and Percentages of Total Points with 5-Year and 10-Year Probabilities for Risk of CHD Table (RCHDT) for HRT and No HRT in Postmenopausal Women (N = 77)

Scores	HRT		No HRT		Total	
	n	%	n	%	n	%
2	2	4.4	0	0.0	2	2.6
3	1	2.2	0	0.0	1	1.3
4	1	2.2	0	0.0	1	1.3
5	5	11.1	1	3.1	6	7.8
6	2	4.4	1	3.1	3	3.8
7	1	2.2	3	9.3	4	5.2
8	8	17.8	1	3.1	9	11.7
9	10	22.2	2	6.3	12	15.6
10	3	6.7	2	6.3	5	6.5
11	1	2.2	1	3.1	2	2.6
12	6	13.3	4	12.5	10	13.0
13	2	4.4	4	12.5	6	7.8
14	0	0.0	2	6.3	2	2.6
15	2	4.4	2	6.3	4	5.2
16	0	0.0	1	3.1	1	1.3
17	1	2.2	1	3.1	2	2.6
18	0	0.0	1	3.1	1	1.3
20	0	0.0	2	6.3	2	2.6
21	0	0.0	2	6.3	2	2.6
22	0	0.0	1	3.1	1	1.3
23	0	0.0	1	3.2	1	1.3
Total	45	100.0	32	100.0	77	100.0
Mean 12.5	8.7		12.8		10.6	
SD	3.4		5.0		4.7	

Note: The total number of questions was 7.

Table D

Frequency Distribution and Percentages of 5-Year and 10-Year Risk for CHD Table (RCHDT) for HRT and No HRT in Postmenopausal Women (N = 77)

Percent of Risk	HRT n	HRT %	No HRT n	No HRT %	Total n	Total %
5-Year Risk						
1%	12	26.7	5	15.6	17	22.1
2%	21	46.7	5	15.6	26	33.8
3%	9	20.0	9	28.1	18	23.4
4%	2	4.4	4	12.6	6	7.8
5%	0	0.0	1	3.1	1	1.3
6%	1	2.2	1	3.1	2	2.6
7%	0	0.0	1	3.1	1	1.3
8%	0	0.0	2	6.3	2	2.6
9%	0	0.0	2	6.3	2	2.6
11%	0	0.0	1	3.1	1	1.3
12%	0	0.0	1	3.1	1	1.3
Total	45	100.0	32	100.0	77	100.0
Mean ± SD	2% ± .01		4% ± .03		3% ± .02	
10-Year Risk						
2%	4	8.9	0	0.0	4	5.2
3%	7	15.6	2	6.3	9	11.7
4%	9	20.0	4	12.5	13	16.9
5%	10	22.2	2	6.3	12	15.6
6%	4	8.9	3	9.3	7	9.1
7%	6	13.3	4	12.5	10	13.0
8%	2	4.4	4	12.5	6	7.8
9%	0	0.0	2	6.3	2	2.6
10%	2	4.4	2	6.3	4	5.2
12%	0	0.0	1	3.1	1	1.3
13%	1	2.2	1	3.1	2	2.6
14%	0	0.0	1	3.1	1	1.3
18%	0	0.0	2	6.3	2	2.6
19%	0	0.0	2	6.3	2	2.6
21%	0	0.0	1	3.1	1	1.3
23%	0	0.0	1	3.1	1	1.3
Total	45	100.0	32	100.0	77	100.0
Mean ± SD	5% ± .02		9.5% ± .06		7% ± .05	

Table E

Frequency Distribution and Percentages of Food Habits Questionnaire (FHQ) Scores for Surgical and Natural Postmenopausal Women (N = 77)

Scores	Surgical		Natural		Total	
	n	%	n	%	n	%
1.00-1.49	0	0.0	1	3.2	1	1.3
1.50-1.99	6	13.0	2	6.5	8	10.4
2.00-2.49	8	17.4	12	38.7	20	26.0
2.50-2.99	21	45.7	13	41.9	34	44.1
3.00-3.49	11	23.9	3	9.7	14	18.2
Total	46	100.0	31	100.0	77	100.0
Mean ± SD	2.6 ± .45		2.5 ± .48		2.55 ±.46	

Note: The total number of questions is 22.

Table F

Frequency Distribution and Percentages of Food Habits Questionnaire (FHQ) Scores for HRT and No HRT Postmenopausal Women (N = 77)

Scores	HRT		No HRT		Total	
	n	%	n	%	n	%
1.00-1.49	1	2.2	0	0.0	1	1.3
1.50-1.99	5	11.1	3	9.4	8	10.4
2.00-2.49	10	22.2	10	31.2	20	26.0
2.50-2.99	21	46.7	13	40.6	34	44.1
3.00-3.49	8	17.8	6	18.8	14	18.2
Total	45	100.0	32	100.0	77	100.0
Mean ± SD	2.55 ± .48		2.54 ± .45		2.55 ±.46	

Note: The total number of questions is 22.

Table G

Frequency Distribution and Percentages of Stage of Change to Dietary Fat Reduction (SCDFR) Scores for Surgical and Natural Postmenopausal Women (N = 77)

Stages	Surgical		Natural		Total	
	n	%	n	%	n	%
Precontemplation (5)	3	6.5	3	9.7	6	7.8
Contemplation (4)	1	2.2	1	3.2	2	2.6
Decision Making (3)	0	0.0	1	3.2	1	1.3
Action (2)	4	8.7	5	16.1	9	11.7
Maintenance (1)	38	82.6	21	67.8	59	76.6
Total	46	100.0	31	100.0	77	100.0
Mean		1.41		1.71		1.53
SD		1.09		1.30		1.18

Note: The total number of questions was 5.

Table H

Frequency Distribution and Percentages of Stage of Change to Dietary Fat Reduction (SCDFR) Scores for HRT and No HRT Postmenopausal Women (N = 77)

Stages	HRT		No HRT		Total	
	n	%	n	%	n	%
Precontemplation (5)	4	8.9	2	6.3	6	7.8
Contemplation (4)	1	2.2	1	3.1	2	2.6
Decision Making (3)	0	0.0	1	3.1	1	1.3
Action (2)	3	6.7	6	18.8	9	11.7
Maintenance (1)	37	82.2	22	68.8	59	76.6
Total	45	100.0	32	100.0	77	100.0
Mean		1.50		1.59		1.53
SD		1.21		1.13		1.18

Note: The total number of questions was 5.

Table I

Frequency Distribution and Percentages of the Self-Efficacy for Health-Related Diet Behaviors (SEHRDB) for Surgical and Natural Postmenopausal Women (N = 77)

Scores	Surgical		Natural		Total	
	n	%	n	%	n	%
0.50-0.99	0	0.0	1	3.2	1	1.3
1.00-1.49	1	2.2	1	3.2	2	2.6
1.50-1.99	5	10.9	1	3.2	6	7.8
2.00-2.49	19	41.3	8	25.8	27	35.1
2.50-2.99	8	17.4	11	35.5	19	24.6
3.00-3.49	9	19.5	6	19.4	15	19.5
3.50-3.99	4	8.7	3	9.7	7	9.1
Total	46	100.0	31	100.0	77	100.0
Mean ± SD	2.6 ± .61		2.6 ± .65		2.6 ± .63	

Note: The total number of questions was 34.

Table J

Frequency Distribution and Percentages of the Self-Efficacy for Health-Related Diet Behaviors (SEHRDB) for HRT and No HRT Postmenopausal Women (N = 77)

Scores	HRT		No HRT		Total	
	n	%	n	%	n	%
0.50-0.99	0	0.0	1	3.1	1	1.3
1.00-1.49	1	2.2	1	3.1	2	2.6
1.50-1.99	6	13.3	0	0.0	6	7.8
2.00-2.49	14	31.1	13	40.6	27	35.1
2.50-2.99	10	22.2	9	28.2	19	24.6
3.00-3.49	8	17.9	7	21.9	15	19.5
3.50-3.99	6	13.4	1	3.1	7	9.1
Total	45	100.0	32	100.0	77	100.0
Mean ± SD	2.7 ± .64		2.5 ± .60		2.6 ± .63	

Note: The total number of questions was 34.