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A comparison of the effects of moderate- and high-carbohydrate diets on coronary heart disease risk factors: A pilot study

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A COMPARISON OF THE EFFECTS OF MODERATE- AND HIGH-CARBOHYDRATE DIETS ON CORONARY HEART DISEASE RISK FACTORS: A PILOT STUDY

A Thesis

by

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This pilot study compared the effects of two diets on total cholesterol to high-density lipoprotein cholesterol ratio, body mass index, and waist circumference. A diet of 15% protein, 55% or more carbohydrate, and 30% or less fat was compared to a diet of 30% protein, 40% carbohydrate, and 30% fat. Lipid levels and anthropometric measurements were obtained on 18 adult males with moderately elevated lipid levels before and after 12 weeks of dietary intervention. Analyses showed variances between and within the study groups in the areas of anthropometric measurements and lipid levels; no statistically significant differences between the groups were found.

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CHAPTER I

INTRODUCTION

Statement of the Problem and Research Hypothesis

The purpose of this pilot study was to compare the effects of two diets on total cholesterol to high-density lipoprotein cholesterol (TC/HDL-C) ratio, body mass index (BMI), and waist circumference in adults in an outpatient setting. The two diets were low-fat high-carbohydrate and low-fat moderate-carbohydrate. The hypothesis was that there is a relationship between the amount of carbohydrate intake in the diet and the degree of coronary heart disease (CHD) risk as indicated by the following risk factors: total cholesterol to high-density lipoprotein cholesterol ratio, body mass index, and waist circumference. The dependent variable was the degree of CHD risk. The independent variables were the high- and moderate-carbohydrate diets. Study participants included 18 adults from the ages of 25 to 65 who met the criteria for primary prevention with diet therapy as outlined by the 1993 National Cholesterol Education Program (NCEP) Expert Panel on Detection, Evaluation, and Treatment of High Blood Cholesterol in Adults.

Significance of the Problem

Coronary heart disease is currently responsible for approximately 24% of total deaths in the United States (Bendich & Deckelbaum, 1997). The peak incidence of clinical manifestations for men is between the ages of 50 and 60 and for women between the ages of 60 and 70 (Tierney, McPhee, & Papadakis, 1997). The annual national economic toll of CHD is currently estimated at 80 billion dollars and the overall total cardiovascular disease costs exceed 138 billion dollars per year. CHD became a major cause of death in 1920 and increased

between 1 and 2% per year to 300 deaths per 100,000 population in the mid 1960s. By 1968, mortality rates due to CHD were beginning to decline again, and decreased to 152 deaths per 100,000 population in 1997 (Bendich & Deckelbaum).

The identification of risk factors, behavior modification, and improved treatment have played a major role in the decrease of mortality related to CHD (Mahan & Escott-Stump, 1996). Certain risk factors, such as gender, age, and family history, are not amenable to change. Other factors such as smoking, alcohol intake, obesity, hypertension, diabetic control, and blood lipid levels (ie. cholesterol, triglycerides, low-density lipoprotein cholesterol [LDL-C], HDL-C) are able to be influenced by lifestyle choices. Of these risk factors that can be modified, the second report of the National Cholesterol Education Program Expert Panel on Detection, Evaluation, and Treatment of High Blood Cholesterol in Adults identified elevated LDL-C levels (a positive risk factor) as the primary target for cholesterol lowering therapy (Expert Panel on Detection, Evaluation, and Treatment of High Blood Cholesterol in Adults [Expert Panel], 1993). Behavior modification, primarily in the form of dietary therapy, is considered by the NCEP to be "the first line of treatment of high blood cholesterol, and drug therapy is reserved for patients considered to be at high risk for CHD" (p. 3015). The recommended method of primary prevention for CHD is the Step I diet, developed from the recommendations of the NCEP expert panel. The Step I diet is low in fat and and high in carbohydrate, with a goal of lowering LDL-C and maintaining a desirable weight.

The NCEP also focused on the protective aspects of elevated HDL-C levels (a negative risk factor) (Expert Panel, 1993). Various trials of lipid-lowering agents, such as the West of Scotland Coronary Prevention Study (Shepherd et al., 1995) and the Scandinavian Simvastatin Survival Study (The Scandinavian Simvastatin Survival Study Group, 1994) have demonstrated that drug-induced

increases in HDL-C levels independently predicted decreases in the risk of CHD. A study by Vega and Grundy (1996) confirmed the NCEP's findings that there is a strong association between low HDL-C levels and increased risk of CHD. The NCEP classified low HDL-C levels (< 35 mg/dl) as a major positive risk factor for CHD and encouraged clinicians to consider the effects of therapeutic treatment on HDL-C levels (Expert Panel).

Studies have demonstrated that the low-fat high-carbohydrate diet recommended by the NCEP not only lowers the undesirable LDL-C but lowers the protective HDL-C as well (Mensink & Katan, 1992). Sears (1995) proposed that this may be due to the effect that carbohydrate consumption has on insulin levels in the blood. A chronic excess consumption of carbohydrates stimulates the pancreas to maintain insulin production at above-normal levels in order to deal with the glucose generated by the metabolism of the carbohydrates. The result is hyperinsulinemia which has been shown to be associated with decreased HDL-C levels (Sears, 1993). The current dietary recommendations of the NCEP encourage Americans to decrease their total fat intake and substitute for the caloric loss by increasing carbohydrate intake. This has led to the perception by the general public that carbohydrates are harmless and can be eaten without concern for serving size. The market has been saturated with "fat-free" foods which frequently add additional sweeteners to maintain the flavor lost by the removal of fat. A United States Department of Agriculture report on food-consumption data revealed an increased intake of sugar and other refined sweeteners from about 120 lb per person per year in 1960 to 150 lb per person per year in 1995 (Katan, Grundy, & Willett, 1997). If Sears' hypothesis is correct, then current dietary recommendations may be contributing to the problem of CHD rather than helping solve it. Vega and Grundy (1996) also noted the need to critically evaluate diets that lower HDL-C levels. The ideal dietary therapy

would be one that lowers LDL-C while raising HDL-C and helps the client achieve a desirable weight.

Research about diet and its effect on health in general and heart disease specifically is of particular importance to nurses. Concern about obesity is epidemic, and cholesterol has become a household word. Unfortunately, although the awareness is there, consumers usually lack complete and accurate information regarding what constitutes obesity and when to be concerned about being overweight. They are also usually unfamiliar with any of the blood lipids other than cholesterol, and are not able to accurately interpret blood lipid results in terms of their health and the need for lifestyle changes.

Nurses are in a prime position to deal with this lack of knowledge, as they are the health professionals most frequently seen by consumers of health services. Since nurses are available in a variety of health care settings (doctor's offices, outpatient clinics, hospitals, rehabilitation facilities, nursing homes, home health care services, schools, etc.), they often field questions concerning obesity and heart disease and methods of decreasing risk. Being visible and available puts nurses in an excellent position to respond to these questions, as well as to reinforce clients' efforts at lifestyle change.

Nurses have also, over the years, engendered an image of trust and caring in the eye of the general public. This trust encourages clients to be willing to discuss lifestyle choices and changes with nurses. Nurses can use that sense of trust to work with clients to make the dietary changes needed to decrease the risk of heart disease.

As nurses move into advanced practice positions in the health care system, the need to be knowledgeable about diet and heart disease becomes even more crucial. As advanced practitioners (either nurse practitioners or clinical nurse specialists), they will be interpreting diagnostic tests and recommending treatment modalities directly to clients. Up-to-date clinical knowledge will enable advanced

practice nurses to work effectively with clients to implement lifestyle changes and improve patient outcomes.

Availability, frequent contact, and trust are crucially important in any effort to make a lifestyle change, such as changing the diet. Changing the habits of a lifetime is not easy, and requires much encouragement and reinforcement over a long period of time. Nurses are in a position to help clients make these changes, but only if they have up-to-date knowledge of the relationships between diet and CHD. Too often nurses are influenced by popular literature about diet, as is the general public, and may inappropriately use this information in health teaching in place of scientifically-based research. A thorough and current understanding of dietary metabolism, blood lipids, and the pathophysiology of heart disease is necessary if nurses are to effectively respond to clients with concerns in this area.

Clinical Framework

This pilot study followed the 1993 NCEP guidelines for the selection of appropriate subjects for dietary therapy. To determine a subject's potential for inclusion in the study, the NCEP's algorithms for primary prevention of CHD based on blood lipid levels and CHD risk factors were used.

The NCEP algorithms are a visual means of determining appropriate treatment modalities for patients with specific blood lipid levels. The initial classification of patients into treatment categories is based on total cholesterol and HDL-C (see Appendix A, Initial Classification Based on Total Cholesterol and HDL-C). An evaluation of cholesterol and HDL-C levels and positive and negative CHD risk factors are used to determine if a patient receives education on risk-factor reduction and repeat lipid evaluation at a later date, or has an immediate lipoprotein analysis done (Expert Panel, 1993).

After the lipoprotein analysis, patients are further classified on the basis of the LDL-C level (see Appendix A, Primary Prevention Algorithm Based on LDL-C). Lipid levels, this time LDL-C, are again evaluated in conjunction with

CHD risk factors to determine treatment. Low- and moderate-risk patients are given risk-factor education and/or diet instruction and scheduled for repeat lipid evaluation at a later date. High-risk patients are immediately started on dietary therapy (Expert Panel, 1993). Patients meeting the criteria for initiation of dietary therapy based on LDL-C and CHD risk factors were considered for inclusion in this pilot study.

Definitions

The defined terms will be divided into the following categories: blood lipids and related terms, anthropometric measurements, diets and related terms, types of risk factors, categories of preventive care, and medical disorders.

Blood Lipids and Related Terms

Cholesterol is one of the two main blood lipids (triglyceride is the other). It is transported in the serum in lipoproteins. It is essential for the body's production of bile salts, for the manufacture of steroid hormones, and for the composition of cell membranes. Saturated fats in the diet are metabolized to form cholesterol (Tierney et al., 1997). Serum cholesterol is an important laboratory test that assesses CHD risk, and is reasonably stable over time (Corbett, 1992). The reference values for serum cholesterol in adults are classified into risk categories: (a) desirable values are less than 200 mg/dl, (b) borderline risk values are between 200 and 239 mg/dl, and (c) high-risk values are greater than or equal to 240 mg/dl (Expert Panel, 1993).

Chylomicrons are lipoproteins that are made in the intestine and found in the blood after fat-containing foods have been eaten. The function of chylomicrons is to transfer the energy from food into muscle and fat cells. Chylomicrons are normally completely metabolized, and their blood levels are not considered in the determination of CHD risk (Tierney et al., 1997).

Dyslipidemia is defined as low levels of high-density lipoproteins, high levels of triglycerides, and unusually small-sized low-density lipoproteins (Wingard, Barrett-Connor, & Ferrara, 1995).

High-density lipoprotein cholesterol is made in the liver and intestine. HDL-C levels are inversely related to risk of CHD. The anti-atherogenic effect of HDL-C, known as reverse cholesterol transport, involves the removal of excess cholesterol from the body and prevents the build-up of LDL-C in the arteries (Mahan & Escott-Stump, 1996). The reference values for HDL-C in adults are classified by the NCEP into the following categories: (a) HDL-C less than 35 mg/dl indicates a higher risk for CHD and is considered a positive risk factor, and (b) HDL-C greater than or equal to 60 mg/dl indicates a decreased risk for CHD and is considered a negative risk factor (Expert Panel, 1993). Measurements of HDL-C levels can vary considerably due to laboratory error and real day-to-day variations (Tierney et al., 1997).

The lipid profile is a diagnostic laboratory test that by direct measurement and calculation determines the following: serum cholesterol, serum triglycerides, and lipoprotein levels (HDL-C, LDL-C, very low-density lipoprotein cholesterol) (Tierney et al., 1997).

Lipoproteins are combined protein-fat particles in the blood that carry lipids. They are classified by the varying density of the particles: alpha lipoproteins (high-density lipoproteins), beta lipoproteins (low-density lipoproteins), pre-beta lipoproteins (very low-density lipoproteins) and chylomicrons (the least dense lipoprotein) (Tierney et al., 1997).

Low-density lipoproteins carry most of the cholesterol in the plasma, and are referred to as low-density lipoprotein cholesterol. Excess amounts of LDL-C are taken up by the liver and the cholesterol within the particles is excreted into the bile. Epidemiological studies have established that as LDL-C levels increase, the risk of CHD also increases (Expert Panel, 1993). This may be related to the smaller size of the LDL-C particles and their particularly atherogenic properties when oxidized (Tierney et al., 1997). The NCEP guidelines for LDL-C levels in adults are as follows: (a) desirable values are less than 130 mg/dl, (b) borderline

risk values are between 130 and 159 mg/dl, and (c) high risk values are greater than or equal to 160 mg/dl (Expert Panel).

The total cholesterol to high-density lipoprotein cholesterol ratio is used as a summary measure of lipid-related CHD risk. As the TC/HDL-C ratio decreases, the risk of coronary heart disease also decreases (Tierney et al., 1997). A TC/HDL-C ratio of greater than 5.6 for women and 6.4 for men is associated with high risk for coronary heart disease (Mahan & Escott-Stump, 1996).

Triglyceride is one of the two main blood lipids (cholesterol is the other). It is normally present in the serum and is transported in lipoproteins. It is formed from the metabolism of neutral fats and oils that come from both animal and vegetable sources. Excess triglycerides are stored in the body as adipose tissue and used for energy. Measurement of triglyceride levels in the serum is used to identify some types of hyperlipidemia and as a variable in calculating LDL-C. Triglyceride levels may vary significantly in relation to diet and alcohol use (Corbett, 1992), as well as laboratory error and real day-to-day variations (Tierney et al., 1997). The reference range for serum triglyceride in adults is between 90 and 150 mg/dl. Hypertriglyceridemia is defined by the NCEP as levels greater than 500 mg/dl (Expert Panel, 1993).

Very low-density lipoprotein cholesterol is manufactured in the liver from fat and carbohydrate stores. These particles transport triglyceride for cellular use. As the particles lose triglycerides, their density decreases and they eventually become LDL-C particles. Very low-density lipoprotein cholesterol has an inverse relationship to HDL-C: the higher the very low-density lipoprotein level, the lower the HDL-C level (Tierney et al., 1997).

Anthropometric Measurements

Body mass index is a measure of adiposity and, by taking into account both height and weight, is correlated with total body fat in adults. It is determined by the following formula: weight in kilograms divided by height in

meters squared. BMI values between 25 and 29 kg/m^2 are considered overweight and greater than or equal to 30 kg/m^2 are considered obese (National Heart, Lung, and Blood Institute [NHLBI], 1998). Obesity is further classified as Class I (30 - 34.9 kg/m^2), Class II (35 - 39.9 kg/m^2), and Class III ($\geq$ 40 kg/m^2) (Flegal, Carroll, Kuczmarski, & Johnson, 1998).

Waist circumference is a method of classifying risk of heart disease. Individuals with a waist circumference over 40 inches in men and over 35 inches in women indicates an increased risk for cardiac disease for those who have a BMI of 25 to 34.9 kg/m^2 (NHLBI, 1998).

Diets and Related Terms

The low-fat high-carbohydrate diet (the NCEP's Step I diet) has a macronutrient breakdown by percent of total calories as follows: fat caloric intake less than 30% (8 - 10% of calories from saturated fat, 10% of calories from polyunsaturated fat, and 15% of calories from monounsaturated fat), protein caloric intake of 15%, and carbohydrate caloric intake of 55% or more (Expert Panel, 1993).

The low-fat moderate-carbohydrate diet (Sears' Zone™ diet) has a macronutrient breakdown by percent of total calories as follows: fat caloric intake of 30% (20% calories from monounsaturated fats), protein caloric intake of 30%, and carbohydrate caloric intake of 40% (Sears, 1995).

A 24-hour recall is a method of determining actual food intake when assessing the diet. In this method, the client is asked to recall all foods, liquids, and supplements ingested in the past 24 hours (Barkauskas, Stoltenberg-Allen, Baumann, & Darling-Fisher, 1994).

Types of Risk Factors

A positive risk factor is one which increases the risk of contracting a specified condition. In this case, it refers to the independent risk factors identified by the NCEP that increase the risk of CHD. The positive risk factors for CHD are:

(a) age greater than or equal to 45 for men and 55 for women, (b) family history of premature CHD, (c) smoking, (d) hypertension, (e) HDL-C less than 35 mg/dl, and (f) diabetes (Expert Panel, 1993). Each identified positive risk factor is associated with doubling the risk of CHD (Tierney et al., 1997).

A negative risk factor is one which decreases the risk of contracting a specified condition. In this case, an HDL-C level equal to or greater than 60 mg/dl is an independent negative risk factor, and indicates a decreased risk of CHD (Expert Panel, 1993). One positive risk factor may be subtracted in the presence of the negative risk factor which means a two-fold decrease in the risk of CHD (Tierney et al., 1997).

Categories of Preventive Care

Primary prevention involves the identification and treatment of patients without CHD who are at high risk for developing CHD. Determination of the need for primary prevention is based on an evaluation of CHD risk factors and total cholesterol and HDL-C values and/or LDL-C values. Clinical management includes repeat evaluations of the lipid levels at specified intervals, education about diet, exercise and other life-style modifications that will lower the risk of CHD, and initiation of the Step I diet (Expert Panel, 1993) (see Appendix A for clinical algorithms).

Secondary prevention includes the treatment of elevated LDL-C in patients with CHD and/or other atherosclerotic disease processes. Dietary treatment is initiated for those individuals with an LDL-C greater than 100 mg/dl. Drug treatment is recommended when the LDL-C is greater than or equal to 130 mg/dl (Expert Panel, 1993).

Medical Disorders

Coronary heart disease, also known as coronary atherosclerotic heart disease, is a condition in which the blood vessels supplying the heart with oxygen gradually become occluded with fatty deposits known as atherosclerotic

plaques. This can lead to angina pectoris, in which partially-occluded vessels cause temporary chest pain, and eventually to acute myocardial infarction (heart attack) (Tierney et al., 1997).

Insulin resistance precedes hyperinsulinemia and involves transient increases in serum insulin levels (Sears, 1995; Tierney et al., 1997).

Hyperinsulinemia involves continuous elevations in serum insulin levels in response to excess dietary carbohydrate (DeFronzo & Ferrannini, 1991; Sears, 1995).

Assumptions and Limitations

Dietary choices are habits of a lifetime, and as such are not easy to modify. A basic assumption of this study was that the participants would adhere to their prescribed dietary therapy. Estey, Tan, and Mann (1990) noted a significant increase in compliance with a prescribed dietary regimen when there was follow-up after the initial instruction. In order to promote compliance among the study participants, unstructured follow-up telephone calls were made by one of the registered nurse researchers on approximately the second, sixth, and eighth weeks after the initial diet instruction. The same researcher maintained telephone contact with the participants throughout the study. During these conversations, participants were asked for an informal 24-hour diet recall. Participants were also encouraged to ask questions in order to further promote adherence to the prescribed diet. Suggestions for adhering to the diet were offered as needed, and the participants were supported in their efforts. If necessary for further reinforcement, family members were involved in the discussions about diet. In addition to these nursing interventions, a follow-up appointment with the dietitian for additional diet counseling was scheduled for the fourth week.

One of the limitations of this pilot study was the small sample size, which limited its generalizability and caused some skewing of results. Initially, the researchers had planned to recruit 35 participants, but too few patients met the

study criteria within the necessary time frame. Results, however, will be useful as a basis for further research in this area with larger populations.

Additional limitations were the geographic location of the study and the gender of its participants. The pilot study was conducted in the Rio Grande Valley of South Texas and had primarily Hispanic participants. There were also no female participants fitting the study criteria at the clinic where the study was conducted. Both of these factors further limit the study's applicability to the general population.

The Hawthorne effect was another area of concern. The Hawthorne effect occurs when subjects are aware that they are participating in a study and behave in a particular manner while in the study that may affect the dependent variable but cannot be generalized to a natural setting (Polit & Hungler, 1995). For example, a subject might follow the prescribed dietary therapy but significantly increase activity level or initiate vitamin or herbal product supplementation without notifying the investigators in hopes of improving results. The self-prescribed therapies could cause changes in the final lipid levels that would be incorrectly attributed to the prescribed diet. Since participants in this study were informed of its purpose, findings may have been affected in this manner. The follow-up phone calls by the registered nurse and the fourth-week dietitian visit were an attempt to stay in touch with what the participants were doing and to discourage the addition of alternative therapies during the course of the study.

Another limitation concerned the use of 24-hour dietary recall during the follow-up phone calls to determine dietary compliance. The 24-hour recall is a method of assessing food intake which involves having the participant recall everything that has been ingested in the past 24 hours (Barkauskas et al., 1994). Self-reporting of caloric intake, however, may lack validity and accuracy. Lichtman et al. (1992) found that a majority of obese subjects misreported their actual food intake, usually underestimating amounts consumed. In addition to

this, Wynder (cited in Barkauskas et al.) mentioned that there are day-to-day variations in food consumption that, when reported, may not reflect the diet as a whole. For these reasons, the 24-hour recall done during the telephone follow-ups was not used to quantify dietary compliance, but only as a means of determining if the patient was staying on track with the diet. During the dietitian visits (initial, fourth-week, and final), compliance was determined by quantifying as a percentage the adherence to the prescribed diet, as determined by a food frequency evaluation.

CHAPTER II

REVIEW OF LITERATURE

Introduction

With the advent of new research and new technology, many of yesterday's accepted standards for the treatment of CHD are being challenged. Criteria for quantifying risk have also changed, as have the emphases on various lipid factors for diagnosis. In this review of literature, recent research about weight, HDL-C, and diet will be examined in relation to the effect on CHD. The review will be organized into the following sections: weight and CHD, HDL-C and CHD, low-fat high-carbohydrate diets and CHD, low-fat moderate-carbohydrate diets and CHD, and nursing research.

Weight and Coronary Heart Disease

The prevalence of overweight persons in the United States from 1988 to 1991 was 33.4% of adults aged 20 and older. During that same time period, the mean BMI increased to 26.3 kg/m^2 and the mean weight gain was 3.6 kg (Kuczmarski, Flegal, Campbell, & Johnson, 1994). Overweight and obesity are major public health problems that affect 55% of the American population and 97 million American adults. Approximately 100 billion dollars annually is attributed to obesity-related disease (NHLBI, 1998).

Weight changes have been associated with adverse effects on arteries and blood pressure. The Atherosclerosis Risk in Communities study examined the association between cardiovascular disease and weight change from young adulthood to middle adulthood. Study participants included 13,282 white men and women and black men aged 45 to 64 years. The results indicated that a 10 kg weight gain in adulthood promoted significant thickening of the

intimal-medial wall in coronary arteries, the area of the blood vessel where fatty plaques form (Stevens, Tyroler, et al., 1998).

Weight loss can significantly decrease cardiovascular and other disease risk. An 8 kg weight loss in obese normotensive and hypertensive individuals was found to significantly lower blood pressure, plasma glucose, and insulin concentrations in response to an oral glucose challenge. Weight loss was also associated with a decrease in insulin resistance (Su et al., 1995).

In 1998, the National Institutes of Health established guidelines for the assessment of overweight and obesity. The guidelines recommended: (a) evaluation of a patient's risk factors, such as blood pressure, blood cholesterol, and family history of obesity-related disease; (b) measurement of BMI; and (c) measurement of waist circumference. Of these three, the standards for BMI and waist circumference differ from previous National Institutes of Health recommendations. Originally, obesity was categorized from mild to severe in three steps: mild (BMI of 27.5 - 30 kg/m^2), moderate (BMI of 30 - 40 kg/m^2), and severe (BMI > 40 kg/m^2) (as cited in Tierney et al., 1997). The new guidelines created a category for overweight/pre-obese (BMI of 25 - 29 kg/m^2) and considered values greater than 30 kg/m^2 obese without further divisions (NHLBI, 1998). Flegal (1998) describes three additional categorizations of obesity: Class I (BMI of 30 - 34.9 kg/m^2), Class II (BMI of 35 - 39.9 kg/m^2), and Class III (BMI $\geq$ 40 kg/m^2).

A BMI over 25 kg/m^2 for both men and women is strongly associated with risk for cardiovascular and other diseases. As BMI increases, average blood pressure and total cholesterol increase while HDL-C decreases. When the BMI surpasses 30 kg/m^2, men have twice and women have four times the risk of hypertension, high blood cholesterol, or both, and the risk of premature death increases (NHLBI, 1998). It is unclear how obesity affects atherogenesis but it is

probably related to coexisting risk factors seen in obese individuals, such as glucose intolerance, diabetes, hypertension, and dyslipidemia, the latter being directly related to an increased BMI (Mahan & Escott-Stump, 1996).

Stevens, Cai, et al. (1998) analyzed mortality as a function of BMI across age groups over a 12-year time frame. The sample included 62,116 white men and 262,019 white women who had never smoked and had no history of heart disease, stroke, or cancer. The results indicated that greater BMI was associated with higher mortality from all causes and from cardiovascular disease in particular in men and women up to 75 years of age. Furthermore, the relative risk associated with greater BMI was higher among younger adults. An exception to these findings was found in some very muscular individuals, indicating that BMI alone does not always adequately describe the relationship of body composition and body size to health outcomes (Michels, Greenland, & Rosner, 1998).

Waist circumference was the other major change in the National Institutes of Health recommendations. Previously, a ratio of both waist and hip measurements (waist-to-hip ratio) had been standard for helping determine the risk of heart disease. The new guidelines focused only on the waist circumference as it was found that abdominal adiposity by itself was more significantly related to CHD risk. This was especially true in women, in which the hip measurements sometimes gave false negative indications of risk when combined with the waist measurements (NHLBI, 1998).

Weight distribution, specifically excess abdominal fat, is another factor that is predictive of CHD risk, as well as affecting glucose tolerance and serum lipid levels (Mahan & Escott-Stump, 1996). Waist circumference is strongly correlated with amount of abdominal fat. An excess amount of abdominal fat is an independent predictor of disease risk. Waist circumferences over 40 inches in men and over 35 inches in women associated with a BMI of 25 to 34.9 kg/m^2 signifies increased risk of obesity-related diseases (NHLBI, 1998). This finding is

supported by Lean, Han, and Seidell's study (1998), which assessed the risks of chronic disorders in people with large waist circumferences. The sample included 5887 men and 7018 women between the ages of 20 and 59. The variables assessed were respiratory insufficiency, low back pain, degree of physical function, Type 2 diabetes, and cardiovascular risk factors. The findings indicated that all symptoms and risks increased with waist circumferences of 102 cm (40.8 in.) for men and 88 cm (34.6 in.) for women.

Body-fat distribution was found to be an important factor in screening for diabetes, a condition in which fifty percent of deaths are due to CHD (Mahan & Escott-Stump, 1996). The San Antonio Heart Study, a population-based study of diabetes and cardiovascular disease, examined body-fat distribution as a diabetes screening tool. The study participants included 1,965 Mexican-American men and women who were assessed for Type 2 diabetes comparing the BMI and the waist-to-hip ratio as screening tools. The waist-hip ratio was found to be a more sensitive method of screening for Type 2 diabetes than the BMI, as the pattern of adipose tissue distribution was more predictive of metabolic consequences than total adipose tissue mass (Haffner, Mitchell, Stern, Hazuda, & Patterson, 1992).

The key element in the San Antonio study was the association of body fat distribution, specifically excess abdominal fat, with increased prevalence and incidence of Type 2 diabetes. The pathophysiology related to visceral adiposity, also described as central obesity, includes several unique features. First, there is a rapid release of stored triglycerides from the visceral adipose tissue into the circulation, which leads to increased hepatic production of both very low-density lipoproteins and glucose, and decreased insulin clearance. There is also an increased secretion of cortisol which may be directly related to or cause an increase in insulin resistance (Arnesen, 1992). These pathophysiologic changes directly affect the severity of diabetes and point to the connection between diabetes and CHD.

High-density Lipoprotein Cholesterol and Coronary Heart Disease

A follow-up of the Lipid Research Clinics Prevalence Study evaluated the role of HDL-C in the epidemiology of CHD and other cardiovascular disease mortality. The study sample included 8,825 male and female participants between the ages of 30 and 99. The findings indicated that HDL-C was inversely related to both CHD and other cardiovascular disease mortality for both men and women. Furthermore, in women HDL-C levels were found to be more closely related to cardiovascular disease than LDL-C levels (Jacobs, Mebane, Bangdiwala, Criqui, & Tyroler, 1990).

As has been noted previously, obesity is a major public health concern. Derby et al. (1998) surveyed 12,223 members of the Pawtucket Heart Health Program and found that during the period from 1981 to 1993, the prevalence of obesity was increased. This was associated with a concurrent decrease in the prevalence of smoking and alcohol use. The trends in these cardiovascular risk factors were examined in relation to HDL-C levels, which were found to be decreased. Even though two cardiovascular risk factors saw improvement, the decreasing HDL-C levels seen in light of the increasing obesity points to an association between the two factors and shows little improvement in total CHD risk.

The importance of HDL-C levels is apparent by their incorporation into the newest NCEP guidelines. The 1993 guidelines recommend that more emphasis be placed on HDL-C by (a) measuring HDL-C as part of initial cholesterol testing, (b) designating elevated HDL-C levels as a negative risk factor, and (c) considering the effect of drug therapy on HDL-C levels when determining treatment regimens (Expert Panel, 1993).

Low-fat High-carbohydrate Diets and Coronary Heart Disease

The NCEP's strategy for control of high blood cholesterol in Americans is based on the compilation of four expert panel reports in the field of cholesterol.

The Adult Treatment Panel I report, published in 1988, provided the first national recommendations for the detection, evaluation, and treatment of high blood cholesterol in adults (Expert Panel, 1993). In 1990, the Laboratory Standardization Panel report provided recommendations for improving the accuracy of cholesterol measurement (Expert Panel). In that same year, the Population Panel report identified a public health approach to decrease cholesterol in the entire population through dietary change (Expert Panel). The Children's Panel report was published in 1991 and provided the guidelines for cholesterol management in children (Expert Panel). The Adult Treatment Panel II, the second report of the Expert Panel on Detection, Evaluation, and Treatment of High Blood Cholesterol, provided the current updated recommendations (Expert Panel).

The NCEP recommendation for primary prevention is the Step I diet. The Step I diet consists of 30% or less of total daily calories from dietary fat, 15% of total calories from protein, and 55% or more of total calories from carbohydrates. The rationale for recommending a low-fat high-carbohydrate diet is that by reducing dietary fat to 30% or less of total calories, with saturated fat 10% or less of total calories and dietary cholesterol less than 200 - 300 mg/day, total cholesterol and LDL-C will be lowered (Expert Panel, 1993). Total cholesterol has been shown to decline 10 - 15% in response to a sustained low-fat high-carbohydrate diet (Bendich & Deckelbaum, 1997).

Other research has supported the use of low-fat high-carbohydrate diets. Epidemiological studies have demonstrated that compared to populations that eat a high-fat diet (> 30%), populations that normally eat a low-fat (< 25%) high-carbohydrate diet, such as in developing nations, have fewer diet-related chronic diseases, especially CHD (Bendich & Deckelbaum, 1997). Low-fat high-carbohydrate diets based on complex carbohydrates consisting of cereal grains, vegetables, and fruit are advantageous for both sedentary obese and active lean populations in decreasing CHD risk (Rimm et al., 1996).

Although low-fat high-carbohydrate diets reduce some CHD risk factors, they also are associated with negative effects such as an increase in plasma triglycerides and a decrease in HDL-C (Grundy & Denke, 1990). The impact on HDL-C has been a particular source of concern for investigators. A meta-analysis of 27 trials evaluated the effects of dietary fats on serum lipids and lipoproteins and concluded that low-fat high-carbohydrate diets lower both LDL-C and HDL-C (Mensink & Katan, 1992). Schaefer et al. (1995) compared the effects of weight maintenance low-fat diets and unrestricted low-fat diets on body weight and plasma lipid levels in hypercholesterolemic subjects. In the weight maintenance phase, a low-fat diet of 15% fat and 68% carbohydrate was prescribed and caloric intake was adjusted to keep body weight constant. In the unrestricted phase, the same low-fat diet was prescribed, but patients could adjust their caloric intake, primarily in the form of carbohydrates. The low-fat weight maintenance diet effectively reduced total cholesterol and LDL-C, but there was a greater decrease in HDL-C levels than in total cholesterol levels, resulting in a worsening of the TC/HDL-C ratio. In contrast, in the unrestricted diet phase, subjects chose to eat less, primarily by eating fewer carbohydrates, and significant weight loss and LDL-C lowering without adverse effects on triglycerides, total cholesterol, or HDL-C were observed. The results indicated that a low-fat high-carbohydrate diet reduces the risk of coronary artery disease as long as it is accompanied by weight loss.

In addition to the effects on total cholesterol and LDL-C, a major argument for the use of low-fat diets is that they promote weight reduction. This position has not been entirely supported, however. In one study of diet and weight loss, the researchers found that a few months after initiating a fat-restricted diet, weight reduction stopped, even though the diet was continued. The net weight loss was only 0.8 to 2.6 kg (Willett, 1994).

Low-fat Moderate-carbohydrate Diets and Coronary Heart Disease

The low-fat moderate-carbohydrate diet recommended by Sears consists of 30% of total calories from fat (20% of calories from monounsaturated fats), 30% of total calories from protein, and 40% of total calories from carbohydrates. The rationale for the low-fat moderate-carbohydrate diet is based on the effects of elevated insulin levels and their relationship to CHD risk. The key enzyme that controls cholesterol synthesis, HMG-CoA reductase, is strongly activated by insulin. Reductions in insulin levels tend to lower triglycerides and LDL-C and increase HDL-C. Sears (1995) emphasized that CHD risk is related to hyperinsulinemia. The risk of CHD associated with hyperinsulinemia can be decreased by using specific proportions of food to control the secretion of insulin. The macronutrient ratio between fat, protein, and carbohydrate provided by the diet facilitates the hormonal balance of insulin.

Hyperinsulinemia is a compensatory mechanism in which the body increases the production of insulin to counterbalance insulin resistance. The body becomes markedly resistant to the action of insulin when an individual consumes excessive calories and gains weight (DeFronzo & Ferrannini, 1991). The effect of insulin resistance on the body includes a reduction in glucose uptake by muscle and fat cells and a reduction in the inhibitory effect of insulin on the liver which results in an increase in hepatic glucose output. The net effect is an increase in blood glucose, which stimulates the pancreatic beta cell to release more insulin. The result is hyperinsulinemia (Vaaler, 1992).

Insulin resistance and hyperinsulinemia are associated with a high-risk plasma lipid profile. Elevated insulin concentrations increase the development of very low-density lipoproteins, leading to the development of hypertriglyceridemia and increasing the formation of low-density lipoproteins. The hormone insulin is recognized to be atherogenic independent of its effects on blood lipids. Insulin enhances cholesterol transport into arteriolar smooth muscle cells and increases

endogenous lipid formation by these cells. Insulin also increases the formation of lipid plaques and decreases the break-down of lipid plaques (DeFronzo & Ferrannini, 1991).

Several studies have demonstrated the relationship between insulin and CHD. The findings of the Paris Prospective Study, the Busselton Study, the Quebec Cardiovascular Study, and the Helsinki Policeman Study all concurred in establishing that hyperinsulinemia predicted CHD risk (Després et al., 1996; Fontbonne et al., 1991; Pyörälä, Miettinen, Laakso, & Pyörälä, 1998; Welborn & Wearne, 1979). However, the predictive value of hyperinsulinemia decreased over time. In the Helsinki Policeman Study, two explanations were considered for this phenomenon. First, the strong association of hyperinsulinemia to CHD may have caused selective morbidity and mortality early in the study. Second, long follow-up periods allow for the development of a cumulative impact of other risk factors and may have overshadowed the impact of hyperinsulinemia (Pyörälä et al.).

Not all studies agree that there is a difference between low- and high-carbohydrate diets. Golay et al. (1996) found no significant difference in weight loss between subjects following the two diets; weight loss was more closely related to total caloric energy intake. On the other hand, variations in dietary composition did alter the beneficial effects of weight loss. Low-carbohydrate diets led to an improvement in insulin levels and lipid metabolism compared with reduced-calorie diets that were low in fat and high in carbohydrate. These findings concur with Sears' research.

Nursing Research

Although there is a wealth of information about diet and CHD in the literature, very little of that literature is from the nursing perspective. A thorough computer search of nursing and allied health literature revealed no research about blood lipids and diet attributed to nursing since 1982. Nursing studies have been

done related to diet or to cardiovascular health, but none were located that examined the effects of diet on CHD.

A total of nine nursing research studies were located that examined disease and weight, exercise, or diet. Of those nine studies, only three were related to cardiovascular health. Gillett, Caserta, White, and Martinson (1995) described the responses of 49- to 59-year old sedentary overweight women to exercise conditioning and/or fitness education. Brunt and Shields (1996) used the preventive behaviors questionnaire to determine the effect of a nurse-managed cholesterol screening program. Lipp, Deane, and Trimble (1996) focused their study on the identification of cardiovascular disease risk factors in adolescent males.

Summary

There is a consensus in the literature that increased weight, BMI, and abdominal adiposity increase the risk of CHD. National assessment standards have been revised to reflect the most recent findings in those areas. The standards have also been revised to emphasize the importance of HDL-C as a risk factor. Low-fat high-carbohydrate diets, the accepted dietary therapy for primary prevention, are known to lower cholesterol and LDL-C levels, but there has been increased concern about their effect of lowering HDL-C levels as well. It has been suggested that by preventing hyperinsulinemia, low-fat moderate-carbohydrate diets may provide the benefits of the high-carbohydrate diets without affecting HDL-C levels, but studies have not conclusively supported that hypothesis.

CHAPTER III

METHODS

Research Design, Population, Sample, and Setting

This pilot study was a quasi-experimental before-after design using quantitative analysis with a non-equivalent control group. Polit and Hungler (1995) define a quasi-experiment as one in which the research participants are not randomly assigned, although the independent variable is manipulated and certain controls are maintained to enhance internal validity. In before-after experiments data are collected from the participants before the experimental intervention and again afterwards. Quantitative analysis indicates that numerical data, as opposed to narrative or nonnumerical data, are being statistically manipulated to assess the magnitude and reliability of relationships between the data. A non-equivalent control group is a comparison group that is not necessarily equivalent to the experimental group since randomization is not used.

The pilot study was conducted over a period of 12 weeks in an outpatient clinic in the Rio Grande Valley of South Texas. Approval for work with human subjects was granted by both the University of Texas-Pan American Human Subjects Review Committee (see Appendix B for approval form) and with telephone approval from the institutional review board of the Audie Murphy Veteran's Administration Hospital in San Antonio. Participants were selected from patients at the clinic who were scheduled for initial dietary consultation. From that group, 37 subjects who met the criteria for primary prevention for CHD with dietary therapy according to the 1993 NCEP guidelines were identified for possible inclusion in the study. Subjects with medical conditions or taking medications known to affect lipid levels were excluded. Of the original 37

who were identified as possible subjects, a group of 20 males with ages ranging from 25 to 65 met all study criteria and were willing to participate. The low-fat high-carbohydrate diet of 30% fat, 15% protein, and 55% carbohydrate recommended by the NCEP (the Step I diet) was prescribed to the control group and the low-fat moderate-carbohydrate of 30% fat, 30% protein, and 40% carbohydrate proposed by Sears in 1995 (the Zone™ diet) to an experimental group. Study participants were assigned to the control and experimental groups by alternate enrollment. The independent variables were the two diets, and the dependent variables were the CHD risk assessment measures: TC/HDL-C ratio, BMI, and waist circumference. The dependent variables were assessed prior to the initiation of the dietary interventions and again after 12 weeks.

Sampling Plan

Patients at the clinic received wellness screening as part of their routine care. Part of this wellness screen included a lipid profile with lipoprotein analysis. Primary prevention, as per the 1993 NCEP guidelines, was automatically initiated for patients that fell into moderate- or high-risk categories. The primary prevention included, among other measures, an appointment with the clinic dietitian for instruction in a diet to decrease the risk of CHD. The dietitian's list of patients scheduled for new appointments thus included a large number of patients who were candidates for inclusion in the pilot study.

Using this list provided by the dietitian, the investigators were able to determine the subjects meeting the specific study criteria. Since lipoprotein analyses were routinely done with the lipid profiles of the wellness screen, only the primary prevention algorithm based on LDL-C was used. Inclusion criteria for the study followed the 1993 NCEP clinical guidelines: (a) LDL-C of 130 - 159 mg/dl with two or more positive CHD risk factors, or (b) LDL-C > 160 mg/dl. The positive CHD risk factors included the following: (a) age for men greater than 45 years, for women greater than 55 years, or premature menopause

without estrogen replacement therapy, (b) family history of premature CHD, (c) smoking, (d) hypertension, (e) HDL-C < 35 mg/dl, and (f) diabetes (Expert Panel, 1993) (see Appendix A for the clinical algorithms).

The exclusion criteria included the presence of the following disease processes known to have effects on lipid levels: (a) hypothyroidism, (b) nephrotic syndrome, (c) hepatic disease, (d) congestive heart failure, or (e) CHD treated with lipid-lowering agents. Female subjects who were pregnant or on hormone replacement therapy were also excluded due to the effects of the hormones on lipid levels. Subjects with diabetes mellitus Type 2 or hypertension were included in the study with the exception of those who were currently taking medications known to affect lipid levels (ie. glucophage, insulin, diuretics, thiazides, beta-blockers). The primary care providers at the clinic helped determine subject appropriateness with consideration for disease process and medication interaction on the lipoprotein analysis.

After determining eligibility for the study, participants signed an informed consent. The consent was modeled after the format required by the clinic, and included a summary of the study; expected risks, benefits, and cost of participation; an assurance of care in the event of non-participation or injury; statements of confidentiality and voluntary participation; and information about contact persons (see Appendix C, Consent Form).

The desired sample size for the study was determined by evaluating the outpatient clinic dietitian's appointment list over a one-year period. The investigators noted an average of 90 scheduled appointments for initial diet consultation per month. Out of the 90 new diet appointees, an average of 36.6% per month met the study criteria, yielding a tentative sample size of 33 patients. Due to time constraints, this size sample was unable to be obtained. This pilot study of 20 subjects will help researchers determine the effect size in this population and an appropriate sample size for a future study.

Data Collection Methods and Procedures

Within approximately 12 weeks of the lipid profile used to determine eligibility for the study, subjects met with the clinic's registered dietitian and had a one-hour appointment during which initial anthropometric measurements (height and weight to determine BMI, and waist circumference) were taken and the assigned diet was explained. The dietitian determined each participant's current energy expenditure as a means of determining any changes in activity level over the course of the study. Subjects were cautioned to avoid the initiation of any alternative therapies (such as new exercise programs or supplementation with any vitamins, minerals, herbs, or other similar products) that might obscure the results of the study. Any supplements that were currently in use were noted.

A second visit with the dietitian at approximately four weeks enhanced the participants' knowledge of and compliance with their assigned diets. This visit was for teaching and reinforcement only, and no measurements or blood analyses were taken.

The study concluded at the end of 12 weeks with a final lipoprotein analysis and dietitian appointment. During the appointment, a summary dietary review was done to determine overall adherence to the prescribed diet, energy expenditure was assessed and compared to the initial determination, use or non-use of alternative therapies was verified, and final anthropometric measurements were taken.

All lipoprotein analyses were collected at the outpatient clinic laboratory and sent to the same diagnostic lab for processing. Blood work was collected after having the subjects fast overnight. Blood samples were drawn by laboratory technicians using standard techniques.

The dietitian collected all the anthropometric measurements. The same instrumentation was used for both the initial and follow-up data collections. The following anthropometric data was obtained: weight (without shoes) in pounds

on an electronic scale, and height (without shoes) in inches. These measurements were used to calculate the BMI, which is a measure of adiposity that takes into account both height and weight and is correlated with total body fat in adults (NHLBI, 1998). The waist circumference was measured in centimeters with a plastic measuring tape over clothing. Individuals with a waist circumference over 40 inches in men and over 35 inches in women have an increased risk for cardiac disease if they also have an increased BMI (NHLBI).

At the initial visit, the dietitian also determined each participant's total energy expenditure in order to assess any changes in activity level over the course of the study. Total energy expenditure was determined by estimating resting energy expenditure (weight in kg x 30 - 35 kcal) and multiplying by each individual's activity level (based on a summation of reported daily activities in minutes). These results were then categorized into four levels: sedentary (up to 2.5 kcal/min), moderately active (2.5 - 4.9 kcal/min), active (5.0 - 7.4 kcal/min), and very active (7.5 - 12.0 kcal/min) (Mahan & Escott-Stump, 1996). The dietitian determined the total energy expenditure at each diet visit and calculated degree of change.

The information collected by the dietitian at the initial, fourth-week, and final diet appointments (ie. anthropometric measurements and calculations, activity level, dietary compliance) was documented on forms routinely used at the clinic (see Appendix C, Nutritional Care Information form). Calculation of BMI and waist measurements were not routinely documented, but were incorporated into the visits by the dietitian for the purposes of the study. The pertinent information was summarized (as necessary) and transcribed to a data collection worksheet.

A data collection worksheet was maintained for each participant to keep pertinent information in one location. The worksheet included demographic data, pertinent medical history, initial and final lipid profile values, initial and final

anthropometric measurements and activity level, and summaries of the follow-up appointment and phone calls. The information on the worksheet was transcribed by the nurse researchers from various sources: the participants' medical records (which included the laboratory results), the Nutritional Care Information form, and the researcher's notes of the follow-up phone calls (see Appendix C, Data Collection Worksheet).

A flowsheet was also used to keep track of each participant's progress in the study. The flowsheet included identifying information for all the participants and dates on which each of the critical steps of the study was completed (see Appendix C, Flowsheet).

Dietary Instruction

The control and experimental diets were explained at the initial visit with the dietitian. The low-fat high-carbohydrate control group was instructed in ways of decreasing sources of fat and cholesterol in the diet. Since no restrictions were placed on quantity of intake, the diet was not considered a weight reduction diet. The low-fat moderate-carbohydrate experimental group was provided detailed instruction on meal patterns and maintaining the proper balance of protein, carbohydrate, and fat. Since Sears (1995) recommended that the total grams of protein, carbohydrate, and fat be based on the individual's lean body mass and physical activity level (ie. intake would be limited), this diet was considered a weight reduction diet. Standardized sample menus for each diet were given to the participants to help them incorporate the new diets into their lifestyles.

Dietary Compliance

Participants were followed throughout the course of the study to determine their progress with their diets. Dietary compliance was evaluated by information collected by both the primary investigators and the dietitian. The investigators assessed general compliance by using an informal 24-hour diet recall. Burke (as cited in Frank-Stromborg& Olsen, 1997) stated that the

advantages of the 24-hour recall are its ease of use for both researcher and participant. Only short-term memory recall is required of the participant, and the interviewer can obtain the data quickly and over the telephone. One of the registered nurse researchers made telephone contact with each participant or an informed family member approximately two, six, and eight weeks after the initial visit with the dietitian. During the unstructured phone conversations, the diet recall was obtained and rated as poor, fair, good, or excellent based on the participant's description of foods consumed. The nurse also answered questions, helped make arrangements for follow-up appointments for the study at the clinic, and offered encouragement.

Approximately four weeks after the initial dietitian visit, the participants had a second visit with the clinic dietitian lasting about 45 minutes. At this time, the dietitian evaluated compliance more specifically through a detailed review of each subject's 3-day diet diary. From the diary, the frequency of consumption of cholesterol, fat, and carbohydrate was documented on a food frequency record in order to determine the degree of change in consumption of these items between each visit. To evaluate these items, the dietitian looked specifically at the following areas: (a) number of meals and snacks per day; (b) number of eggs consumed per day; (c) servings of organ meats per day; (d) servings of shellfish; (e) amount of high-fat food consumption, which included number of high-fat foods eaten outside the home; (f) servings of sweets per day which included sugar-sweetened drinks; (g) amount of alcohol intake; and (h) starchy carbohydrates. Compliance levels were rated as poor (0 - 20%), fair (21 - 50%), good (51 - 75%), and excellent (76 - 100%) based on the amount of deviation from the prescribed diet. Activity level was also evaluated for degree of change and rated accordingly. To enhance the participants' understanding of their diets, the dietitian assisted them in the creation of personalized menus incorporating the guidelines of the specified diet.

Other methods were used to enhance dietary compliance. In addition to the phone calls by the nurse, a letter of encouragement written by one of the nurse researchers was sent to each participant at approximately 10 weeks. This letter included a pamphlet about CHD to reinforce the importance of reducing the risk of heart disease by following the prescribed diet.

Data Analysis

Descriptive statistics were used to describe the study sample. Demographic data collected included age, gender, ethnic background, and work status (employed/student or unemployed). Information was also collected about relevant medical history, medication use, activity level, and smoking status.

Quantitative measures were used to assess the effect of the independent variables on the dependent variable. The independent variables were the low-fat high-carbohydrate and low-fat moderate-carbohydrate diets. The dependent variables included the following CHD risk assessment factors: TC/HDL-C ratio, BMI, and waist circumference. To evaluate the differences between the dependent variables of the two groups of patients before and after dietary therapy intervention, *t*-tests were liberally used. A descriptive analysis of dietary compliance was also performed.

Collaboration Among Disciplines

This study required considerable collaboration between healthcare disciplines. Medicine was involved in the development of the clinical framework for the study, with the clinic physicians providing information about CHD and treatment modalities. The physicians' involvement in this area enhanced their sense of participation in the study, so much so that they were still recommending patients for inclusion in the study long after the enrollment period was over.

Dietetics obviously played a large part in the study. The clinic dietitian not only provided all the comprehensive dietary instruction and performed the measurements, but also provided information about the diets to the researchers,

assisted in the initial enrollment of subjects, and helped ensure that patients were seen on schedule for their follow-up and final visits.

Nursing played a coordinating role. As researchers, the registered nurses acted as organizers and intermediaries, taking the medical and dietetic information on CHD and the moderate- and high-carbohydrate diets and forming them into a cohesive research plan emotionally owned by all three disciplines. As clinicians and educators, they brought the plan down to the level of the research participants so that the they could incorporate the dietary changes into their lifestyles.

CHAPTER IV

RESULTS

Of the 37 subjects initially identified for the study, 20 male subjects between the ages of 25 and 65 met the enrollment criteria and were willing to participate in the study. The clinic population was primarily male, and only two women met the initial criteria for inclusion in the study. Neither were eventually included in the study as one did not meet the final study criteria (taking estrogen supplements) and the other was not willing to participate. The reasons for exclusion of the remaining possible participants were the presence of Type 2 diabetes treated with insulin and/or glucophage ($n = 4$), hypertension treated with beta-blockers ($n = 1$), elevated liver functions ($n = 1$), and unwillingness to participate ($n = 9$). Subjects were assigned by alternate enrollment to either the low-fat high-carbohydrate diet or the low-fat moderate-carbohydrate diet. Ten subjects were assigned to each diet group. Of the 20 subjects enrolled, 18 eventually completed the 12-week study.

The Microsoft Excel computer program was used to perform the statistical analysis. A two-tailed t-test assuming unequal variances was used to determine whether there were differences in BMI, waist circumference, and TC/HDL-C ratio between the two diet groups. Values for $p < 0.05$ were considered to denote significant differences. The two-tailed probability (p value) for BMI, waist circumference, and TC/HDL-C ratio was calculated at $p = 0.9$, 0.9, and 0.75 respectively, which demonstrated that there were no significant differences observed between the two samples.

The characteristics of the study population were examined for the low-fat high-carbohydrate control and low-fat moderate-carbohydrate experimental diet

groups (see Table 1). Variances between the two groups for the variables gender, age and employment status were not observed. The variables for ethnic origin, smoking, and medical diagnosis, however, demonstrated differences in distribution between the two groups. The control group was comprised of 90% Hispanics, compared to the 75% Hispanic population in the experimental group. There were 30% who identified themselves as current smokers; all three were members of the control group. Distribution of medical diagnoses also varied greatly between the two groups. In the control group, 40% had a diagnosis of hypertension currently treated with either an ACE inhibitor (n = 1) or calcium channel blocker (n = 3). The experimental group had a diagnosis distribution of 13% hypertension currently treated with a calcium channel blocker and 13% Type 2 diabetes treated with dietary therapy.

Table 1

Characteristics of the Study Population

Attribute	Control Group $n = 10$	Experimental Group $n = 8$
Gender	male	male
Age (years)	mean = 45.9 $\pm$ 11.8	mean = 46.6 $\pm$ 10.3
Ethnic Origin	Hispanic $n = 9$	Hispanic $n = 6$
Medical History	hypertension $n = 4$	hypertension $n = 1$ diabetes Type 2 $n = 1$
Current Smoker	$n = 3$	$n = 0$
Currently Employed	$n = 8$	$n = 6$

Values for height, weight, BMI, and waist circumference before and after diet intervention are provided in Table 2, with BMI and waist circumference being the variables of interest. There was a notable variance in the BMI between the two diet groups. The low-fat high-carbohydrate control group was on average slightly taller in height and weighed less than the low-fat moderate-carbohydrate

experimental group by 15 lb (6.8 kg). The control group also had a pre-intervention mean BMI of 29.0 kg/m^2, which is considered overweight. In comparison, the experimental group had a pre-intervention mean BMI of 32.4 kg/m^2, which is considered obese. The categories for obesity remained the same for each group post-intervention with an average weight loss for the control group of 5.2 lb (2.3 kg) and an average weight loss for the experimental group of 4.4 lb (2.0 kg).

Table 2
Anthropometric Measurements Pre- and Post-Intervention

	Control Group $n = 10$		Experimental Group $n = 8$	
Attribute	Before	After	Before	After
Height (in.)				
mean	68.3 ± 2.5	68.3	67.1 ± 0.7	67.1
range	65.3 - 71.8		65.3 - 71.8	
Weight (lb)				
mean	191.9 ± 22.3	186.7 ± 20.7	206.9 ± 23.9	202.5 ± 26.3
range	161.3 - 230.5	158.5 - 227.0	179.5 - 239.5	172.3 - 242.8
BMI (kg/m^2)				
mean	29.0 ± 2.7	28.2 ± 2.8 [1]	32.4 ± 3.3	31.7 ± 3.8 [1]
range	24.7 - 34.1	24.1 - 33.6	28.8 - 36.6	26.8 - 37.8
Waist Circumference (in.)				
mean	40.4 ± 3.0	39.6 ± 3.3 [1]	43.0 ± 2.5	42.3 ± 3.8 [1]
range	36.5 - 45.5	35.6 - 44.4	40.0 - 46.4	37.3 - 48.0

[1] two-tailed t-test $p = 0.9$

Waist circumference also varied between the two groups before and after diet intervention. The initial waist circumference for the low-fat high-carbohydrate control group was 2.6 in. (6.6 cm) smaller than the low-fat moderate-carbohydrate experimental group. Upon final measurement, waist circumference decreased an average of 0.8 in. (2.0 cm) for the control group and 0.7 in. (2.8 cm) for the

experimental group. The variance in waist circumference between the two groups remained the same at 2.7 in (6.9 cm).

Lipid and lipoprotein levels were evaluated between the two groups before and after diet intervention (see Table 3); the variable of interest was the TC/HDL-C ratio. The initial lipid and lipoprotein levels between the two diet groups were very similar, but notable post-intervention differences were observed. The low-fat high-carbohydrate control group demonstrated a mean decrease in total cholesterol of 29 mg/dl, a mean decrease in LDL-C of 13 mg/dl, and a mean decrease in HDL-C of 9 mg/dl. In comparison, the low-fat moderate-carbohydrate experimental group demonstrated a mean decrease in total cholesterol of 8 mg/dl, a mean increase in LDL-C of 3 mg/dl , and a mean decrease of HDL-C of 5 mg/dl. A worsening of the TC/HDL-C ratio was observed for both diet groups.

Table 3
Lipid Levels Pre- and Post-Intervention

	Control Group $n = 10$		Experimental Group $n = 8$	
Lipid Factor	Before	After	Before	After
Total Cholesterol (mg/dl)				
mean	250 ± 33.2	221 ± 51.4	250 ± 36.0	242 ± 34.1
range	213 - 307	158 - 343	212 - 307	184 - 299
HDL-C (mg/dl)				
mean	47 ± 10.6	38 ± 7.0	48 ± 12.1	43 ± 8.8
range	29 - 61	27 - 50	32 - 70	28 - 51
LDL-C (mg/dl)				
mean	167 ± 27.8	154 ± 44.8	165 ± 29.8	168 ± 26.1
range	140 - 214	101 - 264	132 - 210	129 - 207
TC/HDL-C				
mean	5.6 ± 1.9	6.2 ± 2.6 [1]	5.5 ± 1.4	5.8 ± 1.6 [1]
range	3.8 - 10.5	3.5 - 12.7	4.1 - 8.3	4.5 - 9.3

[1] two-tailed t-test $p = 0.75$

In summary, the low-fat high-carbohydrate control group was taller and leaner and demonstrated greater improvement in weight loss and lipid metabolism than the low-fat moderate-carbohydrate experimental group. The exception to this trend was seen in the HDL-C levels, which decreased in both groups. The decrease in HDL-C led to a worsening of the TC/HDL-C ratio for both groups, but it was noted that the worsening of the TC/HDL-C ratio was greater for the control group. Although the findings were not significant, the negative impact of a low-fat high-carbohydrate diet on the TC/HDL-C ratio was observed in this sample.

CHAPTER V

DISCUSSION AND SUMMARY

The findings of the pilot study demonstrated no statistical differences ($p > 0.05$) between the low-fat high-carbohydrate control group and low-fat moderate-carbohydrate experimental group for the CHD risk assessment factors: BMI, waist circumference, and TC/HDL-C ratio. The small sample size and the variability between and within the two samples significantly decreased the power of the pilot study to detect differences between the control and experimental groups.

Initially, sample size justification was determined by the average number of lipid profiles performed monthly at the research site ($n = 188$). Since Schaefer et al. (1995) found that hypercholesterolemic subjects placed on a low-fat high-carbohydrate diet experienced an increase in the TC/HDL-C ratio, it was expected that a significant percentage of the participants placed on the low-fat high-carbohydrate diet would have a TC/HDL-C ratio greater than 4.0. At least 50% of the participants on the low-fat moderate-carbohydrate diet would have to demonstrate a TC/HDL-C ratio of less than 4.0 to determine significance. A Fisher's exact test with a 0.05 one-sided significance level would have had an 80% power to detect a difference of 45% between the proportion of participants on the two diets with TC/HDL-C ratios less than 4.0 when the sample size in each group was 15. The actual sample size of the pilot study was 18 participants which lessened the ability to determine statistical significance.

Weight loss was observed in both diet groups. The low-fat high-carbohydrate control group had an average weight loss of 5.2 lb (2.3 kg) and the low-fat moderate-carbohydrate experimental group had an average

weight loss of 4.4 lb (2 kg). This correlated with the 0.8 - 2.6 kg weight loss found by Willett (1994). There was no significant difference in weight between the control group and the experimental group which concurred with the observations made by Golay et al. (1996).

Variances in BMI and waist circumference between the two diet groups affected the results of the study. Before diet intervention, the low-fat high-carbohydrate control group had both a lower BMI and a smaller waist circumference compared to the low-fat moderate-carbohydrate experimental group. The National Institutes of Health guidelines recognize that the risk of CHD, other diseases, and death increase with increased BMI and waist circumference (NHLBI, 1998). Therefore, the relative risk of CHD was already greater at the outset of the study for the experimental group, which had a greater average BMI and waist circumference.

Compliance was another factor that influenced the results of the pilot study. Sixty percent ($n = 6$) of the participants in the low-fat high-carbohydrate control group followed their diet 51 - 75% of the time (the "good" rating), compared to 37.9% ($n = 3$) of the participants in the low-fat moderate-carbohydrate experimental group. None of the participants in either group were rated in the "poor" or "excellent" categories. Possible reasons for the lower compliance level of the experimental group may have been the complexity of following the diet and/or the difficulty of incorporating the diet into a busy working schedule. Also, the lower compliance level of the experimental group explains the smaller changes in weight loss and lipid levels compared to the control group.

Physical activity also had an effect on the results. Even though levels of physical activity did not change over the 12-week time period, there was a notable initial difference in the level of physical activity between the two groups. Fifty percent ($n = 5$) of the low-fat high-carbohydrate control group identified themselves as active, compared to 25% ($n = 2$) of the low-fat

moderate-carbohydrate experimental group. Since a higher degree of physical activity has been shown to improve HDL-C (Expert Panel, 1998), it is interesting to note that the control group, which had a greater number of active subjects than the experimental group, demonstrated a greater decrease in HDL-C and a worsening of the TC/HDL-C ratio. A larger sample size would be needed to decrease the variance between the two groups in order to prove any significance to the findings.

Although the results of the pilot study were not statistically significant, an interesting observation involved the TC/HDL-C ratio. After the 12-week period, LDL-C decreased considerably for the low-fat high-carbohydrate control group. HDL-C also decreased, which led to a worsening of the TC/HDL-C ratio. These findings correlate with those of Schaefer et al. (1995), in which it was found that HDL-C was lower in all subjects on low-fat (15%) high-carbohydrate (68%) diets compared to a baseline diet of 35% fat and 48% carbohydrate. The studies by Vega and Grundy (1996) and Mensink and Katan (1992) concur.

In the low-fat moderate-carbohydrate experimental group, LDL-C increased and HDL-C decreased slightly, leading to a worsening of the TC/HDL-C ratio. An explanation for the increase in LDL-C may be that the experimental group was instructed to consume 15% more protein than the low-fat high-carbohydrate control group. Evaluation of the food diaries revealed that many of the protein sources chosen by the participants were high in saturated fat.

Two extreme findings were observed in the lipid profiles of the low-fat high-carbohydrate control group. Over the course of the study, two participants in this group decreased their total cholesterol from 285 to 207 mg/dl and 285 to 158 mg/dl, and their LDL-C from 207 to 148 mg/dl and 194 to 105 mg/dl, respectively. Although these are positive findings, the likelihood of achieving these types of results over a 12-week period with diet alone is very remote. These values skew the results of the study by improving the average lipid values for the

control group. Oddly, even with the skewed results of the total cholesterol levels, the TC/HDL-C ratio for the control group was not improved.

The TC/HDL-C ratio for the low-fat high-carbohydrate control group worsened to a greater extent than that of the low-fat moderate-carbohydrate experimental group. This was an extremely interesting observation considering that the control group demonstrated greater improvements in BMI, waist circumference, LDL-C and total cholesterol than the experimental group. Furthermore, the control group started off taller, leaner, and more active, and demonstrated greater compliance with their prescribed diet. The unusual improvement in total cholesterol levels, which should have improved the lipid profile for the control group, did not decrease the TC/HDL-C ratio, which in turn did not decrease risk for CHD.

This pilot study did not provide a sufficient sample size to adequately compare the differences between the low-fat high-carbohydrate control group and the low-fat moderate-carbohydrate experimental groups. Although the hypothesis was not statistically supported, the observations seemed to indicate that there was a relationship between the amount of carbohydrate intake in the diet and the degree of CHD risk as indicated by the following risk factors: TC/HDL-C ratio, BMI, and waist circumference.

There is a strong need to replicate this pilot study. Recommendations for improvement include increasing the sample size in order to decrease the variability between the two groups and enhance the significance of the findings. Also, integration of a valid research tool to measure compliance/non-compliance would hopefully provide insight into ways to decrease the barriers that prevent healthy lifestyle changes.

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APPENDIX A
APPROVAL FORM

NURSING DEPARTMENT
THE UNIVERSITY OF TEXAS - PAN AMERICAN
1201 West University Drive • Edinburg, Texas 78539-2999 • (956) 381-3491 / 316-7032 • Fax (956) 381-2384

MEMORANDUM

TO: George Avellano
Associate Vice President for Graduate Programs
And Planning

FROM: Barbara Tucker, Coordinator BT
Master of Science in Nursing Program

DATE: July 22, 1998

RE: Human Subjects Review Committee

Margaret Fernandez and Julia Soper are currently enrolled in NURS 7300 Thesis I (Proposal). They will present their proposal to their committee the first week in August.

The proposed dietary intervention has the approval and support of the patients' attending physician at the McAllen Audie Murphy Veteran's Administration Outpatient Clinic and is awaiting approval from the Institutional Review Board at the regional veteran's facility.

Attached is the abstract for the proposal and the informed consent form. I would appreciate review of the study in order for the students to be ready to proceed with data collection when facility approval is received.

UTPA HUMAN SUBJECTS REVIEW:

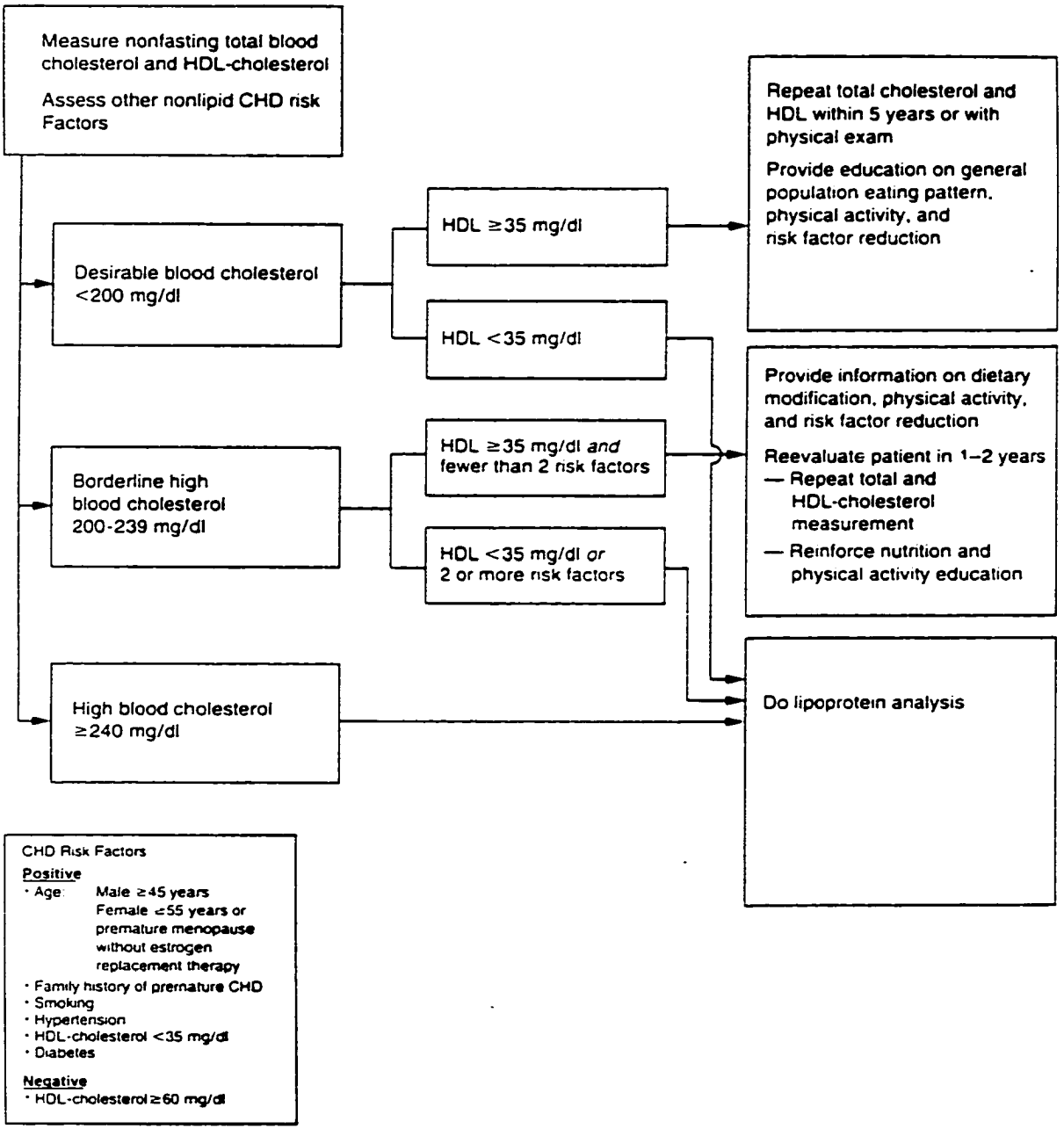
Proposal is ✓ Approved _____ Disapproved

[signature] 7-22-98
Signature Date

Provost
Title

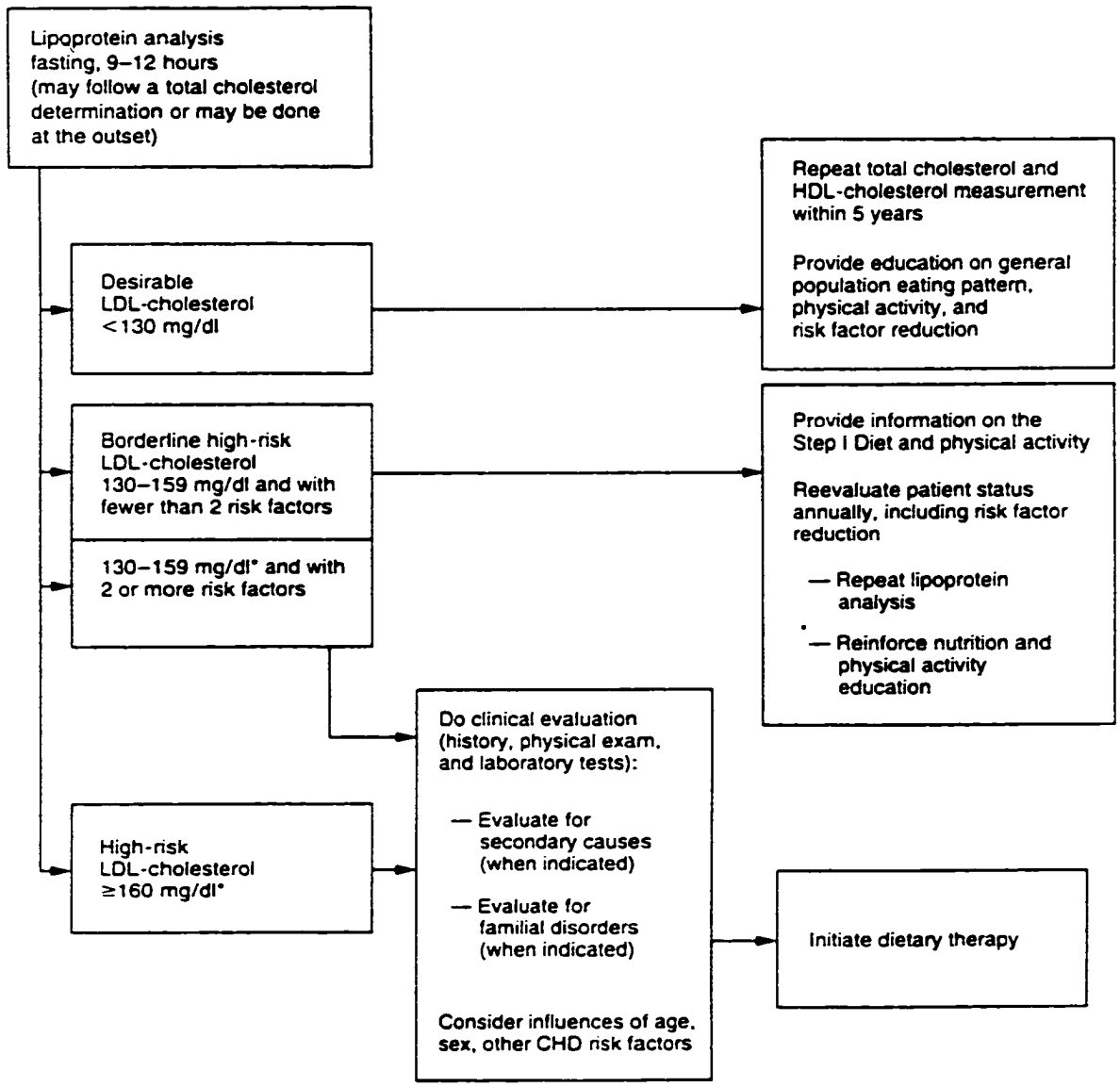
APPENDIX B

CLINICAL ALGORITHMS



Primary prevention algorithm for adults without evidence of CHD: Initial classification based on total cholesterol and HDL-C.

From "National Cholesterol Education Program: Second Report of the Expert Panel on Detection, Evaluation, and Treatment of High Blood Cholesterol in Adults (Adult Treatment Panel II), National Institutes of Health, Publication No. 93-3095, National Heart, Lung, and Blood Institute, 1993," as found in K. L. Mahan and S. Escott-Stump, 1996, Krause's food nutrition and diet therapy (9th ed.), Philadelphia: Saunders, p. 532.



* On the basis of the average of two determinations. If the first two LDL-cholesterol tests differ by more than 30 mg/dl, a third test should be obtained within 1-8 weeks and the average value of three tests used.

Primary prevention algorithm for adults without evidence of CHD: Primary prevention algorithm based on LDL-C.

From "National Cholesterol Education Program: Second Report of the Expert Panel on Detection, Evaluation, and Treatment of High Blood Cholesterol in Adults (Adult Treatment Panel II), National Institutes of Health, Publication No. 93-3095, National Heart, Lung, and Blood Institute, 1993," as found in K. L. Mahan and S. Escott-Stump, 1996, Krause's food nutrition and diet therapy (9th ed.), Philadelphia: Saunders, p. 533.

APPENDIX C
STUDY FORMS

Department of Veterans Affairs

VA RESEARCH CONSENT FORM

Subject Name: _____ **Date** _____

Title of Study: A Comparison of the Effects of Moderate- and High-Carbohydrate Diets on Coronary Heart Disease Risk Factors

Principal Investigator: Margaret Fernandez, RN, BSN **VAMC:** San Antonio, TX

DESCRIPTION OF RESEARCH BY INVESTIGATOR

You are being asked to take part in a pilot research study of diet and coronary heart disease risk factors. We want to learn the effects of two different diets on certain factors that show if a person is at risk for heart disease, specifically lipid levels (total cholesterol, HDL and LDL cholesterol, and triglycerides), weight, and waist measurements. You are being asked to take part in this study because your doctor has determined that you are at risk for heart disease. Your laboratory results indicate that your lipid levels are high enough to require a change in your diet in order to reduce your risk of heart disease, but are not high enough to require more definitive treatment at this time (such as medication, further diagnostic testing, or surgery).

If you decide to take part in this study, you will be assigned to receive instruction in one of the two diets. Both diets have been shown to have some beneficial effects in reducing weight and lipid levels. At your first visit with the dietitian, you will be weighed and measured (height, waist). You will have two visits with the dietitian: the initial visit, and a follow-up visit in four weeks. You will receive phone calls at two, six, and eight weeks to review your progress in following your diet and to answer any questions you may have. The study will be completed at twelve weeks, at which time you will again have blood drawn and be weighed and measured to compare to your initial results.

Discomforts, inconveniences, and other risks that may be reasonably expected are as follows: The discomfort and normal risks of having blood drawn, and the inconvenience of coming to the clinic for an additional visit with the dietitian.

Benefits that may be reasonably expected from participation in this study are that if the diets are followed appropriately, your lipid levels could be lowered, which would reduce your risk of coronary heart disease.

Whether or not you choose to participate, you will receive appropriate care for your condition and be informed of your lab results and the success of your efforts in changing your diet by your physician.

SUBJECT'S IDENTIFICATION (If different from previous form)

As a veteran, your only cost for treatment as a subject in this research study will be the copayment that may be required based on your category of eligibility for medical care.

_____
Signature of Subject

Department of Veterans Affairs

VA RESEARCH CONSENT FORM

Subject Name: _____ **Date** _____

Title of Study: A Comparison of the Effects of Moderate- and High-Carbohydrate Diets on Coronary Heart Disease Risk Factors

Principal Investigator: Margaret Fernandez, RN, BSN **VAMC:** San Antonio, TX

If you are pregnant, you cannot take part in this study.

If you are injured as a result of the research procedures, medical care will be provided. You will be responsible for all charges. We are not able to give you money if you are injured.

Everything we learn about you in this study will be confidential. If we publish the results of the study in a scientific journal or book, you will not be identified in any way.

You consent to the examination or use of your blood sample for research purposes.

Your decision to take part in the study is voluntary. You are free to choose not to take part in the study or to stop taking part at any time. If you choose not to take part or stop at any time, it will not affect your future medical care at the McAllen Outpatient Clinic (VA).

If you have questions now, feel free to ask us. If you have additional questions later or you wish to report a medical problem which may be related to this study, Margaret Fernandez, RN, can be reached at 956-618-7100 (M-F, 8-5), or Julia Soper, RN, can be reached at 956-702-5706 (M-F, 8-4). The University of Texas Health Science Center committee that reviews research on human subjects (Institutional Review Board) will answer any questions about your rights as a research subject (210-567-2351).

You will be given a copy of this form to keep.

YOUR SIGNATURE INDICATES THAT YOU HAVE DECIDED TO TAKE PART IN THIS RESEARCH STUDY AND THAT YOU HAVE READ, HAVE HAD EXPLAINED TO YOU, AND UNDERSTAND THE INFORMATION GIVEN ABOVE.

_____ _____
Signature of Subject Signature of Witness

_____ _____
Signature of Investigator Date/Time

VA FORM NO. 10-1086

Nutritional Care Information

NUTRITIONAL CARE INFORMATION

PATIENT'S NAME: SC NSC | WARD | IDENTIFICATION NO.

PATIENT'S PHYSICIAN | PATIENT'S ADDRESS | BED | AGE | SEX

NEW FOLLOW-UP WALK-IN SCHEDULED

DIAGNOSIS (ES)

PATIENT'S OCCUPATION

CURRENTLY PRESCRIBED MEDICATIONS

Current exercise level
SED. MOD. ACTIVE

PHYSICAL DATA

HEIGHT | PRESENT WEIGHT

IDEAL WEIGHT | RECENT CHANGE

%IBW

MAX. WT. | MIN. WT.

Usual wt. %usual wt.

Frame size:

TRICEPS SKINFOLD ____ MM

MID UPPER ARM CIRCUMFERENCE ____ CM

MID UPPER ARM MUSCLE CIRCUMFERENCE (DERIVED) ____ CM

TRICEPS SKINFOLD AS % OF STANDARD

MID UPPER ARM MUSCLE CIRCUMFERENCE AS % OF STANDARD

PRESENT DIET RX

CURRENT LAB. VALUES:

SELF-PRESCRIBED MEDICATIONS AND DIETARY SUPPLEMENT

Exercise Goal

PREVIOUS DIET COUNSELING

PREVIOUS DIET RX

WEIGHT HISTORY:

KIND OF DIET

RECEIVED INDIVIDUALIZED DIET INSTRUCTION ☐ YES ☐ NO

RECEIVED PRINTED DIET INFORMATION ☐ YES ☐ NO

PHYSICAL HANDICAPS WHICH INFLUENCE FOOD INTAKE (✓)

☐ VISUAL ☐ AUDITORY

☐ MOBILITY ☐ DEXTERITY

☐ DENTAL ☐ OTHER

SELF-IMPOSED DIET REGIMENS

OTHER NUTR. RELATED PROBLEMS

Taste vomiting diarrhea
Smell chewing constipation
Nausea swallowing

INSTRUCTOR

LOCATION

COLLECTION OF DIET INSTRUCTION ☐ GOOD ☐ POOR

FACTORS INFLUENCING FOOD INTAKE

YES	NO	(Items 1 through 13: check yes or no)
		1. Has a good appetite
		2. Resides alone
		3. Prepares own food
		4. Has cooking facilities
		5. Has refrigeration
		6. Has adequate funds for food
		7. Shops in supermarket for food
		8. Buys dietetic/Health foods
		9. Follows 3-meal pattern daily
		10. Eats at normal pace
		11. Is a smoker
		12. Has elimination problems
		13. Has food allergies

(14 through 17: check applicable items and note frequency when indicated)

14. Type of food preparation	Conventional cooking
	Convenience foods
	Snack foods
15. Location of Meals	Home
	Restaurant
	Other
16. Food cravings	Sweets
	Fried
	Salty
17. Food preferences (Check & identify)	Ethnic
	Regional
	Religious

(Use space below for additional information and for comments or explanations about answers to questions 1-17. Reference number of question if comment relates to questions 1-17)

DIET HISTORY/ RECALL

BREAKFAST LUNCH SUPPER SNACKS

Chips -
ETOH-

Milk-
Sodas-
Tea-
KoolAid-
Tortillas-
Lunch Meats-

OP 120-88-1
July 1988

SIGNATURE | TITLE | DATE

VA FORM 10-7979 MAR 1977 SUPERSEDES VA FORM 10-2336, AUG 1970, AND VA FORM 10-7979 (TEST), AUG. 1974, WHICH WILL NOT BE USED

PAGE 1 OF 4

NUTRITIONAL CARE FINDINGS: After reviewing diagnostic procedures, laboratory values, clinical findings, physical impairments, and nutritional care information, summarize pertinent Subjective and Objective findings and make an Assessment of patient's nutritional care needs.

SUBJECTIVE			
Significant changes from previously identified problems			
NEW DIAGNOSES:			
Weight Goal			
OBJECTIVE			
Type diet ordered			
Type diet given			
Food/Drug interact Type Drug			
New Medications			
ASSESSMENT			
Nutrition Status			
Calories required			
Protein required			
Comprehension			
Compliance level			

NUTRITIONAL CARE PLAN: After this is formulated, the plan should always be documented in the patient's medical record. Plan should include:

Dx (Diagnostic) — Such as laboratory work-up, consultations.

Rx (Therapeutic) — Such as diet modifications and adjustments, recommendations for improving dietary habits, goals in relation to nutritional problems, referrals to other health care team members.

Pt. Ed. (Patient Education) — Such as diet instruction (individual and group) including family member and/or caretaker, handout materials to be given, and follow-up visits for continuing care.

PLAN			
DX:			
Weight			
Recent Wt. Change			
Glucose			
Cholesterol			
Triglycerides			
Albumin			
RX: HgB			
HCT			
GOALS SET w/pt.			
Return Appt.			
PT. ED.			
Food/Drug Ed.			
Smoke Ed.			
Exercise Ed.			
Other Ed.(specify)			
COLLATERAL VISIT(S) Name: Birthday: SS No.			

SIGNATURE (Dietitian)	TITLE	DATE

Data Collection Worksheet

DIET GROUP: STEP I ZONE

DEMOGRAPHIC DATA

Medical record no: _____
Name: _____
Gender: M F Age: _____
Race: _____
Contact phone no: _____
Consent signed? _____

HISTORY

Medical hx: _____
_____
_____
Current medications: _____
_____
Smoking? _____

Initial lipid profile: date: _____
chol: _____
tri: _____
LDLC: _____
HDLC: _____

Final lipid profile: date: _____
chol: _____
tri: _____
LDLC: _____
HDLC: _____

Initial diet visit: date: _____
Ht (in.): _____
Wt (lb): _____
waist (in.): _____
WHR (calculated): _____
BMI (kg/m2): _____
Activity level: sedentary mod. active active very active

Final visit: date: _____
Ht (in.): _____
Wt (lb): _____
waist (in.): _____
WHR (calculated): _____
BMI (kg/m2): _____
Activity level: sedentary mod. active active very active

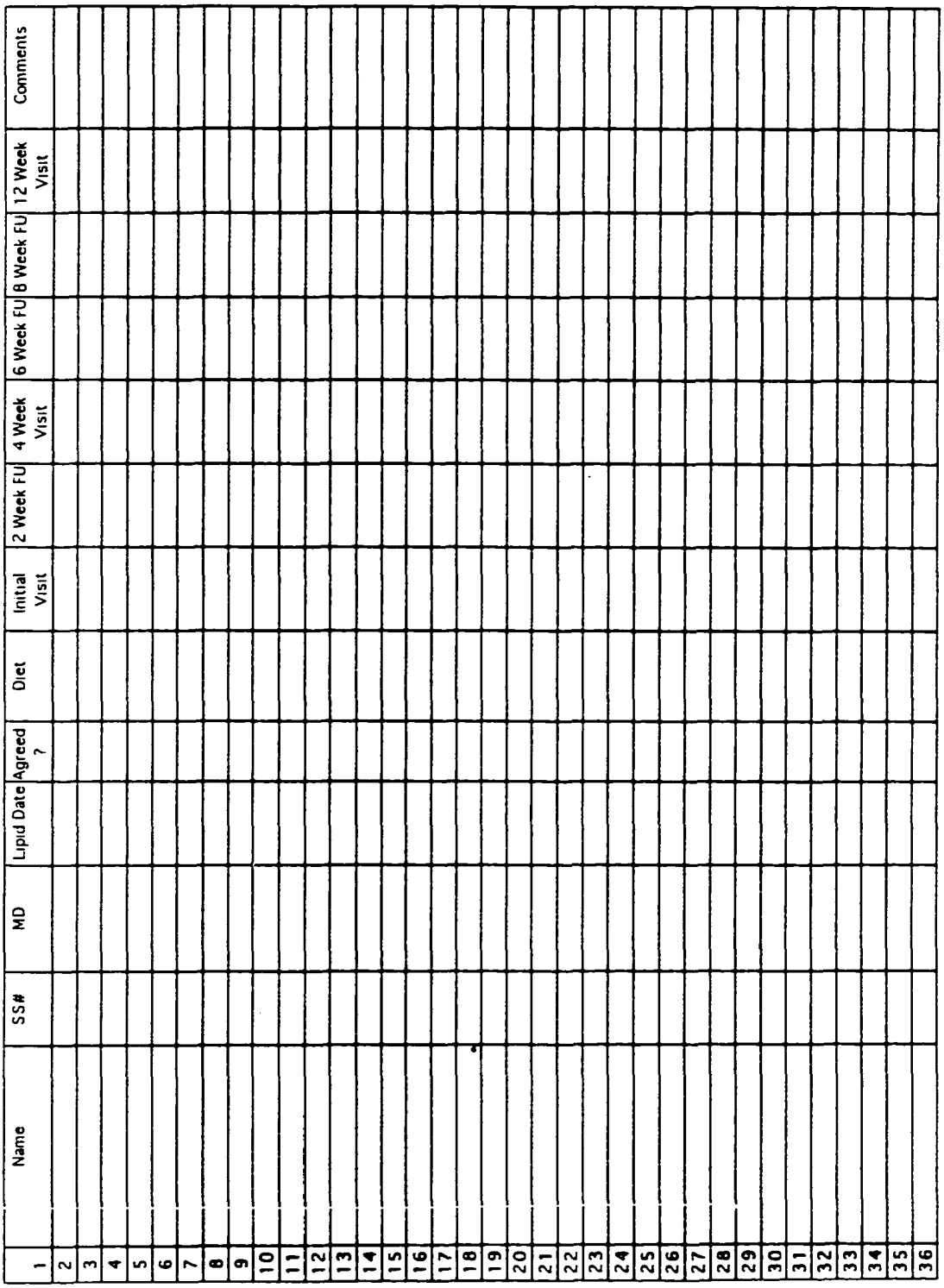
2nd wk phone call: date/initials: _____ 24 hr recall done? _____
compliance with diet: poor fair good excellent
comments: _____

4th wk diet appt: date: _____
compliance with diet: poor fair good excellent
comments: _____

6th wk phone call: date/initials: _____ 24 hr recall done? _____
compliance with diet: poor fair good excellent
comments: _____

8th wk phone call: date/initials: _____ 24 hr recall done? _____
compliance with diet: poor fair good excellent
comments: _____

Flowsheet



	Name	SS#	MD	Lipid Date	Agreed ?	Diet	Initial Visit	2 Week FU	4 Week Visit	6 Week FU	8 Week FU	12 Week Visit	Comments
1													
2													
3													
4													
5													
6													
7													
8													
9													
10													
11													
12													
13													
14													
15													
16													
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25													
26													
27													
28													
29													
30													
31													
32													
33													
34													
35													
36													

VITAE

Margaret H. Fernandez

Higher Education Institutions:

San Antonio College	May 1986 - Aug 1987
University of Texas Health Science Center - San Antonio	Sep 1987 - May 1989
University of Texas - Pan American	Jun 1995 - Dec 1998

Degrees Awarded:

Bachelor of Science in Nursing, University of Texas Health Science Center - San Antonio, 1989

Major: Nursing

Current Academic Program:

Master of Science in Nursing

Major: Adult Health Nursing with a focus on Health Promotion

Professional Experience:

Villa Rosa Psychiatric Hospital	May 1989 - Apr 1991
Southwest Texas Methodist Hospital	May 1991 - May 1992
Edinburg Hospital	Jul 1992 - Oct 1993
South Texas High School for Health Professions	Aug 1992 - Aug 1993
McAllen Kidney Center	Oct 1993 - Feb 1995
Veteran's Administration Outpatient Clinic	Feb 1995 - current

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Julia Ava Niehaus Soper

Higher Education Institutions:

University of Texas Health Science Center - San Antonio	Aug 1994 - Aug 1995
University of Texas - Pan American	Jul 1980 - Dec 1998

Degrees Awarded:

Associate of Applied Sciences, Pan American University, 1982
Major: Nursing Education

Bachelor of Science in Nursing, University of Texas - Pan American, 1987
Major: Nursing

Current Academic Program:

Master of Science in Nursing
Major: Adult Health Nursing with a focus on Alternative/ Complementary Therapies

Professional Experience:

Edinburg Hospital	Aug 1982 - May 1991
Pharr-San Juan-Alamo ISD/ Sorensen Elementary	Aug 1991 - current

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