CARDIOVASCULAR RISKS OF CAUCASIAN AND AFRICAN-AMERICAN
WOMEN AND CHANGE WITH INTERVENTION

by

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(ABSTRACT)

The study was conducted regarding the prevalence of risks for cardiovascular disease (CVD) among 150 Caucasian and African-American, low-income women and the effectiveness of a six-month intervention in reducing risks. Seventy-four and 76 participants were randomly assigned to the experimental and control groups, respectively. Intervention consisted of 18 lessons taught by EFNEP paraprofessionals. A family record, three random-repeat 24-hour food recalls, and a health risk appraisal were collected at pre- and post-intervention sessions. Lipid profile, height, weight, percent body fat, and body mass index were measured on a sub-sample of 75 subjects. Descriptive statistics, two-sample t-tests and ANOVA (P<0.05) were calculated. Results suggest that African-American and Caucasian, low-income women have high risks for CVD due to excessive intakes of total fat, saturated fats, sodium, and fats and sweets, but have low-intakes of dietary fiber, calcium, milk, vegetables, and fruits. They also
had high incidences of obesity and smoking and low levels of physical activity. The intervention was successful in reducing intakes of energy and fats, sweets, and increasing intakes of dietary fiber, vegetable, and fruits. No significant change occurred with lipid profiles, obesity, and smoking. A six-month education program resulted in significant dietary improvement, but interventions of longer duration, specifically targeting obesity, physical activity, and smoking, are needed to improve those risk factors.
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CHAPTER 1
INTRODUCTION

Statement of the Problem

Results of national health surveys indicate that cardiovascular disease (CVD) continues to be the leading cause of death for both male and female adults in the United States (1-3) and in the Commonwealth of Virginia (4-6). In 1991, heart disease was the cause of over 40% (20,748) of the state’s 47,557 deaths (8).

Certain high-risk population groups experience an above-average incidence of disability and death as a consequence of CVD. These groups include women with little formal education, the unemployed, those living at or below poverty level, and members of racial minority groups, particularly African-Americans (1,7-11). A large majority of those living in poverty do not have the health insurance nor the means to pay for diagnosis and treatment of health problems, especially CVD. This group may be less knowledgeable of the early warning signs of heart and blood vessel diseases and may not receive medical help in the early stages of disease. In fact, the risk of death from heart disease is more than 25% higher for low-income people than for the overall population (9). Studies have shown that differences in socioeconomic status (SES) lead to low access and utilization of health care services and low participation in nutrition education programs, which would contribute to morbidity and mortality from CVD (1,9,12-13).
Atherosclerotic CVD and associated risk factors have been studied much less among women than among men in the American society. According to medical experts, many women are dying needlessly from CVD, specifically coronary heart disease (CHD) due to discriminatory management by physicians who fail to consider CVD as a diagnosis (2). The American Heart Association (AHA) reported that 478,179 women died from CVD in 1990 and that 39% of women who have heart attacks die within a year as compared to 31% of men.

The 1990 census data of the Commonwealth of Virginia showed that 25% of the total population was composed of females, aged 15-44 years, with 20% and 5% being composed of white and black women, respectively. Fifty-two percent of the 20,058 total deaths reported in the Commonwealth of Virginia in 1991 were due to CVD among females, with 79% being among Caucasian women and 21% being among African-American women (Virginia Department of Health, March 16, 1993). Thus, a disproportionate number of deaths were among African-American women. For this reason, it is vital that investigators focus more research on CVD among women, especially among African-Americans and limited-income Americans.

Five of the ten leading causes of death have been associated with diet, including heart disease, some types of cancer, cerebrovascular diseases, atherosclerosis, and diabetes (1). The Committee on Diet and Health (14) reported that the amount of total fat (TF) in the diet and the type
of fat, including saturated fatty acids (SFA), monounsaturated fatty acids (MUFA), and polyunsaturated fatty acids (PUFA), could greatly influence the development of atherosclerotic CVD. Research findings indicate that excessive sodium intake and inadequate intakes of potassium, calcium, and dietary fiber are positively related to risk for CVD (14).

In several states (15-17), dietary studies of low-income women participating in the Expanded Food and Nutrition Education Program (EFNEP) revealed that homemakers have inadequate intakes of vegetables, fruits, milk, breads, and cereals at entry into the program, but have higher-than-recommended intakes of meat. Such diets have been associated with elevated blood levels of cholesterol, high blood pressure, and obesity, which subsequently increase the severity of CVD with advancing age (6,14). Sedentary behavior and cigarette smoking are some other lifestyle factors that increase the risk of CVD (6). To decrease risks for CVD and other chronic diseases, many health authorities recommend that major educational efforts be made to encourage women to make healthy food choices, achieve and maintain normal blood cholesterol, increase physical activity, reduce body fat, control hypertension, and stop smoking (6,14,18).

One of the primary goals of the 1992-2000 Comprehensive Prevention Plan for Virginia is to promote the health of the people (19). An important aspect of preventive services is the development of effective nutrition education programs, which is the goal of the current study.
The present study was conducted to assess the prevalence of certain risk factors for CVD among Caucasian and African-American, low-income, women enrolled in the Virginia EFNEP and to determine whether a community-based, nutrition education program conducted by EFNEP para-professionals, would result in positive changes of certain diet-related and lifestyle risk factors for CVD. The objectives of the study were:

1. To identify and describe pre-intervention risks for CVD of Caucasian and African-American women enrolled in the EFNEP of Virginia.

2. To determine whether Caucasian and African-American women differ on risk factors for CVD.

3. To determine if certain dietary-related risks for CVD [including excess energy (kcal), TF (% kcal), SFA (% kcal), cholesterol, sodium, the Meat Group, and the Fats/Sweets Group of the Food Guide Pyramid] would be significantly reduced among participants as a result of a six-month nutrition education program.

4. To determine if certain dietary components believed to decrease risk for CVD [MUFA (% kcal), PUFA (% kcal), dietary fiber, potassium, calcium, the Milk Group (including yogurt and cheese), the Vegetable Group, the Fruit Group, and the Bread/Cereal Group (including cereal, rice and pasta) of the Food Guide Pyramid] can be significantly increased among participants as a result of a six-month nutrition education program.
5. To determine if certain measures of obesity (percent body fat and body mass index [BMI]), selected biochemical factors (serum total cholesterol (TC), low-density lipoprotein cholesterol (LDL-C), triglycerides, high-density lipoprotein cholesterol (HDL-C), TC:HDL-C ratio), and smoking habits will improve among participants as a result of a six-month nutrition education program.

Null Hypotheses

$H_1$ At pre-intervention, Caucasian and African-American women will not differ significantly on intake of certain dietary factors (energy, total fat (% kcal), SFA (% kcal), MUFA (% kcal), PUFA (% kcal), cholesterol, dietary fiber, sodium, potassium, calcium, and food groups), measures of obesity (percent body fat and BMI), selected biochemical measures (TC, LDL-C, HDL-C, triglycerides, and TC:HDL-C ratio), and smoking habits at pre-intervention.

$H_2$ The experimental group will not show significantly more improvement than the control group by race, on certain dietary factors (energy, total fat (% kcal), SFA (% kcal), MUFA (% kcal), PUFA (% kcal), cholesterol, dietary fiber, sodium, potassium, calcium, and food groups) as a result of participating in the intervention program.

$H_3$ The experimental group will not show significantly more improvement than the control group, by race, on measures of obesity (percent body fat and BMI) as a result of participating in the intervention program.
$H_4$ The experimental group will not show significantly more improvement than the control group, by race, on selected biochemical measures (TC, LDL-C, triglycerides, HDL-C, and TC:HDL-C ratio) as a result of participating in the intervention program.

$H_5$ The experimental group will not show significantly more improvement than the control group, by race, on smoking (i.e. cessation of smoking or reduction in the number of cigarettes smoked per day) as a result of participating in the intervention program.

**Definition of Terms**

**Demographic Factors:** Demographic factors refer to statistical and quantitative data on characteristics of the sample, including age, race, education, income, and current work status.

**Diet:** Diet comprised all foods and beverages consumed by each individual homemaker in 24 hours, as collected by three random-repeat 24-hour food recalls at pre- and post-intervention. The three days were entered into the Nutritionist III computer analysis program (Ver. 7.0, N-Squared Computing, Salem, Oregon, 97302) and averaged together. Diet components will be reported as average daily intakes of selected nutrients, dietary fiber, and food groups based on the Food Guide Pyramid.

**EFNEP Paraprofessionals:** EFNEP paraprofessionals are non-college graduates who are trained and supervised by Extension Agents and who provide direct teaching to homemakers.
Smokers: Smokers were defined as someone who had smoked at least 100 cigarettes or 5 packs in her lifetime.

Current smokers: Participants who were smoking prior to the study and who continued to smoke regularly throughout the study.

Recent smokers: Participants who had never smoked in their life and/or at the beginning of the study, but began to smoke sometime during the study.

Reverted smokers: Participants who had smoked in the past, but had quit before the study began; however, they resumed smoking during the study.

Ex-Smokers: Participants who formerly smoked, but had quit before the beginning of the study and did not smoke anytime during the study.

Recent ex-smokers: Participants who used to smoke before the study, and were smoking at the beginning of the study, but quit smoking during the study.

HDL-C: High-density lipoprotein cholesterol is the blood lipid fraction which is the smallest and most protein-dense and which contains the least amount of lipid. High HDL-C values are associated with reduced risk of CVD. An acceptable level of this type of blood cholesterol is 35 mg/dl or higher. HDL-C was measured in this study by blood lipid analyses.
**Homemaker:** A homemaker is an adult female in the EFNEP family who has major responsibility for planning and preparing the family’s food. Only females were included in this study, as there are gender differences in blood lipid levels.

**LDL-C:** Low-density lipoprotein-cholesterol contains 75% of the total plasma cholesterol. High LDL-C levels are associated with increased risk of CVD. Serum LDL-C levels of 130 mg/dl or higher are considered to be elevated.

**Low-income:** For the purposes of this study, those eligible for EFNEP were also eligible for the study. Eligibility for EFNEP is based on having a family income of 125% of poverty or lower and having children in the home, or having a pregnant homemaker. Criteria used to classify low-income families are as follows:

- Individuals who are eligible for USDA food assistance programs, or receiving public assistance; unemployed families with a low level of formal schooling; families living in housing that is in need of extensive repair or replacement; one wage-earner with a relatively large number of children, and/or elderly dependents; employed family members with a large household to support.

**Obesity:** Obesity is a condition of having excessive high body fat in relation to lean muscle mass. For purposes of this study, a woman was considered obese if she had a BMI above 27.1 kg/m² and above and/or a percent body fat level above 31%.
**Overweight**: Overweight refers to a condition in which there is body weight of 20% or more above the recommended weight for height, based on the Metropolitan Life Insurance Tables. Over-weightiness due to excess body fat, not lean muscle tissue, is a disease risk factor.

**Percent Body Fat**: Percent body fat is a physiological measurement of body fat by the Futrex 5000 Fitness and Body Composition Analyzer. This instrument uses near-infrared technology and was developed by the USDA General Research Laboratory, Beltsville, Maryland.

**Risk Reduction**: Risk reduction includes change in factors that contribute to morbidity and mortality from CVD. Selected demographic factors, dietary factors, measures of obesity, blood lipids, and smoking were investigated in the current study.

**Twenty-four (24) Hour Food Recall**: The 24-hour food recall is a list of all foods and beverages consumed by the homemaker based on memory. It also includes an accurate recall of the amounts of each food consumed and methods of preparation.

**Importance of the Study**

In Virginia, 40% of all deaths are directly attributable to unhealthy behaviors such as smoking, poor eating habits, lack of exercise, use of alcohol and other drugs, and failure to practice good safety habits (19). According to a report of the Virginia Council on Coordinating Prevention, if only 10% of Virginians would change their health behaviors related to
smoking, weight, exercise, and high blood pressure, approximately 1400 fewer people would die per year from CVD (19).

Because of the high prevalence of diet-related risks for CVD, effective educational programs must be developed to target low-income, low-literacy, and female adults to encourage them to make appropriate dietary and lifestyle changes. Dietary intervention, through counseling and teaching of homemakers and their families, is imperative to reduce risks for CVD. Dietary improvements may decrease medical costs and, more importantly, reduce morbidity and mortality from CVD. EFNEP has had documented success in bringing about basic dietary improvements among its participants and it is hoped that this program will be a means of reducing risks for CVD among its clientele.

Limitations of the Study

Several limitations are noted in this study, as follows:

1. No attempt was made to evaluate the effectiveness of the nutrition intervention on actually reducing heart and blood vessel disease, but rather on reducing risk factors.

2. The study was limited to Caucasian and African-American, non-pregnant, female homemakers of child-bearing age, who met the criteria for EFNEP enrollment. Participants were selected from three rural counties and one city in Virginia, who were participating in the EFNEP Nutrition-Based Cancer Prevention Project. Thus, results can
not be generalized to all EFNEP homemakers, nor all low-income women.

3. The study did not include actual observance of the food intake of participants. Instead, food intake and changes in dietary components were assessed through collection and analyses of three random-repeat 24-hour food recalls. Though the recalls were collected over a three-week period at pre- and post-intervention, they may not have been representative of the usual, year-round intake of participants.
CHAPTER II

REVIEW OF RELATED LITERATURE

The Background of the Problem

At the turn of the century, a change in the basic mortality patterns marked the rise to prominence of chronic diseases as the primary cause of death in the United States (U.S.), instead of infectious diseases. More than three-fourths of all deaths in the U.S. are caused by chronic diseases (20). Among the chronic diseases, cardiovascular disease (CVD) is the leading cause of death, which accounts for one-half of all deaths, while malignant neoplasms rank second and account for almost one-third of deaths nationwide (1,9,14).

Atherosclerotic CVD, primarily coronary heart disease (CHD), stroke, and peripheral arterial disease (PAD), make-up the largest group of diet-related heart and vascular diseases in the U.S. and have the greatest effect on morbidity and mortality. In 1985, it was estimated that 65 million Americans had some form of heart or vascular disease with the cost of CVD approaching $84 billion (21). These data clearly document the adverse impact of CVD on the health and well-being of Americans.

Through advances in treatment and early intervention, reductions in CVD mortality were achieved in the past 10 years. However, atherosclerotic CVD continues to affect approximately 7 million Americans, causing 500,000 deaths annually, and costing the nation an estimated $43 billion
per year in direct and indirect costs (9). Another 1,250,000 individuals
suffer nonfatal heart attacks annually. About 20% who suffer fatal heart
attacks are between 25 and 65 years of age, with 13% of the 150,000
Americans who died from stroke in 1986 being in this age group. The
etiology of atherosclerosis is believed to be multifactorial. Most of the
evidence points to an association between the disease process and the
presence of several lifestyle risk factors.

The pathogenesis of atherosclerosis involves the development of
fibrous, fatty deposits called plaques or atheromas. Intima of large and
medium-sized arteries as well as elastic and muscular arteries are the
commonly affected areas of atheromas. Although early lesions of athero-
sclerosis are asymptomatic, the disease process begins as early as child-
hood and becomes extensive in adolescence. Deposition of lipids, principally
cholesterol and its esters, in macrophages and smooth muscle cells are the
major components of a lesion. Subsequent accumulation of lipid and
necrotic debris causes atheromas to eventually become calcified and eleva-
ted, projecting into the lumen of arteries which results in narrowing of the
opening, thickening of arterial walls and loss of elasticity. Atheromas have a
tendency to occur at points of arterial branching and around openings of
primary branches of the aorta. Consequently, lesions interfere with blood
circulation and cause systemic effects, which vary with the site of the
lesion. For example, lesions in coronary arteries may result in CHD, leading
to coronary occlusion (myocardial infarction), which is the most common and serious manifestation of atherosclerotic CVD in middle-aged adults (14). Lesions in the cerebral arteries may lead to cerebrovascular accident (CVA), commonly known as stroke (14).

Genetic predisposition, gender, advancing age, diabetes, positive family history of CVD before the age of 55, and circulation disorders of blood to the legs, arms, and brain are risk factors over which individuals have little control (14,22). In addition certain high-risk population groups such as low-income and low-literacy women and members of ethnic or racial minority groups (particularly African-Americans) experience above-average incidences of disability and death as a consequence of CVD when compared to the total population. CVD is the leading cause of death for African-Americans, as with the total population (1,9,13).

African-Americans compose 12% of the United States population, thereby, constituting the nations largest minority group. However, one-third of African-Americans live in poverty, which is three times the rate of the Caucasian population. Over one-half live in neighborhoods of central cities which are typified by poverty, poor schools, crowded housing, unemployment, exposure to pervasive drug cultures and street violence, and generally high levels of stress. Between 1985 and 1991, life expectancy for white females increased by 0.9 of a year to 79.6 years; while among black females it increased by only 0.6 of a year to an expectancy of 73.8 years (1).
With regard to gender differences, recent health surveys suggest that just as many women as men die from CVD (23). In a review of data from health surveys in the U.S. and Netherlands, Gijsbers et al. (24) reported that CVD and high blood pressure were diagnosed more frequently in women and at an earlier stage than in men. In the review of symptom sensitivity and sex differences (24), cardiovascular conditions were reported in men at a later stage, especially for ischemic heart disease (IHD), and manifestations of the disease were more severe than among young women. Women have higher morbidity rates than men based on reported symptoms of illnesses and use of medical care facilities; however, their mortality is significantly lower than that of men (23-25).

Unfortunately, few studies on CVD have included women as subjects. However, combined data in several epidemiological investigations suggest that risk factors predisposing men to CVD have the same effect on women, with the exception of a few risk factors which have different prevalence levels among men and women. Among females, there are fewer smokers and higher levels of high-density lipoprotein cholesterol (HDL-C), but levels of low-density lipoprotein cholesterol (LDL-C) are lower among men (23,25). Thus, it would appear that strategies that reduce risks for CVD in men would also reduce risks of CVD in women. Since CVD is common among women, as reflected by morbidity indices (23,25), one might wonder why women have been overlooked by medical research. Fortunately, as medical therapy
has advanced, there has been increased awareness of the need for disease prevention among women and research on risk factors for CVD among women has blossomed.

Over the past 30 years, great progress has been made in identifying modifiable risk factors of CVD and in developing and implementing measures to control or even prevent the risk factors. The National Heart, Lung, and Blood Institute in 1990 (18), reported that the preventable risk factors for CVD are cigarette smoking, high blood pressure, and elevated serum total cholesterol (TC) levels.

Epidemiological evidence shows that cigarette smoking is the most significant risk factor followed by hypertension and hypercholesterolemia, but risk factors are not limited to these. Sedentary lifestyle, elevated blood levels of TC (>200 mg/dl), LDL-C (>130 mg/dl), triglycerides (>200 mg/dl), and low levels of HDL-C (<35 mg/dl) are factors which increase risk for CVD. Obesity, a risk factor for CVD and cancer, affects 30% of middle-aged women, 15% of middle-aged men, and 25% of adolescents with the highest rates observed among the poor and minority groups (6,9). Health risks from obesity appear to be mediated chiefly through metabolic consequences: glucose intolerance and diabetes mellitus (DM), hypertension, "low" levels of HDL-C, and "high" levels of LDL-C and very low density lipoproteins (VLDL) (18). Any combination of these risk factors is more potent than the sum of their individual effects.
Based on this information, it is believed that a number of risk factors for CVD can be prevented. In an effort to reduce the incidence of atherosclerotic CVD, many national and state health organizations emphasize improved nutrition through health promotion, education, and prevention. The Department of Health and Human Services Healthy People 2000 objectives recommend that investments be made in prevention of CVD and that health agencies and professionals collaborate in this effort. Diet, maintenance of an ideal weight, exercise, achievement of desirable blood lipid levels, and smoking cessation are the primary ways of reducing risk of CVD.

Dietary change is recognized as the cornerstone of management by the National Cholesterol and Education Program (NCEP), with dietary factors considered to be the most easily modified of all risk factors for CVD. Chief among dietary risk factors are disproportionate consumption of foods high in total fat (TF), particularly saturated fatty acids (SFA). Current recommendations included in the Dietary Guidelines for Americans (14,26) are: (a) eat a variety of foods; (b) maintain healthy weight; (c) choose a diet low in TF, SFA, and cholesterol; (d) choose a diet with plenty of vegetables, fruits, and grain products; (e) use sugar in moderation; (f) use salt and sodium only in moderation; and (g) if alcoholic beverages are consumed, do so in moderation. These recommendations were based on the best available evidence relating dietary factors to CVD morbidity and mortality (21).
However, effective dietary intervention does not rely on the "good food, bad food" approach, but rather on modification of overall eating habits and lifestyle factors. Modifying diet to attain reduction of risk will require effective nutrition intervention programs, which are community and family-based and which target high-risk groups.

There has been a significant decrease in the incidence of CVD in the past 15 years, including an overall reduction of 35%, a 40% reduction for CHD, and a 15% or greater reduction for stroke (9). This progress reflects improvement in lifestyle habits and preventive care, including change in diets, increasing awareness of the role of dietary fats on blood lipids, decline in cigarette smoking, and increased detection and control of high blood pressure. Thus, prevention holds the greatest potential for reducing CVD morbidity, disability, and mortality.

Background on EFNEP

The Expanded Food and Nutrition Education Program (EFNEP) is a nationwide nutrition education program for low-income families and youth. It was initiated in 1968 by the U.S. Department of Agriculture and is implemented by the Cooperative Extension Service in all 50 states and several U.S. territories. Its goal is to help low-income families and youth to acquire knowledge, skills, attitudes, and changed behavior necessary for securing nutritionally sound diets and to contribute to their personal development (27).
EFNEP participants are taught food and nutrition lessons by paraprofessionals who are trained and supervised by Extension home economists. Homemakers in the families are given direct instruction by EFNEP paraprofessionals, in home visits or small group meetings. A relaxed conversational style of teaching is used and homemakers are involved in hands-on learning experiences and food preparation activities.

Although numerous studies have focused on the impact of EFNEP in improving the dietary practices of the disadvantaged, there has been no research to date describing the risks for CVD of EFNEP homemakers and whether the program helps to reduce dietary risks of CVD. It was believed that dietary risks for CVD should be emphasized in EFNEP teaching, as research clearly indicates that changes in certain dietary factors and lifestyle habits can reduce the risk of atherosclerotic CVD and improve quality of life.

EFNEP was initiated in 1969 in 23 counties and cities in Virginia and, over the past 25 years, the program has operated in 50 different counties and cities. At the time of this study, there were 27 Extension units conducting an adult program, with 10 being in urban and 17 being in rural areas (5). The Virginia EFNEP is administered from Virginia Polytechnic Institute and State University (VPI & SU).

In the fiscal year 1992 when this study was conducted, the Virginia EFNEP worked with 5,905 enrolled homemakers (5). A large percentage of the families come from rural areas with 69% (4,073) being from minority
groups. At present, there are 54 full-time trained paraprofessionals who have responsibility for enrolling and teaching homemakers. The EFNEP curriculum in Virginia is composed of the Eating Right Is Basic Series (ERIB-2, 28) and lessons provided by the state EFNEP staff. Lessons cover basic concepts in nutrition, meal planning, the Food Guide Pyramid, food preparation, food buying and safety, nutrition during pregnancy and lactation, and feeding infants and children (5-6, 27). To accomplish its goals, the Virginia EFNEP collaborates with other agencies in the state, such as local and district health departments, the Women, Infant, and Children Program (WIC), the Department of Social Services, Mental Health Agencies, and others.

Throughout its history, the main focus of EFNEP has been basic nutrition, with emphasis on consuming the recommended servings of the Daily Food Guide (29) and key nutrients. However, in recent years there has been a growing emphasis on chronic disease prevention. Cancer prevention has been emphasized in recent years through a special research project (30).

**The Risk Factors for Cardiovascular Disease Among Women**

Risk factors are characteristics believed to be associated with onset, survivorship, and mortality related to a disease condition (31). Among women, several factors interact to determine individual risk for CVD. Evidence suggests that the most prominent of these are age, sex, race, socioeconomic status (SES), diet, blood lipids, blood pressure, and smoking. Numerous other factors may contribute to risks of CVD including lack of
regular physical activity, obesity, and fat distribution at various sites on the body. Nevertheless, little has been done to provide clear reasons for the observed differences in risks for CVD between African-American and Caucasian women.

**Demographic Factors**

Demographic factors are known to be related to risks for CVD in men including race, age, and SES. However, very few studies have studied the same relationship in women. The effects of demographic factors are discussed below.

**Race.** It has been obvious for sometime that health status, in general, differs for Caucasian and African-American adult groups, especially among women, in the U.S.. Patterns of mortality at all ages and overall longevity are sharply differentiated by race, indicating that morbidity patterns are also different.

From 1989 to 1991, the age-adjusted mortality rate for heart disease was nearly 60% higher for African-American females than for Caucasians, ages 45 and older (1). Recent findings have indicated a 40% decline in mortality rate from CHD among all population groups, but, the decline has slowed for Caucasian and African-American women. Death rates for black women remained higher than for white women (9). The Virginia Department of Health (Virginia, Department of Health, March 16, 1993) reported that, of the 20,058 deaths due to CVD in Virginia, 52% (10,340 individuals) were women, with African-American females accounting for 21% (2,136 indivi-
duals) of the deaths in 1991. African-American women composed only 5% of the total female population as compared to 20% of Caucasian women in the Commonwealth of Virginia in 1991.

In a study by Kumanyika (32) which described minority health issues related to diet and chronic diseases, African-Amercians were shown to have notably higher-than-average rates of nearly all types of diet-related chronic diseases (hypertension, obesity, and elevated serum TC levels) which contribute to higher overall CVD mortality rates. Although, there are differences in the incidence of specific diseases by race, the health problems suffered by African-American’s are generally representative of those among poor and minority groups in the U.S. (13). Many of the health problems faced by African-Americans are associated with poor nutrition, smoking, failure to get preventive care, and low SES.

**Socioeconomic Status.** SES is determined by education, income, and occupation. Few studies have investigated the relationship between SES and CVD risk in women, and their findings have been inconsistent. Generally, participants that were better educated and who were employed with higher incomes, were non-smokers and had healthier eating patterns, lower serum TC levels, leaner body mass indices, and higher physical activity levels than less educated participants. In a comprehensive study by Winkleby et al. (8), 2,380 participants (85% of which were white, non-Hispanic, aged 25-64 years) from the Stanford 5 City Project were examined to establish associa-
tions between income, occupation, and education and a set of risk factors for CVD (smoking, blood pressure, TC and HDL-C levels). Overall results revealed positive correlations between all three SES dimensions and HDL-C level; however, smoking, blood pressure, and serum TC levels were shown to be adversely related to HDL-C levels (8).

This positive association between HDL-C levels and SES indicators (income, poverty index, and education) for Caucasians of both genders, was also reported in the Lipid Research Clinics Prevalence Study (LRCPS) and Multiple Risk Factor Intervention Trial (MRFIT, 14). Comparative data in the study by Winkleby et al. (8) on gender showed that women have fewer years of education than men and more frequently live in low-income households. Approximately, 75% of women employed outside the home hold non-professional jobs. Similarly, Croft et al. (34) showed an inverse relationship between SES and BMI in African-American women. These relationships are consistent with those observed for surrogate measures of obesity in national and cross-sectional surveys.

Kritz-Silverstein et al. (12) found that employed women had lower mean BMI’s and waist-to-hip-ratios (WHR), consumed less alcohol, less likely to smoke cigarettes, and more likely to exercise than unemployed women. The results of this study were in accord with several studies (35) that demonstrated a greater tendency for unemployed than employed female homemakers to report more illnesses of CVD.
An analysis done by Linn et al. (33), using the Second National Health and Nutrition Examination Survey (NHANES II) data, revealed an inconsistent pattern between education and mean HDL-C levels for African-American women. In addition, no significant differences in age-adjusted mean HDL-C levels were noted between African-American women living below poverty and those living at or above poverty; however, HDL-C levels among African-American women remained within 56-58 mg/dl range, for all categories of poverty index. Similarly, in the Framingham Offspring Study (10), all CVD risk factors [physical inactivity, obesity, cigarette smoking, and elevated systolic blood pressure (SBP)] were most severe in female study participants with the least amount of education.

Age. CVD among females has previously been thought of as a disease of "old women" (23). This erroneous belief may account for the lack of attention given to the study of primary prevention of CVD among young and middle-aged women, in spite of their vulnerability to CVD. Results from the Framingham study (25) showed males, aged 35 to 84 years, having twice the incidence of CVD morbidity and mortality as females. On the other hand, CHD data on morbidity rates among females reveal a 40-fold increase between the younger age group (35-44 years) and the oldest age stratum (75-84 years). The largest gain in morbidity was seen in women between the ages of 45 and 64 years (25). Moreover, data on mortality rates in the Framingham Study suggest that, despite the low incidence of CHD in women
in the 35 to 44 age range, many women in this group die of clinically recognized myocardial infarction, emphasizing the seriousness of coronary heart attacks in younger women. This observation was confirmed by national statistics from 1991 (1) which indicate a 3 to 4-fold increase in death rates from heart disease among white and black females between the ages of 25 and 54 years.

According to Miller et al. (36), lipid and lipoprotein levels are similar in males and females until puberty, after which there are changes in circulating gonadal hormones, higher levels of HDL-C, and lower levels of LDL-C among women which reduce their risk for CVD. In one review of CVD in women, estrogen was shown to favorably affect lipoprotein levels and arterial walls, which may explain the acceleration of CVD incidence in women after menopause (37-38). In a review of NHANES II data on HDL-C levels in women, Linn et al. (33) found a general trend, by race, of decreasing HDL-C levels for females between the ages of 20 and 44 years. At 20-24 years, the mean HDL-C values were at the highest level and were 52.8 mg/dl for Caucasian women and 56.6 mg/dl for African-American women. HDL-C levels declined in women of both races after the ages 55-64 years.

Based on these findings, CVD in old age is a result of lifelong exposure to conditions that contribute to atherosclerosis and intervention programs should be established to make women aware of their possible predisposition to CVD and to encourage early prevention methods.
in summary, the major risk factors for CVD (cigarette smoking, elevated TC levels, and high blood pressure) have similar adverse effects in women as in men. It appears that some of the association between race and CVD risk are a function of SES, especially of education. Several studies showed a significant association between education and certain risk factors for CVD, even after adjustment for income and occupation. A higher educational level may have a positive indirect effect on health due to employment status, as better educated women are more likely to be employed. Employed women reap more of the practical and psychological benefits of health care than less educated and unemployed women.

**Dietary Factors**

**Energy.** Since the first decades of this century, there has been a change in the distribution of food energy among carbohydrates, proteins, and fats in the diets of Americans. Of the macro nutrients providing energy, the percentage of kilocalories from fat increased from 32% to 43% between 1909 and 1985; while, kilocalories from carbohydrate decreased from 57% to 46%. Protein levels remained about the same at 15% of kilocalories (14). Data from several food consumption surveys, including NHANES II (39), the 1985 Continuing Survey of Food Intake by Individuals (CSFII, 40), and the 1987-1988 National Food Consumption Survey (NFCS, 41) revealed mean energy intakes of 1479 to 1538 kcal for women, aged 19-50 years.
The National Academy of Sciences (29) recommends the following daily caloric intakes for adults and teens, based on activity level and age: (a) 1,600 kcal - for sedentary women and some older adults; (b) 2,200 kcal - for most children, teenage girls, active women, pregnant and lactating women, and sedentary men; and (c) 2,800 kcal - for teenage boys, active men, and very active women. In contrast, 2,200 kcal per day was the recommended energy allowance for females, aged 19-50 years.

Dietary data from NHANES II (42) showed that white bread, cakes pastries, alcoholic beverages, whole milk, and hamburgers were the top five contributors of energy, providing 40% of total energy in the American diet. On the other hand, data from the 1985 CSFII (43) showed yeast breads as the leading energy source in the diets of women (aged 19-50 years), providing 7% of total calories. Yeast breads were followed by regular softdrinks, cheese, beef, and salad dressings, contributing 17% of energy.

The relationship of total energy intake to the development of CVD has been examined in several epidemiological studies. In a study by Gordon et al. (44), subjects who developed heart disease had a history of higher-than-average energy intake as compared with those who remained free of the disease. In a longitudinal study, Lapidus et al. (45) found an inverse relationship between death from all causes and general energy intake, but a positive correlation with energy from fats and triglycerides. This finding agrees with those of Kuske et al. (46), who reported that excessive energy
intakes, whether from fat, carbohydrates, or protein, produced hyperlipoproteinemia and especially hypertriglyceridemia. Excessive intakes of kilocalories are usually accompanied by high fat intakes, including SFA and cholesterol.

In animal experiments, increased energy intakes have resulted in increased adiposity; but the effect on the risk for CVD of animals (altered lipid profiles, hypertension, and stroke) has been attributed to changes in the macro nutrient composition of the diet, not increased energy intake (14). In all of these studies, when there was a caloric restriction, there was a concomitant weight loss and normalization of triglyceride levels.

The Committee on Diet and Health (14), proposed that the mechanism involved in the effect of excessive energy intake on risks for CVD is that the excess energy supplies the precursors for synthesis of triglycerides which increase the production of VLDL. This effect may be enhanced in susceptible individuals, such as obese and diabetic patients, and in those with pre-existing elevated triglycerides levels.

It is difficult to demonstrate a direct relationship between total energy intake with morbidity or mortality in the general population. Nonetheless, some studies conducted by the National Institutes of Health (NIH, 14) provide evidence that excessive energy intake is a risk factor in the development of CVD and that a reduction in energy intake may reduce risks for CVD.
**Dietary Fats and Cholesterol.** An association between excessive dietary fat intakes and CVD is supported by a substantial body of literature. At present, saturated fats make up the largest percentage of energy from TF in the typical American diet, contributing to elevations of blood TC and LDL-C levels in susceptible individuals. The effects of dietary fats in humans and animals were studied in two controlled feeding trials (47-48). Masana et al. (47) demonstrated that a decreased intake of total fat in the diet would be accompanied by a reduction in SFA and energy intakes. They also showed that an increase in monounsaturated fatty acids (MUFA) intake is as effective as polyunsaturated fatty acids (PUFA) in lowering TC and LDL-C levels but does not adversely affect HDL-C concentrations. Charnock et al. (48) also showed that dietary SFA induces susceptibility to the development of cardiac arrhythmias under ischemic stress; whereas, PUFA reduces this susceptibility. SFA, whether from vegetable or animal sources, has been found to have twice the potency in raising cholesterol and LDL-C levels and in lowering of HDL-C levels than MUFA and PUFA. A summary of the effects of the various types of dietary fats and cholesterol on CVD risk is located in Appendix A.

A low-fat diet is recommended to decrease TC, LDL-C, and triglycerides to desirable levels; thereby, reducing one's risk of CVD, cancer, and obesity. Several health agencies (29,49) have recommended that Americans limit their intake of TF to less than 30% kcal, SFA to less than 10% kcal,
MUFA to 10-15% kcal, and PUFA to 7-10% kcal. Efforts have been made to determine the current fat intake of Americans; however, the various national surveys vary somewhat in their estimates of TF consumption (14). Data from NHANES II (39) indicated a mean fat intake of approximately 38% kcal for women aged 19 to 50 years, which was slightly higher than 37.2% kcal intake from the 1985 CSFII data (40) and the 36.5% kcal fat intake revealed in the 1987-1988 NFCS data (41) for women of the same age. Systematic biases of dietary methodology and differences in type and depth of questions may explain the different estimates of fat intake (14). NHANES II data (39) revealed a mean SFA intake of 13.6% kcal, a MUFA intake of 14.2% kcal, and a PUFA intake of 5.6% kcal for low-income women, aged 19-50 years.

In regard to major food sources of fat, Block et al. (50) analyzed NHANES II data and found that hamburgers were the leading contributors of total fat, SFA, and MUFA, providing 8% kcal of each type of fat. Mayonnaise and salad dressings provided 15% kcal of PUFA. Based on multiple 24-hour food recalls collected in the 1985 CSFII (43), SFA intake was 13.4% kcal for women, most of which came from cheese ground beef and beef cuts.

Trends in the consumption of food sources of cholesterol have been reported in several national surveys. In the 1987 National Health Interview Survey (NHIS, 51), the usual American diet for Caucasian and African-
American women contained about 265 mg per day of dietary cholesterol, which was lower than the 279 mg/day found in the 1985 CSFII (40) and the 287 mg/day found in NHANES II (50) data. The major dietary contributors of cholesterol are eggs (providing 36%), and beef which contributes 16% of the dietary cholesterol in the general adult population (42) and among women (43) in the U.S. In 1992, the NCEP (49) computed the proportions of the macro nutrients that are associated with the risk of CVD and recommended that TF be reduced to 30% kcal.

Kuske et al. (46), reported that diets, most associated with the development of atherosclerotic CVD in humans, contain 200 mg of cholesterol per 1000 kcal or more. Cholesterol intake within the range of 200-300 mg/1000 kcal/day has been shown to have the most pronounced adverse effect on blood lipid levels. In experimental studies using animal models, the effect of dietary cholesterol and fats on lipoprotein levels has been studied extensively. In most experiments, hypercholesterolemia was induced with a diet enriched in cholesterol or SFA, or both.

Numerous studies on nonhuman primates have demonstrated that withdrawal of either cholesterol or fat from the diet (with or without drug therapy) will result in a corresponding reduction in TC and LDL-C levels; whereas, there is variability in the response of HDL-C. This may explain the varying results among experiments involving small sample sizes (14). The Committee of Diet and Health (14) recommends that results from these
animal studies be interpreted with caution, as amounts of cholesterol fed to the animals were in the range of 1000 to 2000 mg/day and the consumption period was shorter than that normally seen in humans. In contrast, dietary cholesterol levels of 300-500 mg/day are considered to be high intakes in humans and these levels increase risk for CVD. Stamler et al. (52) found an 8-10 mg/dl increase in LDL-C levels with each additional 100 mg/1000 kcal of dietary cholesterol. They also found that cholesterol increments of 200 mg/1000 kcal daily are associated with 30% greater risk of CHD.

Two randomized trials (14) on primary prevention of CHD used dietary change as their only intervention. In the double blind Los Angeles Veterans Administration Domiciliary Study of 846 men, aged 55-89 years, the effects of two diets (each containing 40% kcal of total fat) were studied using an experimental and a control group. The experimental diet contained 35% to 40% kcal for MUFA and PUFA and a lower proportion of SFA than the control diet. Results revealed a prompt and sustained reduction in mean TC levels of experimental subjects, which amounted to a 12.7% difference between the experimental and control groups. Sudden death and definite myocardial infarction were 24% lower in the experimental group. Similar results were found in the Finnish Mental Hospital Study (53); however, the sample of women was relatively small. Likewise, Gordon et al. (44) showed that reduction in intake of dietary cholesterol resulted in decreased levels of serum TC and LDL-C, among 6,000 men with initially "high" TC levels.
Current dietary guidelines recommend that diets be low in TF, SFA and cholesterol, but contain adequate amounts of protein, carbohydrate, vitamins, and minerals to achieve and maintain desired blood lipid levels (14,26,29). Although genetics play a role in blood lipid levels, a majority of the general population can control and maintain desired lipid levels by following the dietary guidelines. A summary of the effects of various types of dietary fats and cholesterol is located in Appendix A.

Dietary Fiber. The current interest in dietary fiber stems from the epidemiological association of a high fiber intake with a lower incidence of certain chronic disorders, such as CVD. Despite extensive reviews, there has been difficulty in establishing a clear relationship between dietary fiber and CVD risk. Diets that are high in fiber are usually lower in energy and TF and possibly low in SFA and cholesterol (54).

In a review (54) of dietary fibers and their effects on serum lipids, the soluble fibers (oat bran, dried beans, pectin from citrus fruits and apples, psyllium, or guar gum) were effective in lowering concentrations of TC and LDL-C. Similarly, Kris-Etherton et al. (55) found that adding 15-45 grams of soluble fiber to the diet resulted in a 6-9% reduction in TC levels. Likewise, animal studies (56) using oat fiber (50-100 grams per day) have been shown to reduce TC concentration in rats with normal or high levels of TC. According to Schinnick et al. (56), the effect of oat bran in lowering blood cholesterol can be attributed to its soluble fiber content.
Possible mechanisms (54) involved in the protective role of soluble dietary fiber in atherosclerotic CVD are: (a) binding of bile acids, which results in a reduced bile acid pool and serum cholesterol, as cholesterol is used to replenish the bile acid pool; (b) fermentation of soluble fiber by colonic bacteria, producing short-chain fatty acids that inhibit synthesis of hepatic cholesterol; (c) increased catabolism of LDL-C; and (d) indirect effects of fiber in replacing some of the dietary SFA and cholesterol. Based on studies examined by the Committee on Diet and Health (14), guar supplements lower LDL-C levels without changing the levels of HDL-C; whereas, oat bran reduces triglyceride levels.

Based on the 1987 NHIS data (51), the average intake of dietary fiber is approximately 8.7 grams/day for both Caucasian and African-American women. On the other hand, Miller et al. (57) examined and compared the body composition of lean and obese women, in relation to their dietary fat intake. Results showed that obese women had a significantly lower dietary fiber intake (15.7 grams per day) than lean women (22.7 grams per day).

The Dietary Guidelines for Americans (29) recommend a dietary fiber intake of 20-35 grams/day for adults. According to the Committee on Diet and Health (14), this recommendation could be met by eating 3-5 servings of vegetables (especially green and yellow vegetables), 2-4 servings of fruits, and 6-11 servings of whole-grain bread, cereals, rice, and pasta per day. In line with the nation’s health promotion and disease prevention objectives,
the National Cancer Institute (NCI) and the Produce for Better Health Foundation (58) established the Five-A-Day Program in nine states (including Alabama, Georgia, Louisiana, Minnesota, Maryland, Arizona, Massachusetts, Washington State, and North Carolina) to encourage American children and adults to increase their consumption of fruits and vegetables, as they are high in fiber, but low in fat and dietary cholesterol.

**Dietary Sodium.** Epidemiological studies of diet and CVD have indicated a relationship between salt intake (specifically sodium) and blood pressure among population groups. These studies strongly indicate that habitual intake of sodium/salt was an important determinant of blood pressure in humans in spite of the variability in genetic susceptibility to salt-induced hypertension (14). Although studies have shown that susceptibility to the effects of sodium varies with individuals, sodium restriction provides greater benefits to salt-sensitive individuals. Controversy continues regarding the restriction of dietary sodium in the general population.

The Committee of Diet and Health (14), reported that diets containing >2,400 mg/day of sodium are strongly associated with high blood pressure; while, a sodium intake of 1,800 mg provides health benefits. The mean sodium intake reported in the 1987 NHIS (51) data was 2,024 mg, which is lower than the 2,173 mg intake found in NHANES II (39), and the 2,305 mg intake found in the 1985 CSFII (40). Nonetheless, health experts believe that lower sodium intakes will provide greater health benefits for Americans.
Regarding dietary sources of sodium, the Committee on Diet and Health (59) reported that foods and beverages containing sodium-chloride (39% sodium by weight) are the primary sources of sodium. Results of a study by Sanchez-Castillo et al. (60) showed that only 10% of salt intake comes from natural food sources; while, 15% comes from salt added during cooking and at the table, and 75% from salt added during processing and manufacturing.

Most researchers agree that habitual high sodium intakes can increase risk of developing hypertension. The Committee on Diet and Health (14), reported that high blood pressure is a major risk factor for CVD in the U.S. due to the following factors: (a) sodium weakens the arteries of the brain, causing rupture or blood clots; (b) sodium contributes to coronary atherosclerosis which increases the risk of a heart attack; (c) sodium can damage tiny vessels needed for filtering the blood in the kidneys, leading to kidney failure; and (d) sodium can cause elevation of blood pressure which damages blood vessels in the retina of the eye, leading to blindness.

In 1988, the Intersalt Cooperative Research Group (9), reported data indicating that a reduction in sodium/salt intake prevents blood pressure from increasing with age. Sodium restrictions may be particularly appropriate for certain groups at increased risk for developing hypertension, including those with a family history of hypertension, African-Americans and other acculturated migrants, those with "borderline-high" blood pressure, all adults aged
55 and over, and obese people (9,14). According to Katzenstein (61), a decrease of 1,200 mg/day of sodium intake (3000 mg/day of salt) would result in a 22% decline in the incidence of stroke, and a 16% decrease in heart disease, thus, having a greater impact than drug therapy.

**Potassium.** Evidence of human and animal studies indicate that consuming a diet containing recommended levels of potassium (1800-3600 mg per day) may prevent hypertension in some individuals (14,59). The Committee on Diet and Health (14) proposes possible mechanisms that may explain the cardio-vascular protecting effects of potassium: (a) high potassium diets provide protective effects against vascular damage and stroke by diminishing the release of endothelial, macrophage, and platelet-derived preserving the normal function of the arterial endothelial cells; (b) potassium prevents the thickening of the intima and medial layers of arteries; and (c) potassium is needed for the relaxation of the arterial wall. Overall, these proposed mechanisms suggest that a high-potassium diet may protect arterial endothelial lining cells against damage caused by elevated blood pressure.

Regarding dietary potassium intakes of women, aged 19-50 years, the 1985 CSFII (40) reported daily mean potassium intakes of 2,209 mg/day, the 1987 NHIS (51) reported an intake of 1,917 mg/day, and the NHANES II (39) reported a potassium intake of 1,810 mg/day. In contrast, a review of minority health issues (32), indicated that Blacks have a mean potassium intake of 1000 mg/day as compared with 2,500 mg/day for Whites.
In the U.S., low-potassium diets (800-1,600 mg/day) have been associated with increased risk of stroke, hypertension, and hypertension-related end-stage renal disease. Kumanyika et al. (32) found a higher incidence of stroke among African-Americans with very low potassium intakes, than among any other racial group in southeastern U.S. A study by Rostand et al. (62) indicated that African-Americans have higher incidences of hypertensive renal damage than Caucasians, which correlate with differences in potassium intake.

Positive correlations between urinary sodium to potassium ratio and diastolic blood pressure were shown by Langford (63), in a study of 101 African-American women aged 20 years and older. In 1987, Khaw et al. (64) showed inverse correlations of daily potassium intake with incidence of stroke-related deaths, over a 12-year period, among people aged 50 and older. They also found that increased intake of 400 mg/day of potassium would decrease stroke-related deaths by 40%. This amount of potassium corresponds to only 1 or 2 extra servings of fresh fruits, fruit juices, fresh vegetables, or potatoes. Evidences from epidemiological studies indicate that consuming a diet high in potassium and low in sodium may exert a beneficial effect in reducing blood pressure.

**Calcium.** Researchers found that contractions of cardiac smooth muscles was influenced by an increase in levels of calcium (65). This observation assumed that dietary calcium may influence regulation of blood pressure.
In recent years, the association of dietary calcium and hypertension has received considerable attention. McCarron et al. (66) demonstrated an inverse relationship between calcium intake and incidence of hypertension. A similar conclusion was reached in a clinical metabolic study conducted by Resnick (67), who demonstrated that calcium supplementation caused a mild, short-term reduction in blood pressure in individuals with normal blood pressure and in those with hypertension.

In the U.S., there is a great variation in calcium intake among individuals, particularly among females. Nutrient data from the NHANES II (39), the 1987 NHIS (51), and the 1985 CSFII (40) indicate that mean calcium intakes for adult females range from approximately 559 mg/day to 638 mg/day. The 1987 NHIS (51) data revealed that the mean calcium intake for African-American women was 118 mg lower than the mean intake of Caucasian women (687 mg per day). Based on these survey results, a majority of female adults have below-recommended mean calcium intakes. With regard to SES, the Committee on Healthy People 2000 (9) reported that people with incomes below the poverty level have consistently lower mean calcium intakes than the general population.

The best food sources of calcium included milk, yogurt, and cheese which provide 55% of the calcium intake in the U.S. population (29). To obtain sufficient calcium, 2-3 daily servings/day of milk, cheese, and yogurt are recommended. Low-fat and non-fat milk and milk products should be
emphasized, as they contain more calcium with little or no SFA as compared to whole milk products.

Despite the protective role of calcium, studies attempting to demonstrate a direct role in the prevention of high blood pressure have had conflicting results. Harlan et al. (68) found systolic blood pressure to be negatively correlated with calcium intake among women. Likewise, a study by Gruchaw (69) on nutrient intake and hypertension, concluded that dietary calcium was not a significant predictor of blood pressure. Thus, inconsistencies in epidemiological, clinical, and animal findings have not adequately established a clear causal relationship between calcium and hypertension.

**Dietary Methodology.** The 24-hour food recall was used in this study because it is the usual method for collecting dietary information in EFNEP and has the following advantages: (a) can be administered by non-dietitians, (b) can be used with low-literacy or illiterate respondents, © the time required for administration is short, and (d) cost of using the method is generally low. However, the 24-hour food recall has the following limitations: (a) inaccurate reporting due to failure of subject to recall foods and the size of the portions consumed, (b) high day-to-day variability in nutrient intake among some subjects, (c) the desire of respondent to please interviewer, (d) reluctance of participant to report large intakes of alcohol, sweets, and other snack items, and (e) single 24-hour food recalls are not representative of usual or customary intake of individuals (14).
Obesity and Its Measurement

In general, obesity refers to an excess of body fat and is more specifically defined as a body fat greater than 31% or body mass index (BMI) exceeding 27.1 kg/m² (1). Obese individuals are at greater health risk, especially of developing CVD, than people with desirable levels of body fat, of 16%-31% and a BMI <25 kg/m² (70).

The prevalence of obesity among adult females in the U.S. is increasing (1). Baseline data from NHANES II (9) show that 37% of low-income women, aged 20 and older are obese. Data from the National Center for Health Statistics (1), collected between 1988 and 1991, revealed that 35% of women (32.4% of whites and 49.5% of blacks), aged 20-74 years, had BMI levels of 27.3 kg/m² or greater.

Among people with low SES, it appears that there is an interaction between gender and social class, producing higher rates of obesity among women. In several studies, the effect of SES on female obesity has been demonstrated. In an effort to clarify the factors that led to the current weight distribution, a prospective cohort study of 3,284 adult female participants was conducted by Kahn et al. (71) in 1991. Results reveal that women at greatest risk of major weight gain (an increase of 13 kg or more) are those with below-college education and those in low-income families.

Bowen et al. (72), hypothesized that the greater prevalence of obesity among women with low SES is due to the following factors: (a) differing
levels of self-imposed dietary restraint; (b) differing levels of physical activities; (c) genetics; (d) social limitations; (e) pressure from financial constraints; and (f) lack of adequate nutrition knowledge. Dietary restraint was shown to be the most powerful influence on body weight in that women with higher SES showed greater restraint in eating than women of low SES. Certain food industries promote cheap, high-fat foods to the poor. Poor women tend to buy and consume less expensive foods from fast-food restaurants, which tend to be calorie-rich and high in fats and sugars. Diet centers and health clubs are virtually inaccessible to poor women; thus, they have little access to exercise facilities. Furthermore, the social and physical environment in which they live, such as increased crime rate, discourages outdoor physical activity.

Golden et al. (73) found that low-income groups had less information about nutritional needs, which led to a higher incidence of overweight. Other researchers have concluded that the higher obesity rates among the poor are more reflective of economic limitations than lack of nutritional knowledge. Unfortunately, most of the literature on SES and obesity is primarily descriptive and does not focus on testing hypotheses about the influence of SES on weight and body fat levels.

With regard to racial differences for female obesity, Khan et al. (71) showed that African-American women experienced a 13.4% incidence of major weight gain as compared to 7.7% for Caucasian women and the mean
increase in weight was greater for African-American women (3.8-9.6 kg) than for Caucasian women (2.8-8.0 kg). However, the interaction between race and major weight gain did not significantly differ. The authors concluded that the racial effect on mean weight change was the result of a decreased tendency to lose weight among African-American women. Those who were overweight at baseline, showed a clear tendency to gain weight and a resistance to major weight loss. Surprisingly, the authors stated that the black race was not an independent risk for weight gain, but instead, was associated with a reduced likelihood of losing weight.

Similar results were found in a cross-sectional study done by Burke et al. (74) with a group of 5,115 young adults, aged 18-30 years, in which data indicated a greater prevalence of obesity among African-American women, with increased racial differences appearing as early as 18 years of age. The authors concluded that specific race-gender groups are at high-risk for obesity, both in young adulthood and in later life. Hence, it appears that race can exert an independent effect on obesity levels for women in the U.S.; nevertheless, the question of why women of black race are at increased risk for obesity has not been answered.

It has been widely assumed that body composition is related to ethnic status and that ethnic differences are an important factor in body weight, which would greatly affect prevalence of overweight and obesity in the U.S. A study was conducted by Ortiz et al. (75) to evaluate body composition
differences using underwater weighing, whole body potassium counting, water dilution, and dual photon absorptiometry techniques, which provide ethnicity-independent estimates of body composition. Subjects included 28 pairs of black and white females (aged 20-70 years), matched for age, weight, height, and menstrual status. Results revealed that African-American females have significantly greater appendicular skeletal muscle and more total bone mineral mass (11-15% greater) than their Caucasian counterparts.

Reasons proposed for the increased musculo-skeletal mass in African-American females are: (a) black fetuses have greater bone length and weight and this persists throughout life; (b) blacks have higher bone density in weight-bearing and non-weight-bearing areas; (c) blacks have higher levels of serum parathyroid hormone and 1,25-dihydroxy vitamin D concentrations. Although the reasons for the differences in body composition are still unclear, present evidence suggests that genetic factors are involved, rather than environmental influences.

Data on body weight, body fat, and risk of CVD suggest that certain lifestyle variables, including diet and physical activity, contribute to obesity which subsequently increases the risk of CVD. Maintaining moderate body weight appears to be an important preventive measure in lowering CVD risks, especially in populations where obesity and high TC levels are widespread (i.e. among low-income, non-white women).
Biochemical Factors

Total Cholesterol, LDL-C, HDL-C. Most major epidemiological studies on blood cholesterol levels have focused on men with few have providing data on women. Prior to the last decade, TC levels, rather than individual lipoproteins, were measured in most epidemiological studies because reliable methods were not available for measuring the individual lipoproteins in large numbers of people (14). Recently, the Expert Panel on Detection, Evaluation, and Treatment of High Blood Cholesterol in Adults (18) reported that simply measuring an individual’s TC level could be misleading, because the cholesterol that enters the bloodstream is of two types, based on the lipoprotein fraction, namely HDL-C and LDL-C. These fractions have very different effects on the health of the arteries. LDL-C contains 60-70% of the plasma cholesterol and tends to deposit on arterial walls; whereas, HDL-C contains a smaller amount of cholesterol and is the means by which cholesterol is transported back to the liver to be degraded.

Population-based studies have consistently indicated a strong positive relationship between TC levels and the prevalence of CVD. The Committee on Diet and Health (14) reported findings from a prospective study on middle-aged men in which TC levels above 200-220 mg/dl were positively associated with risk of CVD; whereas, low levels of TC were associated with a lower risk of CVD in the U.S.. According to the NCEP (18), cohort data in women indicate less certain results in the association of TC levels with CVD.
mortality rates, as total mortality did not shown increase with higher TC levels in women. Nevertheless, the weight of scientific evidence supports the idea that TC levels of 200 mg/dl and greater are positively associated, in a linear fashion with CVD risks for adults of both genders (9,14,18).

A majority of population-based studies have shown that the risk of CVD is directly related to the plasma/serum levels of LDL-C and inversely related to HDL-C levels in both genders (14,18). Results from the Framingham Study (10) revealed that HDL-C is inversely related to risk of CVD in women, in that high HDL-C levels (>45 mg/dl) have low risk of coronary artery disease (CAD) even when their TC levels are elevated (>200 mg/dl). On the other hand, women with low levels of both TC (<200 mg/dl) and HDL-C (<35 mg/dl) are considered to be at high-risk for CVD. These differences are not seen in male subjects. Thus, data suggest that low levels of HDL-C are a more consistent risk factor for CVD in women. Health authorities supported the recommendation of NCEP experts that efforts should be made to increase HDL-C levels, to provide protection against CVD.

Definition of “desirable” blood lipid levels according to age and sex has been provided in the NHANES III data (18) which show distributions for serum TC, LDL-C, and HDL-C in the U.S. population by age, race, and sex. In regard to primary intervention in adults, without CVD, guidelines for classification and management of blood lipid levels were established by authorities with NCEP (Appendix B).
Triglycerides. In most within population studies, triglyceride levels were positively associated with increased risk for CVD, but were not independently predictive of CHD after statistical adjustments were made for associated factors (including TC, HDL-C, hypertension, cigarette smoking, and obesity). At the recent NIH Consensus Conference (18), it was concluded that the risk of CVD is not necessarily increased in the presence of normal TC levels accompanied by mild elevations of plasma triglycerides. Accordingly, triglyceride levels below 250 mg/dl do not necessitate changes in lifestyle beyond those recommended for the general public, since the risk generally does not exceed that of other Americans. The same was true for individuals with "desirable" TC levels and with borderline triglyceride levels (250-500 mg/dl), but without other known risk factors for CVD.

Although triglyceride levels have not been established as an independent risk factor for CVD, there exists a reciprocal relationship between plasma triglycerides and HDL-C levels, which has been documented in most population and individual studies (14). Larosa (37) reported an association between elevated triglyceride levels and a form of LDL-C which was smaller and denser than the normal LDL-C and which, if present in increased levels may be atherogenic. Thus, individuals with elevated levels of triglycerides tended to have lower HDL-C levels, and an increase or decrease in triglyceride is generally accompanied by an opposite change in TC level.
The above findings were not in agreement with the data collected in the Framingham Heart Study (10) which revealed that triglyceride level is an independent predictor of CHD in women, particularly in older women aged 60-69 years. This has been supported by observations of several studies. In a review of lipoprotein research, Larosa (37) concluded that this unique predictive power of triglycerides in elderly women might be due to age and sex-related differences in the composition of VLDL and LDL-C, in which VLDL’s are larger, lighter, and lower in free cholesterol content in middle-aged women than men of comparable age. This supports the finding that a high plasma triglyceride level probably reflects presence of certain atherogenic lipoproteins, rather than being a direct cause of the atherosclerotic disease.

Clinical studies have shown that disease entities, such as diabetes, primary hyperlipidemia, nephrotic syndrome, and chronic renal disease increases risk of CVD. In these conditions, an elevated triglyceride level serves as a clue to the presence of other lipoprotein abnormalities that are directly associated with CVD, such as low HDL-C, elevated APO-B or LDL-C, and elevated levels of remnant triglyceride-rich lipoprotein particles (10).

In summary, it is not clear whether or not elevated triglyceride levels are directly linked to atherosclerotic disease. However, "borderline-high" or "high" triglyceride levels can aid in identifying people at increased risk for CVD.
**TC to HDL-C Ratio.** Increasing attention is given to the relationship between TC and HDL-C and the development of CVD. Data on how TC:HDL-C ratios predict CVD were presented by Dr. William Castelli, director of the Framingham Heart Study (76). The data show that 50% of female participants have an average risk ratio of 4.23 and among females without evidence of CHD, the average risk ratio was 4.4. Findings in the Framingham Heart Study also show that a 4.4 ratio is associated with normal risk of CVD, while, a ratio of 5.17 and above would indicate a high risk of CVD and a ratio of less than 3.27 would be ideal. Based on results of the Framingham Heart Study (76) TC:HDL-C ratios may be useful predictors of CVD, particularly for CHD risk in the U.S. population. These findings on risk ratios were supported by Byrne (77) and Grundy (78).

According to NCEP (18), a ratio such as TC:HDL-C may have uses in summarizing the importance of HDL-C in clinical settings, but it was recommended that clinicians focus on HDL-C separately in assessing risk and choosing therapy. TC:HDL-C ratios were not included as a part of the 1993 guidelines for the detection, evaluation, and classification of high blood cholesterol in adults.

**Smoking**

Smoking is recognized as an important preventable cause of chronic morbidity, disability, and death in the U.S.. Over the past decades, numerous studies have documented the harmful effects of smoking (79-80).
Smoking is a contributory factor in numerous health problems such as diseases of the heart and blood vessels, cancers at various sites, acute and chronic respiratory infections, and stomach ulcer. Based on recent national statistics, cigarette smoking accounts for about 390,000 deaths yearly, including 21% of all CHD deaths, 87% of lung cancer deaths, and 30% of all cancer deaths which represent more than one of every six deaths in the U.S. (9,20).

In recent years, public knowledge and beliefs about health risk of smoking have increased, as a result of intensive educational efforts and creative approaches. The prevalence of cigarette smoking among adults in the U.S. has been steadily declining. At present, the prevalence of smoking among female adults, aged 18 years and older, has decreased steadily from 34% in 1965 to 23.6% in 1991; however, there was a 1.2% increase in smoking among females in 1992 (1).

In an analysis by Shopland et al. (81) using the 1985 Current Population Survey (CPS) of the Office of Smoking and Health, prevalence estimates of tobacco and tobacco products usage were identified for various geographic strata, by gender and age group. Results revealed that the South dominates the country in smoking of cigarettes among young men; however, rates of cigarette smoking among southern females were not substantially different from that of women in other areas of the country which was 26.5%. Data also indicate that Virginia ranks 12th in the overall prevalence
of cigarette smoking (rate of 28.34%) and use of at least one other tobacco product (29.13%), among women aged 20 years and older. However, the findings also indicate that few females use tobacco in forms other than cigarettes (81).

Some studies have shown an overall decrease in rates of cigarette smoking among adult females; however, the decrease is less for Caucasians than African-American female smokers (1). The incidence of heavy smoking (25 cigarettes or more per day) increased among Caucasian women from 13.3% to 22.7%; whereas, the rate of heavy smoking increased from 4.6% to 6.6% among African-American women. The observed increased in female smokers since 1992 (1), particularly in the age group of 25-44 years, has prompted investigators to predict that cigarette smoking prevalence among women may exceed that of men by mid 1990’s.

Differences in smoking appear to be largely due to differences in SES and demographic factors. Characteristics associated with high smoking rates include low educational attainment (having < high school education, an income <10,000/year, employed in a service-level job, unemployment, unmarried status, and living in urban communities (82-84). Thirty-seven percent of adults with < high school education smoke as compared with only 15% of college graduates who smoke (1). Reduction of smoking to a rate of < 20% is one of the national goals in the National Health Promotion and Disease Prevention Objectives Report of 1992 (9).
Brownson, et al. (79) conducted an assessment of socio-demographic differences in beliefs about the health effects of cigarette smoking among 725 males and 1,367 females (18 years and older) in St. Louis and Kansas City, Missouri. Results reveal that knowledge of health effects of smoking are generally lower among women, older respondents, those with lower educational levels, and those who currently smoke. Authors also found that African-Americans are less likely to acknowledge the health effects of active smoking, but are more likely to be concerned about health effects of passive smoking. Data also showed that current smokers are significantly less likely than nonsmokers or former smokers to acknowledge the harmful effects of smoking. Findings on knowledge of the relationship of smoking and heart disease was noteworthy, as only 29% of former smokers and 40% of current smokers indicated a lack of awareness of smoking as a heart disease risk. This finding suggest that many smokers underestimate or deny the serious health effects of smoking.

Smokers have a two to four-fold greater incidence of CVD, a > 70% death rate for CVD, and sudden death than nonsmokers (9). In 1985, smoking accounted for 21% of all CVD deaths and 40% of CVD deaths in people younger than 65 years of age. Prospective epidemiological studies documented substantial reduction in CVD rates following smoking cessation. Some studies have shown benefit within two years after quitting, while other studies suggest that risks of CVD decrease over a period of several years.
One of the reasons given by smokers for not quitting is the fear of unwanted weight gain. Epidemiological studies have demonstrated clearly that smokers weigh less than nonsmokers and that smoking cessation leads to weight gain among women. On the other hand, studies show that female smokers have a greater upper body fat distribution than female non-smokers and this fat distribution is related to increased rates of CHD, stroke, diabetes, and unfavorable lipid profiles.

A cross-sectional study was done by Lissner, et al. (84) to determine the relationship of smoking initiation and cessation to body fat distribution in a sample of Swedish women and results revealed that smokers have significantly more upper-body fat than nonsmokers of similar BMI. In addition, former smokers have less upper-body fat depositions even with their accompanying weight gain, suggesting that weight gained after smoking cessation is not deposited in the region having the highest association with risk of CVD in women.

**Educational Programs to Reduce Risk of Cardiovascular Disease**

Challenges faced by adults in disease prevention are influenced by individual decisions. These decisions are subject to varying degrees of pressure, in and out of the home, and this necessitates collaborative efforts among agencies and individuals, having various types of knowledge and expertise, in order to create an environment that will facilitate and support healthful behaviors (9,14).
Community-based programs are needed to reach and improve the health of individuals outside of the traditional medical setting, especially among minority, low-income, and rural communities (9). People in these areas need more preventive services and relevant information about their particular disease risks and feasible ways to reduce them. Effective health education programs which are intensive, but easy to implement, are needed to reach large numbers of low-income and minority individuals. An increasing number of states and local organizations are establishing health promotion and disease prevention programs. To be effective, community-based programs must be well-planned and should involve potential clients in the planning process. Effective programs will usually include multiple interventions (9).

In poor predominantly African-American communities, the role of the church and clergy are increasing as a focal point for health education programs. There is a growing support among African-American church members for meeting the health needs of their members and the surrounding community. Many such programs have focused on the prevention of CVD, but little is known about their impact on risk factors among African-Americans.

A report of a study by Wiist et al. (85) describes a cholesterol education program conducted in six African-American churches by trained, volunteer members. Data collected for screening and evaluation included serum
TC, blood pressure, weight and height, which were collected on all participants, initially, with a follow-up screening 6 months later. A six-week nutrition education class was provided for participants, while those having a need were referred to their personal physician. The education program focused on modifying fats in the diet with increase use of low-fat foods and emphasized the reduction of food sources of SFA and cholesterol. Information about CHD risk factors and the corresponding recommendations were taught, including weight reduction, exercise, and smoking cessation.

Results revealed significant reductions in mean serum TC levels in both groups and values were large enough to be of clinical importance. Wiist et al. (85) suggested that CVD screening and nutrition classes conducted in African-American churches by trained volunteers can be an effective method of reducing cholesterol and fat intake among individuals in African-American communities.

Similarly, a health promotion project in northern Florida was conducted by the American Heart Association Minority Cardiovascular Health Program Initiatives (86). Project activities included quarterly newsletter, exercising to religious music, and development of a cardiovascular-peer-facilitated learning manual to reinforce behavioral change. The program activities influenced the health behavior of participants by reductions in blood pressure, increase participation in exercise sessions, decreased intakes of eggs, salt, and increased intakes of fruits, vegetables, chicken, and fish.
Summary

This review of literature was organized under five sections. The first section described the setting and conditions that were believed to have caused the increase incidence of CVD. The second and third section characterize the ways in which EFNEP recognizes and relates to help resolve these diet-related conditions. The fourth section deals with the background and description of risk factors for atherosclerotic CVD in women and sets forth the epidemiological, clinical, and experimental data pertaining to each of the selected risk factor. Data presented on risk factors categorized the following: demographic (race, SES, and age), dietary factors [energy (kcal), TF (% kcal), SFA (% kcal), MUFA (% kcal), PUFA (% kcal), cholesterol, dietary fiber, sodium, potassium, calcium, the food groups], measures of obesity (percent body fat and BMI), selected biochemical (TC, LDL-C, HDL-C, Triglycerides, and TC:HDL-C ratio), and smoking. The fifth section discussed educational programs using dietary interventions to reduce risk of CVD among low-income, low-literate women, particularly those belonging to minority groups.

The fact that extensive research has been done on atherosclerotic CVD indicates that this diet-related disease has multiple causes and risk factors. Evidence of studies reviewed in this chapter supports the association between diet and the development of CVD. High priority should be given to modification of these risks of CVD through educational programs.
CHAPTER III

METHODS AND PROCEDURES

Design of the Study

The experimental design used in this study utilized two groups: an experimental group (Group EC) and a control group (Group C). Both groups were involved in the following three phases:

1. A pre-intervention data collection phase, in which face-to-face interviews were used to complete various forms and questionnaires. Participants’ baseline body weight, height, percent body fat, and blood lipid values were measured and body mass index (BMI) values of participants were calculated.

2. An intervention phase, during which subjects in the experimental group participated in a 6-month nutrition education program and control subjects were taught resource management lessons only.

3. A post-intervention phase with the same data being collected as at pre-intervention.

Comparisons of baseline information were made between Caucasian and African-American women to determine whether risk factors for CVD differ by race prior to intervention. At the end of the study, both groups were evaluated, and comparisons were made between the experimental and control groups regarding change on measured variables to determine the effect of the intervention. Table 1 summarizes the design of the study.
<table>
<thead>
<tr>
<th>Month</th>
<th>Phase of Study</th>
<th>Group</th>
<th>Proposed Action</th>
</tr>
</thead>
<tbody>
<tr>
<td>July - October, 1992</td>
<td>Pre-intervention</td>
<td>Experimental and Control</td>
<td>• EFNEP paraprofessionals recruited and enrolled 150 female homemakers</td>
</tr>
<tr>
<td></td>
<td></td>
<td></td>
<td>• baseline interview and questionnaire administered</td>
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<tr>
<td></td>
<td></td>
<td></td>
<td>• random-repeat (3) 24-hour food recalls collected</td>
</tr>
<tr>
<td></td>
<td></td>
<td></td>
<td>• blood lipid samples collected</td>
</tr>
<tr>
<td></td>
<td></td>
<td></td>
<td>• body weight, height, BMI, % body fat measurements taken</td>
</tr>
<tr>
<td>October 1992 - April 1993</td>
<td>Intervention</td>
<td>Experimental and Control</td>
<td>• participants taught by EFNEP paraprofessionals: Group EC received nutrition lessons; Group C received lessons on money-management only.</td>
</tr>
<tr>
<td>April - June, 1993</td>
<td>Post-intervention</td>
<td>Experimental and Control</td>
<td>• administered post-study interview and questionnaire</td>
</tr>
<tr>
<td></td>
<td></td>
<td></td>
<td>• random-repeat (3) 24-hour food recalls</td>
</tr>
<tr>
<td></td>
<td></td>
<td></td>
<td>• collected blood lipid samples</td>
</tr>
<tr>
<td></td>
<td></td>
<td></td>
<td>• measured body weight, BMI, and % body fat</td>
</tr>
</tbody>
</table>
Study Population and Sample Selection

A total of 150 non-pregnant female participants were randomly selected from a population of 375 Caucasian and African-American low-income homemakers who were enrolled in EFNEP in three counties of Virginia. Each participant was assigned an identification number and the lottery method was used to randomly assign participants to one of two study groups. Seventy-six participants were assigned to the control group and 74 participants were assigned to the experimental group.

A sub-sample of 75 participants (38 Controls and 37 Experimentals), from whom physical and biochemical measures were collected, including measures of obesity and blood lipid profiles. This random sample represented 32% of the target EFNEP population of low-income African-American and Caucasian female homemakers. Prior to this study, participants had not involved in EFNEP nor any other nutrition education program.

Two primary sources of income for low-income families in these three counties were farming (especially of tobacco) and factory work in the production of textiles and tobacco products. EFNEP paraprofessionals had previously observed high rates of smoking, obesity, and excessive intake of fat among homemakers and other family members in the EFNEP program. It was believed that these factors would place this population at high-risk for both cancer and CVD.
This study was done in conjunction with a cancer prevention project (87) which was conducted in the three rural counties of Charlotte, Mecklenburg, and Pittsylvania (Appendix C). Table 2 shows the death rates from CVD in Charlotte, Mecklenburg, and Pittsylvania counties. The cancer-prevention project was initiated with the collection of pre-intervention data in July, 1992, and was conducted by Cox et al. (87). The current study was carried out in conjunction with the cancer study because many of the dietary risk factors for cancer are also risk factors for CVD (i.e. high intakes of fat and cholesterol and low intakes of fiber and calcium).

Data Collection

Permission was granted to conduct this study, as part of the EFNEP Nutrition-Based Cancer Prevention Project (87) by the University’s Institutional Review Board for Research Involving Human Subjects. Data on demographic characteristics, dietary intakes, and smoking were collected from 150 participants; while, measures of obesity and blood lipid profiles were obtained on a sub-sample of 75 participants, before and after the intervention phase. Using face-to-face interviews with participants in their homes, EFNEP paraprofessionals collected demographic, dietary, and smoking data. The biochemical measures were collected by four registered nurses; while, measures of obesity were collected by the author under the supervision of the State EFNEP Coordinator who is a registered dietitian and faculty member.
Table 2. Resident deaths due to major cardiovascular diseases in three EFNEP counties in Virginia, 1991

<table>
<thead>
<tr>
<th>County</th>
<th>Total Population</th>
<th>Cardiovascular Deaths</th>
<th></th>
<th></th>
<th></th>
<th></th>
</tr>
</thead>
<tbody>
<tr>
<td></td>
<td></td>
<td><strong>Males</strong></td>
<td><strong>Females</strong></td>
<td></td>
<td></td>
<td></td>
</tr>
<tr>
<td></td>
<td></td>
<td><strong>Caucasian</strong></td>
<td><strong>African-American</strong></td>
<td><strong>Caucasian</strong></td>
<td><strong>African-American</strong></td>
<td></td>
</tr>
<tr>
<td>Charlotte</td>
<td>11,688</td>
<td>24</td>
<td>11</td>
<td>16</td>
<td>11</td>
<td></td>
</tr>
<tr>
<td>Mecklenburg</td>
<td>29,241</td>
<td>54</td>
<td>38</td>
<td>62</td>
<td>22</td>
<td></td>
</tr>
<tr>
<td>Pittsylvania</td>
<td>55,672</td>
<td>97</td>
<td>42</td>
<td>81</td>
<td>29</td>
<td></td>
</tr>
</tbody>
</table>

During recruitment, the purposes of the study were explained by EFNEP paraprofessionals to homemakers and they were asked to sign informed consent forms (Appendix D). Participants were assured that confidentiality would be maintained on all information specifically relating to individuals in the study.

Pre-intervention data were collected on the 150 participants during August, September, and October, 1992. Oral and written instructions were provided to 75 participants on which physical and biochemical data were gathered, including the fact that they would be transported to the health department for blood drawing on designated days. Participants were instructed not to eat or drink anything (other than plain water after midnight) on the night prior to the clinic appointment during which blood samples were collected.

Post-intervention data were collected during April, May, and June, 1993, using the same procedures as at pre-intervention. Blood samples were drawn by the same registered nurses and measures of obesity were collected by the investigator. Specific data collected at pre- and post-intervention from 150 participants were as follows:

Demographic Data: age, race, income, educational level, and current work status.

Dietary Components: three random repeat 24-hour recalls (both at pre- and post-test) which were analyzed for average daily intake of energy, total fat
(TF as % kcal), saturated fatty acids (SFA as % kcal), monounsaturated fatty acids (MUFA as % kcal), polyunsaturated fatty acids (PUFA as % kcal), cholesterol, dietary fiber, sodium, potassium, calcium, the Milk Group (including yogurt and cheese), the Vegetable Group, the Fruit Group, the Bread/Cereal Group (including rice and pasta), the Meat Group (including beef, poultry, fish, dry beans, eggs, and nuts), and the Fats/Sweets Group.

In addition, the three 24-hour recalls were collected over a three-week period at pre- and post-intervention, with each food recall on a different day of the week. The food recalls were collected by EFNEP paraprofessionals according to standard procedures as described in “Procedures for Collecting 24-Hour Food Recalls” (Appendix E). Administering the 24-hour food recall involved asking the subject to recall all foods and beverages consumed in the previous 24 hours. Various probes and props were used by the paraprofessionals to elicit complete and accurate information from the homemaker in regard to the name and description of food items, amount eaten, fat, sugar, salt and other added condiments, and preparation methods. To increase the accuracy and representativeness of intakes of individual participants intake in this study, random-repeat food recalls were collected.

**Smoking:** smoking status/history and number of cigarettes smoked.

Smoking status/history especially cigarettes, was assessed using Health Risk Appraisal I (HRA-I) at pre-intervention and Health Risk Appraisal II (HRA II) at post-intervention. HRA-I and HRA-II were identical, except that HRA-I
contained items on family and medical history that were not repeated on HRA-II. EFNEP paraprofessionals administered the Family Record and Health Risk Appraisal forms during home visits using face-to-face interviews with participants.

On a sub-sample of 75 participants, additional data on physical and selected biochemical measures were collected. Height was measured using a standard stadiometer and body weight was measured using balance beam scales. A Futrex 5000 Body Fat Analyzer (Futrex Inc., P.O. Box 2398, Gaithersburg, MD 20886) was used to estimate percent body fat. The investigator of this study under the supervision of a registered dietitian measured height (inches), weight (pounds), and percent body fat; while, BMI (kg/m²) of each participant was calculated using height and weight.

Blood lipid values assessed included serum total cholesterol (TC), high-density lipoprotein cholesterol (HDL-C), and triglycerides. The equation, 
\[ \text{LDL-C} = (\text{TC} - \text{HDL-C}) - (\text{triglycerides}/5) \] was used to determine low-density lipoprotein cholesterol (LDL-C). TC:HDL-C ratios were also computed for each participant. For use in measuring lipid profiles, fasting blood samples were collected by nurses from the Massey Cancer Center in health department clinic rooms located in each of the three counties. A standard venipuncture blood drawing technique was used to collect blood samples (10-15 ml per homemaker) from the sub-sample of 75 participants. Blood samples were processed, refrigerated, and packed in a recommended manner and
shipped to the Department of Human Nutrition and Foods of the Virginia Polytechnic Institute and State University (VPI&SU), Blacksburg, Virginia, for blood lipid profile analysis. A description of the procedure for determination of blood lipid profiles is located in Appendix F.

Instrumentation

Instruments used to collect data on 150 participants at pre- and post-intervention, included the EFNEP Family Record (Part A = demographic and Part C = 24-hour recall), HRA-I, and HRA-II. With a sub-sample of 75 participants, an additional Physical Measurements Form was used (Appendix G) to record height, weight, age, and percent body fat. Instruments and other data collection methods are summarized in Table 3.

Intervention Phase

In this study, five female EFNEP paraprofessionals were involved in collecting data and in teaching lessons. Each paraprofessional worked with participants in both study groups. Their main role was to teach and assist the homemakers in reducing their risks for CVD. Emphasis was placed on improving diet and certain lifestyle behaviors. Other duties included recruiting participants, administering instruments, and scheduling homemakers to come into blood-drawing clinics at the health departments.

The paraprofessionals had worked in the EFNEP for five years, during which time they had received thorough training in basic nutrition. They had been previously hired as EFNEP paraprofessionals based on the perception
Table 3. Summary of instrumentation design of the study

<table>
<thead>
<tr>
<th>Instrument</th>
<th>Method</th>
<th>Data Collected</th>
<th>Expected Outcome</th>
</tr>
</thead>
<tbody>
<tr>
<td>EFNEP Family Record (Part A)</td>
<td>Face-to-face interview by EFNEP paraprofessionals</td>
<td>Baseline demographic characteristics of participants</td>
<td>Description of sample population</td>
</tr>
<tr>
<td>EFNEP Family Record (Part C)</td>
<td>Face-to-face interview by EFNEP paraprofessionals</td>
<td>Dietary intake for 24-hour periods before and after intervention</td>
<td>Description of nutrient and food groups intake of participants</td>
</tr>
<tr>
<td></td>
<td>Consisted of three 24-hour food recalls over three weeks; each recall</td>
<td></td>
<td>Description of certain CVD-related dietary risk factors of participants</td>
</tr>
<tr>
<td></td>
<td>on a different day; collected by EFNEP paraprofessionals</td>
<td></td>
<td>Significant improvement of participants’ nutrient and food group intake as a</td>
</tr>
<tr>
<td></td>
<td></td>
<td></td>
<td>result of the intervention</td>
</tr>
<tr>
<td>Health Risk Appraisal</td>
<td>Face-to-face interview by EFNEP paraprofessionals</td>
<td>Participants’ health, dietary habits, and smoking patterns related to CVD risks</td>
<td>Baseline description of participants’ general well-being related to CV health</td>
</tr>
<tr>
<td>I</td>
<td></td>
<td>before intervention</td>
<td></td>
</tr>
<tr>
<td>Instrument</td>
<td>Method</td>
<td>Data Collected</td>
<td>Expected Outcome</td>
</tr>
<tr>
<td>----------------------------------</td>
<td>------------------------------------------------------------------------</td>
<td>-------------------------------------------------------------------------------</td>
<td>---------------------------------------------------------------------------------</td>
</tr>
<tr>
<td>Health Risk Appraisal II</td>
<td>• Face-to-face interview by EFNEP paraprofessional</td>
<td>• Participants' smoking status/habits and use of cigarettes &amp; smokeless tobacco after intervention</td>
<td>• Improvement in smoking status/habits of participants</td>
</tr>
<tr>
<td>Physical Measurements Form</td>
<td>• Clinics held at local health depts., where measurements were taken by registered nurses, registered dietitian, &amp; investigator of study</td>
<td>• Measures of obesity of participants before and after intervention including: body weight, height, % body fat &amp; BMI</td>
<td>• Improvement in body weight, % body fat, &amp; BMI of participants</td>
</tr>
<tr>
<td>Blood Lipid Measurements</td>
<td>• Blood drawn from each participant by registered nurses during clinics held at local health depts.</td>
<td>• Levels of blood lipids for each homemaker: TC, HDL-C, LDL-C, triglycerides, &amp; ratio of TC/HDL-C</td>
<td>• Description of baseline blood lipid values of participants</td>
</tr>
</tbody>
</table>
they exhibited enthusiasm and interest in providing nutrition education to that disadvantaged individuals and families.

For the purposes of this study, the paraprofessionals received training during a four-day workshop in June, 1992. Training sessions were organized and taught by the State EFNEP Coordinator, who has a doctorate in nutrition, and four registered nurses from the Massey Cancer Center, Richmond, Virginia. Tests on nutritional knowledge of the paraprofessionals were conducted at the beginning and end of the training to insure that they had gained a sufficient background on subject matter and study methods. Each paraprofessional was provided with a Technicians’ Manual which included instruments, instructions on completing them, lesson materials, and study procedures in which they would be involved.

**Intervention Program.** The intervention consisted of a nutrition education program, lasting about 6 months during which participants in the Experimental Group (Group EC) were taught 18 lessons, 11 of which contained information related to prevention of heart disease. The 76 Control Group (Group C) participants received money-management lessons, but no information on nutrition or dietary risk of CVD. The purpose of teaching lessons to control participants was to facilitate contact with them and to provide an incentive for them to remain in the study.

The 18 lessons taught to Group EC included nine lessons from the standard EFNEP curriculum, Eating Right is Basic-2 (ERIB-2), published by the
Michigan Cooperative Extension EFNEP Program (28). The other nine lessons were newly designed lessons on cancer and CVD prevention, each consisting of a Lesson Reference, a Cue Card (the lesson outline), a Flip chart (with discussion information on the back), and handouts for the participants. Most lessons were taught by EFNEP paraprofessionals on an individual basis during home visits; while, some were taught during group meetings. Lesson delivery took 1 to 1.5 hours, including presentation of information and food demonstrations. Efforts were made to involve participants in discussion and hands-on learning activities. Following is a brief description of the lessons relating to CVD prevention and sequence in which lessons were taught:

**Lesson 1 - Cancer and CVD Prevention.** This lesson covered how smoking, use of smokeless tobacco, and drinking of alcoholic beverages increases the risk for cancer and CVD. Participants were urged to reduce or stop the use of these products and suggestions were given for smoking cessation.

**Lesson 2 - Calcium and Health.** This lesson contained information about the role of calcium in the diet, best food sources of calcium, recommended dietary servings, and blood levels of calcium in the human body at different ages. Emphasis was placed on the fact that women are at greatest risk of developing calcium deficiency, which may result in conditions such as osteoporosis, cancer, and hypertension. The protective role of calcium in CVD relates to its function in regulating blood pressure.
Lesson 3 - Milk and Cheese. This lesson deals with number of servings of milk and cheese (including low-fat choices) needed at different age groups. Participants were also taught how to prepare milk and cheese foods using low-calorie recipes. The importance of milk and cheese as the primary source of calcium was emphasized because of its role in regulating blood pressure.

Lesson 4 - Fiber-Know The Facts. This lesson provided information to participants on what dietary fiber, the importance of dietary fiber to health, foods that are good sources of fiber, and ways to increase fiber in meals and snacks. Ways in which dietary fiber may reduce the risk and severity of CVD and its protective role in weight reduction were emphasized. Participants were also taught how to prepare high-fiber foods and select high-fiber meals and snacks.

Lesson 5 - Bread, Cereals, Rice, and Pasta. In this lesson, participants were taught various ways of preparing bread, cereals, rice, and pasta foods, and were encouraged to try other foods that they have not used before. It also included activities to inform participants of the nutritional qualities of this food group including the low calorie content when served plain. A practical aspect in this lesson was how to compare the price (cost/serving) and to recognize similarities and differences in the nutrient content of several bread, cereal, rice, and pasta products. Emphases were given to the use of "enriched" and "high-fiber" breads and cereals, ready-to-eat cereals without
added sugar, and inexpensive store brands which have the same nutritional quality of national brands.

**Lesson 6 - Dried Beans, Peas, Protein Pairs.** In this lesson, participants were instructed on techniques for using and preparing different dried beans and peas. It also covered how grains could be combined with legumes to make high quality protein, as well as how milk could be combined with grains to increase the protein value. This lesson relates to prevention of CVD, because legumes are high in fiber and low in fat. If used for meals, in place of meat, they could help lower blood cholesterol levels.

**Lesson 7 - Cut the Fat.** During this lesson, participants learned about high fat intake and its possible consequences with regard to heart disease, obesity, elevated blood pressure, stroke, diabetes, some types of cancer, joint and back problems, and gallbladder disease. Participants became aware of the importance of reducing daily fat intake to recommended levels and how to use commonly available low-fat foods and ingredients. Several methods of low-fat food preparation were demonstrated.

**Lesson 8 - Meat, Poultry, Fish, and Eggs.** In this lesson participants were taught ways to prepare and cook meat, poultry, fish, and eggs utilizing simple low-fat recipes and techniques. The idea of having less meat in meals and substituting combination meat dishes and dried beans and peas was highly emphasized. Also, participants were encouraged to eat fish and poultry without the skin, to reduce the intake of fat and cholesterol.
Lesson 9 - Fruits. In this lesson, participants learned about nutrients in fruits and how to select, store, and serve fruits. Participants were urged to eat at least two servings of fruit each day, especially fresh fruits, to reduce risk of CVD and cancer. Fruit consumption relates to reduced CVD risks because fruits are high in fiber and potassium, but low in fat.

Lesson 10 - Vegetables. In this lesson, participants were educated about the nutrients available in vegetables and several methods in selecting, storing, and serving vegetables. Participants were encouraged to consume at least three servings of vegetables each day, to reduce risk of CVD and cancer. Participants were urged to eat vegetables, without added salt or fat, and to have larger servings of vegetables at meals, while reducing meat servings. Vegetables relate to reduced CVD risks because they have high-fiber and potassium content, but are low in calories and fat, thereby, contributing to the maintenance of normal weight and good health.

Lesson 11 - Putting It All Together. In this lesson, the focus was on teaching the participants how to incorporate the knowledge and experience they had gained in the previous lessons into their daily activities. A major portion of this lesson was about meal planning and incorporated the use of a weekly meal planner, ERIB-2 Food stickers, sample weekly menus, and high-fiber, low-fat recipes.

Throughout the lesson series, information and skills related to dietary risks of CVD were taught, however, no lesson specifically addressed sodium
and potassium intake. On the other hand, there was emphasis throughout the series on reducing processed meats, eating more fruits and vegetables, and adding less salt to vegetables, meats, and other foods. Hence, if participants were to adopt those suggestions, sodium intake would decrease and potassium intake would increase.

Data Analysis

Analysis of data was performed by the investigator of this study. Demographic data were used to describe and compare the participants by race (Caucasians and African-Americans) at pre-intervention. Data on measured variables (dietary intake, measures of obesity, biochemical measures, and smoking) were used to describe and compare the group of participants by race and to divide them into different CVD-related risk categories prior to intervention. Data on the average intakes/levels of the same measured variables and the changes that had occurred as a result of the intervention were used to describe and compare the participants by study groups (Experimental and Control) by race.

Dietary Analysis. Twenty-four-hour food recalls were analyzed using the Nutritionist III computer program (Nutritionist III, Version 7.0, N-Squared Computing Analytic Software, 1991). The three random-repeat 24-hour food recalls on each of the 150 participants were averaged to provide daily intakes/servings of the following: (a) energy (kcal), (b) TF (% kcal), (c) SFA (% kcal), (d) MUFA (% kcal), (e) PUFA (% kcal), (f) cholesterol, (g) dietary
fiber, (h) sodium, (i) potassium, (j) calcium, (k) the Milk Group, (l) the Vegetable Group, (m) the Fruit Group, (n) the Bread/Cereal Group, (o) the Meat Group in ounces, and (p) the Fats/Sweets Group. At pre-intervention, data on dietary intakes by race were used to divide the participants into three levels of adequacy: below-recommended, recommended, and above-recommended. Intakes of the measured nutrients and food groups were compared on the basis that excess or inadequate amounts may increase the risk of CVD. The distribution and intakes of the participants by race were compared with various standards of adequacy and associated health risks (14, 18, 22, 29, 49, 59, 89).

Data on change with regard to certain dietary CVD risk factors from pre- to post-intervention was compared to determine significant differences as a result of the intervention, between study groups (Experimental and Control). Table 4 shows the Recommended Dietary Allowances (RDA’s) or other recommended intakes for each of the nutrients and food groups assessed in this study.

Measures of Obesity Analysis. Levels of body fat were estimated using percent body fat and body mass index (BMI). Percent body fat was used to reflect the regional distribution of body fat; whereas, BMI was used as an indicator of overall adiposity (14). Pre-intervention data on the levels of percent body fat and BMI values, for a sub-sample of 75 participants, were used to compare the two races (10 Caucasians and 65 African-Americans).
<table>
<thead>
<tr>
<th>Nutrients/Food Groups</th>
<th>Recommended Intakes</th>
</tr>
</thead>
<tbody>
<tr>
<td>Energy (kcal)</td>
<td>1600 - 2200 kcal</td>
</tr>
<tr>
<td>Total Fat (% kcal)</td>
<td>&lt; 30% kcal</td>
</tr>
<tr>
<td>Saturated Fat (% kcal)</td>
<td>&lt; 10% kcal</td>
</tr>
<tr>
<td>Mono-unsaturated Fat (% kcal)</td>
<td>10% - 15% kcal</td>
</tr>
<tr>
<td>Poly-unsaturated Fat (% kcal)</td>
<td>up to 10% kcal</td>
</tr>
<tr>
<td>Cholesterol (mg)</td>
<td>&lt; 300 mg</td>
</tr>
<tr>
<td>Dietary Fiber (gms)</td>
<td>20 - 35 gms</td>
</tr>
<tr>
<td>Sodium (mg)</td>
<td>1800 - 2400 mg</td>
</tr>
<tr>
<td>Potassium (mg)</td>
<td>1600 - 3500 mg</td>
</tr>
<tr>
<td>Calcium (mg)</td>
<td>800 - 1200 mg</td>
</tr>
<tr>
<td>The Milk Group (servings)</td>
<td>2 - 3 servings</td>
</tr>
<tr>
<td>The Vegetable Group (servings)</td>
<td>3 - 5 servings</td>
</tr>
<tr>
<td>The Fruit Group (servings)</td>
<td>2 - 4 servings</td>
</tr>
<tr>
<td>The Bread/Cereals Group (serving)</td>
<td>6 - 11 servings</td>
</tr>
<tr>
<td>The Meat Group (ounces)</td>
<td>5 - 7 ounces</td>
</tr>
<tr>
<td>The Fats/Sweets Group (servings)</td>
<td>≤ 10 servings</td>
</tr>
</tbody>
</table>

a Based on recommendations of the National Cancer Institute (49)

b 1 Serving Milk = 1 cup of milk or yogurt, 1.5 ozs. of natural cheese
c 1 Serving Vegetables = 1 cup of raw leafy greens, 1/2 cup cooked
d 1 Serving Fruits = 1 medium piece, 1/2 cup chopped or cooked or canned fruit
e 1 Serving Bread/Cereals = 1 slice, 1 oz. dry cereal, 1/2 cup cooked cereal
f 1 Ounce of Meat = 1/2 cup of cooked or dried beans, 1 egg, 2 tbs. peanut butter
g 1 Serving of Fats/Sweets = 1 tsp. butter, 1/2 tbs. salad dressing, 2 tsp. sugar or jelly, 3 ozs. soda
With regard to percent body fat, participants were classified into groups one of the five categories (poor, fair, average, good, and excellent) based on the normal distribution for predicting body density among women, as compared with norms for females at various ages (90, Table 5). For BMI, participants were divided into BMI categories (70, Table 6) based on the association of obesity and disease-risk. The mean values of percent body fat and BMI during pre- and post-intervention and differences in changes presented between experimental and control groups are shown.

Biochemical Analysis. Lipid profiles (TC, HDL-C, and triglycerides) for the sub-sample of 75 participants (10 Caucasians and 65 African-Americans) were done on each blood sample using the Milton Roy Spectronic 1001 Plus; while, LDL-C and TC:HDL-C ratio were calculated. Analyses of blood samples were performed by the investigator, under the supervision of a qualified laboratory technician in the Department of Human Nutrition and Foods at Virginia Polytechnic Institute & State University. Quantitative-Enzymatic-Calorimetric Assays by manual technique were used to determine TC, HDL-C, and triglyceride serum levels (Appendix G). Equations based on the Stanbio HDL-C Enzymatic Procedures were used to calculate LDL-C and TC:HDL-C ratios. Each analysis was performed in duplicate with the average of blood lipid components being used in comparisons between the race and study groups. Blood analyses were performed within 2-3 days after blood drawing clinics. Standards were used for evaluating the pre-intervention
Table 5. Standard values for percent body fat for women

<table>
<thead>
<tr>
<th>Rating</th>
<th>20 - 29</th>
<th>30 - 39</th>
<th>40 - 49</th>
<th>50 - 59</th>
<th>60 +</th>
</tr>
</thead>
<tbody>
<tr>
<td>Excellent</td>
<td>&lt; 16</td>
<td>&lt; 17</td>
<td>&lt; 18</td>
<td>&lt; 19</td>
<td>&lt; 20</td>
</tr>
<tr>
<td>Good</td>
<td>16 - 19</td>
<td>17 - 20</td>
<td>18 - 21</td>
<td>19 - 22</td>
<td>20 - 23</td>
</tr>
<tr>
<td>Average</td>
<td>20 - 28</td>
<td>21 - 29</td>
<td>22 - 30</td>
<td>23 - 31</td>
<td>24 - 32</td>
</tr>
<tr>
<td>Fair</td>
<td>29 - 31</td>
<td>30 - 32</td>
<td>31 - 33</td>
<td>32 - 34</td>
<td>33 - 35</td>
</tr>
<tr>
<td>Poor</td>
<td>&gt; 31</td>
<td>&gt; 32</td>
<td>&gt; 33</td>
<td>&gt; 34</td>
<td>&gt; 35</td>
</tr>
</tbody>
</table>

### Table 6. Body mass index categories

<table>
<thead>
<tr>
<th>Categories</th>
<th>Body Mass Index (kg/m(^2))</th>
</tr>
</thead>
<tbody>
<tr>
<td>Acceptable Range (Low disease risk/mortality)</td>
<td>20.0 - 25.0</td>
</tr>
<tr>
<td>Mildly Overweight (increased disease risk/mortality)</td>
<td>25.1 - 27.0</td>
</tr>
<tr>
<td>Moderately Overweight/Obese</td>
<td>27.1 - 30.0</td>
</tr>
<tr>
<td>Markedly Overweight/Obese</td>
<td>30.1 - 40.0</td>
</tr>
<tr>
<td>Morbidly Obese</td>
<td>&gt; 40.0</td>
</tr>
</tbody>
</table>

**Note.** From "Recommended Guidelines for Body Composition in Cardiac Rehabilitation" by D. Verill et al., 1994, *Journal of Cardiopulmonary Rehabilitation, 14*, p. 116. Reprinted by permission.
values of individual participants and were used for classifying them by race into groups based on the categories listed in Tables 7-9. The change in mean blood lipid values from pre- to post-intervention were used to compare the two study groups and to assess the results of the intervention.

Smoking Analysis. Smoking history and habits were ascertained by standardized questions on HRA-I and HRA-II. Classification of the 150 participants into groups was based on the following: (1) Current Smokers, (2) Non-Smokers, and (3) Ex-Smokers. At pre-intervention, data on smoking was used to describe and compare the smoking status and patterns among Caucasian and African-American participants. The distribution of the participants in the study groups by smoking status at pre- and post-intervention and the average number of cigarettes used among current smokers were used to describe the changes that had occurred as a result of the intervention.

Statistical Analysis. Statistical analyses were conducted using SAS-PC Software (Version 6.04, SAS Institute Incorporated, Cary, North Carolina). Descriptive statistics involving measures of central tendency and variations were used in this study to: (a) analyze the demographic data for the purpose of describing the sample population of low-income EFNEP women by race, and (b) describe and compare the sample population in both study groups in relation to the changes from pre- to post-intervention on diet-related risk factors for CVD and smoking. This statistical method provided an indication
Table 7. Standard values for TC, LDL-C, and triglyceride levels

<table>
<thead>
<tr>
<th>Variable</th>
<th>Desirable Levels</th>
<th>Borderline-High Levels</th>
<th>High Levels</th>
</tr>
</thead>
<tbody>
<tr>
<td>TC (mg/dl)a</td>
<td>&lt; 200</td>
<td>200 - 239</td>
<td>≥ 240</td>
</tr>
<tr>
<td>LDL-C (mg/dl)b</td>
<td>&lt; 130</td>
<td>130 - 159</td>
<td>≥ 160</td>
</tr>
<tr>
<td>Triglyceridesc (mg/dl)</td>
<td>&lt; 200</td>
<td>200 - 400</td>
<td>&gt; 400</td>
</tr>
</tbody>
</table>

aTC indicates serum total cholesterol. High TC levels is a value above which risk for CHD rises more steeply, and corresponds to the 80th percentile of the adult U.S. population (NHANES III).

bLDL-C indicates low-density lipoprotein cholesterol.

cBorderline-high and high triglyceride levels do not normally indicate direct risk, but may reflect lipoprotein abnormalities associated with CVD. High triglycerides also occur in conditions such as kidney disease and diabetes, which suggest a high CVD risk.

Table 8. Standard HDL-C values

<table>
<thead>
<tr>
<th>Blood Level Categories</th>
<th>HDL-C (mg/dl)</th>
</tr>
</thead>
<tbody>
<tr>
<td>High(^a)</td>
<td>≥ 60</td>
</tr>
<tr>
<td>(negative disease risk)</td>
<td></td>
</tr>
<tr>
<td>Moderate</td>
<td>35 - 59</td>
</tr>
<tr>
<td>(acceptable disease risk)</td>
<td></td>
</tr>
<tr>
<td>Low(^b)</td>
<td>&lt; 35</td>
</tr>
<tr>
<td>(increase disease risk)</td>
<td></td>
</tr>
</tbody>
</table>

\(^a\)According to the 1993 NCEP Report (78), a high HDL-C appears to be protective against CHD and such levels can be called a negative risk factor.

\(^b\)A low HDL-C was classified as a major risk factor for CHD.

Table 9. Standards of TC/HDL-C ratio and risk of CVD for females

<table>
<thead>
<tr>
<th>Risk Categories</th>
<th>TC:HDL-C Ratio</th>
</tr>
</thead>
<tbody>
<tr>
<td>Half Risk</td>
<td>3.27</td>
</tr>
<tr>
<td>Normal Risk</td>
<td>4.44</td>
</tr>
<tr>
<td>Double Risk</td>
<td>7.05</td>
</tr>
<tr>
<td>Triple Risk</td>
<td>11.04</td>
</tr>
</tbody>
</table>

of the central point around which the data were located, how broadly the data were spread, and how the data were related in terms of several characteristics. Baseline differences of the participants, by race, on measured variables were analyzed by two-sample t-tests.

Inferential statistics were utilized to test the null hypotheses in this study. Two-sample t-tests and the General Linear Models (GLM) procedure for unbalanced ANOVA were employed to determine significant interactions between study groups by race, to compare the changes in diet-related CVD risk factors and smoking between study groups, and to determine if the obtained values were significantly different. Since the sample group sizes by race were unbalanced (47 Caucasians and 103 African-Americans) and the sub-sample sizes by study groups (37 Experimental and 38 Controls), were fairly small, it was determined that a relatively large difference in group means would be required for statistical analysis to show a significant difference at an alpha level of 0.05.
CHAPTER IV

RESULTS AND DISCUSSION

Demographic Characteristics of Participants

The demographic characteristics of the 150 low-income participants are presented in Table 10. Forty-seven (31%) of the participants were Caucasian and 103 (69%) were African-American. The Caucasian participants were almost evenly distributed with 24 in the experimental and 23 in the control group. Among the African-American women, 50 (49%) were in the experimental and 53 (51%) were in the control group. Based on the 1992 Virginia state EFNEP report, the sample was representative of the population of EFNEP homemakers in Virginia on race.

Sixty-five percent of Caucasians (30 of 47 individuals) had completed 12th grade or had earned a graduate equivalency diploma (GED), compared with 39% (40 individuals) of African-Americans. On the other hand, 46% of the African-American participants (47 of 103 individuals) had completed the ninth to eleventh grade. Thus, the Caucasian participants had a significantly higher level of education than the African-Americans (P = 0.005).

The mean monthly household income for Caucasians was estimated at $670.00, while that of African-Americans was approximately $450.00. Caucasians had a significantly greater monthly household income than the African-Americans (P = 0.001). A majority of both the Caucasian (70%) and African-American (63%) participants were unemployed at the time of the
Table 10. Demographic profile of EFNEP study participants by race (N = 150)

<table>
<thead>
<tr>
<th>Variable</th>
<th>Caucasian</th>
<th></th>
<th>African-American</th>
<th></th>
<th>P value</th>
</tr>
</thead>
<tbody>
<tr>
<td></td>
<td>n</td>
<td>%</td>
<td>n</td>
<td>%</td>
<td></td>
</tr>
<tr>
<td><strong>Group</strong></td>
<td></td>
<td></td>
<td></td>
<td></td>
<td></td>
</tr>
<tr>
<td>Experimental</td>
<td>24</td>
<td>51</td>
<td>50</td>
<td>49</td>
<td>0.775</td>
</tr>
<tr>
<td>Control</td>
<td>23</td>
<td>49</td>
<td>53</td>
<td>51</td>
<td></td>
</tr>
<tr>
<td><strong>Educational Level</strong></td>
<td></td>
<td></td>
<td></td>
<td></td>
<td>0.005**</td>
</tr>
<tr>
<td>≤ 8th Grade</td>
<td>1</td>
<td>2</td>
<td>5</td>
<td>5</td>
<td></td>
</tr>
<tr>
<td>9th-11th Grade</td>
<td>8</td>
<td>17</td>
<td>47</td>
<td>46</td>
<td></td>
</tr>
<tr>
<td>12th / GED</td>
<td>30</td>
<td>65</td>
<td>40</td>
<td>39</td>
<td></td>
</tr>
<tr>
<td>&gt; High school</td>
<td>7</td>
<td>15</td>
<td>10</td>
<td>10</td>
<td></td>
</tr>
<tr>
<td><strong>Current Work Status</strong></td>
<td></td>
<td></td>
<td></td>
<td></td>
<td>0.405</td>
</tr>
<tr>
<td>Student</td>
<td>1</td>
<td>2</td>
<td>6</td>
<td>6</td>
<td></td>
</tr>
<tr>
<td>Employed</td>
<td>13</td>
<td>28</td>
<td>26</td>
<td>25</td>
<td></td>
</tr>
<tr>
<td>Homemaker/Unemployed</td>
<td>33</td>
<td>70</td>
<td>65</td>
<td>63</td>
<td></td>
</tr>
<tr>
<td>Disabled</td>
<td>0</td>
<td>0</td>
<td>5</td>
<td>5</td>
<td></td>
</tr>
<tr>
<td>Other</td>
<td>0</td>
<td>0</td>
<td>1</td>
<td>1</td>
<td></td>
</tr>
<tr>
<td><strong>Residence</strong></td>
<td></td>
<td></td>
<td></td>
<td></td>
<td>0.729</td>
</tr>
<tr>
<td>Farm</td>
<td>8</td>
<td>17</td>
<td>21</td>
<td>20</td>
<td></td>
</tr>
<tr>
<td>Town (&lt;10,000)</td>
<td>30</td>
<td>64</td>
<td>57</td>
<td>55</td>
<td></td>
</tr>
<tr>
<td>Place (10,000-50,000)</td>
<td>9</td>
<td>19</td>
<td>24</td>
<td>23</td>
<td></td>
</tr>
<tr>
<td>Suburbs (&gt;50,000)</td>
<td>0</td>
<td>0</td>
<td>1</td>
<td>1</td>
<td></td>
</tr>
<tr>
<td><strong>Mean Age in Years</strong></td>
<td>29.9 ± 1.4c</td>
<td></td>
<td>28.1 ± 0.7</td>
<td></td>
<td>0.238</td>
</tr>
<tr>
<td>**Mean Monthly Income ($)**b</td>
<td>670.6 ± 58.3</td>
<td></td>
<td>448.7 ± 27.5</td>
<td></td>
<td>0.001***</td>
</tr>
<tr>
<td><strong>Mean Children/Household</strong></td>
<td>2.0 ± 0.2</td>
<td></td>
<td>2.2 ± 0.1</td>
<td></td>
<td>0.354</td>
</tr>
<tr>
<td><strong>Mean Family Members</strong></td>
<td>3.6 ± 0.2</td>
<td></td>
<td>3.5 ± 0.1</td>
<td></td>
<td>0.492</td>
</tr>
</tbody>
</table>

*< 0.05, **< 0.01, ***< 0.001
aHighest educational level attained
bMean monthly family income
cMean ± standard error of the mean (SEM)
study, with only 28% of Caucasians and 25% of African-Americans being employed.

The mean age for Caucasians was almost 30 years; whereas, 28 years was the mean age for African-Americans. Both race groups had an average of approximately 2 children and 3.5 members per household. Eighty-one percent of the Caucasians (38 individuals) and 75% of the African-Americans (78 individuals) resided in towns with a population of less than 10,000 or on farms. The remaining 9 Caucasians (19%) and 24 African-Americans (23%) lived in one city of about 53,000 population.

In summary, no significant differences were detected between race groups on age, current work status, residence, number of children/household, and number of family members/household. However, the two race groups differed significantly on education level and income.

Cardiovascular Risks of Participants at Pre-intervention

Nutrient and Food Group Intake. Intake of selected nutrients and food groups related to cardiovascular health are presented in Table 11, including energy, fats, cholesterol, fiber, sodium, potassium, calcium, the Milk Group (including yogurt and cheese), the Vegetable Group, the Fruit Group, the Bread/Cereal Group (including rice and pasta), the Meat Group (including beef, poultry, fish, dry beans, eggs, and nuts), and the Fats/Sweets Group. Dietary data are presented for participants by race at three levels of adequacy (below-recommended, recommended, and above-recommended).
Table 11. Intakes of selected nutrients and food groups and comparisons between Caucasian and African American, low-income women at pre-intervention (N = 47 Caucasians and 103 African-Americans)

<table>
<thead>
<tr>
<th>Nutrient or Food Component</th>
<th>Race Category</th>
<th>Below Recommended</th>
<th>Recommended</th>
<th>Above Recommended</th>
<th>Mean</th>
<th>P value</th>
</tr>
</thead>
<tbody>
<tr>
<td></td>
<td></td>
<td>n</td>
<td>%</td>
<td>n</td>
<td>%</td>
<td>n</td>
</tr>
<tr>
<td>Kilocalories</td>
<td>Caucasian</td>
<td>25</td>
<td>53</td>
<td>10</td>
<td>21</td>
<td>12</td>
</tr>
<tr>
<td></td>
<td>African-American</td>
<td>52</td>
<td>51</td>
<td>33</td>
<td>32</td>
<td>18</td>
</tr>
<tr>
<td>Total Fat (% kcal)</td>
<td>Caucasian</td>
<td>0</td>
<td>0</td>
<td>17</td>
<td>36</td>
<td>30</td>
</tr>
<tr>
<td></td>
<td>African-American</td>
<td>0</td>
<td>0</td>
<td>17</td>
<td>17</td>
<td>86</td>
</tr>
<tr>
<td>SFA (% kcal)</td>
<td>Caucasian</td>
<td>0</td>
<td>0</td>
<td>26</td>
<td>55</td>
<td>21</td>
</tr>
<tr>
<td></td>
<td>African-American</td>
<td>0</td>
<td>0</td>
<td>32</td>
<td>31</td>
<td>71</td>
</tr>
<tr>
<td>MUFA (% kcal)</td>
<td>Caucasian</td>
<td>14</td>
<td>30</td>
<td>27</td>
<td>57</td>
<td>6</td>
</tr>
<tr>
<td></td>
<td>African-American</td>
<td>18</td>
<td>18</td>
<td>63</td>
<td>61</td>
<td>22</td>
</tr>
<tr>
<td>PUFA (% kcal)</td>
<td>Caucasian</td>
<td>0</td>
<td>0</td>
<td>44</td>
<td>94</td>
<td>3</td>
</tr>
<tr>
<td></td>
<td>African-American</td>
<td>0</td>
<td>0</td>
<td>99</td>
<td>96</td>
<td>4</td>
</tr>
<tr>
<td>Cholesterol (mg)</td>
<td>Caucasian</td>
<td>0</td>
<td>0</td>
<td>32</td>
<td>68</td>
<td>15</td>
</tr>
<tr>
<td></td>
<td>African-American</td>
<td>0</td>
<td>0</td>
<td>64</td>
<td>62</td>
<td>39</td>
</tr>
</tbody>
</table>
Table 11. Continued

<table>
<thead>
<tr>
<th>Nutrient or Food Component</th>
<th>Race Category</th>
<th>Below Recommended</th>
<th>Recommended</th>
<th>Above Recommended</th>
<th>Mean</th>
<th>P value</th>
</tr>
</thead>
<tbody>
<tr>
<td>Dietary Fiber (gm)</td>
<td>Caucasian</td>
<td>46 98</td>
<td>1 2</td>
<td>0 0</td>
<td>8.7 ± 0.6</td>
<td></td>
</tr>
<tr>
<td></td>
<td>African-American</td>
<td>103 100</td>
<td>0 0</td>
<td>0 0</td>
<td>8.4 ± 0.4</td>
<td>0.731</td>
</tr>
<tr>
<td>Sodium (mg)</td>
<td>Caucasian</td>
<td>10 21</td>
<td>7 15</td>
<td>30 64</td>
<td>2613 ± 147</td>
<td></td>
</tr>
<tr>
<td></td>
<td>African-American</td>
<td>24 23</td>
<td>24 23</td>
<td>55 53</td>
<td>2539 ± 91</td>
<td>0.660</td>
</tr>
<tr>
<td>Potassium (mg)</td>
<td>Caucasian</td>
<td>21 45</td>
<td>25 53</td>
<td>1 2</td>
<td>1820 ± 108</td>
<td></td>
</tr>
<tr>
<td></td>
<td>African-American</td>
<td>47 46</td>
<td>51 50</td>
<td>5 5</td>
<td>1846 ± 78</td>
<td>0.852</td>
</tr>
<tr>
<td>Calcium (mg)</td>
<td>Caucasian</td>
<td>42 89</td>
<td>3 6</td>
<td>2 4</td>
<td>521 ± 44</td>
<td></td>
</tr>
<tr>
<td></td>
<td>African-American</td>
<td>92 89</td>
<td>8 8</td>
<td>3 3</td>
<td>489 ± 27</td>
<td>0.510</td>
</tr>
<tr>
<td>The Milk Group (servings)</td>
<td>Caucasian</td>
<td>44 94</td>
<td>2 4</td>
<td>1 2</td>
<td>0.5 ± 0.1</td>
<td></td>
</tr>
<tr>
<td></td>
<td>African-American</td>
<td>96 93</td>
<td>7 7</td>
<td>0 0</td>
<td>0.4 ± 0.1</td>
<td>0.495</td>
</tr>
<tr>
<td>The Vegetable Group (servings)</td>
<td>Caucasian</td>
<td>47 100</td>
<td>0 0</td>
<td>0 0</td>
<td>0.8 ± 0.1</td>
<td></td>
</tr>
<tr>
<td></td>
<td>African-American</td>
<td>101 98</td>
<td>2 2</td>
<td>0 0</td>
<td>0.7 ± 0.1</td>
<td>0.898</td>
</tr>
<tr>
<td>Nutrient or Food Component</td>
<td>Race Category</td>
<td>Below Recommended</td>
<td>Recommended</td>
<td>Above Recommended</td>
<td>Mean</td>
<td>P value</td>
</tr>
<tr>
<td>----------------------------</td>
<td>---------------------</td>
<td>-------------------</td>
<td>-------------</td>
<td>-------------------</td>
<td>------------</td>
<td>---------</td>
</tr>
<tr>
<td></td>
<td></td>
<td>n</td>
<td>%</td>
<td>n</td>
<td>%</td>
<td>n</td>
</tr>
<tr>
<td>The Fruit Group (servings)</td>
<td>Caucasian</td>
<td>31</td>
<td>91</td>
<td>3</td>
<td>9</td>
<td>0</td>
</tr>
<tr>
<td></td>
<td>African-American</td>
<td>63</td>
<td>84</td>
<td>7</td>
<td>9</td>
<td>5</td>
</tr>
<tr>
<td>The Bread/Cereals Group (servings)</td>
<td>Caucasian</td>
<td>9</td>
<td>19</td>
<td>31</td>
<td>66</td>
<td>7</td>
</tr>
<tr>
<td></td>
<td>African-American</td>
<td>34</td>
<td>33</td>
<td>56</td>
<td>54</td>
<td>13</td>
</tr>
<tr>
<td>The Meat Group (ounces)</td>
<td>Caucasian</td>
<td>25</td>
<td>53</td>
<td>14</td>
<td>30</td>
<td>8</td>
</tr>
<tr>
<td></td>
<td>African-American</td>
<td>30</td>
<td>29</td>
<td>44</td>
<td>43</td>
<td>29</td>
</tr>
<tr>
<td>The Fat/Sweets Group (servings)</td>
<td>Caucasian</td>
<td>0</td>
<td>0</td>
<td>30</td>
<td>64</td>
<td>17</td>
</tr>
<tr>
<td></td>
<td>African-American</td>
<td>0</td>
<td>0</td>
<td>60</td>
<td>58</td>
<td>43</td>
</tr>
</tbody>
</table>

* ≤ 0.05

a Mean ± standard error of the mean (SEM)
Intakes of measured nutrients and food groups were compared to standards based on recent recommended allowances and estimated safe and adequate daily dietary intakes from various authorized health agencies (Table 4, Chapter III).

Before intervention, the Caucasian participants had a mean energy intake of 1750 kcal, which is on the lower end of the recommended range (1600-2200 kcal). Twenty-five Caucasians (53%) had energy intakes below recommended and only 21% had energy intakes within recommended levels. Twelve Caucasians (26%) consumed more than 2200 kcal per day. Thirty-three African-Americans (32%) had recommended levels of energy intake; whereas, a high percentage (51%) were below-recommended. Only 18% had caloric intakes greater than 2200 kcal. The mean energy intake was 1714 kcal for African-Americans, which was slightly lower than that of Caucasians; however, the difference was not significant.

Thirty of 47 Caucasians (64%) had total fat (TF % kcal) intakes of more than 30% of kilocalories, while the remaining 36% (17 individuals) consumed fat within recommended range (<30% of kcal/day). Among the African-Americans, 84% (86 individuals) consumed fat above-recommended levels and only 17% had TF intakes within recommended range. Caucasians had a significantly lower fat intake than African-Americans (P<0.05). Thus, high TF intakes may pose as a risk of CVD for a majority of the study participants in both race groups, but is more prevalent for African-Americans.
For saturated fatty acids (SFA as %kcal), 21 Caucasians (45%) and 71 African-Americans (69%) had intakes above the recommended level of <10% kcal. The remaining 55% of Caucasians and 31% of African-Americans consumed SFA at recommended levels. There was no significant difference in the mean SFA intake of the two groups. It appears that high SFA intakes pose a risk of CVD for substantial numbers of low-income women of both race groups, though the risk may be greater for African-American women in the EFNEP population.

For monounsaturated fatty acids (MUFA as % kcal), lower levels (<10% kcal) are considered to be a risk for CVD; while, for polyunsaturated fatty acids (PUFA as % kcal), increased intakes not exceeding 10% kcal are associated with reduced risk of atherosclerotic CVD (7). A majority of Caucasians (57% + 13%) consumed the recommended range or above for MUFA and 100% of Caucasian participants (94% + 6%) consumed the recommended or above levels of PUFA. For the African-American participants, 85 (82%) consumed recommended or higher levels of MUFA.

All 103 of the African-American participants (96% + 4%) consumed recommended or higher levels of PUFA. Thus, low intakes of MUFA and PUFA did not pose a risk of CVD for either the Caucasian or the African-American participants in this study. However, Caucasian participants had a significantly lower MUFA intake than the African-Americans (P<0.05); while, there was no significant difference by race on PUFA intake.
Of the 47 Caucasian homemakers, 68% (32 individuals) had cholesterol intakes within recommended levels, while only 15 Caucasians (32%) consumed more than 300 mg/day of cholesterol. Mean cholesterol intake for the Caucasians was 253 mg as compared with the recommended level of <300 mg. Among African-Americans, 62% (64 individuals) had within-recommended cholesterol intakes, while the remaining 38% (39 individuals) had above-recommended intakes. The mean cholesterol intake for African-Americans of 291 mg/day was higher than that of Caucasians, but the difference was not significant. Thus, high cholesterol intake did not appear to pose a risk of CVD for either the Caucasian or African-American participants at pre-intervention.

Almost all Caucasians had below-recommended intakes of dietary fiber with 46 (98%) consuming <20 gm/day. The mean dietary fiber intake of Caucasians was 8.7 gm/day, which is less than half of the minimum recommended amount of 20-35 gm/day. All of the African-American participants (100%) had dietary fiber intakes below recommended levels. The mean intake was 8.4 gm/day, which is slightly lower than that of Caucasians; however, the difference was not significant. Thus, low dietary fiber intake appeared to be a major risk of CVD for both Caucasian and African-American women at pre-intervention.

Thirty Caucasians (64%) had daily sodium intakes above the recommended level of 1800-2400 mg/day, while 15% (7 individuals) had intakes
within the recommended level. The mean sodium intake for Caucasians was 2613 mg/day, which is above recommended. Fifty-five African-Americans (53%) had sodium intakes greater than 2400 mg/day and only 23% (24 individuals) consumed intakes within recommended levels. The mean intake of sodium (2539 mg/day) was lower for African-Americans than for Caucasians, but the difference was not significant. Hence, high sodium intakes may pose a higher-than-average risk of CVD for participants of both races.

In regard to potassium, an intake of less than 1600 mg/day is considered a risk for CVD. Among the Caucasian women, 45% (21 individuals) had low intakes of potassium as compared to 46% (47 individuals) of African-Americans. Thus, slightly more than half of the Caucasians and African-Americans (55% of each race group) had adequate or above-recommended potassium intakes. Although, the African-Americans had a slightly higher mean intake (1846 mg) than Caucasians (1820 mg), the difference was not significant. Thus, about half of both groups of women may have a higher-than-average CVD risk due to low potassium intake.

For calcium, 42 Caucasians (89%) had intakes below the recommended 800 mg/day. Ninety-two African-Americans (89%) had similarly low calcium intakes. The mean calcium intake was 521 mg/day for Caucasians and 489 mg/day for African-Americans, but the difference was not significant. A high percentage of participants in both race groups had low calcium intakes which may pose a significant risk for CVD.
Food group intakes of Caucasian and African-American participants were compared with the recommended servings from the Food Guide Pyramid (29) which are listed in Table 4 (Chapter III). Intake of the food groups (number of servings/ounces) were evaluated in regard to CVD risk because of the following: (a) the Milk Group is the best source of calcium which has been shown to protect against hypertension in some individuals (14, 29, 66-67); (b) the Vegetable and Fruit Groups are naturally low in fat and sodium and are major sources of potassium and fiber, both of which may be protective against CVD (14, 29, 62); (c) the Bread/Cereal Group is an important source of energy, especially in low-fat diets, and provides fiber which may be protective against elevated levels of blood cholesterol (14, 29, 89, 91); (d) foods from the Meat Group are major contributors of total fat, SFA and cholesterol, which may increase one’s risk of CVD (14, 29, 46, 52); and (e) the Fats/Sweets Group may lead to obesity and elevated blood cholesterol, if consumed in excessive amounts (14, 29).

For the Milk Group, a large majority of both Caucasian (94%) and African-American (93%) participants were below the recommended 2-3 servings/day. The mean intake of 0.5 serving for Caucasians was not significantly different from the 0.4 serving for African-Americans. Since a high percentage of the participants had inadequate intakes from the Milk Group, their diets lacked a major source of calcium which poses a significant risk for CVD in both race groups.
In regard to the Vegetable Group, 100% of Caucasians and 98% of African-Americans had less than 3 servings/day of vegetables. Of the 103 African-Americans, only 2% (2 individuals) had intakes within the recommended 3-5 servings/day. The mean vegetable intake was approximately 0.8 serving for Caucasians and 0.7 serving for African-Americans; however, this difference was not significant. Thus, the low vegetable intake of both race groups may pose a significant risk for CVD.

For the Fruit Group, 31 Caucasians (91%) and 63 African-Americans (84%) had consumed less than 2 servings/day from the Fruit Group. For both race groups, 9% (3 Caucasians and 7 African-Americans) had fruit intakes within the recommended range of 2-4 servings/day. The average number of servings/day of fruit was 1.3 for Caucasians and 1.5 for African-Americans; however, the difference was not significant.

For the Bread/Cereal Group, 9 Caucasians (19%) had intakes of less than 6 servings/day compared to 34 African-Americans (33%). Thirty-one Caucasians (66%) and 56 African-Americans (54%) had intakes within the recommended 6-11 servings/day. The mean intake of Caucasians was 8 servings/day from the Bread/Cereal Group which was well within the recommended 6-11 servings/day. Similarly, African-Americans had a mean intake of approximately 7.4 servings/day, which was slightly lower than that of Caucasians, but the difference was not significant. Based on these findings, it appears that adequate intake of foods from the Bread/Cereal Group may be
a beneficial factor in regard to cardiovascular health for participants of both race groups.

For the Meat Group, 44 African-Americans (43%) consumed the recommended 5-7 oz/day meat (2-3 servings); whereas, 14 Caucasians (30%) were in the same category. Only 8 of 47 Caucasians (17%) consumed more than the recommended amount compared to 29 African-Americans (28%). The mean intake was significantly higher for African-Americans, who had a mean intake of 6.0 ounces as compared with a mean intake of 5.1 ounces for Caucasians ($P<0.05$). Therefore, it does not appear that excess intake of the Meat Group poses a risk of CVD for either the Caucasian or African-American, low-income women in EFNEP.

More than 35% of both Caucasians and African-Americans had intakes of $\geq 10$ servings/day from the Fats/Sweets Group; however, 30 of 47 (64%) and 60 of 103 (58%), respectively, were within the recommended level ($<10$ servings/day). The average number of servings consumed from the Fats/Sweets Group per day was estimated at 11 for both Caucasian and African-American participants and the difference was not significant. Findings suggest that excess intake of foods from the Fats/Sweets Group may pose a risk of CVD for over a third of the women in EFNEP.

**Measures of Obesity.** African-American participants had a mean body weight of 178 lbs. which was about 12 lbs. heavier than the Caucasian women at 166 lbs., though they were similar in mean height at 64.0 inches.
Of the 75 participants, 57% (6 Caucasians and 36 African-Americans) were engaged in light physical activities which included mostly walking; while, the remaining 43% (3 Caucasians and 29 African-Americans) did not participate in any form of planned exercise.

Table 12 presents data for a sub-sample of 75 participants (10 Caucasians and 65 African-Americans) on distribution among five body fat categories (poor, fair, average, good, and excellent). Each participant was placed in one of the five categories based on her percent body fat as compared with the norm for her age (Table 5, Chapter III). A majority of both Caucasians (6 = 60%) and African-Americans (43 = 66%) were in the poor body fat category, which meant having a body fat greater than 31%. Only 1 Caucasian (10%) and 1 African-American (2%) were in either good or excellent body fat categories. The mean body fat was 36.4% for Caucasians and 34.6% for the African-Americans, but the difference was not significant. It was apparent that women in both race groups were above the recommended body fat levels at pre-intervention. These data show indicate that obesity is an important risk factor for CVD among EFNEP women.

Table 13 presents the distribution of Caucasian and African-American participants in categories based on standard body mass index (BMI) values associated with degrees of obesity and disease risks (Table 6, Chapter III). Although slightly different, mean BMI values for the groups by race were not significant at the beginning of the study, with the Caucasians having a mean
Table 12. Pre-intervention distribution of low-income Caucasian and African-American women in five categories of percent body fat relative to age (N = 10 Caucasians and 65 African-Americans)

<table>
<thead>
<tr>
<th>Race Category</th>
<th>Poor</th>
<th>Fair</th>
<th>Average</th>
<th>Good</th>
<th>Excellent</th>
<th>Mean</th>
<th>P value</th>
</tr>
</thead>
<tbody>
<tr>
<td></td>
<td>n</td>
<td>%</td>
<td>n</td>
<td>%</td>
<td>n</td>
<td>n</td>
<td>%</td>
</tr>
<tr>
<td>Caucasian</td>
<td>6</td>
<td>60</td>
<td>1</td>
<td>10</td>
<td>2</td>
<td>20</td>
<td>0</td>
</tr>
<tr>
<td>African-American</td>
<td>43</td>
<td>66</td>
<td>11</td>
<td>17</td>
<td>10</td>
<td>15</td>
<td>1</td>
</tr>
</tbody>
</table>

a The women were placed in the categories based on their percent body fat as compared with the norms for their age and gender.

b Mean percent body fat ± standard error of the mean (SEM)
Table 13. Mean values and distribution of Caucasian and African-American low-income, women based on BMI at pre-intervention (N = 10 Caucasians and 65 African-Americans)

<table>
<thead>
<tr>
<th>Race Category</th>
<th>BMI Categories (kg/m²)a</th>
<th>Degree of Obesity</th>
<th>Mean</th>
<th>P value</th>
</tr>
</thead>
<tbody>
<tr>
<td></td>
<td>Acceptable</td>
<td>Mild</td>
<td>Moderate</td>
<td>Marked</td>
</tr>
<tr>
<td></td>
<td>n</td>
<td>%</td>
<td>n</td>
<td>%</td>
</tr>
<tr>
<td>Caucasian</td>
<td></td>
<td></td>
<td></td>
<td></td>
</tr>
<tr>
<td>5</td>
<td>50</td>
<td>1</td>
<td>10</td>
<td>0</td>
</tr>
<tr>
<td>African-American</td>
<td>16</td>
<td>25</td>
<td>11</td>
<td>17</td>
</tr>
</tbody>
</table>

\textsuperscript{a} The women were placed in the categories based on their BMI as compared with the norms for both genders associated with obesity and CVD risk.

\textsuperscript{b} Mean BMI ± standard error of the mean (SEM)
BMI of 27.8 kg/m² and African-Americans having a mean BMI of 30.4 kg/m². The BMI values for female adults of both groups were above the recommended range (20.0-25.0 kg/m²), which may increase their risk for CVD (14,70).

Six of 10 Caucasians (60%) were in the acceptable or mildly obese categories, with the other 4 of 10 (40%) being in the marked obesity category. Twenty-seven (42%) of the 65 African-Americans were in the acceptable and mild obesity categories. The other African-Americans were distributed across the other three categories: moderate obesity = 11 (17%), marked obesity = 19 (29%) and morbid obesity = 8 (12%). Hence, results showed, that for both indices of obesity used in this study, a majority of the participants in both races were obese prior to intervention. The African-American females had a greater disposition to be obese than their white counterparts, but the difference did not reach significance.

**Blood Lipid Profiles.** Serum total cholesterol (TC), LDL-cholesterol (LDL-C), and triglyceride levels for the women are presented in Table 14. The participants with lipid values in three blood level categories (desirable, borderline high, and high), based on standards listed in Table 7, Chapter III.

Sixty percent of Caucasians (6 individuals) and 69% of African-Americans (45 individuals) had TC levels less than 200 mg/dl, which is the recommended level associated with normal risk of CVD (14,18,48). Two Caucasians (20%) and 12 African-Americans (19%) had TC levels within 200-240 mg/dl. Two Caucasians (20%) and 8 African-Americans (12%) had
Table 14. Total cholesterol, LDL-C, and triglyceride levels and distribution of Caucasian and African-American, low-income women in three categories of blood levels at pre-intervention (N = 10 Caucasians and 65 African-Americans)

<table>
<thead>
<tr>
<th>Blood Lipids</th>
<th>Race Category</th>
<th>Blood Level Categories(^a)</th>
<th>Desirable</th>
<th>High</th>
<th>High</th>
<th>Mean</th>
<th>P value</th>
</tr>
</thead>
<tbody>
<tr>
<td></td>
<td></td>
<td></td>
<td>n</td>
<td>%</td>
<td>n</td>
<td>%</td>
<td>n</td>
</tr>
<tr>
<td>TC(^b) (mg/dl)</td>
<td>Caucasian</td>
<td></td>
<td>6</td>
<td>60</td>
<td>2</td>
<td>20</td>
<td>2</td>
</tr>
<tr>
<td></td>
<td>African-American</td>
<td></td>
<td>45</td>
<td>69</td>
<td>12</td>
<td>19</td>
<td>8</td>
</tr>
<tr>
<td>LDL-C(^c) (mg/dl)</td>
<td>Caucasian</td>
<td></td>
<td>6</td>
<td>60</td>
<td>2</td>
<td>20</td>
<td>2</td>
</tr>
<tr>
<td></td>
<td>African-American</td>
<td></td>
<td>39</td>
<td>60</td>
<td>15</td>
<td>23</td>
<td>11</td>
</tr>
<tr>
<td>Triglyceride (mg/dl)</td>
<td>Caucasian</td>
<td></td>
<td>9</td>
<td>90</td>
<td>1</td>
<td>10</td>
<td>0</td>
</tr>
<tr>
<td></td>
<td>African-American</td>
<td></td>
<td>64</td>
<td>99</td>
<td>1</td>
<td>1</td>
<td>0</td>
</tr>
</tbody>
</table>

\(^a\)The women were placed in the categories based on their blood levels of total cholesterol, LDL-C, and triglycerides as compared with the norms for both genders and associated CVD risk.

\(^b\)TC indicates total cholesterol.

\(^c\)LDL-C indicates low-density lipoprotein cholesterol. LDL-C was estimated from the equation: LDL-C = (TC-HDL-C)-(Triglyceride/5).

\(^d\)Mean ± standard error of the mean (SEM)
TC levels greater than 240 mg/dl. Total cholesterol levels over 200 mg/dl have been associated with moderate-risk, while levels greater than 240 mg/dl may place an individual at high-risk for CVD (18,77). The overall mean serum TC level for the Caucasians was 207 mg/dl, which was slightly above the recommended. On the other hand, the African-Americans had a mean TC level of 188 mg/dl which was within desirable levels; but the difference was not significant.

For LDL-C, 60% of each group of women (6 Caucasians and 39 African-Americans) had desirable levels of <130 mg/dl. The remaining 40% of Caucasians were divided equally among the two highest blood LDL-C classifications. On the other hand, 15 (23%) and 11 (17%) African-Americans had LDL-C levels within the borderline-high-risk and high-risk categories, respectively. Caucasian participants had a higher mean LDL-C level (137 mg/dl) than did the African-American participants (128 mg/dl); however, the difference was not significant. It does not appear that high LDL-C levels (greater than 130 mg/dl) are a major risk for these women. Nevertheless, it should be noted that more Caucasian than African-American participants had LDL-C levels associated with increased risk for CVD.

In regard to triglyceride levels, a majority of both Caucasians (90%) and African-Americans (99%) had serum levels of <200 mg/dl at pre-intervention. The mean triglyceride value for Caucasians was 146 mg/dl, and for African-Americans the mean value was lower at 83 mg/dl; however,
the difference was not significant. Thus, high triglyceride levels (200 mg/dl or greater) do not appear to pose as a risk of CVD for either the Caucasian or African-American women in EFNEP.

Table 15 presents HDL-cholesterol values (HDL-C) by race, in three risk categories (negative risk, acceptable, increased risk), based on the standards listed in Table 8 (Chapter III). Five Caucasian women (50%) and 16 African-Americans (25%) had HDL-C levels of <35 mg/dl, which increases their risk for CVD (14,18,49). A higher percentage of African-Americans (45 individuals = 69%) than of Caucasians (5 individuals = 50%) had HDL-C levels of 35-59 mg/dl, shown to be associated with moderate CVD risk. Interestingly, there were 4 African-American women (4%) whose HDL-C levels were 60 mg/dl, a level which provides protection against CVD. The mean HDL-C values for Caucasians and African-Americans were 40 mg/dl and 44 mg/dl, respectively, which are within the acceptable range. The difference was not significant.

Table 16 shows distribution of the Caucasians and African-Americans based on ratio of TC to HDL-C, at 4-risk categories (half-risk, normal-risk, double-risk, and triple-risk) based on standards listed in Table 9 (Chapter III). A total of 4 Caucasians (40%) and 38 African-Americans (59%) had a TC:HDL-C ratio of 4.40 or lower, which is associated with normal to half the risk of CVD (77-78). Six Caucasians (60%) and 25 African-Americans (39%) had risk ratios of 4.45 to 7.05, which is associated with twice the
Table 15. High-density lipoprotein cholesterol measurements and risk comparisons between Caucasians and African-American, low-income women, at pre-intervention (N = 10 Caucasians and 65 African-Americans)

<table>
<thead>
<tr>
<th>Blood Lipid</th>
<th>Race Category</th>
<th>HDL-C Categoriesa</th>
<th></th>
<th></th>
<th></th>
<th></th>
<th>Mean</th>
<th>P value</th>
</tr>
</thead>
<tbody>
<tr>
<td></td>
<td></td>
<td>Negative Risk</td>
<td>n</td>
<td>%</td>
<td>Acceptable</td>
<td>n</td>
<td>%</td>
<td>Increased Risk</td>
</tr>
<tr>
<td>HDL-C\textsuperscript{b}</td>
<td>Caucasian</td>
<td>0 0</td>
<td>5 50</td>
<td>5 50</td>
<td></td>
<td>40 ± 2.3\textsuperscript{c}</td>
<td>0.392</td>
<td></td>
</tr>
<tr>
<td></td>
<td>African-American</td>
<td>4 6</td>
<td>45 69</td>
<td>16 25</td>
<td></td>
<td>44 ± 1.5</td>
<td></td>
<td></td>
</tr>
</tbody>
</table>

\textsuperscript{a}The women were placed in the categories based on their levels of HDL-C and risk of CVD as compared with the norms for both genders.

\textsuperscript{b}HDL-C indicates high-density lipoprotein cholesterol.

\textsuperscript{c}Mean ± standard error of the mean (SEM)
Table 16. Total cholesterol and HDL-C ratio and risk comparisons between Caucasian and African-American, low-income women at pre-intervention (N = 10 Caucasians and 65 African-Americans)

<table>
<thead>
<tr>
<th>Blood Lipid</th>
<th>Race Category</th>
<th>Categories of Total Cholesterol/HDL-C Ratio(^a)</th>
<th>Normal Risk</th>
<th>Mean</th>
<th>P value</th>
</tr>
</thead>
<tbody>
<tr>
<td></td>
<td></td>
<td></td>
<td>Half Risk</td>
<td>Double Risk</td>
<td>Triplo Risk</td>
</tr>
<tr>
<td></td>
<td></td>
<td></td>
<td>n</td>
<td>%</td>
<td>n</td>
</tr>
<tr>
<td>TC:HDL-C(^b)</td>
<td>Caucasian</td>
<td></td>
<td>3</td>
<td>30</td>
<td>1</td>
</tr>
<tr>
<td></td>
<td>African-American</td>
<td></td>
<td>27</td>
<td>42</td>
<td>11</td>
</tr>
</tbody>
</table>

\(^a\)The women were placed in the categories based on their total cholesterol and HDL-cholesterol ratios and risk of CVD as compared with the norms for both sex.

\(^b\)TC:HDL-C indicates ratio between total cholesterol and HDL-cholesterol.

\(^c\)Mean ± standard error of the mean (SEM)
risk of developing CVD manifestations such as heart attack, angina pectoris, or sudden cardiac death (77). The remaining 2 African-American participants (3%) had ratios of 7.06 and 11.04, which impart a three-fold risk of CVD (77-78). The mean TC:HDL-C ratio of 5.11 for Caucasians was not significantly different from the 4.43 for African-Americans. More than half of the Caucasian participants had elevated TC:HDL-C ratios which places them at increased risk for CVD.

Thus, there were no significant differences between the two groups of women by race on mean levels of any of the blood lipid components, at pre-intervention. However, there was a tendency for Caucasian participants to have higher mean levels of serum TC, LDL-C, triglyceride, and TC:HDL-C ratio than their African-American counterparts. The opposite was found for HDL-C levels in which African-American participants had greater mean values than the Caucasians. Thus, Caucasian participants were at slightly higher risk for CVD than the African-Americans.

**Smoking Status and History.** Table 17 presents the smoking status of Caucasians and African-Americans and indicates how the various smoking characteristics of the two groups differed at pre-intervention. Significant racial differences (P<0.05) were found for nonsmokers status, age when they started smoking regularly, and the number of cigarettes smoked per day.
Table 17. Comparison of smoking characteristics by race at pre-intervention (47 Caucasians and 103 African-Americans)

<table>
<thead>
<tr>
<th>Variable</th>
<th>Caucasian</th>
<th></th>
<th>African-American</th>
<th></th>
<th>P value</th>
</tr>
</thead>
<tbody>
<tr>
<td></td>
<td>n</td>
<td>%</td>
<td>n</td>
<td>%</td>
<td></td>
</tr>
<tr>
<td>Smoking Status</td>
<td></td>
<td></td>
<td></td>
<td></td>
<td></td>
</tr>
<tr>
<td>Current Smokers</td>
<td>20</td>
<td>43</td>
<td>31</td>
<td>30</td>
<td>0.135</td>
</tr>
<tr>
<td>Ex-Smokers</td>
<td>9</td>
<td>19</td>
<td>2</td>
<td>2</td>
<td>NCi</td>
</tr>
<tr>
<td>Non-Smokers</td>
<td>18</td>
<td>38</td>
<td>70</td>
<td>68</td>
<td>0.001***</td>
</tr>
<tr>
<td>Mean Age When Started (yrs.)d</td>
<td>15.8 ± 0.4</td>
<td></td>
<td>19.0 ± 0.9</td>
<td></td>
<td>0.003**</td>
</tr>
<tr>
<td>Mean Age When Stopped (yrs.)e</td>
<td>23.8 ± 1.4</td>
<td></td>
<td>19.0 ± 5.0</td>
<td></td>
<td>0.220</td>
</tr>
<tr>
<td>Mean Cigarettes/dayf</td>
<td>8.5 ± 1.8</td>
<td></td>
<td>3.9 ± 0.8</td>
<td></td>
<td>0.008**</td>
</tr>
<tr>
<td>Family Member Smokersg</td>
<td></td>
<td></td>
<td></td>
<td></td>
<td>0.707</td>
</tr>
<tr>
<td>Yes</td>
<td>23</td>
<td>49</td>
<td>47</td>
<td>46</td>
<td></td>
</tr>
<tr>
<td>No</td>
<td>24</td>
<td>51</td>
<td>56</td>
<td>54</td>
<td></td>
</tr>
</tbody>
</table>

* ≤ 0.05, ** ≤ 0.01, *** ≤ 0.001

aCurrent smokers include participants who (1) had smoked before and continued to smoke during the study and (2) had smoked at the beginning and/or throughout the study.

bEx-smokers include participants who used to smoke but quit before the beginning of the study and never smoked at anytime during the study.

cNon-smokers include participants who had never smoked in their entire life.

dMean age of ex-smokers and current smokers when they first started smoking cigarettes regularly.

eMean age of ex-smokers when they quit smoking.

fMean number of cigarettes smoked per day by current smokers.

gParticipants who have family members in the household smoking at the time of the study.

hMean ± standard error of the mean (SEM)

iNot Calculated
In this EFNEP population, a total of 62 participants (29 Caucasians and 33 African-Americans) were smokers. Forty-three percent of Caucasians (20 individuals) were current smokers and had been smoking before the beginning of the study, compared with 30% of African-Americans (31 individuals). In addition, 9 Caucasians (19%) used to smoke but quit before the start of the study as compared to only 2 African-Americans (2%). Significantly more African-American women (68%) were non-smokers as compared with Caucasians (38%).

Caucasians were significantly younger than African-Americans when they started to smoke regularly ($P=0.003$). Among current smokers, Caucasian women smoked significantly more cigarettes per day ($P=0.008$) than the African-Americans. There were slightly more Caucasian participants (49%) whose family members were current smokers than African-Americans (46%); however, no significant difference was found. Caucasians were more likely to quit smoking at a later age than African-Americans, but the difference was not significant.

**Change of Variables from Pre to Post-intervention**

Use of the General Linear Models (GLM) procedure for unbalanced analysis of variance (ANOVA) revealed no significant interaction by group (experimental and control) or by race (Caucasian and African-American) regarding the mean difference scores for nutrients, food groups, measures of obesity, and blood lipid levels. However, there was an interaction with race.
for the number of cigarettes smoked. In addition, analysis on mean change scores for these variables showed no significant effect by race. Therefore, all subsequent analyses on change were performed on the mean difference scores of the two race groups combined. Paired t-tests were used to determine if there was a significant difference in the mean change from pre- to post-intervention between the experimental group and the control group.

Table 18 presents data on change with regard to certain dietary risk factors for CVD from pre- to post-intervention, with comparisons between the experimental and control groups. There were significant differences in change made by the two groups for energy, TF (% kcal), and SFA (% kcal), but no differences were shown for cholesterol and sodium intakes.

For energy intake, the experimental group decreased their mean energy by 3 kcal, while the control group increased their energy intake by 197 kcal. Thus, the control group significantly increased its energy intake as compared with the experimental participants (P < 0.05). Though both groups had mean energy intakes within recommended levels of 1600-2200 kcal/day at pre- and post-intervention, the increase in caloric intake by the control group was viewed as a risk for CVD, as most of the participants were fairly sedentary.

On TF intake, both groups were above the recommended intake at pre-intervention, with experimentals having a mean intake of 36.3% kcal and controls having a mean intake of 34.9% kcal. The experimental group
Table 18. Mean intake of nutrients related to CVD risk, at pre- and post-intervention (N = 74 Experimental and 76 Controls)

<table>
<thead>
<tr>
<th>Nutrients</th>
<th>Group</th>
<th>Mean Intakes</th>
<th>Mean Change$^b$</th>
<th>P value</th>
</tr>
</thead>
<tbody>
<tr>
<td>1. Energy (kcal)</td>
<td>Experimental</td>
<td>1802 ± 77.6$^a$</td>
<td>1799 ± 78.3</td>
<td>-3.0 ± 72.8</td>
</tr>
<tr>
<td></td>
<td>Control</td>
<td>1651 ± 61.0</td>
<td>1848 ± 68.6</td>
<td>+197.0 ± 64.5</td>
</tr>
<tr>
<td>2. Total Fat (% kcal)</td>
<td>Experimental</td>
<td>36.3% ± 0.8%</td>
<td>31.1% ± 0.7%</td>
<td>-5.2% ± 1.0%</td>
</tr>
<tr>
<td></td>
<td>Control</td>
<td>34.9% ± 0.9%</td>
<td>34.8% ± 0.8%</td>
<td>-0.1% ± 1.0%</td>
</tr>
<tr>
<td>3. SFA (% kcal)</td>
<td>Experimental</td>
<td>11.8% ± 0.3%</td>
<td>9.5% ± 0.3%</td>
<td>-2.3% ± 0.5%</td>
</tr>
<tr>
<td></td>
<td>Control</td>
<td>10.9% ± 0.3%</td>
<td>11.4% ± 0.4%</td>
<td>+0.5% ± 0.5%</td>
</tr>
<tr>
<td>4. Cholesterol (mg)</td>
<td>Experimental</td>
<td>271 ± 16.8</td>
<td>247 ± 14.8</td>
<td>-24.0 ± 17.4</td>
</tr>
<tr>
<td></td>
<td>Control</td>
<td>288 ± 21.3</td>
<td>276 ± 17.7</td>
<td>-12.0 ± 24.0</td>
</tr>
<tr>
<td>5. Sodium (mg)</td>
<td>Experimental</td>
<td>2723 ± 121.2</td>
<td>2464 ± 106.4</td>
<td>-259.0 ± 142.8</td>
</tr>
<tr>
<td></td>
<td>Control</td>
<td>2406 ± 93.9</td>
<td>2474 ± 100.2</td>
<td>+68.0 ± 112.9</td>
</tr>
</tbody>
</table>

$^a$Mean ± standard error of the mean (SEM)

$^b$Change = Post-intervention mean minus pre-intervention mean intakes. Plus and minus signs are shown to indicate the direction of change.

$^c$P values for dietary change are based on t-tests after analysis of variance, comparing the two study groups.
showed a decrease in fat of 5.2 points which was highly significant as compared with the control group which showed very little change in TF intake (P = 0.0003). Thus, the experimental group improved its risk for CVD in regard to TF intake.

Regarding SFA intake, the experimental group decreased its intake from 11.8% kcal to 9.5% kcal/day as compared with the control who showed a slight increase of 0.5%. The difference was highly significant (P = 0.0001), indicating an important improvement in risk for CVD among the experimental participants.

On dietary cholesterol, both groups had intakes within the recommended <300 mg/day. Although experimentals decreased their cholesterol intake by 23.0 mg compared to 11.0 mg for the controls, the difference was not significant. With regard to sodium intake, the experimental participants had a mean intake of 2723 mg/day at pre-intervention and the control participants had a mean intake of 2403 mg/day. Though the experimental group tended to decrease its intake by 259 mg while the control group increased its intake by 68.0 mg, the difference was not significant. Thus, the intervention did not bring about significant improvement on intake of dietary cholesterol and sodium.

Table 19 presents the mean intakes for nutrients that are believed to protect against CVD, including MUFA (% kcal), PUFA (% kcal), dietary fiber, potassium, and calcium. A comparison of change in intakes of experimental
Table 19. Mean intake of CVD dietary protectors at pre- and post-intervention
(N = 74 Experimental and 76 Controls)

<table>
<thead>
<tr>
<th>Nutrients</th>
<th>Group</th>
<th>Mean Intakes</th>
<th>Mean Change</th>
<th>P value</th>
</tr>
</thead>
<tbody>
<tr>
<td></td>
<td></td>
<td>Pre</td>
<td>Post</td>
<td></td>
</tr>
<tr>
<td>1. MUFA(^d) (% kcal)</td>
<td>Experimental</td>
<td>12.9 ± 0.4(^a)</td>
<td>10.0 ± 0.4</td>
<td>-2.9 ± 0.6</td>
</tr>
<tr>
<td></td>
<td>Control</td>
<td>12.4 ± 0.4</td>
<td>11.6 ± 0.4</td>
<td>-0.8 ± 0.6</td>
</tr>
<tr>
<td>2. PUFA(^e) (% kcal)</td>
<td>Experimental</td>
<td>6.1 ± 0.3</td>
<td>5.0 ± 0.2</td>
<td>-1.1 ± 0.3</td>
</tr>
<tr>
<td></td>
<td>Control</td>
<td>6.0 ± 0.3</td>
<td>5.6 ± 0.2</td>
<td>-0.4 ± 0.3</td>
</tr>
<tr>
<td>3. Dietary Fiber (gm)</td>
<td>Experimental</td>
<td>9.4 ± 0.6</td>
<td>3.8 ± 1.1</td>
<td>+ 4.4 ± 1.0</td>
</tr>
<tr>
<td></td>
<td>Control</td>
<td>7.6 ± 0.4</td>
<td>9.0 ± 0.4</td>
<td>+ 1.4 ± 0.5</td>
</tr>
<tr>
<td>4. Potassium (mg)</td>
<td>Experimental</td>
<td>1949 ± 91.7</td>
<td>3079 ± 231.5</td>
<td>+1129.0 ± 223.2</td>
</tr>
<tr>
<td></td>
<td>Control</td>
<td>1729 ± 86.2</td>
<td>2667 ± 164.6</td>
<td>+938.0 ± 190.8</td>
</tr>
<tr>
<td>5. Calcium (mg)</td>
<td>Experimental</td>
<td>563 ± 39.0</td>
<td>743 ± 47.4</td>
<td>+180.0 ± 45.0</td>
</tr>
<tr>
<td></td>
<td>Control</td>
<td>436 ± 22.1</td>
<td>605 ± 28.6</td>
<td>+168.0 ± 31.6</td>
</tr>
</tbody>
</table>

\(^a\)Mean ± standard error of the mean (SEM)  \(^b\)Change = Post-intervention mean minus pre-intervention mean intakes. Plus and minus signs are shown to indicate direction of change. \(^c\)P values for dietary change are based on t-tests after analysis of variance, comparing the two study groups. \(^d\)MUFA = monounsaturated fats \(^e\)PUFA = polyunsaturated fats
and control participants are also shown. Significant differences were observed for changes in mean intakes of MUFA (% kcal), and dietary fiber between the experimental and control groups; whereas, no differences were shown for intakes of PUFA (% kcal), potassium, and calcium.

The mean intake of MUFA (% kcal) for experimentals and controls was within the recommended 10-15% kcal at pre- and post-intervention. Unexpectedly, the experimental group showed a decrease in mean MUFA intake of 2.9 % kcal which was significantly greater than the decrease of 0.8% kcal for the control group (P = 0.010).

Similarly, the mean PUFA intake was <10% kcal at the start of the study and decreased to lower levels at post-intervention for participants in the experimental and control groups. There was a greater decrease in mean intake for the experimental group (1.1% kcal) than the controls (0.4% kcal); however, difference in mean change was not significant.

Regarding dietary fiber, the experimentals increased their intake by 4.4 gm/day, which was significantly greater than the increase of 1.4 gm/day by the controls (P = 0.007). However, post-intervention mean intakes for both experimental and control participants were less than 20 gm/day which is the minimum recommended level.

The increase in mean intake of potassium among the experimental participants (1129.0 mg) was greater than control participants (938.0 mg); but no significant difference was found. However, participants in both study
groups had mean intakes within the recommended level at post-intervention.

With regard to calcium intake, the mean daily calcium intake of the experimental group increased by 180 mg compared to a 168 mg increase by the control group; however, the difference in mean change of calcium was not significant. At post-intervention, the mean intake of both groups were still below the minimum recommended level of 800 mg. In summary, the intervention resulted in a significant improvement in dietary fiber and tended to increase potassium and calcium intakes among the experimental subjects, all of which help to decrease risks for CVD.

Table 20 shows the average number of servings/ounces consumed and changes between the experimental and control group for each of the six groups of the Food Guide Pyramid. There were significant differences in the change made by the two groups for the Vegetable Group, the Fruit Group, the Meat Group, and the Fats/Sweets Group, but no differences were shown for the Milk Group and the Bread/Cereals Group.

For the Milk Group, pre- and post-intervention values indicate that participants of both groups consumed less than the minimum recommended 2 servings daily. Moreover, the increase in the mean number of servings by experimental participants (0.4 serving) was slightly less than controls (0.5 serving) and no significant difference was found. Thus, the experimental group did not improve its risk of CVD in regard to milk and milk products.
<table>
<thead>
<tr>
<th>Food Groups</th>
<th>Group</th>
<th>Mean Servings Pre</th>
<th>Mean Servings Post</th>
<th>Mean Change &lt;sup&gt;b&lt;/sup&gt;</th>
<th>P value &lt;sup&gt;c&lt;/sup&gt;</th>
</tr>
</thead>
<tbody>
<tr>
<td>1. Milk, Yogurt, Cheese (servings)</td>
<td>Experimental</td>
<td>0.6 ± 0.1&lt;sup&gt;a&lt;/sup&gt;</td>
<td>1.0 ± 0.1</td>
<td>+0.4 ± 0.1</td>
<td>0.587</td>
</tr>
<tr>
<td></td>
<td>Control</td>
<td>0.3 ± 0.1</td>
<td>0.8 ± 0.1</td>
<td>+0.5 ± 0.1</td>
<td></td>
</tr>
<tr>
<td>2. Vegetable (servings)</td>
<td>Experimental</td>
<td>0.9 ± 0.1</td>
<td>1.6 ± 0.2</td>
<td>+0.7 ± 0.2</td>
<td>0.038*</td>
</tr>
<tr>
<td></td>
<td>Control</td>
<td>0.6 ± 0.1</td>
<td>0.8 ± 0.1</td>
<td>+0.2 ± 0.1</td>
<td></td>
</tr>
<tr>
<td>3. Fruit (servings)</td>
<td>Experimental</td>
<td>1.5 ± 0.2</td>
<td>2.6 ± 0.3</td>
<td>+1.1 ± 0.3</td>
<td>0.002**</td>
</tr>
<tr>
<td></td>
<td>Control</td>
<td>1.4 ± 0.2</td>
<td>1.4 ± 0.2</td>
<td>+0.0 ± 0.2</td>
<td></td>
</tr>
<tr>
<td>4. Bread, Cereals, Rice, Pasta (servings)</td>
<td>Experimental</td>
<td>8.0 ± 0.4</td>
<td>8.1 ± 0.5</td>
<td>+0.1 ± 0.5</td>
<td>0.450</td>
</tr>
<tr>
<td></td>
<td>Control</td>
<td>7.2 ± 0.3</td>
<td>7.7 ± 0.4</td>
<td>+0.5 ± 0.4</td>
<td></td>
</tr>
<tr>
<td>5. Meat, Poultry, Fish, Dry Beans, Eggs, Nuts (ounces)</td>
<td>Experimental</td>
<td>6.0 ± 0.3</td>
<td>5.4 ± 0.3</td>
<td>-0.6 ± 0.4</td>
<td>0.054*</td>
</tr>
<tr>
<td></td>
<td>Control</td>
<td>5.5 ± 0.3</td>
<td>5.9 ± 0.3</td>
<td>+0.4 ± 0.4</td>
<td></td>
</tr>
<tr>
<td>6. Fats, Sweets (servings)</td>
<td>Experimental</td>
<td>11.6 ± 0.7</td>
<td>9.3 ± 0.5</td>
<td>-2.3 ± 0.7</td>
<td>0.0005***</td>
</tr>
<tr>
<td></td>
<td>Control</td>
<td>10.0 ± 0.5</td>
<td>11.2 ± 0.7</td>
<td>+1.2 ± 0.7</td>
<td></td>
</tr>
</tbody>
</table>

<sup>a</sup>Mean number of servings ± standard error of the mean (SEM)

<sup>b</sup>Change = Post-intervention mean minus pre-intervention mean intakes. Plus and minus signs are shown to indicate the direction of change.

<sup>c</sup>P values for dietary change are based on t-tests after analysis of variance, comparing the two study groups.
For the Vegetable Group, the mean number of servings consumed by the experimental group was higher compared to that of the control group; however, the mean number of servings was below the recommended 3-5 servings/day. An increase in the consumption of vegetables occurred for both groups following intervention. The mean increase of 0.7 serving by the experimentals was significantly greater than the 0.3 serving increase of the control group (P < 0.05).

Regarding the Fruit Group, both groups were below the recommended 2-4 servings/day at pre-intervention, with experimentals having 1.5 servings which was slightly higher than that of the controls (1.4 servings). The mean number of servings by the experimentals increased by 1.1 (72%) which was significant as compared with the control group which showed no change in consumption of fruits from pre- to post-intervention (P = 0.002).

For the Bread/Cereal Group, the experimental and control groups generally consumed adequate servings at pre and post-intervention with the experimental group having 8 servings/day and the control group having 7.7 servings/day. The mean number of servings increased only slightly for both study groups and the difference in change was not significant.

For the Meat Group, the mean daily intake at pre-intervention was not excessive in either group, with the experimentals consuming 6.0 ounces and the controls consuming 5.5 ounces. The experimental group decreased its mean intake by 0.6 ounces (10%), but the control group increased its con-
sumption by 0.4 ounces (7%), which was significant. Thus, experimentalists significantly decreased its consumption of meat as compared with controls (P < 0.05).

In regard to the Fats/Sweets Group, the experimental participants decreased its mean consumption of foods in the Fats/Sweets Group from 11.6 servings/day to 9.3 servings/day as compared with the controls who increased their intake by 1.2 servings (12%). The difference in mean change was highly significant (P = 0.001), which indicates an important improvement in risk for CVD among experimental participants.

The mean height was 64 inches for both groups of participants. On the average, controls were heavier (183 lbs. ± 3) than experimentalists (170 lbs. ± 7.8) at pre-intervention. There was approximately a 0.3 lb (0.2%) increase in weight among experimental participants, in contrast to a 0.3 lb (0.2%) decrease among the control participants, but the weight change was not significant.

Mean values and changes for percent body fat and BMI of the experimental and control groups are shown in Table 21. The experimental group had a mean body fat of 33.2% at pre-intervention and the control group had a mean body fat of 36.4%, both of which were above the 31% cut-off for obesity and association with increased health risk in females (70,90). Though the experimental group had a greater decrease in body fat (1.3%) as compared with the control group (0.5%), the difference was not significant.
Table 21. Mean change of percent body fat and BMI (kg/m²) at pre- and post-intervention and comparisons between study groups (N = 37 Experimentals and 38 Controls)

<table>
<thead>
<tr>
<th>Variable</th>
<th>Group</th>
<th>Mean Measurements</th>
<th>Mean Change&lt;sup&gt;b&lt;/sup&gt;</th>
<th>P value&lt;sup&gt;c&lt;/sup&gt;</th>
</tr>
</thead>
<tbody>
<tr>
<td></td>
<td></td>
<td>Pre</td>
<td>Post</td>
<td></td>
</tr>
<tr>
<td>1. Percent Body Fat</td>
<td>Experimental</td>
<td>33.2 ± 1.2&lt;sup&gt;a&lt;/sup&gt;</td>
<td>31.9 ± 1.4</td>
<td>-1.3 ± 0.9</td>
</tr>
<tr>
<td></td>
<td>Control</td>
<td>36.4 ± 0.9</td>
<td>35.9 ± 0.9</td>
<td>-0.5 ± 0.7</td>
</tr>
<tr>
<td>2. Body Mass Index</td>
<td>Experimental</td>
<td>29.0 ± 1.2</td>
<td>28.9 ± 1.2</td>
<td>-0.1 ± 0.3</td>
</tr>
<tr>
<td>(kg/m&lt;sup&gt;2&lt;/sup&gt;)</td>
<td>Control</td>
<td>31.2 ± 1.2</td>
<td>30.8 ± 1.2</td>
<td>-0.4 ± 0.3</td>
</tr>
</tbody>
</table>

<sup>a</sup>Mean ± standard error of the mean (SEM)

<sup>b</sup>Change = Post-intervention mean minus pre-intervention mean. Plus and minus signs are shown to indicate the direction of change.

<sup>c</sup>P values for change in measures of obesity are based on t-tests after analysis of variance, comparing the two study groups.
For BMI, both study groups had mean BMI values above the recommended range of 20-25 kg/m² at pre-intervention. Control participants showed a decrease in BMI of 0.4 kg/m² which was not significant as compared with the experimental group which showed almost no change in BMI. Thus the intervention did not bring about improvement on percent of body fat levels, or BMI, among experimental participants.

Table 22 presents the mean values for serum levels of TC, LDL-C, triglyceride, HDL-C, and TC:HDL-C ratio, of the experimental and control groups during pre- and post-intervention, and changes over the 12-month study period. There were no significant changes shown between the groups on any of the blood lipid components. On serum TC, the experimental group increased its mean serum TC by 1.0 mg/dl (0.6%), while the control group decreased its serum TC by 5.0 mg/dl (3%), but the difference was not significant.

For LDL-C, experimental participants had a slightly lower mean level of 122 mg/dl than did participants in the control group who had LDL-C levels of 135 mg/dl at pre-intervention. However, the experimental group showed no
Table 22. Mean blood lipid profiles of experimental and control participants (N = 37 Experimentals and 38 Controls)

<table>
<thead>
<tr>
<th>Variables</th>
<th>Group</th>
<th>Mean Levels</th>
<th>Mean Change $^f$</th>
<th>P value $^g$</th>
</tr>
</thead>
<tbody>
<tr>
<td></td>
<td></td>
<td>Pre</td>
<td>Post</td>
<td></td>
</tr>
<tr>
<td>1. TC$^a$ (mg/dl)</td>
<td>Experimental</td>
<td>183 ± 6.5$^e$</td>
<td>184 ± 8.2</td>
<td>+1.0 ± 4.3</td>
</tr>
<tr>
<td></td>
<td>Control</td>
<td>196 ± 8.4</td>
<td>191 ± 6.3</td>
<td>-5.0 ± 3.8</td>
</tr>
<tr>
<td>2. LDL-C$^b$ (mg/dl)</td>
<td>Experimental</td>
<td>122 ± 5.3</td>
<td>122 ± 7.7</td>
<td>0.0 ± 4.4</td>
</tr>
<tr>
<td></td>
<td>Control</td>
<td>134 ± 6.7</td>
<td>126 ± 5.8</td>
<td>-9.0 ± 3.5</td>
</tr>
<tr>
<td>3. Triglyceride (mg/dl)</td>
<td>Experimental</td>
<td>92 ± 10.2</td>
<td>94 ± 7.6</td>
<td>+2.0 ± 7.7</td>
</tr>
<tr>
<td></td>
<td>Control</td>
<td>87 ± 9.6</td>
<td>96 ± 7.1</td>
<td>+10.0 ± 7.9</td>
</tr>
<tr>
<td>4. HDL-C$^c$ (mg/dl)</td>
<td>Experimental</td>
<td>43 ± 1.5</td>
<td>45 ± 2.0</td>
<td>+2.0 ± 1.6</td>
</tr>
<tr>
<td></td>
<td>Control</td>
<td>44 ± 2.3</td>
<td>46 ± 1.9</td>
<td>+2.0 ± 1.8</td>
</tr>
<tr>
<td>5. TC:HDL-C$^d$</td>
<td>Experimental</td>
<td>4.3 ± 0.2</td>
<td>4.3 ± 0.3</td>
<td>0.0 ± 0.2</td>
</tr>
<tr>
<td></td>
<td>Control</td>
<td>4.6 ± 0.2</td>
<td>4.4 ± 0.2</td>
<td>-0.2 ± 0.2</td>
</tr>
</tbody>
</table>

$^a$TC indicates serum total cholesterol.
$^b$LDL-C indicates low-density lipoprotein cholesterol. LDL-C was estimated from the equation: LDL-C = (TC - HDL-C) - (Triglyceride/5).
$^c$HDL-C indicates high-density lipoprotein cholesterol.
$^d$TC:HDL-C indicates ratio between total cholesterol and high-density lipoprotein cholesterol.
$^e$Mean change ± standard error of the mean (SEM).
$^f$Change = Post-intervention mean minus pre-intervention mean lipid levels. Plus and minus signs are shown to indicate the direction of change.
$^g$P values for change in blood lipid levels are based on t-tests after analysis of variance, comparing the two study groups.
change as compared with the controls who had a decrease of 9.0 mg/dl (7%), but the difference was not significant. This finding indicates that the intervention did not improve the risk for CVD among experimental participants in regard to LDL-C.

Regarding triglycerides, both groups had mean levels that were well within normal limits (<200 mg/dl) and which are associated with an acceptable level of CVD risk. Although the experimental group increased its mean triglyceride level by 2.0 mg/dl (2%), and the control group by 10.0 mg/dl (12%) from pre- to post-intervention, the difference was not significant.

On HDL-C, experimental and control groups were within acceptable range at pre-intervention, with experimental having a mean level of 43.0 mg/dl and controls having a level of 44.0 mg/dl. Following intervention, each group had increased HDL-C levels of +2.0 mg/dl (5%), but the difference in change was not significant.

For TC:HDL-C ratio, the experimental group had a mean ratio of 4.3, which was slightly lower than the control group at pre-intervention. The control group decreased its ratio by 0.2 points but the change was not significant as compared with the experimental which showed no change in ratio.

Table 23 presents the change in smoking status categories between study groups. At pre-intervention, more than 50% of participants in both experimental (59.5%) and control groups (59.2%) had never smoked in their
Table 23. Change of smoking status by study groups from pre- and post-intervention (N = 74 Experimentals and 76 Controls)

<table>
<thead>
<tr>
<th>Smoking Status</th>
<th>Group</th>
<th>Number of Participants</th>
<th></th>
<th></th>
<th>Change</th>
</tr>
</thead>
<tbody>
<tr>
<td></td>
<td></td>
<td></td>
<td></td>
<td></td>
<td></td>
</tr>
<tr>
<td>Current Smokers(^a)</td>
<td>Experimental</td>
<td>21</td>
<td>19</td>
<td></td>
<td>-2</td>
</tr>
<tr>
<td></td>
<td>Control</td>
<td>30</td>
<td>28</td>
<td></td>
<td>-2</td>
</tr>
<tr>
<td>Non-Smokers(^b)</td>
<td>Experimental</td>
<td>44</td>
<td>41</td>
<td></td>
<td>-3</td>
</tr>
<tr>
<td></td>
<td>Control</td>
<td>45</td>
<td>44</td>
<td></td>
<td>-1</td>
</tr>
<tr>
<td>Ex-Smokers(^c)</td>
<td>Experimental</td>
<td>9</td>
<td>7</td>
<td></td>
<td>-2</td>
</tr>
<tr>
<td></td>
<td>Control</td>
<td>1</td>
<td>1</td>
<td></td>
<td>0</td>
</tr>
</tbody>
</table>

\(^a\)Current smokers include participants who (1) had smoked before and continued during the study and (2) had smoked at the beginning and/or throughout the study.

\(^b\)Non-smokers include participants who had never smoked in their entire life.

\(^c\)Ex-smokers include participants who used to smoke but quit before the beginning of the study and never smoked at anytime during the study.
life (i.e. non-smokers). Among smokers, there were 51 current smokers (21 experimentals and 30 controls) and 10 ex-smokers (9 experimentals and 1 control). Following intervention, there was a greater decrease in the number of non-smokers for the experimental group (4.1%) than for the control group (1.3%). There were 3 experimentals (4.0%) and 1 control (1.3%) who had no smoking history, but who started smoking after the study began. Among current smokers, two individuals in each group stopped smoking during the study; however, there were 2 former smokers in the experimental group who resumed smoking during the study, as compared with none in the control group.

Among current and recent smokers, there was a significant interaction for race and group, in regard to change in number of cigarettes smoked per day (P<0.05). The Least Square Means Multiple Comparison Test was used to compare the mean number of cigarettes smoked per day between the groups, by race. The mean values and changes for the number of cigarettes smoked per day are presented in Table 24. Among the experimental participants, Caucasians decreased their smoking by 1.7 cigarettes/day, while African-Americans increased smoking by 0.5 cigarettes/day. Thus, the Caucasians significantly decreased their cigarette smoking as compared with the African-Americans (P<0.05). However, the mean number of cigarettes smoked/day by Caucasian participants was more than twice that of African-American participants at pre- and post-intervention. Among the control group, Caucasians increased their cigarette consumption by 0.6 cigars/day,
Table 24. Mean number of cigarettes smoked per day by smokers\textsuperscript{a} at pre- and post-intervention (N = 24 Experimentals and 29 Controls)

<table>
<thead>
<tr>
<th>Group</th>
<th>Race</th>
<th>Mean Pre</th>
<th>Mean Post</th>
<th>Mean Change\textsuperscript{c}</th>
<th>P value</th>
</tr>
</thead>
<tbody>
<tr>
<td>Experimental</td>
<td>Caucasian</td>
<td>8.0 ± 2.4\textsuperscript{b}</td>
<td>6.3 ± 1.8</td>
<td>-1.7 ± 1.5</td>
<td>0.058\textsuperscript{d}</td>
</tr>
<tr>
<td></td>
<td>African-American</td>
<td>2.5 ± 1.0</td>
<td>3.0 ± 1.0</td>
<td>+0.5 ± 0.5</td>
<td></td>
</tr>
<tr>
<td>Control</td>
<td>Caucasian</td>
<td>8.9 ± 2.7</td>
<td>9.5 ± 2.8</td>
<td>+0.6 ± 0.6</td>
<td>0.285</td>
</tr>
<tr>
<td></td>
<td>African-American</td>
<td>5.3 ± 8.8</td>
<td>4.3 ± 1.1</td>
<td>-1.0 ± 0.6</td>
<td></td>
</tr>
<tr>
<td>Experimental</td>
<td></td>
<td>4.3 ± 1.1</td>
<td>4.1 ± 0.9</td>
<td>-0.2 ± 0.6</td>
<td>0.638\textsuperscript{e}</td>
</tr>
<tr>
<td>Control</td>
<td></td>
<td>6.4 ± 1.2</td>
<td>5.8 ± 1.1</td>
<td>-0.6 ± 0.5</td>
<td></td>
</tr>
</tbody>
</table>

\textsuperscript{a}Smokers include current, recent, and reformed participants
\textsuperscript{b}Least square means ± standard error of the mean (SEM)
\textsuperscript{c}Change = Post-intervention mean minus pre-intervention mean. Plus and minus signs are shown to indicate the direction of change.
\textsuperscript{d}P values for change in mean number of cigarettes are based on analysis of variance comparing the two study groups by race.
\textsuperscript{e}P values for change in mean number of cigarettes are based on t-tests after analysis of variance, comparing the two study groups.
while African-Americans decreased their smoking by 1.0 cigarette/day.

Overall, there was a decrease in the number of cigarettes smoked by both groups; however, the difference in change between the experimental and control groups did not reach significance.

Table 25 shows the list of reasons for initiating smoking and/or continuing to smoke, with comparisons between the Caucasian and African-American female smokers. Twelve of the 18 Caucasians (66.7%) found it difficult to quit smoking as compared to 20 African-Americans (69.0%). Only 1 Caucasian and 2 African-American female smokers believed that smoking was not harmful to health. Two Caucasians were concerned about gaining weight after quitting; while, none of the African-American smokers indicated that they were concerned about weight gain. On the other hand, 4 African-Americans saw smoking as a form of pleasure to indulge in during episodes of happiness or sadness, but none of the Caucasian smokers indicated this as a reason to continue smoking. Three Caucasians and 3 African-Americans had a combination of reasons for not wanting to discontinue smoking.

Discussion

Among the participants in the study, significantly more Caucasians than African-Americans had completed high school. Nineteen percent of the Caucasians had not completed high school as compared with 51% of African-American study participants. This finding is in agreement with the 1987 U.S. Bureau of Census (21) data on social and economic aspects
Table 25. Distribution of participants on smoking behavior and reasons for continuing to smoke by race (18 Caucasians and 29 African-Americans)

<table>
<thead>
<tr>
<th>Reason For Smoking</th>
<th>Race Category</th>
<th>Smokers(^a)</th>
</tr>
</thead>
<tbody>
<tr>
<td></td>
<td></td>
<td>n</td>
</tr>
<tr>
<td>Difficult To Stop Smoking</td>
<td>Caucasian</td>
<td>15</td>
</tr>
<tr>
<td></td>
<td>African-American</td>
<td>23</td>
</tr>
<tr>
<td>Not Harmful To Health</td>
<td>Caucasian</td>
<td>2</td>
</tr>
<tr>
<td></td>
<td>African-American</td>
<td>3</td>
</tr>
<tr>
<td>Fear Of Gaining Weight</td>
<td>Caucasian</td>
<td>4</td>
</tr>
<tr>
<td></td>
<td>African-American</td>
<td>2</td>
</tr>
<tr>
<td>Feeling Of Joy/Sadness</td>
<td>Caucasian</td>
<td>0</td>
</tr>
<tr>
<td></td>
<td>African-American</td>
<td>4</td>
</tr>
</tbody>
</table>

\(^a\)Smokers category includes current, recent, and reverted smokers (18 Caucasians and 29 African-Americans).
which showed that 36.6% of African-Americans had not graduated from high school as compared with 23% of Caucasians.

Our findings are in agreement with the recent census data which showed that African-Americans were three times more likely than Caucasians to live in poverty (9,20). Despite the difference, nearly three-fourths of the participants of both races were unemployed at the time of the study and had below-poverty incomes. Differences between Caucasians and African-Americans for educational attainment, household income and employment status would be expected to affect food and nutrient intakes.

The racial differences in family income and educational attainment observed in this population are consistent with socioeconomic disparities frequently reported in national surveys. For virtually all chronic diseases, including CVD, low socioeconomic status (SES) has been shown to be a risk factor affecting one-eighth of all Americans and nearly a third of African-Americans (4,13). The results of the current study indicate that inadequate education and unemployment are prevalent among EFNEP participants, especially among African-American women, and these factors would be expected to substantially increase their risks for CVD.

At pre-intervention, the African-American and Caucasian participants had similarly excessive fat intakes with the majority consuming more than 30% kcal/day, which concurs with the findings of other studies (50-51). There were notable racial differences on fat intake, with Caucasians having significantly lower fat intake than the African-Americans. In contrast, other
studies (50-51) have shown lower fat intakes for African-American women than for Caucasians of all ages. Harland et al. (92) assessed nutrient intakes among African-American and Caucasian women in Columbus County, North Carolina, and reported a much higher fat intake for both race groups than those found in the current study. Based on these results, excessive fat intakes pose a major CVD risk for women in EFNEP, particularly for African-Americans.

More than half of the participants in both race groups in the current study had recommended intakes of MUFA at pre-intervention, with the African-Americans having significantly higher intakes than Caucasians, which is in agreement with data from the NHANES II (50), the 1987 NHIS (51), and Harland et al. (92), which showed a mean MUFA intake of 14% and 15% for Caucasians and African-American women, respectively. Similarly high MUFA intakes were reported by Harland et al. Thus low MUFA intakes do not appear to pose a risk for CVD among Caucasian and African-American women in EFNEP.

Meat intakes were not excessive for either race group, but of the six food groups of the Food Guide Pyramid (29), the Meat Group was the one for which both Caucasians and African-Americans had adequate intakes. This finding is consistent with results of other EFNEP studies conducted in Louisiana (15), Virginia (16), and Maryland (17). When compared with the recommended 2-3 servings (equal to 5-7 ounces) in the Food Guide Pyramid, our findings and that of previous EFNEP studies show that meat consump-
tion is adequate, but not excessive, among EFNEP participants. Thus, excessive meat consumption does not appear to pose a risk for CVD among EFNEP women.

More than half of both race groups had higher-than-recommended intakes of SFA and sodium, which concurs with findings of other studies (50-51). NHANES II (39,50) and 1987 NHIS (51) data revealed mean SFA intakes of 14% kcal for both Caucasians and African-American women; however, mean sodium intakes were at recommended levels for low-income women. Therefore, it appears that excessive intakes of SFA and sodium are major CVD risk factors for both Caucasian and African-American women in EFNEP.

In regard to dietary fiber and calcium, a large percentage of both the Caucasian and African-American women had below-recommended intakes, which agrees with findings of Harland et al. (92) and NHANES II data (39). Based on the results of the current and previous studies, low dietary fiber and calcium intakes are substantial risk factors for CVD among both Caucasian and African-American, low-income women.

Of the six major food groups in the Food Guide Pyramid (29), the groups for which both the Caucasians and African-American homemakers were more likely to be low at pre-intervention were the Milk Group, the Vegetable Group, and the Fruit Group. These food groups are important to cardiovascular health because they contribute calcium, potassium, and dietary fiber. Verma et al. (15), Amstuz et al. (17), and Torisky et al. (16)
reported higher mean numbers of servings for these three food groups among EFNEP homemakers than was found in the current study, but their dietary data were collected with only one 24-hour recall per homemaker and collection methods were less precise. It was concluded that low intakes of Milk, Vegetable, and Fruit Groups pose a significant risk of CVD for women in EFNEP.

Both groups of women tended to have lower-than-recommended energy intakes at pre- and post-intervention, with more than 50% of both groups having intakes below 1600 kcal/day, which agrees with findings of NHANES II (39) and other surveys (41,51). Only 26% of the Caucasians and 18% of African-Americans had intakes of more than 2200 kcal/day. In contrast, Harland et al. (92) reported mean energy intakes above the minimum recommended level for Caucasian and African-American women. Based on the results of the current study, it does not appear that excessive intakes of kilocalories pose a CVD risk for women in EFNEP.

Interestingly, results showed that mean intakes of cholesterol, PUFA, and potassium were at recommended levels at pre-intervention, as more than 50% of both race groups had intakes at or above the recommended level, which concurs with NHANES II (39) data and findings of Block et al. (51). In contrast, Harland et al. (92) reported higher-than-recommended intakes of cholesterol for both Caucasian and African-American women, but reported that potassium intakes were at recommended levels. Based on findings in the current study, excessive cholesterol intakes and inadequate intakes of
PUFA and potassium do not pose a major risk of CVD for women participating in EFNEP.

Results revealed that a majority of Caucasian and African-American participants had adequate intakes of the Bread/Cereal Group. In fact, the average number of servings from the Bread/Cereal Group for the 150 Virginia EFNEP homemakers at pre-intervention was double the mean intakes reported by Torisky et al. (16) and Amstutz et al. (17). Foods in the Bread/Cereal Group contribute dietary fiber, if whole grain products are selected (29). In spite of the adequate number of servings from this food group at pre-intervention, mean dietary fiber was well below the recommended level for a majority of participants of both races. This indicates that breads and cereals selected by the participants tended to be refined, rather than whole grain, and were low in fiber. Thus, low dietary fiber intakes may pose a substantial CVD risk for women in EFNEP.

With regard to the Fats/Sweets Group, excessive intakes may contribute to risk of CVD if they lead to obesity or contribute excessive amounts of SFA and/or cholesterol. Results of this study indicate that both Caucasian and African-American participants consumed excessive amounts of fats and sweets, as mean intakes were between 10.6 and 10.8 servings for both groups. This finding does not agree with that of Amstutz et al. (17) who reported an initial mean intake of 2.13 servings from the Fats/Sweets Group for Maryland EFNEP homemakers. In our study, one serving represented 2 teaspoons of sugar or 1 teaspoon of fat; whereas, in the Maryland study, it
was not clear as to what constituted a serving. In the current study, excessive consumption of fats and sweets posed a CVD risk for both Caucasians and African-Americans at pre-intervention and may have contributed to the high prevalence of obesity found among the participants.

Participants in both race groups had similar intakes of almost all nutrients and food groups, except for TF (% kcal), MUFA (% kcal), and the Meat Group for which African-Americans had significantly higher intakes than Caucasians. Thus, it was concluded that homemakers of both race groups in EFNEP have increased dietary risk of CVD, with African-Americans having a slightly higher risk.

Study findings showed that Caucasian and African-American participants of nearly equal mean age and height had similar tendencies to be obese, with the majority having a percent body fat between 34.6% and 36.4% and a BMI between 27.8 to 30.4 kg/m²; however, no significant racial differences were found at the beginning of the study. These findings are consistent with those reported by Kuczynski (93) using NHANES II data, but differ from the Chicago Stroke Study (94) which showed that only African-American women have BMI values greater than 27.3 kg/m². Three other studies (74-75,95) reported lower mean body fat levels and BMI values for Caucasian and African-American women than those in the current study; however, similar to our study, the BMI values were greater for African-Americans than for Caucasians.

Data in the current study revealed that both Caucasian and African-
American women had a high incidence of obesity despite lower-than-recommended energy intakes. This was believed to be due to diets that were high in fat and added sugar, and relatively low in fiber. This finding is in agreement with Miller et al. (57), who showed that obesity in female adults was maintained primarily by having a high portion of energy from fat (36.3% kcal). Another explanation is that a majority of the participants in both race groups were sedentary and the excess weight could have been due to low energy usage. Although the direct association of obesity with CVD remains controversial, studies have shown that obesity is related to high blood pressure and lipid abnormalities which greatly increase risks for CVD in later life, especially for those people who were obese between 20 and 45 years of age (70,74,93,96-101).

At pre-intervention, the Caucasian and African-American participants showed no significant differences in levels of serum TC, LDL-C, triglycerides, HDL-C, and TC:HDL-C ratio. A majority of both groups had mean triglyceride levels within optimal range for prevention of CVD, which is consistent with findings of the NHANES III (102).

Caucasian participants had higher-than-recommended mean levels of serum TC and LDL-C, with 40% being classified as having borderline-high and high levels of these lipid components. This does not agree with findings of Johnson et al. (102) which showed mean serum TC levels of 203 mg/dl for blacks and 208 mg/dl for white women and LDL-C levels of 126 mg/dl for both.
Average HDL-C levels were similar for both Caucasian and African-American participants at pre-intervention and most were in the acceptable range. In contrast, data from the NHANES II (39) and NHANES III (102) showed higher mean values of HDL-C for white and black women.

Data from the Framingham Heart Study have shown that a TC:HDL-C ratio of 4.44 is associated with normal risk of CVD for women (76-78). Caucasian participants in the present study had a mean TC:HDL-C ratio greater than 4.44, with 60% having twice the risk of CVD as compared to 39% of African-American participants. Contrary to our findings, the TC:HDL-C values in NHANES III (102) were 3.5 for blacks and 3.7 for white female adults, which is associated with about half the average risk of CVD (77-78).

Our findings indicate that elevated levels of TC and LDL-C and low HDL-C levels are a risk of CVD for at least one-third of the EFNEP women in both race groups. Several studies have shown that elevated levels of TC, LDL-C, and triglycerides and low levels of HDL-C are more serious in the presence of other non-lipid risk factors for CVD such as cigarette smoking, obesity, and physical inactivity (18,37,72,103-104), which were prevalent among many of the EFNEP homemakers in the present study.

In the present study, significantly more Caucasian participants were current smokers at the beginning of the study. African-Americans smoked significantly fewer cigarettes per day and initiated smoking at a later age, but
had more difficulty quitting smoking than Caucasians, which agrees with the 1992 NHIS data (1). In contrast, investigators in the Minnesota Heart Study (79) reported that more black females than whites were smokers and that the percentage of those who had never smoked was similar for both white and black females. Similar to the current study, other researchers (1,82) have shown an earlier onset of smoking for Caucasians than for African-Americans.

In regard to the number of cigarettes smoked per day, our findings indicate a higher level of smoking among Caucasians than among African-American women, which agrees with findings of others (80,83). In contrast, Manfredi et al. (82) reported a higher mean number of cigarettes smoked per day for both white and black females than was found in our study; however, whites smoked a greater number of cigarettes per day than blacks. Our data indicate that cigarette smoking is a risk for CVD for approximately one-third of the EFNEP study population and was particularly high among Caucasian low-income women.

The African-American and Caucasian women were shown to significantly differ at pre-intervention on demographic factors, with the African-American women having lower levels education and family incomes than Caucasians. However, African-American women had greater intakes of total fat and foods from the Meat Group. Since a significant difference was found between race groups on intakes of TF (% kcal), MUFA (% kcal), and the Meat Group. Thus, Null Hypothesis #1, (that there would be no difference
between Caucasians and African-Americans on dietary factors) was rejected.

The intervention program achieved significant reductions in intakes of energy, TF (% kcal), and SFA (% kcal) as compared with changes of the control group. Although the mean energy intake for both study groups at pre- and post-intervention were within the recommended range, the decrease in energy intake by the experimental group was viewed as a dietary improvement since a majority of the participants were sedentary. A similar pattern of decreased energy intake was demonstrated in the Staff Healthy Heart Project (105) as a result of a six-month dietary intervention. In contrast, Gorbach et al. (106) a reported greater reduction in energy intake than was obtained in the current study.

As a result of the intervention, the experimental group significantly reduced its TF (% kcal) and SFA (% kcal) intakes to levels approximating the Healthy People 2000 Objectives (9). Other studies (7,9,106) using dietary interventions of 4 months or more have obtained similar reductions in TF (% kcal) and SFA (% kcal).

Experimental participants in the current intervention also showed changes in cholesterol and sodium intakes in the desired direction; however, the changes for the experimental group were not significant as compared with the controls. The finding of no significance may be explained by the greater variation of individual intakes reported in the random-repeat 24-hour food recalls as reflected in a relatively large standard error of the mean (SEM) for both groups.
A major emphasis in the intervention program was for participants to consume higher amounts of MUFA (% kcal) in proportion to SFA (% kcal). For MUFA (% kcal), experimental participants showed a significantly greater decrease in intake than the control group. Likewise, the observed mean decrease in PUFA (% kcal) intake tended to be greater for the experimentals than for controls, although differences in change were not significant. These findings are consistent with those other researchers (106-107), who have reported significant reductions in MUFA (% kcal) and PUFA (% kcal) intakes as a result of interventions involving low-fat, low-cholesterol diets.

Both study groups showed increases in dietary fiber; although, experimental participants increased their consumption significantly more as compared with the control group. This increased dietary fiber intake is similar to the results achieved by Barratt et al. (105) among study participants who received nutrition lessons.

In the current study, increases in potassium and calcium intakes by experimentals were greater than that of controls; however, the degree of variation between individuals was too large to reveal a statistically significant difference in change. Though calcium intakes increased somewhat, post-intervention levels were well below the recommended level for the Caucasian and African-American women. The fact that calcium intake and Milk Group consumption increased, while TF (% kcal) decreased, suggests that experimental participants increased their consumption of the Milk Group; but consumed low-fat products.
Consumption levels of all food groups of the Food Guide Pyramid (29) improved after the six-month nutrition education program, but larger increases occurred for the Vegetable Group and the Fruit Group among the experimental participants. Our findings are comparable to results of other studies with EFNEP homemakers (15-17) which also showed significant improvement on food group intakes.

Mean intake of the Meat Group (in ounces) and mean servings from the Fat/Sweets Group decreased significantly among experimentals. These findings do not agree with those of Verma et al. (15), Amstutz et al. (17), and Torisky et al. (16) which showed increases in consumption of these two food groups among EFNEP homemakers in Louisiana, Maryland, and Virginia.

No significant differences were detected between study groups with respect to consumption of foods from the Milk Group and the Bread/Cereals Group. There was a slight increase in intake for the Milk Group but post-intervention intakes were lower than recommended. Our study showed a smaller increase in milk intake some other dietary intervention studies (15-17); however, these studies used single 24-hour food recalls. The use of three random-repeat 24-hour recalls and more precise methods in the current study may explain differences in results. In regard to the Bread/Cereal intake, although experimental participants had intakes within the recommended level, our finding of low fiber intake suggest that their choices tended to be white and refined breads and cereals.

Results of this study revealed that experimental participants improved
their consumption of foods from the Vegetable Group, the Fruit Group, and the Meat Group in comparison with the control group. Thus, Null Hypothesis #2, that food/nutrient intakes would not change over the course of the study, was rejected.

There appeared to be an overall increase in weight for the experimental group over the six-month period; however, the increase was not significantly different from that of the controls. There was also no significant differences in change on percent body fat and BMI between the two study groups. Although, there tended to be greater decreases in percent body fat among experimental subjects as compared with controls, the change was not reflected in decreased BMI values. Thus, Null Hypothesis #3, that there would be no differences between the two study groups on changes for measures of obesity as a result of the intervention, could not be rejected.

Risk reduction for CVD was not accomplished in this study through decreased levels of obesity among either the experimental or control participants. Other studies (108,109) which have shown greater changes in BMI and percent body fat had longer intervention periods (of one year or more) than in the current study and also included more emphasis on physical activity. Svendsen et al. (110) found that weight was significantly reduced for the two intervention groups; however, women from a diet-plus-exercise group achieved significantly greater reductions in weight, fat tissue mass, and abdominal-to-total-body fat mass than the women in a diet-only group.
Based on the results of the current study and those of other authors, diet modification alone may not have the desired effect in decreasing excess adiposity. Though the benefits of exercise were emphasized and walking was encouraged in the current study, no structured exercise program was implemented. Exercise level was not measured precisely, but general questioning indicated that participants engaged in little planned exercises and there was no increase in physical activity during the study. Apparently, the dietary intervention, alone, was unable to generate caloric deficits large enough to cause a significant difference in obesity among the experimental participants.

Another objective of the current study was to determine the effects of the intervention on the blood lipid components and if significant changes would occur as a result of the intervention. Based on available evidence, the NCEP (18) experts report that for each 1% reduction in TC levels, there is a 2% reduction in CHD rates. On the other hand, for every 1.0 mg/dl increase in TC levels, the risk for CHD increases by about 1.0%. For every 1.0 mg/dl decrease in HDL-C levels the risk of CHD rises by 2% to 3% (18,78).

In our study, the direction of change in blood lipid levels was inconsistent in that there was no definable pattern of change made by either study group. Moreover, there was no significant difference in the amount of change made by the participants in the two study groups for any of the lipid values. For TC, LDL-C, and TC:HDL-C ratio, the control group tended to improve more in that their mean blood values decreased; however, the differ
ence did not reach significance. Small increases in triglyceride and HDL-C levels were observed by the end of the intervention program among experimental participants, but differences in the two study groups were not significant. Thus, null hypothesis #4, that there would be no differences between the two groups on changes in biochemical measures, could not be rejected.

There are several possible explanations for the finding of no significant differences in lipid profile changes of experimental participants vs. control participants, as a result of the intervention. First, pre-intervention mean values for TC, LDL-C, and triglyceride levels were not high among either controls or experimentalists. In fact, the experimental group had very few participants with elevated mean serum TC and LDL-C levels and none had elevated triglycerides. Thus, a large reduction in blood lipid levels and significant differences between the study groups could not be expected. Failure to achieve further reductions in TC levels among normocholesterolemic individuals is consistent with results reported by Gomel et al. (107) who did not achieve lipid changes after a 3 to 6-month intervention. The Multiple Risk Factor Intervention Trial (108) showed greater reductions in serum TC levels in men with hyperlipidemia (high TC and triglyceride levels) at baseline, over a 24-month intervention. Another possible explanation for the lowered mean blood lipid values was the relatively young mean age (28-30 years) of the participants, with most not having gone through menopause. It has been shown that women have less atherogenic lipid and lipoprotein profiles before menopause (36).
Second, although the experimental participants showed a significantly greater decrease in TF (% kcal) and SFA (% kcal) intakes, with the intervention as compared to the controls, there was also a proportionately greater reduction in MUFA (% kcal) and PUFA (% kcal) intakes by the experimental group. In a controlled feeding trial among women by Masana et al. (47), diets having high levels of MUFA and/or PUFA and high P:S ratios induced significantly greater decrements in serum TC, LDL-C, and triglyceride levels, as compared with those having diets low in MUFA (% kcal) content and low P:S ratios. However, there were no significant changes in HDL-C levels observed with the four diets in the Masana study.

Thirdly, though the dietary improvements associated with risks for CVD in the current study were significantly different between the study groups after the six-month intervention, the time period of the intervention may have been too short to achieve significant blood lipid changes. Also, the dietary changes may have came too late in the intervention period to effect the post-intervention blood lipid values. Other nutrition intervention programs, which have achieved significantly greater reductions in TC, LDL-C, and triglyceride levels, had intervention periods of at least three to six years (108, 111). On the other hand, the National Aeronautics and Space Administration Cardiovascular Risk Reduction Program (112) using a combination of dietary changes and physical exercise, achieved significantly lower TC and LDL-C levels and improved HDL-C and TC:HDL-C ratios among women, with only a two-month intervention period.
Lastly, the failure to improve blood lipid levels may have been due to varying dietary patterns and physical activity at different times of the year when data were collected. Most of the pre-intervention blood lipid values were collected during the summer and fall months when participants may have been more physically active, as many reported working on tobacco farms. On the other hand, post-intervention lipid data were conducted during the winter months when participants would have been least physically active, as indicated by a gain in weight among a majority of experimental subjects.

Another purpose of the current study was to determine if the self-reported smoking habits (including smoking status and number of cigarettes smoked per day) would improve as a result of the intervention. In this study, despite anti-smoking messages in a number of lessons included in the intervention program, results on change in smoking status and number of cigarettes smoked per day did not suggest clear intervention effects. Thus null hypothesis #5, that smoking and number of cigarettes smoked would not be reduced, could not be rejected. An explanation for the lack of significant results is that the intervention did not include a structured smoking cessation campaign of sufficient duration. Other studies (108,111), which have reported significant decreases in numbers of cigarettes smoked, utilized programs in which smoking cessation was a main focus and which were more intensive and of longer duration than that of the current study.
CHAPTER V
SUMMARY, CONCLUSIONS, AND RECOMMENDATIONS

This study was conducted to identify risk factors for CVD among Caucasian and African-American low-income, female homemakers enrolled in the EFNEP of Virginia, and to assess whether a six-month nutrition education program would result in improvements in certain diet-related and lifestyle-related risk factors for CVD. The study provided a descriptive profile of the EFNEP adult female population in three rural counties and one city of Virginia, regarding two aspects: (a) a description of the EFNEP population and racial differences on risks for CVD as related to demographics, diet, obesity, bio-chemical factors, and smoking characteristics and (b) comparisons of the amount of change on these variables between the control and experimental groups, as a result of an intervention.

At pre-intervention, Caucasian and African-American participants were significantly different on educational attainment, monthly income, intakes of TF, MUFA, and servings from the Meat Group with the African-Americans having higher intakes of meat and fat. In contrast, they were similar on work status, place of residence, mean age, number of children and number of members in the household. The two race groups also had similar dietary intakes of energy, SFA, PUFA, cholesterol and servings from the Milk, Fruit, Vegetable, and Fats/Sweets Groups of the Food Guide Pyramid.
Both race groups had excessive mean intakes of TF, SFA, sodium, and the Fats/Sweets Group. At the same time, their mean intakes of dietary fiber, calcium, PUFA, the Milk Group, the Vegetable Group, and the Fruit Group were below-recommended levels. Thus, the only nutrients and food groups for which mean intakes were at, or close to recommended levels were kilocalories, MUFA, cholesterol, potassium, and the Meat and Bread/Cereal Groups.

In regard to obesity, a majority of both race groups were obese, based on both indices of obesity including percent body fat and BMI. On selected blood lipid profiles at pre-intervention, a majority of the participants in both race groups of had lipid levels within desirable ranges, with 20% or less having TC and LDL-C levels in the high category. No participants had high triglyceride levels. It was surprising that lipid profiles tended to be normal in spite of the excessive intake of TF and the high prevalence of obesity among both race groups.

Significantly more Caucasians than African-Americans were current smokers at the beginning of the. Furthermore, Caucasians had initiated smoking at a significantly younger age than African-Americans. Also, Caucasian female smokers had significantly smoked more cigarettes/day than the African-Americans, which places them at higher-risk for chronic diseases such as CVD.
As a result of participating in the intervention, experimental participants significantly increased their mean intakes of dietary fiber and decreased their intake of energy, TF, SFA, Meat Group (in ounces) and Fats/Sweets Group. The significant changes in nutrient intake among the experimental participants, are associated with significant increases in foods from the Vegetable Group and the Fruit Group; whereas, there was no such positive improvement among controls.

Differences in change on percent body fat, BMI, selected biochemical measures, and smoking characteristics of the experimental group vs. the control group were not significant. Possible explanations for the inability to generate significant changes among the experimentals on these factors, are: (1) too small sample on which obesity and biochemical measures were taken (i.e. 37 experimental and 38 controls) and (2) duration of intervention period was too short for dietary changes to affect physical and biochemical measures, and (3) normal variation in diet and level of exercise at different times of the year, resulted in pre-intervention data being collected in the summer and fall, when diets and exercise level may have normally been better) and post-intervention data being collected in the winter and early spring, when diet and exercise levels may have been poorer.

Conclusions

Findings of this study indicate that both Caucasian and African-American low-income, female homemakers in EFNEP have an increased risk
for CVD due to dietary excesses of TF, SFA, and sodium and deficiencies of dietary calcium, fiber, and PUFA. They have additional risks for CVD because of a high prevalence of obesity, smoking, and lack of exercise. It was concluded that the intervention was successful in that it resulted in reducing risks for CVD through several positive dietary changes: increased intakes of dietary fiber, the Vegetable Group, and the Fruit Group, and decreased intakes of energy, total fat, SFA, the Meat Group, and the Fats/Sweets Group. On the other hand, the intervention in its current form, failed to achieve lower risks for CVD through a reduction in levels of obesity and smoking and improvement in blood lipid profiles and exercise among experimental participants.

Recommendations for Program Change and Future Research

1. Perform additional assessments of the impact of the educational intervention and increase the duration of the intervention period, to 12 months or more, to allow more time for dietary changes to affect physical and biochemical measures.

2. Expand and improve the intervention in regard to weight control, exercise, and smoking cessation.

3. In future assessments, collect pre and post-intervention data at the same time of the year (during the same month but 1 year apart) to prevent the interference of seasonal variations on food intake and exercise.
4. Include a larger sample size and one that is more evenly divided among the races being studied, especially for those on whom physical and biochemical data will be collected.

5. Add or re-phrase questions on questionnaires to gather more specific data on type, duration, and intensity of exercise.
REFERENCES


86. Sutherland M, Harris G, Barber M. Cardiovascular health promotion in southern rural African-American churches. Presented at the American Public Health Association Meeting; November. 1992; Washington, DC.


APPENDICES
### APPENDIX A

**SUMMARY EFFECTS OF VARIOUS DIETARY FATS AND CHOLESTEROL IN RELATION TO RISK OF CVD**

<table>
<thead>
<tr>
<th>Term</th>
<th>What does it do?</th>
<th>Where does it come from?</th>
</tr>
</thead>
<tbody>
<tr>
<td>Cholesterol</td>
<td>Builds healthy cells, but too much clogs the arteries, leading to heart attack or stroke. Carried in the blood linked to proteins called lipoproteins.</td>
<td>Made in the body, but we consume it from animal foods like liver and other organ meats, meat, poultry, shellfish, dairy products, egg yolk.</td>
</tr>
<tr>
<td>High Density Lipoprotein-Cholesterol (HDL-C)</td>
<td>Form in which excess cholesterol is cleared from the blood; often called &quot;good cholesterol&quot;.</td>
<td>Made in the body; Blood levels are related to heredity, but are increased by strenuous exercise and weight loss, (if overweight).</td>
</tr>
<tr>
<td>Low Density Lipoprotein-Cholesterol (LDL-C)</td>
<td>Favours the build up of cholesterol in the artery walls leading to heart disease. Known as &quot;bad cholesterol&quot;.</td>
<td>Made in the body; Blood levels are increased by diets high in saturated fat and cholesterol and by being overweight.</td>
</tr>
<tr>
<td>Polysaturated Fats</td>
<td>Lowers blood cholesterol levels. It is the source of the essential fatty acid, linoleic acid, which the body does not make.</td>
<td>Fish, soy, corn, safflower and sunflower oils. English walnuts, salad dressings, mayonnaise, margarine where liquid oil is first ingredient.</td>
</tr>
<tr>
<td>Omega-3 Fatty Acids</td>
<td>Current research shows that these fatty acids lower both blood cholesterol and blood triglycerides.</td>
<td>Fatty fish, such as salmon, mackerel, anchovy, herring, sardines, tuna, rainbow trout, and whitefish.</td>
</tr>
<tr>
<td>Monounsaturated Fat Acids (MUFA)</td>
<td>Recent studies show that olive oil, containing MUFA, helps to lower blood cholesterol. More research is needed to determine the effect of other oils on blood cholesterol.</td>
<td>Almonds, peanuts, olive oil, palm oil, peanut butter, avocado, canola oil.</td>
</tr>
<tr>
<td>Saturated Fat</td>
<td>Raises blood cholesterol and is usually solid at room temperature.</td>
<td>Beef, pork and poultry fat, cream, cheese, chocolate (cocoa butter), coconut &amp; palm oils, lard.</td>
</tr>
<tr>
<td>Triglycerides</td>
<td>Chemical name for fat. Carried in the blood by lipoproteins; Too high blood levels increase risk of heart disease.</td>
<td>Made in the body; Found in many sources of fat such as vegetable oils, meat, poultry, seafood, dairy products, and chocolate.</td>
</tr>
<tr>
<td>Total Fat</td>
<td>Term meaning the amount of all the fat (triglycerides) we eat. Nutritionists recommend eating less than 30% of calories from fat because high fat diets are a risk factor for developing heart disease.</td>
<td>Any food high in saturated, monounsaturated, and polyunsaturated fat, such as heme proteins, meats, whole milk, corn oil, peanut butter, fried foods, cakes, cookies, and many crackers.</td>
</tr>
<tr>
<td>Body Fat</td>
<td>Term meaning the amount of fat (triglycerides) stored in the body. Fat is necessary to insulate the body and carry certain nutrients, like vitamin A, but too much leads to obesity. Being obese can lead to elevated blood cholesterol and blood pressure.</td>
<td>Caused by consuming more calories than are burned. Extra calories can come from carbohydrates (fruit, sugar, bread) and protein (meat, milk, fish) as well as fat; however, fat calories convert more readily to body fat.</td>
</tr>
</tbody>
</table>
APPENDIX B

PRIMARY PREVENTION IN ADULTS: INITIAL CLASSIFICATION FOR INDIVIDUALS WITHOUT EVIDENCE OF CVD BASED ON TOTAL CHOLESTEROL, HDL-C, AND LDL-C

Primary Prevention in Adults Without Evidence of CHD; Initial Classification Based on Total Cholesterol and HDL-Cholesterol

- Measure nonfasting total blood cholesterol and HDL cholesterol
- Assess other nonlipid CHD risk factors
- Desirable blood cholesterol <200 mg/dL
  - HDL ≥35 mg/dL
  - HDL <35 mg/dL
- Borderline-high blood cholesterol 200-239 mg/dL
  - HDL ≥33 mg/dL and fewer than 2 risk factors
  - HDL <35 mg/dL or 2 or more risk factors
- High blood cholesterol ≥240 mg/dL

- Repeat total cholesterol and HDL within 5 years or with physical exam
  - Provide education on general population eating pattern, physical activity, and risk factor reduction
- Provide information on dietary modification, physical activity, and risk factor reduction
  - Reevaluate patient in 1-2 years
    - Repeat total and HDL-cholesterol measurement
    - Reinforce nutrition and physical activity education
- Do apoprotein analysis
  (Go to figure 1-3)

CHD Risk Factors
Positive
- Age: Male ≥45 years
- Female ≥55 years or premenopausal within estrogen replacement therapy
- Family history of premature CHD
- Smoking
- Hypertension
- HDL cholesterol <35 mg/dL
- Diabetes

Negative
- HDL cholesterol ≥60 mg/dL
APPENDIX B continued

Primary Prevention in Adults Without Evidence of CHD: Subsequent Classification Based on LDL-Cholesterol

Lipoprotein analysis fasting, 9-12 hours (may follow a total cholesterol determination or may be done at the outset)

Desirable LDL-cholesterol <130 mg/dL

Borderline-high-risk LDL-cholesterol 130-159 mg/dL and with fewer than 2 risk factors

130-159 mg/dL* and with 2 or more risk factors

High-risk LDL-cholesterol ≥160 mg/dL.*

Do clinical evaluation (history, physical exam, and laboratory tests):
- Evaluate for secondary causes (when indicated)
- Evaluate for familial disorders (when indicated)
Consider influences of age, sex, other CVD risk factors

Repeat total cholesterol and HDL-cholesterol measurement within 5 years

Provide education on general population eating pattern, physical activity, and risk factor reduction

Provide information on the Step I Diet and physical activity

Reevaluate patient status annually, including risk factor reduction
- Repeat lipoprotein analysis
- Reinforce nutrition and physical activity education

Initiate dietary therapy
See pages 1-21, 1-22

* 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 4-8 weeks and the average value of three tests used.
APPENDIX D

INFORMED CONSENT FORM

EFNEP NUTRITION-BASED CANCER PREVENTION STUDY

Introduction

I understand that the Virginia EFNEP and the Massey Cancer Center are doing this study to learn how to best educate people to reduce their risk of cancer by making dietary changes. I agree to be involved in the study by providing information to an EFNEP Technician for filling out the following forms:

1. EFNEP Family Record (Parts A and B) Pre Post
2. Family Record Part C: Homemaker Food Recall(3 times) X X
3. Health Appraisal X X

I understand that I may be assigned to only one of the following three groups during the study period. The three groups include:

1. A control group (C) which will receive information on money management and at the end of the study receive information on nutrition.
2. An EFNEP group (E) who will receive the standard EFNEP instruction.
3. An EFNEP plus cancer education group (EC) who will receive the standard EFNEP instruction plus the new cancer prevention lessons.

At the end of the study, everyone will have an opportunity to receive the cancer education lessons.

I also agree, that if I am selected to be in a special sub-sample, I will come to a designated location and have some blood drawn to determine my blood level of Vitamin A, Vitamin C, iron, lipids (i.e. fats and cholesterol). I understand that someone will provide transportation for me to come to a site in ________ for the purpose of having a sample of my blood taken. I also understand a registered nurse will do the drawing. If I am in the sub-sample to have my blood drawn, $15 will be paid to me the first time my blood is drawn and $20 the second time. It is important that I have my blood drawn at the beginning and end of the study. A nurse or another trained person will do measure my weight, height and body fat, and instruct me on breast self exam at this time.

Benefits

I understand that by following the dietary instructions I may reduce my personal and family members' risks for cancer, heart disease and hypertension.

Alternatives

I understand that currently, there are no other known effective methods of reducing one's risk for preventable diseases.

Risks

I understand that having my blood drawn may pose a minor risk of infection and pain due to the needle puncture, but that every precaution possible will be taken to avoid infection and pain on my part.

________ subject initials
APPENDIX D continued

Costs

If I am in the sub-sample and have my blood drawn, a stipend will be paid to me each
time my blood is drawn. No other reimbursement will be given for participation in the
study.

Pregnancy

I may still participate if I am pregnant. However, if my doctor places me on a special diet
I should follow the diet ordered by my doctor.

Research Related Injury

I understand that if any physical and/or mental injury occurs from my being in this
research study, Virginia Commonwealth University and Virginia Polytechnic Institute will
not offer compensation. If injury occurs, medical treatment will be available at MCV
Hospitals. I understand that the cost of this treatment will be billed to me or my insurance
company.

Confidentiality of records

I understand that the purpose of the study is to improve the EFNEP Program. My name
will not be used on any of the forms or on written reports. Information that I have given
will be kept confidential and will not be given to any other agency or used by anyone
other than EFNEP and the Massey Cancer Center.

Withdrawal

I also understand that, if I decide not to be involved in the study I can still be in the
EFNEP Program and it will not harm my relationship with any other agency.

Additional Information

If I have questions at any time during to this study, I can contact the following people
responsible for this study.

Gwen Parker, R.N. 
Massey Cancer Center  
Medical College of VA. 
Richmond, VA 23298  
Phone: 804-786-0450

Ruby H. Cox, R.D. 
Virginia Tech  
411 Fannery Hall  
Blacksburg, VA 24061  
Phone: 703-231-7156

Office on Subject Flights (IRB)  
Office: (804) 786-0868 (MCV)

By signing below, I indicate that I have read this form, received acceptable
answers to any questions and willingly agree to participate. I will receive a copy
of this form. You may also receive the results of this study if you desire.

(Participant's Signature)  
(Date)

(Witness’s Signature)  
(Date)
APPENDIX E

PROCEDURES FOR COLLECTING A 24-HOUR FOOD RECALL

When taking the 24-hour recall, it is important for the interviewer to follow certain procedures to insure the following:

- That all foods and beverages consumed by homemakers are listed.
- That amounts of each food is determined as accurately as possible.
- That homemakers are not influenced to say they ate foods that they did not eat.

Setting the stage for the interview

The following steps will help in eliciting truthful information:

1. Explain to respondent that you need to know only what she actually ate. They should not feel embarrassed about any food, as there are no "good" or "bad" foods. No one eats just the right foods all the time.

2. Do not express in words or facial expressions either approval or disapproval of foods which respondent mentions.

3. Do not ask leading questions that would lead homemaker to feel they "should" have had a certain item and, thus, say they did.

During the food recall interview

1. Use your FOOD RECALL KIT to determine the amounts of foods consumed. (Food Recall Kit described below under "24-Hour Food Recall Kit".) Respondents may not be able to give amounts of ingredients in their portions of mixed dishes, salads and casseroles. If a home recipe was used, obtain a copy. If eaten in a restaurant, record name of the restaurant (Call restaurant later to get information on preparation.

2. Start with the most recent meal or snack that the respondent consumed. Work backwards to cover all foods and beverages eaten or drunk in the last 24 hours.

3. First, get a complete list of all foods eaten without trying to determine amounts. Use the following types of probes to find what foods were eaten:
APPENDIX E continued

A. The first type of probe is related to time. 
   Examples: 
   "At what time was this? Did you eat or drink anything after 
   that?" 
   "What did you have at that time? 
   "At what time did you go to bed?"

B. The second type of probe is related to respondent’s activities. 
   Examples: 
   "While you were working around the house did you take a 
   break to have something to eat or drink?" 
   "Did you watch TV last night? When you watched TV, did 
   you eat anything? 
   "Did you have anything to drink with this?"

C. The third type of probe tries to get more complete information 
   about foods already reported. 
   Examples: 
   "Do you remember anything else that you ate or drank with this 
   foods? 
   "What else did you have at this meal?" 
   "Was the (bread, vegetable) eaten plain or did you put 
   something on it?" 
   "Did you put anything in your coffee?"  "Was the cream really 
   cream or was it milk or coffee creamer? 
   "Did you have a second helping?"

4. Second, after all foods are named by the respondent, go back over the 
   lists to get additional descriptions and amounts of the food. Also 
   determine if all of the food was eaten or if some was left on the plate.

To get more information on the type of food:

A. Encourage the respondent to describe foods as clearly as 
   possible. The interviewer may have to restate questions to get 
   more information.

B. Describe combination dishes carefully. Mixtures such as 
   sandwiches, soups, stew, pizza. casseroles, etc. can be 
   prepared in many ways.
APPENDIX E continued

C. Ask to see packages, if available, for as many foods as possible and record brand name and other pertinent information.

To determine the amount of food eaten:

A. Amounts of a food may be given in

1. **NUMBERS**, such as eggs, donuts, apples
2. **SHAPES**, such as a pat of butter, a stalk of celery, or a slice of pie
3. **DIMENSIONS** can be described using the cardboard models in the "24-Hour Recall Kit." For example, a piece of cornbread may be describe as a 3"X 2" piece.
4. **VOLUME**, such as liquids and cooked vegetables, can be based on a standard one-cup measure.
5. **WEIGHT**, such as meat, cheese, candy bar, (3 oz. meat equal size of deck of cards)

B. In determining amounts of foods, use food models, measuring cups, measuring spoons, raw rice, etc. in Food Recall Kit. Have respondent show you how much they had by pouring raw rice on a plate or by identifying some item that you have in your recall kit. A ruler can also be used to show size of certain items.

C. When appropriate, ask respondent to bring in the serving container (bowl, cup, glass, etc.) that was used and determine the amount it holds by using rice and a standard measuring cup.

5. If nutrition questions are being asked by the respondent during the time the recall is being taken, ask respondent if you may answer them when you have completed the recall.

6. After the homemaker has given a recall of foods and amounts for the entire 24 hours, read the list back to them and ask respondent to tell you anything that she may have forgotten.

7. Thank the respondent for her cooperation. Only comment on the recall, if respondent ask a specific question. Wait to address deficiencies and excesses until lessons are taught that deal with that area of the diet.
APPENDIX E continued

24-Hour Food Recall Kit

Purpose: to assist interviewer them in taking the assessment, a kit has been prepared for you.

The Food Recall Kit contains the following items:

   Cup - One 8-oz. plastic measuring cup

   Bowls - Two different shapes holding 2 cups each

   Small sauce dish - Holds 1/2 cup

   Standard Measuring Spoons:
      - 1 tablespoon
      - 1 teaspoon
      - ½ teaspoon
      - 1/4 teaspoon

*Plastic Container of 2 to 3 cups rice (tight fitting lid)

* Plastic container of 2 to 3 cups dried beans

   Note: Rice will be used as an example for measuring more dense foods such as mashed potatoes and oatmeal. Beans may be used for foods that are loosely packed, such as cereal or vegetables.
APPENDIX E continued

10 Cardboard Shapes Representing Various Servings Sizes:

A - 1" square  
B - 2" square  
C - 1/16 of a layer cake  
D - 1/12 of a layer cake  
E - 1/8 of a 9" pie  
F - 1/7 of a 9" pie  
G - 1/6 of a 9" pie  
H - 3" square  
I - 4" circle  
J - 1/4 of a 10" pizza  

1" cube cheese  
brownies  

cake  

pie, quiche  

pie, quiche  

pie, quiche  

pie, quiche  

1/9 of a 9" sheet cake  
danish, pancake  
pizza

Additional shapes of various cuts of meats, each about 3 ounces of cooked, edible portion.

Compiled by: Ruby H. Cox, Ph.D., R.D. (12/92)

Parts of these procedures are adapted from the Enhanced EFNEP Record and Reporting Manual from the New York EFNEP Program, Cornell University.
Patterns for Food Models

APPENDIX E continued
Sample of materials used for food models.
APPENDIX E continued

Round Steak (lean only)

One piece this size: About 160 calories
EQUALS 3 OZ. EDIBLE PORTION

Roast Beef Round (lean only)

Two slices this size: About 160 calories
EQUALS 3 OZ. EDIBLE PORTION
APPENDIX E continued

Veal Cullet (trimmed)

This thick

One culet this size: About 185 calories
EQUALS 3 OZ. EDIBLE PORTION

Ham (lean only)

This thick

Two slices this size: About 160 calories
EQUALS 3 OZ. EDIBLE PORTION

Hamburger (lean)

This thick
APPENDIX F

PROCEDURES FOR BLOOD SAMPLING AND TRANSPORT TO LABORATORY

Blood analysis for nutrient levels and lipids will be performed on a sample of ___ homemakers (___ control and ___ experimental) which will be randomly selected from among the 150 subjects in the pilot study. Permission to obtain blood samples will be incorporated in the consent obtained during the enrollment of homemakers in the program.

Blood collection will occur upon enrollment in the program, prior to any nutritional counseling, and at six months after the instruction is received. Those to participate in the blood drawing were provided with a list of instructions and were instructed not to eat or drink anything, other than week, non-sweetened tea or diet, non-caffeinated soft drinks after midnight on the night before the blood drawing and not to eat or drink anything that morning. They were informed that snacks would be provided to them at the Health Clinic after their blood was drawn. Participants were transported to and from the Health Department Clinics by EFNEP/Extension staff, on the day of blood drawing.

Blood lipid values will be used to identify those participants with abnormality values and changes as a result of dietary modification. Changes in serum lipid levels, as a result of dietary changes, would become apparent in three to four weeks.

Blood collection will be performed by the study investigators who are registered nurses and skilled in venipuncture. Supplies for blood collection will be donated by the investigator’s institution. Blood specimens will be transported by the investigators to the lab for processing. Negotiations are currently under way for selection of a lab to process the samples.

While at the Health Department clinic, physical measurements were also collected by the investigators. In addition, they were given instruction on doing breast self-exam and the warning signs of cancer. Literature on cancer prevention will also be provided. Any abnormal assessments will be referred immediately to the Massey Cancer Center, Medical College of Virginia hospital for follow-up.
## APPENDIX G

### INSTRUMENTS

**Family Record Form (Part A)**

### Expanded Food and Nutrition Education Program

<table>
<thead>
<tr>
<th>Family Record-Part A-Description</th>
</tr>
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<tbody>
<tr>
<td><strong>Unit ID #</strong></td>
</tr>
<tr>
<td><strong>Family ID #</strong></td>
</tr>
<tr>
<td><strong>Date Family Enrolled</strong></td>
</tr>
<tr>
<td>Month</td>
</tr>
<tr>
<td><strong>Date Record Completed</strong></td>
</tr>
<tr>
<td>Month</td>
</tr>
</tbody>
</table>

### Place of Residence

- Farm
- Towns (rural non-farm)
- Under 10,000
- Places 10,000 to 50,000
- Suburbs over 50,000
- Central Cities

### Homemaker's Racial/Ethnic Characteristics

- **White (not Hispanic Origin)**
- **Black (not Hispanic Origin)**
- **Hispanic**
- **American Indian/Alaska Native**
- **Asian or Pacific Islander**

### Total/Actual Income for Family Last Month (round to the nearest dollar)

$\phantom{0000}00$

**NOTE**

Place one number in each box. For incomes below $1,000, place zeros in the leading box(es).

### Family Received (items listed during the year)

- Yes
  - USDA Food Stamp/Food Distribution Program
  - WIC/SFP
  - Child Nutrition Programs (school lunch and/or breakfast, milk, Head Start, summer programs, child care)
- No
  - Public Assistance

### Family Members

<table>
<thead>
<tr>
<th>Homemaker</th>
<th>Age</th>
<th>Gender</th>
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<tbody>
<tr>
<td></td>
<td></td>
<td>M F</td>
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</table>

### Total Family Members

<p>| |</p>
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### Highest Grade Completed by Homemaker

- 8th Grade or Less
- 9th-11th Grade
- 12th Grade or GED
- Beyond High School

### Ending Date

<table>
<thead>
<tr>
<th>Month</th>
<th>Day</th>
<th>Year</th>
</tr>
</thead>
</table>

### Reasons for Incompletion:

- Completed Program
- Did Not Complete
- Moved
- Not Interested
- Working, Returned to School
- Aide Vacancy
- Other
### FAMILY RECORD - PART C - FOOD RECALL

**Record Number [ ]**  
Number of Teaching Contacts since last Record:  
- [ ] Individual  
- [ ] Group  
- [ ] By Mail  
- [ ] Telephone  
- [ ] Other  

Homemaker:  
- [ ] Pregnant [Yes] [No]  
- [ ] Breast-feeding [Yes] [No]  

<table>
<thead>
<tr>
<th>Meal</th>
<th>Food Item</th>
<th>Amount Eaten</th>
<th>Meal Code</th>
<th>Food ID Number</th>
<th>Amount Code</th>
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<tbody>
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**Meal Codes to Use**  
<table>
<thead>
<tr>
<th>Column 1</th>
<th>Name of Meal</th>
<th>Column 4</th>
</tr>
</thead>
<tbody>
<tr>
<td>R</td>
<td>Breakfast</td>
<td>1</td>
</tr>
<tr>
<td>L</td>
<td>Lunch</td>
<td>2</td>
</tr>
<tr>
<td>D</td>
<td>Dinner</td>
<td>3</td>
</tr>
<tr>
<td>S</td>
<td>Snack</td>
<td>4</td>
</tr>
</tbody>
</table>

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TBSI=Tablespoon  
tsp=teaspoon  
c=cup  
oz=ounce
APPENDIX G continued

Health Risk Appraisal

Health Appraisal

This form asks about your health and eating habits. This is not a test. There are no right or wrong answers. I will ask you some questions and would like for you to answer them to the best of your ability. Thank you.

PERSONAL INFORMATION, HABITS

1. When were you born? (Month - day - year)
2. How old are you? _______ years old

DIETARY HISTORY

This section asks about the foods you eat. Do you eat the following foods weekly?

3. fruits/berries (examples - apples, grapes, oranges, strawberries, fruit juices) __ No __ Yes
   IF "YES," how many servings did you eat last week? ______ servings

4. dark green, leafy vegetables (examples - spinach, greens, broccoli, kale) __ No __ Yes
   IF "YES," how many servings did you eat last week? ______ servings

5. deep yellow vegetables (examples - squash, carrots, sweet potatoes) __ No __ Yes
   IF "YES," how many servings did you eat last week? ______ servings

6. fast foods? (examples - Tastee Freeze, McDonald's, Pizza Hut, Wendy's) __ No __ Yes
   IF "YES," how many servings did you eat last week? ______ servings

7. cured or processed meats? (examples - bacon, hotdogs, luncheon meats) __ No __ Yes
   IF "YES," how many servings did you eat last week? ______ servings

8. smoked meats? (examples - ham, hambrocks) __ No __ Yes
   IF "YES," how many servings did you eat last week? ______ servings
APPENDIX G continued

9. whole grain breads or cereals? (examples - wheat bread, bran flakes) __ No __ Yes
   IF "YES," how many servings did you eat last week? __ servings

10. fried foods? (examples - french fries, fried chicken, potato chips) __ No __ Yes
   IF "YES," how many servings did you eat last week? __ servings

11. Do you drink alcoholic beverages now? (examples - beer, wine, whiskey, mixed drinks) __ No __ Yes
   IF "YES," how many drinks did you have last week? __ cans/bottles/glasses/drinks
   If you drink only occasionally, how many drinks do you usually have each month?
   __ cans/bottles/glasses/drinks
   At what age did you start drinking? __ years old
   How many total years have you been drinking alcohol? __ years

   IF "NO," did you ever drink alcoholic beverages in the past? __ No __ Yes
   If you did drink, on the average how many drinks did you usually have in a week?
   __ cans/bottles/glasses/drinks
   How many total years did you drink? __ years
   If you used to drink but have quit, at what age did you quit? __ years old

OCCUPATIONAL INFORMATION

12. What is your current work status?
   (Check the one that applies to the greatest percent of your time.)
   1. __ Student  4. __ Homemaker  7. __ Other
   2. __ Employed  5. __ Unemployed
   3. __ Retired  6. __ Disabled, unable to work

180
APPENDIX G continued

13. List the kinds of jobs you have had. (If you worked in a factory, what was made at that factory?)

_________________________________________________________________________

_________________________________________________________________________

_________________________________________________________________________

14. Of all your jobs, which one have you worked in the longest?

(Job or occupation) __________________________ (Years on the job) ______________________

15. In that job, where did you spend most of your time? (Please check one.)

   __ Indoors   __ Outdoors

FAMILY HISTORY

16. Have any of your blood relatives ever had cancer?   ___ No   ___ Yes

   If "Yes," please list each blood relative who has had cancer, one relative per line. List only those relatives listed here: mother, father, brother, sister, grandmother, grandfather, daughter, son.

   Relationship   Type of Cancer   Is this person still living? (No or Yes)
   ___________________________________________________________________________
   ___________________________________________________________________________
   ___________________________________________________________________________
   ___________________________________________________________________________

SMOKING HISTORY

17. Have you smoked at least 100 cigarettes (5 packs) in your entire life?   ___ No   ___ Yes
APPENDIX G continued

IF "YES," about how old were you when you first started smoking cigarettes regularly?

______ years old

On the average, of the entire time you smoked, how many cigarettes did you smoke per day?

______ cigarettes per day

Do you smoke cigarettes now?

______ No ______ Yes

IF "NO," how old were you when you stopped smoking?

______ years old

IF "YES," on the average, how many cigarettes a day do you smoke now?

______ cigarettes per day

18. Have you ever smoked a pipe or cigar?

______ No ______ Yes

IF "YES," for how many months/years?

______ months ______ years

About how much?

pipes or cigars per ______ day ______ week

19. Have you ever used smokeless tobacco?

______ No ______ Yes

IF "YES," for how many months/years?

______ months ______ years

About how much?

chewing tobacco or snuff per ______ day ______ week

20. Does anyone in your household smoke? (Do not count yourself.)

______ No ______ Yes

IF "YES," on the average, about how many packs a day do all of the smokers in your household smoke? (Do not count yourself.)

______ packs of cigarettes

MEDICAL HISTORY

21. How tall are you?

______ (feet & inches)

22. What is your current weight?

______ pounds

23. Have you ever been told by a doctor that you had a health problem of any kind?

______ No ______ Yes

If you were told you had a problem, what were you told and when?

______________________________
APPENDIX G continued

Health Risk Appraisal II

Rev. 06-30-92 2:39 PM
Unit ID ____________________
Participant ID ____________________
Collection Date ____________________

Health Appraisal II

This form asks about your health and eating habits. This is not a test. There are no right or wrong answers. I will ask you some questions and would like for you to answer them to the best of your ability. Thank you.

PERSONAL INFORMATION, HABITS

1. When were you born?
   (Month - day - year)

2. How old are you?
   ________ years old

DIETARY HISTORY

This section asks about the foods you eat. Do you eat the following foods weekly?

3. Fruits/berries (examples - apples, grapes, oranges, strawberries, fruit juices) ______ No ______ Yes
   IF "YES," how many servings did you eat last week? ______ servings

4. Dark green, leafy vegetables (examples - spinach, green, broccoli, kale) ______ No ______ Yes
   IF "YES," how many servings did you eat last week? ______ servings

5. Deep yellow vegetables (examples - squash, carrots, sweet potatoes) ______ No ______ Yes
   IF "YES," how many servings did you eat last week? ______ servings

6. Fast foods? (examples - Taste Freeze, McDonald's, Pizza Hut, Wendy's) ______ No ______ Yes
   IF "YES," how many servings did you eat last week? ______ servings

7. Cured or processed meats? (examples - bacon, hotdogs, lunchmeat meats) ______ No ______ Yes
   IF "YES," how many servings did you eat last week? ______ servings
APPENDIX G continued

8. smoked meats? (examples - ham, ham hocks) ____ No ____ Yes
   If "YES," how many servings did you eat last week? ______ servings

9. whole grain breads or cereals? (examples - wheat bread, bran flakes) ____ No ____ Yes
   If "YES," how many servings did you eat last week? ______ servings

10. fried foods? (examples - french fries, fried chicken, potato chips) ____ No ____ Yes
    If "YES," how many servings did you eat last week? ______ servings

11. Do you drink alcoholic beverages now? (examples - beer, wine, whiskey, mixed drinks) ____ No ____ Yes
    If "YES," how many drinks did you have last week? ______ cans/bottles/glasses/drinks
    If you drink only occasionally, how many drinks do you usually have each month?
    ______ cans/bottles/glasses/drinks

    If "NO," did you quit drinking alcoholic beverages in the last year? ____ No ____ Yes
    If you have quit, when did you quit? ______ approximate date

SMOKING HISTORY

12. Do you smoke? ____ No ____ Yes
    If "YES," how many cigarettes do you smoke per day? ______ cigarettes per day
    If "NO," have you stopped in the last year? ____ No ____ Yes
    If you have quit, when did you quit? ______ approximate date

13. Do you use smokeless tobacco regularly? ____ No ____ Yes
    If "YES," about how much chewing tobacco or snuff per ______ day ______ week
    If "NO," have you stopped in the last year? ____ No ____ Yes
    If you have quit, when did you quit? ______ approximate date
MEDICAL HISTORY

14. What is your current weight? __________ pounds

15. Have you ever had a mammogram? ________________  No  Yes
   IF "YES," when was your last one? __________________
   Were you ever told you had an abnormal one?  No  Yes

16. Have you ever had a pap smear? ________________  No  Yes
   IF "YES," when was your last one? __________________
   Were you ever told you had an abnormal one?  No  Yes
APPENDIX G continued

Physical Measurements Form

1. Date of birth _______ (Month - day - year)
2. Age _______ years old

MEDICAL HISTORY

3. Current weight _______ pounds
4. Height _______ inches
5. Percent body fat _______ %

Please list any health education materials distributed.

________________________________________

________________________________________

Comments:
________________________________________

________________________________________

Materials Given:
The Pap Test: It Can Save Your Life __________
Facts About Cervical Cancer __________

Do The Right Thing: Get A Mammogram __________
Check Your Breasts __________
Myths About Mammography __________

You Can Quit Smoking __________
Don't Smoke Around Your Children __________
Stop Smoking Now: The Benefits are Immediate __________
Can They Stop Smoking? __________

Cancer Facts For Women __________
APPENDIX H

BLOOD LIPID ANALYSIS TECHNIQUES

Stanbio Enzymatic Cholesterol
Procedure No. 1010
Quantitative-Enzymatic-Clinicochemical
Determination of Total and HDL Cholesterol in Serum or Plasma

Summary and Principle

The enzymatic method for cholesterol determination was introduced in 1977 by Nagamine and Tomita, using cholesteryl esters of cholesterol esters. Following chromatographic separation of the cholesterol esters, Nagamine modified the technique and added in 1979 adding the first (and enamine esters, the method presented in this section in essence follows the Nagamine method presented in the reference provided and satisfies all three criteria to be combined with the Unlock enzyme-hydroxyapatite system of Trendler.2

Cholesterol esters (CE) hydrolyzed to free cholesterol and fatty acids. The free cholesterol as produced plus the free cholesterol esters are then hydrolyzed in the presence of a cholesteryl esterase-salicylate reaction mixture to form free cholesterol which is quantitated enzymatically with an oxidase reagent in the presence of a coenzyme, 7-acetoxy flavine. The assay is a two-phase and similar to the cholesterol assay procedure presented in this appendix.

Cholesterol Standards: Stanbio Cholesterol + Test Kits

Formula:

\[ \text{Cholesterol} + \text{CoO} \rightarrow \text{Cholesterol Oxi-Dose} + \text{H}_2\text{O} + \text{CO}_2 + \text{ATP} + \text{ADP} \]

A technique for high-density lipoprotein (HDL) cholesterol utilizing Stanbio HDL-Purification Reagent, Cat. No. 2559, used by present examiner.

Reagents:

Phosphate Cholesterol Extraction Reagent, Cat. No. 2511
Reagent contains the following active ingredients at stated concentrations:
- 0.015 mg/mL sphingomyelin
- 0.030 mg/mL cholesterol
- 0.010 mg/mL cholic acid
- 0.005 mg/mL ATP
- 0.001 mg/mL ADP
- 0.001 mg/mL sodium fluoride
- 0.001 mg/mL EDTA
- 0.001 mg/mL sodium pyrophosphate

Phosphate Cholesterol Standard Kit, Cat. No. 251202
Reagent contains the following active ingredients at stated concentrations:
- 0.050 mg/mL cholesterol
- 0.010 mg/mL sphingomyelin
- 0.005 mg/mL cholic acid
- 0.002 mg/mL ATP
- 0.001 mg/mL ADP
- 0.001 mg/mL sodium fluoride
- 0.001 mg/mL sodium pyrophosphate

Specimen Collection and Preparation

Patient should avoid high fat diet for 24 hours prior to venipuncture. Patient should avoid strenuous exercise for 24 hours prior to venipuncture. Patient should avoid smoking prior to venipuncture. Specimen should be collected in a sterile heparinized tube. Allow specimen to clot for 1 hour. Centrifuge at 3000 rpm for 10 minutes. Discard supernatant. Collect plasma in a 5-mL glass vial and store at 4°C.

Protocol:

1. Collect plasma in a 5-mL glass vial and store at 4°C.
2. Centrifuge at 3000 rpm for 10 minutes.
3. Discard supernatant.
4. Collect plasma in a 5-mL glass vial and store at 4°C.

Expected Values:

Total Cholesterol

- Men: 140-220 mg/dL
- Women: 120-200 mg/dL

HDL Cholesterol

- Men: 35-65 mg/dL
- Women: 45-75 mg/dL

Free Cholesterol

- Men: 25-45 mg/dL
- Women: 20-40 mg/dL

Triglycerides

- Men: 25-200 mg/dL
- Women: 20-100 mg/dL

Sample Precautions:

1. Samples should be stored at 4°C.
2. Samples should be kept at 4°C and analyzed within 24 hours.
3. Samples should be analyzed within 24 hours.

Sample Stability:

Samples should be stored at 4°C and analyzed within 24 hours.

Sample Weight:

Samples should be stored at 4°C and analyzed within 24 hours.

Sample Volume:

Samples should be stored at 4°C and analyzed within 24 hours.

Sample Temperature:

Samples should be stored at 4°C and analyzed within 24 hours.

Sample pH:

Samples should be stored at 4°C and analyzed within 24 hours.

Sample Sugar:

Samples should be stored at 4°C and analyzed within 24 hours.

Sample Alcohol:

Samples should be stored at 4°C and analyzed within 24 hours.

Sample Fatty Acids:

Samples should be stored at 4°C and analyzed within 24 hours.

Sample Amino Acids:

Samples should be stored at 4°C and analyzed within 24 hours.

Sample Carbohydrates:

Samples should be stored at 4°C and analyzed within 24 hours.

Sample Enzymes:

Samples should be stored at 4°C and analyzed within 24 hours.

Sample Hormones:

Samples should be stored at 4°C and analyzed within 24 hours.

Sample Vitamins:

Samples should be stored at 4°C and analyzed within 24 hours.

Sample Trace Elements:

Samples should be stored at 4°C and analyzed within 24 hours.

Sample Electrolytes:

Samples should be stored at 4°C and analyzed within 24 hours.

Sample Oxygen:

Samples should be stored at 4°C and analyzed within 24 hours.
APPENDIX H continued

Stanbio

HDL-Cholesterol

Procedure No. 0599

For use in the Determination of High-Density Lipoprotein (HDL) Cholesterol in Plasma or Serum

Summary and Principle

The densitometric (LDG) cholesterol and very low density lipoprotein (VLDL) cholesterol are determined by the successive precipitation of their respective lipoprotein fractions, according to the procedure of Orientation Laboratory (OL) cholesterol in the same medium, using a modified technique known as the Lowenstein technique.

The precipitating reagent is designed to be used in Stanbio's Cholesterol Precipitation Reagent, which is a mixture of a polyacrylamide resin and a detergent. The precipitating reagent is added to the sample, and the mixture is centrifuged. The resulting supernatant is then analyzed for cholesterol.

Equipment

- HDL-Precipitating Reagent, Cat. No. 0569
- Lowenstein Cholesterol Precipitation Reagent, Cat. No. 0570
- Test tubes
- Centrifuge
- 85°C water bath

Reagents

- HDL-Precipitating Reagent
- Lowenstein Cholesterol Precipitation Reagent
- 85°C water bath

Procedure

1. Add 1 ml of HDL-Precipitating Reagent to 1 ml of sample. Incubate at 85°C for 1 hour, and then centrifuge at 1500 rpm for 5 minutes. The supernatant is then discarded.

2. Add 1 ml of Lowenstein Cholesterol Precipitation Reagent to the precipitate. Incubate at room temperature for 1 hour, then centrifuge at 1500 rpm for 5 minutes. The supernatant is then discarded.

3. Repeat steps 1 and 2 until the supernatant is clear.

4. The final supernatant is then analyzed for cholesterol.

Results

The results are expressed as the percentage of total cholesterol.

HDL Separation Procedure

1. To 4 ml of serum, add 4 ml of 0.8% sodium deoxycholate, and mix well.

2. Centrifuge at 10,000 rpm for 30 minutes.

3. The supernatant is then analyzed for cholesterol.

4. The final supernatant is then analyzed for cholesterol.

HDL Analysis

1. Add 1 ml of HDL-Precipitating Reagent to 1 ml of sample.

2. Centrifuge at 10,000 rpm for 30 minutes.

3. The supernatant is then analyzed for cholesterol.

4. The final supernatant is then analyzed for cholesterol.

Stable Constituents

- Reagents
- Procedures

References

- Stanbio Laboratories, Inc.
- Lowenstein Cholesterol Precipitation Reagent
- 85°C water bath
- Test tubes

STANBIO
APPENDIX H continued

Stanbio Enzymatic Triglycerides
Procedure No. 2000
For the Quantitative Enzymatic Colorimetric Determination of
Triglycerides in Serum or Plasma

Summary and Principle
Measurement of triglyceride levels is performed in conjunction with
other laboratory tests to provide information on composition of
serum lipoproteins. Triglyceride concentration is the sum of triglyceride
esters known to be present in serum. This procedure employs triacylglycerol
hydrolysis enzyme to convert triglyceride esters to free fatty acids.

Materials Required But Not/Provided

1. Serum or plasma samples
2. Hydrochloric acid (HCl) solution
3. Sodium hydroxide (NaOH) solution
4. distilled water

Procedure

1. To each sample tube, add 0.5 ml of sample, 0.5 ml of 0.1 M HCl, and 1.0 ml of 0.1 M NaOH.
2. Incubate the samples at 50°C for 30 minutes.
3. Add 0.5 ml of 0.1 M NaOH to each tube and mix.
4. Add 0.5 ml of 0.1 M HCl to each tube and mix.
5. Read the absorbance at 510 nm.

Quality Control

1. Prepare a standard curve using known concentrations of triglycerides.
2. Perform the test as described above.
3. Calculate the triglyceride concentration in the sample.

Results

1. The results are expressed in milligrams per deciliter (mg/dL) or millimoles per liter (mmol/L).

Expected Values

- Normal: 50 to 150 mg/dL
- High: 150 to 200 mg/dL
- Very High: > 200 mg/dL

References

5. American Heart Association.}

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VITA

The author of this thesis was born in the city of Manila, Philippines, on 5 December 1962; the first child of Amado Gonzales and Ma. Stella Velasco. She obtained her primary and secondary education from Canossa School of Sta. Rosa, Laguna.

In 1979, she entered the University of Sto. Tomas to pursue a degree in medicine. She graduated in April 1983 with the degree of Bachelor of Science in Biology, and then obtained the degree of Doctor of Medicine in May 1987. She took the Philippine Medical licensure examination in August 1988 and became a licensed physician in January 1989.

In October 1988, she joined the Rural Health Physician Program by the Department of Health. As a volunteer, she was involved in community-based nutrition programs, as well as rendering curative and preventive services to local residents for six months. In August 1989, she became the Rural Health Physician of District IV, National Capital Region, Department of Health. She worked for 11 months as the head of a medical team that provided health services in towns and barrios without health facilities.

In August 1990, she left for the United States and entered the Virginia Polytechnic Institute and State University, where she is currently pursuing her master’s degree in Human Nutrition and Foods. She was married in January 25, 1992 and currently has one daughter.